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# Epidemiology Of Lumpy Skin Disease In Ethiopia And Its Economic Impact

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## Abstract

Lumpy skin disease (LSD) is one of the most economically significant transboundary, emerging viral diseases that affect cattle of all ages and breeds. The disease was first seen in Zambia in 1929 and in Ethiopia in 1983 in the western part of the country southwest of Lake Tana. It is an economically devastating viral disease that causes several financial problems in livestock industries as a result of significant milk yield loss, infertility, abortion, and death. It is caused by a lumpy skin disease virus of capripoxvirus. The disease is characterized by fever, enlarged lymph nodes, firm, and circumscribed nodules in the skin, and ulcerative lesions. 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. It occurs in all agroclimatic conditions but is common, particularly in low-lying areas and along watercourses. It is transmitted by insect vectors among the cattle sharing similar grazing and watering areas and those congregate in the same barn. A good understanding of epidemiology, economic significance, and control mechanisms of the disease enables to design of suitable control measures. Effective control measure of the disease is achieved through mass vaccination through separation and culling of infected animals.

**Keywords:-** Epidemiology, Lumpy Skin Disease, Poxvirus, Vectors

## Introduction

Ethiopia has an estimated 53.4 million cattle (55.2% are female and 44.8% are males) distributed within the different agroecological zones (CSA, 2011); about 99% of cattle populations are of local Zebu breed. Genetically and geographically the main breed classifications in Ethiopia are Arsi, Fogera, Horro, Borana, Nuwer, Sheko, and Afar breeds, Barka/ Begaitbreed. The remaining 1% of exotic breeds is kept mainly for dairy production in and around urban areas. Livestock diseases are the major cause of economic losses to the peasant farmers and pastoralists in Ethiopia amounting to hundreds of millions of birr annually. Because livestock is the chief source of cash income for smallholders, up to 88% in the highland livestock-cropping system, diseases are an important cause of reduced productivity of meat and milk as well as draft, hides, and dung fuel (Rgbe, 2012).

Lumpy Skin Disease (LSD) also called (Pseudourticaria, Neethling Virus Disease, Exanthema Nodularis Bovis, Knopvelsiekte) is one of the most economically significant transboundary, emerging viral diseases that affect cattle of all ages and breeds. It's caused by a virus of the family *Poxviridae,* genus *Capripoxvirus,* and species *of* Lumpy Skin Disease Virus (*L*SDV), which is antigenically closely related to sheep and goat poxviruses (OIE, 2010).

Lumpy Skin Disease causes significant economic impacts as a result of reduced milk production, temporary or permanent sterility, deaths, beef loss, loss of draft animals' power, abortion, loss of condition, and damage to the hide. There is no antibiotic treatment for LSD, but supportive treatment

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can be available. Ring vaccination, quarantine, movement, and insect vectorscontrol, are the major control and prevention strategies for LSD (Tuppurainen and Oura, 2011).

Even though the disease is highly contagious and has a
great economic impact there is no sufficient and updated information regarding this disease.
Therefore, this seminar paper aims to review and provide updated information on epidemiological aspects of lumpy skin disease at the national level in Ethiopia and its economic impacts of lumpy skin disease.

## 2. LITERATURE REVIEW

### 2.1. Historical Background of Lumpy Skin Disease

Lumpy skin disease was first seen in Zambia in 1929, and at that time it was considered as it was caused by either plant poisoning or an allergic response to insect bite. Then 15 years later it was observed from Zambia into Botswana and South Africa, where it affected over eight million cattle causing major economic loss. In 1957 it entered Kenya, associated with an outbreak of sheep pox. In 1970, LSD spread north into Sudan, by 1974 it had spread west as far as Nigeria, and in 1977 was reported from 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 in affected cattle of 20%. In subsequent years, Bahrain, Kuwait, Oman, Yemen, and Israel were reported. 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; OIE, 2014).

### 2.2. Definition

LSD is an acute to chronic viral disease of cattle that can cause mild to severe symptoms including fever, nodules in the skin, in the mucous membranes and the internal organs, edema of the leg and brisket, lymphadenitis, and sometimes death. It results in economic losses due to decreased milk production, traction power loss, weight loss, poor growth, abortion, infertility, and skin damage (OIE, 2010).

