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Review

Peste des petits ruminants: past, present, and future scope

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Abstract

Introduction: Peste des petits ruminants (PPR) is a highly contagious and fatal disease affecting small ruminants, particularly goats and sheep, and is caused by *Morbillivirus caprinae*, a virus in the genus *Morbillivirus*, family *Paramyxoviridae*. PPR has significant economic and social impacts, especially in Africa, Asia, and the Middle East, where small ruminants are vital to rural livelihoods and food security. This disease is a priority for global eradication due to its disproportionate impact on low-income farmers and wildlife conservation.

Methodology: A literature review was conducted to capture recent information on the epidemiology, diagnosis, host range, transmission, pathogenesis, economic impact, vaccination strategies, and eradication efforts for PPR. This review also explores future perspectives to address gaps in the current understanding and control of the disease.

Results: The review highlights that PPR remains a severe challenge in low-resource areas, causing notable economic loss and endangering wildlife. Vaccination efforts have shown effectiveness, though limited accessibility and high costs persist as barriers. The disease has gained attention from international organizations aiming for global eradication by 2030, with ongoing advancements in diagnostics and surveillance methods showing promise in control efforts.

Conclusions: This review underscores recent advancements in PPR research, focusing on disease distribution, diagnostic improvements, and control strategies. These findings are valuable for regional and global eradication initiatives, providing a foundation for policies that support sustainable livestock economies and biodiversity conservation. International collaboration, effective vaccination programs, and strategic surveillance are essential to achieve the 2030 eradication goal and secure the health of vulnerable livestock populations.

Key words: *Morbillivirus*; livestock; pathogenesis; eradication.

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Introduction

Peste des petits ruminants disease (PPR) is an acute, highly contagious, transboundary disease affecting small ruminants such as sheep and goats [1]. PPR is an economically important disease and is notifiable by the World Organization of Animal Health (WOAH). The disease is endemic in large parts of Africa, the Middle East, and Asia, causing severe socioeconomic losses, especially in developing countries where small ruminant production is a significant contributor to the economy [2]. Nearly 80% of the global small ruminant population is at risk due to the threat of PPR [2]. The approximate estimation of the global PPR losses is 1.45–2.1 billion USD annually [2].

PPR is a disease of small ruminants caused by a virus that has changed its name several times. Until 2015, it was known as pest des petits ruminants virus (PPRV). The following year, it was renamed small ruminant morbillivirus. In 2022, it received its current name, *Morbillivirus caprinae* virus (MCV). This virus is part of the *Morbillivirus* genus and has a negative-sense single-stranded RNA genome. It is classified in the *Orthoparamyxovirinae* subfamily of the *Paramyxoviridae* family [3]. The MCV genome encodes eight proteins including the nucleocapsid protein (N), phosphor-protein (P), matrix protein (M), fusion protein (F), haemagglutinin protein (H), polymerase protein (L), and two non-structural proteins C and V [1].

The recent findings in prevalence, molecular biology, diagnosis, and pathogenesis — which are essential for improving our understanding of PPR and developing effective strategies to combat the disease — are reviewed in this article.

Current geographical distribution of PPR

Based on recent epidemiological data, the virus is present in 70 countries, with an additional 50 countries being categorized as “at risk”. The first official report of MCV infection was in Ivory Coast, West Africa, in

1942 [3]. The increased awareness and attention to the disease since its first report has led to its reporting in several neighboring countries, including Senegal, Chad, Togo, Benin, Ghana, and Nigeria [3,4]. Subsequently, MCV exhibited a northwards leap into northern Africa, invading several countries such as Tunisia in 2006, Morocco in 2008 and 2015, and Algeria in 2011 and 2016 [5]. Alongside this, the disease was also reported in Sudan, Ethiopia, Kenya, Uganda, Angola, Burkina Faso, Djibouti, Egypt, Eritrea, Gabon, Gambia, Guinea, Liberia, Libya, Mali, Mauritania, Niger, Sierra Leone, Somalia, South Sudan, Cameroon, Jordan, Palestine, Israel, Oman, Saudi Arabia, India, and Pakistan [1,6,7]. In East and Central Asia, the virus spread to the Ngari region of western Tibet in 2007 [8] and caused several outbreaks in China during 2013–2014 [9]. Currently, the disease is present in Iran, Turkey, Mongolia, Georgia, and Tajikistan [10].

