should include virus culture. Physicians should be aware of the limitations of laboratory diagnostic assays for LASV.

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Pandemic (H1N1) 2009 in Skunks, Canada

To the Editor: In March 2009, a novel influenza virus A (H1N1) emerged in Mexico, and, because of widespread human-to-human transmission, a global pandemic was declared in June 2009 (*1*). Although most cases have involved humans, pandemic (H1N1) 2009 has sporadically infected swine and turkeys and has also been reported in a small number of pet ferrets, cats, and captive cheetahs, and in a dog (*2*). Many of these animals were cared for by persons who experienced influenza-like illness and the owner of 1 cat who died had confirmed pandemic (H1N1) 2009 respiratory disease before the cat became ill, which suggests probable human-to animal-transmission of the virus (*2*).

During mid-December 2009–mid-January 2010, eight striped skunks (*Mephitis mephitis*) died on a mink farm near Vancouver, British Columbia, Canada. On January 12, 2010, two of the skunks were brought to the Animal Health Centre in Abbotsford, British Columbia, for postmortem examination. One skunk exhibited purulent nasal exudates. In both skunks, investigators observed splenomegaly and severe pneumonia, characterized by heavy, dark red to purple, lung lobes involving >70% of the lung field. Microscopic examination showed moderate rhinitis and severe bronchopneumonia with intralesional bacteria, areas of interstitial pneumonia, and occasional nematode larvae. Also observed were splenic extramedullary hematopoiesis, plasmacytosis of both lymph nodes and spleen, and mild plasmacytic glomerulonephritis with proteinuria.

Routine bacteriologic culture of lung showed heavy growth of *Streptococcus dysgaslactiae* subsp. *equisimilis*, *Staphylococcus aureus,* and *Hafnia alvei*. That death was caused

by uncomplicated mixed bacterial bronchopneumonia in 2 (and possibly up to 8) adult skunks over a 6-week period was considered unlikely. The presence of lungworm was considered incidental. However, the areas of interstitial pneumonia suggested that a primary viral pathogen was likely.

Molecular testing was conducted initially on fresh lung, liver, kidney, and spleen for canine distemper virus and, subsequently, for influenza A virus. The splenic and nodal plasmacytosis and plasmacytic glomerulonephritis also prompted testing for Aleutian disease virus (ADV). Organ samples were negative for canine distemper virus and positive for ADV.

Detection of influenza A virus nucleoprotein and matrix genes and hemagglutinin and neuraminidase typing was performed with real-time reverse transcription–PCR. Organ samples were positive for pandemic (H1N1) 2009, which was confirmed by sequence analysis of DNA fragments obtained in the hemagglutinin, neuraminidase, and matrix gene testing.

Primary viral interstitial pneumonia is frequently complicated by opportunistic bacterial bronchopneumonia and influenza virus A infection has been shown to predispose to pulmonary bacterial toxicity (3). Thus, we concluded that primary pandemic (H1N1) 2009 interstitial pneumonia had predisposed the 2 skunks to mixed bacterial bronchopneumonia and death. The skunks were also infected with ADV, presumably as a result of viral shedding by the minks, which are known to be ADV carriers. Striped skunks can be experimentally infected with ADV, and antibodies to ADV have been detected in wild skunks (4). Although ADV does not cause pneumonia (4), co-infection with ADV and influenza A virus is associated with higher mortality rates in minks with respiratory disease (5). Thus, ADV co-infection may have contributed to the severity of the pneumonia and the death of the skunks.

The source of the pandemic (H1N1) 2009 virus is unclear. Nasal discharge was also observed in many of the minks, which suggests that they had a respiratory viral infection. However, no diagnostic workup was undertaken. Although severe outbreaks of interstitial pneumonia on mink farms can occur (6), most natural influenza A virus infections in minks are either mild or asymptomatic (5). Thus, the minks may also have been infected with pandemic (H1N1) 2009. Many of the pandemic (H1N1) 2009 infections reported in animals are believed to have been the result of exposure to infected humans (2). Workers on the mink farm did not experience influenza-like illness. However, humans with asymptomatic pandemic (H1N1) 2009 infection may have transmitted it to the mink. Because the skunks visited the mink farm daily, transmission of pandemic (H1N1) 2009 from humans to minks to skunks is a possibility.

In view of the detection of pandemic (H1N1) 2009 virus in 2 striped skunks with fatal pneumonia, this species should now be regarded as a potential source of influenza A virus. Wild animals participate in the transmission of influenza A viruses between species, and the presence of wildlife on farms is known to be a risk factor for infection of poultry (7). Similar to raccoons, skunks express both α2,3 and α2,6 sialic acid receptors for avian and human influenza viruses in the respiratory tract (M. Shrenzel, San Diego Zoo, pers. comm.), which is believed to create the opportunity for mixed influenza infections with potential for genetic reassortment (8). Skunks, like raccoons, are highly mobile animals with large home ranges in rural and urban areas, which provides numerous opportunities for influenza A virus exposure and transmission to poultry, livestock, pets, and, ultimately, humans. The inclusion of striped skunks in wildlife influenza surveillance programs may be warranted.

