

## LUMPY SKIN DISEASE: A COMPREHENSIVE REVIEW

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## ABSTRACT

Lumpy skin disease (LSD) is an economically devastating viral disease of cattle caused by Lumpy Skin Disease Virus (LSDV), a member of the genus Capripoxvirus within the family Poxviridae. Historically confined to sub-Saharan Africa, the disease has undergone dramatic geographical expansion over the past two decades, spreading through the Middle East, Eastern Europe, Central Asia, and South and Southeast Asia, culminating in catastrophic outbreaks across the Indian subcontinent since 2019. The disease inflicts severe production losses through mortality, drastically reduced milk yield, hide damage, reproductive failure, and international trade embargoes. This comprehensive review synthesizes current knowledge on the etiology, molecular biology, epidemiology, transmission dynamics, pathogenesis, clinical presentation,

differential diagnosis, laboratory confirmation, treatment protocols, prevention strategies, and future research directions for LSD. Special emphasis is placed on emerging diagnostic tools, next-generation vaccine platforms, and integrated One Health approaches. Sequential citations<sup>[1–20]</sup> are preserved throughout.

**KEYWORDS:** Lumpy skin disease; Lumpy skin disease virus (LSDV); Capripoxvirus; cattle; transboundary animal disease; epidemiology; PCR; real-time PCR; vaccination; vector-borne disease; One Health; poxvirus; economic impact; India; Africa.

## 1. INTRODUCTION

Lumpy skin disease (LSD) is a notifiable transboundary animal disease (TAD) of cattle (*Bos taurus* and *Bos indicus*) and water buffalo (*Bubalus bubalis*) caused by Lumpy Skin Disease

Virus (LSDV). First described in Zambia in 1929, the disease was initially believed to be a nutritional or toxic disorder before its viral aetiology was established.<sup>[1,2]</sup> For decades, LSD remained largely confined to sub-Saharan Africa; however, since the 1980s it has progressively expanded northward and eastward, crossing into the Middle East in the 1980s–1990s, entering Europe (Greece, Bulgaria, and beyond) in 2015–2016, and reaching Russia, Kazakhstan, and the Caucasus region by 2017. The most alarming recent development has been the explosive spread across South and Southeast Asia, with India reporting its first large-scale outbreak in 2019, followed by devastating epizootics in 2021–2022 that affected millions of cattle across nearly every state.<sup>[1,2]</sup>

The economic burden imposed by LSD is multifaceted: direct losses arise from mortality (case fatality rates of 1–5% in naïve populations, occasionally exceeding 10%), precipitous declines in milk production (up to 50%), permanent hide damage reducing leather value, reproductive losses (abortion, temporary infertility in bulls), and the cost of treatment and nursing care. Indirect losses stem from trade restrictions, quarantine costs, and the substantial investment required for mass vaccination campaigns.<sup>[1,2]</sup> The World Organisation for Animal Health (WOAH) lists LSD as a reportable disease, and its emergence in previously free regions triggers immediate international trade bans, compounding economic damage.<sup>[1]</sup>

This review aims to provide a consolidated, up-to-date reference on all major aspects of LSD for veterinary students, practitioners, researchers, and policy-makers, synthesising evidence from foundational texts, international agency reports, and recent peer-reviewed literature.



**Figure:** Cattle infected with Lumpy Skin Disease showing characteristic skin nodules on the head, neck and body.

**Reference:**

- Tuppurainen, E. S. M. (2017). Lumpy skin disease: an emerging threat to Europe, the Middle East and Asia. *Transboundary and Emerging Diseases*, 64(1), 40–48. <https://doi.org/10.1111/tbed.12444>

## 2. Etiology and Molecular Biology

LSDV belongs to the genus Capripoxvirus within the subfamily Chordopoxvirinae, family Poxviridae.<sup>[3,4]</sup> The genus also includes Sheep Pox Virus (SPPV) and Goat Pox Virus (GTPV), with which LSDV shares approximately 96% nucleotide identity across the genome. Despite this high sequence homology, host range is generally species-specific under natural conditions, though experimental cross-infections have been documented.<sup>[3,4]</sup>

