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Review on Epidemiology and Economic Importance of Lumpy Skin Disease

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Abstract: Lumpy skin disease (LSD) is a generalized skin disease which is an infectious, eruptive, occasionally fatal disease of cattle caused by a virus associated with the Neethling poxvirus in the genus Capri poxvirus of the family Poxviridae. LSD was first described in Zambia and occurs in other most African countries and sporadically in the Middle East region. Mechanical vector insects might play a major role in the epidemiology of LSD and wildlife plays a potential role in its maintenance. The disease causes serious economic losses in most African countries including Ethiopia due to the prolonged loss of productivity from high morbidity, restrictions to the global trade of live animals and animal products, costly control and eradication measures. Factors associated with communal grazing and watering point and introduction of new cattle are significantly high risk of LSD occurrence. The diagnostic tests currently available like the clinical examinations and laboratory tests (Virus isolation / identification and Serological tests) are needed to confirm it. LSD needs to stop if occurred and to block if new either by sanitary (import restrictions on livestock, carcasses, hides, skins and semen) or medical prophylaxis (Vaccination).

Key words: LSD · LSDV · Cattle · Epidemiology · Risk Factors · Economic Impact · Transmission

INTRODUCTION

Livestock production constitutes one of the principal means of achieving improved living standards in many regions of the developing world [1].The livestock sector globally is highly dynamic, contributes 40% of the global value of agricultural output and support the livelihoods and food security of almost a billion people [2]. In many developing countries(In Sub-Saharan African countries), livestock keeping is a multifunctional activity and plays a crucial role both in national economies and the livelihood of rural communities. Beyond their direct role in generating food and income, livestock are a valuable asset, serving as a store of wealth, provides drought power, clothing, transport and serve as a source of manure for energy and soil fertility, collateral for credit and an essential safety net during times of crisis [1, 3, 4].

In Ethiopia Livestock production is an integral part of the agricultural system. The livestock sub sector accounts for 40% of the agricultural gross domestic product (GDP) and 20% of the total GDP without considering other contribution like traction power, fertilizing and mean of transport [5, 6]. The livestock sector is now has significant contribution to the total foreigner currency of the country.

In the future, livestock production will increasingly be affected by competition for natural resources, particularly land and water, competition between food and feed and by the need to operate in a carbon-constrained economy [7]. Currently the overall livestock production constraints in Ethiopia are feed shortages, livestock diseases, low genetic potential of indigenous livestock and lack of marketing infrastructure and water shortages [8, 9]. Among the many other diseases, which

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are known in causing economic losses and of poor productivity in livestock specifically in cattle is the presence of lumpy skin disease in many parts of the country [10-12].

Lumpy skin disease (LSD) is a generalized skin disease which is an infectious, eruptive, occasionally fatal disease of cattle caused by a virus associated with the Neethling poxvirus in the genus Capri poxvirus of the family Poxviridae [13- 16].The economic losses due to this disease is associated with decreased milk production, traction power loss, weight loss, poor growth, abortion, infertility and skin damage. Pneumonia is a common sequel in animals with lesions in the mouth and respiratory tract [17-22].

LSD was first observed in the western part of Ethiopia (Southwest of Lake Tana) in 1983 [23]. It has now spread to almost all the regions and agro ecological zones [10, 24]. Some epidemiological studies have been carried out since the disease has become established in the country, with the diverse agro-ecological and production systems [10].

Study based on seroprevalence in southern Ethiopia reported a prevalence of 6% [25]. Targeted sampling from outbreak areas around Southern Range land, Wolliso town and north Ethiopia reported prevalence's of 11.6%, 27.9% and 28%, respectively [25-27]. A recently prevalence study [12] results showed higher herd prevalence recorded in Afar (51%) and Tigray (37%) regions.

Published information on the factors that influence the occurrence of LSD are not many as general, however some studies indicated that LSD is a disease which affect all age group, in Africa imported *Bos Taurus* appear to be more susceptible than the indigenous breeds [17]. The LDSV was found to be associated with Capri poxvirus outbreaks in Kenya [28].

A clinical case of LSD has been reported in other animals: Asian water buffalo from Egypt [29]. Antibodies have been demonstrated in black and blue wild beasts, Elan, Giraffe, greater Kudu and others [30, 31]. Some researchers have made attempt the transmission of the disease with different flies [13, 32-34]. Recently, Tuppurainen *et al*. [35] reported the potential role of ixodic tick in the transmission of LSDV. Weather changes such as cold may adversely affect the insect vector and infected saliva may contribute to the spread of the disease [36].

However, there is a gap in epidemiological disease information (Taransboundry diseases) particularly lumpy skin disease in West Wollega zone except few outbreak reports from the area. The study area interfaces with the pastoralists often crossing the border to other African courtiers (Sudan and South Sudan) and Benishangul Gumuz Regional State of Ethiopia. Thus, the objective of this paper is to assess the risk factors that contribute to the occurrence of lumpy skin disease in the study area.