Although Europe is free of PPR, several outbreaks were reported in Turkey from 1999 to 2018, which represent a significant threat to mainland Europe as a source of disease spread [11]. These outbreaks were reported in Thrace (the European part of Turkey), including Istanbul in 2000, Edirne (bordering Greece) in 2004, and Kırklareli (bordering Bulgaria) in 2006 [11]. In July 2018, seven PPR outbreaks involving nine herds were reported in the Yambol region, which is located 10 kilometers from the Turkish border, by the Bulgarian authorities to the European authorities [12].

The exponential spread and wide geographical expansion of PPR over the last two decades; beyond its original endemic region in Africa to areas of the Middle East, Central and South Asia, and most recently to Bulgaria; indicates its huge transboundary potential.

Host range and susceptibility

Sheep and goats are considered the main hosts for MCV. The morbidity rate can reach up to 100%, and the mortality rate can reach up to 90% [13]. In one field study in India, MCV infection was slightly more prevalent in sheep [13]. Further studies established that the disease was more severe in goats than in sheep; however, the recovery rate was higher in sheep [14]. The greater susceptibility of the goat population to MCV infection can be explained by the fact that goats were exposed to the disease at an earlier age than sheep [14]. In addition, different breeds of sheep and goats may differ in their susceptibilities to the infection. In one study, West African dwarf goats showed severe symptoms of MCV infection, while those belonging to the West African long-legged species had milder symptoms [15]. In addition, the Saanen goat breed was found to be more susceptible to the disease when infected with different MCV strains [16]. Moreover, Guinean and British sheep breeds have been reported to be highly susceptible, whereas Sudanese sheep breeds failed to develop characteristic clinical symptoms [17]. This variation in susceptibility to MCV infection represents a promising area for continued research, which may substantially affect the dynamics of disease transmission.

Over the last two decades, the host range of MCV has expanded dramatically and included other livestock and wild animal species. Some of these animals are infected clinically by MCV in a similar manner to domestic sheep and goats, while others show no apparent clinical symptoms but produce antibodies against MCV. There have been several reports of MCV infection in cattle and buffaloes. In these studies, varying seroprevalence rates of MCV antibodies were reported. For example, in southern peninsular India, 5.21% of cattle and 4.82% of buffaloes were MCV seropositive, whereas in Pakistan, 41.86% of cattle and 67.42% of buffaloes were MCV seropositive [18,19]. However, in all these studies, infection in cattle and buffaloes was sub-clinically not apparent or non-lethal. Therefore, it was suggested that cattle and buffaloes were unlikely to act as MCV reservoirs and did not play a role in the maintenance and transmission of MCV; hence, cattle and buffaloes were considered dead-end hosts for MCV [18]. Seropositive camels for MCV have been documented in several countries including, Ethiopia, Nigeria, and Sudan [20]. A fatal disease in camels caused by MCV was also reported in Sudan in 2014. The disease mainly affected pregnant and recently delivered camels, causing diarrhea, abortion, and sudden death. The average mortality rate was 7.4% [21]. In another study, it was found that camels can develop a subclinical infection with MCV and can transmit it to sheep and goats [22]. These findings indicate that camels are not a dead-end host, and the virus transmissibility between sheep, goats, and camels needs to be considered when designing control measures for PPR.

There have been several reports of natural MCV infection in wild ruminants, including gazelles, ibexes, bharals, wild goats (*Capra aegagrus*), wild sheep (*Ovis orientalis*, *Pseudois nayaur*), chowsingha (*Tetracerus quadricornis*), and water deer (*Hydropotes inermis*) [23]. White-tailed deer (*Odocoileus virginianus*) were experimentally infected with two strains of MCV, and the response varied from fatal consequence to subclinical infection. The reported clinical signs and gross lesions were similar to those reported in goats

[24]. In another study, West African dwarf goats were experimentally infected with different isolates of MCV, which resulted in acute to mild disease [25]. The susceptibility of wild ruminants to the disease is of high importance because this disease can pose a serious threat to the survival of endangered species of wild ruminants, and interaction between these infected wildlife and domestic species may increase the risk of interspecies transmission and thus play an important role in PPR epidemiology, especially in areas of high wild ruminant density [26].

