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A COMPREHENSIVE REVIEW OF PESTE DES PETITS RUMINANTS IN SHEEP AND GOATS: CLINICAL FEATURES, DIAGNOSIS, VACCINATION, AND CONTROL

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ABSTRACT

Peste des petits ruminants (PPR) is a highly contagious morbilliviral disease of sheep and goats that remains one of the most consequential transboundary animal diseases for pastoral, agropastoral, and smallholder systems. Although effective live attenuated vaccines and sensitive molecular tests are available, PPR continues to persist through gaps in surveillance, incomplete vaccination coverage, animal movement, weak cold chains, wildlife and peri-domestic interfaces, and delayed outbreak recognition. This review critically synthesizes the current evidence on PPR epidemiology, clinical presentation, pathogenesis, diagnosis, vaccination, and control, with emphasis on the operational requirements for the global eradication target. PPR virus infection is characterized by fever, ocular and nasal discharge, erosive stomatitis, respiratory disease, diarrhea, lymphoid depletion, and immunosuppression. Diagnostic confirmation increasingly relies on RT-PCR or RT-qPCR, supported by antigen ELISA, serology, virus isolation, sequencing, and field-adapted assays. Existing vaccines, especially Nigeria 75/1 and Sungri/96 derivatives, can provide strong protection, but eradication requires strategic, risk-based campaigns with post-vaccination evaluation, reliable animal identification, movement governance, and sustained surveillance after vaccination ceases. The review concludes that PPR control should move from reactive outbreak vaccination toward integrated, intelligence-led eradication systems that combine laboratory networks, participatory epidemiology, thermostable vaccines, DIVA-compatible tools, genomic surveillance, and One Health-informed wildlife risk assessment.

Keywords: *Peste des petits ruminants; PPR virus; disease-control; pathogenesis; vaccination;*

Introduction

Peste des petits ruminants is an acute, contagious, and frequently fatal disease of small ruminants caused by small ruminant morbillivirus, historically called PPR virus. It is a notifiable transboundary animal disease because outbreaks can spread rapidly through susceptible sheep and goat populations and disrupt trade, food security, household income, and national disease-control programmes. The World Organisation for Animal Health (WOAH) describes PPR as a priority disease for global

control and eradication, and the Food and Agriculture Organization of the United Nations (FAO) frames it as a major threat to more than 1.7 billion sheep and goats globally (FAO, 2024; WOAH, 2025). In highly susceptible flocks, morbidity can be very high and mortality can be devastating, particularly when animals are immunologically naive, stressed, poorly nourished, or exposed to concurrent infections (Banyard et al., 2010; Parida et al., 2015).

The global importance of PPR has increased because small ruminants are central to the livelihoods of many low- and middle-income households. Goats and sheep provide meat, milk, manure, hides, social capital, and emergency income in environments where crop farming may be unreliable. Consequently, PPR is not only a veterinary virology problem but also a food-system and poverty-reduction problem. The eradication of rinderpest demonstrated that morbilliviral diseases can be eliminated when sensitive surveillance, effective vaccines, political commitment, and coordinated field implementation converge (Mariner et al., 2016). PPR is often discussed as the next feasible livestock-virus eradication target because it has no known persistent carrier state, effective vaccines exist, and diagnosis is technically achievable. However, PPR is operationally more complex than a purely laboratory problem because the disease is embedded in informal animal movement, pastoral mobility, cross-border trade, vaccine hesitancy, weak veterinary infrastructure, and under-resourced surveillance systems (Baron et al., 2011; Kwiatek et al., 2011; Munir et al., 2013).

This review critically examines PPR in sheep and goats with a focus on clinical features, pathogenesis, diagnosis, vaccination, and control. Rather than treating each topic as a separate technical domain, the manuscript emphasizes how field recognition, laboratory confirmation, vaccine delivery, and governance interact. A high-impact PPR review must therefore ask not only what is known about the virus, but why preventable outbreaks continue to occur despite available vaccines and tests.

