Chapter 14
Sapelovirus

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Abstract *Sapelovirus* (SPV), an emerging virus in the family *Picornaviridae*, is detected in several animal and bird species irrespective of their age. Amid all SPVs, porcine sapeloviruses (PSVs) are more ubiquitously present all over the world in porcine population. These viruses are highly stable in different environmental conditions and spread easily within the susceptible animals mainly through faeco-oral route. Usually, PSVs cause asymptomatic infections but are also clinically associated with encephalomyelitis, respiratory distress, fertility disorders and skin lesions. PSV-associated outbreaks have been reported where death occurs due to polioencephalitis and respiratory paralysis. Till date, PSVs have been detected from several European and Asian countries with moderate-to-high prevalence and clinical course. Viral capsid proteins are immunogenic and mutations in these pro-

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teins are responsible for the diverse nature of the viruses. Further, genomic analysis shows the varied evolutionary patterns and the presence of recombination within PSV strains. These viruses also exist as concurrent infections with several enteric bacterial, viral and parasitic pathogens. Classical to modern biotechnological assays are in use to detect PSV involving virus isolation in cell culture, immunohistochemistry, conventional nucleic acid amplification techniques, quantitative real-time amplification assays and isothermal amplification molecular techniques. Till date, there is no vaccine available against PSVs.

Keywords Sapelovirus · Picornaviridae · Porcine · Encephalomyelitis · Mutation · Diversity · Recombination · Diagnosis · Epidemiology

14.1 Preamble

Sapeloviruses, formerly known as porcine enterovirus (PEV)-8, are ubiquitous and most commonly responsible for causing asymptomatic infection of the gastrointestinal tract of animals. The genetic analysis revealed unique markers based on which PEV-8 viruses were reclassified under a new genus named *Sapelovirus* (Krumbholz et al. 2002). As on date, these viruses have been found to infect porcine, avian and simian species. Porcine sapelovirus (PSV)-associated clinical symptoms include pneumonia, reproductive defects, skin lesions, enteritis and encephalomyelitis. As the information available on avian sapelovirus (ASV) and simian sapelovirus (SSV) is limited, this chapter comprehends the information on PSV only.

14.2 Virus Characteristics

PSVs belong to the genus *Sapelovirus* under the family *Picornaviridae*. The size of virion particles is small (~35 nm) and they are non-enveloped possessing icosahedral symmetry with a distinct surface morphology visible under the electron microscope (Bai et al. 2018). Mature infectious virions are stable in the environment and could resist acidic pH. Furthermore, virions are not destabilised at elevated temperature (60 °C for 10 min), lipid solvents and some disinfectants. Sodium chlorite or 70% ethanol is effective to inactivate PSV (Horak et al. 2016).The *Sapelovirus* exhibits buoyant density in CsCl of 1.32–1.34 g/cm^3.

14.3 Virus Taxonomy

The current *Sapelovirus* taxonomy is the result of efforts from the picorna study group and the International Committee on Taxonomy of Viruses (ICTV) Executive Committee (www.picornastudygroup.com) that are contributing their expertise to provide a classification scheme that is supported by verifiable data and expert consensus. The genus *Sapelovirus* contains three species, namely *Avian sapelovirus* (ASV), *Sapelovirus A* (SV-A, porcine) and *Sapelovirus B* (SV-B, simian) (ICTV 10th report, https://talk.ictvonline.org/ictv-reports/ictv_online_report/positive-sense-rna-viruses/picornavirales/w/picornaviridae/701/genus-sapelovirus). ASV and SV-A consist of only a single antigenically heterogenous serotype, respectively, known as ASV-1 and PSV-1. However, SSVs are highly divergent and are classified into three genetically defined *Sapelovirus* types as SSV-1 to SSV-3. PSV (strain csh) was first isolated from China in 2011 (Lan et al. 2011). As on date, 50 complete genomic sequences of PSV strains have been published from China, South Korea, Japan, Germany, France, India and the United Kingdom (Lan et al. 2011; Chen et al. 2012; Schock et al. 2014; Son et al. 2014; Ray et al. 2018; Piorkowski et al. 2018; Bai et al. 2018). Different virus strains exhibit affinity towards different target organs, as the PSV-csh strain (China, Shanghai) and PSV-G5 (the United Kingdom) affect mostly nervous system (Lan et al. 2011; Schock et al. 2014), whereas strains like KS0515, KS04105 and KS0552179 and Chinese PSV strain YC2011 are associated with diarrhoea. Notably, PSV-csh is a diarrhoeagenic strain that leads to respiratory distress before the onset of polioencephalomyelitis in infected animals (Lan et al. 2011).

