

Communicable Disease Report

CDR Weekly and *CDR Review*: five years old

S Handysides

The *Communicable Disease Report (CDR)* is the direct descendent of the summaries of weekly returns from public health laboratories circulated by the Emergency Public Health Laboratory during the second world war. Five years ago the *CDR* became available on subscription. At the same time it divided into the *CDR Weekly* – carrying news on its front page and current laboratory data; and the four weekly *CDR Review* – with surveillance reports and papers.

Many further developments have occurred in the past five years. Several sections of the *CDR Weekly* have been revised since a working party drawn from throughout the PHLS and beyond surveyed readers in 1993. This exercise is continuing to bear fruit, with the help of colleagues responsible for particular groups of infections¹. *Notifications of infectious diseases* first appeared in the *CDR* in September 1995, presenting data from the Office of Population Censuses and Surveys that were previously published in the registrar general's weekly return. The *CDR* can now be said to provide 'one stop shopping' for data on communicable diseases in England and Wales. A twice yearly report on the surveillance of waterborne infections is planned for 1996, along with further changes in the presentation of gastrointestinal and meningococcal infections, and a commitment to improve the presentation of data on bacteraemias. News items on the front page of *CDR Weekly* usually represent a synthesis of expert opinion and are not, therefore, attributed to individual authors. We believe, however, that those who write first drafts for the front page deserve a special thanks for unsung work undertaken at short notice. Their names will be published in the *CDR Index* for 1995.

Another development in 1995 was the publication of the *CDR Weekly* front page on the PHLS Communicable Disease Surveillance Centre's world wide web home page (<http://www.open.gov.uk/cdsc/cdschome.htm>)². We are making the entire *CDR Weekly* and four weekly *CDR Review* available on the web in 1996 on an experimental basis to discover the size of our potential electronic market. A steady stream of readers has downloaded files each week³, and we hope to increase our electronic readership as our service becomes comprehensive, and as more potential readers gain access to the internet. We believe that this will be of particular value to readers overseas, for whom postal delivery may be slow, and to non-commercial subscribers ineligible for free subscriptions. *CDR* papers are identified by Medline searchers worldwide and the internet facility will enable them to read them, even if paper copies are unavailable.

In its first five years the *CDR Review* has acquired a reputation as a purveyor of high quality papers on communicable disease surveillance and a source of guidance on the control and prevention of communicable diseases. In 1995 we received many requests to reprint papers and reproduce tables and figures, and

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An outbreak of *Escherichia coli* O157 infection linked to padding pools

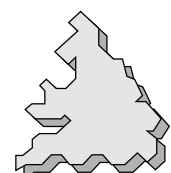
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the *CDR* was cited in numerous publications. A third of the papers submitted to the *CDR Review* are written by authors outside the PHLS. All papers undergo rigorous peer review, both within and outside the PHLS, using a growing number of referees (listed in the *CDR Index*). We are grateful to our referees for the time and effort they spend on our behalf. We publish many papers very quickly, occasionally within four weeks, and on average four months after submission.

Authors are welcome to discuss proposed papers with the editor, and papers should be submitted with reference to 'Information for authors' published in the *CDR Index*.

References

1. CDSC. Look inside: *CDR Weekly* is changing. *Communicable Disease Report* 1995; 5: 61.
2. CDSC. *CDR Weekly* front page: on a screen near you. *Communicable Disease Report* 1995; 5: 64.
3. CDSC. Electronic *CDR*: the story so far. *Communicable Disease Report* 1995; 5: 129.

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General outbreaks of Vero cytotoxin producing *Escherichia coli* O157 in England and Wales from 1992 to 1994

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 H R Smith, B Rowe*

Summary

We have reviewed all general outbreaks of infection due to Vero cytotoxin producing *Escherichia coli* (VTEC) O157 reported in England and Wales from 1992 to 1994. One hundred and seventy-three people were affected in 18 outbreaks, compared with 76 people in seven outbreaks in the preceding three years (1989 to 1991). Outbreaks occurred throughout England and Wales. Thirty-eight per cent of cases were admitted to hospital, 21% developed haemolytic uraemic syndrome, and 3% died. VTEC O157 infection causes particular concern because of its serious complications – haemorrhagic colitis and haemolytic uraemic syndrome, its capacity to spread from person to person as well as by food and water, and its reservoir in dairy and beef cattle.

Introduction

Vero cytotoxin producing *Escherichia coli* O157 was first identified as a human pathogen in 1982 in two outbreaks associated with the consumption of hamburgers from a fast food chain in the United States¹. Outbreaks have since been documented in several countries, including the United Kingdom (UK)²⁻⁹. Transmission of VTEC O157 can be by food^{2-5,10,11}, particularly undercooked ground beef^{1,12,13} and unpasteurised milk¹⁴, waterborne¹⁵⁻¹⁷, through person to person¹⁸⁻²² and zoonotic²³⁻²⁶ spread, and has occurred in laboratories^{27,28}.

VTEC O157 is associated with illness that ranges from non-bloody diarrhoea to haemorrhagic colitis and haemolytic uraemic syndrome (HUS)²⁹. Between 2% and 7% of cases of infection with VTEC O157 usually develop HUS, and the incidence is higher in children than in adults³⁰. The proportion of cases that develop HUS has been as high as 30% in some outbreaks⁴.

The virulence mechanisms of VTEC O157 include the production of Vero cytotoxin and adhesins^{31,32}. The phage types (PT) most frequently isolated in the UK are PT2, PT49, PT1, PT8, and PT4³, and the commonest Vero cytotoxin type is VT2.

The incidence of this infection varies within the UK. The highest annual incidence so far was observed in 1994 in Scotland: 4.73 per 100 000 population compared with 0.8/100 000 in England and Wales and 0.18/100 000 in Northern Ireland.³⁴

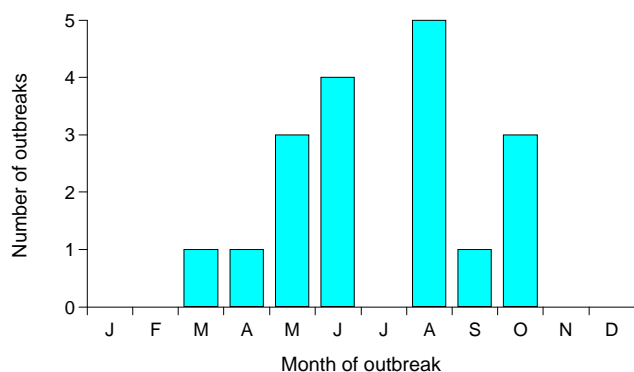
Evidence of infection with *E. coli* O157 can be obtained by faecal culture on sorbitol MacConkey agar (SMAC) or improved derivatives³⁵. Unlike most *E. coli* of human origin, VTEC O157 do not ferment sorbitol within 24 hours³⁶. Non-sorbitol fermenting colonies are tested for agglutination with an O157 antiserum or with an O157 latex agglutination kit³⁷. Serological tests for antibodies to the lipopolysaccharide of *E. coli* O157 may also provide evidence of infection³⁸.

Methods

An outbreak is defined as an incident in which two or more people who experience a similar illness or proven infection (at least one of them being ill) are thought to have had a common exposure³⁹. A general outbreak is defined as an outbreak affecting members of more than one private residence, or residents of an institution.

A surveillance system for all documented general outbreaks of infectious intestinal disease was developed by the PHLS Communicable Disease Surveillance Centre (CDSC) in collaboration with consultants in communicable disease control (CCDCs), in response to a recommendation by the Committee on the Microbiological Safety of Food (Richmond Committee)⁴⁰ and introduced in 1992. CDSC becomes aware of possible general outbreaks of infectious intestinal disease from various sources, including the national laboratory reporting scheme⁴¹, CCDCs, environmental health officers, microbiologists, and the national reference laboratory, the PHLS Laboratory of Enteric Pathogens (LEP). A questionnaire is then sent to the appropriate CCDC with a request that the lead investigator completes it when the outbreak investigation is over. The questionnaire seeks a basic or minimum set of data on all outbreaks, including details of the setting, mode of transmission, causative organism, morbidity, mortality, the proportion of cases whose infection is laboratory confirmed, and details of epidemiological and laboratory investigations. A computer package, Epi Info

Figure 1 Seasonality of outbreaks of E. coli O157 infection: England and Wales 1992 to 1994 (n = 18)



version 5, is used to store and analyse data from the questionnaires⁴².

The duration of an outbreak is defined as the interval between the date of onset of the first case and the date of onset of the last case.

This paper reports on an analysis of the minimum data sets on outbreaks of VTEC O157 infection from this surveillance system's first three years of operation: 1992 to 1994. Supplementary information was obtained from detailed reports of the investigations which accompanied the minimum data set in six outbreaks and from published reports in four^{14,26,43,44}. In addition, we reviewed the laboratory reports of VTEC O157 received by CDSC from LEP to see what proportion of isolations arose from outbreaks.

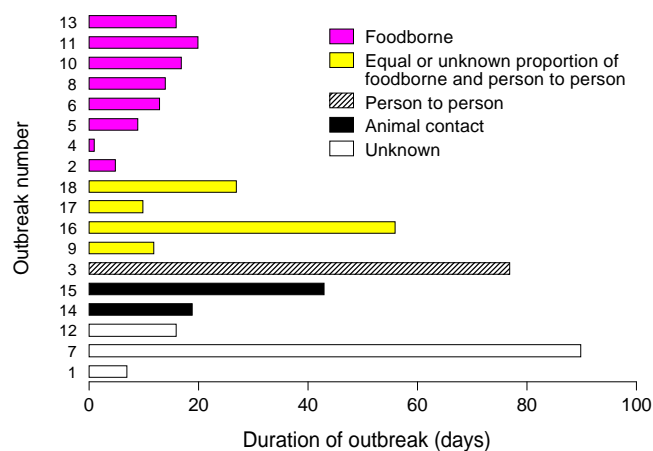
Results

From 1 January 1992 to 31 December 1994, 1590 general outbreaks of infectious intestinal disease in England and Wales were identified and a minimum data set was obtained for 80% (1280/1590), 18 of which were outbreaks of infection with VTEC O157 affecting 173 people.

