Introduction

In 1987, a new reproductive and respiratory disease of pigs was recognized in the United States. A specific causal agent could not be identified at the time, so the syndrome was called “Mystery Swine Disease.” The disease appeared in Europe in 1990 and investigators in the Netherlands succeeded in isolating a previously unrecognized virus in 1991. European workers introduced the term “porcine reproductive and respiratory syndrome” (PRRS), but because of its taxonomic classification the agent is also sometimes identified as “swine arterivirus.” At present, PRRS is the most economically important viral disease of swine in North America, with production costs to pig producers in the United States estimated at $560 million annually. The cost of the disease has led to the intensification of efforts to control and/or eliminate the virus from domestic swine herds.

Objectives

- Review the cause, clinical signs and pathology
- Explain techniques of diagnosis
- Describe methods for immunity, control and eradication

The cause

PRRS virus is an enveloped RNA virus classified in the family Arteriviridae within the order Nidovirales. Continual genetic mutation and recombination, a requirement for replication in macrophages, and persistent infection (carrier animals) despite an active immune response are characteristics shared by viruses in this family. These characteristics drive the clinical expression and epidemiology of the virus. The presence of an “envelope” on the exterior of PRRS virus is important because it increases the virus’ susceptibility to inactivation by quaternary ammonium, chlorine-based, and gluteraldehyde-based disinfectants.

As a consequence of mutation and recombination, new genetic variants of the PRRS virus appear frequently. Constant genetic change in PRRS virus may explain: 1) the wide variation in clinical disease observed in the field; 2) why prior exposure to one PRRS virus variant may not provide protective immunity against other variants; and 3) why certain vaccines may not be protective in some herds or under certain circumstances. Research has produced a great deal of information concerning the PRRS virus genome and viral proteins, but a genetic basis for the variation in virulence and antigenicity among PRRS virus isolates has not yet been identified.
One of the most important characteristics of PRRS virus is its requirement for replication in macrophages. Present in a variety of tissues, macrophages are specialized immune cells that play a key role in the pigs’ defense against infectious agents. Viruses that replicate in macrophages weaken the host’s immune response to secondary pathogens. This is why pigs infected with PRRS virus often have severe secondary infections with any of a variety of bacterial or viral pathogens including, Mycoplasma hyopneumonia, Salmonella choleraesuis, Haemophilus parasuis, Streptococcus suis, Pasteurella multocida, influenza virus, and others.

Another characteristic of viruses that infect macrophages is their ability to replicate in the host for an extended time despite an active immune response. Thus, PRRS virus-infected pigs are able to transmit the virus for several months.

In the environment, PRRS virus is inactivated by heat, drying, and low pH. The virus survives best in cold, moist environments, and almost indefinitely when frozen. PRRS virus remains infectious for 1 to 6 days at 68° F, 3 to 24 hours at 98° F and 6 to 20 minutes at 132° F. The ability of the virus to survive in cold temperatures may partially explain why the virus seems to be more easily transmitted in winter months in cold climates. Infectivity is rapidly lost when pH falls below 6 or rises above 7.5.

**Clinical signs**

PRRS exists in two distinct forms, reproductive and respiratory, and infected farms may experience one or both. A variety of factors, including swine genetics, the specific PRRS virus variants in the herd, health status of the herd, ages of the animals involved, and pregnancy status, may influence the clinical signs.

