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Peste des petits ruminants (PPR) disease control and prevention

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Abstract The Peste des petits ruminant virus (PPRV) poses a significant threat to small ruminants, causing substantial economic losses in affected regions. The virus exhibits pleomorphic morphology and possesses a single-stranded, negative-sense RNA genome. Its structural proteins, including the nucleocapsid (N), phosphoprotein (P), matrix (M), fusion (F), hemagglutinin (H), and large (L) proteins, play crucial roles in viral replication, assembly, and immune evasion. The virus also encodes non-structural proteins, C and V, involved in modulating host immune responses. PPRV primarily infects sheep and goats, transmitting through respiratory aerosols and bodily fluids. The virus replicates in lymphoid tissues, leading to systemic spread and clinical manifestations such as fever, diarrhea, and pneumonia. The immune response to PPRV involves innate and adaptive components, with antibodies and T cells playing crucial roles in viral clearance. Various diagnostic techniques, including clinical examination, rapid tests, and laboratory assays, aid in PPRV detection and differentiation from other diseases. Control and prevention strategies encompass vaccination, quarantine, and biosecurity measures. Live-attenuated vaccines are widely used, but challenges persist, including heat stability and differentiation of infected from vaccinated animals. The development of thermotolerant and DIVA vaccines is crucial for effective PPR control. Ongoing research explores alternative vaccine approaches, such as subunit, vector, and nucleic acid, to enhance PPR prevention and contribute to global eradication efforts. The pursuit of innovative vaccine technologies and improved vaccination strategies holds promise for achieving the ambitious goal of eradicating PPR by 2030, safeguarding small ruminant populations and promoting food security in vulnerable regions.

Keywords: Immune response, PPR, Vaccination, virus replication

Introduction

Peste des petits ruminants is a highly infectious viral disease primarily impacting sheep and goats, posing a substantial risk to livestock. Certain wild ungulates are also vulnerable to infection. While cattle and buffalo can harbor the virus without displaying symptoms, they do not contribute to its further transmission (1). this easily transmissible disease has spread across Africa, the Near and Middle East, and Asia, causing significant economic losses due to livestock mortality (2). first described in 1942 in Côte d'Ivoire, Africa, by Gargadennec and Lalanne. Before this, PPR was likely misdiagnosed as rinderpest due to their similar clinical manifestations. Reports from Senegal (1871) and French West Africa (1927) probably documented PPR but were incorrectly identified as rinderpest (3) Since its initial identification, PPR has expanded beyond West Africa, reaching other parts of Africa, Asia, and the Middle East, with the potential for further spread (4). The current global initiative spearheaded by the FAO and OIE aims to eradicate PPR by 2030. To achieve this ambitious goal, it is crucial to understand the disease's specific epidemiological characteristics and identify the socio-economic factors that play a role in its transmission (5). By addressing these factors, effective strategies can be implemented to halt the spread of PPR and work towards its eradication.

Etiology

PPRV in genus Morbillivirus and family Paramyxoviridae. It has a single-stranded, negative-sense RNA genome exhibiting genetic variability (6). The virus shares close ties with other notable morbilliviruses that impact various hosts, such as the rinderpest in cattle, the measles in humans, and the canine distemper in dogs (7). The structural dimensions of PPRV virions encompass a diameter ranging from 150 to 700 nm, and their genome

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consists of 15,948 nucleotides. (8). They have six structural proteins (N, P, M, F, H, and L) that are essential for virion assembly and function, along with two non-structural proteins (C and V) produced through alternative open reading frames within the P gene (9). The viral envelope is adorned with spikes formed by the H and F glycoproteins, which are instrumental in the early stages of host cell infection (10). Although PPRV is categorized as a single serotype, phylogenetic analysis based on partial N or F protein sequences has revealed four distinct lineages (I, II, III, and IV). Historically, lineages I and II were predominant in West Africa, while lineage III was primarily located in East Africa and parts of India. The Asian lineage (IV) was mainly restricted to Asia, but recent molecular epidemiology studies have indicated its presence in Africa since 2008(11).

