

The role of heightened surveillance in an outbreak of *Escherichia coli* O157.H7

C. L. ROBERTS^{1,2*}, P. A. MSHAR^{1†}, M. L. CARTTER¹, J. L. HADLER¹,
D. M. SOSIN³, P. S. HAYES⁴ AND T. J. BARRETT⁴

¹Connecticut Department of Public Health and Addiction Services,
Epidemiology Program, Hartford, CT

²Centers for Disease Control, Division of Field Epidemiology,
Epidemic Intelligence Service, Atlanta, GA

³Centers for Disease Control, Division of Field Epidemiology, Atlanta, GA

⁴Centers for Disease Control, Foodborne and Diarrheal Diseases Branch,
Atlanta, GA

(Accepted 22 June 1995)

SUMMARY

After instituting laboratory screening for *Escherichia coli* O157.H7, a Connecticut hospital isolated the organism from four persons in September 1993. As a result, an outbreak of *E. coli* O157.H7 associated with a country club was detected. The club had served hamburger from the same shipment at two picnics. Attendees of two picnics were interviewed, stool cultures were obtained from symptomatic persons, and the remaining hamburger was cultured. Twenty (22%) of 89 persons who ate hamburger became ill, compared with 1 of 60 who did not eat hamburger (relative risk = 13.5, 95% confidence interval 3.2–56.3). Among persons who ate hamburgers, illness was strongly associated with eating hamburger that was not thoroughly cooked ($P < 0.001$). All 20 samples from 5 remaining boxes of patties yielded *E. coli* O157.H7. Isolates from hamburger and case-patients were indistinguishable by pulsed-field gel electrophoresis. Heightened surveillance can rapidly identify outbreaks and may mitigate their impact. However, continued review of food safety issues is necessary if *E. coli* O157.H7 outbreaks are to be prevented.

INTRODUCTION

A decade has passed since the association between outbreaks of *Escherichia coli* O157.H7 and undercooked ground beef was first reported [1]. Outbreaks have continued to occur, and ground beef has remained the most frequently implicated source of infection caused by *E. coli* O157.H7 [2–4]. Outbreaks of *E. coli* O157.H7

* Address correspondence to: Dr Christine Roberts, National Centre for Epidemiology and Population Health, The Australian National University, Canberra, ACT 0200, Australia.

† Address for reprint requests: Pat Mshar, Epidemiology Program, Department of Public Health, 150 Washington St. Hartford CT 06106, USA.

can result in considerable morbidity, including haemorrhagic colitis, haemolytic uremic syndrome (HUS), and thrombocytopenic purpura (TTP) [2]. The case fatality rate in *E. coli* O157.H7 outbreaks has ranged from 0–26% [2].

In September 1993, an outbreak of *E. coli* O157.H7 colitis occurred among attendees of two picnics at a Connecticut country club. The outbreak was caused by hamburger patties that were heavily contaminated with *E. coli* O157.H7. We describe the epidemiologic investigation of the first recognized outbreak of *E. coli* O157.H7 in Connecticut and highlight the importance of laboratory surveillance for *E. coli* O157.H7 and the need for emphasis on prevention.

METHODS

Background

On 14 September 1993, the Connecticut Department of Public Health and Addiction Services (DPHAS) was notified that *E. coli* O157.H7 had been isolated from four persons on the same day by a hospital laboratory. The laboratory had begun screening all unformed stools for *E. coli* O157.H7 in June 1993. Another isolation of *E. coli* O157.H7 was identified by calling other laboratories in the area. Four of these five persons reported eating hamburgers at either of two country club picnics over Labor Day weekend. The picnics took place on Sunday, 5 September 1993, and Monday, 6 September 1993. Hamburgers and salads left over from the Sunday picnic were served on Monday.

Epidemiologic investigation

A case-patient was defined as a person who had three or more loose stools per day for more than 1 day, with onset of diarrhoea after eating at either picnic. Persons with chronic diarrhoea were excluded from analyses.

We identified all persons who attended the picnics by contacting all club members, their guests and staff of the country club attending the picnics. Attendees were interviewed by telephone using a standard questionnaire. Information was obtained about which foods were eaten, how well the meat was cooked (i.e. rare or red in the centre, medium or pink in the centre, or well-done or brown in the centre), symptoms (including onset and duration), physician visits, hospital admission, collection of stool specimens and demographic data.

