Vibrio cholerae SXT Element, Laos

To the Editor: The SXT element is a Vibrio cholerae–derived ICE (integrating and conjugative element), which has also been referred to as a conjugative transposon (1) or a conjinst (2). ICEs excise from the chromosomes of their hosts, transfer to a new host through conjugation, and then integrate into the chromosome again. SXT element was originally isolated in 1993 from a V. cholerae O139 clinical isolate (SXT\textsuperscript{MO10}) (1). The ~100-kbp SXT element confers resistance to sulfamethoxazole, trimethoprim, chloramphenicol, and streptomycin (1). Since 1994, V. cholerae isolates from Bangladesh, India, and Mozambique have also contained the SXT element (2–4). In SXT\textsuperscript{MO10}, resistance genes are embedded near the 5′ end, in a ~17.2-kbp composite transposon-like element that interrupts the SXT-encoded \textit{runAB} operon. In contrast, in El Tor O1 V. cholerae strains isolated in India and Bangladesh, the resistance genes are located in SXT\textsuperscript{ET}, which is closely related but not identical to SXT\textsuperscript{MO10} (2). Comparison of 2 related ICEs, SXT of V. cholerae and R391 of \textit{Providencia rettgeri} (5), showed that the conserved backbone apparently contains 3 hot spots for insertions of additional DNA sequences: the first between S043 and traL, the second between traA and S054, and the third between S073 and traF. R391 contains an intact \textit{runAB} operon and a transposon-associated kanamycin resistance gene located ~3.5 kbp from the \textit{runAB} operon (6). Mobile genetic elements such as SXT have a crucial role in spreading antimicrobial drug resistance genes among microbial populations, and our understanding of these genetic elements would help to control the emergence of antimicrobial drug resistance.

We have been monitoring the drug sensitivity pattern in the Lao People’s Democratic Republic (Laos) since 1993, and we have found that \textit{V. cholerae} O1 strains isolated after 1997 were resistant to tetracycline, sulfamethoxazole, trimethoprim, chloramphenicol, and streptomycin (7). Analysis of the genetic determinants encoding antimicrobial drug resistance showed an SXT element (SXT\textsubscript{LAOS}), which is different from the previously reported SXTs (8). SXT\textsubscript{LAOS} contains 2 novel open reading frames (ORFs) in the third hot spot (between S073 and traF). SXT\textsuperscript{ET} contains a class 9 integron in hot spot S073-traF that harbors \textit{dfrA1} as a gene cassette (2). In SXT\textsuperscript{MO10}, the gene encoding trimethoprim resistance (\textit{dfr18}) is encoded in the ~17.2-kbp composite transposon-like element that interrupts the SXT-encoded \textit{runAB} operon. SXT\textsubscript{LAOS} does not encode \textit{dfr18} or \textit{dfrA1}, and the gene encoding trimethoprim resistance has not been identified. In this study, we analyzed hot spot S043-traL and hot spot traA-S054 to better characterize SXT\textsubscript{LAOS}.

Two sets of primers were designed to amplify the hot spot regions. Primer HS1-F, which anneals to S043, was 5′ GGC TAT GCC ACC GGT GGT G 3′; primer HS1-R, which anneals to traL, was 5′ TGC CGA TCA CTA GCC CCA AC 3′; primer HS2-F, which anneals to traA, was 5′ ATG GCT GTC TAC AAC GCG CC 3′; and primer HS2-R, which anneals to S054, was 5′ GGA GAC AGC GCA AGC GCC AG 3′. Polymerase chain reaction (PCR) amplifications on genomic DNA extracted from the \textit{V. cholerae} O1 strain isolated in Laos (strain 00LA1) with primers HS1-F and HS1-R yielded an amplicon of ~1100 bp, which is slightly different from the amplicon obtained with DNA extracted from \textit{V. cholerae} O139, strain MO10 (~1,000 bp). PCR amplification using primers HS2-F and HS2-R gave amplicons of similar size (~2,200 bp) for both strains. The

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

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References


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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,