Lake-Associated Outbreak of Escherichia coli O157:H7 in Clark County, Washington, August 1999

Michael G. Bruce, MD, MPH; Michael B. Curtis, MD, MPH; Melanie M. Payne, MPH; Romesh K. Gautom, PhD; Eric C. Thompson, BS; Aimee L. Bennett, BA; John M. Kobayashi, MD, MPH

Context: Escherichia coli O157:H7, one of hundreds of strains of the gram-negative bacterium E coli, has been implicated in numerous lake-borne outbreaks of infection during the past decade. In August 1999, several children who later became ill with E coli O157:H7 infection reported swimming in a lake in Clark County, Washington. The lake was closed and an investigation begun.

Objectives: To identify the source of the outbreak and determine risk factors for infection with E coli O157:H7.

Design, Setting, and Patients: Two case-control studies were performed among residents of and visitors to Clark County in August 1999 by using community and campground-registrant control subjects.

Main Outcome Measure: Risk factors for infection with E coli O157:H7 among Clark County residents or visitors.

Results: We identified 37 case patients (including 29 primary-case patients) with a median age of 5 years (age range, 1-14 years for primary-case patients). Eight children were hospitalized, 3 with hemolytic uremic syndrome; none died. With analysis restricted to primary-case patients, illness was strongly associated with swimming in the lake (18 of 18 case patients vs 1 of 18 neighborhood-matched and age-matched control subjects; matched odds ratio undefined; P <.001). All primary-case patients were children younger than 15 years who swam in the lake. Illness was associated with placing the head underwater, getting lake water in the mouth, or swallowing lake water (26 of 27 case patients vs 43 of 62 control subjects; matched odds ratio = 11.5; P = .005). Cultures of lake water yielded E coli O157:H7 that matched the outbreak strain according to results of pulsed-field gel electrophoresis.

Conclusions: To date, this is one of the largest documented outbreaks of E coli O157:H7 infection associated with unchlorinated recreational water and represents the first outbreak in which the strain was isolated from lake water. Guidelines are needed to decrease the risk of enteric illness associated with swimming in recreational lakes.

tion increased in Clark County in southwestern Washington. Initial interviews indicated that all cases occurred in children who swam in a popular recreational lake in a nearby state park. An investigation was conducted to identify the extent of the outbreak and determine its source.

### METHODS

#### BACKGROUND

Battle Ground Lake is a freshwater volcanic lake 28 acres (11.3 hectares) in area that is fed by underground springs that flow through large lava tubes. There is a steep caldera rim surrounding the lake. The lake sits within Battle Ground Lake State Park, a multiuse facility that includes campgrounds with a capacity of 200, picnic areas, playgrounds, a concession area, a shallow swimming area for children (stagnant water ≤2 ft [0.6 m] deep with a mud bottom) and an adult swimming area (≥4 ft [1.2 m] deep), with the total beach area measuring 125 ft (38.1 m) long. The toilet facilities closest to the beach are 300 ft (91.4 m) up the steep caldera rim. The park is visited by an estimated 400 day users and 200 overnight campers on an average summer day; however, in August 1999, warm weather and low precipitation levels were noted, and the park had as many as 2500 weekend visitors.

#### DEFINITIONS AND CASE FINDING

We defined a case patient as any resident of or visitor to Clark County with diarrhea onset after August 15, 1999, and either isolation from stool of the outbreak strain of *E. coli* O157:H7, as determined by means of PFGE, or serologic diagnosis with an IgM or IgG antibody titer of 1:320 or more. A case was defined as primary if there was no close contact with another case patient during this period. Case patients were identified through routine laboratory and physician reports; telephone calls to the health department by physicians, laboratories, and the public after extensive news coverage; and telephone calls to persons who had visited the state park between August 13 and August 28, 1999. Once the outbreak was recognized, we encouraged area physicians and laboratories to consider *E. coli* O157:H7 infection and use appropriate cultures.

We interviewed all residents of or visitors to Clark County with reported *E. coli* O157:H7 infection and onset of diarrhea after August 15, 1999. Serum samples were collected from persons who met all aspects of the case definition but from whom stool samples were not available.

For primary-case patients, the incubation period was defined as the number of days between the swim date and the date of symptom onset. When multiple swim dates were given, the date closest to symptom onset was used.

