

PORCINE SAPELOVIRUS



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SUMMARY

Etiology

- Porcine sapelovirus (PSV) is an RNA virus belonging to the genus *Sapelovirus* in the family *Picornaviridae*. PSV is closely related to members of the *Enterovirus* genus and has previously been known as porcine enterovirus 8 (PEV-8).
- There are three species within the *Sapelovirus* genus: simian, avian and porcine (sapelo: simian, aavian, and porcine entero-like viruses). The porcine and avian sapeloviruses consist of a single serotype, while the simian sapelovirus has three.

Cleaning and Disinfection

- Sapeloviruses survive well in the environment. The virus is resistant to elevated temperatures (60°C for 10 minutes) and acidic pH.
- Sodium chlorite or 70% ethanol are effective methods for inactivation of PSV.

Epidemiology

- Domestic and wild swine are the only known hosts for PSV.
- PSV is not known to be zoonotic.
- PSV has been found in the feces of healthy swine in Brazil, Australia, Spain, Italy, and the United States. The virus has been identified as a cause of disease in pigs in China, Australia, and the United Kingdom.
- There is little data on morbidity and mortality associated with PSV. Experimental inoculation of gilts with PSV on day 30 of gestation resulted in 94.4% fetal mortality in one study.

Transmission

- Fecal-oral is the primary mode of transmission. Fomites may also play a role.

Infection in Swine/Pathogenesis

- PSV infections are often subclinical though the virus can cause neurologic disorders, diarrhea, and pneumonia. The term SMEDI syndrome (stillbirth [S], mummified fetus [M], embryonic death [ED], infertility [I]) is used to describe the variety of reproductive disorders caused by PSV and other picornaviruses.

Diagnosis

- PSV can be cultured and identified by virus neutralization or immunofluorescence assays.
- Reverse transcriptase polymerase chain reaction (RT-PCR) is most commonly used to detect PSV in fecal samples and can distinguish between PSV, porcine teschovirus (PTV) and enterovirus-G (EV-G).

Immunity

- Maternal antibody is not protective against transplacental infection, although colostral antibody may be.
- There is no vaccine for PSV-1.

Prevention and Control

- Documented methods for controlling PSV are not available.

Gaps in Preparedness

- The role of PSV as a pathogen, particularly as a cause of polioencephalomyelitis, is unclear.

OVERVIEW

Porcine sapelovirus (PSV) is a non-enveloped, positive-sense single-stranded RNA virus belonging to the genus *Sapelovirus* in the family *Picornaviridae*. PSV is closely related to the genus *Enterovirus* and was previously classified as porcine enterovirus 8 (PEV-8). There are three species within the *Sapelovirus* genus: porcine, simian and avian. Pigs, monkeys and ducks are the only known hosts for each species. PSV is not known to infect humans and there have been no reports of pig-to-human transmission.

Similar to porcine teschovirus (PTV), PSV is resistant to environmental conditions, surviving well in heat and over a wide pH range. Disinfection can be accomplished using sodium chlorite or 70% ethanol. PSV has a worldwide distribution and has been isolated from the feces of healthy swine in Brazil, Italy, Spain, Australia and United States. Pathogenic strains of PSV causing clinical illness have been reported in China, South Korea, and the United Kingdom. There is no specific data available on the morbidity and mortality of PSV infections in swine.

Fecal-oral transmission of PSV is most common. However, fomites likely play a role in transmission as well. PSV infections are commonly asymptomatic; however, they can cause clinical syndromes including diarrhea, polioencephalomyelitis, pneumonia, and reproductive disorders. Little is known about the pathogenicity of neurotropic PSV strains. PSV infection begins in the mucus membranes of the small intestine where it replicates, resulting in villous atrophy. In one study, inoculation of 50–60 day old pigs with the pathogenic strain PSV-csh resulted in diarrhea two days post-infection. Ataxia and limb paralysis followed five days later. Reproductive disorders associated with PSV infection have been termed ‘SMEDI syndrome’ (stillbirth [S], mummified fetus [M], embryonic death [ED], infertility [I]).

Humoral immunity seems to be important in protection against PSV-induced disease. Experimental studies show high titer IgA is produced early in infection. Maternal colostral antibody likely plays a role in protection of pre-weaned swine as well. There is no treatment or vaccine for PSV.