### 2.3. Etiology

LSD is caused by *the Lumpy Skin Disease virus* (LSDV), which is a member of the family *Poxviridae*, subfamily *Chordopoxvirinae*, genus C*apri poxvirus*, the prototype strain *Neethling Virus*.LSDV is a pleomorphic, enveloped, brick- or oval-shaped dsDNA virus with a molecular size of 350×300nm and a molecular weight of 73 to 91 (Kilodalton) KDa. An LSDV genome sequence is 145 to 152(Kara *et al.*, 2003).

### 2.4. Epidemiology

Lumpy skin disease is an important, economically devastating, notifiable disease that causes production loss in cattle due to generalized malaises and chronic debility (Tuppurainen and Oura, 2011). A good understanding of epidemiological aspects of LSD related to pathogen, host, and environment might aid in control and prevention mechanisms. Particular emphasis should be given to the exposure of hosts to pathogens in suitable environments that facilitate the transmission and

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distribution of the disease. LSD is more prevalent during the wet summer and autumn months and occurs particularly in low-lying areas and along watercourses (OIE, 2010).

2.4.1. Geographic distribution

In Ethiopia, LSD was first observed in 1983 in the western part of the country southwest of Lake Tana (Gari, 2011). After its first appearance, an explosive sudden epidemic spread from the north through the central to the southern part of the country. The national disease report showed LSD has spread virtually to all the regions in the country and different agro-climatic zones. Because of the wide distribution of the disease and the size and structure of the cattle population in Ethiopia, LSD is one of the most economically important livestock diseases in the country (Gari *et al.*, 2010, 2012).

A recent study across different agroecological zones in Ethiopia showed an overall observed LSD prevalence of 8.1% and a mortality of 2.12%. The case fatality is estimated to be 2% (Gari *et al*., 2010). The highest frequency of LSD outbreaks in the country have been reported between September and December, with the highest numbers in October and November; 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) (Figure 1). Among indigenous local zebu cattle Fogera breed located in the northwest of Ethiopia is reported to manifest severe clinical disease in 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 (Getachew *et al*., 2010).

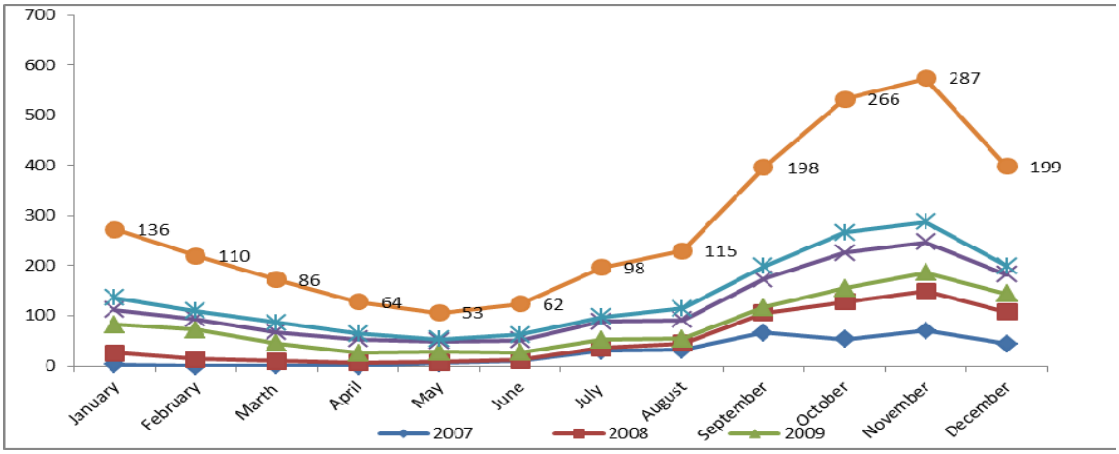


**Figure 1**: Occurrence and seasonality of lumpy skin disease outbreaks in Ethiopia, 2007–2011 (Ayelet *et al*., 2014).

According to Gari *et al.*, (2010), LSD is one of the reported diseases in Ethiopia that deserves outbreak notification to the National Veterinary Services. 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 (Figure 2). 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-

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feeding insect vectors of LSD and has the highest population density of livestock in Ethiopia (Gari *et al.*, 2010).

**No- of LSD outbreak**

**Illubabor**

**Jimma**

**Southwest Shoa Arsi**

**Figure 2**: Map of Ethiopia showing the distribution of lumpy skin disease outbreaks, 2007–2011. The highest number of outbreaks from the black shaded to the lowest number in white-shaded areas (Ayelet *et al*., 2014)

In Ethiopia, limited work has been done on this disease so far and few works have been reported on risk factors assessments, epidemiological aspects, seroprevalence, and financial impacts (Getachew *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 strain is widely used for all cattle, sheep, and goats. The vaccine protection lasts for a minimum of three years (Gari *et al.*, 2011).

