SciMedCentral

Annals of Virology and Research

Review Article

Capripoxviruses: Transboundary Animal Diseases of Domestic Ruminants

Moustafa Kardjadj*

Ecole Supérieure en Science de l'Aliments (ESSA), Algeria

***Corresponding author**

Kardjadj Moustafa, Ecole Supérieure en Science de l'Aliments (ESSA), Algeria, Tel: 213542173600; Email: drkardjadj@live.fr

Submitted: 08 November 2016

Accepted: 06 December 2016

Published: 07 December 2016

Keywords

- Lumpy skin disease
- Sheeppox
- Goatpox
- Capripoxvirus

Abstract

Lumpy skin disease (LSD), sheeppox (SPP) and goatpox (GTP) are economically important Capripoxvirus (CaPV) diseases of domestic ruminants with substantial impact on the livelihoods of small-scale farmers and poor rural communities in endemic regions. The most striking similarity between different poxviruses is the clinical disease which they induce, characterized by pox lesions in the skin. LSD, SPP and GTP cannot be differentiated serologically, although distinct host preferences exist with most strains. This review briefly summarizes what is known about Capripoxviruses, including their current geographical distribution, economic impact, epidemiology, pathogenicity and control measures. Capripoxviruses have the potential to become emerging disease threats because of global climate change and changes in patterns of trade in animals and animal products.

INTRODUCTION

Livestock play an important socio-economic role in many marginal rural areas of the world. These herds provide food, hides and fiber for the inhabitants, making the economic survival of subsistence grazing systems possible. Therefore milk, and above all the meat obtained from goatherds meet the nutritional needs of the rural population in developing countries [1]. The livestock industry has the ability to improve the living standards of farmers and households, as well as increase animal protein for the inhabitants and consequently alleviating poverty [2]. However, transboundary animal diseases (TADs) pose a significant challenge to optimal and efficient management and profitable production of livestock. TADs are defined by FAO as those diseases that are of significant economic, trade and/or food security importance for a considerable number of countries; which can easily spread to other countries and reach epidemic proportions; and where control/management, including exclusion, requires cooperation between several countries [2].

Lumpy skin disease (LSD), sheeppox (SPP) and goatpox (GTP) are economically important *Capripoxvirus* (CaPV) diseases of domestic ruminants with substantial impact on the livelihoods of small-scale farmers and poor rural communities in endemic regions [3]. LSD, SPP and GTP are categorized by the OIE as notifiable diseases due to their potential for rapid spread and substantial economic impact. Diseases caused by the capripoxviruses are transboundary being significant impediments to trade in livestock and livestock products. This particularly affects the economic wellbeing of farmers in developing countries and would have substantial economic impacts on industrialized countries should the diseases be introduced to them [2-4].

Sheeppox virus (SPPV), goatpox virus (GTPV), and lumpy skin disease virus (LSDV) of cattle are the three species in the genus *Capripoxvirus,* subfamily *Chordopoxvirinae,* family *Poxviridae* [3,4]. Capripoxviruses are difficult to distinguish morphologically from orthopoxviruses and their DNA genomes have much in common, including the closed hairpin loops at their termini [5]. These are enveloped, double stranded DNA viruses, which show different levels of host adaptation for either sheep or goats in different parts of the world [4]. Others have a similar pathogenicity for both sheep and goats. The most striking similarity between different poxviruses is the clinical disease which they induce, characterized by pox lesions in the skin. LSD, SPP and GTP cannot be differentiated serologically, although distinct host preferences exist with most strains [3-5].

This review briefly summarizes what is known about capripoxviruses, including their impact on livestock production, their geographic range, host-specificity, clinical disease, transmission and control measures. Capripoxviruses have the potential to become emerging disease threats because of global climate change and changes in patterns of trade in animals and animal products.

