An attempt to eradicate porcine reproductive and respiratory syndrome virus (PRRSV) after an outbreak in a breeding herd: eradication strategy and persistence of antibody titers in sows

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Summary

A sow multiplier herd of high health status experienced an outbreak of porcine reproductive and respiratory syndrome (PRRS). In an effort to eradicate the PRRS virus, breeding animals were deliberately infected with the farm strain by exposing them to whole piglets and minced tissues of piglets that died in the farrowing crates. The herd was then closed for 23 weeks. No clinical signs were observed after the initial outbreak and there was no evidence of virus circulation in the herd, verified through serological testing by PRRS ELISA, during the 2 years after the eradication attempt. Approximately one third of sows present at the time of the outbreak were still seropositive 20 months after the deliberate infection.

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Porcine reproductive and respiratory syndrome (PRRS) is one of the most significant swine diseases worldwide. Reproductive and particularly respiratory problems associated with PRRS may persist for months or even years.[1,2] Several methods have been used to eliminate the virus (PRRSV) from infected swine farms.[3–7] A test and removal procedure was reported by Dee et al[3,4] to be effective in several farms. Torremorell et al[5] also had success eliminating the virus by changing the pig flow and by introducing PRRS-negative replacements into PRRS-positive herds. In Denmark, Hassing et al[6] successfully used an eradication strategy based on partial depopulation. Plomgaard[7] reported eradication of the virus by temporarily closing the herd and eliminating PRRS-negative animals, so that only PRRS-positive and immune animals remained on the farm. We evaluated an eradication program initiated while a herd suffered an acute PRRS outbreak.

Case description

The farm studied was a 570-sow, three-site multiplier herd (ie, the facility raised and sold replacement gilts). The herd was of high health status and was populated with sows and boars from a PRRS-negative source. Blood samples from case herd sows were obtained in June (24 sows), October (22 sows), and early December (26 sows) of 1998, and had average PRRS ELISA sample:positive (S:P) ratios of 0.01, 0.00, and 0.01, respectively (HerdCheck PRRS IDEXX ELISA; IDEXX Laboratories, Westbrook, Maine). An acute outbreak of PRRS, confirmed by serology, polymerase chain reaction (PCR), and isolation of the virus, occurred in December 1998. The virus was isolated and the sequence of open reading frame 5 confirmed that it was a field strain. No direct source of contamination could be found. Both the herd supplying gilts and boars and the boar stud supplying semen remained PRRS-negative. The sow farm was relatively close to other swine farms of unknown health status, and to a road used by trucks transporting pigs. It was hypothesized that contamination might have come from one of these sources.

As the producer wished to eradicate the virus so that sales of PRRS-negative replacement females could be resumed as quickly as possible, the following program was carried out.

In January 1999, as many gilts as possible (41) were introduced, and the herd was then closed. All breeding boars were culled, and all matings were by artificial insemination. An attempt was made to deliberately infect all gilts and sows with the PRRSV field strain. Sows showing clinical signs (eg, abortions) were moved to parts of the buildings where no clinical signs had been observed. On one occasion during the month of January, minced tissues of dead piglets from the farrowing crates were mixed with the feed of lactating sows and sows in the breeding area. Four times within the month, one whole piglet that died of PRRS in the farrowing area was placed in each gestation pen.

The 24 sows tested by PRRS ELISA in April 1999 were positive (values ≥0.4 were considered positive) and had an average S:P ratio of 1.22. Sixty-eight of the 70 sows tested in May were positive (average S:P ratio 1.01). The two negative sows had S:P ratios of 0.32 and 0.15. Since values this high had rarely been found in the herd before the outbreak, and since clinical signs had first been observed 5 months previously, it was hypothesized that these two animals were in the declining phase of their antibody response. Testing 94 of 520 sows (the average inventory in April and May 1999) allowed detection of one negative animal with a confidence level of >80%, if the prevalence of negative animals was ≥2%.

Beginning in July 1999, PRRS-negative gilts were introduced into the herd on a regular basis. Between April 1999 and October 2001, no clinical signs suggestive of PRRS were observed. Blood samples from

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gilts introduced into the herd were obtained on six occasions, the last time in May 2001, and all tested negative for PRRS (IDEXX ELISA). Five to 13 females that had been in the herd 2 to 16 months were tested on each occasion. In September 2000, 15 piglets weaned at about 18 days of age were mixed with piglets from a PRRS-negative herd. When tested at 10 weeks of age, all 15 piglets were negative by PRRS ELISA.

The apparent lack of virus circulation in this herd made it possible to examine the long term serological response of sows to the initial challenge with a field strain of PRRSV, without the interpretation problems associated with re-exposure of sows to the same strain, to a mutation of that strain, or to other strains of PRRSV. Twenty-five sows tested serologically in May 1999 were tested again in September 2000, 20 months after deliberate contamination of the herd. Nine sows (36%) were still positive (average S:P ratio 0.42, range 0.03 to 2.01).

The two positive sows with the highest S:P ratios (1.06 and 2.01) in September 2000 were tested again in January 2001 (S:P ratios 1.19 and 1.87) and May 2001 (S:P ratios 1.09 and 1.64). These two sows were still clearly seropositive 28 months after the presumed exposure date. The May 2001 samples were also tested by indirect fluorescent antibody (IFA) and both were positive (titer 64). The sow with the S:P ratio of 1.09 in May was culled in June. Samples of lungs, spleen, and tonsils, and tracheobronchial, mesenteric, and superficial inguinal lymph nodes, were pooled and tested by PCR and found to be negative for PRRSV.