2.4.2. Species of animal affected

LSD is primarily a disease of all cattle, particularly thin-skinned European breeds, who are susceptible (Brenner, 2006). CaPVs 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 sheep pox, or goat poxvirus (CFSPH, 2008; Tuppurainen and Oura, 2012).

Natural cases have not been seen in an impala and a giraffe but, demonstrated in both of them after experimentally inoculation with LSDV. The absence of a reservoir host for LSD virus might lead us to the assumption that infection might persist in the endemic areas at a low level as unapparent or mild form in the cattle population(OIE, 2008).

2.4.3. Source of infection

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

2.4.4. Mechanism of transmission

*2.4.4.1. Direct transmission*

Direct transmission can occur when the animals share the same feeding and drinking trough due to contamination by nasal and salivary discharges from infected animals or ingestion of the already contaminated food or by iatrogenic agents (Lefèvre and Gourreau, 2010). Suckling calves may be

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infected through infected milk. 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 were found infected by LSDV, and 93 % of cows suffered from ovarian inactivity and showed no signs of estrus(EFSA, 2015). There is an assumption that the virus is also secreted in vaginal secretions. Generally, transmission of the virus by contact is inefficient and field evidence reported that the disease is not contagious (Salib and Osman, 2011).

#### *2.4.4.2. Role of vectors*

The transmission of LSDV occurs mechanically by blood-feeding biting arthropod vectors including hard ticks, biting flies, and mosquitoes (Chihota *et al.*, 2001; Getachew *et al*., 2010; 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 mouth parts 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 2-6 days post-feeding from infected cattle and transfers this to susceptible cattle by female mosquito, *Aedesegypti* 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 transstadial and transovarian transmission of the LSD virus by *Boophilusdecoloratus* and mechanical transmission by *Repicephalusappendiculatus* and *Ambylomahebraeum* (Tuppurainen *et al*., 2011).

Mosquitoes (female *Aedesegypti and Culexquinquefasciatus)* and other flies such as *tabanids* (horse flies), biting midges (*Culicoidesnubeculosus*), 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 flies (Hippoboscidae), and blowflies (Calliphoridae) are also very commonly associated with sucking of infected lachrymal, nasal, or other secretions and transferring the virus to another susceptible animal. Vermin, predators, and wild birds might also act as mechanical carriers of the virus (AUSVETPLAN, 2009).

Epidemiological evidence suggests outbreaks of LSD are highly associated with the prevalence of high insect vector populations and with the upcoming rainy season. Epidemics of LSD are associated with rainy seasons, river basins, and ponds during which cattle grazed and humid areas that are conducive to insect multiplication (OIE, 2010).

### 2.4.5. Risk factors

#### *2.4.5.1. Host risk factors*

Lumpy skin disease 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 *Bosindicus* (Radostits *et al.*, 2007). Among *Bos Taurus*, fine-skinned, high-producing dairy Channel Island breeds are highly susceptible to LSDV (EFSA, 2015). Lactating cows appear to be

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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 and Oura, 2011). Whereas indigenous breeds such as zebu and zebu hybrids are likely to have some natural resistance against the virus (Gari *et al*., 2011).

It is not known what genetic factors influence the disease severity (Babiuk *et al*. 2008). High ambient temperatures, farming practices, and cows that produce high milk yields could be deemed to stress the animals and contribute to the severity of the disease in Holstein–Friesian cattle (Tageldin *et al*., 2014).

Young animals are severely affected and clinical symptoms are rapid to appear. However 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 stress factors of exhaustion and fatigue rather than to a biological reason. The majority of male animals are draft 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). Generally, the clinical severity of the disease depends on susceptibility, immunological status, and age of the host population and the dose and route of virus inoculation (CFSPH, 2008).

*2.4.5.2. Pathogen risk factors*

LSDV is one of the species of capripoxviruses that is resistant to different chemical and physical agents (Murphy *et al.*, 1999). Capri poxviruses have lipid-containing envelopes and are 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. LSDV is susceptible to temperatures of 55 °C/two hours, 65 °C/30 minutes, alkaline or acid pH.No significant reduction in titer when held at pH 6.6–8.6 for five days at 37 °C(OIE, 2014b).