Lumpy Skin Disease: The range of viral skin disease in animals is very wide and involves many agents who also have significant systemic effects [24, 37, 38].Of these Viral Skin Diseases, Lumpy Skin Disease is the most serious, infectious, eruptive and occasionally fatal viral skin disease and other parts of the body of cattle caused by a virus of the family *Poxviridae* and economically, a significant cattle disease(production losses). It is therefore defined as a notifiable disease by the World Organization for Animal Health (OIE) [10, 17, 22, 39-42].

It is an endemic in parts of Africa and has the potential to become established in other parts of the world [43]. It is characterized by disseminated appearance of skin lesions, 2-5 cm in diameter and lymphadenopathy, accompanied by high fever which can sometimes exceed 41°C and may last up to 2 weeks [42].

Historical Background of the Disease: Historically the disease was confined to South Africa, it has moved north into Kenya, Sudan and Ethiopia [44, 45]. According to Haig [46], the clinical syndrome of lumpy skin disease (LSD) was first described in Zambia in 1929. Initially, it was considered to be the result either of poisoning or a hypersensitivity to insect bites. Between 1943 and 1945, cases occurred in Botswana, Zimbabwe and the Republic of South Africa [38, 47, 48].

In 1970 LSD spread north into the Sudan, by 1974 it had spread west as far as Nigeria and in 1977 was reported from Mauritania, Mali, Ghana and Liberia [49]. Another epidemic of LSD between 1981 and 1986 affected Tanzania, Kenya, Zimbabwe, Somalia and the Cameroon, with reported [17,48] Lumpy skin disease (LSD) is confined and enzootic in all Sub-Saharan African countries in which it has occurred and has proved impossible to eradicate and Middle East with recent incursion into Israel [50,51].

From 1929 to 1986, the disease was restricted to countries in sub-Saharan Africa, although its potential to extend beyond this range had been suggested [17]. In 1988, LSD was recognized clinically in the Suez Governorate of Egypt, where it was thought to have arrived at the local quarantine station with cattle imported from other countries of Africa [43]. In 1989, a focus of LSD was identified in Israel and subsequently eliminated by the slaughter of all infected cattle as well as contacts. Ring vaccination with a sheep pox strain was carried out

around the focus area and no further clinical cases have occurred [17, 43]. It reappeared in the summer of 1989 and, in a period of five to six months, spread to 22 of the 26 governorates of Egypt.

A rapid reaction to the problem led to the vaccination of nearly two million cattle with a sheep pox vaccine [47]. Lumpy skin disease outbreaks tend to be sporadic, depending upon animal movements, immune status and wind and rainfall patterns affecting vector populations and as a result of this, the most recent outbreaks outside Africa occurred in the Middle East in 2006 and 2007 and in Mauritius in 2008 [22]. In summer of 2006, the most recent outbreak of LSD was recorded in several Egyptian governorates [52].

As mentioned by Mebratu *et al*. [23]. Lumpy Skin Disease was rst observed in the Western part of Ethiopia (Southwest of Lake Tana) in 1983. According to Gari *et al.* [10], a major epidemic outbreak of LSD occurred in different regions of in different years Ethiopia like Amhara and W/ Oromiya Regions in 2000/2001, Oromiya and SNNP regions in 2003/2004 and Tigray, Amhara and Benishangul regions in 2006/2007.

Causative Agent of the Disease: Lumpy Skin Disease (LSD) is one of a serious poxvirus disease of cattle caused by Lumpy Skin Disease Virus (LSDV), a DNA virus of the genus Capri poxvirus and of the family *Poxviridae*. The prototype strain is Neethling virus [10, 22, 24, 42].

It is closely related to Sheep pox virus (SPPV) and Goat pox virus (GTPV) [41]. However, although all three viruses are considered distinct species, they cannot be differentiated serologically [42, 53, 54].Therefore, the only molecular techniques to distinguish LSD from SPPV and GTPV have been developed.

Epidemiology

Geographic Distribution: LSD occurs in most African countries (Including Madagascar) and sporadically in the Middle East region. Recent outbreaks of LSD in Egypt, Israel (2006 and 2007), Oman and Bahrain (2009) raise the possibility that LSDV might become established in the Middle East and spread to Asia and Europe [55, 56].

In addition, the occurrence of the disease in some districts (Adola and Yabello districts) in the years 2003-2005 was reported [10]. Recently one thousand five hundred herds of cattle have died of suspected lumpy skin disease in Zambia (Mazabuka's Nega Nega area) [57].

Sources of the Virus: LSDV is present in cutaneous lesions and crusts. Virus is also present in blood, nasal and lachrymal secretions, milk, semen and saliva, which may be sources for transmission [17, 58].

All secretions contain LSD virus when nodules on the mucous membranes of the eyes, nose, mouth, rectum, udder and genitalia ulcerate. Shedding in semen may be prolonged since viral DNA has been found in the semen of some bulls for at least 5 months after infection [59].

