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Review on Epidemiology and Diagnosis of Lumpy Skin Disease

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Abstract: Lumpy skin disease is an emerging infectious disease of cattle caused by lumpy skin disease virus. It is World Organization for Animal Health notifiable list A disease. The disease is endemic in most African countries. It is transmitted primarily by biting insects and its incidence is high during wet seasons. The course of the disease may be acute, sub-acute, chronic or subclinical. The clinical disease is characterized by a biphasic febrile reaction, depression, disinclination to move, inappetence, salivation, lachrymation, nasal discharge, which may be mucoid or mucopurulent. The superficial lymph nodes, especially prescapular, precrural and subparotid are usually enlarged. Skin nodules are classical manifestation of lumpy skin disease. These nodules are usually widespread and they may be very numerous and cover the entire body of the animal. The diagnosis of the disease is made based on characteristic clinical signs and it is confirmed by various diagnostic techniques including serological and molecular diagnostic methods. Restrictions to the global trade of live animals and animal products, costly control and eradication measures such as vaccination campaigns as well as the indirect costs because of the compulsory limitations in animal movements cause significant financial losses; especially it is important disease to the cattle industry due to chronic debility in infected cattle, reduction in milk production, abortion, temporary or permanent sterility, damaged hides and deaths. In endemic areas mass vaccination against lumpy skin disease is the only effective method to control the disease.

Key Words: Lumpy Skin Disease Virus, Cattle, Epidemiology, Diagnosis

1. Introduction

Lumpy skin disease is an emerging infectious disease of cattle caused by a double stranded enveloped DNA lumpy skin disease virus called *Neethling virus* which belongs to the family *Poxviridea* and genus *Capripox* [1, 2]. The disease presents as an acute, sub-acute or inapparent infectious, generalised skin disease of cattle and it is characterized by rapid eruption of multiple circumscribed skin nodules, and generalized lymphadenitis, fever and with other signs [3]. Lumpy skin disease is an Office International des Epizooties of the World Organization for Animal Health notifiable list A disease. The disease has significant economic importance to cattle industry sectors which causes chronic debility in infected cattle, reduction in milk production, abortion, temporary or permanent sterility, damaged hides and deaths [4]. Animals which have recovered from the disease develop neutralizing antibodies which persist for at least 5 years [5]. The immunity to reinfection is predominantly cell mediated. Animals that have been vaccinated or showed mild disease develop low levels of neutralizing antibodies [6].

Lumpy skin disease usually occurs at regular intervals in endemic areas or it may cause epidemics, which spread fairly rapidly throughout a region (country) or its epidemiology is characterized by periodic outbreaks and the Outbreaks of the disease are much more common during wet seasons and along watercourses where the insect population is high [7]. The transmission of the disease is primarily by biting insects and its occurrence is high during wet seasons when biting insect populations are abundant and decreases during the dry season [8].

The diagnosis of Lumpy skin disease is based on characteristic clinical signs, and the clinical diagnosis is confirmed by various diagnostic techniques including serological and molecular diagnostic methods [9-11]. In endemic areas mass vaccination against Lumpy skin disease is the only effective method to control the disease [1, 12]. The most likely mode of entry of Lumpy skin disease into a new area is by the introduction of infected animals and contaminated materials [13, 14]. Since it is considered that LSD will probably continue to be endemic after an outbreak, certain measures have been used with limited success, and these include proper hygiene, quarantine methods, slaughter policies and vaccination [7, 15, 16]. The objective of this

review is to give an overview of Lumpy skin disease on its etiology, epidemiology, diagnosis, control and prevention, pathogenesis and Economic impact.

2. Epidemiology of Lumpy Skin Disease

2.1. Etiology

Capripox viruses represent one of the eight genera within the *Chordopox virus* of the family *Poxviridae* (Table 1*).*The morphology of the viruses of the genera of the *Chordopox viruses* are similar with the exception of the *Parapox viruses*. The genus *Capripox virus* comprised of sheep pox virus, goat pox virus, and lumpy skin disease virus [15]. Lumpy skin disease (LSD) is caused by Lumpy skin disease virus (LSDV). It is a large (300nm) pleomorphic, double-stranded, unsegmented DNA virus. It has only one serotype and is closely related to sheep pox and goat pox viruses, the only other members of the genus *Capripox virus* [16-18].

