Outbreak of Shiga Toxin–Producing *Escherichia coli* O111:H8 Infections among Attendees of a High School Cheerleading Camp

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Few US clinical laboratories screen stool specimens for Shiga toxin–producing *Escherichia coli* (STEC) other than *E. coli* O157. An outbreak of STEC O111:H8 infections indistinguishable from *E. coli* O157:H7 at a youth camp highlights the need to improve non-O157 STEC surveillance. Interviews of 521 (80%) of 650 attendees revealed 55 (11%) were ill; 2 developed hemolytic-uremic syndrome. Illness was associated with consuming salad during the camp’s first lunch meal (hazard ratio [HR], 4.68; *P* < .01), consuming ice provided in barrels on the camp’s final day (HR, 3.41; *P* < .01), eating cob corn (HR, 3.22; *P* < .01), and eating a dinner roll (HR, 2.82; *P* < .01). Cultures of 2 of 11 stools yielded *E. coli* O111:H8. Results of serologic testing and additional stool cultures demonstrated no evidence of infection with other bacterial pathogens, including *E. coli* O157, and supported infection with *E. coli* O111. Clinical laboratories should routinely screen suspect specimens for non-O157 STEC and should serotype and report Shiga-positive isolates.

Shiga toxin–producing *Escherichia coli* (STEC) are diarrheagenic bacteria termed “enterohemorrhagic *E. coli*” when pathogenic to humans. Infection may be asymptomatic. Illness ranges from mild diarrhea to more profound watery or bloody diarrhea with severe abdominal cramping and can result in hemolytic uremic syndrome (HUS) [1, 2]. In the United States, STEC cause an estimated 110,000 illnesses and 90 deaths annually [3]. *E. coli* O157:H7 is the STEC most commonly associated with human illness in North America [4, 5]. However, non-O157 STEC have been isolated with a frequency similar to that of *E. coli* O157:H7 from diarrheal stool specimens [6–17] and can also cause HUS [18–20]. Outbreaks may go unrecognized because the diagnostic methods routinely used in most US clinical laboratories will not differentiate non-O157 STEC from harmless *E. coli*. How infections are transmitted is not well characterized, and the true burden of disease (e.g., incidence rate and economic cost) attributable to non-O157 STEC is unknown.

In mid-June 1999, the Texas Department of Health learned that many attendees of a cheerleading camp held 9–11 June had developed gastroenteritis, including bloody diarrhea. Two persons were hospitalized with HUS. Routine stool cultures failed to yield a pathogen, including *E. coli* O157. However, reevaluation of stool specimens identified STEC *E. coli* O111:H8.

**MATERIALS AND METHODS**

**Epidemiologic investigation.** To enhance case find-
ing, we alerted all health departments, hospitals, clinical laboratories, and physicians in Texas about the outbreak. We screened other groups that had used the same facilities shortly before or after the cheerleading camp for possible STEC-related illness. A 20% random sample of persons in each group was selected from registration records and interviewed by telephone about recent diarrheal illness. No other group used the same facilities during the cheerleading camp.

To identify the source of the outbreak and to describe the clinical illness, we conducted a cohort study of the camp’s 650 attendees (which consisted of adolescents and their adult chaperones). Interviews occurred during 14–21 July 1999. The cohort was defined by means of registration records. We excluded persons aged ≤11 years (<5% of all camp attendees) to reduce recall bias. No contact information was available for the camp’s counselors, and successive attempts to locate them were unsuccessful.

We defined illness as either bloody diarrhea (i.e., ≥3 bowel movements in any 24-h period) or diarrhea accompanied by nonmenstrual abdominal cramps occurring ≤14 days after the start of camp. The questionnaire (derived from hypothesis-generating interviews and review of menus) asked about all foods and beverages consumed during attendance at the camp, preexisting medical conditions, medical treatment received, diarrheal illnesses occurring before attendance at the camp, contact with ill persons, travel, and animal and recreational water exposures. We administered >95% of questionnaires by telephone and the remainder in person. We made up to 3 attempts to contact each person. To reduce recall bias, trained interviewers conducted standardized, scripted interviews, and press releases before disclosure of the study’s results did not discuss suspected modes of transmission.

