A fatal waterborne disease epidemic in Walkerton, Ontario: comparison with other waterborne outbreaks in the developed world

S.E. Hrudey*, P. Payment, P.M. Huck***, R.W. Gillham**** and E.J. Hrudey***

* University of Alberta, Dept of Public Health Sciences, Edmonton, Alberta, T6G 2G3, Canada (E-mail: *steve.hrudey@ualberta.ca*)

** Université du Québec, INRS-Institut Armand-Frappier, 532 Boul des Prairies, Laval PQ, Canada H7V 1B7

*** Dept of Civil Engineering, University of Waterloo, 200 University Ave W, Waterloo, Ontario, N2L 3G1, Canada

**** Dept of Earth Sciences, University of Waterloo, 200 University Ave W, Waterloo, Ontario, N2L 3G1, Canada

Abstract An estimated 2,300 people became seriously ill and seven died from exposure to microbially contaminated drinking water in the town of Walkerton, Ontario, Canada in May 2000. The severity of this drinking water disaster resulted in the Government of Ontario calling a public inquiry by Mr. Justice Dennis O'Connor to address the cause of the outbreak, the role (if any) of government policies in contributing to this outbreak and, ultimately, the implications of this experience on the safety of drinking water across the Province of Ontario. The circumstances surrounding the Walkerton tragedy are an important reference source for those concerned with providing safe drinking water. Although some circumstances are obviously specific to this epidemic, others are uncomfortably reminiscent of waterborne outbreaks that have occurred elsewhere. These recurring themes suggested the need for attention to broad issues of drinking water security and they present the challenge for how drinking water safety can be managed to prevent such tragedies in the future.
Keywords *Campylobacter jejuni*; *Escherichia coli* 0157:H7; health risk; multiple barriers; outbreaks; Walkerton Inquiry

Introduction

The public health implications of serious drinking water contamination that occurred in May 2000 in Walkerton, Canada (a community of 4,800 residents located about 175 km northwest of Toronto) hold important lessons for the water industry. More than 2,300 individuals experienced gastroenteritis, 65 were hospitalised, 27 developed haemolytic uraemic syndrome (HUS; a serious and potentially fatal kidney ailment) and seven died. The pathogens identified as being primarily responsible were *Escherichia coli* 0157:H7 and *Campylobacter jejuni* although other pathogens were likely to have been present. The Ontario government established a public inquiry to determine the causes and responsibility for this tragedy (Part 1) and to examine broader questions relating to the safety of drinking water in Ontario (Part 2).

We were retained by the Walkerton Inquiry to assist in establishing the physical cause of the contamination for Part 1 (Huck, Payment and Gillham were expert witness panel members) and to advise the Commissioner on technical matters for Parts 1 and 2 (S.E. Hrudey was a member of the Research Advisory Panel). The determination of cause, based on all of the evidence before the Inquiry, was the purview of the Commissioner, Justice Dennis O'Connor. Part 1 of the Inquiry was held in Walkerton over a period of nine months during which evidence was heard from 114 witnesses, including residents, local officials, senior civil servants, two former ministers of environment and the Premier of Ontario.

Water Science and Technology **Vol 47 No 3 pp 7–14** © IWA Publishing 2003

The Part 1 report (O'Connor, 2002) was made public on January 18, 2002. This landmark document provided a detailed and sobering account of the many factors that contributed to this tragedy and its serious consequences. Suffering was not limited to those who were ill but included all those who struggled to cope with the severe illness of their loved ones. This report should become required reading for all parties involved in the delivery of drinking water to reinforce the magnitude of personal responsibilities and range of challenges that are inherent in undertaking to provide safe drinking water to the public.

Waterborne disease outbreaks continue to occur in North America and Western Europe despite wealthy economies and access to proven drinking water treatment technologies. The obvious question is: *Why do serious failures continue to occur?* Our analysis considered selected waterborne disease outbreaks reported in the accessible literature in relation to the failures documented by the Walkerton Inquiry. In seeking to prevent this type of tragedy from recurring, a minimum requirement ought to be to ensure that the main factors contributing to the Walkerton disaster, and those that can be readily recognised from elsewhere, are fully understood and considered as a basis for recommending improvements.