LSDV is a large, enveloped, brick-shaped double-stranded DNA virus approximately 290 × 270 nm in size. Its genome is approximately 151 kilobase pairs (kbp) in length and encodes around 156 putative open reading frames (ORFs).<sup>[3,4]</sup> The genome contains a central coding region flanked by identical inverted terminal repeat (ITR) sequences, a hallmark of poxviruses. Key virulence factors include genes encoding kelch-like proteins, ankyrin repeat proteins, and immunomodulatory proteins that help the virus evade host innate and adaptive immune responses. The LSDV030 (RPO30), GPCR, EEV, and P32 genes are commonly targeted for diagnostic PCR assays and phylogenetic analyses.<sup>[3,4]</sup>

LSDV is relatively stable in the environment, surviving for weeks in dried scabs and for extended periods at low temperatures. It is inactivated by common disinfectants including sodium hypochlorite (2–3%), phenol compounds, iodophors, and detergents. Formalin (1%) and ether effectively inactivate the virus, while it remains viable at pH 6.6–8.6.<sup>[3,4]</sup>

## 3. Epidemiology

LSD was first recorded in Zambia in 1929 and subsequently spread throughout sub-Saharan Africa over the following decades.<sup>[5,6]</sup> The disease crossed the Sahara into North Africa in the 1980s and entered the Middle East (Israel, Kuwait, Bahrain, United Arab Emirates, Oman) between 1986 and 1989. After a period of relative containment, a second major wave of expansion began around 2012–2013, with outbreaks in Turkey, Iran, Iraq, and Jordan. By 2015, LSD had reached Greece and Bulgaria in southeastern Europe, marking its first incursion into the European Union.<sup>[5,6]</sup>

Between 2016 and 2019, the disease spread rapidly through the Balkans, Eastern Europe, the Caucasus, and Central Asia. Russia, Kazakhstan, Armenia, and Azerbaijan reported outbreaks during this period. The most recent and arguably most consequential expansion has occurred in South and Southeast Asia. India reported its first confirmed LSD outbreak in Odisha in 2019; by 2021–2022, the disease had become epizootic across the entire

subcontinent, with millions of cattle affected in states including Rajasthan, Gujarat, Punjab, Haryana, Uttar Pradesh, and Maharashtra.<sup>[5,6]</sup> Bangladesh, Nepal, Sri Lanka, Pakistan, China, Vietnam, Thailand, and Indonesia have also reported outbreaks in this period.

Risk factors for disease introduction and spread include movement of infected livestock and their products, presence of competent insect vectors, inadequate biosecurity at farm and national borders, and low herd immunity in naïve populations. Seasonality is pronounced in tropical regions, with peak incidence coinciding with the wet season when insect vector populations are highest.<sup>[5,6]</sup>

**Table 1: Key Milestones in the Global Spread of LSD.**

| Period     | Region / Countries                          | Significance                         |
|------------|---------------------------------------------|--------------------------------------|
| 1929       | Zambia (sub-Saharan Africa)                 | First recorded outbreak              |
| 1943–1970s | Sub-Saharan Africa (widespread)             | Endemicity established across Africa |
| 1986–1989  | Middle East (Israel, Kuwait, UAE, Oman)     | First extra-African spread           |
| 2012–2014  | Turkey, Iran, Iraq, Jordan                  | Re-emergence in Middle East          |
| 2015–2016  | Greece, Bulgaria (EU)                       | First EU incursion                   |
| 2017–2019  | Russia, Caucasus, Central Asia              | Northward/eastward expansion         |
| 2019–2022  | India, Bangladesh, Nepal, Pakistan, SE Asia | Massive Asian epizootic              |

#### 4. Transmission

The primary route of LSDV transmission is mechanical vector-borne spread by haematophagous (blood-feeding) arthropods.<sup>[7,8]</sup> Laboratory and field studies have implicated multiple species as competent mechanical vectors, including mosquitoes (*Aedes aegypti*, *Culex quinquefasciatus*), stable flies (*Stomoxys calcitrans*), biting midges (*Culicoides* spp.), and hard ticks (*Rhipicephalus appendiculatus*, *Amblyomma hebraeum*, *Rhipicephalus decoloratus*).<sup>[7,8]</sup> The relative contribution of each vector species varies by region and season, but *Stomoxys calcitrans* is considered particularly important in many outbreak settings.

Direct contact transmission can occur through contaminated saliva, nasal and ocular secretions, milk, semen, and skin lesion exudates from clinically affected animals. LSDV has been detected in semen of infected bulls for up to 42 days post-infection, raising concerns about venereal transmission and the safety of artificial insemination programmes.<sup>[7,8]</sup> Indirect transmission via contaminated fomites (water troughs, feeding equipment, milking machines) and shared housing is also possible but generally considered less important than vector-borne

routes.

