Avian influenza viruses occasionally cross the species barrier, infecting humans and other mammals after exposure to infected birds and contaminated environments. Unique among the avian influenza A subtypes, both low pathogenicity and highly pathogenic H7 viruses have demonstrated the ability to infect and cause disease in humans (1,2). In the eastern and northeastern United States, low pathogenic avian influenza (LPAI) A(H7N2) viruses circulated in live bird markets periodically during 1994–2006 (3) and caused poultry outbreaks in Virginia, West Virginia, and North Carolina in 2002 (4). During an outbreak in Virginia in 2002, human infection with H7N2 virus was serologically confirmed in a culler with respiratory symptoms (5). In 2003, another human case of H7N2 infection was reported in a New York resident (6); although the source of exposure remains unknown, the isolated virus was closely related to viruses detected in live bird markets in the region. Because of the sporadic nature of these and other zoonotic infections with influenza H7 viruses throughout the world, the World Health Organization (WHO) recommended development of several candidate vaccine viruses for pandemic preparedness purposes, including 2 vaccines derived from North American lineage LPAI viruses, A/turkey/Virginia/4529/2002 and A/New York/107/2003 (7).

The Study
On December 19, 2016, the New York City Department of Health and Mental Hygiene collected a respiratory specimen from a veterinarian experiencing influenza-like illness after exposure to sick domestic cats at an animal shelter in New York, NY, USA. The specimen tested positive for influenza A but could not be subtyped. Specimen aliquots were shipped to the Wadsworth Center, New York State Department of Health (Albany, NY, USA), and to the Centers for Disease Control and Prevention (CDC; Atlanta, GA, USA). Next-generation sequencing performed at the New York State Department of Health generated a partial genomic sequence (6 of 8 influenza A virus gene segments) that aligned most closely with North American lineage LPAI A(H7N2) viruses. North American lineage H7 real-time reverse transcription PCR (rRT-PCR) testing and diagnostic sequence analysis performed at CDC confirmed the sample to be positive for influenza A but could not be subtyped. Specimen aliquots were shipped to the Wadsworth Center, New York State Department of Health (Albany, NY, USA), and to the Centers for Disease Control and Prevention (CDC; Atlanta, GA, USA). Next-generation sequencing performed at the New York State Department of Health generated a partial genomic sequence (6 of 8 influenza A virus gene segments) that aligned most closely with North American lineage LPAI A(H7N2) viruses. North American lineage H7 real-time reverse transcription PCR (rRT-PCR) testing and diagnostic sequence analysis performed at CDC confirmed the sample to be positive for influenza A(H7N2) virus. Virus isolation was attempted by inoculating the sample in 10-day-old embryonated chicken eggs and MDCK CCL-34 and CRFK (Crandell-Rees Feline Kidney) cell lines (American Type Culture Collection). A/New York/108/2016 was successfully isolated from eggs but not from MDCK or CRFK cells. Codon complete sequencing of the egg-isolated virus (GISAID accession nos. EPI944622–9; http://www.gisaid.org) showed no nucleotide changes compared with the hemagglutinin (HA) and neuraminidase (NA) gene segments sequenced directly from the clinical specimen. The virus was nearly identical (99.9%) to a virus isolated...
from a cat, A/feline/New York/16-040082-1/2016, from a New York shelter where the veterinarian had worked; the cat died of its illness. Phylogenetic analysis of the cat and human viruses showed that their genomes were closely related to LPAI A(H7N2) viruses that were circulating in the northeastern United States in the early 2000s (online Technical Appendix Figure, https://wwwnc.cdc.gov/EID/article/23/12/17-0798-Techapp1.pdf).

Analysis of the HA gene segments revealed that A/New York/108/2016 and A/feline/New York/16-040082-1/2016 were phylogenetically related to H7N2 viruses that were circulating in the northeastern United States in the early 2000s (online Technical Appendix Figure, https://wwwnc.cdc.gov/EID/article/23/12/17-0798-Techapp1.pdf).