Morphological and Physicochemical Characteristics of PPRV

Electron microscopy and ultrastructural studies reveal that PPRV exhibits a pleomorphic morphology, appearing as either spherical or ovoid particles enveloped within a membrane (12). These intact virions range in size from 130 to 390 nanometers in diameter. The viral envelope, measuring 8 to 15 nanometers thick, is studded with glycoprotein spikes of 8.5 to 14.5 nanometers in length (13). Two key surface glycoproteins, designated F and H, are embedded within this membrane, as visualized in electron micrographs. The virions are further enclosed by eight lipoprotein bilayers, adorned with peplomer spikes ranging from 8 to 20 nm, and encapsulate a helical nucleocapsid with a characteristic herringbone pattern. (14). lipid envelope, make PPRV susceptible to inactivation by various environmental factors, including elevated temperatures, lipid solvents, and non-ionic detergents; exposure to 50°C for a mere 60 milliseconds or 37°C for two hours is sufficient to render the virus inactive. However, refrigerated or frozen tissues can persist for extended periods (15). PPRV is sensitive to pH extremes, losing infectivity at pH levels below 4 or above 11. It remains stable and infectious within a pH range of 5.8 to 9.5. Notably, the virus loses its activity postmortem, particularly during the acidic conditions of rigor mortis. (16).

Genome Organization

The PPRV genome, a linear RNA molecule, features untranslated regions (UTRs) at both its 3' and 5' ends, acting as promoters for replication and transcription (17). The 3' end houses the genomic promoter (GP), responsible for mRNA and antigenome synthesis, while the 5' end contains the antigenomic promoter (AGP), driving genomic RNA production. Both promoters possess conserved 3' terminal sequences essential for their activity (18). Transcription proceeds in a 3'-to-5' direction, initiated by the viral polymerase binding at the genome's 3' end. Each gene is demarcated by a conserved intergenic region (IG) typically bearing the sequence GAA, with exceptions at the L gene-trailer junction (GAU) and potentially between the H and L genes (GCA) in some strains. A U-rich region preceding the IG likely serves as a polyadenylation signal, and a conserved UCCU/C sequence marks the gene start. The intricate interplay of sequences at the genome's 3' end, IG regions, and gene flanks regulate the stop-start transcription mechanism (19). The PPRV proteins are arranged in the order 3'-N-P/C/V-M-F-HN-L-5'. Each mRNA transcript initiates with a conserved AGG trinucleotide and contains UUUU preceding each IG. Genes also possess variable-length UTR flanking their ORF (13)

Viral structural proteins

(N) protein

The nucleocapsid protein, a critical component of PPRV, is positioned at the 3' terminus of the viral genome; it is the first transcribed and generates the most abundant mRNA within infected cells. This protein comprises 525 amino acids, resulting in a molecular weight of approximately 58 kDa, and exhibits a conserved N-terminal core domain alongside a variable C-terminal domain(20); the primary role of this protein involves encapsulating the viral genomic RNA, forming a protective helical nucleocapsid structure that resists degradation by cellular ribonucleases(21). Furthermore, its significant abundance in infected cells underscores its crucial involvement in viral replication and assembly processes (22). The function of this protein is to disrupt the host's innate immune response by counteracting antiviral defenses, mainly through the inhibition of beta interferon (IFN-β) production and the subsequent activation of interferon-stimulated genes (ISGs) (23). With an essential role and conserved sequence, it has emerged as a valuable target for molecular characterization and differentiation of PPRV strains (24)

(P) Protein

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The phosphoprotein, encoded by a second gene within the viral genome, is comprised of 509 amino acids having an approximate MW of 55 kDa (25) and exhibits the lowest degree of genetic conservation compared to other viral proteins, possessing only limited, scattered regions of conserved sequence contributes to virus replication and pathogenicity, demonstrated by its ability to interact with the C-terminal domain of eIF2α, an essential protein in regulating fundamental gene expression (26). Additionally, it has been shown to inhibit interferon signaling by interfering with the JAK-STAT pathway, significantly reducing both IFN-β- and IFN-γ-induced phosphorylation of STAT1 (24).