Materials and methods

We attempted an analysis of the major factors contributing to drinking water disease outbreaks by searching the published English language literature over the past 30 years. We screened papers for those that discussed specific disease outbreaks and which described some of the failure modes contributing to the outbreak. We did not attempt to review all outbreaks and this paper is a very selective description of the relevant literature. There was substantial variation in the quality and detail of description for failure mechanisms among the papers that we retrieved. The multiple barrier approach was adopted as our reference framework for analysing the outbreak literature.

Results and discussion

Other outbreaks evaluated

Table 1 summarises a selection of 15 outbreaks from four countries each affecting between 47 and a potential of >400,000 people including a total of at least 11 deaths attributed to waterborne disease. The factors reported to have contributed to these selected outbreaks are considered in relation to the documented failures contributing to the Walkerton outbreak.

Multiple barrier failures

The Commissioner of the Walkerton Inquiry (O'Connor, 2002) adopted the expert evidence that a multiple barrier approach was necessary for providing safe drinking water, consisting of effective and robust measures (Huck *et al.*, 2001) dealing with the following main elements: (1) source, (2) treatment, (3) distribution, (4) monitoring and (5) response. The Walkerton tragedy involved evident or potential failures in barriers for all of these elements. Review of other outbreaks revealed that they commonly involved failures in more than one barrier, a finding that reinforced the need for the multiplicity of barriers and the assured effectiveness of each.

Source. Contamination commonly affects the raw water supply and constant vigilance is required to minimise sources of contamination. Recognition of patterns that are associated with contamination potential is essential. The pathogens (*E. coli* 0157:H7 and *C. jejuni*) causing the Walkerton outbreak were attributed to contamination of the shallow (5–8 m) well #5 arising from cattle manure from a nearby farm following a period of heavy spring rainfall. Recently, a review (Curriero *et al.*, 2001) of 548 outbreaks in the US over almost 50 years found that 51% were preceded by precipitation events at the 90th percentile of

Table 1 Summary of selected waterborne disease outbreaks

Location and dates	Characteristics	Reference
Richmond Height, FL, USA January–March 1974	1,200 cases of gastroenteritis, likely shigellosis served by chlorinated shallow (6–15 m) groundwater	Weissman *et al*., 1976
Bradford, PA, USA September–December 1979	3,500 cases of gastroenteritis of unidentified aetiology in a chlorinated groundwater supply	Akin & Jakubowski, 1986
Bramham, England July 1980	3,000 cases of gastroenteritis of unidentified aetiology in a direct filtered and chlorinated supply	Short, 1988
Eagle-Vail, CO, USA March 1981	80 cases of gastroenteritis likely rotavirus in a direct filtered and chlorinated supply	Hopkins *et al*., 1986
Orangeville, ON, Canada April 1985	241 cases of campylobacteriosis in an unchlorinated groundwater supply	Millson *et al*., 1991
Pittsfield MA, USA November 1985–January 1986	3,800 cases of giardiasis in a chlorinated but unfiltered water supply	Kent *et al*., 1988
Disraeli, PQ, Canada August 1986	50 cases of gastroenteritis associated with three cases of campylobacteriosis in an unchlorinated, unfiltered surface supply	Tessier *et al*., 1987
Penticton, BC, Canada June 1986	3,000 cases of giardiasis in a chlorinated, unfiltered, surface/groundwater supply	Moorehead *et al*., 1990
Oakcreek Canyon, AZ, USA April 1989	11/240 guests surveyed had gastroenteritis likely caused by a Norwalk-like virus in an unchlorinated private well	Lawson *et al*., 1991
Cabool, MO, USA December 1989–January 1990	243 cases of gastroenteritis including 86 cases of bloody diarrhoea, 2 cases of HUS and 4 deaths caused by *E. coli* 0157:H7 in an unchlorinated community water supply	Swerdlow *et al*., 1992
Uggelose, Denmark November 1992–February 1993	1,400 cases of gastroenteritis of suspected viral aetiology in filtered, unchlorinated municipal supply	Laursen *et al*., 1994
Warrington, England November 1992–February 1993	47 confirmed cases of cryptosporidiosis in a water supply zone serving 38,000 consumers by groundwater with chlorination only	Bridgman *et al*., 1995
Milwaukee, WI, USA March–April 1993	Possibly 400,000 cases of cryptosporidiosis in a filtered, chlorinated surface supply	MacKenzie *et al*., 1996
Gideon, MO, USA December 1993	600 cases of salmonellosis, 15 hospitalisations and 7 deaths in an undisinfected groundwater supply	Clark *et al*., 1996
North Battleford, SK, Canada April 2001	1,900 cases of cryptosporidiosis in a chlorinated, filtered surface supply	Stirling *et al*., 2001

