CASE REPORT

Eradicating porcine reproductive and respiratory syndrome (PRRS) virus using two-site production and nursery depopulation

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Summary: *We attempted to eradicate porcine reproductive and respiratory syndrome (PRRS) virus using early weaning and two-site production. We chose this method over depopulation or test-and removal procedures because the infection was highly prevalent in the breeding herd (63%), the breeding herd was very valuable (a seedstock operation), and because another farm was concurrently available. Modified medicated early weaning (at 14 days) was also used to try to eradicate other chronic respiratory diseases detected in the herd.*

Samples taken from pigs raised on-site were positive by indirect-fluorescent antibody (IFA) assay. Animals tested at the off-site facility (90 head) were negative by IFA for 4 months, but then infection occurred. PRRS virus was isolated from one pig in the nursery and all pigs tested were seropositive to PRRS virus. The source of the virus was thought to be a carrier pig.

No evidence of pneumonia was detected at slaughter, and this observation in conjunction with a lack of clinical signs indicated that in this high health herd, PRRS infection was subclinical. Five to 6 months after infection, IFA titers in 5- to 6-month-old pigs decreased from 1024 to undetectable (0–16) over 5 months. Nursery pigs were seronegative at weaning and seroconverted to PRRS virus at 8–10 weeks of age. Because the nursery appeared to be the area where virus was recirculating, we attempted to eradicate PRRS by depopulating the nursery. After normal pig flow resumed, all pigs tested have remained negative for PRRS antibody.

Few practitioners in the United States have been able to completely avoid an experience with a herd infected with porcine reproductive and respiratory syndrome (PRRS). While our profession has made great strides in identifying, characterizing, and diagnosing PRRS virus in the past few years,[1-3] we unfortunately still lack both a vaccine to prevent the disease and adequate knowledge of its epidemiology to be sure how to proceed with an eradication program once a herd is infected. We investigated the feasibility of eradicating PRRS virus from a herd using early weaning and multi-site production.

A 140-sow farrow-to-finish seedstock herd became infected with PRRS virus in November 1990. The farm, which used all-in/all-out animal flow in both the farrowing rooms and the nursery, experienced high levels of stillborns (14%), mummified fetuses (31%), preweaning mortality (44%), and an increased level of pneumonia in pigs in the finishing barn. In the nurseries, *Escherichia coli* scours and *Streptococcus suis* meningitis became more prominent despite adequate vaccination programs. All serum samples from the breeding herd were negative for known reproductive pathogens, including porcine parvovirus, pseudorabies virus (PRV), brucellosis, leptospirosis, and encephalomyocarditis virus (EMCV). As soon as the producer observed clinical signs consistent with PRRS, he closed the herd and sold animals only to slaughter.

Due to the potential legal ramifications of selling PRRS-infected seedstock, we decided to attempt an eradication program. The best alternative appeared to be using multi-site production because:

- the breeding herd was too valuable to depopulate;
- the high percentage of positive breeding animals made test and removal procedures impractical; and
- the cost and time involved in testing individual offspring prior to sale was prohibitive.

A second site was already available, located 2.4 kilometers from the home farm. Other diseases present in the herd (such as atrophic rhinitis, sarcoptic mange and *Actinobacillus pleuropneumonia*) had deterred seedstock sales in the past. Multi-site production along with modified medicated early weaning (MMEW) can eliminate such pathogens[4] and the opportunity to eradicate several diseases at once was highly attractive.

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Before and during experiment

Facilities

We recommended that the nursery and grower be established on the second site and the home farm be confined strictly to breeding, gestation, and farrowing. The producer rented contract finishing sites within a 10-mile radius to provide enough finishing space to raise all pigs to 230 lb.

Biosecurity

Each site had separate labor forces and feed delivery vehicles. Employees showered and changed clothes prior to entry. Weaned pigs passed from the farrowing rooms through a PVC tube into a van on the outside of the barn, which was driven by the individual from the off-site nursery, thereby eliminating any contact between the employees. After delivering the pigs to the nursery, the van was thoroughly washed and disinfected and was not used for 5-7 days.

Seroprevalence: April 1992

In order to determine the seroprevalence of PRRS in the herd, blood samples were collected from 30 pregnant sows/gilts randomly selected from the breeding herd and 64 6- to 7-month-old pigs randomly selected from the finishing herd. Samples were centrifuged at 1500 rpm for 10 minutes. The serum was separated and frozen at -20°C. The samples were sent to the University of Minnesota for IFA testing and virus isolation.[3]

Of the 30 breeding herd samples, 19 (64%) were seropositive for PRRS virus. Of the 64 finishing samples, 34 (54%) were seropositive for PRRS virus. We then attempted to isolate virus from six of the seropositive finishing gilts with the highest IFA titers (1024). Five of these six gilts (83%) had detectable viremia.

Passive immunity: June 1992

Because the appropriate weaning age at which to eradicate PRRS virus from pigs was unknown, it was critical to determine the duration of passive immunity and whether viremia could be detected in pigs prior to weaning. Blood samples were collected from:

- 30 suckling piglets ranging in age from 3-20 days; and
- 30 weaned pigs ranging in age from 3-10 weeks.