Data were collected and analyzed as part of an outbreak investigation. Outbreak investigations represent an urgent response to a public health emergency and do not require institutional review board approval. Their conduct is part of the routine duties of the Foodborne and Diarrheal Diseases Branch of the Centers for Disease Control and Prevention and US Department of Health and Human Services in conjunction with state and local health departments.

**Environmental investigation.** We inspected the camp’s cafeteria, interviewed food handlers, and reviewed food service records. With engineering and custodial staff, we inspected all other facilities used by the cheerleading camp (e.g., the gymnasium and dormitory), examined maintenance records, and reviewed plumbing diagrams.

**Microbiologic investigation.** Stool specimens obtained from camp attendees were initially evaluated at local clinical laboratories; all were screened for *Salmonella*, *Shigella*, and *Campylobacter* species and *E. coli* O157. Remaining stool specimens were forwarded to the Texas Department of Health laboratory for non-O157 STEC testing. Stools were streaked onto sorbitol-MacConkey agar (SMAC) plates and inoculated into enrichment broths, and these media were incubated for 24 h. Colony sweeps from the plates and broth supernatants were tested for the presence of Shiga toxin by EIA with a commercial kit (Meridian Diagnostics). Shiga toxin–positive specimens were subcultured, and toxin-producing *E. coli* colonies were identified by the same EIA. *E. coli* isolates from these colonies were serotyped for O and H antigens. By use of PCR, we screened isolates for the gene sequences of Shiga toxins 1 and 2 (*stx1* and *stx2*) [21], intimin (*eae*) [22], enterohemolysin (*Ehly*) [23], and *E. coli* O157:H7–specific β-glucuronidase (*uidA*) [24]. Stool specimens collected from 20 asymptomatic cafeteria staff 4 weeks after the outbreak were evaluated for STEC by these same methods. No foods consumed during the outbreak were available for microbiological testing.

**Serologic investigation.** Thirty-eight days after the first illness occurred, we collected serum specimens from a convenience sample of 58 volunteer camp attendees from 2 high schools with high attack rates (>20% for both). We tested serum samples by ELISA for IgM and IgG antibodies to *E. coli* O157 lipopolysaccharide (LPS), as described elsewhere [25], and to *E. coli* O111 LPS with the same procedure. The threshold titer for a “borderline positive” result was 1:160 and for “definitely positive” result was 1:320 for both IgM and IgG, as established by published studies [26], the experience of the US Centers for Disease Control and Prevention (CDC) laboratory, and the results from control specimens used for these assays.

**Statistical methods.** We investigated associations between food exposures using χ² analysis and Fisher’s exact test (Epi Info software, version 6.04c; CDC). We created composite variables (e.g., “ate any salad”) for successive exposures at multiple meals. We considered associations with 2-tailed *P* values of ≤.05 to be significant. We constructed a multivariable logistic regression model incorporating those events significant in univariate analysis to which >25% of case patients had been exposed, and we applied forward-selection and backward-elimination model-building strategies with a 5% retention and insertion rule to identify independently significant associations (SAS, version 9.0; SAS Institute).

We also used time-varying hazard analysis to determine whether risks changed during the 3 days of the camp. Exposures were introduced in the order in which they occurred, and ill persons were removed from the pool of susceptible individuals as they became ill. We could not include composite variables, which lacked discrete times of occurrence. We applied the same model-building strategies as were used in our logistic regression analysis.

We compared distributions of antibody concentrations by the Wilcoxon rank-sum test and considered differences with 2-tailed *P* values of ≤.05 to be significant. We examined the
association of illness with a positive serology result by Fisher’s exact test using SAS, version 9.0.

RESULTS

Epidemiologic investigation. We interviewed 521 (80%; 489 adolescents and 32 adults) of the 650 camp attendees located throughout Texas; 97% were girls, and the median age was 16 years (range, 12–53 years). One hundred thirty-one persons (25%) reported having at least 1 symptom; of these, 55 (11%) met the case definition for illness (table 1); ill persons did not differ significantly from other attendees according to age, sex, or conditions that can modify risk for bacterial enteric infections (e.g., immunomodulating therapy and gastric hypochlorhydria). Illnesses began during the first day of camp and peaked on the third (i.e., final) day of the camp (figure 1). Two women, aged 16 and 19 years, developed HUS and required dialysis. There were no deaths.