Data from both picnics were combined for analyses. The χ^2 test or Fisher's exact test were used to determine the association between risk factors and illness. Relative risks [RR] and 95% confidence intervals [CI] were calculated with Statistical Analysis System (SAS) software [5].

Environmental investigation

The kitchen and barbecue areas were inspected, dietary staff were interviewed and food preparation practices were reviewed. All remaining uncooked hamburger patties were collected from the country club for testing. There were no other leftover foods or ingredients from the picnics. The United States Department of Agriculture Food Safety and Inspection Service (USDA-FSIS) attempted to trace the source of the implicated beef.

Laboratory investigation

Stool specimens were collected from case-patients when possible. *E. coli* O157 isolates were sent to the state laboratory for confirmation using slide (O antigen) and tube agglutination (H antigen) [6].

Samples of hamburger patties from each of five remaining boxes collected from the club were tested for *E. coli* O157:H7 by the state laboratory and the Centers for Disease Control and Prevention (CDC) [7]. At the CDC laboratory, hamburger samples were homogenized 1:1 in 0.1% peptone, and the homogenate was directly plated on sorbitol-MacConkey agar (SMAC). The hamburger homogenates were selectively enriched for *E. coli* O157 in dmTSB-CA broth before plating on SMAC [8]. Quantitative estimation of coliforms and *E. coli* in samples from each of five boxes of culture-positive patties and from uncontaminated (control) hamburger from the same beef production plant was done by the 3-tube most probable number technique [9]. The control sample of ground beef and chuck was taken from the production line 2 weeks after notification of the outbreak, on 30 September 1993.

Stool and hamburger isolates of *E. coli* O157:H7 were subtyped by pulsed-field gel electrophoresis of genomic DNA restricted with *Xba* I enzyme [10].

RESULTS

Epidemiologic investigation

One or both picnics were attended by 166 persons, and 162 (98%) were interviewed. Seven persons who did not eat and two persons who had chronic diarrhoea were excluded from analyses. Of the remaining 153 attendees interviewed, 75 (49%) were women. Ages ranged from 1–72 years (median: 34 years). Eighty-three persons ate on Sunday only, 54 persons ate on Monday only, and 16 persons ate at both picnics.

Twenty-three persons met the case definition, for an attack rate of 16%. The attack rate was similar for women (17%) and men (13%) ($\chi^2 = 0.61$, $P = 0.43$). The median age of ill persons was 33 years (range: 1–72 years). The attack rate among children < 5 years of age was 29% (5/17) ($\chi^2 = 3.1$, $P = 0.07$). The attack rate for persons who ate only on Sunday was 13% (11/83), for those who ate only on Monday was 15% (8/54), and for those who ate at both picnics was 25% (4/16) ($\chi^2 = 1.45$ 2 D.F., $P = 0.48$).

Incubation periods were determined by excluding the four case-patients who ate at both picnics. The median incubation period from first exposure was 68 h or 2.8 days (Fig. 1). In addition to diarrhoea, symptoms included abdominal cramps (83%), bloody stools (39%), nausea (30%), fever (26%), headache (26%) and vomiting (10%). The median duration of diarrhoea was 5 days (range: 2–15 days) and the median number of loose stools in a 24-h period was six (range: 3–15). Nine persons (39%) saw a physician, four (17%) were hospitalized, and six (26%) were sufficiently incapacitated to take time off from work or school. Three children who attended the picnic also attended day care, but none of them became ill. No cases of haemolytic uremic syndrome (HUS) or thrombotic thrombocytopenic purpura (TTP) occurred. No deaths were associated with this outbreak.

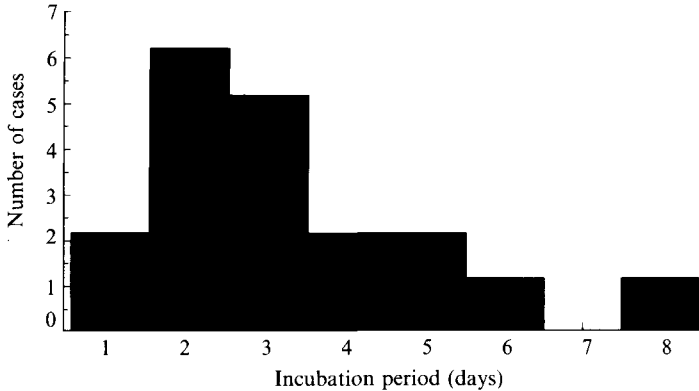


Fig. 1. Incubation period to onset of diarrhoea, *E. coli* O157.H7 outbreak, Connecticut, 1993.

