of 54°C, and 45 amplification cycles. PCR blanks containing all reagents except for DNA and extraction blanks were included in every PCR set.

Results of the amplification reactions are listed in the Table. All accompanied extraction and PCR controls remained free of amplification products. All amplicons resulting from suicide PCRs were sequenced. Amplicons resulting from the use of primer pairs YP14F/YP13R and pst-F/pst-R matched the reference sequence to 100% (GenBank accession no. AL109969.1). Amplicons resulting from the use of primer pair PCP-F/PCP-C matched this reference sequence to only 97.78%. This deviation is because of a 2-bp insertion (2 Ts, positions 8531 and 8532, GenBank accession no. AL109969.1) at Y. pestis strain CO92 plasmid pPCP1. The sequences obtained from 3 persons’ remains showed in the pPCP1 sequence section between nucleotide positions 8528–8532 only 3 Ts instead of 5 Ts described for Y. pestis strain CO92 plasmid pPCP1 (GenBank accession no. AL109969.1). The sequences found in this study were deposited in GenBank under accession nos. HQ290521–HQ290523.

To conclude, the successful recovery of several Y. pestis plasmid pPCP1 DNA sequences in skeletal finds from the mass burial site excavated in Manching-Pichl suggests that these persons died of plague. Moreover, our findings constitute a molecularly supported confirmation for the presence of Y. pestis, the etiologic agent of plague, in late medieval (1250–1500 CE) southern Germany. In future studies, we will attempt to recover chromosomal Y. pestis DNA from the mass grave skeletal remains to obtain clues as to the specific Y. pestis strain and the microbiology of past plague in Europe.

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References


Letters

Two Clusters of HIV-1 Infection, Rural Idaho, USA, 2008

To the Editor: Prevalence of HIV-1 infection in rural areas of the United States has been increasing (1). During 2003–2007, an average of 30 (range 24–42) cases of new HIV-1 infection diagnoses per year among Idaho residents were reported. Of the 152 reported cases during this period, 54 (36%) were related to a person living in a rural area of ≤75,000 residents and a 60-minute drive from an urban area (2). Of these 54 cases, 19 (35%) were in men who have sex with men (MSM), 5 (9%) were in injection drug users (IDU), and 2 (4%) were in those in both categories.

In March 2008, a cluster of newly identified HIV-1 infections that included 5 cases (cluster A) in a rural southeastern Idaho city (city A) was reported to the Idaho Department of Health and Welfare. Two patients were men and the median age was 26 years (range 18–32 years). One patient was an IDU (Table). Through epidemiologic investigation, 3 additional patients were suspected to be IDUs, but confirmation was not practicable. All reported methamphetamine use. One man and 2 women reported both male and female sex partners.

During September–December of that year, another increase in newly identified HIV-1 infections in southeastern Idaho (cluster B) was reported
to Idaho Department of Health and Welfare. Cluster B included 10 cases, all among men who reported living within a 50-mile radius of city A, with most in a rural city (city B) located <30 miles from city A. The median age of the men in cluster B was 24 years (range 18–37 years). Each case was epidemiologically linked to at least 1 other case in the cluster; each patient reported having had unprotected sex with male partners. Although we suspected transmission of HIV-1 between persons in clusters A and B, whether the clusters were linked epidemiologically remained unclear after an initial investigation.

Although the primary use of HIV-1 sequence data is to assist clinicians in selecting antiretroviral (ARV) therapy, public health practitioners can use HIV-1 sequences from cases and compare those with HIV-1 sequences from others living in the region to explore phylogenetic associations and possible HIV transmission clusters (3). To evaluate links between clusters A and B, HIV-1 pol consensus sequence data for 4 of the 5 cases from cluster A and 6 of the 10 cases from cluster B were obtained from 5 commercial laboratories. No case-patients had received ARV. Additionally, we used sequence data from a patient residing in city B who had received an HIV-1 diagnosis in December 2008 but was not epidemiologically linked to either cluster. HIV-1 control sequences were obtained from 2 local clusters had genetic similarity previously believed to be unrelated to 2 local clusters had genetic similarity to cluster A. Limitations of this investigation include the inability to obtain HIV sequences from all persons identified in clusters A and B and an inability to confirm high-risk behaviors for all identified case-patients.

