(H1N1) became resistant to both oseltamivir and amantadine in a short span of 1 month. Oseltamivir-resistant A/Brisbane/59/2007-like clade 2B virus that had reassorted with A/Hong Kong/2625/2006-like clade 2C virus had apparently spread in the community and to other regions of the world. The possibility of reassortment with pandemic (H1N1) 2009 virus is a major concern. Resistance to oseltamivir of pandemic (H1N1) 2009 virus will compromise its use in treatment and render the billion-dose stockpile useless. Although the recently detected oseltamivir-resistant pandemic (H1N1) 2009 virus in Hong Kong was not a reassortant virus, we will continue to closely monitor antiviral drug resistance among circulating viruses, including pandemic (H1N1) 2009 virus and seasonal influenza virus A (H1N1), as well as influenza A (H3N2) viruses, to track how antiviral resistance evolves.

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Pandemic (H1N1) 2009 Reinfection, Chile

To the Editor: Since March 2009, influenza A pandemic (H1N1) 2009 has spread worldwide (1), and in South America, Chile was 1 of the countries most affected by the pandemic, with 21.4 cases among every 1,000 persons. Treatment guidelines in Chile recommended antiviral drug treatment with oseltamivir or zanamivir for 5 days for all patients with confirmed or suspected virus subtype H1N1 infection (2). In persons with seasonal influenza, specific antibody responses reach peak titers by 4 weeks after infection and confer protection against the infecting strain and closely related strains (3). Reinfection is rarely seen in nonpandemic influenza A. We report on 3 patients with confirmed influenza A pandemic (H1N1) 2009 reinfection after successful treatment with oseltamivir.

Patient 1, a healthy 14-year-old girl, had fever, sore throat, and nasal congestion on clinical examination. Pandemic (H1N1) 2009 infection was diagnosed by viral culture and confirmed by PCR specific for subtype H1N1 (LightMix Kit Influenza A swine H1; TIB MOLBIOL, Berlin, Germany, for Roche Diagnostic, Indianapolis, IN, Light Cycler 2.0 instrument). The patient received oseltamivir, and symptoms resolved 48 hours after treatment. Twenty days later, fever, muscle aches, and vomiting developed in the patient. Influenza A virus was isolated by viral culture. The patient received a preliminary diagnosis of seasonal influenza and was treated with amantadine. She recovered from the infection before PCR results confirmed it was caused by pandemic (H1N1) 2009 virus.

Patient 2, a 62-year-old woman, experienced a high fever, cough, and nasal congestion during a prolonged hospitalization for bowel resection after intestinal ischemia. Pandemic (H1N1) 2009 was confirmed by PCR and viral culture. Oseltamivir was administered 5 days after the onset of symptoms, and the symptoms resolved within the following 5 days. The patient had a new episode of fever, productive cough, and bronchial obstruction 2 weeks later while still hospitalized. Culture results were positive for influenza, and PCR results were positive for pandemic (H1N1) 2009. The patient was again treated with oseltamivir, and PCR results were negative for influenza after 48 hours of antiviral treatment.

Patient 3, a previously healthy 38-year-old man, underwent mitral and aortic valve replacement while hospitalized for acute mitral and aortic endocarditis due to Staphylococcus aureus. Eleven days after surgery, he had a sore throat, nasal congestion, cough, and low-grade fever. PCR test results were positive for pandemic (H1N1) 2009. The patient received oseltamivir, and respiratory symptoms resolved within 5 days. He was discharged from the hospital but was
readmitted 18 days later with nasal congestion, cough, and high fever. PCR results were again positive for pandemic (H1N1) 2009, and the patient was successfully treated with oseltamivir. 

Patient 2 and probably patient 3 acquired their infections while hospitalized, suggesting potential nosocomial transmission. No other respiratory viruses were found in any of these patients. The viral isolates were all tested (LightMix for detection of influenza A virus oseltamivir resistance [H274Y]; TIB MOLBIOL) for possible resistance to oseltamivir, but none had the resistance-implicated H274Y mutation in the neuraminidase gene.

Shedding of seasonal influenza A virus ceases within 5–7 days during natural infection and during infections treated with neuraminidase inhibitors (4). Although clearing of virus after the first infection was not documented in the 3 patients described here, it is unlikely that virus persisted between the 2 episodes of influenza since each of the patients fully recovered after specific antiviral drug treatment. However, we cannot rule out that patient 2 may have never cleared the virus due to her immune suppression. As described by mathematical modeling (5), the 3 patients described were susceptible to reinfection with pandemic (H1N1) 2009 due to the high rate of community infection and to their incomplete immunologic protection within the period of reexposure. During the current pandemic of influenza subtype H1N1, healthcare workers and patients should be aware that symptomatic reinfection might occur after a first episode of infection.

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Skin Lesion Caused by ST398 and ST1 MRSA, Spain

To the Editor: Human infections caused by methicillin-resistant Staphylococcus aureus (MRSA) sequence type 398 (ST398) have been emerging in recent years in Europe (1,2). Pigs represent a common reservoir of MRSA ST398, and working with these animals may constitute a risk factor for MRSA carriage and possible infection (2–4). We report a case of human infection caused by MRSA ST398 in Spain.

A 12-year-old girl living close to a pig farm, where her father and mother worked, sought treatment for a skin lesion on her chin. Two types of MRSA were recovered from the lesion, and it resolved after topical treatment with mupirocin over 10 days. MRSA isolates recovered were characterized by multilocus sequence typing (MLST) and by staphylococcal cassette chromosome (SCC) mec, spa, and agr typing as described (3). The presence of genes encoding Panton-Valentine leukocidin (PVL) ( lukF/lukS), toxic shock syndrome toxin-1 (tsst1), and exfoliative-toxin A and B (eta, eth) was investigated by PCR (2,3). Antimicrobial susceptibility tests were carried out by using the VITEK-2 system (bioMérieux, Marcy-l’Etoile, France), and mecA, ermA, ermB, ermC, mrsA, tetK, tetL, tetM, ant(4′)-Ia, aph(3′)-III, and aph(2″)-aac(6) resistance genes were studied by PCR (5). dfrK gene detection was performed by using primers designed from the sequence FM207105 included in GenBank (dfrK-F 5′-GAGAATCCCAGAGGATTGGG; dfrK-R, 5′-CAAGAAGCTTTTCGCTCATAAA), and the obtained ampliﬁcons were sequenced for conﬁrmation. Mutations in quinolone targets were determined by sequence analysis of grlA and gyrA genes (6).

One of the clinical MRSA isolates recovered from the lesion and typed as ST398-spa-t011 showed resistance to tetracycline, macroclides, and lincosamides and harbored 5 antimicrobial resistance genes. The second MRSA strain was typed as ST1-spa-t127 and showed a multiresistance phenotype with 11 resistance genes, as well as the Ser80Phe and Ser84Leu amino acid changes in GrlA and GyrA proteins, respectively.

To establish the MRSA nasal colonization status of the patient and of her relatives (father, mother, and brother, all of whom had contact with

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