14.4 Viral Genome

PSVs are positive-sense RNA viruses containing a genome-linked viral protein (VPg) at the 5′-terminus and a 3′-polyA tail. The complete genome size of the virus is 7.5–8.3 kb, coding a single polyprotein which is further cleaved post-translationally by virus-encoded proteinases into 12 individual proteins, viz. a leader protein (L), four structural proteins (VP1-4) and seven non-structural proteins (2A-2C, 3A-3D). The deduced polyproteins of the *Sapelovirus* range from 2322 to 2521 amino acids (aa). The layout of a complete genome of PSV (strain V13) with the nucleotide length of each protein is depicted in Fig. 14.1. PSVs have unique L and 2A protein (Krumbholz et al. 2002). Though the exact function of these two proteins is not known yet, both are presumed to be a protease. All three *Sapeloviruses* contain Internal Ribosomal Entry Site (IRES) type IV. ASVs have the longer L protein (451 aa) compared to PSV (84 aa) and SSV (88 aa). Regarding 2A protein, ASVs have very small protein (12 aa) whereas PSV and SSV have 226 aa and 302 aa, respectively. A species demarcation criteria is followed for the genus *Sapelovirus* that is founded on above 70% aa identity in the polyprotein, greater than 64% aa identity in P1 (VP4, VP2, VP3, VP1), greater than 70% aa identity in 2C + 3CD, a similar genome base composition which varies by no more than 1% and a common genome organisation (ICTV 10th report, 2018).

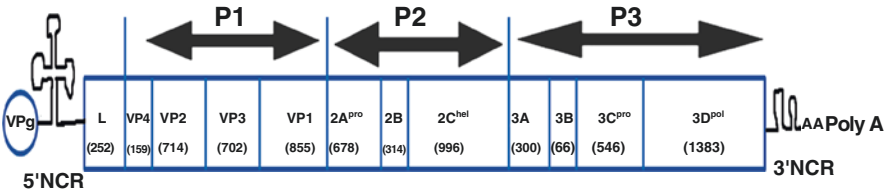

Fig. 14.1 A schematic layout of the porcine Sapelovirus genome (nucleotide lengths in base pair of different genes are given as per the first isolated PSV strain V13, NC_003987). *VPg* genome-linked viral protein, *NCR* noncoding RNA, *L* leader protein, *VP* viral protein, *P1* structural protein, *P2–P3* non-structural protein, *pro* protease, *hel* helicase and *pol* polymerase are depicted in schematic diagram

14.5 Epidemiology

14.5.1 Species Affected

Sapeloviruses have been isolated from pigs, wild boars, monkeys and ducks whereas sapelovirus-like-specific genome has been detected in a variety of species like bats (several species), cats, cattle, dogs, pigeons, quails, and sea lions (Li et al. 2011). Notably, these viruses to date are not known to cause any zoonotic transmission to human.

14.5.2 Geographic Distribution and Strain Variability

PSVs are being detected from different parts of the world in both healthy and diarrhoeic piglets and regarded as emerging enteric pathogens in porcine species. The countries that confirmed the presence of PSVs are shown in Fig. 14.2 (Forman et al. 1982; La Rosa et al. 2006; Buitrago et al. 2010; Lan et al. 2011; Shan et al. 2011; Cano-Gomez et al. 2013; Schock et al. 2014; Donin et al. 2014, 2015). The most immunodominant protein VP1 in the capsid of PSVs is used for estimating the divergence among the strains. Though only a single type of PSV-1 is present, fewer similarities are observed between PSV strains from different geographical regions. Phylogenetic analysis based on VP1 gene sequences shows that PSVs are distributed in different clusters depending upon the country of origin. Both full-length (855 bp) and partial-length (544 bp) VP1 genes have been used for the analysis of PSV diversity. The analysis based on VP1 sequences by SplitsTree4 software segregates three species within the genus *Sapelovirus* (Fig. 14.3) in three different clusters. Variations within PSV isolates are quite evident that indicates that several PSVs are circulating all over the world, and they are different from each other based on VP1 sequence. Studies from China reveal that recombinations occur between PSV strains, which further contribute to the antigenic diversity among PSVs. Recently, identification and genomic characterisation of *Sapelovirus* have been documented from France and India (Ray et al. 2018; Piorkowski et al. 2018;

