

EMERGENCE AND TRANSBOUNDARY SPREAD OF LUMPY SKIN DISEASE IN SOUTH ASIAN COUNTRIES: A REVIEW

Syed Ehtasham Amin¹, Naveed Ul Haq², Shahzad Haider³, Malik Faizan Shaukat⁴, Aiman Arif¹, Arslan Fareed¹, Arslan Khan¹, Abid Mehmood³, Qammer Shahzad¹

1. Department of Food Science and Technology, The University of Haripur, Pakistan
2. Department of Food Science, The University of Guelph, Canada
3. Department of Agronomy, The University of Haripur, Pakistan
4. Department of Horticulture, The University of Haripur, Pakistan

ABSTRACT

In livestock Transboundary pox viral disease known as Lumpy skin disease (LSD) is OIE notifiable. It is severely increasing and affecting the livestock economics. There hasn't been much research and reporting on the LSD virus's zoonotic potential. After initially appearing in Zambia, the virus spread to many parts of the world in around 90 years. In eastern, southern and southeastern countries the estimated impact of LSD is 1.45 billion due to direct losses and production. The same disease was observed for the first time in 2019, from countries such as Bangladesh, Nepal, China, India Pakistan and next year in 2020 it was reported in Bhutan, Srilanka, Bangladesh Southeast China and Vietnam. In 2021, LSD expanded to other countries such as Malaysia, Cambodia and Thailand. Typically, LSD-affected cattle have a nodular lesion or skin mass that covers their entire body. Occasionally, systemic symptoms are also present. The major and most frequent way of mechanical transmission is believed to be hematophagous arthropods; nevertheless, additional transmission routes connected to the illicit animal trade have contributed to the rise of LSD in nations that were previously available from it. The OIE recommends viral neutralization as the industry-standard gold test for serological diagnostics. The OIE recommends viral neutralization as the industry-standard gold test for serological diagnostics. Virus isolation and its sequencing requires in LSD free countries. It is possible to control LSD by applying rapid control measures for viral infection such as vaccination. Specific vaccines for the control of LSD are suitable for the protection of castles and buffalo as compared to other heterologous vaccines. Lack of a specific LSD policy at the time the disease first appeared, a high density of susceptible, unvaccinated populations, ignorance among farmers and veterinarians, and existing laws prohibiting the slaughter of cattle all contributed to favourable conditions for the disease's spread to numerous states in nations like India. Recently, whole world is affected by the COVID- 19 and now LSD is further affecting the economies of the

countries. So it should be reviewed to save the economy of the developing countries in Southeast Asia.

Introduction:

Lumpy skin disease (LSD) is a poxviral illness of cattle that must be reported to the Office International des Epizooties (OIE). Knopvelsiekte, pseudourticaria, exanthema nodularis bovis, and Neethling viral illness are further names for LSD (Abutarbush 2017, CFSPH 2008, MacDonald 1931). Most farmers in South Asia's emerging tropical nations fall into the marginal and tiny category and raise livestock as an extra, sustainable source of income. For the health and prosperity of the farmer milk and dung fuel are obtained from the livestock. The cattle industry is essential for reducing poverty, boosting resilience, and battling hunger and food insecurity (Enahoro et al. 2019). Due to productivity losses, draught power loss, lower feed intake, illness management, trade restrictions, and long-term convalescence, livestock owners in South Asian nations have been more concerned about the LSD virus.

Animals with LSD have a distinctive nodular lesion or skin lump throughout their entire body, which is occasionally accompanied by systemic symptoms (Gupta et al. 2020). The virus initially appeared in Zambia, Africa, in 1929 and spread quickly, in a period of around 90 years, to many different parts of the world (MacDonald 1931). It has spread rather quickly to new nations free of this viral disease. In 1988, first case was reported in the Middle East from the Egypt and then from Bahrain in 2005, and to the Middle East countries it remained restricted till 2018 (OIE 2021, Stram et al. 2008). In 2019 it was reported in the South Asian countries such as Bangladesh, Nepal, China and India (Hasib et al. 2021, OIE 2021). First reported case in India was from Odisha and then other states of the country was affected (EFSA et al. 2020). India, Bhutan, Bangladesh, Vietnam and Southeast China reported the cases of LSD in 2020 (Acharya and Subedi 2020, Roche et al. 2020, Tran et al. 2021). LSD continued to spread in 2021 and first cases was detected in Malaysia, Cambodia and Thailand (OIE 2021). Cattle are thought to be the animal most vulnerable to the LSD virus, and India has the largest global inventory of them. Since the LSD virus remained absent in this area until 2019, there was no government LSD control strategy or contingency in place. India has special rules that prohibit the killing of cattle, stakeholders are ignorant of this illness, and there is no LSD immunisation programme. With this context, it was necessary to study this condition more thoroughly in terms of its recent onset and potential treatments.

LSD Virus:

The LSD virus is a member of the family Poxviridae, in the genus Capripox, subfamily

Chordopoxvirinae (poxviruses of vertebrates). The LSD virus is an enclosed double-stranded DNA virus with a genomic size of around 151 kilobase pairs (Kbps) and a putative 156-gene core coding area (Tulman et al. 2001). LSD virus causes catastrophic illness in sheep and goats, respectively, and shares antigenic similarities with sheeppox virus (SPPV) and goatpox virus (GTPV) (Abutarbush and Tuppurainen, 2018). All three species of Capripoxviruses showed 98% sequence similarity according to genomic research (CaPVs) (Gershon 1988, Tulman et al. 2002). Genomic similarity offers the chance to prevent this illness when the LSD vaccine is not approved for use by using GTPV and SPPV vaccines as prophylactic.

Both experimentally and spontaneously, sheep and goats can become infected with the SPPV and GTPV strains, respectively. Contrarily, the LSD virus can only be experimentally transmitted to sheep and goats, demonstrating that it is host-specific and constrained (El-Kenawy and El-Tholoth 2010). LSD virus is stable for the longer periods and can persist in contaminated animal shelters, particularly when there is no sunlight present. Similar to dry scabs, necrotic skin nodules, and desiccated crusts, the LSD virus has been shown to remain steadily for up to a month or longer at room temperature. Ether (20%), chloroform, formalin (1%), phenol (2% for 15 min), sodium hypochlorite (2-3%), iodine compounds (1:33 dilution), and quaternary ammonium compounds (0.5%) were all effective chemical control agents against the virus. The virus, however, was astonishingly stable and lasted longer at room temperature (OIE 2013). **Disease outbreaks in South**

Asian nations:

including India and Pakistan. Currently, LSD is prevalent in the majority of African nations, a few Middle Eastern nations, and Turkey. Recently, an assessment of the LSD outbreak timeframe and its dissemination (Fig. 1) was conducted (Kayesh et al. 2020). In Zambia, LSD was discovered for the first time in 1929. (MacDonald 1931). Following that, Kenya reported the SPPV outbreak and LSD frequency at a farm (Burdin 1959). Israel recorded the LSD epidemic outside of Africa in 1989 (Zeynalova et al. 2016), reaching Egypt, which is regarded as a nation connecting northeast Africa with the Middle East, where the illness was initially detected in 1988 (House et al. 1990). LSD outbreaks often peak in the summer and autumn, when vectors are at their best for mating, and then decline in the winter (EFSA et al. 2020).