#### CASE-CONTROL STUDIES

Two consecutive case-control studies were performed to determine risk factors for infection. Only primary-case patients were included in this analysis. Using a standardized questionnaire administered by telephone, we obtained demographic and risk factor information from all case patients and control subjects (or their parents). Because most cases were among children, control subjects were limited to children younger than 15 years. Parents of children without symptoms were interviewed about their child's potential exposures, including lake water, drinking water, and food consumption while at Battle Ground Lake State Park. They were also asked about other potential exposures, such as consumption of undercooked meat, raw milk, and sprouts; contact with diapered children; and handling livestock.

#### Community Control Subjects

An age-matched and neighborhood-matched case-control study was performed in the Battle Ground municipal area. We per-

---

### Table 1. Reported *Escherichia coli* Outbreaks Associated With Recreational Water

<table>
<thead>
<tr>
<th>Authors</th>
<th>Year of Outbreak</th>
<th>State/Country</th>
<th>Serotype</th>
<th>No. of Cases</th>
<th>Median Age, y</th>
<th>Age Range, y</th>
<th>Case Patients With HUS, %</th>
<th>Risk Factor for Case-Control Study</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>Keene et al(^1)</td>
<td>1991</td>
<td>Oregon</td>
<td>O157</td>
<td>2</td>
<td>6</td>
<td>1-16</td>
<td>14</td>
<td>Swim, swallow water Lake</td>
<td></td>
</tr>
<tr>
<td>Brewster et al(^3)</td>
<td>1992</td>
<td>Scotland</td>
<td>O157</td>
<td>6</td>
<td>3</td>
<td>NA</td>
<td>17</td>
<td>NA Padddling pool</td>
<td></td>
</tr>
<tr>
<td>Hildebrand et al(^2)</td>
<td>1993</td>
<td>England</td>
<td>O157</td>
<td>6</td>
<td>4</td>
<td>3-8</td>
<td>50</td>
<td>NA Padddling pool</td>
<td></td>
</tr>
<tr>
<td>Cramberg et al(^4)</td>
<td>1993</td>
<td>The Netherlands</td>
<td>O157</td>
<td>4</td>
<td>3</td>
<td>NA</td>
<td>100</td>
<td>NA Lake</td>
<td></td>
</tr>
<tr>
<td>Ackman et al(^5)</td>
<td>1994</td>
<td>New York</td>
<td>O157</td>
<td>12</td>
<td>7</td>
<td>1-13</td>
<td>17</td>
<td>Swallow water Lake</td>
<td></td>
</tr>
<tr>
<td>Centers for Disease</td>
<td>1995</td>
<td>Illinois</td>
<td>O157</td>
<td>12</td>
<td>NA</td>
<td>2-12</td>
<td>25</td>
<td>Swim, swallow water Lake</td>
<td></td>
</tr>
<tr>
<td>Levy et al(^6)</td>
<td>1995</td>
<td>Wisconsin</td>
<td>O157</td>
<td>8</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA Lake</td>
<td></td>
</tr>
<tr>
<td>Levy et al(^6)</td>
<td>1995</td>
<td>Minnesota</td>
<td>O157</td>
<td>5</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>Lake</td>
<td></td>
</tr>
<tr>
<td>Levy et al(^6)</td>
<td>1995</td>
<td>Minnesota</td>
<td>O157</td>
<td>5</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>Lake</td>
<td></td>
</tr>
<tr>
<td>Friedman et al(^7)</td>
<td>1996</td>
<td>Georgia</td>
<td>O157</td>
<td>18</td>
<td>13</td>
<td>NA</td>
<td>NA</td>
<td>Trailer park pool</td>
<td></td>
</tr>
<tr>
<td>Barwick et al(^8)</td>
<td>1997</td>
<td>Missouri</td>
<td>O157</td>
<td>8</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>Lake</td>
<td></td>
</tr>
<tr>
<td>Pauino et al(^9)</td>
<td>1997</td>
<td>Finland</td>
<td>O157</td>
<td>14</td>
<td>3-8</td>
<td>NA</td>
<td>NA</td>
<td>Lake</td>
<td></td>
</tr>
<tr>
<td>Barwick et al(^10)</td>
<td>1998</td>
<td>Minnesota</td>
<td>O157</td>
<td>5</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>Lake</td>
<td></td>
</tr>
<tr>
<td>Barwick et al(^11)</td>
<td>1998</td>
<td>Georgia</td>
<td>O157</td>
<td>26</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>Water park</td>
<td></td>
</tr>
<tr>
<td>Feldman et al(^12)</td>
<td>1999</td>
<td>California</td>
<td>O157</td>
<td>7</td>
<td>NA</td>
<td>6-7</td>
<td>0</td>
<td>Swallow water Lake</td>
<td></td>
</tr>
<tr>
<td>McCarthy et al(^13)</td>
<td>1999</td>
<td>Connecticut</td>
<td>O121</td>
<td>11</td>
<td>6.5</td>
<td>1-62</td>
<td>27</td>
<td>Swallow water Lake</td>
<td></td>
</tr>
<tr>
<td>Lee et al(^14)</td>
<td>1999</td>
<td>Wisconsin</td>
<td>O157</td>
<td>5</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA Lake</td>
<td></td>
</tr>
<tr>
<td>Lee et al(^15)</td>
<td>1999</td>
<td>Florida</td>
<td>O157</td>
<td>2</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA Ditch water</td>
<td></td>
</tr>
<tr>
<td>Lee et al(^16)</td>
<td>1999</td>
<td>Nebraska</td>
<td>O157</td>
<td>7</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA Wading pool</td>
<td></td>
</tr>
<tr>
<td>Health Protection Agency(^17)</td>
<td>1999</td>
<td>England</td>
<td>O157</td>
<td>14</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA Beach</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: HUS, hemolytic uremic syndrome; NA, not available.
formed a 1:1 match for age (±1 year) and neighborhood to distribute these 2 potential confounding variables in an identical manner among both groups. Telephone numbers were chosen by using a forward-digit dialing protocol that involved dialing the first 5 digits of the case patient’s 7-digit telephone number, then replacing the last 2 digits sequentially starting at 00, then 01, and so on, up to 99 until neighborhood-matched and age-matched control subjects were selected. Parents were interviewed about children without symptoms living in the household who were closest in age to the case patient. No child with an episode of diarrhea in the preceding 3 weeks was included as a control subject. If 2 children within the household matched the age criterion, then the control subject closest in age to the case patient was selected.

