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Porcine Reproductive and Respiratory Syndrome (PRRS) Virus

This NebGuide explains Porcine Reproductive and Respiratory Syndrome--its symptoms, transmission and diagnosis; and it discusses methods of prevention, management and control.

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History

Porcine Reproductive and Respiratory Syndrome (PRRS) was first observed in 1986 in the United States and in 1990 in Europe. The syndrome was initially called Mystery Pig Disease in the United States since no known swine pathogens could be implicated. A number of titles have been used to describe this disease syndrome: swine infertility and respiratory syndrome (SIRS), porcine epidemic abortions and respiratory syndrome (PEARS), blue-eared pig disease as well as others. Since 1992, PRRS has been the internationally recognized name applied to the syndrome.

Cause

The etiological agents of PRRS was confirmed to be a positive-stranded enveloped RNA virus in 1991 at the Central Veterinary Institute in the Netherlands. Classification in the *Arteriviridae* family has been proposed. Other viruses included in this new virus family include lactate dehydrogenase elevating virus (LDV), equine arteritis virus (EAV), and simian hemorrhagic fever virus (SHFV). Numerous strains of

PRRSV have been reported within the United States and Europe.

Clinical Disease--Breeding Herd

Production losses and clinical signs will vary considerably between farms depend on the strain of virus, management, and the herd immune status. Disease in a naive herd exposed to a virulent strain can be devastating. Clinical signs may include anorexia, fever, and lethargy in sows or gilts for one to seven days. Cyanotic (blue) ears, vulva, tails, abdomens, and snouts are most often seen with infection caused by European strains. Reproductive failure is characterized by late-term abortions, increased numbers of stillborn fetuses, and/or premature weak pigs. Infection during mid-gestation may be followed by abortions, mummified fetuses, early embryonic death, and infertility. Acute phases of disease typically last two to three months after which reproductive parameters often return to normal. A persistent reproductive form exhibits decreases in the farrowing rate and irregular returns to estrus.

The enzootically infected breeding herd may appear normal on clinical evaluation. Cycles of high conception failure (15-20 percent), stillbirths (10-15 percent), and preweaning mortality (15-20 percent) are common. Chronic PRRS problems are typical in seropositive herds which add seronegative, susceptible animals without proper isolation and acclimatization.

Clinical Disease--Growing Pigs

Piglets can be infected in *utero* and be born weak. This is often associated with premature farrowing. Infected litters are weak, unthrifty, or splay-legged. Signs in congenitally infected pigs may manifest as muscle tremors, eyelid edema, conjunctivitis and/or fever. Pigs may display respiratory dyspnea ("thumping"). Increasing incidence of bruising and hemorrhaging during processing has been reported. Preweaning mortality has been reported as high as 80 percent.

Clinical signs in 3- to 10-week old pigs are quite variable. Many seropositive herds have no clinical signs of PRRS in growing pigs, particularly when weaned early and strict all-in-all-out management is adhered to. Most consistent clinical signs observed are associated with respiratory disease or ill-thrift. Concurrent infections in nursery/grower pigs are often caused by *Salmonella choleraesuis, Streptococcus suis, Haemophilus parasuis, Actinobacillus pleuropneumoniae*, swine influenza virus, porcine respiratory coronavirus, and pseudorabies virus.

Continuous-flow facilities typically have substantial losses due to respiratory infection and subsequent increases in poor-doing animals.

Prevalence and Transmission

Prevalence of PRRSV in the United States swine herds is estimated at 60-80 percent. Direct contact is the most prominent route of exposure. Persistence of virus in the respiratory tract and lymphoid tissues, predominantly tonsil, may occur for up to 157 days post infection. This is true for at least some strains of PRRSV. Further investigation into other strains is needed to provide a firm grasp of persistence of the virus. Logical means of transmission include movement of the infected pigs into a naive unit and local airborne spread. Constant cycling of the PRRSV from older viremic pigs to incoming younger naive pigs will likely occur in all continuous-flow facilities.

One strain of PRRSV has been detected in semen with a polymerase chain reaction (PCR) test up to 92 days post-infection. Fresh semen from an infected boar used to inseminate gilts was shown to cause infection and failure to conceive. Alternately, extended semen from an infected boar did not cause

infection of gilts. This may be due to dilution of the virus in a higher volume of fluid.

