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Editors

W. Christopher Scruton

Stephen Claas

Layout

David Brown

Cover Design

Ruth Cronje, and Jan Swanson;

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Eradication of PRRS: Past, present and future

SA Dee, TW Molitor, HS Joo, and M Bierk

University of Minnesota

Introduction

When Dr. Bob Morrison invited me to give this lecture, he requested that I provide an overview of the work I published prior to coming to the University of Minnesota. Since PRRS has been my sole area of study during that time—and continues to be the focus of my research in the Swine Group—I thought the topic of PRRS Eradication: Past, Present, and Future would be appropriate. Please note that the next 30 minutes will not only be a review of the literature, but also an update on our current investigations and a look into the future of this complex subject.

I witnessed my first case of porcine reproductive and respiratory syndrome (PRRS) on April 24, 1990. The date is easy to remember, owing to the severity of the clinical signs and the subsequent impact that the disease had on a previously productive and profitable swine farm. From that moment, I was consumed by PRRS, and my primary focus in veterinary medicine became the control of this disease. To generate more time in which to work with PRRS, I re-applied to graduate school in 1993, and accepted a faculty appointment in 1999, both at the University of Minnesota College of Veterinary Medicine.

Based on the state of the industry today, one might logically conclude that the effort I have put forth over the last 10 years has been wasted, and that I should consider myself a failure. Some days, I would be the first to agree. At this time, PRRS control in the United States is in a state of total chaos. The biology of the PRRS virus is winning the battle, and perhaps the war. Undetermined methods of entry into negative herds still exist. Mutation, strain variation, and recombination events are taking place at high frequencies. Vaccine efficacy across farms is inconsistent, and the future of modified-live vaccines for the control of PRRS is being questioned. Many infected breeding stock companies still ignore the problem. Those that have uncovered the ugly truth of their status generate catchy phrases to soften the blow, such as “PRRS Stable,” or “Yes, we’re positive but we have non-infectious titers,” or “There are only two types of pig farms: those that have PRRS and those who are going to get it.”

On the other hand, there is a growing segment in the industry, consisting of practitioners, producers, and

seedstock companies who wish to eradicate the disease. Various groups have calculated the cost of the disease within their respective systems, and now realize that they cannot afford to live with PRRS. Seedstock companies are feeling the pressure, both on a national and international basis to produce non-infected pigs for sale. Progressive veterinarians are coming to our group with large-scale virus elimination projects.

With this in mind I would like to begin in 1992, and tell you my continuing story of the “virus you love to hate” and the ongoing hunt for the cure. I will base the lecture on nine selected papers, provide a brief summary of the results, and describe the effect they had on my educational process.

Part 1: The past

Paper one

Title

Eradicating PRRS virus using two-site production and nursery depopulation

Reference

Swine Health and Production, 1993, 1:20–23.

Summary

The development of serologic assays for detection of PRRS virus antibodies allowed practitioners to apply principles of serum profiling to PRRS, to determine the decay pattern of colostral antibodies and detection of seroconversion post-weaning. The Indirect Fluorescent Antibody (IFA) test was the test of choice at the time. This paper summarized a field study in which we attempted to eliminate PRRS virus through segregated production and early weaning at the age of 12–14 days.

Results and implications

- Colostral antibodies were not detectable at 4 weeks of age.
- Seroconversion took place at 6–7 weeks of age.
- SEW will not consistently prevent vertical transmission of PRRS virus.

Paper two

Title

Prevention of the spread of PRRS virus in endemically infected pig populations using nursery depopulation

Reference

Veterinary Record. 1994. 135: 6–9.

Summary

A nursery depopulation protocol was devised and implemented on 3 infected farms. The purpose of the protocol was to prevent horizontal transmission of PRRS virus from older, infected pigs to those recently weaned. Improvements in production parameters and no evidence of seroconversion to PRRS virus was detected 6 months post-depopulation.

Results and implications

- A specific serologic pattern was detected in all 3 farms indicating a lack of vertical transmission, with subsequent horizontal transmission in the nursery.
- PRRS virus spread was prevented and potentially eliminated
- Strategic adjustments in nursery pig flow resulted in improved performance.