These samples were processed in the same way as those taken to determine seroprevalence. Virus was not detected in any of the suckling piglets (0 of 30). Pigs became IFA negative at 4 weeks of age and appeared to seroconvert at 6-7 weeks of age. Forty-eight percent (14 of 30) of the weaned pigs were seropositive at 6-7 weeks of age.

Performance and slaughter monitoring

To assess growth performance, 20 randomly selected pigs from both the on-site and off-site nursery were weighed at 2 weeks of age, 10 weeks of age, and at slaughter.

Bimonthly, the snouts of 30 hogs at slaughter were visually checked for turbinate atrophy and their lungs were checked for lesions consistent with pneumonia.

Phase I: multi-site protocol

Methods

Based on previous data suggesting that colostral antibodies to PRRS virus persist at least 4-5 weeks,[4] pigs were weaned at 14 days of age to simultaneously eradicate *A. pleuropneumoniae*, toxigenic *Pasteurella multocida* type D, and sarcoptic mange. We used the following MMEW protocol:

- Sow Vaccination (5 & 2 weeks prefarrowing)—
  A. pleuropneumoniae
  Bordetella bronchiseptica
  Toxigenic *P. multocida* types A and D
  Erysipelothrix rhusiopathiae
  E. coli
- Piglet Medication—
  Day 1: ½ ml oxytetracycline (100mg), ½ ml ivermectin (.27%)
  Day 7: 1 ml oxytetracycline (200mg), 1 ml ivermectin (.27%)
  Day 14: 1 ml oxytetracycline (200mg), 1 ml ivermectin (.27%)
- Postweaning medication—
  Tiamulin (35gm/ton) for 7 days
  Trimethoprim(80mg)-sulfamethaxozole (400mg) 30 ml/4.54 liters stock for 7 days
- Vaccination at 8 and 10 weeks of age—
  Haemophilus parasuis
  E. rhusiopathiae

Pigs were moved to the off-site nursery for 12 weeks. They were then moved to contract finishing barns located on other farms until marketing age. As a control, 30 pigs that went through the MMEW procedure were weaned into the conventional (on-site) nursery. These pigs were tested for antibodies to PRRS by IFA at 8 weeks of age, while 30 MMEW pigs raised off-site were randomly selected for IFA testing every month for 6 months. These off-site pigs ranged in age from 8 weeks to 6 months.

Results

All pigs tested (90 head) from August to October 1992 raised in the off-site nursery were seronegative for PRRS antibodies. All pigs tested that were raised in the on-site nursery (30/

Table 1.— Summary of PRRS IFA antibody titers and virus isolation over time

Location tested	Date	Number of swine tested for serum antibody	Percent testing positive	IFA range	Number of swine tested for viremia	Percent testing positive
Gestation	4/92	30	63	0–1024	(NT)	0
	11/92	30	25	16–64	(NT)	0
	12/92	30	5.3	16–64	(NT)	0
	1/93	30	5.3	16–64	(NT)	0
Suckling piglets	6/92	30	100	16–256	30	0
	11/92	40	9	0–16	pool	0
	12/92	40	0	0	pool	0
On-site nursery	6/92	30	48	0–1024	pool	0
	8/92	30	100	1024	pool	0
Off-site nursery	8/92	30	0	0	30	0
	9/92	30	0	0	10	0
	10/92	30	0	0	10	0
	11/92	30	100	256–1024	30	3.3
	12/92	10	100	256–1024	pool	0
	1/93	10	100	256–1024	pool	0
	2/93	10	100	256–1024	pool	0
On-site finisher	4/92	64	53	0–1024	6	83
Off-site finisher	12/92	10	100	256–1024	pool	0
	1/93	10	100	16–64	pool	0
	2/93	10	80	0–16	pool	0

NT = not tested; pool= 30 samples pooled together resulting in 5 testable samples

30) were seropositive to PRRS at 8 weeks of age (Table 1). Approximately 1200-1400 head went through the multi-site system during this period and all of the off-site pigs tested were seronegative using IFA techniques.

Phase 2: reinfection

In November 1992, it became serologically evident that PRRS virus had infected pigs in the off-site nursery and grower (Table 1). Thirty of 30 pigs tested had IFA titers to PRRS virus. Virus was isolated from one serum sample of 30. We conducted further testing in an attempt to discover how the virus had entered the off-site facility (Table 1).

Methods

Weekly groups of 10 piglets were blood tested at weaning (14 days of age) just prior to being moved to the offsite nursery. To assess the possibility that the labor force was transmitting the virus, the tonsils of employees on each site were swabbed twice daily for 7 days. A cotton-tipped swab was rubbed 3–4 times over the surface of the tonsil, placed in Minimal Essential Media, frozen at -20°C, and sent to the University of Minnesota for virus isolation, using the method previously described.[3] Two seronegative sentinel pigs (from a herd with no clinical or serological history of PRRS) were placed in the finishing site in a pen near the known positive pigs and were tested for the presence of PRRS antibodies.

To monitor any change in the serostatus of offsite pigs, 10 finishing pigs and 10 nursery pigs were identified with ear tags and bled in December, January, and February. Samples were processed as previously described.

