

PRRS serology: A critical component of the diagnostic workup

Scott Dee, DVM, MS, Dipl, ACVM; and HanSoo Joo, DVM, PhD

Since the development of the indirect-fluorescent antibody (IFA) test in 1992, serologic profiling has been extremely valuable in improving the understanding of PRRS virus (PRRSV) patterns of transmission within infected farms.^{1,2} The IFA test detects IgG antibodies and indicates infection, not protection. IFA titers are initially detected 7–10 days post-infection and persist for up to 3–4 months.³ Recently, an ELISA test has been developed.⁴ It too measures IgG; however, ELISA antibodies are not detectable until 9–10 days post infection and persist for longer periods (at least 5–6 months). Both tests appear to be very useful when initially profiling an infected herd.

The concept of serologic profiling is based on monitoring the dynamics of an antibody response within populations over time. Such strategies have been widely practiced in the poultry industry, but have been slow to gain acceptance for solving disease problems of swine. Potential reasons include:

- cost;
- the lack of understanding where and how to sample;
- accuracy and consistency of tests; and
- difficulty in interpreting results.

PRRSV has changed all of this! This disease has taught us how important serology can be. In our opinion, it is impossible to truly understand farm-specific PRRS problems without a serologic profile. The purpose of this paper will be to relate the aforementioned problems to a field case of PRRS and attempt to demonstrate the value of serology.

History

A 500-sow, farrow-to-finish, one-site production farm in southern Minnesota was originally infected with PRRSV in 1990. Upon cessation of reproductive signs of the disease, recurrent episodes of postweaning pneumonia began to take place in the nursery. Pigs appeared healthy at weaning, but became anorexic and demonstrated signs of respiratory distress 2–3 weeks later. Clinically, an elevated level of sneezing, coughing, and periocular edema were initially detected, followed by increases in meningitis and peracute death loss. Mortality rose from 2% to 10% in the nursery, while average daily gain in this stage decreased from 0.75 lb to 0.25 lb per day. Commercial vaccines were not available at the time.

SD: Swine Health Center, 621 Pacific Avenue, Morris, Minnesota, 56267; HSJ: Department of Clinical and Population Sciences, College of Veterinary Medicine, 1988 Fitch Avenue, Room 385, University of Minnesota, St Paul, Minnesota, 55108.

Diagnostic notes are not peer reviewed

Diagnostic workup

Four piglets demonstrating respiratory problems and/or meningitis were submitted to a diagnostic laboratory. Bacteriology culture indicated the presence of *Streptococcus suis*, isolated in pure culture from the brain. Histopathological examination indicated lesions suggestive of interstitial pneumonia. Finally, PRRSV was isolated from the sera of all pigs.

Based on these results, it was decided to determine the epidemiology of the PRRSV infection on this farm. A serologic profile consisting of 10 gestating sows, 10 recently weaned (3- to 4-week-old) piglets, 10 8-week-old piglets, and 10 6-month-old finishing pigs was collected. By sampling the farm in this manner, one can assess the activity of the virus in each pig population, i.e., breeding (sows), farrowing (4 weeks), nursery (8 weeks), and finishing.

The lack of an antibody response in sows and recently weaned pigs indicated the absence of PRRSV infection in the breeding and farrowing populations (Table 1). In contrast, the high prevalence of antibodies detected in 8-week-old nursery piglets indicated recent infection. Finally, the low titers detected in the finishing pigs indicated antibody decay following infection early in life.

Discussion

This profile demonstrates transmission of PRRSV in the nursery but not in the breeding or finishing populations. The ability to determine stage-specific patterns of virus transmission is very helpful prior to making decisions about how to implement control measures. This problem was easily solved by depopulating the nursery to interrupt the spread of virus in this stage. Levels of performance and mortality returned to normal after depopulation. Let's revisit the aforementioned concerns about serology and see if we have adequately assessed each one:

Cost

Forty samples were collected, and the cost to the producer was \$6.00 per sample. Therefore a total bill of \$240 for serological analysis was submitted. This producer has been spending over \$3.50 per nursery pig produced with antibiotic costs to control the *S. suis* and enzootic pneumonia prior to depopulation. Twelve months later, records indicated that this cost had averaged \$1.50 per pig. This farm produced over 10,000 nursery piglets each year, so the cost of the testing appeared to be worthwhile.

Where and how to sample

It has been our basic approach to profile pigs from areas that best

represent what takes place within a population over time. Using the previously described sampling method, one can assess the PRRS status in the adults (10 sows), in the farrowing and weaning populations (3- to 4-week-old piglets), any changes in serostatus that take place in the nursery (3- to 4-week-old piglets versus 8- to 10-week-old piglets), and in the finisher (10 weeks to 6 months of age).

