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Dr. Barkathullah N
M.V.Sc., Ph.D., Veterinary Assistant Surgeon, Livestock Farm Complex, Directorate of Advanced Institute for Integrated Research on Livestock and Animal Sciences (AIIRLIVAS), Salem, Tamil Nadu, India

Dr. Joyshikh Sonowal
M.V.Sc., Ph.D., Subject Matter Specialist (Animal Science), KVK Sribhumi, Assam Agricultural University, Assam, India

Dr. Chhabhi Lal Patel
M.V.Sc., Ph.D., Principle Scientist, Division of Veterinary Virology, ICAR-Indian Veterinary Research Institute, Mukteswar, Uttarakhand, India

Dr. Alimudeen S
M.V.Sc., Veterinary Assistant Surgeon, O/o the Directorate, Directorate of Advanced Institute for Integrated Research on Livestock and Animal Sciences (AIIRLIVAS), Salem, Tamil Nadu, India

Dr. Kathiravan S
M.V.Sc., Veterinary Assistant Surgeon, Model Farm Complex, Directorate of Advanced Institute for Integrated Research on Livestock and Animal Sciences (AIIRLIVAS), Salem, Tamil Nadu, India

Dr. Sowmiya L
M.V.Sc., Veterinary Assistant Surgeon, Model Farm Complex, Directorate of Advanced Institute for Integrated Research on Livestock and Animal Sciences (AIIRLIVAS), Salem, Tamil Nadu, India

Dr. Hareesh Aravind
M.V.Sc., Assistant Professor, Department of Food/Dairy Biochemistry, Directorate of Advanced Institute for Integrated Research on Livestock and Animal Sciences (AIIRLIVAS), Salem, Tamil Nadu, India

Dr. Venkatasami M
M.Tech., Ph.D., Assistant Professor, Department of Dairy Engineering, Directorate of Advanced Institute for Integrated Research on Livestock and Animal Sciences (AIIRLIVAS), Salem, Tamil Nadu, India

Dr. Krishnakumar K
M.V.Sc., Ph.D., FISSAR, Director, Directorate of Advanced Institute for Integrated Research on Livestock and Animal Sciences (AIIRLIVAS), Salem, Tamil Nadu, India

Dr. Bina Mishra
M.V.Sc., Ph.D., Principle Scientist, Division of Animal Biotechnology, ICAR-National Bureau of Animal Genetic Resources, Karnal, Haryana, India

Corresponding Author:

Dr. Bina Mishra
M.V.Sc., Ph.D., Principle Scientist, Division of Animal Biotechnology, ICAR-National Bureau of Animal Genetic Resources, Karnal, Haryana, India

Sheep pox disease: A comprehensive review of disease dynamics, epidemiological patterns, and preventive intervention

Barkathullah N, Joyshikh Sonowal, Chhabhi Lal Patel, Alimudeen S, Kathiravan S, Sowmiya L, Hareesh Aravind, Venkatasami M, Krishnakumar K and Bina Mishra

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Abstract

Sheep pox, caused by the sheep pox virus (SPPV), is a highly infectious disease that significantly affects sheep health and leads to substantial economic losses, with case fatality rates ranging from 10 to 100 percent, depending on the outbreak severity. Similar to other poxviruses, SPPV invades the host immune system. Vaccination and effective management are the primary methods for controlling its spread. The virus is of research interest because of its similarities with the Modified Vaccinia Virus Ankara (MVA) vector and its persistence despite the availability of vaccines. Therefore, innovative and sensitive strategies for viral eradication are needed. This review examines sheep pox in detail, covering its transmission, epidemiology, virion characteristics, viral life cycle, host interactions, and immune response.

