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Epidemiological Status and Economic Impact of Lumpy Skin Disease-Review

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ABSTRACT

Lumpy skin disease is an acute infectious disease of cattle endemic in almost African countries, Middle East countries, and Mediterranean regions. It is caused by a virus associated with the Neethling poxvirus in the genus Capripoxvirus of the family Poxviridae. Clinically sick animals are the main source of infection. Generally, the clinical severity of the disease depends on susceptibility, immunological status, and age of the host population and dose and route of virus inoculation. The disease is characterized by fever, enlarged lymph nodes, firm and circumscribed nodules in the skin, and nodules are particularly noticeable in the hairless areas. It occurs in all agro-climatic conditions but it is common in low lying areas in particular and along watercourses. It is transmitted by insect vectors in cattle sharing similar grazing and watering areas and those congregated in the same barn. It is economically devastating viral diseases that cause several financial problems in livestock industries as a result of significant milk yield loss, infertility, weight loss, abortion, reduced output of animal production, increase production costs due to increased costs of disease control, trade limitation, and sometimes death in most African countries including Ethiopia. An effective control measure of the disease is achieved through mass vaccination through the separation and culling of infected animals. Good understanding of the epidemiology, economic significance, and control mechanisms of the disease is needed to design suitable control measures.

Keywords: *Economic Impact, Epidemiology, Lumpy Skin Disease, Lumpy Skin Disease Virus, Vectors*

INTRODUCTION

Lumpy skin disease (LSD) is a severe, systemic disease of cattle associated with the Neethling poxvirus, from the family *Poxviridae*, genus *Capripoxvirus*, and species lumpy skin disease virus (LSDV). It is one of the most economically significant of trans-boundary disease, which is emerging a viral disease that affects cattle of all ages and breeds.

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It has close antigenic relationship to sheep pox and goat pox viruses which are also in the same genus. There appears to be a difference in virulence between strains (OIE, 2010). It is also called *Pseudo* urticaria, Neethling virus disease, Exanthema Nodularis Bovis, Knopvelsiekte. It is an acute to chronic infectious viral disease which is characterized by fever, movable nodules on the skin, mucous membranes and internal organs, high morbidity, low mortality, emaciation, enlarged lymph nodes, edema of leg and brisket, mastitis in female, orchitis in male animals and sometimes death (Radostitis et al., 2007).

The first Lumpy skin disease was seen in Zambia in 1929 that was associated with either plant poisoning or an allergic response to an insect bite (Tuppurainen & Oura, 2011). Later, it was spread from Zambia into Botswana and South Africa, where it affected over eight million cattle causing major economic loss. In 1957, it was reported in Kenya associated with an outbreak of sheep pox. In the between 1970-1977, LSD spread in to different part of the continent including Sudan, Nigeria, Mauritania, Mali, Ghana, and Liberia (OIE, 2010). Another epidemic of LSD between 1981 and 1986 affected Tanzania, Kenya, Zimbabwe, Somalia, Cameroon, and Ethiopia with reported mortality rates of 20% in affected cattle. In subsequent years, it was reported from Bahrain, Kuwait, Oman, Yemen, and Israel. Until 1989, LSD occurrence was restricted to sub-Saharan Africa, but Egypt reported its first LSD outbreak in 1988 and Israel in 1989 (OIE, 2010).

The most common method of transmission of the disease is mechanical through biting flies. Therefore, the incidence of LSD is high during the wet season when the biting fly population is abundant and it decreases during the dry season. Direct transmission can also occur between infected animals, but such transmission is rare and it is of low epidemiological significance (OIE, 2015b). Lumpy skin disease is usually diagnosed based on characteristic clinical signs, epidemiology, histopathology, virus isolation, and laboratory diagnosis using Polymerase Chain Reaction (PCR) (Tuppurainen & Oura, 2012).

The disease causes significant economic impacts on the livestock industry, as a result of reduced milk production, temporary or permanent sterility, loss of beef, loss of draft animals' power, abortion, loss of body condition and damage to the hide, and lastly death. There is no specific treatment for LSD, but supportive treatment is available. Due to the lack of effective treatment for LSD, there are many practices implemented to prevent the disease. The most important control and prevention strategies method for LSD are ring vaccination, quarantine infected animals, control movement of animals, and vector control (Tuppurainen & Oura, 2011). Therefore, this is to review the epidemiological aspects and economic impact of lumpy skin disease.

2. EPIDEMIOLOGY OF LUMPY SKIN DISEASE

2.1. Epidemiology

Lumpy skin disease is an important, economically overwhelming, clinical disease that causes production loss in cattle due to generalized malaises and chronic debility (Tuppurainen & Oura, 2011). A good understanding of the epidemiological aspects of LSD related to pathogen, host, and environment might aid in control and prevention mechanisms. Particular emphasis should be given to exposure of hosts to the pathogen in a suitable environment that facilitates the transmission and distribution of the disease. Lumpy skin disease is more predominant in the wet summer and autumn months and occurs principally in low land areas and along with watercourses (OIE, 2010).

2.1.1. Geographic distribution

Lumpy skin disease has a different geographical distribution (OIE, 2010). The disease was first originated from Zambia in 1929 and then it extended its range to include all countries in sub-Saharan Africa as well as Madagascar. It is endemic to all most all African countries and occurs in various

ecological zones except Libya, Algeria, Morocco, and Tunisia which are still considered free of the disease (Figure 2). Outbreaks outside the African continent have occurred in the Middle East in 2006 and 2007, in Mauritius in 2008 (OIE, 2014b) and Israel has reported LSD outbreaks (Brenner et al., 2006). The epidemiological trend of LSD suggests that it is currently endemic in most African countries and spreading further into North Africa, Middle East countries and Mediterranean regions because of global trade movement in animals and animal products (Tuppurinen & Oura, 2011; 2012).

