Control of porcine reproductive and respiratory syndrome (PRRS) virus

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uccessful control of porcine reproductive and respiratory syndrome virus (PRRSV) has proven to be a challenge for swine practitioners throughout the world. While there have been numerous control programs published or reported, no single document has attempted to provide concise summaries of each strategy. The purpose of this paper is to review the primary methods of PRRS control and assess the applications, advantages, and disadvantages of each one.

Remember that before you implement any type of control program, it is important to serologically profile the herd by stage of production. Profiling identifies herd-specific patterns of viral transmission and susceptible populations of pigs that may perpetuate viral circulation. A review of the steps involved in serologic profiling for PRRS is included in the Diagnostic Notes in this issue of *Swine Health and Production* (see pages 100–101).

Epidemiology and viral characteristics

Although the etiologic agent of PRRS has only recently been identified, much information has been published concerning its viral characteristics and epidemiology. It is important to understand these concepts when designing control programs. A brief summary of key points is included below, with references provided if more information is needed.

- The primary mode of transmission is the infected pig. Virus has been detected in saliva, feces, and urine.^{1,2} Porcine reproductive and respiratory syndrome virus persists in infected animals for a long time. Virus has been isolated from tonsillar tissue for up to 157 days post challenge exposure, while shedding has been documented for up to 99 days post infection.^{3,4}
- Porcine reproductive and respiratory syndrome virus can be transmitted via fresh and diluted semen, and infected boars can become long-term carriers of the virus.^{5,6} Porcine reproductive and respiratory syndrome viral RNA has been detected in semen for up to 93 days post challenge using polymerase chain reaction.⁷
- Pig-to-pig transmission is the most important means of virus spread. Studies have indicated the virus may be spread to pigs by certain species of waterfowl, but not by rodents.^{8,9} However, the importance of nonswine vectors for the transmission of PRRSV is unknown.
- Transmission of PRRSV via aerosols has been difficult to reproduce

- under controlled conditions, but reports from the field suggest it may take place.¹⁰
- The environmental stability of PRRSV is poor. Porcine reproductive and respiratory syndrome virus is readily inactivated by heat and chemical disinfection (chloroform, ether, formaldehyde, and phenols). Infectivity has been shown to be greatly reduced (> 90%) after exposure to pH levels <5 and >7.11 The PRRSV survives for only a short time on nonliving substances (fomites). Porcine reproductive and respiratory syndrome virus was inactivated in less than 30 minutes after exposure to fecal slurry, porcine urine, saliva, alfalfa, wood shavings, straw, plastic, boot rubber, and stainless steel when temperatures were maintained at 25°–27°C. In the same study, virus was isolated for up to 9 and 11 days post inoculation from well water and city water, respectively. 12
- Porcine reproductive and respiratory syndrome virus is stable when frozen or when held under cool conditions; however, duration of infectivity decreases with increasing temperature:¹³

at -70°C (-39°F), 3 years duration of infectivity;

at 4°C (39°F), 30 days;

at 20°C (68°F), 6 days; and

at 50°C (122°F), 20 minutes.

PRRS control: Step I—Breeding herd stability

Prior to deciding which PRRS control strategy to use, you must understand the importance of controlling viral circulation in the breeding herd. No program will be effective if adult swine are spreading the virus among themselves or to their piglets. High IFA titers (1:256–1:1024) or high ELISA ratios (2.0–4.0)¹⁴ detected in the sera of sows or recently weaned pigs may indicate actively circulating virus in the breeding herd, particularly if seroprevalence exceeds 20%.¹⁵ Potential reasons for persistent viral shedding in the breeding herd are summarized below:

- introduction of naive seedstock into an infected population;¹⁶
- continued introduction of actively infected seedstock; 17 and/or
- the presence of recently infected or uninfected subpopulations in large (>1000 sow) breeding herds.¹⁸

Control of ongoing viral shedding is dependent on two management practices:

Herd closure

 cease introduction of outside replacement stock for a 4-month period;

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- begin to select replacements from finishing facilities; or
- introduce 4 months of gilts into the herd at one time.
 - —Different ages of gilts may be introduced from a segregated gilt development facility based on planned replacement strategies. An example of this type of gilt management program for a 500-sow herd has been published.¹⁷

Isolation of incoming stock

Before re-opening the herd to seedstock from an outside source, it is critical to establish proper isolation/acclimatization facilities. Isolation facilities should be located offsite, whenever possible. If offsite facilities are not available, on-site facilities can be used, but should not share airspaces (hallways) with existing buildings. Isolation/acclimatization periods need to be extended from 25–30 days to 45–60 days when PRRS is involved, because periods of viremia and shedding are extensive. Finally, these facilities need to be managed on an all-in–all-out (AIAO) basis, cleaning and disinfecting between groups.

