The severity of PRRS virus infection can vary widely and range from a near complete lack of clinical signs to devastating outbreaks of reproductive and respiratory disease.

PRRS virus infection occurs mainly in a cell of the immune system called a macrophage. Macrophages are found throughout many tissues in the body.

Clinical signs in the breeding herd may include anorexia (off-feed), fever, lethargy, nervous signs, purplish discoloration of the ears and vulva, and abortion. Litters born to recently infected dams may have increased rates of mummification, still births, and weak-born piglets. Piglets in these litters may carry the virus for an extended period.

Boars infected with PRRS virus can show similar signs as sows. Infection does not usually impact the fertility of the semen but virus can be found in the ejaculate of boars for several months after being infected.

Newborn piglets infected with the virus can demonstrate severe respiratory disease. Nervous signs as well as anorexia and lethargy have also been reported. Other common diseases of young pigs may become more prevalent and severe during a PRRS outbreak.

In an experimental setting, piglets infected with PRRS virus often show minimal clinical signs. However, in a field setting, PRRS virus infection frequently has a more pronounced effect due to its interaction with the pig’s environment, immune status, and concurrent diseases.
PRRS Virus Infection and Disease
B Thacker

Introduction

The severity of disease and clinical manifestations of PRRS virus infection vary widely. Subclinical infections can occur in a herd over a long period of time based on field observations of herds that remain clinically normal despite ample serological evidence that PRRS virus (PRRSV) circulation occurs continuously (B Thacker, personal observations). On the other hand, the upper limits of severity can be extreme. For example, some outbreaks that occurred in 1996 were initially considered so severe that other agents beside PRRS virus, or "super virulent" strains of PRRS virus, were suspected to be the cause (Hurd et al., 2001).

This marked variation in the severity of PRRS in a herd can be explained by several factors. These factors include PRRS virus strain variation, the presence of other pathogens, age of the pig at the time of infection, stage of reproduction at the time of infection, level of immunity in the herd, herd size, housing, and environment. This paper will describe the clinical manifestations of PRRS virus induced disease by animal category and/or phase of production.

PRRS virus infection occurs mainly in a cell called a macrophage. Macrophages are widespread throughout all tissues. Accordingly, systemic disease resulting in fever and reduced appetite is obvious after infection with virulent strains. Additionally, the infection of macrophages found in the lungs results in respiratory disease signs manifested primarily as elevated respiration rates and a pronounced breathing pattern. Cough is not a prominent feature of uncomplicated PRRS virus infection. Infection of developing embryos and fetuses results in a full range of reproductive problems in pregnant females (unexpected returns to estrus, abortion) along with the expected symptoms of a generalized, systemic disease (fever, anorexia, lethargy). Infection in boars can result in abnormal semen and shedding of virus in semen.

Characterization of Infection and Disease

Unfortunately for the clinician, none of the clinical signs attributed to PRRS are specific only to infection with PRRS virus. Understanding the clinical severity of PRRS within an infected herd is confusing at best, and a nightmare at worst. The challenge for the clinician is determining the contribution of PRRSV infection to poor herd performance when numerous other stressors are known to already exist on the farm. In most infected herds, PRRS virus will continue to circulate until a concerted effort is made to eliminate the virus from the herd (Joo and Dee, 1993).

The severity of an outbreak on farms previously infected (or vaccinated) with PRRSV can be similar to herds that were previously naïve. This was clearly illustrated by the severe disease observed in previously infected herds involved in the "atypical" or "acute PRRS" outbreaks in late 1996 (to be discussed in more detail later in this chapter). To the contrary, evidence suggests that asymptomatic infections occur in breeding herds. This is based on the absence of obvious clinical disease and reproductive failure in adult animals, or infection of the offspring prior to weaning, when serum antibody levels suggest that the herd was recently infected.