Keywords: Poxvirus, sheep pox, capri pox virus, trans-boundary disease, endemic area

Introduction

Sheep pox is a severe viral skin disease in sheep, with higher mortality rates in young animals than in adults. In endemic areas, native breeds often experience mild infections [1]. Clinical signs include fever, skin eruptions, and generalized nodules on wool-free areas, such as the nose, ears, tail, groin, axilla, perineum, and udder [2]. The incubation period ranges from 8 to 13 days, with lesions appearing within 2-4 days in experimental infections [3]. SPPV belongs to the family Poxviridae and genus Capripoxvirus and is one of the largest enveloped double-stranded DNA viruses [4]. Transmission occurs via aerosols, abraded skin, mechanical vectors (such as *Stomoxys calcitrans*), contaminated equipment, vehicles, litter, and fodder [5]. The acute phase presents with papules within 24 h, accompanied by respiratory and ocular symptoms, lymphadenopathy, and mucous membrane ulcers [6, 7]. Lesions can impair mobility and feeding, and severe lymph node swelling may cause labored breathing [8]. SPPV is classified as risk group II and is recognized by the OIE as a potential biological warfare agent [6]. Morbidity rates in endemic regions are 70-90 percent, with mortality rates of 5-10 percent, but can reach 100 percent in imported breeds [1]. Infection rates depend on the virus strain, host species, breed, and immunity [9]. Economic losses stem from reduced milk production, skin damage, and trade restrictions [8]. Uncontrolled animal movement complicates the control of diseases. Annual vaccination with a safe and effective vaccine is essential, starting at 2 weeks of age, with a booster at 2 months and yearly re-vaccination [10].

Genetic and Molecular Profiling of Pathogen

Sheep pox virus (SPPV) is a Capri poxvirus (CaPV) belonging to the Chordopoxvirinae subfamily of the Poxviridae family [11]. SPPV is antigenically related to the Goat pox virus (GTPV) and bovine lumpy skin disease virus (LSDV). It has 96 percent nucleotide identity with GTPV and 97 percent with LSDV [12, 13]. At least 148 potential genes are shared by wild-type genomes, including conserved poxvirus replicative and structural genes, as well as genes presumably implicated in virulence and host range [17]. The virion has a brick-shaped exterior surface with ridges in parallel rows and an extraordinarily complex interior structure

that includes roughly 150kb of a single linear, double-stranded DNA genome (73-91 MDa), related enzymes, and codes for approximately 200 distinct proteins^[14]. The virion measures approximately 270 nm in diameter, 300 nm in length, and 200 nm in height. The virulent strain of SPPV measures 170-260 nm in length, whereas the less virulent strain measures 250-380 nm^[15]. Thin sections examined by electron microscopy indicated that the exterior of the poxvirus is composed of proteins and lipids and covers the biconcave (dumbbell-shaped) core with two "lateral bodies," according to the ICTV^[16]. The sheep pox virus has high adenine-thymine content (approximately 75 percent)^[12]. A core coding region for pathogenic factors is present in the linear genome. Hairpin loops join the two linear dsDNA strands into a continuous molecule in the terminal region. The terminus sequences are lengthy inverted terminal repeats (ITRs) containing sets of small, tandem repeating sequences. The ITR sequence is 2.1-2.3 kb in length^[17]. Hairpins and IRTs are required for viral DNA replication^[18]. The core comprises a bundle of closely packed nucleoproteins. Antigenically, poxviruses are highly complex and elicit both cross-reactive and specific antibodies^[16]. Virions create intracellular and extracellular particles during their life cycle; however, infection is triggered by mature extracellular virions. Intracellular virus particles proliferate autonomously by budding through the host cell membrane^[19]. The viral genome encodes both structural and non-structural proteins. Most genes in the central genomic core are involved in fundamental replication processes and viral morphogenesis. Genes in terminal genomic regions may influence pathogenesis and host range^[20]. SPPV virions are vulnerable (resistant) to both very acidic and alkaline pH (hydrochloric or sulfuric acid at 2 percent for 15 min) as well as 56 °C for 2 h or 65 °C for 30 min. The isolate was inactivated after 60 min at 56 °C. The virus is sensitive to several disinfectants and chemicals, including 2 percent phenol, sodium dodecyl sulfate (SDS), formalin (1 percent), chloroform, sodium hypochlorite (2-3 percent), ether (20 percent), iodine compounds (at a 1:33 dilution), and quaternary ammonium compounds (0.5 percent)^[21]. SPPV is photosensitive but can survive for months in wool, hair, and dry scabs. It can withstand freeze-thaw cycles, although its infectivity is diminished^[22]. Many poxvirus genomes have been sequenced, and their genome sizes vary from strain to strain^[23]. The complete genome sequences of SPPV strains Jaipur (MG000156.1), Srinagar passage-40 vaccine (MT137384.1), TU (NC_004002.1), NK (AY077834.1), V293 (MW167071.1), V123 (MW167070.1), V104 (MW020571.1), Turkey vaccine (MN072631.1), Saudi Arabia (MN072630.1), and Nigeria (MN072628.1) are recorded in the NCBI database^[12]. These strains are named after the locations where they were isolated, such as Ranipet, Jaipur, Hyderabad, Mysore, Karnal, Mathura, Indo-China (India), Roumanian, Roumanian-Fanar (RF), Cairo, Chinese, K Strain, Pergo, RM/65, Kazakhstan, Stavropol, Nairobi, Kenyan, SP8, Pakistan, Turkey, Russian, and Sheep pox A strains. Attenuated vaccine strains include Egyptian, Romanian-Fanner (RF), RM/65, and Nishiki^[14].

