within SXT\textsuperscript{LAOS}. However, the gene was not found in any of the proposed hot spot regions. The possibility that the trimethoprim resistance determinant is located on the chromosome outside the SXT element and cotransfers with the SXT in an Hfr-like manner cannot be ruled out (9). Therefore, additional hot spot regions may exist in SXT elements for insertion of DNA; otherwise the trimethoprim resistance gene is not encoded within SXT\textsuperscript{LAOS}.

The nucleotide sequence data reported in this study will appear in the DDBJ/EMBL/GenBank nucleotide sequence databases with the accession numbers AB185252 for the hot spot \textit{sO43-traL} and AB186353 for the hot spot \textit{traA-sO54}.

Claudia Toma,* Noboru Nakasone,* Tianyan Song,* and Masaaki Iwanaga*
*University of the Ryukyus, Okinawa, Japan

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Address for correspondence: Claudia Toma, Division of Bacterial Pathogenesis, Department of Microbiology, Graduate School of Medicine, University of the Ryukyus, Nishihara, Okinawa 903-0215, Japan; fax: 81-98-895-1408; email: k950417@med.u-ryukyu.ac.jp

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**Modeling the Impact of Pandemic Influenza on Pacific Islands**

To the Editor: Many Pacific Island countries and areas have been severely impacted in influenza pandemics. The 1918 pandemic killed substantial proportions of the total population: Fiji = 5.2%, Tonga = 4.2% to 8.4%, Guam = 4.5%, Tahiti = 10%, and Western Samoa = 19% to 22% (1,2). Thirty-one influenza pandemics have occurred since the first pandemic in 1580 (3); another one is likely, if not inevitable (4). The potential use of influenza as a bioweapon is an additional concern (5).

The scale of an influenza pandemic may be projected on the basis of the available historical data that have been built into a computer model, e.g., FluAid (6). FluAid uses a deterministic model to estimate the impact range of an influenza pandemic in its first wave. Given the lack of accessible data for specific Pacific Island countries and areas, the default values used in FluAid were used for the proportion of the population in the high-risk category for each age group, for the death rates, hospitalizations, and illness requiring medical consultations. Country-specific population data were obtained from the Secretariat of the Pacific Community, and hospital bed data were obtained from the World Health Organization (WHO) (7,8). The FluAid model was supplemented by a model of an 8-week pandemic wave and modeling of hospital bed capacity. Further methodologic details are provided in the online Appendix (available from http://www.cdc.gov/ncidod/EID/vol11no02/04-0951_app.htm).

The results indicate that at incidence rates of 15% and 35%, pandemic influenza would cause 650 and 1,530 deaths, respectively, giving crude death rates of 22 to 52 per 100,000 (see the Table in the online Appendix). Most deaths (83%) would occur in the high-risk group, 60% of whom would be 19–64 years of age, and 22% would be ≥65 years of age. Additionally, 3,540 to 8,250 persons would be hospitalized, most of whom (78%) would not have high-risk conditions. Also, 241,000 to 563,000 medical consultations would occur. Most (87%) consultations would be for patients without high-risk conditions (50% birth–18 years of age and 46% 19–64 years of age).

In the peak week of the pandemic (week 4), from 15% to 34% of all hospital beds would be required for patients with influenza (Table). The upper end of impact on hospital beds at >40% would occur for Guam,
Kiribati, Marshall Islands, Northern Mariana Islands, and Tonga. Assuming all consultations required doctors, 42 to 99 influenza consultations per doctor would be required during the peak week (Table). The upper end of impact on consultations for individual Pacific Island countries and areas would vary from 31 (New Caledonia) to 524 (Vanuatu); Fiji, Kiribati, Samoa, Solomon Islands, Tonga, and Vanuatu would have rates >150 consultations per week.

The uncertainties associated with pandemic influenza mean that any modeling of its future impact is relatively crude. For example, the new strain may be particularly infectious, virulent, or both. In contrast, the use of international-level public health interventions as recommended by WHO (9) may prevent pandemic influenza from reaching some Pacific Island countries and areas or particularly remote island groups. These issues and other limitations with the model are detailed in the online Appendix.

