illness among school A households originated from sources other than the school and support the approach of considering school dismissal only in conjunction with other community mitigation strategies.

In Hong Kong Special Administrative Region, People's Republic of China, where all primary schools, kindergartens, and child care centers were immediately closed for 14 days after identification of the first local case of pandemic (H1N1) 2009, school closures were concluded to have substantially decreased transmission (*4*). The applicability of these findings to communities where such sweeping measures might be less acceptable is unclear.

If school dismissal is considered as a strategy, dismissal early in the pandemic most likely would have the most impact, depending on duration of dismissal, other mitigation measures, and compliance with social distancing recommendations (which was mixed during the 2009 pandemic [*3*,*5*]). Polling of parents whose children experienced school dismissal showed high acceptance of short-term (3–5 days) dismissals and low economic impact, especially on lower income families (*3*,*6*). However, dismissal for longer periods needs to be balanced by the adverse impact on education, loss of student services, and socioeconomic impact on families (*7*–*9*).

This investigation was limited by the relatively low response rate; however, demographics for the sample in our study were similar to those of the school as a whole (*3*). Other limitations included the exclusive use of reported symptoms to document illness, possible unrecognized asymptomatic cases, and absence of similar data from later in the pandemic. The 1-week closure period might not have provided enough information to capture any effect, and comparative data were not available from schools that were not dismissed during the pandemic. Further investigation is needed to evaluate the efficacy and impact of school dismissal, including the timing of dismissal in relation to recognition of cases in a school or community and the impact of school dismissal relative to other community mitigation strategies.

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Pandemic (H1N1) 2009 Virus in Swine Herds, People's Republic of China

To the Editor: During March and early April 2009, a new swine-origin influenza A (H1N1) virus emerged in Mexico and the United States; this virus subsequently spread across the globe by human-to-human transmission at an unprecedented rate. Pandemic (H1N1) 2009 virus also affected pigs. On May 2, 2009, the Canadian Food Inspection Agency notified the World Organisation for Animal Health that the novel influenza A virus had been confirmed on a pig farm in Alberta, Canada. Infection of pigs with pandemic (H1N1) 2009 virus has been observed in multiple

countries (*1*). In this study, we report transmission of pandemic (H1N1) 2009 virus from humans to pigs in the People's Republic of China.

During August 2009–April 2010, swine influenza virus surveillance was conducted in the provinces of central and eastern China, including Henan, Hubei, Hunan, Jiangxi, and Anhui. A total of 1,021 samples, comprising tracheal mucus swabs and lungs, from pigs on 30 farms, were collected, and dozens of swine influenza viruses, H1N1, H1N2, H3N8, H9, and H10 subtypes, were isolated. Eight isolates were subtype H1N1, including 4 novel pandemic (H1N1) 2009 viruses: A/swine/Nanchang/3/2010 (H1N1) (Gen Bank accession nos. JF275917–24), A/swine/Nanchang/5/2010 (H1N1) (GenBank accession nos. JF275933–40) and A/swine/Nanchang/6/2010 (H1N1) (GenBank accession nos. JF275941–48), which were isolated from tracheal mucus, and A/swine/Nanchang/F9/2010 (H1N1) (GenBank accession nos. JF275925–32), isolated from the lung. Pigs from which the novel viruses were isolated showed mild respiratory signs, including depression, cough, and transient increase in body temperature. Compared with the sequence of A/California/04/2009 (H1N1), genomic sequencing of the 4 pandemic viruses showed 15 common point mutations, such as polymerase basic protein 2, T588I; polymerase acidic protein, A70V, P224S, D547E; hemagglutinin, P100S, D103E, S145P, T214A, S220T, I338V; nucleocapsid protein, V100I, H289Y; neuraminidase, V106I, N248D; and nonstructural protein, I123V.

Recent studies have shown that the novel pandemic (H1N1) 2009 human influenza viruses were almost avirulent for mice (50% mouse lethal dose $\geq 10^6$ PFU for A/CA/04/09 [*2*,*3*]). In this study, mice were anesthetized with ketamine/xylazine, as described (*4*) and 50 μL of phosphate-buffered saline containing the indicated doses (10^5 50% egg infectious dose) of the 4 viruses were instilled into anesthetized mice through nostrils. Interestingly, the 4 pandemic (H1N1) 2009 viruses isolated from pigs could cause systemic infection on mice. All mice had extensive loss of body weight, and some of the infected mice died within 2 weeks postinfection (data not shown).

To detect whether the 4 isolated pandemic (H1N1) 2009 viruses could cause clinical diseases in pigs and what kinds of pathologic damage could be induced in infected pigs, pigs were intratracheally challenged with the 4 pandemic (H1N1) 2009 viruses at a dose of 10^7 50% egg infectious dose and monitored for clinical signs and pathologic changes. Clinical signs in pigs were mild, and no deaths occurred. Except for slightly labored breathing, no infected pigs showed dominant signs, such as cough, nasal discharge, facial edema, and dyspnea. Body temperatures of infected pigs started to increase at 2 days postinfection (dpi), peaked ≈3 dpi at 41.5°C, and returned to the initial temperature by 5 dpi.

