

Outbreaks of waterborne infectious intestinal disease in England and Wales, 1992–5

C. FURTADO^{1,2}, G. K. ADAK^{1*}, J. M. STUART³, P. G. WALL¹, H. S. EVANS¹
AND D. P. CASEMORE⁴

¹ PHLS Communicable Disease Surveillance Centre, London

² European Programme for Intervention Epidemiology Training

³ PHLS Communicable Disease Surveillance Centre (South and West), Gloucester

⁴ Cryptosporidium Reference Unit, Public Health Laboratory, Rhyl

(Accepted 23 February 1998)

SUMMARY

Following the introduction of an improved surveillance system for infectious intestinal disease outbreaks in England and Wales, the Public Health Laboratory Service Communicable Disease Surveillance Centre received reports of 26 outbreaks between 1 January 1992 and 31 December 1995 in which there was evidence for waterborne transmission of infection. In these 26 outbreaks, 1756 laboratory confirmed cases were identified of whom 69 (4%) were admitted to hospital. In 19 outbreaks, illness was associated with the consumption of drinking water from public supplies (10 outbreaks) or private supplies (9 outbreaks). The largest outbreak consisted of 575 cases. In 4 of the remaining 7 outbreaks, illness was associated with exposure to swimming pool water. *Cryptosporidium* was identified as the probable causative organism in all 14 outbreaks associated with public water supplies and swimming pools. *Campylobacter* was responsible for most outbreaks associated with private water supplies. This review confirms a continuing risk of cryptosporidiosis from chlorinated water supplies in England and Wales, and reinforces governmental advice to water utilities that water treatment processes should be rigorously applied to ensure effective particle removal. High standards of surveillance are important for prompt recognition of outbreaks and institution of control measures. As microbiological evidence of water contamination may be absent or insufficient to implicate a particular water supply, a high standard of epidemiological investigation is recommended in all outbreaks of suspected waterborne disease.

INTRODUCTION

Between 1911 and 1950, 25 outbreaks of waterborne and water-associated disease were recorded in England and Wales. In these outbreaks, at least 14 181 people reported illness and 171 (1·2%) died. Sixteen out of the 25 outbreaks were due to *Salmonella typhi* or *paratyphi*, 4 to shigella, and 5 were reported as gastro-enteritis. Between 1951 and 1980 the incidence

of reported outbreaks of typhoid and paratyphoid declined dramatically, with only one recorded outbreak of paratyphoid fever during this period. This was attributed to the success of water treatment programmes, particularly chlorination of public water supplies [1, 2].

Since 1980, *Cryptosporidium*, *campylobacter* and *giardia* have emerged as important causes of outbreaks of waterborne disease in England and Wales [3, 4]. In the period 1980–91, 9 of the 17 reported outbreaks of waterborne disease were due to *Cryptosporidium* [3]. With no evidence of a general de-

* Author for correspondence: G. K. Adak, PHLS Communicable Disease Surveillance Centre, 61 Colindale Avenue, London NW9 5EQ, UK.

terioration in the quality of tap water, the observed increase in the reporting of waterborne cryptosporidiosis was considered to be due, at least in part, to improved detection. This was influenced by an increasing awareness which led to and followed the report of a group of experts chaired by Sir John Badenoch [5].

In 1990 the Committee on the Microbiological Safety of Foods (Richmond Committee) recommended that more accurate and detailed information should be collected on outbreaks of infectious intestinal disease (IID) [6]. In response to this recommendation, the Public Health Laboratory Service (PHLS) Communicable Disease Surveillance Centre (CDSC), in collaboration with consultants in communicable disease control (CCDCs), developed an enhanced surveillance system for outbreaks of IID. The system was introduced at the beginning of 1992.

We review the microbiological and epidemiological features of outbreaks of waterborne IID in England and Wales in the 4 years 1992–5, and discuss possible contributory factors.

METHODS

Outbreak ascertainment

Consultants in communicable disease control, regional epidemiologists, environmental health officers, clinical microbiologists and hospital control of infection officers were encouraged to telephone the Gastrointestinal Diseases Section, PHLS CDSC with preliminary details of outbreaks of IID under investigation. Outbreaks of infection were also detected through analysis of weekly computer summaries from the national laboratory reporting database [7]. National or localized increases in the reporting of particular pathogens and/or increased age-specific incidence were followed up. PHLS and NHS laboratories reported outbreaks to CDSC via specific laboratory report forms. Outbreaks identified by PHLS reference laboratories were reported to CDSC in regular meetings or through specific reference laboratory report forms. Information on outbreaks reported to the Department of Health was routinely copied to CDSC to ensure that surveillance was as complete as possible.