LSD 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 for 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 to the environment and can remain viable for long periods on or off the animal host. They may persist for up to 6 months in a suitable environment, such as shaded animal pens. 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(AUSVETPLAN, 2009).

*2.4.5.3. Environmental risk factors*

Environmental determinants play a great role in the epidemiology of lumpy skin disease. It has a major impact on the agent, host, and vectors as well as the interaction between them. These predisposing

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factors have a great role in the maintenance of arthropod vectors 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, and other reasons for cattle movement from place to place and the presence of water bodies are some of the potential risk factors of LSD (Tuppurainen and Oura, 2011).

LSD is associated with an increased number of insects which are 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 zones and along watercourses (OIE, 2010). The warm and humid climate in midland and lowland agro-climates has been considered a more favorable environment for the occurrence of large populations of biting flies than the cool temperature in the highlands (Tuppurainen *et al*., 2012).

2.4.6. 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 the average ranges from 3% to 85% (CFSPH, 2008, Tuppurainen, *et al.*, 2012). However, it can reach as high as 100% in natural outbreaks while the mortality rate rarely exceeds 5% but may sometimes reach 40% (Babiuk *et al*., 2008; Irons *et al*., 2005).

2.5. Pathogenesis

LSD is developed by entry of infectious LSDVthroughskin 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 specific cells such as endothelial cells of lymphatic and blood vessel walls with the development of inflammatory nodules on the skin (Vorster, 2008).

LSD is a 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 (Radostits *et al.*, 2007).

LSD 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 and 14 days in experimentally inoculated animals and 2–4 weeks in naturally infected animals (OIE, 2010).

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2.6. Clinical Sign

The course of lumpy skin disease may be acute, sub-acute, and chronic. The virus causes from unapparent infection to severe clinical symptoms and those animals which develop clinical disease may have a biphasic febrile reaction. The major visible clinical signs are; a fever of 40-41.5$^{o}$C which may last 6-72 hours, lachyrimation, increased nasal and pharyngeal secretion, loss of appetite, reduced milk production, some depression and movement reluctance, nodules in the skin, mucous membrane and internal organs and swelling of superficial lymph nodes (Figure 3). 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 and Oura, 2011).

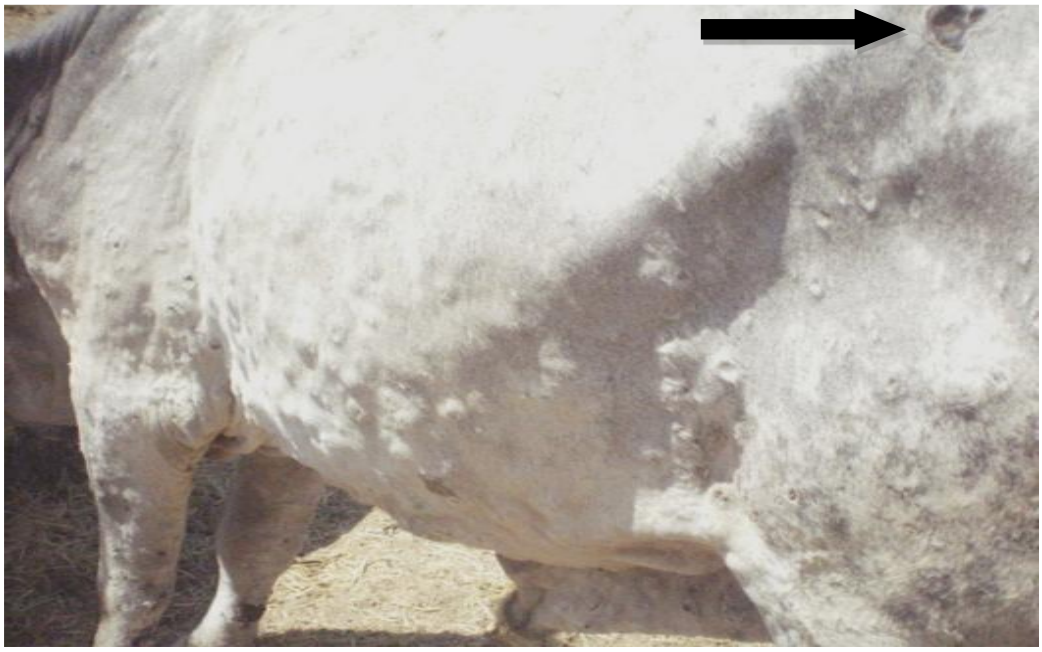

**Figure 3:** Large circumscribed skin nodules in LSD-affected cattle and deep eroded lesions after removal of the skin nodule (Rgbe, 2012).