Vertical (transplacental) transmission has been documented, and infected cows may give birth to calves with generalised skin lesions. The role of wildlife reservoirs remains incompletely characterised, though African buffalo (*Syncerus caffer*) and impala have been experimentally infected, suggesting potential maintenance hosts in endemic African ecosystems.<sup>[7,8]</sup>

## 5. Pathogenesis

Following inoculation via insect bite or direct contact, LSDV undergoes primary replication at the site of entry and in regional lymph nodes. An initial viraemia (primary viraemia) occurs approximately 4–14 days post-infection, during which the virus disseminates haematogenously to target tissues including skin, mucous membranes, lymph nodes, lungs, liver, kidneys, testes, and ovaries.<sup>[9]</sup>

Viral replication in dermal endothelial cells and pericytes leads to vasculitis, thrombosis, and ischaemic necrosis, producing the characteristic firm, circumscribed skin nodules (necrotic plugs or 'sit-fasts'). Histologically, lesions show epidermal hyperplasia, ballooning degeneration of keratinocytes, intracytoplasmic inclusion bodies (Bollinger bodies), and severe lymphoplasmacytic perivascular infiltration.<sup>[9]</sup> Systemic inflammation, lymphadenopathy, and oedema of the limbs and brisket result from widespread immune activation and lymphatic obstruction.

The host immune response involves both innate (type I interferon, NK cells) and adaptive (virus-specific CD4<sup>+</sup> and CD8<sup>+</sup> T cells, neutralising antibodies) components. Neutralising antibodies develop within 2–3 weeks of infection and are considered the primary correlate of protection for vaccine efficacy. LSDV encodes multiple immunoevasion proteins that modulate interferon signalling, complement activation, and apoptosis, contributing to its ability to establish prolonged viraemia.<sup>[9]</sup>

## 6. Clinical Signs

The incubation period ranges from 2 to 5 weeks (typically 4–14 days under experimental conditions). The clinical course varies from subclinical to severe, depending on viral strain, host breed (*Bos taurus* generally more susceptible than *Bos indicus*), immune status, and concurrent infections.<sup>[10]</sup>

The acute phase is characterised by a sudden onset of high fever (40–41.5°C), depression, anorexia, and profuse lacrimation and nasal discharge. Within 48 hours, characteristic firm, raised, circumscribed skin nodules (2–5 cm in diameter) appear on the head, neck, perineum, genitalia, and limbs, and may become generalised across the entire body surface. The nodules involve the full thickness of the skin and underlying subcutaneous tissue; they may ulcerate and become secondarily infected, leaving permanent scars.<sup>[10]</sup>

Generalised lymphadenopathy is a consistent finding, with superficial lymph nodes (prescapular, precrural, parotid) becoming markedly enlarged. Oedema of the limbs, brisket, dewlap, and genitalia is common and may cause lameness. Ocular signs include conjunctivitis, keratitis, and corneal ulceration. Respiratory signs (coughing, dyspnoea) occur when lesions involve the oral and nasal mucosae and larynx. Milk production may fall by 50% or more in lactating cows, and affected bulls may remain temporarily or permanently infertile due to orchitis and epididymitis. Pregnant cows may abort.<sup>[10]</sup>

Morbidity rates in naïve populations typically range from 5% to 45%, with case fatality rates of 1–5% (occasionally higher in severe outbreaks or immunocompromised animals). Recovery from uncomplicated cases occurs over 4–6 weeks, though deep skin lesions may persist for months.

**Table 2: Summary of Clinical Signs and Their Frequency in LSD.**

| Clinical Sign                       | Frequency     | Notes                                |
|-------------------------------------|---------------|--------------------------------------|
| Fever ( $\geq 40^{\circ}\text{C}$ ) | Very common   | Early sign; may precede skin lesions |
| Skin nodules                        | Pathognomonic | Full-thickness; 2–5 cm; may ulcerate |
| Lymphadenopathy                     | Very common   | Generalised; all superficial nodes   |
| Reduced milk yield                  | Common        | Up to 50% reduction in dairy cattle  |
| Limb/brisket oedema                 | Common        | Lymphatic obstruction                |
| Ocular/nasal discharge              | Common        | Lacrimation, rhinitis                |
| Lameness                            | Moderate      | Due to limb oedema or podal lesions  |
| Abortion/infertility                | Moderate      | Bulls and cows affected              |
| Respiratory signs                   | Moderate      | Mucosal lesions; secondary pneumonia |
| Mortality                           | Variable      | 1–5%; higher in naïve herds          |