Five of the 18 outbreaks occurred in 1992, eight in 1993, and five in 1994. To assist identification the outbreaks are numbered in chronological order from 1 to 18 in the text, tables and figures.

CDSC was aware of three further outbreaks, or possible outbreaks, of VTEC O157 infection. The first occurred in a psychiatric hospital, where three of the 14 cases died and four cases were confirmed bacteriologically⁴⁵. This outbreak started in December 1991, but some cases were ill at the beginning of January 1992. In 1993 a possible outbreak was identified comprising four cases of VTEC O157 PT28 infection that were clustered in time. The cases had no geographical or epidemiological links, but PT28 had not been seen before and has not been seen since in England, although it has been isolated in Scotland in 1993, 1994, and 1995. The third outbreak was a cluster of five cases identified in 1994. On investigation, these cases had all recently returned from Majorca.

Figure 2 Duration and attributed mode of transmission of outbreaks of E. coli O157 infection: England and Wales 1992 to 1994 (n = 18)



Seasonality and duration

Thirteen of the 18 outbreaks occurred during the months May to August, five of them in August alone (figure 1). The duration of outbreaks varied between one and 90 days (figure 2).

Region

Over the three year period, the outbreaks were widely distributed throughout England and Wales. The highest proportion of outbreaks (4/18) occurred in the catchment area of Trent Regional Health Authority. No more than two outbreaks occurred in any region in any one year (table 1).

Setting of the outbreak

Two outbreaks occurred after 'functions': an evening dinner in a hotel and an evening dinner in a function room. The other outbreaks were linked to a barbecue, a scout camp, a farm visit, a farm, a home for elderly people, and a preschool nursery. Ten community outbreaks occurred, one of which also affected children in a nursery (tables 1 and 2)⁴⁴.

Mode of transmission

Of the 18 outbreaks, eight were considered by the reporter to be foodborne, four were reported to have been transmitted by food and from person to person in equal or unknown proportions, two were transmitted by contact with cattle, one from person to person, and in three the route of transmission was unknown.

Foodborne outbreaks

Food was submitted for microbiological examination in five of the eight outbreaks reported as being foodborne. Contamination with VTEC O157 was confirmed in two outbreaks: unpasteurised milk in outbreak 6¹⁴ and a beefburger in outbreak 10⁴³. In both outbreaks PTs, VTs, and plasmid profiles of isolates from food were identical to isolates from the human cases^{14,43}.

Food samples were obtained but were negative in three outbreaks. In the first of the three an opportunity

Table 1 Outbreaks of *E. coli* O157 infection in England and Wales 1992 to 1994 date, region, setting, and laboratory results

Outbreak	Date	Region	Setting	Number ill (culture positive)	Serodiagnosis only	Asymptomatic culture positive
1	March 1992	Trent	Home for elderly people	5 (5)	–	–
2	May	Trent	Hotel/reception	19 (3)	–	–
3	June	Oxford	Community nursery ⁴⁴	15 (12)	1	2
			outside nursery	22 (14)	1	
4	August	Wessex	Scout camp	3 (3)	–	–
5	August	Northern	Community	4 (4)	–	–
6	May 1993	Trent	Community ¹⁴	7 (6)	–	–
7	May	Yorkshire	Community	9 (3)	–	1
8	June	W Midlands	Function room:reception	9 (4)	1	2
9	June	SW Thames	Community ⁴⁶	7 (6)	1	–
10	August	Wales	Community ⁴³	17 (7)	–	–
11	August	W Midlands	Barbecue	4 (2)	1	1
12	September	Yorkshire	Community	17 (9)	–	–
13	October	SE Thames	Community	5 (2)	–	–
14	April 1994	Wales	Farm	2 (1)	–	–
15	June	Trent	Farm visit ²⁶	7 (4)	2	–
16	August	Northern	Community	6 (6)*	–	–
17	October	W Midlands	Community	3 (3)	–	–
18	October	Mersey	Nursery	12 (1)	11	–
Total				173 (95)	18	6

* No samples sent to LEP for confirmation and further identification.

existed for cross contamination between raw and cooked meat (outbreak 5). In the second outbreak all cases had attended a barbecue (outbreak 11): foods consumed included chicken portions, beefburgers, and sausages, but samples of leftover food were negative for VTEC O157. In the third outbreak three out of five cases had eaten in the same restaurant (outbreak 13).

In outbreak 8 a cohort study revealed a significant association between illness and the consumption of chilli con carne. In another outbreak reported as foodborne all 19 people who were ill had attended a dinner reception at a hotel (outbreak 2). Rare beef was one of the foods served but a cohort study did not show a significant association between consumption of any particular food and illness, and no food remained for microbiological examination. The remaining outbreak reported as mainly foodborne (outbreak 4) occurred in a scout camp. No food was examined.

In three of these outbreaks (5, 6, 10) illness was associated with the consumption of food from particular butchers and a farm dairy. In outbreak 6, VTEC O157 of the same PT, VT, and plasmid profile was identified in the specimens from human cases, in raw milk, and in rectal swabs from 10 out of 105 cows in the dairy herd¹⁴.

Mainly person to person spread

The mode of transmission was reported as 'mainly person to person' in outbreak 3, a community outbreak of 37 cases, 15 of whom were children in a preschool nursery⁴⁴. A cohort study in the nursery found no significant association between risk of illness and strength of preference for any of the main food items served during

the fortnight before the outbreak began⁴⁴. Food hygiene measures in the nursery were found to be adequate. The mother and elder sibling of one nursery case and 20 people unconnected with the nursery were also diagnosed as cases. Four of these cases had eaten meat products bought from a particular butcher in the fortnight before they became ill but no common food product was identified. A case control study incorporating 15 cases who were not associated with the nursery failed to identify any significant associations between illness and consumption of any items from a wide range of foods or shopping at specific food outlets⁴⁴.

Equal or unknown proportion of foodborne and person to person spread

In four outbreaks (9,16,17,18) the mode of transmission was reported as 'equal or unknown proportion of foodborne and person to person'. Three were community outbreaks and one occurred in a preschool nursery. In the nursery outbreak (18) no suspect food was identified and inspection of the nursery kitchen revealed no problems with facilities or food handling practices. Although the nursery maintained high standards of hygiene, person to person transmission could have occurred. In the community outbreaks the cases were clustered in time and place, but food and purchasing histories failed to identify a common food or food outlet. In outbreak 9, three of seven cases used the same paddling pool, which was poorly maintained (coliforms were identified in water samples but not VTEC O157): the mother of one case became ill after caring for her infected child⁴⁶.

Table 2 Outbreaks of *E. coli* O157 infection in England and Wales 1992 to 1994: morbidity, mortality, setting, and reported mode of transmission

Outbreak	Setting	Total ill	Children	Bloody diarrhoea	HUS	Admitted to hospital	Died	Stated mode of transmission
1	Home for elderly people	5	–	4	–	2	2	Unknown
2	Hotel: reception	19	–	5	–	–	1	Foodborne
3	Community nursery	15	15	7	3	14	–	Person to person ⁴⁴
	outside nursery	22	–	–	2	–	–	
4	Scout camp	3	3	3	–	1	–	Foodborne
5	Community ^a	4	–	–	–	2	–	Foodborne
6	Community ^b	7	4	–	3	4	–	Foodborne ¹⁴
7	Community	9	8	5	6	6	–	Unknown
8	Function room: reception	9	–	3	2	2	–	Foodborne
9	Community ^c	7	6	6	3	6	1	Foodborne/person to person ⁴⁶
10	Community ^d	17	–	6	1	1	–	Foodborne ⁴³
11	Barbecue	4	3	1	1	1	–	Foodborne
12	Community	17	6	16	3	8	–	Unknown
13	Community ^e	5	5	5	5	5	–	Foodborne
14	Farm ^f	2	2	–	–	–	–	Contact with cattle
15	Farm visit ^f	7	5	4	4	5	–	Contact with cattle and goats ²⁶
16	Community	6	3	–	1	6	1	Foodborne/person to person
17	Community	3	–	1	–	1	–	Foodborne/person to person
18	Nursery	12	9	4	2	2	–	Foodborne/person to person
Total		173	69	70	36	66	5	

a. Cases ate meat from the same butcher.

b. Cases drank milk from the same farm dairy.

c. 3/7 cases used same paddling pool.

d. Cases ate food from same butcher.

e. 3/5 cases ate at same restaurant.

f. Contact with livestock positive for VTEC O157.

Contact with livestock

This mode of transmission was reported in two outbreaks. In outbreak 14, two children were affected and two out of 50 cattle tested on the associated farm were positive for VTEC O157 of the same PT and VT as the human cases. In outbreak 15, investigations failed to reveal a common foodstuff but all seven cases had attended a farm visitor centre before becoming ill²⁶. Faecal specimens from animals on the farm were positive for VTEC O157 and strains from four human cases and nine animal strains (four cattle and five goats) were indistinguishable on phage typing, Vero cytotoxin subtyping, and restriction fragment length polymorphism analysis²⁶.

Unknown mode of transmission

Outbreak 1 occurred in a home for elderly people and outbreaks 7 and 12 occurred in the community. Cases were clustered in time and place, but no source or mode of transmission was identified.

Morbidity

One hundred and seventy-three people were affected in the 18 outbreaks. Diarrhoea was reported in all outbreaks, with bloody diarrhoea in 14 (table 2). Abdominal pain was reported in 13 outbreaks and fever in seven. Forty per cent of cases (70/173) were reported to have bloody diarrhoea, 38% of cases (66) were admitted to hospital (the range among outbreaks was 0% to 100%), and there were five deaths (table 2). Twenty-one per cent of cases (36) developed HUS (table 2).

