

Preventing community-wide transmission of *Cryptosporidium*: a proactive public health response to a swimming pool-associated outbreak – Auglaize County, Ohio, USA

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SUMMARY

The incidence of recreational water-associated outbreaks in the United States has significantly increased, driven, at least in part, by outbreaks both caused by Cryptosporidium and associated with treated recreational water venues. Because of the parasite's extreme chlorine tolerance, transmission can occur even in well-maintained treated recreational water venues (e.g. pools) and a focal cryptosporidiosis outbreak can evolve into a community-wide outbreak associated with multiple recreational water venues and settings (e.g. childcare facilities). In August 2004 in Auglaize County, Ohio, multiple cryptosporidiosis cases were identified and anecdotally linked to pool A. Within 5 days of the first case being reported, pool A was hyperchlorinated to achieve 99.9% Cryptosporidium inactivition. A case-control study was launched to epidemiologically ascertain the outbreak source 11 days later. A total of 150 confirmed and probable cases were identified; the temporal distribution of illness onset was peaked, indicating a point-source exposure. Cryptosporidiosis was significantly associated with swimming in pool A (matched odds ratio 121.7, 95% confidence interval 27.4-∞) but not with another venue or setting. The findings of this investigation suggest that proactive implementation of control measures, when increased Cryptosporidium transmission is detected but before an outbreak source is epidemiologically ascertained, might prevent a focal cryptosporidiosis outbreak from evolving into a community-wide outbreak.

Key words: Cryptosporidium, outbreaks.

INTRODUCTION

The incidence of recreational water-associated outbreaks in the United States has significantly increased

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since national surveillance began in 1978 [1]. This increase has been driven, at least in part, by outbreaks both caused by *Cryptosporidium* and associated with treated recreational water venues (e.g. pools). The first detected treated recreational water-associated outbreaks of cryptosporidiosis were epidemiologically linked to a learner pool in Doncaster, UK and a school pool in Los Angeles, United States in 1988

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[2–4]. During 2009–2010, *Cryptosporidium* caused 24 (68·6%) of 35 US-treated recreational water-associated outbreaks with an identified infectious aetiology [1].

Cryptosporidium is transmitted by the faecal-oral route and results from the ingestion of oocysts through the consumption of faecally contaminated food or water or through contact with an infected person or animal. Following ingestion the typical incubation period is 7 (range 1–12) days [5]. Cryptosporidium is extremely chlorine-tolerant with inactivation taking 3.5-10.6 days at free chlorine levels typically mandated or recommended in the United States, i.e. 1–3 mg/l or ppm [pH 7.5, temperature 77 °F (25 °C)]† [6]. Thus, Cryptosporidium transmission can occur even in well-maintained treated recreational water venues, and a focal outbreak associated with one such venue can expand into a community-wide outbreak associated with additional recreational water venues or other settings (e.g. childcare facilities) [7, 8].

There are many species within the genus Cryptosporidium, of which C. hominis and C. parvum cause >95% of human infections [9]. Identifying the Cryptosporidium species can provide key epidemiological clues. For example, C. parvum can infect both humans and ruminants (e.g. pre-weaned calves), each with their own transmission cycle that intersects in zoonotic disease; C. hominis primarily exists in a human-to-human transmission cycle [10]. C. hominis and C. parvum are morphologically indistinguishable, thus molecular typing must be done to speciate and subtype isolates. The increasing use of molecular typing of Cryptosporidium spp. has helped elucidate the epidemiology of cryptosporidiosis (e.g. differentiating clusters during outbreak investigations) [11, 12].

Outbreak detection

On 9 August 2004, Ohio's Auglaize County Health Department (ACHD) was notified of a laboratory-confirmed case of diarrhoea caused by *Cryptosporidium*. After accessing the Ohio Disease Reporting System and noting multiple additional cryptosporidiosis cases in adjacent counties, ACHD contacted the health departments in neighbouring Mercer and Shelby counties to ask if they had found any common exposures in their reported cases. Preliminary interviews revealed that several additional unreported

cases of diarrhoea had occurred in pool A's swim team members. Because this outbreak was detected a few days before a festival, with >9000 anticipated attendees who could amplify transmission, an investigation with the Ohio Department of Health (ODH) and the U.S. Centers for Disease Control and Prevention (CDC) was launched. The investigators aimed to prevent further transmission; determine the magnitude of the outbreak; and identify risk factors for cryptosporidiosis in Auglaize, Mercer, and Shelby counties.