Table 1**: Genera within the *Poxviridae* family.**

Genus	Viruses
Capripoxvirus	Sheep pox, goat pox, lumpy skin disease viruses
Orthopoxvirus	Buffalopox, camelpox, cowpox, vaccinia, ectromelia, monkeypox, rabbitpox, raccoonpox, taterapox, variola and velopox viruses
Parapoxvirus	Pseudocowpox, bovine papular stomatitis, contagious pustular dermatitis (orf), squirrel parapox viruses and parapoxvirus of red deer
Suipoxvirus	Swinepoxvirus
Avipoxvirus	Fowlpox, canarypox, juncopox, pigeonpox, guailpox, sparrowpox, starlingpox, turkeypox, mynahpox, and pcittacinepox viruses
Leporipoxvirus	Hare fibroma, myxoma, rabbit (shope) fibroma and squirrel fibroma viruses
molluscipoxvirus	Molluscum contagiosum virus
Yatapoxvirus	Yata and tanapox viruses

Source: Carn, [15]

Capripox viruses are double-stranded DNA viruses with genomes approximately 150 kilobase pair in size. Goat pox and sheep pox viruses share at least 147 putative genes [19]. Lumpy skin disease virus has an additional nine genes that are non-functional in sheep pox and goat pox viruses, some of which are likely responsible for their ability to infect cattle [20]. *Capripox virus*

isolates are extremely conserved with genome identities of at least 96% between sheep pox virus, goat pox virus and LSDV [19]. A comparative study of the genomes of two field isolates of LSDV with the genome of the South African Onderstepoort vaccine strain suggests that *Capripox virus* virulence is linked to a number of genes putatively involved in host immuno-modulation [21].Terminal genomic sequences contain a unique complement of at least 34 genes which are in gene families or likely function in virulence, host range, and immune evasion. LSDV encodes at least 30 homologues of poxviral proteins known to be structural or involved in virion morphogenesis and assembly. These include proteins present in the virion core, proteins present in the intracellular mature virus and associated membranes, potential enzymes involved in protein modification, DNA packaging, and redox activity, proteins found in or associated with the release of extracellular enveloped virions [20].

Poxvirions are brick or oval shaped. Within the virion there are over 100 polypeptides, which are arranged in a core, two lateral bodies, a membrane and an envelope. The membrane and envelope are important structures for the interaction with the host cell. Mature virions that are released from the cell without cell disruption are enveloped. The envelope contains two layers of cellular lipids and several virus-specific polypeptides. Most of the virions released by the rupture of the host cell are therefore not enveloped. Both enveloped and non-enveloped virions are infectious. The outer membrane is a lipoprotein bilayer that protects the core and lateral bodies. It has irregular arrangements of tubular protein called filaments. The core is dumbbell shaped and there are two lateral bodies of unknown nature. The core of the viruses contains proteins that include a transcriptase and several other enzymes [22].

2.2. Occurrence

The distribution of the disease has extended from sub-Saharan countries to Egypt and Western Africa or in general occurs in most African countries. The only African countries still considered free of the disease are Libya, Algeria, Morocco and Tunisia [4]. LSD outbreaks occur almost in each year in most African countries [23]. The disease is also reported outside the African continent like in some parts of Europe, Asia, Middle East countries [1, 24, 25].

2.3. Host range

The host range of lumpy skin disease virus includes sheep, goats and cattle breeds of all ages and sexes, even though some wildlife has also been implicated; but it is primarily a disease of cattle. In Africa imported *Bos taurus* breeds appear to be more susceptible than indigenous *Bos indicus* cattle. Very young calves, lactating cows and malnourished animals seem to be most susceptible to the disease which might be due to an impaired humoral immunity [7, 14, 18].