Transmission

MCV is transmitted mainly through aerosols and direct contact with ocular, nasal, and oral secretions, as well as fecal material from infected animals [27,28]. In addition, disease transmission can occur through the oral route by ingestion of contaminated feed, pasture, and water [29,30]. A recent study reported that the peak MCV nucleic acid detection in different bodily fluids from infected goats was between 5 and 10 days post-infection. Therefore, this period must be considered the most infectious period for contact transmission [31].

In experimentally infected goats with MCV, a high viral load was observed in nasal excretions from two days post-infection until at least two weeks post-infection. On the other hand, percentage sample positivity was low in both eye swabs and saliva samples during the early stage of infection [31]. Moreover, MCV was detected later post-infection in fecal material than in other body fluids. However, the detection was intermittent; therefore, nasal swabs are considered the best sample for early diagnosis of the disease [31]. Fecal samples were suggested as a better alternative to standard ocular or nasal swabs for MCV surveillance in wildlife and livestock [32]. In addition, it was reported that fecal shedding of the viral antigen continued for 12 weeks post-recovery in goats [33]. Nevertheless, it is not well-known whether exposure of other animals to excreted viruses can transmit the disease since MCV is quickly inactivated in the environment [34].

Pathogenesis — pathological and clinical manifestations

Even though MCV can induce high mortality and morbidity in livestock, the pathogenesis of PPR remains poorly understood and most of the knowledge is based on comparison with the closely related rinderpest virus (RPV) and other morbilliviruses [35].

In an experimental study, goats were challenged with a virulent MCV isolate by the intranasal route which mimics natural infection [36]. The initial site for virus replication was not within the epithelial cells of the respiratory mucosa, as has been previously reported, but was within the tonsillar tissue and lymph nodes draining the site of inoculation. The virus appeared to be taken up by immune cells within the respiratory mucosa which then transported it to lymphoid tissues where primary virus replication occurred, and from there the virus entered circulation [36]. This idea is supported by a recent study in which viral RNA was detected in serum samples from experimentally infected goats; suggesting the role of circulating immune cells in transporting the virus to host target organs [16]. The lung is regarded as the main target organ for most morbilliviruses, except RPV [36]. However, lung infection was found to be a late event in PPR and occurred only when there was a high viral load [37]. Like other morbilliviruses, MCV is both lympho- and epithelio-tropic [38]. Infection of sheep with virulent MCV causes immunosuppression, as indicated by leukopenia and lymphopenia during the acute phase of the disease, providing a window of opportunity for the virus to replicate and spread [38,39]. Previous studies have reported an association of MCV with extensive necrosis and collapse of Peyer's patches in both natural and experimental infections. Additionally, the disease was associated with conjunctivitis, rhinotracheitis, and erosive-ulcerative lesions in the alimentary tract including ulcerative stomatitis, gastroenteritis, and bronchointerstitial pneumonia [40]. Affected animals often developed erosive and necrotic papules in the oral cavity. In severe cases, these necrotic papules appeared with fibrin deposits on the tongue. In the later stages, there was diarrhea and coughing with labored abdominal breathing. In the final stages of the disease, the infected animals showed dyspnea, weight loss, and eventually died. In mild infections, spontaneous recovery occurred within 10–15 days of infection [41]. The reported incubation period in natural as well as in experimental MCV infection was typically 4–6 days, although it may range from 3–10 days [5,37–39]. It is generally accepted that the severity of the disease depends on the MCV strain, host species, breed, health, and immune status of infected animals [42].