To control transmission, prevention measures were proactively and concurrently undertaken in the tricounty area. Within 5 days after the first cases were reported, suspect pool A was hyperchlorinated on 12 and 13 August [6]. Two neighbouring pools were also closed and hyperchlorinated on the same days as pool A. All pools were immediately re-opened posthyperchlorination. Other control measures were implemented 2 days after the first case was reported to ACHD. These included conducting prevention outreach to childcare facilities (e.g. banning all water play or swimming activities, such as any play or activities involving water tables, temporary inflatable or rigid fill-and-drain swimming pools and slides, or public pool visits) public schools, physician offices, medical facilities, and nursing homes and creating a press release for the local media to convey prevention messages to the public. Signage was also created for distribution to local pools reminding patrons not to swim while ill with diarrhoea and for those diagnosed with cryptosporidiosis for an additional 2 weeks after symptoms had completely resolved. Additional measures focused on the festival and included teaching proper handwashing technique to festival food handlers, instructing food handlers to refrain from food preparation if ill, placing signage in and around festival restrooms to encourage handwashing, and strategically locating handwashing stations. Informational packets on cryptosporidiosis and the importance of good hygiene practices (e.g. handwashing), especially when ill, were distributed to festival attendees.

METHODS

Case-finding and case-control study

For this outbreak investigation, a confirmed case was defined as a resident of Auglaize, Mercer, or Shelby counties, Ohio, who (1) had diarrhoea (i.e. ≥3 loose

 $[\]dagger$ Free chlorine levels mandated or recommended in European countries are typically lower than those in the United States.

[‡] Infected persons can shed *Cryptosporidium* oocysts for up to 60 days after complete symptom resolution; however, most stop shedding oocysts within the 2 weeks after complete symptom resolution [5].

stools in a 24-h period) during July–August 2004, (2) had laboratory-based evidence of *Cryptosporidium* infection, and (3) did not travel to South Bass Island, Ohio, where another cryptosporidiosis outbreak simultaneously occurred [13]. A probable case was defined as a resident of Auglaize, Mercer, or Shelby counties, Ohio, who (1) met the above criteria but did not have laboratory-based evidence of infection and (2) was epidemiologically linked to a confirmed case. The tri-county health departments contacted healthcare providers (e.g. physicians and hospitals) in their respective jurisdictions to encourage testing of symptomatic persons and reporting of positive test results. Based on reports called into the tri-county health departments, a line list of case-patients was generated.

To identify community risk factors for cryptosporidiosis, a case-control study was conducted. The study protocol was institutionally reviewed and determined not to be human-subject research, as it was part of a public health response. A parent or guardian answered questions on behalf of a child aged <12 years; the parent's or guardian's permission was obtained prior to speaking with an assenting child aged ≥ 12 years. Case-patients thought to be the first in their respective household to become ill were recruited from the aforementioned line list. Prior to the start of the interview, the interviewer attempted to verify that the case-patient was in fact the first person in the household to become ill. Two controls, who reported no gastrointestinal illness during July-August 2004, were matched to each case-patient by age range (i.e. 0-<2, 2-<6, 6-<18, ≥ 18 years) and county of residence. Area phone books were used to identify potential controls. A person in a randomly selected household listed on the first page of each section was recruited for the study. Subsequently, a person from every tenth listed household was recruited. Only one person per household was interviewed as a control.

Investigators administered a standardized questionnaire, to case-patients and controls, asking about exposures in the 2 weeks prior to the matched case-patient's date of illness onset. Questions about the following were included: food and drinking water consumption; recreational water, childcare, house-hold, farm, and animal exposures; attendance at events with ≥50 persons; and travel history. Because hypothesis-generating interviews indicated a common recreational water exposure among case-patients, detailed questions were asked about recreational water activities in general and at specific pools, including pool A. Additionally, data were collected on the symptoms case-patients experienced and time lost

from work and from daily activities due to illness. The case-control study was launched 25 August, 16 days after the first case was reported to ACHD. All data analyses were performed using SAS version 9.3 (SAS Institute Inc., USA). Odds ratios (ORs), 95% confidence intervals (CIs), and *P* values were calculated using conditional logistic regression to account for matching.

