



# Lumpy skin disease: Preliminary vaccine efficacy assessment and overview on outbreak impact in dairy cattle at Debre Zeit, central Ethiopia



Gelagay Ayelet<sup>a,\*</sup>, Yebeyen Abate<sup>c</sup>, Tesfaye Sisay<sup>c</sup>, Haileleul Nigussie<sup>c</sup>, Esayas Gelaye<sup>a</sup>, Shiferaw Jemberie<sup>a</sup>, Kassahun Asmare<sup>b</sup>

<sup>a</sup> National Veterinary Institute, P.O. Box 19, Debre Zeit, Ethiopia

<sup>b</sup> School of Veterinary Medicine, Hawassa University, P.O. Box 005, Hawassa, Ethiopia

<sup>c</sup> School of Veterinary Medicine, Addis Ababa University, P.O. Box 34, Debre Zeit, Ethiopia

## ARTICLE INFO

### Article history:

Received 14 November 2012

Revised 30 January 2013

Accepted 4 February 2013

Available online 19 February 2013

### Keywords:

Lumpy skin disease  
Vaccine efficacy  
Economic impact  
Dairy animals  
Ethiopia

## ABSTRACT

This study was conducted in and around Debre Zeit town to assess the field efficacy of LSD vaccine in use and overview associated disease impact. The study comprised cross-sectional and retrospective study design which employed active disease follow-up, semi-structured questionnaire survey and molecular techniques. The finding revealed that the Kenyan sheep pox vaccine strain used for the control of LSD did not confer expected protection. From the total of 476 animals observed, 22.9% and 2.31% cattle were found sick and dead due to LSD, respectively. Breed specific morbidity rate was 22.5% in Holstein Friesian–zebu cross and 25.9% in local zebu breed. The disease was observed to be more serious in young animals and also in females. A trend of seasonality was also observed in its occurrence. The study finding urges the need for investigation of vaccine failure including vaccine matching and alternative vaccine development.

© 2013 Published by Elsevier B.V.

## 1. Introduction

Lumpy skin disease (LSD) is a pox of cattle characterized by fever, nodules on the skin, mucous membrane and internal organs. LSD is caused by a double stranded DNA virus in the genus *Capripoxvirus* of the family *Poxviridae*, which is antigenically closely related to sheep and goat poxviruses (OIE, 2010). LSD was first described in 1929 in Zambia from where the disease spreads to other southern African countries by the 1940s. In 1970 it spread north into the Sudan and between 1981 and 1986 reached to Tanzania, Kenya, Zimbabwe, Somalia and Cameroon (Davies, 1982; Lefevre et al., 1979; Nawathe et al., 1982, 1978; OIE, 2010; Pierre, 1978). The disease is now enzootic throughout sub-Saharan Africa including Madagascar (Tuppurainen and Oura, 2012). In Ethiopia lumpy skin disease was first observed in the northwestern part of the country (southwest of Lake Tana) in 1983 (Mebratu et al., 1984). It has now spread to almost all regions and agro-ecological zones of the country. Because of the wide distribution of the disease and the size and structure of the cattle population in Ethiopia, it is likely that LSD is among one of the most economically impor-

tant livestock diseases in the country (GARI et al., 2011, 2012, 2010).

LSD is more prevalent during the wet summer and autumn months and occurs particularly in low-lying areas and along water-courses (Carn and Kitching, 1995b; Davies et al., 1971; OIE, 2010). Transmission is predominantly via arthropod vectors including hard ticks, biting flies and/or mosquitoes. Direct transmission is also possible through saliva, semen, milk, or contact directly with lesions of infected cattle, however the direct course is less efficient in the absence of insect vectors (Carn and Kitching, 1995a). The mathematical modeling recently conducted is also concluded that transmission mostly occurs by indirect contact, probably by flying and blood-sucking insects (Magori-Cohen et al., 2012). LSD virus has been found in mosquitoes in the genera *Aedes* and *Culex* during some outbreaks (Chihota et al., 2001). Flies such as *Stomoxys calcitrans* and other insects might also be involved in transmission (Babiuk et al., 2008; Chihota et al., 2003). Recently, new evidence has been reported about a possible role of ticks in the transmission of LSDV (Tuppurainen et al., 2011). The study showed molecular evidence of trans-stadial and trans-ovarial transmission of LSDV by *Rhipicephalus ecoloratus* ticks and mechanical or intrastadial transmission by *Rhipicephalus appendiculatus* and *Amblyomma hebraeum* ticks (Tuppurainen et al., 2011).

