

Swimming-associated outbreak of *Escherichia coli* O157:H7

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SUMMARY

In 1997 the first outbreak of *Escherichia coli* O157:H7 infections involving 14 cases occurred in Finland. A case was defined as a resident of Alavus with an episode of diarrhoea between 5 and 17 July 1997, and from whom *E. coli* O157:H7 was isolated from stool. The investigation included case searching and a population-based case control study. Five primary and eight symptomatic secondary cases of *E. coli* O157:H7 illness were detected. In the 10 days before the outbreak, all 5 primary patients (aged 3–8 years), but only 6 of 32 population controls from the same age range (Fisher's test, $P < 0.001$) and 4 of 10 sibling controls ($P < 0.05$) had visited (but had not necessarily bathed in) a shallow beach popular among young children. Four out of 5 primary cases had remained within 5 m of the beach while swimming and had swallowed lake water compared to 1 of 5 population controls. These analytical epidemiologic findings incriminated fresh lake water as the vehicle of *E. coli* O157:H7 transmission.

INTRODUCTION

Escherichia coli O157:H7 outbreaks have frequently been associated with the consumption of contaminated food, particularly ground meat [1]. Person-to-person transmission is also well described [1]. Waterborne outbreaks have been less commonly reported amongst children swimming in paddling pools [2, 3] and lakes [4–7].

Finland has had few sporadic cases of domestically acquired enterohaemorrhagic *E. coli* O157:H7 infection until recently. An enhanced laboratory-based

national survey between February 1996 and January 1997 detected three such infections, and few sporadic patients with *E. coli* O157:H7 had been diagnosed before 1996. Another three sporadic cases were detected in April and May 1997. When omitting the cases of the present outbreak the cumulative number of *E. coli* O157:H7 infections diagnosed in Finland until the end of 1997 was 62, and it now seems that some level of endemicity has been established by sporadic cases of mostly unknown sources.

On 5 July 1997, 7 children with diarrhoea (5 from Alavus and 2 from Kurikka, a town 60 km away) were referred to the clinicians of the Central Hospital of Seinäjoki. The onset of Kurikka cases was 3–5 days

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prior to the Alavus cases. All presented with bloody diarrhoea from which *E. coli* O157:H7 was subsequently isolated. An investigation was conducted to determine the vehicle and source of the Alavus outbreak.

METHODS

Background of the outbreak

Alavus is a rural municipality in central Finland with a population of approx. 10 000. The Ryskööt fair, which drew 2000–3000 visitors and many vendors from the surrounding area, took place on 27–29 June 1997 in the centre of Alavus. During the fair six temporary closed system public lavatories were provided to the visitors. The local lake, bordering the town centre, is about 2 km long and 0.5 km wide. Tusa beach is very popular among young children as it is shallow and has many small, still pockets close to the beach line. There is farmland nearby, with grazing cattle, but physical barriers and buffer zones have been created to prevent direct faecal contamination of lake-water from cattle. Tusa beach is about 200 m from the Ryskööt fair main area, and many attendees visited the beach during the fair. Heavy rainfall occurred 2–3 days before the fair after a month-long dry period, resulting in some land erosion. Two days after the Ryskööt fair, a heat wave occurred, lasting for 3 days (Fig. 1). This ended with local thunderstorms and heavy rainfall on 4 July.

Case definition and findings

A case was defined as a person living in Alavus with an episode of diarrhoea starting between 5 and 17 July 1997, from whom *E. coli* O157:H7 was isolated from the stool. A case was defined as primary if there was no close contact with another case in the 2–10 days prior to symptom onset and secondary if there was close contact with another case during this period. As part of case findings, local physicians in the health care centre were advised to culture stool from all patients with diarrhoea and from all close contacts of *E. coli* O157:H7 patients, irrespective of whether they had symptoms. Demographic and clinical information was collected from all cases. The household secondary attack rate was calculated by dividing the total number of secondary cases by the total number of household contacts.

