Academia Journal of Microbiology Research 11(1): 001-011, May 2023
DOI: 10.15413/ajmr.2023.0004
ISSN 2315-7771

Academia Publishing

Research Paper

Review on the epidemiology of lumpy skin disease of cattle and associated risk factors in Ethiopia

Accepted 30th May 2023

Samson Terefe Kassa

Addis Ababa University College of Veterinary Medicine and Agriculture P. O. Box 34, Bishoftu, Ethiopia.

E-mail: samsonterefe1@gmail.com.

ABSTRACT

Lumpy skin disease (LSD) is an economically important disease of cattle and can produce a chronic debility in infected cattle. Lesions in the mouth, pharynx and respiratory tract commonly occur, resulting in a rapid deterioration in condition and sometimes severe emaciation, which can persist for months. Severe and permanent damage to hides results from the skin lesions. Serious economic losses can follow outbreaks that have a high morbidity. Morbidity of LSD varies greatly which ranges from 3 to 85% in different epizootic situations and even 100% in rare cases. The mortality rate of this disease is mostly considered not very high from 1 to 2% and sometime up to 10%. However, mortality may reach up to 40% in severe outbreak cases. There is no specific antiviral treatment available for LSD-infected cattle. Two vaccines, however, Neethling and Kenya sheep and goat pox virus, have been widely and successfully used in Africa. LSD is an endemic disease in Ethiopia, where it first appeared in 1983 in the southwest of Lake Tana. At present, the disease has spread widely across the country regardless of the altitude as well as husbandry systems of the location and farming system, respectively. All cattle breeds, both sexes and all ages groups are susceptible. The disease is distributed almost in all regions and is regarded as one of the most economically important livestock diseases in the country.

Key words: Cattle, epidemiology, Ethiopia, LSD, risk factors

INTRODUCTION

In Ethiopia, livestock roles are irreplaceable in the livelihood of rural communities and country's economy that put cattle production at the center in its impact (FAO, 2019). These resources of livestock are backed by the fact that the country is leading in cattle population from Africa and fifth in the world (World Cattle Inventory (WCI), 2015). Despite its large population size, the contributions of livestock production to agriculture and the overall economy of the country are low. This is associated with a number of complex and inter-related factors such as widespread diseases, limited genetic potential and husbandry standard (Nagassa et al., 2011). Livestock diseases are the major production constraints in Ethiopia in addition to poor nutrition, low genetic potential of indigenous livestock, lack of marketing infrastructure and water shortages (Gebre Egziabhare, 2010). Lumpy skin disease (LSD) is one of the most economically important viral diseases listed as notifiable trans-boundary animal diseases by the World Organization for Animal Health (OIE) and the second significantly important cattle disease in Ethiopia (Gelaye et al., 2015; OIE, 2017).

Lumpy skin disease (LSD) is an infectious disease of cattle, caused by a Lumpy Skin Disease Virus (LSDV), which is found in the family Poxviridae, genus Capripoxvirus. It is closely related antigenically to sheep and goat poxvirus. LSD Causes considerable economic losses due to emaciation, damage to hides, infertility, mastitis, loss of milk production, and mortality of up to 20% (Al- Salihi, 2014). Lumpy skin disease (LSD) is an emerging viral transboundary disease which can spread beyond the

outbreak area and become epidemic (Jordi et al., 2018; **K, 2014; K et al., 2021**).

The first reported cases of LSD in the world was in Africa, Northern Rhodesia (now Zambia) in 1929 and spread to the rest of African countries as well as other parts of the world by different means. For example, introduction of the disease in to Middle East in 1991 was correlated with live animal importation from affected areas (Brenner et al., 2006; El-Kholy et al., 2008). However, the way of LSD introduction in to Ethiopia is unknown, it was first observed in the North-western part of the country (Southwest of Lake Tana) in 1983 (Mebratu et al., 1984). It has been spread to almost all the regions and agro-ecological zones of the country. This is related to the existence of LSDV host, cattle in almost all agro-ecological parts of the country that mainly rely on cattle production. Due to the fact that almost all farmers depend on their cattle population as a means to accomplish their livelihood. These include food and agricultural processes, with a higher impact on country's economy as far as livestock disease is concerned (Gari et al., 2010).

Lumpy skin disease also called Pseudo-urticaria, Neethling virus disease, exanthema nodularis bovis, and knopvelsiekt. LSD is an acute to chronic viral disease of cattle characterized by skin nodules that may have inverted conical necrosis (sitfast) with lymphadenitis accompanied by a persistent fever (Grooms, 2005; James, 2004; Alaa et al., 2008; Vorster and Mapham, 2008). CaPV infections are generally host specific and not reported on CaPV infecting all three species: sheep, goats and cattle (Bhanuprakash et al., 2010; Tuppurainen et al., 2014). They have also specific geographic distributions in which diseases of GTP and SPP are prevalent in Africa above the equator, Asia, the Middle East, and occasional outbreaks occur in regions of Europe surrounding the Middle East. In contrast, LSD is endemic in Africa and outbreaks have been occurred in the Middle East countries surrounding Egypt and in some parts of Europe like Greece (Bhanuprakash et al., 2011; Tuppurainen et al., 2015; Babuik et al., 2008).

Experimental and field evidence indicates that LSDV is inefficiently transmitted between animals through direct contact. LSD is a vector-borne disease transmitted by different biting and blood-feeding arthropods. Circulation of LSDV is often, but not necessarily, associated with warm and humid weather conditions and with a high density of biting insects (Ali et al., 2012). The disease is more prevalent in low-lying areas and along water courses. Fever is the initial sign that is followed within two days by the development of nodules on the skin and mucous membranes (Brenner et al., 2006; Tuppurainen and Oura, 2012). A presumptive diagnosis of the disease can be made based on highly characteristic clinical signs of LSD. However, mild and asymptomatic disease may be difficult to diagnose and rapid laboratory methods are needed to confirm the diagnosis. Different molecular tests are the preferred diagnostic tools (Kumar, 2011).

The mode of transmission of LSD has not been described fully but the biting flies and some tick species are probably the most important method of transmission of LSD and therefore, quarantine and movement control is usually not very effective (Abdulqa et al., 2016). The disease is usually more prevalent during wet summer and autumn months, particularly in low-land and mid land areas and around water courses, but outbreaks may also occur during the dry season and winter months (Coezer and Tuppurainen, 2004; Gari et al., 2010). Consequently continuous surveillance on the status of the disease and genetic information on circulating field viruses is mandatory in order to take effective measures for the control and thereby eradication of the disease in the country (Body et al., 2011).

The disease is now the problem of almost all the regions and agro ecological zones of Ethiopia. Major outbreaks of LSD have been occurred in different regions of Ethiopia (Ayelet et al., 2013). LSD is an OIE listed disease because of considerable financial losses and in Ethiopia due to the endemic nature of LSD; the country is facing serious difficulties in exporting live cattle and their products. In addition, this situation contributes a negative impact on the national economic growth through the loss of meat and milk production and poor quality of skin and hides (Gelaye et al., 2015).