Diagnosis

Diagnosis of PRRSV infection can be hindered by many factors. Factors include: lack of characteristic clinical signs, poor tissue preservation, inappropriate sample selection, and concurrent disease which may mask symptoms of infection. A further hindrance to diagnosis of disease caused by PRRSV is vaccination. The modified-live vaccine available at this time cannot be differentiated from wild-type virus by diagnostic tests now used.

Serum is the diagnostic specimen of choice in most cases. This is particularly true in instances of suspected reproductive forms of the disease. Paired samples obtained from sows as well as randomly selected samples from nursery and feeder pigs should be submitted to a laboratory for serological evaluation and/or virus isolation. Thoracic fluid from fresh aborted or stillborn fetuses may also be useful. Fresh and formalinized tissue samples for histopathology, immunohistochemistry, and virus isolation include: serum, tonsil, bronchial lymph nodes, lung, heart, and other tissues indicated by gross necropsy observations. Isolation of PRRSV is typically done with MARC-145 cells available at diagnostic laboratories nationally. It is important to use only fresh tissues that have been chilled (NOT FROZEN) as the virus rapidly loses infectivity at room temperatures and above.

A number of serological assays have been developed including serum neutralization and enzyme immunosorbent assays (ELISA) for detection of antibodies. ELISAs are being used in most diagnostic laboratories at this time to identify positive animals.

Prevention, Management, and Control

Prevention of PRRS in a naive herd depends on the ability to prevent introduction of infected animals. This is a challenge considering the number of routes of transmission possible. Primary transmission is via direct contact but aerosol and fomite transmission are possible. Pigs can be nonclinical carriers for several months. Since there are multiple strains of the PRRSV, serological tests must be correctly selected. The ELISA will identify all known U.S. and most European/international strains.

A procedure consisting of a strict quarantine schedule is recommended. Minimum quarantine time is 45 to 60 days. Sentinel animals should be exposed to the quarantine area for detection of disease and acclimation of the new pigs. Serological testing must be done on the new animals and sentinels. If all tests remain negative after the 60-day quarantine, risk of PRRS introduction is reduced.

To control disease associated with PRRSV the suggested protocols have included: stopping feedback of tissues and feces from the farrowing house to the sow herd, stopping cross-fostering after 24 hours of age, strict all-in-all-out pig movement, elimination of weak-born pigs, destruction of small poor-doing pigs at all stages of production, and halting reverse movement of pigs through the production chain (i.e. grower to nursery or nursery to farrowing).

Basic biosecurity measures should be strictly enforced. They include:

- Strict all-in-all-out production.
- Thorough washing and disinfection procedures.
- Limit all visitors.
- Provide boots and coveralls to visitors.
- Employees should not own or work with other swine herds.

- Changing boots and clothes after marketing hogs.
- Eliminating stray animals and rodents.
- Appropriate disposal of placentas, fetuses, and dead piglets.
- All transport vehicles washed and disinfected routinely.

Other means of prevention and control include:

- Two- or three-site production
- Segregated early weaning
- Nursery Depopulation
- Medicated early weaning

Control strategies must be tailored to individual farms. Strategies must be based on epidemiology of the virus, facilities available, pathogenicity of virus strain involved, and management resources. Close consultation with an attending veterinarian familiar with your herd and PRRS management is essential.

Treatment

Treatment consists of supportive therapy and antimicrobials for secondary infections such as pneumonia (Haemophilus, Pasteurella, Streptococcus, Salmonella, respiratory coronavirus), rhinitis (Bordetella, Pasteurella), diarrhea (Salmonella, *E. coli*), and Chlamydia. Farm-specific diagnostics should be done to select an appropriate antimicrobial therapy.

Vaccination

One modified-live PRRSV vaccine is currently available. It is licensed for use in three to 16 week-old pigs. The vaccine is widely used by veterinarians in all ages of swine in an off-label capacity. Protocols vary by farm. Recommendations of the attending veterinarian should be strictly followed due to potential risk incurred by vaccines use in breeding stock. Research reports also indicate that limited cross protection between PRRSV strains exist. Benefits and risks of vaccine use should be discussed with a veterinarian.

Conclusion

New and emerging infectious disease should make us reevaluate our management practices. PRRSV is a reminder that farm management protocols are more effective than the best vaccine or antimicrobial.

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