products that were identical to those of N315 or MW2. These findings call into question the accuracy of the published Mu50 genome and its use as a comparator in vancomycin resistance studies.

Although these four disrupted genes could be responsible, at least in part, for the vancomycin-intermediate resistant phenotype of Mu50, as suggested by Avison et al., these changes are not essential for the vancomycin-resistant phenotype because the genes were not disrupted in any of the other clinical VISA and heteroVISA investigated. Furthermore, in the clinical VISA and heteroVISA, the sequences of all the genes studied, except the mrp-homologue, were predicted to encode the same products as those in the VSSA strains N315 and MW2, indicating that vancomycin resistance cannot be attributed to loss of these functions. In five of the VISA and six of the heteroVISA tested, the predicted products of the mrp-homologue sequences differed from those produced by N315, MW2 and any clinical VSSA tested by the same four amino acid substitutions (T142A, N147T, D172N and A211V); the remaining VISA and heteroVISA strains produced an Mrp product identical to N315. These findings suggest some degree of clonality between VISA/ heteroVISA isolates.

Mu50 and other clinical VISA and heteroVISA strains from around the world share common phenotypic characteristics (e.g. thickened cell walls and reduced cross linking of glycan chains).6 However, the disrupted genes identified in Mu50 and Mu3 appear to be functional in many VISA, indicating that the VISA phenotype can be effected by various means. A study of the expression of genes involved in the biosynthesis and turnover of peptidoglycan in these isolates would be expected to provide a better understanding of their vancomycin resistance.

Nucleotide sequence accession numbers


Acknowledgements

We are grateful to all suppliers of VISA and heteroVISA strains. This work was kindly funded by a grant from the British Society for Antimicrobial Chemotherapy (BSAC).

References


60. Sir, Non-typhoidal salmonellae are one of the principal pathogens implicated in cases of food poisoning worldwide. In the UK, Europe and USA, Salmonella enterica serovar Enteritidis (S. Enteritidis) is one of the most commonly isolated serotypes, and is thought to be spread to humans through the food chain from reservoirs in food-producing animals.

Antibiotic resistance is relatively uncommon in S. Enteritidis. During 1996–2000, the overall incidence of multidrug resistance (resistance to four or more antibiotics) in this serovar was less than 1% in England and Wales. Although antibiotics are rarely required in cases of salmonella enterocolitis, they are crucial if the infection spreads from the intestine. In the treatment of extra-intestinal salmonella infections, the antibiotics of choice are extended-spectrum cephalosporins and fluoroquinolones. Recently, Salmonella isolates harbouring extended-spectrum β-lactamases (ESBLs) capable of hydrolysing third-generation cephalosporins have been reported. This is of particular concern for the treatment of salmonellosis in children, because fluoroquinolones cannot be used in this age group. Here we report the presence of the ESBL TEM-52 in an S. Enteritidis strain isolated during a hospital outbreak in Scotland. Previously, TEM-52 has only been reported in salmonellae isolated in Hungary and Korea. An outbreak of salmonellosis was identified in a general hospital in Glasgow, Scotland, in the period 20 December 2001–21 January 2002. During this outbreak, nine isolates were obtained from five patients with salmonella gastroenteritis, and two asymptomatic members of staff. Isolates were identified as S. enterica serovar...
Enteritidis PT21 by agglutination to polyclonal antiserum and phase typing. Isolates from two patients and one member of staff were susceptible to 14 common antibiotics. Isolates from the remaining patients and one member of staff were resistant to ampicillin and cefotaxime, and carried a plasmid of 95 kb. Plasmid profiling and PFGE revealed these isolates to be clonal, one of which (020003) was used to determine the mechanism of resistance.

Susceptibility testing to ampicillin, cefaclor, cefotaxime and ceftazidime was performed using standard methods, and plasmid transfers carried out in broth-mating experiments with Escherichia coli K12 J53-2 (lactose positive, rifampicin resistant). The β-lactamase genes blaTEM, 5 and blaSHV, 5 were sought by PCR using a Taq polymerase kit (Promega) and the following oligonucleotide primer pairs: blaTEM, 5′-ATGAGTTATCCAACATTTCCG-3′, 5′-CCATGCTT-TATTGCATGGG-3′; and blaSHV, 5′-GCCCGGGTTATTCTTATT-TGTCGC-3′, 5′-CTTTCCGATGCCGCCCGCATCA-3′. Sequencing was performed in forward and reverse directions, and the sequences were compared with sequences in public databases.

The salmonella isolates were resistant to ampicillin, cefaclor (both >128 mg/L), cefotaxime (8 mg/L) and ceftazidime (>32 mg/L). All these resistances were carried on the 95 kb conjugative plasmid. PCR with blaSHV, 5 primers failed to detect a gene encoding an SHV-1 derivative. The blaTEM, 5 PCR yielded an 858 bp product. DNA sequencing showed that this gene encoded TEM-52, 3,4 which differs from TEM-15 by amino acid substitutions at positions Glu-104 and Gly-238. Enteritidis 020003) Met (ATG) Lys (AAG) Ala (GCG) Thr (ACG) Ser (AGT) 4

This work was presented at the Forty-third Interscience Conference on Antimicrobial Agents and Chemotherapy, Chicago, IL, USA, September 2003, and was supported by the International Partnership Research Award in Veterinary Epidemiology (IPRAVE), funded by the Wellcome Trust (grant number GR068596/A/02/Z).

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