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A review of the epidemiology, diagnosis and economic impacts of Peste des Petits Ruminants (PPR) in Nigeria, and the case for early detection

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Abstract

Global food security faces multi-faceted challenges posed by poverty, economic mismanagement, flawed production systems and emerging infectious diseases amongst others. Livestock production in Nigeria plays a vital role in meeting food (protein) demands, socioeconomic and ecological balance, and relies significantly on the small ruminant component (sheep and goats), which accounts for around 28% of Nigeria's total livestock population. The livestock sector constantly grapples with endemic diseases that lead to significant losses for farmers, stakeholders and the community. Among these diseases is Peste des Petits Ruminants (PPR), which is endemic in Nigeria and is associated with very high mortality. A regular update on the status of PPR in Nigeria is essential to identify vulnerabilities and devise effective preventive and control measures. The present review provides insights into PPR's epidemiology, diagnosis and economic impacts in Nigeria, as well as the benefits of timely detection of the disease.

Keywords: Peste des Petits Ruminants; Goats and Sheep; Review; Epidemiology and Diagnosis; Economic impact; Timely/Early detection.

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Introduction

Peste des Petits Ruminants (PPR) is an acute and contagious viral disease that significantly impacts on small ruminants' production economics in Nigeria and Africa. It poses a serious threat to sheep and goat production across Nigeria, Asia and the Middle East (Kang-Seuk *et al*., 2012; Ahaduzzama, 2020). PPR, also known as stomatitis-pneumo-enteritis complex or Kata, and is caused by the rapidly spreading PPR virus (PPRV), a Mobillivirus belonging to the family of Paramyxoviridae (Dhar *et al*., 2002). The disease may present acute, sub-acute and sub-clinical manifestations, depending on the virulence of the virus.

The PPR virus is classified as a pleomorphic organism that is sensitive to ether, and its nucleic acid is RNA, similar to other viruses like that of Measles, Rinderpest, Canine distemper and Parainfluenza III (Golchinfar *et al*., 2011). The PPR virus is a linear, single-stranded and enveloped negatively-sensed RNA organism, akin to other RNA viruses affecting various animals, including cattle, buffaloes, humans, dogs, wild carnivores and aquatic mammals. Although there is only one single serotype of PPR virus, phylogenetic analysis based on the small region of the N/F gene has revealed four distinct lineages, namely, I – IV, with Lineages I and II present in West Africa, Lineage III in Arabia and East Africa, and Lineage IV in Asia and the Middle East (Prajapati *et al*., 2019).

Historically, PPR disease was first reported in the early 1940s during an outbreak in sheep and goats in West Africa (Dhar *et al*., 2002). Since then, the disease has reportedly been endemic throughout sub-Saharan Africa and has spread to other parts of the world, including Asia and the Middle East (Balamurugan *et al*., 2014). Quantifying the losses incurred due to the menace of PPR disease in Nigeria's small ruminant sector presents a challenging task, encompassing both agricultural dimensions and economic implications (Jones *et al*., 2016). The occurrence of PPR disease in other parts of the world has been speculated to be due to spread through international trade from sub-Saharan Africa, raising concerns about small ruminant livestock trade (Balamurugan *et al*., 2014). PPR is recognized as one of the five most destructive trans-boundary diseases in Africa, Asia and the Middle East by the Food and Agriculture Organization (FAO) and the World Organization for Animal Health (OIE). (OIE-FAO, 2015). Nigeria, with its substantial population of approximately 113.8 million sheep and goats, has the potential for large-scale exportation of these animals. However, factors like PPR disease hinder such opportunities (Esonu *et al*., 2022).

The clinical manifestations of PPR have been consistently described across Nigerian states, with classical signs including oculo-nasal discharges, conjunctivitis, coughing, and pyrexia (Lawal *et al*., 2011; Emikpe *et al*., 2013; Woma *et al*., 2015a; Mantip *et al*., 2016; Okwelum *et al*., 2017; Adedeji *et al*., 2019; Akanbi *et al*., 2020). Additional reports have noted abortion (Chukwudi *et al*., 2020) and mastitis (Adedeji *et al*., 2019; Akanbi *et al*., 2020) as part of the spectrum of clinical signs.