HISTORY OF LSD

The first description of the clinical signs of LSD was reported in 1929 in Zambia (Morris 1931). Same clinical signs occurred in Botswana, Zimbabwe and the Republic of South Africa between 1943 and 1945, where the infectious nature of the disease was recognized in these outbreaks. In South Africa, LSD occurred as a panzootic, which affected eight million cattle. In1957, LSD was identified in East Africa in Kenya. In 1972, the disease was reported in Sudan and West Africa in 1974. At present, LSD occurs in most of African country (Tuppurainen and Oura, 2012). Lumpy skin disease was limited to African continent until 1989 but later it moved outside Africa to Madagascar and the Middle East and caused a serious economic loss to the livestock production. Prior to 2012, only sporadic LSDV outbreaks were reported in the Middle East region (Tuppurainen and Oura, 2011).

In the Middle East, the outbreaks of the LSD were reported in Oman in 1984 and 2009 (Kumar, 2011; Tageldin, 2014).

Kuwait in 1986 and 1991, Egypt in 1988 and 2006 (Fayez and Ahmed, 2011; Ali and Amina, 2013), Israel in 1989 and 2006 (APHIS, 2006), Bahrain in 1993 and 2002-2003, Yemen, United Arab Emirates in 2000 and the West Bank also reported LSD invasion (Kumar, 2011; Sherrylin et al., 2013). LSD is exotic to the European Union (EU), but incursions of LSD have occurred in EU neighboring areas (EFSA Journal, 2015).

ETIOLOGY

LSDV is grouped under the family of *poxviridae*. The family *Poxviridae* is subdivided into two subfamilies: *Chordopoxvirinae* (poxviruses of vertebrates) and *Entomopoxvirinae (*poxviruses of insects). Lumpy skin disease virus (LSDV) belongs to the genus Capripoxvirus and the subfamily Chordopoxvirinae. There is only one serotype of LSDV which is prototype strain of LSDV is the Neethling virus and it is closely related antigenetically to sheep and goat poxvirus and can be distinguished by routine virus neutralization or other serological tests. The LSDV primarily affects cattle but can affect sheep and goats, experimentally. Lumpy skin disease virus will grow in tissue culture of bovine, ovine or caprine origin; although maximum yield is obtained using lamb testis cells (Gelaye et al., 2015). The members of this family are among the largest of all viruses. It is an envelope, linear ovoid shape with a molecular brick shaped or ovoid virions measuring 220-450 nanometer (nm) by 140-266nm. LSDV has ds DNA genome of about 151kb (Yehuda et al., 2012).

EPIDEMIOLOGY OF LSD

Occurrence of the disease

LSD is an endemic disease of most African countries particularly in those of the sub-Saharan region. After 2012 it has spread rapidly through the Middle East, south-east Europe, the Balkans, Caucasus, Russia and Kazakhstan (OIE, 2017; Coezer and Tuppurainen, 2004). Field outbreaks typically involve a severe, generalized infection with high rates of morbidity and mortality, though in some cases there may be few affected animals and few or no deaths reported. In general, outbreaks are more severe when the infection is first introduced to a region and then tend to subside, most likely due to the spread of widespread immunity. The morbidity rate for animal diseases reaches 80%, but is almost 20% in endemic areas (Radostits et al., 2006).

Sources of the virus

Capripox viruses are highly resistant viruses to physical and chemical action. They can survive in scab or tissue fragments for very long periods of time (Davies, 1991). It can be recovered from skin nodules kept at –80°C for ten years and from infected tissue culture fluid stored at 4 °C for about six months (Coezer and Tuppurainen, 2004). LSDV can be isolated for up to 35 days or longer from skin nodules, scabs and crusts which are known to contain relatively high amounts of virus. It can also be isolated from blood, saliva, ocular and nasal discharges, and semen (Irons et al., 2005) of infected animals. LSDV is found in the blood intermittently from approximately 7 to 21 days post-infection at lower levels than present in skin nodules. Viral shedding in semen can be prolonged and it has been isolated from the semen of an experimentally infected bull after 42 days (OIE, 2017).

Hosts and susceptibility

Domestic cattle and Asian water buffalo are the animals affected by LSDV naturally during field outbreaks (El-*Nahas* et al., 2011*;* Al-Salihi, 2014). Some strains may replicate in sheep and goats but to date no epidemiological studies have evidenced small ruminants as reservoirs for the virus (Tuppurainen, 2017).Very little is known about the susceptibility of wild ruminants to LSDV. The susceptibility of host animals mostly depends on immune status, age and breed rather than the virulence of the virus. European cattle breeds are generally more susceptible than indigenous African and Asian breeds (Tageldin et al., 2014).

Lumpy skin disease is host-specific, causing natural infection in cattle and Asian water buffalo (*Bubalus bubalis*) though the morbidity rate is significantly lower in buffalo (1.6%) than in cattle (30.8%) (El-Nahas et al., 2011). Both cattle and Buffalo are susceptible regardless of their breed and age differences. However, some specific breeds such as *Bos taurus* is particularly more susceptible to clinical disease than zebu cattle. Furthermore, fine skinned Channel Island breeds develop more severe disease among *Bos taurus,* (OIE, 2010). Production status is also incriminated to have an impact on susceptibility. Accordingly, lactating cows appeared to be severely affected with a consequence in a sharp drop of milk production. Most of these effects are related to high fever caused by LSDV infection and secondary bacterial mastitis. Even though mixed herds of cattle, sheep and goats are common, no epidemiological evidence on the role of small ruminants as a reservoir for LSDV has been reported yet (Tuppurainen et al., 2017b).

Experimental infection done on impala (*Aepyceros melampus*) and giraffe (*Giraffa camelopardalis*) result in clinical signs of LSD. Additionally, the disease has also been reported in an Arabian oryx (*Oryx leucoryx*) and springbok (*Antidorcas marsupialis*). However, the susceptibility of wild ruminants or their possible role in the epidemiology of LSD is not known with this disease also unable to affect humans (Tuppurainen et al., 2017b).

Transmission

Studies have shown that the main route of transmission for LSD is through vectors whereas transmission ways like direct contact are not effective (Magori-Cohen et al., 2012). Stomoxys, Musca confiscate and *Aedes egypti* mosquitos and the three common African hard tick species, namely, *Rhipicephalus appendiculatus*, *Amblyomma hebraeum* and

the African blue tick *Rhipicephalus (Boophilus) decoloratus*, were reported to have a great role in the transmission of LSD (Chihota et al., 2003). It is possible to transmit LSDV by *Aeidesa egypti* to susceptible animals without the subsequent development of clinical disease in the animals. Transstadial and transovarial transmission of LSDV by *Boophilus decoloratus* ticks and mechanical or intrastadial transmission by *Rhipicephalus appendiculatus* and *Amblyomma hebraeum* ticks has been shown (Tuppurainen et al., 2011). Studies also showed that the disease can also transmit when common drinking troughs are used, thus confirming the suspicion that infected saliva might contribute towards the spread of the disease. The disease is transmissible to young calves through infected milk (Coezer and Tuppurainen, 2004).

