that these strains are most similar to strains characterized in 2014–2015 in Brazil, which were associated with idiopathic vesicular disease and neonatal death. Little diagnostic testing is performed on culled animals, which may in part explain the discrepancy between 1% of oral fluids submitted for diagnostic testing being positive for SVA (7), compared with 72% of culled swine swab specimen pools in this study. The sole sample from primary markets that was positive for SVA by qRT-PCR had a Ct of 36.9, just below the negative cutoff of 37.

Further research is needed to address possible correlation between SVA and health status of animals sold at lower value to cull markets. A notable distinction between contemporary SVA in the United States and Brazil, however, is that all the US samples originated from healthy animals that showed no clinical symptoms. Given the high genetic similarity between contemporary US SVA sequences and those from Brazil, additional cofactors likely affect clinical disease.

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References


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Novel Senecavirus A in Swine with Vesicular Disease, United States, July 2015


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To the Editor: Senecavirus A (SVA; formerly known as Seneca Valley virus [SVV] belongs to the genus Senecavirus, family Picornaviridae (1,2). SVA was first isolated in 2001 as a contaminant of the PER.C6 cell line and designated as SVV-001 (1,3). Since its discovery, SVA has been infrequently detected in swine with idiopathic vesicular disease (IVD) (4–6), which clinically resembles foot-and-mouth disease, swine vesicular disease, vesicular exanthema of swine, and vesicular stomatitis. The virus has also been retrospectively detected in previous cases with various clinical conditions in the United States during 1988–2001 (7). However, the clinical significance of SVA in swine could not be determined (7,8).

In late July 2015, the Iowa State University Veterinary Diagnostic Laboratory (ISUVDL) received reports of 4 apparently unrelated cases of IVD affecting exhibition and commercial swine. The first 3 cases originated from unrelated farms located in southwest and central Iowa and were observed at 2 county fair exhibitions. The fourth case was observed in a commercial finisher farm in South Dakota. Affected animals exhibited acute lameness, anorexia, lethargy, and transient fever without associated mortality; they also exhibited coronary band hyperemia and vesicles, which occasionally progressed to cutaneous ulcers, as previously reported (5,6). Small vesicles were also evident on the snout, within the oral cavity, or both; these vesicles variably progressed to ulceration. No specific microscopic lesions beyond the ulcerative changes were present in specimens submitted to ISUVDL.

We collected vesicular lesion swab specimens and blood samples from all affected animals, and all tested negative for the viruses causing vesicular diseases mentioned previously (foot-and-mouth disease, swine vesicular disease, vesicular exanthema of swine, and vesicular stomatitis). No other common swine pathogens except
SV A were detected at ISUVDL. By using a quantitative real-time reverse transcription PCR assay, we targeted a conserved region between the 5′ untranslated region and protein L (602–710 bp) and detected SV A RNA in vesicular fluids, epithelial scrapings of the snout, coronary band lesions, and/or hoof lesions with quantities ranging from $2 \times 10^7$ to $1.2 \times 10^{11}$ genomic copies/mL. We also identified the virus in serum and fecal samples, indicating SV A viremia and shedding. In a follow-up submission from the South Dakota premise, we detected SV A in nearly all of the tissues tested; inguinal lymph nodes and tonsils contained the highest SV A loads. Seroconversion to SV A in all affected swine was evident by indirect fluorescent antibody test titers ranging from 1:160 to 1:1,280 at 2–3 weeks after the clinical outbreak.

Our attempts to isolate the virus by using ST cells (ATCC CRL-1746; ATCC, Manassas, VA, USA) and NCI-H1299 (ATCC CRL-5803) (8) yielded cytopathic SV A isolates with titers up to $1 \times 10^9$ PFU/mL from multiple vesicular lesion swabs or scrapings. We designated a representative isolate from each Iowa case as SV A15-39812IA, SV A15-40380IA, and SV A15-40381IA and the South Dakota case as SV A15-41901SD. Sequencing of viral protein 1 (VP 1) as previously described (7) demonstrated that each SV A isolate had a VP1 sequence identical to that of the virus in clinical specimens.

We obtained almost full-length genomic sequences (7,116–7,221 nt) of the 4 SV A isolates from multiple vesicular lesion swabs or scrapings. We designated a representative isolate from each Iowa case as SV A15-39812IA, SV A15-40380IA, and SV A15-40381IA and the South Dakota case as SV A15-41901SD. Sequencing of viral protein (VP) 1 as previously described (7) demonstrated that each SV A isolate had a VP1 sequence identical to that of the virus in clinical specimens.

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Veterinary Services Laboratories of the US Department of Agriculture’s Animal and Plant Health Inspection Service in Plum Island, New York. The next-generation sequencing was performed at the Iowa State University DNA Facility.

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References


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