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.

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

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To the Editor: Senecavirus A (SVA), formerly Seneca Valley virus, is a single-stranded positive-sense, nonenveloped RNA virus (*1*). 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).

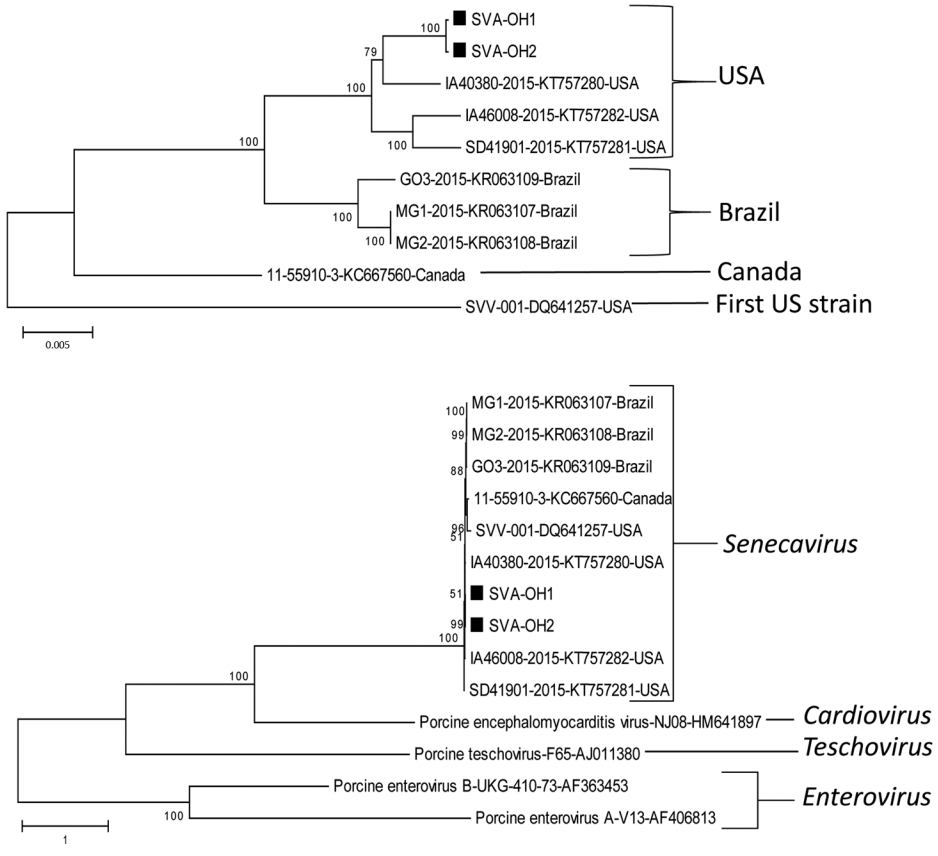

Figure. Phylogenetic trees constructed on the basis of the whole-genome sequences of isolates from the genera *Senecavirus* (SVA), *Cardiovirus*, *Teschovirus*, and *Enterovirus* of the family *Picornaviridae*, including the SVA-OH1 and -OH2 isolates (black squares) from pigs in Ohio, USA. Dendrograms 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

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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

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

Technical Appendix

Technical Appendix Table 1. Primers used for amplification and sequencing of the SV-A complete genome*

