An Outbreak of *Escherichia coli* O157 Infection Following Exposure to a Contaminated Building

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**Context**
Infection with *Escherichia coli* O157 causes an estimated 70,000 diarrheal illnesses per year in the United States and can result in hemolytic-uremic syndrome and death. Environmental contamination with *E coli* O157 may be a public health problem.

**Objectives**
To determine risk factors for *E coli* O157 infection during an outbreak investigation at a county fair and to evaluate environmental contamination as a possible cause of the outbreak.

**Design, Setting, and Participants**
Case-control study of 23 patients (median age, 15 years) and 53 age-matched controls who had attended the Lorain County, Ohio, fair between August 20 and August 26, 2001. Case-patients had laboratory-confirmed *E coli* O157 infection, hemolytic-uremic syndrome, or bloody diarrhea within 7 days of attending the fair; controls attended the fair and did not have diarrhea.

**Main Outcome Measures**
Risk factors for infection and isolates of *E coli* O157 from environmental specimens.

**Results**
Six (26%) case-patients were hospitalized and 2 (9%) developed hemolytic-uremic syndrome. Case-patients were more likely than controls to have visited building A (a multipurpose community facility on the fairgrounds; matched odds ratio [MOR], 21.4 [95% confidence interval {CI}, 2.7-170.7]). Among visitors to building A, illness was independently associated with attending a dance in the building (MOR, 7.5; 95% CI, 1.4-41.2), handling sawdust from the floor (MOR, 4.6; 95% CI, 1.1-20.0), or eating and/or drinking in the building (MOR, 4.5; 95% CI, 1.2-16.6). Twenty-four (44%) of 54 specimens collected from building A 6 weeks after the fair grew Shiga toxin-producing *E coli* O157. Isolates from sawdust, the rafters, and other surfaces were identical by molecular fingerprinting to patient isolates. Sawdust specimens collected 42 weeks after the fair also grew the same *E coli* O157 strain.

**Conclusions**
Absence of evidence implicating specific food or beverage sources and the recovery of *E coli* O157 from the rafters suggest that airborne dispersion of bacteria contributed to the contamination. Because *E coli* O157 can survive in the environment for more than 10 months, humans may be at risk of infection long after an environment is initially contaminated.

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METHODS

In September 2001, Ohio health officials identified a cluster of *E coli* O157 infections in Lorain County, Ohio. The Lorain County General Health District sought cases by telephoning clinical laboratories, hospital infection control practitioners, and health departments in 5 surrounding counties. For surveillance, a case-patient was defined as having diarrheal illness, including laboratory-confirmed *E coli* O157 infection, or HUS occurring after August 15. The Lorain County General Health District interviewed all individuals with laboratory-confirmed infection or HUS, as well as 88 persons who called to report illness. Most ill persons attended the Lorain County fair, which ran from August 20 to August 26, 2001.

We conducted a case-control study of fair attendees to identify risk factors for infection. To reduce the likelihood of including patients with secondary infection or other illnesses, we defined a case-patient for analytic purposes as a person who (1) developed diarrheal illness (≥3 loose stools in a 24-hour period) within 7 days after attending the fair, (2) had laboratory-confirmed *E coli* O157, HUS, or bloody diarrhea, and (3) represented the first diarrheal illness in their household.

Controls were persons who attended the fair and did not have diarrhea between August 20 and September 1, 2001. To reduce the possibility of enrolling individuals with latent infection, we excluded controls if any member of their household had diarrhea during this time interval. Controls were identified using 2 rosters of fair attendees available to the health department: children who entered a bicycle raffle that was held on each day of the fair and members of the local 4-H club. Each name on the list was considered a household; entries for multiple persons from the same household were deleted so that lists included only unique residences. Households were randomly selected for telephone interview. One person per household was used as a control. Controls were age-matched to cases. The goal was to obtain 2 controls per case with 1 control from each roster.

