An extensive gastroenteritis outbreak after drinking-water contamination by sewage effluent, Finland

J. LAINE[1,2]*, E. HUOVINEN[1], M. J. VIRTANEN[1], M. SNELLMAN[1], J. LUMIO[2,3],
P. RUUTU[1], E. KUJANSUU[4], R. VUENTO[3,5], T. PITKÄNEN[1], I. MIETTINEN[1],
J. HERRALA[2], O. LEPISTÖ[6], J. ANTONEN[2,4], J. HELENIUS[3], M.-L. HÄNNINEN[7],
L. MAUNULA[7], J. MUSTONEN[2,4], M. KUUSI[1] and the Pirkanmaa Waterborne Outbreak
Study Group†

[1] *National Institute for Health and Welfare, Helsinki and Kuopio, Finland*
[2] *Tampere University Hospital, Tampere, Finland*
[3] *University of Tampere, Tampere, Finland*
[4] *Nokia Health Centre, Nokia, Finland*
[5] *Centre for Laboratory Medicine, Tampere, Finland*
[6] *Environmental Health Control Unit, Pirkkala, Finland*
[7] *University of Helsinki, Helsinki, Finland*

SUMMARY

An inappropriate cross-connection between sewage- and drinking-water pipelines contaminated tap water in a Finnish town, resulting in an extensive waterborne gastroenteritis outbreak in this developed country. According to a database and a line-list, altogether 1222 subjects sought medical care as a result of this exposure. Seven pathogens were found in patient samples of those who sought treatment. To establish the true disease burden from this exposure, we undertook a population-based questionnaire investigation with a control population, infrequently used to study waterborne outbreaks. The study covered three areas, contaminated and uncontaminated parts of the town and a control town. An estimated 8453 residents fell ill during the outbreak, the excess number of illnesses being 6501. Attack rates were 53% [95% confidence interval (CI) 49·5–56·4] in the contaminated area, 15·6% (95% CI 13·1–18·5) in the uncontaminated area and 6·5% (95% CI 4·8–8·8) in the control population. Using a control population allowed us to differentiate baseline morbidity from the observed morbidity caused by the water contamination, thus enabling a more accurate estimate of the disease burden of this outbreak.

Key words: Gastroenteritis, outbreak, waterborne infections.

INTRODUCTION

Safe water is essential to life. Globally, the lack of clean water is estimated to cause about 4% of total mortality [1], predominantly in the developing world. However, waterborne outbreaks also remain a source of concern in developed countries. In the USA, 20 million people are estimated to contract a waterborne infection annually [2]. Over 400 000 people fell ill with cryptosporidiosis in Milwaukee in 1993 as a consequence of inadequate water filtration [3]. In Canada, the Walkerton outbreak resulted in seven fatalities

attributable to *Escherichia coli* O157:H7 infection [4]. Waterborne outbreaks have been reported from many European countries including England and Wales [5], Norway [6], Sweden [7–10], Italy, Spain and Switzerland [11].

Finland is a large country (338 424 km²) with a small population (5·4 million). The number of waterworks is high (almost 2000) and most serve small communities. The majority (95%) use groundwater. However, almost half of the population are served by surface-water plants of the largest cities. Groundwater is usually not disinfected for use, while surface water is filtered and disinfected before distribution to consumers [12].

Although drinking water is generally considered safe in Finland, a number of waterborne outbreaks have occurred. Twenty-four outbreaks affecting 7700 inhabitants were spontaneously reported during the period 1980–1992 [13]; since 1997 reporting of outbreaks has been mandatory, and 14 outbreaks affecting 7300 people were registered during the first 2 years [12]. The most widespread outbreak to date affected 5500 people [14], and a number of other substantial waterborne outbreaks have been reported [15–17]. The most common microbes have been norovirus [18] and *Campylobacter* [19].

We report the investigation of a serious waterborne gastroenteritis outbreak that occurred in a Finnish town in autumn 2007.

Setting

Nokia (population 30 016) is a town located in southern Finland. Over 90% of the population are served by the municipal drinking-water supply. The average daily water consumption in Nokia is 4250 m³ (year 2006). The waterworks uses groundwater, which is treated with sodium hypochlorite for disinfection and pH adjustment. Sewage is processed in one municipal waste water plant, which covers 86% of the population.