Review approach and scope

This article was developed as a comprehensive critical review. Literature was prioritized from peer-reviewed articles, WOA and FAO technical documents, major reviews, and recent studies on PPR diagnostics, vaccines, field epidemiology, and control. Emphasis was placed on sources addressing sheep and goats, although evidence from wildlife, camels, and broader morbillivirus biology was included where it informs spillover risk, surveillance design, or pathogenesis. The review gives preference to studies and guidance that are directly relevant to eradication, outbreak management, and routine veterinary decision-making.

The manuscript is organized around six questions: (a) what epidemiological conditions sustain PPR, (b) how does PPRV cause disease, (c) which clinical features should trigger suspicion, (d) which diagnostic methods are fit for different decision points, (e) what are the strengths and limitations of current vaccines, and (f) how can integrated control programmes close the gap between technical feasibility and eradication implementation?

Etiology and viral biology

PPRV is an enveloped, negative-sense, single-stranded RNA virus within the genus *Morbillivirus* and the family *Paramyxoviridae*. The viral genome encodes structural proteins that support attachment, fusion, replication, transcription, assembly, and immune modulation. The hemagglutinin and fusion glycoproteins are particularly important because they mediate host-cell entry and are major targets of neutralizing antibodies (Bailey et al., 2005; Banyard et al., 2010). The nucleocapsid gene is widely used for molecular epidemiology because it is abundant, diagnostically useful, and informative for lineage assignment.

PPRV has traditionally been divided into four genetic lineages, I to IV. Lineages I and II were historically associated largely with West Africa, lineage III with East Africa and the Middle East, and lineage IV with Asia; however, lineage IV has expanded geographically and is now detected in wider regions, including parts of Africa and Europe (Kwiatek et al., 2011; Dundon et al., 2014; Oboegbulem et al., 2024). Lineage classification is epidemiologically useful but should not be mistaken for serotype diversity: PPRV is considered to have a single serotype, which is one reason why existing live attenuated vaccines can protect across lineages (Hodgson et al., 2018).

Epidemiology and transmission

The main hosts of PPR are domestic goats and sheep, although infection has been documented in wild small ruminants, camels, and other susceptible species. Goats often show more severe clinical disease than sheep in many field contexts, but species differences vary with breed, age, immune status, virus strain, nutritional status, and exposure intensity (Banyard et al., 2010; Parida et al., 2015). Transmission occurs mainly through close contact with infected animals and exposure to infectious secretions and excretions, including nasal and ocular discharge, saliva, feces, and respiratory droplets. Because infectious animals may move through markets, seasonal grazing routes, and informal trade networks, outbreaks often reflect social and economic movement patterns as much as viral biology.

Risk is highest in immunologically naive flocks, recently assembled animals, young stock after waning maternal antibodies, and herds exposed to stressors such as transport, drought, parasitism, malnutrition, or other infections. WOA notes that outbreaks are often associated with contact between naive animals and animals from endemic areas (WOA, 2025). The disease can occur in village, peri-urban, pastoral, and migratory systems, and control is especially difficult where animal ownership is distributed, borders are porous, and veterinary services lack real-time disease intelligence.

PPR is geographically entrenched across parts of Africa, the Middle East, and Asia, with repeated incursions into areas where freedom is fragile. Recent reports in Europe

highlight the importance of rapid detection and border-aware surveillance. For example, field investigations in Romania in 2024 documented typical clinical signs and substantial flock-level mortality, underscoring the consequences of introduction into vulnerable populations

(Dărăbuș et al., 2025). Such events strengthen the argument that eradication requires not only endemic-country vaccination but also regional preparedness, surveillance in trade corridors, and contingency planning in officially free areas.

Table 1. Epidemiological features of PPR relevant to sheep and goat control programmes.