\* Corresponding author.

E-mail address: [gelagayayelet@yahoo.com](mailto:gelagayayelet@yahoo.com) (G. Ayelet).

Lumpy skin disease in cattle can range from acute to sub-clinical forms (Babiuk et al., 2008; OIE, 2010). Morbidity and mortality of the disease vary considerably depending on the breed of cattle, the immunological status of the population and insect vectors involved in the transmission. *Bos taurus* (European cattle breed) develop more severe disease than local Zebu (*bos indicus*). Also, lactating cows of either breed are severely affected by LSD (OIE, 2010). The morbidity can reach as high as 100% in natural outbreaks while mortality rate rarely exceeds 5% (Babiuk et al., 2008; Irons et al., 2008). Decreased milk production, abortion, infertility, loss of condition and damaged hides cause enormous economic losses (Babiuk et al., 2008).

Four live attenuated capripox virus strains are currently used for vaccine production. They are Kenyan sheep pox, Yugoslavian RM 65, Romanian sheep pox, and South African neethling strains (OIE, 2010). A major neutralizing site of the capripox virus strains is shared by all three strains so that all have been used in vaccinations of cattle to protect against LSD infection in different parts of the world (OIE, 2010). However the control intervention for 2006 and 2007 LSD epidemic in Israel could not effectively limit the occurrence of LSD after RM65 strain vaccination (Brenner et al., 2009). Similarly, in Ethiopia, veterinarians and other animal health professionals have been observing a lack of efficacy of LSD vaccine when massive epidemic occurred among the vaccinated cattle population (NVI Personal communication). This incident made the authors question the validity of the assumption that has been in place for decades. Therefore, this study was initiated to assess the efficacy of the vaccine in field conditions and determine the associated risk factors for the occurrences of the disease.

## 2. Materials and methods

### 2.1. Description of the study area

The study was conducted from November 2010 to March 2011 in and around Debre Zeit town. The town is located at 8°45'N latitude and 38°59'E longitude at an altitude of 1,880 m above sea level. The mean annual rain is approximately 1,150 mm of which 84% rains from June to August. The district has a mean annual minimum and maximum temperature of 8.5 °C and 30.7 °C, respectively.

### 2.2. Study design and sampling

In response to LSD vaccination failure feedback obtained from farm owners and animal health professionals, the National Veterinary institute (NVI, Ethiopia) launched a preliminary field investigation in and around Debre Zeit. The assessment was a combination of active disease follow-up, questionnaire and retrospective data collection focused on two governmental dairy farms and 60 urban and peri-urban dairy farms involving 476 animals with different breeds (Local Zebu, Holstein Friesian–Zebu cross), age and sex groups. The farms under investigation were all reported to have suffered LSD outbreak.

### 2.3. Questionnaire survey and epidemiological data collection

A semi-structured questionnaire format was used to generate information related to LSD occurrence, history of vaccination, individual animal biodata, and disease associated losses including death, abortion, and reduction in milk yield. Clinical investigation of sick animals was also conducted when encountered. Retrospective investigation of outbreaks between 2007 and 2010 was also made.

### 2.4. Sample collection, submission and preparation

The farms under investigation were selected based on the outbreak report made during the study period to the institute. In addition to the collection of data in questionnaires, biopsy samples of skin nodules were collected for virus isolation and antigen detection. Samples for virus isolation were collected within the first week of the occurrence of clinical signs, before the development of neutralizing antibodies (OIE, 2010). The collected samples were kept at –20 °C until processed for virus isolation.