The town has a municipal health centre for primary care and a hospital for primary in-patient care and limited secondary care. Secondary and tertiary care are provided by Tampere University Hospital 20 km distant from Nokia. The Centre for Laboratory Medicine provides the municipalities with clinical laboratory services. Environmental health surveillance and monitoring of water quality is organized in collaboration with five neighbouring municipalities.

The outbreak

On Wednesday, 28 November 2007, maintenance work was conducted at the waste water plant in Nokia. The same day, technical problems were encountered in the municipal drinking-water system, and during the days following, customers complained of a bad smell and taste in their tap water. These were first misinterpreted as being caused by harmless deposits loosened from the inner walls of the pipeline and discharged during the maintenance work on 28 November.

From 30 November, environmental health and municipal waterworks officials received reports of bouts of diarrhoea and vomiting among residents, and during that same day an increase in the number of patients with gastroenteritis was observed at the health centre. Extensive water and pipeline sampling was initiated and a boil-water notice issued, and water chlorine concentration was increased.

The water contamination and its source became evident the same day. A valve in the waste-water plant connecting the drinking-water line and a waste-water effluent line had been opened during the maintenance work and inadvertently left open for 2 days. As a consequence, the drinking-water network became contaminated with sewage effluent. According to flow data, an estimated 450 m³ of waste water was mixed with the drinking water and distributed to customers, heavily contaminating household water and pipelines in southern parts of the town. The population in this area numbered over 9500.

Distribution of clean water was organized by municipal officials, the military and volunteers, and the health centre focused on treating outbreak patients, cutting back on other activities. The University Hospital prepared to open a temporary ward, which was in fact eventually not needed. Schools and day-care centres were closed from 6 to 9 December owing to teachers' and children's sick leave and the need to reduce the possible horizontal spread of gastrointestinal viruses.

After 12 days, the boil-water notice was withdrawn in the uncontaminated area. In the contaminated area, adenoviruses and noroviruses were repeatedly detected in water-supply samples despite chlorination and pipeline flushing. Finally, after extensive decontamination procedures including shock chlorination, precautionary restrictions were discontinued in the contaminated area on 18 February 2008, almost 3 months after the incident.

We conducted a comprehensive investigation to determine the magnitude and consequences of the waterborne outbreak including describing the clinical aspects, reviewing microbiological findings and estimating the disease burden using a population-based survey.

METHODS

Descriptive epidemiology

Patients visiting the municipal health centre between 28 November and 31 December 2007 because of acute gastroenteritis were registered on a line-list, including name, age, gender, address and personal identity number. The timing and range of symptoms were recorded. Prospective line-listing was initiated 6 days after the beginning of the contamination visits prior to that being added retrospectively from patient charts. The information collected from the patient register included the number of primary-care visits to the centre because of gastroenteritis as defined by the international classification of primary care (ICPC-1, WHO, Geneva) codes D01, D09, D10, D11, D25, D70, D73 or D96. The information on reported symptoms was collected from the line-list. Daily admissions to Tampere University Hospital were counted by emergency-department personnel.

Analytical epidemiology

The town was divided into two study areas, contaminated (population 9538) and uncontaminated (population 20 478). The division was based on microbiological data from distribution system samples and technical modelling of water-flow directions within the network. Another municipality (population 27 259) 35 km distant from Nokia was chosen as a control population. One thousand persons for each study group were randomly selected from the population register. Groups were matched by age and gender and all age groups were included. Only one participant per household was included. The survey was conducted using postal questionnaires, the form being posted 8 weeks after the contamination had occurred. A repeat questionnaire was sent out 3 weeks later to non-responders.

Participants were asked about the timing and nature of gastrointestinal symptoms. A case was defined as a person suffering acute diarrhoea ($\geqslant 3$ loose stools/day) or vomiting between 28 November 2007

and 20 January 2008. The occurrence of gastroenteritis during a 10-month period prior to the outbreak was also enquired about to ensure the comparability of the study groups.

Microbiological investigation

Faecal specimens for bacterial pathogens were not investigated from all the patients or from representative samples from consecutive cases. Instead, bacterial samples were available from cases with more severe symptoms. Specimens for detection of norovirus were collected from five randomly selected patients within the first week of the outbreak. Other viral pathogens were studied in samples from children admitted to the emergency department of the University Hospital. In order to detect enteric parasites specimens were taken from ten consecutive patients during the second week of the outbreak. Subsequently, when cases with giardiasis were detected, parasites were investigated in all symptomatic patients. Specimens were taken at the health centre or in the University Hospital.