Data collection and analysis

When a report was received from any source, a questionnaire was sent from CDSC to the appropriate

CCDC with a request that it should be completed by the lead investigator at the end of their investigation. The questionnaire was designed to collect the following information on each suspected outbreak in a uniform manner: mode of transmission; setting; causative organism; test laboratory; number of cases (as defined by the outbreak control team); number of people at risk; number of cases admitted to hospital; number of deaths; number of laboratory confirmed cases; dates of onset; suspected vehicles of infection; evidence implicating vehicles of infection. In addition, a request was made to the investigating CCDC to return a copy of the local investigation report when it became available. Data from returned questionnaires were entered onto a database [8].

Definitions

Waterborne outbreak. An outbreak of IID for which there is epidemiological or microbiological evidence showing water to be the likely vehicle of infection.

Date of onset of outbreak. The earliest date of onset of symptoms in identified cases.

Duration of outbreak. The interval between the onset of symptoms in the first and last case.

Total cases. The number of individuals with symptoms conforming to the case definition for the outbreak.

Strength of association. Outbreaks were classified into strong, probable or possible association with water according to epidemiological and microbiological evidence [9].

Public supply. Water supply operated by a water utility/ company.

Private supply. Water supply, usually small, not owned or operated by a water utility/ company.

RESULTS

In the period between 1 January 1992 and 31 December 1995, CDSC received reports of 2636 outbreaks of IID in England and Wales. Outbreak summary forms providing a minimum dataset of information were returned for 2090 of these outbreaks. Waterborne transmission of disease was suspected in 26 (1.2%). In 19 (73%) of the waterborne outbreaks, illness was associated with the consumption of drinking water (10 outbreaks with water from public supplies and nine from private supplies). Exposure to swimming pool water was associated

Table 1. Outbreaks associated with the consumption of drinking water from public supplies, England and Wales, 1992-5

Outbreak number	Date of outbreak	Pathogen	Region	Duration (days)	Cases		Hospitalized	Epidemiological evidence (type of study)	Water microbiology	Strength of association
					Total	Laboratory confirmed				
1	Apr 1992	<i>Cryptosporidium</i> sp.	Northern	60	63	8	Case-control	Negative	Probable	
2	Oct 1992	<i>Cryptosporidium</i> sp.	Yorkshire	41	125	9	Case-control	<i>Cryptosporidium</i> sp.	Strong	
3	Nov 1992	<i>Cryptosporidium</i> sp.	Mersey	85	47	5	Case-control	Negative	Probable	
4	June 1992	<i>Cryptosporidium</i> sp.	S. Western	168	108	NR*	Case-control	Negative	Probable	
5	Apr 1993	<i>Cryptosporidium</i> sp.	Wessex	32	40	4	Case-control	Negative	Probable	
6	June 1993	<i>Cryptosporidium</i> sp.	Yorkshire	34	97	1	Case-control	Negative	Probable	
7	July 1993	<i>Cryptosporidium</i> sp.	Wessex	75	27	0	Case-control	Negative	Probable	
8	Aug 1994	<i>Cryptosporidium</i> sp.	S. England	153	224	5	Case-control	<i>Cryptosporidium</i> sp.	Strong	
9	Oct 1994	<i>Cryptosporidium</i> sp.	Trent	NR*	33	0	Descriptive	Negative	Possible	
10	July 1995	<i>Cryptosporidium</i> sp.	S. Western	63	575	25	Descriptive	<i>Cryptosporidium</i> sp.	Strong	

* NR, Not reported.

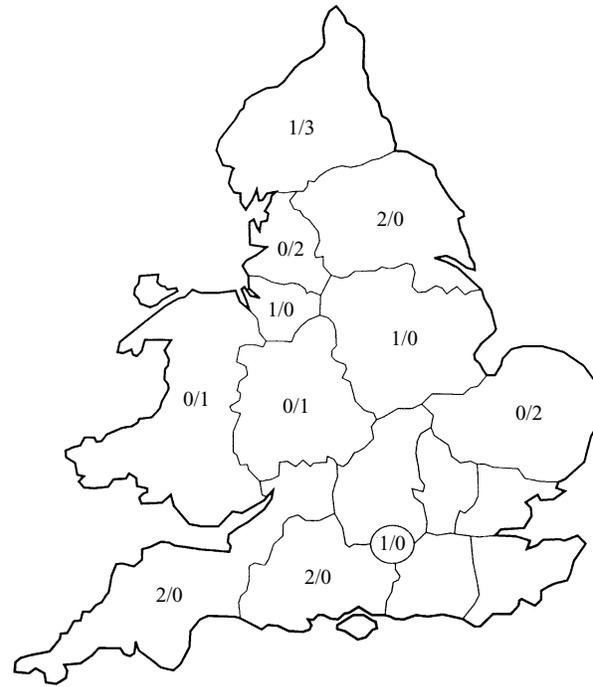


Fig. 1. Geographical distribution of outbreaks of IID associated with the consumption of water from public and private supplies in England and Wales. Numbers indicate public supplies/private supplies, 1992-5.

with illness in 4 (15%) outbreaks. One outbreak was found to be associated with the consumption of drinks containing contaminated ice, another with consumption of water from a sewage treatment plant and a third outbreak was found to be associated with immersion in river water while canoeing. The median number of laboratory confirmed cases per outbreak was 38 (range 3-575). No deaths were reported.

