

States, Guatemala, or Brazil. Last, we counted Associated Press news wire stories as a proxy for daily volume of Zika news coverage in the Western Hemisphere (online Technical Appendix, <http://wwwnc.cdc.gov/EID/article/22/7/16-0415-Techapp1.pdf>).

Using a day as our unit of analysis (i.e., $n = 60$ in the analysis), we first assessed Pearson product-moment correlations between news coverage, social media mentions, and online search behavior and then fit a time series model. Results suggested prominent but ephemeral peaks in salience and attention, with some variation over time in searches by country (Figure). We found strong positive correlations between news (daily volume) and tweets for all 3 countries (United States, $r = 0.86$, $p < 0.001$; Guatemala, $r = 0.78$, $p < 0.001$; Brazil, $r = 0.60$, $p < 0.001$). We also found strong positive correlations between news and Google searches for all 3 countries (United States, $r = 0.86$, $p < 0.001$; Guatemala, $r = 0.74$, $p < 0.001$; Brazil, $r = 0.48$, $p < 0.001$). Because time series data can reflect autocorrelation that makes observed relationships spurious, interpretation of bivariate correlations alone to link time series data is inadvisable. To assess the relationship between news coverage and online searching related to Zika virus, we used time series analysis to predict US Google searches as a function of other observed trends and date. We fit an autoregressive integrated moving average (0, 1, 3) model to address dependence between residuals, resulting in a Ljung-Box statistic that was not significant ($p > 0.05$). This finding indicated that we sufficiently reduced the time series to white noise to assume no autocorrelation in residuals. Our model achieved an R^2 value of 0.90 and stationary R^2 value of 0.53. Associated Press wire stories emerged as a significant and positive predictor (coefficient = 1.52, $t = 3.24$, $p < 0.01$). No other predictor predicted variance greater than that of news stories ($p > 0.05$). Daily news story volume predicted departures from the expected trend in US search behavior related to Zika virus.

Our results suggest that news coverage of public health authority announcements opens brief windows of information sharing, engagement, and searching that offer opportunities to address perceptions and provide preparation and vector control recommendations through education. Sharing and searching are less apparent outside these windows, especially in contexts in which an emerging infectious disease is not yet prevalent. Our findings may not generalize beyond the initial stages of Zika virus transmission in the United States, and future work could obtain appropriate data for investigating the tone of news coverage and online communication in various countries. Nevertheless, recent trends in online information-seeking about Zika virus has been sensitive to official announcements, suggesting the usefulness of pairing announcements with provision of information resources that can be found through search engines.

References

1. Dudo AD, Dahlstrom MF, Brossard D. Reporting a potential pandemic: a risk-related assessment of avian influenza coverage in U.S. newspapers. *Sci Commun*. 2007;28:429–54 <http://dx.doi.org/10.1177/1075547007302211>.
2. Southwell BG, Reynolds BJ, Fowlie K. Communication, media relations and infectious disease surveillance. In: M'ikanatha N, de Valk H, Lynfield R, Van Benden C, editors. *Infectious disease surveillance*. 2nd ed. Oxford: John Wiley & Sons; 2013. p. 607–17.
3. Southwell BG, Hwang Y, Torres A. Avian influenza and US TV news. *Emerg Infect Dis*. 2006;12:1797–8. <http://dx.doi.org/10.3201/eid1211.060672>
4. Kelly B, Squiers L, Bann C, Stine A, Hansen H, Lynch M. Perceptions and plans for prevention of Ebola: results from a national survey. *BMC Public Health*. 2015;15:1136. <http://dx.doi.org/10.1186/s12889-015-2441-7>
5. Prue C, Southwell B, Williams P, Alexander J, Zulkiewicz B. Threat, trust, and tools for action: what matters in helping travelers from countries with Ebola outbreaks to follow government monitoring and reporting guidelines? Presented at: National Conference on Health Communication, Marketing, and Media; 2015 Aug 11–13; Atlanta, Georgia, USA.
6. Weeks BE, Friedenber LM, Southwell BG, Slater JS. Behavioral consequences of conflict-oriented health news coverage: the 2009 mammography guideline controversy and online information seeking. *Health Commun*. 2012;27:158–66. <http://dx.doi.org/10.1080/10410236.2011.571757>
7. Towers S, Afzal S, Bernal G, Bliss N, Brown S, Espinoza B, et al. Mass media and the contagion of fear: the case of Ebola in America. *PLoS One*. 2015;10:e0129179. <http://dx.doi.org/10.1371/journal.pone.0129179>
8. Rowan KE. Why some health risks upset us and others do not: risk perception and risk communication. *Spectra*. 2015;51:13–7.
9. Southwell B. Risk communication: coping with imperfection. *Minnesota Medicine*. 2003;86:14–6.
10. Tavernise S. W.H.O. sounds a global alarm over Zika Virus. *New York Times*. 2016 Jan 29 [cited 2016 Mar 1]. <http://www.nytimes.com/images/2016/01/29/nytfpage/scan.pdf>