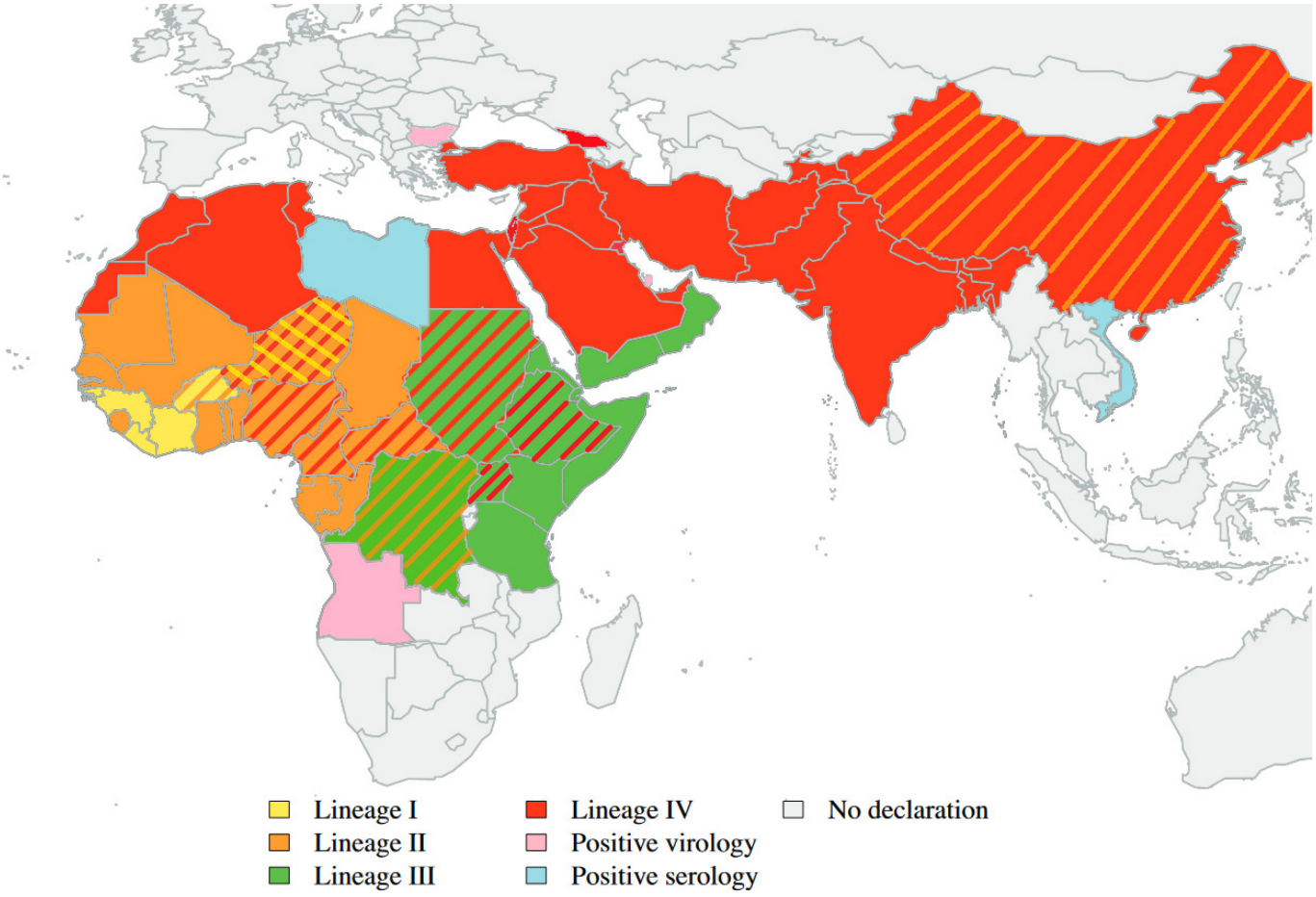
Molecular biology

MCV is an enveloped RNA virus with a negative-sense, single-stranded RNA genome [3]. The virion genome size is 15,948 nucleotides, encoding six structural proteins, including the nucleocapsid protein (N), phosphoprotein (P), matrix protein (M), fusion protein (F), hemagglutinin (H) protein, and large (L) protein [1]. The P and L proteins form the viral RNA-

dependent RNA polymerase for the viral transcription of mRNA [34]. The nucleocapsid protein N plays a central role in viral replication and transcription by binding to the viral RNA genome, forming a helical nucleocapsid structure that protects the viral RNA from degradation and serves as a template for replication and transcription. The N protein also triggers the formation of stress granules (SGs), which are dynamic cytoplasmic structures that promote viral replication by facilitating the assembly of viral replication complexes. Additionally, the N protein interacts with other viral proteins, such as the P and L proteins, to facilitate the packaging of the viral RNA into new viral particles [43]. The fusion glycoprotein (F protein) plays a vital role in virus-induced hemolysis, cell fusion, and the initiation of infection [44]. The matrix protein M serves as a link between the glycoproteins and ribonucleoprotein complex (RNP), mainly containing the viral RNA and the N protein surrounding it [45]. The L protein is involved in elongation, capping, cap methylation, and polyadenylation, and possesses kinase activity [46].