Domain	Key points	Implication
Virus	Small ruminant morbillivirus; enveloped negative-sense RNA virus	Lineage assignment supports source tracing and outbreak reconstruction.
Primary hosts	Domestic goats and sheep	Control must prioritize small-ruminant production systems rather than only formal farms.
Transmission	Close contact, aerosols, secretions, excretions, contaminated environments over short periods	Movement control and market surveillance are crucial during outbreaks.
High-risk settings	Markets, communal grazing, pastoral mobility, border zones, drought aggregation points	Risk-based vaccination should target network hubs and mobile populations.
Disease impact	High morbidity and mortality in susceptible flocks; trade and livelihood disruption	Economic benefits of eradication extend beyond animal health.
Eradication feasibility	Single serotype, effective vaccines, no recognized long-term carrier state	Technical feasibility depends on operational delivery and surveillance quality.

Pathogenesis

After exposure, PPRV initially replicates in epithelial and lymphoid tissues of the upper respiratory tract and oropharynx. The virus disseminates through infected immune cells and establishes systemic infection. A central feature of morbillivirus pathogenesis is immunosuppression, which contributes to secondary bacterial infections and amplifies respiratory and gastrointestinal disease. Lymphoid depletion, mucosal epithelial injury, and inflammatory lesions explain many of the classical clinical and pathological findings (Banyard et al., 2010; Parida et al., 2015; UI-Rahman et al., 2025).

The respiratory and alimentary tracts are major targets. Damage to oral and gastrointestinal mucosa produces erosive stomatitis, salivation, diarrhea, dehydration, and

wasting. Respiratory involvement produces nasal discharge, coughing, dyspnea, and pneumonia. The combination of fever, mucosal damage, diarrhea, and pneumonia can rapidly become fatal, particularly in young, malnourished, or parasitized animals. Immunosuppression also complicates field diagnosis because secondary infections may obscure the primary viral syndrome.

Pathogenesis is best understood as a cascade linking exposure, lymphoid infection, systemic spread, epithelial injury, clinical disease, and onward transmission. The practical implication is that field control must act early. Waiting for pathognomonic lesions or large numbers of deaths wastes the short window in which movement restriction, sample collection, and emergency vaccination can prevent wider spread.

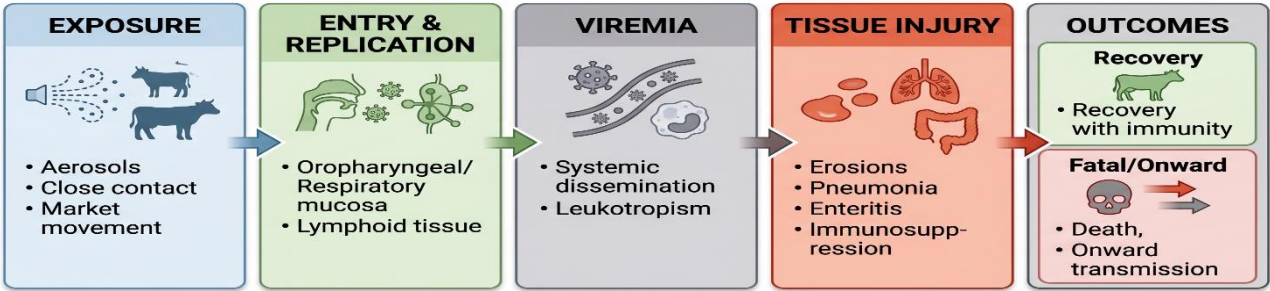


Figure 1. Schematic pathogenesis cascade of PPRV infection and major control points.

Clinical features and differential diagnosis

The incubation period is commonly several days, but observed timing varies with dose, host susceptibility, and field conditions. The classical syndrome includes high fever, depression, anorexia, ocular and nasal discharge, erosive or necrotic stomatitis, foul breath, excessive salivation, coughing, pneumonia, diarrhea, dehydration, and death. Discharges may become mucopurulent, and oral erosions may involve gums, dental pad, lips, tongue, and palate. Diarrhea can be profuse and may contribute to perineal soiling and rapid dehydration (Merck Veterinary Manual, 2026; WOA, 2025).