Not all outbreaks reported the number of children affected, but this information was available for eleven outbreaks and for nursery cases of a community outbreak (3)⁴⁴. Forty-five per cent of children in these outbreaks (31/69) developed HUS (table 2). The proportion of these children who developed HUS ranged from 20% (3/15) in outbreak 3 to 100% (5/5) in outbreak 13, whose recognition was based on the identification of five cases of HUS admitted in a ten day period to the same renal unit.

The mean number of cases in each outbreak was 10 (range 2 to 39). The attack rate was reported for eight of the 18 outbreaks; the median attack rate was 40% (range 1% to 100%).

Laboratory isolates: sporadic cases and cases from outbreaks

From 1 January 1992 to 31 December 1994, laboratories in England and Wales referred 1266 isolates of VTEC O157 to LEP for confirmation and further identification: 470 in 1992 (0.92/100 000 population), 385 in 1993 (0.75/100 000), and 411 in 1994 (0.8/100 000)^{34,47}. LEP confirmed 89 laboratory isolations of VTEC O157 in the 173 cases identified in outbreaks (51%) (table 1). In addition, outbreak investigations revealed six asymptomatic people with positive specimens which were confirmed by LEP (table 1). Therefore, 7.5% of all laboratory isolations of VTEC O157 (95/1266) arose from the 18 general outbreaks reviewed. Eighteen further cases were diagnosed by detecting antibodies to *E. coli* O157 lipopolysaccharide and the remainder on clinical grounds alone.

Discussion

In this review, 173 people were affected in 18 outbreaks of VTEC in England and Wales, compared with 76 people in seven outbreaks in the preceding three year period (1989 to 1991)⁶. It must be noted, however, that the current surveillance system only became operational in 1992. Subsequently, this system and increased awareness of infections caused by VTEC O157 may have resulted in better ascertainment of outbreaks. This may, in part, explain the difference in numbers of outbreaks recorded in the two periods. The mean number of cases per outbreak was ten in both three year periods.

The majority of outbreaks took place in the summer months, notably in August. In England and Wales VTEC O157 is most commonly isolated (both from outbreaks and sporadic cases) in the third quarter of the year^{6,48}. The same pattern is seen in the United States, Canada, and elsewhere^{26,49,50}. This seasonal variation may reflect a variation in the prevalence of VTEC O157 in cattle, the barbecuing of beef, or the effect of higher ambient temperatures on food preparation and storage⁵¹.

In the present surveillance system the stated mode of transmission depends on the assessment of the reporting investigator. Alternative modes of transmission may have existed for two of the outbreaks reported as foodborne; in one (outbreak 4), scouts had camped in a field grazed by cows, creating an opportunity for direct contact with cow faeces. Outbreak 13, reported as foodborne, was suspected when five children from the same geographical area were admitted with HUS to a London teaching hospital in a period of 10 days. Only two faecal specimens were positive for VTEC O157. Three cases had eaten at the same restaurant – two siblings on one day and the other child the following day. Food samples, taken from the restaurant several days after the suspected meals, were negative. The restaurant was in a zoo with a children's corner, where children could have been in contact with calves. The other two cases had not been to the zoo and had had no previous contact with the other affected children. The cases all lived in the South East Thames region, which is a large geographical area, and the latter two cases may be unrelated to the three who had been to the zoo.

VTEC O157 may not be found in food because current culture techniques are suboptimal for the detection of the organism in food samples or because not all laboratories test food. In addition, all the food may have been eaten or discarded by the time an investigation begins.

The mode of transmission in three community outbreaks (9,16,17) was reported as 'equal or unknown proportion of foodborne and person to person spread'. Cases were clustered in time and place but no evidence was reported in support of foodborne transmission. In two further community outbreaks (7 and 12) cases were clustered in time and place, and the mode of transmission was described as 'unknown'. The features of these outbreaks were similar to outbreaks 16 and 17, and indicate that the reported mode of transmission varied with the reporting investigator.

The identification of a cluster of cases is not the same as the identification of an outbreak. The cluster may represent a coincidental occurrence of unrelated cases within the space time coordinates that serve as the frame of reference for the cluster. A cluster becomes of

epidemiological interest if the cases in the cluster share some of the same causal mechanisms. Investigators must search for epidemiological links to establish vehicles of infection and trace the source of the infection. In several of the clusters reported as outbreaks in this review, the boundaries in time and space that encompassed the cluster were defined after the cases were identified. This technique, in the absence of additional evidence, can produce clusters of causally unrelated cases⁵² by capturing a population that has experienced a high incidence of disease.

Thirty-eight per cent of cases were admitted to hospital, 21% developed HUS, and 3% died. These figures are higher than those recently reported for outbreaks in the United States, where 23% of cases were admitted to hospital, 6% developed HUS, and 1.2% died⁵¹. A third of VTEC O157 cases in Scotland from 1984 to 1992 were admitted to hospital⁵³. A similar proportion was admitted to hospital in the largest reported single outbreak of infection with VTEC O157, which affected more than 700 people in several western states of the United States in 1993¹². In this review, the proportion of cases admitted to hospital may reflect the proportion of children affected, due to their greater susceptibility to HUS. Likewise, in the United States, the proportions of cases that develop HUS in outbreaks has varied with the population affected, with lower rates in communities and (generally) higher rates in children and elderly people³⁰. HUS, which is thought to account for at least 70% of acute renal failure in children in the UK⁵⁴, leads to long term residual impairment in renal function in a substantial proportion of survivors⁵⁵ and has a reported case fatality rate of between 3% and 7%^{49,56}.

Cases of HUS may be more likely than other VTEC O157 infections to be identified and reported: outbreak 13 was reported because five cases of HUS were identified. In this review, the proportion of the total number of cases who developed HUS (21%) was relatively high compared with other reports (2% to 7%)³⁰ and with the proportion in England and Wales from 1989 to 1991 (15%)⁶. Using data from eleven outbreaks and the nursery cases of a community outbreak⁴⁴ we calculated that 45% of affected children developed HUS. The number of HUS cases in this review peaked in the summer months, in parallel with the overall summer increase in VTEC O157 infections, as previously observed in the United States²⁵ and the UK⁵⁷.

Children in preschool nurseries were involved in two outbreaks in this review, and other outbreaks in nurseries have been documented^{9,58,59}. In this setting it is important to exclude children with diarrhoea until their symptoms have resolved. The most important preventive measure within the nursery is to supervise handwashing. Interim guidelines, recently published by the PHLS, recommend that confirmed cases of VTEC O157 should be excluded until two consecutive negative specimens have been obtained⁶⁰. Excluding asymptomatic children whose stools are culture positive until microbiological clearance may be necessary to control outbreaks. Transmission ceased in one outbreak (3) when an exclusion criterion of one negative stool specimen was applied⁴⁴. In one outbreak lasting seven days in a home for elderly people, the mode of transmission was reported as 'unknown'. It may have been foodborne, but secondary transmission can easily occur in such a setting. It is therefore important that effective control policies exist and are implemented, staff

are adequately trained in infection control, and simple surveillance mechanisms are in place to detect and control outbreaks quickly⁶¹⁻⁶³. Two outbreaks occurred on farms, outlining the risks of acquiring zoonotic infections through direct contact with farm animals and emphasising the need to follow simple hygienic measures⁶⁴.

Infected cattle were identified as a possible source of infection in three outbreaks and a contaminated beefburger was associated with one outbreak, indicating that cattle are a reservoir for VTEC O157^{14,23-26}. VTEC O157 has previously been isolated from apparently healthy cattle in the course of herd investigations linked to outbreaks of human infection^{25,66}. One recent report described a faecal carriage rate of 9% in a dairy herd⁶⁷. The organism has also been recovered from cattle in the course of surveys unrelated to human infections^{68,69}. Infected animals can introduce infection into abattoirs and contaminate carcasses of healthy animals⁷⁰. The process of mincing beef may transfer pathogens from the surface of the meat to the interior. Since mince may include meat from several carcasses, a single contaminated carcass may contaminate a large amount of mince. Beefburgers and other minced meat products, therefore, pose a greater hazard than intact joints of meat³⁰. It is essential that effective prevention and control measures are put into practice from the farm to the kitchen, and that consumers are aware of advice from the Department of Health that beefburgers should be adequately cooked before consumption^{34,71}. The outbreak associated with raw milk supports the argument for banning the sale of raw milk in England and Wales³⁴.

Only 40% of cases identified in general outbreaks had bloody diarrhoea. If the presence of blood in the stool is used as the selection criterion for examining for VTEC O157, a large proportion of cases will be missed. To establish the true magnitude of the problem the working group on VTEC of the Advisory Committee on the Microbiological Safety of Food recommended in 1995 that all diarrhoeal stools should be tested for VTEC O157³⁴. In conjunction with the detailed typing of VTEC O157, this approach will enable a better understanding of the epidemiology of this important pathogen.

Less than 8% of laboratory isolations of VTEC O157 identified by LEP in England and Wales between 1992 and 1994 were associated with general outbreaks. It is likely that some of the remainder were sporadic cases but others arose from family outbreaks or, possibly, from unrecognised or unreported general outbreaks. The threat to public health from infections with VTEC O157 requires the introduction of an effective national surveillance system to obtain exposure histories from all cases and not only those in recognised general outbreaks. If this pathogen is to be controlled, more information is needed on its epidemiology in humans and animals and its distribution in food and the environment. Good quality epidemiological data combined with detailed typing of all isolates will help to clarify the problem.