Sheep and goat pox

Sheep and goat pox (SGPX) is probably the most serious infectious disease of small ruminants in many parts of the world [6]. The disease inflicts substantial losses in terms of reduced productivity and lower quality of wool and leather. It poses a

major obstacle in the intensive rearing of sheep and goats and also greatly hampers international trade. It is suggested that goat pox is the most important of all pox diseases of domestic animals causing high mortality in kids and significant economic losses [7]. The disease is characterized by skin lesions, occurring over the whole of the body, or restricted to the hairless areas of the perineum, head, groin, axillae and mammary glands. The lesions are also found in the oropharynx, in the lungs, alimentary tract and other organs. The morbidity and mortality rates can be very high, especially in totally susceptible populations with many neonates and young animals. The disease is highly infectious and the virus is resistant to desiccation, it remains viable in the environment and in scab debris for a long time [6,8,9].

Distribution: Sheep and goat pox are found in the extensive pastoral systems in the arid and semi arid zones of Asia and Africa, but also in the more settled livestock management systems in South East Europe. Animal movements for grazing and watering, for shearing and marketing, and trade movements are all associated with the mixing of large numbers of animals, which increases the risk of transmission. Infected flocks remain a source of the virus for several months after apparent recovery [6]. The infection was eradicated from Great Britain in 1866 and subsequently most western European countries have eliminated the disease by the slaughter of infected animals and the enforcement of strict movement control measures [9]. Irregular introductions occur from the adjacent endemic countries. Sporadic outbreaks still occur in Eastern European Mediterranean islands, probably originating from imported animals [9].

Historically, the global distribution of SPP and GTP has been wider than LSD. Indeed, cases of SPP and GTP regularly occur in northern and central Africa, across the Middle East and the Indian subcontinent, Iran, Iraq, Russia, Kazakhstan, Kyrgyzstan, Afghanistan, Pakistan, Nepal, Mongolia, China, Bangladesh, Vietnam and Chinese Taipei [10]. Many of these countries produce vaccines against sheep pox and against goat pox, and some conduct national disease control programs. The diseases are also endemic in Turkey and between 2013 and 2015 four outbreaks occurred in Bulgaria and several outbreaks were reported in Greece [10]. According to the OIE WAHID database [10], the incidence of SPP in Greece is still continuing in 2015 despite implementation of an extensive stamping out policy.

Hosts/Species affected: Capripoxviruses only infect ungulates, and most strains of virus tend to cause clinical disease in only one species [4]. Sheep and goats are the main hosts at risk from SGPX. Virtually all of the known species of goats and sheep from different parts of the world are susceptible to host-specific and other strains of the virus [6]. Only local lesions may follow the inoculation of some of the well adapted, host-specific strains in the alternative host, such as a host-adapted sheep pox into goats or vice versa. Of some of the viruses found in goats, Kenyan and Yemen isolates, as well as an Oman sheep isolate, infect sheep and goats equally [11,12]. Usually, Middle East and Indian isolates are host specific and do not infect sheep [13-15].

There is some variation in the susceptibility of different breeds and strains of sheep and goats to SGPX virus; this difference appears to be of genetic origin [13]. It is possible that the host preference shown by different strains is due to their adaptation to either goats or sheep in a restricted geographical area [6,9]. European breeds are particularly susceptible [9]. The disease causes high mortality, particularly if the infection is associated with other diseases such as peste des petits ruminants, or bad management [14]. Some strains of SGPX virus produce local necrotic lesions at the site of inoculation in cattle; this is particularly notable with the East African strains of SGPX virus, which are very closely related to lumpy skin disease virus [14]. Some mild generalization of the infection may follow the use of the Kenyan modified live virus SGPX vaccine strain for the prophylaxis of lumpy skin disease in Bos taurus breeds exotic to Africa (Bos indicus breeds are relatively resistant) [11].