Laboratory investigation

Stool testing for *Cryptosporidium* was requested for individuals who were experiencing diarrhoea or whose diarrhoea had resolved within the previous 2 weeks. Testing for *Cryptosporidium* was performed by ODH by direct immunofluorescent assay (DFA) and results were confirmed by DFA at CDC. *Cryptosporidium* isolates were subtyped at CDC using DNA sequence analysis [14]. Stool specimens from 25 initially identified case-patients were tested by ODH to rule out bacterial and viral aetiologies of diarrhoeal illness.

Environmental health investigation

The environmental health investigation focused on pool A, which consisted of a main pool and a wading pool and was anecdotally implicated early in the outbreak investigation. Maintenance records and daily attendance numbers were reviewed for the months of July and August. On 11 August 2004, 2 days after the first case was reported to ACHD, pool A was closed and a private company took water samples from the wading pool and the deep and shallow ends of the main pool. These samples were processed by ODH using U.S. Environmental Protection Agency Method 1622 [15]. The resulting immunomagnetic separation beads were sent to CDC for molecular analysis [16]. Additionally, aquatic staff were interviewed, using a standardized questionnaire asking about whether or not they had diarrhoea, their activities and duties at the pool, and pool patron behaviour observed during July-August 2004.

RESULTS

Case-control study

A total of 150 confirmed and probable cases were identified. The temporal distribution of illness onset was peaked, indicating a point-source exposure (Fig. 1). Of 63 case-patients who were thought to be the first to become ill in their respective household,

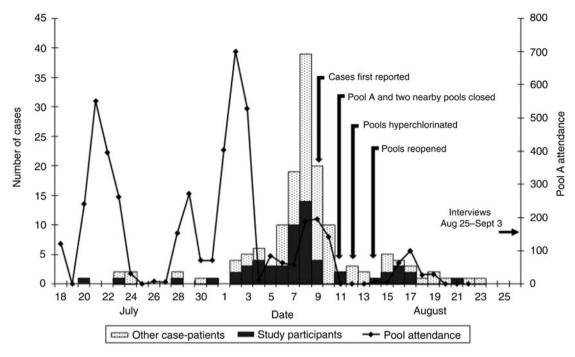


Fig. 1. Number of cryptosporidiosis cases (N = 150) by date of illness onset and pool attendance by date, Auglaize County, Ohio, July-August 2004.

55 (87.3%) were successfully recruited to participate in the case-control study and matched with 105 controls. Of the 55 enrolled case-patients (median age 8.0, range 1.3-61 years), 48 (87.3%) were children and 26 (47.3%) were male; reportedly none were immunocompromised. The most commonly reported symptoms were diarrhoea, tiredness/fatigue, loss of appetite, and abdominal cramps (Table 1). Three (5.5%) case-patients reported being seen in an emergency department; two (3.6%) reported being hospitalized. Fifty (90.9%) case-patients reportedly experienced a median of 7 (range 3–25) stools maximum in 24 h. Diarrhoea reportedly lasted a median of 6.5 (range 1–19) days. Of 52 case-patients (denominators vary due to missing, unknown, or refused values), 32 (61.5%) reported diarrhoea that was absent for ≥1 day and then returned a median of two (range 1-8) times before complete resolution. Three (5.5%) case-patients reportedly continued recreational water activities while they had diarrhoea, and 10 (18·2%) case-patients reportedly participated in recreational water activities within the 2 weeks after their diarrhoea completely resolved.

Of seven adult case-patients, six (85.7%) reported not being able to perform daily activities for a median of 7.5 (range 2–17) days; two (50%) of four employed adult case-patients reported missing work due to illness. Of 42 paediatric case-patients, 32 (76.2%) were reportedly

Table 1. Clinical symptoms reported for cryptosporidiosis case-patients recruited for case-control study, Auglaize County, Ohio, 2004

Symptom	Case-patients ($N = 55$)		
	n*	%	
Diarrhoea	55/55	100	
Tiredness/fatigue	52/54	96.3	
Loss of appetite	47/53	88.7	
Abdominal cramps	48/55	87.3	
Nausea	37/52	71.2	
Headache	35/50	70.0	
Vomiting	35/54	64.8	
Body/muscle aches	30/49	61.2	
Bloating/gas	29/49	59.2	
Fever	32/54	59.3	
Weight loss	24/46	52.2	
Blood in stool	0/53	0	

