Breeding herd Senecavirus A infection: understanding its persistence

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- Senecavirus A (SVA) continues to be responsible for an important number of FAD investigations.
- SVA continues to circulate in breeding herds for up to 21 weeks after clinical signs had been detected.
- Heat check boars may contribute to population persistence of this virus.

Senecavirus A (SVA) is well known in the United States (U.S.) industry for causing a vesicular disease — visually indistinguishable from other high-impact and government-monitored vesicular diseases, such as foot-and-mouth disease (FMD). For this reason, this virus has been responsible for a rampant increase in the number of foreign animal disease (FAD) investigations in the U.S. since the initial SVA outbreaks in swine operations in 2015. Based on the USDA’s FAD Investigation report, in 2019 a total of 2,517 FAD investigations were conducted and 1,845 (73%) of them were for swine vesicular diseases.

One critical question that is yet to be answered is how long the virus remains active within a sow farm after its introduction, as vertical transmission of the virus leads to having infected litters carrying the virus down the production flow. At the University of Minnesota (UofM) we began addressing this question by conducting a study funded by the AASV Foundation, in which processing fluids samples collected from 10 sow farms undergoing an SVA outbreak were tested for SVA using PCR. One farm had semen and tissues from heat check boars tested in order to initially assess their role in the persistence of the virus in the breeding herd. Farms were monitored for a period ranging from 16 to 26 weeks. Some farms provided samples that had been stored and that were collected even before SVA signs were detected.

The virus was detected in processing fluids from all farms and intermittent detection with suspect samples ranged between 1 and 21 weeks after the first SVA clinical signs were seen, with an average of 11.8 weeks. Interestingly, one farm was SVA suspect 3 weeks before clinical signs were evident, while 3 were suspect 2 weeks before clinical signs, and another farm suspect 1 week before clinical SVA signs were seen (Table 1). Heat check boars could be potentially acting as a source of SVA infection to naive gilts and sows over time and after an outbreak. In two semen collection time-points, 7 out of 9 and 1 out of 16 boars tested had SVA positive semen at 7 and 18 weeks after the outbreak had SVA respectively. Furthermore, the boar with SVA-positive semen in the second collection time-point was euthanized and had its testicles tested at 22 weeks after the outbreak. Testicular tissues contained a significant amount of SVA genetic material.

The data collected in this study shows that SVA continues to circulate in breeding herds for a significant amount of time after clinical signs have been detected. Considering that SVA signs in breeding herds are usually seen for 2 weeks after the outbreak, producers and practitioners should be cautious when classifying the herd as stable based upon weaning SVA-negative piglets. Our study clearly demonstrated that the absence of clinical signs does not necessarily indicate that the virus is no longer present within the herd.

Processing fluids have been successfully used for monitoring PRRS in disease elimination protocols, and it appears that this tool might be useful for the control of SVA. Heat-check boars also appear to play an important role in the persistence of SVA in a sow farm, so strategic sampling and testing of these animals is advised. This study contributes to the SVA-epidemiology knowledge in sow farms by increasing the understanding of SVA persistence and transmission, which can help lead to the development of strategies to control SVA and its impact on pig production systems.