Discussion

Several months after completion of the eradication attempt, the farm became a commercial facility selling market hogs instead of a multiplier unit selling replacement gilts, and the need for serological testing on a regular basis was thus reduced. Although the number of samples tested to determine that introduced PRRS-negative gilts had remained negative was not adequate to reach definitive conclusions, testing was conducted over a long period of time (October 1999 to May 2001), always with negative results. This does suggest that no virus circulation occurred in the herd after the eradication attempt. Nevertheless, definitive proof that the virus is no longer present on the farm will require that the herd remain negative after all sows present at the time of the outbreak have been culled.

Lager et al[8] showed that gilts experimentally infected with a field strain of PRRSV were totally protected (no clinical signs, no viremia, no seroconversion) when challenged with the same strain 200 to 604 days later. The hypothesis behind the strategy used in the case herd was that if all animals came in contact with the PRRSV field strain as quickly as possible, with no new additions of susceptible pigs, they would all become immune to it and would eventually stop shedding the virus.

Several investigators have studied the length of time after infection that animals remain carriers of PRRSV or shed the virus and contaminate other pigs. In a recent study, PRRSV was detected by bioassay in two of ten pigs 150 days after experimental infection.[9] Wills et al [10] isolated the virus from oropharyngeal samples up to 157 days after experimental challenge. However, hypothetically, an animal can carry a virus for extended periods of time, without shedding it and transmitting it to other pigs. Zimmerman et al[11] showed that sows were able to transmit PRRSV 99 days after experimental infection. In a study by Wills et al,[12] experimentally infected pigs were able to transmit PRRSV for no more than 69 days. Benfield et al[13] showed that pigs infected in utero transmitted the virus at 64, 84, 98, and 112 days of age, but not at 260 days of age.

The strategy described in this case report resembles that described by Plomgaard,[7] in that it was initiated during an acute outbreak, and the goal was to have left on the farm only sows that presumably were immune to the field strain. However, in this case, the strategy included deliberate infection of the animals, introduction of as many gilts as possible at the start of the program to reduce the financial impact of the subsequent closure period, a shorter closure period (23 weeks instead of 30), and, finally, less than 100% of sows tested to determine whether they had contacted the virus. Dee et al[3,4] reported a successful test and removal procedure in breeding herds that had been infected with PRRSV 12 to 24 months before. Seroprevalence in these herds was 6 to 12% before the test and removal program was initiated. Immediately after all sows and boars had been tested both by serology and PCR, the positive animals were culled, leaving a population of negative sows and boars. Since the farm in the present case was acutely infected, a considerable amount of time would have passed before such a low seroprevalence was achieved, and the herd owners preferred to try a strategy that might allow them to sell PRRS-negative breeding stock earlier.

It could be argued that deliberate infection of all animals on the farm is a debatable and risky procedure, and that the outcome might have been the same by just closing the herd for 23 weeks. The feedback method might have exacerbated the losses related to PRRSV and, potentially, to other pathogens. However, as the study farm was of high health status, the role of other pathogens was of less concern, and it was thought that all animals might not contact the virus in the desired period of time if the outbreak was left to follow its natural course. Terpstra et al[14] showed that during an outbreak, some sows may escape infection, remain susceptible, and contribute to the long term persistence of the virus in the herd. In addition, having all animals infected in a short time might increase the chances of successful eradication because of the period of PRRSV excretion after infection. If, for example, virus is rarely excreted for more than 150 days, the possibility that any animal in a herd is shedding the virus after day 150 should be reduced when all animals are infected on day 0, compared to having some animals infected on days 30, 90, or later.

It is not possible to predict with accuracy what the losses associated with PRRS would have been if the outbreak had been left to run its natural course. Abortions were reported in about 2% of the sows, although more may have occurred early in gestation and were not reported. One percent or less of the sows died, and the number of piglets born dropped from 5779 for the period June to November 1998 to 3482 for the period January to June 1999.

Although the results associated with this eradication strategy appear promising, only one herd was involved. The case herd was of high health status and was probably infected with only one strain of PRRSV. Data from other farms are needed before the reported protocol can be considered a

safe and effective method of PRRSV eradication.

Few studies have looked at the long-term persistence of antibodies after challenge with PRRSV. Assuming a constant rate of antibody decay, Yoon et al[15] estimated that ELISA antibody titers would approach the lower limit of detection on day 137 post infection. Benfield et al[16] reported that the antibody kinetics of the PRRS ELISA, IFA, and immunoperoxydase monolayer assay tests were similar, and that antibodies reached their maximal titers 30 to 50 days post infection, then gradually declined and reached low or undetectable levels about 4 to 6 months post infection. Using the IDEXX ELISA test, Wills et al[10] showed that three of four pigs were still seropositive 213 days post challenge. Lager et al[8] reported that 11 experimentally infected pigs were all seropositive when tested by IFA 233 to 604 days post challenge.

In this study, the apparent lack of virus circulation after the attempt to eradicate the virus allowed us to evaluate the persistence of antibodies in sows after infection. Twenty months after the presumed time of infection, 36% of sows tested were still positive by PRRS ELISA, and 8 months later, two sows remained positive both by ELISA and IFA. In the apparent absence of virus circulation in the herd, some animals had persistent positive serological titers for at least 2 years, while titers of most animals gradually decreased. Reasons for this are unknown. One could speculate that animals remaining highly seropositive might be carriers of the virus, while those that become seronegative have cleared the infection. In this case, it appeared that either the seropositive animals were not carriers, or they were not shedding the virus, as susceptible pigs were not infected.

Implications

- If validated in more units, a PRRS eradication strategy that might stop virus circulation in an acutely infected herd includes deliberate infection of the breeding herd during the outbreak, then temporary herd closure.
- Some sows may remain seropositive to PRRSV for more than 28 months post infection.

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