Stool specimens were cultured for the presence of *Campylobacter, Salmonella, Yersinia* and *Shigella* spp. Norovirus was investigated using electron microscopy and PCR; rotavirus antigens were detected by enzyme immunological assay (EIA). Protozoan pathogens were investigated using microscopy of formalin-fixed samples. Subsequently, because no other protozoan pathogens were identified, only antigen detection of *Giardia* was used instead of microscopy. If a patient reported bloody stools, Shiga toxin-producing *Escherichia coli* (STEC) was examined for by culture and toxin detection (EIA). Only samples taken between 28 November and 31 December 2007 were included. For *Giardia* the time-period for inclusion was extended to 31 May 2008 (6 months) because of the longer incubation time and gradual case detection.

The methods used in water and pipeline analysis will be described in detail in a forthcoming paper. Briefly, samples from the drinking-water distribution system were analysed by cultivation for the presence of *Campylobacter, Salmonella* and *Clostridium* spp. PCR was utilized to analyse enteric viruses as previously described by Maunula and colleagues [20]. The presence of *Giardia* cysts and *Cryptosporidium* oocysts was detected using concentration, immunomagnetic separation and epifluorescence microscopy as described in detail by Rimhanen-Finne and colleagues [21].
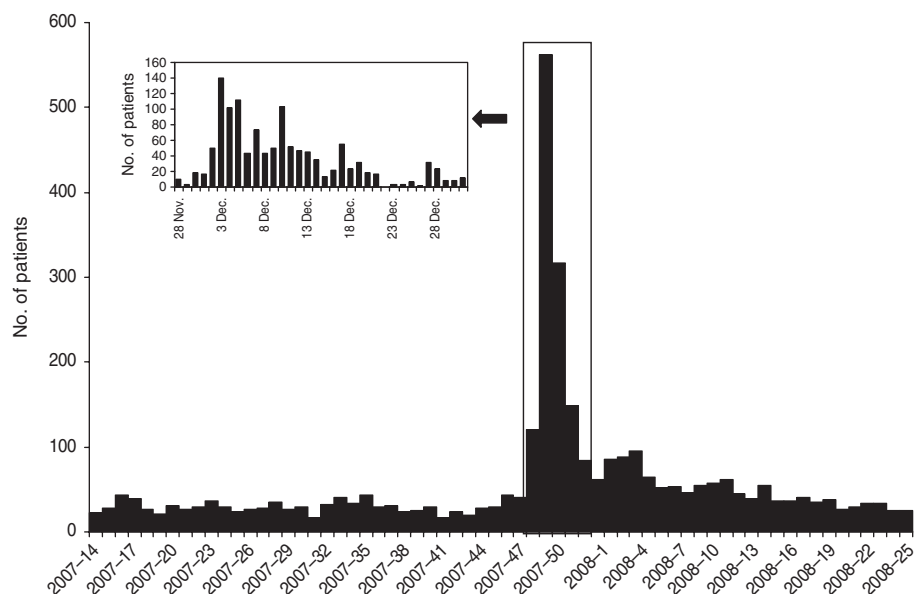
Fig. 1. Weekly number of patients attending with gastrointestinal symptoms at Nokia health centre from 1 April 2007 to 22 June 2008 (main figure). The inset represents the daily number of patients attending with gastrointestinal symptoms during the outbreak period.

Data analysis

The study areas were compared using χ^2 or Kruskal–Wallis rank sum tests whenever appropriate. Attack rates were calculated as percentage of cases in a sample population. Total numbers of cases were calculated by extrapolating the age- and sex-adjusted attack rates to the total population of each area. To assess baseline rates of gastroenteritis, the attack rates from the control population were used to estimate the number of cases in the contaminated population that would have occurred in the absence of the water contamination. Estimations were made adjusting for age and sex. Confidence intervals for the total numbers were obtained using bootstrap methods [21]. All statistical analyses were performed using R version 2.9 (or later versions) [22].

