Epidemiol. Infect. (2001), **126**, 389–396. Printed in the United Kingdom

A statewide outbreak of cryptosporidiosis in New South Wales associated with swimming at public pools

M. C. PUECH[1]*, J. M. McANULTY[2], M. LESJAK[1], N. SHAW[3], L. HERON[1] AND J. M. WATSON[4]

[1] *Department of Public Health and Community Medicine, Westmead Hospital, Westmead NSW 2145, Australia*
[2] *Communicable Diseases Surveillance and Control Unit, Public Health Division, NSW Health Department*
[3] *General Environmental Health Unit, Public Health Division, NSW Health Department*
[4] *NSW Health Department; currently, Consultant Epidemiologist, PHLS Communicable Disease Surveillance Centre, London, UK*

(*Accepted 13 January 2001*)

SUMMARY

From December 1997 to April 1998, 1060 laboratory-confirmed cryptosporidiosis cases were reported in New South Wales, Australia. In a case-control study, compared with 200 controls, the 100 cases were younger (mean age 4·2 versus 7·1 years; $P < 0{\cdot}0001$), more likely to report swimming at a public pool (59% versus 38%; adjusted OR and 95% CI = 2·7; 1·4–5·1) and swimming in a dam, river or lake (OR = 4·8; 1·1–20·3) but less likely to report drinking bottled water (OR = 0·4; 0·2–0·9). In subgroup analyses, in rural areas illness was associated mainly with contact with another person with diarrhoea, and in urban areas illness was associated with swimming in a public pool. *Cryptosporidium* oocysts were more commonly detected in pools to which at least two notified cases had swum ($P = 0{\cdot}04$). Outbreaks of cryptosporidiosis can be prolonged, involve multiple pools and be difficult to control.

INTRODUCTION

Cryptosporidium parvum infections cause watery diarrhoea [1], that while usually benign, can be chronic and debilitating in immunocompromised persons [2, 3]. The infection is acquired by ingesting infectious oocysts, and manifests after an incubation period of 1–12 days [4]. The infective dose in humans is thought to be very small [5], and infected people excrete large numbers of oocysts and excretion may last for weeks after resolution of symptoms [4]. Oocysts can remain infectious up to several months in moist environments and are highly resistant to standard levels of chemical disinfectants such as chlorine [6]. Contaminated drinking water supplies [7–9] are well documented sources of infection, as are individual swimming pools [10–16]. Other reported modes of infection include person-to-person transmission [17–19], animal-to-person transmission [20] and rarely, contaminated food [21].

In New South Wales (NSW), laboratories are required to notify all cases of cryptosporidiosis to Health Areas' Public Health Units.

In January 1998, the Australian Capital Territory (ACT), experienced a cryptosporidiosis outbreak largely associated with swimming at a single pool [22]. By the end of January 1998, a marked increase in notifications of cryptosporidiosis cases was also observed across NSW, of which only 5% were linked to the ACT outbreak. We report the results of the investigations undertaken to assess the extent of the outbreak, and identify and control its causes.

* Author for Correspondence.

METHODS

We defined a case as a NSW resident who was notified with laboratory-confirmed cryptosporidiosis with onset of symptoms between 1 December 1997 and 30 April 1998.

For hypothesis-generating purposes, we interviewed 23 primary cases by telephone, inquiring into a broad range of potential risk factors, including exposure to water (drinking and recreational) and consumption of an extensive range of fresh and manufactured foods and drinks.

CASE-CONTROL STUDY

Cases were eligible for the case-control study if they also: (1) were aged less than 15 years; (2) had a stool specimen collected on or after 1 March 1998; and (3) had their main carer(s) available on the telephone. We restricted the study to these age groups because nearly three-quarters of previous notifications cases were aged less than 15 years old.

Two controls were selected for each case and frequency-matched to the Health Area (HA) of residence of the cases. Potential controls were randomly selected from all potentially active telephone numbers for the relevant HA, using a technique developed for the NSW Health Survey Program [23]. To be eligible, controls had to be a NSW resident, aged less than 15 years and have their main carer(s) available on the telephone. Controls were included regardless of history of recent diarrhoea or gastrointestinal illness. Potential controls were called in HA-specific batches, until the required number of controls, frequency-matched to the corresponding batch of cases from the same HA, was reached. Cases' exposure period was the 2-week period prior to onset of symptoms. For a given HA, controls were asked about exposure to risk factors in a 2-week period that was frequency-matched to the pooled exposure periods of the cases. We did not match cases and controls on age to allow the assessment of age as a potential risk factor.

Cases and controls whose main carer had poor command of English were excluded. Cases' and potential controls' telephone numbers were called up to four times to invite them to participate in the study.

Trained interviewers, using standard questionnaires, conducted telephone interviews of the main carer. For cases and controls, questions covered demographics, recent gastrointestinal illness, and exposure to potential risk factors, including attendance at childcare, swimming, sources of drinking water, contact with animals and with persons with diarrhoea, in the 2-week exposure period. A primary case was defined as a case that reported no contact with a confirmed or presumed (on the basis of a watery diarrhoea) case of cryptosporidiosis in his/her exposure period.

ENVIRONMENTAL STUDY

To determine whether *Cryptosporidium* oocysts were more likely to be detected in the pools that cases reported swimming in, we sampled public pools around NSW. Pools were classified as case or control pools in early March 98. A 'case' pool was defined as a pool where two or more notified cases had swum during their exposure periods and a 'control' pool one where no notified case, as known at the time of selection, reported swimming during his/her exposure period. Pools were not eligible if they had been superchlorinated in the 2 weeks prior to sampling. The sample was a convenience sample, although, when possible, for each 'case' pool tested, a 'control' pool in the same HA was also sampled.