Papers three and four

Titles

Evaluation of the effects of nursery depopulation on the persistence of PRRS virus and the productivity and profitability of 34 farms

References

Veterinary Record. 1997. 140: 247–248
Veterinary Record. 1997. 140: 498–500

Summary

Nursery depopulation was carried out on 34 farms in the US. Parameters measured included nursery average daily gain, percent mortality, medication/vaccination costs, feed efficiency and seroprevalence at 10 weeks of age for 12 months prior and 12 months following completion of the protocol.

Results and implications

- Nursery depopulation was a cost-effective protocol for PRRS control in 94% of the cases.
- Nursery depopulation did not consistently eliminate PRRS virus.
- The duration of downtime (7 vs. 14 days) did not affect performance.
- A seronegative breeding herd, as determined by IFA, was essential for the maintenance of a PRRS negative nursery.

Paper five

Title

Recurrent reproductive failure associated with PRRS in a swine herd.

Reference

JAVMA. 205(7): 1017–1018

Summary

A clinical investigation into recurrent reproductive failure was initiated on two matched swine farms. The role of the naïve replacement gilt purchased from an outside source was determined to be essential for propagation of virus spread and clinical PRRS in one farm. The other farm employed an internal replacement program following natural exposure to the virus during the nursery stage.

Results and implications

- Recurrent reproductive failure secondary to PRRS virus infection may be parity related.
- Naïve gilts require proper acclimation to farm-specific PRRS virus strains prior to breeding.
- Introduction of replacement gilts at non-traditional ages (weaning to 25 kg) may improve reproductive performance when PRRS virus is involved.

Paper six

Title

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

Reference

Swine Health and Production. 1994. 3(2): 64–69

Summary

Two methods of managing infected replacement gilts were evaluated for their effect on the PRRS serostatus of infected breeding herds. The methods included internal replacement and the establishment of an off-site gilt developer to raise 4 months of age-staggered gilts.

Results and implications

- Direct introduction of actively infected replacement stock exacerbates existing PRRS problems.
- Both methods of gilt management resulted in significant reductions in breeding herd seroprevalence.

Paper seven

Title

Detecting subpopulations after PRRS virus infection in large breeding herds using multiple serologic tests.

Reference

Swine Health and Production. 4(4): 181–184

Summary

Ten farms, >1000 sows in size, with a history of nursery depopulation failure were selected for this study. Randomly selected sows were serially profiled and assayed using IgM/ IgG IFA and serum neutralizing (SN) tests. Sows were categorized into 1 of 3 subpopulations—not infected, acutely infected, and chronically infected—according to changes in antibody dynamics over time.

Results and implications

- Breeding herd subpopulations persist in chronically infected herds.
- Animals may shift from one subpopulation to another based on the spread of virus over time.
- Management of subpopulations was considered to be a key factor for the control of viral transmission in the breeding herd. Based on this assumption, the field study in paper #8 was initiated.

Paper eight

Title

Attempted elimination of PRRS virus from a seedstock farm by vaccination of the breeding herd and nursery depopulation.

Reference

Veterinary Record. 1998.142: 569–572.

Summary

Modified-live PRRS virus vaccine was used to mass vaccinate an infected breeding herd to eliminate subpopulations and prevent vertical transmission. Horizontal transmission was prevented through the depopulation of the nursery and finishing sites. The farm was serologically monitored on a monthly basis (n=30/site).

Results and implications

- The farm was serologically negative for 15 months post-completion.
- The finishing site became seropositive during month 16 with suspected sources being infected weaners or a breakdown in biosecurity.
- One of 10 piglets IgM positive at 3 weeks of age raised questions about carrier animals and the sample size necessary to detect them.

Part 2: The present

Following completion of the last study, the quest for understanding the role of the carrier sow was initiated. The central hypothesis was that a small subpopulation of infected animals existed within infected breeding herds that were responsible for maintenance of vertical transmission. The ability to detect and remove these “high-risk”

animals became the basis for the test and removal (T&R) protocol and led to the following paper:

Paper nine

Title

Elimination of PRRS virus using a test & removal process.

Reference

Veterinary Record. 1998. 143:474–476.

Summary

A combination of antibody (ELISA) and antigen (PCR) tests were used to identify previously exposed sows. Sera were collected from all breeding animals on a given day. Suspect sows were removed from the herd and the breeding and finishing populations monitored monthly.

Results and implications

- Eight percent of the breeding herd was removed, all of 5th parity or greater.
- Two PCR positive animals were detected, with respective ELISA s/p ratios of .35 and .55.
- The farm remains ELISA negative for 24 months post-completion.