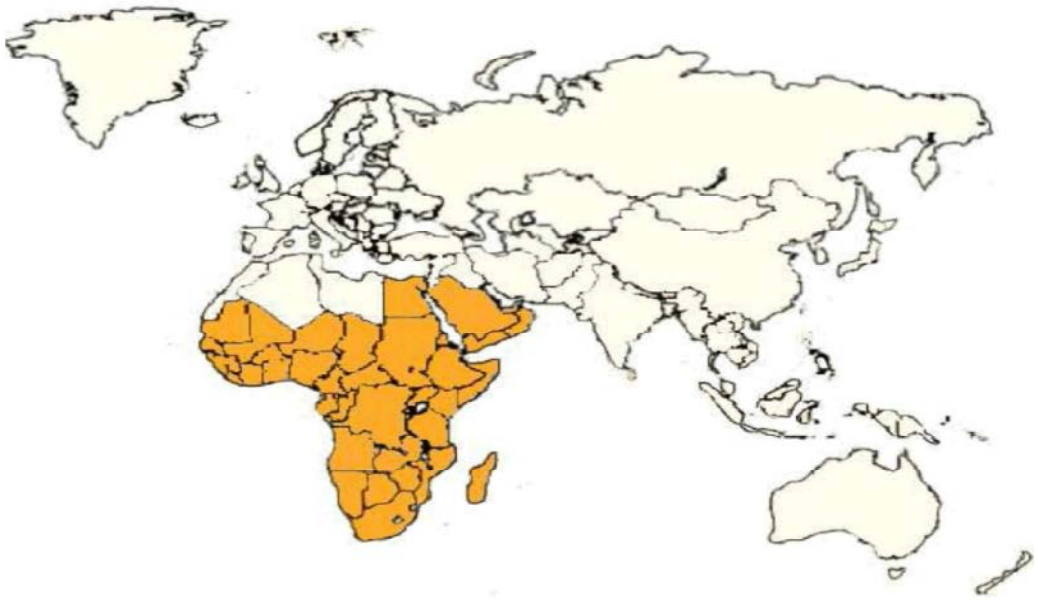

Fig. 1: Global Distribution of lumpy skin disease in the World (2012).
Note: The shaded area shows LSD positive countries
Source:http://www.epizone-eu.net/publicdocs/animal diseases/website LSD

Approximately 50% of infected animals are likely to show clinical signs; the majority of experimentally infected animals become viraemic and source of the virus. In experimentally infected cattle LSD virus was demonstrated in saliva for 11 days, semen for 22 days and in skin nodules for 33 days, but not in urine or faeces [54, 59-61].

Occurrence of the Disease: Lumpy skin disease has been suggested that, while extending its geographical distribution, the virus has increased in pathogenicity causing extensive epidemics and pandemics on the African continent with sporadic cases occurring during inter-epidemic years [62].

There is, also the possibility that LSDV might become established in the Middle East and spread to Asia and Europe [54]. It may be present in other Middle Eastern countries, which are lacking active surveillance [22, 54]. As mentioned by Greth *et al*. [62], the only African countries still considered free of the disease are Libya, Algeria, Morocco and Tunisia.

According to Gari *et al*. [25], about 90% respondents replied that the disease occurs from July to November, which is the season of high moisture and also extends up to December in Ethiopia. However, lack of an understanding of why LSD is not yet established outside of Africa and the Middle East is the point would be seen as a gap.

Species Involved and Role of Wildlife in the Spread of LSDV: Most of the time, Capri poxviruses are highly host specific. LSD is primarily a disease of all cattle, particularly thin-skinned European breeds, are susceptible [43]. Very little data are available on the susceptibility of wild ruminants to LSD [54].

Capri pox disease has been reported in domestic Asian water buffalo (Bubalus bubalis) and Arabian Oryx (Oryx leucoryx) [62], but could have been caused by closely related poxviruses [43, 50, 54]. However, it was not differentiated if these animals were infected with LSDV or sheep pox or goat poxvirus [54, 63].

Recently, the persistence of LSDV nucleic acid was reported in skin samples collected from springbok (Antidorcas marsupialis) in South Africa [64]. Natural infections were reported in Asian water buffalo (Bubalus bubalis) during the LSD outbreak in Egypt in 1988, but the morbidity was significantly lower in buffalo (1.6%) than in cattle (30.8%) [29]. Natural cases have not been seen in an impala (Aepycerosmelampus) and a giraffe (*Giraffa camelopardalis*) but, have been success fully infected experimentally [40,43] and clinical signs of LSD have been demonstrated in both of them after experimental inoculation with LSDV [65].

LSDV will replicate in sheep and goats following inoculation, but the role of these species as potential reservoirs of the virus is yet to be clarified [54, 66-68]. No carrier status is recognized in cattle following infection with LSDV although live virus can be detected up to 39 days post infection in the skin of infected animal [15, 40, 54, 69].