Cattle movements between farms, regions or countries can play a significant role in the introduction of LSD. In this regard, the first outbreak reported by Israel in 1989 was associated with cattle introduction from Egypt. Additionally, vectors were also incriminated to carry the virus supported by winds to even go that further. There are different vectors species that plays a greater role in LSD transmissions. These vectors are the common stable fly (*Stomoxys calcitrans*), mosquito (*Aedes aegypti*), and some African tick species (*Rhipicephalus* and *Amblyomma* spp.). These vectors could also transmit the virus from infected carcasses to naive live animals (Tuppurainen et al., 2017b). Even though vectors transmissions are playing the central role, transmission via animal products (milk), fomites (equipment and clothing) and personnel are also possible means of LSD transmission (Tuppurainen et al., 2005).

The virus persists in the semen of infected bulls so that natural mating or artificial insemination may be a source of infection for females. Additionally, there was a report related to the transmission of LSDV to calves that suckle on infected milk as well as teats lesions. Another transmission way is related to iatrogenic intra- or inter-herd transmission. This is due to the use of contaminated needles during vaccination or other injections with a common use of needles between animals or herds. Eventually, affected animals clear the agent from their body which made impossible to stay cattle in their carrier state (Tuppurainen et al., 2017b).

Geographic distribution

Northern Rhodesia (Zambia) was the first country to report LSD in 1929 (MacDonald, 1930). This endemic disease remained till 1990 to sub-Saharan Africa including Ethiopia that reported in 1983 for the first time (Mebratu et al., 1984). The spread continue into North Africa and then into the Middle East. Furthermore, the geographical range continues to include many countries of Middle Eastern countries mostly since 2013 (Tuppurainen et al., 2017b). More recently, LSD has spread into parts of southeast Europe, with outbreaks reported in Turkey and Russia amongst other countries (Artem, 2016; Standing Group of Experts (SGE, 2017). Accordingly, Tuppurainen and Oura (2012) identified LSD as a potential global phenomenon affecting most countries and continents of the world.

LSD is endemic disease in Ethiopia with an increase in its range from its existence in 1983 from Southwest of Lake Tana (Mebratu et al., 1984). In current situation, the disease wide spread throughout the country regardless of the altitude as well as husbandry systems of the location and farming system, respectively. Taking this in to consideration, Ministry of Agriculture kept this disease as a reported disease that must be reported as outbreak notification to the National veterinary service office under the ministry that receive the specific information from the specified site. Different reports of outbreaks were reported from almost all parts of the country including Central parts of the country (Ayelet et al., 2014).

Morbidity and Mortality rates

Morbidity of LSD varies greatly that ranges from 3 to 85% in different epizootic situations and even 100% in rare cases. The mortality rate of this disease mostly considered not very high from 1 to 2% and sometime up to 10%. However, mortality may reach up to 40% in severe outbreak cases. These broad ranges for morbidity and mortality are related to different factors including cattle breed, health status, viral isolates and insect vectors involved in the transmission. For instance in Africa, imported breeds from Europe or Australia has showed high susceptibility to LSD (Coetzer, 2004; Paolo et al., 2018).

Other than breeds, host species can also have an impact on those rates. Accordingly, the morbidity rate is significantly lower in buffalo (1.6%) than in cattle (30.8%) (El-Nahas et al., 2011). According to Tuppurainen and Oura (2012), in the later animal (Cattle), LSD morbidity varies significantly depending on the immunity status and the abundance as well as the distribution of mechanical arthropod vectors. To this extent, African trypanosomosis contribute greatly to the severity of LSD infection and hence to morbidity and mortality (Babiuk et al., 2008b).

Pathogenesis

Pathogenesis differs in field and experimental conditions due to a significant difference in their incubation period. Accordingly, a longer incubation period has been seen in natural (2 to 4 weeks) than experimental infections (about five days) (OIE, 2010). During natural infection of field condition, the most efficient ways of LSDV introduction is through the skin via the bite of arthropods, such as mosquitoes, ticks, *Culicoides* spp., or sand flies. The introduction site of skin is expected to swell after a week

followed by enlargement of regional lymph nodes. However, generalized eruption of skin nodules usually occurs between 7 to 19 days. In most cases of LSD, introduction of the virus in reaching blood vessel (intravenous) or limited to skin (intrademal/subcutaneous) determine the characteristic lesions. Accordingly, localized and generalized skin lesions are expected if the inoculation limited to skin surface and reaches blood vessels, respectively. Furthermore, this site of inoculation to reach intravenous circulation produces viremia that causes more severe diseases (Tuppurainen et al., 2017b).

The basic pathogenic mechanism by which the virus seems to cause lesions is viral replication in cells such as the pericytes and endothelial cells in lymphatic‟s and blood vessels walls; giving rise to vasculitis and lymphangitis. In some more severe cases thrombosis and infarction may be the end result. Other cells such as macrophages, fibroblasts and keratinocytes may also be infected. Most animals that recover from clinical disease seem to develop a lifelong immunity. Immunity to LSD seems mostly cell- mediated but maternal antibodies acquired by calves may protect them from clinical diseases for approximately six months (Vorster and Mapham, 2008).

Different cell are expected to be affected by LSDV in the course of disease development. From these various cells, pericytes, fibroblasts, epithelial and endothelial cells can be infected by the virus are the one infected with this virus. Consequently, the attack of those cells by this virus could lead to severe vasculitis, lymphangitis and even infarction in severe cases. Furthermore, LSD inflammatory effect due to viral infection causes fever which is followed shortly by the development of nodular lesions in the skin that subsequently undergo necrosis. Generalized lymphadenitis and edema of the limbs are also common. Lacrimation, nasal discharge, and loss of appetite are reported to occur in the early stages of this disease. Additional signs are from the skin nodules involving the dermis and epidermis appeared to be raised and later ulcerated, and may even become infected with bacteria as a secondary complication (Barthold et al., 2011).

Experimental infection of cattle with LSDV showed the existence the virus in saliva after 11 days after fever development. On other specimens such as semen and skin nodules, the virus has been recovered after relatively longer period. Likewise, virus appeared after a double (22 days) and triple (33 days) folds of dates of the one seen in saliva when appeared in semen and skin nodules, respectively. However, the virus is not recovered from urine or fecal samples (Tuppurainen et al., 2017b).