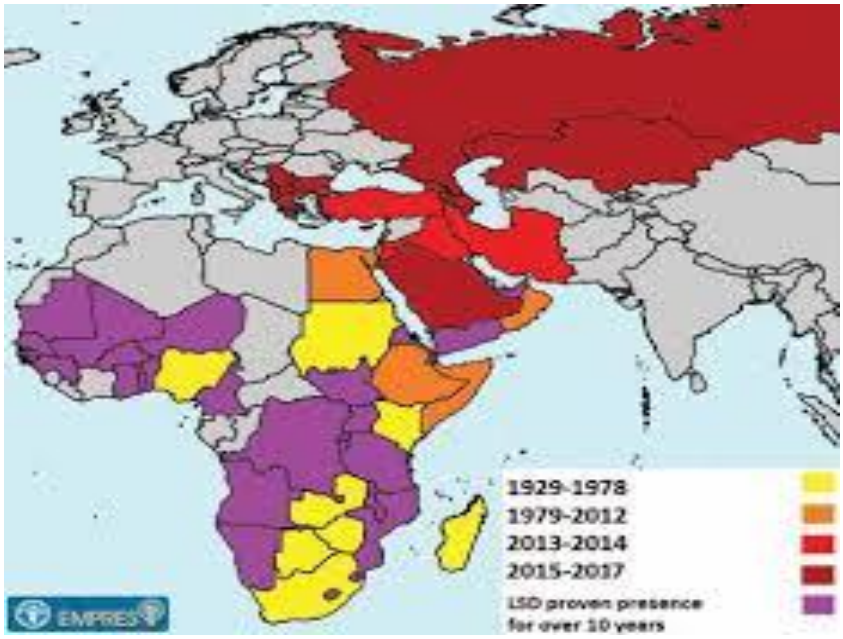

Fig. 1: Geographical distribution of LSD in the world

Source: FAO (2017).

2.1.2. Species of animal affected

Lumpy skin disease is primarily a disease of all cattle; in particular thin-skinned European breeds are susceptible (Brenner, 2006). Capripox viruses are highly host-specific, with only a few known exceptions. Very few data are available on the susceptibility of wild ruminants to LSD. Capri pox disease has been reported in domestic Asian water buffalo and Arabian Oryx. However, it was not differentiated if these animals were infected with LSDV or sheep pox or goat pox virus (CFSPH, 2008; Tuppurainen & Oura, 2012). Natural cases have not been seen in Impala and Giraffe but demonstrated in both of them after experimental inoculation with LSDV. The absence of a reservoir host for LSD virus might lead to the assumption that infection might persist in the endemic areas at a low level as an unapparent or mild form in the cattle population (OIE, 2008).

2.1.3. Source of infection

Clinically infected animals are the main source of infection to other healthy animals. However, LSD virus can be present in the blood, cutaneous lesions, saliva, nasal discharge, lachrymal secretions, milk, semen and very rarely drinking water, which may be sources for transmission (Irons et al., 2005; Babiuk et al., 2008b; Abera et al., 2015).

2.1.4. Risk factors

Host risk factors

LSD is a disease of cattle and causes several disorders. Though all breeds and age groups are susceptible, *Bos Taurus* is particularly more susceptible to clinical disease than zebu cattle and *B. indicus* (Radostits et al., 2007). Among *Bos taurus*, fine-skinned, high-producing dairy channel Island breeds are highly susceptible to LSDV (EFSA AHAW Panel, 2015). Lactating cows appear to be severely affected and result in a sharp drop in

milk production because of high fever caused by the viral infection itself and secondary bacterial mastitis (Tuppurainen & Oura, 2011). Whereas indigenous breeds such as zebu and zebu crosses are probable to have some natural resistance against the virus (Gari et al., 2011). It is not known what genetic factors influence disease severity (Babiuk et al., 2008). High ambient temperature, farming practices, and cow which produce high milk yield could be deemed to stress animals and contribute to the severity of the disease in Holstein-Friesian (HF) cattle (Tageldin et al., 2014).

Young animals are mostly clinically affected presented with most clinical symptoms. But traditional calf management practices that segregate calves from the herd might have contributed to a decreased exposure risk of calves to the source of infection. Calves in the endemic area can obtain certain protective passive immunity from their dam. An animal recently recovered from an attack is not susceptible to LSDV because there is a solid immunity lasting for about 3 months (Gari et al., 2011).

In local zebu cattle, male animals have higher cumulative incidence than females due to the stress factor of exhaustion and lethargy rather than a biological reason. The majority of male animals are working oxen used for heavy labor, which might contribute to an increase in susceptibility. Another reason is that draft oxen cannot protect themselves well from biting flies when harnessed in the yoke, and the beat scratches on their skin induced while plowing may attract biting flies capable of transmitting LSD infection (Gari et al., 2011; Tageldin et al., 2014). Generally, the clinical severity of the disease depends on susceptibility, immunological status, and age of the host population and dose and route of virus inoculation (CFSPH, 2008). There is no evidence or report that the virus can affect humans (OIE, 2011).