### 2.5. Cell culture

Vero cell was used for LSD virus isolation. The biopsy samples were thawed at room temperature and washed three times using sterile phosphate buffer saline (PBS) at a pH of 7.2 under laminar air flow hood. About 1 g of the samples was homogenized using sterile mortar and pestle by adding 10 ml of sterile PBS containing antibiotic (gentamycin). The tissue suspension was centrifuged at 1500 rpm for 15 min. The supernatant was collected and filtered by filter of 0.45 µm pore size (Millipore, USA). About 1 ml of filtered tissue suspension was inoculated on Vero monolayer cells grown on 25 cm<sup>2</sup> tissue culture flask and then flashed with growth media and incubated at 37 °C and 5% CO<sub>2</sub> in a humidified incubator for 48 h. Cells were monitored for cytopathic effect (CPE) daily, and frozen when CPE was exhibited.

### 2.6. Polymerase chain reaction

Tissue samples collected from LSD sick animals and cell culture positive samples were tested with polymerase chain reaction (PCR) as described on OIE (2010). DNA was extracted by using the DNeasy Tissue Kit (Qiagen, Germany) according to the manufacturer's instructions. The PCR tests were performed targeting for amplification of a specific segment of 192 pb at gene responsible for viral attachment protein of capripox virus (Ireland and Binepal, 1998; Irons et al., 2008). DNA amplification was carried out in a final volume of 50 µl containing: 5 µl of 10 × PCR buffer, 1.5 µl of MgCl<sub>2</sub> (50 mM), 1 µl of dNTP (10 mM), 1 µl of forward primer, 1 µl of reverse primer, 5 µl of DNA template, 0.5 µl of *Taq* DNA polymerase and 35 µl of nuclease-free water. The PCR was conducted using capripox virus specific primers: Forward primer 5'-TTT-CCT-GAT-TTT-TCT-TAC-TAT-3' and Reverse primer 5'-AAA-TTA-TAT-ACG-TAA-ATA-AC-3' the reaction conditions were: 95 °C for 1 min, 35 cycles of 94 °C for 45 s, 50 °C for 50 s and 72 °C for 1 min and a final extension step of 72 °C for 5 min. For further confirmation the PCR product was sequenced.

### 2.7. Data management and analysis

The collected data were entered and stored into Microsoft Excel spread sheet 2007. The data were thoroughly cleaned and properly coded before subjecting to statistical analysis. The analysis was done using Stata version SE 11.0 (StataCorp., Texas, USA). Descriptive statistics was used to the findings.

## 3. Results

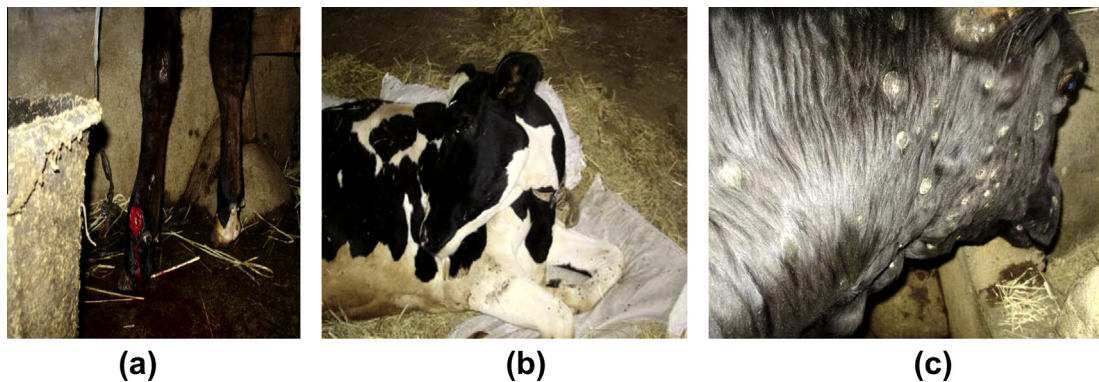
### 3.1. Vaccination and clinical LSD

Only 39 out of 476 animals were not vaccinated. In the investigation, it was recorded that 22.9% (109/476) of them reported to have suffered the disease and 2.31% (11/476) of them eventually died of it. Among the sick ones 95.4% (104/109) of them and all the dead were vaccinated against LSD (Table 1).

**Table 1**

Morbidity, mortality and case fatality rate of LSD and their association with different risk factors.