(M) protein

Matrix protein is a crucial structural component with a well-conserved sequence and a defined role in viral assembly and release. Encoded by the ORF spanning nucleotides 3438 to 4442, translates into a relatively small protein of 335 amino acids, making it one of the smaller structural proteins within the morbillivirus (28), a high degree of conservation observed in its sequence, particularly the 92.5% similarity and 85.0% identity with a protein of RPV, underlines its functional significance in a virus lifecycle (26). This functional importance manifests in its multifaceted role during virion assembly and release. Firstly, it interacts with a ribonucleoprotein complex (RNP), which houses the viral RNA genome. This interaction is believed to be critical for forming and properly assembling new viral particles (30). Secondly, it is located on the viral envelope's internal surface, dealing with the cytoplasmic domains of two H and F glycoproteins (H and F) embedded within an envelope (10). This relation likely contributes to anchoring the glycoproteins within the viral envelope, potentially influencing their function during viral entry, and exhibits a unique feature, a triple ATG sequence, which is absent in some other morbilliviruses (31)

(F) protein

The fusion (F) protein plays a critical role in the virus's ability to infect host cells, making it a key target for the development of vaccines and antiviral therapies. The F protein facilitates the merging of the viral envelope with the host cell membrane, allowing the virus to enter and initiate infection (32). It consists of 456 amino acids and has a MW of 59.137 kDa. The F protein exhibits a high degree of conservation between PPRV and RPV and across all members of the Morbillivirus genus. This shared characteristic probably plays a role in the observed cross-protection between PPRV and RPV. (13). The F protein is a transmembrane glycoprotein situated on the viral envelope, forming spike-like projections. It is synthesized in the ribosomes of the rough endoplasmic reticulum and possesses a unique functional capability. In contrast to other paramyxoviruses that require multiple proteins for fusion, PPRV only needs the F protein to mediate both virus-host cell fusion and the fusion of infected cells (33). Additionally, research has identified hemolytic factors associated with PPRV F proteins (34). and their ability to trigger autophagy (31). Important role in viral infection has made it a subject of extensive research, with studies focusing on its nucleotide sequence, its contribution to disease development, and its role in eliciting immune responses, such as antibody-dependent cell-mediated cytotoxicity (ADCC) (35).

(H) protein

It is glycoprotein type II, composed of 609 amino acids. Characterized by a C-terminal ectodomain and an N-terminal transmembrane domain. Unlike other Morbillivirus proteins, it exhibits hemagglutination (HA) and neuraminidase (NA) activities, leading to HN designation. This dual functionality distinguishes it from the Measles virus, which only displays HA activity, and the RPV, which lacks both activities (36). It is implicated in various stages of viral pathogenesis and is pivotal in activating the innate immune response through Toll-like receptor 2 signaling (37). Furthermore, it mediates viral entry by interacting with two distinct cell receptors: the signaling lymphocyte activation molecule (SLAM) in immune cells and the Nectin-4 receptor in epithelial cells (10).

(L) protein

The substantial 247 kDa L protein of PPRV, comprised of 2,183 amino acids, is encoded by a gene located at the 5' terminus of the genome, occupying 40% of the total genomic sequence. It has enzymatic function as a polymerase, which requires only catalytic amounts as enzymes are not consumed during reactions. It is produced in the least quantity, so it is essential for gene replication and transcription, displaying RNA-dependent RNA polymerase activity that contributes to viral mRNA modifications such as capping and polyadenylation (30). It appears to have highly conserved sequences and functions across the Paramyxovirus family, with well-defined protein domains (38). Structurally, three major conserved

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domains are interconnected by hinge regions with variable sequences (39). The L protein exhibits a multi-domain structure with distinct functionalities. Its N-terminal domain mediates interactions with both N and P proteins. The central domain houses the polymerase active site, responsible for phosphodiester bond formation. Finally, the C-terminal domain facilitates ATP binding, enabling the protein's kinase activity (40).

