Chapter 1
Peste des Petits Ruminants: An Introduction

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Abstract Peste des petits ruminants virus (PPRV) is an acute, highly contagious, and economically important transboundary disease of sub-Saharan Africa, Middle East, Indian subcontinent, and Turkey. It is one of the World Organization for Animal Health (WHO) notifiable diseases and is considered important for poverty alleviation in PPRV-endemic regions. Significant research has been directed toward improved vaccine, diagnosis, and epidemiology of the virus in recent years; however, research on fundamental aspects of the virus is required, especially when disease spectrum and distributions patterns are increasing. This chapter is designed to provide an overview of each chapter that is describing comprehensively a specific aspect of PPRV in the book.

1.1 An Overview

Peste des petits ruminants virus (PPRV), the causative agent of peste des petits ruminants (PPR), is a member of genus *Morbillivirus* within subfamily *Paramyxovirinae* of the family *Paramyxoviridae* (Gibbs et al. 1979). PPRV is relatively recently diagnosed virus; therefore, most of our understanding on virus structure and molecular biology is based on the comparison with other morbilliviruses such as measles virus (MV), canine distemper virus (CDV), and rinderpest virus (RPV). Based on this comparison, PPR virions are pleomorphic particles and are enveloped (Fig. 1.1). The genome (15,948 nt in length) encodes sequentially for the nucleocapsid (N) protein, the phosphoprotein (P), the matrix protein (M), the fusion (F) and the hemagglutinin–neuraminidase (HN) membrane glycoproteins, and the large (L) protein (viral RNA-dependent RNA polymerase, RdRP) (Fig. 1.1) (Michael 2011; Munir et al. 2013). As with other morbilliviruses, it is only the P gene that encodes for two or three non-structural proteins, V, W, and C, through

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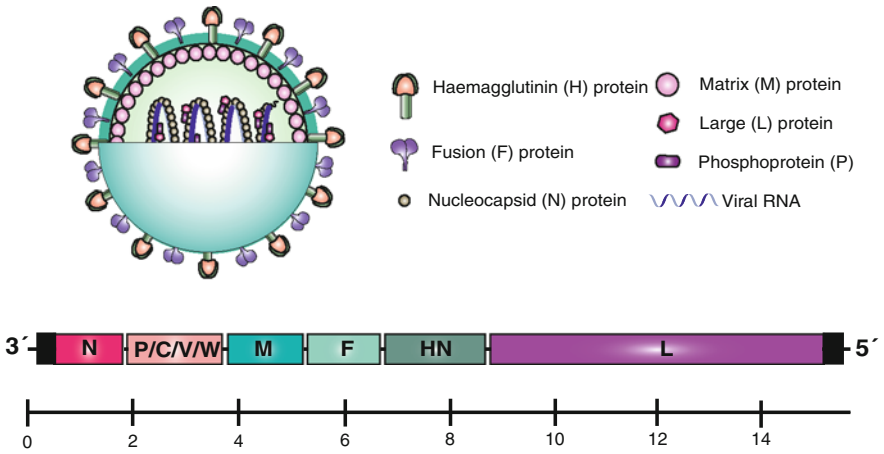

Fig. 1.1 Schematic diagrams of a Morbillivirus and its genome. Modified from Munir (2014b) with permission

"gene editing" or "alternative ORF" mechanisms. The available information on functions of each of these genes is recently reviewed by Munir (2014b), and Michael (2011) and is described compressively in the next chapter (see Chap. 2). Two essential components of PPRV life cycle, replication and transcription, are essentially regulated by genome promoter (3′ end of the genome), antigenome promoter (5′ end of the genome), and intergenic sequences between individual genes. Our understandings on the preference over replication or transcription mode are insufficient; however, different hypotheses have been proposed due to functional similarities of PPRV with other morbilliviruses (see Chap. 2). With the availability of complete genome sequences from all lineages of PPRV (Bailey et al. 2005; Muniraju et al. 2013; Dundon et al. 2014) from both vaccine strains and filed isolates, and due to the availability of reverse genetics (Hu et al. 2012), it is expected to see a surge in the research on the biology of PPRV and its pathogenic potentials in diverse hosts.