The question of how to sample deals primarily with sample size. The sampling of 10 animals per stage allows you to detect at least a 30% prevalence with a 95% degree of confidence. In order to improve sampling sensitivity, it may be necessary to bleed 30 breeding animals. Prevalence levels of 10% or less may exist in chronically infected adult populations. Based on this fact, we may sample 30 breeding animals, submit 10 samples, while separating and freezing the remaining 20 sera. If further analysis is required, the stored samples can be submitted. If 30 samples are tested, you should include at least five samples over a range of parities, including gilts from the replacement pool, prior to breeding.

Accuracy and consistency of results

No serologic assay today is 100% sensitive and specific. All tests have inherent disadvantages, but these can be minimized if a few steps are consistently followed:

Work with a designated laboratory

This will allow for improved familiarity with testing methods and personnel involved.

Provide the serologist with an accurate history and inform him/her of your reasons for testing

Providing serologists with some background information on the case may help them interpret the results. For example, if you suspect that there is PRRSV circulating in a particular stage of production (as observed above), include a short summary of clinical signs, onset of disease problem, etc.

Prior to sampling, establish a plan of action

What happens if the results are positive? Negative? What will be the course of action following receipt of the data? Having a predetermined course of action will not only help increase the value of the results, it will impress the client! A mistake people frequently make is to go through all the work of collection, spend a lot of money, and then have no idea what to do after the results are reported.

Interpreting the results

This, of course, is the critical step. As demonstrated in the previous case, it is important to understand the type of antibody detected and what the presence of that antibody means. The presence of antibodies rarely indicates that the animal is protected, but rather that it has been at some time.

The magnitude of the titer response may help determine the chronology of infection. For example, titers of 1:1024 suggest recent infection. In contrast, titers of 1:16 may either indicate antibody decay after infection, or that the animal sampled was recently infected and is undergoing seroconversion. This demonstrates the weakness of a one-time

Table 1

A summary of a serologic profile indicating PRRS virus transmission in nursery pigs

Pig	Breeding	Stage sampled		
		4 wk	8 wk	6 mo
1	—	—	1:1024	1:16
2	—	—	1:256	1:64
3	1:64	—	1:16	—
4	—	—	—	—
5	—	1:16	1:1024	1:16
6	—	—	1:1024	1:16
7	—	—	1:1024	1:64
8	—	—	1:256	—
9	—	—	1:64	—
10	—	—	—	1:16

Analysis via indirect fluorescent antibody testing

sampling procedure; therefore, a farm may need to be reassessed 1–2 months later. While this does increase cost, it also increases confidence and accuracy. Finally, knowledge of the type of antibody detected is helpful as well. IgG antibodies endure longer than do those of the IgM class. An IgM IFA test has been developed for PRRS, allowing us to detect acute infection and a potentially viremic condition.⁵ Negative results generated from other IgM tests, such as the complement-fixation test for *Actinobacillus pleuropneumoniae* or the agglutination test for leptospirosis, may cause confusion. This is usually due to false negative results, following rapid decay of the IgM. Again, it is critical to understand the test that is chosen.

Conclusions

In our opinion, serology is an essential part of a PRRS diagnostic and control program. We hope we have demonstrated the need for serologic profiling and answered some of the frequently encountered questions or concerns. Needless to say, serologic results need to be strengthened by other components of a thorough diagnostic workup including microbiology, histopathology, and clinical history. However, if used properly, serology can be a very cost-effective method of generating meaningful data on the epidemiology of PRRSV for a particular farm situation, as well as other infectious diseases.

References

1. Yoon IJ, Joo HS, Goyal SM, Molitor TW. An indirect-fluorescent antibody test for the detection of antibody to PRRS virus in swine sera. *J Vet Diagn Invest.* 1992; 4:144–147.
2. Dee SA, Joo HS. Prevention of the spread of PRRS virus in endemically infected pig herds by nursery depopulation. *Vet Rec.* 1994; 135:6–9.
3. Yoon KJ, Zimmerman JJ, Swenson SL, et al. Characterization of the humoral immune response to PRRS virus infection. *J Vet Diagn Invest.* 1995; 7:305–312.
4. Snyder MC, Mermer B, Anderson PR, et al. Evaluative data for an immunodiagnostic ELISA for PRRS. *Proc 2nd Intl Symp on PRRS, Copenhagen, 1995.* pp 15.
5. Joo HS. Interpretation of PRRS serology. *Proc AASP, 1995,* pp 397–400.