Pathogen risk factors

LSD virus is one of the species of capripox viruses that is resistant to different chemical and physical agents (Murphy et al., 1999). Capripoxviruses have lipid-containing envelopes and susceptible to a range of detergents containing lipid solvents like ether (20%), chloroform, formalin (1%), phenol and sunlight. They are also susceptible to sunlight but survive well at cold temperatures. Lumpy skin disease virus is susceptible to a temperature of 55°C/two hours, 65°C/30 minutes, alkaline, or acid pH. No significant reduction in titer when held at a pH of 6.6-8.6 for five days at 37°C (OIE, 2014b).

Lumpy skin disease virus is present in nasal, lachrymal and pharyngeal secretions, semen, milk, and blood. However, the virus may persist in saliva for up to 11 days, in semen for 22, in necrotic tissue remaining at the site of a skin lesion for 33 days and 6 months on fomites, including clothing and equipment but there is no evidence that virus can survive more than four days in insect vectors. There is no evidence of the virus persisting in the meat of infected animals, but it might be isolated from milk in the early stages of fever (Babiuk et al., 2008a). Capri poxviruses are very resistant in the environment and can remain viable for long periods on or off the animal host. Capri poxviruses may persist for up to 6 months in a suitable environment such as shaded animal pens and can be recovered from skin nodules kept at -80°C for 10 years and infected tissue culture fluid stored at 4°C for six months (Animal Health Australia, 2009).

Environmental risk factors

Environmental determinants play a great role in the epidemiology of LSD. It has a major impact on the agent, host, and vectors as well as the interaction between them. These predisposing factors have a great role in the maintenance of the arthropod vector and transmission of the virus to susceptible animals. Animals sharing the communal grazing lands and watering points, uncontrolled cattle movements across different borders due to trade and pastoralism, rainfall and wet climate which favor insect multiplications, other reasons of cattle movement from place to place and presence of water bodies are some of the potential risk factors of LSD (Tuppurainen & Oura, 2011).

LSD is associated with an increased number of mechanical vectors (Magori-cohen, 2012). It is more prevalent during the wet and warmer conditions of summer and autumn months and occurs particularly in low lying agro-climate zone and along watercourses (OIE, 2010). The warm and humid climate in midland and lowland agro-climates has been considered as a more favorable environment for the occurrence of large populations of biting flies than the cool temperature in the highland (Tuppurainen et al., 2012).

2.2. Mechanism of Transmission

2.2.1. Direct transmission

Direct transmission can occur when the cattle share the same feeding and drinking trough that is infected by nasal, and salivary discharges, but ingestion and direct contact transmission are not common routes, even though the virus is present in nasal and lacrimal secretions, semen, and milk of infected animals. Most cases are believed to result from transmission by an arthropod vector (Lefèvre and Gourreau, 2010). Suckling calves may be infected through infected milk. The transmission of LSDV through semen has been experimentally demonstrated (Annandale et al., 2013). A more recent study demonstrated the persistence of the live virus in bovine semen for up to 42 days post-infection and viral DNA was detected until 159 days post-infection (Irons et al., 2005).

During the natural outbreak of LSD in Egypt in 2006-2007, 25% of cows had been found with infected ovary by LSDV, and 93% of cows suffered from ovarian inactivity and showed no signs of estrus (EFSA AHAW Panel, 2015). There is an assumption that the virus is also secreted in vaginal secretions. Generally, the transmission of the virus by contact is inefficient and field evidence reported that the disease is not contagious (Salib & Osman, 2011).

2.2.2. Role of vectors

The transmission of LSDV occurs mechanically by blood-feeding biting arthropods vectors including hard ticks, biting flies, and mosquitoes (Chihota et al., 2001; Magori-cohen, 2012). This vector related transmission is mechanical, rather than biological. This distinction is important because infectious organisms do not generally survive in vectors for long periods for multiplication. In the mechanical mode of transmission, the virus is transmitted via contaminated mouthparts of vectors without actual replication of the virus in arthropod cells or tissues. A study by Chihota et al. (2001) indicated that the virus can survive for 2-6 days post-feeding from infected cattle and transfers this to susceptible cattle by the female mosquito, *Aedes egypti* during experimental infection. Recently, new evidence has been published reporting a possible role of hard ticks in the transmission of LSDV. The study showed molecular evidence of trans-stadial and transovarian transmission of LSDV by *Boophilus decoloratus* and mechanical transmission by *Repicephalus appendiculatus* and *Ambyloma hebraeum* (Tuppurainen et al., 2011).

Mosquitoes (female *Aedes egypti and Culex quinquefasciatus)* and other flies such as *tabanids* (horse flies), biting midges (*Culicoides nubeculosus*) and *Glossina* species like tsetse fly are among the other arthropod vectors that play a great role in the transmission of the virus. Non-biting flies, including houseflies (*Muscidae*), bush fly (*Hippoboscidae*), and blowflies (*Calliphoridae*) are also very commonly associated with sucking of infected lachrymal, nasal or other secretions and transfer the virus to another susceptible animal. Vermin, predators, and wild birds might also act as mechanical carriers of the virus (Animal Health Australia, 2009). Epidemiological evidence suggests outbreaks of LSD are highly associated with the prevalence of high insect vectors population and with the upcoming of the rainy season. Epidemics of LSD are associated with rainy seasons, river basins, and ponds during which cattle grazed and humid areas that is conducive to insect multiplication (OIE, 2010).

2.3. Morbidity and Mortality

The morbidity of the disease is highest in wet, warm weather and decreases during the dry season (OIE, 2008). In outbreaks of the disease, the morbidity rate varies widely depending on the immune status of the hosts and the abundance of mechanical arthropod vectors and averagely ranges from 3 to 85% (CFSPH, 2008, Tuppurainen, et al., 2012). But it can reach as high as 100% in natural outbreaks while mortality rate rarely exceeds 5% but may sometimes reach 40% (Irons et al., 2005; Babiuk et al., 2008).