Epidemiology: Close contact with infected or recovered animals is probably the most important mechanism for the transmission of SGPX; high titres of virus are present in the pustular exudates from lesions and in epithelial tissue and scab debris. Inhalation of infected droplets or aerosols from infected animals has been shown to cause infection [16]. Direct contact with infected sheep is the main means of infection, although the time taken for infection to become manifest in newly introduced sheep is surprisingly long, 20-40 days [6]. Infection of abrasions at shearing or other times with infected debris from scabs and lesions may also occur. The virus can be transmitted by scarification, by intradermal, subcutaneous, intranasal and intravenous inoculation. Mixing of sheep at markets and at watering holes is a common cause of spread of infection [9,16]. Indirect transmission may follow contact with infected premises such as pens or yards, the use of lorries, boats and shearing clippers, which have also been used by infected sheep or goats. Once a flock is infected, the disease will spread through all the animals within 6 to 12 weeks. The virus is very resistant and remains viable for long periods, on or off the animal host. They may persist for up to 6 months in shaded animal pens, and for at least 3 months in dry scabs on the fleece, skin and hair from infected animals [6,9,16]. There is no evidence of animals in a carrier state that are persistently infected with goat and sheep poxvirus [16].

Animals are most infectious soon after the appearance of papules, during the 10 days before the development of significant levels of protective antibody [16,19]. High titres of virus are present in papules, and those on the mucous membranes quickly ulcerate and release virus in nasal, oral and lachrymal secretions, and into milk, urine and semen, which all constitute important sources of virus dissemination [16]. Transmission of the disease frequently occurs by aerosols during direct or intimate contact between infected and susceptible animals, aerosol transmission may also occur from infected pustules and skin debris, although direct contact of virus with skin abrasions on the mouth or elsewhere is thought to play the major role [19]. Animals that develop generalized lesions produce considerable quantities of virus and are highly infectious. Transmission by fomites is probably not of major importance [18]. Experimentally, the disease can also be transmitted by intradermal, intravenous and subcutaneous inoculation as well as by artificially produced virus aerosols. The virus may be transmitted by Stomoxys [17], but the outbreaks are not exclusively confined to the fly biting seasons where these are short-lived and distinct. Other transmission

mechanisms are much more important.

Pathogenicity: The introduction of virus may be through the oral, nasal or respiratory epithelium or via the skin dermis or epidermis [6,16]. Local viral replication will occur in these affected epithelial tissues. Infected macrophage type cells are thought to transport the virus to the regional lymph nodes, where further viral replication takes place [18,19]. There is a marked proliferative response in the affected lymph nodes, which greatly increase in size [20]. A strictly cell-associated viraemia then occurs, which introduces infected cells (probably macrophages) throughout the body. The virus then replicates further in the epithelial tissues of the skin, lungs, endothelium, muscle and, more rarely, in nervous tissue. The basic lesions in the various affected tissues are due to a vasculitis, thrombosis and the resulting necrosis [6,20].

The incubation period for SGPX is 3-12 days following virus contact, a fever of between 40 and 41°C, which may not be detected, then occurs. Lymphadenopathy may be noticed at this stage and the skin lesions appear after a further 24-48 h. The course of the disease is then acute for 5-15 days, during which time the fever persists and the skin lesions remain for 4-12 weeks. Pneumonia is also common. Various degrees of debility and emaciation develop, depending on the extent and severity of the lesions. Healing of the skin lesions is complete after 2-3 months [6,9,19,20].

The initial clinical signs are of fever, depression, a disinclination to move, often lachrymitis and conjunctivitis, and rhinitis with serous nasal discharge [6,20]. Disease may appear more rapidly in goats than in sheep. Some enlargement of the superficial lymph nodes may be detected at this stage. After a further 24 to 48 h, local erythematous skin papules, irregularly round, of 3-25 mm in diameter may erupt, particularly on the hairless areas of the body such as the perineum, scrotum, axilla and groin, prepuce, mammary glands, muzzle and ears. The red areas are slightly raised above the areas of surrounding normal skin often with some serum exudation at the surface or oedema [6,9,20]. These may swell to form small papules with vesicular fluid or become hard and necrotic with scab formation at the surface within 6-12 days. Often the whole lesion becomes hard and indurated and gradually separates from the surrounding areas of normal unaffected skin over 4-8 weeks [20].

