

## SHORT REPORT

# Cryptosporidiosis from a community swimming pool: outbreak investigation and follow-up study

T. K. BOEHMER<sup>1,2\*</sup>, N. B. ALDEN<sup>2</sup>, T. S. GHOSH<sup>2</sup> AND R. L. VOGT<sup>2</sup>

<sup>1</sup> Epidemic Intelligence Service, Centers for Disease Control and Prevention, Atlanta, GA, USA

<sup>2</sup> Tri-County Health Department, Greenwood Village, CO, USA

(Accepted 8 April 2009; first published online 11 May 2009)

### SUMMARY

Tri-County Health Department investigated an outbreak of cryptosporidiosis linked to a community swimming pool. A cohort study was conducted in 37 persons who were invited to the pool party; 12 (57%) of 21 attendees had primary cryptosporidiosis infection. Risk factors for illness included swimming, getting water in mouth, and swallowing water. The pool met chlorination guidelines and used UV light irradiation, a supplemental disinfection technology that inactivates *Cryptosporidium*. A follow-up survey of the cohort was completed 7–8 weeks after the pool party; four (25%) of 16 non-attendees had secondary cryptosporidiosis infection. The median duration of illness, including patients with recurring symptoms, was 26 days. Clinical response rate to nitazoxanide, a therapeutic agent, was 67%. This study is unique because it describes a cryptosporidiosis outbreak from a well-maintained community swimming pool using supplemental disinfection. It also reports information on disease burden and treatment response.

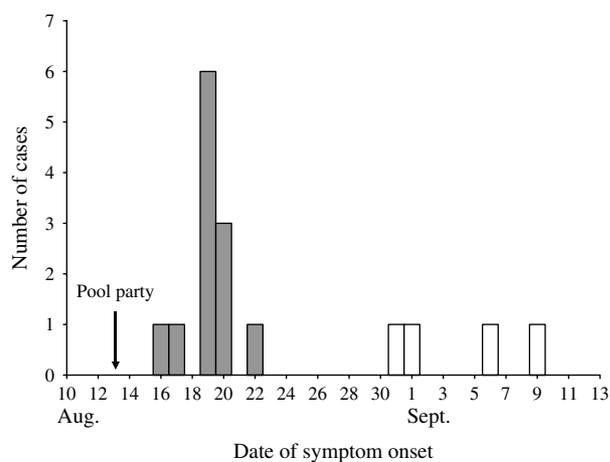
**Key words:** Community outbreaks, *Cryptosporidium*, disinfection, epidemiology, water-borne infections.

Cryptosporidiosis, a parasitic disease that causes watery diarrhoea, affects 1–2 persons/100 000 population annually in the USA [1]. Faecal–oral transmission of *Cryptosporidium* occurs through ingestion of contaminated drinking or recreational water, consumption of contaminated food, or contact with infected persons or animals [2, 3]. The typical incubation period for cryptosporidiosis is 7 days (range 1–12 days). *Cryptosporidium* oocysts are highly resistant to chlorine disinfection and can survive for days in water with recommended residual chlorine levels (1–3 ppm) [4]. Because of its chlorine resistance,

environmental stability, and low infectious dose, *Cryptosporidium* is the leading cause of gastroenteritis outbreaks associated with chemically treated swimming venues in the USA [2, 5]. Nitazoxanide, the only licensed treatment for *Cryptosporidium*-induced diarrhoea, was Food and Drug Administration-approved for use in children aged 1–11 years in 2002 and in persons aged  $\geq 12$  years in 2004 [6, 7].

In August 2006, a parent notified Tri-County Health Department (TCHD), Colorado, USA, of children experiencing gastrointestinal symptoms; the only common exposure was a birthday party held at an indoor community swimming pool. We investigated the outbreak to identify the illness aetiology and implement environmental control measures. After *Cryptosporidium* was identified, TCHD received

\* Author for correspondence: Dr T. K. Boehmer, Centers for Disease Control and Prevention, 4770 Buford Hwy NE, MS F-58, Atlanta, GA 30341, USA.  
(Email: tboehmer@cdc.gov)



**Fig. 1.** Date of symptom onset for primary (■) and secondary (□) cryptosporidiosis cases in persons invited to the pool party, August–September 2006.

additional notifications about household transmission and persistent symptoms after treatment. In response, we conducted a descriptive follow-up survey to assess disease burden and treatment response. This report describes the outbreak investigation and follow-up survey.