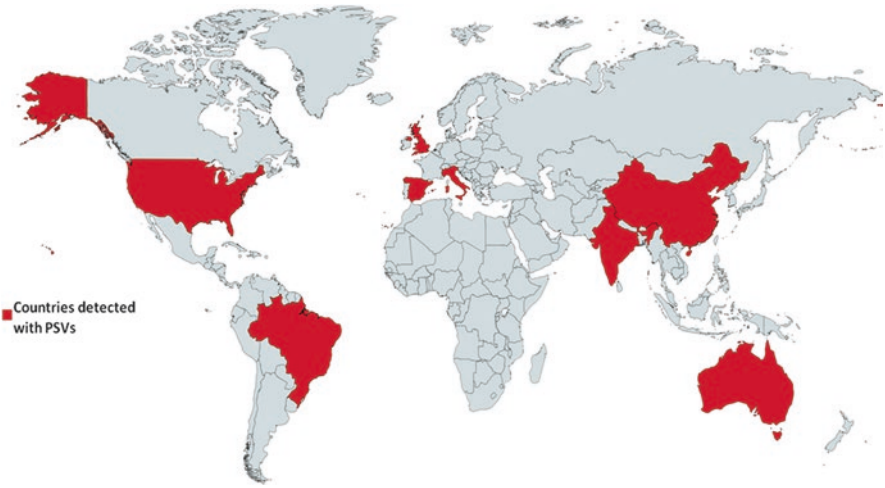

Fig. 14.2 Distribution of PSV throughout the world. The colour-shaded portions are where PSV incidences have been reported

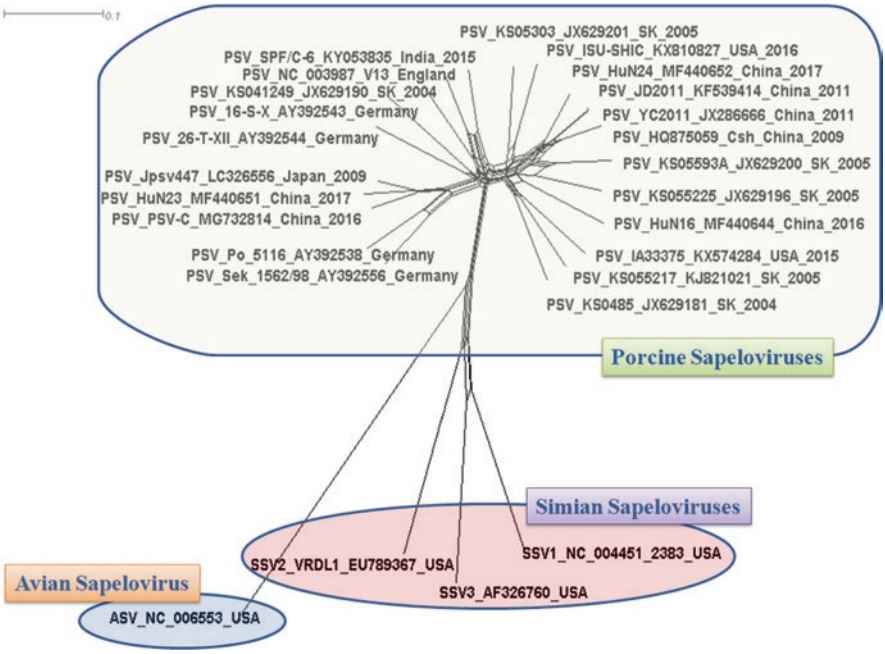

Fig. 14.3 Unrooted maximum-likelihood phylogenetic tree based on partial VP1 (544 bp) nucleotide sequences of the representative members of *Porcine Sapelovirus* PSV, *Simian Sapelovirus* SSV and *Avian Sapelovirus* ASV from China, Japan, England, India, the United States, Germany and SK (South Korea). Tree was developed using SplitsTree4 (Huson and Bryant 2006)

Kumari et al. 2019). A Chinese study reported the recombination hotspot near 3′ of the VP1 gene and showed six interclades within PSV recombinant strains (Yang et al. 2017).

14.6 Transmission and Pathogenesis

The most common route of transmission of the virus is faecal-oral; however, due to its high stability the infectious virions spread through fomites also (Huang et al. 1980; Lan et al. 2011). Several intestinal epithelial cell lines are commonly used for the pathogenicity and virulence studies of PSVs (Lan et al. 2013).

After ingestion through the oral route, PSVs primarily replicate in the intestinal tract. Receptor used by the PSV is unknown, but one recent study shows that a 2,3-linked terminal sialic acid (SA) on the cell surface GD1a ganglioside could be used for PSV binding and infection as a receptor (Kim et al. 2016). Experimental data shows that PSV utilises caveolae-dependent endocytosis pathway for PK-15 cell internalisation and depends upon lower pH, dynamin, Rab7 and Rab11 (Zhao et al. 2019). PSV strains are known to cause viremia through which they access the central nervous system (CNS). The incubation periods vary from 5 to 14 days.

