Senecavirus A (SV A) 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 SV A 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

ACKNOWLEDGMENTS

We completed sequencing the whole genomes for the vesicle tissue (SV A-OH1) and vesicle swab sample (SV A-OH2). On the basis of BLAST (http://blast.ncbi.nlm.nih.gov/Blaster.cgi) searches, the SV A-OH1 and -OH2 isolates had 99% nt identity to 3 new US strains (USA/IA40380/2015; USA/SA41901/2015; 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 SV A strain (SVV-001), respectively. Further analysis showed that, in comparison with these 8 strains with complete genome sequences available in GenBank, the 2 Ohio SV A 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 SV A-OH1 and 1 unique synonymous mutation only in SV A-OH2.

Phylogenetic analysis of the complete genome further supports that the 2 Ohio SV A 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 SV A strain reported from United States (Figure). Consistent with the previous findings (1), all SV A 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 SV A and our genetic analysis demonstrating that the 2 Ohio SV A isolates are closely related to the other SV A strains from different countries provide further support for SV A involvement in IVD in pigs. More support could be provided by future studies, including continued surveillance of SV A and confirmation of the Koch postulates.

References

1. Hales LM, Knowles NJ, Reddy PS, Xu L, Hay C, Hallenbeck PL. Complete genome sequence analysis of Seneca Valley virus-001, a novel oncolytic picornavirus. J Gen Virol. 2008;89:1265–75. http://dx.doi.org/10.1099/vir.0.83570-0
2. Knowles NJ, Hales LM, Jones BH, Landgraf JG, House JA, Skene KL, et al. Epidemiology of Seneca Valley virus: identification and characterization of isolates from pigs in the United States. XIVth meeting of the European Study Group on the Molecular Biology of Picornaviruses; 2006 Nov 26–Dec 1; Saariselka, Inari, Finland; 2006. p. G2 [cited 2016 Apr 26]. http://www.euronic.org.uk/Euronic2006/euronic_2006_posters.htm
3. Singh K, Corner S, Clark SG, Scherba G, Fredrickson R. Seneca Valley virus and vesicular lesions in a pig with idiopathic vesicular disease. J Vet Sci Technol. 2012;3:123.
4. Amass SF, Schneider JL, Miller CA, Shawky SA, Stevenson GW, Woodruff ME. Idiopathic vesicular disease in a swine herd in Indiana. Journal of Swine Health and Production. 2004;12:192–6.
5. Montgomery JF, Oliver RE, Poole WS. A vesiculo-bullous disease in pigs resembling foot and mouth disease. I. Field cases. N Z Vet J. 1987;35:21–6. http://dx.doi.org/10.1080/00480169.1987.35369
6. Munday BL, Ryan FB. Vesicular lesions in swine—possible association with the feeding of marine products. Aust Vet J. 1982;59:193. http://dx.doi.org/10.1111/j.1751-0813.1982.tb16008.x
7. Pasma T, Davidson S, Shaw SL. Idiopathic vesicular disease in swine in Manitoba. Can Vet J. 2008;49:84–5.
8. Gibbs EPJ, Stoddard HL, Yeldoluchng R, House JA, Legge M. A vesicular disease of pigs in Florida of unknown etiology. Florida Veterinary Journal. 1983;12:25–7.
9. Kresse JI, Taylor WD, Stewart WW, Eernisse KA. Parvovirus infection in pigs with necrotic and vesicle-like lesions. Vet Microbiol. 1985;10:525–31. http://dx.doi.org/10.1016/0378-1135(85)90061-6
10. Vannucci FA, Linhares DC, Barcellos DE, Lami HC, Collins J, Marthaler D. Identification and complete genome of Seneca Valley virus in vesicular fluid and sera of pigs affected with idiopathic vesicular disease. Transbound Emerg Dis. 2015;62:589–93. http://dx.doi.org/10.1111/tbed.12410