^{*} Denominator varies due to missing, unknown, or refused responses.

not able to perform daily activities for a median of 3.0 (range 1–18) days; parents or guardians of 29 (60.4%) of 48 paediatric case-patients reported not being able to perform daily activities due to their child's illness for a median of 3.0 (range 1–14) days. Of 32 employed parents or guardians of paediatric case-patients, 10 (31.3%)

Table 2. Case-control study: bivariate conditional analysis of exposures, Auglaize County, Ohio, 2004

Factor	Case-patients $(N = 55)$		Controls $(N = 105)$		
	n‡	%	$n\ddagger$	%	mOR (95% CI)
Demographics					
Gender (% male)	26/55	47.3	45/98	45.9	1.1 (0.5-2.3)
Age (years)					
0-<2	2/55	3.6	5/103	4.9	
2-<6	14/55	25.5	25/103	24.3	
6-<18	32/55	58.2	59/103	57.3	
≥18	7/55	12.7	14/103	13.6	
Human contact†					
Adult household contact ill with diarrhoea	13/55	23.6	4/82	4.9	5.4 (1.6–23.0)**
Child household contact ill with diarrhoea	26/55	47.3	3/94	3.2	41.2 (6.7->999)***
Any household contact ill with diarrhoea	31/55	56.4	6/83	7.2	10.5 (3.6–41.9)**
Dietary exposures†					
Cider or juice	25/55	45.5	72/104	69.2	0.3 (0.2–0.7)**
Recreational water exposures†					
Swim or enter recreational water	54/55	98.2	62/104	59.6	43⋅3 (9⋅5–∞)***
Swim, wade in, or enter a swimming pool	53/55	96.4	53/103	51.5	48.4 (10.7–∞)***
Pool A	49/55	89.1	6/103	5.8	121.7 (27.4–∞)***
Private/residential pool	3/55	5.5	23/103	22.3	0.2 (0.0-0.8)**
Animal contact†					
Cat or dog	35/53	66.0	88/103	85.4	0.3 (0.1-0.8)**
Farm animal contact	12/50	24.0	43/98	43.9	0.4 (0.2–0.9)**
Animal waste/manure	11/49	22.4	41/99	41.4	0.4 (0.1–0.9)**

mOR, Matched odds ratio; CI, confidence interval.

reported missing work due to their child's illness for a median of 2.5 (range 1-14) days.

Multivariable analysis

In bivariate analysis (Table 2), cryptosporidiosis was significantly associated with swimming in pool A [matched odds ratio (mOR) 121.7, 95% CI $27.4-\infty$, P < 0.0001]. Swimming in any other individual recreational water venue was not a significant risk factor for cryptosporidiosis nor was childcare. Having an adult (mOR 5.4, 95% CI 1.6–23.0, P = 0.004) and a child (mOR 41·2, 95% CI 6·7–> 999, P < 0.0001) household contact ill with diarrhoea in the 2 weeks prior to illness onset were also each significant risk factors for cryptosporidiosis. When case-patients who reported any ill household contacts in the 2 weeks prior to illness onset were excluded from the analysis, swimming in pool A remained a significant risk factor (mOR 36·7, 95% CI 7·9 $-\infty$, P < 0.0001) as did swimming in any swimming pool (mOR 15.3,

95% CI $3 \cdot 1 - \infty$, P = 0.0012) or recreational water (mOR 141.0, 95% CI $2 \cdot 2 - \infty$, P = 0.0073).

Swimming in a private/residential pool; consumption of any cider or juice; contact with a cat or dog, farm animal, or animal waste or manure were each significantly protective. More than 95% (22/23) of controls who swam in a private/residential pool did not swim in pool A. When the aforementioned protective factors were individually modelled as predictors along with swimming in pool A, they remained protective but were no longer statistically significant.

Laboratory investigation

Twenty-eight stool specimens were positive for *Cryptosporidium* by DFA at CDC. The DNA of 18 $(64\cdot3\%)$ isolates amplified. Molecular typing of 18 isolates identified *C. hominis*, subtype IdA15G1. Stool specimens from 25 $(39\cdot7\%)$ out of 63 initial case-patients were tested for the presence of bacterial and viral pathogens, of which 24 $(96\cdot0\%)$ were negative; one $(4\cdot0\%)$ had

[†] In the 2 weeks before illness onset

[‡] Denominator varies due to missing, unknown, or refused responses.

