

## Chapter 2

# Replication and Virulence Determinants of Peste des Petits Ruminants Virus

**Abstract** The first interaction of the host and pathogen is initiated by receptor binding, which is mediated by the hemagglutination-neuraminidase (HN) protein of Peste des Petits Ruminants Virus (PPRV) and sialic acid on the host cell membrane. A siRNA-mediated study has confirmed that signal lymphocyte activating molecules (SLAM) could be a putative co-receptor for PPRV. As in all paramyxoviruses, RNA-dependent-RNA polymerase (RdRp) binds to the genome promoter, which is a stretch of the nucleotides before the nucleocapsid open reading frame that initiates transcription in a “stop-start” fashion with the contribution of other viral proteins such as matrix, nucleocapsid, and phosphoprotein. Viral budding occurs through the neuraminidase activity, which cleaves sialic acid residues from the carbohydrate moieties of glycoproteins. Some of these steps in the replication of PPRV are not fully defined yet. However, among morbilliviruses, PPRV is unique in which the HN protein performs both hemagglutination and neuraminidase actions, so better reflected as an HN protein instead of H protein. The virus propagation and pathogenicity is directly proportional to that of the host’s immune response, parasitic infection, the nutritional level of host, and the age of the animal. This chapter highlights recent studies on PPRV replication, transmission, and the factors, both host and non-host, affecting virus propagation in the host.

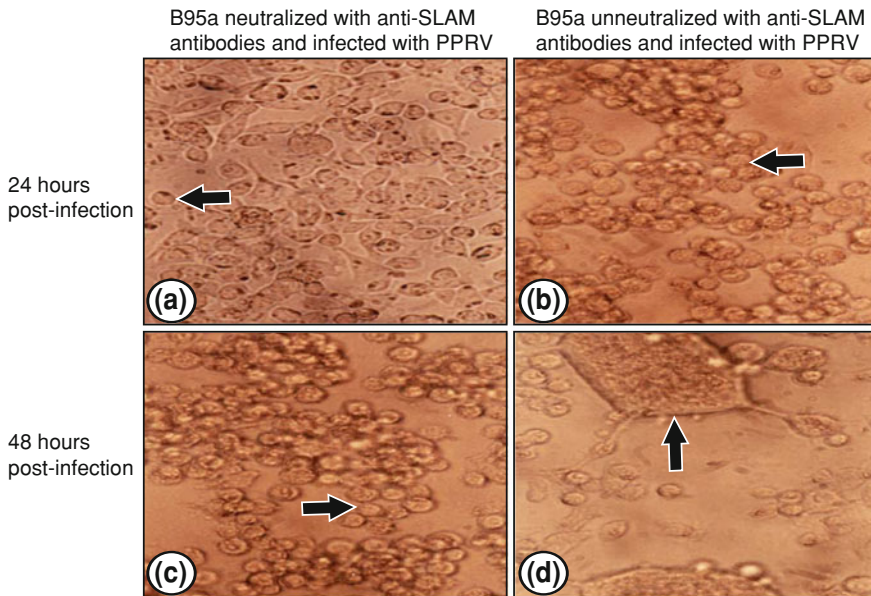
**Keywords** Virus lifecycle • Replication • Virulence • Host determinants of pathogenesis

## 2.1 Introduction

The first interaction of the host and pathogen is mediated by receptor binding. PPRV interacts with the host cell membrane through the hemagglutinin-neuraminidase (HN) protein and sialic acid receptors. However, other receptors are also likely to exist for PPRV. Our understanding of PPRV replication and transmission is not fully elucidated yet, although there are significant contributions that make the study of PPRV life cycle possible. These findings have also provided bases to establish further studies and make reliable comparisons to other morbilliviruses. A highly valuable tool, reverse genetic system, will be needed to make the virus life cycle definite, and to investigate the roles of host and non-host factors in virus replication and virulence. This chapter will provide an overview of PPRV with regard to its replication, transmission, and virulence, while highlighting recent studies that have expanded our knowledge about the molecular biology of PPRV and finally lead to establish bases for its possible control.