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

Pneumonia is a common bacterial complication and usually fatal disease. The absence of an estrus cycle, painful genitalia that prevents bulls from serving, and abortion that are frequent in the early stages are due to prolonged fever (Ahmed and Zaher, 2008; AUSVETPLAN, 2009).

The most common sites of nodules are the head, neck, perineum, genitalia, limb, and udder; involve skin, cutaneous tissues, and sometimes underlying parts of the muscle. The severity of clinical signs depends on the strain of Capripoxvirus and the breed of the host cattle and in case of experimental infection route of transmission and dose of the virus also have a determinant factor (OIE, 2010).

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2.7. Pathological Lesion

2.7.1. Gross lesions

On autopsy, nodules may be found in the subcutaneous tissue, muscle fascia, and muscles, which are grey-pink with caseous 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 (AUSVETPLAN, 2009). Bronchopneumonia may be present and enlarged superficial lymph nodes are common. In severe cases, there is synovitis and tendosynovitis with fibrin in the synovial fluid (CFSPH, 2008).

2.7.2. 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; AUSVETPLAN, 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).

**2.8. Diagnosis**

According to OIE (2010), LSD can be diagnosed based on epidemiology, clinical signs, necropsy findings, and laboratory diagnosis. Clinically it is diagnosed by its pathognomic 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). Also, there is edema of the leg and swelling of the superficial lymph nodes (Tuppurinen and Oura, 2011).

At necropsy, LSD can be diagnosed by looking at the nodules on the skin, in the mouth, nostrils, vulva, and prepuce and, on mucous membranes, swelling of the superficial lymph nodes and systemic involved symptoms (CFSPH, 2008). 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 the virus can be made from 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 (OIE, 2010; CFSPH, 2008). 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 have 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 and Oura, 2011).

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2.8.1. Differential diagnosis

Lumpy skin disease can be suspected whenever clinical signs indicate 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). According to AUSVETPLAN (2009) and OIE(2010), some of the diseases of cattle that can be mentioned as differential diagnoses for LSD are Bovine herpes mammillitis, Hypodermal bovis, hotosensitization, Ringworm and Streptotrichosis.

## 2.9. Control, Prevention and Eradication

2.9.1. 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 flies from worrying and prevent secondary infections (CFSPH, 2008, Tuppurainen, and Oura, 2012).

2.9.2. Control and prevention

*2.9.2.1. In endemic areas*

Control and prevention of LSD in endemic countries like Ethiopia relies mainly on the vaccination of cattle over six months every year. Because calves born to immunized cows will have passive immunity that persists for about six months. The experience in the major parts of the country showed that the vaccination approach is commonly chosen and is often that of ring vaccination around a local foci outbreak when it occurs (Gari*et al.*, 2011).

Four live attenuated strains of capripoxvirus are currently used as vaccines to control LSD; these include 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 passed 16 times in pre-pubertal lamb testes or fetal muscle cell cultures. Mostly 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. Animals that have recovered from natural infection or are vaccinated with one of the strains have lifelong protection and are resistant to infection with any other strain and do not become carriers (AUSVETPLAN, 2009). Protective immunity will develop from 10 to 21 days post vaccination, and then require an annual booster dose (OIE, 2010).

*2.9.2.2.Innew areas*

Risks of introduction of the disease into the new areas are the introduction of infected animals animal products and contaminated materials (Irons *et al.*, 2005). If the occurrence of LSD is confirmed in new

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areas, before the spread of the disease to other areas extensively, quarantine of the area, slaughtering of the diseased and in contact animals and contacted equipment must be cleaned and disinfected (AUSVETPLAN, 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 to minimize mechanical transmission of the virus, 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 LSD (AUSVETPLAN, 2009; OIE, 2010).

2.11. Economic Importance

Lumpy skin disease is one of the economically significant diseases in African and Middle Eastern 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 lumpy skin disease can broadly be 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, 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, include less immediate impacts of animal 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 stakeholders of the agriculture sector, for example, farmers (EFSA, 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 impacts observed on single farms. 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 resources that may be needed for the implementation of the control program(EFSA, 2015).

Major consequences of the disease are regarded as genetic improvement, the 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 became endemic, continuing economic loss and poor productivity would occur due to stock losses, reduced production in cattle industries, a ban on international trade of livestock, and costs of

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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 LSD outbreak 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. LSD 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. The 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).

