PORCINE TESCHOVIRUS

Swine Health Information Center

The mission of the Swine Health Information Center is to protect and enhance the health of the United States swine herd through coordinated global disease monitoring, targeted research investments that minimize the impact of future disease threats, and analysis of swine health data.

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SUMMARY

IMPORTANCE

- Porcine teschovirus (PTV) is an enteric picornavirus of swine. Most infections are subclinical, but some strains cause severe central nervous system disease (polioencephalomyelitis) with high mortality. Why some PTVs cause polioencephalomyelitis and others circulate inapparently is not known.

PUBLIC HEALTH

- PTV is not considered to be zoonotic.

INFECTION IN SWINE

- Many PTV infections are subclinical. Severe polioencephalomyelitis (teschovirus encephalomyelitis, Teschen disease) causes high morbidity and mortality in pigs of all ages. Milder cases of polioencephalomyelitis (benign enzootic paresis, Talfan disease) occur in post-weaning pigs.
- Some PTVs are linked to SMEDI syndrome (stillbirth [S], mummified fetus [M], embryonic death [ED], infertility [I]). PTV has been associated with diarrhea, but the virus can also be found in the feces of healthy pigs. PTVs are also linked to respiratory disease, pericarditis, myocarditis, and congenital microphthalmic syndrome.

TREATMENT

- There is no treatment for pigs infected with PTV.

CLEANING AND DISINFECTION

- PTV persists in the environment, surviving at moderate temperatures and pH extremes.
- Sodium hypochlorite can be used to inactivate PTV.

PREVENTION AND CONTROL

- Teschovirus encephalomyelitis is notifiable to the U.S. Animal and Plant Health Inspection Service.
- SMEDI syndrome can be prevented by exposing gilts to endemic viruses at least one month prior to breeding (i.e., feedback). PTV cannot likely be excluded from farm environments. However, cleaning and disinfection protocols should be in place.

TRANSMISSION

- Transmission is mainly fecal-oral. Fomites also likely play a role in disease spread.
- PTV has been detected in urine. Transplacental transmission has been demonstrated experimentally, although fetal infection does not always occur.

PATHOGENESIS

- PTV replicates primarily in the tonsils and intestinal tract. Virulent strains cause viremia and travel to the central nervous system via the blood.

DIAGNOSIS

- PTV can be grown in swine-origin cell lines and identified by virus neutralization, complement fixation, or immunofluorescence. Immunohistochemistry can be used to detect virus in fixed tissue samples.
- Multiple reverse transcriptase polymerase chain reaction (RT-PCR) assays have been described targeting the 5ˈNTR, VP1, and VP2 regions of PTV.
- Enzyme-linked immunosorbent assays (ELISAs) have been developed for PTV. One was tested with oral fluids, but no anti-PTV antibodies were detected in study samples.

EPIDEMIOLOGY

- Swine, including wild boar, are the only known hosts for PTV.
- Periodic polioencephalomyelitis outbreaks occur. Less virulent PTVs have been found many regions.
- Teschen disease causes high morbidity and mortality in all age groups. Talfan disease mainly affects younger, post-weaning pigs, and lower morbidity and mortality rates are observed.

ETIOLOGY

- PTV is a non-enveloped RNA virus in the genus *Teschovirus*, family *Picornaviridae*. Until recently, all PTVs belonged to the species *Teschovirus A*; however, a second species, *Teschovirus B*, has now been described.
- There are at least 13 distinct PTV serotypes. New PTVs are being frequently detected.

HISTORY IN SWINE

- PTV-1, which causes teschovirus encephalomyelitis, was first reported in Czechoslovakia in 1929; outbreaks continued during the 1940s and 50s across Europe.
- North American outbreaks include the United States (2003), Haiti (2009), and Canada (2011).

IMMUNITY

- Maternal antibodies are protective against PTV-induced reproductive disorders.
- Both live and attenuated vaccines were used during European outbreaks of Teschen disease but are no longer commercially available.
- Cross-protection between PTV strains is unlikely.

GAPS IN PREPAREDNESS

- In the United States, diagnostic laboratory submissions with a history of posterior paresis seem to be increasing in pigs from a wide age range. Additionally, in other countries, sporadic outbreaks of severe polioencephalomyelitis continue to occur.
- Teschen disease causes high morbidity and mortality in pigs of all ages and can result in serious economic losses. More research is needed to understand why some PTVs induce severe polioencephalomyelitis, and others do not.
- Additionally, no PTV vaccines are currently available.