## 2.2 PPRV Replication and Life Cycle

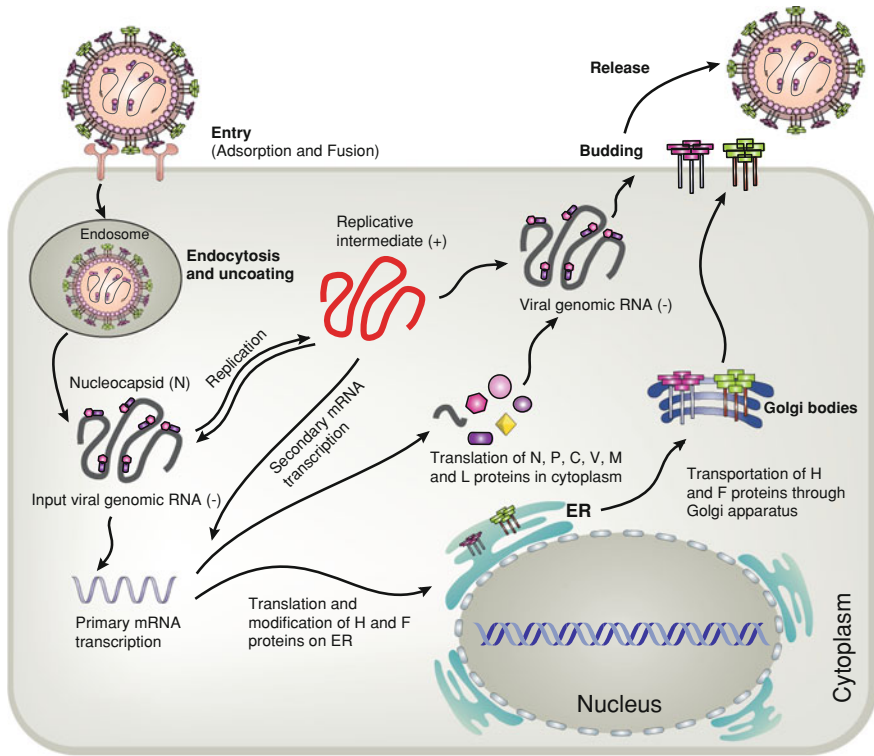
The first and the foremost step in viral replication is the virus–host receptor interaction. PPRV interacts through the HN protein with sialic acid on the host cell membrane (linked in  $\alpha$  2–3 linkage), which is suggested by the hemagglutinating activities of the PPRV for pig and chicken erythrocytes (Renukaradhya et al. 2002). Recently, a small interfering RNA (siRNA)-mediated study confirmed that SLAM could be a putative co-receptor for PPRV (Pawar et al. 2008). It was revealed that suppression of the SLAM receptor lead to the reduction of the PPRV titer by  $\log_{10}$  1.09–2.28 fold (Fig. 2.1). Additionally, sheep and goat SLAM expressing monkey CV1 cells showed high susceptibility for PPRV growth, and proved to be a reliable source for virus propagation from pathological samples (Adombi et al. 2011). The property to grow in these cells remained identical for other members of morbilliviruses, such as measles virus (MV), canine distemper virus (CDV), and rinderpest virus (RPV). This virus–host interaction, followed by F protein-mediated fusion, leads to release of the nucleocapsid from the viral envelope (illustrated in Fig. 2.2). The large (L) protein then works as an RdRp and initiates the transcription of messenger RNAs (mRNAs) in the cytoplasm. As in all paramyxoviruses, RdRp binds to the genome promoter, which is a stretch of the nucleotide before the nucleocapsid (N) open reading frame (ORF) that initiates transcription in a “stop-start” fashion. There is a series of transcription attenuations across each gene junction, a natural justification for the protein quantity required for the viral replication. The mRNA for the N protein, being at the N-terminus, is most abundantly transcribed and is required most, while the L protein, which is required only in a small amount, is least transcribed due to its being far away from the genome promoters. A quantitative estimation has been made for MV, where P, M,



