

## **Illness in a community associated with an episode of water contamination with sewage**

J. FOGARTY<sup>1</sup>\*, L. THORNTON<sup>2</sup>, C. HAYES<sup>1</sup>, M. LAFFOY<sup>1</sup>,  
D. O'FLANAGAN<sup>1</sup>, J. DEVLIN<sup>2</sup> AND R. CORCORAN<sup>2</sup>

<sup>1</sup>*Department of Public Health Medicine, Eastern Health Board, Dublin*

<sup>2</sup>*Department of Public Health Medicine, Eastern Health Board, Naas, Co. Kildare*

*(Accepted 27 October 1994)*

### SUMMARY

Following an episode of water contamination with sewage in a rural Irish town, a community-wide survey of gastrointestinal-associated illness and health service utilization was conducted. Random sampling of households yielded residents who were surveyed using a self-administered questionnaire. Of 560 respondents from 167 (84%) households, equal proportions lived in areas known to have been exposed and unexposed to the contaminated water, although 65% of subjects reported using contaminated water. Sixty-one percent of subjects met the case definition. The most common symptoms among cases were abdominal cramps (80%), diarrhoea (75%), appetite loss (69%), nausea (68%) and tiredness (66%). Mean duration of illness was 7·4 days. Only 22% of cases attended their general practitioner. Drinking unboiled water from the exposed area was strongly associated with being a case. A substantial degree of community illness associated with exposure to contaminated water was observed. The episode ranks as one of the largest reported water-borne outbreaks causing gastrointestinal illness in recent times.

### INTRODUCTION

The availability of safe water supply for human consumption is taken for granted in the developed world. When sewage contamination of drinking water occurs there is a risk of gastrointestinal infection due to a range of potential pathogens [1]. In early October 1991 an episode of sewage contamination occurred which affected the water supply to approximately half the population of a rural town of 11 141 people in Ireland [2].

### BACKGROUND

The existing town water supply (supply A) had become inadequate to meet demand due to population growth. An artesian well (supply B) was brought into use in December 1990 to supplement supply A. Supply B, which was chlorinated

\* Correspondence and reprint requests to: Dr J. Fogarty, Western Health Board, 25 Newcastle Road, Galway.

at source, exclusively supplied approximately half the town's water supply. Throughout 1991 the town's water supply was extensively monitored and on all occasions met water quality guidelines [3] indicating that it was fit for human consumption. The most recent sampling had been on 23 September 1991. On 7 October a consumer complained of a foul smell from a domestic water supply. Water samples were taken for bacteriological analysis. No further complaints were received on that day. On 8 October, when further complaints were received, inspection of the well borehole (supply B) revealed visible signs of sewage contamination. Supply B was closed, not used again and substituted with supply A. Water sampling revealed a total coliform count of  $1.1 \times 10^7$  per ml and an *E. coli* count of  $1.5 \times 10^5$  per ml. The pipe system was flushed out and hyperchlorinated. From 9 October an alternative drinking water supply was provided for the town by means of tankers. Water from supply A continued to be monitored on a daily basis from an average of 25 sampling points in the town. On 25 October, after repeated satisfactory results the local authority was advised that the water was again fit for human consumption. Daily sampling for a further month revealed satisfactory results.

The well (supply B) had become contaminated with untreated human sewage which leaked from an adjacent sewage conduit. It is not known precisely when the contamination commenced but it was reported after the event came to notice that a consumer had noted a foul smell from the water on 5 October. Communication with the public and medical practitioners suggested widespread symptoms of illness among residents. The object of this study was to establish the effects of the episode on the health of residents of the town.

#### SUBJECTS AND METHODS

A random sample of 200 households in the entire town was selected from the electoral register. Each member of each selected household was included in the sample. A self-administered questionnaire was delivered to residents, accompanied by a detailed list of areas supplied by the contaminated water so that respondents could determine their own exposure to the water. Exposure to the contaminated water by area of residence was determined by reference to street lists provided by the local authority. Data were collected on (a) demographic factors (e.g. age, sex, address), (b) exposure to the contaminated water, (c) illness symptoms (e.g. nausea, vomiting, diarrhoea, abdominal cramps), (d) general practitioner attendance and hospital service utilization and (e) absenteeism from work or school. A case was defined as a subject with onset of specified symptoms, namely diarrhoea, vomiting or abdominal cramps between midnight on 4 October and the day of questionnaire completion on 24–26 October 1991.

