

INFECTION WITH PORCINE EPIDEMIC DIARRHOEA VIRUS

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Porcine epidemic diarrhoea (PED), also occasionally referred to as porcine epidemic diarrhoea syndrome, is a non-zoonotic viral disease of pigs caused by a coronavirus and characterised by watery diarrhoea and weight loss. It was first identified and reported in 1971 but has now been diagnosed in naïve swine populations in countries previously not known to be affected by the disease. It affects pigs of all ages, but most severely neonatal piglets, reaching a morbidity and mortality of up to 100% with mortality decreasing as age increases. It is a contagious disease transmissible mainly by the faecal-oral route. The disease is clinically similar to other forms of porcine gastroenteritis including anorexia, vomiting, diarrhoea and dehydration. The prevention and management control are focussed on strict biosecurity and early detection. There is no specific treatment for the disease.

PED is not included in the OIE List of Diseases. However, consistent with the reporting obligations of Member Countries outlined in Article 1.1.3. 1) e) of the OIE *Terrestrial Animal Health Code* relating to emerging diseases, there has been an increase in the number of disease notifications received by and distributed through the OIE's World Animal Health Information System.

The information presented in this technical factsheet reflects the epidemiological observations and research done to date (June 2014) and will be updated when additional information is available.

AETIOLOGY

Classification of the causative agent

PED virus is an enveloped RNA virus that belongs to the Alphacoronavirus genus of the Coronaviridae family. It does not demonstrate cross-immunity with other porcine enteric coronaviruses such as the virus responsible for transmissible gastroenteritis (TGE).

Susceptibility to physical and chemical action

PED virus is susceptible to

- Formalin (1%),
- Anhydrous sodium carbonate (4%), lipid solvents, iodophores in phosphoric acid (1%),
- Sodium hydroxide (2%).

Survival:

- The virus can survive for variable periods outside the host depending on the temperature and relative humidity, for example, it can survive at least 28 days in slurry at 4°C, 7 days in faeces-contaminated dry feed at 25°C, up to 14 days at 25°C in wet feed and at least 28 days in wet feed mixture at 25°C,
- The virus loses infectivity above 60 °C,
- It is stable at pH 6.5-7.5 at 37°C and pH 5-9 at 4°C.

Epidemiology

Host

Pigs are the only known host of PED virus. The occurrence of PED in wild pigs is unknown.

PED is not a zoonosis and does not pose a risk to human health or to food safety.

Transmission

Direct transmission occurs through ingestion of virus-contaminated faeces.

Indirect transmission occurs through vehicles which may be contaminated including feed trucks, service vehicles as well as personnel, equipment or other types of faeces-contaminated objects including feed.

Contaminated pig blood products, such as spray-dried plasma, that are incorporated into rations for feeding piglets have been suspected as a possible means to spread the virus. However, multiple experimental studies suggested that spray-dried porcine plasma is not a likely source of infectious virus provided that good manufacturing practices and biosecurity standards are followed.

Contaminated vehicles used for the movement of pigs have been identified as an important risk factor for spreading the disease.

Viraemia, incubation and infectious period

The incubation period is estimated to be between 1 and 4 days. The infectious period can last between 6 and 35 days after the first onset of clinical signs. Viraemia has been detected on multiple days in pigs 2-4 weeks of age experimentally infected with PED virus.

Sources of virus

The main source of this enteric virus is faeces.

Pathogenesis

Oral ingestion results in viral replication in the epithelial cells of the small intestinal and colonic villi resulting in degeneration of enterocytes leading to shortening of the villi. This causes clinical manifestations of the disease including watery diarrhoea.

Occurrence and impact

PED was first reported in the United Kingdom in 1971 and has since then been identified in several European countries, large parts of Asia and the Americas. PED virus has been associated with large-scale outbreaks of diarrhoea with severity depending on pig age. In endemic countries, the impact has been limited to scenarios with occasional clinical outbreaks. However PED can produce important losses in naïve populations. There have been increased reports since 2011 regarding high morbidity and mortality mostly in young pigs. In outbreaks described in 2013 and 2014, mortalities in suckling piglets ranging from 50 to 100% were detected at the farm level.

Diagnosis

Clinical diagnosis

The clinical presentation of PED virus infection in pigs can be variable in its severity and is not distinguishable from other causes of diarrhoea. The clinical signs are dependent on age of the pigs, previous exposure and the immunological status of the pigs, presence of secondary infection, etc.