**Fig. 2.1** Inhibition of the SLAM receptor suppresses the replication of PPRV. **a** PPRV infection in the B95a cells for 24 h in which the SLAM neutralized by anti-SLAM antibodies shows no deformities in the cells. **b** PPRV infected for 48 h and non-neutralized SLAM B95a cells shows rounding. **c** PPRV infection in the B95a cells for 24 h in which the SLAM was neutralized by anti-SLAM antibodies shows rounding of the cells. **d** PPRV infected for 48 h and an unneutralized SLAM B95a cell shows giant cell formation. Note the *arrows* in all the figures (**a-d**). These figures were adapted from Pawar et al. (2008), with permission

F, and H proteins are produced with a percentage of 81, 67, 49, and 39 %, respectively if N protein is taken as 100 % (Horikami and Moyer 1995). Due to the fact that inaccurate transcription occurs in the negative-strand RNA genome, where both mono- and poly-cistronic mRNA are produced, no estimation has been made for the transcription efficacy of each gene in PPRV. As is typical for negative-sense RNA viruses, the RNA produced needs to undergo 5' capping and 3' polyadenylation to be efficiently translated by the host ribosomes. These mechanisms have not been well described for the morbilliviruses.

During the course of infection, by an as yet unclear mechanism, transcription switches to replication and produces a full-length antigenome RNA, which is encapsidated by the N protein (Gubbay et al. 2001). Although this transfer of transcriptase to replicase ability of RdRp is complicated, Kolakofsky et al. (2004) have proposed that there are two distinct forms of RdRp in Sendai virus, a member of the same family (Kolakofsky et al. 2004). One form of RdRp is required for transcription while the other acts as a replicase. The contribution of other viral proteins (M, N, and P) in the activity of RdRp cannot be ignored. In MV, the M proteins regulate the function of RdRp but this regulation does not depend on the role of M protein in viral assembly and budding (Suryanarayana et al. 1994;



**Fig. 2.2** A generalized morbillivirus replication cycle. Interaction of the viral HN and F proteins with the host plasma membrane leads to viral entry by binding of the HN protein to receptors (SLAM and other unidentified receptors) for PPRV. The rest of the proteins are involved in replication of virus. Briefly, the P protein regulates transcription and replication and assembly of the N protein to nucleocapsids, the M proteins mediate viral assembly, and the HN protein facilitates the budding process when it acts as a neuraminidase in PPRV. Viral genome copies are formed from the minigenome through replicative intermediates. The role of the C and V proteins in PPRV is still not clear. It is believed that these proteins have abilities to abrogate the cellular interferon (IFN- $\alpha/\beta$ ) responses and hence contribute in the virulence of PPRV

Barrett et al. 2006). In other morbilliviruses, the role of the M protein apart from assembly and budding has not been investigated. Viral budding occurs through neuraminidase activity, which cleaves sialic acid residues from the carbohydrate moieties of glycoproteins (Scheid and Choppin 1974). Among morbilliviruses, PPRV is unique in that the H protein performs both hemagglutination and neuraminidase actions (Seth and Shaila 2001), hence, better reflected as an HN protein instead of an H protein.

The relative levels of the P, V, and C proteins are most likely also regulated in the same way. The editing process can clearly regulate the relative levels of P and V proteins. It is tempting to speculate, based on the functions of these proteins in other morbilliviruses, that during various stages of infection these proteins are

expressed at various levels and perform crucial roles in facilitating the virus replication by downregulating the host immunity. However, such functions of these PPRV proteins yet need to be confirmed.

