Technical Note

Porcine Epidemic Diarrhea (PED)

OVERVIEW

The porcine epidemic diarrhea (PED) virus was first recognized in the United Kingdom in 1971 and had spread throughout much of Europe and Asia by 2013. The United States Department of Agriculture's (USDA) National Veterinary Services Laboratories (NVSL) confirmed the first PED diagnosis in the United States on May 17, 2013 in Iowa. PED's pathogenic agent is a coronavirus, which is distinguishable from transmissible gastroenteritis of swine (TGE) only by laboratory tests. PED is most serious in neonatal piglets where morbidity and mortality can be 80 to 100 percent. Transmission of PED is fecal-oral; no vector or reservoir has been implicated in its spread.

Economic loss occurs directly in the form of death and production loss in swine. Further monetary loss occurs because of the cost of vaccination and biosecurity. There is no effective treatment other than control of secondary infections. Vaccines exist in Japan, South Korea, and China, but not in Europe or the United States. PED is not a listed disease for either the World Organization for Animal Health (OIE) or the USDA, so no quarantines or movement restrictions are in place either internationally or interstate.

AGENT CHARACTERISTICS

Location of Outbreaks

Worldwide—Porcine Epidemic Diarrhea was first reported as a clinical entity in England in 1971 and was determined to be separate from porcine TGE in 1977 (Wood, 1977). The agent was further identified as a coronavirus-like particle in Belgium in 1978 (Pensaert and de Bouck, 1978). Although no evidence of PED is currently reported from Canada, similar coronavirus-like particles were reported from herds in Quebec in 1980 (Turgeon et al., 1980). Since then, PED has been found in the Czech Republic, Hungary, Korea, the Philippines, China, Italy, Thailand, Germany, Spain, and Japan (Song and Park, 2012; Pospischil et al., 2002).

United States—On May 17, 2013, USDA NVSL confirmed the finding of PED virus in Iowa swine. Additional occurrences of PED have been confirmed in multiple states.

Biologic Classification

PED virus is a single-stranded, positive-sense RNA group 1 Coronavirus with a diameter which averages 130 nm. It contains a centrally located electron-opaque body and club-shaped projections of 18 to 23 nm. The internal structure of PED is not known (Park et al., 2013). The structural gene "spike" or "S" is a 28-kb genome portion that encodes the multifunctional virulence factor (Li et al., 2012).

Significant genetic variation of PED has been documented in reemerging PED in China (Yang et al., 2013). PED is sensitive to ether and chloroform and loses infectivity when heated to or above 60˚C. It is reported to be susceptible to formalin (1%), anhydrous sodium carbonate (4%), lipid solvents, iodophores in phosphoric acid (1%), and sodium hydroxide (2%) (Pospischil et al., 2002).

Related Agents—PED virus is a coronavirus most closely related to TGE virus and can only be differentiated by confirmatory laboratory tests (Song and Park, 2012).
Natural Host—Swine are the only known hosts of PED virus (Harris, 2012). Mice are specifically demonstrated to be non-competent vectors (Kamau et al., 2010).
Alternate or Reservoir Hosts—Alternate or reservoir hosts are not reported.
Clinical Signs—The incubation period of PED is 3 to 4 days (Harris, 2012). The clinical presentation of PED is not distinguishable from TGE. Clinical signs of PED may vary widely and are dependent on previous exposure and the immunological and endemic status of the farm, region, or area affected. The primary clinical finding is watery feces that may be flocculent and fetid. Vomiting may occur. Dehydration and metabolic acidosis may be secondary signs. PED may spread more slowly than TGE. If swine recover, it is usually within 7 to 10 days (Pospischil et al., 2002).
Pathologic Signs—Histopathologic lesions characteristically include small intestinal villous blunting. Ultrastructural colon lesions have been observed. Acute back muscle necrosis occurs but is not pathognomonic (Pospischil et al., 2002). At the cellular level, PED protein E becomes localized in the endoplasmic reticulum with small amounts being found in the nucleus of infected cells (Xu et al., 2013).
Morbidity—Morbidity can approach 100% in all ages of susceptible swine (Turgeon et al., 1980).
Mortality—Mortality may approach 100% in highly susceptible swine of any age. Piglets less than 7 days old may have a mortality rate of about 50%, with mortality decreasing as age increased. In suckling pigs, mortality commonly reaches 50 to 80% but declines to 1 to 3% in grower pigs (Pospischil et al., 2002).