## 7. Diagnosis

A presumptive diagnosis is often made on the basis of characteristic clinical signs (fever, generalised skin nodules, lymphadenopathy) in conjunction with epidemiological history (recent introduction of animals, proximity to outbreaks, presence of insect vectors).<sup>[11,12]</sup> However, laboratory confirmation is essential, particularly for the first reported case in a

previously free region, for differentiation from clinically similar diseases, and for official reporting to WOAHA.

### 7.1 Differential Diagnosis

LSD must be differentiated from several conditions that produce skin nodules or systemic illness in cattle. Key differentials include:

- Pseudo-lumpy skin disease (Bovine herpesvirus 2 / Allerton virus): milder lesions, no systemic signs, restricted to skin
- Bovine papular stomatitis (Parapoxvirus): oral lesions predominate; no generalised nodules
- Dermatophilosis (*Dermatophilus congolensis*): superficial crusty lesions; bacterial aetiology; responds to antibiotics
- Ringworm (*Trichophyton* spp.): circular alopecic lesions; no fever; fungal aetiology
- Insect bites / urticaria: transient; no necrotic nodules; no fever
- Bovine besnoitiosis (*Besnoitia besnoiti*): scleroderma; thickened skin; protozoal aetiology

### 7.2 Laboratory Methods

Conventional PCR and real-time PCR (qPCR) targeting the LSDV-specific P32 (envelope protein), RPO30 (RNA polymerase subunit 30), or GPCR genes are the gold-standard diagnostic tools, offering high sensitivity and specificity with results available within hours.<sup>[11,12]</sup> PCR can be performed on skin biopsy, blood (EDTA), nasal swabs, or saliva collected during the acute febrile phase. Multiplex PCR assays allow simultaneous differentiation of LSDV from SPPV and GTPV, which is critical given the high genomic homology among Capripoxviruses.

Virus isolation in susceptible cell lines (primary bovine skin cells, Vero cells, BHK-21 cells) remains the reference method but is time-consuming (5–14 days), requires BSL-2 containment, and is less sensitive than PCR in the field setting.<sup>[11,12]</sup> Characteristic cytopathic effect (CPE) includes cell rounding, syncytium formation, and intracytoplasmic inclusion bodies visible on Giemsa or haematoxylin-eosin staining.

Serological methods include virus neutralisation test (VNT), indirect ELISA, and immunofluorescence assay (IFA). VNT is highly specific but requires live virus and BSL-2 facilities. Commercially available blocking ELISAs offer a practical alternative for seroprevalence surveys and post-vaccination monitoring.<sup>[11,12]</sup> Histopathology of skin

biopsies reveals pathognomonic intracytoplasmic eosinophilic inclusion bodies (Bollinger bodies) within keratinocytes and dermal cells, supporting diagnosis when PCR is unavailable.

Transmission electron microscopy (TEM) can visualise poxvirus particles directly in lesion material, providing rapid presumptive confirmation. Whole-genome sequencing (WGS) is increasingly used for molecular epidemiology, strain characterisation, and identification of recombinant field strains, which have emerged as a significant concern following the widespread use of live attenuated capripoxvirus vaccines.<sup>[11,12]</sup>

## 8. Treatment

There is currently no licensed specific antiviral therapy for LSD. Management is therefore entirely supportive and symptomatic, aimed at reducing morbidity, preventing secondary complications, and maintaining animal welfare.<sup>[13]</sup>

Non-steroidal anti-inflammatory drugs (NSAIDs) such as flunixin meglumine (2.2 mg/kg IV or IM, BID) or meloxicam (0.5 mg/kg IV or SC, SID) are administered to control fever, pain, and systemic inflammation. Broad-spectrum antibiotics (oxytetracycline, procaine penicillin, amoxicillin-clavulanate, or fluoroquinolones) are indicated to prevent or treat secondary bacterial infections of skin lesions, which are common and can lead to deep tissue necrosis, myiasis, and septicemia.<sup>[13]</sup>

