

A community outbreak of *Cryptosporidium* infection associated with a swimming pool complex

Russell Stafford,¹ Gerard Neville,^{1,2} Chris Towner,¹ Brad McCall¹

Abstract

A case-control study was conducted to investigate the cause of a sudden increase in cases of cryptosporidiosis notified to the Brisbane Southside Public Health Unit from January to March 1998. Fifty-two eligible cases were identified over a three-week period early in 1998. Thirty-one of these cases and 21 control subjects participated in the study. Swimming in the 2 weeks before onset of illness was identified as a likely risk factor for cryptosporidiosis infection (OR 3.1, CI 0.8-12.6, P=0.06). Analysis of swimming pool attendance identified swimming at Pool Complex A as a significant risk factor for the acquisition of cryptosporidiosis (OR 8.9, CI 1.5-67.4, P=0.004). No other potential risk factors were significantly associated with illness. The detection of cryptosporidium oocysts in three of the four pools at Pool Complex A supported the findings of the case-control study. As a response to this outbreak, Queensland Health has developed a Code of Practice outlining measures for the control and prevention of future outbreaks of swimming pool-associated cryptosporidiosis and/or giardiasis. *Commun Dis Intell* 2000; 24:236-239

Keywords: gastroenteritis, cryptosporidiosis, giardiasis, oocysts, swimming pool, water-borne

Introduction

Cryptosporidium parvum, a protozoan parasite, was identified as a human pathogen in 1976 and has since been identified as the cause of outbreaks of gastroenteritis associated with drinking water and swimming pools.¹⁻⁴ It is transmitted mainly by ingestion of water or food contaminated with oocysts excreted in animal or human faeces. The infective dose of *C. parvum* has been shown to be as low as 30 oocysts and may even be lower.⁵ The onset of clinical symptoms generally occurs within 2 to 12 days (average 7 days) of exposure. The main clinical symptom is diarrhoea, which may be profuse and watery, often accompanied by abdominal pain, fever, malaise and vomiting. The illness usually lasts between 1 and 3 weeks, and while it is generally self-limiting, may be prolonged or even fatal in immunocompromised individuals. Available treatment is limited to providing relief of symptoms.

Routine surveillance undertaken by the Brisbane Southside Public Health Unit (BSPHU) detected a sudden increase in human cryptosporidium notifications, particularly in bayside and eastern suburbs of Brisbane, in January and February 1998. During these months there were 104 reports compared with 37 notifications for January and February in 1997. A further 168 notifications were received for the month of March 1998 (Figure). An outbreak investigation was commenced by BSPHU on Friday 6 March 1998.

Methods

A case-control study was conducted using all cases notified to the BSPHU between 17 February and 9 March 1998. A case was defined as any person residing in the Redland Shire or the southern suburbs of Brisbane City who had an

illness characterised by diarrhoea, vomiting or abdominal pain and had laboratory-confirmed *Cryptosporidium* oocysts detected in their stools during this period. Persons diagnosed before this period were not interviewed because of the potential for poor recall. Secondary household cases were excluded from the case-control study.

Cases were asked to nominate two control subjects who were of similar age (0-4, 5-12, 13-24, 25-39, 40+ years) and area of residence. Only one control per case was included in the study (the first control agreeing to participate and be interviewed). Controls were excluded if they had a history of enteric illness in the preceding 2 weeks. Whether these controls were infected with *Cryptosporidium* and had subclinical illness or were asymptomatic carriers was not ascertained.

Data were collected from cases and controls by telephone interview. A parent or guardian was interviewed if the case was aged 17 years or less. Cases were excluded from the study if three telephone contact attempts were unsuccessful. A standardised questionnaire sought information on basic demographics, symptoms and exposure to potential risk factors during the 2 weeks prior to onset of illness. Within 24 hours of the corresponding case interview, controls were interviewed about exposures during the two-week period immediately before their interview. Potential risk factors included exposure to treated and untreated drinking water, consumption of unpasteurised milk products, contact with swimming pool or surface water (such as lakes, rivers), attendance at childcare facilities, contact with animals, and recent overseas travel.