## 2.3 Viral Transmissions and Propagation

### 2.3.1 *Non-Host Factors*

PPR outbreaks can occur by the close contact of infected and non-infected animals, which are likely to happen in common grazing places. Animals affected by PPR shed the virus in exhaled air, in secretions and excretions (from the mouth, eye and nose, and in feces, semen, and urine) approximately 10 days after the onset of fever. Sneezing and coughing by the infected animal can spread infection, while the transmission between animals in the vicinity can occur through inhalation (over a distance of 10 m) or, unlikely, through inanimate objects (fomite) due to its rapid inactivation in external dry conditions. Spread through ingestion and conjunctival penetration, and by licking of bedding, feed, and water troughs, is also not uncommon. Infection may spread to offspring by feeding them the milk of an infected dam. The exact viral survival in milk has not been demonstrated for PPR, but like rinderpest it is believed that the virus is present in the milk from 1–2 days before the signs appear until 45 days after complete recovery. Like rinderpest, recovered animals show strong immunity and there is no chronic and convalescent carrier state in PPR, but infection is likely to be spread in the subclinical infection during the incubation period. Recently, in an attempt to find out whether the incubatory carriers, as in RPV, could shed PPRV. Couacy-Hymann et al. (2007) confirmed in an experimental infection that infected animals could transmit PPRV before the onset of clinical signs (Couacy-Hymann et al. 2007). The year after, Ezeibe et al. (2008) studied the shedding of virus during the post-recovery state of the animal, and realized that goats infected with PPRV can shed HA virus antigens in feces for 11 weeks after complete recovery (Ezeibe et al. 2008). There is little known about the fragility of PPRV in the external environment. Comparison with rinderpest is likely to be reliable because there are many features in common. Although transmission is not impossible through fomites it is not common either, due to the short life of the virus in dry environment (above 70 °C) and acidic (>5.6) and basic (<9.6) pH. It also cannot resist for a longer time outside the host, due to its short half-life, which is estimated to be 2.2 min at 56 °C and 3.3 h at 37 °C (Rossiter and Taylor 1994).

### 2.3.2 *Host Factors*

Although cattle, pig, buffalo, and wild ruminants are susceptible to infection, only wild ruminants such as white-tailed deer are fully susceptible and may have a role in the epizootiology of PPR. Little information is available about susceptibility, occurrence, and severity of the disease in wild ungulate species. But a recent report has suggested the role of wildlife in the PPR spread. In this study, Kinne et al. (2010) isolated the virus from different wild small ruminants kept under semi-free-range conditions in the United Arab Emirates (UAE) (Kinne et al. 2010). Sequence analysis of the N gene indicated that the virus belongs to lineage IV, and was different from the viruses already isolated from the Arabian Peninsula (Kinne et al. 2010). Further analysis indicated that these isolates are more closely related to Chinese ones rather than the expected Saudi Arabian isolates. The origin of this new PPRV strain in the region has not been investigated, but it highlights the role of wild ruminants as a possible threat to domestic small ruminants.