Table 1. Attack rates by reported degree of cooking, *E. coli* O157.H7 outbreak, Connecticut, 1993

Reported degree of cooking	Ill	Total	Attack rate (%)	Relative risk (95% CI)
Well-done	1	35	3	1.0 (Referent)
Medium	9	29	31	10.9 (1.5–80.8)
Rare	9	21	43	15.0 (2.0–110.2)

χ^2 for trend = 13.1. $P < 0.001$.

Food-specific attack rates for foods served at both picnics indicated a substantially increased risk of illness among persons who ate hamburgers. Twenty (22%) of 89 persons who ate hamburger became ill, compared with 1 of 60 persons who did not eat hamburger (RR = 13.5, 95% CI = 3.2–56.3). Four persons, including two who were ill, could not recall whether they had eaten a hamburger. The one person who became ill but did not eat a hamburger became ill only 3 h after the picnic; a stool culture was not obtained from this person. Among those who ate hamburgers, risk increased as a function of under-cooking (Table 1). Compared with persons who ate well-done hamburgers, those eating medium hamburgers were 11 times more likely to become ill and those eating rare hamburgers were 15 times more likely to become ill. No other foods were implicated as a possible source of the outbreak.

Environmental investigation

No illness was reported by the dietary staff, which consisted of four foodhandlers and one dishwasher. Specific information on duration and temperature of cooking the hamburgers could not be obtained. The hamburger patties were removed from the freezer just before the picnic began and placed in chafing dishes over ice until they were cooked. According to the chef, some hamburger patties were still frozen in the centre at the start of the barbecue and may not have been cooked thoroughly. The chef also reported that some people requested rare hamburgers.

Table 2. Quantification testing of implicated and control hamburger. *E. coli* O157.H7 outbreak, Connecticut, 1993

Quantification test*	Implicated hamburger samples (n = 5)	Control hamburger sample (n = 1)
Total plate count (CFU/g)	4.5–28 × 10 ⁵	6.2 × 10 ⁵
Total coliforms (MPN/g)	230–1500	< 3
Sorbitol negative coliforms (MPN/g)	90–930	< 3
<i>E. coli</i> O157 (MPN/g)	40–930	< 3
<i>E. coli</i> O157/5 oz patty	0.6–12.5 × 10 ⁴	< 3

* (CFU, colony forming units; MPN, most probable number (of organisms)).

The club stopped using hamburger patties from the suspect shipment on Sunday, 12 September after some members reported illness that they attributed to eating at the Labor Day picnics. All remaining frozen raw patties (approximately 45 pounds), were submitted to the state laboratory. No cooked hamburger patties were available for testing.

The hamburger patties were produced and distributed (refrigerated, not frozen) by a local ground beef production plant. The fresh chuck came via a New York distributor from one of two large mid-western beef packing companies. The chuck could have originated from any of six large slaughter houses, owned by either of the two packing companies.

The ground beef from the shipment of hamburger patties supplied to the country club on 2 September was also distributed to nine other local establishments in three Connecticut counties. None of these establishments had any of the ground beef or hamburger patties remaining. Seven cases of *E. coli* O157.H7 infection were reported from these counties for September 1993, and five of these were cases in this outbreak. We could not establish any association between the remaining two reported infections and any of the establishments that received the implicated ground beef. Isolates from these two persons were not available for further testing.

Laboratory investigation

Of the 23 persons meeting the case definition, 10 had stool cultures collected. Five of these cultures were positive for *E. coli* O157.H7. All five positive specimens were collected as part of an initial diagnostic work-up in a physician's office. None of the four specimens collected by investigators was positive. All five persons with positive isolates had bloody diarrhoea. Of the five persons who had negative cultures, three had bloody diarrhoea and four had specimens collected ≥ 10 days after symptom onset.

All 20 samples of hamburger patties tested yielded *E. coli* O157.H7. Total plate counts were similar for the implicated and control hamburger patties (Table 2). The numbers of *E. coli* O157.H7 in the five hamburger samples tested ranged from 40–930 organisms per gram, or 5600–125000 per 142 gram (5 ounce) hamburger patty. All isolates from case-patients were indistinguishable from the hamburger isolates by PFGE.