Outbreaks associated with the consumption of drinking water from public supplies

In 10 outbreaks, epidemiological and microbiological investigations suggested that the vehicle of infection was unboiled tap water from chlorinated public water supplies (Table 1). In nine outbreaks, the strength of association was strong or probable. *Cryptosporidium* was found to be the causative organism in all ten outbreaks. The median number of confirmed cases per outbreak was 80 (range 27-575). The hospital admission rate was 4.2% (57/1339), range 0-25. Outbreaks were not concentrated in any particular area (Figure 1). One outbreak affected individuals in three regions, South West Thames, Wessex and Oxford. No outbreaks starting in the first quarter of the year were reported.

Analytical epidemiological investigations were carried out in 8 of the 10 reported outbreaks. Cryptosporidial oocysts were isolated from treated water in three outbreaks.

Outbreak 1

Date: April 1992. Setting: a rural community in Northern region. A case-control study demonstrated a positive association between illness and the consumption of unboiled domestic tap water from a filtered and chlorinated public supply. A clear dose response relationship was also demonstrated (OR range 5.61–30.61; *P*-value 0.002). No cryptosporidial oocysts were identified in pre-treatment surface supplied waters tested by the local water company or in post-treatment waters examined by the local PHL during the outbreak.

Outbreak 2

Date: October 1992. Setting: a mainly urban community in Yorkshire region. A positive association between cryptosporidiosis and the consumption of unboiled filtered and chlorinated tap water from a public surface water supply was demonstrated (OR 13.5, 95% CI 3.8–51.7; *P*-value 0.000002). Cryptosporidial oocysts were identified in treated and raw water samples and also in a sample of sand taken from the filter beds at the implicated treatment plant.

Outbreak 3

Date: November 1992. Setting: a mainly urban community in one health district in Mersey region. A case-control study demonstrated a positive association between illness and the consumption of unboiled filtered and chlorinated tap water from a public ground water supply. A clear dose response relationship was also demonstrated (OR range 8.1–45.5; *P*-value < 0.01). Prior to the outbreak there was no routine testing of raw water from the implicated supply, as this is not recommended practice for ground water sources. No cryptosporidial oocysts were identified in samples of reservoir sediment, raw or treated water taken during the outbreak investigation.

Outbreak 4

Date: June 1992. Setting: a seaside town in South Western region. A case-control study demonstrated a

positive association between illness and the consumption of filtered and chlorinated tap water from a public supply. A dose response relationship was also demonstrated (OR range 1.09–8.32; *P*-value 0.06). No cryptosporidial oocysts were identified in a variety of samples taken from the implicated treatment plant or from treated water taken during the outbreak investigation. The plant had a surface water supply.

Outbreak 5

Date: April 1993. Setting: area around a coastal town in Wessex region. A case-control study demonstrated a positive association between illness and the consumption of chlorinated tap water from a public ground water supply. A dose response relationship was also demonstrated (OR range 4.4–13.8; *P*-value 0.05). Cryptosporidial oocysts were identified at low levels on four occasions in a service reservoir after the peak of the outbreak. Data from water company records showed no positive identification of oocysts from treated water either before or during the outbreak [10].

Outbreak 6

Date: June 1993. Setting: the same community as outbreak 2. A case-control study demonstrated a positive association between cryptosporidiosis and the consumption of filtered and chlorinated tap water from one local public supply (*P*-value 0.000001). No cryptosporidial oocysts were identified in a variety of samples taken from the implicated treatment plant or from treated water taken during the outbreak investigation. Water company records showed that the quality of raw surface water entering the treatment works in the period at the time of the outbreak was exceptionally poor as measured by coliform counts. In addition there was a chlorination failure at the treatment works which led to unchlorinated water entering the system for a period of over 10 h during the period of the outbreak.

Outbreak 7

Date: July 1993. Setting: an urban community in Wessex region. A case-control study demonstrated a positive association between illness and the consumption of filtered and chlorinated tap water from a public surface supply. A dose response relationship was also demonstrated (no OR or *P*-values given in report). Data from water company records show no

positive identification of oocysts from treated water either before or during the outbreak.