Clinical diagnosis is important for early suspicion but cannot stand alone for confirmation. PPR can resemble contagious caprine pleuropneumonia, bluetongue, foot-and-mouth disease, orf, pasteurellosis, coccidiosis, salmonellosis, rinderpest-like syndromes historically, and mixed respiratory-enteric disease complexes. Oral lesions, pneumonia, and diarrhea should therefore trigger sample collection rather than definitive field labeling. In free areas, any compatible syndrome in small ruminants should be treated as an emergency until laboratory testing rules out PPR.

Table 2. Clinical patterns of PPR in sheep and goats and their diagnostic implications.

Pattern	Common observations	Interpretation
Peracute/acute	High fever, depression, ocular/nasal discharge, rapid respiratory distress	Early warning; collect swabs and blood before severe decomposition or antimicrobial treatment.
Oral phase	Erosive stomatitis, salivation, foul breath, necrotic debris	Differentiate from FMD, bluetongue, orf, and toxic/traumatic lesions.
Respiratory phase	Coughing, dyspnea, pneumonia, mucopurulent discharge	Secondary bacterial pneumonia may dominate clinical appearance.
Enteric phase	Watery or bloody diarrhea, dehydration, weight loss	Can be confused with parasitism, coccidiosis, salmonellosis, or malnutrition.
Recovery	Resolution with durable immunity in survivors	Recovered animals can indicate past exposure but serology must be interpreted with vaccination history.
Flock-level pattern	High morbidity, clustered deaths, recent movement or market contact	Epidemiological context is often more informative than a single animal examination.

Diagnosis

PPR diagnosis should be aligned with the decision that must be made. In a suspected outbreak, the immediate requirement is rapid confirmation or exclusion to guide quarantine, movement control, and emergency vaccination. In surveillance, the requirement may be herd-level evidence of virus circulation or immunity. In eradication, the requirement expands to include proof of absence, lineage tracing, and discrimination between vaccination and infection where possible.

Recommended specimens include ocular, nasal, oral, or rectal swabs; anticoagulated blood during the febrile phase; lymph nodes, spleen, lung, and intestinal tissues from fresh carcasses; and sera for antibody detection. Sample quality and timing are decisive. RT-PCR and RT-qPCR are now central to laboratory confirmation because they are sensitive, specific, and faster than virus isolation. WOA laboratory guidance includes molecular assays, antigen

detection, virus isolation, and serological tests as part of the diagnostic toolkit (WOA, 2024).

Antigen-capture ELISA can be valuable for screening clinical samples where molecular infrastructure is limited, although sensitivity may be lower than RT-qPCR in poorly collected or late samples. Competitive ELISA is widely used for detecting antibodies and is essential for serosurveillance and post-vaccination monitoring. However, serology alone does not confirm active disease in vaccinated populations and must be interpreted with vaccination history, age structure, and sampling design. Sequencing provides lineage information and can support reconstruction of transboundary spread, but it requires stronger laboratory capacity and representative sampling (Kwiatk et al., 2011; Oboegbulem et al., 2024).

New diagnostic development is moving toward broader lineage coverage, faster field deployment, and better integration with genomic surveillance. Recent RT-qPCR work targeting conserved genomic regions illustrates the

continuing need for assays that detect all lineages with high sensitivity, especially as lineage IV expands geographically (Zhang et al., 2025). Nevertheless,

diagnostic innovation will not improve control unless sample transport, biosafety, data reporting, and outbreak-response authority are strengthened simultaneously.

