Escherichia coli O157:H7 Outbreak Associated with an Improperly Chlorinated Swimming Pool

Michael S. Friedman, Thierry Roels, Jane E. Koehler, Lynne Feldman, William F. Bibb, and Paul Blake

From the Epidemic Intelligence Service, Notifiable Disease Unit, and Epidemiology Section, Epidemiology and Prevention Branch, Georgia Division of Public Health, Atlanta; Epidemiology Program Office and the Foodborne and Diarrheal Diseases Branch (Preventive Medicine and Immunology Laboratory, Laboratory Section), Division of Bacterial and Mycotic Diseases, National Center for Infectious Diseases, Centers for Disease Control and Prevention, Atlanta; and Health District 8-1, Georgia Division of Public Health, Valdosta, Georgia

A cluster of gastrointestinal illnesses, including one case of hemolytic-uremic syndrome and one culture-confirmed Escherichia coli O157:H7 infection, followed a trailer park pool party. We interviewed a cohort of party attendees and park residents. A primary case was defined as the first gastrointestinal illness within a household between 5 July and 20 July in which the titer of IgG antibody to E. coli O157 (if determined) was elevated. Of 51 party attendees and trailer park residents, 18 developed a gastrointestinal illness, including 10 who met the definition of a primary case. Swimming in the pool significantly increased the risk of primary illness (relative risk = 6.3; 95% confidence interval = 1.8–18.9). No other exposure was significantly associated with primary illness, after pool exposure was controlled for. The implicated pool had little to no chlorine added during the period of 4–10 July. This outbreak provides new evidence of the importance of proper pool maintenance in controlling the spread of E. coli O157:H7.

Since recognition of this strain in 1982, investigations of outbreaks of Escherichia coli O157:H7 infection have increased knowledge of the epidemiology and spread of this emerging pathogen. These investigations have documented that E. coli O157:H7 can be transmitted by consumption of undercooked beef [1–3], unpasteurized milk [4, 5] and apple cider [6], lettuce [7, 8], alfalfa sprouts [9], and municipal water [10, 11]; by swimming in contaminated lakes [12, 13]; and by person-to-person contact [14–16]. Although there have been anecdotal reports of case patients exposed to paddling (shallow) pools [17, 18], we know of no previous evidence that swimming pools can transmit this disease. This report describes an outbreak of E. coli O157:H7 infections associated with swimming in a poorly chlorinated pool.

On 16 July 1996, public health officials in Georgia were informed of a 2-year-old resident of southern Georgia hospitalized for hemolytic-uremic syndrome. The child became ill with bloody diarrhea on 9 July, 5 days after she attended a Fourth of July party at her grandmother’s semicommercial trailer park/recreational center in rural Georgia. Two of the child’s cousins also reported illness with bloody diarrhea of onset around 9 July. The only exposure common to all three children was the party, which included swimming in a pool, a small cookout, and a nearby fireworks display.

On 15 July the grandmother of the index patient developed bloody diarrhea. Two days later, a culture of her stool yielded E. coli O157:H7. At that time, the local health department had not been notified of any other recent cases of E. coli O157:H7 infection in the district. Furthermore, passive surveillance data indicated no cases of E. coli O157:H7 infection had been reported in the district during the previous 12 months. On 19 July we began an investigation to determine the extent of the outbreak, the source of infection, and the means of controlling its spread.

Methods

Epidemiological Investigation

Active surveillance for E. coli O157:H7 infections and hemolytic-uremic syndrome was initiated in the health district and was centered around the county where the party was held. We contacted the three primary care physicians in that county and all pediatricians in a neighboring county to determine whether the incidence of diarrhea (bloody or nonbloody) had increased during July 1996. Because verbal reports of disease activity can be inaccurate, we audited patient logs at the one hospital in the county and at an “after hours” pediatric-care center and an emergency department in nearby cities, comparing the number of patients who had either a chief complaint of diarrhea or a discharge diagnosis of gastroenteritis in June vs. July 1996. In addition, we surveyed the microbiology labora-
E. coli O157:H7. Illnesses meeting the clinical criteria for a primary case were considered cases. A secondary case was defined as a diarrheal illness (three or more loose stools in a 24-hour period) in a member of the cohort who had the first such illness in his or her household (onset, between 5 and 20 July) and had not submitted a specimen for serological confirmation. Additional diarrheal illnesses in family members were included as primary clinical cases if illness began no more than 1 day after onset of the first illness.