Case control studies

Two consecutive case control studies were conducted. In the first, two sets of controls were selected. Sibling controls were culture negative brothers and sisters of five primary cases. Population controls were a systematic sample of age group matched healthy children and their siblings drawn from Health Centre cards of the three administrative units of Alavus stratified according to population size.

A self-administered questionnaire for use by the parents of the primary cases was developed after in-depth questioning of the parents of two primary cases [8]. The questionnaire explored potential exposures in the 10 days before disease onset, such as consumption of selected foods, water drinking habits (tap water, or own well), and participation in activities at the festival, including specific enquiry about swimming in local lakes.

After identifying a common exposure (the beach at Tusa), a new round of questions was conducted by telephone among parents of cases and population controls (these were not cultured, but none had close contact with cases) who had visited Tusa beach within the 10 days prior to the outbreak to establish differences in risk behaviour. Exact beach visit dates were asked to determine the period of transmission.

Microbiological methods

Preliminary tests were carried out at the microbiological laboratory of the central hospital by culturing of faecal samples on sorbitol–MacConkey (SMAC) agar plates for sorbitol negative *E. coli* O157:H7. All positive samples were sent for confirmation at the Laboratory of Enteric Pathogens, Helsinki using PCR for bacteria carrying genes encoding Shiga-toxin 1 (Stx1) and/or (Stx2) [9]. The specific colony giving PCR-positive result was subsequently isolated and identified by standard methods. The O and H antigens of the strains were also assayed using standard methods [10].

Environmental investigations

Various food specimens taken from the local shops and from patient homes and faeces of cattle grazing in the vicinity of the beach and lake water were cultured for *E. coli* O157:H7 using enrichment in modified tryptone soya broth (MTSB) and SMAC supple-