**Gilts and sows.** Gilts and sows infected with PRRS virus may show may show inappetence (off feed) and fever, or may show no clinical signs. “Rolling inappetence” and fever (104° F to105° F) are often observed and may involve up to 30% of the infected animals. During this acute stage, abortions may occur in 1 to 3% of pregnant animals, with abortion most common in females infected during the last trimester of pregnancy. An increase in the number of open sows, irregular returns to heat, and reduction in farrowing rate may be observed. Some PRRS virus variants are more virulent than others; highly virulent variants may cause abortion rates of 10 to 50% and mortality in up to 10% of Gilts and Sows. Signs of central nervous system involvement, including ataxia, circling, and falling to one side, may follow early abortions. In the acute infection enters a second phase approximately one week after the onset of clinical signs. As a result of PRRS virus infection of the fetuses, late-term reproductive failure occurs; both in pregnant females with and those without previous clinical signs. This phase typically lasts 1 to 4 months and causes 5 to 80% reproductive failure in pregnant females at 100 to 118 days of gestation. Most affected sows abort or farrow prematurely, but some farrow at full term. The number of premature farrowings may increase 1 to 4 weeks after the outbreak begins. These litters will contain variable numbers of normal, weak, and dead pigs in various stages of mummification. Over the course of this 1- to 4-month phase, the pattern of abnormal pigs in affected litters shifts. Initially, affected litters contain stillborn and large, partially mummified late-term fetuses. With time, this shifts to smaller, more completely mummified pigs; then to small, weak-born pigs; and finally, to pigs of normal size and vigor. Typically, 1 to 2% of litters are born dead. Gilts/sows infected with PRRS virus that progress through this four-stage disease process are difficult to rebreed. Often, these animals exhibit delayed return to estrus and low conception rates, resulting in depressed farrowing rates.

**Boars.** In general, few clinical signs are observed in boars infected with PRRS virus. If present, signs include fever, lethargy, inappetence, decreased libido, and occasionally, respiratory signs. Infrequently, boar deaths have been attributed to highly virulent PRRS virus variants. Some field and experimental studies report a decline in semen quality (reduced motility and proximal and distal cytoplasmic droplets) 2 to 10 weeks after infection. PRRS virus in semen from infected boars can be transmitted to susceptible females by natural mating or artificial insemination, so producers need to monitor sources of boars and semen for PRRS virus infection. The likelihood of transmission of PRRS virus via semen is dependent on the quantity of virus. As compared to intranasal inoculation, a 100 to 1000-times higher dose of PRRS virus is required to infect guilts by virus-contaminated semen. Of course, infected boars also shed virus in saliva and other secretions and excretions.
Suckling pigs. Symptoms of acute PRRS virus infection in pigs infected in utero or shortly after birth include severe dyspnea (labored breathing or thumping), lethargy, inappetence, fever, edema or swelling of the eyelids, and a blue or red discoloration of the ears or hindquarters. Pre-weaning mortality may approach 100%.

Weaned and grow-finish pigs. The most consistent clinical signs in weaned and grow-finish pigs include inappetence, lethargy, dyspnea (labored breathing and thumping), and blue or red discoloration of the skin on the ears or hindquarters. Many of these pigs are visibly unthrifty and have rough hair coats. Commonly, the presence of secondary bacterial and viral infections increases the severity of the clinical disease, i.e., porcine respiratory disease complex (PRDC). The economic impact of infection in these animals results from lower average daily gain and feed efficiency.

Pathogenesis and pathology

Lesions of PRRS virus most often occur in lung and lymphoid tissues where macrophages, the primary target of PRRS viral replication, are concentrated. The severity of lesions and clinical disease depends on the relative virulence of the PRRS virus involved. Thus, both clinical disease and lesions range from negligible to severe.

Infection of pregnant females with PRRS virus often causes them to be off feed for a few days, but is usually not fatal and gross lesions are usually mild or absent. PRRS virus crosses the placenta most efficiently in the third trimester of pregnancy and infects a proportion of fetuses. Fetuses may die, grow slowly in utero, or continue to thrive. Most PRRS virus-infected litters are aborted at over 100 days of gestation or are delivered at full-term. Infected litters are composed of variable proportions of 1) normal, live, uninfected pigs; 2) live, normal appearing PRRS virus-infected pigs; 3) live, weak, often small (the size of a small rat or larger) virus-infected pigs; and 4) dead pigs varying in size and the degree of sterile in utero autolysis. In fetuses that died recently, autolysis may appear as dark red-to-brown discoloration. Fetal pigs that have been dead longer will be partially decomposed or partially dehydrated with reddish discoloration of organs and reddish fluid in body cavities. Most PRRS virus-infected fetuses or new-born pigs lack lesions specific for PRRS virus infection. A few fetuses or live-born pigs may have segmental hemorrhagic enlargement of the umbilical cord, but this must be differentiated from trauma caused by sows stepping on the umbilical cord. Some fetuses have clear-to-amber fluid around abdominal organs and in body cavities. Neonatal PRRS virus-infected pigs may have swelling of eyelids or tissues around the eyes, as well as pneumonia similar to that more commonly seen in post-natal infections. Microscopic lesions may be observed in umbilical cord, lung, heart, kidney, liver and brain, but they are non-specific and non-diagnostic.

