

Role of stress on early pathogenesis of Senecavirus A in pigs

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Key points:

- Impact of a stress model on the early pathogenesis of Senecavirus A was evaluated.
- Time to detect replicating virus in snout skins and coronary bands was 48 hours post inoculation.
- Naïve animals likely do not have enough time to develop vesicles during most transportation events, if exposed via the nasal route.

Senecavirus A (SVA) has been detected in many pork processing plants from different countries and is responsible for great concern due to the similarities with other vesicular diseases, such as foot-and-mouth disease. Interestingly, vesicular lesions are commonly detected in finishing pigs and sows after arrival at packing plants, while going undetected at the farms or buying stations. This lack of detection prior to the transportation of the animals raises questions about the timeline between exposure and first presence of vesicles, and whether stressful events such as transportation could shorten the incubation period and result on early appearance of vesicular lesions.

The present study aimed to investigate the impact of stress from a simulated transportation model on the early pathogenesis of the SVA infection, and development of vesicular lesions. Animals were allocated in three groups and housed in separate rooms: 1) SVA stressed (n=8), 2) SVA non-stressed (n=8), and 3) control (n=2). SVA stressed and non-stressed groups were both inoculated with SVA via the intranasal route. Inoculation of the SVA stressed group coincided with the beginning of the stress event in order to mimic an exposure event of naïve pigs at load out or in the truck en route to the packing plant. Blood samples were collected at 6, 12, 24 and 48 hours. Two animals from each of the inoculated groups were euthanized at 6, 12, 24 and 48 hours post-inoculation (hpi). Samples were collected from each animal and tested by the UMN Veterinary Diagnostic Laboratory for SVA RNA by RT-PCR testing and *in-situ* hybridization (ISH).

Samples collected at necropsy yielded positive RT-PCR and ISH with the first detections in the palatal tonsils and mandibular lymph nodes of the four animals necropsied at 6hpi. Retropharyngeal lymph nodes first revealed positive results at 12hpi. At 24hpi, the first detections of RT-PCR positive sera were recorded in 4/4 SVA stressed and 3/4 SVA non-stressed animals, indicating viremia started between 12 and 24hpi. Finally, snout skins and coronary bands first showed positive results for both RT-PCR and ISH at 48hpi, with 2/2 SVA stressed animals being positive in both tissues and 1/2 from the SVA non-stressed group being positive only from the skin in the dorsal aspect of the snout. No vesicles developed in any of the animals during this 48-hour period.

Based on these results, naïve animals exposed via the nasal route likely do not have enough time to develop vesicles during most transportation events if they get infected at load out at the farm, or en route to the packing plant. This is because under our experimental conditions it took between 12-24 hours to develop viremia, and between 24-48 hours to be detectable in the skin, where the visible lesions occur typically between 4-6 days post-inoculation under experimental conditions. Additional research into the time-course of the development of lesions within models utilizing other modes of transmission and routes of inoculation (i.e. intradermal) will be valuable to further understand the early pathogenesis of SVA in pigs.