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The role of the replacement gilt in the maintenance of the PRRS infectious process

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Before the advent of PRRS control programs, gilt development practices were often overlooked. Despite a proven role in the infectious process of TGE, Aujesky's Disease, and porcine parvovirus, a recent NAHMS survey indicated that isolation of replacement animals was practiced in only 37.4% of surveyed farms.[1] Since PRRS has re-educated practitioners and producers on the importance of handling replacement stock, a number of systems, concepts, theories, and schemes have been devised. Working with gilt programs since 1992, it is my opinion that gilt development can "make or break" a PRRS control program. Prior to recommending a specific protocol to a producer, it is important to separate science from speculation, and attempt to understand the facts that are known regarding replacement gilts and PRRS. The purpose of this paper is to present new observations on four critical points of gilt development and provide solutions to these problems.

Problems

1. The positive gilt has a negative effect

Introducing actively infected replacement animals enhances virus transmission within the breeding herd, and exacerbates PRRS problems in the nursery. Published data has described the change in breeding herd PRRS seroprevalence and nursery pig performance following introduction of viremic replacement females.[2] This paper also examined a gilt development center which held animals during a 4-month period in which no replacements entered the breeding herd. "Monitor" animals from the gilt center and the breeding herd were serologically assayed on a monthly basis. When significant differences were not detectable between the seroprevalence of both populations, the gilts were introduced and the nursery depopulated.

2. Consistent natural exposure is inconsistent

Currently, a challenge to gilt development programs is the process of acclimation of the animals to the farm-specific isolates of PRRS virus. Molecular diagnostics, such as polymerase chain reaction (PCR) and viral sequencing have enabled practitioners to differentiate field isolates from modified live virus vaccine isolates. A recently completed field study indicated that the ability to consistently expose gilts to wild type virus through direct contact with infected nursery pigs is inconsistent. (Dee, S.A. unpublished data 1998) Thirty replacement gilts, derived from a PRRS negative source were housed in pens of 3 gilts/pen. Two seeder pigs were placed within each pen for a 30 day period. Evaluation of viremia of replacement animals indicated that shedding of wild type virus from seeder pigs took place at irregular intervals during the acclimation period (days 17, 23, and 30). Furthermore, viremia was detected in only 50% (15/30) of the replacements (**Table 1**). As viral isolates were identified, RFLP analysis indicated a 2–6–2 "cut pattern" and their molecular sequences differed by 15 base pairs when compared to the RespPRRS vaccine strain. Identical results had been obtained from commercial nursery pigs tested prior to their introduction to the developer facility. One dose of RespPRRS modified live virus (mlv) vaccine was sequenced as a control. As expected, its RFLP pattern was 2–5–2 and differed by 15 base pairs when compared to the wild type isolate.

Table 1: Shedding patterns of wild type PRRS virus from seeder pigs to replacement gilts during acclimation

Days Tested Post-Exposure	# Detected	# Tested	RFLP	Bases*
1, 5, 9, 13	0	30	—	—
17	3	30	262	15
21	0	27	—	—
23	3	27	262	15
25 - 29	0	24	—	—
30	9	24	262	15

* = Base pair differences when compared to RespPRRS mlv vaccine

3. Subclinical shedding of wild type virus to suckling piglets occurs in gilt litters

Trends in the global industry have focused on the introduction of younger replacement gilts, in larger groups, less frequently throughout the year.[4] Typically, gilts are purchased as weaners, vaccinated on arrival (16–18 days) to the developer facility, and acclimated for 3–4 months. While a second vaccination is frequently given at selection, (5 months of age) if animals are allowed to gestate and lactate prior to re-vaccination, a 6.5–7.5 month interval exists between vaccinations. Since duration of immunity studies following vaccination with RespPRRS mlv vaccine were terminated after 4 months post-vaccination, the question arose whether this extended period between vaccinations, in combination with inconsistent exposure to wild type virus during acclimation, would result in the development of potentially naive first parity animals in late gestation.

Although current serologic tests cannot be correlated with protective immunity following PRRS virus infection, they can be used to assess the chronicity of the infection. Previously published data described subpopulations of noninfected, acutely infected, and chronically infected sows via serologic profiling using a battery of serologic tests.[3] The data in **Table 2** summarizes cross-sectional profiling of third trimester gilts, and gilts during lactation from 13 PRRS positive farms. Gilts had been vaccinated and acclimatized as previously described and sampled using ELISA and serum neutralization (SN) tests. Based on these data, it appears that 22.3% of the animals tested were seropositive on ELISA, but negative on the SN test. These results indicate that a subset of the sampled population had been exposed to PRRS virus within 2–3 weeks prior to sampling. Whether they were naive to begin with cannot be adequately assessed, but the serologic response seems to indicate recent exposure to virus at a critical stage.

