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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References

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 ferment 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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The Journal of Infectious Diseases 2002;186:582-3
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0022-1899/2002/18604-0023$15.00