

sensitive strain P3343110. When tested by long PCR, all 20 *S. Java* isolates produced a 10,041-bp fragment identical to that produced by P3170700.

PCR was used to determine whether the pentaresistant phenotype was due to the presence of the *Salmonella* genomic island 1 (SGI1) as previously described (5). All 20 strains produced amplicons with primers U7-L12 and LJ-R1 for the left junction and primers 104-RJ and 104-D for the right junction. These results indicate that the SGI1 in the strains of *S. Java* was located in the same chromosomal location as previously described for DT 104 ACSSpSuT but lacks the retronphage found to date only in DT104 strains (6).

These findings demonstrated that the ACSSpSuT resistance gene cluster in *S. Java* isolated from patients in the United Kingdom from 2000 to March 2003 appeared to be chromosomally located and was almost indistinguishable from that found in the epidemic clone of DT104 ACSSpSuT. This resistance gene cluster has also been identified in strains of *S. Agona* from poultry in Belgium (6), in a strain of *S. Paratyphi B* from tropical fish in Singapore (7), and a variant cluster in a strain of *S. Albany* from fish food from Thailand (8). It also appears to be present in isolates of *S. Paratyphi B* of R-type ACSSpSuT from cases of human infection in France in 2003 (F. Xavier-Weill, pers. comm.). The strains of *S. Paratyphi B* from Singapore and France were not tested additionally to identify the Java variant.

The strains of *S. Java* with chromosomally integrated ACSSpSuT resistance identified in the United Kingdom are not those associated with poultry in Germany (9) or the Netherlands (10), which have also caused infections in humans in the United Kingdom (1). The latter strains have different phage types, resistant spectrums, pulsed-field profiles, and they possess plasmid-mediated drug-

resistance. However, several *S. Java* infections in the United Kingdom have been associated with tropical fish tanks (L.R. Ward, unpub. data), although the strain has not been isolated from this medium. A substantive increase in multiresistant *S. Java* has also been reported in Australia (D. Lightfoot, pers. comm.). The antibiogram of these isolates are indistinguishable from the isolates of R-type ACSSpSuT made in the United Kingdom.

These results suggest either a common origin of the ACSSpSuT-resistance gene cluster in epidemic multiresistant DT104 and multiresistant *S. Java* or the horizontal transfer of the cluster from DT104 to other *Salmonella* serovars with a worldwide distribution. In either event, the increasing occurrence of the DT104 resistance gene cluster in potentially epidemic serovars other than *S. Typhimurium* DT104 is concerning.

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1990s *Vibrio cholerae* Epidemic, Brazil

To the Editor: We read with interest the letter by Sarkar et al. on new *Vibrio cholerae* phages (1). The description of new *V. cholerae* phages is a welcome tool for epidemiologic

studies of this species. Our main concern about their work is the inaccurate picture that is presented of the cholera epidemic in Brazil. Some of the statements made in the final paragraphs are in disagreement with the official epidemiologic records and the characteristics of the *Vibrio* bacteria that occurred in Brazil during the 1990s epidemic (2).

In 1991, the seventh cholera pandemic reached South America by the Pacific coast, spreading to Brazil in the same year (3). In Brazil, the first cholera cases were reported in the Amazon region bordering Peru; within a few months a large number of cholera cases were recorded in states facing the Atlantic Ocean in the northeastern region (2). According to the official figures of the Brazilian Ministry of Health (2), 168,598 cases of cholera caused by a *V. cholerae* O1 El Tor strain occurred in Brazil from 1991 to 2001. Of these, 155,363 (92.1%) occurred in the northeastern area of the country, with 2,037 deaths. From 2001 to 2003, the number of confirmed cases was 4,756, 734, and 7, respectively.

Sarkar et al. (1) indicate that 60,000 cases occurred from 1991 to 2001 in Rio de Janeiro, a city localized in the southeastern region; the official records report only 349 cases. The statement that "since 1993, no cholera cases caused by O1 have been reported" is also perplexing. From 1994 to 2001, the official records report 68,583 cases of cholera in Brazil (51,324 of these in 1994, the second major year of cholera incidence). Are the authors suggesting that this number of cases was caused by non-O1 *V. cholerae*? The official records state that the cholera epidemic in Brazil was caused by an El Tor O1 strain (4,5).

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Fluoroquinolone-resistant *Salmonella* Paratyphi A

To the Editor: Fluoroquinolones have been the drug of choice for treating typhoid and paratyphoid fever since the beginning of the 1990s. Multidrug-resistant strains began to prevail in disease-endemic areas, and former first-line antimicrobial drugs, such as chloramphenicol, were sometimes ineffective (1). In recent years,

however, strains with decreased susceptibility to quinolones have emerged, and clinical treatment failure is a serious concern (2–5).

An 87-year-old woman was referred from a local clinic to Yokohama Municipal Citizen's Hospital in July 2002 because *Salmonella enterica* serovar Paratyphi A was detected in her urine. She had no subjective symptoms such as pain on urination or urinary urgency, and her temperature was normal. She had never had paratyphoid fever, and she had not traveled abroad. No other person in the community had paratyphoid. Before being admitted to the hospital, she had experienced frequent episodes of urinary tract infection and had been empirically treated each time with oral antimicrobial drugs, including ciprofloxacin. She had been given a dose of 600 mg/day for 7 days, 25 times in the last 4 years.

The patient did not display any abnormal findings on physical examination. *S. Paratyphi A* was not detected in the urine but was confirmed in the stool; therefore, the previous report of bacteriuria could have been due to contamination of a urine sample with feces. An ultrasound showed a polyp and multiple stones in her gallbladder. A carrier state was suspected. Bile was obtained by duodenal aspiration and was positive for *S. Paratyphi A*. The patient was considered to be an asymptomatic cholecystic carrier of *S. Paratyphi A*.

On disk diffusion susceptibility testing, the isolate was resistant to nalidixic acid (NA) and to ofloxacin. The MIC of ofloxacin was as high as 256 µg/mL, and the MIC of ciprofloxacin was 128 µg/mL (Table). An open cholecystectomy was performed for treatment of the polyp, the stones, and the highly quinolone-resistant bacteria. A routine perioperative intravenous antimicrobial agent, cefmetazole, was administered as surgical prophylaxis. The polyp was malignant, and the operation was