Nevertheless, reports from Azerbaijan indicating an epidemic in June, July, October, and November of 2015 (OIE 2013). LSD may start as an epidemic in a fresh location during the hot and humid time of the year, according to recent appearances from India in August 2019 and unconfirmed cases reported during most months from various areas of India, in July to December from

Bangladesh, and in June to September in Nepal. But after that, regardless of the season, it spreads. A likely transboundary spread of diseases was suggested by the timing of illness outbreaks in China, Bangladesh, India, and by recent reports from Nepal, Bhutan, and Malaysia (Burdin 1959, Acharya and Subedi 2020, EFSA. et al. 2020, Roche et al. 2020). The unauthorised movement of animals for trade and trafficking or the transmission of the illness by vectors from outbreak regions may also be contributing factors to its development. All parties believe that irregular cross-border trafficking of livestock from India's neighbouring states, such as Bihar, to Nepal may be responsible for the LSD outbreak there (Acharya and Subedi 2020). However, there hasn't been any formal documentation of the Bihar LSD outbreak to far. Odisha and Jharkhand have identified recent occurrences in India (Kumar et al. 2021a, Sudhakar et al. 2020). In addition, several unconfirmed LSD cases from 14 Indian states have been suspected (Vora and Kulkarni 2020, Kumar et al. 2021a). LSD generally has a low death rate (10%) and a high morbidity rate (2-45%). LSD generally has a low death rate (10%) and a high morbidity rate (2-45%) (Tuppurainen et al. 2017a). LSD morbidity in Odisha was found to be 7.1%, with no deaths (Sudhakar et al. 2020). In two separate epidemics, the LSD outbreak in China reported 6.6-100% morbidity and 0-16.7% fatality (Lu et al. 2020). Similar to this, LSD mortality in Bangladesh varied from 1.0 to 2.0%, with a morbidity of 0.01 to 8.26%. (Kayesh et al. 2020).

Susceptible host:

Initially, the cattle are the initial hosts for the LSD virus (Tuppurainen et al. 2015). The LSD virus has a higher host specificity, which prevents it from causing clinical illness in domesticated animals including sheep, goats, pigs, and horses. However, other domesticated animals like yaks and water buffalo may also be impacted (USDA 2016). According to legend, Asian water buffaloes (*Bubalus bubalis*) have a relatively low vulnerability to LSD, but few clinical cases have been reported (Neamat-Allah and Mahmoud 2019). Regarding age and sex, there was no association seen in the incidence of LSD in cattle. Nevertheless, variations are found in breed type. The more susceptible are the exotic cattle as compared to native cattle and buffaloes (Kiplagat et al. 2020). Malnourished animals, breastfeeding cows, and young calves (early age group) tend to naturally get more severe illness (Carn 1995, Mulatu and Feyisa 2018). It could result from compromised humoral immunity. African buffalo from Kenya (*Synerus caffer*) may also serve as reservoir hosts. Although infected buffaloes did not exhibit any LSD symptoms, the virus's antibody titre was found (Davies 1991, Gibbs 2013).

Several animal species, including an Arabian oryx (*Oryx leucoryx*) and springbok (*Antidorcas marsupialis*), as well as experimental infections in impalas (*Aepyceros melampus*) and giraffes, have been documented to acquire clinical LSD (*Giraffa camelopardalis*) (Tuppurainen et al. 2018) and Thomson's gazelle (*Eudorcas thomsonii*) (Davies 1991). In addition, LSD antibodies were detected in springbok, eland (*Taurotragus oryx*), African buffalo (*Syncerus caffer*), black wildebeest (*Connochaetes gnou*), and blue wildebeest (*Connochaetes taurinus*) in South Africa. Due to limited access to the wild population for clinical examination, diagnosis, and monitoring, the potential contribution of wildlife to disease epidemiology is yet unclear.

OIE has not yet reported any zoonotic potential for the LSD virus. However, Kamal observed rare and anthroponotic transmission of the LSD virus to people after the massive epidemic of LSD in cattle in Cairo and Egypt in 2018-2019 (Kamal 2019). According to the paper, the LSD virus may likely infect humans by inhalation and through direct contact with insects, sick people, or a workplace hazard. Although the symptoms in humans do not mirror an abscess on limbs in cattle, they are comparable to the development of skin nodules and can occasionally be fatal. Herpesvirus infection in both humans and cattle is linked to LSD virus infection (Kamal 2019). Human herpesvirus infection may be a protective factor against poxvirus illness.

Disease transmission:

Transmission of illness LSD is a transnational illness. The discovery of the LSD virus in India and surrounding nations, where this disease was previously nonexistent, emphasises the significance of understanding its method of transmission. Human herpesvirus infection may be a protective factor against poxvirus illness. propagation of disease A transnational sickness is LSD. A host infected with the LSD virus-like poxvirus can spread it directly and indirectly. Epidemiology of the LSD virus and potential routes of transmission have been documented by Carn and Kitching (Carn 1995), and Sprygin and colleagues have eloquently analysed these findings (Sprygin et al. 2019).

Climate warming and the current COVID-19 epidemic have caused extraordinary changes in biodiversity and ecological dynamics. Such transition has helped the vectors and the related developing illnesses to flare up. Although experimental evidence of disease transmission is limited, hematophagous arthropod-borne mechanical transmission is thought to be the primary and frequent mechanism for LSD infection (Sohier et al. 2019, Weiss 1968). While *Rhipicephalus appendiculatus* and *Amblyomma hebraeum* spread the virus mechanically, Ixodid ticks (*Rhipicephalus decoloratus*) can transmit this virus by transstadial and transovarial pathways (Lubinga et al. 2014, Tuppurainen

et al. 2011). The same cow may get infected with LSD by vector-borne transmission, which can be compounded by the presence of other hemoparasitic diseases. Tick-transmitted hemoparasitic infection affects the cattle herd in India (Kumari et al. 2019, Roy 2021) and is sometimes mixed infected (Kumar et al. 2021b). Recent studies show experimental proof of mechanical LSD virus transmission in bulls caused by *Stomoxys calcitrans* and *Haematopota* spp. (Sohier et al. 2019). The propagation of the LSD virus from Egypt to Israel despite the total ban on animal transportation suggests the possibility of airborne transmission via the associated vectors (CFSPH 2008). This virus may spread via infected vectors across distances of up to 300 kilometres (Australia 2009). There have also been reports of the LSD virus being transferred intrauterinally (Rouby and Aboulsoud 2016). Pregnant cows infected with LSD have had calves with skin lesions. The source of transmission may include the diseased animal's fluids, including blood, saliva, semen, and nasal and lachrymal secretions.