LITERATURE REVIEW: PORCINE TESCHOVIRUS

IMPORTANCE

Porcine teschovirus (PTV) is an enteric picornavirus of swine. Most infections are subclinical, but some strains cause severe central nervous system disease (polioencephalomyelitis) with high mortality. Why some PTVs cause polioencephalomyelitis and others circulate inapparently is not known. PTVs are also associated with reproductive, gastrointestinal, and respiratory disease.

PUBLIC HEALTH

PTV is not considered to be zoonotic.[1] In 2013, a report from Bolivia described an outbreak of acute flaccid paralysis (AFP) in children that was temporally associated with neurologic disease in domestic pigs.[2] Fecal samples were collected from AFP cases and pigs in nearby communities. Testing via reverse transcriptase polymerase chain reaction (RT-PCR) showed that none of the human specimens were positive for PTV, but human picornaviruses belonging to the genera *Parechovirus*, *Cosavirus*, and *Cardiovirus* were found in swine fecal samples.[2]

INFECTION IN SWINE

Many PTV infections are subclinical in domestic and wild pigs.

POLIOENCEPHALOMYELITIS

Polioencephalomyelitis caused by virulent PTV-1 (teschovirus encephalomyelitis, Teschen disease) is the most serious form of PTV, affecting pigs of all ages. Initial signs include fever, anorexia, and listlessness, followed by ataxia.[1] Nystagmus, convulsions, opisthotonus, and coma may occur in severe cases.[1] Some pigs become hypersensitive, grinding their teeth, smacking their lips, and squealing.[3] Paresis or paralysis can be seen as early as 2–3 days post-infection (dpi), and pigs may be found dog-sitting or in lateral recumbency.[1] Commonly, death occurs 3–4 days after the onset of clinical symptoms due to paralysis of respiratory muscles.[1, 3, 4] An outbreak in 6–7 week-old pigs attributed to PTV-13 caused sudden death in some animals as well as central nervous system (CNS) signs. Necrosis of the ear, keratoconjunctivitis, and corneal opacity were also noted.[5]

Milder cases of polioencephalomyelitis (benign enzootic paresis, Talfan disease) are caused by PTV-1, 2, 3, 5, and 11.[1] Younger animals are most affected, once maternal antibodies have waned.[1] Disease rarely progresses to complete paralysis.[1] Piglets with mild cases may recover.[1]

Outbreaks of polioencephalomyelitis occur periodically, and at least one has occurred in the United States[6] (see *History in Swine*). Experimentally, PTV-induced polioencephalomyelitis has been demonstrated in colostrum-deprived piglets inoculated with PTV-2 and PTV-11.[7]

There are no gross lesions associated with polioencephalomyelitis.[8] Histology usually shows non-suppurative polioencephalomyelitis, with lymphocytic vascular cuffing distributed throughout the CNS. Changes may be more prominent and widespread in cases of Teschen disease compared to Talfan disease.[1] Neurons in the cerebellum may show progressive diffuse chromatolysis with focal areas of gliosis and perivascular lymphocytes.[1]

REPRODUCTIVE DISEASE

Reproductive disease has been linked to PTV-1, 3, and 6.[1] In the 1960s, PTVs were associated with SMEDI syndrome (stillbirth [S], mummified fetus [M], embryonic death [ED], infertility[I]).[9] Infection in early to mid-gestation (40–70 days) leads to embryonic death and mummification, while infection during later stages of development can cause stillbirth.[1, 10] An association with abortion has been confirmed experimentally and in the field.[1, 11, 12] PTV must be distinguished from porcine parvovirus, which induces similar losses and is another cause of SMEDI syndrome.[1]

There are no clinical signs in PTV-infected sows or gilts.[10] PTV has been isolated from the male reproductive

tract, but experimental intrauterine inoculation did not result in infection of embryos or prevent conception.[1, 10, 13] No gross lesions are seen in stillborn or neonatal piglets affected by SMEDI syndrome.[1] Occasionally, mild focal gliosis and perivascular cuffing are found in the brain stem.[1] Placental degeneration is non-specific.[1]