Acknowledgements

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References

- Riley LW, Remis RS, Helgerson SD, McGee HB, Wells JG, Davis BR, et al. Hemorrhagic colitis associated with a rare *Escherichia coli* serotype. *N Engl J Med* 1983; **308**: 681-5.
- Belongia EA, MacDonald KL, Parham GL, White KE, Koriath JA, Lobato NM. An outbreak of *Escherichia coli* -O157:H7 associated with consumption of precooked meat patties. *J Infect Dis* 1991; **164**: 338-43.
- Upton P, Coia J. Outbreak of *Escherichia coli* O157 infection associated with pasteurised milk supply. *Lancet* 1994; **334**: 1015.
- Morgan D, Newman CP, Hutchinson DN, Walker AM, Rowe B, Majid F. Verotoxin producing *Escherichia coli* O157 infections associated with the consumption of yoghurt. *Epidemiol Infect* 1993; **111**: 1 81-7.
- Besser RE, Lett SM, Weber JT, Doyle MP, Barrett TJ, Wells JG, et al. An outbreak of diarrhea and hemolytic uremic syndrome from *Escherichia coli* O157:H7 in fresh pressed apple cider. *JAMA* 1993; **269**: 2217-20.
- Thomas A, Chart H, Cheasty T, Smith HR, Frost JA, Rowe B. Vero cytotoxin producing *Escherichia coli*, particularly O157, associated with human infections in the United Kingdom: 1989-91. *Epidemiol Infect* 1993; **110**: 591-600.
- Rowe PC, Orrbine E, Wells GA, McLaine PN. Epidemiology of hemolytic-uremic syndrome in Canadian children from 1986-1988. *J Pediatr* 1991; **13**: 60-98.
- Isaacson M, Canter PH, Etter P, Arntzen L, Bomans P, Heenan R. Haemorrhagic colitis epidemic in Africa. *Lancet* 1993; **341**: 961.
- Lerman Y, Cohen D, Gluck A, Ohad E, Sechter I. A cluster of cases of *Escherichia coli* O157 infection in a day-care centre in a communal settlement (kibbutz) in Israel. *J Clin Microbiol* 1992; **30**: 520-1.
- CDC. *Escherichia coli* O157:H7 outbreak linked to commercially distributed dry cured salami - Washington and California 1994. *MMWR* 1995; **44**: 157-60.
- Cieslak PR, Barrett TJ, Griffin PM. *Escherichia coli* O157:H7 infection from a manured garden. *Lancet* 1993; **342**: 367.
- Update: Multistate outbreak of *Escherichia coli* O157 infections from hamburgers - Western United States, 1992-1993. *MMWR* 1993; **42**: 258-63.
- Bell BP, Goldoft M, Griffin PM, Davis MA, Gordon DC, Tarr PI, et al. A multistate outbreak of *Escherichia coli* O157:H7-associated bloody diarrhea and hemolytic uremic syndrome from hamburgers. *JAMA* 1994; **272**: 1349-53.
- Chapman PA, Wright DJ, Higgins R. Untreated milk as a source of verotoxigenic *E. coli* O157. *Vet Rec* 1993; **133**: 171-2.
- Dev VJ, Main M, Gould IM. Waterborne outbreak of *Escherichia coli* O157. *Lancet* 1991; **337**: 1412.
- Brewster DH, Brown MI, Robertson D, Houghton GL, Bimson J, Sharp JCM. An outbreak of *Escherichia coli*

- O157 associated with a children's paddling pool. *Epidemiol Infect* 1994; **112**: 441-7.
17. Keene WE, McAnulty JM, Hoesly FC, Williams LP Jr, Hedberg PHK, Oxman GL, et al. A swimming associated outbreak of hemorrhagic colitis caused by *Escherichia coli* O157:H7 and *Shigella sonnei*. *N Engl J Med* 1994; **331**: 579-84.
 18. Salmon RL, Farrell ID, Hutchison JG, Coleman DJ, Gross RJ, Fry NK, et al. A christening party outbreak of haemorrhagic colitis and haemolytic uraemic syndrome associated with *Escherichia coli* O157. *Epidemiol Infect* 1989; **103**: 249-54.
 19. Belongia EA, Osterholm MT, Soler JT, Ammend DA, Braun JE, MacDonald KL. Transmission of *Escherichia coli* O157:H7 infection in Minnesota child day care facilities. *JAMA* 1993; **269**: 883-8.
 20. Spika JS, Parsons JE, Nordenberg D, Wells JG, Gunn RA, Blake PA. Hemolytic uremic syndrome and diarrhea associated with *Escherichia coli* O157:H7 in a day care center. *J Pediatr* 1986; **109**: 287-91.
 21. Pavia AT, Nichols CR, Green DP, Tauxe RV, Mottice S, Greene KD, et al. Hemolytic uremic syndrome during an outbreak of *Escherichia coli* O157:H7 infections in institutions for mentally retarded persons: clinical and epidemiologic observations. *JPediatr* 1990; **116**: 544-51.
 22. Ryan CA, Tauxe RV, Hosesk GW, Wells JG, Stoesz PA, McFadden HW, et al. *Escherichia coli* O157:H7 diarrhea in a nursing home: clinical, epidemiological, and pathological findings. *J Infect Dis* 1986; **154**: 631-8.
 23. Renwick SA, Wilson JB, Clarke RC, Lior H, Borczyk A, Spika JJ, et al. Evidence of direct transmission of *Escherichia coli* O157:H7 infection between calves and a human. *J Infect Dis* 1993; **168**: 792-3.
 24. Synge BA, Hopkins GF, Reilly WJ, Sharp JCM. Possible link between cattle and *E. coli* O157 infection in a human. *Vet Rec* 1993; **133**: 507.
 25. Martin ML, Shipman LD, Potter ME, Wachsmuth IK, Wells JG, Hedberg K, et al. Isolation of *Escherichia coli* O157:H7 from dairy cattle associated with two cases of haemolytic uraemic syndrome. *Lancet* 1986; **ii**: 1043.
 26. Shukla R, Slack R, George A, Cheasty T, Rowe B, Scotter J. *Escherichia coli* O157 infection associated with a farm visitor centre. *Communicable Disease Report* 1995; **5**: R86-90.
 27. Booth L, Rowe B. Possible occupational acquisition of *Escherichia coli* O157 infection. *Lancet* 1993; **342**: 1298-9.
 28. Burnens AP, Zbinden R, Kaempf L, Heinzer I, Nicolet J. A case of laboratory acquired infection with *Escherichia coli* O157:H7. *Zbl Bakt* 1993; **279**: 512-7.
 29. Tarr PI. *Escherichia coli* O157:H7: clinical, diagnostic, and epidemiological aspects of human infection. *Clin Infect Dis* 1995; **20**: 1-10.
 30. 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.
 31. Ostroff SM, Tarr PI, Neill MA, Lewis JH, Hargnett-Bean N, Kobayashi JM. Toxin genotypes and plasmid profiles as determinants of systemic sequelae in *Escherichia coli* O157:H7 infections. *J Infect Dis* 1989; **160**: 994-8.
 32. Scotland SM, Willshaw GA, Smith HR, Rowe B. Properties of strains of *Escherichia coli* belonging to serogroup O157 with special reference to production of Vero cytotoxins VT1 and VT2. *Epidemiol Infect* 1987; **99**: 613-24.
 33. Frost JA, Cheasty T, Thomas A, Rowe B. Phage typing of Vero cytotoxin-producing *Escherichia coli* O157 isolated in the United Kingdom: 1989-91. *Epidemiol Infect* 1993; **110**: 469-75.
 34. Advisory Committee on the Microbiological Safety of Food. *Report on Verocytotoxin-producing Escherichia coli*. London: HMSO, 1995.
 35. Chapman PA. Isolation, identification and typing of Verocytotoxin-producing *Escherichia coli* O157. *PHLS Microbiology Digest* 1994; **11**: 13-7.
 36. Farmer JJ, Davis BR. H7 antiserum-sorbitol fermentation medium for detecting *Escherichia coli* O157:H7 associated with hemorrhagic colitis. *J Clin Microbiol* 1985; **22**: 620-5.
 37. Smith HR, Scotland SM. Isolation and identification methods for *Escherichia coli* O157 and other Verocytotoxin-producing strains. *J Clin Pathol* 1993; **46**: 10-7.
 38. Chart H, Smith HR, Scotland SM, Rowe B, Milford DV, Taylor CM. Serological identification of *Escherichia coli* O157:H7 infection in haemolytic uraemic syndrome. *Lancet* 1991; **337**: 138-40.
 39. Department of Health. *Management of outbreaks of foodborne illness*. London: HMSO, 1994.
 40. Committee on the Microbiological Safety of Food. *The microbiological safety of food: part 1*. London: HMSO, 1990.
 41. Grant AD, Eke B. Application of information technology to the laboratory reporting of communicable disease in England and Wales. *Communicable Disease Report* 1993; **3**: R75-8.
 42. Dean AG, Dean JA, Burton AH, Dicker RC. *Epi Info, Version 5: a word processing, database and statistics programme for epidemiology on microcomputers*. Centres for Disease Control and Prevention, Georgia: USD Inc, 1990.
 43. Willshaw GA, Thirlwell J, Jones AP, Parry S, Salmon R, Hickey M. Vero cytotoxin producing *Escherichia coli* O157 in beefburgers linked to an outbreak of diarrhoea, haemorrhagic colitis and haemolytic uraemic syndrome in Britain. *Letters in Applied Microbiology* 1994; **19**: 304-7.
 44. Allaby MAK, Mayon-White R. *Escherichia coli* O157: outbreak in a day nursery. *Communicable Disease Report* 1995; **5**: R4-6.
 45. CDSC. *Escherichia coli* O157 in Stockport. *Communicable Disease Report* 1992; **2**: 13.
 46. Hildebrand J, Maguire H, Holliman R, Kangesu K. An outbreak of *Escherichia coli* O157 linked to paddling pools. *Communicable Disease Report* 1996; **6**: R33-6.
 47. CDSC. Vero cytotoxin producing *Escherichia coli*: which specimens should be tested? *Communicable Disease Report* 1995; **5**: 147.
 48. Cheasty T, Thomas A, Chart H, Smith HR, Rowe B. Vero cytotoxin producing *Escherichia coli* O157 in the United Kingdom. *Communicable Disease Report* 1992; **2**: R140-1.
 49. Ostroff SM, Kobayashi JM, Lewis JH. Infections with *Escherichia coli* O157:H7 in Washington State: the first