The presence of skin lesions over the whole of the body greatly restricts the movement of the affected animal. Lesions in the oropharynx affect the ability to feed, drink and move. The papules on the mucous membranes quickly ulcerate, and the secretions of rhinitis and conjunctivitis become mucopurulent. Upper respiratory and pneumonic lesions may cause stertorious respiration and respiratory distress [20]. Lesions on the udder and teats greatly interfere with suckling and even cause mastitis. The debility results in agalactia. The disease is accompanied by emaciation, and the inability to feed may result in mortality from associated causes. The respiratory lesions are often accompanied by a secondary pneumonia, which may be fatal. A hyperacute syndrome may occur in lambs and kids, with generalized lesions and death within 2-4 days of onset [6,9,20].

Prevention and control: Most live SGPX vaccines produce the lifelong immunity. In enzootic areas, both live attenuated and inactivated vaccines are useful in the prevention and control of goat pox, but inactivated vaccines give only short-term immunity [21,22]. Vaccination is recommended for animals of all ages and thereafter lambs and kids should be vaccinated annually, at 12-16 weeks of age, when the maternal antibody has disappeared. Where enclosed farming systems exist, the use of vaccine for several years will eliminate the disease completely, as long as vaccination is maintained on an annual basis for all young stock and great care is taken to introduce only vaccinated stock from clean areas [21,22]. Individual farms can maintain complete freedom from disease in this way and coordinated national programs can have a dramatic effect upon the disease. Coordinated control of movements from uninfected foci and complete restriction of movements from the infected areas will maintain the disease-free situation. Ring vaccination is frequently practiced during outbreaks in enzootic areas, but usually only the species that are clinically affected are vaccinated [21-23].

If national vaccination programs are established with strict quarantine and movement controls, and if disease foci are identified, they will have a dramatic effect upon SGPX in 3-5 years. A stage will be reached where a stamping out policy can be adopted for any new foci of disease. Absolute integrity and enforcement of movement controls is critical when the infected foci have been identified. If this can be achieved, SGPX eradication programs can be successful in quite a short time frame [22,23].

In endemic areas, a regular cleaning program for winter housing is essential to eliminate any residual virus that may remain dormant. Poxviruses are capable of long intervals between animal to animal transmissions. Owners often report the appearance of cases when they house the animals for the winter period. Virus may persist for several months in organic matter and this is even more essential, if there have been cases of disease. Thorough cleaning and removal of the dung and subsequent treatment with phenol, alkali or other suitable disinfectants is advisable to eliminate any residual virus [23].

Lumpy skin disease

A disease was first described in Northern Rhodesia (Zambia) in 1929, which was initially thought to be due to an allergic reaction in cattle to biting insects [24]. This was because it appeared usually at that time of year when populations of biting insects were at their greatest. It recurred fairly frequently there, and in 1943 the same syndrome was described in Botswana [25]. This raised further questions about its aetiology, and then in 1945 it was reported in Southern Rhodesia (Zimbabwe), Mozambique, and in South Africa [26].

LSDV is an occasionally fatal disease of cattle with morbidity averaging 10% and mortality 1% in affected herds, although mortality rates over 75% have been recorded [27]. Production losses are similar to SGPX with decreased weight gain, reduced milk production and damage to hides. The reasons for the wide ranges in mortality following infection with LSDV could be attributed to numerous factors that include the cattle breed, virus isolate, secondary bacterial infections, state of health of the animal, as well as the type of insect vector involved in

transmission [27-29].