CLINICAL SIGNS OF THE DISEASE

An incubation period of 2-4 weeks is common in field outbreaks and 7-14 days following experimental challenges. The clinical signs range from in apparent to severe. Host susceptibility, dose and route of virus inoculation affect the severity of disease (Knopvelsiekte, 2008). In severe cases there is an initial rise of temperature, which lasts for over a week, sometimes accompanied by lacrimation, nasal discharge, salivation, and lameness. Multiple nodules appear suddenly about a week later, the first ones usually appearing in the perineum. They are round and firm, varying from 1 to 4 cm in diameter, and are flattened and the hair on them stands on end. They vary in number from a few to hundreds; they are intradermal and, in most cases, are confined to the skin area. Other manifestations that may be observed in severe cases include lesions in the nostrils and on the turbinates, causing mucopurulent nasal discharge, respiratory obstruction and snoring; plaques, later ulcers, in the mouth causing salivation; nodules on the conjunctiva, causing severe lacrimation, and on the prepuce or vulva, and spreading to nearby mucosal surfaces. The limbs may become grossly distended with edema fluid (Radostits et al., 2006; Salib and Osman, 2011). Feed intake decreases in affected cattle, milk yield can drop markedly, and animals may become emaciated. Rhinitis, conjunctivitis and keratitis can also be seen; ocular and nasal discharges are initially serous but become mucopurulent (Knopvelsiekte, 2008).

Secondary bacterial infections can cause permanent damage to the tendons, joints, teats and mammary gland. Abortions and temporary or permanent sterility may occur in both bulls and cows. A few animals die, but the majority slowly recovers. Recovery can take several months, and some skin lesions may take a year or two to resolve. Deep holes or scars are often left in the skin (Grooms, 2005). The post mortem lesions can be extensive. Characteristic grayish-pink deep nodules with necrotic centers are found in the skin (Grooms, 2005). Similar lesions on the skin are present in the mouth, pharynx, trachea, skeletal muscle, bronchi and stomachs, and there may be accompanying pneumonia. The superficial lymph nodes are usually enlarged. Respiratory distress and death are often the result of respiratory obstruction by the necrotic ulcers and surrounding inflammation in the upper respiratory tract and/or concurrent aspiration pneumonia. Fever, skin nodules, enlarged lymph nodes, lameness, depression, lacrimation and salivation were the major and characteristic clinical features of LSD found during these outbreaks (Radostits et al., 2006).

DIAGNOSIS OF LSD

The tentative diagnosis of LSD is usually based on the characteristic clinical signs, differential diagnosis, and confirmation is done by laboratory tests using molecular techniques of conventional or real time polymerase chain reaction (PCR) and cell culturing. LSD should be suspected clinically when there are characteristic skin nodules, fever and enlargement of superficial lymph nodes (Abdulqa et al.,

2016; Tuppurainen, 2017a; OIE, 2017).

Virus isolation

Confirmation of LSD in a new area requires virus isolation and identification. Virus isolation is the method used to investigate the viability of the virus in the samples (Tuppurainen, 2017a). LSDV will grow in tissue culture of bovine, ovine or caprine origin, although maximum yield is obtained using lamb testis or bovine dermis cells. In cell culture, LSDV causes a characteristic cytopathic effect (CPE) and intracytoplasmic inclusion bodies that is distinct from infection with Bovine herpesvirus 2, which causes pseudo-lumpy skin disease and produces syncytia and intranuclear inclusion bodies in cell culture (Abdulqa et al., 2016; OIE, 2017).

Molecular detection methods

Laboratory confirmation of LSD virus can be done very rapidly using a 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 microscopy (TEM). Genome detection using Capri pox virus-specific primers for the attachment protein and fusion protein gene has been reported, and several conventional and real-time PCR methods have been established to be used on blood, tissue and semen specimens (Abdulqa et al., 2016; OIE, 2017; Abera et al., 2015). Recently, a capripoxvirus real-time PCR method using primers and a probe has been validated (Bouden et al., 2009; Tuppurainen and Oura, 2011). Molecular tests using loop-mediated isothermal amplification to detect capripoxvirus genomes are also reported to provide sensitivity and specificity similar to real-time PCR with a simpler method and lower cost (Das et al., 2012; Murray et al., 2013).

Serological tests

Serological tests that can be used for LSDV include an indirect fluorescent antibody test (IFAT), virus neutralization, enzyme-linked immunosorbent assays (ELISA) and immune blotting (Western blotting) (Abera et al., 2015). The virus neutralization test (VNT) is the only validated serological test available. The agar gel immune diffusion test (AGID) and IFAT are less specific than the VNT due to cross-reactions with antibody to other poxviruses. Western blotting using the reaction between the P32 antigens of LSDV with test sera is both sensitive and specific, but is difficult and expensive to carry out. Some antibody-detecting ELISAs have been described but none is sufficiently validated to be recommended for use (OIE, 2017; Babuik et al., 2008).

Differential diagnosis

The main differential diagnosis is pseudo-LSD caused by bovine herpesvirus 2 (BoHV-2). This is usually a milder clinical condition, characterized by superficial nodules, resembling only the early stage of LSD. Intra-nuclear inclusion bodies and viral syncytia are histopathological characteristics of BoHV-2 infection not seen in LSD (OIE, 2017; Radostits *et al*., 2006). Other differential diagnoses (for integumentary lesions) include: dermatophilosis, dermatophytosis, bovine farcy, photosensitisation, actinomycosis, actinobacilosis, urticaria, insect bites, besnoitiosis, nocardiasis, demodicosis, onchocerciasis, pseudo-cowpox, and cowpox. Differential diagnoses for mucosal lesions include: foot and mouth disease, bluetongue, bovine viral diarrhoea, malignant catarrhal fever, infectious bovine rhinotracheitis, and bovine popular stomatitis (OIE, 2017; Abera et al., 2015).

ECONOMIC IMPORTANCE OF THE DISEASE

Lumpy skin disease is one of the economically significant diseases in Africa and the Middle East countries that cause severe production loss in cattle. The world organization for animal health (OIE) categorizes the disease as notifiable diseases because of its severe economic losses. The economic importance of the disease was mainly due to having high morbidity rate rather than mortality (Tuppurainen and Oura, 2011). The financial implication of these losses is greatly significant to the herd owners, consumers and the industrial sectors which can process the livestock products and by products. In intensive farming of cattle, the direct and indirect production losses caused by LSD were estimated to be as high as 45-60%. It was reflected that the severity of the disease was much more in developing countries where the poorest small scale farmers was found. The disease was mainly affects cattle with subsequent effects on production through the morbidity and reduced productivity (CFSPH, 2008). Major consequences of the disease are retarded genetic improvement, limits the ability of the animal to work, draught power and traction loss, abortion in pregnant cows, marked reduction of milk yield during the active case of the disease, sterility and infertility in both sexes of cattle, permanent damage to hide and chronic debility in beef cattle (Tuppurainen, 2005; OIE, 2010).

The morbidity and mortality rates for LSD vary greatly in different endemic areas depending on the severity of strain, prevalence of insect vectors and susceptibility of the host (Getachew et al., 2010). An outbreak in a previously free country could be expected to result in a high morbidity rate. If LSD became endemic, continuing economic loss and poor

productivity would occur due to stock losses, reduced production in cattle industries and cost of preventative vaccination. Permanent loss of some markets would also be expected, with associated downturn in rural economy and increased rural unemployment (Tuppurainen and Oura, 2011).

