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A christening party outbreak of haemorrhagic colitis and haemolytic uraemic syndrome associated with *Escherichia coli* O 157.H7

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SUMMARY

A point source outbreak of haemorrhagic colitis due to *Escherichia coli* O 157.H7 producing verocytotoxin (VT), took place following a christening party in Birmingham in June 1987. Twenty-six people were affected, six were admitted to hospital and one developed haemolytic uraemic syndrome: there were no deaths. VT+ *E. coli* O 157.H7 was isolated from 13 (57%) of 23 faecal specimens from affected people and from 3 (9%) of 33 specimens from asymptomatic people. Free VT was detected in the faeces of one further asymptomatic person. Illness was associated with eating turkey-roll sandwiches ($P < 0{\cdot}001$) suggesting that cold meats might be an important source of infection.

INTRODUCTION

Verocytotoxin-producing strains of *Escherichia coli* (VT+ *E. coli*) of serotype O 157.H7 were first shown as the cause of haemorrhagic colitis (HC) in the United States in 1982 (1). Subsequently, outbreaks and sporadic cases have been reported from North America (2–7) and Japan (8). Some cases have developed haemolytic uraemic syndrome (HUS) (3–5, 9). In England and Wales sporadic cases of HUS (10) and HC (8) and one outbreak of HC in East Anglia in 1985 (11) due to *E. coli* O 157.H7 have been reported. *E. coli* O 157.H7 was retrospectively implicated as the cause of widespread outbreaks of HUS in children in the West Midlands in 1982 and 1983 (12, 13). We describe the first point source outbreak of HC due to *E. coli* O 157.H7 in the UK.

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PATIENTS AND METHODS

The outbreak

Three adults who attended a reception held at a public house in Birmingham on 28 June 1987, were admitted with acute onset bloody diarrhoea to the Infectious Diseases Unit at East Birmingham Hospital between 1 and 3 July 1987. Preliminary inquiries of these patients and their visitors confirmed that other reception guests, which included a child admitted to The Childrens' Hospital, Birmingham, had suffered from diarrhoea. The reception, attended by 93 people from a close-knit community within Birmingham, was provided with a 'finger buffet' at which some 15 cold items were available.

Epidemiological investigations

Attenders at the function were interviewed using a standard questionnaire, to determine symptoms, dates of illness, and what they ate and drank at the reception. Clinicians and laboratories in the West Midlands were contacted to identify additional cases unconnected with the function but none were found. Presumptive cases were individuals with diarrhoea (three or more motions per day) or with frank blood in their stool. Risk factors were analysed using the Mantel–Haenzsel formulation of the χ^2 test (14).

Laboratory investigation

Faecal specimens were requested from all attenders. Faeces were cultured for *Campylobacter*, *Salmonella* and *Shigella* spp. by conventional methods and were also cultured on sorbitol-MacConkey agar. Those colonies which were non-sorbitol fermenters were further tested with a specific antiserum for *E. coli* O 157. From the platings of each faecal sample several hundred well separated colonies were transferred onto nylon membranes and these were tested in DNA hybridization experiments with probes for VT1 and VT2 genes (15). Up to five probe-positive colonies from each faecal sample were identified biochemically, serotyped and examined for verocytotoxin production (10). The presence of free neutralizable verocytotoxin in faecal specimens was examined as described by Scotland and colleagues (10).

When possible, 25 g samples of left-over buffet food, as well as suspect foods subsequently bought in from the supermarket were macerated and cultured in 'MacConkey broth purple' (Oxoid): these were subsequently sub-cultured onto MacConkey's agar and colonies from these sub-cultures were then transferred onto nylon membranes and tested as above for VT1 and VT2 genes.

RESULTS

Of the 93 people who attended the reception, 83 (89%) were interviewed (mean age 25 years, range 6 months–71 years). Twenty-six (28%) were presumptive cases; six were admitted to hospital and one woman aged 35 years developed HUS one week after the onset of severe haemorrhagic colitis.