A primary serologically confirmed case was an illness in which the patient had, in addition to the above, either an IgM titer of \( \geq 1:320 \) or an IgG titer of \( \geq 1:160 \) specific for E. coli O157:H7. Illnesses meeting the clinical criteria for a primary case but with antibody titers below both cutoff points were not considered cases.

A secondary case was defined as a diarrheal illness (three or more loose stools in a 24-hour period) in a member of the cohort who had the second or third such illness in his or her household and whose onset of illness was \( \geq 1 \) day after the onset of the first illness in that household. If no serum or culture specimen was obtained, the clinical case definition was sufficient. When appropriate specimens were obtained, only illnesses in which antibodies to E. coli O157 were identified or a stool culture yielded E. coli O157:H7 were considered cases.

Laboratory Investigation

No food was served at the Fourth of July party. Blood specimens were drawn from all consenting persons within the cohort. For symptomatic persons, blood specimens were drawn 14–24 days after onset of illness. The specimens were sent to the Foodborne and Diarrheal Diseases Laboratory at the Centers for Disease Control and Prevention (CDC, Atlanta) to test for titers of specific IgM and IgG antibody to E. coli O157 lipopolysaccharide antigen, by means of ELISA. On the basis of published seroprevalence studies [19–22] as well as the laboratory’s previous experience, an IgM antibody titer of \( \geq 1:320 \) or an IgG antibody titer of \( \geq 1:160 \) is considered to be sensitive and specific for recent E. coli O157:H7 infection.

Case Definitions

A primary probable case was defined as a diarrheal illness (three or more loose stools in a 24-hour period) in a member of the cohort who had the first such illness in his or her household (onset, between 5 and 20 July) and had not submitted a specimen for serological confirmation. Additional diarrheal illnesses in family members were included as primary clinical cases if illness began no more than 1 day after onset of the first illness.

A primary serologically confirmed case was an illness in which the patient had, in addition to the above, either an IgM titer of \( \geq 1:320 \) or an IgG titer of \( \geq 1:160 \) specific for E. coli O157:H7. Illnesses meeting the clinical criteria for a primary case but with antibody titers below both cutoff points were not considered cases.

Environmental Investigation

We conducted an environmental investigation to evaluate possible sources of the outbreak, such as contamination of drinking water or pool water. The trailer park’s water came from an on-site well. Water samples collected at park taps on 24 and 31 July were sent to the state laboratory to be tested for the presence of coliform bacteria. Pool and well water specimens collected on 29 July in bottles coated with thiosulfate were sent to the Environmental Protection Agency in Cincinnati for testing for E. coli.

We interviewed the person responsible for maintaining the trailer park pool and requested pool maintenance records. The trailer park was inspected, and the pool was tested for free chlorine on 27 July.

Analysis

We restricted our analysis to (1) persons who had an illness that met the primary case definition and (2) persons who were disease-free, i.e., persons in the cohort who were not ill during 5–20 July and either did not have an elevated titer of antibody to E. coli O157 or had not submitted a serum specimen. Thus, to minimize classification bias, we omitted persons who had no illness during July 5–20 but an elevated antibody titer; secondary cases; and reported illnesses that were not associated with elevated titers.

Analyses were performed with use of Epi Info, version 6.02 (CDC, Atlanta); relative risks, 95% confidence intervals, and Fisher’s exact two-tailed \( P \) values were calculated. For stratiﬁed analyses, Mantel-Haenzel summary relative risks were calculated. We also performed analyses using a strictly clinical deﬁnition for cases and noncases. Persons who had the ﬁrst or second diarrheal illness (if onset of the second case was \( \geq 24 \) hours after the ﬁrst) in a household, with onset during 5–20 July, were considered primary clinical case-patients, regardless of serological results or whether a specimen was submitted. All persons not meeting these clinical criteria, including those with secondary illnesses, were included in the analyses as noncases.