RESULTS

Healthcare visits

According to the health centre database, 1222 visits due to gastrointestinal symptoms were made between 28 November and 31 December 2007. During the busiest week, the number of patients presenting with gastrointestinal complaints was 53-fold higher than expected compared to the weekly median number of cases in a 7-month period preceding the outbreak. Children aged <10 years constituted the largest group (Figs 1, 2).
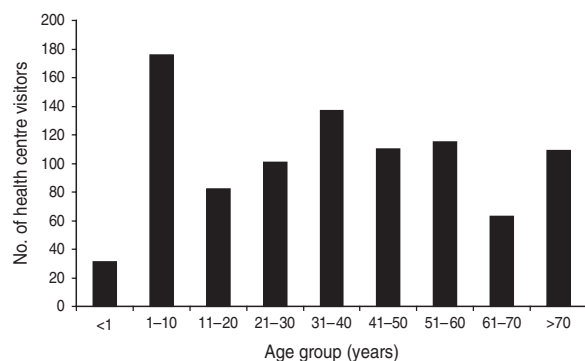

Fig. 2. Age distribution of visitors attending Nokia health centre with gastrointestinal symptoms, 28 November to 31 December 2007; data from patient register.

Line-list data were available on 1024 patients. Only one visit per person was included. The most common symptoms reported were diarrhoea ($n=813$, 79%), vomiting ($n=601$, 59%), fever ($n=337$, 33%) and abdominal pain ($n=291$, 28%). Altogether 204 visits were made to emergency rooms in Tampere University Hospital, 145 (71%) of them involving children.

To our knowledge, the outbreak did not lead directly to fatalities.

Questionnaire study

A total of 2154 participants returned the questionnaire. Thirty-one were excluded as being inadequately

Table 1. *Background characteristics of the three populations in the questionnaire study (only respondent data included)*

|	Nokia, contaminated ($n = 808$)	Nokia, uncontaminated ($n = 717$)	Control population ($n = 598$)
Male gender (%)	44·8	44·5	45·8
Age (yr), median (min–max)	41 (0–92)	44 (0–94)	42 (0–96)
Having gastroenteritis between 1 January and 31 October 2007 (before the outbreak), n (%)	132 (17)	128 (18)	104 (17)
Daily consumption of unboiled tap water (glasses/day), mean (S.D.)	4·9 (3·1)	4·9 (3·3)	4·5 (3·0)
Tap water provided from municipal waterworks, n (%)	783 (98)	587 (84)	433 (82)
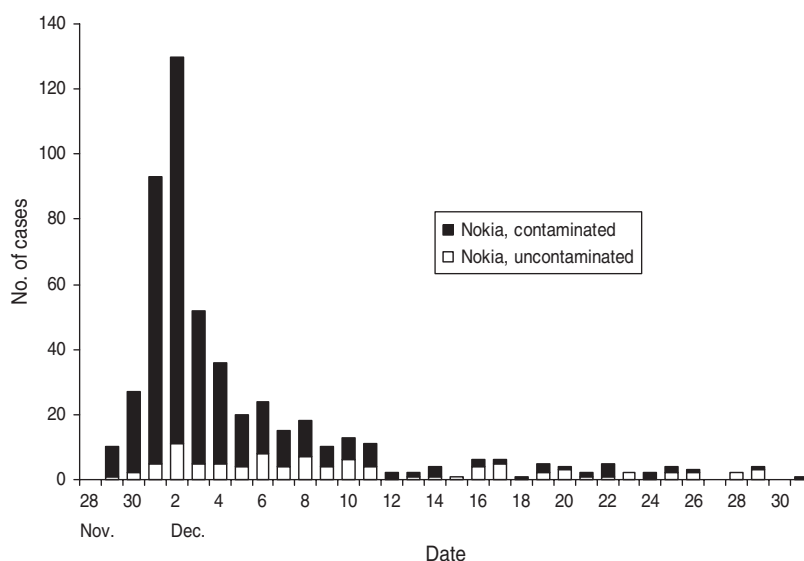
Fig. 3. The epidemic curve. Cases of acute gastroenteritis by onset of illness in two areas of Nokia, 28 November 2007 to 20 January 2008, according to the questionnaire study.

completed or because the respondent could not be identified; 2123 forms were finally accepted for the analysis (response rate 71%). The response rate was highest in the contaminated area and lowest in the control population. The background characteristics of the study groups showed no significant demographic differences between the study populations (Table 1). Altogether, 17% of the responders reported having gastroenteritis during the 10 months prior to the outbreak.