**Camper Control Subjects**

The campground registration list, which included advance reservations, was used to identify another group of control subjects. Each telephone number on the list was called in an effort to identify children with no diarrhea in the preceding 3 weeks who were younger than 15 years and had either camped or spent 1 or more days at Battle Ground Lake State Park during the outbreak from August 13 through August 28, 1999. Any child who had not been to the park during the outbreak or who had had a diarrheal illness in the preceding 3 weeks was excluded.

**LABORATORY INVESTIGATION**

**Clinical Isolates**

Standard procedures were followed at the WPHL for the recovery of *E coli* O157:H7 from clinical samples. Stool samples from Cary-Blair transport medium were cultured on sorbitol-MacConkey agar. Sorbitol-negative colonies were identified as *E coli* O157:H7 by means of latex agglutination (*E coli* O157 Test Kit; Oxoid Limited, Basingstoke, England) and confirmed biochemically. The flagellar antigen H7 was identified by means of agglutination (RIM *E coli* O157:H7 Latex Kit; Remel Inc, Lenexa, Kan). Serum specimens obtained by means of venipuncture were sent to the Centers for Disease Control and Prevention in Atlanta, Ga, and were assayed for antibodies to *E coli* O157:H7 lipopolysaccharide antigens as described elsewhere. Any IgM or IgG titers of 1:320 or more were considered positive for *E coli* O157:H7.

**Environmental Isolates**

At the WPHL, 10-g sediment samples and 10-mL water samples were incubated overnight at 42°C in A-1 broth. The initial concentration of A-1 broth was such that the addition of environmental samples did not reduce ingredient concentrations to less than that of the standard medium. Immunomagnetic separation with beads coated with anti-O157:H7 (Dynabeads anti-*E.coli* O157; Dynal Biotech, Oslo, Norway) was performed on overnight enrichments from A-1 broth and plated on tellurite cefixime sorbitol-MacConkey agar. Typical *E coli* O157:H7 colonies were picked from tellurite cefixime sorbitol-MacConkey agar plates and confirmed by using standard biochemicals and latex agglutination. In addition to the A-1 method, lake water samples were tested by using the standard membrane filtration procedure for drinking water. The total volume of water filtered was 100 mL. Four filters were made: 1 was used after the usual procedure with m-endu broth (MilliQone Corp, Billerica, Mass), 2 were placed on selective plating media (tellurite cefixime sorbitol-MacConkey agar and Fluorocult *E coli* O157:H7 agar [Merck & Co Inc, Darmstadt, Germany]), and the remaining filter was placed in a modified trypticase soy broth with agar.