DISCUSSION

The epidemiologic, laboratory and environmental data provide conclusive evidence that contaminated hamburger was the source of this *E. coli* O157.H7 outbreak. Not only was there significant risk associated with eating hamburger, especially rare hamburger, but all the hamburger patties tested yielded high levels of *E. coli* O157.H7, and hamburger and human isolates were indistinguishable by PFGE. Other investigations indicate that contaminated ground beef products have been the most frequently implicated source of outbreaks of *E. coli* O157.H7 [2]. However, these investigations have been less successful in demonstrating contamination of the implicated ground beef samples [1, 11, 12].

Only one other investigation has quantified the concentration of *E. coli* O157.H7 in the implicated hamburger [3, 13]. In the 1993 outbreak in the western United States, *E. coli* O157.H7 was grown from some, but not all, of the implicated hamburger samples. Although the highest reported number of organisms per gram was 15, most of the contaminated hamburger samples had only 1–4 organisms per gram, suggesting a small inoculum is capable of causing illness in susceptible persons [13]. Our findings of 40–930 organisms per gram, and the contamination of all hamburger patties tested, suggest that the hamburger in this outbreak was more heavily contaminated than the hamburger in the western US outbreak.

This was the first outbreak of *E. coli* O157.H7 infections identified in Connecticut. Without routine laboratory screening this outbreak may not have been detected by public health authorities. The likelihood of *E. coli* O157.H7 detection increased markedly in Connecticut in 1993 because of attention given to the western US outbreak and because DPHAS requested laboratories to screen at least all bloody stools for *E. coli* O157.H7 [14]. By November 1993, 91% of Connecticut laboratories that perform on-site stool testing were routinely screening at least bloody stools for *E. coli* O157.H7 compared with 16% in April 1993 [14]. Routine laboratory screening can facilitate more timely public health investigation and institution of control measures, as well as prevent unnecessary diagnostic investigation of patients (e.g. barium enema, colonoscopy and laparotomy) to determine the cause of intestinal illness.

The DPHAS was notified of this outbreak 9 days after the first picnic, at the time of the first positive stool cultures. The detection and investigation of this outbreak began as rapidly as possible. However, even a rapid response was too late for additional public health action because the country club had stopped serving the implicated hamburger patties, and subsequent USDA-FSIS recall of the implicated shipment found that the remaining hamburger had been consumed. The lack of opportunity for health authorities to prevent consumption of contaminated meat emphasizes the importance of measures to eliminate *E. coli* O157.H7 contamination of food products prior to distribution to consumers.

The hamburger served at the country club was approved for consumption by the USDA. To date, approval has been based primarily on visual inspection [13]. No government microbiological food safety standard for *E. coli* O157.H7 currently exists [15]. However, in September 1994, USDA-FSIS declared that 'we consider raw ground beef that is contaminated with *E. coli* O157.H7 to be adulterated

within the meaning of the Federal Meat Inspection Act' and announced a programme of targeted sampling and testing of raw ground beef which commenced on 17 October 1994 [16, 17]. USDS-FSIS also recognizes that the ultimate solution to the O157.H7 problem lies in preventive measures rather than end-product testing [16].

Primary prevention (which requires no action by the consumer) could be achieved by ensuring that distributed foods are free from *E. coli* O157.H7 contamination. In response to the western US outbreak, the USDA-FSIS has implemented a programme to address such issues as which animals and animal husbandry practices are associated with *E. coli* O157.H7 and which slaughtering and processing practices result in contaminated meat [13]. Precooking sterilization, such as irradiation, is another way of reducing exposure to contaminated meat [18]. Secondary prevention by thoroughly cooking ground beef will also eliminate *E. coli* O157.H7 [19]. However, measures such as mandated labelling and recommendations for cooking ground beef [20] have not prevented further outbreaks from occurring because of lack of compliance by consumers [4, 21, 22].

Heightened surveillance using routine laboratory screening for *E. coli* O157.H7 allowed for the rapid detection and investigation of an outbreak of illness associated with hamburger. However, continued review of food safety issues is necessary if *E. coli* outbreaks are to be prevented.

ACKNOWLEDGEMENTS

We thank Patricia Griffin, MD, and Frederick Angulo, PhD for encouragement, advice and assistance during this investigation. We also acknowledge the support of Dr Bala Swaminathan and Mary Ann Lambert-Fair (Foodborne and Diarrheal Diseases Branch, CDC), and Thomas Furgalack and Roger Mshar (Food Protection, DPHAS).