^{**} P < 0.05, *** P < 0.001.

no growth, including of normal inte

no growth, including of normal intestinal flora, and thus was considered an invalid clinical specimen.

Environmental health investigation

The main pool had a spread finger layout; a diving well, water slide, children's slide, rain drop water feature, and zero-depth entry; and a capacity of 994 477 litres. Disinfection of the main pool was achieved through treatment with sodium hypochlorite (NaOCl) adjusted by automatic controllers; two sand filters† provided a required flow rate of 2071 litres per minute. The wading pool had a capacity of 11 708 litres. It had a separate circulation system and sand filter; it was also disinfected with NaOCl. The samples taken from the deep and shallow ends of the main pool were positive for Cryptosporidium spp. by real-time PCR, while the wading pool samples were negative. However, conventional PCR, which requires a longer segment of DNA to be amplified, was negative and therefore, molecular typing was not possible. A review of pool A attendance records showed a peak in pool attendance on 2 August (Fig. 1), or 6 days prior to the illness onset peak in the epicurve as would be expected with cryptosporidiosis. Further breakdown of the types of patrons in attendance revealed a peak in attendance by pre-school-aged children on 2 August. During the pool inspection conducted on 18 August, Auglaize County environmental health specialists noted no violations.

Of 38 pool employees, 32 (84·2%) were interviewed. All interviewed pool employees were lifeguards. Eighteen (56.3%) were also swim instructors; three (9.4%) also worked as managers. The median age of interviewed employees was 17 (range 15-21) years. Eight (25.8%) of 31 employees reported having diarrhoea since 1 July 2004; two (25.0%) of the eight reported that they continued to go into the water or participated in other recreational water-related activities in any pool while ill with diarrhoea. Four (50.0%) of the eight employees reported that they went into the water or participated in other recreational water-related activities in pool A within the 2 weeks after their diarrhoea completely resolved. Eight (25.0%) of 32 reported observing mothers changing diapers in the area around the wading pool; four (13·3%) of 30 reported observing mothers changing diapers around the main pool. No employees

reported observing mothers cleaning or rinsing their child's bottom in the wading or main pool. Fifteen (48·4%) of 31 employees reported faecal incidents in the main pool and three (10·0%) of 30 reported faecal incidents in the wading pool during July–August. Managers anecdotally reported that the majority of 'babies' were in the main pool on a typical day.

DISCUSSION

This is the first US recreational water-associated cryptosporidiosis outbreak for which a proactive public health response has been documented. The lack of transmission beyond pool A suggests that such a proactive response might have prevented subsequent community-wide transmission to other recreational water venues or settings, despite the large festival. This outbreak followed a 2003 Kansas communitywide recreational water-associated cryptosporidiosis outbreak, which affected >600 persons [18]. Lessons learned from the Kansas outbreak investigation were applied to this Ohio outbreak investigation, namely proactively implementing control measures, i.e. once an outbreak is suspected or an increase in cryptosporidiosis case reporting has been detected but before the case-control study was conducted; the data were analysed; and the outbreak source was epidemiologically ascertained. Unfortunately, this Ohio outbreak was followed by community-wide recreational water-associated cryptosporidiosis outbreaks in Utah in 2007 and Texas in 2008, which each affected thousands, as well as Victoria, Australia in 2013 and Florida in 2014 [7, 19–21]. The ongoing occurrence of community-wide recreational waterassociated outbreaks of cryptosporidiosis in >10 years since this Ohio outbreak argues for the reporting of the proactive implementation of control measures and subsequent lack of community-wide transmission and provides an example for response to future recreational water-associated cryptosporidiosis outbreaks.