A standardized questionnaire was administered by telephone between September 23 and September 25, 2001. The questionnaire focused on clinical history (ill persons), food and drinks consumed, vendors visited, animal contact, and fair activities. Between October 10 and October 12, 2001, a follow-up questionnaire focusing specifically on activities involving a building on the fairgrounds was administered to persons who completed the first questionnaire.

Fecal specimens from case-patients were sent to the Ohio Department of Health Laboratory for culture. Isolates were confirmed as O157 serologically, tested for Shiga toxin with the use of an immunoassay, and subtyped using standardized pulsed-field gel electrophoresis (PFGE) methods. After immunomagnetic separation and a second enrichment with mTSB at 42°C, followed by immunomagnetic separation, 25 μL of bead-bacteria complex was streaked onto cefixime-tellurite sorbitol-MacConkey agar. All isolates were confirmed serologically with anti-O157 latex reagent. PFGE was performed on selected isolates.

In selected patient and environmental isolates, Shiga-toxin genes were detected by multiplex polymerase chain reaction (PCR) with the use of established primers. A PCR restriction fragment length polymorphism test was used to identify the *H7*-specific allele of the flagellin gene (*fliC*). In univariate analysis, maximum likelihood estimates for the matched odds ratio (MOR) and 95% confidence intervals (CIs) were calculated using the Wald χ² test.

RESULTS

Case finding identified 111 Lorain County residents with diarrheal illness between August 15 and September 15. Twenty-three persons met the case definition and were enrolled. Fourteen (65%) had laboratory-confirmed *E coli* O157 infection, 1 (4%) had HUS, and 8 (35%) had a history of bloody diarrhea. Most case-patients developed illness after the fair ended (FIGURE).

The median age of case-patients was 15 years (range, 1-64 years); 13 (57%) were female. Illness lasted a median of 7 days (range, 3-22 days). All 23 patients reported diarrhea; 19 (86%) had bloody diarrhea; 22 (96%) had abdominal cramps; and 8 (34%) had a fever. Six
(26%) patients were hospitalized, 2 (9%) developed HUS, and no one died.

Of the 14 laboratory-confirmed cases, 12 had culture-confirmed infection and 2 had O157 antigen detected in stool specimens. Of 12 culture-confirmed cases, all were sorbitol nonfermenting *E. coli* O157:nonmotile (NM). Ten shared an indistinguishable PFGE pattern; 2 had different but closely related PFGE patterns. Two patient isolates tested with PCR had Shiga toxin–producing gene sequences (stx1, stx2) and the H7 gene sequence.

We enrolled 53 controls. Eating a hamburger or drinking liquids made from fairground water was not associated with illness (Table 1. Case-patients were less likely than controls to have visited a cow barn, pet or fed a cow, or handled cow manure (matched OR [MOR], 0.3; 95% CI, 0.1-1.8).

Patients were more likely than controls to visit building A, which is a multipurpose community facility located on the fairgrounds (MOR, 21.4; 95% CI, 2.7-170.7). Among persons visiting building A, case-patients were more likely than controls to eat or drink in the building (MOR, 4.5; 95% CI, 1.2-16.6), handled sawdust from the building’s floor (MOR, 4.6; 95% CI, 1.1-20.0), or attended a dance held in the building (MOR, 7.5; 95% CI, 1.4-41.2). Held on the last night of the fair, the dance was attended by approximately 900 persons, mostly aged 10 to 18 years. Some of those who attended the dance complained that air in the building was particularly dusty during the dance. Showing an animal in building A was less common among case-patients than controls (MOR, 0.1; 95% CI, 0.01-0.80).

Building A is a wooden frame structure with open rafters and bleacher seats that surround a central show area. The floor is clay, covered with approximately 2.5 cm to 5 cm of sawdust. The building hosts auctions, exhibits, concerts, dances, and animal shows year-round. Animals never sleep or eat in building A and only spend a few hours in the facility during shows or practice. During the 2001 fair, animal shows involved dairy and beef cattle, sheep, horses, and dogs. A total of 599 cattle were registered at the fair. Cattle were in building A on all days, but no more than 30 were in the facility at a time. No hand-washing facilities are available in or immediately outside the building.