Wound care is an important component of management: skin lesions should be cleaned with antiseptic solutions (povidone-iodine, chlorhexidine), debrided if necrotic, and protected from fly strike using repellents or fly-proof dressings. Severely affected animals may require intravenous fluid therapy to correct dehydration and electrolyte imbalances. Animals with severe respiratory involvement may benefit from bronchodilators and mucolytics. Nutritional support, provision of easily accessible feed and water, and separation from herd-mates to reduce stress and secondary trauma are recommended.<sup>[13]</sup>

Experimental use of cidofovir (a nucleoside analogue with broad-spectrum antiviral activity against DNA viruses) has shown promise in vitro and in small animal models, but clinical data in cattle are lacking and the drug is cost-prohibitive for field use. Recovery in uncomplicated cases typically occurs over 4–8 weeks; however, permanent scarring, hide damage, and chronic weight loss may persist.

## 9. Prevention and Control

An integrated control strategy combining vaccination, movement control, surveillance, vector management, and biosecurity measures is essential for sustainable LSD management.<sup>[14,15,16]</sup>

### 9.1 Vaccination

Vaccination is the most effective tool for LSD control. Homologous live attenuated LSDV vaccines (e.g., Neethling strain-based vaccines) provide robust, long-lasting protection ( $\geq 3$  years) and are the preferred option where available.<sup>[14,15]</sup> Heterologous live attenuated vaccines based on SPPV or GTPV strains (e.g., Romanian SPPV strain, Kenya SPPV O-240 strain) have been widely used in LSD-affected countries outside Africa due to their availability and cross-protective immunity; however, protection is generally less complete than with homologous vaccines, and some SPPV/GTPV-based vaccines have been associated with recombination events generating novel field strains.<sup>[14,15]</sup>

Mass vaccination campaigns targeting  $\geq 80\%$  of the susceptible bovine population are recommended to achieve herd immunity. Ring vaccination around newly confirmed outbreaks is employed as an emergency measure. Annual booster vaccination is recommended in endemic regions. Calves born to vaccinated dams may carry maternal antibodies that interfere with vaccine response for the first 3–6 months of life, necessitating delayed primary vaccination.<sup>[14,15]</sup>

Next-generation vaccine platforms under development include recombinant subunit vaccines (targeting the P32 envelope protein), DNA vaccines, virus-like particle (VLP) vaccines, and DIVA (Differentiating Infected from Vaccinated Animals)-compatible marker vaccines. These offer advantages in safety, thermostability, and compatibility with serological surveillance but are not yet commercially available.<sup>[15]</sup>

### 9.2 Movement Control and Quarantine

Immediate imposition of movement restrictions on livestock, livestock products (hides, semen, embryos), and fomites from affected premises is a critical first-line response.<sup>[14,16]</sup> Quarantine zones (typically 3–10 km radius around confirmed cases) and surveillance zones (20–50 km radius) should be established in accordance with WOAHP guidelines. All susceptible animals within quarantine zones should be examined, and suspect cases reported to veterinary authorities. International movement of cattle and cattle products from affected countries should be suspended pending disease-free certification.<sup>[16]</sup>

### 9.3 Surveillance and Reporting

Passive and active surveillance systems are essential for early detection, rapid response, and monitoring of vaccine coverage and efficacy.<sup>[16]</sup> Clinical surveillance should be complemented by systematic serological surveys and PCR-based screening of high-risk populations (recently imported animals, animals at livestock markets). Molecular epidemiology using whole-genome sequencing enables tracing of outbreak origins and spread, identification of recombinant strains, and evaluation of vaccine-field strain relatedness. All confirmed outbreaks must be reported to WOAHA via the World Animal Health Information System (WAHIS).<sup>[16]</sup>

### 9.4 Vector Control and Biosecurity

Integrated vector management strategies include the use of insecticide-treated ear tags, pour-on insecticides, residual spraying of housing with pyrethroids, and installation of insect-proof netting in high-risk premises.<sup>[16]</sup> Reducing standing water near livestock facilities minimises mosquito breeding sites. Strict biosecurity measures — including controlled access to farms, footbaths, disinfection of vehicles, and dedicated protective clothing for farm workers — reduce the risk of mechanical introduction by fomites. Isolation of newly purchased animals for a minimum of 28 days before integration into the main herd is strongly recommended.<sup>[14,16]</sup>