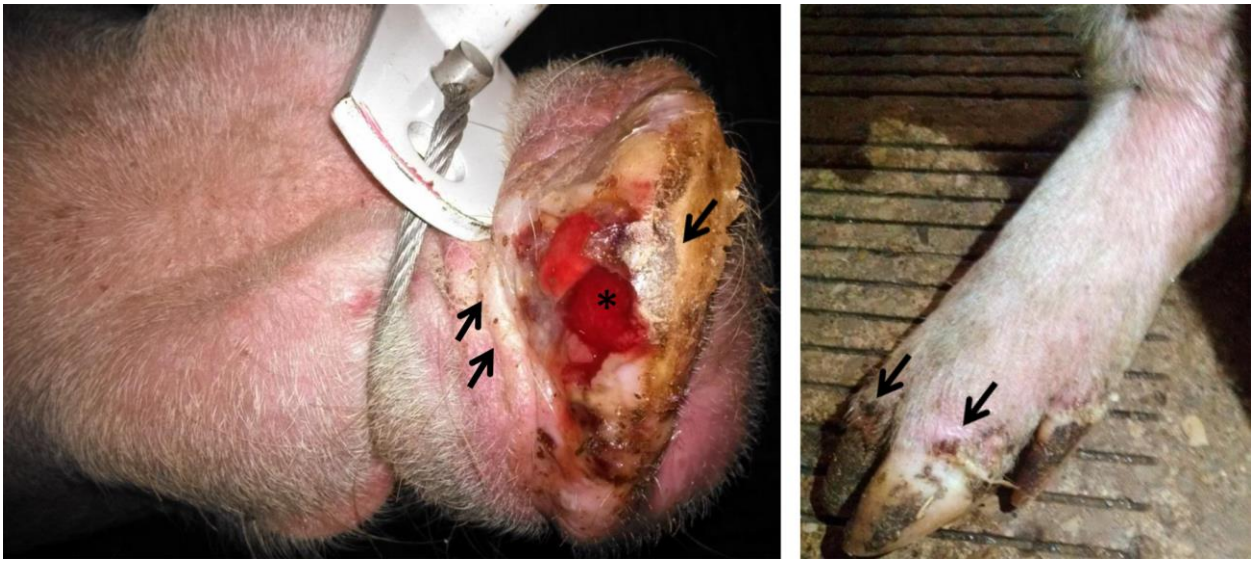
Name	Sequence, 5′→3′†	Sense	Position‡	Amplicon size
P338	AACCGGCTGTGTTTGCTAGAGGC	Forward	59–81	
P339	gcgcTCTTCATTCAGCTCCTGTA	Reverse	1571–1589	1,531
P340	CAGGGAGGCCTCAACGGAGG	Forward	1393–1412	
P341	ACGGCGTCCTTCTCCAGTACCTT	Reverse	2824–2846	1,454
P342	TCTGCTGGTGAGGATTACAC	Forward	2623–2642	
P343	TTGGCATTGATCATAGTGGTGAGAC	Reverse	4184–4208	1,586
P344	GACCTAGTCAAAGAGGTGGTCGA	Forward	3958–3980	
P345	GCCCGGTCCGAGACGTACCATCA	Reverse	5447–5469	1,512
P346	CTTACACAGTCCGCTCTTCT	Forward	5281–5300	
P347	TCCACGTCATACGTGTTCTTTCT	Reverse	6511–6533	1,253
P348	gCTTAGACGGTGACTACTCTGA	Forward	6261–6281	
P349	TTTTTTTCCCTTTTCTGTTCCGACTGAGTTC	Reverse	7257–7287	1,027
P353	GGGGGCTGGGCCCTGATGCCCAG	Forward	10–32	
P355	GAGAACCATGTACTCATGGTGGTAGC	Reverse	466–491	482
P356§	TGGAAGCCATGCTCTCCTAC	Forward	7031–7050	Not applicable

*SV-A, Senecavirus A.
†Lowercase nucleotides are nonspecific sequence used to increase GC contents of the primers.
‡Positions correspond to SVV-001 strain (GenBank accession no. DQ641257).
§Primer used to sequence the 3′ end of SV-A.

Technical Appendix Table 2. Summary of unique mutations in SV-A strains*

Gene	No. mutations	Position†
VP4	1	902
VP2	5	1448, 1565, 1721, 1781, 1871
VP3	2	1988, 2082‡
VP1	1	3449
2B	4	3519§, 3524, 3746, 3788
2C	3	4163, 4442, 4688
3A	3	4961, 5037,‡¶ 5063
3B	1	4165
3D	2	6149, 6875
Total	22	Not applicable

*SV-A, Senecavirus A; VP, viral capsid protein.
†Positions correspond to strain SV-A-OH1 (GenBank accession no. KU058182).
‡Nonsynonymous mutation.
§Unique mutation only present in SV-A-OH2.
¶Unique mutation only present in SV-A-OH1.



Technical Appendix Figure. Photographs of snout (left) and affected foot (right) of sow with idiopathic vesicular disease. Note ruptured vesicle on snout revealing red ulcerated region of exposed dermis (asterisk) with flaps of devitalized epidermis at margins (arrows). The affected foot on the right shows 2 focal ulcerations covered by brown scabs at the coronary band of each digit (arrows).