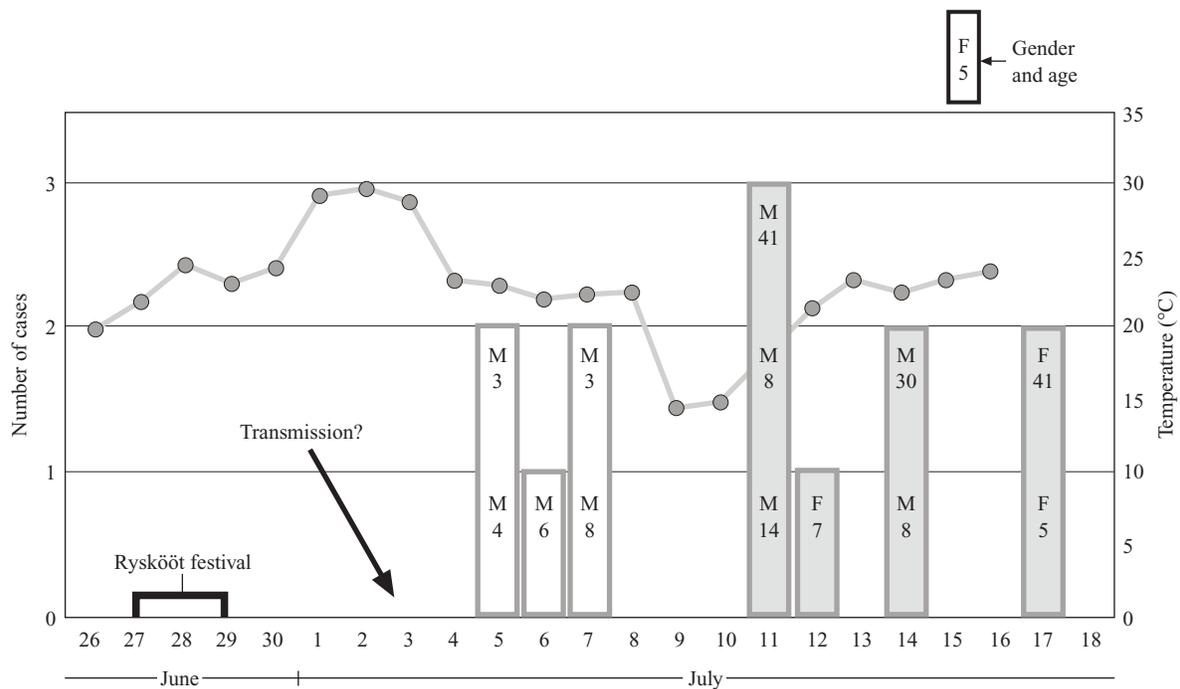


Fig. 1. Number of microbiologically confirmed cases of *E. coli* O157 by date of onset of first symptom, in Alavus, June–July 1997 (13 symptomatic cases). ■, Secondary case; □, primary case.

mented with cefixime and tellurite (CT) at the National Veterinary and Food Research Institute. Lake water was tested for faecal coliforms and faecal enterococci by standard methods at the Municipal Food and Water Research Laboratory [11].

Statistical methods

Differences in exposure frequencies of cases and controls were compared using Fisher's exact test [12].

RESULTS

Descriptive epidemiology

We identified 13 *E. coli* O157:H7 cases with bloody diarrhoea (attack rate 14.0/10000) in Alavus. Five primary cases fell ill between 5 and 7 July (Fig. 1). They came from different families – four from the central district, and one from the northernmost administrative district. All five cases from Alavus were male, aged 3–8 years. Eight more cases occurred 6–10 days after the initial onset of symptoms of the primary cases and *E. coli* O157:H7 was also detected from one asymptomatic person through screening of family contacts. The secondary cases were aged 9–41 years and five were male. Seven of these secondary cases and the asymptomatic person (not included in

Fig. 1) were siblings or parents of a primary case living in the same house. One had contact with a case in a day care unit and the 15th (not included in Fig. 1) case was a nurse treating cases in the Central Hospital of Seinäjoki.

Case control studies

The response rate among sibling controls was 83% (10/12) and among all population controls and their siblings irrespective of age was 82% (67/82). There were 32 population controls aged 3–8 years. All five primary cases reported visiting the local beach at Tusa and there were no other common exposures. Six of 32 population controls aged 3–8 years (OR undefined, Fisher's $P < 0.001$), and 4 of 10 of sibling controls (OR undefined, Fisher's $P < 0.05$) had visited Tusa beach. Visiting the beach does not automatically mean that the control swam in the lake.

In the second round of questioning among primary cases and population controls who had reported visiting Tusa beach, 5/5 primary cases and 5/5 controls were interviewed by telephone (one control was discarded as his visit to Tusa beach was before the period 27 June to 3 July). The parents of four of the five primary cases reported that their children had swallowed lake water and swum less than 5 m from the water-line and three had swum in small, warm,

still and sequestered ponds of the lake water near the water line. Only one population control had any of these exposures during this period, but differences were not statistically significant.

All but one primary case reported visiting the beach on Thursday 3 July, and in one case that was the only day. A subsequent in-depth interview with the father of the only primary case who initially reported not visiting the beach on this date, revealed he had visited the beach on 2 or 3 July. Thus, the most probable transmission time window was Thursday 3 July (Fig. 1). The incubation period of primary cases was thus 2–4 days. When assuming that the population control series gives a reasonable percentage estimate of 3–8-year-old Tusa beach visitors in Alavus before the outbreak, the *E. coli* O157:H7 attack rate among the same age range would be *c.* 7%.

Secondary cases occurred 6–10 days after the first primary case fell ill. The secondary attack rate (including the asymptomatic person) within families was 32%, but this was strongly influenced by one household where 5 of 6 contacts fell sick.

Microbiological investigations

All isolates of *E. coli* O157:H7 carried genes for Stx2, but not for Stx1. *E. coli* O157:H7 was not isolated from the swimming water (sample was taken 16 July from Tusa) nor in any of the food or animal samples. The number of indicator microbes (heat resistant faecal coliforms) in the Tusa beach swimming water on 4 June and 1 July were 2 and 11 per 100 ml respectively, compatible with high quality (Council of European Communities – Directive on water quality 76/160/EEC/1975) of swimming water. Routine samples, as well as the sample for *E. coli* O157:H7 were taken several metres from the beach line at 0.3 m depth.