Diagnostic confirmation typically relies on a combination of clinical evaluation, post-mortem findings and laboratory testing. Virus neutralization tests (VNT), enzyme-linked immunosorbent assay (ELISA) and polymerase chain reaction (PCR) remain the most commonly used tools in both field investigations and laboratory settings (Mantip *et al*., 2022). Using these techniques in parallel reportedly enhances diagnostic sensitivity and specificity, thereby improving response to outbreaks.

At the global level, the Food and Agriculture Organization (FAO) recognized PPR as a trans-boundary animal disease of concern in 2007. By 2009, the FAO reported that over one billion small ruminants were at risk globally,

with African countries like Nigeria bearing a substantial proportion of that risk due to high livestock density, inadequate vaccination coverage, and weak surveillance infrastructure (FAO, 2009).

In the light of the global eradication of rinderpest in 2011, there were calls for the control or eradication of PPR at regional and global levels. This strategy has garnered support from 45 African countries and the African Union Inter-African Bureau for Animal Resources (AU-IBAR) (AU-IBAR, 2015). Proactive efforts to prevent and control PPR are essential not only for Nigerian farmers but also for enhancing the supply of animal protein across Africa and beyond (Dilli *et al*., 2011; Britton *et al*., 2019).

Despite concerted control efforts, PPR remains endemic in Nigeria, contributing substantially to the global burden of the disease. The present review provides a critical evaluation of the current epidemiological trends, diagnostic tools, pathological insights and economic implications of PPR in Nigeria. By highlighting key knowledge gaps and operational challenges, it emphasizes the urgent need to strengthen early detection systems as part of the broader strategy to achieve global eradication of the disease by 2030.

Research Methodology

A structured literature search was conducted to gather data on the epidemiology, pathology, diagnosis and economic impacts of Peste des Petits Ruminants (PPR) in Nigeria. The search covered publications from 2018 to 2023 and utilized online databases such as PubMed, Scopus, Google Scholar, and African Journals Online (AJOL). Keywords used included “Peste des Petits Ruminants,” “Nigeria,” “epidemiology,” “diagnosis,” “economic impact,” “surveillance,” and “PPR rapid diagnostic kits.” Additional information was obtained through consultations with researchers specializing in livestock production, animal health and veterinary public health, who were contacted via email and invited to share relevant literature. The reviewed sources included peer-reviewed journal articles, national reports, conference proceedings, books of abstracts, online textbooks and scientific monographs. Non-English materials and non-Nigerian studies were excluded unless they provided significant contextual relevance.

Epidemiology of PPR in Nigeria

In support of more effective control strategies for PPR in Nigeria, there has been a growing body of research focused on the disease's prevalence, risk factors, diagnosis and prevention (Table 1). According to Esonu *et al*. (2022), at least one PPR-related study has been conducted in 30 of the 36 states, with most investigations centering on small ruminants. A notable serological survey by El-Yuguda *et al*. (2013) involving 2,879 animals (including 1,571 goats, 1,008 sheep, 192 cattle, and 108 camels) in the semi-arid North-East region, reported PPRV antibody seroprevalence rates of 76.5% in sheep, 51.6% in goats, 27.8% in camels and 16.7% in cattle. These findings underscore the endemicity of PPR among both ruminants and dromedaries in the region, particularly given their frequent co-habitation and inter-species interactions.

Regional studies across Nigeria (Table 1) have highlighted the persistent and geographically variable burden of PPR, reflecting a complex interplay of ecological, socio-economic and institutional factors that sustain the disease. The North-Western region of Nigeria, characterized by large-scale pastoralism and trans-boundary livestock movement, has shown measurable levels of PPR exposure in non-traditional hosts. Bello *et al*. (2013) reported a seroprevalence rate of 18.25% among dromedary camels in the region. Although camels are not considered primary hosts, accumulating evidence suggests that

both camels and large ruminants such as cattle may serve as subclinical carriers of the virus. This finding is significant because these species often co-graze with small ruminants in communal pastures and watering points, creating opportunities for inter-species virus transmission. The presence of sub-clinical infections further complicates disease surveillance and poses challenges to containment strategies, as these animals may not display clinical signs while still shedding the virus (Sen *et. al*., 2014).