Address for correspondence: Brian G. Southwell, RTI International, 3040 E Cornwallis Rd, Research Triangle Park, NC 27709, USA; email: bsouthwell@rti.org

Detection and Genomic Characterization of Senecavirus A, Ohio, USA, 2015

Leyi Wang, Melanie Prarat, Jeff Hayes, Yan Zhang

Author affiliation: Ohio Department of Agriculture, Reynoldsburg, Ohio, USA

DOI: <http://dx.doi.org/10.3201/eid2207.151897>

To the Editor: Senecavirus A (SVA), formerly Seneca Valley virus, is a single-stranded positive-sense, non-enveloped RNA virus (*I*). The RNA genome of SVA is 7.2 kb long and is translated into a polyprotein in a host

cell. The polyprotein is then posttranslationally cleaved into mature proteins, including 4 structural viral capsid proteins (VP 1–4) in the N terminus and 7 nonstructural proteins (2A, 2B, 2C, 3A, 3B, 3C^{pro}, and 3D^{pol}) in the C terminus (1). SVA was discovered as a contaminant of PER.C6 cells and is closely related to viruses in the genus *Cardiovirus* (1). Genomic characterization has led to classification of SVA in a new genus, *Senecavirus*, family *Picornaviridae*. A retrospective study conducted in the United States showed that the samples collected during 1988–2001 were SVA positive, and genetic analysis revealed that the sequences of all 7 SVA isolates are considerably similar to the first US SVA strain (SVV-001), suggesting that SVA may have been circulating in the US pig population for a long time (2).

Idiopathic vesicular disease (IVD) is a vesicular disease of pigs, and etiology is unknown (3). The clinical signs of IVD are fever, lameness, and vesicular lesions on various body parts including the oral cavity, snout, and coronary bands (3). Despite not being a debilitating disease, IVD is noteworthy because it causes lesions clinically indistinguishable from those of other vesicular animal diseases, including foot-and-mouth disease (FMD), vesicular stomatitis, swine vesicular disease, and vesicular exanthema of swine. IVD has been reported in several countries, including the United States (4–7), and has been recognized

in several US states, including Florida, Indiana, and Iowa (4,8,9). Several lines of evidence show that SVA may be associated with IVD outbreaks in Canada, the United States, and Brazil (3,7,10). We describe the detection and genomic characterization of SVA isolated from pigs with vesicular lesions in Ohio.

In October 2015, the Animal Disease Diagnostic Laboratory of the Ohio Department of Agriculture received vesicle tissue, a vesicle swab sample, and whole blood from a sow with vesicular disease for rule-out testing for FMD virus (FMDV). The sow was lame on both front feet and had ruptured vesicular lesions on the snout and coronary bands of both front feet (online Technical Appendix Figure, <http://wwwnc.cdc.gov/EID/article/22/7/15-1897-Techapp1.pdf>). FMDV-specific real-time reverse transcription PCR was applied to the nucleic acid samples extracted from the 3 samples by using a MagMAX Pathogen RNA/DNA kit (Life Technologies, Carlsbad, CA, USA). All samples were negative for FMDV. We then performed 2 conventional reverse transcription PCRs with primers targeting 2 regions of the SVA genome (VP3/VP1, 3D/3' untranslated region) on the same set of samples; the vesicle tissue and swab samples were SVA positive. Subsequently, we determined the whole-genome sequence of SVA by using 7 pairs of SVA-specific primers (online Technical Appendix Table 1).

