CASE REPORT

Cessation of porcine reproductive and respiratory syndrome (PRRS) virus spread in a commercial swine herd

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Summary: *We report a case of porcine reproductive and respiratory syndrome (PRRS) virus infection that appeared to spontaneously stop spreading. We compared serologic profiles of this 250-sow farm (herd A) with those of another 300-sow farm (herd B), both with a previous history of PRRS. In November 1992, we collected serum samples from pigs of different age groups (from 1 week to 3 years old) from both herds. We tested PRRS virus antibody titers by an indirect-fluorescent antibody (IFA) method. IFA titers of ≥1:16 were detected in 20 of 129 pigs (15.5%) from herd A and 73 of 158 pigs (46.2%) from herd B. In April 1993, none of 30 pigs between 4-26 weeks of age from herd A were seropositive, while all 60 pigs of the same age group from herd B were seropositive. A group of 15 gilts in herd A, introduced in January 1992 from a farm showing no clinical signs of PRRS, remained seronegative. No PRRS virus was isolated in April 1993 from 30 serum samples from pigs 5-10 weeks old in herd A, while virus was isolated from 4 of 28 sera from pigs of a similar age in herd B. PRRS virus stopped spreading in herd A, while the infection is still endemic in herd B.*

Porcine reproductive and respiratory syndrome (PRRS) virus infection occurs in swine farms throughout the United States[1] and Europe.[2,3,4] The virus causes reproductive failure in pregnant sows and respiratory disease in young pigs. Following an initial outbreak of PRRS, the viral infection appears to be endemic in nursery pigs because investigators have been able to repeatedly isolate virus from nursery pigs for many months.[5]

No investigators have reported a case of a swine farm spontaneously returning to PRRS virus-free status. Here, we report a case in which PRRS virus infection appeared to spontaneously stop spreading in a commercial swine herd (herd A) without purposeful intervention. We include serologic data from a second herd (herd B) that has experienced continued virus spread to compare with the serologic profile from herd A.

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Farm history

Herd A

Herd A is a 250-sow confinement herd that had been repopulated in the spring of 1989. Its first farrowing took place in the fall of 1989. The breeding animals were hand mated every 2 weeks in a crated breeding/gestation facility. After the piglets were weaned at 28 days of age, they were moved into:

- an all-in/all-out first-stage nursery for 10 days; then
- into a continuous flow nursery for 28-35 days; and then
- into continuous flow grower/finisher buildings.

New breeding stock was obtained from a single source and was kept in on-farm quarantine before being placed in the herd.

Between January and March of 1990, the herd experienced clinical signs typical of PRRS, including a low farrowing rate, increased stillbirths and mummies, and increased preweaning mortality. We first diagnosed PRRS virus infection in November 1992 by testing serum samples that had been collected in November 1990. Through 1991 and 1992, production values gradually improved until they reached the ranges recorded prior to infection. At the time of this writing, no PRRS-related disease problems are occurring in any stage of the operation.

Herd B

Herd B is a 300-sow, farrow-to-finish farm. Sows were housed in a breeding/gestation building and bred and farrowed on a weekly basis. Piglets were weaned at 21 days of age into an all-in/all-out first stage nursery and then transferred to an all-in/all-out second-stage nursery. The producer then continuously flowed pigs into the grower/finisher rooms. The producer selected replacement gilts from the finishing barn. In early 1992, a new source of seedstock was brought directly

into the unit and commingled with the existing breeding herd. Shortly after they arrived, the herd experienced an outbreak of PRRS virus infection. We isolated PRRS virus from weakborn and nursery pigs.

Methods

In November 1992, we collected 129 blood samples from pigs of 13 different age groups (1 week to 3 years old) from herd A and 158 samples of 16 different age groups in herd B (Figure 1). No more than two suckling pigs per litter were bled. We took additional samples in December 1992 and April 1993 from herd A. We used an indirect fluorescent antibody (IFA) test to detect antibody titers to PRRS virus as previously described.[6] We also used swine alveolar macrophages to examine the serum samples for the presence of PRRS virus.[7]

Results

Herd A

In November 1992, 20 (15.5%) of 129 sera tested were seropositive (Table 1). Of the 20 positive samples, 19 (95.0%) had IFA titers of 1:16 and 1 (5.0%) had an IFA titer of 1:64 (Table 2). Seropositive pigs were distributed throughout the 16 age groups (Figure 1). None of the 30 pigs collected in December 1992 or the 30 pigs sampled in April 1993 were seropositive. Fifteen gilts that had been introduced in January 1992 (from a farm with no history of PRRS) remained seronegative. No virus was isolated from the 30 sera of 5- to 10-week-old pigs that were collected in April 1993.

Table 1

PRRS virus antibody in pigs of different age groups in herds A and B.

Herd	Age group	Nov '90	Nov '92	Dec '92	Apr '93
A	1-3 weeks	30/30	7/30	NT	NT
	4-8 weeks	NT	4/39	NT	0/20
	10-26 weeks	NT	0/20	0/30	0/10
	> 8 months	6/6	9/40	NT	NT
	Total	**36/36**	**20/129**	**0/30**	**0/30**
B	1-3 weeks	NT	4/30	NT	NT
	4-8 weeks	NT	3/40	NT	30/30
	10-26 weeks	NT	57/58	NT	30/30
	> 8 months	NT	9/30	NT	NT
	Total		**73/158**		**60/60**

Number of seropositive pigs by IFA test/Number of pigs tested.
NT= not tested.