The endemicity of PPR in these settings (as outlined above) is sustained by multiple factors, including unrestricted animal movement, insufficient veterinary oversight and gaps in farmer awareness. Such conditions are common in rural northern Nigeria, where small ruminant production remains a critical component of household income and regional trade. De Nardi *et al*. (2012) highlighted the importance of understanding these socio-ecological dynamics when designing control programs. Compounding the problem is the porous nature of Nigeria's international borders, which allows trans-boundary animal movements to proceed, largely unchecked. This further exacerbates the circulation of PPRV strains across regions and between neighboring countries.

Table 1. Summary of epidemiological findings on Peste des Petits Ruminants (PPR) in Nigeria.

Region/State	Study (Author, Year)	Species sampled	Sample size	Seroprevalence (%)	Key findings & Risk factors
North-East Nigeria	El-Yuguda *et al*. (2013)	Sheep, goats, camels, cattle	2,879	76.5% (sheep), 51.6% (goats), 27.8% (camels), 16.7% (cattle)	High cohabitation; evidence of subclinical infections in large ruminants and camels
North-West Nigeria	Bello *et al*. (2013)	Camels	160	18.25%	Low-level exposure; PPRV circulation confirmed among camels
South-East Nigeria	Chukwudi *et al*. (2020)	Sheep and goats	420	60.2% (Enugu), 40.5% (Anambra), 32.1% (Ebonyi)	Lack of vaccine access; low awareness; poor veterinary service infrastructure
North-Central Nigeria	Adedeji *et al*. (2019)	Sheep and goats	246	Not specified	Co-infection with goatpox; abortion and mastitis observed
South-West Nigeria	Lawal *et al*. (2011); Emikpe *et al*. (2013)	Goats	80	~35 – 40%	Clinical outbreaks: ocular and nasal discharge, coughing, fever; pneumonia and abortion common
North-Central (Camels)	Woma *et al*. (2015b)	Camels	Not specified	Detected	Camels may serve as reservoirs or spill-over hosts

In South-Eastern Nigeria, studies (Table 1) have further emphasized the significant seroprevalence of PPR among susceptible livestock species. Chukwudi *et al*. (2020) documented notably high seroprevalence rates among goats and sheep in Enugu State, with comparatively lower rates in neighboring Anambra and Ebonyi States. These variations are reportedly reflective of differences in herd size, movement patterns, veterinary service coverage, and farmer practices (Chukwudi *et al*., 2020). Several risk factors were identified, including a general lack of awareness among livestock farmers regarding the benefits of PPR vaccination, irregular access to veterinary services, and inconsistent availability of vaccines at local veterinary clinics (Chukwudi *et al*., 2020)

A key epidemiological concern in South-East region is the continued use of manure from infected animals as organic fertilizer, which may inadvertently contribute to environmental transmission of the virus. Ezeibe *et al*. (2008) reported that goats recovering from PPR infection can shed viruses in their faeces for a reasonable period of time post-recovery. When this faecal material is applied to farmland as manure (a common practice among smallholder farmers), it can serve as a potential source of indirect transmission. This underscores the need for greater education and risk communication efforts targeting rural communities.

In the South-West region (Table 1), serological surveys have consistently demonstrated moderate levels of exposure. For example, studies conducted between 2010 and 2013 reported true seroprevalence of 17 – 19% in states such as Ogun and Ondo (Esonu *et al*., 2022). More recent molecular surveillance (2017–2018) confirmed active viral circulation, with PPRV detected in goats and sheep from Oyo, Ondo, and Osun States (Mantip *et al*., 2022). These findings highlight that, despite ongoing vaccination campaigns, small ruminant populations in the South-West Nigeria remain at significant risk of infection.