STATUS OF LUMPY SKIN DISEASE IN ETHIOPIA

LSD was first reported in 1983 in the northwestern part of the country near Lake Tana (Mebratu et al., 1984). It has now spread to almost all the regions and agro-ecological zones. Because of the wide distribution of the disease and the size and structure of the cattle population in Ethiopia, it is likely that LSD is one of the most economically important livestock diseases in the country (Gari et al., 2010). There were frequent outbreak reports of LSD in the county that are highly associated with seasonal peak of mechanical vectors in wet and warm weather conditions (Getachew et al., 2010). One of the outbreaks of LSD was occurred in central Ethiopia in 2007 to 2011. These outbreaks were described as active. It was investigated in four districts: Adama, Wenji, Mojo and Welenchiti. The totally 1,675 outbreaks were reported over 5 years period from 2007 to 2011, with 62,176 cases and 4,372 deaths. The Oromia represented the highest numbers of outbreaks (1,066), followed by Amhara (365) and the Southern Nations, Nationalities and People's Region (123). The 2010 were reported the highest number of outbreaks that were frequently seen between September and December. The morbidity and mortality rates were 13.61% (296) and 4.97%, respectively (Ayelet et al., 2014).

Its spread was mainly enhanced by cattle movements, communal grazing and watering, and pastoralist ways of life (Tuppurainen and Oura, 2011; Gari *et al*., 2012). In Ethiopia from 2007-2011 a total of 1352 disease outbreaks of LSD have been reported and highest frequency was documented in Oromia region and the least in Afar region (Gumbe, 2018). Another study also showed that a total 3811 LSD outbreaks reported in Ethiopia between 2000 and 2015. Most of these outbreaks were from Oromia (54.5%), Amhara (27.9%), SNNP (10.1%) and Tigray regional states (3.6%) No out breaks were reported from Harari and Dire dawa. It also shows that LSD affects districts for one or two years and then spreads to other nearby areas with a susceptible cattle population with a trend of LSD outbreaks increased over time (Molla *et al*., 2017a).

Data investigations from the national disease outbreak report database during the period 2000-2009 showed that major epidemic outbreaks of LSD occurred in 2000/2001 in the northern parts of the country: Amhara and West Oromia regions. Then it extended to the central and the southern parts of the country in 2003 and 2004 covering large parts of Oromia and Southern Nation, Nationalities and Peoples (SNNP) regions. In 2006 and 2007 another extensive outbreak reappeared in Tigray, Amhara and Benishangul regions in the northern and north-western parts of the country. From 2007 up to 2009 the outbreak number progressively increased in Oromia Region situated in the central part of the country while it seemed to be gradually decreasing in the northern part of the country including Tigray, Amhara and Benishangul regions. This showed that an epidemic reoccurs after an interval of 5-6 years cycle in unvaccinated cattle population (Gari, 2011).

According to the 2010 Annual Report of the Ministry of Agriculture, Animal and Plant Health Regulatory Directorate in the Department of Epidemiology shows us the prevalence of the disease in different regional states of the country; 1.63, 0.49, 5.2, 2.69, 0.37, 0.7 and 3.8% in Addis Abeba, Amhara, Gambela, Oromia, SNNP, Somali and Tigray regions respectively. The 2011 annual report shows prevalence of; 0.36, 1.13, 0.22, 0.65, 0.24 and 0.30% in Amhara, Gambela, Oromia, SNNP, Somali and Tigray regions, respectively. This demonstrates how widely the disease has spread across the country (Molla et al., 2017a).

Since the country has no a well-designed control strategy for this disease, it continues to be a major problem. Even if the animal health authorities undertake vaccination campaigns when outbreak is reported, researches have shown that the different vaccines used in Ethiopia are not fully effective (Molla et al., 2017b; Ayelet et al., 2013). There have been repeated concerns reported to NVI on the insufficient protection provided by the vaccine, for cattle against LSDV. In addition to this, lack of genetic information on the circulating isolates in the field and their relation to the vaccine strain in use, which is essential for better vaccine matching, is also a great problem in the country (Gelaye et al., 2015).

RISK FACTORS OF LSD IN ETHIOPIA

Host risk factor: All ages and types of cattle are susceptible to the causative virus, except animals recently recovered from an attack, in which case there is a solid immunity. In outbreaks, very young calves, lactating and malnourished cattle develop more severe clinical disease. British breeds, particularly Channel Island breeds, are much more susceptible than zebu types, both in numbers affected and the severity of the disease because of their thin skin. Wildlife species are not affected in natural outbreaks, although there is concern that they might be reservoir hosts. Serological evidence of naturally acquired infection has been observed only in African buffalo (Syncerus caffer). There is only one report of the natural occurrence of LSD in a species other than cattle, in water buffalo (Bubalis), but no further such cases are recorded (Radostits et al., 2006; Vorster and Mapham, 2008).

Environmental risk factor: Outbreaks tend to follow

waterways and extensive epizootics are associated with high rainfall and concomitant high levels of insect activity with a peak of disease in the late summer and early autumn (Radostits et al., 2006). Other environmental risk factors associated with spread of LSD were found to be worm humid agro-climate, communal grazing/watering and introduction of new animals in a herd. The incidence of LSD occurrence is high during wet seasons when biting-fly populations are abundant and it decreases or ceases during the dry season (Gari et al., 2010).

Pathogen risk factor: LSDV is generally resistant to drying, survive freezing and thawing. Resistance to heat is variable but most are inactivated at temperatures above 60°C (Radostits et al., 2006). LSDV is very resistant to physical and chemical agents. The virus persists in necrotic skin for at least 33 days and remains viable in lesions in air-dried hides for at least 18 days at ambient temperature (Vorster and Mapham, 2008).

In Ethiopia, outbreaks were seen to highly correlate with the wet and warm weather that are related to the favorability to mechanical vectors (Gari et al., 2010). In addition, husbandry related issues can also have a role in this disease transmission. For example, sharing of the same feeding and watering troughs gives an opportunity of contaminating feed and/or water with the saliva of infected animals. In support to this, Gari et al. (2010) find out the communal grazing and watering points in Ethiopia were found to be associated with the occurrence of LSD. Additionally, those points were a potential higher risk to existing herd with an introduction of new infected animals. The putative risk factors altitude, breed, sex, and presence of animal trade route showed no significant association with LSD sero-status. Generally, cattle population with many adult animals and that live in wet areas are at highest risk, whereas cattle in frequent contact with other animals and animal species have lower risk, potentially due to a dilution effect of vectors.

Many other authors have also recorded the same symptoms in natural and experimental infections (Body et al., 2011; Jalali et al., 2017). Susceptibility of host animals due to factors like age, immunological status, and dose and route of virus inoculation also affected disease severity (Ayelet et al., 2014). In which animals with severe cases or died of LSD observed in these outbreaks had a poor body conditions which might affect the immunity to the disease. The morbidity, mortality and case fatality rates of LSD indicating an important impact posed by the disease in central Ethiopia may be due to the farming and management system practiced in the area which favors the vector transmission and poor nourishment of diseased animals which will die due to secondary disease.