Geographical Spread and Seasonal Fluctuations

Sheep pox is an ancient disease that dates back to the 2nd century A.D. During the nineteenth century, researchers investigated the infectivity of sheep pox in sheep. The

disease was first recorded in African, Asian, and European nations in the mid-twentieth century. Between 1847 and 1866, Britain experienced many epidemics of sheep pox^[6]. Although SPPV has been eliminated in many countries, it remains present in central and northern Africa, central Asia, the Middle East, and parts of the Indian subcontinent^[24]. It is enzootic in Africa north of the Equator, the Middle East, Turkey, Iran, Iraq, Afghanistan, Pakistan, India, Nepal, parts of the People's Republic of China, and Bangladesh. Sporadic outbreaks have been reported in Southern Europe and globally^[1]. The disease was initially documented in India in 1936 in Tamil Nadu, Bombay, the United Provinces, and Mysore^[25]. Although highly effective vaccines are available, cases of this disease persist. Due to the frequency of Sheep pox in Karnataka, Tamil Nadu, Himachal Pradesh, Makhdoom, Jammu, and Kashmir, India is now considered enzootic^[26]. The most recent outbreaks occurred in Vietnam in 2005, Mongolia in 2008 and 2009, and Azerbaijan in 2009. The first outbreak in Chinese Taipei occurred in 2008, followed by outbreaks in Bulgaria, Greece, and Turkey in 2013, Sudan in 2015, and Pakistan in 2018^[27]. Between 2013 and 2019, 663 sheep and goat pox epidemics were documented across all major regions of the Eastern Amhara region^[8]. The sheep pox virus is considered exotic to the EU and is listed on the OIE notifiable disease list. The virus has been absent from countries in central and Western Europe for many years, but following the outbreak of sheep pox in Southern Europe, there is a potential for further spread of these viruses into Europe^[28]. Outbreaks of Sheep pox occur throughout the year but are more frequent between November and May, with a peak in March^[29]. During the cold season, sheep are exposed to low temperatures, which can cause stress that may suppress the immune system and ultimately make them vulnerable to infection^[30]. The observed seasonality of sheep pox disease may be attributed to the virus's prolonged survival in wet, cold weather, its association with the lambing season, or the compromised physiological condition of herds during autumn^[21].