In the North-Central region, the disease burden appears higher. Retrospective records from the National Veterinary Research Institute (NVRI), Vom (2013–2022) documented 600 suspected outbreaks, of which 34% were laboratory-confirmed as PPR, involving over 15,000 small ruminants with a mortality rate of approximately 23% (Chabiri *et. al.*, 2023). Molecular surveys further detected PPRV in goats from Plateau, Benue, and Kwara States, confirming active transmission in the zone. Importantly, phylogenetic studies indicate that circulating strains belong to Lineage II and IV, closely related to existing vaccine strains, suggesting that gaps in vaccination coverage, implementation and herd immunity rather than vaccine inefficacy may account for persistent outbreaks (Woma *et. al.*, 2015a).

Overall, these data indicate that South-West Nigeria maintains a moderate but sustained level of PPR endemicity, while North-Central Nigeria experiences higher outbreak frequency and mortality, reinforcing the need for strengthened vaccination programs, improved surveillance, and cross-border control measures.

Pathology of PPR

The clinical signs of PPR typically manifest within six days of natural infection, beginning with depression and a sudden onset of pyrexia, with rectal temperatures often exceeding 40°C. Following this initial phase, various signs usually emerge, including oculo-nasal discharges, pneumonia and diarrhea, typically occurring 5 – 8 days prior to death or recovery (Megersa *et al*., 2011; Truong *et al*., 2020). Other signs may include restlessness, decreased appetite and serous nasal discharge, which can progress from mild to mucopurulent, occasionally leading to severe

catarrhal exudate that crust over and occludes the nostrils, resulting in respiratory distress.

At post-mortem examination (Table 2), the carcass of an affected animal is often emaciated, with the hindquarters soiled by soft, watery feces and sunken eyeballs. Dried-up discharges can be found in the eyes and nose. Lesions in the respiratory system may include petechiation, necrosis and erosions of the nasal mucosa, turbinate and trachea, alongside pulmonary oedema and bronchopneumonia (Balamurugan *et al*., 2014). Additionally, there may be profuse catarrhal conjunctivitis, necrotic stomatitis and congestion with enlargement of the spleen, lymph nodes, and various lymphoid organs (Kumar *et. al*., 2014). [Table 2]

The pathology of PPR is characterized by retrogressive and necrotic changes in lymphoid tissues and the epithelial cells of the gastrointestinal and respiratory systems. Various studies have documented gross pathological changes in naturally occurring PPR in goats and sheep (Table 2). Prominent lesions in infected animals include consolidation and discoloration of the lungs, where frothy mucus may be observed upon squeezing cut lung samples, particularly in the antero-ventral areas of the right lung. The lungs may appear dark red or purple, firm to the touch, with lesions predominantly on the anterior and cardiac lobes. Consolidation of lung lobes and airway occlusion due to secondary bacterial pneumonia are common occurrences. Congested alveolar borders represent one of the most characteristic clinical and pathological changes in the lungs of goats with PPR.

Table 2. Common pathological findings in Peste des Petits Ruminants (PPR).

System/Organ	Gross Pathology	Histopathology	References
Respiratory system	Congestion of nasal mucosa; Mucopurulent oculonasal discharge; Pneumonia (especially bronchointerstitial).	Necrosis and desquamation of respiratory epithelium; Infiltration of mononuclear cells.	Emikpe *et al*., 2013; Mantip *et al*., 2016
Gastrointestinal tract	Ulcerations on dental pad, lips, gums, tongue, and esophagus; Zebra striping of colon.	Necrosis of epithelial lining; Lymphoid depletion in Peyer's patches.	Okwelum *et al*., 2017; Woma *et al*., 2015
Lymphoid tissues	Enlargement and hemorrhage of lymph nodes and spleen.	Lymphoid necrosis and depletion; Proliferation of reticuloendothelial cells.	Ezeibe *et al*., 2008; Chukwudi *et al*., 2020
Skin and mucosae	Crusting around mouth and eyes; Ulcers and erosions on mucocutaneous junctions.	Dermatitis with inflammatory infiltrates.	Adedeji *et al*., 2019
Lungs	Consolidation of cranial lobes; Fibrinous pneumonia; Pulmonary edema.	Interstitial pneumonia with thickened alveolar septa; Syncytial cells with viral inclusions.	Mantip *et al*., 2022
Other	Emaciation and dehydration; Abortion (in some outbreaks); Mastitis in mixed infections.	Mononuclear infiltration in mammary glands.	Akanbi *et al*., 2020; Chukwudi *et al*., 2020