GASTROINTESTINAL DISEASE

Enteric disease has been linked to PTV-1, 2, 3, and 5. Co-infection with other enteric pathogens is common.[14-20] PTVs have been detected in feces from healthy pigs and pigs with diarrhea (see *Morbidity and Mortality*). Isolation from the intestinal tract may be of no significance. However, PTV gastrointestinal disease has been experimentally induced in pigs believed to be free of other pathogens.[1, 21] Diarrhea is usually mild to moderate, with watery yellow feces.[1, 3] An outbreak of acute diarrhea, respiratory disease, and death in 7–10 week-old pigs was reportedly caused by PTV-8 in China.[21] A similar PTV was linked to sows with reproductive failure.[12]

ADDITIONAL SYNDROMES

PTV-1, 2, and 3 have been associated with respiratory disease.[1] Signs including pneumonia, rhinitis, and dyspnea have been reported.[3] It is unlikely that PTV alone is a cause of respiratory disease in pigs.[1] However, interstitial pneumonia has been seen in some pigs infected with PTV.[3] Additionally, pneumonic lesions have been reproduced experimentally, including grayish-red consolidation in the ventral anterior lung lobes, alveolar and bronchial exudates, and perivascular and peribronchiolar cuffing.[1]

Pericarditis and myocarditis have been linked to PTV-2 and 3.[1] Experimentally, PTV causes diarrhea, pericarditis, myocarditis, and polioencephalomyelitis in germ-free pigs.[1, 22] Grossly, pericarditis may be serofibrinous with a cloudy pericardial effusion that quickly forms a coagulum upon standing. Focal myocardial necrosis with cellular infiltrate may also be present in severe cases.[22]

PTV-5 has been reported as the cause of congenital micropthalmic syndrome in a 17-week-old finishing pig. Reported clinical signs included ataxia, partial flaccid paresis of the pelvic limbs, and skin lesions on the feet and claws. Vision was severely reduced, and both eyes had multiple persistent pupillary membranes and hypermature cataracts.[23]

Teschoviruses were isolated from epithelium and feces during a swine vesicular disease (SVD) outbreak in the United Kingdom.[1, 24]

TREATMENT

There is no treatment for pigs infected with PTV.

CLEANING AND DISINFECTION

SURVIVAL

PTV persists in the environment, surviving at moderate temperatures and pH extremes. The virus is detectable in liquid manure for long periods.[1, 3] PTV has been found in streams that contain water draining from pig farms in Brazil,[25] and water downstream from a pig slurry spill site in Spain.[26] In Iowa, PTV was detected in water about 3.5 miles downstream from a swine manure spill 18 days after the event.[27]

In food safety studies, PTV has been used to indicate fecal contamination since it is cultivable and relatively easy to detect. Experimentally, PTV survived in vacuum packaged pork chops after six weeks at 2°C.[28]

DISINFECTION

Like other picornaviruses, PTV is resistant to heat, lipid solvents, and many disinfectants. Sodium hypochlorite can be used to inactivate PTV.[1, 3, 29] Chloramine, chloride of lime, and caustic soda were reportedly used as disinfectants in historical PTV outbreaks.[3]

PREVENTION AND CONTROL

DISEASE REPORTING

PTV is not currently an OIE-listed disease. There are no restrictions for importation of animals from countries or zones affected by PTV. However, teschovirus encephalomyelitis is considered foreign to the United States, and is therefore notifiable according to the U.S. Animal and Plant Health Inspection Service (APHIS). Any suspicious clinical or necropsy findings should always be reported to the USDA and your State Animal Health Official.

DISEASE PREVENTION

Vaccination has previously been used to control Teschen disease. No vaccines have been developed for PTV serotypes that cause reproductive disorders (see *Immunity*).

PTV persists in the environment and is likely present on most swine farms. To prevent SMEDI syndrome in gilts, they should be exposed to endemic viruses at least one month prior to breeding.[1, 10] Pigs from different litters can be mixed, or if pigs are segregated at an early age, they can be exposed to feces from recently weaned pigs.[1] Replacement animals (sows or gilts) should similarly be exposed to the farm virome via feedback.[1]

It does not seem possible to exclude PTVs from the farm environment. However, cleaning and disinfection protocols should be in place. Additionally, sick pigs should be isolated to minimize disease spread.

DISEASE CONTROL

There are no specific control measures for PTV. Standard biosecurity practices should be in place on all swine premises.