2.4. Pathogenesis

Lumpy skin disease is developed by the entry of infectious LSDV through skin or GIT mucosa then viremia accompanied by a febrile reaction. Then the virus reaches and causes swelling of regional lymph nodes (Gari et al., 2011). The mechanism by which the virus causes skin lesions is due to the replication of the virus in a specific cell such as endothelial cells of lymphatic and blood vessels walls with the development of inflammatory nodules on the skin (Vorster, 2008). Lumpy skin disease is generalized and epitheliotrophic disease that cause localized and systemic reaction and results in vasculitis and lymphadenitis which result in edema and necrosis. In some severe cases, thrombosis and other symptoms will be observed. Nodules of LSD may be changed to grey-pink with caseous necrotic cores. Circumscribed necrotic lesions may ulcerate. Skin localization is due to epitheliotrophic property of LSDV (Radostitis et al., 2007).

Lumpy skin disease skin nodules may exude serum initially but develop a characteristic inverted grayish pink conical zone of necrosis. Adjacent tissue exhibits congestion, hemorrhages, and edema. 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 (Tuppurainen et al., 2012). The incubation period of LSD can vary under field and experimental conditions. It varies from 4-14 days in experimentally inoculated animals and 2–4 weeks in naturally infected animals (OIE, 2010).

2.5. Clinical Signs and Pathological Lesions

2.5.1. Clinical Signs

The course of LSD may be acute, sub-acute, and chronic. The virus causes unapparent infection to severe clinical symptoms and those animals which develop the clinical disease may have a biphasic febrile reaction. The major visible clinical signs are; fever of 40-41.5^{o}C which may last 6-72 hours, lachrymation, increased nasal and pharyngeal secretion, loss of appetite, reduced milk production, some depression and movement reluctance, a nodule in the skin (Figure 2), mucous membrane and internal organs and swelling of superficial lymph nodes. The diameter of the nodular lesion may be up to 1-7 cm diameter appears as round, firm, intradermal, and circumscribed areas of erected hair (OIE, 2010; Tuppurinen & Oura, 2011).

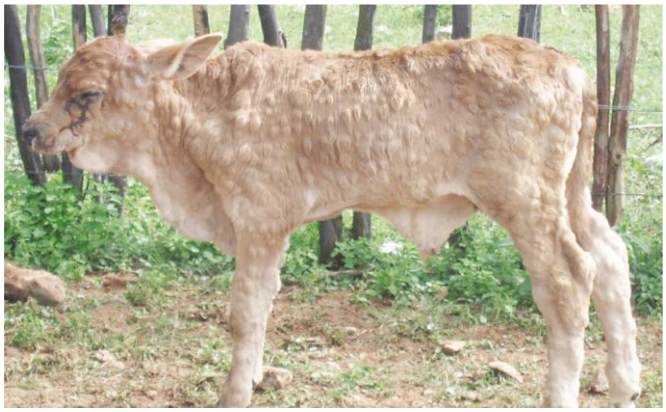

Fig. 2: Multifocal nodules on the entire skin of a calf

Source: Abutarbush (2013).

In severe cases, ulcerative lesions may develop in the mucous membrane of mouth, trachea, and larynx and esophagus (Radostitis et al., 2007). The necrotic cores become separated from the adjacent skin and are referred to as 'sit-fasts' (Figure 3). It might be exacerbated by secondary bacterial complication and infestation of fly worms (CFSPH, 2008). Lesions in skin, subcutaneous tissue, and muscles of limbs, together with severe skin inflammation caused by secondary infection of lesions, greatly reduce mobility as indicated (Murphy et al., 1999).

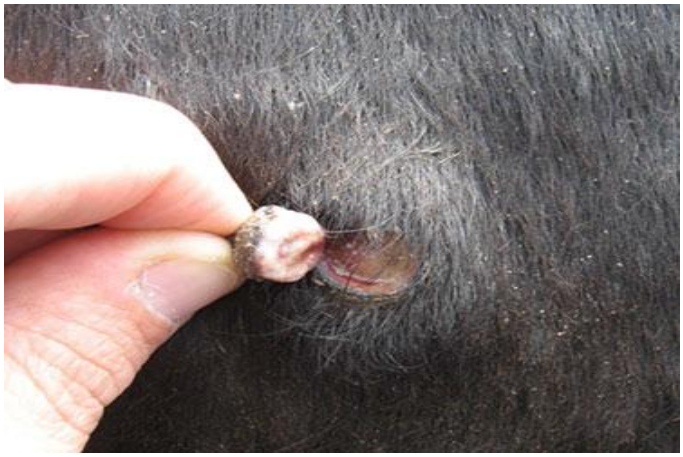

Fig. 3: Inverted conical zone of necrosis (a sitfasts lesion)
Source: Abutarbush (2013).

Pneumonia is a common bacterial complication and usually fatal disease. Absence of estrus cycle, painful genitalia that prevents bulls from serving, and abortion that frequently occurs in the early stages are due to prolonged fever (Ahmad & Zaher, 2008; Animal Health Australia, 2009). The most common sites of nodules are head, neck, perineum, genitalia, limb, and udder; they involve skin, cutaneous tissues, and sometimes the underlying part of the muscle. The severity of clinical signs depends on the strain of Capripoxvirus and breed of the host cattle and in case of experimental infection route of transmission and dose of the virus also has a determinant factor (OIE, 2010).