These results spurred an increased level of interest in T&R as a potential strategy for PRRS eradication. As of this writing, a large project involving 10 farms is currently underway with 5 having exceeded a 12-month period following the protocol’s completion. The following tables provide an overview of study farm characteristics, and detailed data from two representative herds:

# of farms in progress	10		
Breeding herd inventory	mean=87 4	range=200 –1500	
Location	MN (n=7)	SD (n=1)	NE (n=2)
# of three-site facilities	n=5		
# of single site facilities	n=5		
Time following completion of T&R	n=12–24 months (5 farms)		
# of infected farms	0		
Approximate diagnostic cost/sow of T&R protocol	\$9.00		

Diagnostics

The diagnostic protocol consists of a combination of ELISA and serum PCR. All animals are screened by ELISA, and all positives (s/p greater than or equal to .4) are removed from the herd. Suspect sows can be allowed

to farrow, then culled at weaning. Another alternative is to move pregnant suspects to another site or, lastly, culled immediately. Due to the many unknowns regarding PRRS virus and boars, several farms have elected to remove all boars immediately and begin purchasing semen and new boars from PRRS-negative studs. Since all farms in this dataset used AI, replacing a small number of boars has not resulted in great hardship or cost.

The actual diagnostic protocol is as follows:

ELISA	PCR	Interpretation	Action
+	+	viremic	remove
+	-	exposed/infected?	remove
-	+	infected	remove
-	-	negative	retain

At this time, it appears that 2–4 serum PCR positives may be detected throughout a breeding herd inventory of approximately 800 sows. If serum PCR is an indication of viable virus, this may suggest that the frequency of actively infected animals that exist in a herd on a given day is small. The biggest weakness of the protocol is the removal of animals which are ELISA+/PCR-. Based on the lack of ante-mortem tests, one cannot distinguish whether animals within this category are carriers and not viremic at the time of sampling, or are merely animals which were infected at one time, but have cleared the virus and present no risk to the herd. Studies are underway to investigate this issue. A summary of diagnostic data from two farms is provided at this time:

	Farm 1	Farm 2
# of sows	825	792
# removed	88	66
ELISA+/PCR+	3	1
ELISA+/PCR-	85	64
ELISA-/PCR+	0	1
ELISA-/PCR-	777	726