- year of statewide surveillance. *JAMA* 1989; **262**: 355-9.
50. Pai CH, Ahmed N, Lior H, Johnson WM, Sims HV, Woods DE. Epidemiology of sporadic diarrhoea due to verocytotoxin-producing *Escherichia coli*: a two year prospective study. *J Infect Dis* 1988; **157**: 1054-7.
 51. Boyce TG, Swerdlow DL, Griffin PM. *Escherichia coli* O157:H7 and the hemolytic-uremic syndrome. *N Engl J Med* 1995; **333**: 364-8.
 52. Rothman KJ. A sobering start for the cluster busters' conference. *Am J Epidemiol* 1990; **132** (suppl 1): S6-13.
 53. Sharp J, Coia J, Curnow J, Reilly W. *Escherichia coli* O157 infections in Scotland. *J Med Microbiol*, 1994; **40**: 3-9.
 54. Taylor CM, White RHR, Winterborn MH, Rowe B. Haemolytic uraemic syndrome: clinical experience of an outbreak in the West Midlands. *BMJ* 1986; **292**: 1513-6.
 55. Fitzpatrick M, Shah V, Trompeter R, Dillon M, Barrat M. Long term outcome of childhood haemolytic uraemic syndrome. *BMJ* 1991; **303**: 489-92.
 56. Martin DL, Macdonald KL, White KE, Soler JT, Osterholm MT. The epidemiological and clinical aspects of the hemolytic uremic syndrome in Minnesota. *N Engl J Med* 1990; **323**: 116-7.
 57. Hall SM. BPA-CDSC surveillance of haemolytic uraemic syndrome 1983-84. *Communicable Disease Report* 1985 (28): 3-6.
 58. Verotoxin producing *Escherichia coli* O157 phage type 49. *Communicable Disease Report* 1991; **1**: 213.
 59. CDSC. Outbreak of *Escherichia coli* O157 in North Wales. *Communicable Disease Report* 1995; **5**: 175.
 60. Subcommittee of the PHLS Working Group on Verocytotoxin producing *Escherichia coli* (VTEC). Interim guidelines for the control of infections with Verocytotoxin producing *Escherichia coli* (VTEC). *Communicable Disease Report* 1995; **5**: R77-81.
 61. Smith PW, Daly PB, Rusnak PG, Roccaforte JS. Design and dissemination of a multiregional long term care infection control training programme. *Am J Infect Control* 1992; **20**: 275-7.
 62. Smith PW, Rusnak RN. APIC guideline for infection prevention and control in the long term care facility. *Am J Infect Control* 1991; **19**: 198-215.
 63. Public Health Medicine Environmental Group. *Guidelines on the control of infection in residential and nursing homes*. Lewisham: PHMEG, 1995.
 64. Dawson A, Griffin R, Fleetwood A, Barrett NJ. Farm visits and zoonoses. *Communicable Disease Report* 1995; **5**: R81-6.
 65. Borczyk AA, Karmali MA, Lior H, Duncan LMC. Bovine reservoir for Verocytotoxin producing *Escherichia coli* O157:H7. *Lancet* 1987; **i**: 98.
 66. Wells JG, Shipman LD, Greene KD, Sowers EG, Green JH, Cameron DN, et al. Isolation of *Escherichia coli* serotype O157:H7 and other shiga-like toxin producing *Escherichia coli* from dairy cattle. *J Clin Microbiol* 1991; **29**: 985-9.
 67. Anon. Report of Veterinary Investigation Service, England and Wales. *Vet Rec* 1995; **137**: 334-6.
 68. Montenegro MA, Bulte M, Trunpf T, Aleksic S, Reuter G, Bulling E, et al. Detection and characterisation of fecal Verocytotoxigenic *Escherichia coli* from healthy cattle. *J Clin Microbiol* 1990; **28**: 1417-21.
 69. Chapman PA, Wright DJ, Norman P. Verocytotoxin-producing *Escherichia coli* infections in Sheffield: cattle as a possible source. *Epidemiol Infect* 1989; **102**: 439-55.
 70. Chapman PA, Siddons CA, Wright DJ, Norman P, Fox J, Crick E. Cattle as a possible source of Verocytotoxin producing *Escherichia coli* O157 infections in man. *Epidemiol Infect* 1993; **111**: 439-47.
 71. Sir Donald Acheson's advice on cooking beefburgers. Department of Health press notice, 14 February 1991. (H91/66).

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An outbreak of *Escherichia coli* O157 infection linked to paddling pools

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Summary

In a one month period in the summer of 1993 a community outbreak of *Escherichia coli* O157 infection affected six children living within an area of 1.5 miles radius in south west London. Three children developed haemolytic uraemic syndrome, one of whom died.

E. coli O157 phage type 2 was isolated from faeces in five cases and serological tests showed the sixth child had antibodies to *E. coli* O157 lipopolysaccharide. *E. coli* O157

phage type 2 was isolated from a faecal specimen from this child's mother who was a secondary case.

Three of the cases, whose onset dates were within three days of each other, had all been exposed to a paddling pool where disinfection procedures were found to be inadequate. Samples of water taken from this pool contained *E. coli*, although not the O157 serotype. A fourth case had played at a different paddling pool in the three days before becoming ill. Action has been taken to improve disinfection procedures at municipal paddling pools in the London borough concerned.

Introduction

In 1977 it was recognised that some strains of *E. coli* could produce a toxin capable of lysing Vero cells¹. Vero cytotoxin producing *E. coli* (VTEC) was shown to be associated with haemorrhagic colitis in 1982² and with haemolytic uraemic syndrome (HUS) in 1983³.

Serotype O157:H7 is the commonest strain of VTEC identified in human infections⁴. Laboratory isolates of *E. coli* O157 infection have been increasing in recent years in the United Kingdom⁴.

In July 1993 the consultant in communicable disease control (CCDC) for a health authority in south west London was informed of five children with bloody diarrhoea, three of whom had developed HUS. The cases had developed symptoms in June and July and all lived within an area of radius 1.5 miles. *E. coli* O157 had been isolated from stools from three of the children. The cases lived in the health authority's catchment area which has a population of 341 200⁵. In the years 1992 to 1994, 57 *E. coli* O157 isolates were reported to the PHLS Communicable Disease Surveillance Centre (CDSC) from laboratories in the South West Thames region⁶ (mid-1993 population estimate 2 999 279⁷) giving a rate of six isolates per million population each year. One would therefore expect about two cases a year to occur in the health authority in question.

Method

Epidemiology

An incident control team met on 20 July and decided to investigate the cluster of cases of *E. coli* O157 infection using a descriptive study. A structured questionnaire was personally administered to the parents of affected children. The parents were asked about potential common contributory exposures in the ten days before the children became ill.

Case finding

A letter was sent to all general practitioners in the area informing them of the outbreak, and asking them to inform the CCDC if they had any patients with bloody diarrhoea and to test patients' stools appropriately.

An electronic message was sent on 19 July to CCDCs and microbiology laboratories in the South West Thames region, requesting information about any isolates of *E. coli* O157 that might be linked with the local outbreak.

Case definition

A primary case was defined as a person with any gastrointestinal symptoms and/or HUS with onset date in 1993 between mid-June and the end of July, who lived in south west London and in whom there was microbiological evidence of *E. coli* O157 infection. A secondary case was defined as a person who met the above definition but who, in addition, had had household or close personal contact with a case in the previous two weeks.

Microbiology

Faeces, food, and filtrate from pool water were tested using Sorbitol-MacConkey medium⁸ at Tooting Public Health Laboratory (PHL), St George's Hospital. Isolates of *E. coli* O157 were sent to the PHLS Laboratory of Enteric Pathogens (LEP) for confirmation of identity, phage typing,

and additional tests to distinguish strains⁹. Serum from suspected cases, from whom *E. coli* O157 had not been isolated from stool, was also sent to LEP to test for antibodies to *E. coli* O157 lipopolysaccharide⁹.

Environmental

After foods eaten by the children and shops visited by the parents were identified, an effort was made to collect samples of relevant meat and milk products from the shops.

All food items and possible environmental links were fully investigated by staff from the environmental health departments of the two London boroughs concerned. Samples were sent to Tooting PHL for examination.

Results

Epidemiology

A total of six primary cases were identified with onset dates from 28 June to 27 July. The first case was 8 years old and all other cases were aged 3 or 4 years (table). Four were girls and two were boys. The one secondary case identified was the mother of case 3 (table).

Clinical features

All cases had bloody diarrhoea and three had HUS (table). Case 4 developed a left hemiplegia 12 days after becoming ill and then had fits unresponsive to anticonvulsants. Computed tomography showed areas of recent cerebral ischaemia or infarction. The patient died despite extensive efforts at resuscitation. The secondary case (mother of case 3) became ill nine days after her child. She developed bloody diarrhoea and was in hospital for seven days.

Exposures

Three (cases 1, 2, and 3) of the four children who had visited an outdoor paddling pool in the 10 days before becoming ill had visited the same pool (hereafter known as pool X). Two of these three became ill on the same date (1 July) and the other became ill three days earlier (28 June). It was likely, although their parents could not recall accurately, that all three may have been at pool X on the same day. They all paddled in the pool but there was no report of having eaten a common food item while at the pool. The fourth case had used a paddling pool less than 1.5 miles away (pool Y). No parent commented that they had seen faecal matter in any of the paddling pools. Cases 5 and 6 had not visited an outdoor paddling pool and no links were established between them. There were also no links between cases 5 and 6 and any of the first four cases – for example, all six children went to different nursery schools or playgroups. Three families had pet cats but none had dogs. Case 1 had returned from Ibiza a week before she became ill. Five of the families had a microwave oven at home: all used it for reheating, two for defrosting, and three for cooking.