2.5.2. Pathological Lesions

Gross lesions

On autopsy, nodules may be found in the subcutaneous tissue, muscle fascia, and in muscles, which are grey-pink with serious necrotic cores, congestion, hemorrhage, and edema. The subcutis is infiltrated by red, watery fluid. Similar nodules may be scattered through the nasopharynx, trachea, bronchi, lungs, rumen, abomasum, renal cortex, testicles, and uterus (Animal Health Australia, 2009). Bronchopneumonia may be present and enlarged superficial lymph nodes are common. In severe cases, there is synovitis and tenosynovitis with fibrin in the synovial fluid (CFSPH, 2008).

Microscopic lesion

Histopathological sections show typical eosinophilic, intracytoplasmic pox inclusion bodies in cells of epithelioid, hair follicles, and cells of muscles and skin glands at an early stage of skin lesions (CFSPH, 2008; Animal Health Australia, 2009). Prominent lesions of vasculitic necrosis with cell debris and severe diffuse infiltration with inflammatory cells mainly neutrophils, have been seen in the superficial and deep dermis (Gari et al., 2011).

2.6. Diagnosis and Differential Diagnosis

2.6.1. Diagnosis

LSD can be diagnosed based on epidemiology, clinical signs, necropsy findings, and laboratory diagnosis. Clinically it is diagnosed by its pathognomonic nodular lesions like multiple skin nodules with circumscribed areas of erected hair, nodules around nostrils, turbinate, mouth, vulva, and prepuce that can

persist as hard lumps or become moist, necrotic and slough (Gari et al., 2011). Diagnosis is most commonly made by electron microscopic demonstration of typical capripox virions in full-thickness skin biopsies or scabs coupled with the clinical findings of a generalized nodular skin disease with enlarged superficial lymph nodes. Biopsy of lesions reveals a granulomatous reaction in the dermis and hypodermis. In the earlier acute stages, there are intracellular, eosinophilic inclusion bodies. (OIE, 2010).

Also, there is edema of the leg and swelling of the superficial lymph nodes (Tuppurinen & Oura, 2011). At necropsy, LSD can be diagnosed by looking at the nodules on the skin, in mouth, nostrils, vulva, and prepuce and, on mucous membranes, swelling of the superficial lymph nodes and systemic involved symptoms (CFSPH, 2008).

Common serological techniques used are Virus neutralization the indirect fluorescent antibody tests are commonly used. Rapid laboratory diagnoses are needed to confirm the disease. Laboratory diagnosis of LSD can be made by transmission electron microscopic isolation and identification of the agent, serological tests, routine histopathological examination, and immune histological staining (Tuppurainen, 2005; OIE, 2010). Isolation of a virus can be made from the collected biopsy or at post-mortem from skin nodules, lung lesions or lymph nodes within the first week of the occurrence of clinical signs, before the development of neutralizing antibodies (CFSPH, 2008; OIE, 2010). Primary cell cultures are bovine skin dermis and equine lung cells, but the growth of such viruses is slow and requires several passages (Tuppurainen, 2005).

Serological tests are used for retrospective confirmation of lumpy skin disease but they are much more time consuming to be used as primary diagnostic methods and the limited presence of detectable antibodies in serum (Vorster, 2008). Real-time PCR for the diagnosis of LSD has high sensitivity and good specificity and it is the most appropriate technique (OIE, 2010; Tuppurainen & Oura, 2011).

2.6.2. Differential Diagnosis

Lumpy skin disease can be suspected whenever clinical signs indicate towards persistent fever which may exceed 105.8°F, widespread skin nodules (lumps), enlarged peripheral lymph nodes, conjunctivitis, keratitis, corneal opacity, edema in the brisket and legs (Radostits et al., 2007). Histopathology can be an important tool to exclude viral, bacterial, or fungal causes of nodular development in clinical cases and characteristic cytopathic effects which are eosinophilic intracytoplasmic inclusion bodies in cases of LSD are well known (Brenner et al., 2006).

Severe LSD is differentiated based on its typical characteristics, but milder forms can be confused and misdiagnosed with numerous skin diseases of cattle which are to be considered in the differential diagnosis. According to Animal Health Australia (2009) and OIE (2010), the following diseases are considered important in the differential diagnosis of LSD: Bovine herpes mammalians (pseudo-lumpy skin disease) with lesions which are superficial (involving only the epidermis) and occurring predominantly on the cooler parts of the body such as teats and muzzle. However, there is no generalized disease. The second disease is *Hypoderma bovis* where the parasitic fly larvae of the parasite have a predilection to migrate to the skin at the back of the animal. The larvae cause a nodule with a small central hole through which the larvae exit from the body resulting in significant hide damage. The other disease is photosensitization which is characterized by dry, flaky, inflamed areas confined to the un-pigmented parts of the skin. Ringworm (dermatophytosis) is the other disease that is characterized by grayish lesions, raised, plaque-like, and often pruritic. The organism can be demonstrated with a silver stain. The last disease is streptotrichosis (dermatophilosis). The lesions are superficial, often moist, and appear as crusts or 0.5- to 2-cm diameter with an accumulation of keratinized material. The lesions are common in the skin of the neck, axillary region,

inguinal region, and perineum. The organism can be demonstrated by Giemsa staining.

2.7. Treatment

There is no specific antiviral treatment available for LSD infected cattle. Sick animals may be isolated from the herd and given supportive treatment consisting of a local wound dressing to discourage fly infestation and prevent secondary infections. The use of antibiotics or sulfonamides is recommended. (CFSPH, 2008; Tuppurainen & Oura, 2012).

