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Active influenza A virus surveillance among exhibition swine occurred during summer 2016 at 101 agricultural fairs across the midwestern United States; pigs were sampled at the end of exhibition irrespective of clinical signs of respiratory disease (3). Samples obtained using nasal swabs or nasal wipes were stored in viral transport medium at −80°C (4,5). Upon notification from the state animal health official, samples collected from pigs at fairs associated with H3N2v cases were screened for influenza A virus with real-time reverse transcription PCR, and positive samples were inoculated for virus isolation as previously described (6). The genomes of 1 or 2 isolates per fair were sequenced, and the nucleotide sequences were deposited into GenBank (7). Nucleotide sequences of the H3N2v viruses detected in humans were deposited in the GISAID database (online Technical Appendix, https://wwwnc.cdc.gov/EID/article/23/9/17-0847-Techapp.pdf).

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A fair-by-fair comparison of the influenza A virus genomes sequenced from human H3N2v cases and isolates from swine provided strong molecular evidence of zoonotic influenza A virus transmission. The viruses recovered from swine were nearly identical to viruses identified in humans, and human virus gene segment sequences were nested within monophyletic swine virus clades. We identified 2 distinct H3 lineages in the pigs and humans across the implicated fairs (Figure 1). An influenza A virus from the well-established H3 cluster IV-A, found in the pigs at fair C, was responsible for 2 (11.1%) human cases. This cluster IV-A H3N2 genome belonged to the previously described H3 genotype 1 (Table 2) and was similar to the viruses responsible for the H3N2v infections detected in 2011–2013 (12). The influenza A virus detected in swine at the 6 fairs associated with the remaining 16 (88.9%) human H3N2v cases was a relatively new H3 lineage in swine. The HA gene of this virus descended from the human seasonal H3N2 virus circulating in 2010–11, which has since reassorted with enzootic swine influenza A viruses to produce novel viruses in the US swine herd (13). The other 7 gene segments in this human-like H3 reassortant virus were of the same lineages as those segments found in the cluster IV-A virus (Table 2).

Irrespective of the fair of origin, the genomic sequences of all 11 human-like H3N2 virus isolates from swine were ≥99.89% identical to each other, demonstrating clonal expansion of 1 virus across 2 states. This pattern of virus dissemination within the exhibition swine population was a hallmark of the 2012 fair season, when 306 H3N2v human cases were reported (6).

Influenza A virus was detected in pigs at each fair at least 1 day before each H3N2v virus infection was detected in humans (Figure 2). The observed lag time between the collection of human and swine samples is probably a function of the timing for active surveillance in swine (i.e., swine are sampled at the end of the fair), whereas specimens were collected from humans when they showed symptoms of influenza-like illness (Figure 2). Retrospective investigations of infections in the swine from these fairs would not have been possible if the pig sampling relied on protocols triggered by the detection of H3N2v virus cases in humans because fairs typically run for 1 week and infected swine would have been dispersed before sampling could have occurred.

Conclusions

Variant influenza infections in humans continue to occur through contact with exhibition swine; often, the cases are in swine exhibitors with close and prolonged swine exposure. The concurrent detection of genetically identical influenza A viruses from exhibition swine across 2 states illustrates the rapidity with which this virus, and potentially other pathogens, can move within the highly mobile exhibition swine population. In addition to the zoonotic risks of influenza A virus, this pattern serves as a warning of possible dissemination of other emerging or high-consequence diseases in swine. Management practices common in the exhibition swine industry (i.e., frequent exhibition and relaxed biosecurity) facilitate the rapid dissemination of influenza virus across a large geographic landscape (14). Collaboration between animal and public health officials facilitated this investigation. Methods to control intraspecies and interspecies influenza virus transmission during swine shows have been outlined by the National Association of State Public Health Veterinarians (http://nasphv.org/Documents/Influenza_Transmission_at_Swine_Exhibitions_2016.pdf).

The recovery of human-like H3 influenza A viruses from exhibition swine supports previous studies demonstrating that the US commercial swine herd can serve as an influenza A reservoir for the much smaller exhibition swine population, which is more accessible to humans. Within the US commercial herd, the proportion of H3 isolates containing human-like H3 nearly doubled to 46% in spring and summer 2016 (data not shown). Whereas human-like H3s have been circulating, reassorting, and becoming more prevalent in the commercial swine population since 2012, introduction and expansion of the human-like H3 reassortant influenza A viruses in exhibition swine facilitated documented zoonoses from this genotype. The path traversed by this human-like H3, from initial introduction from humans to swine until the zoonotic transmission events of 2016, demonstrates how novel viruses can be generated and maintained in animal populations and, subsequently, can infect humans through specific ecologic niches like swine exhibitions or live-animal markets (15). Therefore, continued surveillance in swine populations is imperative for detecting novel influenza A viruses that threaten swine and human health.

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|---------------------------------|------------------|------------------|-------------------|------------------|</p>
<table>
<thead>
<tr>
<th>Fair</th>
<th>ILI among swine reported</th>
<th>No. swine sampled</th>
<th>rRT-PCR</th>
<th>Isolation</th>
</tr>
</thead>
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<tr>
<td>A</td>
<td>Yes</td>
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<td>20 (100)</td>
<td>18 (90)</td>
</tr>
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<tr>
<td>C</td>
<td>No</td>
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<td>18 (90)</td>
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<tr>
<td>D</td>
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<td>20</td>
<td>20 (100)</td>
<td>18 (90)</td>
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<tr>
<td>F</td>
<td>No</td>
<td>20</td>
<td>15 (75)</td>
<td>12 (60)</td>
</tr>
</tbody>
</table>

*Nasal swab or nasal wipe samples were collected from swine at the end of the fair. ILI, influenza-like illness; rRT-PCR, real-time reverse transcription PCR.
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<table>
<thead>
<tr>
<th>Genotype</th>
<th>PB2</th>
<th>PB1</th>
<th>PA</th>
<th>HA</th>
<th>NP</th>
<th>NA</th>
<th>M</th>
<th>NS</th>
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<tbody>
<tr>
<td>H3 genotype 1, n = 2</td>
<td>trig</td>
<td>trig</td>
<td>trig</td>
<td>Swine cluster IV-A</td>
<td>trig</td>
<td>2002</td>
<td>pdm</td>
<td>trig</td>
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<td>Human-like H3, n = 11</td>
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Figure 2. Timeline of detection of human and swine influenza A virus isolates at agricultural fairs in 2016. Isolates recovered are shown as squares for swine and circles for humans; colors indicate the fair attended. One person was exposed to pigs at 3 fairs (C, E, and H). Fair H is an eighth location not described in this study.
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<th>No. swine sampled</th>
<th>No. (%) positive rRT-PCR</th>
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<tbody>
<tr>
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<td>14 (70)</td>
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Influenza A(H3N2) Virus at Agricultural Fairs, USA

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