CORRESPONDENCE

Verocytotoxigenic *Escherichia coli* Serologic Responses in Patients with Hemolytic Uremic Syndrome

To the Editor—We read with interest the recent paper by Jelacic et al. [1], in which they unsuccessfully sought a link between ABO and P1 blood groups and outcomes of childhood infections by *Escherichia coli* O157. We would like to draw readers' attention to our observations during an outbreak of hemolytic-uremic syndrome (HUS), in which we compared the numbers of seroreactivities to potential enterohemorrhagic *E. coli* (EHEC)–related *E. coli* O antigens versus the total complications and the mean total complication score as determined clinically by an independent observer.

Our results clearly show that, as the number of complications increased or as the complication score increased, all mean seroreactivities also increased [2, 3]. This strongly supports the conclusion that multiple infections with a variety of verocytotoxigenic *E. coli* (VTEC) contributed to the outbreak. It also appears that the larger the variety of infecting VTEC, the greater the possibility of complication.

We do not doubt the significance of EHEC O157 as an important cause of HUS and other human infections; however, until such infections are thoroughly investigated—including testing for the presence of non-O157 VTEC, which are known to be present on meat derived from ruminant animals [4–6]—false interpretations are likely.

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Reply

To the Editor—We thank Goldwater and Bettelheim [1] for raising the possibility that some of the patients we reported [2] from whom *E. coli* O157:H7 was isolated were also infected with Shiga toxin–producing *Escherichia coli* (STEC) other than *E. coli* O157:H7. However, two lines of evidence suggest that the dual presence of O157:H7 and non-O157:H7 STEC is uncommon in the population we studied.

First, we recently completed a study in the Seattle Children's Hospital and Regional Medical Center (CHRMC) Emergency Department, focusing on all STEC and not just on *E. coli* O157:H7 [3]. We analyzed 5 sorbitol-fermenting colonies from the sorbitol-MacConkey agar plates of 22 of the 28 children infected with *E. coli* O157:H7 who presented to this facility during a 3-year period (stool cultures from the remaining 6 patients lacked sorbitol-fermenting colonies). None of the sorbitol-fermenting coisolated coliforms possessed *stx* genes. Eleven of these 28 patients were also studied in our report [1]. Thus, if non-O157:H7 STEC, which usually ferments sorbitol, infected the patients whose stools contained sorbitol-nonfermenting *E. coli* O157:H7, these organisms would comprise <20% of the aerobic non-O157:H7 gram-negative flora

Second, in a 1991 study conducted in the CHRMC Microbiology Laboratory, 5 lactose-fermenting colonies were selected and probed for the presence of *stx* genes, without their serotype being known [4, 5]. In no case was a mixed STEC infection identified. Thus, the use of a selection protocol that does not bias toward the recovery of *E. coli* O157:H7 (non-O157:H7 STEC and *E. coli* O157:H7 almost always ferment lactose) did not identify coinfections.

We agree that a subset of non-O157:H7 STEC are human pathogens; they are underdetected by present screening techniques, which usually rely on sorbitol-MacConkey agar. However, our data suggest that, if non-O157:H7 STEC are also excreted by children in the Pacific Northwest who are infected with *E. coli* O157:H7, then these organisms are considerably less frequent among the coliform flora than are the *E. coli* O157:H7 identified on the sorbitol-MacConkey agar plate.

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