Similarly the mucous membranes of the eyes, nose, mouth, rectum, udder, and genitalia can also develop ulcerated LSD virus nodules, which are a significant source of transmission (Babiuk et al. 2008). Bulls with subclinical infection can ejaculate virus in semen for at least 12 days while bulls with clinical indications of LSD infection can ejaculate virus in semen for up to 22 days (Weiss 1968). Possible biosecurity threats include LSD sex-to-seminal transmission and artificial insemination (Annandale et al. 2014). It has been shown that viruses may be transmitted intravenously and intradermally (Carn 1995). Another method of spreading LSD is through the potential for iatrogenic intra- or inter-herd transmission using infected needles during vaccinations or other injections caused by sharing needles between animals or herds (Tuppurainen et al. 2017b). There has been speculation regarding the function of migratory and wild birds in mechanical transmission, but no proof has been found. Co-infections caused by vectors.

Vector associated co-infections:

The major method of LSD transmission is thought to be mechanical transfer by the vectors of the LSD virus. However, it might also lead to the spread of additional pathogens that are connected to these vectors. The clinical state and the disease's ultimate outcome may be complicated by an infection. The main vectors for the spread of hemoparasitic infections are ticks and flies. Particularly in tropical and subtropical regions, this might lead to the likelihood of co-infection with the LSD virus and hemoparasites. There have been reports of mixed blood parasite infections (babesiosis, theileriosis, and anaplasmosis) in Iraqi cows infected with the LSD virus (Jameel 2016). There is limited research and data available on the coinfection of other diseases with vector-associated

transmission. To shed light on the potential connection between the clinical presentation of LSD and the immunocompromised state of hosts coinfecting with hemoparasites, more study is needed. The disease's progression, the case fatality rate, and productivity losses might all be prolonged by infection.

Parthenogenesis and clinical findings:

Pathophysiology and medical findings During host feeding, arthropod vectors inject the LSD virus into the animal's skin. The vulnerable host's bloodstream is then infected by the virus. Hyperplasia and ballooning degeneration of the epithelium are caused by the LSD virus's keratinocyte-specific tropism (Coetzer 2004). For regulatory purposes, the OIE Terrestrial Animal Health Code specifies a maximum incubation time of 28 days. However, in experiments, the virus's incubation time is just 5 days (Woods 1990), and the incubation time of this virus in a spontaneous infection ranges from 4 to 28 days (Barnard et al. 1994). All age groups of susceptible animals are susceptible to infection, and immunocompromised animals and young age groups are likely to have cases.

High temperature (103–106°F) is the first clinical symptom postincubation that is seen in cattle. Fever is often present for 1-3 days, however it can last longer if additional tick-transmitted diseases are present. Anorexia, lacrimation, nasal discharge, decreased milk production, and indifference to the surroundings are all possible signs of the febrile period. These symptoms are the result of several tissues in infected animals being inflamed by viremia. (El-Mandrawy and Alam 2018). It coincides with or follows a skin nodule's spontaneous emergence. Skin nodules up to 5 cm in diameter may begin as localised forms on the legs, udder, perineum, head, and neck, or they may become widespread and enclose the entire body. These elevated, hard, rounded, and constrictive skin nodules affect the skin, subcutaneous tissue, and occasionally even the underlying muscles (OIE 2013). Both intravenous and intradermal experimental inoculation of LSD virus suspension in calves led to the development of a firm, well-circumscribed, raised cutaneous nodule that was 4–8 cm in diameter by 7 days after inoculation and moderately to noticeably enlarged prescapular lymph nodes at 5 days after inoculation (SanzBernardo et al. 2020). A febrile reaction to an experimental infection was seen in calves. 7-9 days after vaccination. Large nodules on the skin of the infected animal may turn necrotic (sitfast) and subsequently fibrotic in 2–3 weeks. It's possible for these fibrotic signs to last for several months or fade with time (OIE 2019). The oropharynx and nares of the muzzle may develop typical ring-like lesions that enlarge the local lymph nodes (Davies 1991). Some infected cattle have been reported to experience myiasis and mastitis as LSD-related consequences (Al Salihi

and Hassan 2015).b There have been cases of abortions accompanied by LSD skin lesions in the acute phase of LSD virus infection in pregnant cattle (Brenner et al. 2006). Bulls and cows with the infection may become sterile permanently or temporarily (Sohier et al. 2019 , Tuppurainen and Oura 2012).. Pneumonia may attack due to lung inflammation and nasal discharge. Large LSD lesions and limb edoema may result in lameness symptoms in the animal. Cattle with LSD infection may exhibit distinctive clinical indications, which are highly helpful in raising the possibility of the illness. However, due to their low sensitivity to the LSD virus, LSD-infected water buffaloes could not exhibit any clinical signs (Mulatu and Feyisa 2018). Even there are no clinical symptoms in the susceptible cattle infected by LSD.

Histopathological analysis of infections caused by the LSD virus in both natural and artificial settings has revealed that it can produce significant vascular alterations in skin lesions, including vasculitis (Prozesky and Barnard 1982, Tageldin et al. 2014, Sanz-Bernardo et al. 2020). Only the CaPV family of poxviruses has undergone these alterations; other poxviruses have not. The mucous membranes of the mouth, abomasum, trachea, and lungs may have vesicles, erosions, or ulcers. During necropsies on 33 deceased Azerbaijani cattle, nodules and lung congestion were seen throughout the internal organs (Zeynalova et al. 2016).

Diagnosis:

Diagnosis Characteristic skin lesions and related clinical indicators can be used to a significant degree to support the probable diagnosis of LSD. Clinical-based diagnosis, however, is limited in the case of mild and asymptomatic illness, requiring laboratory procedures for confirmation. In addition to viral isolation, confirmation calls for molecular and serological testing. LSD must also be confirmed in order to be distinguished from other illnesses with comparable clinical symptoms, such as pseudolumpy skin disease, bovine papular stomatitis, pseudopox, foot-and-mouth disease demodiosis, tick bites, insect bites, photosensitization, urticaria, and other dermal disorders (Gupta et al. 2020, OIE 2013).

Although the procedure is labor- and time-intensive, the OIE recommends viral neutralisation as the gold standard among serological diagnostic techniques (Kreiae et al. 2020). Madin-Darby bovine kidney (MDBK) cells were used in a modified viral neutralisation test that was published by Kresic and colleagues (Krei et al. 2020). They were effective for identifying antibodies that specifically neutralised the LSD virus and showed a good correlation with the outcomes of

commercial ELISA. Serological tests are suggested as practical techniques to look at current outbreaks. The isolation of viruses is possible from blood, scab, skin nodules, and biopsy skin tissues (Kumar et al. 2021a) For the diagnosis of LSD to be confirmed, virus isolation is necessary. Although the diagnostic test is sensitive and trustworthy, it takes a while to get the findings (Tuppurainen et al. 2005).

The virus might be identified for a longer length of time using molecular techniques based on PCR and quantitative real-time approaches, which have been reported as being quicker and more sensitive (Tuppurainen et al. 2005, Balinsky et al. 2008, Kumar et al. 2021a). Due of the extremely conserved genomic sequence of the capripox virus, detection by PCR is based on primers tailored to comparable sequences found in sheep pox and LSD viruses (Kara et al. 2003, Tuppurainen et al. 2005). PCR has been shown to identify viral nucleic acids 53 days after virus isolation in skin lesions (Tuppurainen et al. 2005). The phenetic link between the LSD virus and other isolates and CaPVs was determined using phylogenetic analysis. However, this association analysis needs the PCR amplification result to be sequenced (Ochwo et al. 2020, Kumar et al. 2021a). The most resemblance to Kenyan LSD virus strains was found, according to a published study on phylogenetic studies of circulating Indian LSD virus strains from the states of Odisha and Jharkhand (Kumar et al. 2021a, Sudhakar et al. 2020).