intensity and 68% were preceded by precipitation events at the 80th percentile. In six of the outbreaks in Table 1 (Orangeville, Warrington, Bradford, Uggelose, Disraeli and Penticton) either heavy rainfall or runoff from heavy snow-melt was directly implicated. Clearly, severe weather should demand heightened awareness and active inquiry by water personnel into the potential for contamination.

Well #5 at Walkerton was located close to two farms posing a water contamination risk, one of which was judged to be the source of the 2000 outbreak, notwithstanding having followed model environmental management farming practices. Concerns about contamination from farming were raised at the commissioning of the well in 1978 but no explicit source protection measures were ever adopted. Poor location was a factor at Disraeli where

the buried intake was subject to contamination from a park that (a) had hosted a large public event attended by 3,000 people without adequate sanitation facilities, (b) was adjacent to three open abandoned wells and (c) was subject to influence from a nearby river downstream from pig farms. Orangeville was supplied by six unchlorinated wells, one of which was found to be under the influence of surface drainage (faecal coliforms at >600/L) from mixed farming operations. Penticton had a surface water intake for an unfiltered, chlorinated supply in a location accessible to and frequented by *Giardia*-infected beavers as well as domestic animals. Uggelose had its wellhead exposed to backup and flooding by a drain connected to a sanitary sewer.

Poor geological conditions existed at Walkerton well #5 which was shown to be subject to surface contamination during pump testing (faecal coliforms appearing after 24 h) in 1978. The shallow production zone consisted of highly weathered bedrock with closely spaced horizontal and vertical fractures. Point source breaching of soil overburden followed by rapid horizontal transport in the fractured bedrock was the most plausible contamination route. Richmond Heights and Oakcreek Canyon were both outbreaks where sewage effluent infiltration was found to have rapid travel times (9 h and 3–11 respectively) to well intakes during subsequent dye tests. Bramham had geology that allowed a sanitary sewer overflow to reach the well intake in 8 h.

Treatment. The Walkerton operators were asked to provide a chlorine residual (majority to be free chlorine) of 0.5 mg/L after 15 min. However, monitoring for chlorine residual was only expected once a day, in accordance with 1978 policy for groundwater that showed bacterial contamination. These conditions were never adopted as express conditions of the Certificate of Approval, the formal regulatory approval for this well. Evidence at the Inquiry revealed that chlorine dosage practice at Well #5 was insufficient to achieve a 0.5 mg/L residual even in the absence of any chlorine demand. Although the evidence did not allow for an estimate of the chlorine demand at the time Well #5 was contaminated, it was reasonable to assume that the contamination causing this outbreak was accompanied by a chlorine demand sufficient to consume entirely, or almost entirely, the low chlorine dose thereby allowing inadequately disinfected water into the distribution system. [For reference, maintaining a 0.5 mg/L free chlorine residual after 15 min would have satisfied the CT requirement for 99% kill of *E. coli* O157 at 5°C, pH 7.0 (Rice *et al*., 1999) by 20-fold and the CT for 99.99% kill by 7.5-fold.] Inadequate chlorine disinfection was evident in the Bradford, Pittsfield, Eagle-Vail, Richmond Heights and Bramham outbreaks and no disinfection was required for contamination-susceptible systems at Orangeville, Disraeli, Oakcreek Canyon, Gideon and Uggelose.

The protozoan pathogen *Cryptosporidium* is resistant to chlorine disinfection and *Giardia* demands optimal chlorine disinfection. Despite the recently demonstrated efficacy of UV inactivation, robust management of outbreak risks from these pathogens requires an effective filtration treatment barrier, either chemically assisted granular media filtration or membrane filtration. Accordingly, several outbreaks have occurred because of inadequate filtration performance including Milwaukee and North Battleford.