Four of the children ate beefburgers; two beefburgers were from the same manufacturer but were bought at different shops. Three children had eaten foods that contained minced beef but the beef was bought from different shops. All had drunk milk but it was always pasteurised. Two had eaten yoghurt and three had eaten "fromage frais" (two of the same brand). Four children ate cheese but it was of different types and from different

Table Primary cases: sex, age, onset date, clinical features and length of stay in hospital

Case number	Age (years)	Onset date in 1993	Bloody diarrhoea	HUS	Complications of HUS	Days in hospital
1	8	28 June	Yes	No	No	Nil
2	4	1 July	Yes	No	No	3
3	4	1 July	Yes	Yes	No	19
4	4	9 July	Yes	Yes	Yes	11
5	3	10 July	Yes	Yes	No	10
6	3	27 July	Yes	No	No	3

sources. All had eaten ice cream but there was no common supplier. Three children ate sliced ham and coleslaw, but these were purchased from different shops.

The number of days between the onset of illness and interview ranged between nine and 24 days and several mothers found it difficult to remember details of exposures.

Microbiological

E. coli O157 phage type (PT) 2 was isolated from faecal specimens from five cases. The strains were indistinguishable using plasmid analysis and Vero toxin (VT) gene analysis with DNA probes. The pattern identified was a plasmid of 56 megadaltons and VT 2. *E. coli* O157 infection in the other case (case 3 in the table) was confirmed by serological tests indicating the presence of antibodies to *E. coli* O157 lipopolysaccharide. Faecal specimens from the mother of this case (a secondary case) revealed that she had been infected with *E. coli* O157 PT 2, although further analysis to identify plasmid and Vero toxin type was not performed on this specimen.

Environmental

Food

Four London boroughs took part in a sampling exercise between 2 and 26 August. Sixty samples of fromage frais or yoghurt and 33 meat samples were examined; none grew *E. coli*.

Paddling pools

Seven local paddling pools (including pools X and Y) were inspected and procedures for maintenance and disinfection were reviewed. Written guidelines that had been prepared and issued locally were not being followed by many of the park keepers and many were unaware of their existence. The guidelines themselves were inadequate. For example, one set of guidelines recommended dosing with disinfectant (hypochlorite) at three hourly intervals but subsequent testing showed that chlorine levels dropped below the minimum acceptable concentration of 1 part per million (ppm) after only 90 minutes.

Chemical results: 18 samples of water taken between 15 June and 2 September from six pools in the area were measured for their free chlorine levels. The chlorine concentration was unsatisfactory (<1ppm) in nine of the 18 samples. Water samples from pool X were unsatisfactory on 23 July and 3 August and a sample taken from pool Y was unsatisfactory on 21 June.

Microbiological results: 10 samples were taken from four pools (including pools X and Y) between 23 July and 2 September. *E. coli* (not O157) was detected in four of the samples. The chlorine level was <1ppm in all four

contaminated samples. Water samples from pool X showed that *E. coli* was present on the two occasions (23 July and 3 August) when the chlorine level was <1ppm.

Discussion

This was a small outbreak of *E. coli* O157 infection (five of the six confirmed as PT 2) in a geographically defined area. PT 2 is one of the commonest phage types in England and Wales¹⁰. The isolates were found to be indistinguishable using plasmid analysis but about half of all PT 2 strains in England and Wales have the particular plasmid pattern found, this being one plasmid of 56 megadaltons (Dr H Smith, personal communication). Isolates were also indistinguishable using VT gene analysis, but workers at LEP have reported that 390 (99%) of 394 PT 2 strains examined had VT2 only¹¹. It was difficult, therefore, to conclude with certainty that the strains were 'identical'.

It is possible that some cases may not have been identified. General practitioners practising outside the area were not informed by letter about the outbreak, unlike those in the locality. Furthermore, mild symptoms and signs caused by *E. coli* O157 but unaccompanied by bloody diarrhoea may not have been reported or diagnosed. Laboratories in this area examined only bloody stools for *E. coli* O157.

There was epidemiological and supportive microbiological evidence that a paddling pool acted as a focus for infection in three cases in this outbreak. It is possible that cases 2 and 3 and possibly case 1 all acquired infection at pool X. Case 1 may have contaminated the pool, giving rise to subsequent infection in cases 2 and 3.

Disinfection and maintenance was inadequate at many paddling pools in the local area (including pool X). Although *E. coli* (not O157) was isolated from pool X visited by three cases (1, 2, and 3) the earliest sample was taken on 23 July. The finding was confirmed in a later test on 3 August and we believe that conditions in early July are likely to have been similar, as the tests were carried out before any recommendations to improve maintenance were implemented. Guidance on the quality of pool water (including paddling pools)¹² recommends that *E. coli* should be absent from all water samples.

A link between *E. coli* O157 and a plastic paddling pool has recently been reported in a study of an outbreak in Scotland¹³, and natural bodies of water have also been implicated¹⁵.

Evidence for the role of the environment in facilitating transmission of *E. coli* O157 has also come from a report of illness that followed eating vegetables that had been manured and then inadequately washed before consumption¹⁵. The manure came from cattle that were

found to have antibodies to *E. coli* O157 lipopolysaccharide, and *E. coli* O157 was isolated from manured soil in the garden where the vegetables were grown.

Contaminated food appears to be a major vehicle of transmission of *E. coli* O157⁴. Products implicated have included milk, milk products, and inadequately cooked minced beef. Outbreaks associated with beef have included a school outbreak in Minnesota in 1988 associated with precooked meat patties¹⁶ and an outbreak in California in 1993 linked to a hamburger cooked at home¹⁷ (where there was microbiological evidence of contamination of ground beef). An outbreak in north west England in 1991 was associated with eating yoghurt¹⁸. Our outbreak revealed no clear indication of a likely common food source, although two cases were reported to have eaten the same brand of fromage frais. Our study was uncontrolled, however, and it was impossible to judge how commonly young children in the area ate this brand or indeed how often they visit paddling pools.

Person to person transmission of *E. coli* O157 infection can occur⁴ and there was evidence of this in our outbreak. A mother who had been caring for her sick child (case 3) became ill nine days after her child became ill.

In conclusion, it is likely that at least three of the children in this outbreak became infected while playing in a local paddling pool where disinfection procedures were found to be inadequate. We recommend that this type of exposure be sought when investigating sporadic cases of *E. coli* O157 infection in young children.

One of the difficulties encountered in this investigation was the failure of recall due to time between the onset of illness and interview. We recommend that cases are investigated promptly so that any links between them can be determined as soon as possible and appropriate control measures can be instituted as necessary. Prompt investigation depends critically upon early laboratory confirmation and reporting to the local CCDC, who should act promptly in response.

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References

1. Konowalchuk J, Speirs JI, Stavric S. Vero response to a cytotoxin of *Escherichia coli*. *Infect Immun* 1977; **18**: 775-9.
2. Riley LW, Remis RS, Helgerson SD, McGee HB, Wells JG, Davis BR, et al. Haemorrhagic colitis associated with a rare *Escherichia coli* serotype. *N Engl J Med* 1983; **308**: 681-5.
3. Karmali MA, Steele BT, Petric M, Lim C. Sporadic cases of haemolytic uraemic syndrome associated with faecal cytotoxin and cytotoxin producing *Escherichia coli* in stools. *Lancet* 1983; **i**: 619-20.
4. Advisory Committee on the Microbiological Safety of Food. *Report on Vero cytotoxin producing Escherichia coli*. London: HMSO, 1995.
5. Rawaf S. *For better health: annual report of the Department of Public Health Medicine, Merton and Sutton Health Authority*. Canterbury: Parkers, 1993.
6. Laboratory of Enteric Pathogens, PHLS Central Public Health Laboratory, Colindale, unpublished data.
7. Department of Health. *Public health common data set 1994: England, volume III*. Guildford: Institute of Public Health, University of Surrey, 1995.
8. March SB, Ratnam S. Sorbitol - MacConkey medium for detection of *E. coli* O157:H7 associated with haemorrhagic colitis. *J Clin Microbiol* 1986; **23**: 869-72.
9. Smith HR, Scotland SM. Methods to provide evidence of infection by Vero cytotoxin producing *Escherichia coli*. *PHLS Microbiology Digest* 1990; **7** (4): 128-32.
10. Frost JA. Phage typing of *Escherichia coli* O157 isolated in the UK. *PHLS Microbiology Digest* 1990; **7** (4): 154-6.
11. Frost JA, Cheasty T, Thomas A, Rowe B. Phage typing of Vero cytotoxin producing *Escherichia coli* O157 isolated in the United Kingdom: 1989-91. *Epidemiol Infect* 1993; **110**: 469-75.
12. Pool Water Treatment Advisory Group. *Pool water guide. The treatment and quality of swimming pool water*. Diss: Micropress Printers Ltd, 1995.
13. Brewster DH, Brown MI, Robertson D, Houghton GL, Bimson J, Sharp JCM. An outbreak of *Escherichia coli* O157 associated with a children's paddling pool. *Epidemiol Infect* 1994; **112**: 441-7.
14. Keene WE, McAnulty JM, Hoesly FC, Williams LP Jr, Hedberg K, Oxman GL, et al. A swimming associated outbreak of hemorrhagic colitis caused by *Escherichia coli* O157:H7 and *Shigella sonnei*. *N Engl J Med* 1994; **331**: 579-84.
15. Cieslak PR, Barrett TJ, Griffin PM, Gensheimer KF, Beckett G, Buffington J, Smith MG. *Escherichia coli* O157: H7 infection from a manured garden. *Lancet* 1993; **342**: 367.
16. Belongia EA, MacDonald KL, Parham GL, White KE, Korlath JA, Lobato MN, et al. An outbreak of *Escherichia coli* O157:H7 colitis associated with consumption of precooked meat patties. *J Infect Dis* 1991; **164**: 338-43.
17. Turney C, Green-Smith M, Shipp M, Mordhorst C, Whittingslow C, Brawley L, et al. *Escherichia coli* O157 outbreak linked to home cooked hamburger - California, July 1993. *MMWR* 1994; **43**: 213-5.
18. Morgan D, Newman CP, Hutchinson DN, Walker AM, Rowe B, Majid F. Verotoxin producing *Escherichia coli* O157 infections associated with the consumption of yoghurt. *Epidemiol Infect* 1993; **111**: 181-7.

