We completed sequencing the whole genomes for the vesicle tissue (SVA-OH1) and vesicle swab sample (SVA-OH2). On the basis of BLAST (http:blast.ncbi.nlm.nih.gov/Blast.cgi) searches, the SVA-OH1 and -OH2 isolates had 99% nt identity to 3 new US strains (USA/IA40380/2015, USA/SD41901/2015, USA/IA46008/2015) and 98% nt identity to 3 Brazil strains (SVV/BRA/MG1/2015, SVV/BRA/MG2/2015, SVV/BRA/GO3/2015) from GenBank. The Ohio isolates also shared 96% and 94% nt identity with Canada strain (11-55910-3) and the first US SVA strain (SVV-001), respectively. Further analysis showed that, in comparison with these 8 strains with complete genome sequences available in GenBank, the 2 Ohio SVA isolates had 22 unique nucleotide mutations in the genome: 1 in the VP4 gene, 5 in VP2, 2 in VP3, 1 in VP1, 4 in 2B, 3 in 2C, 3 in 3A, 1 in 3B, and 2 in 3D (online Technical Appendix Table 2). Among the 22 unique mutations, there were 2 nonsynonymous mutations at position 2082 in the VP3 gene of both isolates and position 5037 in the 3A gene of SVA-OH1 and 1 unique synonymous mutation only in SVA-OH2.

Phylogenetic analysis of the complete genome further supports that the 2 Ohio SVA isolates are closely related to each other and clustered together with the 3 recently isolated US strains, were less closely related to the isolates of the Brazil cluster, and were more distantly related to the isolate from Canada and the original SVA strain reported from United States (Figure). Consistent with the previous findings (*1*), all SVA isolates from different countries clustered together under the genus *Senecavirus*, which is most closely related to the genus *Cardiovirus* of the family *Picornaviridae* (Figure).

Our findings that a pig with clinical signs of IVD was infected with SVA and our genetic analysis demonstrating that the 2 Ohio SVA isolates are closely related to the other SVA strains from different countries provide further support for SVA involvement in IVD in pigs. More support could be provided by future studies, including continued surveillance of SVA and confirmation of the Koch postulates.

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# Senecavirus A in Pigs, United States, 2015

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**To the Editor:** Senecavirus A (SVA) has been sporadically identified in pigs with idiopathic vesicular disease in the United States and Canada (*1*–*3*). Clinical symptoms observed include ruptured vesicles and erosions on the snout and lameness associated with broken vesicles along the coronary band. A recent report characterized SVA in pigs in Brazil with similar clinical symptoms in addition to a higher proportion of deaths than would be expected in pigs 1–4 days of age (*4*,*5*). Several outbreaks of this infection in pigs were reported in the summer of 2015 in the United States; the more severe clinical features resembled those seen in outbreaks in Brazil (*6*). Subsequent testing by PCR of 2,033 oral

fluid samples from material submitted during 441 routine diagnostic testing procedures (from 25 states) identified 5 SVA-positive cases (1%) (*7*). Besides affecting animal health, SVA infection is notable because its clinical symptoms resemble those caused by foot-and-mouth disease and vesicular stomatitis viruses. When vesicular disease is observed in US swine, mandatory reporting and testing of animals for foreign animal diseases are required.

In June 2015, we collected 25 nasal and 25 rectal swab specimens from healthy pigs at 5 pig markets in North Carolina (250 total samples), representing pigs from 5 producers per market; the pigs were commingled for <12 hours. Primary markets 1 and 2 were slaughterhouses that purchased top quality pigs. Secondary market 3 was a slaughterhouse that purchased lower quality pigs (primarily underweight or herniated pigs). Market 4 was a broker that purchased pigs for culling and resold them for slaughter. Market 5 was a culled pig slaughterhouse. At markets 1–4, animals were ≈20 weeks of age; at market 5, animals were >10 weeks of age.