Twenty-three presumptive cases and 33 well guests submitted faecal specimens. VT+ *E. coli* O 157.H7 was isolated from faeces of 13 presumptive cases compared with only 3 well guests (in addition free neutralizable verocytotoxin was identified

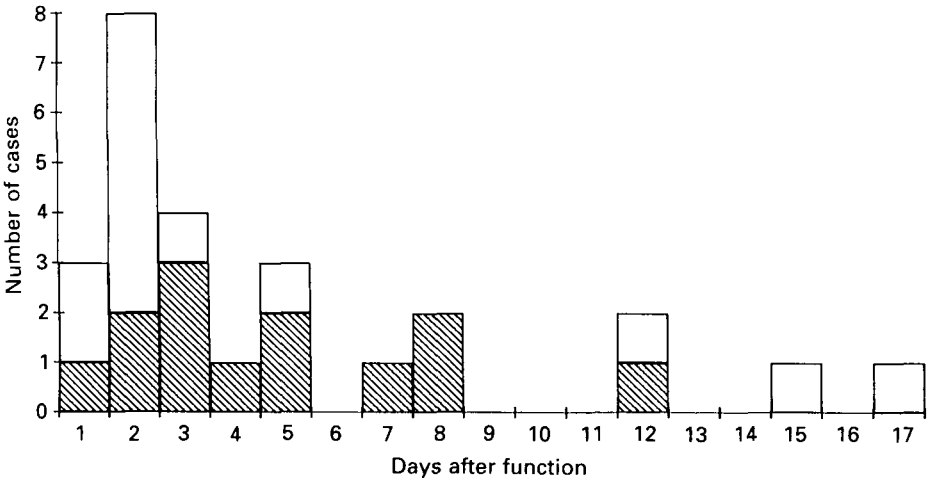

Fig. 1. Onset of illness of microbiologically confirmed (▨) and presumptive (□) cases.

in the faeces of one other well guest) ($\chi^2 = 10{\cdot}4$, $P < 0{\cdot}001$). Of the 17 individuals with confirmed infection 12 suffered diarrhoea; 11 abdominal pain; 8 blood in faeces; 3 feverishness and 4 were asymptomatic. Eight individuals had abdominal pain and bloody diarrhoea. VT+ *E. coli* infection was not confirmed in the patient with HUS.

Faeces samples were obtained a median of 15 days (range 4–23 days) after the reception. Faeces samples from which VT+ *E. coli* O 157. H7 was identified or free verocytotoxin was present were submitted at a median of 11 days (range 8–16 days) compared with 16 days (range 4–23 days) for other persons (χ^2 for difference in distribution of groups about the median = 9·3, $P < 0{\cdot}001$).

The epidemic curve (Fig. 1) was consistent with a point source outbreak with a modal interval from exposure to onset of illness of 2 days (median 3 days: range 1–15 days). No secondary household cases in people who had not been at the reception were identified. However, all microbiologically confirmed cases and 4 of 7 presumptive but not confirmed cases, occurring more than 5 days after the reception were in household contacts of earlier microbiologically confirmed cases and may have been due to secondary spread. The age and sex distribution of cases and well persons was similar.

Clinical illness was associated (Table 1) with eating turkey-roll sandwiches ($P < 0{\cdot}001$), tinned frankfurter sausages ($P < 0{\cdot}025$) and pork pie ($P < 0{\cdot}05$). Stratification of consumption of turkey-roll sandwiches by consumption of pork pie and, or tinned sausages showed an independent association of turkey-roll sandwiches and illness ($\chi^2 = 6{\cdot}2$, $P < 0{\cdot}025$). Consumption of tinned sausages and pork pie were not significantly associated with illness when stratified by turkey-roll consumption ($P > 0{\cdot}1$ for both). Seven of 17 microbiologically confirmed people with infection ate turkey-roll sandwiches compared with 3 of 38 microbiologically negative people ($\chi^2 = 6{\cdot}5$, $P < 0{\cdot}025$). In people who did not report eating turkey-roll sandwiches neither clinical illness nor infection were significantly associated with consumption of any other individual food item.

The majority of the foods, including those associated with illness, were bought

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Table 1. *Food-specific attack rates for foods consumed at christening party*

Food consumed	Attack rate (ill/nos who ate)	Relative risk
Salmon rolls	8/26 (31%)	0·97
Egg mayonnaise sandwiches	6/13 (46%)	1·62
Chicken nuggets	9/29 (31%)	0·98
Pork pie	14/29 (48%)	2·18, $P < 0·05$
Sausage rolls	12/34 (35%)	1·23
Ham sandwiches	13/29 (45%)	1·86
Turkey roll sandwiches	10/14 (71%)	3·15. $P < 0·001$
Tinned frankfurter sausages	12/22 (55%)	2·30, $P < 0·025$
Black pudding	8/19 (42%)	1·50
Pâté	6/14 (43%)	1·48
Tomatoes	4/11 (36%)	1·19
Cheese rolls	10/32 (31%)	1·00
Gâteaux	10/31 (32%)	1·05
Coleslaw	6/18 (33%)	1·08

