Controlling the spread of PRRS virus in the breeding herd through management of the gilt pool

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Summary: *This article discusses two strategies used in field trials to control the spread of porcine reproductive and respiratory syndrome (PRRS) virus in a breeding herd. In the first experiment, two breeding herds were closed to outside replacements and replacement males and females were raised on the farms. In the second experiment, replacement gilts were held in an off-site holding facility and the nursery was depopulated. None of the experimental farms showed evidence of PRRS spread by IFA serology for the 6 months of the experiment.*

Porcine reproductive and respiratory syndrome (PRRS) was first reported in 1987.[1] A devastating viral disease, it is capable of producing severe losses through both reproductive failure and poor postweaning performance.[1-3] Nursery depopulation (ND) has been shown to be an effective strategy for controlling postweaning PRRS problems,[4-7] but one that requires an absence of viral shedding in the sow herd.

The primary method of assessing the serostatus of a herd is the indirect fluorescent antibody (IFA) test.[4-8] The IFA test measures exposure to PRRS virus, but has no correlation with protective immunity.[8] High titers (>1:256) indicate recent exposure and may take up to 144 days to become undetectable.[8,9] Animals with titers of this level may be viremic and ND has failed if viremia is not under control in the adult population or the suckling piglets.[6] If there is evidence of seropositive sows or recently weaned pigs (3–4 weeks of age), viral shedding and subsequent exposure may take place, reinfecting the nursery pigs. Therefore, controlling PRRS virus transmission within the sow herd is of the utmost importance.

It has been reported that viral persistence in the breeding herd may be due to introducing seronegative, naive gilts into an endemically infected population.[11] Similarly, if replacement gilts are originating from a PRRS-positive herd, they may be viremic upon arrival.[6] Virus can be shed via oro-nasal, fecal, and urinary secretions.[12-14] While regularly introducing replacement gilts is important to maintain a biologically productive sow herd, it may be a mechanism for persistently transmitting virus in this area.

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Freese, et al., described a case of spontaneous elimination of PRRS virus.[15] The farm they worked with housed replacement gilts in a separate facility from the existing herd during the gestation period. No new animals were added to the herd for nearly 4 months. Serologic profiling via IFA testing revealed that viral transmission had apparently ceased and exposure had decreased. As of this writing, follow-up testing in this herd has indicated no evidence of reinfection. While effective, this management practice may not be applicable to all production systems. In this paper, we describe other strategies we developed to manage the gilt pool to control PRRS virus shedding in the breeding herd and report the results of testing these strategies on three commercial swine farms.

Methods

All three operations included in this study (Farms A, B, and C) were chosen based on the owners' willingness to participate and a positive diagnosis of PRRS virus infection via serology and virus isolation.[8,10]

Experiment 1: Control of PRRS virus exposure in the breeding herd using an on-site internal replacement program

Experiment 1 was conducted with Farms A and B, whose owners had decided to implement a closed-herd genetic program. Prior to this decision, the source of the replacement gilts had been a commercial seedstock company that had also recently been confirmed to be PRRS positive via serology and virus isolation. After the herds were closed, the owners planned to raise replacement males and females on the farm and select among them at 6–7 months of age for inclusion in the breeding herd. Based on this decision, we attempted to investigate what effect this type of replacement program would have on the PRRS serostatus of the adult population.

At the time the farms were closed, we tested 10 randomly selected monitors from each of the following groups of animals:

- sows;
- replacement gilts;
- 4-week-old pigs;
- 10-week-old pigs;

- 16-week-old pigs; and
- 6-month-old pigs.

Serum was separated via centrifugation at 2500 rpm and tested using the IFA method previously described.[8]

After the farms were closed, 10 randomly selected samples were collected monthly over a 5-month period from the breeding herd on each farm. During the sixth month, we collected 30 randomly selected samples from both breeding herds and we again tested 10 randomly selected monitors from the following age groups of pigs:

- 4-week-old pigs;
- 10-week-old pigs;
- 16-week-old pigs; and
- 6-month-old pigs.

Gilts were coming in from the finisher, so we did not resample the replacement gilts. The sample sizes collected during the sixth month were calculated assuming a seroprevalence of 10%. Therefore, 30 samples per farm were collected in order to be 95% confident of detecting at least one positive pig. Decreases in titer levels before and after the herd was closed were tested for significance using the Student's T-test.