Twenty-four (44%) of 54 specimens taken from building A 6 weeks after the fair ended grew Shiga toxin–producing *E. coli* O157:NM (Table 2). All isolates were sorbitol nonfermenting. PCR demonstrated that isolates carried Shiga toxin–production and *H7* flagella gene sequences. PFGE was performed on 12 (50%) of 24 isolates from building A. All 12 shared a PFGE pattern indistinguishable from the pattern found in 10 patient isolates.

We retested building A 14 weeks and 42 weeks after the fair ended grew Shiga toxin–producing *E. coli* O157:NM, including all 4 sawdust specimens. One (13%) of 8 specimens from a rafter located approximately 8 m above the ground grew Shiga toxin–producing *E. coli* O157:NM with a PFGE pattern that matched the outbreak pattern. Forty-two weeks after the fair ended, all 6 sawdust specimens were positive; 5 (83%) matched the outbreak pattern by PFGE.

Building A was not cleaned until 42 weeks after the fair. At that time, building A’s inside surfaces were disinfected with a cleaning agent (Sani-T-10, Spartan Chemical Co, Maumee, Ohio) approved for use on food contact surfaces. All sawdust was removed and replaced. Concrete walkways were installed. We did not test the building after cleaning. We did not collect specimens from animals exhibited at the fair.

**COMMENT**

We describe the first, to our knowledge, reported outbreak of *E. coli* O157 infections in which both epidemiologic-
cal and microbiological data implicate a contaminated building as the source of infection. It is particularly noteworthy that E. coli O157 survived for 42 weeks in an environment identified as the source of human infections.

We speculate that building A initially became contaminated when at least 1 E. coli O157–shedding animal, probably a cow, defecated on the building’s sawdust. E. coli O157 survived and possibly multiplied in the sawdust.18 The sawdust may have become airborne during a large event such as the dance. Individuals who touched contaminated surfaces in the building became infected when they ate or drank without adequately washing their hands. It is possible that some may have swallowed bacteria that landed directly into their mouths or onto their food or drink. E. coli O157 was probably dispersed through the air. The evidence supporting airborne dispersion includes attendance at the dance as an independent risk factor, anecdotes of dust conditions during the dance, and widespread contamination of the building, including the rafters, which were out of the reach of humans and animals. Washing hands does reduce the risk of infection after contact with contaminated surfaces but would be ineffective if airborne bacteria landed directly into an individual’s mouth or onto food or drink. Direct contact with cows and exhibition of an animal were protective. The protective effect could reflect more careful hygiene among those that handle animals or, more likely, acquired immunity from repeated exposure to E. coli O157. Previous studies suggest that prior exposure may reduce the risk of symptomatic infection.19-21 This finding raises the question of whether selection bias affected our epidemiological study. We do not believe that controls were a biased subset of the fair population because patterns of attendance, food and beverage consumption, non-cattle animal contact, and hand washing were similar across case and control groups.

Options for preventing outbreaks in settings in which the public interacts with E. coli O157–shedding animals are limited. Important research priorities include studying ways to reduce E. coli O157 carriage and shedding in animals, assessing the practicality of installing inorganic ground covers (eg, sand) and redesigning venues to limit public traffic through animal areas, and evaluating the efficacy of surface decontamination in reducing the risk of human infection. For now, fair managers and health officials should educate the public about the hazards of contact with animals and their environments and that such hazards could persist for months after an animal has left an environment. Frequent, meticulous hand washing with soap and water remains the only practice known to reduce the risk of E. coli O157 infection, but further research is needed to understand whether this is sufficient when environmental contamination is widespread and bacteria may be dispersed in the air.22

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REFERENCES