Most subjects fell ill within 1 week of the contamination of the water supply (Fig. 3). The most common symptoms were diarrhoea, vomiting, nausea and abdominal pain (Fig. 4). Overall, 579 respondents met the case definition, of whom 540 (93%) were residents of Nokia. Extrapolating the result for the whole population, an estimated 8453 (95% CI 7568–9363) residents fell ill with gastroenteritis in the whole of

Nokia (Table 2). The excess number of cases was 6500 (95% CI 5613–7370) in Nokia, compared to the control population. The gastroenteritis attack rate was eightfold higher in the polluted area (53·0%) and 2·4-fold higher in the unpolluted area (15·6%) compared to the control municipality (6·5%). The median duration of gastroenteritis was 3 days for cases in the polluted area, 2 days in the unpolluted area and 2 days in the control population ($P < 0.001$). The probability of falling ill increased along with the quantity of tap water consumed in the contaminated area, but not in the uncontaminated area or in the control population.

Microbiological findings

Seven enteropathogens were detected in faecal specimens (Table 3). The most frequently encountered

Table 2. *Background populations, response rates and main findings in the three study populations according to questionnaire data*

	Nokia, contaminated	Nokia, uncontaminated	Control population
Total population	9538	20 478	27 259
Sample size*	1021	979	1000
Accepted responses	808	717	598
Accepted response rate	79 %	73 %	60 %
Total number of cases in the study	428	112	39
Estimated total number of cases (95 % CI)	5174 (4834–5513)	3279 (2731–3850)	1788 (1267–2362)
Estimated excess number of cases (95 % CI)	4519 (4118–4911)	1981 (1295–2671)	0
Attack rate (95 % CI)	53·0 % (49·–56·4)	15·6 % (13·1–18·5)	6·5 % (4·8–8·8)
OR (95 % CI)	7·5 (4·3–10)	1·95 (1·05–3·6)	

OR, Odds ratio; CI, confidence interval.

A gastroenteritis case is defined as a person with ⩾3 loose stools/day or vomiting between 28 November 2007 and 20 January 2008.

Odds ratio: logistic regression for fulfilling the case criteria, compared to the control population.

* The original sample size was 1000 persons in both the uncontaminated and the contaminated groups. Assessment of the contaminated area was later defined according the data accumulating, and 21 persons were then moved from the uncontaminated to the contaminated group.
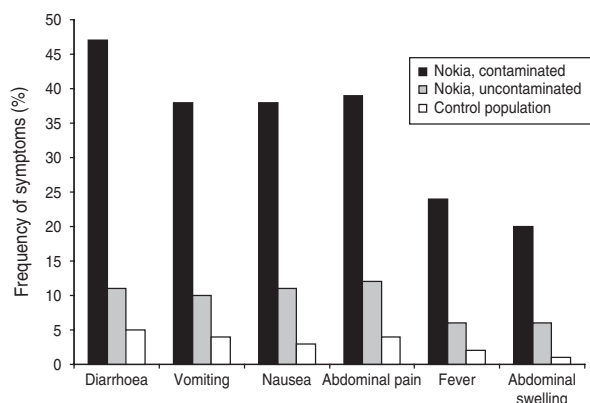

Fig. 4. Frequency of the most common symptoms in the three study populations, according to the questionnaire study.

pathogen was *Campylobacter* sp., with *C. jejuni* representing most of them, followed by *Giardia*. Five random samples investigated for norovirus were collected on 2 December 2007 and all were positive. Other findings included non-typhoidal salmonellas, *Clostridium difficile* and rotavirus. Four stool cultures grew *Shigella boydii*. In ten persons, two different bacterial pathogens were found in faecal specimens. Except for *Shigella boydii*, the same pathogens also were detected in water-distribution system samples. Ninety-eight percent (158/162) of *Campylobacter*- and *Salmonella*-positive cultures occurred within the first 3 weeks of the outbreak.

Table 3. *Microbial findings in water and stool specimens*

	Found in water samples	Found in faecal samples n (number of persons tested)
Campylobacter sp.	+	148 (539)
Norovirus	+	7 (10)
Giardia	+	55 (872)
Salmonella sp.	+	14 (689)
Clostridium difficile	+	6 (65)*
Rotavirus	+	17 (31)
Shigella boydii	n.d.	3

n.d., Not done.

Data represent findings from 28 November to 31 December 2007, except for *Giardia*, where the time period was extended to 31 May 2008. Five of the ten samples for norovirus detection were collected within 1 week after onset of the outbreak; all five were positive. Microbiology was not studied in all patients attending healthcare.