All-in—all-out requires careful planning, particularly in herds that have previously depended on weekly shipments of gilts. In these cases, it becomes important to adjust the flow of replacement stock so that herds begin to receive shipments of gilts on a monthly basis. Holding areas should consist of an offsite isolation facility, and a separate acclimatization facility, each with the capacity to house 4 weeks of gilts. Thus, monthly groups of animals could be moved throughout the isolation, acclimatization, and breeding (gilt pool) facilities on an ongoing basis using AIAO pigflow. Within each shipment are four groups of

Table I

Facility location and specific actions per site for isolation/acclimatization programs based on monthly shipments of replacement stock

Isolation facility

Location = offsite

Action = PRRS vaccination day I and day 30 post arrival (may involve extra label use)^a

Duration of stay = 30 days

Acclimatization facility

Location = onsite^b or offsite

Action = Feedback^c, contact with cull sows and boars and farm specific vaccination programs should be implemented during this stage to enhance exposure of new animals to farm specific microflora

Duration of stay = 30 days

- ^a Using the product in this way constitutes extra-label use and is not approved by the USDA nor recommended by Boehringer-Ingelheim.
- ^b Onsite facility should be physically separated from main breeding building.
- ^cTissue feedback for PRRS virus exposure acceptable **only** during the first week of this stage. No tissue feedback should be provided to pregnant sows at any stage of gestation.

gilts with each group representing 1 week's worth of replacements. Groups may be identified by using a different-colored eartag per weekly shipment of animals. While this type of management strategy may not be conducive to all production systems, it has been successful where implemented. Site-specific action plans are provided in Table 1.

PRRS Control: Step 2—Selecting a strategy

The following are brief summaries of commonly practiced control programs. References have been provided if further information is required.

Nursery depopulation19

- Purpose: Nursery depopulation is a control strategy for postweaning PRRS. It consists of a strategic, temporary adjustment in nursery pigflow to prevent the spread of virus from older, previously infected pigs to those recently weaned.
- Protocol: After attaining a serologic profile that indicates an absence of viral transmission in the breeding and finishing populations:
 - —depopulate all nurseries at one time. Move pigs to another site until marketed. Do not re-introduce these animals to the primary herd site;
 - —wash and disinfect all nursery rooms three times using 90°–94° C (194°–200° F) water and a rotation of chemical disinfectants, i.e., formaldehyde- and phenol-based products. During the disinfecting procedure, be sure that gloves and adequate protective clothing (face and eyewear) are worn at all times. Byproducts of the disinfecting process (fumes) can be extremely irritating to the eyes, skin, and respiratory tract;
 - —rooms should remain free of pigs for a minimum of 2–3 days. Nursery pits should be emptied between cleaning and disinfecting cycles.
 - —After the cleaning program is completed, resume use of nursery rooms.

• Pros

- —low-cost approach to controlling PRRS and improving nursery performance;
- -minimal disruption of pigflow; and
- —minimal stress on the labor force.

• Cons:

- —depends heavily on a specific serologic pattern that indicates lack of viral transmission in the breeding and finishing populations:
- —may be difficult in large (> 1000 sow) herds;
- —requires a temporary offsite facility for housing depopulated pigs.

Vaccination²⁰

RespPRRSTM: a federally-licensed modified-live virus vaccine (Boehringer-Ingelheim, St. Joseph, Missouri)

- Purpose: To induce a protective immune response in weaned and growing pigs.
- Protocol: Vaccinate pigs intramuscularly with a 2.0-mL dose.

RespPRRS™ is approved for pigs from 3–18 weeks of age.

- Pros
 - product is safe and efficacious when applied according to label directions.
- Cons:
 - —product cost and administration effort;
 - —the degree of heterologous protection among PRRS virus isolates is unclear;
 - —due to the inability to consistently distinguish antibodies produced after vaccination from those produced after infection with field virus, it may be difficult to interpret serological results after the vaccine is used.