The disease was reported in the Asian water buffalo (*Bubalus bubalis*) in Egypt [26, 27]. The authors also indicated that domesticated buffaloes (*Bubalus* species) appeared to be more susceptible to LSD than wild buffaloes (*Syncerus* species). Antibodies against LSD have been detected in blue wildebeest (*Connochaetes taurinus*), springbok (*Aepyceros melampus*), eland (*Taurotragus oryx*) and black wildebeest (*Connochaetes gnou*); but the prevalence of antibodies to the virus in these animals were low. So that wildlife species do not play a significant role in the spread or maintenance of LSDV and there is no strong evidence of a wildlife reservoir for *Capripox viruses.* However, wildlife infected with LSDV could be at a distinct disadvantage for survival, and their potential involvement remains unknown since the high rate of removal of infected animals by death and predation would result in a low seropositivity rate in the remaining population [28, 29].

2.4. Transmission

The virus has been isolated from nasal, ocular, and pharyngeal secretions, semen, milk and blood, which might be the source for transmission [16, 30]. Lumpy skin disease is not particularly contagious, and direct transmission by contact between animals is inefficient [31]. Infection by contact can occur, though it is said to be at a low rate and not considered a major role in transmission during epizootics [14]. Most infection is thought to be the result of blood sucking arthropods mechanically [32]. The multiplication of LSDV in the vector insects has not been demonstrated. Biting flies have been incriminated in most epidemics, which have been well defined and have occurred at regular intervals [31]. A report by Davis and Otema, [17] alluded to the possibility of the involvement of arthropod vectors but also suggested that husbandry methods where cattle are crowded together would predispose them to aerosol transmission.

Biting flies (*Stomoxys calcitrans* and *Biomyia fasciata*) and mosquitoes (*Culex mirificens* and *Aedes natrionus*) can be a source for transmission of the disease [32]. Tuppurainen *et al.*, [33] found molecular evidence suggesting that LSD can be transmitted through hard (Ixodid) ticks (*Rhipicephalus decoloratus, Rhipicephalus appendiculatus* and *Amblyomma hebraeum*). Other risk factors associated with spread of LSD were found to be warm humid agro-climate, communal grazing/watering and introduction of new animals in a herd [8].

2.5. Pathogenesis

Poxviruses are generally epitheliotrophic and can cause localized or systemic disease. Initial multiplication of the virus occurs at the entry site of the virus into the body of the host. In systemic infections, further viral replication takes place in the draining lymph nodes, followed by viraemia and further viral multiplication in many different organs including the liver, spleen and lungs. The latter multiplication leads to establishment of secondary viraemia and subsequent infection and development of disseminated focal lesions in the skin. Viral replication takes place in the cytoplasm of cells. Viral particles are enveloped when mature virus particles move to the Golgi complex; most particles are however non-enveloped and are released by cell disruption. Both enveloped and non-enveloped particles are infectious [22].

Even though the exact pathogenesis of the development of the lesions associated with lumpy skin disease is not as well understood as the pathogenesis of sheep poxvirus, LSDV exerts its pathogenic effects by infiltrating a variety of cell types, including epithelial and endothelial cells, pericytes and fibroblasts, resulting in lymphangitis and vasculitis. During the acute stage vasculitis and lymphangitis with concomitant thrombosis and infarction resulted in edema and necrosis [34]. The lesions were initially infiltrated by neutrophils and macrophages, and later on these cells were gradually replaced by lymphocytes, plasma cells and macrophages, as well as fibroblasts [14, 34].

3. Clinical signs

The clinical manifestations of LSDV in experimental and naturally occurring infections have been documented. Under field conditions, the incubation period is 1-4 weeks; with experimental inoculation, it is between 7-14 days [3, 9, 14]. The course of the disease may be acute, sub-acute, chronic or subclinical. The clinical disease is characterized by a biphasic febrile reaction that can

reach up to 41^{o}c. This may persist for 4 to 14 days. Clinical signs observed during this stage includes: depression, disinclination to move, inappetence, salivation, lachrymation and a nasal discharge, which may be mucoid or mucopurulent. Lachrymation may be followed by conjunctivitis and, in some cases, by corneal opacity and blindness. The superficial lymph nodes, especially prescapular, precrural and subparotid are usually markedly enlarged [3, 7, 14, 31, 34].