Risk factors	No. at risk	No. of sick	No. of death	Morbidity rate	$\chi^2$	Mortality rate	$\chi^2$	Case fatality rate	$\chi^2$
<i>Breed</i>									
Cross	422	95	11	22.51		2.61		11.58	
Local	54	14	0	25.93		0.00		0.00	
Total	476	109	11	22.90	0.32	2.31	1.44	10.09	1.8
<i>Age</i>									
<2 years	132	80	5	60.61		3.79		6.25	
2–4 years	156	18	1	11.54		0.64		5.56	
>4 years	186	11	5	5.91		2.69		45.45	
Total	476	109	11	22.90	76.6	2.31	2.94	10.09	15.2
<i>Sex</i>									
Female	355	97	7	27.32		1.97		7.22	
Male	122	12	4	9.84		3.28		33.33	
Total	476	109	11	22.90	10.2	2.31	0.69	10.09	8.03
<i>Vaccination</i>									
Vaccinated	437	104	11	23.80		2.52		10.58	
Non-Vaccinated	39	5	0	12.82		0.00		0.00	
Total	476	109	11	22.90	2.44	2.31	0.90	10.09	1.16

**Fig. 1.** Eroded lesion on the foot of LSD infected Heifer (a), LSD infected cow which was unable to stand (b), and nodular lesion on the skin of LSD infected heifer (c).

Upon clinical examination, animals were febrile, dull, limping and in some case reluctant to move and, in some cases, unable to stand. Gross lesions seen on the skin were nodules of variable size (Fig. 1). Breed specific morbidity, mortality, and case fatality rates were analyzed as depicted (Table 1). The morbidity rate was nearly similar with 22.5% (95/422) and 25.9% (14/54) for cross and local breed, respectively. The mortality and case fatality rate was 2.61% (11/422) and 11.58% (11/95) respectively in cross breed, while death was not recorded in local breed of cattle.

The protection rate of the vaccine within the breed was compared (Table 2). In Holstein Fresian–Zebu cross breed the morbidity rate was 21.8% (94/418) in vaccinated and 25% (1/4) in non-vaccinated animals, which is not statistically significant ( $P > 0.05$ ). But among local Zebu cattle the morbidity rate was 58.8% (10/17) in vaccinated and 10.8% (4/37) in non-vaccinated cattle, which is highly statistically significant ( $P < 0.001$ ).

**Table 2**

Morbidity rate of LSD in local zebu and Holstein fresian cross breed cattle.

Breed	Vaccination status	No. at risk	No. of sick	Morbidity	P-value
Local	Vaccinated	17	10	58.8	0.0003
	Not vaccinated	37	4	10.8	
	Total	54	14	25.9	
Cross	Vaccinated	418	94	21.8	0.87
	Not vaccinated	4	1	25	
	Total	422	95	22.5	

The observed morbidity rate was higher in age category less than 2 years (80/132, 60.6%) and lower in adults (11/186, 5.9%). The mortality rate was higher in female (97/355, 27.32%) than male (12/122, 9.84%) whose difference was statistically significant ( $P < 0.05$ ), while the case fatality was higher in male (4/11, 33.3%). The highest (5/132, 3.8%) mortality rate was recorded in age category less than 2 years. In terms of case fatality, the highest proportion was recorded among adults above 4 years of age (5/11, 45.45%). Details are given in Table 1.

### 3.2. Impact of LSD on milk yield

According to the respondents, the reduction in milk production of 2–14 l per lactating cow with an average of 5 l/day for duration of 7 to 30 days (mean 19.3 days) occurred. The monetary loss was estimated to be 778 birr (44.7 USD, 1USD = 17.4 birr) per cow since milk prices are roughly 8 birr/l. Farmers estimated the mean veterinary costs per sick animals to be up to 75 birr (4.3 USD). Losses of 17,000 birr (~997 USD) per cow occurred due to death. In addition, abortion and still birth of calves could produce more economic losses due to a decreased reproduction (Table 3).

### 3.3. Seasonal occurrence of LSD

Regarding seasonality, most respondents observed a sporadic form of LSD from August 2008 to May 2009, but an epidemic occurred from August 2010 to November 2010. The respondents also replied that the incidence increased during rainy season. The

**Table 3**

Impact on milk production and reproduction of cows infected with LSD as per interviewees' respondents.