Based on nucleotide sequence analysis of the F and/or N genes, MCVs are divided into four distinct genetic lineages (types I to IV) [47]. The lineages I, II, and III were found to circulate in Africa [47]. The fourth lineage (type IV) was until recently confined to Asia, including Turkey and the Arabic peninsula. However, in 2008, it was isolated from a PPR outbreak in Morocco, indicating its first introduction into Africa [47]. It is now found from Sudan to northern Africa (Algeria, Morocco), as well as central Africa and the Gulf of Guinea [48]. In Senegal and Mauritania, a similar scenario has occurred with lineage II, originating from Central Africa and moving to West Africa, whereas in the 1980s, lineage I was the dominant, if not the only lineage, found there [48]. The updated geographic distribution of MCV lineages is shown in Figure 1.

Figure 1. Updated geographic distribution of MCV lineages. Different colors show different lineages. The pink color indicates virological evidence of PPR infection. The blue color indicates serological evidence of PPR infection but no virus isolated. The grey color indicates missing information or disease never reported. Figure adapted from [48].



Diagnosis

Early and accurate diagnosis of MCV infection; together with the availability of simple, practical, and inexpensive diagnostics and laboratory-based tests; is critical for prompt control of PPR, especially in most endemic, low-income countries of Africa and Asia [41]. The presumptive diagnosis of PPR is based on clinical observations, characteristic symptoms, epidemiology, and post-mortem lesions. However, a definitive diagnosis of the disease using laboratory tests is the key to achieving accurate results because MCV lesions are often mistaken for other diseases such as bluetongue, contagious caprine pleuropneumonia, capripox, and foot-and-mouth disease [49].

Since its discovery, a series of serological and molecular diagnostic assay tools have been developed for the diagnosis of PPR with continuous development in recent years. Serological tests such as agar gel immunodiffusion (AGID) and counter-immunoelectrophoresis (CIE) were among the earliest developed diagnostic tests. Progress was made to develop highly sensitive ELISA techniques such as immunocapture enzyme-linked immunosorbent assay (IC-ELISA), sandwich-ELISA, and dot-ELISA based on conventional antibodies; these techniques exhibited high diagnostic sensitivity and specificity [41].

Due to the advances made in nucleic acid-based testing (NAT) methods, various molecular assays, including conventional reverse transcription-polymerase chain reaction (RT-PCR) and real-time RT-PCR (RT-qPCR) are currently used for the diagnosis of PPR [50]. Nested RT-PCR assay for the detection of the MCV nucleic acid by targeting the N-protein gene was recently developed. This technique has the advantage of yielding high-quality DNA that is necessary for conducting sequence analysis to identify circulating lineages [51]. These molecular assays are rapid, sensitive, and more reliable as they do not rely on the isolation and identification of a live virus; nevertheless, these techniques are time-consuming and prone to high risk of cross-contamination. In addition, the high cost of equipment and technical demands impede its utility in low-income countries [41].

Economic impact, vaccination, and control

In Asia and Africa where PPR is endemic, outbreaks of disease impose significant economic costs, threaten food security, and affect the livelihood of smallholder livestock farmers for whom sheep and goats are often the main assets [2]. MCV can affect up to 90% of exposed animals in the flock with a mortality rate that can reach 90% [34]. Losses are classified as direct production losses associated with mortality and morbidity; and indirect losses, which include treatment costs, loss of animal body condition, reduction in market value, disease control, and vaccination costs. In addition, PPR can add restrictions to international trade in livestock and their products [52]. It is estimated that the global annual loss due to PPR ranges from USD 1.45 billion to USD 2.1 billion [53]. This huge impact can be reduced through control and eradication of the disease, which will increase the profitability and productivity of small ruminant herds [53].

Mass vaccination, and control of sheep and goat movement, in addition to vaccination monitoring and disease surveillance are good approaches to controlling the disease. However, problems related to logistical constraints and knowledge gaps prevent the implementation of this strategy. These challenges include the development of cost-effective thermotolerant vaccines because most of the PPR-endemic regions have a hot climate and they usually have poor infrastructure that cannot maintain the cold chain needed to preserve vaccine potency and efficacy, lack of knowledge on the roles of wildlife populations and domestic species other than small ruminants in PPR spread, the socio-economic and biodiversity impacts of PPR disease, efficiency of PPR related animal health services, farmers perceptions and acceptance of PPR vaccination, and their decision making around disease control [54].

