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Virulence study in mice of O26:H11 *Escherichia coli* strains that encode stx2d and intimin

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Background: Shiga toxin (Stx)-producing *E. coli* strains (STEC) are the underlying cause of diarrhoea-associated disease outbreaks, with the potential for the life-threatening complication of hemolytic uremic syndrome (HUS). There are two types of Stx, Stx type 1 (Stx1) and Stx type 2 (Stx2) that are typically encoded within inducible lysogenic bacteriophages. One of the most common *E. coli* serotypes associated with STEC infection after O157:H7 is O26:H11. A recent report on several HUS-associated O26:H11 isolates from France found that some of those strains encoded an intestinal mucus-activated Stx2 variant called Stx2d and the adhesin intimin. In this study we evaluated the virulence in mice of six of those O26:H11 clinical isolates. Methods: Serogroup and toxin subtyping was confirmed by PCR, toxin gene copy number was determined by Southern blot. We assessed Stx2d activity on Vero cells. The activation capacity or inducibility of the toxin produced by the strains was measured on Vero cells after incubation with mouse intestinal mucus at a concentration of 1mg/ml, or treatment of overnight cultures with 5-15 ng/mL ciprofloxacin (Cip). In vivo studies were performed in streptomycin (Str)- or Str- and Cip-treated male BALB/c mice. Results: In vitro all strains showed similar cytotoxicity and activation parameters. However, the capacity of the toxin to be induced by Cip was different: some strains did not appear to be inducible, while others were at least 10-fold more cytotoxic after Cip-treatment. Although each strain was isolated from a patient with HUS, we observed significant differences in mouse virulence: some strains caused slight weight loss or moderate lethality only in Str- and Cip-treated mice, whereas one strain exhibited 80% mortality with or without Cip treatment. Conclusion: Our study highlights the difference in the mouse pathogenicity of six O26:H11 STEC strains that make Stx2d. We hypothesize that the virulence differences among the strains might be associated with variable induction by Cip and in vivo toxin levels. The fact that some of these strains only caused mortality when induced by Cip underscores the importance of proper primary treatment of any diarrhoea-associated disease, where the possibility of an STEC infection arises.

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