2.8. Control and Prevention

2.8.1. Vaccination

Vaccination is the only effective method to control the disease in endemic countries like Ethiopia. The experience in the major parts of the country showed that the vaccination approach commonly chosen and is often that of ring vaccination around a local foci outbreak when it occurs. Animals that recover from virulent LSD infection generate lifelong immunity consisting both of a humoral and cell-mediated protective immunity (Kitching et al., 2003).

According to (Rushton, 2009), prevention of LSD can be carried out either by sanitary prophylaxis or medical prophylaxis. In the case of sanitary prophylaxis, import restrictions on livestock, carcasses, hides, skins, and semen can help to prevent the introduction of LSD into the disease-free countries or through medical prophylaxis like vaccination. Animals older than six months must be vaccinated against lumpy skin disease during spring. It is safe to vaccinate pregnant cows. All animals must be vaccinated once a year, preferably before the summer rain to ensure good protection. When vaccinating the animals during a disease outbreak, it is important to use one needle per animal so that the virus is not spread from sick to healthy animals (Gari et al., 2012).

Professional help and recommendation on vaccines must be carefully followed and practiced. Antibiotics also given to prevent the secondary bacterial complication as the defense mechanism of the body weakened which can prolong the complete recovery of the diseased animals (CSFPH, 2008). There are four commercially available vaccines for LSD, the Neethling strain LSDV (nLSDV), Herbivac, Lumpyvax, and Kenyan strain sheeppox virus (KS-1) (Gari et al., 2012).

2.8.2. In endemic areas

Control and prevention of LSD in endemic countries like Ethiopia rely mainly on annual vaccination of cattle older than six months because calves born to immunized cows will have passive immunity that persists for about six months (CSFPH, 2008). Four live attenuated strains of capripoxvirus are currently used as vaccines to control LSD; that includes the Kenyan sheep- and goat-pox strain (KS-1), the Yugoslavian RM 65 sheep-pox strain, the Romanian sheep-pox strain, and the South African Neethling LSDV strain. Two different vaccines have been widely and successfully used for the prevention of LSD in cattle populations in Africa. In southern Africa, the Neethling strain of LSD was passaged 50 times in tissue cultures of lamb kidney cells and then 20 times in embryonated eggs. It is produced in tissue culture and issued as a freeze-dried product. In Kenya, the strain of sheep and goat pox virus was passaged 16 times in pre-pubertal lamb testes or fetal muscle cell cultures. Mostly, the Neethling strain vaccine is used to vaccinate cattle in Africa (Brenner et al., 2006; OIE, 2010).

Because of antigenic homology and cross-protection between sheep pox, goat pox, and LSD viruses, any of these viruses can be used as a vaccine strain to protect cattle against LSDV (CSFPH, 2008). Animals that have recovered from natural infection or vaccinated with one of the strains have lifelong protection and are resistant to infection with any other strain and do not become carriers (Animal Health Australia, 2009). Protective immunity will develop from 10 to 21 days post-vaccination, and then require an annual booster dose (OIE, 2010).

2.8.3. In a new area

The risk of introduction of the disease into new areas is through the introduction of infected animals, animal products, and contaminated materials (Irons et al., 2005). If the occurrence of LSD is confirmed in new areas, before the spread of the disease to other areas extensively, quarantine of the area,

slaughtering of diseased animals and any in-contact animals and equipment must be cleaned and disinfected (Animal Health Australia, 2009).

Proper disposal of an infected animal and animal products to remove the source of infection, quarantine and movement controls of animals; products and other potentially infected items to prevent the spread of infection; control of insect vectors by insect repellent, insect-proof housing for animals and application of insecticides; tracing and surveillance to determine the source and extent of infection and ring vaccination are the major control and prevention strategies of the disease (Animal Health Australia, 2009; OIE, 2010).

3. ECONOMIC IMPACT OF LUMPY SKIN DISEASE

The office international des epizootics consider LSD as a list A of disease that has the potential for a rapid spread with the ability to cause serious economic loss (OIE, 2010). Morbidity and mortality of the disease vary considerably depending on the breed of cattle, the immunological status of the population, and insect vectors involved in the transmission. Morbidity rates generally varying between 1% and 20%. In a few outbreaks, it was reported to be more than 50% although the mortality rates are usually less than 10%. Cows in 1% to 7% of cases may abort. (Vorster & Mapham, 2008).

Lumpy skin disease is one of the economically significant diseases in Africa and Middle East countries that cause severe production loss in cattle. The economic importance of the disease is mainly due to having a high morbidity rate rather than mortality (Tuppurainen and Oura, 2011). The impact of LSD can be broadly divided into direct losses, i.e. the direct impact on animal health and productivity and indirect losses, which include mitigation or control efforts and lost export opportunities (EFSA AHAW Panel, 2015).

Direct losses include visible losses such as animal death and illness or stunting that result from disease or subsequent control methods. Invisible losses, on the other hand, are due to impacts of the disease, such as reduced productivity or changes in herd fertility, which result in the need to have a higher proportion of animals in a breeding group rather than in production. In resource-limited countries, the slaughter of infected and in-contact animals is usually seen as a waste of a valuable source of food and is not usually feasible. This kind of loss primarily affects the agriculture sector, mainly farmers and pastoralists (EFSA AHAW Panel, 2015).

Among indirect losses, forgone revenues should be considered, namely the indirect economic impact of animal diseases resulting from the ban on international trade of livestock, losses in consumer confidence, and negative effects on other sectors of the economy. The dynamics of supply and demand of animals and animal products can be disturbed by large outbreaks and their impact can be much larger than combining the impact observed on a single farm. Furthermore, the mitigation and control costs should be also considered, i.e. the costs of the drugs, vaccines, surveillance, and labor needed to carry out control measures. These costs may also have an impact on taxpayers because of the supplementary resource that may be needed for the implementation of a control program (EFSA AHAW Panel, 2015).