Economic Implications

Sheep pox causes considerable economic losses owing to its high morbidity and mortality, reduced milk and meat production, abortion, depreciation of wool and skin quality, and trade restrictions^[31]. Economic investigations have indicated that variables such as the number of adult animals affected, the number of days of illness, and flock size significantly influence economic losses due to sheep pox disease^[32]. There is a large trade in hides, skins, and wool from countries where Sheep pox is persistently present to other countries worldwide^[21]. Hides and skins processed only by salting or drying may pose a risk of introducing the sheep pox virus into other countries. Economic investigations have indicated that variables such as the number of adult animals affected, the number of days of illness, and flock size significantly influence economic losses due to sheep pox^[33]. Economic losses arise due to reduced milk production and deterioration of the quality of skin and wool. Sheep pox leads to significant economic losses due to high morbidity and mortality, reduced milk and meat production, abortions, lower wool and skin quality, and trade restrictions^[31]. Factors such as the number of affected adult animals, duration of illness, and flock size influence the extent of these losses^[33]. International trade in

hides, skins, and wool from endemic countries poses a risk of spreading the virus, mainly when products are processed only by salting or drying^[8, 21]. Skin lesions heal slowly and often leave permanent scars on the skin. The disease hampers efforts to improve local breeds through importation and reduces productivity, particularly in young, immunologically naïve animals, where mortality can exceed 50 percent^[34] based on breed type, whether wool or mutton. Mutton breeds are more prone to infection than wool breeds. Exotic breeds are more resistant than crossbred and indigenous breeds^[35]. This wide range is attributable to variables such as the host, etiological agent, and environmental conditions. An animal's physiological state, such as immunological naïveté, weakness, or stress due to blood protozoan or tick infection, might impact the epidemiology of Sheep pox illness^[36].

Transmission

Transmission of Sheep pox between and among herds occurs through the movement of sheep. While the disease can affect all age groups, it manifests with greater severity in young animals than in adults^[37]. Systemic signs include fever, conjunctivitis, rhinitis, lymphadenopathy, anorexia, and depression. Necrosis of the mucous membranes may occur, and affected animals may exhibit mucopurulent nasal or ocular discharge. Sheep pox is transmitted via the respiratory route when there is close contact between sick and healthy animals^[38]. The disease-causing virus may also enter the body through other mucous membranes or skin abrasions produced either iatrogenically or by insects^[6]. Intrauterine infection can occur, and in such cases, lambs can be born with fully developed lesions. Viruses are shed in the secretions and excretions of infected animals. However, they are not believed to be significant sources of transmission during outbreaks because it is challenging to recover live viruses from scab material in tissue culture^[21]. Movement of infected animals is the leading cause of the spread of the Sheep pox virus. Indirect transmission of the disease occurs through contaminated litter, fodder, and other objects, and transmission by insects and mechanical vectors has been reported. The virus can linger in animal pens and remain infectious for up to 6 months^[1].

Clinical signs

Clinical signs vary in intensity depending on host factors (such as age, breed, and immunity) and viral factors. The incubation period ranges from 4 to 21 days, with most cases occurring within 1-2 weeks. The disease is more severe in lambs and kids than in adult animals^[39]. Some very young lambs and kids may die before exhibiting any signs of the disease. Most affected animals become weak and lose their appetites. They may exhibit a high fever (104-108°F) for a short time^[40]. The skin lesions follow a typical poxvirus progression from macules to papules. Skin lesions appear as small red patches, usually around the mouth, on the head, under the tail, and between the legs. The centers of the patches become depressed and turn greyish in color due to necrosis^[41]. These patches form blisters that break, becoming open sores that develop scabs. Ruptured lesions often form hard, black scabs. These skin lesions render infected sheep prone to fly strikes, and scabs may persist for several weeks. Animals often have labored breathing due to blisters inside the respiratory tract and lungs. Lesions in the mouth, nose, and eyes can cause discharge and excessive

salivation. Necrosis and ulceration can occur in the affected mucous membranes^[42]. Intestinal nodules can cause diarrhea. Some animals may exhibit depression and emaciation symptoms. Additionally, abortion can occur. Lesions may require several weeks to heal and often result in permanent scars. During the healing process, the animals remain vulnerable to fly strikes. Secondary bacterial infections, including pneumonia, frequently occur, and mortality can occur at any stage of the disease. Recovery is typically prolonged in cases of severe affliction^[25].