Modes of Transmission

Direct Transmission—Fecal-oral transmission is the main, and perhaps only, mode of transmission. Clinical signs of PED may occur within 4 to 5 days following introduction of infected swine to farms with susceptible animals. Following an outbreak, PED may subside but may become endemic if sufficient litters are produced to overcome lactogenic immunity (Pospischil et al., 2002).
Indirect Transmission—Contaminated personnel, equipment, or other fomites may introduce PED into a susceptible herd (Pospischil et al., 2002).

CASE DEFINITION

Suspect Case

Acute, malabsorptive diarrhea with high morbidity (greater than 50 percent) affecting one or more age groups of swine AND atrophic enteritis demonstrated by histopathology.

Presumptive Positive Case
A suspect case that tests negative for TGE virus by current diagnostic protocols.

Confirmed Positive Case
First confirmed positive case in a State: A pig that has tested positive for PED virus by PCR or virus isolation and has been identified by sequencing. Secondary confirmed positive cases in a State: A pig that has tested positive for PED virus by PCR or virus isolation (USDA, 2013).

TESTS AVAILABLE

Immunofluorescence (IF) tests, immunohistochemical tests, electron microscopy, and enzyme-linked immunosorbent assays (ELISA) are available but time consuming and often of insufficient sensitivity and specificity. Reverse Transcriptase Polymerase Chain Reaction (RT-PCR) using M-gene derived primers to obtain PED-specific fragments have been shown to identify PED more frequently than other methods. A protein-based ELISA is also useful (Song and Park, 2012).

TREATMENT AND PREVENTING INFECTION

Most growing swine recover without treatment unless secondary infections occur. Specific treatment is of uncertain value because the agent is a virus for which there is no specific or economically feasible medication. When secondary infections occur, neomycin, framycetin, trimethoprim/sulpha, lincomycin or tiamulin may be effective (The Pig Site Article 345, 2013).

Prevention
Natural Immunity—Maternal antibodies via colostrum from PED immune sows may protect neonates against oral infection until about 4 to 13 days of age, but may not protect against intestinal infection (Song and Park, 2012).
Biosecurity—Excellent biosecurity should always be practiced. If PED becomes endemic in finishing units, it may be helpful to break the cycle by suspending additions for three weeks. "All-in-all-out" practices may also be helpful in breaking the transmission cycle (The Pig Site Article 345, 2013).
Vaccination—No PED vaccine exists for use in the United States. Licensed PED vaccines are currently available in South Korea, Japan, and China (The Center for Food Security and Public Health, 2013). In Europe, PED economic losses did not justify vaccine development (Song and Park, 2012). There is recent evidence from China that because of three new PED variants discovered in 2011, the CV777 based vaccine is no longer effective (Li et al., 2012).

ZOONOTIC POTENTIAL

PED is not transmissible to humans thus poses no danger to human health (Pospischil et al., 2002).

FOREIGN ANIMAL DISEASE STATUS

PED is not an "OIE listed" disease and is not internationally reportable (World Organization for Animal Health, 2013). PED is not a nationally reportable disease (United States Department of Agriculture, 2011).

INDUSTRY RISKS

Economic Effect of PED Presence

Significant economic loss in the swine industry is possible because of the high morbidity and mortality that occurs in immunologically naïve neonatal piglets. Vaccination and increased biosecurity further increase production costs (Song and Park, 2012). Acute outbreaks may become less common, but marginally less costly, if PED becomes widespread (Pospischil et al., 2002).

Value of U.S. Swine Industry

In 2011, United States agriculture produced 110.9 million hogs and 22.8 billion pounds of pork (The American Meat Institute, 2013). Pork products constitute the second largest segment of the United States meat and poultry production, which is in itself the largest segment of United States agriculture. The negative economic effect following discovery of PED in the United States cannot be estimated at this early stage, but the disease has been manageable in Europe and Asia.