Exposure to the contaminated water was considered to have occurred if a respondent, between midnight 4 October and midday 10 October, used water from the affected area in any of a number of ways (e.g. by drinking unboiled or boiled water, brushing teeth, having a bath or shower, washing raw fruit or vegetables prior to eating, cooking food). The chi-square ( $\chi^2$ ) test was used for comparison of proportions with the use of Fisher's Exact test where appropriate, and Student's *t* test was used for comparing means between two groups. Hypothesis testing was

performed by determining relative risks and confidence intervals. Data were analysed using Epi Info version 5 [4].

## RESULTS

Completed questionnaires were returned by 560 persons from 167 (83.5%) households surveyed. Almost equal proportions of respondents lived in exposed (278/560, 49.6%) and unexposed (282/560, 50.4%) areas. Of 543 respondents, 350 (64.5%) reported using the contaminated water. Although 371 (66.7%) of 556 respondents classified themselves as ill, 340 (60.7%) subjects fulfilled the case-definition criteria. Two hundred and fifty-two (74.1%) cases were under 40 years of age. Of 280 cases who provided adequate information on occupation, 209 (74.6%) were from social class categories 1–3 [5]. In terms of age, sex and social class, cases did not differ from non-cases.

The attack rate was 77.3% for subjects living in the exposed area and 75.7% for those who reported using the contaminated water (Table 1). Cases were more likely to live in the exposed area (215/278, 77.3%) than in the unexposed area (125/282, 44.3%) (relative risk (RR) = 2.2; 95% confidence interval (CI) = 1.8–2.8,  $P < 0.001$ ). Cases were also more likely to report having used the contaminated water (265/350, 75.7%) than not having used it (65/193, 33.7%) (RR = 2.0; 95% CI = 1.7–2.4,  $P < 0.001$ ).

The frequency of symptoms among cases is presented in Table 2. The most common symptoms were abdominal cramps (79.5%), diarrhoea (74.6%), appetite loss (68.5%), nausea (67.8%) and fatigue (66.1%). The mean frequency of diarrhoea was 7.5 episodes per day.

Cases were more likely to report drinking unboiled water from the contaminated area (225/295, 76.3%) than were non-cases (43/196, 21.9%) (RR = 3.5, 95% CI = 2.7–4.6;  $P < 0.001$ ). A dose–response relationship to drinking unboiled water from the contaminated supply was observed with a median daily consumption of 5.0 glasses in cases compared to 2.5 glasses in non-cases. However, this did not reach statistical significance. Two hundred and twenty-three respondents reported that they did not drink unboiled water during the period specified. Of these, cases were more likely to drink boiled water (44/68, 64.7%) than were non-cases (43/152, 28.3%) (RR = 2.3, 95% CI = 1.7–3.1;  $P < 0.001$ ).

In subjects reporting no consumption of unboiled or boiled water, other, presumably lesser, forms of exposure to water (brushing teeth, washing dishes, cooking food in water and bathing or showering) were associated with being a case. However, it was not possible to conclude that any of these factors were independently associated with increased risk of being a case as most subjects carried out many of these activities. Of 287 cases who provided an exact date of onset of illness 243 (84.7%) had onset of symptoms in the week 5–11 October, with a peak of 119 cases on 7 and 8 October (Fig. 1).

The mean duration of illness in cases was 7.2 days. One hundred and forty-nine (43.8%) cases reported taking time off work or school due to illness with a mean absence of 3.9 days.