In a laboratory setting, nursery pigs infected with a moderately virulent PRRS virus causes respiratory disease that resolves within 28 days - if no other pathogens are present and under ideal environmental and animal care conditions. With regard to the persistence of PRRSV infections, determining a fixed endpoint where infected animals no longer shed virus has been elusive. Viral shedding following experimental inoculation of young pigs has been observed for at least 157 days after challenge (Wills et al., 1997). Latency, the potential for an infectious virus to be carried by a pig not showing any signs of the disease (as seen with pseudorabies), has not been demonstrated with PRRS virus. However, fetuses that survive being infected in late term gestation (80 to 90 days of gestation) have been reported to be persistently infected. Piglets born under these conditions can carry the virus in their bloodstream for up to 11 weeks and intermittently shed virus for up to 30 weeks.

Clinical Disease in the Breeding Herd - Sows and Gilts

Clinical signs most frequently observed in adult animals include anorexia, fever, and lethargy (Keffaber, 1989; Loula, 1991). Occasionally,
subcutaneous and hind limb edema, nervous signs, and skin lesions, such as purplish discoloration of the ears and vulva, are observed (Hopper et al., 1992, Rosow et al, 1998). Diseases with similar presentations include pseudorabies (PRV) and swine influenza virus (SIV) infections. Unlike viral diseases such as PRV, SIV, and transmissible gastroenteritis (TGE) virus, which typically spread rapidly through the herd so that the onset of clinical disease is relatively synchronous in the population, PRRS virus can move slowly through the herd. This slow spread can result in a “rolling” or periodic anorexia that persists for several days to several weeks on a herd basis (B Thacker, 1992). PRRS virus infection in adults can be fatal, especially in late-term gestating females.

The slow progression of the disease on some farms has created some confusion regarding the effects of PRRS virus at various stages of gestation. Many of the clinical signs of reproductive failure are not specific to one stage of gestation. Initially, the main effect of PRRS virus-induced reproductive failure was believed to be late-term abortions (Cromwijk, 1991; Hill, 1990). Early experimental studies confirmed that reproductive failure was relatively easy to induce in late pregnancy while early pregnancies were relatively resistant. However, in many severe field outbreaks, reproductive failure occurred regardless of the stage of gestation and later studies were able to demonstrate reproductive failure in early gestation. Accordingly, PRRS virus-induced reproductive failure can present clinically as increased regular and delayed returns to estrus, not-in-pig sows, abortions, mummified fetuses, stillbirths, and weak-born pigs. The increased rate of regular returns to estrus may be attributable to reduced fertility in boars following PRRS virus infection. Insemination of naïve gilts with PRRS virus-contaminated, but otherwise fertile semen, had little impact on fertility although the gilts did become infected (Lager et al., 1997). Mean-to-estrus intervals can be prolonged and the intensity of estrus can be reduced when clinical disease occurs in sows during and after lactation. Cycling of gilts can be delayed or disrupted, as well. Reduction in feed intake due to generalized illness is most likely responsible for these outcomes.

Clinical Disease in the Breeding Herd - Boars

Boars can show generalized clinical disease similar to sows, although the severity and the percentage of boars exhibiting clinical disease may be lower than sows. The impact of infection in boars on semen quality and mating ability is highly variable. Several studies have demonstrated increased sperm abnormalities, while several other studies found no influence on semen quality. Of particular importance is the finding that experimental infection of boars can induce sperm abnormalities even though the boars exhibit minimal clinical signs.

Fietsma et al. (1992) reported their observations on field infection in five artificial insemination centers. Out of 230 boars, approximately 25 percent exhibited reduced appetite, fever, and in some cases, diminished libido, with recovery in one week. Sperm counts were not affected in any of the boars, but motility was reduced in boars that exhibited clinical disease. After experimental challenge of four boars, Swenson et al. (1995) observed mild respiratory signs (sneezing and coughing) for one day, but appetite, behavior, libido, and semen quality remained normal.

The impact of PRRS virus on semen quality is highly variable and may or may not play a significant role in PRRS virus-induced reproductive failure. The influence of genetics on the severity of PRRS virus infection in boars has been studied but the low number of boars evaluated precluded any meaningful statistical analysis of the data. In that study, Landrace, Yorkshire, and Hampshire boars were evaluated and the data suggested that Yorkshire boars were more resistant to shedding PRRS virus in semen compared to Landrace boars (Christopher-Hennings et al., 2001). Contamination of semen with PRRS virus following infection or immunization with modified live vaccine is a common event (Swenson et al., 1994). The duration of shedding in semen however, can be quite variable. The maximum length of virus shedding has been reported to be 92 days (Christopher-Hennings et al., 1995).