Value of Trade in Swine and Swine Products

In 2011, the United States exported 1.75 billion metric tons of pork and related products worth $5.32 billion (The American Meat Institute, 2013). Japan and Mexico are the two leading importers of U.S. pork products by value.

FOR MORE INFORMATION, CONTACT:

USDA APHIS Veterinary Services
Center for Epidemiology and Animal Health
NRRC Building B, M.S. 2W4
2150 Centre Avenue
Fort Collins, CO 80526-8117
970-494-7000
E-mail: vs.ceah@aphis.usda.gov

REFERENCES

Harris, H.D.L., 2012. Porcine Epidemic Diarrhea. The Merck Manual. Merck, Sharpe and Dohme Corp.

Kamau, N.A., Park, J.Y., Park, J.E., Hyun, B.H., 2010. Susceptibility of Mice to Porcine Epidemic Diarrhea Virus. Journal of Animal and Veterinary Advances 9, 3114-3116.

Krasny, R., 2013. Virus found in Iowa hog population, possibly beyond. Reuters. Thomson Reuters, Washington, D.C., 1.

Li, W., Li, H., Liu, Y., Pan, Y., Deng, F., Song, Y., Tang, X., He, Q., 2012. New variants of porcine epidemic diarrhea virus, China, 2011. Emerging infectious diseases 18, 1350-1353.

Park, S.J., Song, D.S., Park, B.K., 2013. Molecular epidemiology and phylogenetic analysis of porcine epidemic diarrhea virus (PED) field isolates in Korea. Archives of virology.

Pensaert, M., de Bouck, P., 1978. A New Coronavirus-like Particle Associted with Diarrhea in Swine. Archives of virology 58, 243-247.

Pospischil, A., Stuedli, A., Kiupel, M., 2002. Diagnostic Notes Update on porcine epidemic diarrhea. J Swine Health Prod 10, 81-85.

ProMed, 2013. Porcine Epidemic Diarrhea-USA (Iowa) First Report. International Society for Infectious Diseases.

Song, D., Park, B., 2012. Porcine epidemic diarrhoea virus: a comprehensive review of molecular epidemiology, diagnosis, and vaccines. Virus genes 44, 167-175.

The American Meat Institute, 2013. The United States Meat Industry at a Glance.

The Center for Food Security and Public Health, 2013. Vaccines : Porcine Epidemic Diarrhea. Iowa State University.

The Pig Site, 2013. USDA Confirms Porcine Epidemic Diarrhoea Virus. The Pig Site.

The Pig Site Article 345, 2013. Porcine Epidemic Diarrhea. The Pig Site.

The Pig Site Article 453, 2013. Porcine Epidemic Diarrhoea. The Pig Site.

Turgeon, D.C., Morin, M., Jolette, J., Higgins, R., Marsolais, G., DiFranco, E., 1980. Coronavirus-like particles associated with diarrhea in baby pigs in Quebec. The Canadian veterinary journal. La revue veterinaire canadienne 21, 100-xxiii.

United States Department of Agriculture, 2011. National Animal Health Reporting System Reportable Disease List.

USDA-APHIS-VS-CEAH National Surveillance Unit, 2013. Case definition for porcine epidemic diarrhea. Date accessed: May 21, 2013.

Wood, E.N., 1977. An apparently new syndrome of porcine epidemic diarrhoea. The Veterinary record 100, 243-244.

World Organization for Animal Health, 2013. OIE-Listed diseases, infections and infestations in force in 2013.

Xu, X., Zhang, H., Zhang, Q., Dong, J., Liang, Y., Huang, Y., Liu, H.J., Tong, D., 2013. Porcine epidemic diarrhea virus E protein causes endoplasmic reticulum stress and up-regulates interleukin-8 expression. Virology journal 10, 26.

Yang, X., Huo, J.Y., Chen, L., Zheng, F.M., Chang, H.T., Zhao, J., Wang, X.W., Wang, C.Q., 2013. Genetic variation analysis of reemerging porcine epidemic diarrhea virus prevailing in central China from 2010 to 2011. Virus genes 46, 337-344.