Knowledge of the mechanism of PPR virus propagation and dissemination in the host cells is not complete yet. Few studies have demonstrated the sequence of events during virus propagation and its likely ways of spread in the host cell (Scott 1981; Gulyaz and Ozkul 2005). Like other morbilliviruses, PPRV is both lymphotropic and epitheliotropic, and thus the pathological lesions are likely to be severe in organs rich in lymphoid and epithelial tissues (Scott 1981). The PPR virus after invading the host through the respiratory system mainly localizes in the regional lymph nodes (pharyngeal and mandibular) and tonsils, resulting in lymphopenia. The febrile stage may occur on the fifth day and may persist until the sixteenth day post-infection. The resultant viraemia facilitates the dissemination of the virus to all visceral lymph nodes, bone marrow, spleen, and the mucous membranes of both the respiratory and digestive tracts. The virus can be isolated from nasal discharges from the day ninth of virus infection. PPRV then starts multiplying in the gastrointestinal tract, which leads to stomatitis and diarrhea. Scraping from the mucosa of the large intestine and extraction of the mesenteric lymph node can also be used to identify the virus at this moment. An unsuccessful attempt to isolate the virus from the blood of affected animals can be explained by the presence of PPRV-specific neutralizing antibodies that might form a complex with the virus and hence inhibit its recovery. We have recently amplified PPRV nucleic acids directly from filter papers impregnated with the blood of infected animals (Munir et al. 2012), indicating the stability of viral RNA and its presence in the blood. Virus spread to oral lesions has been reported in several studies (Brindha et al. 2001; Gulyaz and Ozkul 2005). Al-Naeem and Abu-Elzein (2008) demonstrated the presence of viral antigen in papules around the oral cavity, which is an indication of the predilection site for viral replication and tropism like the measles virus, a skin lesion-causing virus in humans (Al-Naeem and Abu-Elzein 2008). Although this prediction is helpful to understand the pathogenesis of the disease, further studies are required to confirm that this is not due to other concurrent infections. Bundza et al. (1988) have, for the first time, reported the

release of virus particles from the microvilli of intestinal epithelial cells and its shedding in feces (Bundza et al. 1988).

Recently, an unusual staining of PPRV antigen was demonstrated in cortical vessels, proximal tubules, and the epithelium of the renal pelvis. This probably explains the glomerulus filtration of the virus, after pooling in the kidney from the blood stream, and hence secretion in the urine (Kul et al. 2007). A similar localization of other morbilliviruses such as canine- and seal-distemper virus in the urinary system is well established (Kennedy et al. 1989). All morbilliviruses are neurovirulent, and severity depends upon host immunity, specificity of the receptors (such as CD46), and the extent of nervous system infection (Cosby et al. 2002; Kennedy et al. 1989; Yarim and Kabakci 2003). Although this characteristic is not well established in PPRV and RPV, a study conducted by Galbraith et al. (2002) indicates that RPV (Saudi/81) and PPRV (Nigeria 75/1) are neurovirulent when experimentally inoculated into mice (Galbraith et al. 2002). Moreover, a recent study also detected the viral antigen in ependymal cells and meningeal macrophages in natural PPRV infection in 4-month-old sheep (Kul et al. 2007). This interesting feature of PPRV requires further confirmation, because only 1 out of 21 animals showed this sign, but this at least indicates the ability of PPRV to reach cerebrospinal fluid.

## 2.4 Host Determinants of Pathogenesis

To pinpoint the factors required to predispose the animal to infection, it is important to study the epidemiology of the disease and hence its control. Several studies explored factors such as age, sex, breed, and seasons (Amjad et al. 1996; Brindha et al. 2001; Dhar et al. 2002; Munir et al. 2009). Extensive species based antibody surveys have indicated that the level of antibodies against the PPRV N protein was higher in sheep than in goats. Furthermore, it was also observed that goats are more susceptible to infection than sheep in terms of clinical signs. This explains why the virus might have more affinity in goats than sheep. Wosu (1994) has observed that the rate of recovery is lower in goats than in sheep (Wosu 1994). Recently, we have presented a corresponding trend of antibodies in the sheep and goats of governmental livestock farms in Pakistan (Munir et al. 2009). This determinant of pathogenesis needs to be investigated at the molecular level.

The difference in pathogenicity between sheep and goats may not be due to viral affinity, but may be due to a high recovery rate in sheep. In tropical areas, the fertility rate is higher in goats than sheep, which accounts for larger flock replacement by goat offspring. The newborn kids are susceptible to infection after 4 months of age, due to decrease in maternal protective antibodies (Srinivas and Gopal 1996; Ahmed et al. 2005). Waret-Szkuta et al. (2008) recently conducted a serological survey in Ethiopia and declared that age is the main risk factor for the seropositivity in small ruminants (Waret-Szkuta et al. 2008). Bodjo et al. (2006) have suggested the vaccination of the kids and lambs at 75–90 days after birth