Outbreak 8

Date: August 1994. Setting: mixed urban and rural communities in a wide area of South East England. The affected communities were served by a number of different water companies using several water treatment plants. A case-control study demonstrated a positive association between illness and the consumption of filtered and chlorinated tap water from public supplies deriving some of their water from a common river source. A dose response relationship was also demonstrated (OR range 1.86–5.03; *P*-value 0.03). Low levels of contamination of raw and treated water with cryptosporidial oocysts were recorded from some of the works on a small number of occasions in the period before and during the outbreak.

Outbreak 9

Date: October 1994. Setting: a small community in Trent region. All the cases lived in the same public water supply zone. No cryptosporidial oocysts were identified in samples taken from a storage reservoir or from filtered and chlorinated water taken during the outbreak investigation. The plant had a surface water supply.

Outbreak 10

Date: July 1995. Setting: the same community as in outbreak 4. A descriptive study showed an association between illness and the consumption of filtered and chlorinated tap water from the same public surface supply implicated in the earlier outbreak. The highest attack rates were recorded amongst those served by the trunk main compared with those who lived outside the area (RR 9.8; *P*-value < 0.000001). Cryptosporidial oocysts were identified in samples of treated water taken during the outbreak investigation.

Outbreaks associated with the consumption of drinking water from private supplies

In 9 outbreaks, epidemiological and microbiological investigations suggested that the vehicle of infection was tap water from private water supplies (Table 2). In seven, the association was considered strong or probable. The median number of cases per outbreak

was 36 (range 8–56). Two hospital admissions were reported, both from the same outbreak. Illness was found to be caused by infection with campylobacter strains in 6 of the 8 outbreaks where a pathogen was identified. The geographical distribution of the outbreaks is shown in Figure 1.

As in public water supply outbreaks, none was recorded in the first quarter of the year. Analytical epidemiological investigations were conducted in 2 of the 9 outbreaks. Pathogens or indicator organisms were isolated from water samples in eight of the outbreaks.

Outbreak 11

Date: November 1991. Setting: a rural community in the West Midlands. *Giardia* cysts (3 cysts/100 l) were found in water from a reservoir supplying water to a village. The water supply was found to be irregularly chlorinated. All of the cases live in a single village. Interviews revealed that the only common factor linking the cases was consumption of water from the village's private supply. It is thought that a reservoir had become contaminated as a result of livestock grazing in the area, and that inadequate treatment of the supply allowed cysts to contaminate the drinking water. The water was abstracted originally from a ground water supply.

Outbreak 12

Date: April 1992. Setting: a boarding school in the North Western region. Pupils were found to be suffering from cryptosporidiosis and no cases were identified in the wider local community. The school had a private chlorinated surface water supply. As soon as the outbreak was identified, the school arranged for water from a nearby mains supply to be delivered by tanker. The onset date of the last case was 5 days after the school stopped using its private supply. Environmental investigations suggested that the reservoir supplying the school may have become contaminated with cryptosporidial oocysts from slurry spraying on adjacent land.

Outbreak 13

Date: April 1993. Setting: a birthday party at a farm. *Campylobacter* infection was confined to children who drank water from the farm's private unchlorinated ground water supply. Adults who did not drink this water were unaffected.

Table 2. Outbreaks associated with the consumption of drinking water from private supplies, England and Wales, 1992–5

Outbreak number	Date of outbreak	Pathogen	Region	Duration (days)	Cases			Epidemiological evidence (type of study)	Water microbiology	Strength of association
					Total	Laboratory confirmed	Hospitalized			
11	Nov 1991	<i>Giardia lamblia</i>	W.Midlands	175	31	5	0	Descriptive	<i>Giardia lamblia</i>	Strong
12	Apr 1992	<i>Cryptosporidium</i> sp.	N.Western	24	42	12	0	Descriptive	Negative	Possible
13	Apr 1993	<i>Campylobacter</i> sp.	Northern	2	8	6	0	Descriptive	Not reported	Possible
14	May 1993	<i>Cryptosporidium</i> sp. & <i>Campylobacter</i> sp.	Northern	8	43	5	2	Descriptive	<i>Escherichia coli</i>	Probable
15	Sept 1993	<i>Campylobacter coli</i>	N.Western	10	36	17	0	Descriptive	<i>Campylobacter coli</i>	Strong
16	Apr 1994	<i>Campylobacter jejuni</i>	East Anglia	11	53	14	0	Cohort	<i>Campylobacter jejuni</i>	Strong
17	Sept 1994	<i>Campylobacter</i> sp.	Northern	7	8	2	0	Descriptive	<i>Campylobacter</i> sp.	Strong
18	Sept 1994	<i>Campylobacter jejuni</i>	Wales	61	22	4	0	Cohort	Faecal coliforms	Strong
19	May 1995	None identified	East Anglia	20	56	0	NR*	Descriptive	<i>Escherichia coli</i>	Probable