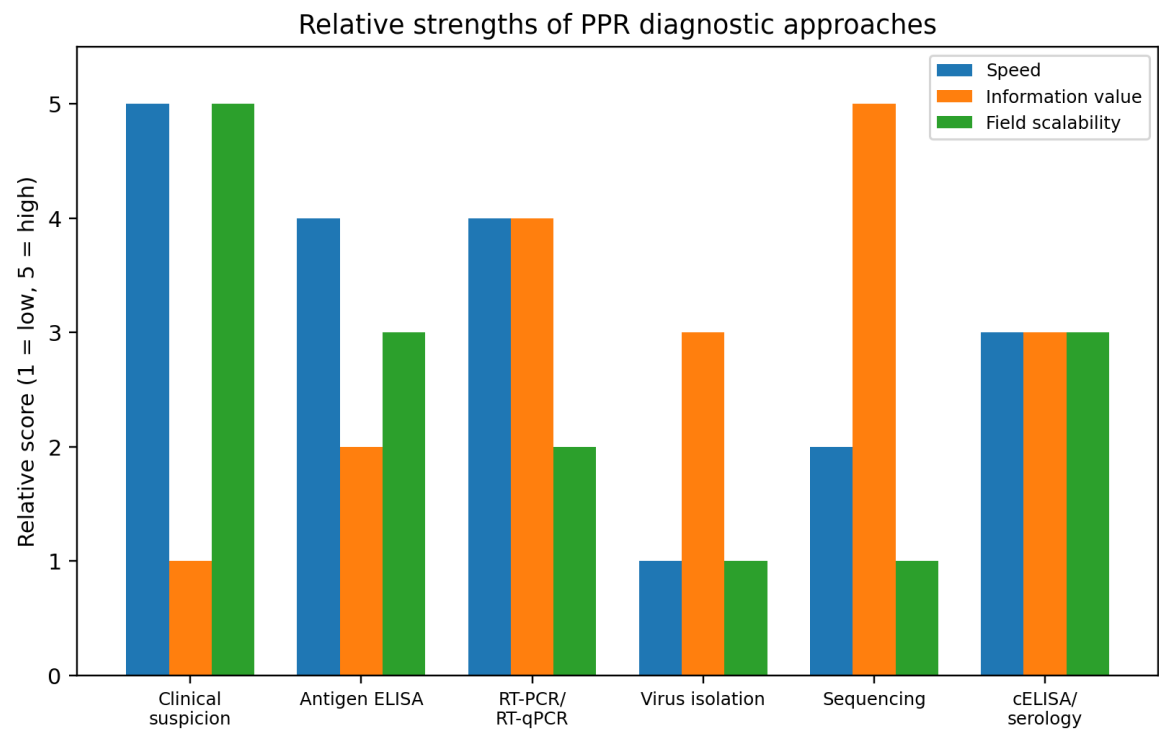


Figure 2. Relative strengths of major PPR diagnostic approaches.

Table 3. Diagnostic methods for PPR and their most appropriate uses.

Method	Main use	Strengths	Limitations	Best fit
Clinical examination	Early suspicion and triage	Immediate, inexpensive, identifies syndromic clusters	Low specificity; cannot confirm disease	All field investigations
Antigen ELISA	Detection of viral antigen in clinical samples	Useful where molecular tests are unavailable; suitable for outbreaks	Lower sensitivity than optimized RT-qPCR; sample quality dependent	District or regional laboratories
RT-PCR/RT-qPCR	Detection of viral RNA	High sensitivity and specificity; fast confirmation; can support quantification	Requires equipment, trained staff, cold chain for reagents	National/reference laboratories and advanced regional labs
Virus isolation	Recovery of infectious virus	Useful for research, characterization, assay validation	Slow; requires cell culture and biosafety capacity	Reference laboratories
Sequencing	Lineage identification and molecular epidemiology	Tracks spread, introductions, and evolution	Requires representative sampling and bioinformatics	Reference laboratories and genomic surveillance networks

cELISA/serology	Antibody detection	Post-vaccination monitoring and serosurveillance	Cannot distinguish infection from vaccination with conventional vaccines	Herd immunity assessment and freedom claims
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Vaccination

Vaccination is the central technical tool for PPR control. The most widely used vaccines are live attenuated PPRV strains, especially Nigeria 75/1 and Sungri/96 derivatives. These vaccines can induce strong protective immunity, and comparative studies show cross-lineage protection despite the existence of multiple genetic lineages (Hodgson et al., 2018; Mahapatra et al., 2020). WOAHP notes that effective PPR vaccines are available and can induce long-lasting protective immunity (WOAHP, 2025).