Both clinical and environmental isolates were subcultured on appropriate media and incubated at 37°C overnight. Genomic DNA from the isolates was prepared and analyzed with the PFGE method described by Gautom.

**ENVIRONMENTAL INVESTIGATIONS**

Environmental health specialists inspected the waste water systems closest to the swimming beach to assess their integrity and placed fluorescein dye in these systems to look for leakage into the swimming area. Operations and food-handling procedures of the sole food concessionaire were reviewed. Drinking water from water fountains near the swimming area was tested for evidence of fecal contamination by using a fluorescent reagent test (Colilert; IDEXX Laboratories Inc, Westbrook, Me) on August 29, 1999. Before the identification of the outbreak, water from the child and adult swimming areas of the lake was not routinely tested. However, after 2 children reported becoming ill after swimming at the lake, 2 samples of lake water were collected on August 26, 1999 (2 days before the outbreak was identified and the lake closed) and tested for fecal coliforms by using standard methods. Once the outbreak was identified, additional samples of lake water and sediment were collected on August 29 and September 3, 1999, from multiple sites in and around the child and adult swimming areas of the lake. All samples were sent to the WPHL and tested for *E coli* O157:H7.

**STATISTICAL ANALYSIS**

Univariate odds ratios and univariate matched odds ratios were calculated by using commercially available software (Epi Info version 6.04b; Centers for Disease Control and Prevention). *P* values were 2-tailed, and values less than .05 were considered statistically significant.

**RESULTS**

**DESCRIPTIVE EPIDEMIOLOGIC FINDINGS**

We identified 37 cases of *E coli* O157:H7 infection (35 culture confirmed, 2 serologically confirmed) (Figure 1). All 29 primary-case patients had visited the lake from August 13 through August 28, 1999. Swim dates were available in 28 of the 29 primary-case patients; the median incubation period from swim date to symptom onset was 3.5 days (range, 1-11 days). Median age in all case patients was 5 years (age range, 8 weeks to 39 years); all primary-case patients were younger than 15 years. Twenty-four case patients (65%) were male. Eight secondary cases occurred among household or other close contacts. According to self-report, all 37 case patients had diarrhea, 31 (84%) had abdominal cramps, 24 (65%) had bloody diarrhea, 17 (46%) had headache, 17 (46%) had fever, and 8 (22%) received antibiotics during their illness. Eight children were hospitalized, including 3 with hemolytic-uremic syndrome; none died.

**EXCLUSION OF POTENTIAL CONTROL SUBJECTS**

Thirty-eight of 100 potential camper control subjects contacted were excluded because of diarrheal illness. No community control subjects reported diarrheal illness.
CASE-CONTROL STUDIES

Community Control Subjects

Of the 29 total primary cases identified in this outbreak, 18 case patients were available when the first case-control study was performed. In a matched univariate analysis, illness was strongly associated with swimming in the lake (18 of 18 case patients vs 1 of 18 neighborhood-matched and age-matched control subjects; matched odds ratio undefined; \( P < 0.001 \)). No other risk factors were significantly associated with disease.

Camper Control Subjects

Of the 29 total primary cases identified in this outbreak, 27 case patients were available at the time that the second case-control study was performed. In a univariate analysis, case patients were significantly more likely than camper control subjects to have taken lake water into the mouth (\( P = 0.002 \)) and to have swallowed lake water (\( P = 0.001 \)). Case patients were also more likely to have put their head underwater than were control subjects, although this difference was not statistically significant. When all 3 water exposure variables were combined into 1, case patients were more likely than control subjects to report at least 1 of the 3 (26 of 27 case patients vs 43 of 62 control subjects; matched odds ratio = 11.5; \( P = 0.005 \)). They also tended to spend more time in the water than did control subjects (\( P = 0.007 \)) (Table 2).