Distribution. Possible scenarios whereby the distribution system at Walkerton could have been contaminated were investigated and carefully evaluated by the Inquiry. These included (i) installation of new water mains at three locations, (ii) a fire event with potential for associated depressurisation, (iii) mains breaks and repairs at four locations in March 2000, (iv) potential for contamination of two treated water storage standpipes, (v) cross connections found at eight private wells and many private cisterns, (vi) potential for cross contamination of water mains by sanitary sewers and (vii) surface flooding in the town

during the heavy rainfall of May 12, 2000. Although one or more of these elements offered the potential for causing contamination of the Walkerton distribution system, none provided an adequate explanation for the contamination that caused the outbreak. Rather, Justice O'Connor concluded that the primary, if not the only, source of contamination of the Walkerton water system was cattle manure from a farm near well #5. Residents of Walkerton were probably first exposed to this contamination shortly after the May 12 heavy rainfall.

Previously, the most serious outbreak from *E. coli* 0157 contamination of drinking water occurred in Cabool causing four deaths in 1989. This was determined to have been most likely caused by distribution system contamination with sanitary sewage during repairs of two broken water mains and/or replacement of 43 water meters in this town that was served by an unchlorinated groundwater supply. The sanitary sewer system was found to be prone to infiltration of storm runoff with subsequent flooding of water distribution lines and water meter box sites. The Warrington outbreak was apparently caused by a corroded distribution line that allowed ingress of contaminated water from an adjacent chamber with an unsuspected cross connection to a septic tank. The Uggelose outbreak was caused by a sanitary sewer backup that contaminated the water supply wellhead by flowing back up a drain connecting the wellhead to the sewer. Apparently, the Gideon outbreak was caused by bird faeces contaminating a treated water storage tank. The Pittsfield outbreak was thought to have been caused by bringing an auxiliary reservoir back on-line for the first time in three years. This change caused increased turbidity levels in the distribution system because of flow reversals during the switchover. The auxiliary reservoir itself was found to be vulnerable to contamination by beavers and muskrats.

Monitoring. The quality monitoring requirements for the Walkerton water system were daily measurements of chlorine residual and a specified number of monthly raw and distribution system samples for microbiological testing. For the latter, the number of samples collected (1 raw, 1 treated and 2 distribution samples for each well in operation per month for a typical total of eight samples) was routinely below the required number of 13/month. Samples were routinely mislabeled as to where they were actually taken. Notably, samples from the distribution system were often not taken. Rather, samples were generally taken near the treated well water location so it was not possible to judge the microbiological quality of distribution system samples. Despite this deception, the microbiological monitoring program disclosed adverse results including presence of *E. coli* in treated water on several occasions. This recurring presence of *E. coli* in treated water provided direct evidence of dysfunctional chlorination because of the documented susceptibility of *E. coli* to chlorine disinfection. Even more troubling was the disclosure that chlorine residuals were not measured daily as required and that daily chlorine residual monitoring logs were routinely falsified with repeated fictitious entries of exactly 0.5 or 0.75 mg/L. Chlorine residuals measured by Ministry of Environment inspectors were always lower than 0.5 mg/L yet the discrepancies with the falsified records were neither formally challenged nor rectified.

The Inquiry noted that Ontario Drinking Water Objectives were modified in 1994 to recognise that unfiltered water, supplied from groundwater that was under the direct influence of surface water, required continuous chlorine residual monitoring and effectively continuous turbidity monitoring. Unfortunately, there were no criteria specified to identify such systems and no program was implemented to review Certificates of Approval to water systems that had been granted before the new policy. As a result, well #5, which had shown evidence of being under the direct influence of surface water at commissioning in 1978, was not required to implement the new policy. Significantly, after considering all of the

evidence presented, Justice O'Connor found that if the Walkerton system had been required to satisfy the 1994 policy the fatal outbreak would have been prevented.

Both the Bramham and the Bradford outbreaks documented failures in monitoring chlorine residual that contributed to the outbreaks. At Bramham, the pump for adding chlorine was discovered to be actually bypassing chlorine to the drain. This failure had been missed because personnel did not have the equipment to check chlorine residual levels following adjustment of the chlorine dosage. The Bradford chlorination monitoring program involved improper procedures for determining the applied dosage necessary to produce a residual in the distribution system.