Infection of young pigs most often occurs when maternal antibody wanes in nursery-age pigs or when pigs from multiple sources are mixed in the grower-finisher. Lesions are the same in all ages. Pneumonia is observed from 3 to 28 days after infection and is most severe at 10 days post infection. Lungs are slightly firm and non-collapsing (like a wet sponge) and are mottled grey-tan in color, rather than the normal pink. Lesions may affect the entire lung, but are often most severe in the lower front portions. Secondary bacterial pneumonia is often present in PRRS virus-damaged lungs, complicating the lesions and making interpretation of the pathology more difficult. Many lymph nodes are 2 to 10 times normal size from 4 to 28 days after infection. Enlarged nodes are moist, semi-soft to firm, and on cut-surface examination are tan to white. Microscopic lesions are commonly observed in lungs and lymph nodes; less commonly in the heart, brain and gravid uterus; and infrequently in the kidney, nasal mucosa and stomach. Gross and microscopic lesions in post-natal pigs are suggestive of PRRS virus infection, but are not sufficient for confirming a diagnosis.

Diagnosis

PRRS virus should be included in the differential diagnosis when a herd shows clinical signs of respiratory disease at any production stage, respiratory signs in conjunction with reproductive failure, or when herd performance is suboptimal. Analysis of herd production records can reveal changes in production parameters suggestive of active PRRS virus infection: an increase in abortions or early farrowings, decreased numbers of live-born pigs, increased preweaning mortality, reduced fertility, and increased nonproductive sow days, including increased and delayed returns to estrus. Many PRRS virus variants
produce mild or subclinical disease and infection can only be detected through laboratory tests. Therefore, a definitive diagnosis of PRRS requires integrating clinical history, production record data, clinical signs, and laboratory test results.

The laboratory diagnosis of PRRS virus infection relies on one or more of the following: 1) the demonstration of infectious virus by virus isolation; 2) evidence of viral replication in cells by detection of viral antigens using immunofluorescence or immunohistochemistry; 3) evidence of PRRS virus-specific antibodies; and 4) evidence of the viral nucleic acid. As noted in the previous section, the presence of gross or microscopic lesions in certain tissues may contribute to the diagnosis, but are frequently non-specific and non-diagnostic.

Diagnostic tests. The most common method used to determine if a herd has become infected or to monitor infection in endemic-infected herds, is detection of serum antibodies using the commercial PRRS ELISA (enzyme-linked immunosorbent assay). It is a rapid and inexpensive method of detecting and monitoring of PRRS virus infections on a herd basis. The test detects the presence of antibodies in serum as early as 7 days after infection, but more commonly by 14 days. The ELISA reports results in the form of a sample-to-positive (S/P) ratio and S/P ratios of 0.4 or higher are considered positive.

The polymerase chain reaction (PCR) is a test that detects and amplifies a small, specific segment of the PRRS virus genome. PCR is more sensitive than virus isolation and results are available in a shorter period of time. Samples suitable for analysis by PCR include serum, semen, and tissue. Once collected, samples should be kept cool and shipped to the diagnostic laboratory over night on ice. Chilling (refrigeration) is preferable to freezing. DNA obtained from the PCR reaction can be sequenced to differentiate infection with field virus from modified-live virus vaccine.

