PRRS transmission

Transmission

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An important feature of the PRRS virus is the ability of infected pigs to transmit the virus for up to 100 days. Infected pigs with few or no symptoms are “shedding carriers” and are probably the most common way that the PRRS virus is introduced into a population of pigs. The virus is highly infectious. The infectious dose may be as little as 10 virus particles.

POSSIBLE ROUTES OF PRRS VIRUS TRANSMISSION:
- Feces
- Semen
- Urine
- Mammary secretions
- Nasal secretions

Pregnant sows infected with PRRS can deliver PRRS virus–infected piglets. PRRS can be transmitted from infected piglets or sows to other piglets. The cycle of virus shedding and infection can continue well into the nursery phase in situations in which the sow herd is actively infected.

Older infected pigs held back or crossfostered in farrowing rooms often are a source of virus for younger pigs. Likewise, older pigs and their secretions can be a source of infection to younger pigs on premises where pig flow is continuous/multi-aged or where biosecurity between groups is lacking.

Boars are known to shed PRRS virus in semen. The pattern of shedding via semen can be variable and intermittent among boars with studies reporting shedding periods lasting 21 to 35 days. Infection of sows via infected semen can occur through natural breeding or artificial insemination.

Stability

Because PRRSV posses a lipid envelope, the survivability outside the host is affected by temperature, pH, and detergents. PRRSV is heat labile, but relatively stable at 4°C and -70°C and solvents such as chloroform and ether are particularly efficient at disrupting the lipid envelope. pH values between 6.5-7.5 keeps the virus stable however below and above this pH range the virus’ infectivity is reduced.

Carrier animals.

By definition, PRRSV is not a ‘persistent’ virus. However, PRRS virus produces a chronic, “persistent” infection in pigs (carrier animals). That is, virus continues to replicate 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-Lincoln), 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 complicates all aspects of PRRS prevention and control.

**Transmission between pigs.**
PRRSV has been recovered from a variety of porcine secretions and excretions including blood, semen, saliva, feces, aerosols, milk, and colostrum. Fecal shedding remains a highly debated issue as studies report the presence of PRRSV in feces from 28 to 35 days post infection (PI), whereas others report no detection of virus in fecal samples.

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, particularly 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 result 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.

Some examples of practical processes that can enhance PRRS transmission within herds is enclosed in the figure below.

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<td>Needles</td>
<td>Cross fostering</td>
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<td>New gilts</td>
<td>Nurse sows</td>
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**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.