Major consequences of the disease are a retarded genetic improvement, inability of the animal to work, draught power and traction loss due to lameness, decreased milk production, abortion, infertility, chronic debility in beef cattle and loss of condition and damaged hides cause enormous economic losses (Babiuk et al., 2008). If LSD becomes endemic, continuing economic loss and poor productivity would occur due to stock losses, reduced production in cattle industries, ban on international livestock trade and costs of annual mortality, treatment, and vaccination. Lesions in the skin, subcutaneous tissue, and muscles of limbs, together with severe skin inflammation caused by secondary infection of lesions greatly reduce mobility (Murphy et al., 1999).

According to Gari et al. (2011), annual financial loss following an outbreak of LSD in Ethiopia is calculated as the sum of the values of the annual production losses due to morbidity and mortality and the costs for

treatment and vaccination. The formula is shown as follows:

C=Md+(B+M+Wop) +V+T

Where: C is the total financial costs

M is the milk production losses

B is the beef production losses

Wop is the work output losses

Md is the mortality losses

V is the vaccination costs

T is the treatment costs. Treatment cost represents the expenses incurred by farmers for medication.

Lumpy skin disease incidence interferes with normal herd dynamics, causing a reduction of surplus in the case of mortality, or a reduction of stock for the market in affected herds because of long term morbidity that can lower weight gain. The valuation of the draft power loss depends on the point in the crop season that an ox fell sick and on the corresponding demand for draft power during that specific season. The reduced work output of draft oxen due to LSD is an important loss for the mixed crop-livestock farming system. Morbidity of draft oxen leads to reduced crop production through a reduction in cultivation and lower yields due to inefficient land preparation and timing (Gari et al., 2011).

The financial impact of LSD between local zebu and HF/crossbreds shows that HF/crossbreds have far higher production losses in most parameters compared with local zebu cattle; the financial loss impact thus has a linear relationship with the incidence of the disease in each breed type (Gari et al., 2011). Milk production losses of up to 50% per lactation have been reported in infected herds showing that LSD infection is very important in high producing exotic breeds (Gari et al., 2011).

High economic losses were also incurred by feedlot owners for extra feed bought to assist sick animals during their recovery and the lengthened period required for fattening. Furthermore, animals that recovered were no longer fit for export purposes and were therefore sold at local markets at a lower price. Lastly, the survey found that animals that had recovered from LSD produce less milk and suffered a loss in draught power (Ayelet et al., 2014). Overall, LSD is considered as a disease of high economic pressure because of its ability to compromise food security through protein loss, draft power, reduced output of animal production, increase production costs due to increased costs of disease control, disrupt livestock and their product trade, the result of reduced milk yield, weight loss, abortion, infertility in cows, mastitis and infertility in lactating cows, infertility in bulls (Kumar, 2011).

Permanent damage to the skin and hide greatly affects the leather industry. It causes a ban on international trade of livestock and causes prolonged economic loss as it became endemic and brought serious stock loss (Animal Health Australia, 2009). As a consequence, the financial implication of this disease is greatly significant to the herd owner, consumers, and the industrial sectors which can process the livestock products. Report from Ethiopia (Figure 4) indicated that the financial loss estimated based on milk, beef, draught power, mortality, treatment, and vaccination costs in the individual head of zebu were lost 6.43 USD and for the HF 58 USD.

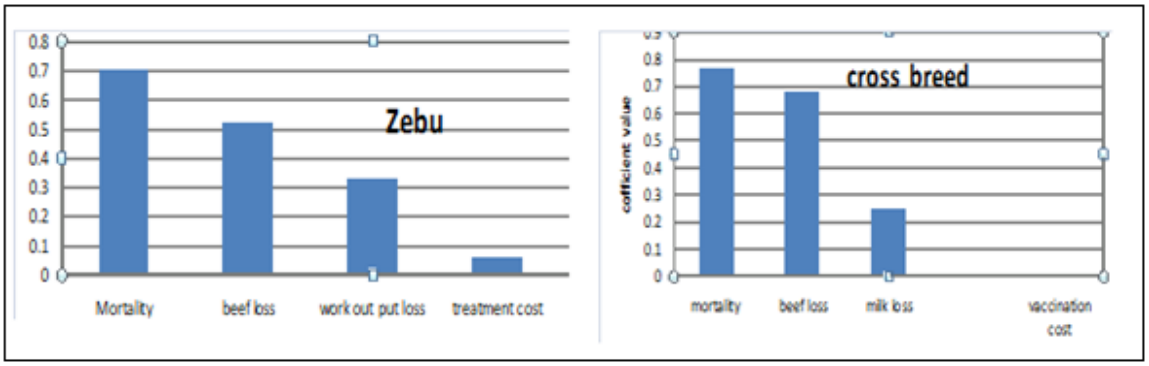

Fig. 4: Sensitivity analysis of the financial cost estimates for local zebu cattle and HF/crossbreds using the regression coefficient

Source: Gari et al. (2011).

4. STATUS OF LUMPY SKIN DISEASE IN ETHIOPIA

Ethiopia has the largest livestock population in Africa. Ethiopian economy is highly dependent on agriculture, which contributed about 48% of the gross domestic product, followed by 39% from the service sector and 13% from the industrial sector. However, livestock disease is one of the major livestock production constraints and lumpy skin disease is one of the most economically important diseases. It is one of the newly emerging diseases of cattle in Ethiopia (Gebreegziabhare, 2010).