Previous HIV clusters have demonstrated that infectious persons can spread HIV quickly within a social network and highlighted the importance of timely prevention activities to limit HIV transmission in a community (6,7). Use of phylogenetic analysis of HIV-1 sequences obtained from commercial laboratories showed that clusters A and B were not epidemiologically related and helped target appropriate and specific HIV prevention activities.

### Acknowledgments

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*ID, identification; WSMW, women who have sex with men and women; SIDU, sex with injection drug users; MSMW, men who have sex with men and women; IDU, injection drug user; MSW, men who have sex with women; ND, not determined; MSM, men who have sex with men; WSM, women who have sex with men.
†Cluster A associated with injection drug use; cluster B associated with MSM.
‡HIV-1 sequence was not available for molecular analysis.
§HIV-1 sequence was similar to controls but not to sequences from either cluster A or B.
¶Case with no epidemiologic link to cluster A or cluster B.
for providing control data; Eoin Coakley, Shannon Utter, and Christopher Lockhart for assistance with acquisition of sequence data; and Alexandra Oster for guidance during this investigation.

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Pandemic (H1N1) 2009 and Oseltamivir Resistance in Hematology/Oncology Patients

To the Editor: Tramontana et al. (1) recently described characteristics and oseltamivir resistance in hematology and oncology patients infected with pandemic (H1N1) 2009 virus. Such cases merit further study because concurrent medical problems in immunosuppressed patients may obscure and delay diagnosis and management of pandemic (H1N1) 2009 infections. Moreover, severe complications of such infection may be more likely to develop in immunosuppressed patients (2). During the winter of 2009, oseltamivir-resistant pandemic (H1N1) 2009 virus infection was diagnosed for 4 patients at Duke University Medical Center. We describe the clinical features of the infections, the challenges associated with diagnosis of pandemic (H1N1) 2009 virus infection, and the clinical outcome for the infected patients.

Four immunocompromised patients who received chemotherapy and immunotherapy for solid-organ and hematologic malignancies were hospitalized at our tertiary care medical center during October–November 2009, a period of peak activity of pandemic (H1N1) 2009 in surrounding communities in North Carolina (3). These 4 case-patients experienced symptoms attributable to pandemic (H1N1) 2009 from 0 to 14 days after hospital admission, and the diagnosis of pandemic (H1N1) 2009 was made 0–28 days after symptom onset. Illness, diagnosis, and treatment of the patients are summarized in the Table. One patient reported contact with a family member who had influenza-like illness. Three other patients likely acquired pandemic (H1N1) 2009 in the hospital. An investigation could not conclusively establish whether transmission of pandemic (H1N1) 2009 occurred between case-patients and healthcare workers or visitors (4). All 4 case-patients ultimately died; 2 patients recovered from pandemic (H1N1) 2009 after antiviral drug therapy but died of underlying disease and subsequent bacterial infections. One case-patient did not receive antiviral drugs because the diagnosis was made posthumously.

We learned valuable lessons regarding diagnosis and management of pandemic (H1N1) 2009 in immunocompromised patients. First, pandemic (H1N1) 2009 infection can be difficult to diagnose in immunocompromised hospitalized patients. Such patients do not exhibit consistent symptoms or signs for pandemic (H1N1) 2009. Consistent with Tramontana et al. (1), fever was the most common feature, followed by progressive dyspnea and intermittent cough. None of our patients reported sore throat. Moreover, such nonspecific symptoms may be inadvertently attributed to concurrent medical problems common in immu-