Univariate analysis revealed that 13 of 202 exposures were statistically significantly associated with illness and accounted for ≥25% of illnesses. Multivariable analysis revealed that 2 exposures remained independently associated with illness: having consumed any ice from 50-gallon plastic trash cans lined with disposable garbage bags that were provided twice daily in the cafeteria lobby for filling water bottles during workouts (adjusted OR [AOR], 3.58; 95% CI, 1.88–6.79; P < .01), and having eaten salad from the cafeteria salad bar on at least 1 occasion (AOR, 3.20; 95% CI, 1.11–9.16; P = .03) (table 2).

Univariate time-varying hazard analysis identified 20 of 211 exposures that were statistically associated with illness and accounted for ≥25% of illnesses; 8 had also been identified by χ² analysis. In the multivariable model, 4 exposures remained independently and significantly associated with illness: having eaten a salad from the cafeteria salad bar at lunch on day 1 of the camp (hazard ratio [HR], 4.68; 95% CI, 1.42–15.4; P = .01), having eaten cob corn at dinner on day 1 (HR, 3.20; 95% CI, 1.11–9.16; P = .03),

Table 1. Clinical characteristics of ill persons involved in an outbreak of Escherichia coli O111:H8 infections in Texas, June 1999.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Persons who met the strict case definitionᵃ</th>
<th>Persons who reported ≥1 symptom but did not meet the strict case definitionᵇ</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, median years (range)</td>
<td>16 (12–53)</td>
<td>16 (12–40)</td>
</tr>
<tr>
<td>Female sex, %</td>
<td>97.1</td>
<td>96.1</td>
</tr>
<tr>
<td>Abdominal cramps</td>
<td>55/55 (100)</td>
<td>46/74 (62)</td>
</tr>
<tr>
<td>Any diarrhea</td>
<td>55/55 (100)</td>
<td>33/76 (43)</td>
</tr>
<tr>
<td>Nausea</td>
<td>35/55 (64)</td>
<td>45/76 (59)</td>
</tr>
<tr>
<td>Headache</td>
<td>30/51 (59)</td>
<td>34/74 (46)</td>
</tr>
<tr>
<td>Chills</td>
<td>25/53 (47)</td>
<td>17/75 (23)</td>
</tr>
<tr>
<td>Vomiting</td>
<td>21/55 (38)</td>
<td>21/75 (28)</td>
</tr>
<tr>
<td>Bloody diarrhea</td>
<td>17/51 (33)</td>
<td>3/76 (4)</td>
</tr>
<tr>
<td>Fever</td>
<td>15/53 (28)</td>
<td>12/75 (16)</td>
</tr>
<tr>
<td>Temperature, median °F/°C (no. of observations)</td>
<td>100/37.8 (8)</td>
<td>99.9/38.0 (4)</td>
</tr>
<tr>
<td>Hemolytic uremic syndrome</td>
<td>2/55 (4)</td>
<td>0/76 (0)</td>
</tr>
<tr>
<td>Maximum no. of bowel movements, median (range)d</td>
<td>5 (3–30)</td>
<td>2 (0–14)</td>
</tr>
<tr>
<td>Duration of illness, median days (range)</td>
<td>5.5 (1–37)</td>
<td>2 (&lt;1–28)</td>
</tr>
<tr>
<td>Received antibiotics for illness</td>
<td>21/54 (39)</td>
<td>11/73 (15)</td>
</tr>
<tr>
<td>Hospitalized</td>
<td>2/54 (4)</td>
<td>2/76 (3)</td>
</tr>
</tbody>
</table>

NOTE. Data are n/N (%) of persons, unless otherwise indicated.
ᵃ Bloody diarrhea (≥3 bowel movements in any 24-h period of illness) alone or diarrhea with nonmenstrual abdominal cramps occurring within 14 days after the start of the camp.
ᵇ Any camp attendee or adult chaperone reporting ≥1 of the following symptoms: nausea, vomiting, diarrhea without cramps, bloody stool (but <3 bowel movements during any 24-h period of illness), nonmenstrual abdominal cramps without diarrhea, subjective or measured fever, chills, or headache occurring within 14 days after the start of the camp.
ᶜ These 3 persons reported <3 bowel movements during any 24-h period of illness and, therefore, did not meet the strict case definition. All 3 reported abdominal cramps. One of the 2 stool specimens that yielded E. coli O111:H8 on culture was submitted by these persons. All 3 persons consumed salad, and 2 also consumed barrel ice.
ᵈ During any 24-h period of illness.
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Figure 1. Bar graph illustrating the onsets of illness of an outbreak of Escherichia coli O111:H8 infections in 55 camp attendees, Texas, June 1999