In Ethiopia, LSD was first observed in 1981 in the north-western part of the country (Mebratu et al., 1984). However, it has now spread to almost all regions and agro-ecological zones of the nation with seroprevalence ranging from 23 to 31% at the animal level and 26-64% at herd level (Gari et al., 2010, 2012). The occurrence of LSD is associated with different agro-climatic conditions and the associated risk factors. There are three variables expected to influence the distribution and occurrence of LSD in Ethiopia: the effect of agro climate, communal grazing/watering management, and introduction of new animals. Moreover, Ethiopia has two major seasons of rainfall: a shorter rainy season that usually begins in mid-February and continues up to the end of April and the long rainy season (75%) starting mid-June and ending mid-September (Alemayehu, 2009).

Hence, this association might be attributed to the availability and abundance of effective mechanical vector insects, thus the temporal involvement between LSD occurrence and increase in the biting-fly population is a positively correlated and significant increase to the occurrence of the disease. Consequently, both biting-flies activity and disease outbreak frequencies begin to increase from April reaching a maximum in September which suggested that mechanical vector insects might play a major role in the disease outbreak of LSD (Abera et al., 2015).

Among the African countries Ethiopia is the major epidemic area for an outbreak of LSD and has also occurred in different regions and agro-ecological zones of the country (Gari et al, 2011). The disease has now spread to almost all regions and agro-ecological zones of the country. Because of the wide distribution of the disease and the size and structure of the cattle population in Ethiopia Major epidemic outbreak of LSD occurred in different years and regions of Ethiopia like Amhara and West Oromiya region in 2000/2001, Oromiya and SNNP region in 2003/2004 and Tigray, Amhara and Benishangul regions in 2006/2007. LSD likely is one of the most economically important livestock diseases in the country (Gari et al., 2011). It commonly occurs the outbreak at the end of summer and the beginning of the autumn season in the country (Gari et al., 2011). In Ethiopia, the highest frequency of LSD outbreaks have been reported between September and December, with the highest numbers in October and November (Figure 5) which is the end of the main rainy season in most parts of the midland and highland agro-ecological zones and the lowest number is reported in May (Ayelet et al., 2014).

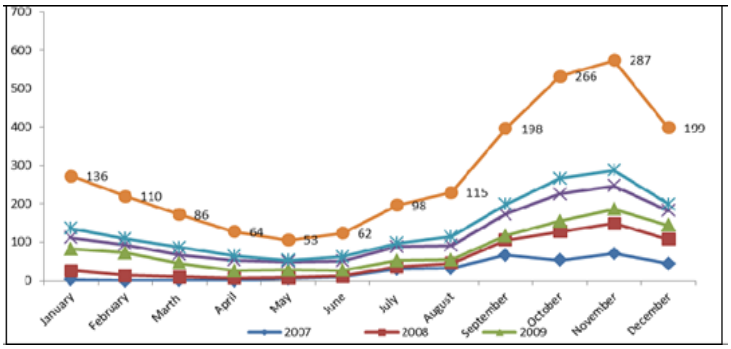

Fig. 5: Occurrence and seasonality of lumpy skin disease outbreaks in Ethiopia, 2007-2011
Source: Ayelet et al. (2014).

Among indigenous local zebu cattle Fogera breed located in the northwest of Ethiopia is reported to manifest severe clinical disease in the epizootic occurrence of LSD (OIE, 2008; Gari et al., 2011). A study in Ethiopia also shows that communal grazing, watering points, and movement of infected stock are associated with the occurrence of LSD. Lumpy skin disease is one of the reported diseases in Ethiopia which deserves outbreak notification to the National veterinary services (Gari et al., 2010).

According to Ayelet et al. (2014), analysis of retrospective data between January 2007 and December 2011 indicated that LSD is reported from all regions of the country except Harari and Dire Dawa. The majority of outbreaks are frequently reported from the midland agro-climate zone of Oromia, Amhara and the Southern Nations, Nationalities and People's Region, which is known to be favorable for the breeding of the blood-feeding insect vectors of LSD and has the highest population density of livestock in Ethiopia (Gari et al., 2010). Control of LSD in Ethiopia relies mainly on ring vaccination carried out at the onset of an LSD outbreak. In Ethiopia, both Kenyan SGPV and Neethling strain vaccines are produced at the National Veterinary Institute (NVI) and the Kenyan SGPV strains are widely used for all cattle, sheep, and goats. The vaccine protection lasts for a minimum of three years (Gari et al., 2011).

CONCLUSION AND RECOMMENDATIONS

Lumpy skin disease is a viral disease for which there is a limited option for the treatment of the disease. The disease is now endemic in most African and Middle Eastern countries. The disease is transmitted by vectors and the dynamics of the vectors in different agro-ecologies are not well established. The severity of clinical signs of LSD may be an acute or subacute form which depends on cattle breed, ages, and sex factors. The disease is more severe in young animals and cows in peak lactation. LSD can be diagnosed using appropriate serological and molecular techniques. Lumpy skin disease is a disease of high economic significance because of its ability to compromise food security through protein loss, draft power, reduced output of animal production, increase production costs due to increased costs of disease control, disrupt livestock and their product trade, the result of reduced milk yield, weight loss, abortion, infertility in cows, mastitis and infertility in lactating cows, infertility in bulls. It results in economic limitations to the global trade of live animals and animal products. To effectively control LSDV in endemic countries, there is limited understanding of the risk factors and ecology of different blood-feeding and biting arthropod species. The control of LSD can be achieved through vaccination, restriction of animal movement, and eradication of infected and exposed animals.

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