We sampled the same sites a second time in August 2015. Again, we performed metagenomic sequencing on swab specimens pooled by producer (5 specimens per pool, 50 total pools per sampling) (*8*). Reads most similar to SVA were identified in numerous pools from samplings and at 4 different markets. Quantitative reverse transcription PCR (qRT-PCR) was performed at the Kansas State Veterinary Diagnostic Laboratory (Manhattan, KS, USA) on the original pooled samples and was positive for SVA (cycle threshold [$C_t$] <37) for 26 (52%) pools from June and 18 (36%) pools from August. Sites 2 (n = 1 pool positive), 3 (n = 10), 4 (n = 5), and 5 (n = 10) had positive results in June, and sites 3 (n = 10), 4 (n = 1) and 5 (n = 7) had positive results in August. Both specimen types had an approximately equal number of positive results. We carried out virus isolation on swine testicle cells (positive samples from the second sampling), and 100% cytopathic effects were observed for 5 samples that tested positive for SVA by qRT-PCR with $C_t$ values 16–21.

Templated assembly of the metagenomic sequencing reads with the SVA prototype strain SVV-001 genome (GenBank accession no. DQ641257) yielded near complete genomes from 5 pools (GenBank nos. KT827249–KT827253). The polyprotein-encoding region of the genomes showed >99% pairwise identity to each other and were most similar to sequences determined from recent outbreaks in Brazil (97%–98% nucleotide and >99% amino acid identity). Analysis of the P1 region of the genome found >99% nucleotide identity between 2015 US SVA sequences and 97% identity to SVA from Brazil. The contemporary US SVA sequences were more distantly related to SVA from an outbreak in Canada in 2011 (95% identity) and to historical US sequences (87%–92% identity). To investigate SVA phylogeny, we performed ClustalW (http://www.genome.jp/tools/clustalw/) alignment of P1 nucleotide sequences, followed by maximum-likelihood analysis using the best-fitting Kimura 2-parameter plus gamma distribution model of evolution. The 2015 US SVA sequences were most closely related to SVA sequences from Brazil; these sequences shared a common ancestor in Canada/11-55910-2011 (Figure).

Our results suggest that SVA commonly circulates in secondary and culled swine markets in North Carolina and

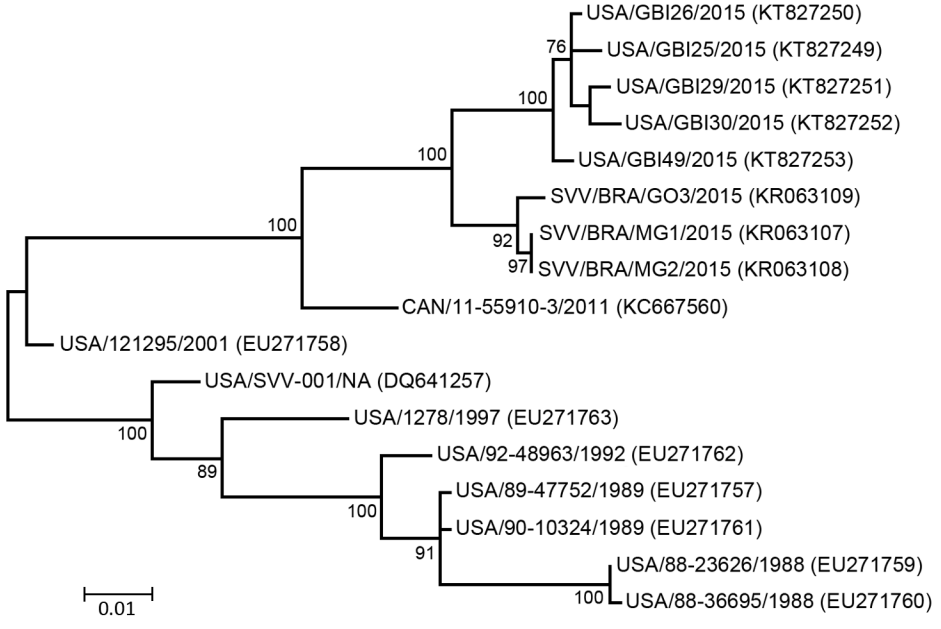


**Figure.** Phylogenetic tree of Senecavirus A P1 sequences. Maximum-likelihood analysis in combination with 1,000 bootstrap replicates as implemented in MEGA 6.06 (http://www.megasoftware.net) was used to derive the tree on the basis of nucleotide sequences. GenBank accession numbers are shown in parentheses. SVV in some isolate names indicates Seneca Valley virus, the original name for Senecavirus A. Scale bar indicates number of nucleotide changes per site.

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 $C_t$ 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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# 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