Immunohistochemistry (IHC) can be used for directly visualizing PRRS virus antigens in tissues. IHC is less sensitive than other tests and lung, lymph nodes and tonsil are usually the preferred specimens because these tissues contain higher amounts of virus.

Samples. In respiratory cases in which PRRS virus is suspected, serum, lung, and lymphoid tissues from pigs with clinical signs or increased body temperatures should be submitted for testing. In cases of abortion, live weak-born pigs should be submitted, rather than stillborn and mummified fetuses, because the virus is rapidly inactivated in necrotic tissues. In suckling, weaned, or growing pigs, virus may be isolated from serum for 4 to 6 weeks post infection; 1 to 2 weeks in sows and boars. In adults, virus can usually be detected for an additional 2 to 4 weeks after infection in cells that have been washed from the lungs (lung lavage).

Early after infection, PRRS virus is present in most tissues of young pigs and sows. As the infection progresses, virus becomes limited to lymph nodes and tonsils. Tonsil scrapings and tonsil biopsies collected during the persistence phase of infection are excellent ante mortem samples for detecting PRRS virus in breeding animals. These samples can be used for virus isolation to demonstrate infectious virus and for detection of viral nucleic acid by PCR.

Diagnosis of PRRS virus in boars. Diagnosis of PRRS virus in boars is important because of the risk of transmission through semen and the frequency with which AI is used in breeding programs.

PCR has been used extensively to detect PRRS virus in semen. PCR amplifies the PRRS virus genome exponentially so that even small amounts of the virus can be detected. Traditional diagnostic techniques, such as virus isolation, are not performed on boar semen samples because semen is toxic to the cells on which the virus is grown. “Swine bioassay” has also been utilized to detect PRRS virus in semen, but is impractical outside of the research setting. Previous work comparing PCR and swine bioassay showed a high correlation between the two tests. That is, semen samples positive for viral nucleic acid via PCR contained infectious virus. Therefore, PCR positive semen samples should be considered highly likely to contain infectious virus and should not be used for artificial insemination. In time course studies, it has been demonstrated that boars may be viremic and shed PRRS virus in semen 1 to 2 weeks prior to the development of PRRS virus antibodies. During the acute stages of infection (1 to 2 weeks after exposure and prior to antibody development) a serum sample for virus isolation and/or PCR and semen for PCR testing should be submitted. Preferably, more than one semen sample should be submitted at different time periods (e.g., approximately one week apart) because PRRS virus is shed intermittently in the semen.
In the later stages of PRRS virus infection, most boars produce PCR-positive semen, but serum is PCR and virus isolation negative. By this time, however, the boar has developed serum antibodies so the ELISA test is positive. Importantly, the absence of infectious PRRS virus and/or nucleic acid in serum or semen does not mean that an infected boar has cleared the virus or is not shedding virus through other secretions. It is known that boars can carry virus in tonsils for extended periods of time and may shed virus in oropharyngeal secretions.

Due to the possible presence of infectious PRRS virus in boars that test PRRS virus negative in ante mortem samples (serum and semen), controlling the spread of PRRS virus in boars may be difficult. Quarantine and strict biosecurity procedures are very important in this regard. One field study in a PRRS virus seropositive boar study found that only 12 of 131 boars (9.1%) were positive for PRRS virus in semen by PCR when quarantine was 25 days or greater and only 7 of 440 boars (1.6%) were positive for PRRS virus in semen by PCR when quarantine was 45 days or greater. Thus, longer quarantine appears to reduce the number of boars shedding PRRS virus in semen. Since it is impossible to identify boars that are actively shedding PRRS virus on the basis of clinical signs, testing semen and serum for the presence of PRRSV is necessary. However, strict biosecurity and extended quarantine periods are equally important and should be used in combination with diagnostic testing.

**Epidemiology**

**Entry into domestic swine.** PRRS virus recently entered the domestic swine population and spread rapidly, thereafter. Based on the detection of PRRS virus-specific serum antibodies, it is known that the virus was present in domestic swine in Canada by 1979, in the United States (Iowa) by 1985, and in Europe (Germany) by 1987. The original source of the virus and the circumstances that led to its entry into the domestic swine population are not known. Feral swine were probably not the original source of the virus. In fact, the virus may have moved into feral swine from domestic swine, rather than the reverse. The question of the origin of the virus is important because of the possibility that the original source could continue to play a role in the epidemiology of the virus.