Experiment 2: Control of PRRS virus exposure in the breeding herd using an off-site gilt-holding facility in combination with nursery depopulation

We conducted Experiment 2 on Farm C. Previous serology had indicated a high prevalence of IFA positive animals in all phases of the operation. Postweaning performance was also poor, with average daily gains of 0.136 kg per day and 10%–15% mortality in the nursery. The owner wanted to solve these problems using nursery depopulation (ND); however, 36.6% of the samples collected from the breeding herd were positive. The commercial seedstock source was also endemically infected. An off-site finishing facility was available for rent, therefore we decided to establish a separate facility to house seropositive replacements prior to entry into the breeding herd. We decided that previously seropositive replacements would only enter the existing herd following evidence of a fourfold reduction in IFA titers. Based on the data in experiment 1, we hypothesized that the seroprevalence to PRRS in the breeding herd would eventually decrease because it would prevent the virus being continuously introduced.

The off-site facility on Farm C was a partially slatted barn which had been empty for 2 years. Based on calculated production parameters, we decided to purchase at one time four groups of gilts (25 gilts per group) ranging in age from 9–12 weeks, and house them in the off-site facility. These 100 gilts were acquired, identified by ear tag, and 30 were randomly selected for testing for antibody to PRRS virus via IFA on arrival. The same day, the breeding herd was closed and 30 sows were selected at random and tested in a similar fashion. Sample sizes were calculated to exceed an estimated 50% prevalence (sows) and 75% prevalence (gilts) with an accuracy of 20% at a 95% confidence level. The production parameters, pig flow schedule, and the effect of the project on breeding herd inventory are summarized in Tables 1A and 1B. Gilts were fed a 16% crude protein corn/soy diet ad libitum. Gilt breeding age was targeted at 7–7.5 months, therefore the time period allotted the project was approximately 4–5 months.

Table 1A

Projected changes in inventory and schedule of pig flow on Farm C: Proposed production parameters

Number of sows:	500
Annual cull rate:	45%
Number culled per year:	225
Number culled per month:	18–20
Reduction in monthly sow inventory:	4%
Gilt conception rate:	80%
Number of gilts required per month:	25
Desired age at first service:	210 d (30 wks)
Proposed age of PG-600® injection	150–160 d
Proposed age of boar exposure	180 d
Gilt groups	
25 gilts in each of four groups:	
Group 1 age at arrival	12 wk
Group 2	11 wk
Group 3	10 wk
Group 4	9 wk

In an attempt to synchronize estrus in the off-site population, each group of gilts was injected intramuscularly with 400 IU of pregnant mare serum and 200 IU of human chorionic gonadotropin (PG-600®, Intervet Inc., Millsboro, Delaware) at approximately 150–160 days of age. Thirty days after the first group was injected, a mature herd boar was placed in the off-site facility to provide olfactory stimulation. The boar had been previously tested for PRRS antibody via IFA and was found to be negative. The boar was housed in the alleyway in order to provide nose-to-nose contact to all four groups.

Of the 30 gilts and 30 sows tested, 10 from each group were chosen at random to be tested monthly. These animals were given a different-colored ear tag to enhance quick identification. We interpreted a fourfold reduction in IFA titers or no evidence of seroconversion to mean that animals were no longer viremic and exposure to virus was declining. If we saw a fourfold increase in the titer of a monitored animal, it was culled. At the end of the experimental period, but prior to the entry of the gilts into the breeding herd, 30 gilts and 30 sows were randomly selected for testing. This sample size was calculated as previously described in this experiment. During the fifth month, the individual groups of gilts entered the breeding herd on a weekly basis and were bred according to plan (Table 1B). Nursery depopulation procedures were carried out as previously described.[4,6,7] Serologic monitoring by IFA of 8- to 10-week-old nursery pigs took place 6 months later to assess whether PRRS virus was still present in the nursery after the ND protocol was completed. Thirty animals were sampled in a similar fashion. Differences in seroprevalence of the

breeding herd and the gilt pool were detected for significance using Chi square analysis.

Results

Experiment 1

Prior to closing the herd(s), all samples collected from the breeding herd, purchased replacement gilts, and 4-, 10-, and 16-week-old pigs from both farms A and B had titers > 1:256. In contrast, only 50% of the finishing pigs sampled from both farms had similar titers; the remainder of titers ranged from negative to 1:64. Over the 6-month testing period, the number of positive samples in all areas of both farms decreased significantly ($P < .005$) until the final testing indicated that all samples collected were negative (<1:16).