Parameters	Mean	Range
Reduction in milk yield/liter during infection	5	2–14
Milk reduction/loss duration (days)	19.3	7–30
Abortion/still birth (%)	16.7	
Treatment cost/USD	4.31	2.87–5.75
Death (%)	10.09 (case fatality)	(11/109)

livestock owners were interviewed about the possible source of infection. Thirty-three percent (6/18) selected a direct contact with sick animals. Twenty-two percent (4/18) selected the introduction of sick animals to the herd. The rest (45%) had no answer.

### 3.4. Virus isolation and PCR test result

Of 10 bovine skin samples collected and cultured on VERO monolayers, 7 developed cytopathic effect (CPE) typical of LSD virus. The infected cells developed a characteristic LSD virus CPE consisting of retraction of the cell membrane from surrounding cells and eventually rounding of cells between 4–6 days post inoculation. PCR analyses were performed on 7 of the CPE positive samples and 5 were positive in confirmation of LSD virus, amplified a DNA fragment of 192 bp equivalents to the expected amplification for LSD virus (Fig. 2). The sequence result also confirmed that the circulating virus is LSD virus (the data not presented in this paper).

## 4. Discussion

The present study showed that the Kenyan sheep pox vaccine strain in use for Lumpy Skin Disease control was not satisfactory in the study area. This finding is in agreement with the report in Egypt (Fayez and Ahmed, 2011) and in Israel (Brenner et al., 2009) who reported LSD outbreak occurrence after vaccination of cattle with sheep pox vaccine. The study in Israel indicated that a cutaneous clinical manifestation appeared, following re-exposure of cattle that had been vaccinated with the RM65 strain, to LSDV infection during an epidemic in 2006–2007 (Brenner et al., 2009). Although LSD virus is genetically and antigenically related to sheep pox virus (Bhanuprakash et al., 2006) and several researchers recommend use of vaccines derived from sheep pox virus, the current finding indicated that sheep pox virus vaccine (Kenyan strain) could not protect cattle against LSD. The result in this study showed that high rate of morbidity was observed in vaccinated groups than unvaccinated one, but the variation is not statistically significant ( $P > 0.05$ ). The high rate of morbidity was observed in vaccinated cattle of local breed compared with non-vaccinated

ones; this might be due to small number of animals observed during the study period. Death was not recorded in unvaccinated group, which might be confounded by breed factors since most local zebu cattle were not vaccinated and death was not recorded in local zebu breed.

Practically vaccine failure can be observed due to various reasons such as; Strain difference between the vaccine and field strain, low titre of the vaccine, vaccination of calves with maternal antibody (less 6 months of age), vaccination of animals already incubating the disease, confusion with 'pseudo lumpy skin' disease caused by *Allerton virus* (which is herpes virus) and mishandling of the vaccine during transport and storage (Hunter and Wallace, 2001). However most of the possible reasons mentioned above are unlikely to be responsible for this failure, as the vaccine used passed NVI quality control test before release and all animals above 6 months of age were vaccinated two months before the outbreak began. The farm records and questionnaire survey result showed that all the vaccinated animals were got vaccination with Kenyan sheep pox vaccine two months before the occurrence of the disease, animals were vaccinated in May/June time but the occurrence of the disease was reported since the end of August 2010. Moreover due to proximity of the study area to vaccine production institute all necessary precaution regarding cold chain and transportation was under follow up of experts. The laboratory investigation made on the virus isolated from outbreak samples was also diagnosed by PCR using capripox specific primers and also further confirmed by sequencing, which avoid confusion with pseudo lumpy skin disease. However observation of vaccine failure in this study might be due to lack of cross protection with vaccinal strain.

The study revealed that LSD is becoming one of the major diseases of cattle that occur as an epidemic in the area since 2008. During the study period the occurrence of LSD and its association with different risk factors were analyzed. Breed specific morbidity, mortality and case fatality rates were analyzed between cross and local breeds of cattle. The morbidity rate was nearly similar with 22.5% and 25.9% for cross and local breed, respectively. The morbidity of LSD varies from 3% to 85% and mortality never exceeds 3% (Babiuk et al., 2008; Tuppurainen and Oura, 2012). The mortality rate and case fatality rate was 2.61% and 11.58%, respectively, in cross breed, while death was not recorded in local breed of cattle. It appeared that the local zebu breeds examined in this study have less susceptibility to the disease than cross breed cattle, which is in agreement with the report (Davies, 1991; Woods, 1988) who reported the presence of breed variation in LSD susceptibility.