The morbidity rate was closer to reported from the study in central Ethiopia with 13.61% (Ayelet et al., 2014). Higher morbidity (21.2%) and mortality rates (4.5%) were previously reported by Wassie et al. (2017) from Ethiopia. On the other hand, morbidity rate of the disease in the current study was higher than the reported results in north-eastern Ethiopia by Hailu et al. (2014) who reported a morbidity rate of 7.4%. Different morbidity, mortality and case fatality rates were recorded in different PAs with the highest morbidity in Kalu/07 (21.42%) and lowest in Tehuledere/023 (11.76%). This might be due to the proximity of Kalu area to rivers which might be suitable for the replication of arthropod vectors. The variations in morbidity rates and mortality rates could be from the differences in geographic location and climate; the management conditions, breed, immune status and condition of the animals, virulence of the virus, and the number and types of putative insect vectors (Tuppurainen and Oura, 2011).

In many studies, the morbidity rate of LSD indicated that calves were more susceptible to infection than adult cattle. This might have occurred due to the natural susceptibility of young animals which may also usually show more severe clinical disease (Coezer and Tuppurainen, 2004). This finding is in line with the reports of Ahmed and Zaher (2008) and Ayelet et al. (2014) but it disagrees with the study done by Kasem et al. (2018).

The mortality and case fatality rates were higher in male animals (3.29 and 28.57%) than in females (2.38 and 11.76%), while the morbidity was higher among female animals (20.23%) than male animals (11.53%). Even if the physiological conditions of female animals (like pregnancy and lactation) might affect their susceptibilities, evidenced by increase in morbidity rates, they are usually kept inside houses.

The farmers in the area use oxen for ploughing their lands and stress and fatigue might have increased the mortality rates. A study also shows that rates of morbidity and mortality were higher in cross breeds than local cattle with statistically significant differences. The genetic differences between breeds may have influenced susceptibility to the disease (Abera et al., 2015). In contrast, high case fatality rates were recorded in local cattle. This might be due to lack of proper nursing and treatment of diseased local animals because of their lower market values.

Even if more number of cattle were vaccinated, the disease has been manifested by high morbidity and mortality rates, regardless of vaccination status. The observed vaccine failure may be due to lack of cross-protection of the vaccine strains against circulating virulent field strains in the area (Kasem et al., 2018). In addition, problems in vaccine management, including transport and storage may have influenced vaccine efficacy. Introduction of animals already incubating the virus or newly vaccinated animals becoming infected before they develop protective immunity due to untimely vaccine campaigns (vaccination after the out breaks have already occurred in the area) might have also a great contribution for vaccine failure (Ayelet et al., 2014).

CONCLUSION AND RECOMMENDATIONS

Lumpy skin disease is a poxvirus disease of cattle characterised by fever, nodules on the skin, mucous membranes and internal organs, emaciation, enlarged lymph nodes, oedema of the skin, and sometimes death. Lumpy skin disease is considered as transboundary and trade banned disease which has significant impendent on livestock market and animal products. This disease, not only economically affects individual cattle owners, but also affects the foreign currency of the country from exporting of live animal and animal product. LSD was found to be the major cattle health problem causes severe economic loss due to permanent damage to hides, a prolonged debilitating clinical course, reduced weight gain, temporary or permanent loss of milk production, temporary or permanent infertility or even sterility in bulls, and abortion of pregnant cows. The disease affected all age groups of cattle regardless of the differences in breed, sex and vaccination status and already caused great economic loss due to high mortality, morbidity and case fatality rates. In Ethiopia vaccination is given for control of LSD in sporadic and endemic area with the vaccine that prepared from Kenyan sheep and goat pox strain but high morbidity and mortality rates reported among vaccinated ones. The efficacy test of the vaccine performed under laboratory protocol revealed that the vaccine is immunologically protective. Vaccine failure which could be due to problems in vaccine storage and efficacy of produced vaccines against the current field strain in the area needs concern. Therefore, the following recommendations were forwarded from the above conclusions:

- ✓ Annual surveillances on the status of the disease should be done in all regions of the country
- ✓ Disease prevention and control measures such as ring vaccination and prophylactic immunization in high risk population should be implemented by effective and well managed vaccines during outbreak of LSD.
- ✓ Broader and detailed investigation including sequence characterization and determination of their evolutionary relationship of the viruses as compared to the vaccine strain in the country.
- ✓ Further isolation and molecular characterization of LSDV should be conducted so as to identify strain of the virus in the country in order to produce new vaccine.
- ✓ Awareness creation for cattle owners to vaccinate healthy animals for control and support the diseased ones to prevent death and disease transmission.

REFERENCES

Abdulqa HY, Rahman HS, Dyary HO, Othman HH (2016). Lumpy Skin Disease. Reproductive Immunology: Open Access.

Abera Z, Degefu H, Gari G, Kidane M (2015) Sero-prevalence of lumpy skin disease in selected districts of west Wollega zone. BMC Vet Res :1–9.

Ahmed WM, Zaher KS (2008). Observations on lumpy skin disease in local Egyptian cows with emphasis on its impact on ovarian function. Afr. J. Microbiol. Res. 2(10):252-257.

Alaa A, Hatem M, Khaled A (2008). Polymerase chain reaction for rapid diagnosis of a recent lumpy skin disease virus incursion to Egypt. J. Arab Biotech, 11:293-302.

Al-Salihi KA (2014). Lumpy Skin disease: Review of literature. Mirror of research in veterinary sciences and animals 3 (3):6-23.

Ali MA, Amina AD (2013). Abattoir-Based Survey and Histopathological Findings of Lumpy Skin Disease in Cattle at Ismailia Abattoir. International Journal of Bioscience, Biochemistry and Bioinformatics. 3(4):372-375.

Ali AA, Esmat M, Attia H, Selim A, Abdel-Hamid YM (2012). Clinical and pathological studies of lumpy skin disease in Egypt. Vet. Rec. 127(22):549–50.

APHIS Veterinary Services Centers for Epidemiology and Animal Health (2006). Lumpy Skin Disease, Israel. (Impact Worksheet).

Artem M (2016). The Russian Federation LSD report.

Ayelet G, Abate Y, Sisay T, Nigussie H, Gelaye E, Jemberie S, Asmare K (2013). Lumpy skin disease: preliminary vaccine efficacy assessment and overview on outbreak impact in dairy cattle at Debre Zeit, central Ethiopia. Antiviral research 98(2): 261-265.

Ayelet G, Haftu R, Jemberie S, Belay A, Gelaye E, Sibhat B, Skjerve E, Asmare K (2014). Lumpy skin disease in cattle in central Ethiopia: outbreak investigation and isolation and molecular detection of the virus. Rev. of Science and Tech. 33**:** 877-887.