Others [22, 23] who have investigated recreational water-associated outbreaks of cryptosporidiosis have also called for proactive response to these outbreaks. These studies advocate for rapid field epidemiology to direct implementation of control measures in response to recreational water-associated outbreaks of cryptosporidiosis. This recommendation fits the traditional model of field epidemiology [24]. However, delaying implementation of control measures, such as hyperchlorination of treated recreational water venues, can cause a focal cryptosporidiosis outbreak to evolve into a community-wide outbreak associated

[†] Flocculants are not typically a part of pool operations in the United States. Sand filters alone do not efficiently remove *Cryptosporidium* from the water (i.e. removals are <0·19 log or 36%) [17]. Flocculants are typically part of pool operations in European countries.

with multiple treated recreational water venues or other settings. This is because *Cryptosporidium* is extremely chlorine tolerant, and the free chlorine levels typically mandated or recommended for treated recreational water venues and other settings do not effectively inactivate the parasite. The pathway for a community-wide outbreak's characteristic transition from waterborne to person-to-person *Cryptosporidium* transmission is likely young children, who use treated recreational water and then transmit the parasite to caregivers in their household and childcare facilities [7, 25, 26].

Since the aforementioned Kansas outbreak and this Ohio outbreak, the U.S. CDC has been recommending proactively implementing control measures once an increase in cryptosporidiosis reporting is detected or an outbreak is suspected, formally so since 2008 [7, 27]. Accordingly, in the scenario reported by Coetzee et al. [23], where an increase in cryptosporidiosis reporting was detected, CDC would have recommended hyperchlorinating any treated recreational water venue used by ≥ 2 case-patients if they are not all from the same household. In the scenario reported by McCann et al. [22] where a cluster of diarrhoeal illness was detected, CDC would have recommended hyperchlorinating the swim club pool as soon as Cryptosporidium was identified as the outbreak aetiology.

In Ohio, pool A and two nearby pools were closed to swimmers, hyperchlorinated, and reopened all within 5 days of ACHD receiving its first report of an outbreak-associated case (Fig. 1). Closing pool A and nearby pools, which might have been used by the same swimmers, and hyperchlorinating the water, after samples from pool A were taken for testing, did not interfere with the concurrent outbreak investigation and prevented further transmission at these pools. Reopening the pools immediately posthyperchlorination potentially prevented those, who typically swim at these pools, from going to other recreational water venues and contaminating them with *Cryptosporidium* oocysts.

Because these extremely chlorine-tolerant oocysts are not efficiently removed by sand filters typically used at US-treated recreational water venues (or in European venues with improper coagulant dosing) and because remediation options for untreated recreational water venues (e.g. lakes) are limited, swimmers, i.e. the *Cryptosporidium* oocyst reservoir, must be engaged in efforts to prevent contamination of recreational water in the first place and recontamination

post-hyperchlorination [28]. While 98.2% of interviewed case-patients reported swimming in the 2 weeks before they became ill, 5.5% reported swimming while ill with diarrhoea. A previously reported study suggests that a healthy swimming education campaign, such as the one conducted in the days following the detection of this outbreak, might help prevent cryptosporidiosis outbreaks associated with recreational water [29]. Before the 2008 summer swim season and during the 2007 statewide recreational water-associated cryptosporidiosis outbreak in Utah, local and state public health officials disseminated healthy swimming messages via the web, television advertisements, public service radio announcements, and posters at pools [7]. Healthy swimming messages were also targeted at schools, competitive water sports teams, and childcare facilities. A 2009 U.S. national survey of an individual from each of >4500 households found that Utah residents were significantly more likely to know that they should not swim while ill with diarrhoea than residents of other states (100% vs. 78·4%). No recreational waterassociated outbreaks were detected in Utah during 2008-2011.

By contrast, 25% of pool employees reported entering the water or participating in other recreational water-related activities in any pool while ill with diarrhoea. Ill pool employees might have found it difficult to abstain from entering the water while performing work duties without admitting to being ill and then potentially losing wages or even work. An investigation of a waterpark-associated cryptosporidiosis outbreak found that illness onset peaked in employees before patrons and Cryptosporidium contamination of the water by employees could have led to the outbreak [30]. Thus, U.S. CDC recommends that the aquatics sector institute policies that allow employees to perform alternate duties that do not require entering the water while they are ill with diarrhoea. Such policies would be analogous to those in the food service sector that deter employees from handling food while ill with diarrhoea.