Nearly all CaPVs may be classified into groups based on the origins of their hosts, according to nucleic acid sequencing (Le Goff et al. 2009). The G-protein-coupled chemokine receptor (GPCR), ankyrin repeat (ANK), RNA polymerase subunit (RPO30), and envelope protein p32 are among the LSD virus genes targeted for PCR amplification (Ireland, 1998, Kumar et al. 2021a, Mafirakureva et al. 2017, Salnikov et al. 2018, Stram et al. 2008, Sudhakar et al. 2020).

Treatment:

Treatment The LSD virus cannot be prevented or treated specifically. The majority of the care given to the infected animals is supportive, with the goal of lessening the severity of the virus's pathogenesis and any related secondary consequences (Al-Salihi 2014). Based on reports of hemoparasite coinfection, the use of supportive therapy and antiparasitic medications is indicated (Jameel 2016). The goal of supportive therapy is to increase appetite by lowering temperature, discomfort, and inflammation (Capstick et al. 1959). In order to avoid additional bacterial problems, it has been reported that anti-inflammatory, antipyretic, and antibiotic medications are used (Woods 1988, Abdulqa et al. 2016). Skin wound-related myiasis, mastitis, pneumonia, lameness, ocular opacity, and coinfection with hemoparasitic illnesses are frequent complications of LSD that need

veterinary care (Salib and Osman 2011). Bulls infected with LSD responded favourably to a combination treatment consisting of dexamethasone (0.2 mg/kg/day) for three consecutive days and 10% oxytetracycline (10 mg/kg/day) for five consecutive days (Feyisa 2018, Biswas et al. 2020). A survey investigation of LSD diagnosis and treatment for each afflicted animal costs USD 5 in Ethiopia (Molla et al. 2017).

Economic impact of LSD:

Economic effects of LSD-related diseases, Due to its widespread spread, LSD has significant economic and cattle production ramifications. Depending on the farmer or the regional or national government organisations, there are direct and indirect losses. Reduced milk production, abortions, stunted body growth, death, concealed injury, etc. are examples of direct losses to farmers. Farmers may suffer indirect losses owing to missed opportunities, lower lifetime output of sick animals, treatment costs, and additional management burdens. The government's direct losses from vaccination and trade restriction control measures, vector control, disease surveillance programs, awareness programs, etc. a ban on commerce, measures to prevent insects that spread disease, campaigns to raise awareness, etc. Numerous factors including LSD epidemiology, LSD virus pathogenesis, breed of cattle, commerce in livestock, and control efforts have been linked to output losses and death (Gari et al. 2011, Molla et al. 2017, Casal et al. 2018, Kiplagat et al. 2020). It has been reported that in nations where attenuated homologous LSD vaccines are used for mass immunisation, milk production can decrease by up to 6-8 kg/week seven days after vaccination (Morgenstern and Klement 2020). Nevertheless, it had little to no impact on milk production during the month following immunisation. According to an estimate from Ethiopia by Molla et al. (2017), death (USD) accounts for the largest portion of economic loss at the herd level. According to Kiplagat et al. (2020), LSD caused economic and production losses in Ethiopia, with variations proportional to the herd size and local vs exotic sources of replacement cattle. They calculated that on farms raising indigenous breeds, the mean farm-level losses were disproportionately larger owing to milk output (97 USD) than death (31 USD). This result was at odds with Molla et al. (2017). In farms with exotic breed cattle, the estimate for indirect losses for treatments and vaccines was higher than in farms with native cattle (Kiplagat et al. 2020). According to Gari et al. (2011), the mean financial cost in Ethiopian herds with sick cattle was greater for Holstein-Friesian/crossbred cattle (about USD 58) than for local breeds (6.43/head). The reports and estimations of losses in Balkan nations. In Albania, Bulgaria, and the Former Yugoslav Republic of Macedonia, the price per animal in the impacted herds was USD 648.51, 176.87, and 310.42, respectively (Casal et al. 2018).

Due to its debut in 2019, research on the economic impact and production losses caused by lumpy skin disease in Bangladesh, China, and India have not been done. The Food and Agriculture Organization (FAO) has published a document that details the economic effects of LSD on nations in the south, east, and southeast.

An estimated 1.45 billion US dollars might be lost directly in livestock and agriculture (Roche et al. 2020). LSD's legalisation in 2019 might have a significant impact on the cattle trade in Asian nations. According to a 2017 estimate, exports of live cattle, buffalo meat, meat products, dairy products, and skins totaled USD 5.5 billion to Asian countries (Roche et al. 2020). APEDA (Agricultural and Processed Food Products Export Development Authority) statistics shows that India alone exported 3,694.29 USD million of which 3175.09 USD million was buffalo meat (APEDA 2021). In general, elements taken into account in the study and country's disease management policies are what cause the estimate's fluctuation.

Prevention and control:

The prevention measures can be taken by immunization, hygienic measures, vector control, and limits on the movement of infected animals. Due to special rules forbidding the killing of cattle, the stamping-out strategy for the management and control of animal illness is not adhered to in nations like India. Additionally, none of these nations have a preventative immunization programme employing recommended vaccinations. Adopting appropriate hygienic procedures, isolating affected animals, restricting their movement and trade, providing insect-proofed quarantine facilities, avoiding communal grazing, disease surveillance, and vector control programmes should be the exclusive focus of the management programme. The entire cost of the control campaign may be greatly impacted by the various national policies regarding the killing and destruction of afflicted animals (Casal et al. 2018). Because there is little more than a week between infection and viremia—during which there is essentially no method to detect infected animals—movement limitations in the LSD control programme are only partially effective.

The only effective and controllable way to reduce LSD in endemic areas and nations with little resources is vaccination. It lessens the financial burden that LSD has on farmers by preventing the clinical signs of the presenting disease and further preventing additional diseases from prevailing. In nations where both viruses are present, SPPV- or GTPV-based immunizations are used to prevent LSD; otherwise, the vaccine may function as a source of a fresh illness. According to reports, immunisation lowers LSD-related expenses by 31%/head for crossbred or Holstein-Friesian herds

and by 17%/head for local zebu herds (Mulatu and Feyisa 2018). The LSD virus Neethling strain, the Kenyan SPVV and GTPV (KSGPV) O-240 and O-180 strains, the Yugoslavian RM65 sheeppox (SPP) strain, the Romanian SPP strain, and the Gorgan goatpox (GTP) strains are among the CPV vaccine strains that are commercially available (Abutarbush, 2017). Table 1 lists the nations from where different vaccine strains used for the prevention and control of LSD have failed. It has been observed that LSD-infected cattle received a 10-fold higher dosage of the sheep- and goatpox vaccination.