* NR, Not reported

Outbreak 14

Date: May 1993. Setting: a university hall of residence in the Northern region. Cases of cryptosporidiosis, campylobacteriosis and mixed infection were identified. The descriptive epidemiology suggested that illness was associated with drinking water from the hall of residence's private unchlorinated surface water supply. *Escherichia coli* was isolated from water samples. Dead lambs were found in the water supply system. Although water was treated using filtration and ultra violet light, the effectiveness of the ultra violet lights was impaired due to the accumulation of deposits over the glass coverings.

Outbreak 15

Date: September 1993. Setting: the same boarding school as outbreak 12. Questionnaires returned by the cases indicated that the only common exposure among cases was the consumption of tap water from the school's private surface water supply. This was supported by the isolation of the outbreak strain of *Campylobacter coli* in reservoir water and faecal coliforms in tap water. Testing of the school's tap water showed that chlorine levels were low.

Outbreak 16

Date: April 1994. Setting: a residential holiday centre in East Anglia. A cohort study showed that illness was associated with the consumption of water from the centre's private unchlorinated supply. A dose response effect was recorded (RR 3.36–4.85; *P*-value 0.03). This was supported by the isolation of the outbreak strain of *Campylobacter jejuni* from drinking water samples taken from the centre. The water, from a ground water supply, was not chlorinated and the ultra violet treatment system had not been adequately maintained.

Outbreak 17

Date: September 1994. Setting: a rural community in the Northern region. Illness was confined to the inhabitants of a large house with a private untreated surface water supply, 8/9 of the inhabitants became ill. This was supported by the isolation of *Campylobacter* spp. from water taken from a storage tank in the flats and the presence of *Escherichia coli* in tap water samples.

Outbreak 18

Date: September 1994. Setting: an armed forces training centre in Wales. A cohort study demonstrated a dose-response relationship between illness and drinking untreated tap water from a ground water supply (RR 0.98–2.84; *P*-value 0.003). Faecal coliforms were also isolated from numerous water samples taken from around the training centre.

Outbreak 19

Date: May 1995. Setting: a research institute in East Anglia. The institute had private untreated ground water supply. Water was pumped from two separate wells on alternate weeks with a routine switch on Monday mornings. Water was reported to be tainted following one of the routine switches of supply. In addition there was evidence of leakage from a sewer which ran close to the implicated ground water supply. *Escherichia coli* was isolated from water samples. No causative organism was identified in the cases.

Outbreaks associated with swimming pools

In four outbreaks, illness was linked to the use of swimming pools; the association was classified as strong in one, and possible in the other three (Table 3). *Cryptosporidium* was the causative organism in all four outbreaks. The number of cases per outbreak varied between three and 23 (median 13). Nine patients required hospital admission. Descriptive epidemiological investigations were conducted. *Cryptosporidial* oocysts were identified in swimming pool water samples in one outbreak.

Outbreak 20

Date: March 1992. Setting: a leisure centre in South Western region. Most of the cases were children who had used a learner pool in the leisure centre. *Cryptosporidial* oocysts were identified in one of the three 10 litre water samples taken from the pool [11].

Outbreak 21

Date: January 1993. Setting: a school swimming pool in West Midlands region. Children at the school were interviewed, and the data suggested that there was an association between illness and using the school swimming pool. No further investigations were reported.

Outbreak 22

Date: October 1994. Setting: a leisure centre in South Western region. The descriptive epidemiology suggested an association between illness and using the leisure centre. There was evidence of secondary spread within the community.

Outbreak 23

Date: October 1995. Setting: a paddling pool in Trent region. The descriptive epidemiology suggested an association between illness in children and using the pool. Investigations revealed that children in nappies (diapers) were allowed to use the pool and that supervision and maintenance standards were poor.

Outbreaks associated with the consumption of water from miscellaneous sources

CDSC received reports of three other outbreaks in which water was implicated as the vehicle of infection (Table 4).

Outbreak 24

Date: August 1993. Setting: a wedding reception at a hotel in North East Thames region. Gastro-enteritis was reported in 55 of the 87 people attending the function. No pathogens were isolated from the specimens taken. A cohort study showed an association between illness and the consumption of drinks containing ice (RR 1.65; *P*-value 0.016). Coliforms were isolated from samples taken from the ice machine.

Outbreak 25

Date: April 1994. Setting: a military camp in Wessex. Four soldiers were reported ill with gastro-enteritis. Astrovirus was identified in the faeces of one of the cases. All the affected soldiers had visited the camp's sewage treatment plant where they had drunk partially treated water from a bottle found in one of the buildings. The water was not for consumption and would have been expected to contain microorganisms.