Skin nodules are classical manifestation of LSD. These nodules are usually widespread and they may be very numerous and cover the entire body or there may be only a few of them. Predilection sites are the skin of the head, neck, perineum, genitalia udder and limbs. Nodules are 5 to 50 mm in diameter, circumscribed, firm and round, raised, and involve the skin, subcutaneous tissue and sometimes even the underlying muscles. Ulcerative lesions may appear on the conjunctiva, muzzle, and nostrils, on the mucous membrane of the mouth, larynx, trachea, oesophagus and abomasum. Small nodules may resolve spontaneously, without any consequences or may become ulcerated and sequestered. Secondary bacterial infection or infestation by fly larvae may occur. Large nodules may become fibrotic and persist for several months [14, 16, 31, 34].

4. Diagnosis

The tentative diagnosis of LSD is usually based on characteristic clinical signs, and the clinical diagnosis is confirmed by various diagnostic techniques including; virus isolation in cell cultures, transmission electron microscopy, immunohistochemistry, direct and indirect fluorescent antibody tests, agar-gel immunoduffusion, and enzyme linked immunosorbent assay (ELISA), western blot and serum neutralization test (SNT). Molecular diagnostic methods being used include conventional PCR [9, 10]; real-time PCR [16] and dot blot hybridization [11].

Lumpy skin disease virus can be cultured in a large variety of tissue cultures: lamb and calf kidney cells, lamb and calf testis cells, sheep kidney cells, lamb and calf adrenal and thyroid cultures, foetal lamb and calf muscle cells, sheep embryonic kidney and lung cells, rabbit foetal kidney and skin cells, chicken embryo fibroblasts, on the chorioallantoic membrane of embryonated chicken eggs, African green monkey kidney (Vero) cells , baby hamster kidney cells, primary cell cultures of bovine dermis and equine lung cells [35-37]. The development of cytopathic effects (CPE) may take up to 14 days during primary isolation and the development of

cytopathic effects (CPE) is characterized by rounding, shrinking and detachment of cells to give a moth-eaten appearance to the monolayer [1, 7].

The SNT is the most specific serological test and gold standard for detecting antibodies against LSDV but it is very time consuming to perform [1, 16]. The sensitivity of the SNT in the presence of low levels of neutralizing antibodies in tested sera has been reported and should always be considered when interpreting the results [16]. Therefore, a negative result does not necessarily indicate the animal has not been exposed to the virus. The sensitivity and specificity of the SNT is 78% and 97% respectively [38]. This is due to the fact that LSDV infection predominantly provokes a cell-mediated immune response [1].

Fluorescent antibody techniques can be used to detect LSDV [17, 38]. However this technique is prone to cross-reaction with other *Parapox viruses.* Such cross-reaction has not been observed with the SNT. This technique is also less specific than the SNT. Western blotting is also used to detect LSDV with reliable specificity and sensitivity; however, these assays are expensive and need specialised equipment and training to be performed [1].

5. Control and Prevention

Different live attenuated strains of *Capripox virus* have been used as vaccines for the control of lumpy skin disease as described as follows. The Kenyan sheep and goat pox vaccine is a freeze-dried live vaccine based on a local strain of sheep and goat poxvirus produced at the Veterinary Research Laboratory, Kabete, Kenya and it was passaged 18 times in pre-pubertal lamb testes or foetal muscle cell cultures and used for vaccination at this level [1].This was shown to immunize cattle against LSD [15]. Local reactions have not been seen, but some *Bos taurus* breeds have shown lymphadenitis with signs of mild, generalized LSD-like lesions following vaccination [39]. The Neethling strain of LSDV vaccine is also a freeze-dried product produced by the Onderstepoort Biological Products, Onderstepoort, South Africa and was passaged 60 times in tissue cultures of lamb kidney cells and then 20 times in embryonated eggs [1]. The strain proved to be innocuous and immunogenic for cattle, although local reactions do occur in a high proportion of animals at the vaccination site [13].