The involvement of the respiratory system is significant, with pneumonia being a predominant lesion observed in over 90% of cases during PPR outbreaks (Abdollahpour *et al*., 2006; Zahur *et al*., 2009). Bronchopneumonia is consistently present, potentially accompanied by pleuritis and hydrothorax. The mediastinal and mesenteric lymph nodes are most commonly affected, appearing enlarged, oedematous and congested. Microscopically, tissues in the respiratory system, including the tonsils, show infiltration with neutrophils, formation of syncytial cells and intra-nuclear inclusion bodies.

The PPR virus shows a strong predilection for lymphoid tissues, resulting in profound immunosuppression in affected animals (Kumar *et. al.*, 2014). The virus replicates extensively in the spleen, thymus, lymph nodes, and Peyer's patches of the ileum, leading to lymphoid depletion, necrosis and follicular atrophy. This lymphoid pathology compromises immune competence and predisposes infected animals to secondary bacterial and parasitic infections, thereby exacerbating disease severity and mortality. In addition to lymphoid lesions, PPR is frequently associated with characteristic erosions in the oral cavity and gastrointestinal tract (Table 2). The gastrointestinal pathology is marked by congestion, hemorrhages and necrotic erosions along the abomasum, small intestine and large intestine (Njaa *et. al*., 2012). The Peyer's patches of the ileum are particularly affected, often showing necrosis and ulceration, which contribute to profuse diarrhea and dehydration which are the major causes of death in PPR outbreaks (Aleksandersen *et. al*., 2002). Collectively, the lymphoid, gastrointestinal and respiratory lesions underscore the systemic nature of PPR infection and its high case fatality.

Diagnosis of PPR Infection

The diagnosis of PPR is achieved through a combination of information on clinical signs, epidemiology, gross and histological tissue lesions and the detection of antibodies, antigens, or viral genomes in infected animals/samples using serological and molecular tests. A clinical presentation featuring oculo-nasal discharges, diarrhoea and deaths with respiratory distress in sheep and/or goats, without symptoms in contact cattle, should raise suspicion of PPR. Observing characteristic post-mortem changes can further strengthen a provisional diagnosis.

Common serological tests for diagnosing PPR include indirect enzyme-linked immunosorbent assay (ELISA), agar gel immunodiffusion and counter-immunoelectrophoresis (Dhinakar Raj *et. al*., 2008). Although useful, these conventional tests are often time-consuming and less sensitive. In contrast, molecular and cell culture techniques provide more rapid, sensitive and specific diagnoses of PPRV infection. Detection of PPRV RNA from clinical samples can be achieved using methods such as monoclonal antibody-based ELISA (Singh *et. al*., 2004), reverse transcriptase polymerase chain reaction (RT-PCR) (Balamurugan *et. al*., 2006), and real-time RT-PCR (Bao *et. al*., 2008).

In terms of cost implications, serological assays such as ELISA and agar gel immunodiffusion are relatively inexpensive, making them suitable for large-scale surveillance in endemic areas, though they may lack sensitivity for early detection (Sharma *et. al*., 2015). Molecular methods, including RT-PCR and real-time RT-PCR, are significantly more expensive due to equipment, reagent and expertise requirements, but they offer superior sensitivity and specificity, making them more appropriate for outbreak confirmation and targeted research (Mahapatra *et. al.*, 2019).

Cell culture techniques are the most resource-intensive and are primarily confined to research and vaccine production rather than routine diagnosis (Balamurugan *et al*., 2014).