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Managing swimming, spa, and other pools to prevent infection

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Summary

Many different kinds of recreational and therapeutic pools now exist and all have the potential to transmit infection. This paper offers a brief review of reports of pool associated infections, along with short descriptions of the different kinds of pool and of the disinfection and filtration systems designed to minimise the possible transmission of infection by pool water. An outline of pool monitoring is included together with the rationale of microbiological testing and current microbiological guidelines. Good management is important; a well managed pool with an adequate disinfectant level, a pH value within the recommended range, regular filter backwashing, and satisfactory microbiological parameters will not present a significant infection risk. If management or design is poor, conditions can occur in which bathers become infected. Most infections reported in association with pools happen in these circumstances.

Introduction

Bathing in one form or another is a popular means of recreation, which can provide exercise and relaxation but is sometimes associated with the acquisition of infections. Swimming pools are found in many schools, leisure complexes, health clubs, hotels, and in homes. As well as traditional rectangular pools with deep and shallow ends there are leisure pools of irregular shape, which may have complex features such as wave machines, flumes (large tubular slides), 'beaches', and even 'islands' with vegetation; splash pools; level deck pools; competition pools; diving pools; learner pools; paddling pools; salt water pools; and domestic (often outdoor) pools. There are therapeutic pools, such as hydrotherapy pools and natural thermal pools. More recently, a range of smaller pools - spa or whirlpool, hot tub, plunge pool, birthing pool, whirlpool bath, and flotation tank - have all appeared (box).

Disinfection and filtration - the circulation system

Any pool may transmit infection. Most pools therefore incorporate some form of disinfection system, which is often automatic but may be manual in some smaller pools. Chlorine, a powerful oxidising agent, is the most commonly used disinfecting agent. It can be supplied as a gas, as sodium or calcium hypochlorite, or as a chlorinated isocyanurate. The active disinfectant in each case is hypochlorous acid, which combines with pollutants to form various compounds, chiefly chloramines, known as 'combined chlorine'. These are responsible for most of the irritant effects of chlorine disinfected pools. If sufficient hypochlorous acid is added a 'breakpoint' is reached at which, with the pollutants 'mopped up', no more combined chlorine can be formed and the hypochlorous acid is free to act as a disinfectant. This concentration of 'free chlorine' is often referred to as the 'disinfectant residual' and should be maintained at an adequate level (at least twice the

combined chlorine level) to deal with any added pollutants from bathers. The sum of the free and combined chlorine is known as the 'total chlorine'. Free and total chlorine are measured routinely during pool use.

Systems using chlorine gas or a hypochlorite are essentially similar: the disinfectant is injected into the circulation system and chemicals are added to correct the pH. Chlorinated isocyanurates stabilise the free chlorine in the presence of ultraviolet light, and are therefore particularly useful for outdoor pools, but the level of cyanuric acid (a dissociation product) must be carefully controlled otherwise it will tend to neutralise the free chlorine.

Chlorination is now often combined with ozonation, which removes many of the pollutants. Combined chlorine levels can be kept low, with improved comfort for bathers. Such systems are more complex than other chlorine systems and require a carbon filter to remove residual ozone from the treated water before it is returned to the pool.

Bromine based disinfectants such as bromo-chlorodimethylhydantoin (BCDMH) are also quite often used. The active disinfectant is hypobromous acid which acts in a similar way to hypochlorous acid. The bromamines formed have disinfecting properties. Skin irritation occurs more often in pools using BCDMH than with chlorine.

Other less well tried systems include a form of chlorine dioxide, ultraviolet radiation with a chlorine residual, and metallic (copper/silver) ions with chlorine. Many small domestic pools use polymeric biguanide, which has a disinfecting action but does not remove organic pollutants: it is unsuitable for heavily used pools.

In laboratory tests the disinfecting activities of chlorine, bromine, BCDMH, iodine, chlorine dioxide, and ozone against *Staphylococcus aureus* have been compared under standard conditions. Ozone was by far the most effective; followed by chlorine, bromine, BCDMH, and chlorine dioxide, all of which had similar levels of activity; iodine was the least effective¹.

Pools used by more than one person at a time also incorporate a filtration system, usually in the form of one or more pressure sand filters. The filtration rate affects the efficiency of the filter - the greater the velocity, the less efficient the filter. Medium rate filters with a filtration velocity of around 10 to 25 metres/hour are the most satisfactory for large, popular pools. High rate filters (above 30 metres/hour) are more likely to allow particulate matter to pass through and should be considered only for small pools used by few bathers. Other materials, such as diatomaceous earth or fibre (as a cartridge or pad), can be used for small pools but require more attention.

Filters remove particulate matter. They also remove colloidal material if a coagulant such as alum is used. A filter is an important means of removing microorganisms such as giardia and cryptosporidium which are relatively or completely resistant to disinfectant. To be fully effective, filters must be backwashed regularly. This is a process in which the flow of water through a filter is reversed and allowed to run to waste. This cleans the filter and allows the pool water to be diluted with fresh water from the

Box**Types of pools**

Splash pool: designed to receive bathers descending a large water slide.

Level deck (or deck level) pool: the water is level with the surround so that much of the surface water, where pollution is greatest, spills over the edge into a channel and thence to a balance tank, which maintains the pool water at a constant level. In many systems, a proportion of the balance tank water returns to the pool through the circulation system (see below).

Competition pool: at least 25 metres long and 1.0 - 1.8 metres deep, marked out into lanes.

Diving pool: deep enough to ensure that diving in will be safe - the depth varies according to the height of the springboard.

Learner pool: generally smaller than competition pools; maximum depth of 0.9 metres.

Paddling pool: small shallow pool for small children. Pollution levels are often high.

Salt water pool: made with corrosion resistant materials; may contain seawater.

Hydrotherapy pool: found in many hospitals. Relatively small, with warm water and special features to facilitate physiotherapy.

Spa pool: designed for sitting in rather than swimming and usually holds up to six people at a time. Water is held at a temperature of 30 to 42°C, and pumped with hydrojets and air induction systems. Includes a disinfection and filtration system. Often called whirlpool spas or 'jacuzzis' (a trade name for one particular type of spa pool).

Whirlpool bath: similar hydrojet and aeration systems to a spa pool, but is designed to hold only one person at a time; there is no disinfection system. Water is drained and refilled between each user.

Hot tub: a form of spa pool made from wood.

Plunge pool: often found in health complexes. Filled with cold water to cool the bathers after a sauna session. It may be large enough for swimming or able to hold only one person.

Birthing pool: small pool of warm water, intended to provide relaxation for women in labour.

Flotation tank: the solitary user floats, often in the dark, in a strong solution of magnesium sulphate. They are intended to induce complete relaxation.

mains supply, so helping to remove undesirable pollutants. Inadequately maintained filters can allow a variety of microorganisms, including *Pseudomonas aeruginosa*, to proliferate, with corresponding contamination of the pool water. Filters that contain activated carbon, used to remove ozone, seem particularly susceptible to microbial colonisation.

The disinfection and filtration systems form part of the pool circulation system - water from the pool is pumped through the filters and returned to the pool with added disinfectant. More detailed information about disinfection and filtration systems can be found in the pool water guide².

Pool associated infections

Infections derived from pool water are comparatively uncommon, considering how often pools are used. They may be grouped into infections of the skin, the respiratory tract, the gastrointestinal tract, and - less commonly - the urinary tract and central nervous system. Reports of the pool associated infections mentioned below have been reviewed elsewhere^{3,4}, unless otherwise indicated.

Skin infections

Skin infection with *Pseudomonas aeruginosa* has often been reported. It usually presents as an erythematous body rash in the form of a folliculitis, which may be pustular in severe cases. Sometimes mastitis is present. The onset is

more than 12 (and usually at least 24 hours) after exposure. This distinguishes it from irritant rashes associated with the disinfectant, which appear sooner⁵. The infection is self limiting in previously healthy people and clears in about a week. This form of pseudomonal infection is usually associated with spa pools, where the relatively high temperature and agitation of the water make the skin more susceptible, as well as enhancing the organism's proliferation. Proliferation is also assisted by the relatively high bathing load, which places greater demands on the disinfection system.

Otitis externa is the other common presentation of *P. aeruginosa* infection, and tends to be associated with swimming pools where heads are immersed⁵. Many pool infections have been associated with serotype 011 and it has been suggested that this serotype has a greater pathogenic potential for the skin, but serotypes 04, 09, and 010 have also been isolated from skin and ear infections in bathers⁶. This organism also causes problems with whirlpool baths⁷.

Outbreaks of swimming pool granuloma caused by *Mycobacterium marinum* have been reported, usually where the pool surface has been cracked and roughened, providing a suitable environment for the organism to proliferate.

Fungal infections of the feet and the viral infection that

causes plantar warts have been associated with swimming pool use but the vehicle of transmission is contaminated floor surfaces around the pool rather than water.

Respiratory infections

Respiratory infections associated with pools include adenoviral infection of the throat and eyes, and one report of *P. aeruginosa* pneumonia in a spa pool user. Conjunctivitis associated with swimming pools is often caused by chemical irritation rather than infection. Infective conjunctivitis is more likely to be spread from person to person by sharing towels. Otitis media has been associated with swimming, but the infection arises from the pharynx through the eustachian tube, and probably results from pressure changes during swimming and especially diving. An unusual respiratory infection with *Mycobacterium chelonae* in children with cystic fibrosis using a poorly maintained hydrotherapy pool has been reported⁸.

