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Case study: Application of sequencing to investigate an infection in a boar stud and the herds that received semen from it

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Clinical history

A 500-head boar stud became infected with PRRS virus (PRRSV) in October 2001. The infection was confirmed on October 30 upon receipt of diagnostic results from a boar submitted on October 23.

At about the same time, on October 16, an infection was confirmed in a finishing unit located 0.8 miles from the stud. This finisher had received PRRSV-negative (naive) pigs and had remained negative for 3 months.

About one third of the herds that received semen from the stud experienced clinical signs attributed to PRRSV infection. Clinical signs in the sow herds appeared between 3 to 5 weeks post-confirmation of the infection in the stud. Of special interest was a case that reported clinical signs 2 weeks after the stud was declared positive. An effort was made to confirm whether the infections originated from the boar stud.

Sequencing was used to compare the isolates recovered from the finishing unit, the boar stud, and the herds that received semen from the study stud. In addition, sequencing results were compared to historical sequences available in a diagnostic database.

Results

Sequencing results suggested that the finishing site and the boar stud had been infected with a similar PRRSV isolate (98.2% homology). Results from reviewing the timeline of the infection and the appearance of clinical signs suggested that the finishing site was infected before the boar stud. In addition, a historical PRRSV isolate recovered in 2000 was found to be 97.2% similar to the one found in the finisher and 99% similar to the one found in the boar stud. This isolate was geographically related to the above-described PRRSV breaks and its similarity was therefore not considered coincidental.

Comparison of the isolates recovered from the sow herds that received semen from the newly infected stud confirmed that in all the evaluated cases, except one, the isolates were >98.0% similar to the one identified in the stud (98.1%, 98%, and 98.1%) and >99.8% similar to the one identified in the finisher site (99.8%, 100%, and 100%).

The sow herd that reported clinical signs 2 weeks post-infection in the stud had become infected with a PRRSV isolate completely unrelated to the boar stud isolate (86.3% homology). Additional investigations suggested that this sow herd had become positive through a lateral infection from a neighboring herd located 2 miles away.

Discussion

PRRSV sequencing has proven to be a very useful tool when investigating PRRSV breaks. The information provided by sequencing allows to establish epidemiological relationships between breaks. However sequencing per se does not prove the mechanism how the virus moves and how the herds become infected.

In the examples described in this study, sequencing could only prove that the infection in the sow herds originated from the boar stud because the mechanism by which the virus traveled was known (semen). Infection in the boar stud itself was believed to have originated from the neighboring finisher because of the similarity of the isolates and the fact that infection in the finisher was present at least 2 weeks earlier than in the stud. Interestingly enough, the isolates recovered from the sow herds had a higher degree of homology with the isolate recovered from the finisher than with the isolate recovered from the boar stud, suggesting that similar variants of the virus (“quasispecies”) existed in the stud. Because of the proximity between the boar stud and the finisher, the route of infection was thought to be airborne given that no other links were identified. Whether this was the true mechanism by which the virus entered the boar stud is unknown.

Also, although there was information that suggested that a geographical relationship existed between the isolate that infected the finisher site and a previous isolate, the mechanism by which the finishing site became positive could not be determined. It is important to consider that even though the geographical information suggested that the boar stud became infected from the finisher site, this information is not conclusive, since the boar stud and the finisher could have been infected simultaneously. However, the order in which the infections appeared suggested that this was not the case.

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In addition, sequencing was a powerful tool in proving where the virus did *not* come from. In the boar stud investigation, sequencing was instrumental in confirming that the virus did not originate from the stud itself. This piece of information was critical since the stud had undergone a PRRSV eradication project and doubt existed as to whether an unidentified—perhaps persistently infected—boar could have been the source of the infection. In another case, sequencing was also helpful in proving that the infection in one of the sow herds was not due to the introduction of PRRS-positive semen.