DISCUSSION

We describe an outbreak of *E. coli* O157:H7 where fresh lake water was incriminated epidemiologically as the most likely transmission vehicle for five primary cases. All the primary cases were young boys in whom swimming skills were not well developed. Swimming in and swallowing lake water may have been the mechanism of transmission, as a significantly smaller proportion of population controls had visited Tusa beach within the 10 days prior to the outbreak compared to primary cases. In addition, primary cases

more frequently reported activity compatible with increased exposure – swallowing water, swimming in shallow water or in small still pockets with stagnant water near the beach line.

The Ryskööt festival crowding the Tusa beach during hot summer days probably resulted in human faecal contamination of fresh lake water with *E. coli* O157:H7. Human contamination has also been suspected in previous reported outbreaks [2, 4]. This is supported by the lack of isolation of *E. coli* O157:H7 from animal and food samples. The probable 2–4-day incubation period is compatible with the previous literature [1, 4, 5, 7]. The tight clustering of the five primary cases supports the idea that the transmission was not sustained, unlike the two USA outbreaks [4, 7]. The lake water in Alavus was probably an effective vehicle for only a short period of time, although *E. coli* O157:H7 has been shown to survive for several weeks in water [13]. Interestingly, transmission seems to have occurred at the end of the heat wave. Perhaps the most important reason for the short transmission window was the observation that bathing activities more or less ceased for quite a while after the air cooled down and the heavy rains exchanged the water in the lake and in the ponds.

Remarkably similar conditions have preceded the three well analysed outbreaks reported in the USA in which lake water was incriminated as the vehicle for *E. coli* O157:H7 transmission [4, 5, 7]. The USA outbreaks also all occurred in July like the present outbreak. These outbreaks and a meticulously analysed Dutch case series [6], in which bathing in a lake was the only common exposure of four girls with haemolytic uraemic syndrome, have all occurred during hot summer days with high water temperature. In all of these outbreaks the beaches had unusually large numbers of bathers in small lakes on a temporary basis. As in this outbreak, none demonstrated *E. coli* O157:H7 in the water [4–7]. The Centre for Disease Control and Prevention has data for an additional four unpublished swimming-associated outbreaks in the USA [14]. The Alavus outbreak, to our knowledge, is the first *E. coli* O157:H7 outbreak in Europe where lake water has been analytically incriminated as the vehicle of transmission.

The 9 secondary cases in Alavus and 1 nurse infected in the Central Hospital of Seinäjoki followed 5 primary cases and seem to indicate high person-to-person transmission in this outbreak (household secondary attack rate 32%), although many of the secondary cases occurred in one household. In the

lake-borne outbreak in Oregon State, USA [4] 7 secondary cases followed 21 primary cases, and in a small outbreak of 2 children possibly infected from a paddling pool, 4 secondary cases were observed [2].

Isolation of *E. coli* O157:H7 requires special techniques in sample preparation and for the microbiological diagnosis [15, 16]. In addition, the intermittent nature of *E. coli* O157:H7 contamination [16] further makes routine use of the microbiological laboratory and for environmental monitoring very difficult. The normal results of routine bacteriological indicators of faecal contamination in Alavus suggest that they provided little help as markers to determine the existence of *E. coli* O157:H7.

Swimming-associated transmission of *E. coli* O157:H7 in fresh water is becoming well established [4–7]. There are thousands of public beaches on lakes in Finland in which hundreds of thousands of Finns bathe frequently during summers. The unusual circumstances, the public fair in a place adjacent to a public swimming bath and the unusually high temperatures probably contributed to the relatively high risk of *E. coli* O157:H7 infection observed during this outbreak. A careful monitoring of the epidemiological situation will indicate whether general recommendations or restrictions must be applied in Finland and other countries with similar swimming conditions.

