The 3 patients infected with W135 in our study did not receive bivalent meningococcal vaccines. W135 meningococcal disease appears to be an emerging problem that should be investigated epidemiologically. These patients highlight the need for further epidemiologic surveillance to monitor changes in the incidence of meningococcal disease caused by W135 and for improved public health disease control strategies in the future.

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Avian Influenza (H5N1) Outbreak among Wild Birds, Russia, 2009

To the Editor: Highly pathogenic avian influenza (HPAI) virus (H5N1) has been endemic in poultry in Southeast Asia since 2003 (*1*). In April 2005, an outbreak of influenza virus (H5N1) infection was detected in wild birds on Qinghai Lake in western China (*2*). Subsequently, the Qinghai-like (clade 2.2) HPAI virus (H5N1) lineage was detected in wild birds and poultry in many countries (*1*,*3*,*4*). The source of these introductions, although still debated, is likely through bird migration (*5*).

In June 2006, an influenza (H5N1) outbreak was detected in wild birds on Uvs-Nuur Lake in western Siberia, Russia. We showed that A/duck/Tuva/01/2006, isolated during the outbreak, was highly pathogenic for chickens and mice and belonged to the Qinghai-like group (2.2 clade) (*6*).

The first case of Fujian subclade 2.3.2 influenza virus (H5N1) lineage in the Russian Far East was recorded in April 2008 (*7*). Before this case, no HPAI (H5N1) outbreaks of the Fujian lineage had been reported in Russia.

In June 2009, an outbreak of HPAI in wild birds was recorded in Mongolia (*4*) and on Uvs-Nuur Lake in Russia. RNA extracted from organs (liver, spleen, intestine) of 10 dead birds belonging to 4 species (great crested grebe [*Podiceps cristatus*], little grebe [*Tachybaptus ruficollis*], black-headed gull [*Larus ridibundus*], and spoonbill [*Platalea leucorodia*]) was positive for type A influenza RNA and for the H5 subtype by real-time reverse transcription–PCR (*8*). We isolated 2 viruses from embryonated specific antibody–negative fowl eggs. Hemagglutination (HA) and neuraminidase (NA) inhibition assays with monospecific antiserum confirmed the H5N1 subtype. Viruses were designated as A/black-headed gull/Tyva/115/2009 and A/great crested grebe/Tyva/120/2009, and sequences of their HA and NA segments were defined. No HPAI virus (H5N1) was found in cloacal swabs obtained from 36 live birds (of the 4 species listed above) from Uvs-Nuur Lake.

Phylogenetic analysis (*9*) of the HA gene (Figure) showed that viruses belong to clade 2.3.2. These viruses are clearly distinguishable from the HPAI viruses previously isolated in this Russian region in 2006, A/duck/Tuva/01/2006 (clade 2.2) but are more related to A/whooper swan/Mongolia/8/2009 and A/whooper swan/Mongolia/2/2009. For the NA

gene, isolated viruses were most closely related to viruses found in Mongolia. Analysis of NA protein determined that the viruses found are sensitive to NA inhibitors.

Both viruses were shown to be highly pathogenic for chickens (intravenous pathogenicity index 3). This finding is consistent with the results of the sequence analysis of the HA gene. The HA protein possesses a series of basic amino acids (PQRERRRKR) at the cleavage site. Several amino acid changes were found between HA of investigated viruses and viruses from clade 2.3.2 that were isolated in Russia in 2008. However, the receptor-binding site of HA (positions 222–224) was not changed.

The spread of HPAI (H5N1) west across the globe has caused serious debates on the roles of migratory birds in virus circulation (*2*,*5*,*7*). In the 2009 outbreak we describe, we doubt that wild birds were infected from local poultry because domestic poultry are not present in the Uvs-Nuur Lake region and there have been no reports of HPAI among poultry in Russia since early 2008. We suggest that wild birds brought the virus to Uvs-Nuur Lake from outside the country. Because prior to June 2009 the only case of new Fujian sub-clade 2.3.2 influenza virus (H5N1) lineage was in the Russian Far East, we believe that the virus isolated in 2009 from Uvs-Nuur Lake was probably introduced by wild birds that wintered in Southeast Asia.

Many different bird species stop at Uvs-Nuur Lake during the spring and fall migrations. Qinghai-like viruses were introduced to the region from central China by wild birds in 2006 (*6*). The introduction of the H5N1 Fujian-lineage to the lake 3 years later shows further evidence that Uvs-Nuur Lake is an major area for wild bird migration and breeding and hence an environment that could potentially support the introduction of influenza virus variants from migrating wild birds. Bodies of water such as Qinghai Lake and Uvs-Nuur Lake may play a major role in the circulation of avian influenza. Therefore, we continue to study new outbreaks thoroughly and take into account the ecology and pathobiology of the species involved. Areas where large numbers of birds congregate should be closely monitored because these areas could serve as the breeding ground for avian influenza virus variants that might spread globally. Additionally, we must keep in mind that wild bird species can vary greatly in their response to HPAI and that naturally resistant waterfowl could serve as vectors for the introduction of HPAI into new locations (*1*,*2*,*5*,*7*).

Because wild birds can be involved in virus introduction, continuing surveillance is warranted. Detection of any influenza A (H5N1) virus in wild birds in a new region should be immediately followed up with efforts to characterize the virus and to control the spread of new HPAI viruses.