Response. The operators at Walkerton were qualified by experience to operate the mechanical aspects of the water system. However, the evidence indicated that they clearly lacked any substantive understanding of the need for disinfection to inactivate pathogens in drinking water and the serious health consequences that could arise from failing to maintain adequate disinfection of the Walkerton water supply. The General Manager of the system and his brother were both granted class 2 operator status as part of a voluntary "grandparenting" program in 1988 with neither having ever taken any training courses nor being required to pass any examinations. They were both upgraded to class 3 operators in 1996, again without any assessment of their knowledge or skills. Neither operator understood even the most basic elements of assuring effective chlorine disinfection. The General Manager testified that one of the reasons that they added less than the required amount of chlorine was because they had received complaints from town residents about the taste of too much chlorine in the water.

The contamination most likely entered well #5 on May 12 when 70 mm of rain fell. On May 13, 14 and 15 no chlorine residuals were measured and the opportunity to detect the contamination of well #5 by the absence of a chlorine residual was foregone. On May 15, well #5 was shut down and well #7 was brought on-line without a chlorinator. Although the latter failing clearly played a role in the evasive behaviour of the General Manager over subsequent days, substantial contamination from well #7 was judged to be unlikely. Microbial monitoring samples were taken on May 15 at sites that were undoubtedly mislabeled but included samples that were most likely taken near to well #5. These Walkerton distribution samples were reported to the General Manager on May 17. They included membrane filter analyses that showed gross contamination with total coliforms and *E. coli*. Gastrointestinal illness began to appear in the health care system on May 18 and public health authorities were alerted on May 19. The local health unit contacted the General Manager to ask whether there was any problem with the water supply. He replied that he thought the water was "okay". Thereafter he began flushing the distribution system and super-chlorinating so that when contacted again by the health unit on May 20 he reassured them that there was a chlorine residual in the distribution system. By midday on May 21, the Owen Sound hospital laboratory had confirmed an identification of *E. coli* 0157:H7 from one patient and announced a presumptive result for another. The local health unit then issued a boil water advisory despite the assurances from the General Manager on the previous two days.

The problem of residents complaining of chlorine taste being mentioned by operators as a reason for keeping chlorine residuals unacceptably low was also cited in the Bramham outbreak. Likewise, a review (Lahti and Hiisvirta, 1995) of waterborne outbreaks in Finland also referred to one unspecified community outbreak in which concerns about formation of chlorinated organic compounds were cited as the reason for lowering chlorination to ineffective levels leading to an outbreak affecting a few hundred people. In the Gideon outbreak leading to seven deaths from salmonellosis, it was later found that many

residents continued to drink unboiled water after the boil water order was issued because they did not understand the severity of the situation. Consumers only responded when information sheets that clearly explained the need and rationale for the boil water order were delivered to their homes. Finally, at the extreme of response failure, from the Eagle-Vail outbreak, chlorination failed for 24 h when a chlorine cylinder emptied, triggering an off-site alarm, but it was turned off with no investigation. Subsequently, a continuous chlorine residual monitor with an alarmed, automatic water system shutoff requiring a manual restart was installed.

Conclusions

A multiplicity of failures occurred in Walkerton, despite the readily accessible experience from elsewhere warning of similar failure modes. The challenge for improving drinking water system safety is to reform the pervasive culture of complacency that was evident among so many key players in the water system. Such complacency must be replaced with a culture of personal responsibility and vigilance. This poses a challenge, in part, because we have been successful in making waterborne disease outbreaks very rare in developed countries. Furthermore, because a single barrier failure may not cause a total system failure leading to an outbreak, there may be inadequate remedial attention given to individual failures because each alone appears to have limited or no consequences. Internal consequences to institute remedial action need to be created by system management. For the multiple barrier framework to succeed in preventing outbreaks, rather than allowing continual feedback indicating that an individual failure will likely not cause catastrophe by itself, all elements must be maintained effective by requiring their individual performance to meet effectiveness criteria.

Acknowledgements

The authors are indebted to Justice Dennis O'Connor for documenting so thoroughly what happened in Walkerton, Dr. Harry Swain for guiding the Research Advisory Panel and Mr. Paul Cavalluzzo, Inquiry Chief Counsel for reviewing earlier publications by the authors.