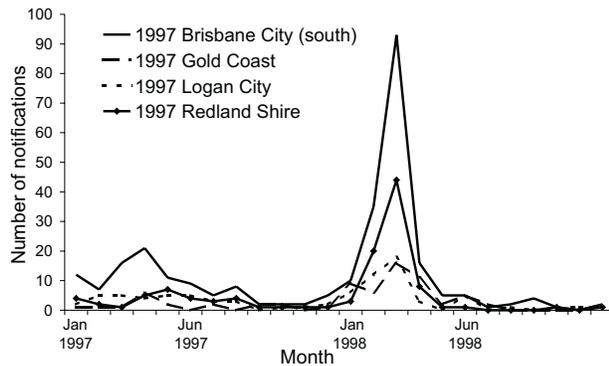
Data were analysed with Epi Info v6.04b (univariate analysis)⁶ and SPSS 7.5 (logistic regression).^{6,7} Unmatched and matched odds ratios and 95% confidence intervals were

1. Brisbane Southside Public Health Unit, Coopers Plains, Queensland

2. Environmental Health Unit, Queensland Health, Brisbane, Queensland

Corresponding author: Russell Stafford, Brisbane Southside Public Health Unit, GPO Box 333, Archerfield, Qld, Australia 4108. Tel: 07 3000 9148. Fax: 07 3000 9121. E-mail: russell_stafford@health.qld.gov.au

Figure. Cryptosporidium notifications, Queensland 1997-1998, by Local Government Area and month



calculated to determine associations between exposure factors and illness and tests of statistical significance applied to the data.

Environmental inspection of Pool Complex A was undertaken to obtain information on chlorination and filtration records and procedures, water quality results and records of faecal accidents.

Microbiological tests were conducted on water samples from the pools at Pool Complex A. The Complex consisted of four indoor swimming pools - a wading pool, 25 and

50 metre pools, and a diving pool. All pools had separate sand filtration systems, except the wading pool, which had a cartridge filter. A total of seven 10L water samples (surface and backwash samples) were collected from the swimming pools. Following a series of concentration steps, immunofluorescent microscopy was used for detection of oocysts in each 10L-water sample. Laboratory tests for oocyst viability and species identification were not available at the time of this investigation.

Results

Fifty-two cases of cryptosporidiosis were notified to the BSPHU during the period of the study. Sixty-two percent were aged 4 years or less (32 cases), with a median age of 2 years (range 4 months to 70 years). The male to female ratio was 1.1:1. Thirty-three were able to be contacted by telephone of whom 31 agreed to interview (median age was 3 years; range 1 to 58 years; male to female ratio was 1.1:1; 18 (58%) were aged 4 years or less). Twenty-one controls agreed to interview.

Symptoms reported by cases were diarrhoea (97%), abdominal cramps (94%), fever (65%), and vomiting (45%). Three (10%) reported blood in their stools. The median duration of symptoms was 11 days (range 3 to 24 days). No case was hospitalised.

The odds ratios for matched and unmatched analyses were similar, so the results of the unmatched analyses are presented in the Table. Age and geographic area were not confounding factors. Swimming during the 2 weeks before onset of illness was identified as a likely risk factor for

Table. Univariate analysis of the major potential risk factors for cryptosporidiosis

Risk factor (exposure during two weeks before illness or interview)	Cases		Controls		Odds ratio ¹	95% CI ²	P value ³
	Number (n=31)	%	Number (n=21)	%			
Male	16	51.6	10	47.6	1	0.3 - 3.4	0.96
Attended childcare centre	12	38.7	7	33.3	1.3	0.3 - 4.7	0.7
Overseas travel	0	0	0	0	-	-	-
Contact with pets	21	67.7	11	52.4	1.9	0.5 - 7.1	0.27
Contact with sick animals	1	3.2	0	0	Undef*	-	1
Contact with zoo animals	1	3.2	0	0	Undef	-	1
Contact with farm animals	0	0	2	9.5	Undef	-	0.16
Raw milk consumed	0	0	0	0	-	-	-
Untreated water consumed	4	12.9	1	4.8	3	0.3 - 76.6	0.64
Mains water consumed	30	96.8	21	100	Undef	-	1
Tank water consumed	1	3.2	0	0	Undef	-	1
Swimming	24	77.4	11	52.4	3.1	0.8 - 12.6	0.06
Swam at Pool Complex A	15	48.4	2	9.5	8.9	1.5 - 67.4	0.004

1. Unmatched odds ratio

2. 95% confidence interval

3. Mantel-Haenszel chi squared test or Fisher Exact test where appropriate,

* Undef = undefined value.

cryptosporidiosis (OR=3.1, 95%CI 0.8-12.6, P=0.06). Analysis of attendance at particular pools identified that swimming at Pool Complex A was a significant risk factor for the acquisition of cryptosporidiosis (OR=8.9, 95%CI 1.5-67.4, P=0.004). This association remained statistically significant when adjusted for other potential confounding variables simultaneously using logistic regression.