Babuik S, Bowden TR, Boyle DB, Wallace DB, Kitching RP (2008). Capripox viruses: An Emerging Worldwide Threat to Sheep, Goats and Cattle. Transboundary and Emerging Diseases 55: 263-272.

Babiuk S, Bowden T, Parkyn G, Dalma B, Manning L, Neufeld J, Embury-Hyatt C, Copps J, Boyle D (2008b). Quantification of Lumpy skin disease virus following experimental infection in cattle. Trans. and Emer. Dis. 55: 299-307.

Barthold SW, Bowen RA, Hedrick RP, Knowles DP, Lairmore MD, Parrish CR, Saif LJ, Swayne DE (2011). Veterinary and Zoonotic Viruses. In: Fenner's Veterinary virology. MacLachlan, N.J. and Dubovi, E.J. (eds), 4th ed. Elsevier Inc. 151-160.

Bhanuprakash V, Venkatesan G, Balamurugan V, Hosmani M, Yogisharadhya R, Chauhan RS, Pande A, Mondal B, Singh RK (2010). Pox outbreaks in Sheep and Goats at Makhdoom (Uttar Pradesh), India: Evidence of Sheeppox Virus Infection in Goats. Transboundary and Emerging Diseases 375-382.

Bhanuprakash V, Hosamani M, Singh RK (2011). Prospects of control and eradication of capripox from the Indian subcontinent: A perspective. Antiviral Research 91: 225-232.

Body M, Singh KP, Hussain MH, Rawahi AA, Maawali AM, Lamki KA, AL-Habsy S (2011). Clinico-Histopathological Findings and PCR Based Diagnosis of Lumpy Skin Disease in the Sultanate of Oman. Pakistan Veterinary J. 32(2):206-210.

Bouden TR, oupar BE, abiuk SL, White JR, Boyda V, Ducha CJ, Shiell BJ, Uedad N, Parkynb GR, Coppsb JS, Boylea DB (2009). Detection of antibodies specific for sheeppox and goatpox viruses using recombinant capripoxvirus antigens in an indirect enzyme-linked immunosorbent assay. J. Virology Methods.

Brenner J, Haimovitz M, Oron E, Stram Y, Fridgut O, Bumbarov V, Kuznetzova L, Oved Z, Waserman A, Garazzi S, Perl S, Lahav D, Edery N, Yadin H (2006). Lumpy skin disease (LSD) in a large dairy herd in Israel. Israel J. veterinary Medicine. 61, 73–77.

CFSPH (2008). The Center for Food Security and Public Health, Iowa State University, College of Veterinary Medicine and Institution of International cooperation in Animal Biologics, an OIE collaborating center.

Chihota CM, Rennie LF, Kitching RP, Mellor PS (2003). Attempted mechanical transmission of lumpy skin disease virus by biting insects Medical and veterinary entomology 17(3)294-300.

Coezer J, Tuppurainen E (2004). Lumpy skin disease Oxford University Press. Cape Town, Southern Africa 2: 1268-1276.