Two other strains of sheep pox vaccine have been also used in cattle in the control of LSD. The Romanian strain, prepared in the skin of lambs for use against sheep pox, was used in cattle in

Egypt and appeared to be immunogenic [40]. But did not provide cattle with complete protection against LSD since outbreaks were reported in cattle in Egypt in 2006 after vaccination with sheep pox vaccine [41]. Another sheep pox strain, the RM 65 prepared in tissue culture, was used in Israel. No complications have followed the use of these strains in cattle. However, outbreak in cattle has been reported in Israel during 2006 to 2007 periods after vaccination with the RM65 sheep pox vaccine [42].

6. Economic impact of lumpy skin disease

The World Organization for Animal Health (OIE) categorizes LSD as a notifiable disease because of the substantial economic impact of an outbreak. The morbidity rate can be varies between 1- 100% and the mortality rate is usually less than 10% but has been as high as 20 - 75% in some outbreaks [2, 3, 16]. The disease is more severe in cows in the peak of lactation and causes a sharp drop in milk yield because of high fever caused by the viral infection itself and secondary bacterial infection causes mastitis [1]. Temporary or permanent infertility may occur in cows and bulls. Emaciation of infected animals and a convalescence period lasting for several months may cause decreased growth rate in beef cattle, the pregnant animals may abort, and deep skin lesions leave permanent scars and decrease the value of hides which affects leather industries [13, 30].

Restrictions to the global trade of live animals and animal products, costly control and eradication measures such as vaccination campaigns as well as the indirect costs because of the compulsory limitations in animal movements cause significant financial losses on a national level. In intensive cattle farming units, direct and indirect production losses caused by LSD have been estimated to be as high as 45–65%. In developing countries, the poorest small-scale farmers and rural communities, whose livelihood is totally dependent on cattle, bear the heaviest burden during outbreaks [33].

7. Conclusion and Recommendation

Lumpy skin disease is an infectious disease of cattle caused by a double stranded lumpy skin disease virus. The disease might be occurring in the acute or sub-acute forms. The disease has significant economic importance to cattle industry. Usually the disease is transmitted by biting insects and the incidence of the disease is high during wet seasons when biting insect populations

are abundant and decreases during the dry season. Appropriate and protective vaccine type with the proper season is very important for the control and prevention of Lumpy skin disease.

8. References

1. OIE (2010): Lumpy skin disease. In: manual of diagnostic tests and vaccines for terrestrial animals. Office International des Epizooties, World Organization for Animal Health, Paris. Pp: 1-13.
2. Salib F.A. and Osman A.H. (2011): Incidence of lumpy skin disease among Egyptian cattle in Giza Governorate, Egypt. *Vet World*, 4: 162-167.
3. Coetzer J.A.W. (2004): Lumpy skin disease. In: Infectious Diseases of Livestock. 2nd ed. Coetzer J.A.W. and Justin R.C. (eds). Oxford University Press, Cape Town, South Africa, pp: 1268-1276.
4. Tuppurainen E.S.M. and Oura C.A.L. (2012): Review; Lumpy skin disease: An emerging threat to Europe, Middle East and Asia. *Transbound Emerg Dis.*, 59: 40-48.
5. Woods J.A. (1988): Lumpy skin disease a review. *Tropical Animal Health and Production,* 20: 11-17.
6. Kithing R.P. and Hammond J.M. (1992): Poxvirus infection and immunity. In: Encyclopaedia of immunology Vol. 3 (Roitt I.M. and Delves P.J. 3rd ed.), Academic press, London pp. 1261-1264.
7. Davis F.G. (1991): Lumpy skin disease, an African *Capripox virus* disease of cattle. *Br Vet J.*, 147: 489-502.
8. Gari G., Waret-Szkuta A., Grosbois V., Jacquiet P. and Roger F. (2010): Risk factors associated with observed clinical lumpy skin disease in Ethiopia. *Epidemiol Infec.*, 138: 1657-1666.
9. Tuppurainen E.S.M., Venter E.H. and Coetzer J.A.W. (2005): The detection of lumpy skin disease virus in samples of experimentally infected cattle using different diagnostic techniques. *Onderstepoort J. Vet. Res.*, 72: 153–164.
10. Zheng M., Liu Q., Jin N.Y., Guo J.G., Huang X., Li H.M., Zhu W. and Xiong Y. (2007): A duplex PCR assay for simultaneous detection and differentiation of *Capripox virus* and *Orf virus*. *Mol. Cell. Probes,* 21: 276–281.
11. Awad W.S., Ibrahim A.K., Mahran K., Fararh K.M. and Abdel-Moniem M.I. (2010): Evaluation of different diagnostic methods for diagnosis of lumpy skin disease in cows. *Tropical animal and health production,* 42: 777-783.