Among PPRV proteins, it is the HN protein that determines the initiation of viral infection and is the main determinant of host range selection through interaction with cellular receptors (sialic acid, signaling lymphocyte activation molecule (SLAM), and ovine Nectin-4) (Pawar et al. 2008; Birch et al. 2013). Beside presence of these receptors in several mammals, sheep and goats are remained to be the natural hosts. However, the host spectrum of PPRV has now expanded from sheep and goats to several wildlife species and to camels (Kwiatek et al. 2011; Munir 2014a). The disease can be equally severe in sheep, goats, or wild small ruminants; however, the clinical manifestation varies widely (Lefevre and Diallo 1990; Wosu 1994; Munir 2014a) (see Chap 3). Briefly, after an onset of high fever and inappetence for 1–2 days, lesion (congestion, serous to mucopurulent discharges) spread over oral and respiratory mucosa. These lesions cause *functio laesa* in these organs and lead to

cough, dyspnea, and diarrhea on third day post-infection. This clinical picture further aggravates and culminates in severe pneumonia and dehydration, and reasons 90 % mortality in immunologically naïve populations within 5–10 days. Multiple studies have revealed comprehensive disease progression, clinical scoring, and virus antigen distribution patterns in multiple organs of small ruminants (Eligulashvili et al. 1999; Munir et al. 2013; Pope et al. 2013) (see Chap. 4). Collectively, these studies indicate that the multiplication and pathogenicity of the virus are proportional to that of the host resistance or innate resistance, host's immune response, host density, the nutritional level of host, the breed, sex, and age of the animal (reviewed in (Munir et al. 2013)) (see Chap 3, 4). PPRV has high tropism for epithelial and lymphoid organs and thus leads to profound immunosuppression, which makes the infected animals vulnerable to secondary infections (Kerdiles et al. 2006). Consequently, concurrent infections aggravate the clinical outcome of PPRV by potentiating the severity of the PPR infection in immunodeficient host resulted from PPRV-induced lymphocytolysis (see Chap. 7). However, interestingly, the convalescent animals develop lifelong immunity despite immunosuppression and infection of opportunistic pathogens.

Beside its natural hosts, PPRV has been reported in cattle, domestic, and wild African buffaloes (*Synceruc caffer*) without severe consequences. Moreover, PPRV is now considered a pathogenic and emerging virus of camelids and wild small ruminants of at least *Gazellinae, Tragelaphinae, and Caprinae* subfamilies. PPRV can cause severe illness in wild small ruminants and camels; however, it is unclear whether these animals shed or transmit virus or play any role in the epizootiology of PPRV (Munir 2014a).

The disease is infectious and of emerging transboudary nature, which expanded from sub-Saharan Africa to Middle East, Turkey, and the Indian subcontinent rapidly. Up to present time, Food and Agriculture Organization (FAO 2009) has estimated that about 62.5 % of the total small ruminant population is at risk to PPR, around the globe, especially those from southern Africa, Central Asia, Southeast Asia, China, Turkey, and southern Europe. Recently, disease has been reported from previously disease-free countries such as China, Kenya, Uganda, Tanzania, Morocco, Eritrea, and Tunisia (Banyard et al. 2010; Cosseddu et al. 2013; Munir et al. 2013; Munir 2014b) (see Chap. 5). Initially, F gene-based classification was adapted for genetic characterization and for phylogenetic analysis, which was later shifted to N gene owing to its potential to depict better epidemiological patterns (Kwiatek et al. 2007). Currently, either N gene or both genes (N and F) are used for classification of PPRV strains into four distinct lineages (I, II, III, and IV). Recently, it is also suggested to use surface glycoprotein, HN, for epidemiological linking in addition to F and N gene-based analysis (Balamurugan et al. 2010). Regardless of the genes used, this classification has been only used for geographical speciation and is not indicative of stain pathogenicity or host preference. Lineages I, II, and III were considered African and the Middle East lineages, whereas lineage IV was reported exclusively from Asian countries. However, (i) this lineage (lineage IV) has been recently reported from several countries of Africa (Sudan, Uganda, Eritrea, Tanzania, Tunisia, and Mauritania) despite being still prevalent in

Asia (Banyard et al. 2010; Kwiatek et al. 2011; Cosseddu et al. 2013; Munir et al. 2013; El Arbi et al. 2014; Munir 2014b; Sghaier et al. 2014); (ii) most recent reports of PPRV in previously PPRV-free countries belong to lineage IV, (iii) countries once exclusively carrying a single lineage are now simultaneously reporting the presence of several lineages, i.e. Sudan and Uganda. In the majority of these cases, the newly introduced lineage is lineage IV (Kwiatek et al. 2011; Luka et al. 2012; Cosseddu et al. 2013) (see Chap. 5); and (iv) it is only lineage IV that is isolated from wild small ruminants (Munir 2014a) (see Chap. 6). These results indicate that lineage IV is a novel group of PPRV, has potential to replace the other lineages, and might be evolutionary more adaptive to small ruminants.