Results

Epidemiological Investigation

Community physicians and hospital microbiologists had not noticed an increase in gastrointestinal illnesses or stool cultures yielding E. coli O157:H7. The number of patients with a clinical diagnosis of “acute diarrhea/gastroenteritis” or “rule-out appendicitis” in the three health care facilities audited were similar during June and July. These data suggest that a larger, community-wide outbreak did not occur.

The three microbiology laboratories surveyed used the proper techniques for culturing stools for E. coli O157:H7. However, stool specimens from only two of the four patients
who presented to the one acute-care center because of bloody diarrhea were cultured for *E. coli* O157:H7, despite the center’s policy requiring *E. coli* O157:H7 testing of all bloody stools. At another center, no such policy existed, and no *E. coli* O157:H7 cultures had been requested in the preceding 3 months.

At the Fourth of July party, the foods served were birthday cake, prepared by the index case’s mother, ice cream, hot dogs, canned beans, chips, dip, and watermelon, but no hamburgers. Beverages served included iced tea and lemonade (both made from powder mixes and well water), as well as bottled soft drinks. Ice for the drinks was obtained from a local supermarket.

Interviews were completed for 43 (98%) of the 44 party attendees and 8 (67%) of the 12 trailer park residents. Overall, 18 (35%) of the 51 persons interviewed had had a gastrointestinal illness that began during the period of 5 July through 20 July (figure 1). Among these 18 reported illnesses, we identified 10 primary cases (1 probable and 9 confirmed) and 4 secondary cases (1 stool culture–positive case, 2 serologically positive cases, and 1 nontested case) with use of our case definitions. The remaining four illnesses occurred in persons with nonelevated serological values and therefore did not meet the case definitions.

After exclusion of asymptomatic persons who had a positive serology (*n* = 6), persons who had secondary illnesses (*n* = 4), and persons who were ill but had negative serologies (*n* = 4), 27 disease-free individuals remained in our cohort.

In the 10 primary cases, the median duration of symptoms was 4 days; all these patients had cramps and diarrhea, and four (40%) had bloody stools. Their median age was 13 years, compared with 23.5 years for persons with secondary illnesses and 34 years for disease-free persons. The male-to-female ratio was 1:1 for primary cases, 1:2 for secondary cases, and 1:1 for disease-free persons.

Swimming in the pool on 4 July or during the following week was significantly associated with illness. Seven (70%) of the 10 persons who reported swimming but only 3 (11%) of the 27 who reported not swimming during 4–10 July developed an illness that met the definition of a primary case (RR = 6.3; 95% CI = 2.0–19.7; table 1). For the seven primary case-patients who swam, the incubation period ranged from 2 to 6 days, with a median of 5 days.

Analyses of other possible exposures to *E. coli* O157:H7 at the Fourth of July party and in the trailer park are summarized in table 1. Cake consumption at the party had a relative risk of 3.6 (95% CI = 0.9–14.6). There was no correlation between the number of pieces of cake eaten and the risk of *E. coli* O157:H7 infection. When stratified by pool use, the association between cake consumption and disease remained nonsignificant (summary RR = 2.0; 95% CI = 0.6–7.0). Conversely, the association between pool use during 4–10 July and disease remained significant when stratified by cake consumption (summary RR = 5.5; 95% CI = 1.7–17.3).

Eating cooked hot dogs during the Fourth of July party was the only other exposure significantly associated with *E. coli* O157:H7 illness. Illness that met the primary case definition occurred in three (75%) of the 4 persons who reported eating hot dogs, compared with 7 (21%) of 33 who reported not eating hot dogs (RR = 3.5; 95% CI = 1.5–8.4). However, stratification by pool use eliminated the association between eating hot dogs and occurrence of *E. coli* O157:H7 illness, with a summary relative risk of 1.1 (95% CI = 0.5–2.5). Conversely, the association between pool use during 4–10 July and disease remained statistically significant when stratified by hot dog consumption (summary RR = 6.0; 95% CI = 1.8–20.0).

Table 1. Association of primary illness with potential exposures, *Escherichia coli* O157:H7 outbreak, Georgia, 1996.