TRANSMISSION

PTV transmission is mainly fecal-oral. However, PTV is hardy in the environment, and fomites likely play a role in disease spread.[1] PTV has also been detected in urine.[30] Transplacental transmission has been confirmed experimentally. In orally inoculated sows, PTV caused intestinal infection, followed by viremia and transplacental spread to fetuses. Not all fetuses became infected, however, due to some protectivity of the blood-placenta barrier.[10]

PATHOGENESIS

Once PTV is ingested, primary replication occurs in the tonsils and intestinal tract.[1, 31-33] Tonsils are believed to be important for viral entry, shedding, and sustained infection.[15] Virus is found in the large intestine and ileum more frequently and in higher amounts than in the duodenum and jejunum. Virulent strains are believed to cause viremia, which allows PTV access to the CNS via blood.[8, 34] When introduced intranasally, PTV travels from the tonsils to the brain in a retrograde fashion.[35] Experimental nasal and oral inoculation have also led to fetal infection in pregnant gilts.[1, 36]

DIAGNOSIS

TESTS TO DETECT NUCLEIC ACIDS, VIRUS, OR ANTIGENS

PTV is easily cultured in cell lines of porcine origin; primary and secondary porcine kidney cells are most commonly used.[1] The virus also replicates in some established cell lines, like IBRS-2.[37] Isolated virus can be identified via virus neutralization, complement fixation, or immunofluorescence.[1] Monoclonal antibodies to detect PTV have been described.[1] Immunohistochemistry on formalin-fixed paraffin-embedded tissue can also be used for virus identification.[38, 39] Isolation of PTV from cases of diarrhea or pneumonia should be interpreted with caution since the virus is common in healthy animals.[1]

Molecular assays described for PTV include:

- RT-PCR targeting the 5ˈNTR [7, 15, 26, 40-47]
- qRT-PCR targeting the 5ˈNTR[26, 35, 48-51]
- RT-PCR targeting VP2 [52]
- RT-PCR targeting VP1[42, 53-55]
- RT-LAMP (loop-mediated isothermal amplification)[56]
- Duplex RT-PCR (sapelovirus and PTV) targeting VP1[57, 58]
- Multiplex RT-PCR (porcine circovirus type 2 [PCV2], transmissible gastroenteritis virus [TGEV], and PTV)[59]
- Multiplex RT-PCR (porcine reproductive and respiratory syndrome virus [PRRSV], classical swine fever virus [CSFV], and PTV)[17]
- Multiplex RT-PCR (porcine sapovirus, porcine deltacoronavirus, porcine kobuvirus, porcine astrovirus, porcine torovirus, and PTV)[60]
- Multiplex qPCR/RT-PCR (PCV2, PRRSV, pseudorabies, and PTV)[61]

TESTS TO DETECT ANTIBODY

Virus neutralization can be used for diagnosis only when paired sera are available, and the PTV serotype is known.[1] An enzyme-linked immunosorbent assay (ELISA) has been developed for teschovirus encephalomyelitis, detecting antibodies to PTV-1.[1]

A pan-PTV indirect ELISA has been tested with serum and oral fluids; however, no anti-PTV antibodies were detected in oral fluids during the study.[62] Even when a fluorescent microsphere immunoassay (FMIA) was used to increase sensitivity, antibody detection in oral fluids was low and highly variable.[62]

A recombinant protein based on a highly conserved epitope (RNNQIPQDF) on the GH loop of VP1 has been developed as a potential pan-PTV diagnostic reageant.[63]

SAMPLES

In cases of Teschen disease, virus can be isolated from the CNS (spinal cord, brain stem, or cerebellum).[1] Infectious virus may not be present in animals that have been paralyzed for several days.[1] Although live virus cannot be reliably detected in stillborn or mummified fetuses, fetal lung is the best sample for virus isolation.[36] Fetal body fluids have been used for virus neutralization.[1]

EPIDEMIOLOGY

SPECIES AFFECTED

Swine are the only known host for PTV; the virus is found in both domestic and wild pigs.[1] A teschovirus-like virus was recovered from a bat in Saudi Arabia.[64] Novel picornaviruses found in cattle and sheep in Hungary were closely related to PTV.[65]

GEOGRAPHIC DISTRIBUTION

Historically, Teschen disease (PTV-1) has occurred mainly in Europe.[1, 3] Periodic outbreaks continue to occur (see *History in Swine*).

