persistently low-level parasitemia among asymptomatic immigrants is probably higher than previous estimates. Screening for malaria among immigrants long after arrival would help determine if there are any factors that influence the development of clinical malaria. Delayed screening could also be particularly relevant in certain risk groups, such as pregnant women and persons who are HIV positive. As a public health measure, such delayed screening could play a role in preventing outbreaks or reintroducing malaria in countries where it has been eradicated.

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Pandemic (H1N1) 2009 Virus Circulating in Pigs, Guangxi, China

To the Editor: A novel swineoriginated influenza A virus known as pandemic (H1N1) 2009 was first isolated from humans in Mexico in April 2009 (1), and a worldwide pandemic followed, which affected >214 countries and resulted in >18,000 deaths (2). In August 2010, the World Health Organization stated that the pandemic caused by this virus had ended. As this virus emerged, animals, including swine, turkeys, ferrets, cats, and cheetahs, were found to have been infected (3). In addition, transmission from humans to pigs in porcine herds has been reported (4).

Swine influenza Α virus belongs to the family Orthomyxoviridae and is a causative agent of respiratory disease in pigs (5). Currently, 3 subtypes of influenza viruses are circulating in the swine population globally: H1N1, H3N2, and H1N2 (6,7). Pigs can be simultaneously infected with avian influenza viruses and human influenza viruses, and the viruses can exchange genes and produce new variants, which suggests that pigs have become mixing vessels for influenza viruses (8). Pandemic (H1N1) 2009, caused by a virus usually circulating in pigs in Europe and Asia, is a triple hybrid that contains swine, human, and avian virus gene segments, which further emphasizes that SIVs pose a serious threat to public health. We describe an outbreak of pandemic (H1N1) 2009 virus, which was isolated from a pig farm in Guangxi Province, People's Republic of China, and report the consequences of subsequent epidemiologic studies.

In January 2011, an outbreak of severe respiratory problems occurred in pigs on a pig farm. Nine hundred growing and fattening pigs exhibited clinical signs of influenza, including fever, cough, runny nose, loss of appetite, lethargy, edema, watery eyes, conjunctivitis, diarrhea, and vomiting. The incidence rate was ≈80%, and the death rate was 22%. The outbreak lasted ≈2 weeks. However, no outbreak of respiratory disease occurred in other pig farms in the same area simultaneously, and no evidence of human-to-pig transmission was found.

We collected lung samples from 3 dead pigs with underlying illness for reverse transcription PCR and virus isolation in 10-day-old specific pathogen-free embryonated chicken eggs. Viral RNA was extracted from the tissue suspension and allantoic fluids. Virus isolation was assessed by hemagglutination inhibition (HI) assay and neuraminidase inhibition assay by using a panel of reference serum samples (National Reference Laboratory for Avian Influenza, Harbin Veterinary Research Institute, Harbin City, China). Later, 3 viruses were isolated. All of them had hemagglutination (HA) activity, and the HA titers ranged from 128-256. HA-positive isolates were further identified as subtype H1N1. Subsequently, nucleotide sequences of the 8 viral genes were amplified and sequenced (GenBank accession nos. JN222372-JN222379). This analysis showed high identities to a pandemic strains A/California/04/2009 (H1N1), hemagglutinin (99.2%),neuraminidase (99.1%), matrix (99.3%), nucleoprotein (99.5%), nonstructural protein (98.5%), polymerase acidic protein (98.5%), polymerase basic protein 1 (99.7%), and polymerase basic protein 2 (99.6%) genes. Bacteria were cultured from spleen, liver, and heart-blood samples from 5 pigs. Four pigs were infected with porcine streptococci, and 1 pig with mild symptoms was negative for the bacteria.

We sought to gain more insight into the epidemiology of pandemic (H1N1) 2009 virus in Guangxi

Province and collected 600 bronchial swab samples and 200 blood serum samples when pigs were slaughtered every month from February through June 2011. The samples were used to isolate virus in 10-day-old specific pathogen-free embryonated chicken eggs, and then the virus isolates were subjected to sequencing of a partial genome of the HA gene. Overall, we obtained 10 strains of subtype H1N1 influenza virus, including 5 strains of classic swine H1N1, 3 strains of Eurasian avianlike H1N1, and 2 strains of pandemic (H1N1) 2009 virus, which were derived from 3,000 bronchial swab samples.

In addition, a serologic survey was implemented by using HI testing with pandemic (H1N1) 2009 virus and SIV (H1N1) antigens. Serologic studies showed that 251 of the 1,000 samples tested had positive HI titers for pandemic (H1N1) 2009 virus, and 248 of these samples had positive HI titers for SIV (H1N1). Notably, crossreactivity of pandemic (H1N1) 2009 virus between H1 subtype viruses has been reported recently in pigs (9). However, the higher rate of positive test results indicated that swine serum samples contained antibodies against pandemic (H1N1) 2009 virus.