(Bodjo et al. 2006). The higher susceptibility in goats may contribute to the severity of PPRV disease in goat populations. It is also true that PPRV infection can spread between goats without affecting sheep in the close vicinity (Animal-Health 2009), but mixed raising of both sheep and goats is considered to be a main risk factor for seropositivity in sheep flocks (Al-Majali et al. 2008). The case fatality rate is also found to be higher in young goats than in adults (Shankar et al. 1998; Atta-ur-Rahman et al. 2004). The sex-biased distribution of antibodies is hard to interpret because of early selling of males and longer maintenance of females.

In subtropical areas, the occurrence of the disease is reported to be more common during winter and rainy seasons (Amjad et al. 1996; Brindha et al. 2001; Dhar et al. 2002). Confinement and restricted movement of the animals, due to rainy seasons in tropical countries, may affect the nutritional status of the animals and hence predispose them to PPRV infection. Some studies have reported major outbreaks in cold and dry weather (Obi et al. 1983; Durojaiye et al. 1983; Opasina 1980), while others reported them soon after the rainy season (Bourdin 1980). This variation is probably explained by the region-dependent differences in animal husbandry conditions and socio-economic status of the farm owner.

## 2.5 Conclusions

Understanding viral replication and the factors influencing it can provide bases for devising the control strategies. Considering that PPRV is a suitable candidate for eradication after RPV, there is a great need to investigate the molecular determinants of PPRV pathogenicity, and to understand the complex interaction between virus and host. Our current knowledge of the virus life cycle shows that both host and environmental factors contribute to the virus transmission and propagation. However, the life of the virus in unusual susceptible hosts such as wildlife and camels remains elusive, but investigation of this could help the efficient planning of animal husbandry and provide a basis for understanding the role of wildlife and camels in the epizootiology of PPRV.

## References

- Adombi CM, Lelenta M, Lamien CE, Shamaki D, Koffi YM, Traore A, Silber R, Couacy-Hymann E, Bodjo SC, Djaman JA, Luckins AG, Diallo A (2011) Monkey CV1 cell line expressing the sheep-goat SLAM protein: a highly sensitive cell line for the isolation of peste des petits ruminants virus from pathological specimens. *J Virol Methods* 173(2):306–313
- Ahmed K, Jamal SM, Ali Q, Hussain M (2005) An outbreak of peste des petits ruminants in goat flock in Okara, Pakistan. *Pak Vet J* 25:146–148