Differentiation from other Diseases

PPR is frequently confused with other diseases presenting similar clinical signs and lesions, such as, Rinderpest, Foot and Mouth Disease (FMD), Contagious Caprine Pleuropneumonia (CCPP), Bluetongue (BT) and Contagious Ecthyma (Orf). Mixed infections with two or more of these viral diseases can occur, as there had been reported cases of PPR and adenovirus co-infection in Nigeria (Adedeji *et al*., 2019)

Rinderpest: Both PPR and Rinderpest affect cloven-hoofed animals and share clinical manifestations like pyrexia, conjunctivitis, oculo-nasal discharges, stomatitis and profuse diarrhea (Golchinfar *et al*., 2011). While some animals may recover from Rinderpest and develop lasting immunity, respiratory involvement is more prevalent in PPR. Additionally, Rinderpest occurs in small ruminants only when in contact with affected cattle or buffaloes, making it vital to examine all species during investigations. Rinderpest has been successfully eradicated in virtually all countries (Morens *et al*., 2011)

Foot-and-Mouth Disease (FMD): FMD is more common in sheep than goats. FMD is distinguished from PPR by the absence of respiratory distress and diarrhoea, alongside notable lameness. Sudden death of young lambs may occur without other signs. Oral lesions, when present, are typically small and hard to detect, and the mouth does not exude the foul odour characteristic of PPR (Balamurugan *et al*., 2014).

Contagious Caprine Pleuropneumonia (CCPP): This goat-specific disease caused by *Mycoplasma* species presents with fever, abnormal breathing and coughing, but lacks oral lesions and diarrhoea seen in PPR (Iqbal *et. al*., 2019).

Bluetongue (BT): Both BT and PPR cause oral lesions; however, in PPR these lesions are usually haemorrhagic, necrotic and erosive, and they often occur on the dorsum of the tongue. Respiratory involvement and severe gastrointestinal disease with diarrhoea are more characteristic of PPR. In contrast, BT is typically associated with coronitis, a feature not seen in PPR (Saminathan *et. al*., 2020)

Contagious Ecthyma (Orf): This condition may be confused with PPR due to the nodules and thick scabs that occur on the lips during later PPR stages. Confusion is particularly likely in severe Orf cases where lesions extend into the mouth and nose. However, uncomplicated Orf typically does not cause oral necrosis, diarrhoea or pneumonia. Furthermore, PPR is characterized by pyrexia, serous to purulent ocular and nasal discharge, diarrhoea and bronchopneumonia. Oral lesions in PPR include necrosis and erosion of the gums and buccal mucosa, while Orf presents with erythematous and ulcerative papules in the peri-labial area (Kumar *et al*., 2015).

Laboratory Diagnosis of PPR

Laboratory tests based on Antibody Detection: The diagnosis of PPR through antibody detection typically requires the collection of two blood samples from the same animal, spaced three weeks apart. While this method is feasible in experimental settings, it poses challenges in field conditions. Antibody surveys are crucial for determining the presence and extent of PPR infection in populations. The competitive enzyme-linked immunosorbent assay (c-ELISA) has largely replaced the virus neutralization test (VNT) due to its laboriousness, cost and requirement for infectious virus, that made it less suitable for large-scale routine testing. A rapid c-ELISA developed by Kang-Seuk Choi (2012) reportedly detects PPRV antibodies in serum

samples by quantifying the monoclonal antibody (MAb) P-3H12 after a 30-minute incubation period with a PPRV recombinant nucleocapsid protein, demonstrating specificity and sensitivity rates of 98.5% and 93.4%, respectively.

Laboratory tests based on Antigen Detection: PPRV is a pleomorphic virus with a ribonucleoprotein core enveloped by a lipoprotein membrane. The initial detection of virus antigens can be accomplished using the agar gel immunodiffusion test; however, this method does not differentiate between PPR and Rinderpest viruses. For more definitive identification, histopathology combined with immunohistochemical staining using specific monoclonal antibodies is employed. Furthermore, immunocapture ELISA provides a rapid and sensitive approach for antigen detection and can effectively differentiate between PPR and rinderpest (Golchinfar *et al*., 2011).