DAS A, Babiuk S, Mcintosh MT (2012). Development of a loop-mediated

isothermal amplification assay for rapid detection of capripoxviruses, J. Clinical Microbiol. 50: 1613–1620.
El-Kholy AA, Soliman HMT, Abdelrahman KA (2008). Polymerase chain reaction for rapid diagnosis of a recent lumpy skin disease virus incursion to Egypt. Arab J. Biotechnol. 11, 293-302.
El-Nahas EM, El-Habbaa AS, El-Bagoury GF, Radwan MEI **(**2011). Isolation and identification of lumpy skin disease virus from naturally infected buffaloes at Kaluobia,Egypt. Global Veterinarian 7:234–237.
FAO (2019). Livestock in balance. Vialedelleterme di Caracalla: Food and Agriculture Organization of the United Nations. 23.
Fayez AS, Ahmed HO (2011). Incidence of lumpy skin disease among Egyptian cattle in Giza Governorate. Egypt Veterinary World 4(4):162-167.
Gari G, Waret-Szkuta A, Grosbois V, Jacquiet P, Roger F (2010). Risk factors associated with observed clinical lumpy skin disease in Ethiopia. Epidemiol. Infections 138(11):1657-1666.
Gari G., Bonnet P., Roger F. and Waret-Szkuta A. S. (2011): Epidemiological aspects and financial impact of lumpy skin disease in Ethiopia. Preventive veterinary medicine 102(4):274-283.
Gari G, Grosbois V, Waret-Szkuta A, Babiuk S, Jacquiet P, Roger F (2012): Lumpy skin disease in Ethiopia: seroprevalence study across different agro-climate zones. Acta Tropicals 123(2):101-106.
Gebreegziabhare B (2010). An overview of the role of Ethiopian livestock in livelihood and Food safety, Ministry of Agriculture and Rural development of Ethiopia; Presented on dialogue on livestock, food security and sustainability, a side event on the session of 22 COAGO, FAO, Rome.
Gelaye E, Belay A, Ayelet G, Jenberie S, Yami M, Loitsch A, Tuppurainen E, Grabherr R, Diallo A, Lamien EC (2015). Capripox disease in Ethiopia: Genetic differences between field isolates and vaccine strain, and implications for vaccination failure. Antiviral Research.
Getachew G, Waret-Szkuta A, Grosbois V, Jacquite P (2010). Risk Factors Associated with observed clinical lumpy skin disease in Ethiopia. PhD thesis. 68-84.
Grooms D (2005). Raising Awareness about Lumpy Skin Disease Department of Large Animal Clinical Sciences. Michigan Dairy Review 48: 824-1225.
Gumbe A (2018). Review on lumpy skin disease and its economic impacts in Ethiopia J. Dairy, Veterinary Anim. Res.
Birhanu HH, Tadele T, Getachew G, Teshale T, Belay B (2014). Estimated prevalence and risk factors associated with clinical Lumpy skin disease in north-eastern Ethiopia. Preventive Veterinary Medicine. 115(1-2):64–68.
Irons PC, Tuppurainen ES, Venter EH (2005). Excretion of lumpy skin disease virus in bull semen. Theriogenology 63, 1290–1297.
Jalali SM, Rasooli A, Seifi Abad, Shapuri M, Daneshi M (2017). Clinical, hematologic, and biochemical findings in cattle infected with lumpy skin disease during an outbreak in Southwest Iran. Arch. Razi Inst. 72, 255–265.
James A (2004). Transmission and geographic distribution of lumpy skin disease. Foreign Animal Disease Diagnostic Laboratory, Greenport, New York. 1-9.
Jordi C, Alberto A, Aleksandra M, Ledi P, Blagojco T, Dimitar T, Alexandrov T, Beltrán-Alcrudo D (2018). Economic cost of lumpy skin disease outbreaks in three Balkan countries: Albania, Bulgaria and the former Yugoslav Republic of Macedonia (2016–2017). Transbound. Emerg. Dis. 65, 1680–1688.
K LK, B S, K R (2021). Clinico-molecular diagnosis and characterization of bovine lumpy skin disease virus in Andhra Pradesh, India. Trop. Anim. Health Prod. 53, 424.
K A Al-Salihi (2014). Lumpy skin disease: Review of the literature. Mirror Res. Vet. Sci. Ani. 3, 6–23.
Kasem S, Saleh M, Qasim I, Hashim O, Alkarar A, Abu-Obeida A, Gaafer A, Hussien R, Al-Sahaf A, Al-Doweriej A (2018). Outbreak investigation and molecular diagnosis of Lumpy skin disease among livestock in Saudi Arabia 2016. Transboundary and emerging diseases 65(2):494-500.
Knopvelsiekte N (2008). Identification and diagnosis of lumpy skin disease. J. Virology, 44:1-4.
Kumar SM (2011). An outbreak of lumpy skin disease in a Holstein dairy herd in Oman: A Clinical report. Asian J. Anim. Veterinary Adv. 6: 851-859.
MacDonald RAS (1930). Pseudo-urticaria of cattle. Northern Rhodesian Department of Cattle Health, Annual Report. 20–21.
Magori-Cohen R, Louzoun Y, Herziger Y, Oron E, Arazi A, Tuppurainen E, Shpigel NY, Klement E (2012). Mathematical modelling and evaluation of the different routes of transmission of lumpy skin disease virus. Veterinary Res. 43: 1, 1.
Mebratu GY, Kassa B, Fikre Y, Berhanu B (1984). Observation on the outbreak of lumpy skin disease in Ethiopia. Review Elev. Med. Veterinary Pays Tropics. 37: 395–399.
Molla W, De Jong M. C. M. and Frankena K. (2017a). Temporal and spatial distribution of lumpy skin disease outbreaks in Ethiopia in the period 2000 to 2015. *BMC Veterinary Research* 13: 310.
Molla W, de Jong MCM, Gari G, Frankena K (2017b). Economic impact of lumpy skin disease and cost effectiveness of vaccination for the control of outbreaks in Ethiopia. Preventive Veterinary Medicine. 147**:** 100-107. 34
Morris JA (1931). Pseudo-urticaria. Northern Rhodesia Department of Animal Health, Annual Report 1930: 12.
Murray L, Edwards L, Tuppurainen ES, Bachanek-Bankowska K, Oura CA, Mioulet V, King DP (2013). Detection of capripoxvirus DNA using a novel loop-mediated isothermal amplification assay. BMC Veterinary Research 9: 90.
Molla Wassie, Frankena Klaas, Gari Getachew, Kidane Menbere, Shegu Dereje, de Jong Mart C.M. (2018) Seroprevalence and risk factors of lumpy skin disease in Ethiopia. Prev. Vet. Med. 160:99–104.
Negassa A, Rashid S, Gebremedhin B (2011). Livestock Production and Marketing. ESSP II Working Paper 26. Int. Food Policy Research Institute/ Ethiopia Strategy Support Program II, Addis Ababa, Ethiopia.
OIE (World Organisation for Animal Health) (2017). Lumpy Skin Disease. Terristerial manual chapter 2.4.13.
OIE (2010). Lumpy skin disease; Manual of Diagnostic Tests and Vaccines for Terrestrial animals. Paris: World Organization for Cattle Health. 1-13.
Tuppuraine ES, Alexandrov T, Beltran-Alcrudo D (2017). Lumpy skin disease field manual - A manual for veterinarians. FAO Animal Production and Health Manual 20: 1-60.
Paolo C, Kris D, Annebel DV, Simon G, Eyal K, Arjan S, Jos CA, Sotiria-Eleni A, Alessandro B, Andrey G (2018). Lumpy skin disease: scientific and technical assistance on control and surveillance activities. EFSA J. 16(10):5452.
Radostits OM, Gay CC, Hinchcliff KW, Constable PD (2006). Veterinary Medicine: A textbook of the diseases of cattle, horses, sheep, pigs and goats. Edition 10th. Sounders Elsevier.
Salib A, Osman H (2011). Incidence of lumpy skin disease among Egyptian cattle in Giza Governorate, Egypt. Vet World. 4: 162-167.
Standing Group of Experts (SGE). (2017). Lumpy Skin Disease in the South East Europe region under the GF-TADs umbrella. Fifth meeting (SGE LSD5). Lumpy skin disease (LSD) epidemiological situation in Europe.
Tageldin MH, Wallace DB, Gerdes GH, Putterill JF, Greyling RR, Phosiwa MN, Al Busaidy RM, Al Ismaaily SI (2014). Lumpy skin disease of cattle: an emerging problem in the Sultanate of Oman. Tropical. Animal. Health and Production. 46:241–246.
Tuppurainen ES (2017a). Diagnostic assays for the detection of lumpy skin disease virus and antibodies. Reaserch Gate accessed on 5-10.
Tuppurainen E, Alexandrov T, Beltrán-Alcrudo D (2017b). Lumpy skin disease field manual –A manual for veterinarians. FAO Cattle Production and Health Manual No. 20, 3-6.Rome.
Tuppurainen E, Venter EH, Shisler JL, Gari G, Mekonnen GA, Juleff N, Lyons NA, De Clercq K, Bouden TR, Babuik S, Babuik LA (2015). Review: Capripoxvirus Diseases: Current Status and Opportunities for Control. Transboundary and Emerging Diseases 64**:** 729-745.
Tuppurainen S, Pearrson CR., Bankowska BK, Knowles NK, Amareen S, Frost L, Henstock MR, Diallo A, Martens PP (2014). Characterization of sheep pox virus vaccine for cattle against lumpy skin disease virus. Antiviral Research. 109**:** 1-6
Tuppurainen ESM, Oura CAL (2012). Review on lumpy skin disease: An Emerging threat to Europe, the Middle East and Asia. Transboundary emerging disease. 59:40-48.
Tuppurainen ES, Oura CA (2011). Review: Lumpy Skin Disease: An Emerging Threat to Europe, the Middle East and Asia. J. Transboundary and Emerging Diseases.

Tuppurainen ES, Venter E, Coetzer J (2005). 'The detection of lumpy skin disease virus in samples of experimentally infected cattle using different diagnostic techniques', On derstepoort J. Veterinary Research **72**, 153–164.

Vorster H, Mapham H (2008). Pathology of lumpy skin disease. Livestock Health and Production Review, 1:16-21.

World Cattle Inventory (WCI). (2015). Ranking of countries (FAO) by Rob Cook..

Yehuda S, Larisa O, Boris G, Hagai Y, Marisol R (2008). The use of lumpy skin disease virus genome termini for detection and phylogenetic analysis. J. Virological Methods. 151: 225–229.

Cite this article as:

Kassa ST (2023). Review on the epidemiology of lumpy skin disease of cattle and associated risk factors in Ethiopia. Acad. J. Microbiol. Res. 11(1): 001-011.

Submit your manuscript at
http://www.academiapublishing.org/ajmr