12. Anon (2010): Lumpy skin disease, http://www.oie.int/wahis/public.php. WAHID Interface-OIE World Animal Health information, Paris.

13. Weiss K.E. (1968): Lumpy skin disease. Virology Monographs. Springer Verlag, Vienna, New York pp. 111-131.

14. Carn V.M. and Kitching R.P. (1995): The clinical response of cattle following infection with lumpy skin disease (Neethling) virus. *Arch Virol.*, 140: 503–513.

15. Carn V.M. (1993). Control of *Capripoxvirus* infections. *Vaccine,* 11: 1275-1279.

16. Babiuk S., Bowden T.R., Boyle D.B., Wallace D.B., and Kitching R.P. (2008): *Capripoxviruses:* an emerging worldwide threat to sheep, goats and cattle. *Transbound Emerg Dis.*, 55:263-272.

17. Davies F.G. and Otema C. (1981): Relationships of *Capripox virus* found in Kenya with two Middle Eastern strains and some *Orthopox viruses*. *Research in Veterinary Science,* 31: 253-255.

18. Radostits O.M., Gay C.C., Hinchcliff K.W. and Constable P.D. (2007): Veterinary Medicine: A textbook of diseases of cattle, horses, sheep, pigs and goat. 10th ed. WB Saunders Co. Philadelphia, USA. Pp 1424-1426.

19. Tulman E.R., Afonso C.L., Zsak Z.Lu, L., Sur J.H., Sandybaev N.T., Kerembekova U. Z., Zaitsev V.L., Kutish G. F. and Rock D. L. (2002): The genomes of sheep pox and goat pox viruses. *J. Virol.,* 76: 6054–6061.

20. Tulman E.R., Afonso C.L., Zsak L.L., Kutish G.F. and Rock D.L. (2001): Genome of Lumpy skin disease virus. *J. Virol.,* 75: 7122-7130.

21. Kara P. D., Afonso C. L., Wallace D. B., Kutish G. F., Abolnik C., Vreede Z. Lu, F. T., Taljaard L. C., Zsak A., Viljoen G. J. and Rock D. L. (2003): Comparative sequence analysis of the South African vaccine strain and two virulent field isolates of lumpy skin disease virus. *Arch. Virol.,* 148: 1335–1356.

22. Fenner F., Bachmann P.A., Gibbs E.P.J., Murphy F.A., Studdert M.J. and White D.O. (1987): *Poxviridae*. Veterinary Virology. New York, London, Sydney, Tokyo, Toronto: Academic Press, 387-405.

23. African Union Inter-African Bureau for Animal Resources (AU-IBAR) (2011): Pan-African Animal Health Yearbook, Kenindia Business Park, Museum Hill, Westlands Road, Kenya, pp 26-27.

24. Kumar S.M. (2011): An outbreak of lumpy skin disease in a Holstein dairy herd in Oman: A clinical report. *Asian Journal of Animal and Veterinary Advances,* 6: 851-859.

25. Anonymous (2011): World Animal Health Information Database. Disease information: lumpy skin disease http://web.oie.int/wahis.

26. Ali A.A., Esmat M., Selim A. and Abdel-Hamid Y.M. (1990): Clinical and pathological studies on lumpy skin disease in Egypt. *Veterinary Record*, 127: 549-550.