According to Barnard [70], animals with mild or in apparent infection with LSDV do not always show antibody levels detectable with a neutralization assay. Therefore, the actual number of LSDV-infected wild ruminants may be considerably higher than that revealed by this test. Wild animals showing clinical signs of LSD are likely to be more susceptible to predators, which could explain the lack of reports of clinical disease in wildlife species. In addition, the presence of clinical signs of LSD in wildlife is easily missed, as the monitoring of the skin lesions is difficult or impossible, especially in mild cases.

Risk Factors Associated with Lumpy Skin Disease: Capri poxviruses have a long incubation period; animals intentionally infected can travel a considerable distance before showing disease and can therefore disperse and spread disease.

Pathogen factors: LSDV is remarkably stable, surviving for long periods at ambient temperature, especially in dried scabs. It is very resistant to inactivation, surviving in necrotic skin nodules for up to 33 days (-80°C for 10 years) or longer, desiccated crusts for up to 35 days, infected tissue culture fluid stored at 4°C for 6 months, (If protected from sunlight) and at least 18 days in air-dried hides [59].

It can remain viable for long periods in the environment, but virus is susceptible to high temperatures (Inactivation is achieved by heating at 55ºC for 2 hours, 65°C/30 minutes) and also to highly alkaline or acidic pH. LSDV is susceptible to sunlight, but survives well at cold temperatures [59, 54].

Environment Factors: There is a dynamic relationship between the “agent” that transmits the diseases, the “host” that entertains on the one side and the “environment” on the other. The host and the agent operate within a particular environment and depending on that environment disease spreads and is controlled [71].

The potential risk of agro-climate variations in LSD occurrence showed that midland and lowland agro-climates were more likely to be at risk for LSD occurrence than the highland agro-climate [10]. The warm and humid climate in midland and lowland agro-climates has been considered a more favourable environment for the occurrence of large populations of biting flies than the cool temperature in the highlands [54, 69].

Herd contact and mixing is likely to occur in communal grazing and watering points and these were found to be significantly associated with LSD occurrence [10]. Post-harvest fields would allow contact and intermingling of different herds that would probably increase the risk of exposure and enhance the virus transmission through the speculated mechanical vectors such as Stomoxys spp. and mosquitoes (*Aedes aegypti*) [10, 32, 72].

The Host Factors: Host susceptibility, dose and route of virus inoculation affect the severity of disease. All breeds, age groups and both sex of cattle are considered to be at risk can be infected with severe and serious complications, while Asian water buffalo are also reported to be susceptible. *Bos taurus* breeds of cattle are more susceptible than *Bos indicus* breeds and young calves often experience more severe disease than adults [22, 43, 54].

The morbidity rate varies widely depending on the immune status of the hosts (Host susceptibility) and the abundance of mechanical arthropod vectors [73]. As more recently reported by Birhanu [12], an introduction of new animals to the herd was highly associated with the occurrence of LSD. According to OIE [74], there is no evidence or report that the virus can affect humans.

Transmission: LSDV is thought to be primarily transmitted by biting and blood feeding arthropods, which are believed to act as mechanical rather than biological vectors [22, 32, 44, 54]. Recently, new evidence has been published reporting a possible role for hard ticks in the transmission of LSDV [54].

The study showed molecular evidence of transtadial and transovarial transmission of LSDV by R. (B.) decoloratus ticks and mechanical or intrastadial transmission by R. appendiculatus and A. hebraeumticks [54]. Female Aedes aegypti mosquitoes were shown to transmit LSDV from infected to susceptible cattle for 2-6 days post-feeding on experimentally infected animals [32].

However, attempts to transmit LSDV between experimentally infected and susceptible cattle by Stomoxys calcitrans have failed [13], as did the transmission of LSDV by two species of mosquito (Anopheles stephensi and Culex quinquefasciatus) and the biting midge (Culicoides nubeculosus) [13, 40, 69].

The host's reaction to the piercing pain from the fly's bite would interrupt the insects' feeding, which would lead to the flies looking for other nearby hosts to complete their feeding, allowing the transmission of the infection from infected to susceptible animals [10].

Tabanidae, *Glossina* and *Culicoides* spp. have all been found in situations where there has been ongoing LSD transmission and have been suspected to be involved. *Stomoxys* spp. has been shown to transmit SGPV successfully [17].

Transmission of LSDV through semen has not been experimentally demonstrated, but LSDV has been isolated in the semen of experimentally infected bulls [54, 60, 61]. Transmission via infected saliva, ocular and nasal discharge, contaminated food and water by infected feces and urine needs to be demonstrated under experimental conditions [75].

Morbidity and Mortality: The incidence of disease is highest in wet, warm weather and decreases during the dry season. New foci of disease can appear at distant sites; in these cases, the virus is thought to be carried by insects [53].

According to Woods [40], recorded morbidity rates have varied greatly from as low as 5% to 100%. Mortality, except in exceptional circumstances, rarely rises above 5%. In outbreaks of the disease, the morbidity rate varies widely depending on the immune status of the hosts (Host susceptibility) and the abundance of mechanical arthropod vectors and usually ranges from 3% to 85% [43, 54,73, 76,77].