Age specific morbidity was higher in cattle less than 2 years compared with those groups above 4 years of ages. The result complies with the findings in Egypt (Fayez and Ahmed, 2011) who reported a higher morbidity in young age category. The higher morbidity rate reported in female may also be due to lactation

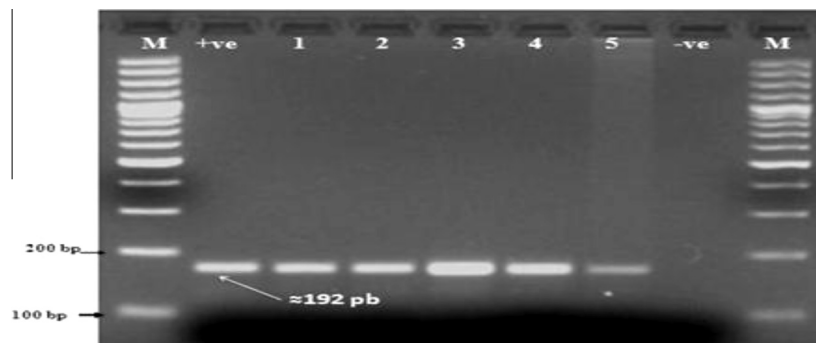


Fig. 2. PCR based detection of LSDV. Lane +ve: positive control; Lane -ve: negative control; Lanes 1 and 5 represent tested samples; M: molecular weight marker.

and/or pregnancy which are physiologically stressful and compromise immunity. The phenomenon further emphasizes disease related economic loss due to decline in milk reduction.

The occurrence of LSD outbreaks were recorded in rainy season which is in agreement with the report of (Fayez and Ahmed, 2011) since LSD is an arboviral viral disease its transmission is largely dependent of insect density which is believed to be highest during rainy season.

In conclusion, the preliminary observation made in this study suggests the need for evaluating the immunogenicity of LSD vaccine strain in use. The overview also highlights the existence of enormous economic loss associated with LSD. The seasonal trend observed was also an important observation to gear strategic approach in establishing herd immunity prior to the anticipated outbreak. Thus, the strain of the LSD virus circulating in the area needs to be characterized and also vaccine matching needs to be conducted. In a nutshell there is a need to develop effective and highly potent vaccine.

### Acknowledgement

The authors are grateful for the animal owners who allowed us to conduct this preliminary study in the area.