Despite the fact that the vaccine is only approved for use in swine from 3–18 weeks of age, the practice of vaccinating breeding and gestating animals is relatively widespread. Intranasal vaccination of piglets prior to weaning is sometimes practiced as well. Using the product in this fashion constitutes extra-label use and is not approved by the USDA nor recommended by Boehringer-Ingelheim or NOBL Laboratories. Practitioners must understand that this requires a valid veterinary-patient-client relationship at all times, and that there is little safety and efficacy data available concerning use of the product in this manner. Recent published research in mature swine has shown that:

- vaccinating pregnant sows during the third trimester of gestation (90–93 days) did not result in reproductive failure under controlled conditions;^{21,22} however, the use of modified-live virus vaccine is not recommended during late gestation;
- piglets may be viremic at birth if sows are vaccinated during the third trimester because the vaccine virus can cross the placenta at this time;²²
- vaccine virus may be shed to contact pigs following glucorticoid treatment of vaccinated animals;²³
- vaccine virus can be shed in the semen.²⁴

MCREBEL^{25,26} (Management Changes to Reduce Exposure to Bacteria to Eliminate Losses)

- Purpose: A systematic approach to reduce the spread of secondary bacteria and PRRSV among farrowing house pigs and to nursery pigs.
- Protocol:
 - —cross foster only during the first 24 hours of life;
 - —do not move sows or piglets between rooms;
 - —eliminate the use of nurse sows;
 - —humanely destroy piglets that become sick and are unlikely to recover;
 - —minimize handling of piglets, especially administration of routine antibiotics or extra iron injections;
 - —do not transfer undersized pigs back to rooms containing younger litters;
 - —immediately stop all feedback of porcine tissue;
 - —move nursery pigs according to strict AIAO principles, allowing for 2–3 days between groups for cleaning and disinfecting.
- Pros:
 - -minimal cost to implement;

- —practical strategy for minimizing infected nursery losses while attempting to stabilize breeding herd PRRSV circulation.
- Cons:
 - —difficult to implement, because labor may resist eliminating cross fostering and piglet euthanasia;
 - —incentive plans need to be re-evaluated before it is implemented;
 - —the strategy is new, and minimal herd data are available concerning duration of effects.

Conclusion

Based on reports from the field, it may not be possible to control all cases of PRRS with a single strategy. Therefore, developing systematic plans involving a group of strategies may be more effective, particularly in large herds. Tables 2 and 3 describe such an approach, highlighting critical decision-making steps and listing potential control options to proceed. Again, this is provided only as an example, and practitioners need to establish control programs based on herd-specific data. However, we believe that if you take a systematic approach to PRRS control, you can obtain positive results under a wide range of farm conditions.

References

- 1. Yoon IJ, Joo HS, Christianson WT, et al. Persistent and contact infection in nursery pigs experimentally infected with PRRS virus. *Swine Health and Prod.* 1993.1(4):50
- 2. Rossow KD, Bautista E, Goyal S, etal. The effect of pig age on clinical disease and immunopathogenesis of SIRS virus infection. AASP Newsl. 1992; 4:26.
- 3. Will R, Zimmerman J, Yoon KJ, et al. PRRS virus—A persistent infection. In: *Proc 2nd Intl Symp PRRS*, Copenhagen; 1995:19.
- 4. Zimmerman J, Sanderson T, Eernisse KA, et al. Transmission of PRRS virus for convalescent animals to commingled penmates under experimental conditions. *AASP Newsl.* 1992; 4:17.

Table 2

PRRS control — Case number I

Clinical signs:

- Poor performance postweaning
- Nursery pigs healthy at weaning, but demonstrate signs of respiratory disease I-2 weeks later
- ADG = 0.3-0.5 lb/day
- % Mortality = 5%-20%

Serologic profile:

		Results	
Stage	Prevalence	IFA	ELISA
Breeding	0%-10%	≤I:I6	≤ 0.4
4 week old	0%-10%	≤1:16	≤ 0.4
8 week old	60%-100%	1:256-1:1024	> 2.5
6 month old	20%-30%	1:16-1:64	0.4-1.0

Interpretation:

- · Virus transmission taking place in the nursery
- · No active infection in other production stages

Control options:

- Nursery depopulation
- Vaccination of weaned pigs

PRRS control — Case number 2

Clinical signs:

- · Anorexia, agalactia in lactating sows
- Respiratory disease in pigs prior to and immediately following weaning
- Preweaning mortality = 10%-15%
- Nursery mortality = 10%-15%
- Nursery ADG = 0.3-0.5 lb/day

Serologic profile:

Resu	lts
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Stage	Prevalence	IFA	ELISA
Breeding	50%-100%	1:256-1:1024	≥ 2.5
4 week old	50%-100%	1:256-1:1024	≥ 2.5
8 week old	50%-100%	1:256-1:1024	≥ 2.5
6 month old	50%-100%	1:256-1:1024	≥ 2.5

Interpretation:

 Active virus infection occurring throughout all stages of production

Control program immediate action: (Day 1–30 post infection)

- · Close herd to outside replacements
- Implement MCREBEL

Intermediate action (I-2 months post infection):