* Only culture-confirmed findings included.

DISCUSSION

This paper describes the largest reported waterborne outbreak in Finland to date. In the town of Nokia, over 8000 residents (28 % of the population) were estimated to have fallen ill with gastroenteritis, 6500 of them as a consequence of water contamination. Over half of the population in the contaminated area suffered from gastroenteritis.

Although faecal contamination is a known cause of waterborne epidemics [8, 23–26], the chain of events in Nokia was exceptional. In most circumstances, faecal contamination occurs when small amounts of sewage are mixed with clean water as a result of an obstructed or broken waste-water line or drainage from surface sources, e.g. after heavy rainfall. However, in the present case, sewage had direct entry to the distribution line through an inappropriate cross-connection. As a result, the magnitude of contamination was substantial.

The purpose and origin of the inappropriate valve was not ascertained in the investigation. It was built some decades ago, and was not prohibited during that time. After the outbreak, the national authorities required waterworks across the country to rule out the presence of similar cross-connection constructions.

Contacts with healthcare are often used to quantify the burden of illness in outbreak investigations. However, experience from previous outbreaks indicates that a large proportion of affected subjects have a self-limiting disease and do not contact healthcare providers [17]. Thus, if only health centre visitors' data are used, the burden of disease would be underestimated. In this study, a population-based survey was employed to avoid this problem. Since gastroenteritis is a common disease, a survey without proper controls might overestimate the magnitude of an outbreak if assessment of background morbidity is not done [27]. To obviate this challenge, we used a control population chosen from the same region but on the opposite side of the Tampere city area. Daily contacts between these communities are likely to be minimal, so that baseline morbidity could be estimated from the control population, lending accuracy to conclusions regarding the morbidity from the water system contamination.

The gastroenteritis attack rate was high in the contaminated area, where over half of the population fell ill. This probably reflects the high concentration of pathogens in the drinking water as well as their infectivity, especially that of viruses [20]. An even higher attack rate has been reported during a large waterborne norovirus outbreak in Finland [14]. In that case, however, the epidemic lasted longer and was investigated using an internet survey, making selection bias more likely.

There was also excess morbidity among residents in the uncontaminated area of the town. This can be explained by exposure to polluted water upon visiting the affected area. Another likely explanation is person-to-person transmission of certain pathogens, especially viruses. The incidence in the uncontaminated area increased later and less sharply than in the contaminated area, which would support the hypothesis of secondary spread. Furthermore, what is referred to here as the affected area is an estimate, which was grounded on a thorough judgement of available network and microbiological data. However, it may not be completely precise.

The duration of illness was longer in those living in the affected area. Bacterial infections were possibly more common in residents in the contaminated area, while in the uncontaminated area secondary spread of viral infections may have been more common. Another reason may be the quantity of exposure. Those living in the affected area were probably more extensively exposed to contaminated water than others, who perhaps ingested contaminated water only occasionally. Furthermore, those most intensively exposed may have been infected with several pathogens, either simultaneously or consecutively.

The frequency of previous episodes of gastroenteritis during the 10 months preceding the outbreak was 17% in the whole study population without differences between study groups. This is in good agreement with the yearly incidence of gastroenteritis (19·4/100 person-years) in a British cohort [28].

The survey was conducted as soon as possible after the outbreak. Since the outbreak took place shortly before the Christmas and New Year holiday season, it still took over 2 months before the survey could be launched. This conceivably resulted in some degree of recall bias. In addition, this outbreak gained wide coverage in the public media, which may have caused over-reporting of symptoms. The response rate of those who fell ill during the outbreak may have been higher, which might lead to overestimation of the disease burden. Nevertheless, the overall response rates in all study areas were relatively good, making significant bias unlikely.