Virus non-structural proteins

(C) protein

This relatively small protein, with a molecular weight between 19 and 21 kDa, comprises 177 amino acids in RPV and PPRV. It is generated by translating the P mRNA from an alternate ORF (41). the impact in modulating host immune responses, particularly by inhibiting the induction of interferon-β (IFN-β) (42). This antagonistic effect on IFN-β expression is believed to be a crucial mechanism for innate immune elusion and subsequent disease advancement (43)

(V) protein

It is descended from the P gene encoded by a specific mRNA., This specificity results from inserting one or more guanine residues during transcription, an event that reflects virus-specific modification (44). Smaller than the P protein, it has the same N-terminal sequence but diverges in the C-terminal region. It has 298 amino acid residues and has an estimated MW 32.3 kDa. Studies have shown that potent interferon antagonists help the immune system, as seen in morbillivirus infection (45).

PPRV replication cycle

The replication process is a multi-stage initiated by viral attachment to a host cell surface receptor (46), its surface, including hemagglutinin (H), facilitating binding to immune cells via sialic acid as well as CD150 and to epithelial cells through Nectin-4 (10). Both viral and host cell membranes blend upon binding, releasing the viral genome complexed with nucleoprotein into the cytoplasm. This ribonucleoprotein complex, consisting of an RNA genome and (N, P, L) proteins, is central to viral replication (2). Its L protein, owning RNA-dependent RNA polymerase activity, drives transcription and viral genome replication in conjunction with the P protein (47). Transcription entails the synthesis of capped and polyadenylated mRNA transcripts, which are translated by host cell ribosomes (48). The resulting viral proteins undergo post-translational modifications and are transported to the cell membrane (49). The replication process then transitions into a replicative mode, wherein the viral polymerase generates a positive-sense RNA template for new negative-sense genome RNA synthesis. This new RNA is encapsulated with viral proteins, forming new virions (50). A matrix protein is essential for the construction of new virions and egress, interacting with ribonucleoproteins and glycoproteins to facilitate budding from the plasma membrane (51).

Replication virus. (1). HN protein binds to receptor cells (SLAM or Nectin-4). (2). F and HN proteins mediate the entry. (3). Genome Release genome enters the cell. (4). Genome Release: genome enters host cell cytoplasm. (5). mRNA Synthesis: Viral RdRp transcribes mRNAs using a 'start-stop' mechanism for protein regulation. (6) Antigenome Synthesis: Full-length positive-sense RNA (cRNA) is synthesized.). (7). Protein Synthesis: two proteins are synthesized on RER (F, H), then modified in Golgi. Other proteins are synthesized on ribosomes. (8) New virions are assembled from viral proteins and genomes. (9). Budding: Progeny virions bud from the plasma membrane, acquiring their envelopes (13)

Epidemiology

History of outbreaks and global distribution

PPR and RPV, while closely related with origins tracing back to the late 4th century AD, are distinct pathogens that primarily affect large ruminants and have historically been associated with devastating outbreaks characterized by high mortality rates (11). PPRV was first documented in 1942 in the Ivory Coast of West Africa by Gargadennec and Lalanne. Initially, it was thought to be a novel virus due to its perceived inability to transmit from small ruminants to cattle, differentiating it from RPV (52). Following this initial identification, PPR awareness grew, with subsequent reports of the disease emerging in neighboring countries, including Senegal, Chad, Togo, Benin, Ghana, Nigeria, Oman, Sudan, Saudi Arabia, India, Jordan, Israel, Ethiopia, Kenya, and Uganda (11). World Organization for Animal Health data indicates that between 2015 and 2019, 12,757 PPR outbreaks were officially reported across 59 countries, with potential underreporting in 7 additional countries. Most outbreaks (75.1%) occurred in Asia and the Middle East, while Africa accounted for 24.8% of cases. Europe experienced a minor outbreak in Bulgaria, comprising 0.1% of the total (53) Historically, OIE and FAO officially reported PPR in Iraq in 1998(54)