* Figures in parentheses are percentages.

at a Birmingham branch of a large national supermarket chain on the morning of 27 June 1987. Turkey-roll was purchased ready sliced and open from the delicatessen counter. It was picked up by the assistant on greaseproof paper and put into a polythene bag and sealed. The foods were then taken to the home kitchens of four women for preparation. In two of these kitchens were joints of raw beef from a local butcher. The foods were prepared over a period of 30 h immediately before the reception: the cold meats, including the turkey-roll, were stored in a single domestic refrigerator. All the food was taken to the reception room at noon on 28 June 1987 when rolls and sandwiches were made up. The sliced turkey-roll was taken from its polythene bag placed in bread, quartered and the sandwiches garnished with cucumber and tomato and then placed on trays. The food was covered in 'cling film', left on a serving table during an exceptionally warm afternoon, and with the room heating inadvertently on, until it was eaten as a buffet after the christening service at about 6.00 pm. None of the 4 women was ill at the time of, or before, preparing the food: 3 of the 4 attended the function, and 2 became ill on 1 and 3 July: they submitted faecal specimens from which VT+ *E. coli* O 157.H7 were isolated on 8 and 9 July respectively. The woman who did not attend the function remained well and faeces samples were negative.

No epidemiologically implicated foods remained for culture. VT+ *E. coli* were not detected in six other foods (commercial mayonnaise, ham, black pudding, pâté, pickled beetroot, pickled cucumber) remaining nor in 17 samples of cooked meats purchased from the same supermarket 6 weeks after the reception.

DISCUSSION

This is the first point-source outbreak of *E. coli* O 157.H7 haemorrhagic colitis documented in the UK. The attack rate (28%) was comparable with other

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outbreaks (2, 4, 5). The modal incubation period of 2 days was shorter than reported elsewhere (1, 5) and similar to that reported for enterotoxigenic *E. coli*. Excretion of the organism was identified up to 16 days after the reception and up to 14 days after onset of illness. Previously (3, 6) VT+ *E. coli* were not detected in faecal samples later than 6 days after onset of illness. The illnesses reported were typical of previous outbreaks with abdominal pain, watery diarrhoea, frank blood in faeces and only 18% of cases reported feverishness. The development of HUS in a young adult, rather than children or the elderly, is unusual but has previously been documented (16). The source of VT+ *E. coli* infection has been traced to hamburger meat (1, 2, 4) and raw milk (17, 18) in North America and Belgium (19), but this association has not yet been established in the UK. We found that consumption of turkey-roll sandwiches was strongly associated with infection and may have been the primary source of infection. However, since only 10 clinically ill cases reported eating turkey-roll, cross contamination of other foods during preparation was probable. Rapid multiplication of bacteria would have been encouraged by the high ambient temperature in which food was left for over 6 h.

The turkey-roll may have been contaminated in the kitchens of the food preparers, at the retail outlet or during manufacture. Raw beef, was found in two of the buffer preparer's domestic kitchens, so that cross contamination to the turkey roll might have occurred but no obvious lapses of hygiene were elicited during detailed interviews with the food handlers. It is more likely that the turkey roll was contaminated before purchase. It might be objected that this would have led to a widespread outbreak. However, without the clustering of cases resulting from the reception, such an outbreak might well escape notice. The attack rate might be low if the initial number of organisms in the purchased food was small, and storage in a refrigerator or rapid consumption, did not allow bacterial multiplication. Furthermore, 4 of 17 microbiologically confirmed infected persons in our outbreak were asymptomatic and a further 5 had mild or atypical illness. Unless frank blood is present in the faeces such patients in the community are unlikely to be investigated for VT+ *E. coli*.

It is necessary to discover more about the sources and mode of spread of VT+ *E. coli*. Surveys are required to establish the prevalence of VT+ *E. coli* in foods but such surveys require highly sensitive methods such as the use of specific DNA probes. Clinical laboratories should consider routinely screening faeces using sorbitol MacConkey agar and *E. coli* O 157 antiserum.

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