However, according to two separate findings, the GTPV (Gorgan strain and G20- LKV) vaccination strain induces a strong protective response and offers 100% protection against LSD in cattle (Gari et al. 2011, Zhugunissof et al. 2020). The majority of phylogenetic analyses revealed that the SPPV and LSD viruses are more distantly related than the goatpox virus (Le Goff et al. 2009, Lamien et al. 2011).

It is also possible to use the goatpox and sheeppox vaccines to stop the disease from spreading to vulnerable animals. In order to protect cattle from the LSD virus, crossprotection within the CaPV genus and SPPV vaccinations have been widely employed (Tuppurainen et al. 2014). According to Kitching, every CaPV strain has a similar antigen, and overcoming an infection with one strain confers protection against all others. Consequently, it is feasible to protect cattle, sheep, and goats with a single vaccine strain (Kitching 2003). These SPPV and GTPV vaccinations, according to a recent study, are ineffective against the LSD virus. The use of homologous strains against LSD over the Romanian SPPV vaccine and/or a combination of SPPV and GTPV was emphasised by Mikhael et al. (2017). The latter vaccinations did not offer enough protection, and there was no evidence of a serological response to LSD. Hamdi et al(2020) .'s most recent research additionally demonstrated that the Romanian SPPV vaccination gave only partial cross-protection to cattle against LSD, while the LSD virus protects cattle against LSD, which suggests that vaccination against LSD virus should be carried out with the homologous strain.

A commercially available LSD vaccine that utilises field viral isolates is called LumpyVax® (MSD Animal Health-Intervet, South Africa). It is a freeze-dried live attenuated virus (SIS Neethling-type) vaccine. The most often used vaccines in the field are live ones, and it is well recognised that using them correctly in target species results in strong immunity. It is safe to administer 1 ml of the indicated dose subcutaneously to cattle of all ages and physiological states. The other 2 commercial vaccines are made by Onderstepoort Biological Products and are Bovivax LSDN® (freeze-dried), MCI Santè Animale, Morocco, and OBP, South Africa (Lumpy Skin Disease

vaccination for Cattle), all of which include cell-adapted strains of the original LSD virus Neethling strain (Morgenstern and Klement 2020). Both of these vaccinations should be administered subcutaneously at a dosage of 2 ml per animal. Calves should receive their first dosage at the age of six months and a booster dose every year after that. According to reports, applying pesticides topically to diseased cattle does not appear to help prevent the spread of illness (Davies 1991).

Additionally, the use of practical and affordable vector control will lessen the effects, impede the disease's future spread into new regions, and lower the cost of the vector control programme. According to a questionnaire-based assessment conducted in Ethiopia, LSD control expenditures were the smallest cause of herd-level losses (Molla et al. 2017, Kiplagat et al. 2020). According to Molla et al. (2017)'s financial study, the LSD vaccination project resulted in a net profit of USD 136 (USD 56 for subsistence farm herds and USD 283 for commercial herds) per herd. According to a recent study conducted at 77 dairy farms in Israel, there are very few negative impacts of this vaccine on productivity indices (Morgenstern and Klement 2020).

Restricted farm visits and awareness campaigns on the spread of the LSD virus directed at people who work with cattle populations directly or indirectly, such as farmers, veterinarians, truck drivers, etc., will aid in the early notification, detection, and prompt response of the authorities for this deadly disease. Due to religious restrictions, infected livestock are present in India and serve as a source of infection.

The key to stopping the spread of this illness will be active surveillance. Israel and Southeastern Europe were among the nations where LSD use was claimed as having the ability to completely eradicate sickness. By strictly killing all diseased and in-contact animals and implementing a ring vaccination programme with the SPVV vaccine, Israel may eradicate LSD (Stram et al. 2008). However, due to a voluntary vaccination campaign against LSD and the spread of viruses in the area, LSD reappeared in Israel in 2019. (EFSA et al. 2020). In Southeast Europe, efforts to prevent invasion were concentrated more on mass immunisation with the LSD homologous vaccine than other methods (EFSA et al. 2020).

Conclusion:

Livestock services are being affected by the present pandemic to some extent; climate change favours the spread of vectors in many fresh places. LSD is a serious developing illness that is anticipated to spread continuously due to all of these variables. There is now a demand for research on this quickly growing virus in developing nations like India. An extra effort should be made to comprehend the function of the vectors that are present among.

Table.1 Vaccination failure with different CaPV strains reported in various countries

Vaccine strain	Remarks	Reference
KS1 O-180 virus strain vaccine	23.8% morbidity in the cattle population in Ethiopia after vaccination.	(Ayelet <i>et al.</i> 2013)
Heterologous vaccine	Jordon (LSD morbidity of 4.7% in cattle vaccinated against it)	(Abutarbush 2014)
Heterologous vaccine	Israel (vaccinated 11% cattle became infected)	(Brenner <i>et al.</i> 2009)
SPPV Bakirkoy strain	Vaccination failure	(Sevik <i>et al.</i> 2016)
Kenyan 67 sheep and goat pox vaccine	Continuous LSD outbreak for > three months in a vaccinated cattle herd in Oman.	(Ayelet <i>et al.</i> 2013)
Romania vaccine	Cases of infected cattle emerging from a vaccinated herd in Egypt	(Abdallah <i>et al.</i> 2018, Zeedan <i>et al.</i> 2019)

DECLARATION OF INTEREST

We declare that this manuscript is original, has not been published before and is not currently being considered for publication elsewhere. The authors certified that there are no conflicts of interest associated with this publication, and there has been no significant financial support for publishing this work that could have influenced its outcome. As corresponding Author, I conform that the manuscript has been read and approved for submission by all the named authors.

REFERENCES

- Abdallah F M, El Damaty H M and Kotb G F. 2018. Sporadic cases of lumpy skin disease among cattle in Sharkia province, Egypt: Genetic characterization of lumpy skin disease virus isolates and pathological findings. *Veterinary World* 11: 1150– 58.
- Abdulqa H Y, Rahman H S, Dyary H O and Othman H H. 2016. Lumpy skin disease. *Reproductive Immunology: Open Access* 1: 25.

Abutarbush S M and Tuppurainen E S. 2018. Serological and clinical evaluation of the Yugoslavian RM 65 sheep pox strain vaccine use in cattle against lumpy skin disease. *Transboundary and Emerging Diseases* 65: 1657–63.

Acharya K P and Subedi D. 2020. First outbreak of lumpy skin disease in Nepal. *Transboundary and Emerging Diseases* 67: 2280–81.

Al-Salihi K A. 2014. Lumpy skin disease: Review of literature. *Mirror of Research in Veterinary Sciences and Animals* 3:6– 23.

Al Salihi K A and Hassan I Q. 2015. Lumpy skin disease in Iraq: study of the disease emergence. *Transboundary and Emerging Diseases* 62: 457–62.

Annandale C H, Holm D E, Ebersohn K, Venter E H. 2014. Seminal transmission of lumpy skin disease virus in heifers. *Transboundary and Emerging Diseases* 61: 443–48. Anonymous. 2020. Lumpy skin disease reported in 3 districts of Kerala. *The Hindu Sect. Section|:Start Page| (col. Column)|. ANIMAL PRODUCTS*. Available from http://apeda.gov.in/apedawebsite/six_head_product/animal.htm

Australia A H. 2009. Disease strategy: Lumpy skin disease (Version 3.0). Australian Veterinary Emergency Plan (AUSVETPLAN). In. 3 ed.