- Al-Majali AM, Hussain NO, Amarin NM, Majok AA (2008) Seroprevalence of, and risk factors for, peste des petits ruminants in sheep and goats in Northern Jordan. *Prev Vet Med* 85(1–2):1–8
- Al-Naeem A, Abu-Elzein EM (2008) In situ detection of PPR virus antigen in skin papules around the mouth of sheep experimentally infected with PPR virus. *Trop Anim Health Prod* 40(4):239–241
- Amjad H, Qamar ul I, Forsyth M, Barrett T, Rossiter PB (1996) Peste des petits ruminants in goats in Pakistan. *Vet Rec* 139(5):118–119
- Animal-Health A (2009) Disease strategy: peste des petits ruminants (Version 3.0). Australian veterinary emergency plan (AUSVETPLAN), 3 edn. Primary Industries Ministerial Council, Canberra, ACT
- Atta-ur-Rahman, Ashfaq M, Rahman SU, Akhtar M, Ullah S (2004) Peste des petits ruminants antigen in mesenteric lymph nodes of goats slaughtered at D.I. Khan. *Pak Vet J* 24(3):159–160
- Barrett T, Ashley C B, Diallo A (eds) (2006) Molecular biology of the morbilliviruses. In: Rinderpest and peste des petits ruminants virus plagues of large and small ruminants, 2nd edn. Elsevier, Academic Press, London
- Bodjo SC, Couacy-Hymann E, Koffi MY, Danho T (2006) Assessment of the duration of maternal antibodies specific to the homologous peste des petits ruminant vaccine “Nigeria 75/1” in Djallonké lambs. *Biokemistri* 18(2):99–103
- Bourdin P (1980) History, epidemiology and economic significance of PPR in West Africa and Nigeria in particular. In: Hill DH (ed) Peste des petite ruminants (PPR) in sheep and goats. In: Proceedings of the international workshop held at IITA Ibadan, Nigeria, 24–26, ILCA (International Livestock Centre for Africa), Addis Ababa, Ethiopia, pp 10–11
- Brindha K, Raj GD, Ganesan PI, Thiagarajan V, Nainar AM, Nachimuthu K (2001) Comparison of virus isolation and polymerase chain reaction for diagnosis of peste des petits ruminants. *Acta Virol* 45(3):169–172
- Bundza A, Afshar A, Dukes TW, Myers DJ, Dulac GC, Becker SA (1988) Experimental peste des petits ruminants (goat plague) in goats and sheep. *Can J Vet Res (Revue canadienne de recherche veterinaire)* 52(1):46–52
- Cosby SL, Duprex WP, Hamill LA, Ludlow M, McQuaid S (2002) Approaches in the understanding of morbillivirus neurovirulence. *J Neurovirol* 8(Suppl 2):85–90
- Couacy-Hymann E, Bodjo SC, Danho T, Koffi MY, Libeau G, Diallo A (2007) Early detection of viral excretion from experimentally infected goats with peste-des-petits ruminants virus. *Prev Vet Med* 78(1):85–88
- Dhar P, Sreenivasa BP, Barrett T, Corteyn M, Singh RP, Bandyopadhyay SK (2002) Recent epidemiology of peste des petits ruminants virus (PPRV). *Vet Microbiol* 88(2):153–159
- Ezeibe MC, Okoroafor ON, Ngene AA, Eze JI, Eze IC, Ugonabo JA (2008) Persistent detection of peste de petits ruminants antigen in the faeces of recovered goats. *Trop Anim Health Prod* 40(7):517–519
- Galbraith SE, McQuaid S, Hamill L, Pullen L, Barrett T, Cosby SL (2002) Rinderpest and peste des petits ruminants viruses exhibit neurovirulence in mice. *J Neurovirol* 8(1):45–52
- Gubbay O, Curran J, Kolakofsky D (2001) Sendai virus genome synthesis and assembly are coupled: a possible mechanism to promote viral RNA polymerase processivity. *J Gen Virol* 82(Pt 12):2895–2903
- Gulyaz V, Ozkul A (2005) Pathogenicity of a local peste des petits ruminants virus isolate in sheep in Turkey. *Trop Anim Health Prod* 37(7):541–547
- Horikami SM, Moyer SA (1995) Structure, transcription, and replication of measles virus. *Curr Top Microbiol Immunol* 191:35–50
- Kennedy S, Smyth JA, Cush PF, Duignan P, Platten M, McCullough SJ, Allan GM (1989) Histopathologic and immunocytochemical studies of distemper in seals. *Vet Pathol* 26(2):97–103
- Kinne J, Kreutzer R, Kreutzer M, Wernery U, Wohlsein P (2010) Peste des petits ruminants in Arabian wildlife. *Epidemiol Infect* 138(8):1211–1214