Experiment 2

Throughout the testing period, declining titers were seen in all but one of the monitored sows and gilts (Table 2). A fourfold rise in titer was seen in one of the monitored sows (#10) and this animal was sold to slaughter. Chi square analysis of the seroprevalence of titers in randomly selected sows and gilts indicated a significant ($P < .005$) decrease in both populations (Table 3). Four of the 30 sows tested remained positive, with titers of 1:16. Two of 30 gilts tested were positive, both with titers of 1:16. Nursery depopulation resulted in improvements in average daily gain (.38 kg per day) and a reduction in mortality to 4%. No evidence of IFA titers to PRRS virus were found in any of the samples collected in 8- to 10-week-old pigs 6 months following the ND procedures (Table 4).

Discussion

The results of both experiments indicate that PRRS virus exposure in the breeding herd can be controlled by managing the gilt pool. By reducing the risk of introducing potentially viremic animals into the existing population, the degree of exposure in the herd appears to decrease. These findings agree with results previously described.[15] We have observed that the desired serostatus may be obtained without manually intervening or controlling animal flow. However, this appears to take place over a 1- to 1.5-year period after infection and depends highly on the degree of exposure in the replacement females. A common finding in self-regulating herds is a lack of high (1:256–1:1024) titers to PRRS virus as