<table>
<thead>
<tr>
<th>Exposure</th>
<th>No. of persons ill/total no. of persons (AR, %)</th>
<th>No. of persons exposed</th>
<th>Not exposed</th>
<th>RR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pool use, 4–10 July Consumption</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cake</td>
<td>8/19 (42)</td>
<td>3/27 (11)</td>
<td>6.3</td>
<td>2.0–19.7</td>
</tr>
<tr>
<td>Hot dogs</td>
<td>3/4 (75)</td>
<td>7/33 (21)</td>
<td>3.5</td>
<td>1.5–8.4</td>
</tr>
<tr>
<td>Ice cream</td>
<td>3/14 (21)</td>
<td>7/22 (32)</td>
<td>0.7</td>
<td>0.2–2.2</td>
</tr>
<tr>
<td>Beans</td>
<td>0/1 (0)</td>
<td>10/34 (29)</td>
<td>?</td>
<td>. . .*</td>
</tr>
<tr>
<td>Watermelon</td>
<td>2/4 (50)</td>
<td>8/33 (24)</td>
<td>2.1</td>
<td>0.7–6.5</td>
</tr>
<tr>
<td>Chips</td>
<td>4/14 (29)</td>
<td>6/23 (26)</td>
<td>1.1</td>
<td>0.4–3.2</td>
</tr>
<tr>
<td>Dip</td>
<td>2/7 (29)</td>
<td>8/29 (28)</td>
<td>1.0</td>
<td>0.3–3.8</td>
</tr>
<tr>
<td>Well water</td>
<td>4/8 (50)</td>
<td>6/28 (21)</td>
<td>2.3</td>
<td>0.9–6.3</td>
</tr>
<tr>
<td>Ice</td>
<td>8/25 (32)</td>
<td>2/11 (18)</td>
<td>1.8</td>
<td>0.4–7.0</td>
</tr>
</tbody>
</table>

NOTE. All food exposures occurred during a Fourth of July party (see text). AR = attributable risk.

* Undefined (*P* = 1.00).
Two confirmed primary cases occurred in trailer park residents who had not participated in the Fourth of July activities. They had had no contact with the party attendees, including the other eight persons who had primary cases. One of these persons swam in the trailer park pool only on 10 July and was hospitalized 2 days later with severe gastroenteritis and diarrhea. The other person denied exposure to the pool.

The alternative analysis, in which a strictly clinical case definition was used, supported the above findings. Eight (53%) of 15 persons who swam in the pool vs. 4 (11%) of 35 persons who did not swim had illnesses that met the primary clinical case definition (RR = 4.7; 95% CI = 1.7–13.2). There was no association between illness and eating cake (RR = 1.4; 95% CI = 0.5–4.1) or hot dogs (RR = 1.5; 95% CI = 0.5–4.5).

Laboratory Investigation

Only two persons had stool specimens cultured on MacConkey-sorbitol medium. One specimen yielded E. coli O157:H7. It was obtained from a woman whose illness began on 15 July, 6 days after her grandchild (the index case) became ill. The other specimen, obtained from the child who had hemolytic-uremic syndrome, did not yield E. coli O157:H7; however, it was collected after treatment with antibiotics.

Blood specimens were obtained from 11 (92%) of 12 persons who had a primary clinical illness and 4 (67%) of 6 persons who had a secondary illness. The one illness meeting the primary case definition but lacking serological confirmation occurred in a child who had bloody diarrhea that began on 9 July. Of the 51 persons in the cohort, 29 (57%) submitted blood specimens. Eight (28%) of these 29 persons had elevated titers of both IgM and IgG antibody to E. coli O157:H7; and 9 (31%) had elevated titers of only IgG antibody. Thus, 17 (59%) of 29 had serological evidence of E. coli O157:H7 infection.

A history of gastrointestinal illness during the period of 22 June through 22 July was significantly associated with elevated antibody titers; 12 (75%) of 16 serologically tested persons with a history of gastrointestinal illness had elevated E. coli O157 antibody titers, vs. 5 (38%) of 13 who remained well ($P < .05$). The median IgG titer among ill persons was 1:320, vs. 1:80 among those who remained well (range, 1:40 to >1:320).