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Host range

Several factors influence an animal's likelihood of contracting PPRV, including species, age, sex, immune status, seasonal changes, and geographic location (30). Sheep and goats are the main carriers of PPRV. However, there have been a few cases of illness outbreaks in camels (55). Cattle and pigs can serve as carriers of the virus without exhibiting any clinical symptoms (56). Infected animals can spread PPRV to nearby susceptible animals through inhaled aerosols, especially when coughing, or clinical excretions such as tears, nasal discharge, saliva, and feces. PPRV is sensitive to changes in temperature and is easily affected by them. It is rendered inactive when exposed to a dry environment outside of its host (57). Animals that have been infected and recovered establish a durable immune response that provides lifelong protection against infection and do not become disease carriers (58).

Pathogenesis

Upon entering the respiratory system, the PPRV replicates within the throat and neck lymph nodes. It then disseminates via immune cells such as macrophages and dendritic cells to lymphoid tissues like tonsils, eventually reaching systemic circulation. This process typically spans 4-6 days but can range from 3 to 10 days (59). The virus can also infect epithelial cells, the digestive tract, and lymphoid organs, leading to replication and necrotic lesions in these tissues. Destruction of lymphoid tissue causes lymphopenia and immunosuppression, increasing susceptibility to secondary infections (60). Clinical signs, including fever and appetite loss, typically manifest 3-4 days after this initial spread. PPR virus-induced necrosis in lymphoid organs like the spleen, thymus, and lymph nodes results in leukopenia. In experimental infections, fever typically emerges 3-7 days post-infection, followed by the onset of clinical symptoms, which can vary depending on the virus strain, inoculation route, and the animal's immune status (13).

Immune response

The innate immune system, the body's initial defense mechanism, identifies and responds to a variety of pathogens, including viruses, bacteria, parasites, and fungi. This recognition process is mediated by pattern recognition receptors (PRRs), which detect specific molecular structures on pathogens, known as pathogen-associated molecular patterns (PAMPs). TLRs, retinoic acid-inducible gene-I (RIG-I)-like receptors (RLRs), and Nod-like receptors (NLRs) are among the most important PRRs that trigger the immune response against invading pathogens (61). In the context of viral infections, various viral molecular signatures, such as double-stranded RNA, uncapped single-stranded RNA, CpG DNA, and specific viral proteins, are recognized by different PRRs, including TLRs 3, 7, and 8. These TLRs are strategically located in endosomal compartments of immune cells like dendritic cells and macrophages, enabling them to effectively detect viral nucleic acids released during viral uncoating and replication (62); these TLRs are located in endosomal compartments of immune cells, such as dendritic cells and macrophages, allowing them to effectively recognize viral nucleic acids released during the process of viral uncoating and replication, upon binding to their respective ligands, TLRs initiate a signaling cascade that leads to the activation of transcription factors like NF-κB and IRF3/7(63) This results in the production of proinflammatory cytokines, chemokines, and type I interferons (IFNs), which play key roles in antiviral defense by directly inhibiting viral replication and activating other immune cells(64). The adaptive immune response to PPRV involves both humoral and cellular components that collaborate to control and eliminate the viral infection. In humoral immunity, B cells encounter PPRV antigens and differentiate into plasma cells, which secrete antibodies targeting viral proteins. These antibodies neutralize the virus by attaching to its surface proteins, preventing it from entering host cells and facilitating its removal by phagocytic cells (65). Additionally, antibodies can trigger the complement system, destroying infected cells. Research has indicated that antibodies against the PPRV attachment (H) and fusion (F) proteins are particularly crucial for protection. (35). n cellular immunity, T cells play a vital role in the adaptive response to PPRV. Helper T cells (CD4+) identify viral peptides presented by antigen-presenting cells (APCs) and activate other immune cells, including cytotoxic T cells (CD8+) and B cells (66), CD8+ T cells directly eliminate virus-infected cells, while CD4+ T cells support B cells, enhancing antibody production and promoting the formation of memory B cells. (67).