Our knowledge on current epidemiology has expanded significantly especially in small ruminants. Beside often distinct clinical picture, the availability of proficient assays for both the serology and genetic detection of the virus has contributed significantly in understanding current epidemiology of the disease. Favorably, convalescent and vaccinated small ruminants develop an early (10 days post-virus–host interaction), strong and lifelong immunity, which favor the detection of PPRV antibodies under comparatively limited resources or when sophisticated equipments for genetic detection are not available (Libeau et al. 1994). The N protein of morbilliviruses is highly conserved and is the most abundant protein owing to promoter-proximal location in the genome. Based on extensive analysis of monoclonal antibodies (mAbs) screening, selective anti-N mAbs have been used in the development of ELISAs for detection and differential diagnosis of PPRV (Libeau et al. 1994, 1995). These assays are currently in use for moderate laboratory diagnosis of PPRV (see Chap. 8). Monoclonal antibodies raised against the HN protein of PPRV have also been used in establishment of both competitive ELISA (c-ELISA) and blocking ELISAs (B-ELISA) (Saliki et al. 1994; Libeau et al. 1995; Singh et al. 2004a, b). Since antibodies against HN protein are virus-neutralizing, per se, detection of mAbs elicited against HN protein of PPRV correlates better with the virus neutralization test and immune status of the host (Saliki et al. 1993; Libeau et al. 1995). Beside antibodies detection, mAbs-based immunocapture ELISA and sandwich ELISAs (s-ELISA) have been developed and are extensively being used for the detection of antigen in both clinical and laboratory specimens (Libeau et al. 1994; Singh et al. 2004b). One of such assays, developed at Centre de Coopération Internationale en Recherche Agronomique Pour le Développement (CIRAD), France, is internationally recognized and applied for antigen detection. These assays have variable sensitivities and specificities, however, are generally at acceptable levels (Balamurugan et al. 2014). Despite availability of efficient serological assays, extensive seromonitoring has not been conducted in unvaccinated animals to estimate the prevalence of the disease. Such seromonitoring setup and information are crucial to assess the efficacy of the vaccination campaigns. However, like rinderpest eradication program, clinical surveillance will be an important marker of success in any campaign leading to disease control.

For the detection of PPRV genome, different polymerase chain reaction (PCR) chemistries, including conventional PCRs, real-time PCRs, multiplex real-time PCRs, and LAMP-PCR, have been developed to easily detect genome of PPRV,

independent of lineage variations. These assays have been designed based on the conserved sequences in the F gene (Forsyth and Barrett 1995), N gene (Couacy-Hymann et al. 2002; George et al. 2006), M gene (Balamurugan et al. 2006; George et al. 2006), and HN gene of PPRV (Kaul 2004). A conventional PCR, targeting the F gene, has extensively been used for the detection of genetic material of PPRV from clinical specimens with great success (Forsyth and Barrett 1995). Moreover, the amplified segment of F gene is long enough to draw epidemiological analysis. Owing to mismatches at the 3′ end of these primers, this PCR may not be suitable for lineages-wide detection in future. As alternatives, PCR assays targeting M and N genes have been established for specific detection of PPRV in clinical samples collected from sheep and goats (Shaila et al. 1996; Couacy-Hymann et al. 2002; Balamurugan et al. 2006; George et al. 2006) (see Chap. 8). Despite high sensitivities and specificities of these diagnostic assays, currently these assays are incapable in differentiating four lineages of PPRV strains. This is of special concern in the countries where more than one PPRV lineages are prevalent or emerging (Chaps. 5 and 9). There is also need of assays that can differentiate PPRV from diseases that show same clinical picture in animals in the event of co-infection. Currently, virus isolation is not a well-adopted model for identification of PPRV, especially for viruses that are causing new outbreaks. However, recently a new cell line that expresses SLAM/CD150 receptor has been demonstrated to be highly permissive for PPRV (Adombi et al. 2011). Moreover, an alpine goat was found to be highly susceptible to a Moroccan strain of PPRV (Hammouchi et al. 2012) and may present an experimental model in future.