Legionella pneumophila is a hydrophilic microbe. Outbreaks of legionnaires' disease and of Pontiac fever⁹ have occurred in spa pool bathers, where the agitation and aeration of the water provide conditions that enable the organism to be inhaled. A Pontiac fever-like illness was also related to the use of a spa pool contaminated with *Legionella micdadei*¹⁰.

Gastrointestinal infections

Given the possibility of faecal contamination it is not surprising that gastrointestinal infections have been associated with pool use. Giardiasis and cryptosporidiosis as a result of swimming in contaminated swimming pools have been reported^{11,12}. Outbreaks of infection with Norwalk¹³ and hepatitis A viruses¹⁴ have been associated with using a swimming pool.

Urinary tract infections

Urinary tract infections are not commonly associated with pool use but urinary infection with *Pseudomonas aeruginosa* has been reported in a spa pool user.

Central nervous system infections

Infections of the central nervous system are not usually associated with pools. The exception is primary amoebic meningoencephalitis, which has occasionally been reported in users of warm water pools.

Other infections

It might be thought that bloodborne infections such as hepatitis B virus and HIV infection could be transmitted in pools, but transmission by this route has not been reported.

Pool associated infections are almost invariably accompanied by evidence that the pool concerned was contaminated as a result of inadequate or no disinfection. Pool associated infections may arise as a result of active proliferation of the organism in the pool water and circulation system, which occurs with *P. aeruginosa*, mycobacteria, and legionellas, or as a result of passive transmission of the organism as with giardia, cryptosporidium, and viruses. A well managed pool with an adequate disinfectant residual, satisfactory water chemistry, and well maintained filters should not be capable of transmitting infection.

Pool water monitoring

Chemical and bacteriological monitoring of pool water is essential to protect the health of bathers. The disinfectant level (free chlorine and combined chlorine or total bromine) and the pH should be monitored at frequent intervals during the day when the pool is in use. The pH is important because disinfectant activity will be impaired if the pH value is not kept within the recommended range: 7.2 to 7.8 (for most disinfectants). If these two parameters are satisfactory, and the filters backwashed regularly, significant microbial contamination is unlikely. It is nevertheless advisable to sample pool water for bacteriological testing once a month as a routine (weekly for hydrotherapy pools), or more often if there is evidence of disinfection failure or infections in the bathers.

As some water is likely to be swallowed during swimming, bacteriological standards for pools have evolved from those appropriate for drinking water. In the past examination for faecally excreted organisms such as coliforms has been emphasised, especially *Escherichia coli*. Microorganisms from the skin and respiratory tract are also shed into the pool. To gain a more complete picture of pool bacteria, the total viable (colony, or plate) count has been found to give useful information¹⁵. A raised count indicates microbial proliferation in the pool water and circulation system, which results from poor management.

The association of *P. aeruginosa* with pool infections has made testing for the presence of this bacterium become commonplace, but most reported infections have occurred in spa pools or hydrotherapy pools where water temperatures are higher than in swimming pools. Testing routinely for *P. aeruginosa* in swimming pool water is unlikely to yield more information than the colony count, but it is advisable to include this test as a routine for spa and hydrotherapy pools, where health risks are greater. As with the raised colony count, the presence of *P. aeruginosa* indicates proliferation within the pool water system.

The presence of *P. aeruginosa* in spa or hydrotherapy pools is clearly undesirable, but the position with swimming pools is less clear cut. At times pools have been closed down when *P. aeruginosa* has been found, even in small numbers. The United Kingdom Pool Water Treatment Advisory Group has considered this issue and advised that any corrections needed to disinfectant levels and pH should be made first, and the filters backwashed. Closing the pool should be considered only if the organism persists after such measures and after consultation with the local consultant in communicable disease control and microbiology laboratory¹⁶. As mentioned above, the presence of *P. aeruginosa* presents less of a health risk to swimmers than to users of spa or hydrotherapy pools. Published reports^{6,17,18} do not indicate a clear threshold level for *P. aeruginosa* above which there is a definite health risk. Laboratory enumeration of *P. aeruginosa* has only recently become subject to external quality control; comparing counts made in the past by different laboratories therefore requires some caution. Further work will be required to resolve this difficulty.

Staphylococci, and particularly *Staphylococcus aureus*, exhibit a greater resistance to pool disinfectants than coliforms, and have been proposed as suitable bacteriological indicators of pool water contamination. As

Table Summary of microbiological guidelines for pool water²

Colony counts*	Less than 10 cfu/ml after 24 hours at 37°C
Coliforms (including <i>E. coli</i>)†	Not detected in 100 ml
<i>P. aeruginosa</i> (spa and hydrotherapy pools)	Not detected in 100 ml

* an occasional count between 10 and 100cfu/ml is acceptable provided no coliforms (or *E. coli*) are present and operational conditions are satisfactory.

† occasionally a few (less than 10) coliforms may be found (with no *E. coli*) and a satisfactory colony count. This may be acceptable provided the residual disinfectant concentration and pH value are satisfactory and coliforms are not found in consecutive samples.

they will be shed by bathers some authors consider that they would provide a useful indication of disinfection efficacy¹⁹. One study has shown that they are almost invariably found when bathers are present in the pool and simply indicate what can be observed²⁰.

Microbiological standards

There are no legally enforceable microbiological standards for recreational or therapeutic pool water in the United Kingdom. For swimming pools, an early study by the PHLS Water Sub-committee in 1953 concluded that appropriate guideline levels would be an absence of coliform bacteria in 100 ml and colony counts of less than 10 per ml (at 37°C) in 75% of samples and not more than 100 per ml in 25%²¹. In 1984, the Department of the Environment gave similar levels but allowed the presence of a few coliforms (up to about 10 organisms per 100ml, but no *E. coli*) provided they were not present in consecutive samples, disinfectant residuals and pH values were within accepted ranges, and the colony count was within normal limits¹. More recently, these guidelines have been updated by the Pool Water Treatment Advisory Group and include recommendations for spa and hydrotherapy pools² (table). The latter are the same as those in the PHLS publications *Hygiene for Spa Pools*²² and *Hygiene for Hydrotherapy Pools* (in preparation).

References

- Department of the Environment. *The treatment and quality of swimming pool water*. London: HMSO, 1984.
- Pool Water Treatment Advisory Group. *Pool water guide: the treatment and quality of swimming pool water*. Diss, PWTAG, 1995.
- Galbraith, NS. Infections associated with swimming pools. *Environmental Health* 1980; **88**: 31-3.
- Jones F, Bartlett CLR. Infections associated with whirlpools and spas. *J Appl Bacteriol* 1985; **59** (suppl): S61-6.
- Penny, PT. Hydrotherapy pools of the future - the avoidance of health problems. *J Hosp Infect* 1991, **18** (suppl A), 535-42.
- Ratman S, Hogan K, March SB and Butler RW. Whirlpool associated folliculitis caused by *Pseudomonas aeruginosa*: report of an outbreak and a review. *J Clin Microbiol* 1986; **23**: 655-9.
- Hollyoak VA, Freeman R. *Pseudomonas aeruginosa* and whirlpool baths. *Lancet* 1995; **346**: 644.
- Basavaraj DS, Hooper WL, Richardson EA, Penny P, O'Mahony M, Begg N. *Mycobacterium chelonae* associated with a hydrotherapy pool. *Communicable Disease Report* 1985; (41): 3-4.
- Bartlett CLR, Macrae AD, Macfarlane JT. *Legionella infections*. London: Edward Arnold, 1986.
- Goldberg DJ, Collier PW, Fallon RJ, McKay TM, Markwick TA, Wrench JG, et al. Lochgoilhead fever: outbreak of non-pneumonic legionellosis due to *Legionella micdadei*. *Lancet* 1989; **i**: 316-8.
- Porter JD, Ragazzoni HP, Buchanon JD, Waskin HA, Juranek DD, Parkin WE. *Giardia* transmission in a swimming pool. *Am J Public Health* 1988; **78**: 659-62.
- Joce RE, Bruce J, Kiely D, Noah ND, Dempster WB, Stalker R, et al. An outbreak of cryptosporidiosis associated with a swimming pool. *Epidemiol Infect* 1991; **107**: 497-508.
- Kappus KD, Marks JS, Holman RC, Bryant JK, Baker C, Gary GW, et al. An outbreak of Norwalk gastroenteritis associated with swimming in a pool and secondary person-to-person transmission. *Am J Epidemiol* 1989; **1169**: 834-9.
- Mahoney F J, Farley TA, Kelso KY, Wilson SA, Horan JM, McFarland LM. An outbreak of hepatitis A associated with swimming in a public pool. *J Infect Dis* 1992; **1659**: 613-8.
- Mood EW. Bacterial indicators of water quality in swimming pools and their role. In: Hoadley AW and Dutka BJ, editors. *Bacterial indicators/health hazards associated with water*. Philadelphia: American Society for Testing and Materials, 1977: 239-46.
- Pool Water Treatment Advisory Group. *Pseudomonas need not close pools*. Press release, June 1994.
- Seyfried PL, Fraser DJ. *Pseudomonas aeruginosa* in swimming pools related to the incidence of otitis externa infection. *Health Laboratory Science*, 1978; **159**: 50-7.
- Price D, Ahearn DG. Incidence and persistence of *Pseudomonas aeruginosa* in whirlpools. *J Clin Microbiol*, 1988; **26**: 1650-4.
- Tosti E, Volterra L. Water hygiene of two swimming pools: microbial indicators. *J Appl Bacteriol* 1988; **65**: 87-91.
- Crone PB, Tee GH. Staphylococci in swimming pool water. *Journal of Hygiene* 1974; **73**: 213-20.
- PHLS Water Subcommittee. The choice of an indicator organism for the bacteriological control of swimming-bath purification. *Monthly Bulletin of the Ministry of Health and the Public Health Laboratory Service* 1953; **12**: 254-67.
- Public Health Laboratory Service. *Hygiene for Spa Pools*. London: PHLS Publications, 1994.

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