14.7 Clinical Signs

PSVs can cause both symptomatic and asymptomatic infections in the natural field cases and experimentally infected piglets. Commonly, co-infections of PSV with other enteric pathogens like porcine kobuvirus (PKV), porcine enterovirus (PEV), porcine teschovirus (PTV), group A-C rotaviruses (RVA-C), porcine sapovirus (PSaV), porcine norovirus (PNoV), transmissible gastroenteritis coronavirus (TGEV), porcine epidemic diarrhoea coronavirus (PEDV), *Escherichia coli* and Salmonella spp., *Lawsonia intracellularis*, *Brachyspira hyodysenteriae*, *Brachyspira pilosicoli*, *Brachyspira intermedia* and *Clostridium perfringens*. Symptomatic manifestations are mainly neurological, which include spinal cord damage, ataxia, mental dullness, paresis, paralysis and a decreased response to environmental stimuli. Polioencephalitis in grower pigs is characterised by ataxia and paraparesis. Experimental infection of polioencephalitis has been documented in 50–60-day-old grower pigs. A novel PSV isolate from the United States was found to be associated with typical polioencephalomyelitis (Arruda et al. 2016). Gastroenteritis and respiratory distress may also be seen with PSV-induced polioencephalomyelitis (Lan et al. 2011). Intravaginal inoculation at day 15 of gestation causes early embryonic death and foetal resorption, but when inoculated at day 30, it results in a significant increase in foetal death (Huang et al. 1980). PSV is also detected from intestinal contents of stillbirth animals from Indian pig population, which suggests the transplacental transmission of the virus (Kumari et al. 2019).

14.8 Morbidity and Mortality

No specific data is available on the mortality and morbidity associated with PSV infection. Experimental infection through intravaginal and intrauterine route in gilts with PSV on day 30 of gestation results in a 94.4% foetal mortality (Huang et al. 1980). An outbreak reported in the United States in 2016 stated 20% morbidity and 30% case fatality rate (Arruda et al. 2016).

14.8.1 Post-mortem and Histopathology

Lesions are mainly seen in the central nervous system (CNS), consistent with the other neurotropic viral infection, characterised as subacute, multifocal and non-suppurative polioencephalomyelitis, accompanied with punctate haemorrhage and hyperaemia in the dura mater (Lan et al. 2011; Schock et al. 2014). Neuronal vacuolisation and perivascular cuffing are also commonly observed (Lan et al. 2011). Congestion is the most common gross lesion seen in the small intestine along with pronounced loss of villi with haemorrhage in the lamina propria histologically (Lan et al. 2011). In the case of clinical pneumonia, consolidation and multifocal haemorrhage are commonly seen in the lung lobes, where erythrocytes infiltrate throughout the interstitium and alveoli with alveolar wall thinning (Lan et al. 2011). Pathological lesions from naturally infected Indian pig show severe clouding, thickening and congestion of meninges, pleural thickening, frothy exudates in the trachea, congested mesenteric lymph nodes, yellowish discolouration of liver, intestinal (ileum) corrugation and thickening of the intestinal mucosal fold (Kumari et al. 2019)

14.9 Diagnosis

14.9.1 Clinical History

A neurological disorder characterised by ataxia and limb paralysis, with or without other clinical symptoms (diarrhoea or pneumonia), is suggestive of PSV infection (Lan et al. 2011). The PSV-induced reproductive disorder can be suspected in litters with a few to several stillborn or mummified foetuses (Huang et al. 1980).

14.9.2 Samples

Samples from live animal include faeces and blood (serum). In the case of the faecal sample, direct collection from the anus is preferred to avoid sample contamination. Spinal cord and brain are the most common preferred samples from the dead animal for PSV-induced CNS infection diagnosis. Other than these, liver, spleen, trachea, lung and intestine samples are also useful in diagnosing PSV infections. Though PSV has not been successfully isolated from the tissues of stillborn or mummified foetuses, molecular detection has been reported from stillbirth cases (Huang et al. 1980; Kumari et al. 2019).