Host immunological responses, in terms of innate and adaptive, are sufficiently investigated (Munir et al. 2013). Relative and definitive contributions of humoral and cell-mediated immunity in protection provided hallmarks of vaccine evaluation and provided bases of protection in both replicating and non-replicating vaccines. Our current knowledge on the immunodominant epitopes on the N and HN proteins, both for B and T cells, can be exploited for the Differentiating Infected from Vaccinated Animals (DIVA) vaccine construction. Efforts have already been started in establishing DIVA vaccine especially with the success of reverse genetic system (Hu et al. 2012) (see Chap. 10). After availability of the heterologous vaccine (RPV-based), which provided long-lasting protection, interests emerged to establish homologous vaccine for PPRV. As a result, a highly efficient vaccine, providing lifelong protection with single injection, became available in 80 (Diallo 2003). Currently, different vaccines have been developed which provide lifelong protection to reinfection and have provided foundations to establish effective control strategies. Homologous marker and subunit vaccines are proven to be effective and are now extended to build multivalent vaccines (see Chap. 12). Most of available vaccines provide lifelong immunity (6-year protection for a life span of 4–6 years in small ruminants) after even a single administration; however, the thermal stability of these vaccines is poor (half-life 2–6 h post-reconstitution at 37 °C), especially in the climatic conditions in tropical countries where disease is endemic. Current efforts have been successful in extending the thermostability (5–14 days at 45 °C in lyophilized form, whereas 21 h at 37 °C in reconstituted form) (Worrall et al. 2000;

Silva et al. 2011). Such improvements are sufficient for the shipment of PPRV vaccines in remote areas without maintaining the cold chain. However, no such vaccine has been launched in the market. Taken together, we have significant understanding of the level of protection, duration of immunity, antigenic profile, and thermostability of PPRV vaccines. While the experimentally proven vaccines are in abundance, there is still need to formulate the mechanism either for domestic production or for easy access to these vaccines especially in countries where disease is endemic.

Beside importance of disease management, availability of diagnostic assays and vaccines, it is imperative to ascertain the factual impact of the disease both at research and government levels. Comprehensive research needs to be conducted to ascertain the economic impact of the PPR on trade, export, and import of new animal breed especially out of the disease-endemic countries and into the disease-free countries. Public awareness is a central component for prioritizing the utilization of public funds in animal research. Since turnover rate of sheep and goat (natural hosts of PPR) is significantly lower than large ruminant, a well-designed cost-benefit analysis will be a critical criterion to plan the disease control program and to prioritize the research interests (see Chap. 12).

Cumulative efforts, initiated by the reference laboratories, and supported and followed on by the national laboratories and policy makers, would determine the fruitful outcome of disease control. Depending on the regional disease surveillance, individual vaccination of susceptible population (lambs and kids over 5 months) every year followed by carpet vaccination of all small ruminants every 3 years, occasional pulse vaccination, establishment of immune belt at the borders, and efficient sero-monitoring are crucial for the success of any efforts in controlling the diseases globally. Moreover, two countries each from Asian and African continents should drive the control and eradication campaign by combining their strengths and should be monitored by the international agencies such as FAO/OIE and GPRA would lead to faster accomplishment of much-needed goal of PPRV eradication (see Chap. 13).

1.2 Conclusions and Future Prospects

Molecular biology of PPRV is poorly understood and requires intensive efforts from developed laboratories to ascertain the host–pathogen interactions and to pinpoint the differences that might exist between PPRV and other morbilliviruses that might help to understand the host restrictions of the virus and its possible future expansion especially when PPRV is currently reported from a lion and when its spectrum is expanding to camels. It has now clearly been established that PPRV is an endemically important disease for poverty alleviation. However, epidemiological features such as transmission dynamics in different agro-climatic conditions require future investigations. The disease transmission has recently become important with the report of disease in wild ruminants and camels. The disease outcome is dependent on multiple factors and studies have just begun to understand any

genetics or non-genetic factors for this outcome. Epidemiologically, PPRV is expanding and this expansion is mainly contributed by the lineage IV of PPRV. Functional studies are required to understand the evolutionary mechanisms for the fitness of lineage IV over other lineages. Development and use of specific diagnostic tests that can distinguish PPR from diseases with similar signs helped unquestionably to improve our knowledge and understanding in the geographical distribution and spread of the disease in specific areas. Moreover, we are currently lacking a real-time assay that can differentiate different lineages of PPRV, which might be prevalent simultaneously in the country for proficient profiling of the lineage distribution.

In conclusion, although we have successful eradication model of rinderpest, it has to be kept in mind that "PPRV is not rinderpest and small ruminants are not large ruminants" for any initiative to be made for the control and eradication of PPRV.

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