The endemic geographic range of LSDV was limited to the continent of Africa (including Madagascar) [28], although recent outbreaks in Egypt spread into Israel [30]. An additional outbreak of LSD occurred in Egypt in 2006, having been introduced with foot and mouth disease by cattle imported from Ethiopia, and spread to Israel (World Animal Health Information Database, OIE) creating a real risk of LSDV establishing itself in the Middle East and spreading into Asia and Europe [10].

Hosts/Species affected: Natural infections with LSD have only been described in cattle in sub-Saharan Africa, and both Bos taurus and Bos indicus breeds, are susceptible. Bos taurus animals exotic to Africa are generally more susceptible than the zebu-type cattle, which are indigenous to sub-Saharan Africa. Sheep and goats develop LSD-like lesions, when inoculated intra-dermally with LSD virus, and cattle likewise develop a similar lesion when inoculated with KSGP virus [31]. Some mild generalised lesions can develop in Bos indicus cattle inoculated with the KSGPV vaccines which are used to protect sheep and goats against this disease, and these can be more serious in highly susceptible Bos taurus breeds [31]. Camels are not normally affected by LSD. A single natural clinical case of LSD was found in an Arabian oryx in Saudi Arabian zoo [32]. Experimental inoculation of impala (*Aepyceros melampus*), of Thomsons gazelle (*Gazella thomsonii*) and the giraffe (*Giraffa camelopardalis*) was followed by the development of LSD lesions in the skin [33]. Such lesions have not been observed during the LSD epizootics in Africa [31].

Epidemiology: Latency or intermittent secretion of virus does not occur with LSD. At the acute stage of the disease the nasal, lachrymal and pharyngeal secretions contain virus for 10-12 days and there may be contagion from such sources early in the course of the disease. Beads of infected serum appear on early skin lesions, which are infective and attract flies. Contagion does not occur readily with LSD, but can happen rarely if animals share water troughs. The duration of viraemia usually varies between 1 to 12 days. Viral DNA may be detected in blood samples using PCR method up to 17 days and in infected skin lesions for 4-6 months or longer. Poxviruses are extremely resistant to desiccation in tissue, and animals have been seen with LSD necrotic skin lesions in-situ 2 years after infection. A more recent study demonstrated the persistence of live virus in bovine [34].

The movement of animals from infected herds, often months after recovery, has regularly resulted in the introduction of infection. The source of the virus is considered to be from old skin lesions. In most of Sub Saharan Africa, the disease has been observed to appear following the seasonal rains, when there is always an increase in the population of different arthropod species. Local movement of the disease in the presence of strict quarantines has been attributed to aerial movement of insect vectors in low-level air currents. Direct contact is considered to be an ineffective means of transmission. Communal cattle grazing and watering points have been associated with the occurrence of LSD. Transmission of LSDV through semen (natural mating or artificial insemination) has not been experimentally demonstrated, but LSDV has been isolated in the semen of experimentally infected bulls [34,35].

The transmission of LSD by insects is thought to be mechanical, as it is with fowl pox; there is a wide range of biting flies with the potential to transmit virus. High morbidities are seen where mosquito populations are abundant, with 50-60% attack rates; and low, 5-15% morbidity in arid environments where there are fewer potential mechanical vectors [35,36]. Any biting fly could theoretically transmit a poxvirus after an interrupted feed on a viraemic host. The virus has been recovered from *Stomoxys* and *Biomyia spp*. in South Africa, and *Stomoxys* have been shown to be capable of transmitting Capripoxviruses. Mosquitoes have been found feeding upon cattle in huge numbers in epizootics and are capable of transmitting many viruses by mechanical means [36]. *Tabanids*, *Culicoides* and *Glossina* spp. may all have the potential to transmit LSD, as all feed voraciously upon domestic cattle [28,35]. Recently, new evidence has been published reporting a possible role for hard ticks in the transmission of LSDV [29]. The study showed molecular evidence of transstadial and transovarial transmission of LSDV by *Rhipicephalus* (*Boophilus*) decoloratus ticks, and mechanical or intrastadial transmission by *Rhipicephalus appendiculatus* and *Amblyomma hebraeum* ticks.