## 10. Economic Importance

LSD imposes a substantial and multidimensional economic burden on affected countries and farming communities.<sup>[17]</sup> Direct production losses include: mortality (1–5% case fatality rate in naïve herds, occasionally higher); severe reduction in milk yield (up to 50% during acute illness, with partial recovery over weeks to months); permanent hide damage rendering hides unsuitable for leather production; reproductive losses including abortion, temporary anoestrus in cows, and prolonged or permanent infertility in bulls due to orchitis; and reduced draught capacity in working animals.<sup>[17]</sup>

Indirect costs are equally significant and include: veterinary treatment, vaccination, and biosecurity expenditure; quarantine and movement restriction costs borne by farmers and governments; trade losses from national and international embargoes on cattle and cattle products; and the administrative and logistical costs of surveillance and outbreak response. The 2021–2022 Indian epizootic, which affected an estimated 15–20 million cattle across 28 states and union territories, resulted in direct losses estimated in the billions of Indian Rupees,

with particularly severe impacts on smallholder and marginal dairy farmers who depend on a single cow or buffalo for household income.<sup>[17]</sup>

Beyond the immediate farm-level impact, LSD undermines food security by reducing the availability of milk and dairy products in affected regions, and its emergence in new territories triggers prolonged trade disruptions that affect entire national livestock sectors. The disease is therefore considered a priority TAD by WOA, FAO, and regional veterinary bodies.<sup>[17]</sup>

## 11. Future Perspectives

Several priority areas have been identified for future research and policy development to strengthen global LSD control.<sup>[18,19]</sup>

**Vaccine innovation:** Development and commercialisation of DIVA-compatible, thermostable, recombinant or subunit vaccines that provide complete protection against diverse LSDV field strains without the risk of recombination-driven emergence of novel pathogenic strains is a top priority.<sup>[18]</sup> Advances in mRNA vaccine technology, as demonstrated during the COVID-19 pandemic, offer a potentially transformative platform for rapid vaccine development against LSDV and other poxviruses.

**Rapid and field-deployable diagnostics:** Point-of-care (POC) diagnostic tools, including loop-mediated isothermal amplification (LAMP) assays, lateral flow immunoassays, and CRISPR-based detection systems, are under development and could enable rapid, on-farm diagnosis without specialised laboratory infrastructure.<sup>[18,19]</sup> These tools are particularly important for resource-limited settings in Asia and Africa where LSD is newly emerging.

**Genomic surveillance and molecular epidemiology:** Expansion of whole-genome sequencing capacity and establishment of open-access genomic databases will facilitate real-time tracking of LSDV spread, identification of recombinant strains, and evidence-based vaccine strain selection.<sup>[18,19]</sup> Integration of genomic data with epidemiological and meteorological data through geospatial modelling will improve outbreak prediction and targeted response.

**Vector biology and control:** A deeper understanding of the ecology, competence, and population dynamics of LSDV vectors across different agroecological zones is needed to design evidence-based integrated vector management programmes. Climate change is expected to alter vector distribution and seasonality, potentially expanding the geographic

range of LSD.<sup>[18,19]</sup>

One Health integration: LSD provides a compelling case study for One Health approaches that bridge veterinary, public health, environmental, and socioeconomic dimensions. Strengthening cross-sectoral collaboration between veterinary services, public health authorities, wildlife agencies, and the agricultural sector will be essential for sustainable TAD control in an era of accelerating globalisation and climate change.<sup>[19]</sup>

## 12. CONCLUSION

Lumpy skin disease has transformed from a regionally confined African disease into a globally significant transboundary animal disease threatening cattle industries across Asia, Europe, and beyond. Its rapid geographical expansion, driven by globalised livestock trade, inadequate biosecurity, and climate-mediated changes in vector distribution, underscores the urgent need for coordinated international action. Effective control requires a multi-pronged strategy: widespread deployment of safe and efficacious vaccines, robust surveillance and early warning systems, stringent movement controls, integrated vector management, and strong international cooperation through WOAHA, FAO, and regional veterinary networks. Advances in molecular diagnostics, next-generation vaccines, and genomic surveillance offer promising tools for the future. Sustainable control of LSD will ultimately depend on political commitment, adequate funding, and the empowerment of frontline veterinary services — particularly in low- and middle-income countries where the burden falls most heavily on vulnerable smallholder farming communities.<sup>[20]</sup>

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