**Prevalence and geographic distribution.** PRRS virus is endemic in all major swine producing areas of the world. Countries considered free of PRRS virus (Argentina, Australia, Cuba, Finland, New Caledonia, New Zealand, Norway, Sweden, Switzerland) have relatively small swine stocks, use more traditional and/or less intensive methods of production, and conduct less trade in live animals and by-products of swine origin.

Accurate estimates of the prevalence of infection with wild-type virus in specific countries or regions are not generally available. Published surveys are usually limited in scope and/or not based on statistically valid population sampling procedures. A larger problem is the fact that, beginning in the mid-1990’s, the widespread use of non-differential vaccines limited the use of serologic assays for determining infection status. In the United States, the best prevalence estimates are based on a 1995 National Animal Health Monitoring System (NAHMS) study in which 8,038 serum samples were collected from 286 herds in 16 swine-producing states. Among unvaccinated herds and animals, 59% of 217 herds and 41% of 6,376 animals were infected. When animals were identified by status, 24% of unvaccinated breeding animals and 52% of unvaccinated finishers were considered infected on the basis of specific anti-PRRS virus antibodies. Likewise, reports from many parts of the world reinforce the impression that the infection is widespread and the prevalence of infection is high (50 to 100%) in swine-dense regions.

**Infection and shedding.** Swine can be infected with PRRS virus by oral, intranasal, intramuscular, or vaginal exposure, but the probability of transmission varies by route and dose. Transmission by parenteral exposure is particularly efficient. That is, exposure to 10 or fewer PRRS virus particles through breaks in the skin is sufficient to transmit infection. Contamination of instruments and medications with body fluids from PRRS virus-infected animals can result in transmission. For example, instruments used for ear notching, tail docking, teeth clipping, or tattooing, as well as needles, syringes, medications, and biologics. Likewise, bites, cuts, scrapes, and/or abrasions inflicted by infected pigs results in transmission. Relative to parenteral exposure, oral, intranasal, or intravaginal routes require much higher virus exposure dose to transmit PRRS virus.
Infected animals shed virus from multiple sites and for a long time. Virus has been detected in saliva at 42 days post infection (PI), nasal secretions at 21 days PI, urine at 28 days PI, semen at 92 days PI, tonsil at 157 days PI, and in the colostrum and milk of susceptible females infected in the last trimester of gestation. Following the acute phase of infection, virus is shed at low levels or perhaps is shed intermittently. For that reason, as discussed in the previous section, it is recommended that semen samples be checked at least twice before concluding that a boar is not shedding virus. Neither the absence of viremia nor serum antibody levels indicate whether an animal is shedding virus.

Shedding of virus by infected animals results in environmental contamination and creates the potential for transmission via fomites. PRRS virus is fragile and quickly inactivated by drying. In addition, the virus has a narrow range of tolerance in fluids and is quickly inactivated in solutions such as urine or fecal slurry. However, PRRS virus suspended in ‘clean’ aqueous solutions at neutral pH under cool-to-frozen conditions can remain infectious for a long time. For example, the half-life (inactivation of 1/2 of the virus population) in a pH 7.5 solution held at 39° F was estimated to be 5.8 days. On the farm, infectious virus may theoretically persist in clean standing water, i.e., in drinking cups, troughs, and puddles, for several days, depending on the ambient temperature. Standard cleaning, drying, and disinfection procedures are effective for inactivation of PRRS virus in facilities and on equipment. When implementing disinfection procedures, ambient temperature and its effect on virus inactivation should be taken into account.