Seventy-three (21.8%) of 335 cases reported attending their general practitioner (GP) due to their illness. Of cases indicating the frequency of GP attendance 56

Table 1. *Distribution of cases and non-cases using two exposure categories; living in exposed area and using contaminated water*

		Case <i>n</i> (%)	Non-case <i>n</i> (%)	Total <i>n</i> (%)
Living in exposed area	Yes	215 (77.3)	63 (22.7)	278 (100)
	No	125 (44.3)	157 (55.7)	282 (100)
Using contaminated water	Yes	265 (75.7)	85 (24.3)	350 (100)
	No	65 (33.7)	128 (68.3)	193 (100)

Table 2. *Frequency of symptoms among the 340 cases*

Symptoms	<i>n</i> (%)*
Abdominal cramps	260 (79.5)
Diarrhoea	250 (74.6)
Poor appetite	222 (68.6)
Nausea	221 (67.8)
Fatigue	213 (66.1)
Headaches	189 (59.1)
Vomiting	164 (50.6)
Fever	156 (48.6)
Aches	153 (48.0)
Sore throat	96 (30.4)
Stiff neck	68 (21.8)
Brown urine	41 (13.1)
Rash	23 (7.3)
Blood in stools	11 (3.6)
Yellow skin	9 (2.8)
Yellow eyes	5 (1.6)
Other symptoms	35 (11.3)

\* Percentages are calculated from varying response rates by cases to individual questions on symptoms.

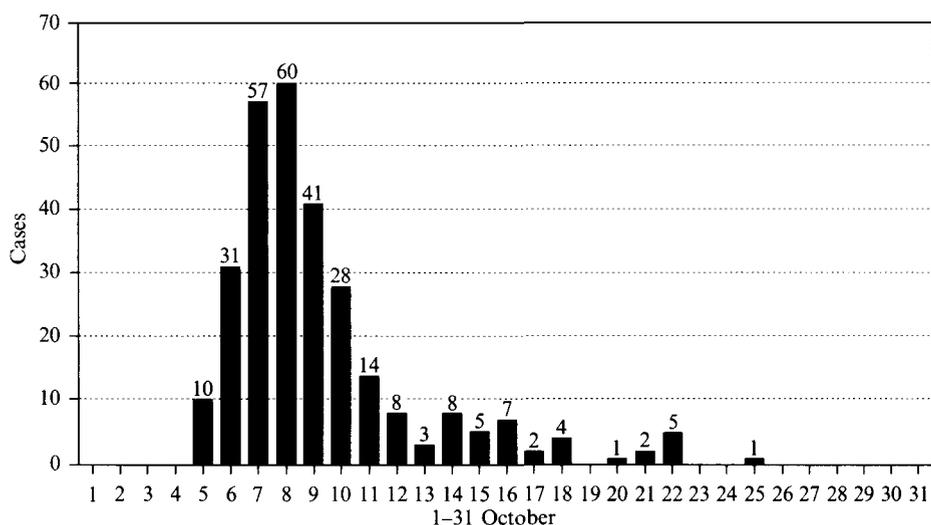


Fig. 1. Onset date of illness among cases during October, based on data from 287 cases.

(76.7%) attended once, 6 twice, 4 three times and 1 person four times. Two cases were admitted to hospital and a further 2 were treated at a hospital accident and emergency department.

While the study did not directly address the aspect of laboratory tests on clinical specimens, members of the public who were ill were encouraged to provide faecal samples for analysis. Considering the extent of symptoms in the community, relatively few samples were provided for analysis. The majority of isolates were of non-pathogenic *Escherichia coli* and other non-pathogenic organisms (e.g. *Providencia alcalifaciens*). There were several isolates of different strains of enterotoxigenic *E. coli* and of *Giardia lamblia*. No isolates of salmonella, shigella, campylobacter, cryptosporidium or rotavirus were demonstrated.

#### DISCUSSION

There was a high level of community co-operation with this study. This was an encouraging recognition by the public of the necessity to investigate the incident scientifically.

The finding that almost three-quarters of the cases were relatively young (< 40 years) and that a similar proportion belonged to social classes 1–3 is consistent with the demographic profile of the sample. This reflects the fact that the contaminated water supplied predominantly the newer owner-occupied housing estates in the town which are mainly inhabited by younger families.