This investigation has at least three limitations. First, the methods of this outbreak investigation cannot prove that proactively implementing control measures prevented a focal outbreak from evolving into a community-wide outbreak. However, the causation criteria of temporality, plausibility, and specificity are met and support this hypothesis. Second, recall bias in study participants might have been introduced into the study given that the questionnaire was administered a few weeks after the outbreak started. Case-patients

might have been able to more accurately report their exposures during the 2 weeks before their illness than controls, who did not have an illness to enhance their memory of the time period of interest. Finally, 31 (56.4%) enrolled case-patients might not have been the first person in their respective household to become ill. This finding could be the result of questionnaire formatting or wording, leading to a misunderstanding on the part of interviewers or respondents. Regardless, pool A remained significantly associated with illness, when data on these 31 case-patients and their corresponding controls were removed from analysis, despite the consequent decreased statistical power. Alternatively, while these 31 case-patients might not have been the first in the household to become ill, they might have had the same exposure as the household member who truly became ill first [e.g. 49 (89·1%) of 55 case-patients reported swimming in pool A in the 2 weeks prior to illness onset].

The findings of this investigation suggest that a proactive public health response might prevent a focal cryptosporidiosis outbreak from expanding into a community-wide outbreak. However, such a proactive public health response is likely to be too late to prevent household transmission. Having ill household contacts has been identified as a risk factor for cryptosporidiosis in multiple studies [25, 26, 31]. While the proactive response to this outbreak provides a model for prevention of community-wide transmission of Cryptosporidium, transmission still potentially occurred within households and highlights the need for education regarding basic hygiene, such as handwashing after toileting, assisting others with toileting, or changing a diaper and before preparing food [32-34]. Given the prolonged nature of diarrhoea caused by Cryptosporidium, time lost from work and daily activities, and healthcare costs associated with emergency department visits and hospitalization, disseminating hygiene messages, which can also prevent the transmission of other enteric pathogens, before outbreaks occur and proactively implementing control measures when increased Cryptosporidium transmission is detected could be a worthwhile public health investment [35].

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DECLARATION OF INTEREST

None.

REFERENCES

- Hlavsa MC, et al. Recreational water-associated disease outbreaks – United States, 2009–2010. Morbidity and Mortality Weekly Report 2014; 63: 6–10.
- Joce RE, et al. An outbreak of cryptosporidiosis associated with a swimming pool. Epidemiology and Infection 1991; 107: 497–508.
- Centers for Disease Control and Prevention. Swimmingassociated cryptosporidiosis – Los Angeles County. Morbidity and Mortality Weekly Report 1990; 39: 343–345.
- Sorvillo FJ, et al. Epidemiologic notes and reports swimming-associated cryptosporidiosis – Los Angeles County. American Journal of Public Health 1992; 82: 742–744.
- Jokipii L, Jokipii AM. Timing of symptoms and oocyst excretion in human cryptosporidiosis. New England Journal of Medicine 1986; 315: 1643–1647.
- Shields JM, et al. Inactivation of Cryptosporidium parvum under chlorinated recreational water conditions. Journal of Water and Health 2008; 6: 513–520.
- Centers for Disease Control and Prevention.
 Communitywide cryptosporidiosis outbreak Utah,
 2007. Morbidity and Mortality Weekly Report 2008;
 57: 989–993.
- 8. Centers for Disease Control and Prevention. Protracted outbreaks of cryptosporidiosis associated with swimming pool use Ohio and Nebraska, 2000. *Morbidity and Mortality Weekly Report* 2001; **50**: 406–410.
- 9. **Chalmers RM, et al.** Long-term *Cryptosporidium* typing reveals the aetiology and species-specific epidemiology of human cryptosporidiosis in England and Wales, 2000 to 2003. *Euro Surveillance* 2009; **14**.
- Xiao L. Molecular epidemiology of cryptosporidiosis: an update. Experimental Parasitology 2010; 124: 80–89.
- Blackburn BG, et al. Cryptosporidiosis associated with ozonated apple cider. Emerging Infectious Diseases 2006; 12: 684–686.
- Valderrama AL, et al. Multiple risk factors associated with a large statewide increase in cryptosporidiosis. Epidemiology and Infection 2009; 137: 1781–1788.
- O'Reilly CE, et al. A waterborne outbreak of gastroenteritis with multiple etiologies among resort island visitors and residents: Ohio, 2004. Clinical Infectious Diseases 2007; 44: 506–512.
- 14. Xiao L, et al. Subtype analysis of Cryptosporidium specimens from sporadic cases in Colorado, Idaho,