Results

All tonsil samples were virus-negative and all blood samples from piglets tested at weaning were negative for PRRS antibodies and virus. PRRS antibody titers in finishing animals declined from 1:1024 in November to undetectable (1:0–16) at the end of February; however, all nursery pigs tested maintained PRRS titers of 1024, indicating constant viral recirculation within the population. Neither of the sentinel pigs placed in the finisher seroconverted (Table 1).

Despite the presence of PRRS virus infection in the offsite nursery, there was no evidence of reduced performance or elevated mortality in these pigs. All samples tested at the time of this writing have been serologically negative to both *A. pleuropneumoniae* (71 samples) and *Mycoplasma hyopneumoniae* (104 samples) by ELISA. Slaughter check data indicated no evidence of pneumonia, all snouts were grade 0 (6–9mm turbinate atrophy) and pigs averaged 103 kilograms at 5 months of age. Mortality from weaning to market weight was 4.0%.

Phase 3: depopulation

In April 1993, we completed a serologic profile of pigs in the off-site nursery. We collected serum from different age group (ranging from 2-week-old to 10-week-old pigs) every 2 weeks and used IFA techniques to test them for PRRS virus antibody. Test results indicated that all samples were negative until pigs reached 8-10 weeks of age. Thirty percent of the 8- to 10-week-old pigs were positive with titers ranging from 1:64–1:256. Similar results were observed when sampling was repeated in May 1993. Because there appeared to be a decreasing prevalence of seropositive pigs in the nursery, we attempted a nursery depopulation. This phase of the experiment also included

biweekly monitoring of the finishing sentinel pigs put in place in phase 2.

Methods

All nursery pigs were moved to a contract finishing site. This site contained only barrows, thereby preventing exposure of the finishing gilts to virus via potential carrier nursery pigs.

Newly weaned litters were housed in straw-bedded pens.

The nursery was pressure washed with hot water (>94°C) and disinfected with formaldehyde three times. All pits were pumped between each washing. The building was left empty for 14 days.

Thereafter, normal pig flow resumed into the cleaned facility. When the first pigs reached 8 weeks of age, and again 1 month later, 30 serum samples were collected and tested for PRRS antibodies using IFA techniques.

Results

All samples, as of July 1993, have been negative for PRRS antibodies in the repopulated nursery.

Discussion

We were never able to determine the means of viral entry into the nursery. The tonsil swab results exonerate the labor force, and fomite transmission also appears unlikely due to the strict biosecurity measures. However, the virus could have been introduced to the off-site pigs via carrier pigs or aerosol transmission.

Aerosol transmission of PRRS virus has been suggested over a distance of 3.2 kilometers.[5] Since the nursery site was located 2.4 kilometers from the sow facility, this may have occurred. However, *M. hyopneumoniae* has been reported to travel 3.2 kilometers in the air,[6] yet all pigs tested (85 head) have been serologically negative to *Mycoplasma* with no gross lesions at slaughter, despite a positive breeding herd. Therefore, we suspect that virus entered the off-site nursery via a carrier pig.

Despite the initial failure, we have an early indication that it may be possible to eradicate PRRS virus if multi-site production is combined with depopulation of the nursery. Partial depopulation of the nursery or grow/finish areas has been documented to be an effective way to improve the health status of the pigs without adversely affecting cash flow or pig flow.[7] Depopulating the nursery may have removed the chronic carrier animals, thereby preventing virus from recirculating to susceptible weaners. We shall continue to test 8- to 10-week-old piglets, which should indicate whether nursery depopulation is a viable eradication technique over the long term.

We have continued to test finishing gilts in this herd. We have consistently observed declining PRRS titers and have failed to isolate virus from serum of selected animals. Placing these animals in contact with known negative pigs has not resulted in transmission of the virus. If we can prevent adding any exposed animals to the finisher, we hope this trend will continue. Thus far, PRRS virus appears not to have spread to the older finishing pigs.

References

1. Dial GD, Hull RD, Olson CC, Hill HT, Erickson GA. Mystery Swine Disease: Implications and needs of the North American swine industry. *Proc. LCI Mystery Swine Disease.* 1990, 3–6.

2. Wensvoort G, Terpstra C, Pol JMA, et al. Mystery Swine Disease in the Netherlands: the isolation of the Lelystad virus. *Vet. Quarterly.* 1991; 13: 121–130.

3. Frey M, Eernise K, Landgrat J, Pearson J, Chladek D. Diagnostic testing for SIRS virus at the National Veterinary Services Laboratories. *Proc. International Symposium on Swine Infertility and Respiratory Syndrome. AASP Newsletter* 4(4) 1991: 31.

4. Collins, J., personal communication, April 1992

5. Connor J. Elimination of specific agents from herds: elimination by medication. *Proc. Minnesota Swine Conference for Veterinarians.* 1992: 151–157.

6. Clanton C. New routes to a high health herd. *National Hog Farmer,* 1992; 37: 10–12.

7. Vansickle J. Put a lock on herd health. *National Hog Farmer,* 1992; 37: 15–16