### References

- Babiuk, S., Bowden, T.R., Boyle, D.B., Wallace, D.B., Kitching, R.P., 2008. Capripoxviruses: an emerging worldwide threat to sheep, goats and cattle. *Transbound. Emerg. Dis.* 55, 263–272.
- Bhanuprakash, V., Indrani, B.K., Hosamani, M., Singh, R.K., 2006. The current status of sheep pox disease. *Comp. Immunol. Microbiol.* 29, 27–60.
- Brenner, J., Bellaiche, M., Gross, E., Elad, D., Oved, Z., Haimovitz, M., Wasserman, A., Friedgut, O., Stram, Y., Bumbarov, V., Yadin, H., 2009. Appearance of skin lesions in cattle populations vaccinated against lumpy skin disease: statutory challenge. *Vaccine* 27, 1500–1503.
- Carn, M., Kitching, P., 1995a. An investigation of possible routes of transmission of lumpy skin disease virus (Neethling). *Epidemiol. Infect.* 114, 214–226.
- Carn, V.M., Kitching, R.P., 1995b. The clinical-response of cattle experimentally infected with lumpy skin-disease (Neethling) virus. *Arch. Virol.* 140, 503–513.
- Chihota, C.M., Rennie, L.F., Kitching, R.P., Mellor, P.S., 2001. Mechanical transmission of lumpy skin disease virus by *Aedes aegypti* (Diptera: Culicidae). *Epidemiol. Infect.* 126, 317–321.
- Chihota, C.M., Rennie, L.F., Kitching, R.P., Mellor, P.S., 2003. Attempted mechanical transmission of lumpy skin disease virus by biting insects. *Med. Vet. Entomol.* 17, 294–300.
- Davies, F.G., 1982. Observations on the epidemiology of lumpy skin-disease in Kenya. *J. Hyg.* 88, 95–102.
- Davies, F.G., 1991. Lumpy skin-disease, an African capripox virus-disease of cattle. *Br. Vet. J.* 147, 489–503.
- Davies, F.G., Krauss, H., Lund, J., Taylor, M., 1971. Laboratory diagnosis of lumpy skin disease. *Res. Vet. Sci.* 12, 123–127.
- Fayez, A., Ahmed, H., 2011. Incidence of lumpy skin disease among Egyptian cattle in Giza Governorate, Egypt. *Vet. World* 4, 162–167.
- Gari, G., Bonnet, P., Roger, F., Waret-Szkuta, A., 2011. Epidemiological aspects and financial impact of lumpy skin disease in Ethiopia. *Prev. Vet. Med.* 102, 274–283.
- Gari, G., Grosbois, V., Waret-Szkuta, A., Babiuk, S., Jacquet, P., Roger, F., 2012. Lumpy skin disease in Ethiopia: seroprevalence study across different agro-climate zones. *Acta Trop.* 123, 101–106.
- Gari, G., Waret-Szkuta, A., Grosbois, V., Jacquet, P., Roger, F., 2010. Risk factors associated with observed clinical lumpy skin disease in Ethiopia. *Epidemiol. Infect.* 138, 1657–1666.
- Hunter, P., Wallace, D., 2001. Lumpy skin disease in southern Africa: a review of the disease and aspects of control. *J. S. Afr. Vet. Assoc.* 72, 68–71.
- Ireland, D.C., Binpal, Y.S., 1998. Improved detection of capripoxvirus in biopsy samples by PCR. *J. Virol. Methods* 74, 1–7.
- Irons, P.C., Luther, I., Ebersohn, K., Bosman, A.M., Annandale, C.H., Van Wilpe, E., Colenbrander, B., Venter, E.H., 2008. Effect of lumpy skin disease virus in bull semen on in vitro embryo production. *Reprod. Domest. Anim.* 43, 44.
- Lefevre, P.C., Bonnet, J.B., Vallat, B., 1979. Lumpy skin diseases. 1. Epizootiology in Africa. *Rev. Elev. Med. Vet. Pay.* 32, 227.
- Magori-Cohen, R., Louzoun, Y., Herziger, Y., Oron, E., Arazi, A., Tuppurainen, E., Shpigel, N.Y., Klement, E., 2012. Mathematical modelling and evaluation of the different routes of transmission of lumpy skin disease virus. *Vet. Res.*, 43.
- Mebratu, G., Kassa, B., Fikre, Y., Berhanu, B., 1984. Observations on the outbreak of lumpy skin disease in Ethiopia. *Vet. Tropicaux* 37, 395–399.
- Nawathe, D.R., Asagba, M.O., Abegunde, A., Ajayi, S.A., Durkwa, L., 1982. Some observations on the occurrence of lumpy skin-disease in Nigeria. *Zbl. Vet. Med. B* 29, 31–36.
- Nawathe, D.R., Gibbs, E.P.J., Asagba, M.O., Lawman, M.J.P., 1978. Lumpy-skin disease in Nigeria. *Trop. Anim. Health Prod.* 10, 49–54.
- 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.
- Pierre, F., 1978. Bovine lumpy skin-disease in Ivory-Coast. *Rev. Elev. Med. Vet. Pay.* 31, 281–286.
- Tuppurainen, E.S.M., Oura, C.A.L., 2012. Review: lumpy skin disease: an emerging threat to Europe, the Middle East and Asia. *Transbound. Emerg. Dis.* 59, 40–48.
- Tuppurainen, E.S.M., Stoltz, W.H., Troskie, M., Wallace, D.B., Oura, C.A.L., Mellor, P.S., Coetzer, J.A.W., 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.
- Woods, J.A., 1988. Lumpy skin-disease – a review. *Trop. Anim. Health Prod.* 20, 11–17.