The practical problem is not simply vaccine efficacy under controlled conditions; it is vaccination effectiveness under field conditions. Campaign outcomes depend on vaccine quality, cold-chain integrity, correct reconstitution and administration, animal availability, coverage, owner participation, timing relative to seasonal movements, and the ability to vaccinate young replacement animals as maternal antibodies wane. The PPR Global Control and Eradication Strategy emphasizes sufficiently high herd immunity, often discussed around an 80% threshold, to interrupt transmission (FAO & OIE, 2015; Mandefro et al., 2024).

Several limitations of conventional live vaccines are important for eradication. First, they generally do not permit differentiation of infected from vaccinated animals using standard serology. Second, thermolability can reduce effectiveness in remote settings with weak cold chains. Third, repeated campaigns may miss mobile, marginal, or conflict-affected populations. Fourth, vaccination without surveillance can conceal ongoing transmission rather than demonstrate freedom. Therefore, the next generation of PPR vaccination strategy must be evaluated not only by immunogenicity, but by its compatibility with surveillance, post-vaccination evaluation, and eventual cessation of vaccination.

DIVA-compatible vaccines, recombinant vectors, and thermostable formulations are attractive because they could improve surveillance during the endgame of eradication. Recombinant Newcastle disease virus-vectored PPR vaccines and other marker-vaccine platforms illustrate the direction of research, but broad field adoption requires demonstration of safety, durable protection, scalability, affordability, and regulatory acceptance (Murr et al., 2020; Rojas et al., 2014).

Table 4. Vaccine platforms and strategic considerations for PPR eradication.

Vaccine/control tool	Primary role	Advantages	Limitations
Nigeria 75/1 live attenuated	Widely used in many endemic regions; strong protection	Proven field use and cross-lineage protection	Cold-chain dependence; no conventional DIVA capability
Sungri/96 live attenuated	Used especially in India and related programmes	Protective in experimental and field contexts	Same DIVA and cold-chain constraints as other live vaccines
Thermostable live vaccine approaches	Remote or hot environments	Potential to reduce cold-chain failure	Requires quality assurance, stability data, and field validation
Recombinant vector vaccines	DIVA-compatible eradication endgame and combined disease control	Can support marker-vaccine strategies	Often still experimental or limited in field deployment
Post-vaccination monitoring	Assessment of herd immunity after campaigns	Identifies coverage gaps and campaign failure	Requires statistically sound sampling and reliable serology