Our findings strengthened previous data by showing that growing and fattening pigs are susceptible to infection of pandemic (H1N1) 2009 virus (4). Analysis of the complete genome sequence of the subtype H1N1 isolates suggests that no gene reassortment occurred. The results of serologic studies demonstrated that uninfected pig farms are also susceptible to pandemic (H1N1) 2009 virus infection. Our results suggest that the pandemic virus is currently circulating in swine populations and posing a challenge to pigs in southern China. Increasing serologic surveillance of pigs for prevention and better control of pandemic influenza is urgently needed in China.

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Cutaneous Melioidosis in Adolescent Returning from Guadeloupe

To the Editor: Melioidosis is an emerging zoonosis caused by a highly invasive and drug-resistant bacterium, *Burkholderia pseudomallei*, that is found in soil and is endemic to Southeast Asia and the Pacific region. Few cases occur in other regions (most imported by travelers) (1-5), but sporadic cases have originated from the Caribbean (6-8).

Melioidosis can manifest many years after exposure to *B. pseudomallei* and can cause severe, systemic infection, including multiple abscesses of internal organs and skin. A less severe manifestation, primary cutaneous melioidosis, causes skin

lesions and milder clinical illness. We describe an adolescent patient who had a benign, cutaneous form of melioidosis; she had recently returned to France from Guadeloupe, a Caribbean archipelago.

A 15-year-old girl without a medical history, except for asthma, was evaluated in September 2010 for muscle weakness, weight loss of 15%, cough, and fever ≥40°C. During a trip to Guadeloupe 3 weeks before, she had been infected by dengue virus, along with her brother and father, who recovered rapidly. Treatment with amoxicillin and clavulanic acid was started after her return to France, despite the lack of a clear diagnosis, and induced a slight decrease in fever.

Clinical examination showed a body mass index <15, multiple adenopathies (<10 mm), papulous skin eruptions, small and an inflammatory 15-mm-wide tumefaction on the upper arm, evoking an adenopathy on ultrasound investigation. Biological screening 2 weeks later showed persistence of inflammation. Results of serologic tests for cytomegalovirus, Epstein-Barr virus, parvovirus B19, chikungunya virus, Rickettsia, Coxiella, Chlamydia, Brucella, and Borrelia spp. did not show acute infectious disease; results were positive for recent mycoplasma infection, despite absence of typical signs and symptoms. A 2-week treatment regimen with spiramycin was started; general improvement followed, and the cough resolved.

The tumefaction of the upper arm persisted, and a biopsy was performed. Histologic results were nonspecific; culture on sheep's blood Columbia agar and chocolate agar produced small colonies of gram-negative bacilli after 24 hours' incubation at 35°C in an atmosphere of 5% CO₂. This bacillus was later identified as *B. pseudomallei* by using the Vitek2 test card (bioMérieux, Marcy l'Etoile, France). Identification was confirmed by sequencing of 16S rRNA.

The patient was treated with intravenous ceftazidime (150 mg/kg for 10 d), followed by oral cotrimoxazole (800 mg of trimethoprim and 160 mg of sulfamethoxazole, $2\times/d$), with a total treatment duration of 12 weeks. Eleven weeks after treatment ended, the patient had recovered, and the tumefaction of the arm had disappeared.

The differential diagnosis for melioidosis primary cutaneous includes pyogenic abscesses, insect bites, mycobacterial other granulomatous lesions, adenopathies, but melioidosis is usually not suspected in these conditions, particularly in patients from non-disease-endemic regions such as the Caribbean. Clinical phenotypes of melioidosis range from asymptomatic carriage to fulminant shock syndrome (1-5); death rates for the latter are $\approx 100\%$ (3). Subacute melioidosis may be associated with pulmonary and general signs; chronic variants could give rise to abscesses or septicemia in cases of concomitant immunodeficiency (1-5),decades after exposure. Signs and symptoms of melioidosis can mimic those of tuberculosis, even though there is no link between the infectious agents (2,4).

Cutaneous melioidosis be primary (a single, nonspecific, ulcerated measuring from several millimeters to several centimeters) or secondary (multiple lesions associated with visceral infection). In a study of 486 melioidosis patients in Australia, 58 (12%) had the primary cutaneous form (9). These cases were characterized by younger patient age (more common among children <16 years of age), higher frequency during the dry season, better prognosis in spite of a possible chronic evolution, and absence of classic risk factors (9) such as diabetes, alcoholism, chronic renal or pulmonary infections, surgery, pregnancy, or cystic fibrosis (1-5).