27. Shawari S.S and Abd El-Rahim I.H. (2011): The utility of polymerase chain reaction for the diagnosis of lumpy skin disease in cattle and water buffaloes in Egypt. *Revue Scintifique et Technique Office International des Epizooties*, 30: 821-830.

28. Hedger R.S. and Hamblin C. (1983): Neutralising antibodies to lumpy skin disease virus in African wildlife. *Comp. Immunol. Microbiol. Infect. Dis.*, 6: 209-213.

29. Hosamani M., Mondal B., Tembhurne P. A., Bandyopadhyay S. K., Singh R. K. and Rasool T.J. (2004): Differentiation of sheep pox and goat poxviruses by sequence analysis and PCR-RFLP of P32 gene. *Virus Genes*, 29: 73–80.

30. Irons P.C., Tuppurainen E.S.M. and Venter E.H. (2005): Excretion of lumpy skin disease virus in bull semen. *Theriogenology*, 63: 1290-1297.

31. Barnard B.J.H, Munz E., Dumbell K. and Prozesky L. (1994): Lumpy Skin Disease. In: Coetzer, J.A.W., Thomson, G.R., Tustin, R.C. (eds) Infectious diseases of livestock with special reference to Southern Africa. Oxford University Press, Cape Town, 604-612.

32. Chihota C.M. Rennie L.F., Kitching R.P. and Mellor P.S. (2001): Mechanical transmission of lumpy skin disease virus by *Ades aegypti* (Diptera: Culicidae). *Epidemiol Infect.*, 126: 317-321.

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

34. Prozesky L. and Barnard B.J.H. (1982): A study of the pathology of lumpy skin disease in cattle. *Onderstepoort J Vet Res.*, 49: 167-175.

35. Binepal Y., Ongadi F. and Chepkwony J. (2001): Alternative cell lines for the propagation of Lumpy skin disease virus. *Onderstepoort J. Vet. Res.*, 68: 151-153.

36. Bagla V.P., Osuagwuh U.I., Annandale C.H., Irons P.C. and Venter E.H. (2006): Elimination of toxicity and enhanced detection of lumpy skin disease virus on cell culture from experimentally infected bovine semen samples. *Onderstepoort journal of veterinary research*, 73: 263-268.

37. Babiuk S., Parkyn G., Copps J., Larence J., Sabara M., Bowden T., Boyle D. and Kitching R.P. (2007): Evaluation of an Ovine testis cell line (OA3.Ts) for propagation of

Capripoxvirus isolations and development of an Immunostaining technique for viral plaque visualization. *J. Vet. Diagn. Invest,* 19: 486-491.

38. Gari G., Biteau-Coroller F., LeGoff C., Caufour P. and Roger F. (2008): Evaluation of indirect fluorescent antibody test (IFAT) for the diagnosis and screening of lumpy skin disease using Bayesian method. *Vet. Microbiol.,* 129: 269-280.
39. Yeruham I., Perl S., Nyska A., Abraham A., Davidson M., Haymovitch M., Zamir O. and Grinstein H. (1994): Adverse reactions in cattle to a Capripox vaccine. *Vet. Rec.,* 135: 330-332.
40. Michael A., Saber M., Sooliman S., Mousa A., Salama S., Fayed A., Nassar M. and House J. (1996): Control of lumpy skin disease in Egypt with Romanian sheep pox vaccine. *Assiut.Vet. Med. J.,* 36: 173-180.
41. El-Kholy A.A. Soliman H.M.T. and Abdelrahman K.A. (2008): Polymerase chain reaction for rapid diagnosis of a recent lumpy skin disease virus incursion to Egypt. *Arab J. Biotechnol.,* 11: 293-302.
42. Brenner J., Bellaiche M., Gross E., Elad D., Oved Z., Haimovitz M., Wasserman A., Friedgut O., Stram Y., Bumbarov V. and Yadin H., (2009). Appearance of skin lesions in cattle population vaccinated against lumpy skin disease: statutory challenge. *Vaccine,* 27: 1500-1503.