- Kolakofsky D, Le Mercier P, Iseni F, Garcin D (2004) Viral DNA polymerase scanning and the gymnastics of Sendai virus RNA synthesis. *Virology* 318(2):463–473
- Kul O, Kabakci N, Atmaca HT, Ozkul A (2007) Natural peste des petits ruminants virus infection: novel pathologic findings resembling other morbillivirus infections. *Vet Pathol* 44(4):479–486
- Munir M, Siddique M, Ali Q (2009) Comparative efficacy of standard AGID and precipitinogen inhibition test with monoclonal antibodies based competitive ELISA for the serology of peste des petits ruminants in sheep and goats. *Trop Anim Health Prod* 41(3):413–420
- Munir M, Zohari S, Suluku R, Leblanc N, Kanu S, Sankoh FA, Berg M, Barrie ML, Stahl K (2012) Genetic characterization of peste des petits ruminants virus, sierra leone. *Emerg Infect Dis* 18(1):193–195
- Obi TU, Ojo MO, Durojaiye OA, Kasali OB, Akpavie S, Opasina DB (1983) Peste des petits ruminants (PPR) in goats in Nigeria: clinical, microbiological and pathological features. *Zentralblatt für Veterinärmedizin Reihe B J Vet Med* 30(10):751–761
- Opasina BA (1980) Epidemiology of PPR in the humid forest and the derived savanna zones. In: Hill DH (ed) *Peste des petits ruminants (PPR) in sheep and goats*. In: *Proceedings of the international workshop held at IITA Ibadan, Nigeria*
- Pawar RM, Raj GD, Kumar TM, Raja A, Balachandran C (2008) Effect of siRNA mediated suppression of signaling lymphocyte activation molecule on replication of peste des petits ruminants virus in vitro. *Virus Res* 136(1–2):118–123
- Renukaradhya GJ, Sinnathamby G, Seth S, Rajasekhar M, Shaila MS (2002) Mapping of B-cell epitopic sites and delineation of functional domains on the hemagglutinin-neuraminidase protein of peste des petits ruminants virus. *Virus Res* 90(1–2):171–185
- Rossiter PB, Taylor WP (1994) *Peste des petits ruminants*. In: Coetzee JAW (ed) *Infectious diseases of livestock*, vol II. Oxford University Press, Cape Town
- Scheid A, Choppin PW (1974) Identification of biological activities of paramyxovirus glycoproteins. Activation of cell fusion, hemolysis, and infectivity of proteolytic cleavage of an inactive precursor protein of Sendai virus. *Virology* 57(2):475–490
- Scott GR (1981) Rinderpest and peste des petits ruminants. In: Gibbs EPJ (ed) *Virus diseases of food animals*, vol II. Academic Press, London
- Seth S, Shaila MS (2001) The hemagglutinin-neuraminidase protein of peste des petits ruminants virus is biologically active when transiently expressed in mammalian cells. *Virus Res* 75(2):169–177
- Shankar H, Gupta VK, Singh N (1998) Occurrence of peste des petits ruminants like diseases in small ruminants in Uttar Pradesh. *Indian J Anim Sci* 68(1):38–40
- Srinivas RP, Gopal T (1996) Peste des petits ruminants (PPR): a new menace to sheep and goats. *Livest Adv* 21(1):22–26
- Suryanarayana K, Baczko K, ter Meulen V, Wagner RR (1994) Transcription inhibition and other properties of matrix proteins expressed by M genes cloned from measles viruses and diseased human brain tissue. *J Virol* 68(3):1532–1543
- Waret-Szkuta A, Roger F, Chavernac D, Yigezu L, Libeau G, Pfeiffer DU, Guitian J (2008) Peste des petits ruminants (PPR) in Ethiopia: analysis of a national serological survey. *BMC Vet Res* 4:34
- Wosu LO (1994) Current status of peste des petits ruminants (PPR) disease in small ruminants. A review article. *Stud Res Vet Med* 2:83–90
- Yarim M, Kabakci N (2003) The comparison of histo-pathological and immunohistochemical findings in natural canine distemper virus infection. *Folia Veterinaria* 47(2):86–90

Molecular Biology and Pathogenesis of Peste des Petits  
Ruminants Virus

Munir, M.; Zohari, S.; Berg, M.

2013, XI, 151 p. 28 illus., 19 illus. in color., Softcover

ISBN: 978-3-642-31450-6