In the female, the reproductive consequences of receiving virus-contaminated semen are minimal, providing semen quality is acceptable. Several studies have been unable to demonstrate reduced fertilization or conception rates following insemination of virus contaminated semen (Lager et al., 1997; Prieto et al., 1997). Of more importance is the likelihood of transmitting PRRS virus to the recipient female resulting in clinical disease in that animal and subsequent spread to other animals in the herd (Prieto et al., 1997; Yaeger et al., 1993). Introduction of PRRS virus-contaminated semen is a constant threat to herd biosecurity.
Clinical Disease in Lactating Sows and Neonates

Clinical signs of PRRS virus infection observed in neonates during acute outbreaks can be quite remarkable (Keffaber, 1989; Loula, 1991). The first sign is often severe respiratory disease in pigs less than two weeks of age. The respiratory rate becomes markedly elevated and the depth of respiration increases to the point where each breath is evident by the pronounced movement of the chest and abdomen. Severely affected pigs will exhibit open-mouth breathing and the respiration will become so rapid as to make it impossible to accurately determine the respiratory rate. Central nervous signs, including drowsiness and anorexia, along with corresponding microscopic lesions in the brain were reported by Rossow et al. (1999). Other clinical signs reported in neonates include edema around the eyes, conjunctivitis, blue discoloration of ears, bruising of the skin, diarrhea, shaking, rough hair coats and profuse bleeding post-injection (Rossow, 1998). Whether some of these clinical signs, such as diarrhea, are directly attributable to PRRS virus infection is debatable and may be due other secondary infections.

Simultaneously, or within a few days of the appearance of disease in neonates, weak-born pigs and stillbirths become more frequent. Weak-born pigs fail to move beyond the rear of the sow and often die within a few hours. The rate of stillborn pigs can reach 75 percent. Often as an outbreak progresses and dead fetuses have more time to undergo the process of mummification, the rate of stillbirths declines and the rate of mummified fetuses increases. Finally, small litters may be observed due to embryonic death during early pregnancy.

The impact on the health status of lactating sows is similar to gestating sows. Loss of appetite and fever leads to agalactia, which results in starvation of the piglets or development of diseases that are controlled by maternal immunity such as colibacillosis. In total, preweaning mortality rates can exceed 80 percent in severe cases (Christianson et al., 1991).

Clinical Disease from Weaning to Market

As with the breeding herd, the impact of PRRS virus infections in pigs after weaning can be highly variable. Pig age influences the severity of disease (Rossow et al., 1994). In the experience of this author, it appears that 8 weeks of age is an important break point with regard to the severity of disease induced by experimental challenge. In the field, most clinicians would agree that younger pigs tend to develop more severe disease, although in an individual herd, the timing of infections with other pathogens will influence disease severity as well. PRRS virus infections of weaned pigs can persist within a herd essentially forever because each new group of young pigs is susceptible to infection following the decay of Colostral immunity (Dee et al., 1997). Infection during pregnancy can sometimes occur without any obvious harm to the pig. Infection with PRRS virus alone can markedly reduce growth rates, although mortality rates are usually only mildly elevated unless other pathogens are present. In field cases, reduced growth rates are frequently observed and increased rates of cull or light pigs occur (Keffaber, 1989). Nursery daily gains can be reduced by 50 to 75 percent and mortality rates can rise to 10 to 25 percent in field situations (Keffaber, 1989). In a recent study, Regula et al. (2000) reported that finishing pigs that seroconverted to PRRS virus gained 40 grams per day less than pigs that did not seroconvert.

Experimentally, infection of high health status pigs results in no, or slight, death loss and a 25 to 40 percent reduction in daily gain in the first 28 days following experimental challenge (Thacker et al., 1999). Clinical signs include fever (>40°C), anorexia, and an increased respiration rate that is especially pronounced after handling the pigs for rectal temperature assessment or blood collection.