- New Mexico, and Iowa in 2007: widespread occurrence of one *Cryptosporidium hominis* subtype and case history of an infection with the *Cryptosporidium* horse genotype. *Journal of Clinical Microbiology* 2009; **47**: 3017–3020.
- 15. **United States Environmental Protection Agency.** Method 1622: *Cryptosporidium* in water by filtration/ IMS/FA. *EPA-821-R-01-026*. 2001.
- Jothikumar N, et al. Detection and differentiation of Cryptosporidium hominis and Cryptosporidium parvum by dual TaqMan assays. Journal of Medical Microbiology 2008; 57: 1099–1105.
- 17. **Amburgey JE**, *et al.* Removal of *Cryptosporidium* and polystyrene microspheres from swimming pool water with sand, cartridge, and precoat filters. *Journal of Water and Health* 2012; **10**: 31–42.
- Dziuban EJ, et al. Surveillance for waterborne disease and outbreaks associated with recreational water – United States, 2003–2004. Morbidity and Mortality Weekly Report. Surveillance Summaries 2006; 55: 1–30.
- Centers for Disease Control and Prevention. Notifiable diseases and mortality tables. *Morbidity and Mortality* Weekly Report 2014; 63: ND-607–ND-620.
- Chief Health Officer, Victoria, Australia. Update: outbreak of cryptosporidiosis extends to regional Victoria – 19 March 2013 (http://www.health.vic.gov. au/chiefhealthofficer/alerts/alert-2013-03-cryptosporidiosisregional-update.htm). Accessed 28 August 2014.
- 21. **Cantey PT**, *et al.* Outbreak of cryptosporidiosis associated with a man-made chlorinated lake Tarrant County, Texas, 2008. *Journal of Environmental Health* 2012; **75**: 14–19.
- McCann R, et al. An outbreak of cryptosporidiosis at a swimming club – can rapid field epidemiology limit the spread of illness? Epidemiology and Infection 2014; 142: 51–55.
- 23. Coetzee N, et al. A swimming pool-associated outbreak of cryptosporidiosis in Staffordshire, England, October to December 2007. Eurosurveillance 2008; 13: 19028.
- 24. **Gregg MB.** The field investigation. In: Gregg MB, ed. *Field Epidemiology*, 2nd edn. New York: Oxford University Press, 2002, pp. 62–77.

- Roy SL, et al. Risk factors for sporadic cryptosporidiosis among immunocompetent persons in the United States from 1999 to 2001. Journal of Clinical Microbiology 2004; 42: 2944–2951.
- Robertson B, et al. Case-control studies of sporadic cryptosporidiosis in Melbourne and Adelaide, Australia. Epidemiology and Infection 2002; 128: 419–431.
- Centers for Disease Control and Prevention. Cryptosporidiosis outbreak response and evaluation (http://www.cdc.gov/parasites/crypto/resources/core_guidelines.pdf).
 Accessed 11 November 2014.
- Amburgey JE, Anderson JB. Disposable swim diaper retention of *Cryptosporidium*-sized particles on human subjects in a recreational water setting. *Journal of Water and Health* 2011; 9: 653–658.
- 29. **Centers for Disease Control and Prevention.** Promotion of healthy swimming after a statewide outbreak of cryptosporidiosis associated with recreational water venues Utah, 2008–2009. *Morbidity and Mortality Weekly Report* 2012; **61**: 348–352.
- 30. **Wheeler C**, *et al*. Outbreak of cryptosporidiosis at a California waterpark: employee and patron roles and the long road towards prevention. *Epidemiology and Infection* 2007; **135**: 302–310.
- Hunter PR, et al. Sporadic cryptosporidiosis casecontrol study with genotyping. Emerging Infectious Diseases 2004; 10: 1241–1249.
- 32. **Ejemot RI**, *et al*. Hand washing for preventing diarrhoea. *Cochrane Database of Systematic Reviews* 2008: Issue no. 1. Art. no. CD004265.
- 33. **Huang DB, Zhou J.** Effect of intensive handwashing in the prevention of diarrhoeal illness among patients with AIDS: a randomized controlled study. *Journal of Medical Microbiology* 2007; **56**: 659–663.
- 34. **Aiello AE**, *et al*. Effect of hand hygiene on infectious disease risk in the community setting: a meta-analysis. *American Journal of Public Health* 2008; **98**: 1372–1381.
- 35. **Collier SA**, *et al*. Direct healthcare costs of selected diseases primarily or partially transmitted by water. *Epidemiology and Infection* 2012; **140**: 2003–2013.