Outbreak 26

Date: October 1994. Setting: a watersports centre in Trent. Seven of 11 members of a party of canoeists became ill with small round structured virus (SRSV)

Table 3. Outbreaks associated with swimming pools, England and Wales, 1992–5

Outbreak number	Date of outbreak	Pathogen	Region	Duration (days)	Cases			Epidemiological evidence (type of study)	Water microbiology	Strength of association
					Total	Laboratory confirmed	Hospitalized			
20	Mar 1992	<i>Cryptosporidium</i> sp.	S.Western	17	12	12	2	Descriptive	<i>Cryptosporidium</i> sp.	Strong
21	Jan 1993	<i>Cryptosporidium</i> sp.	W.Midlands	10	23	21	4	Descriptive	Not reported	Possible
22	Oct 1994	<i>Cryptosporidium</i> sp.	S.Western	31	14	8	0	Descriptive	Not reported	Possible
23	Oct 1995	<i>Cryptosporidium</i> sp.	Trent	5	3	3	3	Descriptive	Not reported	Possible

Table 4. Outbreaks associated with miscellaneous water sources, England and Wales, 1992–5

Outbreak number	Date of outbreak	Pathogen	Region	Duration (days)	Cases			Epidemiological evidence	Water microbiology	Strength of association
					Total	Laboratory confirmed	Hospitalized			
24	Aug 1993	None identified	NE Thames	7	55	0	1	Ice	Faecal coliforms	Strong
25	Apr 1994	Astrovirus	Wessex	1	4	1	0	Partially treated water from a sewage treatment plant	Faecal coliforms	Probable
26	Oct 1994	SRSV	Trent	1	7	3	0	River water	None	Probable

infection. A cohort study showed an association between immersion in river water and illness [12].

DISCUSSION

The introduction of an enhanced surveillance system for outbreaks of IID resulted in a marked increase in the number of outbreaks of waterborne disease ascertained. Twenty six outbreaks were recorded during the period 1992–5, compared with 4, 4 and 9 outbreaks respectively in the three preceding four year periods [3].

Outbreaks associated with the consumption of water from public supplies

All 10 outbreaks associated with consumption of water from public supplies were attributed to infection with *Cryptosporidium parvum*. That no bacterial outbreaks were reported during this period probably reflects the effectiveness of modern water treatment systems in eliminating bacteria from public supplies [1]. However cryptosporidial oocysts are resistant to chlorine at the levels used in water treatment, and filtration remains the only effective method of oocyst removal available to water companies. If there are failures in filtration or if the raw water entering treatment plants is heavily contaminated with cryptosporidial oocysts, the treated water reaching the public may remain contaminated. The surveillance data collected during the period 1992–5 clearly demonstrates that water companies cannot ensure that drinking water is always free of cryptosporidial contamination [13] and that these incidents can result in large numbers of people becoming ill. Indeed the largest of the 2636 outbreaks of IID reported in England and Wales during this 4-year period was probably waterborne (Outbreak 10) with 575 confirmed cases of cryptosporidiosis, 25 (4%) of whom were admitted to hospital. Three other outbreaks of waterborne IID affected more than 100 people.

When outbreaks of disease occur which are suspected to be caused by contamination of public water supplies, outbreak control groups are convened. As part of the investigation process, case definitions are established. These usually specify that cases to be included in the analytical investigation should have laboratory confirmed disease plus appropriate symptoms in which onset dates fall within a defined period. As such when outbreak information is returned to CDSC the number of cases known to be

part of the outbreak will only include the laboratory confirmed cases, as can be seen from Table 1. It is known that some people affected may not consult clinicians and will therefore not have their infection confirmed nor be included in official outbreak figures. Under these conditions it is difficult for local investigators to accurately estimate the number of additional cases that may have occurred in such outbreaks and include these figures in reports to CDSC.

Outbreaks of illness associated with public water supplies are widely perceived as serious events. The emergence of waterborne cryptosporidiosis in Europe and North America is of particular concern given the potentially fatal effects of the disease among the immunocompromised [3, 14, 15]. The need to establish the vehicle of infection to protect the public is a priority to those charged with communicable disease control. Microbiological evidence of contamination of water supplies may not be available unless contamination continues over a long period, and the significance of low levels of cryptosporidial oocysts in water samples is uncertain [4, 13, 16]. When oocysts are recovered from potable water during investigation their presence can rarely be directly related to the presumed time of exposure of cases, even if 'routine' samples were taken. The presence of oocysts can only demonstrate that oocysts are able to penetrate the water treatment system; failure to detect oocysts merely means that they were not detected in that sample, not that they were absent in the water distribution at the relevant time. Similarly, interpretation of viability data is uncertain and little is known of other parasite factors such as origin or relative infectivity of isolates. No supportive microbiological evidence was obtained in seven of these outbreaks even though extensive sampling of water supplies was reported in all but one. This emphasizes the importance of well conducted analytical and/or descriptive epidemiological studies to assess and characterize the risk associated with such outbreaks.