Table 1B

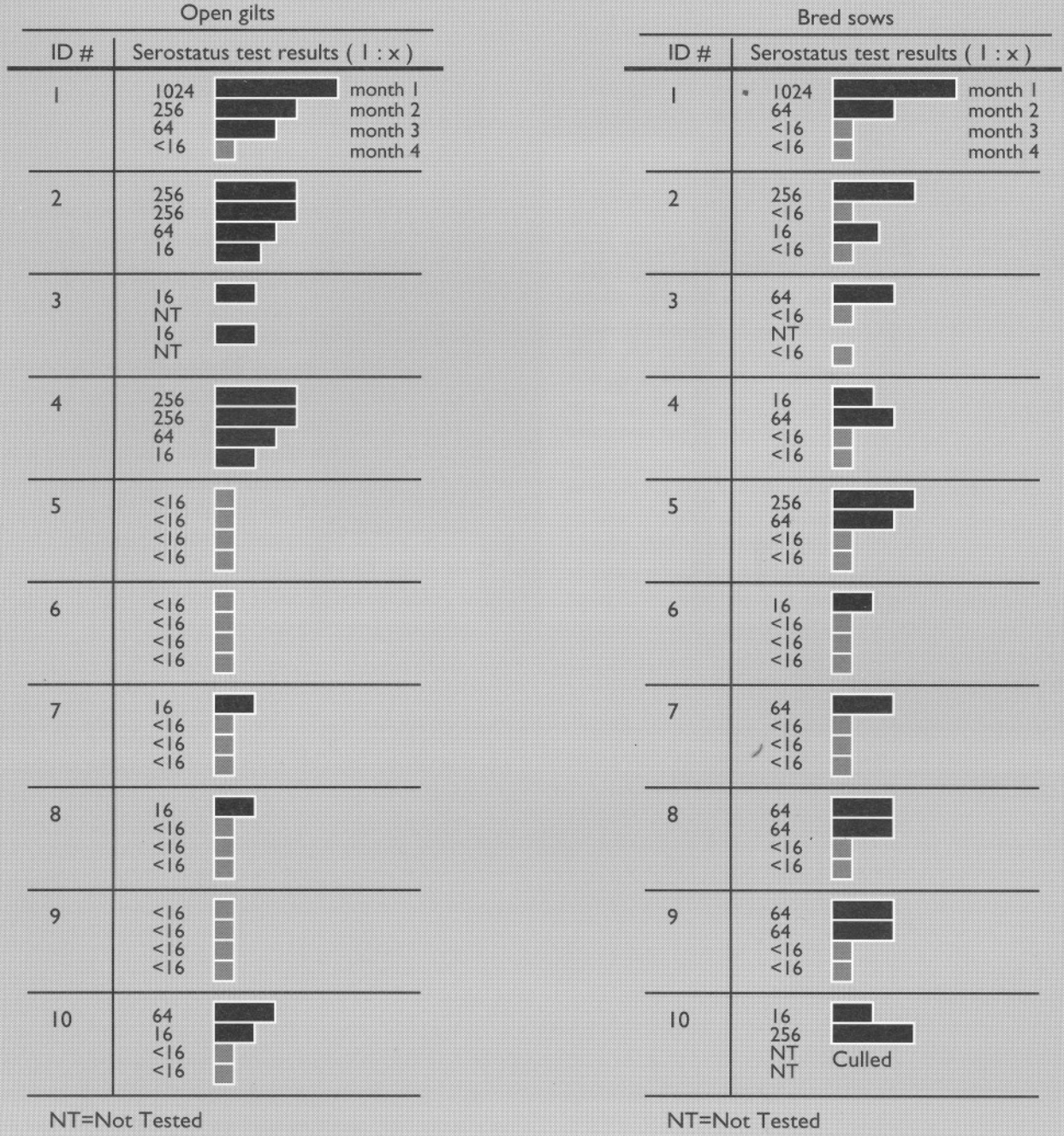
Proposed activity and stage over time

Time	Offsite pool	Breeding herd	Breeding herd inventory
Month 1	Gilts arrive	Close herd	n=500
week 1	Test 30*	Test 30	
week 2	Select 10 monitors	Select 10 monitors	
Month 2	Test 10 monitors	Test 10 monitors	n=481
Month 3	Test 10 monitors	Test 10 monitors	n=462
week 1	Group 1— PG -600®		
week 2	Group 2— PG -600®		
week 3	Group 3— PG -600®		
week 4	Group 4— PG -600®		
Month 4	Test 10 monitors	Test 10 monitors	n=443
week 1	Introduce boar		
Month 5	Test 30	Test 30	n=425
week 1	Move group 1	Introduce and breed group 1	n=450
week 2	Move group 2	Introduce and breed group 2	n=475
week 3	Move group 3	Introduce and breed group 3	n=500
week 4	Move group 4	Introduce and breed group 4	n=525

*Note: All tests are serological assessment via IFA

Table 2

Changes in individual monitor pig serostatus over time



well as an absence of detectable viremia in replacements. In the cases described in experiments 1 and 2, incoming replacement animals demonstrated evidence of recent viral exposure and viremia. Previous studies have demonstrated that animals of this nature may be viral carriers and potential sources of viral reintroduction to the herd.[8] Without some regulation of animal flow, it is unlikely that viral shedding could have been controlled.

It was interesting that the herds in experiment 1 experienced what appeared to be a spontaneous elimination of virus as described by Freese, et al.[15] Differences between the farm Freese, et al., studied and farms A and B in experiment 1 were:

- batch farrowing (Freese, et al.) versus weekly farrowing;
- off-site gestation housing (Freese, et al.) versus on - site; and
- an outside source of seedstock (Freese, et al.) versus the internal replacement program.

The length of time that the negative serostatus can be maintained is unknown. Recent work has demonstrated that the piglets infected in utero may be carriers and potentially shed virus later in life.[17] Also, a long-term carrier state has been described in the adult animal, with the ability to infect seronegative contact controls.[18] Because these farms will be monitoring IFA status on a regular basis, any changes in viral exposure will be detected.

Table 3

Change in seroprevalence in randomly selected individuals from both populations over the 4-month testing period. No attempt to test specific individuals was made