A follow-up finding to the previous point indicated that subclinical shedding of virus from dam to offspring exists in gilt litters. Field studies currently in progress have documented that infected piglets exist in clinically and serologically "stable" breeding herds. (Dee, S.A. unpublished data 1998) Sequencing of viral isolates in vaccinated herds have resulted in verification of wild type virus infection in suckling piglets nursing vaccinated sows. Nursing piglets ranging in age from birth (day 0) to weaning (day 16) were tested. Five litters per parity group were selected and 1 piglet was tested from each selected litter. Wild type virus was detected in 60% of the gilt litters sampled, but only in 16-day-old piglets during the first trial (**Table 3**). No viremic piglets were detected from parity 2 sows or higher. A second repetition of this study resulted in negative results throughout all parities, while a third replicate is currently underway. Vaccine virus transmission has not been detected at this time in any replicates, despite the vaccination of dams at day 16 of lactation.

Solutions

Obviously, all studies are not yet completed. Still, there appears to be some preliminary messages that could be put into practice in the field.

1. Purchase naive, noninfected animals

The introduction of PRRS-negative replacement stock reduces the risks described in Problem #1. It also eliminates the risk of introduction of a new isolate of virus into the system. Overall, it has been my observation that the introduction of mixed sources of PRRS positive gilts into previously well-controlled systems results in disaster.

Table 2: Percentage of ELISA (+)/SN (-) gilts in third trimester or during lactation from 13 herds

Farm#	# Tested	# Animals ELISA (+)/ SN(-)	%
1	10	1	10
2	10	2	20
3	10	1	10
4	10	1	10
5	10	1	10
6	10	2	20
7	10	2	20
8	10	1	10
9	10	2	20
10	10	2	20
11	10	1	10
12	10	5	50
13	10	8	80
Total	*130*	*29*	*22.3*

Table 3: Shedding patterns of wild type PRRS virus by parity throughout lactation

	Days of Lactation								
	0	*2*	*4*	*6*	*8*	*10*	*12*	*14*	*16***
P1	0/5	0/5	0/5	0/5	0/5	0/5	0/5	0/5	3/5
P2 - 7*	0/30	0/30	0/30	0/30	0/30	0/30	0/30	0/30	0/30
RFLP	(-)	(-)	(-)	(-)	(-)	(-)	(-)	(-)	2-6-2
Bases***	(-)	(-)	(-)	(-)	(-)	(-)	(-)	(-)	15

* = 5 litters/parity: 1 pig/litter
** = day of sow vaccination
*** = Base pair differences when compared to RespPRRS mlv vaccine

2. Don't rely solely on natural exposure

Vaccinate incoming naive gilts and allow for a 4-week period post-vaccination prior to acclimation. Vaccination with a modified live virus vaccine will provide consistent exposure to an attenuated live virus, something that natural exposure fails to do on a consistent basis. Killed autogenous products have been ineffective in my experience in North America and Europe, due to the inability of these products to stimulate the cell-mediated immune response.

Although previous publications have described re-vaccination of replacement animals at 30 day intervals[4], recent data have indicated no significant differences in lymphocyte blastogenesis responses following one or two (30-day interval between injections) vaccinations of a modified live virus vaccine. (Molitor, T. personal communication, February 1998) Therefore, one dose is provided at entry and re-vaccination is carried out 4 months later, at the time of selection.

3. Booster gilts at day 50 of gestation to prevent the formation of susceptible animals

If the duration of commercially available mlv vaccines does not consistently exceed 4 months, re-vaccination of gilts at 50 days of gestation may be an option to minimize this risk. This recommendation is not to be confused with the 6–60 program. Unlike the 6–60, it is parity-specific and based on the diagnostic data described. Controlled studies have also indicated that 50 days of gestation is a very safe point in which to vaccinate pregnant sows. Finally, because it is "homologous" challenge, transplacental infection will not take place.[5]

4. Diagnostically document the point of infection of the suckling piglet prior to initiating intervention strategies

Based on these data, it must be remembered that shedding occurs sporadically, and at irregular intervals. If susceptible gilts exist within pig populations to the degree of <22%, targeted intervention strategies and monitoring programs that are focused on parity are important. Specifically, monitoring programs must be in place to assess the time the infection occurs in lactating piglets and if there is a parity involvement. Based on these needs, monitoring programs are evolving from simple serologic profiling by stage of production, to sampling of suckling piglets at 2–3 day intervals, along with immunohistochemistry and histopathological examination for the presence of virus and/or lesions in pigs prior to weaning.

With the ability to accurately detect and sequence PRRS viral antigen, practitioners must attempt to better understand the exact point of infection in the life of the pig. This is critical, particularly if piglet vaccination is being used as a means to control the disease post-weaning. While antibody tests have been helpful, the use of PCR and sequencing will provide diagnostic answers with a greater degree of precision. It will also enhance our ability of understanding whether a breeding herd is truly stable, above and beyond the degree that serology and production data can provide.

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