It is evident that the symptomatology among cases relates to the ingestion of heavily contaminated water, even though laboratory investigations were few and the number of pathogenic isolates small for the entire community during the episode. Rosenberg and colleagues in a U.S. episode [6] report that although sewage probably contaminated the drinking water with multiple organisms, affecting about 2000 staff and visitors to an American national park, only a single pathogenic strain was recovered from ill persons. In the present study the reporting of blood in stools and signs and symptoms of jaundice among cases was uncommon. This is in keeping with the failure to isolate organisms such as shigella and campylobacter. Furthermore, no case of hepatitis A was notified to the Medical Officer of Health during or for the 6 month period following the contamination episode. The reporting of 'brown urine' among a small proportion of cases is likely to reflect the effects of dehydration in the absence of other evidence to support the occurrence of hepatitis.

The failure to link illness associated with contamination of water supplies with causative organisms is not unusual. Anderson and Strenstrom in Sweden [7] reported that 53.2% of water-borne disease outbreaks were caused by unknown agents and similarly in the United States [8] 47% of water-borne illness outbreaks failed to reveal a causative organism. Zmirou and colleagues [9] found poor correlation between the isolation of organisms in the laboratory and symptomatology among those affected by contaminated water. Their findings are in keeping with the high levels of morbidity among subjects in this study despite lack of microbiological confirmation. The syndrome of 'sewage poisoning' which was believed to be responsible for a large water-borne gastroenteritis epidemic in California in 1971 [10] fits closely with the findings of our study. Nevertheless,

disease outbreaks associated with contamination of public water supplies have unearthed causative agents, such as cryptosporidium [11], enteropathogenic *E. coli* [12], bacillary dysentery [13] and phenol [14].

While the difference in amount of unboiled water consumed by cases and non-cases was not statistically significant, the median consumption by cases was twice that of non-cases. Such a dose-response between gastrointestinal symptoms and the number of cold-tap drinks consumed was shown by Williams [15] in a re-analysis of data from a study by Meara [16].

This report details the occurrence of cases in two categories of exposure to contaminated water: (a) self-reported exposure in subjects who used the water and (b) residence in the area known to be supplied by the contaminated water. The occurrence of some cases categorized as non-exposed by either definition may represent background illness in the community due to other factors not addressed in the study, inaccurate recall of actual exposure or to the occurrence of secondary cases in unexposed subjects, a well-recognized factor in such contamination episodes [17].

We may have underestimated the extent of symptoms among residents by not setting a date earlier than midnight 4 October for case definition. It appears from the epidemic curve (Fig. 1) that the contamination episode commenced on 4 or 5 October. However no complaint about the water was received from the public until 7 October. Possible explanations for the continuation of cases late in the epidemic curve may relate to secondary household spread or to late primary cases who continued to use contaminated water.

Differential reporting of symptoms by subjects living in areas exposed and unexposed to the contaminated water is a possibility in this study. The use of lists indicating the geographical areas which were supplied with contaminated water may have contributed to a degree of recall bias, as was also suggested by Mayon-White and colleagues in their report of a water contamination incident in Oxfordshire [18].

An interesting feature of the outbreak was the relatively low level (just over one-fifth) of general practitioner attendance. This may be because cases were satisfied that they knew the origin of their illness and believed that their symptoms would be self-limiting.

Due to the high attack rate for illness experienced by subjects in the study, estimates of morbidity within the town (population of 11 141; Census, 1991 [2]) are extremely high. If no cases occurred among the non-responders, then the morbidity rate of 50.7% (340/671) (671 being the estimated number of subjects in all households sampled) can be applied to the population of 11 141 giving 5648 subjects who would have met the case-definition based on the random sampling conducted. If the case rate of 60.7% of the responders is applied, 6762 subjects would have met the case-definition. Whichever rate is appropriate, it is estimated that approximately 6000 residents met the case-definition of illness associated with this water contamination episode.

A review of records of water-borne and water-associated disease outbreaks in the United Kingdom over a 50-year period revealed a total of 11 794 cases [19]. The present episode exceeds any of the outbreaks cited in the review in terms of the number of persons estimated to have met the case-definition of illness.