Environmental Investigation

Three of four well water samples from different taps in the trailer park tested at the state laboratory yielded coliform bacteria, but none had detectable fecal coliform bacteria. Minor external work had been done on the well ~5 weeks before the outbreak, which could have led to temporary contamination of the well with nonpathogenic organisms. None of the eight water samples sent to the Environmental Protection Agency yielded pathogenic E. coli.

The pool was a 20-year-old indoor facility, measuring 8 × 12 feet and 4.5 feet deep, and was family owned and operated. Although it was frequently rented for parties, the pool was not officially registered as a commercial pool. Therefore, local public health environmentalists had never inspected it. No maintenance logs or records of chlorine level measurements were kept. There was no continuous chlorination system, nor was a chlorine stabilizer used. Intermittent, perhaps infrequent, “shock treatment” of the pool with large quantities of chlorine was the only mechanism used to maintain the pool.

The family stated that no chlorine was added to the pool on 4 July, when 11 persons swam. After recognition of the outbreak, the family hyperchlorinated the pool multiple times. On 24 July, the pool’s free chlorine level measured 1.0 ppm, which is within the recommended level for pool water [23].

The family member responsible for pool maintenance at the trailer park reported having had a gastrointestinal illness with diarrhea and severe cramps on 25 June, for which he went to a nearby emergency department. No cultures were performed. However, 4 weeks following his illness, his titers of IgM and IgG antibody to E. coli O157 were elevated.

Discussion

Our investigation found a strong association between swimming in the pool and infection with E. coli O157:H7. To our knowledge, this is the first report of E. coli O157:H7 infection associated with a chlorinated swimming pool. The only other exposures of interest were eating cake and eating hot dogs. Cake consumption during the Fourth of July party was more common among persons who became ill with E. coli O157:H7, but this exposure was not statistically significant. Those who had eaten cake were more likely to swim at the party and therefore to become ill.

Eating hot dogs at the party was significantly associated with E. coli O157:H7 illness on univariate analysis, but only three of the 10 primary case-patients had eaten hot dogs. The stratified analysis demonstrated that, like cake, hot dogs were indirectly associated with illness since the persons who ate hot dogs (i.e., children) were more likely to swim and thus become ill. Finally, although dry fermented salami has caused one outbreak of E. coli O157:H7, hot dogs are cooked twice and have never been implicated as a source of E. coli O157:H7 infection [24].

The case involving the trailer park resident with delayed pool exposure provides additional evidence that the pool was the source of the outbreak. This resident did not attend the party or consume anything served at the party. His illness was extremely unlikely to be a secondary case, since he had no known contact with any of the other ill persons on or after 4 July. His illness was severe, requiring hospitalization for 2 days because of extreme abdominal pain and diarrhea. No stool cultures were performed, but his titer of IgG antibody to E. coli O157:H7 was elevated, at 1:320.
The body of knowledge regarding *E. coli* O157:H7 supports our finding that poorly chlorinated pool water can be a source of infection. Lakes and unchlorinated paddling pools for toddlers have been implicated in previous outbreaks of *E. coli* O157:H7 infection [12, 13, 17, 18]. Water used to wash produce has been suggested as the means of widespread contamination of apples and lettuce in recently investigated outbreaks [6, 8]. In addition, experimental work has demonstrated the survival of *E. coli* O157:H7 in water for as long as 10 weeks at 25°C [25]. In this outbreak, the interval between pool exposure and onset of symptoms for all swimmers who had primary illnesses was consistent with the known 1–8-day incubation period for *E. coli* O157:H7 [3].

It is uncertain how the pool became contaminated. The family member responsible for the pool’s maintenance had had a severe gastrointestinal illness beginning 25 June and could have been shedding *E. coli* O157:H7 on 4 July, 9 days later. Other possible sources are an infected swimmer in a group that rented the pool on 30 June, an infected swimmer on the day of the party, and a swimmer who had just walked barefoot through one of the nearby cow pastures before entering the pool.