Carrier animals. PRRS virus produces a chronic, persistent infection in pigs (carrier animals). That is, virus continues to replicates in infected individuals for several months. Virus can be recovered from approximately 85% of pigs at 100 days post infection. Beyond 100 days the data is sparse, but researchers have isolated virus from experimentally infected pigs 132 days post infection (University of South Dakota), 2 of 5 pigs at day 150 post-inoculation (University of Nebraska-Lincon), and one of 4 pigs at 157 days post-inoculation (Iowa State University). The primary sites of PRRS virus persistence are lymph nodes and tonsil. Virus has been isolated from ‘tonsil scraping’ samples for up to 157 days after infection and viral nucleic acid (PCR) has been demonstrated in tonsil tissue up to 255 days post inoculation. Even so, pigs do not appear to be persistently infected for life and most animals are believed to eventually clear the infection.

Persistence is the single most significant epidemiological feature of PRRS virus. Carrier animals represent the constant threat of transmission to susceptible herd mates and the initiation of a PRRS outbreak. At present, we do not have the technology to accurately, rapidly, and cheaply identify carriers. Neither the absence of viremia nor serum antibody levels are indicators of carrier status. In fact, some carrier animals have low serum antibody levels, e.g., <0.40 S/P on the commercial ELISA. Thus, the existence of carrier animals profoundly complicate all aspects of PRRS prevention and control.

Transmission between pigs. Transmission most commonly occurs by close contact between pigs or by exposure to contaminated body fluids (semen, virus-contaminated blood, secretions, contaminated needles, coveralls, and boots). Social behavior and pig-to-pig interactions are important in direct transmission, particular the agonistic behavior associated with establishing social order. Typically, such behavior involves scrapes or bites in the shoulders, neck, and head and results in the exchange of blood and saliva. If contaminated with virus, such exchanges result in infection. Other behaviors that results in exchange of blood and saliva, i.e., tail-biting and ear-biting, may also function in transmission.

Transmission within herds. Once infected, PRRS virus tends to circulate within a herd indefinitely. Spontaneous elimination of PRRS virus from commercial herds has been reported, but rarely. The virus is perpetuated by transmission from carrier animals to susceptible animals introduced into the herd through birth or purchase. Maternal antibodies may provide some immunologic resistance to infection in neonatal pigs, but the protection is incomplete and of short duration. Under conditions in which susceptible and infectious pigs are mixed, such as at weaning, a large proportion of the population may quickly become infected. In some cases, pigs escape infection for an extended period of time, even to the extent that seroconversion has been reported in young sows on farms using in-herd gilt replacements. The presence of subpopulations of susceptible gilts or sows in endemically infected breeding herds partially explains periodic outbreaks of PRRS.

Transmission between herds. Herd-to-herd transmission occurs, but is frequently recognized too long after the fact to make it possible to accurately determine the source of the virus. The primary source of herd-to-herd transmission is the introduction of infected animals. Following outbreaks of PRRS in late 1990,
Dr. Scott Dee (University of Minnesota) reported that of 10 farms surveyed, 8 had purchased breeding stock from the same source. In France, it was reported that 56% of herds acquired PRRS virus through introduction of infected pigs, 20% through contaminated semen, 21% through contaminated fomites, and 3% through unidentified sources.

'Area spread,' meaning transmission in the absence of any apparent animal or human source, occurs relatively frequently in swine dense areas. In France, 45% of herds infected through area spread were located within 500 meters (0.3 miles) of an infected herd and only 2% were 1 kilometer (0.6 miles) or more from the initial outbreak. The mechanism(s) of area spread are not clearly identified. Airborne virus was once thought to be the primary means of area spread, but extensive work with aerosols has not substantiated the role of aerosol transmission. Flies and mosquitoes have been shown to transmit PRRS virus under experimental conditions, but whether insects move PRRS virus between herds in the field requires additional evidence. Because of its importance in the regional control of PRRS, area spread is currently an area of active investigation.