A cohort study was conducted among 37 persons from eight households who were invited to the pool party. Initial telephone inquiries determined that those not attending had experienced no gastrointestinal illness. All 21 attendees completed an internet-based questionnaire assessing food and pool exposures and illness status; 95% completed the survey within 2–3 weeks of the pool party. A primary cryptosporidiosis case was defined as diarrhoea (three or more loose stools per day), vomiting, or abdominal pain/cramping that occurred 1–12 days after the pool party in an attendee. Twelve (57%) primary cases were identified in 21 attendees with a median incubation period of 6 days (range, 3–9 days) (Fig. 1). Eighty-three percent of primary cases occurred in children (median age 7 years, range 5–38 years). According to the initial questionnaire, the median illness duration was 6.5 days, but 7/12 patients reported still being sick. Self-reported symptoms included watery diarrhoea (100%), abdominal cramps (92%), nausea (83%), fatigue (83%), loss of appetite (83%), fever (50%), vomiting (33%), and body aches (33%). One patient aged 7 years was immunocompromised.

To assess risk factors for cryptosporidiosis, we calculated unadjusted relative risks (RR) by using SAS<sup>®</sup> version 9.1 (SAS Institute Inc., USA) and *P* values and 95% confidence intervals (CI) by using exact

non-parametric procedures available in StatXact<sup>®</sup> version 7 (Cytel Inc., USA). The risk for illness was greater in attendees who had entered the pool and in swimmers who got water in their mouth or swallowed water (Table 1). Illness was not associated with any food items served at the party.

A follow-up survey of the cohort was conducted 7–8 weeks after the pool party. Thirty-three (89%) of 37 persons from 7/8 households completed a telephone-based questionnaire assessing secondary transmission, symptom duration, and treatment. A secondary cryptosporidiosis case was defined as diarrhoea, vomiting, or abdominal pain/cramping that occurred >12 days after the pool party in a non-attendee. Four (25%) secondary cases (three adults, one child) from two households were identified in 16 non-attendees with onset 18–27 days after the pool party (Fig. 1).

Of the 15 (11 primary, four secondary) cryptosporidiosis patients who completed a follow-up survey, 80% reported having recurring symptoms and the median illness duration including relapses was 26 days (range 10–42 days). Thirteen patients (four adults, nine children) who sought medical care were treated with age-appropriate dosages of nitazoxanide [7]; none were hospitalized. Of nine immunocompetent patients (four adults, five children) who were symptomatic at the start of treatment, the median time from treatment initiation to diarrhoea resolution was 5.0 days (range 3–18 days). Six of nine immunocompetent patients reported diarrhoea resolution by day 7 of treatment for a 67% clinical response rate; two of the remaining three patients received a second round of nitazoxanide. The immunocompromised child had diarrhoea resolution on day 14 of treatment.

Bulk stool samples from seven primary and two secondary cases were tested at the Colorado Department of Public Health and Environment (CDPHE) or a commercial laboratory. Four of seven samples from primary cases were sent to the Centers for Disease Control and Prevention (CDC) for species analysis by polymerase chain reaction and genotyping based on *gp60* gene sequencing [8]. *Cryptosporidium* was detected in all nine samples. Four samples tested at CDC contained *Cryptosporidium hominis* genotype IbA10G2.

An environmental inspection revealed that the pool had consistently met appropriate chlorination standards and had used UV light irradiation disinfection procedures and high-rate sand filtration. Pool logs indicated non-diarrhoeal faecal incidents

Table 1. Risk for primary cryptosporidiosis infection in pool party attendees, August 2006

	Exposed		Unexposed		RR	95% CI	P value
	Ill/Total	% Ill	Ill/Total	% Ill			
Attendees (n = 21)							
Swam or waded in pool	12/17	71	0/4	0	Undefined	1.3–∞	0.01
Ate ice cream	7/12	58	5/9	56	1.1	0.5–2.4	0.97
Drank bottled water	4/8	50	8/13	62	0.8	0.2–1.8	0.69
Ate cake	7/15	47	5/6	83	0.6	0.3–1.3	0.15
Swimmers (n = 17)							
Got water in mouth*	8/8	100	0/4	0	Undefined	1.9–∞	<0.01
Swallowed water*	5/5	100	2/7	29	3.5	1.2–18.7	0.02

CI, Confidence interval; RR, relative risk.

