Sequence Type ST405 *Escherichia coli* Isolate Producing QepA1, CTX-M-15, and RmtB from Detroit, Michigan

Plasmid-mediated fluoroquinolone resistance determinants are increasingly identified worldwide among various isolates of *Enterobacteriaceae* (9). Among them, QepA belongs to the major facilitator superfamily (MFS)-type group of efflux pumps. It was initially identified in *Escherichia coli* clinical isolates from Belgium and Japan (8, 13). QepA (now QepA1) has subsequently been detected in *E. coli* isolates of human (1, 5, 10) and animal (6, 7) origins. QepA2, a two-amino-acid variant of QepA1, has been reported from an *E. coli* clinical isolate in France (2).

Here we report identification of QepA1 in an *E. coli* isolate from the United States. *E. coli* M3006 was isolated from the urine of a 47-year-old female patient with paraplegia, who was admitted from a nursing home to a hospital in Detroit, MI, for treatment of sacral osteomyelitis in January 2010. She had received ciprofloxacin for 10 days just prior to her presentation. The isolate was highly resistant to ceftazidime, cefoperazone, cefepime, ciprofloxacin, and amikacin, all with MICs of >256 μg/ml, and susceptible to all four carbapenems (ertapenem, imipenem, meropenem, and doripenem).

PCR analysis was performed to identify various resistance genes, including β-lactamase genes *bla*<sub>TEM</sub>, *bla*<sub>SHV</sub>, and *bla*<sub>CTX-M-15</sub>; 16S rRNA methylase genes *armA*, *rmfB*, and *rmfC*; pentapeptide repeat protein genes *qnrA*, *qnrB*, *qnrC*, and *qnrS*; the fluoroquinolone-modifying aminoglycoside acetyltransferase gene *aac(6′)-Ib-cr*; and the plasmid-mediated fluoroquinolone efflux pump gene *qepA* (11). PCR products were sequenced on both strands. As a result, *bla*<sub>CTX-M-15</sub>, *bla*<sub>TEM-1</sub>, *qepA1*, and *rmfB* were detected in *E. coli* M3006.

A cefotaxime-resistant transconjugant and a transformant containing *bla*<sub>CTX-M-15</sub> were successfully obtained from *E. coli* M3006 using *E. coli* J53 and DH10B as recipients, respectively. Both of them were positive for *bla*<sub>CTX-M-15</sub> as well as *bla*<sub>TEM-1</sub>, *qepA1*, and *rmfB*, indicating that these four genes were located on the same conjugative plasmid. The MIC of ciprofloxacin for the *E. coli* J53 transconjugant was 0.125 μg/ml, which represented an 8-fold increase over that of the recipient alone. The level of ciprofloxacin resistance was much higher for the parental strain, which was likely due to mutations in the quinolone resistance-determining regions of the *gyrA* and *parC* genes. As expected, this transconjugant was also resistant to cefoxime (MICs, 256 μg/ml) and cefepime (MICs, >256 μg/ml for gentamicin and amikacin).

*E. coli* M3006 was then subjected to phylogenetic typing and multilocus sequence typing (MLST) (3, 12). As a result, *E. coli* M3006 was classified into phylogenetic group D, with its allele combination corresponding to the sequence type (ST) 405. Phylogenetic group D-CTX-M-15 strains, along with phylogenetic group B2-ST131 strains, have been implicated as vehicles driving the international spread of *bla*<sub>CTX-M-15</sub> (4).

To the best of our knowledge, this is the first identification of a QepA-type efflux pump in the United States. The location of the multidrug-resistant, conjugative plasmid carrying *qepA1*, *bla*<sub>CTX-M-15</sub>, and *rmfB* on a globally disseminated *E. coli* clone is a troubling phenomenon, as its presence simultaneously compromises the efficacy of fluoroquinolones, cephalosporins, and aminoglycosides that are commonly used in treatment of *E. coli* infections.

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