Table 2. Significant exposures associated with illness, as determined by multivariable logistic regression analysis, in an outbreak of Escherichia coli O111:H8 infections in Texas, 1999.

<table>
<thead>
<tr>
<th>Exposure</th>
<th>Ill persons, n/N (%)</th>
<th>Not case-defined ill persons, n/N (%)</th>
<th>Adjusted OR (95% CI)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Consumed any barrel ice</td>
<td>39/53 (74)</td>
<td>200/462 (43)</td>
<td>3.58 (1.88–6.79)</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Ate any salad</td>
<td>51/55 (93)</td>
<td>367/466 (79)</td>
<td>3.20 (1.11–9.16)</td>
<td>.03</td>
</tr>
</tbody>
</table>

Infections in the state. Ten other groups had used the camp facilities 2 weeks before or after the cheerleading camp; we located sufficient contact information to interview individuals from 7 of these groups. Notable illness occurred in 1 of these groups: 17 (29%) of 58 persons in this group reported at least 1 gastrointestinal symptom, and 8 (14%) met the case definition for our cohort study. No one reported bloody diarrhea, and the 13 persons (22%) who reported abdominal cramping characterized it as mild. No stool specimens were collected for culture. This group used the same dormitory and cafeteria for a 3-day camp that took place 2 weeks before the event we investigated. During the final days of their event, this preceding group was served the same menu as was served during the first 2 days of the cheerleading camp; this group was also provided barrel ice. We could not demonstrate that any of the same foods had been used to prepare any shared menu items. None of the 6 other groups interviewed had been served the same menu as that served to the cheerleading camp.

Environmental investigation. Three ice machines were used to fill the barrels in the cafeteria lobby, to cool items on the salad bar, and to fill beverage ice dispensers in the cafeteria. None had failed or been repaired in the 30 days before the camp. Ice samples collected 2 weeks after the camp ended contained no coliform bacteria. The ice barrels in the cafeteria lobby had no utensil provided for scooping; camp attendees had dipped their hands and water bottles directly into the ice
and reported occasionally submerging their hands, arms, and heads in the ice to cool off.

The salad bar lettuce was a bagged ready-to-use product dispensed without washing during various meals, including lunch on day 1 of the camp. All other salad bar vegetables were washed and then cut or chopped. Salad dressings were commercially prepared or made on site by mixing dried spices with commercial mayonnaise and pasteurized buttermilk. Other toppings were prepackaged and ready to serve. The cob corn was frozen and reheated with steam. The dinner rolls were baked fresh from refrigerated, preshaped dough. Both the cob corn and rolls were served at multiple meals.

Inspections demonstrated evidence of inadequate food heating. No food handlers reported diarrheal illness during the 2 months preceding or the 1 month after the outbreak. We found no evidence of water-supply interruptions or failures in the facilities we inspected.

Microbiologic investigation. Initial stool specimens, cultures of which were performed at commercial laboratories, were obtained from 21 ill campers and yielded no routine enteric pathogens, including E. coli O157. When infection with non-O157 STEC was considered, only 11 stool specimens remained available for evaluation. Specimens were cultured for non-O157 STEC a mean of 17 days after collection, compared with a mean of 10 days after collection for E. coli O157. Three enrichment broth cultures produced detectable Shiga toxin, and 2 yielded Shiga toxin–producing E. coli O111:H8 that contained gene sequences for stx1, stx2, eae, and E-hly but not uidA. The third broth yielded no bacteria. One of the culture-positive specimens was obtained from an adolescent who developed HUS. The other was obtained from an adolescent with bloody stools (not diarrhea) and abdominal cramping. No stool specimens obtained from food handlers yielded STEC.