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REFERENCES

- Griffin PM, Tauxe RV. The epidemiology of infections caused by *Escherichia coli* O157:H7, other enterohemorrhagic *E. coli*, and the associated hemolytic uremic syndrome. *Am J Epidemiol* 1991; **13**: 60–99.
- Brewster DH, Brown MI, Robertson D, Houghton GI, Bimson JJ, Sharp JCM. An outbreak of *E. coli* O157:H7 associated with a children's paddling pool. *Epidemiol Infect* 1994; **112**: 441–7.
- Hildebrand JM, Maguire HC, Holliman RE, Kangesu E. An outbreak of *E. coli* O157:H7 infection linked to paddling pools. *CDR Rev* 1996; **6**: R33–6.
- Keene WE, McAnulty JM, Hoesly FC, et al. A swimming-associated outbreak of hemorrhagic colitis caused by *E. coli* O157:H7 and *Shigella sonnei*. *N Engl J Med* 1994; **331**: 579–84.
- CDC. Lake-associated outbreak of *E. coli* O157:H7 – Illinois, 1995. *MMWR* 1996; **45**: 437–9.
- Cransberg K, van den Kerkhof JHCT, Bänffer JRJ, et al. Four cases of hemolytic uremic syndrome-source contaminated swimming water? *Clin Nephrol* 1996; **46**: 45–9.
- Ackman D, Marks S, Mack P, Caldwell M, Root T. Swimming-associated haemorrhagic colitis due to *E. coli* O157:H7 infection: evidence of prolonged contamination of a fresh water lake. *Epidemiol Infect* 1997; **119**: 1–8.
- World Health Organization, Food and Safety Unit. Programme of Food Safety and Food Aid, Consultations and Workshops. Prevention and Control of Enterohaemorrhagic *Escherichia coli* (EHEC) Infections. Outbreak investigation. Report of a WHO Consultation Geneva, Switzerland 28 April–1 May 1997.
- Alsvik O, Strockbine NA, PCR detection of heat-stable, heat-labile and Shiga-like toxin genes in *Escherichia coli*. In: Persing DH, Smith TH, Tenover F, White TJ, eds. Diagnostic molecular microbiology: principles and applications. Washington DC: American Society for Microbiology, 1993: 271–6.
- Keskimäki M, Ikäheimo R, Kärkkäinen P, Scheutz F, Puohiniemi RL, Siitonen A. Shiga toxin-producing *Escherichia coli* serotype OX3:H21 causing haemolytic-uraemic syndrome. *Clin Infect Dis* 1997; **24**: 1278–9.
- Finnish Society for Standardization. Membrane filter technique for the enumeration of thermotolerant (fecal) coliform bacteria in water. Standard No. SFS 4088, 3rd edn. Helsinki: Board of Water and Environment, 1988.
- Siegel S, Castellan NJ Jr. Two independent samples. In: Nonparametric statistics for the behavioral sciences, 2nd edn. Singapore: McGraw-Hill Book Company, 1989.
- Rise EW, Johnson CH, Wild DK, Reasoner DJ. Survival of *E. coli* O157:H7 in drinking water associated with a waterborne disease outbreak of hemorrhagic colitis. *Lett Appl Microbiol* 1992; **15**: 38–40.
- Wachsmuth IK, Sparling PH, Barrett TJ, Potter ME. Enterohemorrhagic *Escherichia coli* in the United States. *FEMS Immunol Med Microbiol* 1997; **18**: 233–9.
- Armstrong GL, Hollingsworth J, Morris G. Emerging foodborne pathogens: *E. coli* O157:H7 as a model of entry of new pathogens into the food supply of the developed world. *Epidemiol Rev* 1996; **18**: 29–51.
- Jackson SG, Goodbrand RB, Johnson RP, et al. *E. coli* O157:H7 diarrhoea associated with well water and infected cattle on an Ontario farm. *Epidemiol Infect* 1998; **120**: 17–20.