Pathogenicity: Animals of all age groups can become infected, and cases are common in young calves as well as all older age groups. The clinical picture of LSD in cattle follows an incubation period that varies from 4-12 days, and is usually about 7 days. There is a febrile reaction of 40-41.5°C, which may persist for 6-72 h or more rarely up to10 days. This is accompanied by lachrymation, increased nasal and pharyngeal secretions, anorexia, dysgalactia, general depression and a disinclination to move. There is great variation in the severity of these initial clinical signs, which do not relate to sex or age and they may be missed in extensively managed herds. Channel island breeds tend to more susceptible than others amongst Bos taurus types, and very severe cases are seen in many zebu breeds [24,27,37].

Within 1-2 days, there is a sudden eruption of nodules in the skin of the animals, which may be widespread or restricted to just a few lesions. Predilection sites are the head and neck, the perineum, the genitalia, udder, and the limbs. Frequently the whole of the skin is covered with lesions. These are 5-50 mm in diameter, which are irregularly round, and appear as circumscribed areas of erect hair over a firm and slightly raised area of skin [27]. These nodules later become necrotic and ulcerate giving rise to severe gastro-enteritis. Muco-purulent discharges appear from the nares, persistent dribbling from the mouth, coughing and often stertorious and distressed respiration if the larynx and trachea are involved. Keratitis is a common complication [27,35].

Pneumonia is a common sequel to LSD, which may be fatal. LSD lesions occur in the lungs as areas of grey consolidation measuring 20-30 mm. If these are widespread, interstitial pneumonia with consolidation and a fatal pneumonia may develop. Inhalation of necrotic tissue from lesions higher in the respiratory tract has been fatal many months after the initial infection. Abortion is common sequel of the acute phase of the disease; aborted foetuses and live calves have been observed with skin lesions of LSD. Infertility is a problem following LSD infection; females remain in anoestrous for several months [24,27].

Prevention and control: LSD has frequently been introduced into a previously unaffected area or country by the movement of live animals from a region or a country where LSD virus activity has occurred. A clinical examination of these animals may have been carried out, and they may have been in good health with no obvious signs of disease. To detect cattle with just a few lesions would require careful examination by a trained investigator, and missing recovered cases is all that would be required to allow the disease to be introduced [38]. The level of disease surveillance, which is necessary to detect low levels of LSD virus activity, is not widely available in many of the sub-Saharan countries, where animal health services operate with low budgets; active surveillance activities cannot be funded. The risk of LSD arising from local or regional movement of cattle is therefore high. Quarantine facilities in importing countries, where cattle can be held for inspection, are generally inadequate to control insect-borne diseases. Insect-proofed quarantine facilities are rare, and transmission can occur from the open yards where cattle are usually held or in transit from a ship or lorry [39].

Only live attenuated vaccines against LSD are currently commercially available. Due to antigenic homology and cross protection between sheep pox, goat pox and LSD viruses, any of these viruses can be used as a vaccine strain to protect cattle against LSD [39]. In Egypt, a Romanian strain of sheep pox was used in cattle during the epidemic. It proved to be protective against the rapid epizootic spread of LSD. A tissue culture-adapted Romanian sheep pox strain was used on a limited basis in Israel, and it was found that a 10 times higher immunizing dose was required, compared with that recommended for sheep [39]. During recent LSD outbreaks in the Middle East region it was reported that vaccination did not result in a complete protection against the disease in each vaccinated animal [38]. However, vaccination is currently the only effective way to control the spread of LSDV in endemic countries. In non-endemic areas the use of live attenuated vaccines may compromise the disease-free status of the country and would be highly questionable on grounds of safety. In addition, the use of genetically modified recombinant live vaccines may not be permitted. In non-endemic countries the use of inactivated vaccines could be considered as a short term solution in an emergency; however the protection provided by inactivated vaccines is not solid and is only short-lived [40].