REFERENCES

1. Riley LW, Remis RS, Helgersen SD, et al. Hemorrhagic colitis associated with a rare *Escherichia coli* serotype. *New Engl J Med* 1983; **308**: 681–5.
2. Griffin PM, Tauxe RV. The epidemiology of infections caused by *Escherichia coli* O157.H7, other enterohemorrhagic *E. coli*, and the associated hemolytic uremic syndrome. *Epidemiol Rev* 1991; **13**: 60–98.
3. Bell BP, Goldoft M, Griffin PM, et al. A multistate outbreak of *Escherichia coli* O157.H7-associated bloody diarrhea and hemolytic uremic syndrome from hamburgers. *JAMA* 1994; **272**: 1349–53.
4. CDC. *Escherichia coli* O157.H7 outbreak linked to home-cooked hamburger – California, July 1993. *MMWR* 1994; **43**: 213–5.
5. SAS Version 6.04 [Computer Program]. Cary, NC, SAS Institute Inc., 1991.
6. Balows A, Hausler WJ, Herrmann KL, et al, eds. *Manual of clinical microbiology*, 5th ed. Washington DC: American Society of Microbiology, 1991.
7. FDA. Isolation methods for enterohemorrhagic *E. coli* (EHEC). In: *Food and Drug Administration – bacteriological analytic manual*, 7th ed. Arlington: AOAC International, 1992: 44.
8. Padhye NV, Doyle MP. Rapid procedure for detecting enterohemorrhagic *Escherichia coli* O157.H7 in food. *Appl Environ Microbiol* 1991; **57**: 2693–8.
9. Van der Zant C, Splittstoesser D, eds. *Compendium of methods for the microbiological examination of foods*, 3rd ed. Washington DC: American Public Health Association, 1992.

10. Barrett TJ, Lior H, Green JH, et al. Laboratory investigation of a multistate foodborne outbreak of *Escherichia coli* O157.H7 using pulsed-field gel electrophoresis and phage typing. *J Clin Microbiol* 1994; **32**: 3013–7.
11. Ostroff SM, Griffin PM, Tauxe RV, et al. A statewide outbreak of *Escherichia coli* O157.H7 infections in Washington state. *Am J Epidemiol* 1990; **132**: 239–47.
12. Ryan CA, Tauxe RV, Hisek GW, et al. *Escherichia coli* O157.H7 diarrhea in a nursing home: clinical, epidemiological, and pathological findings. *J Infect Dis* 1986; **154**: 631–8.
13. United States Department of Agriculture. Food and Safety Inspection Service. Report on the *Escherichia coli* O157.H7 outbreak in the western states. FDA Prime Connections, June 1993: 1L93-11.
14. CDC. Laboratory screening for *Escherichia coli* O157.H7. Connecticut 1993. *MMWR* 1993; **43**: 192–4.
15. Snyder OP. Hazard analysis and critical control points: An industry self-control program. Part III. Dairy, Food and Sanitation, March 1992: 164–7.
16. Taylor MR. Change and opportunity: harnessing innovation to improve the safety of the food supply. Presented at 1994 American Meat Institute Annual Convention, San Francisco, 29 September 1994.
17. Microbiological testing program for *Escherichia coli* O157.H7 in raw ground beef. Washington DC: US Department of Agriculture – Food Safety and Inspection Service, FSIS Notice 50-94, 23 December 1994.
18. Kaferstein FK, Moy GC. Public health aspects of food irradiation. *J Public Health Policy* 1993; **14**: 149–62.
19. Line JE, Fain AR, Moran AB, et al. Lethality of heat to *Escherichia coli* O157.H7: D-value and Z-value determinations in ground beef. *J Food Protection* 1991; **54**: 762–6.
20. Public Health Service. Food Code: 1993 Recommendations of the United States Public Health Service/Food and Drug Administration. Washington DC: US Department of Health and Human Services, Public Health Service 1993: Report no. PB94-113941AS.
21. Spencer-Molloy F. Bacteria make 12 people ill – State seeking source of *E. coli*. *The Hartford Courant* 1994: Sect. C1 (col. 6).
22. Levy CJ. 7 reported ill in New Jersey from eating tainted meat. *The New York Times* 1994: Sect. B4 (col. 6).