LITERATURE REVIEW

1. Etiology

1.1 Key Characteristics

Porcine sapelovirus (PSV) is a non-enveloped, positive-sense single-stranded RNA virus belonging to the genus *Sapelovirus* in the family *Picornaviridae*. PSV is most closely related to members of the *Enterovirus* genus and was formerly known as porcine enterovirus 8 (PEV-8), classified as porcine enterovirus A (PEV-A).¹ Similar to porcine teschovirus (PTV), PSV is a ubiquitous virus that commonly results in asymptomatic infection of the gastrointestinal tract.²⁻⁵ Pathogenic infections can lead to a variety of clinical syndromes including diarrhea, respiratory disease, reproductive disorders and polioencephalomyelitis.

1.2 Strain Variability

There are three species within the *Sapelovirus* genus: simian, avian and porcine (sapelo: simian, aavian, and porcine entero-like viruses). PSV has a single serotype, porcine sapelovirus 1 (PSV-1). Avian sapelovirus (ASV) has a single serotype as well, avian sapelovirus 1 (ASV-1). Simian sapelovirus (SSV) has three serotypes, simian sapelovirus 1-3 (SSV 1-3).¹ To remain consistent with genus and species nomenclature, the International Committee on Taxonomy of Viruses has proposed changing the species name of porcine and simian sapeloviruses to sapelovirus A and sapelovirus B, respectively.⁶ The complete genomic sequences of several PSV strains have recently been published.⁷⁻¹⁰ Neurotropic strains include PSV-csh (China, Shang-hei)⁷ and PSV-G5 (United Kingdom).⁸ Diarrheic strains include Korean PSV strains KS0515, KS04105 and KS055217⁹ and Chinese PSV strain YC2011.¹⁰ PSV-csh also caused diarrhea and respiratory distress prior to the onset of polioencephalomyelitis in infected animals.⁷

2. Cleaning and Disinfection

2.1 Survival

PSV isolates are very stable in the environment. The virus is resistant to elevated temperatures (60°C for 10 minutes) and acidic pH.¹ There have been no reports of seasonal outbreaks of PSV.

2.2 Disinfection

Heat, lipid solvents, and some disinfectants are ineffective in destabilizing PSV. Sodium chlorite or 70% ethanol are effective methods for inactivation of PSV.¹¹

3. Epidemiology

3.1 Species Affected

Domestic and wild swine are the only known hosts for PSV.^{5,12} Monkeys and ducks are the only known hosts for simian and avian sapelovirus species, respectively.¹

3.2 Zoonotic Potential

PSV is not known to infect humans; there have been no reports of pig-to-human transmission of PSV.¹

3.3 Geographic Distribution

PSV can be found throughout the world and has been isolated from the feces of healthy swine in Brazil^{12,13}, Australia¹⁴, Spain^{3,5}, Italy² and the United States.¹⁵ PSV has been the causative agent of disease in swine in China⁷, Australia¹⁴ and the United Kingdom.⁸

3.4 Morbidity and Mortality

No specific data is available on the morbidity and mortality associated with PSV infection causing diarrhea, pneumonia and polioencephalomyelitis. Experimental intravaginal and intrauterine inoculation of gilts with PSV on day 30 of gestation resulted in a 94.4% fetal mortality.¹⁶

4. Transmission

Fecal-oral transmission of PSV is most common.^{7,16} However, PSV is hardy in the environment and fomites likely play a role as well.¹

The mucus membranes of the gastrointestinal tract are the target and primary replication site for PSV. A porcine intestinal epithelial cell line (IPEC-J2) was infected *in vitro* with PSV-csh to demonstrate the pathogenicity of PSV within the intestine. IPEC-J2 cells began to spontaneously shrink 24 hours post-infection (hpi), rupture at 60 hpi and slough at 72 hpi. Viral load was highest 48 hpi, prior to cell rupture.¹⁷

5. Infection in Swine/Pathogenesis

5.1 Clinical Signs

PSV infections are often subclinical¹⁸, though PSV can also cause neurologic disorders¹⁴, diarrhea¹⁹, reproductive failures^{16,20} and pneumonia.⁷

Little is known about the pathogenicity of neurotropic PSV strains.⁸ In one study, pigs (50–60 days old) orally inoculated with the PSV-csh isolate developed diarrhea two days post infection. Polioencephalomyelitis syndrome, characterized by ataxia followed by limb paralysis, was observed seven days post infection.⁷

Polioencephalomyelitis induced by PSV has also been reported in pigs 50–60 days old, after maternal colostral antibodies have waned. In one outbreak, front and hind limb ataxia progressed to generalized weakness and lateral recumbency. Affected pigs died two to three days after the onset of clinical signs.⁸ Gastroenteritis and respiratory distress may also be seen with PSV-induced polioencephalomyelitis.⁷