REFERENCES

1. Costales A, Gerber P, Steinfeld H. Underneath the livestock revolution. In Livestock report 2006. Food and Agriculture Organization, Rome. 2006; 15-27.
2. Supporting livelihoods and building resilience through Peste des Petits Ruminants (PPR) and small ruminant diseases control. FOA Animal Production and Health Position Paper. Rome. 2013.
3. RM Buller, BM Arif, DN Black, KR Dumbell, JJ Esposito, EJ Lefkowitz, G McFadden, B Moss, AA Mercer, RW Moyer, MA Skinner, DN Tripathy. 2005; Family Poxviridae. In: CM Fauquet, MA Mayo, J Maniloff, U Desselberger, LA Ball. Virus Taxonomy: Classification and Nomenclature of Viruses. Eighth Report of the International Committee on Taxonomy of Viruses. 117-133. Elsevier Academic Press, San Diego.
4. Diallo A, GJ Viljoen. Genus Capripoxvirus. Birkhäuser Advances in Infectious Diseases. 2007; 167-181.
5. IK Damon. 2007: Poxviruses. In: DM Knipe, PM Howley, DE Griffin, RA Lamb, MA Martin, B Roizman, SE Straus. Fields Virology. 2947-2975. Lippincott Williams & Wilkins, Philadelphia.
6. Singh IP, Pandey R, Srivastava RN, Sheep Pox. A rev. Vet. Bulletin. 1979; 49: 145-154.
7. Upton M, 1980. The economic importance of veterinary viral and tick-borne diseases on livestock production in Kenya. Report. Reading, UK: Pan Livestock Services Limited, Department of Agriculture, University of Reading.
8. Bennet SCJ, Horgon ES, Munsur MA. The pox diseases of sheep and goats. J Comparative Pathol Therapeutics. 1944; 54: 131-160.
9. Davies FG. 1981. Sheep and Goat Pox. In: Gibbs EPJ. Virus Diseases of Food Animals. London, UK: Academic Press, 733-749.
10. OIE, 2016. World Animal Health Information Database (WAHID).
11. Davies FG. Characteristics of a virus causing a pox disease in sheep and goats in Kenya, with observations on the epidemilogy and control. J Hyg. 1976; 76: 163-171.
12. Kitching RP, Taylor WP. Clinical and antigenic relationship between isolates of sheep and goat pox viruses. Tropic Ani Health Produc. 1985; 17: 64-74.
13. Sharma SN, Dhanda MR. Studies on sheep- and goat-pox viruses. Relationship with contagious pustular dermatitis and vaccinia viruses. Indian J Ani Sci. 1971; 41: 864-867.
14. Tantawi HH, Awad MM, Shony MO, Alwan AH, Hassan FK. Preliminary characterization of the 'Sersenk' strain of goat-pox virus. J Trop Ani Health Prod. 1980; 22: 30-34.
15. Kitching RP. Progress towards sheep and goat pox vaccines. Vaccine.1983; 1: 4-9.
16. Kitching RP, Taylor WP. Transmission of capripoxvirus. Res Vet Sci. 1985; 39: 196-199.
17. Kitching PR, Mellor PS. Insect transmission of Capripox viruses. Res Vet Sci. 1986; 40: 255-258.
18. Matumoto M. Mechanisms for the perpetuation of animal viruses in nature. Bacteriol Rev. 1969; 33: 401-413.
19. Kitching RP, 1994. Sheep and goat poxviruses. In: Webster RG, Granoff A. Encyclopedia of Virology. London: Academic Press, 1160-1165.
20. Plowright W, Macleod WG, Ferris RD. The pathogenesis of sheep pox in the skin of sheep. J Comparative Pathol. 1959; 69: 400-413.
21. Prasad IJ, Datt NS. Observation on the use of live and inactivated vaccines against goat pox. Indian Vet J. 1973; 50: 1-10.
22. Carn VM, Timms CP, Chand P, Black DN, Kitching RP. Protection of goats against capripox using a subunit vaccine. Vet Record. 1994; 135: 434-436.
23. Carn VM. Control of capripoxvirus infections. Vaccine. 1993; 11: 1275-1279.
24. MacDonald RAS, 1931. Pseudo-urticaria of cattle. Ann Rpt. 1930, Dept Anim Health, Govt. N. Rhod.
25. Von Backstrom U. Ngamiland cattle disease. Preliminary report on a new disease, the aetiological agent being probably of an infectious nature. JSAVMA. 1945; 16: 29-35.
26. Green HF. Lumpy skin disease, its effect on hides and leather and a comparison in this respect with some other skin diseases. Bull Epiz Dis Afr. 1959; 7: 63-69.