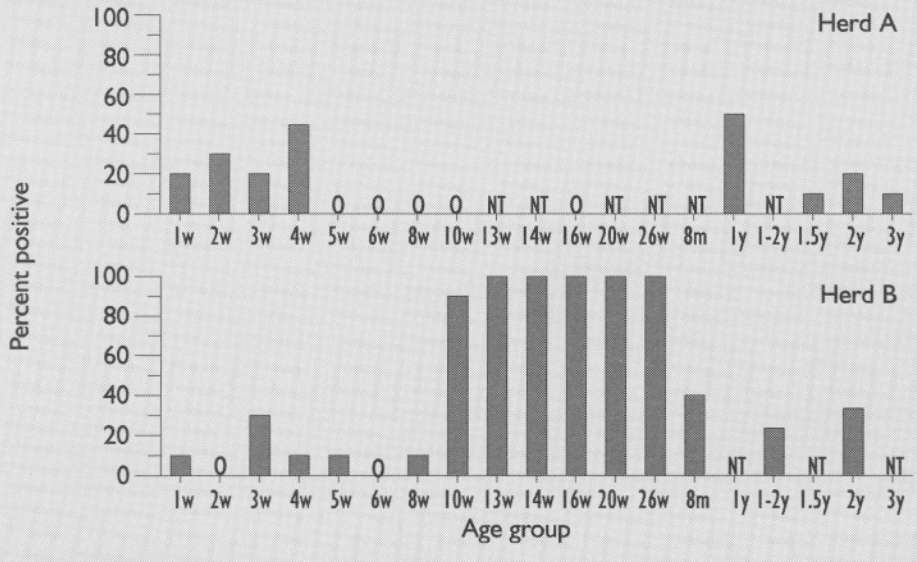
Herd B

In November 1992, 73 (46.2%) of 158 sera had positive antibody titers (≥ 1:16) to PRRS virus (Table 1). Of the 73 positive samples:

- 8 (11.0%) had IFA titers of 1:16;
- 22 (30.1%) had IFA titers of 1:64;
- 38 (52.1%) had IFA titers of 1:256; and
- 5 (6.8%) had IFA titers of ≥1:1024 (Table 2).

In six of the 10- to 26-week-old groups, more than 90% of pigs had positive antibody titers (Figure 1). Fifty seven (78.1%) of the 73 seropositive pigs were in these six age groups. All 60 pigs between 4-26 weeks of age collected in April 1993 were seropositive. We isolated PRRS virus from four of the 28 serum samples we collected in April 1993 from 5- to 10-week-old pigs.

Figure 1



Distribution of PRRS virus antibody-positive pigs in different age groups in November 1992 (herd A — top; herd B — bottom).

Discussion

The seropositive pattern of herd B is typical of a herd endemically infected with PRRS virus. There were probably low levels of maternal antibody in pigs up to 5 weeks old. Active antibody began to appear at 8 weeks of age and declined at 8 months of age. This antibody pattern is typical of swine herds that are endemically infected with PRRS virus.[5,8] When serodiagnosing PRRS with a small sample size, you should sample

Table 2

PRRS virus antibody titer distribution for serum samples collected in November 1992 from herds A and B.

Herd	Age group	No. of sera positive/tested	PRRS virus antibody titer <1:16	1:16	1:64	1:256	>1:1024
A	1-8 weeks	11/69	58	11	-	-	-
	10-26 weeks	0/20	20	-	-	-	-
	≥8 months	9/40	31	8	1	-	-
	Total:	**20/129**	**109**	**19**	**1**	**-**	**-**
B	1-8 weeks	7/70	63	4	2	1	-
	10-26 weeks	57/58	1	1	15	36	5
	≥8 months	9/30	21	3	5	1	-
	Total:	**73/158**	**85**	**8**	**22**	**38**	**5**

pigs between the ages of 10–26 weeks to achieve a high level of confidence of detecting positive pigs, if present.

In contrast, none of 50 sera from five age groups (between 5 and 16 weeks of age) in herd A were seropositive. We believe that PRRS virus had stopped spreading in herd A, particularly because sentinel gilts did not seroconvert and we failed to isolate virus from nursery pigs. All replacement gilts from the source that were introduced into the herd during the first quarter of 1992 have farrowed twice with normal litters and are still negative by IFA testing at this time.

It is difficult to identify specific factors that may have contributed to the cessation of virus spread in herd A. However, we suspect that all-in/all-out pig flows with 2-week intervals from the farrowing house to the nurseries were important. A recent report[9] suggests that a 2-week interval in the nursery causes less opportunity for pig-to-pig transmission of PRRS virus than a 1-week interval did. In addition, the herd-A producer kept replacement gilts in strict isolation and only introduced them to the breeding herd at farrowing. Our observations suggest that the spread of PRRS virus may be controlled by:

- quarantine of incoming stock;
- all-in/all-out pig flows; and
- a 2-week interval in the nursery.

Further research is needed to test this hypothesis.

Acknowledgments

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