Our investigations revealed that the trailer park pool was not being maintained properly. Dumping large quantities of chlorine into a pool, commonly known as “shocking” a pool, is a common practice [23]. Performed intermittently, perhaps weekly, shock treatments often keep the pool water clear and odorless by preventing the growth of algae [23, 26]. However, this will not prevent the survival of bacteria once the chlorine has been neutralized. Neutralization can occur within hours to days, particularly when chlorine stabilizer is not used and a system for continuously adding more chlorine is not in place, as was the situation here.

Most of the bacteriocidal activity of chlorine comes from free, nonionized chlorine. Free chlorine dissipates rapidly in bright sunlight, with high temperatures, and with agitation of the water [25]. Chlorine’s bacteriocidal activity in a pool also weakens with time and with contact with pool contaminants, which quickly neutralize free chlorine [23, 26]. With no continuous chlorinator and no chlorine added to the pool on the day of the party, any free chlorine that remained active that day was probably rendered ineffective by the number of persons in the pool. Therefore, *E. coli* O157:H7 bacteria, once introduced into the pool, may have persisted in the warm, chlorine-free water.

This outbreak emphasizes the role of public health officials in regulating commercial pools and educating owners of residential pools. Although the implicated pool was frequently rented out to large groups, it was not officially registered as a commercial pool and thus was not regulated by the county. Prior inspection of the pool by a knowledgeable public health environmentalist might have revealed the need for a continuous chlorine pump. Efforts to educate pool owners about the importance of proper pool maintenance might help improve the safety of unregulated pools.

This outbreak investigation had several limitations. Comparison of molecular subtypes of strains, a useful technique in outbreak investigations and surveillance, was not feasible, as we had only one isolate [27]. Second, because of the lack of isolates, our case definitions were based on a combination of clinical and serological findings. However, at least one other study has used titers of antibody to lipopolysaccharide to identify outbreak-related cases [8].

Given the 73%–92% sensitivity and 96% specificity of elevated IgG antibody titers in previous studies [19–22], the use of serology was instrumental in confirming that this cluster of illnesses was indeed an *E. coli* O157:H7 outbreak and helped tighten our definition of a primary case and a disease-free person. Since the sensitivity of serology may be lower with milder infections [22], it is reassuring to note the findings were similar when a strictly clinical case definition was used.

Third, there was potential for misclassification of persons who had primary and secondary illnesses and those who were disease-free. Accordingly, we used strict criteria for the primary case and disease-free definitions. However, this eliminated 27% of the cohort from the analysis. To determine if this affected the results, we reanalyzed the data, considering as disease-free all persons in the cohort whose illness did not meet the primary case definition. In this analysis of the entire cohort, use of the swimming pool was the only statistically significant risk exposure (RR = 5.4; 95% CI = 1.6–18.3).

Finally, environmental culture specimens could not be obtained early in the outbreak, when recovery of the organism might have been possible. By the time the outbreak was identified and proper samples could be taken, the pool had been chlorinated multiple times. However, previous experience suggests that the yield from environmental culturing of water may be low. In previous waterborne outbreaks of *E. coli* O157:H7, environmental cultures did not yield *E. coli* O157:H7, despite the fact that cultures were performed while transmission was still ongoing [10–12].

These limitations cast a new light on the common misperception that *E. coli* O157:H7 is not a problem in the southeast region of the United States [28]. Without the index case of hemolytic-uremic syndrome, this outbreak of gastrointestinal illness might have been labeled “endemic summer diarrhea” by the local medical community. Only with heightened clinical suspicion and proper microbiological protocols for handling bloody diarrheal specimens will *E. coli* O157:H7 illnesses and outbreaks be promptly and correctly identified.

Our implication of a poorly maintained swimming pool as the source of an outbreak of *E. coli* O157:H7 adds yet another way in which this organism can be transmitted and strongly suggests that it can survive for at least several days in inadequately chlorinated swimming pools. A recent population-based study of sporadic *E. coli* O157:H7 infections found a statistically significant association between swimming in outdoor pools and *E. coli* O157:H7 illness [29]. In the context of
our findings, this suggests that contaminated pools may play a larger role in the burden of *E. coli* O157:H7 illness than has been recognized and that careful pool maintenance may help prevent these potentially devastating infections.

**Acknowledgment**

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**References**