The following signs could be found in PED virus infection:

- Morbidity: up to 100%,
- Mortality varying according to age:
 - Suckling piglets: up to 100%
 - Piglets older than 10 days: less than 10%
 - Adult and fattening pigs: less than 5%
- Diarrhoea and vomiting
- Dehydration and metabolic acidosis.

Lesions

Post-mortem findings in acutely affected pigs are similar to transmissible gastroenteritis (TGE) and can include:

- Thinning of the intestines, mostly limited to the small intestines,
- Presence of undigested milk in the stomach,
- Watery intestinal contents.

Differential diagnosis

PED is clinically indistinguishable from other pig gastroenteric diseases such as those caused by TGE or rotavirus, by bacteria (*Clostridium spp.*, *E. coli*, *Salmonella spp.*, *Brachyspira spp.*, *Lawsonia intracellularis*, etc.) or by parasites (*Isospora suis*, *Cryptosporidium spp.*, nematodes, etc.).

Laboratory confirmatory tests are thus necessary to make a final and definitive diagnosis.

Laboratory diagnosis

Samples

- Fresh faeces,
- Oral fluids,
- Small intestine,
- Serum can be used to determinate the presence of antibodies.

Procedures

Identification of the agent

- Reverse-transcriptase polymerase chain reaction (RT-PCR),
- Antigen enzyme-linked immunosorbent assays (ELISA),
- Immunohistochemistry (IHC),
- Virus isolation (difficult to isolate the virus).

Serological tests

- ELISA,
- Immunofluorescence,
- IHC,
- Serum neutralisation.

Prevention and control

There is no specific treatment other than symptomatic treatment of diarrhoea and control of secondary infections. Most growing pigs recover without treatment within 7-10 days unless secondary infections occur. Reinfection may occur when the immunity wanes.

Maternal antibodies via colostrum from immune sows can protect neonates against infection.

PED vaccines are available and applied in several countries.

Strict biosecurity is the most effective measure to prevent the introduction and spread of the virus, especially, introduction of pigs of known health status, on-farm movement control of pigs, material and people, disinfection of vehicles, equipment and appropriate disposal of dead pigs and slurry. The implementation and maintenance of high biosecurity programmes has been efficient to control PED in endemic countries. "All-in-all-out" practice has been demonstrated to be effective in breaking the transmission cycle within a farm.

References

1. Guscetti F., Bernasconi C., Tobler K., Van Reeth K., Pospischil A. & Ackermann M. (1988). Immunohistochemical detection of porcine epidemic diarrhoea virus compared to other methods. *Clin Diagn Lab Immunol.*, **5**(3): 412-414.
2. Pospischil A., Stuedli A. & Kiupel M. (2002) Diagnostic Notes Update on porcine epidemic diarrhoea. *Journal Swine Health Production*, **10**, 81-85.
3. Morales R.G., Umandal A.C. & Lantican C.A. (2007) Emerging and re-emerging diseases in Asia and the Pacific with special emphasis on porcine epidemic diarrhoea. Conference OIE 2007, 185-189.
4. Song D. & Park B. (2012). Porcine epidemic diarrhoea virus: a comprehensive review of molecular epidemiology, diagnosis and vaccines. *Virus genes*, **4**, 167-175.
5. Saif L.J. *et al.* (2012). Chapter 35. Coronaviruses. *in: Diseases of swine*. J.J. Zimmerman, L.A. Karriker, A. Ramirez, K.J. Schwartz and G.W. Stevenson, eds. Ames, IA, Wiley-Blackwell: 501-524.
6. Woo P.C.Y., Lau, S.K.P., Lam C.S.F., Lau C.C.Y., Teng J.L.L., Tsang C.C.C., Wang M., Zheng B., Chan K.H. & Yuen K.Y. (2012). Discovery of Seven Novel Mammalian and Avian Coronaviruses in the Genus Deltacoronavirus Support Bat Coronaviruses as the Gen Source of Alphacoronaviurs and Betacoronavirus and Avian Coronaviruses as the Gene Source of Gammacoronavirus and Deltacoronavirus.
7. Dufresne L. & Robbins R. (2014). Field experience with porcine epidemic diarrhoea. *American Association of Swine Veterinarians*. 613-616.