The spectrum of pathogens in both water and patient samples was wide, consistent with the mechanism of contamination [24, 25]. This distinguishes the outbreak from most Finnish waterborne epidemics, where usually only *Campylobacter* and/or norovirus are seen. Although samples were not systematically studied in all symptomatic patients, almost 200 *Campylobacter*-associated cases were confirmed in this outbreak, which is about 200 times more than the mean monthly number ($n = 1$) for *Campylobacter* cases during the 24 pre-outbreak months in Nokia

(National Infectious Disease Register). Only a few samples were collected to detect norovirus in the early days of the epidemic, but all (5/5) were positive. Despite the small number of confirmed cases, we regard norovirus as a major pathogen in this outbreak. A number of faecal specimens collected for other purposes were later analysed for enteric viruses, and almost a third of them contained norovirus [20]. Astroviruses, adenoviruses, rotaviruses and enteroviruses were also detected. In another study connected to this outbreak and covering children attending the University Hospital, 40 % (20/50) of samples yielded norovirus. In the same study, a variety of other viral pathogens were also detected, and a notable proportion of patients yielded mixed viral findings in their stools [29]. In addition to *Campylobacter* and norovirus, *Giardia* was regarded as a major pathogen. Although *Giardia* has been isolated from natural sources in Finland [30], domestic infections are uncommon.

Drinking-water contamination was considered to be the obvious source of this outbreak, and this study provides strong evidence to support the assumption. Most subjects fell ill shortly after the valve was opened and microbes found in stools were in accord with findings in pipeline samples. Finally, the incidence of the cases was highest in the contaminated area.

Regardless of the severity of the event, the clinical consequences were milder than feared. Most of those afflicted in Nokia suffered self-limiting gastroenteritis and no permanent damage to their health is anticipated. To our knowledge, there were no obvious fatal outcomes. However, Contributory influences in the case of deaths mainly for other reasons cannot be excluded. If waterborne outbreaks cause fatalities, these are most often caused by STEC O157:H7. Although O157:H7 was present in Finland during the 1990s, it is nowadays uncommon [31] and was not detected in this outbreak. Hepatitis A virus (HAV) is another severe infection associated with waterborne exposure. HAV is highly uncommon in the Finnish population, but sporadic cases do occur. HAV was not found in water samples during this outbreak, and no hepatitis cases were detected. *Clostridium difficile* was identified, but the hypervirulent ribotype O27 was absent (S. Kotila *et al.*, unpublished observation).

This study demonstrates the value of a population-based controlled survey in estimating disease burden in outbreaks when illnesses are mild or moderate. In this particular case, the survey showed that the actual number of those affected was almost seven times higher than the number of people seeking medical care. Furthermore, the use of a control population affords greater accuracy when assessing excess morbidity connected to an outbreak.

APPENDIX. The Pirkanmaa Waterborne Outbreak Study Group

Pekka Collin[1,2], Markku Korpela[1,2], Anna-Leena Kuusela[1,2], Sami Mustajoki[1,2], Heikki Oksa[1,2], Sirpa Räsänen[1], Terhi Uotila[2] and Tina Katto[2].

([1] University of Tampere, [2] Tampere University Hospital).

ACKNOWLEDGEMENTS

DECLARATION OF INTEREST

None.

REFERENCES

1. **Pruss A, Kay D, Fewtrell L.** Estimating the burden of disease from water, sanitation and hygiene at a global level. *Environmental Health Perspectives* 2002; **110**: 537–542.
2. **Reynolds KA, Mena KD, Gerba CP.** Risk of waterborne illness via drinking water in the United States. *Reviews of Environmental Contamination & Toxicology* 2008; **192**: 117–158.
3. **Mac Kenzie RW, et al.** A massive oubreak in Milwaukee of Cryptosporidium infection transmitted through the public water supply. *New England Journal of Medicine* 1994; **331**: 161–167.
4. **Auld H, MacIver D, Klaassen J.** Heavy rainfall and waterborne disease outbreaks: the Walkerton example.

Journal of Toxicology and Environmental Health 2004; **67**: 1879–1887.

5. **Smith A, et al.** Outbreaks of waterborne infectious intestinal disease in England and Wales, 1992–2003. *Epidemiology and Infection* 2006; **134**: 1141–1149.

6. **Nygård K, et al.** A large community outbreak of waterborne giardiasis – delayed detection in a non-endemic urban area. *BMC Public Health* 2006; **6**: 141.

7. **Neringer R, Andersson Y, Eitrem R.** A waterborne outbreak of giardiasis in Sweden. *Scandinavian Journal of Infectious Diseases* 1987; **19**: 85–90.

8. **Carrique-Mas J, et al.** A Norwalk-like virus waterborne community outbreak in a Swedish village during peak holiday season. *Epidemiology and Infection* 2003; **131**: 737–744.

9. **Martin S, et al.** A case-control study to investigate concomitant waterborne outbreaks of Campylobacter and gastroenteritis in Soderhamn, Sweden. *Journal of Water and Health* 2006; **4**: 417–424.