Transmission by non-porcine species. The role of non-porcine species in the epidemiology of PRRS virus is uncertain. Studies indicate that dogs, cats, skunks, raccoons, opossums, rats, mice, guinea pigs, house sparrows, and starlings are not susceptible to infection. Conflicting evidence exists regarding the susceptibility of avian species to PRRS virus. PRRS virus has been recovered from mosquitoes and house flies captured in swine facilities, but the role of insects in the epidemiology of PRRS virus remains to be determined. Overall, infected pigs and virus-contaminated semen are the primary sources for introduction of PRRS virus into herds.

Immunity - how are pigs protected against PRRS virus infections?

Protective immunity to PRRS virus is a complex and unresolved issue. It is clear that PRRS virus is able to modulate the immune response in such a way that conventional antibody and cell mediated responses are not sufficient to eliminate the virus.

Infection with PRRS virus progresses from acute, to persistent, to convalescent and most pigs eventually clear the virus. However, the mechanism(s) for elimination of PRRS virus by the pig is unknown. Previously, it was believed that neutralizing antibodies (humoral immunity) eliminated PRRS virus viremia during acute infection and that these antibodies, in concert with interferon gamma, eventually cleared persistent PRRS virus infection. However, recent studies suggest that neutralizing antibody is not the primary factor acting to eliminate viremia. The persistence of virus in tonsils and lymph nodes also argues against the effectiveness of neutralizing antibodies in eliminating the virus. Paradoxically, there is also no correlation between T-cell responses (cell-mediated immunity) and the resolution of acute and persistent infections. Even so, it is recognized that sufficiently high levels of serum neutralizing antibodies can prevent infection and/or transplacental infection. This may partially explain why vaccination or prior infection is successful in reducing abortions and preventing infection of fetuses in the field.

Overall, these observations suggest that protection against PRRS virus may be related to something other than immunity. One possibility is that the infection is eliminated because macrophages become less permissive to PRRS virus. Under this hypothetical scenario, the immune response may actually be secondary in helping to eliminate the virus. While it is known that pigs resist future infection with homologous strains of PRRS virus, there is much debate on whether this protection extends to all strains of PRRS virus. Overall, significant challenges remain in understanding immunity and developing second generation vaccines to this virus.

Control and eradication

A variety of management strategies have been used to reduce clinical PRRS or stop the circulation of virus in susceptible populations of pigs. Constant genetic change in the virus combined with a long-term carrier state and ambivalent immunity in the pig frequently confound attempts to control or eradication PRRS. Genetically diverse variants of PRRS virus often co-exist within the same farm and complicate the picture. These factors work together to complicate long-term control measures. The lack of consistent success in controlling PRRS and the costs of the disease has led the U.S. industry to consider eradication of the disease as a possible solution.
The most important component of a control program is the proper use of diagnostic tests to accurately determine the pattern of virus transmission within an infected production system. Serum samples for serologic profiling should be collected from the breeding herd across all parities, recently weaned pigs, 8- to 10-week old nursery pigs, and 5- to 6-month old finishers. Testing by parity may indicate whether programs need to focus on a specific subpopulation within the herd, such as gilts. It is important to use clinical observations and production records in conjunction with diagnostic data to understand the status of the sampled population. Once the pattern of virus spread and the age in which the pig is infected are determined, intervention strategies can be initiated. Additional and extensive information on PRRS control and eradication may be found in The 2003 PRRS Compendium (2nd edition) available through the National Pork Board (Des Moines, Iowa 50306 or www.porkboard.org).

Methods of achieving controlled PRRS virus infection are frequently based on increasing the level of herd immunity through vaccination, exposure of susceptible animals to infectious animals, or exposure of susceptible animals to virus-contaminated material collected from infected animals.

Gilt development. Gilt development is an important component of a PRRS control program. Direct introduction of either susceptible or actively infected replacement stock into the breeding herd will result in clinical PRRS. Therefore, a gilt development program should be established to properly prepare replacement stock for entry into the breeding herd. Essentially, gilt development consists of quarantine and acclimatization periods. Gilt development strategies may involve the introduction of gilts as weaners or 25kg piglets, housing them in designated developer facilities, and establishing vaccination and/or acclimation protocols to equilibrate the health level of this population with the existing breeding herd.