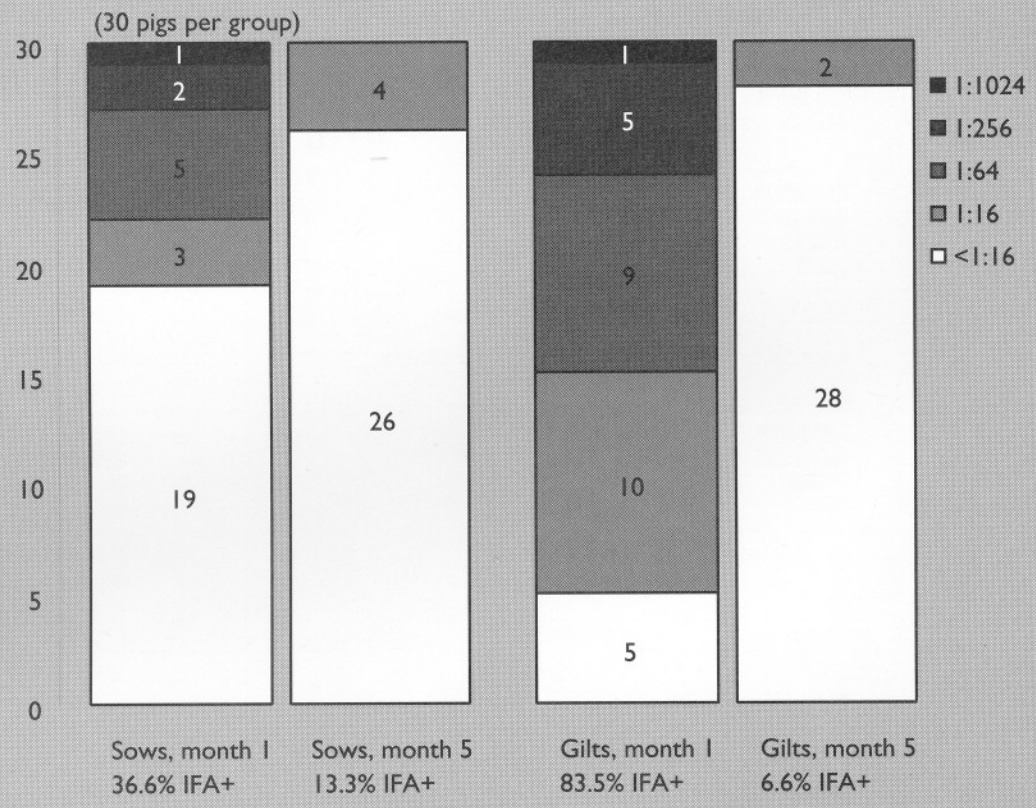


Table 4

Performance and serostatus improvements in young pigs following nursery depopulation

Group tested	# tested	Titer range <1:16	1:16	1:64	1:256	1:1024	ADG	Mortality
4 weeks old	10	10	–	–	–	–	No data	No data
10 weeks old, no nursery depopulation	10	1	1	3	4	1	0.14 kg	15%
10 weeks old, 6 months following nursery depopulation	30	30	–	–	–	–	0.38 kg	4%

Because the herds in experiment 1 are currently IFA negative, we will need to decide how to introduce new genetic lines. The data in experiment 2 indicates that it may work to establish an off-site holding facility for housing seropositive replacements until the proper time for introduction is at hand. One should take at least two tests at 30- to 60-day intervals to assess changes in serostatus and carry out virus isolation on pooled samples. Only animals with evidence of declining titers and negative virus isolation results should be brought into the herd. Handling previously infected seedstock in this manner has been successful.[16] This allows the commercial producer to maintain its genetic program and a viable working relationship with the chosen seedstock source, even if the latter is PRRS positive.

The disadvantage of the system described in experiment 2 is that for a 4-month period, the normal replacement rate and inventory of the existing breeding herd was disrupted. As far as the cost of the reduction in breeding herd inventory (500 to 425 sows), the farm will eventually experience a short-term reduction in income, due to a lower number of marketable pigs than if the inventory had remained constant. However, there will more than likely be an overall increase in herd productivity due to improvement in nursery mortality (15% versus 4%) resulting from the nursery depopulation procedure. It should also be noted that breeding herd inventory at the end of the project actually increased by a total of 25 sows (Table 1B).

It is obvious that accurately interpreting serology is critical to making decisions. The majority of the serology from both experiments was read by the first author. Positive and negative serum controls were included to reduce confusion resulting from nonspecific background fluorescence common with the IFA test.[19] Using consistent personnel and including serum controls appear to be very useful for objective interpretation. In order for practitioners to improve standardization of their results, it may be helpful to submit paired samples to a diagnostic laboratory at the same time, and consistently work with the same laboratory. Finally, providing the serologist with an accurate history of the farm and goals of the project is helpful, too.

In conclusion, managing the gilt pool appears to be an effective way to reduce exposure to PRRS virus and to control viral shedding in the breeding herd. The method chosen depends on the production system, management capabilities, and facility availability of each specific case. However these strategies are useful to enhance the success of postweaning eradication programs. While it appears that this strategy is effective in the study herd, it is unknown at this time whether similar strategies will work elsewhere. It may be difficult to control the PRRS status of all replacement animals, particularly over a long period of time, especially if the replacements originate from highly infected herds where no control measures are currently in place. Other variables, such as herd size, may also have an effect. It appears to be difficult to achieve similar levels of success in larger herds, i.e., ≥ 1000 sows. The results we achieved in this study may not be repeatable if the aforementioned variables are not carefully controlled. Therefore, this management strategy may not be applicable in all cases. Perhaps with the development of commercially available, efficacious, and approved vaccines, a consistent level of immunity and control of viral shedding can be obtained in large breeding herds.

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