Laboratory tests based on the Virus Genome: The PPRV genome is a negative-sense, single-stranded RNA approximately 16 kilobases long. Molecular detection techniques, including conventional PCR, RT-PCR and real-time RT-PCR, are based on this genome structure. The first PCR method for PPR detection was described by Forsyth and Barrett in 1995, utilizing specific primers to amplify a nucleoprotein fragment. Although RT-PCR offers higher sensitivity, it is expensive and technically demanding. The advent of TaqMan technology and fluorescent probe methods has enhanced molecular diagnosis, providing rapid and specific results. Loop-mediated isothermal amplification (LAMP) has emerged as a cost-effective alternative for detecting PPRV (Kumar *et al*., 2014), with real-time RT-PCR currently being the preferred method for clinical samples (Garg *et. al*., 2022)

Isolation of PPR Virus: PPRV exhibits an affinity for lymphoid tissues, allowing isolation from the spleen and lymph nodes of infected animals (Taylor, 2002). Virus isolation can be performed by inoculating clinical samples into susceptible cell cultures, such as Vero or B95a cells, with the latter yielding significantly higher virus titres. Immunocapture ELISA can detect PPRV in cell culture supernatants, while the serum neutralization test, despite being a standard method for identifying neutralizing antibodies against PPRV, is less frequently used due to its labor-intensive nature (Kamal *et al*., 2018).

Globally, four lineages of PPRV (I, II, III, and IV) have been identified through sequence analysis of the nucleoprotein (N) and fusion (F) genes (Alemu *et. al*., 2019). Laboratory diagnosis progresses from antibody detection using c-ELISA to viral amplification via RT-PCR, culminating in virus isolation and molecular characterization through sequencing of the N or F genes, followed by phylogenetic analysis. Lineage I and II are prevalent in Western and Central Africa, lineage III in Eastern Africa and the southern Middle East, and lineage IV primarily in Asia (Munir *et. al*., 2012). There has also reportedly been a noted spread of Asian lineage IV into Central Africa, North Africa, and parts of East Africa (Alemu *et. al*., 2019).

Early Diagnosis of PPR by the use of a Rapid Antibody Test Kit:

Early diagnosis of PPR can be achieved by the use of a rapid diagnostic test kit. Commercially prepared test kits detect PPR virus antibody in blood or serum samples of infected animals. This technique is useful as it can achieve diagnosis in animals with low antibody titre, that have not started to manifest the clinical signs of PPR. Such early diagnosis allows for early management necessary to mitigate death and resultant economic loss. There are a few rapid test kits for PPR produced from isolated viruses. Quantitative or qualitative detection of PPR antibodies can be carried out using a variety of methods. The double

antigen sandwich method was adopted for the production of a rapid diagnostic test kit in which the antibody in the sample binds to the colloidal gold-coated small ruminant-H protein to form an antigen-antibody complex (Nankai Biotech Co., Ltd., 2025).

Economic Impacts of PPR in Nigeria

PPR is associated with severe morbidity, and mortality rates that can reach up to 90% (Govindaraj *et al*., 2023). The annual economic burden of PPR is estimated to exceed USD 1.2 billion globally (OIE/FAO, 2015). The significant economic losses stem from mortality, decreased production targets and the costs associated with disease prevention and management (George *et al*., 2001). These financial strains disproportionately affect smallholder rural farmers, who rely on small ruminants as essential family assets. These animals provide income through sales, organic manure for arable crops, and contribute to food security and the wool industry, making them integral part of their livelihoods (WOAH, 2024).

In Nigeria, the economic losses attributed to PPR are estimated at around USD 10.4 million annually (Fadiga *et al*., 2011; Dilli *et al*., 2011). The disease can decimate entire flocks, either independently or in conjunction with other diseases like pneumonia and gastrointestinal parasitism, directly impacting the livelihoods of rural, impoverished communities (Majiyagbe, 1985; Luka *et al*., 2011). During an epidemic, the mortality rate can exceed 90%, transforming a rural farmer's family into one facing severe poverty and food insecurity (Adamu *et al*., 2005; OIE/FAO, 2015). This loss undermines family livelihoods, affecting their ability to pay for education, healthcare, and essential food items. Furthermore, if an outbreak occurs during the arable farming season, the absence of organic manure can lead to poor crop yields, compounding economic losses as farmers often cannot afford commercial fertilizers.