We observed necrosis of the tracheal wall and pneumonia foci in the 4 pandemic (H1N1) 2009 virus–infected pigs. Hematoxylin and eosin–stained sections of the trachea from the infected pigs showed mild necrotizing tracheitis. The necrotic epithelial cells were present in the lumen, and a mixed inflammatory cell infiltrate was present throughout (Figure). In the lung, we also observed alveolar septal edema and interstitial inflammatory cell infiltrates, as well as histologic changes, including alveolar epithelial hyperplasia, a mixed inflammatory infiltrate, and interstitium broadening (Figure). Additionally, immunohistochemical analysis for the distribution of viral antigens showed positive staining in bronchial epithelial cells and alveolar pneumocytes.

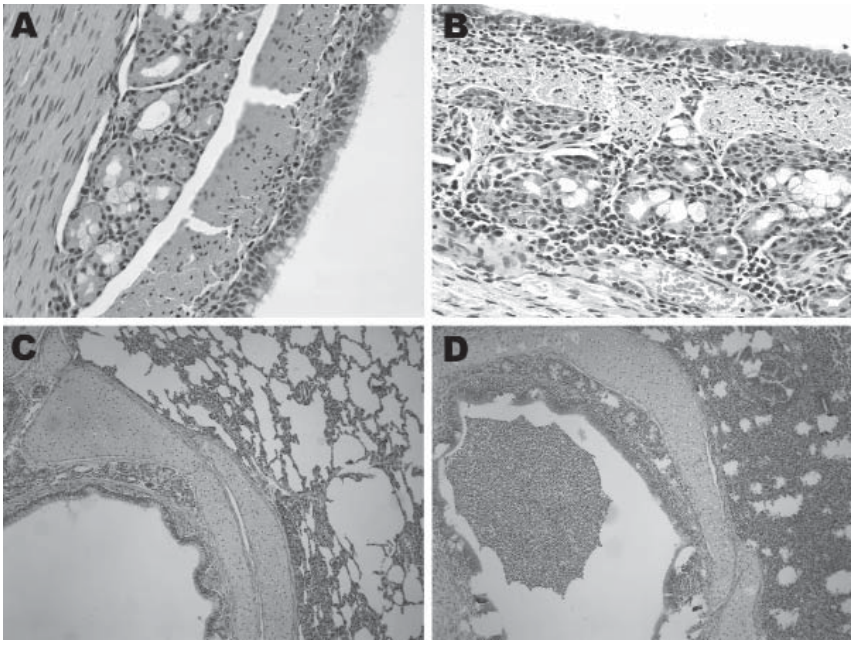

Figure. Hematoxylin and eosin–stained trachea and lung controls and samples from pigs infected with pandemic (H1N1) 2009 virus. A) Control trachea sample; B) mixed inflammatory cell infiltrate present throughout trachea sample from infected pig; C) control lung sample; D) mixed inflammatory infiltrate and interstitium broadening in lung sample from infected pig. Original magnifications: panels A and B, ×40; panels C and D, ×10. A color version of this figure is available online (www.cdc.gov/EID/17/9/101916-F.htm).

Previous studies showed that pandemic (H1N1) 2009 may have become established in swine populations in Canada, Norway, and Hong Kong (*1*,*5*–*8*). The human-to-pig transmission of pandemic (H1N1) 2009 may substantially affect virus evolution and subsequent epidemiology. Although the pandemic was mild, the virus could develop further reassortment in swine and gain virulence. On the other hand, subtype H5N1 and H9N2 viruses have become established in pigs, so the introduction of pandemic (H1N1) 2009 virus to pigs has provided the possibility for the incorporation of avian virus genes into mammalian-adapted viruses. That transmission could occur from humans to pigs and vice versa is especially troublesome. Given the possible production of novel viruses of potential threat to public health, we should emphasize influenza surveillance in pigs and establishment of the genetic basis of the viral genome for rapidly identifying such reassortment events.

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Pulmonary Disease Associated with Nontuberculous Mycobacteria, Oregon, USA

To the Editor: Nontuberculous mycobacteria (NTM) are environmental organisms ubiquitous in soil and water, including municipal water supplies. When inhaled, these organisms cause chronic, severe lung disease in susceptible persons (*1*). Recent epidemiologic studies suggest NTM pulmonary disease is increasingly prevalent in North America, with annual incidence rates of 13 cases per 100,000 population in persons ≥50 years of age and 2–4-fold higher in older age groups (*2*–*4*). The current distribution of pulmonary NTM disease has been poorly characterized with regard to environment, climate, and other factors.

We recently performed a statewide NTM surveillance project in Oregon, United States, where we documented higher pulmonary disease rates within the moister, temperate western regions of the state. Oregon is bisected north-south by mountains into 2 distinct climate zones. Western Oregon, where 87% of the state's population lives, is temperate and wet; eastern Oregon is primarily rural, with an arid, high desert climate. Our goal was to evaluate whether disease clustering within the state could be explained by population density.

For all Oregon residents who had newly diagnosed and existing pulmonary NTM disease during 2005 and 2006, we used case-patient home ZIP code and county of residence to construct statewide disease maps (*4*). We obtained state ZIP code and county-level census data for 2005 and 2006 from the Portland State University Population Research Center and used Oregon Office of Rural Health criteria to designate