Cryptosporidium oocysts were detected in water samples taken from the 25m, 50m, and wading pools. Surface sample counts varied between 13 and 62 oocysts per 10L sample. Backwash counts were 10 to 100 times higher. No *Cryptosporidium* oocysts were detected in diving pool water. *Giardia intestinalis* cysts were detected in surface water samples from the 50m and wading pools and in the backwash of the 50m pool. No records were kept of faecal accidents. Review of 1998 records of chlorination and water quality for each pool met the recommended standards. There was no evidence of filter malfunction preceding the onset of the outbreak.

Over the 3-week notification period from which eligible cases were identified, the notification rates were 7.2 and 20.4/100,000 persons for the Brisbane City South area and Redland Shire, respectively. The same 3-week notification rates for adjacent regions were 0.6/100,000 persons for Gold Coast City and 3.2/100,000 persons for Logan City (Figure).

Discussion

The findings of the case-control study suggest that a community outbreak of cryptosporidiosis occurred in association with swimming at Pool Complex A. The magnitude of this association, the consistency of these findings with other studies,¹⁻³ biological plausibility (oocysts resistant to typical pool chlorine levels and filtration methods),⁸ and the temporal nature of infection following swimming, all support a causal association between swimming at Pool Complex A and developing cryptosporidiosis. In view of the small number of cases and controls it was not possible to determine with certainty which of the four pools in this complex were involved.

This outbreak occurred in the context of a large increase in notifications of cryptosporidiosis in the two local government areas neighbouring Pool Complex A. No other risk factors analysed in this study showed any significant association with cryptosporidiosis. The microbiological results of the water samples were limited by the non-availability of testing for speciation and viability. However, the finding of oocysts in water samples from the complex was considered to lend support to the epidemiological findings. As a result of these findings, Pool Complex A was closed for public use on 11 March 1998 for cleaning and disinfection.

The potential for information bias created by the different time exposure periods for cases and controls must be considered in this study. However, given the strength of the association between Pool Complex A and illness, it is unlikely that this effect could have been explained by information bias alone.

Despite the strong association, 16 of the 31 cases from the case-control study were not linked to that Pool Complex. Nine cases reported swimming at other pools, whilst seven cases did not report swimming in the 2 weeks before their illness. Other pools, person to person transmission, or other

unrecognised exposures are possible sources of infection in these 16 cases.

Children too young to be toilet-trained frequently used the pool complex. The presence of *Giardia* cysts detected in the water samples supports the concept of faecal shedding in the pools. However, because *Giardia* is susceptible to chlorine levels normally found in pools, it is unlikely these were viable cysts.⁸ Because *Cryptosporidium* oocysts are resistant to such chlorine levels⁸ and are small in size (4-6 μ m), their inactivation and removal may not have been effected by the usual filtration and chlorination practices in place at Pool Complex A.

More than half the notified cases were aged less than 5 years. This may reflect a notification bias, as young children are more likely to attend a medical practitioner and to be tested than older children or adults. Furthermore, compared with older children or adults, young children are more likely to swallow pool water when swimming and increase their risk of becoming infected.

Following the identification of the outbreak, all four pools in the complex were treated with chlorine dioxide as a means of inactivating *Cryptosporidium* oocysts.⁸ The pools were subsequently sampled and, following a series of negative results, the complex was reopened 14 days after its closure. No further cases linked to the complex were identified subsequent to its reopening.

Given the public health significance of this outbreak, a Code of Practice for the control of *Cryptosporidium* and *Giardia* in swimming pools, leisure pools, spas and hydrotherapy pools has been developed in Queensland.¹⁰ The Code addresses issues relating to the maintenance of pools, disinfection procedures and preventive measures against future outbreaks of swimming pool-associated cryptosporidiosis and giardiasis. Routine screening of swimming pools for cryptosporidia and giardia is not recommended. Protocols ensuring accurate recording and monitoring of chemical treatment and general pool maintenance - including contingency plans to deal with faecal accidents - are the mainstay of prevention strategies. However, testing is recommended when evidence suggests two or more cases of disease may be associated with a particular pool.

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Editorial statement. Preliminary reports of this work have appeared elsewhere.

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