Two treatment plants were implicated in recurrent outbreaks (Nos. 2 and 6; 4 and 10). This may indicate design or management deficiencies or that the raw water entering these plants was at risk from intermittent heavy contamination with cryptosporidial oocysts. The area feeding one treatment plant was subject to heavy rain and storms in the period preceding one of the outbreaks. This highlights difficulties in preventing contamination of treated water supplies with *Cryptosporidium* when raw waters are heavily contaminated. As recommended [5, 13],

continuous assessment of the quality of the water sources and filtration processes is needed, especially in areas where previous outbreaks suggest an increased risk of waterborne cryptosporidiosis, in conjunction with enhanced local surveillance.

Outbreaks associated with the consumption of water from private supplies

Private supplies usually serve small populations such as farms, institutions and small rural communities. Consequently, the number of cases of illness in outbreaks associated with drinking water from private supplies was relatively small. As well defined cohorts are usually affected, it becomes possible to identify early cases who were sampled after they had ceased to excrete pathogens. Therefore in these outbreaks the reported number of individuals affected tends to exceed the number reported with laboratory confirmed infection. *Campylobacter* was the commonest identified pathogen. This was in marked contrast with the absence of bacterial pathogens in samples from public water supplies associated with outbreaks. All but one of outbreaks associated with private water supplies was attributable to poor maintenance of equipment, particularly systems using ultra violet light, or inadequate treatment procedures. The remaining outbreak (No.12) was thought to have occurred because slurry spraying led to heavy contamination of a water source with cryptosporidial oocysts. Private suppliers do not have the same level of resources as their public counterparts to support rigorous maintenance regimes [17], and many private supplies are not chlorinated [18]. If treatment equipment fails in unchlorinated supplies or if chlorine levels fall below recommended levels, there will be no barrier against bacterial pathogens that may be in the raw water, either from agricultural run off or from sewage ingress. It is likely that most of these outbreaks could have been prevented if the appropriate water treatment and monitoring procedures [18] had been followed.

Microbiological evidence of contamination of water supplies was obtained in 7 of the 9 outbreaks. In 2 of these 7 outbreaks supporting evidence of an association between illness and the consumption of water was obtained from cohort studies. In the two remaining outbreaks, no pathogen was identified in water samples, case-control or cohort studies were not undertaken, and the association was classified as possible. The need to initiate analytical epidemio-

logical studies is clearly greater in the absence of identified problems with the water, its supply or treatment.

Outbreaks associated with swimming pools

Four outbreaks were reported to be associated with using swimming pools. The causative organism in each of these outbreaks was *Cryptosporidium parvum*. Nine out of 52 cases were admitted to hospital. Although proper management and maintenance procedures, including continuous filtration [19, 20], should reduce the risk of prolonged contamination of swimming pool waters by cryptosporidial oocysts it is difficult to prevent point source outbreaks due to faecal contamination, especially from small children. Such contamination was reported in three of the four outbreaks. As cryptosporidial oocysts are particularly resistant to chlorine the risk of acquiring cryptosporidiosis from swimming pools is higher than for other gastrointestinal pathogens. Analytical studies may help in strengthening the evidence for an association with a swimming pool, especially if no pathogen is identified in the water. Given the nature of these outbreaks contamination of pools may not persist long enough to be detectable by the time the outbreak is recognized and sampling of water (including filter backwash) is arranged.

Outbreaks associated with the consumption of water from miscellaneous sources

Only one outbreak was reported to be associated with exposure to other recreational waters. However this probably underestimates the extent of illness, including sporadic infections, caused by ingestion of river and sea water while taking part in water sports. Many participants in water sports will be fit young adults who may not consult their general practitioners when they get gastro-enteritis, even though some water sports centres encourage reporting. Recent studies conducted at the water sports centre implicated in Outbreak 26 indicate that diarrhoeal illness among canoeists is more common than previously recognised [12].

Previous reports have described the emergence of *Cryptosporidium parvum* as an important waterborne pathogen [3, 4]. It is now established as the most important pathogen associated with waterborne outbreaks of IID in England and Wales and the USA

[4,14, 15]. It is an organism that can cause very severe illness particularly among the immunocompromised and the elderly [16]. Among those affected in the 16 outbreaks of cryptosporidiosis reported here, 68 (5%) were admitted to hospital, compared with 2% in all other outbreaks of IID reported to CDSC during the same period.

