

When the first patients with ILI in households were unvaccinated, ILI was observed in 59 of 136 children, yielding an SAR_{b_0} of 43.4%. Among 6 exposures caused by vaccinated first patients, ILI developed in 1 person, yielding an SAR_{b_1} of 16.7%. Although not significant ($p = 0.40$), the reduction in infectiousness by vaccination was estimated to be $1 - SAR_{b_1}/SAR_{b_0} = 61.6\%$ (95% CI -132.3% to 93.6%). The SAR_{o_1} was 0% (i.e., 1 exposure to an unvaccinated person caused by a vaccinated first patient did not result in influenza). Limiting the definition of influenza to confirmed cases, all 8 exposures to vaccinated persons did not result in influenza, and SAR_{o_b} and SAR_{i_b} were 10.8% and 0%, respectively. Similarly, all 5 exposures caused by vaccinated first patients did not result in confirmed cases, and SAR_{b_0} and SAR_{b_1} were 10.5% and 0%, respectively.

Although the CIs of the estimates included zero because of the small sample size, the expected reductions in susceptibility and infectiousness were 75.0% and 61.6%, respectively, which is consistent with findings from a meta-analysis of vaccine efficacy against seasonal influenza (9). Two limitations must be noted, namely, estimates based on nonrandom samples and a case definition that relied on symptoms of case-patients. The former point cannot be explicitly addressed by a retrospective study design, but we enforced strict inclusion criteria for analyses and limited our study to healthy children. Accounting for the latter point (e.g., serologic diagnosis to capture symptomatic and asymptomatic cases) could yield slightly higher estimates than ours, provided that vaccination reduces the probability of clinical illness if infection occurs. Thus, despite these limitations and a critical need for further studies that include estimations of effectiveness (10), our results provide insight into the effects

of vaccination in reducing risks for infection and clinical attack among children exposed to pandemic (H1N1) 2009 virus in their households.

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Pandemic (H1N1) 2009 Virus in 3 Wildlife Species, San Diego, California, USA

To the Editor: The influenza A pandemic (H1N1) 2009 virus rapidly created a global pandemic among humans and also appears to have strong infectivity for a broad range of animal species (1-3). The virus has been found repeatedly in swine and has been detected in a dog, cats, turkeys, and domestic ferrets and in nondomestic animals, including skunks, cheetahs, and giant anteaters (2-4). In some cases, animal-to-animal transmission may have occurred, raising concern about the development of new wildlife reservoirs (2).

In 2009, the first recognized occurrence of pandemic (H1N1)

2009 in southern California in April was followed by a surge of cases during October through November (4). During this time, respiratory illness developed in a 12-year-old male American badger (*Taxidea taxus taxus*), a 19-year-old female Bornean binturong (*Arctictis binturong penicillatus*), and a 7-year-old black-footed ferret (*Mustela nigripes*) housed in a San Diego zoological garden.

The 3 affected animals had clinical signs that included lethargy, inappetance, dyspnea, nasal discharge, and coughing. The severity of disease in the badger and binturong necessitated euthanasia; the ferret recovered with antibiotic and fluid therapy. Postmortem examination revealed bronchopneumonia with diffuse alveolar damage in the badger and interstitial pneumonia with diffuse alveolar damage in the binturong. Bacterial cultures and Gram stains of affected lung samples were negative.

Molecular analyses for several groups of viruses, including *Herpesviridae*, *Paramyxoviridae*,

Adenoviridae, and all influenza A viruses, were performed on frozen lung samples from the badger and binturong and on frozen conjunctival and pharyngeal swabs from the ferret. Results of PCRs specific for segments of influenza A nucleoprotein, matrix protein, hemagglutinin, and neuraminidase genes were positive in samples from all 3 animals, and DNA sequencing of amplicons identified the viruses as pandemic (H1N1) 2009. Influenza A virus was not detected in samples from the ferret after it recovered. Results of PCRs for all other viruses were negative. Immunohistochemical evaluation of lung samples from the badger for antigens of influenza A virus (5) showed rare staining in bronchiolar epithelial cells (Figure).

Respiratory disease in all 3 affected animals seemed to be caused by pandemic (H1N1) 2009 virus. The badger and binturong were generally healthy, no other pathogens were detected, and pulmonary lesions were consistent with influenza pneumonia. In these animals, pandemic (H1N1)

2009 infection was especially aggressive, resulting in irreversible disease. Reports of pandemic (H1N1) 2009 virus in skunks and anteaters also describe severe disease in those species (2,3).

In contrast, the infected black-footed ferret in our study had relatively mild clinical illness, consisting only of lethargy. This finding was surprising given recent experimental studies that reported the current pandemic (H1N1) 2009 virus was more pathogenic in domestic ferrets (*Mustela putorius furo*) than typical seasonal influenza viruses (6). However, several factors could have resulted in the low level of disease in this animal, such as prior immunity to influenza viruses or a low exposure dose. It is also possible black-footed ferrets are innately more resistant to influenza infection than domestic ferrets.