Additional molecular characterization of the HA1 protein showed 20 aa differences between A/New York/108/2016 and A/turkey/Virginia/4529/2002 (26 aa in both HA1 and HA2; Figure 2). The substitution A125S resulted in a gain of glycosylation in the HA protein of A/New York/108/2016, previously correlated with increased replication efficiency and wider tissue distribution of A/Netherlands/219/2003 (H7N7) (12). The substitution of T183I was shown in other avian influenza viruses (e.g., H5N1) to enhance binding to mammalian sialic acid receptors (13). Four of the 20 aa changes were in residues associated with antibody recognition at antigenic site B (E177G, S180N, T183I, and S188N) and antigenic site C (R269G).
To determine the effect of these differences on antigenicity, we assessed the relationships in a 2-way hemagglutination inhibition assay, using a panel of ferret antisera raised to related H7 viruses (Table). The results showed that A/New York/108/2016 and A/feline/New York/16-040082-1/16 reacted with α-A/turkey/Virginia/4529/2002 postinfection ferret antisera (2-fold reduction of the hemagglutination inhibition titer compared with the A/turkey/Virginia/4529/2002 homologous titer) and α-A/New York/107/2003 antisera (8-fold reduction compared with the A/New York/107/2003 homologous titer). These data suggest that the A/turkey/Virginia/4529/2002 candidate vaccine virus would provide cross protection if vaccination against the 2016 H7N2 viruses was needed. Both A/turkey/Virginia/4529/2002 and A/New York/107/2003, however, reacted poorly with the antiserum raised against A/New York/108/2016.

A 20-aa deletion in the NA stalk region, considered a genetic marker of poultry-adapted viruses (I4), was also identified in the human and feline H7N2 viruses. No genetic markers known to reduce susceptibility to the NA inhibitor class of antiviral drugs were identified in the NA gene. Results of the NA inhibition assay indicated that the H7N2 viruses were susceptible to 4 NA inhibitors: oseltamivir, peramivir, and laninamivir (data not shown). The acquisition of many genetic changes throughout the genome of the human and cat H7N2 viruses we report, however, suggests onward evolution of the virus since it was last detected in poultry and wild birds. We found that the human virus bound to α-2,6–linked sialic acid receptors, which are more common in mammals, yet retained α-2,3–linked sialic acid binding, indicating that it has dual receptor specificity; this information can be used in pandemic risk assessment of zoonotic viruses. Although human infections with LPAI A(H7N2) viruses have occurred previously, we know of no other reported instances of direct transmission from a cat to a human.

Dr. Marinova-Petkova is a microbiologist with the Influenza Division, National Center for Immunization and Respiratory Diseases, Centers for Disease Control and Prevention, Atlanta, Georgia, USA. Her research interests include studying influenza viruses at the animal–human interface, influenza virus evolution, and animal models for risk assessment of zoonotic pathogens.

References
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Avian Influenza A(H7N2) Virus in Human Exposed to Sick Cats, New York, USA, 2016

Technical Appendix

Methods

Influenza A Confirmation and Subtyping of the Clinical Specimen

RNA was extracted from the clinical specimen using Qiamp viral RNA purification kit (QIAGEN, Hilden, Germany) at Wadsworth Center, and MagNA Pure Compact Nucleic Acid Isolation Kit I (Roche Diagnostics Corporation, Indianapolis, IN, USA), at the Centers for Disease Control and Prevention (CDC). Influenza A virus was confirmed by using rRT-PCR with primers and probe for detection of universal influenza A Matrix gene (CDC Laboratory Support for Influenza Surveillance, Centers for Disease Control and Prevention, Atlanta, GA, USA); and subtyped using specific primers and probe for North American lineage influenza A(H7) viruses (primer and probe sequences available upon request). The A(H7N2) subtype was further confirmed by Sanger sequencing analysis of PCR products amplified using a single step RT-PCR reaction with H7 and N2-specific primers (available upon request). Illumina next-generation sequencing of the viral RNA was performed using MiSeq analysis (Illumina, San Diego, CA, USA) and sequence data was analyzed with IRMA (1).