Ayelet G, Abate Y, Sisay T, Nigussie H, Gelaye E, Jemberie S and Asmare K. 2013. Lumpy skin disease: preliminary vaccine efficacy assessment and overview on outbreak impact in dairy cattle at Debre Zeit, central Ethiopia. *Antiviral Research* 98: 261–65.

Babiuk S, Bowden T R, Boyle D B, Wallace D B and Kitching R P. 2008. Capripoxviruses: An emerging worldwide threat to sheep, goats and cattle. *Transboundary and Emerging Diseases* 55: 263–72.

Balinsky C A, Delhon G, Smoliga G, Prarat M, French R A, Geary S J, Rock D L and Rodriguez L L. 2008. Rapid preclinical detection of sheep pox virus by a real-time PCR assay.

Journal of Clinical Microbiology 46: 438–42.

Barnard B J H, Munz E, Dumbell K and Prozesky L. 1994. Lumpy Skin Disease, Oxford: Oxford University Press.

Ben-Gera J, Klement E, Khinich E, Stram Y and Shpigel N Y. 2015. Comparison of the efficacy of Neethling lumpy skin disease virus and x10RM65 sheep-pox live attenuated vaccines for the prevention of lumpy skin disease—The results of a randomized controlled field study. *Vaccine* 33: 4837–42.

Biswas D, Saha S S and Sayeed S B M. 2020. Outbreak of lumpy skin disease of cattle in south-west part of Bangladesh and its clinical management. *Veterinary Sciences: Research and Reviews* 6: 100–108.

Brenner J, Bellaiche M, Gross E, Elad D, Oved Z, Haimovitz M, Wasserman A, Friedgut O, Stram Y, Bumbarov V Y and Yadin H. 2009. Appearance of skin lesions in cattle populations vaccinated against lumpy skin disease: statutory challenge. *Vaccine* 27: 1500–03.

Brenner J, Haimovitz M, Oren E, Stram Y, Fridgut O, Bumbarov V, Kuznetzova L, Oved Z, Wasserman A and Garazzi S. 2006. Lumpy skin disease (LSD) in a large dairy herd in Israel, June 2006. *Israel Journal of Veterinary Medicine* 61: 73.

Burdin M. 1959. Lumpy skin disease of cattle in Kenya. *Nature* 183: 949–950.

Capstick P B, Prydie J, Coackley W and Burdin M L. 1959. Protection of cattle against the “Neethling” type virus of lumpy skin disease. *Veterinary Record* 71: 422.

Carn VMaK R. P. 1995. An investigation of possible routes of transmission of lumpy skin disease virus (Neethling). *Epidemiology and Infection* 114: 219–26.

Casal J, Allepuz A, Miteva A, Pite L, Tabakovsky B, Terzievski D, Alexandrov T and Beltrán-

Alcrudo D. 2018. Economic cost of lumpy skin disease outbreaks in three Balkan countries: Albania, Bulgaria and the Former Yugoslav Republic of Macedonia (2016–2017). *Transboundary and Emerging Diseases* 65: 1680–88.

Lumpy Skin Disease. Technical Factsheet. Available from

http://www.cfsph.iastate.edu/Factsheets/pdfs/lumpy_skin_disease.pdf. Coetzer JAW.

2004. *Lumpy Skin Disease*. 2nd Edition ed. Cape Town, South Africa: Oxford University Press.

Davies F G. 1991. Lumpy skin disease of cattle: a growing problem in Africa and the Near East. *World Animal Review* 68: 37–42.

EFSA, Calistri P, De Clercq K, Gubbins S, Klement E, Stegeman A, Cortiñas Abrahantes J, Marojevic D, Antoniou S E and Broglia A. 2020. Lumpy skin disease epidemiological report IV: data collection and analysis. *EFSA Journal* 18: 6010.

El-Kenawy A A and El-Tholoth M S. 2010. Sequence analysis of attachment gene of lumpy skin disease and sheep poxviruses. *Virologica Sinica* 25: 409–16.

El-Mandrawy S A and Alam R T. 2018. Hematological, biochemical and oxidative stress studies of lumpy skin disease virus infection in cattle. *Journal of Applied Animal Research* 46:1073–77.

Elhaig M M, Selim A and Mahmoud M. 2017. Lumpy skin disease in cattle: Frequency of occurrence in a dairy farm and a preliminary assessment of its possible impact on Egyptian buffaloes. *Onderstepoort Journal of Veterinary Research* 84: 1–6.

Enahoro D, Mason-D’Croz D, Mul M, Rich KM, Robinson T P, Thornton P and Staal S S. 2019. Supporting sustainable expansion of livestock production in South Asia and Sub Saharan Africa: Scenario analysis of investment options. *Global Food Security* 20: 114–121.

Feyisa A F. 2018. A case report on clinical management of lumpy skin disease in bull. *Journal of*

Veterinary Science and Technology 9: 538.

Gari G, Abie G, Gizaw D, Wubete A, Kidane M, Asgedom H, Bayissa B, Ayelet G, Oura C A, Roger F and Tuppurainen ESM. 2015. Evaluation of the safety, immunogenicity and efficacy of three capripoxvirus vaccine strains against lumpy skin disease virus. *Vaccine* 33: 3256–3261.

Gari G, Bonnet P, Roger F and Waret-Szkuta A. 2011. Epidemiological aspects and financial impact of lumpy skin disease in Ethiopia. *Preventive Veterinary Medicine* 102: 274– 83.

Gershon P D and Black D N. 1988. A comparison of the genomes of capripoxvirus isolates of sheep, goats, and cattle. *Virology* 164: 341–49.

Gibbs P. 2013. Pox Diseases: Lumpy Skin Disease. Available from:

<http://www.merckvetmanual.com/mvm/index.html>. Gupta T, Patial V, Bali D, Angaria S, Sharma M and Chahota R. 2020. A review: Lumpy skin disease and its emergence in India. *Veterinary Research Communication* 44: 111–118.

Hamdi J, Bamouh Z, Jazouli M, Boumart Z, Tadlaoui K O, Fihri O F and Harrak M E. 2020. Experimental evaluation of the cross-protection between Sheeppox and bovine Lumpy skin vaccines. *Scientific Report* 10: 1–9.

Hasib F M Y, Islam M S, Das T, Rana E A, Uddin M H, Bayzid M, Nath C, Hossain M A, Masuduzzaman M, Das S and Alim M A. 2021. Lumpy skin disease outbreak in cattle population of Chattogram, Bangladesh. *Veterinary Medicine and Science*.

House J A, Wilson T M, Nakashly S E, Karim I A, Ismail I, Danaf N E, Moussa A M and Ayoub N N. 1990. The isolation of lumpy skin disease virus and bovine herpes virus-from cattle in Egypt. *Journal of Veterinary Diagnostic Investigation* 2: 111–115.

Ireland DCaB Y S. 1998. Improved detection of capripoxvirus in biopsy samples by PCR. *Journal of Virological Methods* 74: 1–7.