BACTERIAL SUBTYPING

All 35 clinical isolates of Escherichia coli O157:H7 obtained during the outbreak were subtyped with PFGE and found indistinguishable from one another.

ENVIRONMENTAL INVESTIGATIONS AND CONTROL MEASURES

Inspection of the waste water system closest to the swimming beach by health district environmental specialists revealed an intact system functioning properly, and fluorescein dye study results showed no evidence of leakage into the swimming area. No irregularities were detected in a review of operations and procedures used by the food concessionaire. Drinking water samples from water fountains near the swimming area were negative for coliforms. The 2 samples of lake water collected August 26, 1999, showed fecal coliform levels at 18 and 93 organisms per 100 mL, which is less than the Washington State regulatory standard of 100 organisms per 100 mL for recreational water quality. The WPHL's testing of lake water and sediment samples collected September 3, 1999, from the children's swimming area identified Escherichia coli O157:H7 in the sediment sample. Furthermore, DNA fingerprinting with PFGE of these environmental isolates revealed patterns that exactly matched the outbreak pattern of the clinical isolates obtained from swimmers and their close contacts (Figure 2). In addition, results of a separate laboratory investigation at the University of Washington (Seattle)

Table 2. Univariate Analysis of Risk Factors for Escherichia coli O157:H7 Infection in Case Patients vs Camper Control Subjects

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>III, No. (%) (n = 27)*</th>
<th>Not III, No. (%) (n = 62)</th>
<th>Odds Ratio (95% Confidence Interval)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Head underwater</td>
<td>21 (78)</td>
<td>37 (60)</td>
<td>2.4 (0.8-8.1)</td>
</tr>
<tr>
<td>Water in mouth</td>
<td>24 (89)</td>
<td>33 (53)</td>
<td>7.0 (1.8-39.4)</td>
</tr>
<tr>
<td>Swallowed water</td>
<td>14 (52)</td>
<td>12 (19)</td>
<td>4.5 (1.5-13.4)</td>
</tr>
<tr>
<td>Water exposure†</td>
<td>26 (96)</td>
<td>43 (69)</td>
<td>11.5 (1.6-495.4)</td>
</tr>
<tr>
<td>&gt;2 h per day spent in lake</td>
<td>7 (26)</td>
<td>3 (5)</td>
<td>6.9 (1.4-44.0)</td>
</tr>
</tbody>
</table>

*Of the 29 primary cases, 2 were not included in the case-control study because of delay in diagnosis.
†One or more of the first 3 water exposure variables.
confirmed a match between environmental and clinical isolates by means of PFGE.28

This outbreak of 37 confirmed cases of E coli O157:H7 infection was caused by contamination of a freshwater lake. To our knowledge, this is one of the largest documented outbreaks of E coli O157:H7 infection from unchlorinated recreational water.

Previous investigators have inferred that the dose of E coli O157:H7 required to cause illness is low (<100 bacteria),16,29 and although the major reservoir is cattle and other mammals, the organism can survive for lengthy periods in the environment.30-32 Both these observations are supported by the results of our investigation. Many case patients reported inadvertent ingestion of small amounts of lake water, which could result in a low infectious dose. In this outbreak, the exposures to the implicated lake among ill persons occurred during a 16-day period, and the outbreak strain was recovered from lake samples obtained 21 days after the first reported exposure, which indicates that the organism could have survived in the lake at least 3 weeks.

Our findings are limited by a lack of information about the number of swimmers in the lake per day; therefore, we were unable to calculate attack rates. For the 28 primary-case patients from whom swim dates were available, 7 (25%) swim on August 19, 1999; including these 7 patients, 24 (86%) swim between August 19 and August 26, 1999, which implies a relatively high rate of contamination of the lake for these 8 days (Figure 3).

In both case-control studies, misclassification bias could have occurred from parents inaccurately reporting their child's exposure. Media coverage was intense and may have contributed to recall bias. Sick control subjects were excluded to limit misclassification bias toward the null that could have occurred if persons with E coli O157:H7 infection had been included in the control group; however, persons with asymptomatic E coli O157:H7 infection could have been classified as control subjects, which would result in a misclassification bias toward the null.