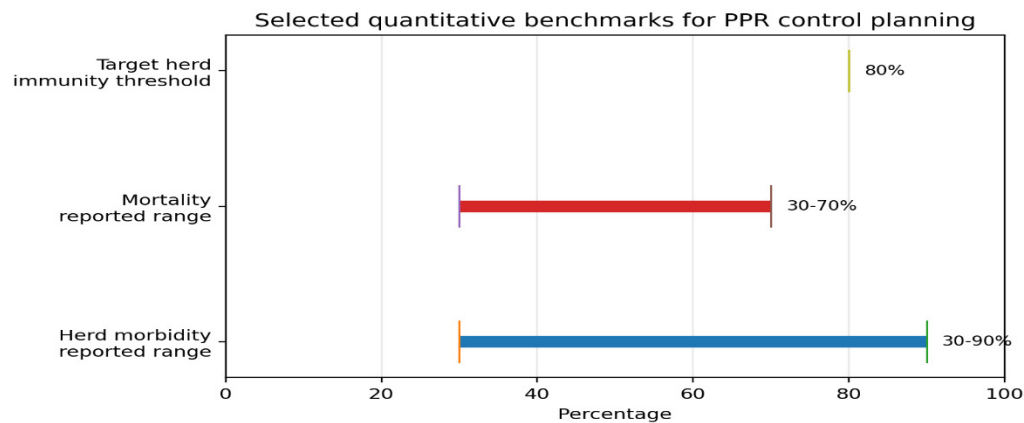


Figure 3. Selected quantitative benchmarks used in PPR planning.

Prevention and control measures

PPR control requires a layered system. At the farm or flock level, prevention includes vaccination, quarantine of new animals, avoidance of mixing with unknown-source animals, isolation of sick animals, improved nutrition, parasite control, and hygiene. At the community level, prevention requires market surveillance, risk communication, reporting incentives, and coordination with animal-health workers. At the national and regional level, prevention requires movement regulation, laboratory networks, vaccine banks, outbreak contingency plans, cross-border data sharing, and legally supported notification systems.

During outbreaks, immediate priorities are suspicion reporting, restriction of animal movement, clinical investigation, sample collection, safe disposal of carcasses, disinfection, tracing of animal movements, and targeted vaccination around risk zones. Stamping out may be used in free countries or high-value settings, but in endemic low-

resource settings, vaccination and movement governance are often more feasible. Because PPRV can spread through social and market networks, epidemiological tracing should include traders, transport routes, watering points, grazing areas, seasonal congregation sites, and informal cross-border flows.

The FAO-WOAH Global Control and Eradication Strategy and the PPR Global Eradication Programme provide the strategic architecture for progressive reduction and elimination, with a global target of eradication by 2030 (FAO & OIE, 2015; FAO, 2024; WOAH, 2025). The most important implementation lesson is that mass vaccination alone is insufficient. Programmes must demonstrate that vaccination produced immunity in the right animals, at the right time, in the right places. This requires post-vaccination evaluation, risk mapping, targeted revaccination, and transparent surveillance data

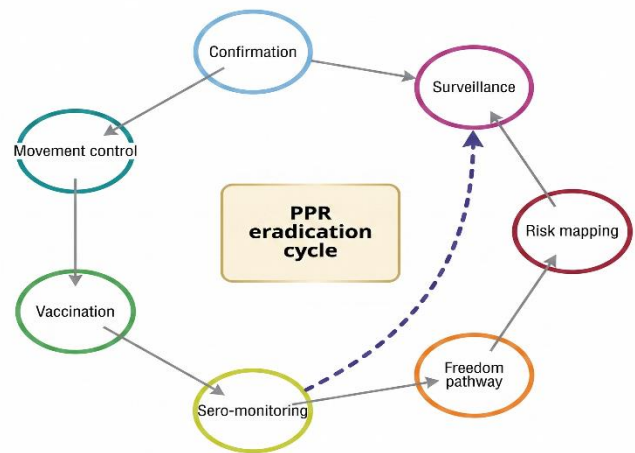


Figure 4. Integrated PPR control cycle linking risk mapping, surveillance, laboratory confirmation, vaccination, monitoring, and freedom pathways.

Wildlife, atypical hosts, and One Health-adjacent considerations

PPR is not considered a zoonosis, but its control has One Health relevance because it affects livelihoods, food security, biodiversity, and resilience of pastoral communities. Wildlife infections raise concerns for conservation and for the interpretation of eradication surveillance. Reports of infection or exposure in wild ungulates and other species do not necessarily mean that wildlife independently maintains PPRV, but they do show that endangered populations may be vulnerable to spillover from domestic small ruminants (Banyard et al., 2010; Lu et al., 2025).