Nevertheless, if the death rate is in the range suggested by the model, this outcome would make it the worst internal demographic event since the 1918 influenza pandemic for many Pacific Island countries and areas. The lower death rate (albeit for a single wave) is similar to the U.S. rates for the 1957 influenza pandemic (22 per 100,000) and the 1968 influenza pandemic (14 per 100,000) (10). The upper end is considerably lower than for the 1918 pandemic, which suggests that the range indicated is reasonably plausible. Although relatively high, the death toll from pandemic influenza would still be less than the typical annual impact for some Pacific Island countries and areas from other infectious diseases (including malaria and diarrheal diseases) and from such fundamental determinants of health status such as poor sanitation, poor diet, and tobacco use.

The predicted range of hospitalizations attributable to pandemic influenza would likely overwhelm hospital capacity in many of the Pacific Island countries and areas. Rapid response at the onset of the pandemic could ensure efficacious use of hospital beds and resources, e.g., cancel elective procedures and early discharge to community care. Other contingency plans by hospitals could facilitate lower hospital admission rates (e.g., strengthening the primary care response).

Planning and capacity building could be provided by WHO, the Secretariat of the Pacific Community, and donor nations and agencies with support for improving surveillance and other preventive measures for disease control (see the online Appendix for details). A combination of national capacity building with international support will maximize the capacity to respond to the next influenza pandemic as well as other potential communicable disease threats.

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Mycotic Brain Abscess Caused by Opportunistic Reptile Pathogen

To the Editor: A 38-year-old, HIV-seropositive Nigerian man sought treatment with an 8-month history of severe parietal headache, impaired memory, fatigue, paresthesia, and left-sided focal seizures. He had no history of neurologic disorders, including epilepsy. On physical examination, the patient appeared well, alert, and oriented, with slurred speech. Evaluation of the visual fields showed left homonymous hemianopsia. All other neurologic assessments were unremarkable. The patient had a blood pressure of 120/80, a pulse of 88 beats per minute, and a body temperature of 37.3°C. Leukocyte count was 8,600/µL, total lymphocyte count was 1,981/µL, CD4+ cell count was 37.3/µL, HIV-load was 50 copies/mL, and a HIV RNA-load was <16µg/mL (Etest, AB-Biodisk Solna, Sweden). Recovery was complicated by a generalized seizure and severe, acute psychosis associated with rapid refilling of the 2 lesions with mycotic abscess fluid. After re-aspiration, the patient’s psychosis improved gradually, and no further seizures occurred. When last seen 4 months later, the patient was healthy and without neurologic deficits. His CD4+ cell count was 233/µL, HIV-load was <50 copies/mL, and a MRI scan of the brain showed partial regression of the 2 brain lesions (Figure D).

*Chrysosporium* spp. are common soil saprobes, occasionally isolated from human skin. Invasive infection is very rare in humans, and most were observed in immunocompromised patients, manifesting as osteomyelitis (3,4) or diffuse vascular brain invasion (5). Here, we report the first case of brain abscesses by the *Chrysosporium* anamorph of *Nannizzioopsis vriesii* (1,2). High-dose antimicrobial treatment with voriconazole (200 mg twice daily, subsequently reduced to 200 mg daily) was added to the antiretroviral (ritonavir, amprenavir, trizivir), anticonvulsive, and adjunct corticosteroid treatment. The isolate was highly susceptible to voriconazole in vitro (MIC, ≤16µg/mL [Etest, AB-Biodisk Solna, Sweden]). Recovery was complicated by a generalized seizure and severe, acute psychosis associated with rapid refilling of the 2 lesions with mycotic abscess fluid. After re-aspiration, the patient’s psychosis improved gradually, and no further seizures occurred. When last seen 4 months later, the patient was healthy and without neurologic deficits. His CD4+ cell count was 233/µL, HIV-load was <50 copies/mL, and a MRI scan of the brain showed partial regression of the 2 brain lesions (Figure D).

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In our patient, we were unable to determine the portal of entry and the sequence of fungal dissemination; no

References