Serologic investigation. Serum concentrations of anti-O111 LPS IgM and IgG in 13 persons who met the case definition were significantly higher than those in 45 campers who did not have case-defined illness (P = .04 and P = .03, respectively; figure 2). The comparable concentrations of anti-O157 LPS antibodies were not significantly different (P = .89 and P = .74). Case-defined illness was associated significantly with positive anti-O111 LPS IgM and IgG titers, using a cutoff concentration of 1:160, and with positive anti-O111 LPS IgM titers, using a cutoff concentration of 1:320. However, illness was not associated with positive anti-O157 LPS IgM or IgG titers, using either cutoff concentration (table 4).

DISCUSSION

In this outbreak, infections due to non-O157 Shiga toxin–producing E. coli caused life-threatening illness in healthy young adults. Analytic methods used routinely to investigate foodborne outbreaks associated illness with consuming salads and ice in open barrels. Time-varying hazard analysis demonstrated a biologically plausible change in risk-associated exposures over time: illness may have been introduced through consumption of food from the salad bar at the first meal of the camp and may have subsequently spread through consumption of ice from open barrels contaminated by direct contact with campers. Coincident independent associations with eating cob corn and a roll may represent risk associated with eating foods handled extensively by campers’ bare hands, which may have previously touched contaminated ice or diarrheal stool or, possibly, other ill attendees. We did not ask whether persons had washed their hands before meals, and there were no samples of hand-eaten foods available for testing at the time of the investigation to more definitively exclude the unlikely possibility that multiple foods had primary contamination with STEC O111.

This outbreak of E. coli O111:H8 infection was clinically indistinguishable from outbreaks due to E. coli O157:H7 infection. The peak of illnesses on the camp’s third day is consistent with the median 2–4-day incubation period reported for E. coli O157:H7 infection [2, 27]; onset of illness has occurred within 24 h after exposure [28, 29]. The symptom profile among ill persons (table 1) was very similar to that reported in a comparable foodborne outbreak of E. coli O157:H7 infections among mostly young adults attending a college graduation banquet [30]. The proportion of ill persons who developed HUS (4%) was the same as that associated with E. coli O157: H7 outbreaks reported to the CDC in 1998 and 1999 [31, 32]. The outbreak reported here is particularly noteworthy because it affected adolescents, which is not one of the groups generally considered most vulnerable to STEC infection (i.e., children and elderly individuals).

Although the risk of bloody diarrhea among ill persons receiving antibiotic therapy was statistically elevated, the timing of administration does not support a causal relationship. Concern exists that antibiotic administration may increase the risk of HUS in children [33, 34]; too few persons developed HUS in this outbreak to assess whether antibiotics may have contributed to this complication.

E. coli O111 is the second most common non-O157 STEC reported in the United States [35] and among the most common reported in Europe [36]. Two US clusters of STEC O111 infections have previously been reported [19, 37]. Outbreaks have been reported from Canada [38], Australia [39], Japan [40], and Europe [41, 42]. E. coli O111 appears to be a non-O157 STEC associated particularly with HUS [43, 44].

A food vehicle for STEC O111 transmission has been determined in only 1 previous outbreak of infections, which was due to consumption of dried fermented beef sausage [39, 45]. Outbreaks of E. coli O111 infections have also resulted from person-to-person contact [37, 46] and possibly from exposure
Table 3. Significant exposures associated with illness, as determined by multivariable time-varying proportional hazard analysis, in an outbreak of *Escherichia coli* O111:H8 infections in Texas, 1999.

<table>
<thead>
<tr>
<th>Exposure</th>
<th>Ill persons, n/N (%)</th>
<th>Not case-defined ill persons, n/N (%)</th>
<th>Hazard ratio (95% CI)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lunch salad bar, day 1</td>
<td>46/53 (87)</td>
<td>305/462 (66)</td>
<td>4.68 (1.42–15.4)</td>
<td>.01</td>
</tr>
<tr>
<td>Dinner cob corn, day 1</td>
<td>23/54 (43)</td>
<td>100/455 (22)</td>
<td>3.22 (1.62–6.38)</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Dinner roll, day 2</td>
<td>43/54 (80)</td>
<td>271/452 (60)</td>
<td>2.82 (1.38–5.76)</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Barrel ice, day 3</td>
<td>30/52 (58)</td>
<td>141/455 (31)</td>
<td>3.41 (1.70–6.83)</td>
<td>&lt;.01</td>
</tr>
</tbody>
</table>

Figure 2. Bar graphs comparing the serum antibody titers to *Escherichia coli* O111 and *E. coli* O157 lipopolysaccharide (LPS) for 13 case-defined ill camp attendees (i.e., Case) with those for 45 not case-defined ill camp attendees (i.e., Not case).

