Multidrug-resistant Salmonella Java

To the Editor: Since 2000, Salmonella enterica serovar Paratyphi B variant Java (S. Java) with resistance to antimicrobial drugs has been isolated with increasing frequency from patients in Scotland, England, Wales, and the Channel Islands. For England, Wales, and the Channel Islands, drug-resistant S. Java was found in humans: 25 in 2000, 36 in 2001, 49 in 2002, and 4 in 2003 (January 1–March 31). These isolates made up 35% of 325 strains of S. enterica isolated from patients in London, New South Wales, and the Channel Islands. For England and Wales, the Channel Islands, drug-resistant S. Java of R-type ACSSpSuT was isolated from human patients in 64 instances from 2000 to 2003 (5 in 2000, 22 in 2001, 34 in 2002, and 3 in 2003 [to March 31]). None of these cases were related to eating contaminated foods, and the ACSSpSuT antibiogram has not been isolated from strains of this serotype from foods in the United Kingdom. This resistance pattern corresponds to that of the epidemic clone of S. Typhimurium definitive phage type (DT) 104 (DT 104 ACSSpSuT), which caused many infections in humans and food production animals throughout Europe, the United States, and Canada in the 1990s (1). In all isolates of DT104 ACSSpSuT studied, from many different countries, the resistant gene cluster has been chromosomally integrated (1). Resistances have been contained in a 13-kb cluster composed of 2 integrons coding for resistance to SSp (1.0 kb) and ASu (1.2 kb), with the genes for resistance to chloramphenicol and tetracyclines located between these integrons (2,3). To investigate the possibility of the horizontal transfer of the ACSSpSuT gene cluster within S. enterica, we have characterized the resistance genes and associated structures in strains of S. Java of R-type ACSSpSuT and compared them with those in a strain of DT104 ACSSpSuT. From 2000 to 2002, a total of 20 isolates of S. Java of R-type ACSSpSuT from patients in England and Wales (18 isolates) and Scotland (2 isolates) were characterized by phage typing, plasmid profile typing, and pulsed-field gel electrophoresis (PFGE). Pulsed-field profiles of 3 closely-related pulsed-field types, – 1 var 3 (6 isolates), 3b var 2 (5), Dundee (2), Worksop (3), and RDNC (4), and 3 closely-related pulsed-field types, differing by only 1 to 3 of 14 bands in the XbaI PFGE profiles, were identified in the 20 isolates studied; 2 of these pulsed-field types were observed in the electronically transmitted images of the 3 isolates from Scotland. These pulsed-field profile types have been designated SPTJXB001 through SPTJXB003. Of these, SPTJXB002 predominated, being present in 11 of the isolates studied, belonging to 3 phage types. SPTJXB001 was identified in 8 isolates of 3 phage types, and SPTJXB003 in the remaining isolate. PFGE type did not change over time. All isolates were plasmid-free, and resistances were not transferable, either directly or by mobilization after a self-conjugative plasmid was introduced into the strains. By PCR, all isolates possessed blaTEM, cmlA, aadA2, sulI, and tetG but were negative for blaCARB-2, catI, and catIII. These results corresponded to those of the control DT104 ACSSpSuT strain P3170700. When tested for class 1 integrons, all S. Java isolates of R-type ACSSpSuT produced characteristic amplicons of 1.0 and 1.2 kb, as did P3170700, but not the drug-
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 retropathage 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. Paratyphi B from tropical fish in Singapore (7), and a variant cluster in a strain of S. Albany from fish food in France in 2003 (F. Xavier-Weill, pers. comm.). The antimicrobial profiles of these isolates are indistinguishable from the isolates of R-type ACSSpSuTT 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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References


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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