Clinical signs

Clinical presentation of PPR varies based on factors like breed and pre-existing immunity, usually manifesting 2-6 days post-infection. Younger animals are more susceptible, though illness tends to be

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rapidly fatal in this group (68). PPR presents in mild, acute, or severe acute forms, with young goats exhibiting a per acute form. A short incubation period (typically 2 days) is followed by a high fever (41-42°C) and a general decline in condition (69). The acute phase is marked by persistent high fever (39.5-41°C), lethargy, loss of appetite, dry muzzle, drooling, eye and nasal discharge progressing to a thicker consistency. Erosive and necrotic lesions develop in the mouth (70). Respiratory signs like rapid breathing, neck extension, nasal flaring, and coughing may indicate pneumonia, developing lesions around the muzzle and lips, leading to dryness and clear ocular and nasal discharge that later becomes mucopurulent. Profuse, watery diarrhea, sometimes containing blood or mucus, is a hallmark of PPR and often leads to severe dehydration. In later stages, small nodules may appear on the muzzle. Death often occurs within 7-10 days due to dehydration, emaciation, and fever. Some animals recover, but prolonged recovery is possible (71). In young animals, PPR often leads to infertility and low birth weight. In adults, it frequently causes abortion, stillbirth, or delivery of weak, potentially deformed, or undersized lambs (72).

Diagnosis

The diagnosis of PPR involves a multifaceted approach, encompassing clinical examination, rapid diagnostic tests, and laboratory confirmation through gross pathology, histological analysis, and specific assays (73). Laboratory confirmation is crucial to differentiate PPR from other diseases with similar clinical presentations. Characteristic clinical signs include developing lesions around the muzzle and lips, leading to dryness and clear ocular and nasal discharge that later becomes mucopurulent. Additional signs include fever, pneumonia, profuse diarrhea, and inflammation of the respiratory and digestive tracts (69). Differential diagnoses include rinderpest, contagious ecthyma, contagious caprine pleuropneumonia, bluetongue, Pasteurella infections, and foot-and-mouth disease (65). Sample collection involves swabs from the eyes and nose and blood and potentially tissue samples from the large intestine, lungs, and spleen. Maintaining a cold chain during transport is essential. Serological and molecular diagnostic methods can detect PPR (74).

Laboratory techniques for virus detection include virus isolation, antigen detection, nucleic acid sequencing, and serum antibody detection (58). Gene detection by PCR, particularly TaqMan-based qRT-PCR targeting the N gene, offers high accuracy and specificity (75). Competitive ELISA using monoclonal antibodies can detect PPR-specific antibodies in the blood, which is valuable for assessing vaccine response. (76) The serum neutralization test, a long-standing serological assay, quantifies protective antibodies by evaluating their ability to neutralize viral infectivity in cell culture(77). This test, mandated by the OIE for international trade, involves incubating serial serum dilutions with a virus before adding it to a susceptible cell culture. The absence of cytopathic effect (CPE) indicates the presence of neutralizing antibodies, and the titer is the highest dilution without CPE (78) .

Control and Prevention

The global strategy for controlling and eradicating PPR with the ambitious goal of complete eradication by 2030 was formally endorsed at the 2015 international conference held in Abidjan, Ivory Coast, under the auspices of the FAO and OIE (79). Early intervention during the acute phase of the disease involves isolating symptomatic animals and administering hyperimmune serum in conjunction with aggressive fluid therapy to counteract the dehydration resulting from severe diarrhea. Such interventions can significantly mitigate mortality rates associated with fluid loss and electrolyte imbalances. Additionally, meticulous cleaning of lesions around the nostrils, lips, and eyes is essential for effective supportive care (80). In regions where PPR is not endemic, proactive measures like stringent import controls, restrictions on animal movement, and targeted vaccination of high-risk populations are paramount. Combined with quarantines, humane depopulation of infected and exposed animals, and comprehensive decontamination of affected premises, these strategies can collectively lead to eradicating this devastating disease (81).