The wildlife question has two practical implications. First, domestic-small-ruminant vaccination near protected areas can protect both livestock and susceptible wildlife by reducing spillover pressure. Second, surveillance systems should include wildlife morbidity and mortality reporting when outbreaks occur near conservation landscapes. However, scarce resources should not be diverted into speculative wildlife programmes at the expense of proven domestic-flock vaccination and surveillance unless risk assessment supports that allocation.

Critical gaps and future directions

Despite substantial progress, several gaps continue to constrain PPR eradication. The first is surveillance sensitivity. Many outbreaks are detected late because early clinical signs are non-specific, farmers may fear trade restrictions, and field services may lack transport or sampling supplies. Participatory epidemiology, mobile reporting, and community-based animal health workers can improve early warning, but they require trust and feedback to livestock keepers.

The second gap is vaccination intelligence. Campaign reports often count doses delivered rather than immunity achieved. High-impact PPR control requires animal-level and herd-level evidence: which populations were missed, whether young animals are becoming susceptible, and whether immunity persists across production cycles. Seromonitoring studies such as Mandefro et al. (2024) show why post-vaccination evaluation is necessary: even apparently successful campaigns may fall below the herd immunity threshold needed to interrupt transmission.

The third gap is DIVA-compatible endgame surveillance. Conventional vaccines are powerful but complicate serological interpretation. Marker vaccines and companion diagnostics could become important when countries approach freedom and need to distinguish residual infection from vaccine-induced antibodies. However, DIVA tools must be affordable and deliverable in the same settings where PPR persists.

The fourth gap is regional governance. PPR does not respect administrative borders. Control programmes fail when neighboring districts or countries vaccinate asynchronously, when trade routes are ignored, or when outbreak data are not shared. Regional synchronization, cross-border vaccination corridors, and harmonized laboratory protocols are essential for durable freedom.

Finally, eradication must be socially legitimate. Livestock owners participate when they trust vaccines, understand the benefits, and can access services without disproportionate costs. Communication should address rumors, adverse-event concerns, timing conflicts, and the economic logic of reporting disease early. The eradication endgame will depend as much on social implementation as on virology.

Proposed research and policy roadmap

Table 5. Research and policy roadmap for high-impact PPR control and eradication.

Area	Priority action	Expected benefit
Diagnostics	Validate rapid, affordable field assays across lineages and sample types	Faster outbreak confirmation and decentralized response
Genomic surveillance	Build regional sequencing networks and shared lineage databases	Better source tracing and monitoring of viral movement
Vaccines	Develop thermostable and DIVA-compatible vaccines suitable for remote settings	Improved field effectiveness and endgame surveillance
Delivery systems	Use risk maps, movement data, and participatory planning to target vaccination	Higher immunity in transmission-critical populations
Post-vaccination evaluation	Standardize seromonitoring and coverage-quality audits	Evidence that campaigns interrupt transmission
Socioeconomics	Quantify incentives, reporting barriers, gendered livestock roles, and livelihood impacts	Control programmes that farmers will actually use

Wildlife interface	Conduct risk-based surveillance near protected areas and outbreak fronts	Protection of biodiversity and better spillover interpretation
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Conclusion

PPR remains a major barrier to small-ruminant health, rural livelihoods, and animal-health security, despite the availability of effective vaccines and reliable diagnostic technologies. The disease is clinically recognizable but not clinically confirmable; it is vaccine-preventable but not eradication-ready without surveillance; and it is technically eradicable but operationally demanding. The central challenge is therefore integration. Successful PPR control in sheep and goats requires early clinical suspicion, rapid

molecular confirmation, strategic vaccination, post-vaccination monitoring, movement-aware outbreak management, and sustained regional coordination. The highest-yield innovations will be those that make eradication systems more field-compatible: thermostable vaccines, DIVA-compatible tools, practical diagnostics, participatory surveillance, and data-driven targeting of mobile and under-served flocks. If these elements are implemented together, PPR eradication remains a realistic and valuable global animal-health goal.

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