This study documents an episode of water contamination which, although not life-threatening, was a source of major morbidity and disruption in a young population. It represents one of the largest reported water-borne illness outbreaks in recent times.

## ACKNOWLEDGEMENTS

We thank the residents of the town of Naas, Co. Kildare; Kildare County Council; the Area Medical Officers and Secretarial staff, Poplar House, Naas; Mr Martin Devine, Principal Environmental Health Officer, Naas; Dr M. E. Dillon, Consultant Microbiologist, Public Health Laboratory, Cherry Orchard Hospital, Dublin; and the management of the Eastern Health Board.

## REFERENCES

1. Levine WC, Stephenson WT, Craun GF. Waterborne disease outbreaks, 1986–1988. *MMWR CDC Surveill Summ* 1990; **39**: 1–13.
2. Central Statistics Office Census 91 – Local Population Report – 1st Series. Dublin: Central Statistics Office, 1993.
3. European Communities (Quality of Water Intended for Human Consumption) Regulations, 1988.
4. Dean AG, Dean JA, Burton AH, Dicker RC. Epi Info, version 5: a word processing, database and statistics system for epidemiology on microcomputers. Stone Mountain, Georgia: USD Incorporated, 1990.
5. O'Hare A. A note on a proposed census based Irish social class scale for epidemiological health research. *Econ Soc Rev* 1982; **13**: 205–16.
6. Rosenberg ML, Koplan JP, Wachsmuth IK. Epidemic diarrhea at Crater Lake from enterotoxigenic *Escherichia coli*. *Ann Intern Med* 1977; **86**: 714–18.
7. Andersson Y, Stenstrom TA. Waterborne outbreaks in Sweden – causes and etiology. *Wat Sci Tech* 1987; **19**: 575–80.
8. Merson MH, Barker WH Jr, Craun GF. Outbreaks of waterborne disease in the United States, 1971–1972. *J Infect Dis* 1974; **129**: 614–15.
9. Zmirou D, Ferley JP, Collin JF, Charrel M, Berlin J. A follow-up study of gastro-intestinal diseases related to bacteriologically substandard drinking water. *Am J Public Health* 1987; **77**: 582–4.
10. Mahoney LE, Friedmann CTH, Murray RA, Schulenburg EL, Heidbreder GA. A waterborne gastroenteritis epidemic in Pico Rivera, California. *Am J Publ Hlth* 1974; **64**: 963–8.
11. Hayes EB, Matte TD, O'Brien TR, *et al*. Large community outbreak of cryptosporidiosis due to contamination of a filtered public water supply. *N Eng J Med* 1989; **320**: 1372–6.
12. Dev VJ, Main M, Gould I. Waterborne outbreak of *Escherichia coli* O157. *Lancet* 1991; **337**: 1412.
13. Green CA, Macleod MC. Explosive epidemic of sonn  dysentery. *BMJ* 1943; **ii**: 259–61.
14. Jarvis SN, Straube RC, Williams ALJ, Bartlett CLR. Illness associated with contamination of drinking water supplies with phenol. *BMJ* 1985; **290**: 1800–2.
15. Williams JH. Associations between water quality and GIT symptoms in country dwellers. *J Publ Hlth Med* 1991; **13**: 135.
16. Meara JR. An investigation of health and lifestyle in people who have private water supplies at home. *Community Med* 1989; **11**: 131–9.
17. Lobel HO, Bisno AL, Goldfield M, Prier JE. A waterborne epidemic of gastroenteritis with secondary person-to-person spread. *Amer J. Epidemiol* 1969; **89**: 384–92.
18. Mayon-White R, Del Fante P, Murphy M, *et al*. A water contamination incident. *Commun Dis Rep* 1992; **2**: R69–70.
19. Galbraith NS, Barrett NJ, Stanwell-Smith R. Water and disease after Croydon: a review of water-borne and water-associated disease in the UK 1937–86. *Water Environ Management* 1987; **1**: 7–21.