Address for correspondence: Yan Zhang or Leyi Wang, Animal Disease Diagnostic Laboratory, Ohio Department of Agriculture, 8995 E Main St, Blvd #6, Reynoldsburg, OH 43068, USA; email: yzhang@agri.ohio.gov or wang.1074@buckeyemail.osu.edu

Senecavirus A in Pigs, United States, 2015

Ben M. Hause, Olivia Myers, Joshua Duff, Richard A. Hesse

Author affiliations: Kansas State University, Manhattan, Kansas, USA (B.M. Hause, R.A. Hesse); North Carolina State University, Raleigh, North Carolina, USA (O. Myers); Maxwell Foods, Goldsboro, North Carolina, USA (J. Duff)

DOI: http://dx.doi.org/10.3201/eid2207.151951

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](http://www.megasoftware.net)
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 cul 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.

Financial support for this study was provided by Zoetis and Boehringer Ingelheim.

References

1. Singh K, Corner S, Clark SG, Scherba G, Fredrickson R. Seneca valley virus and vesicular lesions in a pig with idiopathic vesicular disease. J Vet Sci Technol. 2012;3:123 [2015 Sep 2015]. http://www.omicsonline.org/seneca-valley-virus-and-vesicular-lesions-in-a-pig-with-idiopathic-vesicular-disease-2157-7579.1000123.php?aid=9061
2. Pasma T, Davidson S, Shaw SL. Idiopathic vesicular disease in swine in Manitoba. Can Vet J. 2008;49:84–5.
3. Hales LM, Knowles NJ, Reddy PS, Xu L, Hay C, Hallenbeck PL. Complete genome sequence analysis of Seneca valley virus-001, a novel oncolytic picornavirus. J Gen Virol. 2008;89:1265–75. http://dx.doi.org/10.1099/vir.0.83570-0
4. Vannucci FA, Linhares DCL, Barcellos DESN, Lam HC, Collins J, Marthaler D. Identification and complete genome of Seneca valley virus in vesicular fluid and sera of pigs affected with idiopathic vesicular disease. Brazil. Transbound Emerg Dis. 2015;62:589–93. http://dx.doi.org/10.1111/tbed.12410
5. Leme RA, Zotti E, Alcântara BK, Oliveira MV, Freitas LA, Alfiieri AF, et al. Senecavirus A: an emerging vesicular infection in Brazilian pig herds. Transbound Emerg Dis. 2015;62:603–11. http://dx.doi.org/10.1111/tbed.12430
6. American Association of Swine Veterinarians. Senecavirus A (Seneca Valley virus) in swine-breeding herd cases [cited 2015 Sep 25]. https://www.aasv.org/SVV/SVA_Sow_ISU_9215.pdf
7. American Association of Swine Veterinarians. SVV oral fluids testing sheds light on distribution [cited 2015 Sep 25]. https://www.aasv.org/news/story.php?id=8361
8. Hause BM, Collin EA, Anderson J, Hesse RA, Anderson G. Bovine rhinitis viruses are common in U.S. cattle with bovine respiratory disease. PLoS ONE. 2015;10:e0121998. http://dx.doi.org/10.1371/journal.pone.0121998

Address for correspondence: Ben M. Hause, Kansas State Veterinary Diagnostic Laboratory, Kansas State University, 1800 Denison Ave, Manhattan, KS 66506, USA; email: bhause@vet.k-state.edu

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

Baoqing Guo, Pablo E. Piñeyro, Christopher J. Rademacher, Ying Zheng, Ganwu Li, Jian Yuan, Hai Hoang, Phillip C. Gauger, Darin M. Madson, Kent J. Schwartz, Paisley E. Canning, Bailey L. Arruda, Vickie L. Cooper, David H. Baum, Daniel C. Linhares, Rodger G. Main, Kyoung-Jin Yoon

Author affiliation: Iowa State University, Ames, Iowa, USA

DOI: http://dx.doi.org/10.3201/eid2207.151758

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 finisher 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