If PRRS virus positive sources are employed, it is important to ensure that a change in source does not occur and that actively infected animals do not directly enter the recipient herd. Generally, quarantine periods of 90 days or more are considered adequate. Quarantine facilities should function on an all in-all out basis. The purchase of gilts from PRRS virus-negative sources increases the importance of establishing immunity to PRRS virus prior to herd entry. Several approaches are used:

1. Transmission of PRRS virus to gilts in development by natural exposure to cull sows and/or infected nursery pigs is usually inconsistent, particularly in chronically infected herds, because of lack of active circulation of field virus.
2. The use of vaccination in gilt developer protocols is one approach to reducing the risk of developing PRRS virus-naïve subpopulations. Commercially available PRRS vaccines include modified-live virus (MLV) and inactivated virus (killed) products. Duration of immunity studies indicate that modified-live preparations are protective for up to at least 4 months. Diagnostic profiling is critical when vaccination is employed to avoid immunizing pigs concurrently infected with field virus. Farm-specific vaccination programs should be developed in collaboration with the herd veterinarian and based on individual farm diagnostic data, rather than standardized protocols.
3. “Planned exposure” involves inoculating susceptible animals with virus-contaminated material collected from infected animals in the herd. Most frequently, serum from PRRS virus-infected young pigs is used as the source of the virus. The theory behind this approach is that exposure to the PRRS virus variant(s) endemic in the herd provides the maximum protection against clinical disease. Planned exposure may possess some value for use in gilt acclimatization protocols. In all cases, the risks inherent in planned exposure should be recognized and discussed. Its use in pregnant sows can promote episodes of third trimester abortion, elevated pre-weaning mortality, and lactating sow mortality.

Nursery depopulation. Nursery depopulation consists of emptying all nursery rooms, washing and disinfecting, and allowing the facility to remain empty for 1 to 2 days. This strategy also can be applied to PRRS virus-infected finishing populations. Nursery depopulation (ND) is a cost-effective means to interrupt transmission of PRRS virus from older, infected pigs to those recently weaned. Significant improvements in nursery pig daily gain, mortality, treatment cost, and profitability have been observed following implementation of ND. Despite such improvement, re-infection of nurseries with PRRS virus is a frequent event, often taking place 12 to 18 months following completion of the depopulation protocol. Therefore, it has been speculated that the primary benefit of this strategy may be for the control of opportunistic pathogens, rather than PRRS virus.

McREBEL. McREBEL stands for “Management Changes to Reduce Exposure to Bacteria to Eliminate Losses” and consists of a series of steps to reduce the spread of PRRS virus among piglets. McREBEL has
been most frequently applied to herds undergoing acute outbreaks. The advantages of the protocol are its simplicity and low cost. McREBEL has not yet been quantitatively evaluated over a wide range of farms. In some cases, producers and/or employees may resist the elimination of cross fostering and the euthanasia of poor-doing piglets.

**Eradication.** Elimination of PRRS virus is possible through a number of methods, including whole herd depopulation-repopulation, test-and-removal and herd closure. All methods are of equal efficacy in eliminating the current PRRS virus variant from the infected population; however, re-infection with a unrelated variant is a frequent event. Therefore, the implementation of strict biosecurity measures based on reported routes of PRRS virus transmission is essential.

**Summary**

Steps are being made in the understanding and control of PRRS. The ability of PRRS to produce variants and remain as a persistent infection makes total eradication extremely difficult. However, temporary eradication is possible by implementing management procedures such as whole herd depopulation-repopulation, test-and-removal, and herd closure. The use of diagnostic tests to accurately determine the pattern of virus transmission within an infected production system may be the best method currently available for combating PRRS. Though a great deal of knowledge has been gained from research done to date, much more research is required to conclusively understand the mechanisms by which PRRS can be controlled.

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Additional and extensive information on PRRS may be found in The 2003 PRRS Compendium (2nd edition) available through the National Pork Board (Des Moines, Iowa 50306 or www.porkboard.org)

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