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Address for correspondence: Andrew S. Bowman, The Ohio State University, Department of Veterinary Preventive Medicine, 1920 Coffey Rd, Columbus, OH 4310, USA; email: bowman.214@osu.edu
Influenza A(H3N2) Virus in Swine at Agricultural Fairs and Transmission to Humans, Michigan and Ohio, USA, 2016


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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 interspecies 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).

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Illness among swine was reported at only 2 of the fairs (fairs A and E), suggesting that subclinical influenza A infections in pigs remain a threat to public health (3).

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 genes in the cluster IV-A H3N2 genome 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
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