Again, according to Davies [17], morbidity rates of 1 to 2 % may be contrasted with those of 80 to 90 % in different situations. Mortality rates of 10 to 40 %t and even higher have been reported on occasion but the much lower range of 1 to 5 % is more usual. However, as more recently reported, more severe disease is seen in *Bos taurus,* particularly Channel Island breeds, than zebu cattle. Calves and lactating cows tend to be most susceptible to disease [17, 53, 54]. In general, mortality rate is low (1-3%) but in some occasions up to 75% mortality has been reported [42, 73].

Mechanism of Pathogenicity: During the acute stage of skin lesions, histopathological changes include vasculitis and lymphangitis with concomitant thrombosis and infarction, which result in to oedema and necrosis.

LSD skin nodules may exude serum initially but develop a characteristic inverted greyish pink conical zone of necrosis. Adjacent tissue exhibits congestion, haemorrhages and oedema. The necrotic cores become separated from the adjacent skin and are referred to as 'sit-fasts'. Enlarged lymph nodes are found and secondary bacterial infections are common within the necrotic cores. Multiple virus-encoded factors are produced during infection, which influence pathogenesis and disease [54, 78].

Incubation Period and Clinical Signs: Incubation period: Lumpy skin disease has an incubation period of 2 to 4 weeks in the field. In experimentally infected animals, fever can develop in 6 to 9 days and lesions first appear at the inoculation site in 4 to 20 days [22, 43, 54].

Clinical Signs: LSD is an acute infectious disease of cattle of all ages [17, 79] but young calves often have more severe disease than adults [43, 73]. The severity of clinical signs of LSD depends on the strain of Capri poxvirus and the host cattle breed. It causes acute to sub-acute systemic disease characterized by mild to severe symptoms including fever, nodules on the skin, in the mucous membranes and in the internal organs, skin oedema, lymphadenitis and occasionally death [17, 40, 60].

Fever is the initial sign. It is usually followed within two days by the development of nodules on the skin and mucous membranes [43]. These nodules vary from 1 cm to 7 cm and common on the head, neck, udder, genitalia, perineum and legs. Although they penetrate the epidermis and dermis, subcutaneous tissue and sometimes they extend into the underlying musculature, which are a nidus for secondary bacterial infections and fly infestations [43, 54, 73].

The skin nodules are painful and could involve tissues up to the musculature [22, 42]. Where extensive generalization occurs, animals may become lame and reluctant to move. Abortion may occur because of prolonged fever [21, 40, 42, 47, 50]. In severely affected animals, ulcerative lesions appear in the mucous membranes of eye and oral/nasal cavities causing excessive salivation, lachrymation and nasal discharge. All these secretions may contain LSDV [24,53]. Pox lesions may also be present in the pharynx, larynx, trachea, lungs and throughout the alimentary tract. Raised, circular, firm, coalescing nodules are common and cores of necrotic material called "sit-fasts".

Diagnosis of LSD: At present, there are no diagnostic test kits for LSDV commercially available [54]. The tentative diagnosis of LSD is usually based on characteristic clinical signs, differential diagnosis and the clinical diagnosis is confirmed by laboratory tests (Using conventional PCR) [43, 59, 60, 74].

Clinical Examination: Clinically, LSD should be suspected when the characteristic skin nodules, fever and enlarged superficial lymph nodes are seen and the mortality rate is usually low [43]. The appearance of the skin lumps that characterize the disease follows within 48 hours. Nodules may appear anywhere on the body from the nose to the tail. Distribution is in a random pattern and not linear. Similar lesions appear in the mucosa of the mouth, nose, vagina and conjunctiva. A purulent nasal and ocular discharge is common [39, 44].

Laboratory Tests: Laboratory confirmation of LSD is most rapid using a polymerase chain reaction (PCR) method specific for Capri poxviruses or by the demonstration of typical Capri pox virions in biopsy material or desiccated crusts using the transmission electron microscope in

(a) (b)

Fig. 2: Nodules on the skin of the animals
Source: OIE [74], Iowa State University (B) and Gari et al. [11] (A).

combination with a clinical history of a generalised nodular skin disease and enlarged superficial lymph glands in cattle [22,50].

As mentioned by CFSPH [43], Tuppurainen and Oura [54] and OIE [74], routine Diagnostic Techniques are described in the OIE Manual of Diagnostic Tests and Vaccines including: Identification of the agent and Serological tests.

Identification of the agent: Capri poxvirus is distinct from Para poxvirus, which causes bovine popular stomatitis and pseudo cowpox, but cannot be distinguished morphologically from cowpox and vaccinia virus, both *orthopoxvirus* infections of cattle [22].

Confirmation of lumpy skin disease in a new area requires virus isolation and identification [43, 54, 74]. LSDV will grow in bovine, caprine or ovine cell cultures; the best growth is seen in lamb testis cells [80]. LSDV can be distinguished from the herpes virus that causes pseudo-lumpy skin disease by the cytopathic effect and the intracytoplasmic location of inclusion bodies.