All primary cases occurred in children younger than 15 years, 3 of whom developed hemolytic uremic syndrome. In other reported lake-associated outbreaks of E coli O157:H7, children have also been disproportionately affected.14-24

The source of contamination was likely the bathers themselves. No agricultural sources of contamination were identified, and illness was not associated with food or beverage exposures. Children may have contributed to contamination of the lake through fecal accidents or dirty diapers. During the investigation, we received a report about a feces-laden diaper having been rinsed in the lake near the children's swimming area. It is likely that E coli O157:H7 from feces was introduced into the shallow still water of the children's swimming area sometime in the early to middle part of August 1999 and that the organism survived for a time in the lake or was periodically reintroduced via fecal accidents (Figure 1).

A possible contributing factor in this outbreak was the lack of appropriate diaper-changing stations in the area. Additionally, the distance of toilets from the beach was 300 ft (91.4 m), and the lack of nearby hand-washing facilities may have made it difficult for parents to take their children on bathroom breaks regularly and wash their hands after using the toilet. Human fecal contamination has also been suggested as the likely source of pathogens in other swimming-associated outbreaks.14,15,18,24

Before the outbreak, lake water was not routinely monitored. Testing of lake water after 2 children reported becoming ill and shortly before the lake was closed revealed allowable levels of fecal coliforms. Results from other outbreaks also illustrate that levels of indicator organisms may be within acceptable limits at the time swimmers acquire infection with E coli O157:H7.14,15,17 The US Environmental Protection Agency has recently established guidelines for microbial quality in fresh recreational water, with E coli (monthly geometric mean <126 organisms per 100 mL) or enterococci (monthly geometric mean <33 organisms per 100 mL) as the preferred proxy indicators in lieu of fecal coliforms (http://www.epa.gov/waterscience/beaches/local/sum2.html). Numerous pathogens in addition to E coli O157:H7 have caused outbreaks of gastroenteritis and other syndromes associated with swimming or wading in freshwater lakes, including Shigella, Leptospirosis, viral agents, Cryptosporidium, and schistosomes that cause dermatitis.3,5

Lakes can become contaminated by a variety of mechanisms, and although monitoring may contribute to some level of safety, no freshwater lake should be considered risk free, even with intensive monitoring. Recognizing that complete safety in fresh recreational water cannot be ensured, the challenge is to minimize risk so that families can continue to enjoy lake swimming. Results of this investigation point out the need for guidelines to decrease the risk of transmission of infectious diseases from recreational freshwater sources. Assessing the availability of enough convenient toilets, diaper-changing stations, and hand-washing facilities may be more important than testing the water for microbes.
During the past decade, numerous outbreaks of infection with *E. coli* O157:H7 have been reported in association with swimming in recreational water. This association has until now been made with epidemiologic evidence alone.

Our investigation results demonstrated epidemiologic evidence implicating the lake, as well as laboratory evidence confirming the existence of a strain of *E. coli* O157:H7 in the lake that matched the clinical outbreak strain identified with PFGE. In addition, we were able to determine specific risk factors associated with swimming that may help in future prevention efforts.

Accepted for publication February 21, 2003.

This study was supported by the Washington State Department of Health, Seattle, and Southwest Washington Health Department, Vancouver.

We thank Karen Steingart, MD, Chris Plaster, Fran Cappa, PhD, Rodie Renn Lasher, MS, Maya Bhat, MPH, and other staff members of the Southwest Washington Health District, Vancouver, for their help with the epidemiologic and environmental investigations; Bill Lawrence, BS, Kaye Pride, BS, and Melanie Panoke of the Washington State Public Health Laboratories, Shoreline, for their PFGE work and testing of environmental isolates; Bala Swaminathan, PhD, William Bibb, PhD, Robert Tauxe, MD, Laura Fehrs, MD, and Melanie Panoke of the Washington State Public Health District, Vancouver, for their help with the epidemiologic testing of environmental isolates; Bala Swaminathan, PhD, William Bibb, PhD, Robert Tauxe, MD, Laura Fehrs, MD, and Melanie Panoke of the Washington State Public Health District, Vancouver.

The use of trade names or commercial sources is for identification only and does not constitute endorsement by the Public Health Service or by the US Department of Health and Human Services.

Corresponding author and reprints: Michael G. Bruce, MD, MPH, Arctic Investigations Program, Centers for Disease Control and Prevention, 4055 Tudor Centre Dr, Anchorage, Alaska 99508 (e-mail: mdmb@cdc.gov).

REFERENCES