According to Gari *et al*., (2011), the financial loss impact 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. Milk production losses of up to 50% per lactation have been reported in infected herds. This shows that LSD infection is very important in high-producing exotic breeds.

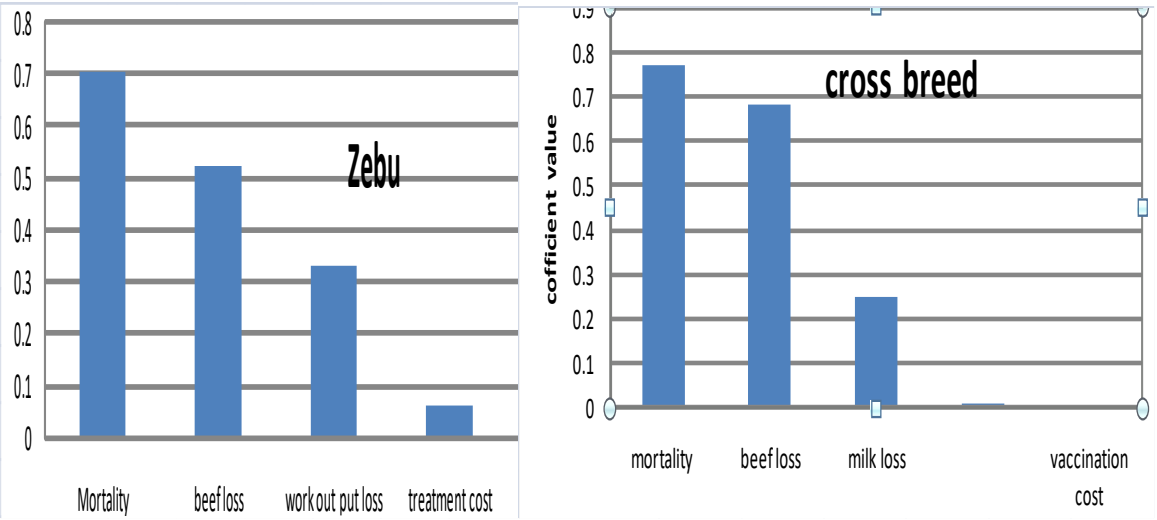


**Figure-4:** Sensitivity analysis of the financial cost estimates for local zebu cattle and HF/crossbreds using regression coefficient (Gari *et al*., 2011).

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. Further, 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 produced less milk and suffered a loss in draught power (Ayelet *et al*., 2014).

Overall, LSD is considered a disease of high economic pressure because of its ability to compromise food security through protein loss, draft power, reduced output of animal production, increased

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production costs due to increased costs of disease control, disruption of 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, 2009). Permanent damage to the skin and hide greatly affects the leather industry. It caused a ban on the international trade of livestock and caused prolonged economic loss as it became endemic and brought serious stock loss (AUSVETPLAN, 2009; Getachew *et al*., 2010).

**3. CONCLUSIONS AND RECOMMENDATIONS**

Lumpy skin disease is one of the most economically significant transboundary, emerging viral diseases of domestic cattle caused by viruses of the genus *Capripoxvirus*. It has significant economic importance in animals, due to chronic debility, reduction in milk production and weight, damaged hides, abortion, and death. LSD is now endemic in most regions of Ethiopia.LSDV transmission among cattle is by the mechanical haematophagus arthropod vectors. The importance of different mechanical vectors in the transmission of LSDV is likely to vary in different geographical regions, depending on the environment, temperature, humidity, and abundance of the vectors. LSD is common during the wet season which is at the end of summer and beginning of autumn. The control of LSD can be achieved through vaccination, restriction of animal movement, and eradication of infected and exposed animals. Based upon the above conclusion the following recommendations are forwarded:

- ✓ Better control of illegal livestock and animal product movements should be considered.
- ✓ The government and/or NGO should facilitate awareness creation and training for farmers and veterinary staff in recognizing the disease under field conditions.
- ✓ If LSD entered a disease-free country, rapid detection and prompt culling of infected herds, carcasses, and ring vaccination should be considered.
- ✓ To effectively control LSDV in endemic countries, a comprehensive understanding of the ecology of different blood-feeding and biting arthropod species in the cattle farming setting is important.
- ✓ Animals suspected of LSD should be isolated and the farm should be quarantined until a definitive diagnosis is determined.

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