Jameel G H. 2016. Determination of complications decrease the risk factor in Cattle infected by lumpy skin disease virus in diyala province, Iraq. *International Journal of Micro Biology, Genetics and Monocular Biology Research* 2:1–9.

Kamal S A. 2019. Comparative studies on lumpy skin disease virus in human. *Medical and Clinical Archives* 3: 1–8. Kara P D, Afonso C L, Wallace D B, Kutish G F, Abolnik C, Lu Z, Vreede F T, Taljaard L C F, Zsak A, Viljoen G J et al. 2003.

Comparative sequence analysis of the South African vaccine strain and two virulent field isolates of lumpy skin disease virus. *Archives of Virology* 148: 1335–56.

Kayesh M E H, Hussan M T, Hashem M A, Eliyas M and Anower A M. 2020. Lumpy skin disease virus infection: An emerging threat to cattle health in Bangladesh. *Hosts and Viruses* 7: 97– 108.

Kiplagat S K, Kitale P M, Onono J O, Beard P M and Lyons N A. 2020. Risk factors for outbreaks of lumpy skin disease and the economic impact in cattle farms of Nakuru county, Kenya. *Frontiers in Veterinary Science* 7: 259.

Kitching R P. 2003. Vaccines for lumpy skin disease, sheep pox and goat pox. *Developments in Biologicals (Basel)* 114: 161– 67.

Krešić N, Šimić I, Bedeković T, Acinger-Rogić • and Lojkić I.2020. Evaluation of serological tests for detection of antibodies against lumpy skin disease virus. *Journal of Clinical Microbiology* 58: e00348–00320.

Kumar N, Chander Y, Kumar R, Khandelwal N, Riyesh T, Chaudhary K, Shanmugasundaram K, Kumar S, Kumar A, Gupta M K, Pal Y, Barua S and Tripathi B N. 2021. Isolation and characterization of lumpy skin disease virus from cattle in India. *PLoS One* 16: e0241022.

Kumar P, Kumar P, Roy R K, Kumari R R, Kumar A, Sarma K, Sharma P and Kumar M. 2021. Mixed infection of tick-borne haemo-parasites in water buffalo and associated pathological responses and treatment. *Indian Journal of Animal Research*.

Kumari R R, Kumar R, Kumar P and Kumar M. 2019. Emergence and variations in disease ecology of tick-borne bovine theileriosis in East India. *International Journal of Livestock Research* 9: 12–25.

Lamien C E, Le Goff C, Silber R, Wallace D B, Gulyaz V, Tuppurainen E, Madani H, Caufour P, Adam T, El Harrak M, et al. 2011. Use of the capripoxvirus homologue of vaccinia virus 30 kDa RNA polymerase subunit (RPO30) gene as a novel diagnostic and genotyping target: development of a classical PCR method to differentiate goat poxvirus from sheep poxvirus. *Veterinary Microbiology* 149: 30–39.

Le Goff C, Lamien C E, Fakhfakh E, Chadeyras A, Aba-Adulugba E, Libeau G, Tuppurainen E, Wallace D B, Adam T, Silber R, Glyaz V, Madani H, Caufour P, Hammami S, Diallo A and Albina E. 2009. Capripoxvirus Gprotein-coupled chemokine receptor: a host-range gene suitable for virus animal origin discrimination. *Journal of General Virology* 90: 1967–77.

Lu G, Xie J, Luo J, Shao R, Jia K and Li S. 2020. Lumpy skin disease outbreaks in China, since 3 August 2019. *Transboundary and Emerging Diseases* 68: 977–980.

Lubinga J C, Clift S J, Tuppurainen E S, Stoltz W H, Babiuk S, Coetzer J A and Venter E H. 2014. Demonstration of lumpy skin disease virus infection in *Amblyomma hebraeum* and *Rhipicephalus appendiculatus* ticks using immunohisto chemistry. *Ticks and Tick-borne Diseases* 5: 113–20. MacDonald RAS. 1931. Pseudo-Urticaria of cattle. pp. 20–21.

Mafirakureva P, Saidi B and Mbanga J. 2017. Incidence and molecular characterisation of lumpy skin disease virus in Zimbabwe using the P32 gene. *Tropical Animal Health and Production* 49: 47–54.

- Mikhael C A, Nakhla O E and Mohamed N A. 2017. Study on the capability of a dual capripox vaccine in protection of cattle against LSD infection. *Journal Of Veterinary Medical Research* 24: 224–33.
- Molla W, de Jong M C, Gari G and Frankena K. 2017. Economic impact of lumpy skin disease and cost effectiveness of vaccination for the control of outbreaks in Ethiopia. *Preventive Veterinary Medicine* 147: 100–07.
- Morgenstern M and Klement E. 2020. The Effect of Vaccination with Live Attenuated Neethling Lumpy Skin Disease Vaccine on Milk Production and Mortality—An Analysis of 77 Dairy Farms in Israel. *Vaccines (Basel)* 8: 324.
- Mulatu E and Feyisa A. 2018. Review: Lumpy skin disease. *Journal of Veterinary Science and Technology* 9: 1–8.
- Neamat-Allah A N F and Mahmoud E A. 2019. Assessing the possible causes of hemolytic anemia associated with lumpy skin disease naturally infected buffaloes. *Comparative Clinical Pathology* 28: 747–53.
- Ochwo S, VanderWaal K, Ndekezi C, Nkamwesiga J, Munsey A, Witto S G, Nantima N, Mayanja F, Okurut A R A, Atuhaire D K and Mwiine F N. 2020. Molecular detection and phylogenetic analysis of lumpy skin disease virus from outbreaks in Uganda 2017–2018. *BMC Veterinary Research*.16:1–10.
- OIE. 2013. Lumpy Skin Disease. Aetiology, Epidemiology, Diagnosis, Prevention and Control References. In: www.oie.int. pp. 1–5.
- OIE. 2019. Infection with Lumpy Skin Disease Virus, In: *Terrestrial Animal Health Code*. In: Paris: www.oie.int. Database Name 2021. World Organisation for Animal Health. Available from: <https://wahis.oie.int/#/dashboards/country-or-disease-dashboard>.
- Prozesky L and Barnard B J. 1982. A study of the pathology of lumpy skin disease in cattle.

Onderstepoort Journal of Veterinary Research 49: 167–75.

Roche X, Rozstalnyy A, TagoPacheco D, Pittiglio C, Kamata A, Beltran Alcrudo D, Bisht K, Karki S, Kayamori J, Larfaoui F, Raizman E, VonDobschuetz, Dhingra M S and Sumption K. 2020. Introduction and spread of lumpy skin disease in South, East and Southeast Asia: Qualitative risk assessment and management. Rome: FAO.

Rouby S and Aboulsoud E. 2016. Evidence of intrauterine transmission of lumpy skin disease virus. *Veterinary Journal* 209: 193–95.

Roy S, Bhandari V, Barman M, Kumar P, Bhanot V, Arora J S, Singh S and Sharma P. 2021. Population genetic analysis of the *Theileria annulata* parasites identified limited diversity and multiplicity of infection in the vaccine from India. *Frontiers in Microbiology* 11: 3471.