27. Davies FG. Observations on the epidemiology of Lumpy skin disease in Kenya. J. Hyg. 1982; 88: 95-102.

28. Carn VM, Kitching RP. The clinical response of cattle experimentally infected with lumpy skin disease (Neethling) virus. Arch of Virol. 1995; 140: 503-513.

29. Tuppurainen ESM, Stoltsz WH, Troskie M, Wallace DB, Oura CAL, Mellor PS, et al. A potential role for ixodid (hard) tick vectors in the transmission of lumpy skin disease virus in cattle. Transboundary and Emerging Diseases. 2011; 58: 93-104.

30. Yeruham I, Nir O, Braverman Y, Davidson M, Grinstein H, Haymovitch M,et al. Spread of lumpy skin disease in Israeli dairy herds. Vet Record. 1995; 137: 91-93.

31. Capstick PB, Coackley W. Protection of cattle against lumpy skin disease. I Trials with a vaccine against Neethling type infection. Res Vet Sci. 1961; 2: 362-368.

32. Greth A, Gourreau JM, Vassart M, Vy NB, Wyers M, Lefevre PC. Capripoxvirus disease in an Arabian Oryx (Oryx leucoryx) from Saudi Arabia. J Wildl Dis. 1992; 28: 295-300.

33. Young E, Basson PA, Weiss K. Experimental infection of the giraffe, impala and the Cape buffalo with lumpy skin disease virus. Onderstepoort J Vet Res.1968; 37:79-86.

34. Irons PC, Tuppurainen ESM, Venter EH. 2005. Excretion of lumpy skin disease virus in bull semen. Theriogenology. 2005; 63: 1290-1297.

35. Weiss KE. Lumpy skin disease. In: Virology Monographs, Vol. 3. Vienna, Austria; New York, USA: Springer-Verlag.1968; 111-131.

36. Chihota CM, Rennie LF, Kitching RP, Mellor PS. Mechanical transmission of lumpy skin disease virus by Aedes aegypti (Diptera: Culicidae). Epidemiol Infect. 2001; 126: 317-321.

37. Ayre-Smith RA. The symptoms and clinical diagnosis of lumpy skin disease in Egypt. Vet Rec. 1960; 127: 549-550.

38. Brenner J, Bellaiche M, Gross E, Elad D, Oved Z, Haimovitz M, e al. Appearance of skin lesions in cattle populations vaccinated against lumpy skin disease: statutory challenge. Vaccine. 2009; 27: 1500-1503.

39. Romero CH, Barrett T, Evans SA, Kitching RP, Gershon PD, Bostock C, et al. Single capripoxvirus recombinant vaccine for the protection of cattle against rinderpest and lumpy skin disease. Vaccine. 1993; 11: 737-742.

40. Romero CH, Barrett T, Kitching RP, Carn VM, Black DN. Protection of cattle against rinderpest and lumpy skin disease with a recombinant capripoxvirus expressing the fusion protein gene of rinderpest virus. Vet Record. 1994. 135: 152-154.

Cite this article

Kardjadj M (2016) Capripoxviruses: Transboundary Animal Diseases of Domestic Ruminants. Ann Virol Res 2(3): 1024.