Other PTV serotypes and less virulent strains of PTV-1 are ubiquitous. Specifically, PTV has been detected in domestic pigs in China,[46, 55, 59, 66-69] Japan,[70, 71] Denmark,[72] Italy,[42, 73], Switzerland,[20] the United Kingdom,[37] the Czech Republic,[14] Spain,[41, 58] India,[44, 54] Brazil,[15, 16] and Kenya and Uganda.[74] PTV has also been isolated from wild boar in Hungary,[75] the Czech Republic,[14] Spain,[57] and Brazil.[45]

MORBIDITY AND MORTALITY

Teschovirus encephalomyelitis causes high morbidity and mortality in all age groups. Morbidity and mortality from the 2009 outbreak in Haiti were 60% and 40%, respectively.[4] In the 2011 Canadian outbreak, reported mortality was 100%.[76] A 2018 Spanish outbreak of atypical neurological disease in 6–7-week-old pigs (sudden death, neurological signs, ear necrosis, and occasional corneal opacity) had a morbidity rate of 20%, a fatality rate of 60%, and a mean batch mortality rate of 10–12%.[5]

Talfan disease is generally less severe, with low morbidity and mortality, affecting young post-weaning animals.[12,13] Pigs with milder presentations of polioencephalomyelitis may survive if their appetite returns after the transient paresis phase.[1]

Reported prevalence estimates for PTV include:

- 10% of fecal samples from U.S. pigs also infected with porcine epidemic diarrhea virus (PEDV)[77]
- 47% of fecal samples from domestic pigs in Spain[41]
- 47–90% of fecal samples from domestic pigs in China[46, 50, 59, 68]
- 7% of fecal samples from North India[54]
- 47% of fecal samples from healthy pigs; 54% of fecal samples from pigs with diarrhea in Switzerland[20]
- 70% of fecal samples from wild boar in Hungary[75]
- 51% of fecal samples from wild boar in Spain[57]
- 45% of fecal samples from wild pigs in Brazil[45]

Several studies have investigated PTV prevalence in pigs of different ages. In Brazil, PTV was found in 25% of diarrheic suckling piglets and 18% of diarrheic nursery pigs.[15] A second Brazilian study found no PTV in feces from suckling pigs; however, PTV was detected in weaned pigs along with enterovirus (15%) and sapelovirus A (52%).[16] In China, PTV was detected in 89% of nursery pigs (6–10-weeks-old) and 90% of fattened pigs (10–12-weeks-old).[50] Another study found that 86% of pigs aged 8–12 weeks were PTV-positive.[68]

ETIOLOGY

CHARACTERISTICS OF PICORNAVIRUSES

Teschoviruses are members of the family *Picornaviridae*.[1] Picornaviruses are small (30 nm), round, single-stranded positive-sense RNA viruses. They have a large open reading frame (ORF) translated into a polyprotein containing a leader (L) protein, four structural capsid proteins (V1–4), and seven nonstructural proteins (2A–2C, 3A–3D).[1, 78, 79] Additionally, picornaviruses have one of five internal ribosome entry sites (IRESs) involved in ribosome recruitment and translation initiation. Type IV IRES is found in PTV.[80]

The family *Picornaviridae* currently contains 68 genera and 158 species.[81] Additionally, picornavirus "supergroups" have been proposed based on phylogenetic clustering. Teschoviruses belong to SG1, which includes fifteen other genera, including *Aphthovirus* and *Senecavirus*.[80] Picornaviruses that infect pigs are found in the genera *Kobuvirus*, *Aphthovirus, Cardiovirus, Cosavirus, Enterovirus, Pasivirus, Parechovirus, Sapelovirus, Senecavirus,* and *Teschovirus*.[1]

CHARACTERISTICS OF TESCHOVIRUSES

The name teschovirus is derived from Teschen disease, named after the Czech/Polish town where it was first discovered.[82] Previously, teschoviruses were known as "porcine enteroviruses" (PEVs). However, based on serotyping and genomic sequencing, PEVs have been reclassified as teschoviruses, sapeloviruses, and true enteroviruses.[1, 82]

There are at least 13 distinct PTV serotypes. An animal can be co-infected with two or more serotypes simultaneously, and natural recombination has been documented.[67, 83, 84]

Former PEV serotypes 1–7 and 11–13 are now known as PTV-1 to PTV-10. These serotypes belong to the species *Teschovirus A* in the genus *Teschovirus*.[1, 82] Requirements for classification as a teschovirus include: < 20% divergence in polyprotein aa sequence, < 30% divergence in P1 aa sequence, and < 10% divergence in 2C+3CD aa sequence, as well as a shared genome organization and natural host range.[82]

Teschovirus A also contains more recently identified serotypes, including:

- PTV-11, isolated from the CNS of a pig with neurological disease in Germany in 1995.[85, 86]
- PTV-12, found in feces of domestic pigs from Spain in 2011.[33, 58, 87]
- PTV-13, first found in the feces of wild boar in Hungary in 2011.[6]
- Putative PTVs 14–22, identified in feces and intestinal contents from domestic pigs in China (samples collected from 2014–2017).[50, 55] PTV-17 has also been described in diarrheic pigs in India.[44] Additionally, PTV-17 and -18 have been detected in China and confirmed as members as *Teschovirus A*.[69]

Recently, a second species has been added to the genus *Teschovirus*. PTV-21 (HuN41-42)[55] is now classified as *Teschovirus B*.[82] An isolate from swine feces in Japan, initially described as "teschovirus A-related," has also been placed in *Teschovirus B*. [71]

HISTORY IN SWINE

Teschovirus encephalomyelitis, caused by PTV-1, was first described in Czechoslovakia in 1929. Extensive outbreaks continued during the 1940s and 50s across Europe.[1, 3] An outbreak of polioencephalomyelitis in nursery pigs in Indiana was attributed to PTV-1 in the early 2000s.[6, 88] Other instances of teschovirus encephalomyelitis in North America occurred in Haiti in 2009[4] and Canada in 2011.[76]

Additional outbreaks of PTV-associated neurological disease include:

- PTV (serotype not determined) in piglets, Japan, 2002[89]
- PTV-1 in piglets, China, 2007[66]
- PTV-3 and -11 in weaning piglets (6–12 weeks-of-age), the Netherlands, 2014–2015[61]
- PTV-13 in 6–7-week-old piglets, Spain, 2017[5]
- PTV-1 in weaning piglets, Brazil, 2021[90]
- PTV-1 in neonatal pigs, Northeast China, 2021[84]

In a few instances, co-infection with other pathogens has been documented in pigs with PTV polioencephalomyelitis. In Japan, both PTV and PCV2 were isolated from a pig with polioencephalomyelitis and respiratory distress.[91] Both atypical porcine pestivirus (APPV) and PTV were detected in the CNS of piglets with congenital tremor type A-II in Brazil.[92]

IMMUNITY

POST-EXPOSURE

The primary immune response to PTV is humoral (IgM, IgG), but mucosal immunity (IgA) may have a protective role in the gastrointestinal tract.[1, 93-95] Cell-mediated immunity is weak and localized with no significant or specific antiviral activity.[1, 96]

Maternal antibodies are likely protective against PTV-induced reproductive loss since they prevent vertical transmission. However, if PTV reaches the uterus, maternal antibodies have no effect on embryonic or fetal infections.[1, 36] Fetal development of anti-PTV antibodies (IgM followed by IgG) begins at 68 days of gestation and reaches maturity around 84–96 days of gestation.[97] Colostral antibodies are protective until weaning.[10] In a prolonged outbreak of polioencephalomyelitis in Indiana, colostral antibody titers were likely not high enough to

protect uncharacteristically large litters, resulting in the development of polioencephalomyelitis.[6]

VACCINES

During European outbreaks of Teschen disease, attenuated and inactivated PTV-1 vaccines were used.[1] Along with restricted imports of swine and pork products from endemic areas, vaccines were effective in limiting the spread of PTV.[1] Vaccination has not been practiced for other syndromes associated with PTV. Since multiple serotypes cause SMEDI syndrome, a multivalent PTV vaccine would be needed for protection.[1]

There are currently no PTV vaccines available. PTV T cell and B cell epitopes (RPVNDE, AYSRSHPQ) have been identified for possible use in virus-like particle (VLP) or subunit vaccines.[98]

CROSS-PROTECTION

Due to the large number of PTV serotypes, cross-protection is not likely.[1, 99] Experimentally, infection with PTV-12 (isolate *CC25*) does not prevent infection with PTV-1 in minipigs.[33]

GAPS IN PREPAREDNESS

Most PTVs described since the 1950s have caused mild polioencephalomyelitis in young animals. However, in the United States, diagnostic laboratory submissions with a history of posterior paresis seem to be increasing in pigs from a wide age range.[7, 100] Additionally, in other countries, sporadic outbreaks of severe polioencephalomyelitis continue to occur. Teschen disease, caused by PTV-1, leads to high morbidity and mortality in pigs of all ages and can result in serious economic losses. More research is needed to understand why some PTVs induce severe polioencephalomyelitis, and others do not. Additionally, no PTV vaccines are currently available.

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