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Community-acquired Oseltamivir-Resistant Pandemic (H1N1) 2009 in Child, Israel

To the Editor: During the spring of 2009, a pandemic influenza A (H1N1) virus emerged and spread globally. Initial testing of the virus found it susceptible to neuraminidase inhibitors and resistant to adamantanes (*1,2*). As of March 5, 2010, only 264 cases of oseltamivir-resistant pandemic (H1N1) 2009 infection had been reported to the World Health Organization, but the number of cases has been steadily increasing (*2*). These viruses were carrying the H275Y mutation, which conferred resistance to oseltamivir (*2*). Most of the reported cases were in immunocompromised patients who had prolonged viral shedding or in patients who had received oseltamivir prophylaxis or treatment (*1–4*). We describe an otherwise healthy 2-year-old boy with oseltamivir-resistant pandemic (H1N1) 2009 infection and a traumatic lung contusion, complicated by acute respiratory distress syndrome (ARDS). He had not received prior chemoprophylaxis or treatment with oseltamivir.

In November 2009, a healthy 2-year-old boy was admitted to the pediatric intensive care unit at the Western Galilee Hospital in Nahariya, Israel, after he had been hit by a car. One day before the accident, he had exhibited fever and cough (for which he was treated with acetaminophen). His 4-year-old brother had recovered recently from an influenza-like illness without antiviral treatment. The other household contacts were his parents, who did not have a respiratory illness.

On admission, small, bilateral lung contusions, right pneumothorax, and liver lacerations were shown on computed tomographic scan. The patient was treated with a chest tube for drainage, supplemental oxygen, and oseltamivir from hospital day 1 (30 mg 2 ×/day; child's body weight = 13 kg) and was placed in droplet isolation. Respiratory swab specimens, obtained on hospital day 1, were sent to the Israel Central Virology Laboratory (ICVL) and found to be positive for pandemic (H1N1) 2009 by real-time reverse transcription–PCR (RT-PCR). On hospital day 3, the child was intubated because of worsening respiratory distress and hypoxemia, and he required a second chest tube drain. His chest film showed bilateral pulmonary infiltrates. His condition was then treated with nitric oxide, dopamine, and milrinone for ARDS and failure of the right side of the heart. The dosage of oseltamivir was doubled on hospital day 4 because of gastric residuals. Antimicrobial drug therapy with vancomycin and piperacillin-tazobactam was added because sepsis and secondary bacterial lung infection were suspected. Because of the severity of his symptoms and persistence of fever, additional lower and upper airway specimens were sent to ICVL on hospital days 5 and 10; they were positive for pandemic (H1N1) 2009.

After these results were received, oseltamivir resistance was suspected, and his respiratory specimens were also checked by ICVL. A mixture of both wild-type and mutant pandemic (H1N1) 2009 was found in the specimens from hospital days 1, 5, and 10 by an in-house q-RT-PCR assay designed to detect the H275Y mutation (*4,5*). Further testing by sequence analysis of the neuraminidase gene showed a mixed population of wild-type and mutant pandemic (H1N1) 2009; the mutant virus was carrying the histidine-to-tyrosine substitution at position 275, which conferred the quantitative RT-PCR result and the H275Y phenotype of oseltamivir-resistant pandemic (H1N1) 2009. By the time these laboratory results were known, the patient's respiratory condition was improving without changing the oseltamivir therapy. Cultures of blood and endotracheal specimens were sterile and antimicrobial drug therapy was stopped. On hospital day 15, he was extubated, oseltamivir therapy was ended, and he was weaned off oxygen a few days later. The respiratory specimen on hospital day 20 was negative for pandemic (H1N1) 2009. No secondary influenza cases were detected among healthcare personnel or patients in the unit.

In Israel, oseltamivir resistance has been detected by ICVL in 6 cases (*5*). The fact that our patient had oseltamivir-resistant pandemic (H1N1) 2009 without a previous oseltamivir exposure is surprising because almost all cases of oseltamivir-resistance have been associated with previous oseltamivir prophylaxis or therapy and with prolonged viral shedding (which is often combined with oseltamivir therapy) in immunocompromised patients (*1–5*). Our patient did not attend daycare and his parents had not been ill recently. Therefore, he likely was infected by his older brother who probably had pandemic (H1N1) 2009 but was neither diagnosed nor treated with antiviral medications. This theory suggests that oseltamivir-resistant viruses circulate in the community with the potential to be transmitted between persons.