- Maintain herd closure and MCREBEL
- · Re-assess serostatus
- -If no change, maintain current program
- If results indicate impending cessation of viral spread in breeding herd and recently weaned pigs, begin planning for initiation of pig vaccination or nursery depopulation programs

Final action (3–4 months post infection):

- Re-assess serostatus. If herd appears to be stabilized, carry out the following strategies:
- -Discontinue MCREBEL
- -Begin pig vaccination program or implement nursery depopulation
- -Re-open breeding herd after establishing proposed isolation/acclimatization program
- -Maintain replacement stock vaccination program on a continuous basis as previously described

- 5. Yeager MJ, Prieve T, Collins JE, et al. Evidence for the transmission or PRRS virus in boar semen. *Swine Health and Prod.* 1993; 1(5):7–9.
- 6. Swenson S, Zimmerman J, Evans L, et al. Exposure of gilts to PRRS virus by artificial insemination. In: *Proc 2nd Intl Symp PRRS*. Copenhagen; 1995:42.
- 7. Christopher-Hennings J, Nelson E, Nelson J, et al. Persistent shedding of PRRS virus in boar semen. In: *Proc 2nd Intl Symp PRRS*. Copenhagen; 1995:53.
- 8. Zimmerman J, Yoon KJ, Pirthe EC, et.al. Susceptibility of four avian species to PRRS virus. In: *Proc Ann Meet LCI*; St.Louis; 1993:107–108.
- 9. Hooper CC, Van Alstine WG, Stevenson GW, Kanitz CL. Mice and rates (laboratory and feral) are not a reservoir for PRRS virus. *J Vet Diagn Invest.* 1994; 6:13–15.
- 10. Zimmerman J. A general overview of PRRS virus: A perspective of the USA. *Proc 2nd Intl Symp PRRS*. Copenhagen; 1995:2.
- 11. Benfield DA, Nelson EA, Collins JE, et al. Characterization of PRRS virus (isolate ATCC VR-2332). *J Vet Diagn Invest*. 1992;4:127–133.
- 12. Pirtle EC, Beran GW. Stability of PRRS virus in the presence of fomites commonly found on farms. *JAVMA*. 1996;208:390–392.
- 13. Albina E, Benfield DA, Haas B. Survival of PRRS virus outside of the host. *Pig Dis Info Ctr.* Cambridge 1992: 1–3.
- 14. Phillips R. Personal communication, December 1995.
- 15. Dee SA, Joo HS, Polson DD. An evaluation of nursery depopulation for controlling postweaning PRRS on 34 farms. 1. Production and diagnostic data. *Vet Rec* (submitted).
- 16. Dee SA, Joo HS. Clinical investigation of recurrent reproductive failure associated with PRRS virus infection in a sow herd. *JAVMA*. 1994; 204:1017–1018.
- 17. Dee SA, Joo HS, Pijoan C. Controlling the spread of PRRS virus in the breeding herd through management of the gilt pool. *Swine Health and Prod.* 1994;64–69.
- 18. Dee SA, Joo HS, Park BK, et al. Demonstration of subpopulations following PRRS virus infection in large breeding herds using multiple serologic tests. *Swine Health and Prod.* (submitted)
- 19. Dee SA, Joo HS. Prevention of PRRS virus spread in endemically infected swine herds by nursery depopulation. *Vet Rec.* 1994;135:6–9.
- 20. Gorcyca D, Spronk G, Morrison R, Polson D. Field evaluation of a new MLV PRRS virus vaccine. *Proc AASP Ann Mtg.* 1994:401–412.
- 21. Gorcyca D, Schlesinger K, Harris L et. al. Safety testing of a modified-live virus vaccine for PRRS. In: Proc 2nd Intl Symp PRRS. Copenhagen; 1995:56.
- 22. Mengeling WL, Lager KM, Brockmeier SL, Vorwald AC. The effect of various strains of PRRS virus on reproductive efficiency of gilts infected in late gestation. *Prod 3rd Ann Dis Conf Swine Pract*. Des Moines, Iowa. November 1995; 103–105.
- 23. PRRS/RespPRRS™ Reference Guide. NOBL Laboratories. February, 1996.
- 24. Shin JH, Torrison J, Choi CS, et al. Monitoring of PRRS virus infection in boars. In: *Proc 2nd Intl Symp PRRS*. Copenhagen; 1995:55.
- 25. McCaw MB. MCREBEL PRRS: Management procedures for PRRS Control in large herd nurseries. In: *Proc AD Leman Swine Conf*, St Paul; 1995:161–162.
- 26. Houghton D. A plan for PRRS (Think like a rebel). *Hogs Today*. November 1995; 11(9):12–13.