\* Total number of exposed and unexposed persons does not add up to 17 because of missing data.

8 days before and 5 days after the pool party. A 1-litre backwash sample of pool water and sand filter was collected 17 days after the pool party (before hyperchlorination) and tested at CDC. No parasites were identified in the sample.

This report describes a cryptosporidiosis outbreak epidemiologically linked to contaminated pool water at a community swimming pool. Identification of the same genotype of *C. hominis* implies a single, human source of faecal contamination. We surmise that a faecal incident occurred but was not reported to the pool management. Undetected faecal incidents (including diarrhoeal accidents and swim diaper leakage) continue to be problematic for maintenance of a healthy swimming environment; thus, patrons should be educated to refrain from swimming while ill with diarrhoea and to report all faecal incidents to pool staff.

This study provides two unique contributions to the literature. First, this cryptosporidiosis outbreak occurred despite a well-maintained, state-of-the-art pool operation system that included UV light irradiation, a supplemental disinfection technology that inactivates *Cryptosporidium* [9, 10]. We are not aware of other published studies describing cryptosporidiosis outbreaks at recreational water facilities that used supplemental disinfection. UV disinfection occurs in line with the pool filtration system, so the speed by which *Cryptosporidium* oocysts are inactivated is dependent upon the pool water recirculation rates. Thus, UV light technology reduces the duration of *Cryptosporidium* transmission from several days to a few hours compared to chlorination alone. Since supplemental disinfection does not eliminate *Cryptosporidium* transmission, pool operators

should adhere to newly revised CDC disinfection recommendations immediately after a diarrhoeal faecal incident (<http://www.cdc.gov/healthyswimming/>).

Second, we were able to conduct a follow-up study that provided detailed information on disease burden and treatment response, neither of which are commonly reported in outbreak investigations. During this outbreak, a high disease burden was observed despite the availability and use of a therapeutic agent. The 6.5-day symptom duration reported during the initial outbreak investigation is in agreement with the 4- to 9-day duration reported during previous outbreaks [11, 12]; however, the follow-up survey revealed that the majority of patients experienced recurring symptoms lasting more than 3.5 weeks. The 25% secondary attack rate observed in this study is considerably higher than the 5–10% reported previously [13, 14], but was derived from a small sample size. The elevated occurrence of secondary transmission in this outbreak is probably a reflection of *Cryptosporidium*'s high infectivity and the parents' reported use of chlorine bleach and other chlorine-based cleaning products, which are ineffective against *Cryptosporidium*. To reduce household transmission, health departments should inform the public that cleaning with hydrogen peroxide [15] or boiling water is recommended to inactivate *Cryptosporidium*.

A 67% clinical response rate for nitazoxanide was observed during our study. Two clinical trials in immunocompetent children and adults in Egypt reported a 80–96% clinical response rate [16, 17]. Additionally, the time from treatment initiation to diarrhoea resolution was 2 days longer in our study compared with published data [16]. Although our cohort has a small sample size and our methodology

is not directly comparable to the clinical trials mentioned above, we believe that reports from the field on practical applications of drugs in outbreak settings can be useful and that the efficacy of nitazoxanide in immunocompetent cryptosporidiosis patients in the USA should be examined in future outbreaks and in clinical settings.

This study is subject to at least two limitations. First, additional case ascertainment might have better determined the extent of the outbreak beyond the cohort of party invitees. Second, the follow-up study was limited by sample size and lack of a comparison group. Nevertheless, results from our study contribute to the literature by documenting disease burden and treatment response during a cryptosporidiosis outbreak linked to a community swimming pool that used supplemental UV disinfection in addition to standard procedures.

