within SXT<sup>LAOS</sup>. 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<sup>LAOS</sup>.

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 *sO43-traL* and AB186353 for the hot spot *traA-sO54*.

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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  $\approx$ 5.2%, Tonga  $\approx$ 4.2% to 8.4%, Guam  $\approx$ 4.5%, Tahiti  $\approx$ 10%, and Western Samoa  $\approx$ 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  $\geq 65$  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,

## LETTERS

FluAid model for the peak week (at incidence rates [IR] of 15% and 35%)						
	Input data		Hospital bed requirement in the peak week (% of bed capacity)		Consultations per physician in the peak week	
		Physicians			·	
Country/area	Hospital beds (n)	per 10,000 population	15% IR	35% IR	15% IR	35% IR
Melanesia						
Fiji Islands	2,097	3.4	16	38	78	182
New Caledonia	935	20.1	11	27	13	31
Solomon Islands	881	1.3	17	39	209	487
Vanuatu	605	1.2	13	29	224	524
Micronesia						
Federated States of Micronesia	329	5.9	13	29	45	106
Guam	225	11.1	31	73	24	56
Kiribati	140	3.0	24	56	90	209
Marshall Islands	105	4.6	18	42	59	137
Nauru	50	15.7	7	16	17	40
Northern Mariana Islands	82	4.5	43	100	58	135
Palau	90	11.0	11	25	24	56
Polynesia						
American Samoa	140	7.0	17	39	38	89
Cook Islands	128	7.8	5	12	34	79
French Polynesia	1,062	17.5	10	23	15	35
Niue	0	13.0	-*	-*	20	48
Samoa	557	3.4	12	29	79	184
Tokelau	36	13.3	2	4	20	47
Tonga	200	3.5	20	47	76	178
Tuvalu	56	5.9	7	17	45	105
Wallis and Futuna	75	9.2	8	19	29	68
Total	7,793	6.3	15	34	42	99
*The single hospital in Niue was completely destroyed in a cyclone in 2004.						

Table. Predicted impact on health services from the next influenza pandemic using the FluAid model for the peak week (at incidence rates [IR] of 15% and 35%)

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 of the left arm, 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  $102/\mu$ L, and CD4/CD8 ratio was 0.07. HIV RNA-load was <50 copies/mL; all other laboratory parameters were normal. The patient had received antiretroviral therapy (stavudine, lamivudine, nevirapine) for 5 months before admission, but no prophylaxis for opportunistic infections. Magnetic resonance imaging (MRI) of the brain disclosed 2 masses, 3.3 and 4.8 cm in diameter, respectively (Figure A), and signs of chronic sinusitis. A computed tomographic chest scan showed infiltration of both lower segments with multiple, small nodules (Figure B).

Blood cultures were repeatedly negative. A computer-guided needleaspiration of the brain lesions yielded yellow-brown, creamy fluid in which abundant septated fungal hyphae were detected microscopically (Figure C). Cytologic investigation was consistent with a necrotic abscess. The cycloheximide-resistant isolate was strongly keratinolytic and identified as a Chrysosporium anamorph of Nannizziopsis 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 adjuvant 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 HIV-load 233/µL. 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 N. vriesii. This fungus has been associated with fatal mycosis in reptiles (6,7)and cutaneous mycosis in chameleons originating from Africa (2).

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