LSDV antigens can be identified with direct immunofluorescence, virus neutralization or enzyme-linked immune sorbent assay (ELISA). LSDV can be detected in cell cultures or directly in tissues by polymerase chain reaction (PCR) assays [22, 43, 54]. Genome detection using *Capri pox* virus-specific primers for the fusion protein gene and attachment protein gene has been reported and several conventional and real-time PCR methods have been published for use on blood, tissue and semen samples [22].

Serological tests: Serological tests include an indirect fluorescent antibody test, virus neutralization, ELISA and immune blotting (Western blotting). Cross-reactions with other poxviruses are seen in some assays. Agar gel immune diffusion is also available, but cross-reactions occur in this test with bovine papular stomatitis and pseudo cowpox virus [22, 43]. The virus neutralisation test is the most specific serological test, but because immunity to LSD infection is predominantly cell mediated, the test is not sufficiently sensitive to identify animals that have had contact with LSD virus and developed only low levels of neutralizing antibody.

The agar gel immune diffusion test and indirect immune fluorescent antibody test are less specific due to cross-reactions with antibody to other poxviruses. Western blotting using the reaction between the P32 antigens of LSD virus with test sera is both sensitive and specific, but is difficult and expensive to carry out [22].

Indirect Fluorescent Antibody Test (IFAT): It demonstrated to be suitable for use in retrospective serological surveys in a study carried out in Ethiopia and it was evaluated test for accuracy [25].The IFAT is a serological test for Capri pox Virus. It was used to detect serum antibody against *Capri pox* Virus and differentiate serological positive and negative animals.

Differential Diagnosis: Sskin lumps appear in cattle due to many conditions other than lumpy skin disease and it is obvious that confusion has been caused by many of them in the past [40]. Misdiagnosis and misreporting have probably been common over the years due to veterinarians not having had previous experience of the disease [22, 40].

Severe LSD is highly characteristic, but milder forms can be confused with: Pseudo lumpy skin disease (Bovine Herpesvirus$_2$), Bovine papular stomatitis (Para poxvirus), Pseudo cowpox (Para poxvirus), Vaccinia virus and Cowpox virus (Orthopoxviruses) uncommon and not generalised infections, Dermatophilosis, Insect or tick bites, Besnoitiosis, Rinderpest, Demodicosis, Hypoderma bovis infection, Photosensitisation, Urticaria, Cutaneous tuberculosis, Onchocercosis [17, 59, 81].

Pathological Lesions: Post mortem lesions can be extensive. Characteristic deep nodules are found in the skin that penetrate into the subcutaneous tissues and muscle with congestion, hemorrhage and edema. Lesions may also be found in the mucous membranes of the oral and nasal cavities as well as the gastrointestinal tract, lungs, testicles and urinary bladder. Bronchopneumonia may be present and enlarged superficial lymph nodes are common [43].

Nodules involving all layers of skin, subcutaneous tissue and often adjacent musculature, with congestion, haemorrhage, oedema, vasculitis and necrosis; Enlargement of lymph nodes draining affected areas with lymphoid proliferation, oedema, congestion and haemorrhage; Pox lesions of mucous membrane of the mouth, the pharynx, epiglottis, tongue and throughout the digestive tract; nasal cavity, trachea and lungs (Fig. 3, 4).

Oedema and areas of focal lobular atelectasis in lungs; Pleuritis with enlargement of the mediastinal lymph nodes in severe cases; Synovitis and tendosynovitis with fibrin in the synovial fluid; Pox lesions may be present in the testicles and urinary bladder [59].

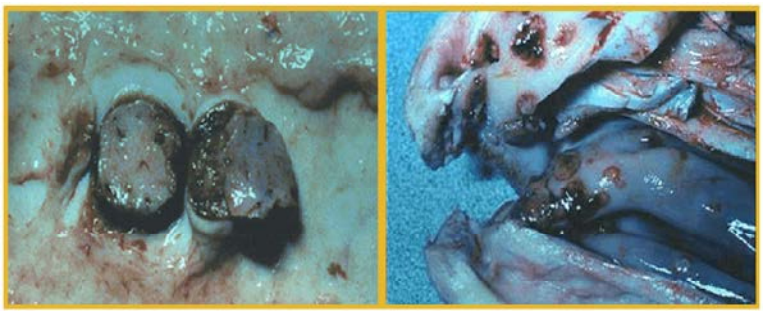

Fig. 3: Nodules in lungs Fig. 4: Lesions in the m/m throughout the GIT
Source: CFSPH [73], Iowa State University

Treatment, Control and Prevention

Treatment: There is no specific antiviral treatment available for LSD infected cattle. Sick animals may be removed from the herd and given supportive treatment consisting of local wound dressing to discourage fly worry and prevent secondary infections [43, 47, 54, 59].

Control and prevention: LSD should appear in cattle in another country beyond its previous range [17]. Therefore, it needs to stop if occurred and to block if new and this can be carried out either by sanitary prophylaxis or medical prophylaxis. In case of sanitary prophylaxis, import restrictions on livestock, carcasses, hides, skins and semen can help to prevent the introduction of LSD in to the disease free countries [71, 82-84].