We cannot exclude the possibility that illness was introduced and spread by an asymptomatic carrier. Interviewing the counselors may have provided clarification. The occurrence of nonbloody diarrheal illnesses 2 weeks before the cheerleading camp in the only other group exposed to the day 1 and 2 menus implies a shared foodborne etiology.

Delayed collection and processing of specimens substantially reduces recovery of *E. coli* O157 [29, 51] and, probably, other STEC, as well. Infection with non-O157 STEC is often first considered after failure to isolate *E. coli* O157. In contrast to *E. coli* O157:H7, the majority of non-O157 STEC cannot be visually distinguished from nonpathogenic *E. coli* on discriminating media (e.g., SMAC). Screening for non-O157 STEC has been facilitated by assays that detect Shiga toxin. Shiga toxin screening should be used as an adjunct and not a replacement to contaminated water [42]. STEC O111 has been isolated from cattle feces [47–49], including cattle feces linked to human illness [50]. It is possible that fields or irrigation water contaminated with feces resulted in contamination of salad items.

Despite conducting a large number of standardized interviews among 31 school groups from across Texas, recall bias may have developed during the month after the camp. The outbreak received news coverage in areas that the attendees returned to, and media reports could have led some respondents to associate illness with a particular exposure. We found no evidence during hypothesis-generating interviews or preliminary surveys that attendees shared any conclusions about the outbreak’s cause. Foods reported by news agencies as associated with previous STEC infections were not statistically associated with illness in this outbreak.
Table 4. Association between case-defined illness and antibody response to Escherichia coli O111 and E. coli O157 lipopolysaccharide for 13 case-defined ill and 45 not case-defined ill camp attendees.

<table>
<thead>
<tr>
<th>Serum antibody</th>
<th>P value, by positive titer cutoff</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anti-O111 LPS IgM</td>
<td>&gt;1:160&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Anti-O111 LPS IgG</td>
<td>&gt;1:320&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Anti-O157 LPS IgM</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Anti-O157 LPS IgG</td>
<td>0.06</td>
</tr>
</tbody>
</table>

NOTE. P values were determined by Fisher’s exact test.
<sup>a</sup> Defined as a borderline-positive serological result.
<sup>b</sup> Defined as a definitively positive serological result.

for bacterial culture and serotyping. Identifying the causative Shiga toxin–positive organism is critical for surveillance and detection of new, emerging pathogens.

Not all non-O157 STEC are pathogenic to humans; increasing surveillance for STEC will capture some of these incidental and nonpathogenic serotypes. Clinicians and epidemiologists should consider this possibility when interpreting culture and serotyping results. Screening for STEC in specimens obtained from persons with more severe clinical illness (e.g., bloody diarrhea and HUS) is nevertheless prudent, particularly because the true burden of disease attributable to non-O157 STEC is unknown. In 2000, the Council of State and Territorial Epidemiologists recommended that isolation of both O157 and non-O157 STEC associated with hemorrhagic colitis or HUS be made notifiable in every state [52]. Surveillance data and case-control studies could substantially advance our understanding of the clinical and microbiological epidemiology of STEC.

We recommend that clinicians consider STEC when evaluating cases of diarrheal illness and that they specifically request testing for O157 and non-O157 STEC in patients with bloody diarrhea or HUS. Clinicians should also inform health departments when they suspect a cluster of STEC infections. All suspect specimens tested for STEC should continue to be screened by culture on SMAC media. Performance of Shiga toxin assays should occur in parallel to SMAC screening; screening specimens with Shiga toxin assays alone without culture and bacterial isolation provides inadequate information for surveillance and other critical public health activities. STEC isolates should be forwarded to public health laboratories for confirmation and serotyping.

Acknowledgments

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