Virus isolation

Virus isolation was attempted in Madin Darby Canine Kidney (MDCK) and Crandell-Rees Feline Kidney (CRFK) cell lines (ATCC), and in 10-day-old embryonated chicken eggs (2). Cell cultures were incubated at 35°C for 72 hours and checked for cytopathic effect twice daily. Inoculated eggs were chilled after 48 hours of incubation at 35°C. Presence of influenza A virus in the allantoic fluids of eggs, but not cell-culture supernatants, was confirmed by positive reaction hemagglutination with 0.5% suspension of turkey erythrocytes.
Viral Genome Sequencing, Phylogenetic Analysis, and HA Monomer Protein Structure Modeling

Codon complete genome sequencing of virus isolated in embryonated chicken eggs was performed using MiSeq analysis. Gene sequences were submitted to GISAID with the following accession numbers: PB2, EPI944626; PB1, EPI944627; PA, EPI944625; HA, EPI944629; NP, EPI944622; NA, EPI944628; MP, EPI944624; and NS, EPI944623. Reference sequences for the phylogenetic reconstruction were retrieved from the GenBank and GISAID databases (3,4). Codon complete genome sequences were aligned via MUSCLE (5) and HA sequences were trimmed to the start of the mature H7 HA protein sequence using BioEdit v7.0 (6). Neighbor-joining phylogenetic trees (Jukes-Cantor model) with 1,000 bootstrap replicates were constructed using MEGA 5.05 (7). The model of HA monomer structure was generated using SWISS – Model (8) with 3M5G as the starting model. All the structural figures were generated using MacPyMOL (9).

Antigenic Characterization

Hemagglutination inhibition (HI) testing was performed by using selected subtype H7 WHO candidate vaccine viruses and CDC reference viruses of the North American lineage, as well as postinfection ferret antisera produced against H7 viruses of the North American lineage (see Table in main article). Turkey erythrocytes at 0.5% concentration were used for the HI test (10). All antisera used in the HI test were treated with receptor-destroying enzyme (Denka Seiken, Tokyo, Japan) according to the manufacturer’s recommendations, and used at 1:10 starting dilution.

Glycan Microarray Analysis

Glycan microarray slides were produced under contract for CDC using a glycan library generously provided by the Consortium for Functional Glycomics (www.functionalglycomics.org), funded by National Institute of General Medical Sciences grant GM62116 (Technical Appendix Table, glycans used for analyses in these experiments). Virus preparations were diluted in phosphate-buffered saline (PBS) with 2% (wt/vol) bovine serum albumin to an HA titer of 128. Virus suspensions were applied to the slides, and the slides were incubated in a closed container (at 4°C) subjected to gentle agitation for 1.5 hours. Unbound virus was washed off with brief sequential rinses in PBS with 0.05% Tween 20 (PBS-T) and PBS. The slides were then immediately incubated with ferret serum raised against A/New York/108/2016 (30 min); a biotinylated anti-ferret IgG antibody (Rockland) in combination with
streptavidin-Alexa Fluor488 conjugate (30 min) (Thermo Fisher, Waltham, MA, USA), with brief PBS-T/PBS washes being performed after each incubation. After the final PBS–T/PBS washes, the slides were washed briefly in deionized water, dried by a gentle steam of nitrogen gas, and immediately subjected to imaging. Fluorescence intensities were detected using an Innoscan 1100AL scanner (Innopsys, Carbonne, France). Image analyses were carried out using ImaGene 9 image analysis software (BioDiscovery, El Segundo, CA, USA).

References


### Technical Appendix Table. Glycan microarray for H7N2 viruses

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*Colors represent glycans that contain α-2,3 sialic acid (SA) (blue), α-2,6 SA (red), α-2,3/α-2,6 mixed SA (purple), N-glycolyl SA (green), α-2,8 SA (brown), β-2,6 and 9-O-acetyl SA (yellow), and non-SA (gray).
**Technical Appendix Figure.** Neighbor-joining phylogenetic trees of the (A) HA, (B) NA, and (C–H) internal genes. The human and feline H7N2 isolates are green. All candidate vaccine viruses are red and HI reference viruses are blue. Amino acid differences were calculated based on A/turkey/Virginia/4529/2002; bootstraps >50 generated from 1,000 replicates are shown at branch nodes. The scale bar represents nucleotide substitutions per site.