The origin of infection in these cases was not determined but was most likely an infected human. All animals had some level of contact with caretakers or veterinarians and were housed separately from other wildlife species. None of the potential human sources of virus had clinical signs before the animals became ill; however, influenza infections in humans can often be mild (7). Wild animals, such as opossums and skunks, that occasionally enter the zoological garden, represent another possible source. Good hygiene and husbandry practices used within the enclosures of the badger, binturong, and ferret failed to prevent infection, which suggests pandemic (H1N1) 2009 is efficiently transmitted to these species. Descriptions of infection in giant anteaters and cheetahs kept under similar conditions also support high transmissibility of influenza A viruses to animals, as do ongoing findings for swine (3,4,8).

Although ferrets are known to be susceptible to influenza A virus, to our knowledge, influenza in badgers and binturongs has not been

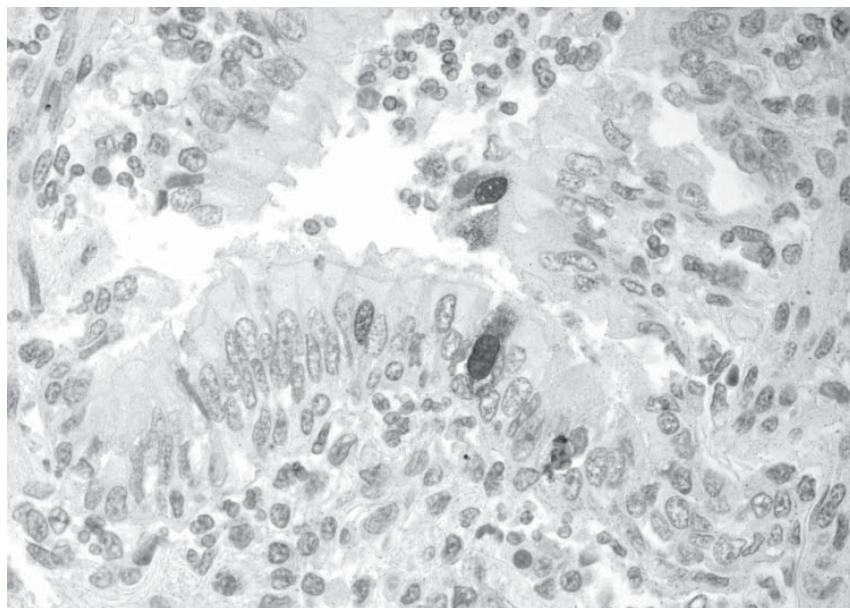


Figure. Lung section from an American badger showing immunohistochemical staining specific for the pandemic (H1N1) 2009 virus within the nucleus and cytoplasm of bronchiolar epithelial cells and concurrent inflammatory cell infiltrates; hematoxylin counterstain. Original magnification $\times 158$. A color version of this figure is available online (www.cdc.gov/EID/content/17/4/747-F.htm).

reported. Badgers and binturong have been housed in zoological gardens for decades without incidence of influenza. Increased surveillance for influenza by the scientific community during the pandemic may have resulted in the novel recognition of infection in these species. Alternatively, the current pandemic (H1N1) 2009 virus may have a broader host range and stronger virulence than viruses in the past.

Pandemic (H1N1) 2009 was first detected in humans in March 2009 and reached pandemic levels by June of that year, rapidly establishing a rich pool for the development of genetic variants. Naturally acquired disease has now been described in 10 animal species, and experimental infection has been reported in an additional 2 animals (mice and cynomolgus macaques) (9). The ubiquity of pandemic (H1N1) 2009 and its ability to infect a diverse range of hosts is worrisome for the health of wildlife and for the possibility of creating additional reservoirs that could alter the evolution of subtype H1N1 viruses by applying varied selection pressures and establishing new ways of generating unique reassortant strains.

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Hemagglutinin 222 Variants in Pandemic (H1N1) 2009 Virus

To the Editor: The biologic role of amino acid variants at position 222 of the hemagglutinin (HA) gene of pandemic (H1N1) 2009 virus in severe infections has been extensively discussed. A recent series of studies (1–3) confirm the initial suggestions that G or N in this position might confer greater pathogenic potential to the virus than to the wild type. In contrast, their data suggest that no particular pathogenicity is associated with the 222E variant because it occurs at the same frequency in severe and mild infections. Most authors also seem to agree that D222G or N appears sporadically in phylogenetically distant viruses, with limited transmissibility.

However, Puzelli et al. (4) reported transmission of a 222G mutant from son to father (with the appearance of an additional G155E mutation). In Italy, the pattern of D222 variants has been peculiar, with extremely rare appearances of 222G and high diffusion of 222E isolates. At the National Institute for Infectious Diseases in Rome, 82 isolates (GenBank accession nos. CY063455–CY063469 for new sequences in this study) were monitored for D222 variants. No 222G or N variants were detected, even in 24 severe infections, nor was the G155E mutation detected. This finding was not surprising, given the worldwide low frequency of this mutation, even in severe infections.

Conversely, D222E was detected in 12 of the 82 cases, peaking in September 2009, when it was present in most of the infections, with no overrepresentation in severe cases. Subsequently, it was substituted by different 222D viruses during the autumn–winter outbreak. The analysis of publicly available sequences from