Pathology

The skin exhibits congested, hemorrhagic, swollen, and necrotic lesions. The most prominent clinicopathological feature is generalized cutaneous eruptions, each involving the full thickness of the skin. Frequently, the centers of these eruptions become necrotic and ulcerated. A significant proportion of affected animals develop pneumonia, which is grossly characterized by multiple foci of subpleural consolidation and edema. Additionally, pox lesions may be present on various serosal surfaces^[43]. The mucous membranes of the mouth, nose, eyes, vulva, and prepuce may be necrotic and ulcerated, and all body lymph nodes are swollen and enlarged. Lymph nodes draining infected regions can increase up to eight times their normal size, become edematous, and may exhibit congestion and hemorrhage^[39]. Necropsy findings in affected sheep include widely distributed, discrete, generally circular, slightly raised, firm papules (1-2 cm) on the lung surface, occasionally in the mucosa of the rumen and abomasum, and rarely, small lesions (2-5 mm) in the renal cortex and liver^[6, 44]. Histologically, skin lesions are characterized by dermal edema and increased cellularity, with variable numbers of "Sheep pox cells," which are histiocytic-like cells with large vacuolated nuclei and poorly defined eosinophilic cytoplasmic inclusions. In the lungs, necrotizing and proliferative bronchiolitis may be observed, often with "Sheep pox cells" distributed throughout the proliferative zone^[45].

Host-SPPV interaction

Host-virus interaction refers to how SPPV sustains itself within the host at the molecular and cellular levels through the action of its immunomodulatory genes. Chibssa, Kangethe^[46] demonstrated the host defense response against SPPV infection by identifying critical innate immune mechanisms through studies of PBMC exposure to SPPV. They revealed RIG-1 as a critical pattern recognition receptor in the signaling pathway of innate immune activation during SPPV infection, possibly through intermediate viral dsRNA^[47]. Another group of researchers used NGS transcriptomics to investigate SPPV interaction with lamb testis cells in cell culture systems at the immediate early stages of infection, revealing key dysregulated genes such as trans membrane receptors (CD40, FAS, ITGβ1, ITGα1, Pak1, Pak2, CD14, ILK), immune-related DEGs MAPK, JNK, ERK, NFκB, IκB, PI3K, and STAT, which provide optimal cellular conditions for early viral protein expression^[36]. Immediate early SPPV infection also revealed DEGs, such as FOXO3, ATF, CDKN1A, TCF, SRF, and BDNF, which favor apoptosis induction, whereas MPTP, BAD, and Tp53 suppress apoptosis. The research team also revealed that TP53 is the top upstream regulator in SPPV-infected LTCs^[48].

Immunity and vaccination

Immunity induced by poxviruses or vaccines is strong and may persist for a long time compared to immunity induced by other pathogens. Poxviruses elicit both cellular and humoral immune responses. Maternal immunity protects against the sheep pox virus for up to 3 months^[5, 49]. Animals that recover from sheeppox virus infection develop lifelong immunity. For the virus to persist, it must continually leap from one infected animal to another that is still vulnerable, making a minimum number of susceptible hosts essential for its survival^[50]. The size of this population depends on the virus strain and the susceptibility of the host population. Active mass vaccination against sheep pox can induce strong herd immunity, effectively controlling the disease^[51]. Previously, SGP viruses considered to be a single virus. However, genetic sequencing has demonstrated that these are distinct viruses. Most SGP strains are host-specific, causing severe disease in either goats or sheep, although some strains exhibit similar virulence in both species^[12, 52]. Indeed, isolates from most regions are host-specific, but those from Kenya, Yemen, and Oman naturally infect both goats and sheep^[53]. Therefore, in most countries, at least two different vaccines containing isolates of either the GP or SP virus are necessary to protect small ruminants against both viruses^[1, 47]. However, recent studies have confirmed that the identity of the Kenyan SGP vaccine (commonly used) virus O-240 is not the SP virus, and that this vaccine may cause clinical disease^[5]. Furthermore, researchers have recently reported that some SP outbreaks occurred in both sheep and goat herds at the exact geographic location in India^[27]. Bhanuprakash, Hosamani^[54] also reported the sheep pox virus infection in goats.