The term SMEDI syndrome (stillbirth [S], mummified fetus [M], embryonic death [ED], infertility [I]) was adapted to describe the variety of reproductive disorders caused by PSV and other picornaviruses.²¹ When seronegative and seropositive gilts were intravaginally inoculated with PSV at day 15 of gestation, early embryonic death and complete resorption occurred. Infection on day 30 of gestation resulted in a significant increase in fetal death.¹⁶

5.2 Postmortem Lesions

Lesions seen with PSV-induced polioencephalomyelitis are consistent with other neurotropic viral infections, such as porcine teschovirus (PTV). In the CNS, punctate hemorrhage and hyperemia are present in the dura mater.⁷ Polioencephalomyelitis is generally characterized as subacute, multifocal and non-suppurative.⁸ Neuronal vacuolization and perivascular cuffing are also commonly observed.⁷

In the small intestine, congestion is seen grossly, and pronounced loss of villi with hemorrhage in the lamina propria can be seen upon histologic examination.⁷

In cases of PSV-induced clinical pneumonia, consolidation and multifocal hemorrhage are seen on lung lobes. Upon histologic examination, erythrocytes are pervasive throughout the interstitium, and alveoli and prominent alveolar ectasia with alveolar wall thinning can be observed. Some alveoli may rupture to form large cysts.⁷

6. Diagnosis

6.1 Clinical History

Polioencephalomyelitis syndrome, characterized by ataxia and limb paralysis, with or without other clinical symptoms (diarrhea or pneumonia) is suggestive of PSV infection.⁷ Litters with few to several stillborn or mummified fetuses is suggestive of PSV-induced reproductive disorder.¹⁶

6.2 Tests to Detect Nucleic Acids, Virus, or Antigens

PSV can be cultured in cell lines of porcine origin. Porcine kidney cells PK-15 and IBRS-2 have been successfully used.^{7,18} Monkey kidney (Vero) and baby hamster kidney fibroblasts (BHK-21) are also appropriate.¹⁹ Cultured PSV can be identified using virus neutralization (VN)¹⁸ and immunofluorescence antibody (IFA) assays.⁹

Reverse transcriptase polymerase chain reaction (RT-PCR) is most commonly used to detect PSV in fecal samples and can distinguish between PSV, porcine teschovirus (PTV) and enterovirus-G (EV-G). It is specific, sensitive, and rapid and does not cross react with other common causes of porcine disease including pseudorabies virus, porcine reproductive and respiratory syndrome, porcine parvovirus, porcine coronavirus, porcine reovirus, or picorna-like virus.²² Nested reverse transcription,²³ real time PCR,²⁴ and reverse transcription loop-mediated isothermal amplification (RT-LAMP) techniques have also been described.²⁵

6.3 Tests to Detect Antibody

Information on assays to detect anti-PSV antibody is unavailable.

6.4 Samples

6.4.1 Preferred Samples

Preferred samples for CNS disease include the spinal cord and brain.²⁶ PSV has not been successfully isolated from the tissues of stillborn or mummified fetuses.¹⁶

6.4.2. Oral Fluids

Specific data regarding the suitability of oral fluids for detecting PSV is unavailable.

7. Immunity

7.1 Post-exposure

Little information is available on the immune response to PSV infection. In a pathogenicity study, infection of IPEC-J2 cells with PSV led to genetic changes largely in innate immunity pathways. The humoral response to PSV has been primarily characterized by IgA early in infection.¹⁷

Intrauterine and intravaginal inoculation of anti-PSV antibody positive gilts resulted in embryonic and fetal infection indicating maternal antibody is not protective against transplacental infection.¹⁶ However, maternal colostral antibody is believed to be protective. An outbreak of polioencephalomyelitis, caused by PSV-G5, on a breeding and grower unit affected only post-weaning animals, suggesting maternal colostral antibody plays a role in protection against infection of neonates.⁸

7.2 Vaccines

There is no vaccine for PSV-1.

7.3 Cross-protection

Specific data regarding cross-protection between PSV-1 strains is unavailable.

8. Prevention and Control

Documented methods for controlling PSV are not available.

9. World Organization for Animal Health (OIE) Terrestrial Animal Health Code

PSV is not included in the 2015 OIE Terrestrial Animal Health Code. There are no restrictions for importation of animals from countries or zones infected with PSV.

10. Gaps in Preparedness

The role of PSV as a pathogen, and more specifically as a cause of polioencephalomyelitis, is unclear. This virus is commonly isolated from the intestinal tract of healthy swine and more research is needed to clarify the pathogenesis of the neurotropic disease caused by PSV strains.⁸ Development of a vaccine may be justified for use in outbreaks of polioencephalomyelitis with high mortality or SMEDI syndrome.

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