PPR Vaccines

There are various types of vaccinations now being used or developed to combat Peste des Petits Ruminants (PPR) table (1); these include live-attenuated PPR virus, as well as vaccines where PPRV proteins are expressed in viral vectors routinely used for other diseases. Multi-valent preparations are also available (Hodgson, 2020). The pursuit of effective PPR mitigation strategies has led to the development and exploration of diverse vaccination approaches. These approaches include using vaccines based on

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live-attenuated PPR virus and those employing viral vectors traditionally associated with other diseases to deliver PPRV proteins. Furthermore, the development of multivalent formulations aims to provide comprehensive protection against multiple pathogens (82); however, the historical practice of employing the Plowright RPV vaccine in small ruminant populations was discontinued. This decision stemmed from concerns regarding potential interference with the maintenance of rinderpest-free zones (83).

Table 1: PPR vaccine (84)

Types	Features
Vector vaccine	
vaccinia virus (RVV)	expressing F and H proteins
Modified Vaccinia virus (M V A)	of PPRV
CPV (capripoxvirus)	
canine	
adenovirus type-2 (rCAV-2)	
adenovirus (red)	
Chimeric kind	
	BV carrying a membrane-bound form of
baculovirus (BV)	H protein of PPRV
	BV carrying immunodominant
	ectodomains of F protein of PPRV
Reverse genetics kind	
Recombinant PPRV (rPPRV)	rPPRV expressing green fluorescent protein GFP) or
	FMDV(VP1)
Subu subunit kind	
H protein	H protein of PPRV expressed in transgenic peanut plants
VLP (Virus-like Particle)	VLP is composed of PPRV M and H (or F) proteins
Nucleic acid kind	
Suicidal DNA	Recombinant pSCA1 plasmid expressing H protein of PPRV

Live attenuated vaccines

1989 marked a turning point in PPR vaccination with the advent of the Nigeria 75/1 strain vaccine. This revolutionary progress significantly reduced the reliance on off-label RPV vaccines for PPRV control. This strain, derived through serial passage on Vero cells (85)., This vaccine, widely used, offers robust and long-lasting protection against PPRV (86); the Sungri 96 is another PPR strain also used as a vaccine strain, both conferring strong protection against all known disease strains when properly administered (22). Numerous other live-attenuated vaccines are employed in various regions (Table 2). However, current vaccines face limitations, including poor heat stability and the inability to distinguish vaccinated from field-infected animals; the development of thermotolerant and DIVA vaccines is paramount to address these challenges. Such advancements would facilitate broader PPR vaccination coverage, strengthening herd immunity and enabling DIVA features (87).

Table 2: PPR attenuated vaccines used in different countries (88)

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Producer	Product Name		Viral Strain/Subtype	Country of Licensure
Botswana Vaccine Institute	PPR-VAC®		Nig 75/1	Botswana
Central Veterinary Control and Research Institute	Freeze-Dried PPR Vaccine			Turkey
Dollvet	Pestdoll-S			Turkey
Intervac (PVT) Ltd.	Pestevac			Pakistan
JOVAC	PESTEVAC			Iraq, Jordan
National Veterinary Institute	PPR Vaccine			Ethiopia
Nepal Directorate of Animal Health				Nepal
Vetal Company	Pestvac K™			Turkey
Veterinary Serum and Vaccine Research Institute	PPR-TC Vaccine		Egypt 87	ARE
IVRI	PPRV Sungri/96		Sungri 96	
TANUVAS	TN MIB /87	Arasur 87		India
	PPR Vaccine - TN/97			
		Coimbatore 97		
Hester Biosciences Limited	PPR Vaccine		Sungri 96	
Biosciences Limited			Nig 75/1	
Indian Immunologicals Limited	Raksha PPR		Sungri 96	
National Veterinary Research Institute	PPR Virus Vaccine		PPR V 75/1	Nigeria