Diagnosis

Sheep pox should be suspected in animals with characteristic full-thickness skin lesions, fever, and lymphadenitis. Skin biopsies, vesicular fluid, scabs, skin scrapings, lymph node aspirates, and whole blood collected in heparin or EDTA are used for diagnosis in live animals^[55]. Skin lesions, lymph nodes, lung lesions, and histology: a whole collection of tissues, particularly those with lesions, is gathered from animals during necropsy^[56]. Polymerase chain reaction (PCR) and a clinical history compatible with widespread Capri pox infection provide the fastest laboratory confirmation of Capri pox. Capripoxviruses can be cultivated in tissue cultures derived from ovine, caprine, or bovine sources, allowing viral isolation^[5]. Laboratory procedures for the diagnosis of sheep pox include electron microscopy (to observe the virus; morphology is characteristic) and virus isolation (identification by immunofluorescence or peroxidase staining). Viral antigens can be detected using agar gel immune diffusion (AGID) or enzyme-linked immunosorbent assays (ELISA)^[57]. However, cross-reactivity occurs in the AGID test with parapoxvirus. Serology is also useful; antibodies can be found one week after skin lesions appear. Serological tests include virus neutralization, AGID, indirect immunofluorescence, ELISA, and Western blotting (immunoblotting)^[58]. Virus neutralization is considered the most specific serological test; however, its sensitivity is insufficient to identify infections in all animals. AGID and indirect immunofluorescence tests may exhibit cross-reactivity with other viruses^[59]. Field isolates, on the other hand, may require 14 days to form or multiple tissue culture

passes. The virus produces intracytoplasmic inclusions that are easily visible with hematoxylin and eosin staining^[54, 60].

Preventive interventions

Following the introduction, disease control is typically accomplished through early detection and notification, rapid implementation of animal movement restrictions, culling of affected and in-contact animals, and ring vaccination. Routine control measures include cleaning and disinfection of depopulated premises and the establishment of protection and surveillance zones with radii of 3 and 10 km, respectively, around the outbreak area^[5, 61]. As noted earlier, the uncontrolled movement of infected animals in SGP-endemic areas poses significant challenges to the efficient control of the disease. Therefore, it is essential to vaccinate sheep flocks annually with a safe and efficient vaccine to control this severe and economically significant disease in endemic regions^[62, 63]. Key factors facilitating the control of SPV in enzootic countries include ease of disease or virus detection, significant economic impact, absence of reservoir hosts beyond domestic ruminants, robust immunity following vaccination, absence of a carrier state, and a relatively low annual turnover rate of animals within herds^[64]. In contrast, factors that may impede control include prolonged viral stability on wool, a long incubation period for the disease, and unregulated introduction of livestock through illegal importation^[54].

Conclusion

Sheep pox is one of the most economically significant infectious diseases in sheep, particularly because of the permanent scarring that affects the tanning industry. Mortality is lower in indigenous breeds in endemic areas but increases in exotic or naïve animals, especially when intensive practices or concurrent infections are present. Annual vaccination with a safe and effective vaccine is essential for controlling the disease in endemic regions.

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Conflict of Interest

The authors declare that they have no conflict of interest.

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