14.9.3 Detection of Virus, Nucleic Acid or Antigens

Routine laboratory diagnosis of PSVs mainly includes cell culture isolation and virus genome characterisation. Pig kidney cell-derived cell lines including EFN, PK-15, IBRS-2 and LLC-PK are susceptible to PSV with a cytopathic effect (CPE) in these cells (Sozzi et al. 2010; Lan et al. 2011). Additionally, monkey kidney (Vero) and baby hamster kidney fibroblasts (BHK-21) are also susceptible to PSVs where a CPE characterised by cell shrinkage, rounding and detachment is commonly seen after 3–5 passages. Recently, a human hepatocarcinoma cell line, PLC/PRF/5, is normally used to isolate the hepatitis E virus, from swine faecal specimens serendipitously isolated from Japanese PSV strain (Bai et al. 2018). Cultured PSVs are generally identified by using virus neutralisation (VN) (Sozzi et al. 2010) and immunofluorescence antibody (IFA) assays (Son et al. 2014). However, these methods are costly, less specific, labour intensive and time consuming. Several molecular methods like reverse transcription PCR (RT-PCR), nested reverse transcription PCR (nRT-PCR) and real-time quantitative PCR have been developed for PSV detection (Zell et al. 2000; Palmquist et al. 2002; Krumbholz et al. 2003). Table 14.1 depicts the list of assays with target genes and primer sequences used by researchers in detecting PSVs. These molecular methods are specific, sensitive and rapid and do not cross-react with other common pathogens of porcine diseases including pseudorabies virus, porcine reproductive and respiratory syndrome, porcine parvovirus, porcine coronavirus, porcine reovirus or picorna-like virus (Palmquist et al. 2002). Real-time PCR assays developed for the detection of PSVs from field samples could detect a range of 10^2–10^3 copies of DNA (Chen et al. 2014; Kumari et al. 2019). Both these real-time PCR assays utilised the TaqMan chemistry, which is quite specific. Another sensitive diagnostic available for PSV is reverse transcription loop-mediated isothermal amplification (RT-LAMP) that was developed by Wang et al. (2014) with a detection limit of ten copies of DNA. All these easy, rapid and sensitive diagnostics will help in better understanding of the epidemiology of PSV and find a better way for prevention.

Table 14.1 RT-PCR assays used for detection of PSVs

Assays	Primer sequence (5′–3′)	Targeted gene	References
RT-PCR	FP-CCCTGGGACGAAAGAGCCTG	5′UTR	Zell et al. (2000)
	RP-CCTTTAAGTAAGTAGTAAAGGG		
nRT-PCR	FP-CCAAGATTAGAAGTTGATTTG		
	RP-GGGTAGCCTGCTGATGTAGTC		
Duplex PCR for PTV and PSV	FP-GTGGCGACAGGGTACAGAAGAG	5′UTR	Palmquist et al. (2002)
	RP-GGCCAGCCGCGACCCTGTCAG		
RT-PCR (genotyping)	FP-AGGATGTGGTGCAAGCAAGCAT	VP1	Son et al. (2014)
	RP-AGGCAGCACCGTTCTGGTCAA		
RT-LAMP	FP-CCATACCCTACCCTCCCTTC	5′UTR	Wang et al. (2014)
	RP-GCCCATAGTTCACTGCCTAC		
Real-time RT-PCR	FP-GGCAGTAGCGTGGCGAGC	5′UTR	Chen et al. (2014)
	RP-CTACTCTCCTGTAACCAGT		
	Minor groove-binding Taqman probe—CGATAGCCATGTTAGTG		
Real-time RT-PCR	FP-GGAAACCTGGACTGGGYCT	5′UTR	Kumari et al. (2018)
	RP-ACACGGGCTCTCTGTTTCTT		
	Taqman probe—CCAGCCGCGACCCTATCAGG		

14.10 Immunity

There is little information available on the immune response to PSVs. The humoral response is primarily based on the IgA. A study done on porcine intestinal epithelial cells (IPEC-J2) reveals the role of PSV infection in changing the innate immune response pathway (Lan et al. 2013). The study shows both upregulation (e.g. TLR3) and downregulations (e.g. TLR4) of several innate immune system molecules (Lan et al. 2013). Maternal antibodies proved not to be protective in the case of transplacental infection (Huang et al. 1980). Seropositive gilts when inoculated intrauterinely and intravaginally resulted in embryonic and foetal infection (Huang et al. 1980). Though maternal antibody in the colostrums is believed to be protective infections are found in post-weaning animals (Schock et al. 2014). As of now, there is no vaccine available for PSVs.

14.11 Conclusion and Prospects

Although PSV is widely distributed all over the world among the porcine population, detailed information is lacking about their evolutionary pattern and mechanisms driving their divergence. However, precise data about the prevalence of PSV infections and

the genetic diversity of this virus in pigs and wild boars have been reported only in a limited number of countries. Moreover, the association of co-infections with other enteric pathogens is exactly not known which raises ambiguity in the pathogenic potential of PSVs. Several recent findings on recombination events between PSV strains emphasise to study genetic variability among PSVs.

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