It is mainly spread to new areas by infected animals, but it could also be transmitted in contaminated hides and other products. However, in infected countries, infected insects are suspected to have spread LSDV to new areas during some outbreaks. Outbreaks can be eradicated by strict quarantines to avoid introduction of infected animals into safe 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 [38, 43, 47,54, 59].

Medically, with the exception of vaccination, control measures are usually not effective [59]. Vaccination will greatly reduce the morbidity and economic effects of an epizootic but may not completely limit the extension of-LSD. Follow-up vaccination of calves and re-vaccination programmes over a period-of two to three years will greatly reduce the incidence of clinical disease.

Two different vaccines have been widely and successfully used for the prevention of LSD in cattle populations in Africa [47]. Homologous live attenuated virus vaccine: Neethling strain: immunity conferred lasts up to 3 years and heterologous live attenuated virus vaccine: Sheep or goat pox vaccine, but can sometimes cause severe local reactions. Not advised in countries free from sheep and goat pox [54, 59].

As reported by Davies [17], two other strains of sheep pox vaccine have recently been used as a prophylaxis against LSD. The Romanian strain, prepared in the skin of lambs for use against sheep pox, was used in several million cattle in Egypt and appeared to be immunogenic. No complications have followed the use of these strains in cattle. No country in sub-Saharan Africa, however, has succeeded in eradicating LSD once it has occurred.

LSDV is susceptible to ether (20%), chloroform, formalin (1%) and some detergents, as well as phenol (2% for 15 minutes). This virus can survive for long periods in the environment: up to 35 days in desiccated scabs and for at least 18 days in air-dried hides [43].

Status of Lumpy Skin Disease in Ethiopia: Ethiopia basically constitutes an agrarian society; the socio-economic activities of about 85% of the population are based on farming and animal husbandry [85].

The Ethiopian economy is highly dependent on agriculture, which in the 2004/05 fiscal year, contributed about 48% of the GDP, followed by 39% from the service sector and 13% from the industrial sector [6]. The country has been earning foreign currency by exporting meat (Mainly chilled shoats' carcass) and live animals namely cattle, sheep, camels and goats to major destination markets of UAE, KSA, Yemen and Egypt. As the country has the largest number of livestock in Africa, Ethiopia has much to gain from the growing global market for livestock products [86].Livestock disease is one of the major livestock production constraints in Ethiopia [6].

Table 1: Official reported and non-reported LSD outbreaks in the different of districts of some Administrative Regions of Ethiopia from years 2000-2007

	Years of an outbreak of the disease								
Region/district	2000	2001	2002	2003	2004	2005	2006	2007	Total
Amhara									
Laygayint		5							5
Farta	5							2	7
Gozamen	1	5						1	7
Kobo								5	5
Oromiya									
Adola					1	14	3		18
Yabello		1	3	13	8	6	4	1	36
Sebeta-Awas		1	2	1	2	1	1	2	8
Bako-Tibe									2
Chora				10					10
Fentale				3					3
SNNP									
Kabiena							1		1
Afar									
Awash-Fentale			1						1
Total	6	12	6	27	11	21	14	6	103

Source: Gari *et al*. [10]

Table 2: Reported outbreaks and LSD affected populations in different regions of Ethiopia from 2007-2011

	Years of reported outbreaks					
Regions	2007	2008	2009	2010	2011	Total
Addis Ababa			3	7	1	11
Afar			3	2	2	7
Amhara	92	68	35	40	22	257
Ben.Gumuz	3				5	8
Gambela				1	9	10
Oromiya	95	154	219	268	160	896
SNNP	18	18	14	32	17	99
Somali			3	9	4	16
Tigray	7	8	2	18	13	48
Grand Total	215	248	276	375	233	1347

Source: Birhanu [12].

Lumpy skin disease has been one of the newly emerging diseases of cattle in Ethiopia. Lumpy Skin Disease was flrst observed in southwest of Lake Tana in 1983 [23].

A major epidemic outbreak of LSD occurred in 2000/2001: in Amhara and Western part of Oromiya Region, in 2003/2004: again in Oromiya and Southern Nations and Nationalities People regions and in 2006/2007: in Tigray, Amhara and Benishangul regions (Table 2).

In terms of the size and magnitude of its occurrence, an epidemic of LSD covering a number of PAs was reported to have occurred in some districts (Adola and Yabello districts) in the years 2003-2005 [10] (Table 1). In Somali regional state, the first case of an epidemic of Lumpy Skin Disease in cattle was reported in Somali Region in 2005 [87].

As reported by Gari *et al.* [10], LSD has been extensively circulating across diverse agro-climatic zones of Ethiopia with large variations between districts that could be attributed to their respective agro-ecological zones and farming practices. Additionally, the same author [11] explained that, animal and herd sero-prevalence was higher in the midland agro-climate than in highland and lowland agro-climate zones and suggested that the prevalence of LSD infection in Ethiopia is higher than what has been previously reported.