#### ACKNOWLEDGEMENTS

The authors thank the following persons for their contributions and assistance during this investigation: Michael Beach, PhD, Centers for Disease Control and Prevention; Alicia Cronquist, MPH, Colorado Department of Public Health and Environment; Cynthia Bruso, Dan Collins, MA, Donna Hite-Bynum, Laura DeGolier, MPH, Jill Swope, Jennifer Patnaik, MHS, Gary Sky, Anita Watkins, MPH, Stacy Weinberg, MA, Lloyd Williams, MA, and Bruce Wilson, MPA, Tri-County Health Department; and Chris Helm and Sarah Rodriguez from the swimming pool facility.

N. B. Alden is currently with Boulder County Public Health, Boulder, Colorado.

The findings and conclusions in this report are those of the author(s) and do not necessarily represent the views of the Centers for Disease Control and Prevention.

#### DECLARATION OF INTEREST

None.

#### REFERENCES

1. Yoder JS, Beach MJ. Cryptosporidiosis surveillance – United States, 2003–2005. *Morbidity and Mortality Weekly Report* 2007; **54**: 1–10.

2. Dillingham RA, Lima AA, Guerrant RL. Cryptosporidiosis: epidemiology and impact. *Microbes and Infection* 2002; **4**: 1059–1066.
3. Centers for Disease Control and Prevention. Cryptosporidiosis outbreaks associated with recreational water use – five states, 2006. *Morbidity and Mortality Weekly Report* 2007; **56**: 729–732.
4. Korich DG, et al. Effects of ozone, chlorine dioxide, chlorine, and monochloramine on *Cryptosporidium parvum* oocyst viability. *Applied and Environmental Microbiology* 1990; **56**: 1423–1428.
5. Dziuban EJ, et al. Surveillance for waterborne disease and outbreaks associated with recreational water – United States, 2003–2004. *Morbidity and Mortality Weekly Report* 2006; **55**: 1–30.
6. Fox LM, Saravolatz LD. Nitazoxanide: a new thiazolidine antiparasitic agent. *Clinical Infectious Diseases* 2005; **40**: 1173–1180.
7. Food and Drug Administration. Nitazoxanide prescribing information [from label approved 15 June 2005] (<http://www.fda.gov/cder/foi/label/2005/021498s003lbl.pdf>). Accessed 23 June 2008.
8. Xiao L, et al. *Cryptosporidium* taxonomy: recent advances and implications for public health. *Clinical Microbiology Reviews* 2004; **17**: 72–97.
9. Bukhari Z, et al. Medium-pressure UV for oocyst inactivation. *Journal of the American Water Works Association* 1999; **91**: 86–94.
10. Rochelle PA, et al. The response of *Cryptosporidium parvum* to UV light. *Trends in Parasitology* 2005; **21**: 81–87.
11. MacKenzie WR, et al. A massive outbreak in Milwaukee of *Cryptosporidium* infection transmitted through the public water supply. *New England Journal of Medicine* 1994; **331**: 161–167.
12. 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.
13. Insulander M, et al. An outbreak of cryptosporidiosis associated with exposure to swimming pool water. *Scandinavian Journal of Infectious Diseases* 2005; **37**: 354–360.
14. MacKenzie WR, et al. Massive outbreak of waterborne cryptosporidium infection in Milwaukee, Wisconsin: recurrence of illness and risk of secondary transmission. *Clinical Infectious Diseases* 1995; **21**: 57–62.
15. Weir SC, et al. Efficacy of common laboratory disinfectants on the infectivity of *Cryptosporidium parvum* oocysts in cell culture. *Applied and Environmental Microbiology* 2002; **68**: 2576–2579.
16. Rossignol JFA, Ayoub A, Ayers MS. Treatment of diarrhea caused by *Cryptosporidium parvum*: a prospective randomized, double-blind, placebo-controlled study of nitazoxanide. *Journal of Infectious Diseases* 2001; **184**: 103–106.
17. Rossignol JFA, et al. Effect of nitazoxanide in diarrhea and enteritis caused by *Cryptosporidium* species. *Clinical Gastroenterology and Hepatology* 2006; **4**: 320–324.