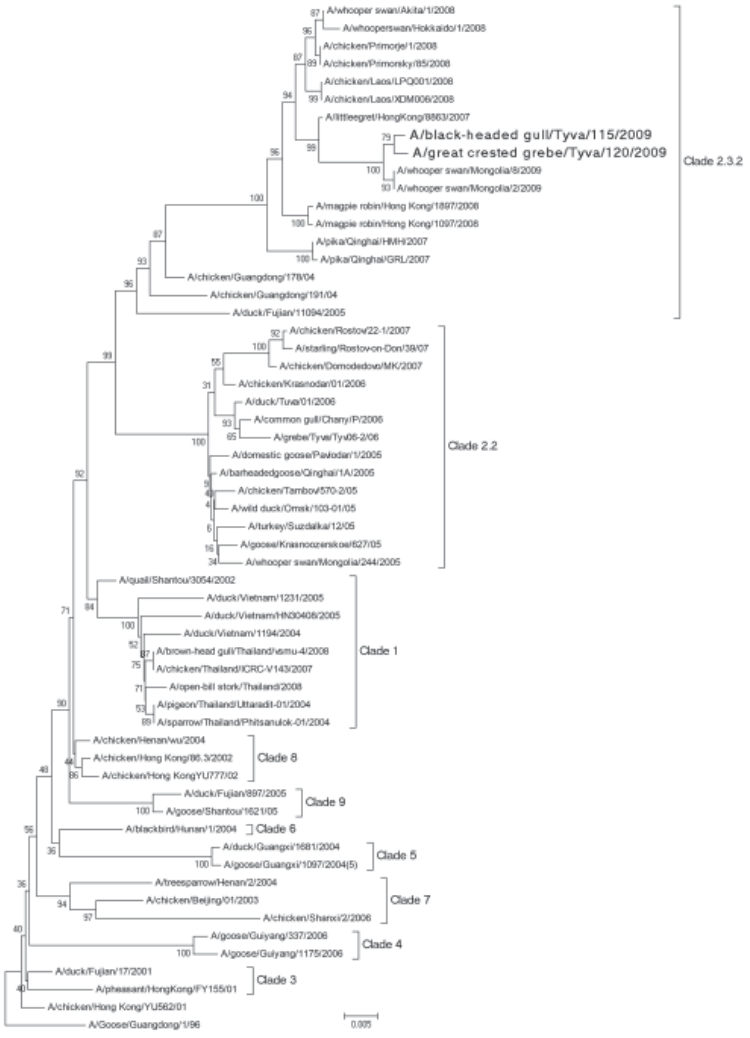

Figure. Phylogenetic tree constructed by neighbor-joining analysis (no. replications ×600) of the hemagglutinin gene segment of representative influenza virus (H5N1) isolates. Taxon names of the viruses isolated in Russia in 2006 and 2009 are in **boldface**. Scale bar indicates genetic distance.

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Detection of Pandemic (H1N1) 2009 Virus in Patients Treated with Oseltamivir

To the Editor: In April 2009, an influenza outbreak caused by a novel strain of influenza virus A (H1N1) was identified in Mexico. The rapid spread of this new virus among humans led the World Health Organization to raise the phase of pandemic alert to 6. We report results from the 2 virology laboratories from university hospitals that were involved in the surveillance network of pandemic (H1N1) 2009 in Paris at the beginning of the outbreak in France.

Patients exhibiting influenza-like illness (i.e., fever, sore throat, cough, asthenia, headache, myalgia) and who recently had traveled to countries where the pandemic (H1N1) 2009 outbreak had started (i.e., Mexico, United States, Canada, Japan) were hospitalized. Symptoms began either the day before or the day of hospitalization. Nasal-swab specimens were collected at admission by using the Virocult system (ELITech; Salon-de-Provence, France), and treatment with oseltamivir was started (75 mg, 2×/day). Pandemic (H1N1) 2009 infection was diagnosed by using rapid test QuickVue Influenza A+B (Quidel, San Diego, CA, USA) and real-time reverse transcription–PCR (RT-PCR) assays from the French National Influenza Centers or the US Centers for Disease Control and Prevention (*1*). In the case of a positive result, influenza virus in nasal secretions from patients was monitored daily by RT-PCR until viral genomes became undetectable.

From April 24 through June 7, 2009, nasal swab specimens from 234 persons (132 men; median age of all patients 33 years) were processed; pandemic (H1N1) 2009 infection was confirmed for 17 men and 15 women (median age 33 years) by RT-PCR. Results of the Quidel rapid tests were available for 27 specimens, with positive results for 9 (33% sensitivity). However, no positive result was observed with the Quidel rapid tests among the nasal swab specimens with negative RT-PCR results (100% specificity). Influenza virus detection in nasal secretions was monitored for 16 patients who had laboratory-confirmed pandemic (H1N1) 2009 infection and were treated with oseltamivir. Viral detection by RT-PCR was absent 2 to >5 days after antiviral treatment began (Figure). Significant differences were not found in sex and age of the patients (data not shown).

These preliminary virologic data obtained during the first 6 weeks of pandemic (H1N1) 2009 in France confirm the poor sensitivity of the Quidel test toward this new virus, as recently reported (*2*). Further studies are needed to evaluate the performances of other rapid tests. Hayden et al. (*3*) demonstrated that treatment with oseltamivir significantly reduced duration of viral shedding among patients infected with seasonal influenza virus A (H1N1), in comparison with a placebo group: 1.5–2.5 days vs. 3.5–5.5 days ($p = 0.003$). In our study, surprisingly, PCR results for sequential nasal swab specimens from 16 patients infected by pandemic (H1N1) 2009 and treated with oseltamivir were negative within 3 days after therapy for only 9 (56%); indeed, for 3 (19%) patients, viral genome could be detected >5 days after antiviral treatment began. These data raise questions about potential virus transmission during antiviral treatment and the possible resistance of