In addition, recent survey which assesses the risk factors and financial impacts of LSD in selected districts of North-eastern Ethiopia (Tigray and Afar Regional States) conducted by Birhanu [12] reported a higher herd prevalence of 51% and 37% was recorded in Afar and Tigray Region respectively.

Economic Importance of the Disease: Lumpy skin disease (LSD) is an economically important disease of cattle, serious economic losses can follow outbreaks that have a high morbidity and can produce a chronic debility in infected Cattle [10, 17, 54].

Even though the morbidity and mortality rates of LSD are usually low, it is an economically important disease of cattle in Africa because of the prolonged loss of productivity of dairy and beef cattle, use of the animals for traction, decrease in body weight, mastitis, severe orchitis, which may result in temporary infertility and sometimes permanent sterility. The valuation of the draft power loss depended on the point in the crop season that an ox fell sick and on the corresponding demand for draft power during that speciflc season. Thus, the draft work output loss in terms of days was taken into account on two levels, when demand for draft power was high and when it was low, with demand determined by the crop calendar prevailing at the onset of the disease [16, 21, 54, 60]. *Capri pox* viruses are classified by the government of the United States as potential agents for agri terrorism [54].

The average quantity of milk production loss and the time duration that LSD affected, lactating cow was subject to milk production loss [53, 73].

The annual beef production losses were estimated as the decrease in the off-take rate in the study groups. LSD incidence interferes with normal herd dynamics, causing a reduction of surplus in the case of mortality, or a reduction in flnished stock for the market in affected herds because of long term morbidity that can lower weight gain [73].

In any epizootic, economic losses clearly depend on the morbidity rates and are brought about by mortality, the loss of production, the depression of growth rates and hide damage [17].Production loss due to mortality was computed based on the weighted average price, determined for each breed, sex and age group, of animals that had died of LSD [73].

Apart from quality degradation of skin and hides skin diseases including LSD induce associated economic losses due to reduction of wool quality, meat, losses as a result of culling and occasional mortalities and related with cost of treatment and prevention of the diseases [20]. The disease is more severe in cows in the peak of lactation and causes a sharp drop in milk yield because of high fever caused by the viral infection itself and secondary bacterial mastitis [54].Even though there is no specific antiviral treatment available for LSD-infected cattle, there will be treatment cost for secondary bacterial infection.

Treatment cost represents the expenses incurred by farmers for medication at the local public veterinary clinics when farmers bring their clinically sick animals for treatment [16].

Restrictions to the global trade of live animals and animal products, costly control and eradication measures such as vaccination campaigns as well as the indirect costs because of the compulsory limitations in animal movements cause significant financial losses on a national level. The disease is listed as notifiable throughout the European Community (82/894/EEC, 89/162/EEC) and should an outbreak occurs, regulation would require the slaughter of affected and in-contact animals and the implementation of a 3-km protection zone and a 10-km surveillance zone around the infected premises [54].

CONCLUSION

LSD has now spread out of the African continent into the Middle East region and there are no geographical or epidemiological reasons why the disease cannot spread further north into some countries, or further east into continent of Asia. It is also difficult to predict with accuracy how global climate change may affect the abundance and distribution of mechanical vector populations, which may have a direct effect on the spread of this disease. The use of insecticides to control biting flies is poor in Ethiopia except using pour-on drugs few areas where infested with tsetse flies. Pathogen factors (LSDV stable, Environment Factors (Grazing and watering points) and the Host Factors (Host susceptibility, all breeds, age groups and both sex of cattle are considered to be at risk can be infected).

As currently available live attenuated vaccine does not provide each individual with solid protection, vaccinated animals can develop skin lesions that contain high virus titres, resulting in the possible dissemination of the virus by blood-feeding arthropods. No alternative safe and effective vaccines, suitable for use in countries free of LSD, sheep pox and goat pox, are currently available. The eradication of the disease is further hampered by a lack of epidemiological data about how long the virus can survive in the environment and whether wildlife plays a role as a reservoir for LSDV between outbreaks. These observations underline the need for the development of improved vaccines that can be used in non-endemic countries and can be combined with sensitive serological assays capable of differentiating vaccinated from infected animals.

Currently there is one project in Ethiopia (NAHDIC with integration of NVI and MoA) on improvement of the efficacy of LSD vaccines. Because of the *Capri pox* viruses are classified as potential agents for agri terrorism and listed as notifiable disease, it causes serious economic losses. Further work is also required to improve the sensitivity and specificity of *Capri pox* antibody ELISAs, especially for LSD and, as rapid recognition of the disease is essential for its successful control and eradication, there is also a need to develop pen-side tests for the swift detection of LSDV at a farm level. The role of specific arthropod vectors in transmission of disease requires clear explanation. Alterations in vector distribution and dynamics caused by climate change need to be considered. Potential mechanical transmission by humans after handling infected animals should be considered.

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