References

Akin, E.W. and Jakubowski, W. (1986). Drinking water transmission of giardiasis in the United States. *Wat. Sci. Tech.*, **18**(10), 219–226.

Bridgman, S.A. *et al*. (1995). Outbreak of cryptosporidiosis associated with a disinfected groundwater supply. *Epidemiol. Infect.*, **115**, 555–566.

Clark, R.M. *et al*. (1996). Tracking a *Salmonella* serovar *typhimurium* outbreak in Gideon, Missouri: role of contaminant propagation model. *Aqua*, **45**(4), 171–196.

Curriero, F.C., Patz, J.A., Rose, J.B. and Lele, S. (2001). The association between extreme precipitation and waterborne disease outbreaks in the United States, 1948–1994. *Am. J. Publ. Hlth.*, **91**(8), 1194–1199.

Hopkins, R.S., Karlin, R.J., Gaspard, G.B. and Smades, R. (1986). Gastroenteritis: case study of a Colorado outbreak. *J. Am. Wat. Wks. Assoc.*, **78**(1), 40–44.

Huck, P.M., Payment, P., Hrudey, S.E. and Anderson, W.B. (2001). A severe waterborne disease outbreak in Walkerton, Ontario: issues relating to treatment and distribution. *Wat. Qual. Technol. Conf.*, Nashville, Am. Water Works Assoc. Paper M8-2 (on CD-ROM).

Kent, G. P. *et al*. (1988). Epidemic giardiasis caused by a contaminated public water supply. *Am. J. Pub. Hlth.*, **78**(2), 139–143.

Lahti, K. and Hiisvirta, L. (1995). Causes of waterborne outbreaks in community water systems in Finland: 1980–1992. *Wat. Sci. Tech.*, **31**(5–6), 33–36.

Laursen, E.O., Mygind, O., Rasmussen, B. and Ronne, T. (1994). Gastroenteritis: a waterborne outbreak affecting 1,600 people in a small Danish town. *J. Epidemiol Comm. Hlth.*, **48**, 453–458.

Lawson, H.W. *et al*. (1991). Waterborne outbreak of Norwalk virus gastroenteritis at a southwest US resort: role of geological formations in the contamination of well water. *The Lancet*, **337** (May 18), 1200–1204.

MacKenzie, W. *et al*. (1994). A massive outbreak in Milwaukee of *Cryptosporidium* infection transmitted through the public water supply. *N. Engl. J. Med.*, **331**(3), 161–167.

Millson, M. *et al*. (1991). An outbreak of *Campylobacter jejuni* gastroenteritis linked to meltwater contamination of a municipal well. *Can. J. Publ. Hlth.*, **82**, 27–31.

Moorehead, W.P. *et al*. (1990). Giardiasis outbreak from a chlorinated community water supply. *Can. J. Publ. Hlth.*, **81**, 358–362.

O'Connor, D.R. (2002). *Report of the Walkerton Inquiry: Part 1 – The Events of May 2000 and Related Issues*. The Walkerton Inquiry, Toronto, 504 pp.

Rice, E.W., Clark, R.M. and Jones, C. (1999). Chlorine inactivation of *Esherichia coli* O157. *Emerg. Inf. Dis.*, **5**(3), 461–463.

Short, C.S. (1988). The Bramham incident 1980 – an outbreak of waterborne infection. *J. Inst. Wat. Environ. Manag.*, **2**, 383–390.

Stirling, R. *et al*. (2001). Waterborne cyptosporidiosis outbreak, North Battleford, Saskatchewan, Spring 2001. *Can. Comm. Dis. Rep.*, **27**(22).

Swerdlow, D.L. *et al*. (1992). A waterborne outbreak in Missouri of *Escherichia coli* 0157:H7 associated with bloody diarrhea and death. *Ann. Intern. Med.*, **117**(10), 812–819.

Tessier, B. *et al*. (1987). Epidemic of campylobacteriosis linked to drinking water – Quebec. *Can. Dis. Wkly. Rep.*, **13–14**, 63–65.

Weissman, J.B. *et al*. (1976). An epidemic of gastroenteritis traced to a contaminated public water supply. *Am. J. Epidemiol.*, **103**, 391–398.