The emotional toil of losing small ruminants is profound, as these animals represent not just a source of income but also a legacy to be passed down through generations. In certain Eastern Nigerian communities, gifting female small ruminants to new brides is a longstanding cultural practice tied to productivity. The death of these prized animals can be devastating, impacting traditional marriage ceremonies (Shamaki *et al*., 2004).

On the international trade front, the exporting of small ruminant meat and products is constrained by the risk of PPR transmission through animal imports (Zhang *et al*., 2022). The global market for small ruminant meat and live animal exports was valued at over USD 9.48 billion and USD 1.91 billion, respectively, in 2022, with significant potential for growth (OEC, 2022). Capturing a portion of this market could bolster national foreign exchange revenue and stimulate economic diversification, creating jobs across the small ruminant value chain.

The adverse impact of PPR on the livelihoods of poor farmers in Nigeria is significant and cannot be overstated. In addressing this challenge, collaboration among farmers, the government and non-governmental organizations is vital to developing effective solutions. Sustainable control and eradication strategies for PPR are projected to enhance returns on investment in sheep and goat husbandry, resulting in increased profitability and improved quality of life for farming communities. It is estimated that such initiatives could yield a benefit-cost ratio exceeding 30 and an internal rate of return surpassing 190% (Jones *et al*., 2016; Jemberu *et al*., 2022). Additionally, these efforts would contribute to the diversification and strengthening of economies in nations engaged in the small ruminant industry.

The Case for Early Detection and the Need for Rapid Diagnostic Tools

Despite existing vaccination campaigns and control efforts, the continued endemicity of PPR in Nigeria and across many parts of Africa underscores a critical need for timely detection of outbreaks. One of the major barriers to effective containment of the disease is the delayed identification of infected animals especially in rural and peri-urban settings where access to laboratory facilities is limited. For smallholder farmers and pastoralists, the early clinical signs of PPR (e.g., fever, ocular and nasal discharge, oral lesions) are often confused with other respiratory or mucosal infections. As a result, infected animals may remain in contact with the rest of the herd, facilitating rapid disease transmission before appropriate intervention is taken. This delay not only increases morbidity and mortality but also escalates the economic burden on livestock owners.

There is, therefore, an urgent need to deploy rapid, affordable and farmer-friendly diagnostic tools, such as lateral flow strip tests, that can be used in the field without requiring electricity, advanced training or sophisticated laboratory infrastructure. These point-of-care diagnostics can help detect PPRV antigens in ocular or nasal swabs within minutes, allowing farmers or community animal health workers to promptly isolate infected goats or sheep and initiate treatment and reporting protocols. The availability of such tools will significantly enhance community-based surveillance and reduce the interval between infection and response. By enabling livestock stakeholders to make real-time decisions on quarantine and treatment, these tests form an essential component of a holistic control strategy. They also align with the One Health principle of early outbreak containment, thereby preventing further spread across herds and potentially into neighboring communities.

Incorporating rapid diagnostic tools into the PPR eradication framework is no longer optional: it is an essential step toward achieving the 2030 global eradication goal. Early detection empowers farmers, strengthens surveillance systems, and minimizes losses, thus enhancing the overall resilience of livestock systems in endemic regions.

Conclusion

The epidemiological landscape of PPR in Nigeria reveals a complex interplay of factors influencing its persistence and spread. Understanding these dynamics is crucial for developing effective diagnostic strategies and implementing control measures. Given the similarities between PPR and other infectious diseases affecting cloven-hoofed animals, accurate differential diagnosis is critical to implement effective control measures. The economic impact of PPR on livestock stakeholders is profound, affecting not only individual farmers but also entire communities and national economies. Continued advancements in diagnostic technologies especially in the production of specific and sensitive rapid test kits, will further enhance livestock farmers' and owners' ability to monitor and combat PPR, ensuring the health and productivity of small ruminant populations. This review will serve as a lead for further research geared towards the development of a rapid and early diagnostic test for PPR.

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Conflict of interest

The authors declare no conflict of interest.

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