Inactivated vaccines

While live-attenuated PPR vaccines are effective in regions where the disease is endemic, veterinary authorities in areas accessible to PPR, like Europe, often discourage their use due to potential risks. Inactivated vaccines represent the only viable alternative in these regions (89). Consequently, recent advancements have been made in developing and testing new formulations of inactivated PPR vaccines. Cosseddu created a vaccine that offered

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sterile immunity and resisted virulent PPRV when tested. However, it was necessary to provide two doses of the vaccine (90)

Subunit vaccine

Subunit vaccines represent a specific vaccine comprised solely of purified pathogen components, including proteins, polysaccharides, or peptides. These components are crucial for eliciting a defense reaction but lack the complete pathogen. Notably, subunit vaccines are considered safer than live-attenuated or inactivated vaccines due to the absence of causing the disease they aim to prevent (91). While these vaccines are widely recognized and suitable for immunocompromised individuals, they may necessitate adjuvants and booster doses to achieve optimal efficacy, and examples of these types of vaccines are recombinant H and F protein vaccines (6).

Vector vaccines

Vector vaccines represent a cutting-edge advancement in vaccinology, harnessing non-pathogenic microbes known as vectors. These vectors serve as a "Trojan horse," delivering specific genetic material into host cells. Through genetic engineering, vectors are modified to incorporate DNA or mRNA fragments encoding a particular antigen from a disease-causing agent. This antigen is subsequently produced within the host cells, triggering an immune response (92). Currently, researchers frequently utilize viral vectors derived from sources such as adenovirus, measles virus, influenza virus, and poxvirus, Viral vectors can be classified into two primary groups: those that retain the capacity for replication within the host (attenuated) and those that lack this ability (inactivated). Non-replicating vectors offer enhanced safety compared to replicating counterparts (93). However, the development of vector-based vaccines presents challenges due to the intricate nature of genetically modifying vectors while ensuring the safety and efficacy of the resulting vaccine. Furthermore, pre-existing immunity to the vector, whether present in the population or acquired through prior immunization, may compromise the vaccine's effectiveness (94).

Nucleic acid vaccines

These vaccines represent a groundbreaking advancement in vaccinology, utilizing DNA or RNA molecules to deliver genetic instructions for producing pathogen-specific antigenic proteins. DNA vaccines primarily employ plasmids as carriers for the genetic material, while RNA vaccines predominantly rely on encapsulation within lipid nanoparticles (95). In DNA vaccines, the introduced DNA is transported to the nucleus of host cells, where it is transcribed into mRNA and then exported to the cytoplasm. After that, ribosomes translate it into the desired antigen, which is then processed and presented to immune cells, eliciting a targeted immune response (96). RNA vaccines, in contrast, bypass the nuclear transcription step, allowing direct translation of the antigen within the cytoplasm (97). This technology has demonstrated particular relevance during the COVID-19 pandemic, with notable examples including the BNT162b2 and mRNA-1273 vaccines (98).

mRNA vaccine

It represents a groundbreaking advance in nucleic acid vaccines, laying the groundwork laid by previous generations of attenuated or inactivated pathogen-based subunit vaccines. This new class essentially includes both mRNA and plasmid DNA vaccines and holds great promise for future development (99). The basic principle underlying mRNA vaccines is the delivery of RNA to host cells, instructing them to produce protein antigens that subsequently induce immunity to these proteins, reinforcing immune mechanisms (92).

Conclusion

Peste des petits ruminant (PPR) is a highly contagious viral disease that primarily affects sheep and goats, causing significant economic losses. The virus is transmitted through respiratory aerosols and bodily fluids, and replicates in lymphoid tissues, leading to systemic spread and various clinical signs. The immune response to PPRV involves both innate and adaptive components. Diagnosis involves clinical examination, rapid tests, and laboratory confirmation. Control and prevention strategies include vaccination, quarantine, and biosecurity measures. Live-attenuated vaccines are widely used, but challenges persist, and the development of thermotolerant and DIVA vaccines is crucial. Ongoing research explores alternative vaccine approaches to enhance PPR prevention and contribute to global eradication efforts.

Conflict of interest

Authors declare no conflict of interest.

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