The MCV Nigeria 75/1 lineage II and Sungri 96 lineage IV live attenuated vaccines, which are currently used in the field against MCV, have been shown to induce protective immunity against all four genetic lineages of MCV when challenged subcutaneously or intranasally in experimental studies [55]. Nevertheless, these live attenuated vaccines prevent differentiation of infected from vaccinated animals, which would hinder the control and surveillance programs in the vaccinated areas. In addition, the low thermal tolerance of the vaccines can be problematic in PPR-endemic countries, which have very limited cold-chain resources for vaccine storage and transport [56]. Currently, a new generation of vaccines against PPR are being developed which address the disadvantages of live attenuated vaccines. These alternative approaches include inactivated vaccines such as the Morocco/2008 MCV [57], DNA vaccine candidates based on a Semliki Forest virus replicon expressing the MCV-F or -H genes [58], and the use of recombinant viral vectors that express MCV immunogenic proteins [59]. Regardless of the technology used, these vaccines must go through strict controls and trials for their safety and efficacy.

To date, control strategies are mainly based on annual national mass vaccination campaigns or focal vaccination in response to overt outbreaks [2]. However, this method in practice is expensive and difficult to achieve because of the higher turnover of sheep and goat populations. A more effective time-bound strategy is therefore required which will achieve eradication and avoid the need for long-term costly control programs.

Progress control pathway for PPR and future perspectives

During the last few years, PPR has spread to other parts of the world and is now circulating in Africa, Asia, and the Middle East [6,7]. The disease is still spreading globally and has emerged in new areas in Mongolia, Georgia, and recently within the European Union in Bulgaria where a PPR outbreak was reported in 2018 [32].

A total of 4,120 PPR outbreaks were reported to the World Animal Health Information System (WAHIS) from 2019 to 2023; among them, 1,810 outbreaks were in Asia and the Middle East, 2,310 outbreaks were in Africa, and no PPR outbreaks were in Europe (except in Bulgaria) [32].

PPR is currently the target of a Global Eradication Program (PPR GEP), launched in 2015, and aimed at eradicating the disease by 2030 based on a progressive reduction in PPR incidence and spread, through targeted vaccination [60]. This program relies on a four-stage stepwise approach: in stage 1, countries are advised to focus on assessing the local epidemiological situation; in stage 2, control activities including vaccination are implemented; in stage 3, eradication is pursued by strengthening surveillance and preventive measures; lastly, in stage 4, vaccination is suspended, and the country must provide evidence that no virus is circulating at the zonal or national level and that it is ready to apply for official World Organisation for Animal Health (WOAH), formerly the Office International des Epizooties (OIE) PPR-free status [32]. Following the end of the first five-year phase of the PPR GEP (2017–2021), an assessment of the status of the stepwise control and eradication process focusing on the Middle East concluded that substantial shortcomings were observed in surveillance and disease reporting, as well as in the implemented control strategies, most notably vaccination [60]. It should be emphasized that the ability of any country to control and eradicate the disease depends on the availability of diagnostic and surveillance systems, appropriate national vaccination programs, improved biosecurity, animal identification and traceability of both within-country and cross-border animal movements, and improved public-private-community partnerships. Although the eradication of PPR seems appealing, a highly concerted effort, including capacity building, technical support, and huge financial commitment are needed to control this disease in endemic areas. Moreover, surveillance and epidemiology in endemic areas are of great significance to control the entry of the virus into new areas.

Conclusions

PPR is a highly contagious transboundary disease affecting domestic and wild small ruminants with a considerable impact on rural economies and the livelihoods of small farmers in Africa, Asia, and the Middle East. PPR is widespread in Asia, Africa, and the Near and Middle East and is emerging as a threat to other continents, such as Europe; therefore, concerted efforts at the national, regional, and global levels are essential to control and ultimately eliminate the disease.

Variations in susceptibility to MCV infection among different breeds and species highlight the complexity of the disease and the need for further research. Additionally, the expanding host range of MCV to include other livestock and wild animals, with varying clinical responses and seroprevalence rates, emphasizes the importance of continued surveillance and study of this infectious disease.

Gaps in understanding transmission factors, host range, epidemiology, and vaccine efficacy pose a risk to the success of the OIE PPR eradication campaign by 2030. The global eradication of PPR by 2030 requires a multi-faceted approach, including robust surveillance systems, effective national vaccination programs, and strong public-private-community partnerships. The successful implementation of these strategies hinges on substantial financial investment, capacity building, and technical support to enhance diagnostic capabilities and improve biosecurity measures.

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