

Natural and experimentally-induced Senecavirus A infections in boars

Matthew Sturos¹, Deborah Murray², Darwin Reicks³, Jean Paul Cano⁴, Stephanie Rossow¹, Fabio Vannucci¹

¹University of Minnesota, ²NFP, ³Reicks Veterinary Research & Consulting, ⁴PIC

Key points

- Naturally-infected boars have been documented to shed Senecavirus A (SVA) RNA in semen for up to three months after exhibiting vesicular disease
- Experimentally-infected boars shed SVA RNA in semen for up to three weeks post-inoculation
- The majority of experimentally-infected boars did not exhibit clinical signs or develop apparent lesions

Background

SVA, also known as Seneca Valley Virus (SVV), has rapidly worked its way into the conversations and herd health management plans of swine producers and veterinarians both in the United States and around the world. However, there are still many questions surrounding this pathogen. SVA is known to cause vesicular disease in pigs of all ages and has been associated with transient neonatal losses in some infected sow farms. Little is currently known about the routes of transmission and likely sources of virus introduction into naïve farms. A potential source of viral introduction which has not been thoroughly investigated is boar semen. This update from will briefly touch on viral shedding and tissue distribution of SVA in two naturally-infected boars as well summarize some preliminary data from a recent experimental trial.

Naturally-infected boars Two heat-check boars became infected with SVA during an outbreak on a Minnesota sow farm in 2017 and developed vesicular disease. Semen was collected from one or both of these boars approximately weekly from the time that they developed lesions until approximately three months after the outbreak occurred on the farm. Semen and other clinical samples were tested for SVA by real-time PCR (RT-PCR) at the University of Minnesota Veterinary Diagnostic Laboratory (UMN VDL). These boars consistently shed viral RNA in their semen during the investigation period, with PCR-positive samples ranging from a threshold cycle (Ct) value of Ct 20 up to Ct 32. The lowest Ct values were obtained approximately 6 weeks after the development of lesions. However, the virus could only be isolated from one semen sample collected approximately two weeks after the outbreak. These boars were euthanized and necropsied approximately three months after the outbreak, which included an extensive evaluation of the reproductive tract. Large amounts of SVA RNA were detected within the testes of both boars by PCR. Histology of the testes revealed an interstitial lymphocytic orchitis and degeneration of seminiferous tubules. SVA RNA was identified within these areas of inflammation by in-situ hybridization (ISH, see figure 1). SVA RNA was also detected by PCR and identified using ISH within the tonsils of both boars.

Experimentally-infected boars

Twelve boars were intra-nasally inoculated with SVA. One group of six boars was inoculated with an historical strain obtained in 1999 from a porcine fetus (historical group) and one group of six boars was inoculated with a contemporary strain obtained in 2017 from boar semen (contemporary group). All clinical sample tissues collected at necropsy were tested for SVA RNA by RT-PCR at the UMN VDL. All boars in the contemporary group shed SVA RNA in the semen on at least collection during the first week post-inoculation, ranging from Ct 30 to Ct 35. One of the contemporary group boars shed in semen for three weeks. One boar in the historical group shed SVA RNA in semen at only one collection during the first week, with a Ct 35. Two of the six boars in the contemporary group developed vesicular/ulcerated dermal lesions on the snout or feet during the first week, which resolved after one week. None of the boars in the historical group developed lesions or exhibited clinical signs. All boars were necropsied approximately 1.5 months post-inoculation. Testis was PCR-positive in more than half of the contemporary group boars (Ct 33-34) and less than half of the historical group boars (Ct 32-34). All boars from both groups were PCR-positive on tonsil tissue (Ct 21-28). There were no histologic lesions in the testes or tonsils of these boars.

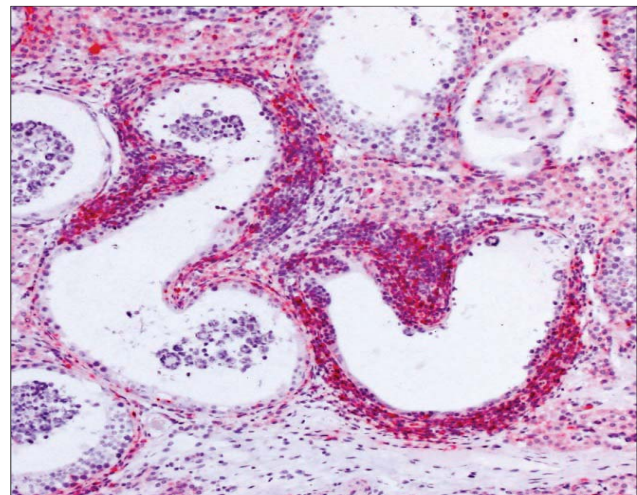


Figure 1. Testis of boar naturally-infected with Senecavirus A (SVA). Infiltrates of lymphocytes are present in the interstitium surrounding degenerate seminiferous tubules. Bright red areas indicate positive signal for SVA by in-situ hybridization.

Conclusions and discussion

This update shows that SVA RNA is shed in semen from both naturally-infected and experimentally-inoculated boars. The prolonged shedding of viral RNA in semen and the presence of SVA RNA in the testes and tonsils of the naturally-infected boars for up to three months are concerning findings and raises the possibility of persistent infection in boars. While the duration of shedding in semen for the experimentally-infected boars was considerably shorter than for the naturally-infected boars, the fact that all contemporary-strain boars had PCR-positive semen on at least one collection indicate that shedding in semen is a repeatable phenomenon and shedding occurred in some boars which did not exhibit clinical signs or develop vesicular lesions. It is currently unknown whether semen from infected boars can serve as a source of infection if used to inseminate susceptible females. These findings provide additional insight on Senecavirus A infections and highlight the need for continued research.