10. **Nygård K, et al.** Emerging genotype (GGIIb) of Norovirus in drinking water, Sweden. *Emerging Infectious Diseases* 2003; **9**: 1548–1552.

11. **Hrudey EH, Hrudey EJ.** Published case studies of waterborne disease outbreaks – evidence of a recurrent threat. *Water Environment Research* 2007; **79**: 233–245.

12. **Miettinen I, et al.** Waterborne epidemics in Finland in 1998–1999. *Water Science and Technology* 2001; **43**: 67–71.

13. **Lahti K, Hiisvirta L.** Causes of waterborne outbreaks in community water systems in Finland: 1980–1992. *Water Science and Technology* 1995; **31**: 33–36.

14. **Kuusi M, et al.** Internet use and epidemiological investigation of gastroenteritis outbreak. *Emerging Infectious Diseases* 2004; **10**: 447–450.

15. **Kukkula M, et al.** Outbreak of viral gastroenteritis due to drinking water contaminated by Norwalk-like viruses. *Journal of Infect Diseases* 1999; **180**: 1771–1776.

16. **Kuusi M, et al.** An outbreak of gastroenteritis from a nonchlorinated community water supply. *Journal of Epidemiology & Community Health* 2004; **58**: 273–277.

17. **Kuusi M, et al.** A large outbreak of campylobacteriosis associated with a municipal water supply in Finland. *Epidemiology and Infection* 2005; **133**: 593–601.

18. **Maunula L, Miettinen I, von Bonsdorff C-H.** Norovirus outbreaks from drinking water. *Emerging Infectious Diseases* 2005; **11**: 1716–1721.

19. **Hänninen ML, et al.** Detection and typing of *Campylobacter jejuni* and *Campylobacter coli* and analysis of indicator organisms in three waterborne outbreaks in Finland. *Applied and Environmental Microbiology* 2003; **69**: 1391–1396.

20. **Maunula L, et al.** Enteric viruses in a large waterborne outbreak of acute gastroenteritis in Finland. *Food and Environmental Virology* 2009; **1**: 31–36.

21. **Rimhanen-Finne R, et al.** Contaminated water caused the first outbreak of giardiasis in Finland, 2007: a descriptive study. *Scandinavian Journal of Infectious Diseases* (in press).

22. **R Development Core Team.** R: A language and environment for statistical computing. Vienna: R foundation for statistical computing, 2009 (http://www. R-project.org).

23. **Anon.** Sewage contamination of drinking water in Bolton. *Communicable Disease Report Weekly* 1998; **8**: 431.

24. **Gallay A, et al.** A large multi-pathogen waterborne community outbreak linked to faecal contamination of a groundwater system, France, 2000. *Clinical Microbiology and Infection* 2006; **12**: 561–570.

25. **Vestergaard L, et al.** Outbreak of severe gastroenteritis with multiple aetiologies caused by contaminated drinking water in Denmark, January 2007. *Eurosurveillance* 2007; **12**: E070329.1.

26. **Häfliger D, Hubner P, Luthy J.** Outbreak of viral gastroenteritis due to sewage-contaminated drinking water. *International Journal of Food Microbiology* 2000; **54**: 123–126.

27. **Hunter P, Syed Q.** Community surveys of self-reported diarrhoea can dramatically overestimate the size of outbreaks of waterborne gastroenteritis. *Water Science and Technology* 2001; **43**: 27–30.

28. **Wheeler J, et al.** Study of infectious intestinal disease in England: rates in the community, presenting to general practise, and reported to national surveillance. *British Medical Journal* 1999; **318**: 1046–1050.

29. **Räsänen S, et al.** Mixed viral infections causing acute gastroenteritis in children in a waterborne outbreak. *Epidemiology and Infection* 2010; **138**: 1227–1234.

30. **Horman A, et al.** Campylobacter spp., Giardia spp., Cryptosporidium spp., noroviruses and indicator organisms in surface water in southwestern Finland, 2000–2001. *Applied and Environmental Microbiology* 2004; **70**: 87–95.

31. **Kuusi M, Siitonen A, Eklund M.** In: Iivonen J, Kela E, Kuusi M, Lyytikäinen O, Ruutu P, eds. *Infectious Diseases in Finland 1994–2004*. Helsinki, Finland: National Public Health Institute, 2005.