Although the second report of the Badenoch committee [13] noted that the incidence of cryptosporidiosis had fallen in England and Wales between 1992 and 1994, the number of laboratory reported cases rose to 5705 in 1995 (the highest annual total since 1989). As chlorination is demonstrably insufficient to remove the risk of cryptosporidial contamination from drinking water, water treatment processes should be rigorously applied in order to remove oocysts effectively from water supplies [5, 13]. High standards of surveillance at local, regional and national level are needed to recognise outbreaks promptly so that investigation and control measures can be instituted rapidly. As microbiological evidence of water contamination may be absent or insufficient to establish whether water is the vehicle of infection, especially in chlorinated supplies, a high standard of epidemiological studies are recommended in all outbreaks of suspected waterborne disease.

ACKNOWLEDGEMENTS

We would like to acknowledge Dr J. M. Cowden, Mrs S. Labaigue and Mrs Di Ross for the design and maintenance of the CDSC surveillance system for outbreaks of infectious intestinal disease. We also thank Dr Mike Rowland, and Dr Alain Moren for their helpful comments on this paper. We are very grateful to all those who took the time to complete and return surveillance forms.

REFERENCES

- Galbraith NS, Barrett NJ, Stanwell-Smith R. Water and disease after Croydon: a review of water-associated disease in UK 1937–86. *J Inst Wat Environ Man* 1987; **1**: 7–21.
- Galbraith NS. Historical review of microbial disease spread by water in England and Wales. In: Golding AMB, Noah N, Stanwell-Smith R, eds. *Water and public health*. Great Britain 1994: 15–37.
- Stanwell-Smith R. Water and public health in United Kingdom. Recent trends in the epidemiology of waterborne disease. In: Golding AMB, Noah N, Stanwell-Smith R, ed. *Water and public health*. Great Britain 1994: 39–54.
- Meinhardt PL, Casemore DP, Miller KB. Epidemiological aspects of human cryptosporidiosis and the role of waterborne transmission. *Epidemiol Rev* 1997; **18**: 118–36.
- Department of the Environment and Department of Health. *Cryptosporidium in water supplies*. Report of the group of experts. London: HMSO, 1990.
- Committee on the Microbiological Safety of Food. *The microbiological safety of food: part 1*. London: HMSO, 1990.
- Grant AD, Eke B. Application of information technology on the laboratory reporting of communicable disease in England and Wales. *CDR* 1993; **3**: R75–8.
- Dean AG, Dean JA, Coulombier D, et al. *Epi Info Version 6: a word processing, database, and statistics program for epidemiology on microcomputers*. Atlanta: Centers for Disease Control and Prevention, 1994.
- Nazareth B, Stanwell-Smith RE, Rowland MGM, O'Mahoney MC. Surveillance of waterborne disease in England and Wales. *CDR* 1994; **4**: R93–5.
- Morgan D, Allaby M, Crook S, Casemore DP, et al. Waterborne cryptosporidiosis associated with a borehole supply. *CDR* 1995; **5**: R93–7.
- Hunt DA, Sebugwawo S, Edmondson SG, Casemore DP. Cryptosporidiosis associated with a swimming pool complex. *CDR* 1994; **4**: R20–2.
- Gray JJ, Green J, Cunliffe C, Gallimore C, Lee JV, Neal K, Brown DWG. Mixed genogroup SRSV infections among a party of canoeists exposed to contaminated recreational water. *J Med Virol*. In press.
- Department of the Environment and Department of Health. *Cryptosporidium in water supplies*. Second report of the group of experts. London: HMSO, 1995.
- CDC. Surveillance for waterborne-disease outbreaks – United States, 1993–1994. *MMWR* 1996; **45** (SS-1): 1–33.
- Casemore DP, Wright SE, Coop RL. Cryptosporidiosis – human and animal epidemiology. In: Fayer L, ed. *Cryptosporidium and cryptosporidium*. Boca Raton: CRC Press, 1997: 65–92.
- Casemore DP. Epidemiological aspects of human cryptosporidiosis. *Epidemiol Infect* 1990; **104**: 1–28.
- Healey MG. Regulating drinking water quality in England and Wales. In: Golding AMB, Noah N, Stanwell-Smith R, ed. *Water and public health*. Great Britain 1994: 101–7.
- Drury D. Private water supplies: classification and monitoring. *CDR* 1995; **5**: R98–9.
- Pool Water Treatment Advisory Group. *Pool water guide: the treatment and quality of swimming pool water*. Diss, PWTAG, 1995.
- Dadswell JV. Managing swimming, spa, and other pools to prevent infection. *CDR* 1996; **6**: R37–40.