CTX-M-15 Extended-Spectrum β-Lactamase in a Shiga Toxin-Producing *Escherichia coli* Isolate of Serotype O111:H8

Charlotte Valat,a Marisa Haenni,a Estelle Saras,a Frédéric Auvray,b Karine Forest,a Eric Oswald,c,d,e and Jean-Yves Madeca

Unité Antibiorésistance et Virulence Bactériennes, ANSES Site de Lyon, Lyon, France; Unité Ecophysiologie et Détection Bactérienne, ANSES Laboratoire de Sécurité des Aliments de Maisons-Alfort, Maisons-Alfort, France; Inserm, Toulouse, France; INRA, Toulouse, France; and CHU Toulouse, Hôpital Purpan, Service de Bactériologie-Hygiène, Toulouse, France

We report the discovery of a CTX-M-15-producing *Escherichia coli* (STEC) of serogroup O111:H8, a major serotype responsible for human enterohemorrhagic *Escherichia coli* (EHEC) infections. In line with the recent CTX-M-15/O104:H4 *E. coli* outbreak, these data may reflect an accelerating spread of resistance to expanded-spectrum cephalosporins within the *E. coli* population, including STEC isolates.

Shiga toxin-producing *Escherichia coli* (STEC) comprises food-borne pathogens producing Stx1 and/or Stx2 (1). O157:H7 is the main serotype responsible for human infections, but O26: H11, O103:H2, O111:H8, and O145:H28 are also frequently incriminated (8). Ruminants are a major source of STEC, and transmission principally occurs through consumption of contaminated food but also through direct or indirect contacts with contaminated animals or persons (4).

Several studies reported on the antimicrobial resistance of STEC, but these pathogens have not been considered so far as a reservoir of extended-spectrum β-lactamases (ESBLs), one of the most widespread mechanisms of transmissible antimicrobial resistance in Gram-negative bacteria. ESBLs confer resistance to all β-lactams but cefotaxim and carbapenems and mostly belong to the TEM, SHV, and CTX-M families, with the last group demonstrating an epidemiological success in recent years (3). To our best knowledge, only five ESBL-producing STEC isolates have been reported so far, including three human isolates belonging to serogroup O26 and carrying either a blaCTX-M-3 (10), blaCTX-M-18 (14), or blaTEM-52 gene (2), one chicken isolate belonging to serogroup O157 and carrying a blaCTX-M-2 gene (23), and the highly virulent O104:H4 isolate harboring the blaCTX-M-15 gene and responsible for the recent outbreak in Germany and France (19).

In this study, ESBL production was detected in an *E. coli* isolate, 22207, recovered in 2008 through the National Network for the Surveillance of Resistance to Antimicrobials in Animals in France (Résapath; www.resapath.anses.fr) from the fecal contents of a calf found in plasmid pO157, i.e., espP (16), e-hlyA (21), and msbB2 (12) encoding a serine protease, the EHEC hemolysin, and an acetyltransferase involved in lipid A biosynthesis, respectively, were also detected. The espP, e-hlyA, and msbB2 genes were not

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Address correspondence to C. Valat, charlotte.valat@anses.fr

C. Valat and M. Haenni contributed equally to the work.

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located on the bla<sub>CTX-M-15</sub>-carrying plasmid, as they were not detected in the transconjugant.

Here we showed the presence of a bla<sub>CTX-M-15</sub>-carrying plasmid in an <i>E. coli</i> of serotype O111:H8 carrying the <i>stx</i><sub>1</sub> and <i>eae</i> genes. This strain is likely to be the cause of the severe diarrhea that occurred before the death of the calf. More importantly, this strain shows the worrying combination of a major determinant of β-lactam resistance in humans with virulence genes typical of one of the major serotypes of STEC responsible for hemorrhagic colitis and hemolytic and uremic syndrome in human beings. In line with the recent CTX-M-15/O104:H4 <i>E. coli</i> outbreak, the emergence of such strains is of great concern. However, in this study, the bla<sub>CTX-M-15</sub> gene was located on a different plasmid with an incompatibility group other than that of the O104:H4 plasmid, suggesting that the two strains did not result from the dissemination of the same plasmid. On the other hand, CTX-M-15 enzymes are recurrently found in <i>E. coli</i> from cattle (13, 20), which are also a major reservoir of STEC. Consequently, CTX-M-15-producing STEC from cattle may expand in the future, and this may indicate a more widespread movement of bla<sub>CTX-M</sub> genes within the <i>E. coli</i> population, including STEC isolates.

Finally, the presence of a bla<sub>CTX-M-15</sub>-carrying plasmid in a Shiga toxin-producing <i>E. coli</i> of serotype O111:H8 points out again the relationship between virulence genotypes, phylogenetic backgrounds, and resistance traits in pathogenic <i>E. coli</i>. It would be valuable to better understand which selective pressures may promote such combinations of virulent and highly resistant pathogens. Current rapid genetic technologies such as those used in this study should facilitate screening of these hazardous strains and help to study their occurrence or emergence in animals.

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