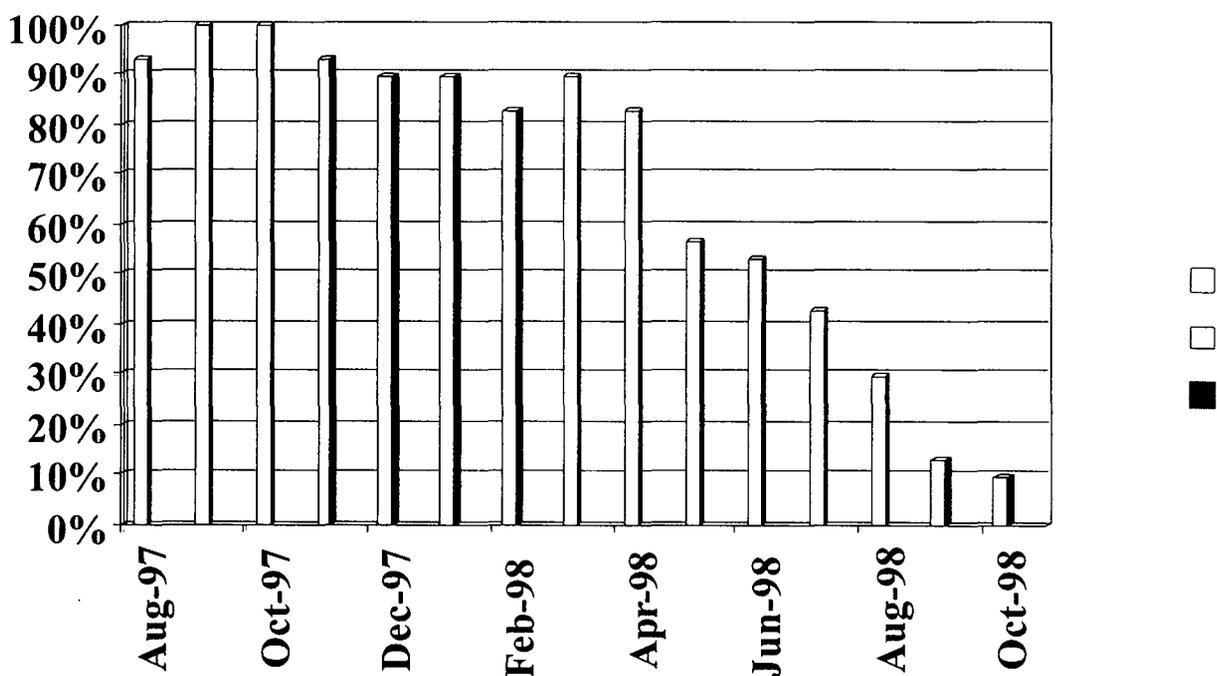
Gilt management

According to data from the herds described above, approximately 6–11% of the breeding herd have been removed. The goal of the T&R protocol is to initiate the whole herd testing when the breeding herd prevalence approaches 10% as determined by ELISA. Surprisingly, we have discovered that a large number of chronically infected farms exist with low breeding herd prevalence. However, based on the aforementioned paper on gilt pool management, the herd prevalence can be manipulated through the addition of replacement gilts. At this time, there appears to be two potential methods, each requiring 14–16 months to prepare a herd for T&R. Both methods require three distinct periods of gilt management, and are outlined as follows (top of next page)

Figure 1 displays the change in seroprevalence over time following implementation of Method 1.

Although the observations are limited to a few herds, there does seem to be a parity effect influenced by the gilt management scheme. Herds that utilize Method 2 have demonstrated that primarily older sows (5+ parities) are re-

Figure 1. Change in breeding herd seroprevalence over time following herd closure with Method 1



	Method 1	Method 2
Phase 1	Close herd (load with gilts)	Close herd (load with gilts)
Duration	4 months	4 months
Phase 2	Introduce naturally exposed gilts	Introduce vaccinated naïve gilts from finisher after 60 day quarantine
Duration	4 months	4 months
Phase 3	Introduce naïve gilts	Introduce naïve gilts
Duration	6 months/800 sows	6 months/800 sows

moved. In contrast, use of Method 1 results in not only removal of older sows, but Parity 2 as well. The difference is due to the fact that the modified-live virus vaccines used in these farms (RespPRRS Repro, BI-NOBL) do not persist for extended periods. This is in contrast to Method 1 that uses naturally infected females, resulting in extended periods of wild-type viral persistence. Finally, removed animals tend to localize as clusters or individuals (**Figure 2**). Clustered animals also have ELISA data that are normally distributed, possibly indicative of previous viral transmission over a short distance.

Part 3: The future

Before we get too far ahead, it is important to summarize what we have learned from our work up to this point in time. As I see it, these studies have brought forth three concepts:

- The PRRS status of the breeding herd drives the system.
- Gilt management should focus on the introduction of younger animals in fewer groups, less frequently throughout the year.
- The ability to conduct a regular partial depopulation of the nursery and finisher should be designed into every swine facility.

Figure 2. Distribution of ELISA S:P Ratios within Infection Foci

Section A

0.0	0.08	0.26	.49	1.37	.51	0.21	0.0
Serum (+)							

Section B

0.0	0.32	0.71	1.15	.30	0.26	0.0	0.0
Tissue Positive							

Section C

0.0	0.41	0.95	1.23	.75	0.39	.58	0.0
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Regarding the future of PRRS eradication, many questions still remain. The T&R protocol must be applied over more farms, and monitoring must continue for a minimum of 18–24 months following completion of the T&R protocol. The true prevalence of infected breeding swine needs to be determined. How can we identify and distinguish between “high risk” and “low risk” animals? The relationship of a positive PCR test on sera or tissue and the presence of viable, infectious virus needs further clarification. Ante-mortem tests, such as tonsillar biopsy need to be inspected in order to calculate levels of sensitivity and specificity. Can less costly diagnostic protocols be devised? Finally, the role of external stimuli, risk factors, and on-farm conditions that enhance replication of persistent virus need identification, as does the amount

of virus necessary for transmission and infection of neighboring swine require quantification.

I hope you have enjoyed the “history lesson” and our vision of PRRS eradication. We are in the process of initiating studies to answer these questions. We look forward to sharing the remainder of our voyage into the “Great Unknown of PRRS” as we return from yet another adventure! Thank you for your time, attention, and support.