Salib F A and Osman A H. 2011. Incidence of lumpy skin disease among Egyptian cattle in Giza Governorate, Egypt. *Veterinary World* 4: 162–67.

Salnikov N, Kolcov T U A, Morgunov S Z Y, Gogin V G A and Yurkov I T S. 2018. Identification and characterization of lumpy skin disease virus isolated from cattle in the Republic of North Ossetia-Alania in 2015. *Transboundary and Emerging Diseases* 65: 916–20.

Sanz-Bernardo B, Haga I R, Wijesiriwardana N, Hawes P C, Simpson J, Morrison L R, MacIntyre N, Brocchi E, Atkinson J, Haegeman A, De Clercq K, Darpel K E and Beard P M. 2020. Lumpy skin disease is characterized by severe multifocal dermatitis with necrotizing fibrinoid vasculitis following experimental infection. *Veterinary Pathology* 57: 388–96.

Sevik M, Avci O, M D and Ince Ö B. 2016. Serum biochemistry of lumpy skin disease virus-infected cattle. *BioMed Research International* 2016: 6257984.

- Sohier C, Haegeman A, Mostin L, De Leeuw I, Van Campe W, De Vleeschauwer A, Tuppurainen E S M, van den Berg T, De Regge N, De Clercq K. 2019. Experimental evidence of mechanical lumpy skin disease virus transmission by *Stomoxys calcitrans* biting flies and *Haematopota* spp. horseflies. *Scientific Report* 9: 1–10.
- Sprygin A, Pestova Y, Wallace D B, Tuppurainen E, Kononov A V. 2019. Transmission of lumpy skin disease virus: a short review. *Virus Research* 269: 197637.
- Stram Y, Kuznetzova L, Friedgut O, Gelman B, Yadin H, Rubinstein-Guini M. 2008. The use of lumpy skin disease virus genome termini for detection and phylogenetic analysis. *Journal of Virological Methods* 15: 225–29.
- Sudhakar S B, Mishra N, Kalaiyarasu S, Jhade S K, Hemadri D, Sood R, Bal G C, Nayak M K, Pradhan S K and Singh V P. 2020. Lumpy skin disease (LSD) outbreaks in cattle in Odisha state, India in August 2019: Epidemiological features and molecular studies. *Transboundary and Emerging Diseases* 67: 408–22.
- Tageldin M H, Wallace D B, Gerdes G H, Putterill J F, Greyling R R, Phosiwa M N, Al Busaidy R M, Al Ismaaily S I. 2014. Lumpy skin disease of cattle: An emerging problem in the Sultanate of Oman. *Tropical Animal Health and Production* 46: 241–246.
- Tran H T T, Truong A D, Dang A K, Ly D V, Nguyen C T, Chu N T, Hoang T V, Nguyen H T, Nguyen V T and Dang H V. 2021. Lumpy skin disease outbreaks in Vietnam, 2020. *Transboundary and Emerging Diseases* 68: 977–80.
- Tulman E, Afonso C, Lu Z, Zsak L, Kutish G, Rock D. 2001. Genome of LSDV *Journal of Virology* 75: 7122–30.
- Tulman E R, Afonso C L, Lu Z, Zsak L, Sur J H, Sandybaev N T, Kerembekova U Z, Zaitsev V L, Kutish G F and Rock D L. 2002. The genomes of sheeppox and goatpox viruses. *Journal of Virology* 76: 6054–61.

Tuppurainen E, Venter E H, Shisler J L, Gari G, Mekonnen G A, Juleff N, Lyons N A, De Clercq K, Upton C, Bowden T R et al. 2017b. Review: Capripoxvirus diseases: Current status and opportunities for control. *Transboundary and Emerging Diseases* 64:729–45.

Tuppurainen E S, Alexandrov T, Beltrán-Alcrudo D. 2017a. Lumpy skin disease-A manual for veterinarians. *FAO Animal Production and Health Manual*.

Tuppurainen E S, Babiuk S and Klement E. 2018. *Lumpy Skin Disease*. Springer, Cham. Tuppurainen E S and Oura CAL. 2012. Review: lumpy skin disease: An emerging threat to Europe, the Middle East and Asia. *Transboundary and Emerging Diseases* 59: 40–48.

Tuppurainen E S, Pearson CR, Bachanek-Bankowska K, Knowles N J, Amareen S, Frost L, Henstock M R, Lamien C E, Diallo A, Mertens P P. 2014. Characterization of sheep pox virus vaccine for cattle against lumpy skin disease virus. *Antiviral Research* 109: 1–6.

Tuppurainen E S, Stoltz W H, Troskie M, Wallace D B, Oura C A L, Mellor P S, Coetzer J A and Venter E H. 2011. A potential role for ixodid (hard) tick vectors in the transmission of lumpy skin disease virus in cattle. *Transboundary and Emerging Diseases* 58: 93–104.

Tuppurainen E S, Venter E H, Coetzer J A and Bell-Sakyi L. 2015. Lumpy skin disease: Attempted propagation in tick cell lines and presence of viral DNA in field ticks collected from naturally-infected cattle. *Ticks and Tick-borne Diseases* 6: 134–40.

Tuppurainen E S, Venter E H and Coetzer J A W. 2005. The detection of lumpy skin disease virus in samples of experimentally infected cattle using different diagnostic techniques. *Onderstepoort Journal of Veterinary Research* 72: 153–164.

USDA. 2016. Lumpy skin disease standard operating procedures: 1, pp. 1–10. Overview of Etiology and Ecology. October 2016 ed. Maryland.

Vora R and Kulkarni V. 2020. Lumpy skin disease becomes worst nightmare for farmers in a dozen States. Business Line Sect. Section|:Start Page| (col. Column)|. Weiss K E. 1968. Lumpy skin disease virus. Cytomegaloviruses. Rinderpest Virus. Lumpy Skin Disease Virus JBHPE Weiss editor Berlin, Heidelberg: Springer. Woods J A. 1988. Lumpy skin disease—a review. Tropical Animal Health and Production 20: 11–17.

Woods J A. 1990. Lumpy skin disease. Virus Infections of Ruminants Amsterdam: Elsevier Science Publishers.

Zeedan G S G, Mahmoud A H, Abdalhamed A M, Abd El KAEH. 2019. Detection of lumpy skin disease virus in cattle using real-time polymerase chain reaction and serological diagnostic assays in different governorates in Egypt in 2017. Veterinary World 12: 1093.

Zeynalova S, Asadov K, Guliyev F, Vatani M AND Aliyev V. 2016. Epizootology and Molecular Diagnosis of Lumpy Skin Disease among Livestock in Azerbaijan. Frontiers in Microbiology 7.

Zhugunissov K, Bulatov Y, Orynbayev M, Kutumbetov L, Abduraimov Y, Shayakhmetov Y, Taranov D, Amanova Z, Mambetaliev M, Absatova Z and Azanbekova M. 2020. Goatpox virus (G20-LKV) vaccine strain elicits a protective response in cattle against lumpy skin disease at challenge with lumpy skin disease virulent field strain in a comparative study. Veterinary Microbiology 245: 108695.