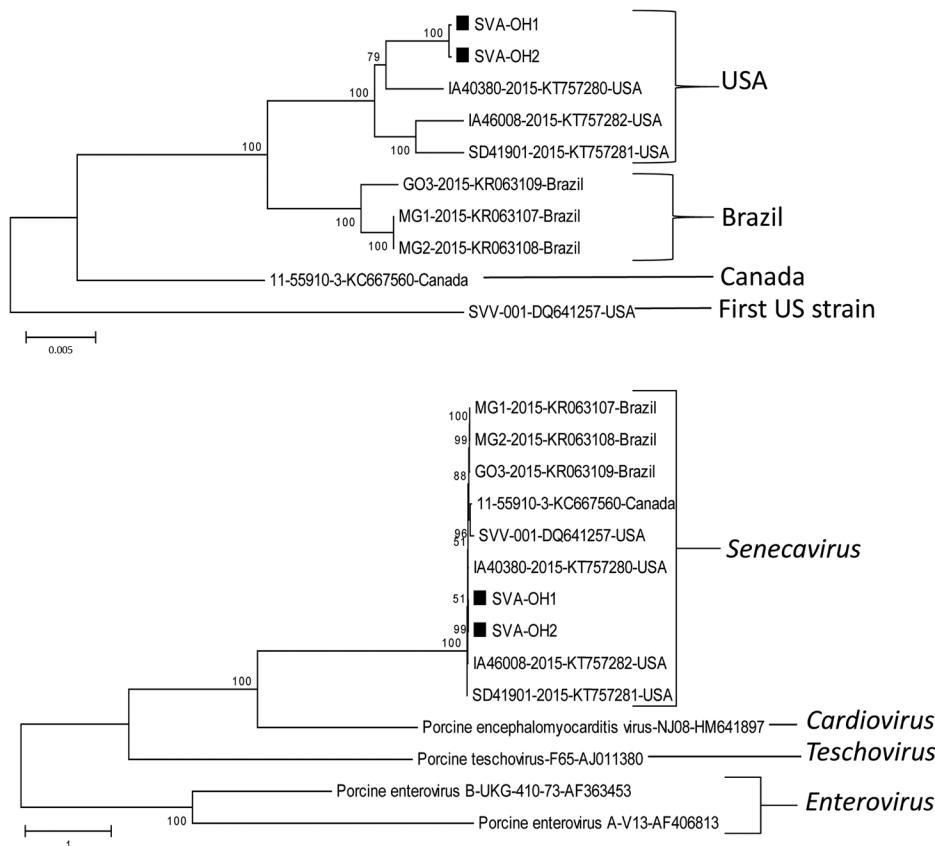


Figure. Phylogenetic trees constructed on the basis of the whole-genome sequences of isolates from the genera *Senecavirus* (SVA), *Cardiovirus*, *Teschovirus*, and *Enterovirus* of the family *Picornaviridae*, including the SVA-OH1 and -OH2 isolates (black squares) from pigs in Ohio, USA. Dendrograms were constructed by using the neighbor-joining method in MEGA version 6.05 (<http://www.megasoftware.net>). Bootstrap resampling (1,000 replications) was performed, and bootstrap values are indicated for each node. Reference sequences obtained from GenBank are indicated by strain name and accession number. Scale bars indicate nucleotide substitutions per site.

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.

Acknowledgments

We acknowledge and appreciate the excellent technical support provided by Jason Herr.

References

- 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>
- Knowles NJ, Hales LM, Jones BH, Landgraf JG, House JA, Skele 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.europic.org.uk/Europic2006/europic_2006_posters.htm
- 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.
- 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.
- 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>
- 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>
- Pasma T, Davidson S, Shaw SL. Idiopathic vesicular disease in swine in Manitoba. *Can Vet J*. 2008;49:84–5.
- Gibbs EPJ, Stoddard HL, Yedloutchnig RJ, House JA, Legge M. A vesicular disease of pigs in Florida of unknown etiology. *Florida Veterinary Journal*. 1983;12:25–7.
- 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](http://dx.doi.org/10.1016/0378-1135(85)90061-6)
- Vannucci FA, Linhares DC, Barcellos DE, 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>

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