As stated above, the earlier in life that a pig becomes infected with PRRS virus, the more severe the clinical outcome will be. This finding is corroborated by field experiences that suggest avoiding circulation of PRRS virus in nurseries appears to be very important with respect to the overall performance of the finishing herd. Under field conditions, pigs are typically infected with other pathogens prior to, during, and/or after infection with PRRS virus. Depending on the relative timing of infection and duration of disease following infection, the severity of clinical signs will vary widely. Agents commonly associated with PPRS infection in the field, or vice versa, include porcine circovirus type 2, Mycoplasma hyopneumoniae, Streptococcus suis, Haemophilus parasuis, Salmonella choleraesuis, swine influenza virus, porcine respiratory coronavirus, Pasteurella multocida and Actinobacillus pleuropneumoniae.

PRRS virus infection also appears to reduce the benefits of medications and vaccinations (Keffaber, 1989; Loula, 1991). In the case of medications, lower feed intake or water consumption can reduce
the effectiveness of mass medication in the diet or drinking water. With regard to vaccination, Thacker et al. (2000) showed that infection with PRRS reduced the effectiveness of vaccinating against *Mycoplasma hyopneumoniae*.

**Risk Factors and Disease Severity**

Risk factors associated with the severity of PRRS outbreaks are a concern and a frequent topic of discussion with clinicians. But with PRRS, it is usually difficult to sort out variations in disease severity resulting from strain variation versus those resulting from associated risk factors. Even over time, it is difficult to determine if a reduction in severity is due to an improved control over risk factors, a change in strain virulence, or perhaps a higher level of population immunity. Herd size appears to be an important risk factor and introduction of large numbers of susceptible or recently infected gilts appears to be especially problematic. Goldberg and co-workers (2000) also reported that large herd size increased the rate of sow deaths and the severity of respiratory disease in nursery pigs. All-in/all-out management of nursery pigs was associated with reduced reproductive disease in the sows and all-in/all-out management of finishing pigs was associated with increased reproductive disease. The influence of environment on the outcome of PRRSV infection has not been rigorously studied.

**Acute PRRS**

In late 1996, severe outbreaks of reproductive failure were described in endemically infected herds (vaccinated and unvaccinated herds) in the U.S. (OIE, 1997; Rossow et al., 1997). Initially termed “sow abortion and mortality syndrome” (SAMS) or “atypical PRRS,” the terminology ultimately accepted to describe these outbreaks was “acute PRRS.” This terminology was considered appropriate because there were no features, characteristics, or clinical signs setting acute PRRS apart from previous severe PRRS outbreaks except perhaps the magnitude of losses on individual farms.

Acute PRRS outbreaks were characterized by mortality greater than 5 percent in sows and boars, and abortion rates greater than 10 percent and as high as 60 percent (Rossow et al., 1997). The reproductive outbreaks were of short duration (2 to 4 weeks) and, at least in the early stages of the disease, did not involve other stages of production (nursery or grower-finisher). Later on, death loss was severe in some nurseries that received pigs following the initial outbreak in the breeding herd.

Because of the sudden onset of these severe outbreaks and the heightened awareness of emerging diseases, the USDA responded to producer’s request for help by sending an Emergency Response Team to investigate outbreaks on ten farms in southeastern Iowa in December 1996. The primary question was whether acute PRRS was caused by a more virulent strain of PRRS virus, a pathogen other than PRRS virus, a combination of PRRS virus plus another pathogen, or whether the outbreaks were compatible with previous reports of PRRS. Although the outbreaks were severe, the investigators found no indication that any agent besides PRRS virus was involved.

A large number of risk factors were evaluated in this study but only three were found to be significant: isolation of PRRS virus, swine influenza reported, and females purchased from PRRS virus-positive or unknown status sources (Bush et al., 1997).

Subsequent to the USDA investigation, PRRS virus isolates from the acute PRRS outbreaks were studied experimentally. These isolates did appear to be more virulent than previous isolates (Halbur et al., 1998). Pregnant gilts inoculated at 85 to 89 days of gestation developed severe clinical disease following inoculation, including death (1 of 8 gilts), reproductive failure (2 of 8 sows aborted), and increased stillborn and mummified pigs (Lager et al., 1998). In this study, the authors implied that PRRS virus was solely responsible for the acute outbreaks.

**References**