One hundred litre samples of pool water were collected from each of the public pool complexes, with at least one sample per complex. If the complex included a toddlers' pool, this was sampled; otherwise, the sample was collected from the shallow end of the main pool. Filtration, concentration, flow-cytometry [24], staining and direct microscopy were used to isolate and identify *Cryptosporidium* oocysts. The pool complex was considered contaminated if oocysts were detected in at least one of the samples.

ANALYSIS

Descriptive statistics and univariate analyses used *t*-test for continuous variables, χ^2 or Fisher statistics for categorical variables and χ^2 test for linear trend (Mantel–Haenszel procedure).

For the case control study's multivariate analysis, adjusted odds ratios were calculated using logistic regression. Significance testing used Wald χ^2 statistic except for multilevel variables where the difference between '-2 log likelihoods' was used. Plausible interactions were assessed, using a significance level of 0·01, to allow for multiple significance testing; sub-

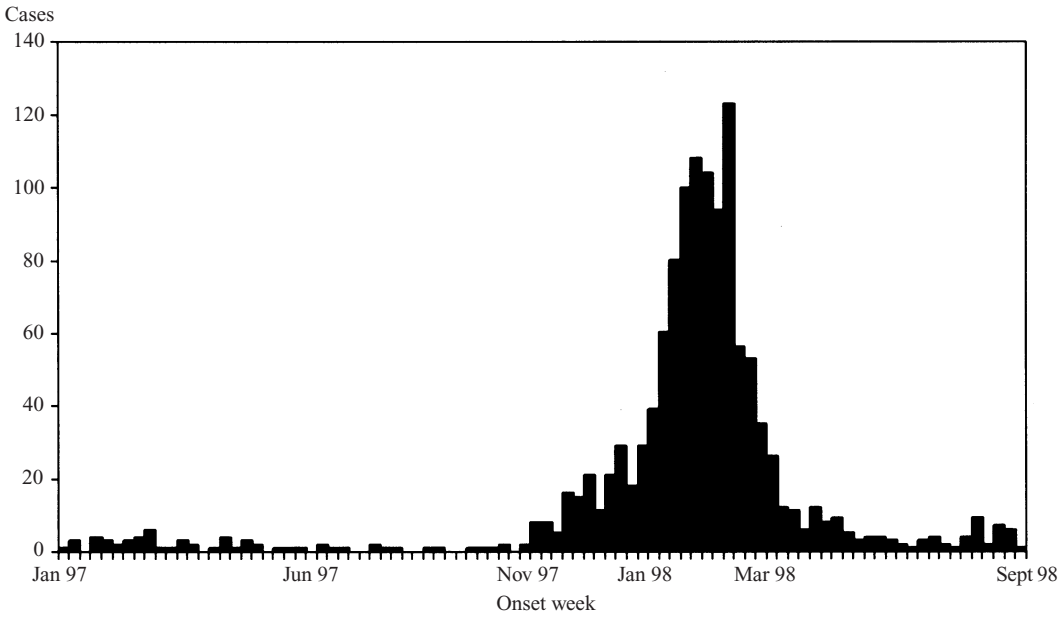

Fig. 1. Laboratory-confirmed cryptosporidiosis cases – NSW notifications by week of onset (January 1997 to September 1998).

group analyses, selected at the study design stage, were also performed.

RESULTS

Between 1 December 1997 and 30 April 1998, 1060 cryptosporidiosis cases in NSW were notified by laboratories. Only 86 cases were notified in the 11 months between 1 January 1997 and 30 November 97 (Fig. 1). Cases were widespread throughout NSW, but rates were higher in rural areas (Fig. 2). The median age of cases was 5·4 years, with 73·3% aged less than 15 years. Males accounted for 49·2% of cases.

Case-control study

The interviews were conducted for all cases and controls from mid-March to early April 1998. The 100 cases that completed the telephone interview were recruited out of 120 consecutive potentially eligible notifications. Nineteen cases were excluded because they could not be contacted by phone and one person was excluded because of an unclear onset date. No one refused to participate. After 2130 phone calls, 237 controls were eligible for the survey. Of these, 37 refused to participate (response rate of 84%).

Symptoms commonly reported by the cases included watery diarrhoea [25] ($n = 97$), anorexia ($n = 93$), abdominal cramps ($n = 70$), fever ($n = 73$) and nausea/vomiting ($n = 65$). Weight loss ($n = 31$), fatigue ($n = 15$) and headaches ($n = 15$) were less common. The average time elapsed between the onset of symptoms and the telephone interview was 23·7 days (range: 6–59 days). Sixty-one cases had recovered at the time of the telephone interview, and among these the mean illness duration was 11·0 days (median = 8·0 days). Twelve cases were admitted to hospital. Twenty-eight (14%) controls reported a gastrointestinal illness in the 4 weeks prior to interview. While abdominal cramps were as frequent as among the cases, other symptoms such as nausea/vomiting, diarrhoea, anorexia and fever, were significantly less frequent. The illness reported was significantly shorter and milder.

Fifty-nine percent of cases and controls resided in the Sydney metropolitan area, 15% in other urban areas, 20% in coastal rural areas and 6% in inland rural areas. In the univariate analysis, there were no significant differences between cases and controls by sex, contact with children in childcare, contact with pets, travelling or drinking town water (Table 1). However, compared with controls, cases were significantly younger, more likely to report contact with another person with diarrhoea, and swimming at a public pool in untreated water in their exposure period, and less likely to report drinking bottled water and being exposed to farm animals. Public pools where cases and controls had swum during their exposure period were distributed widely around NSW, as were the locations of town water supplies and travel destinations reported by cases and controls. No

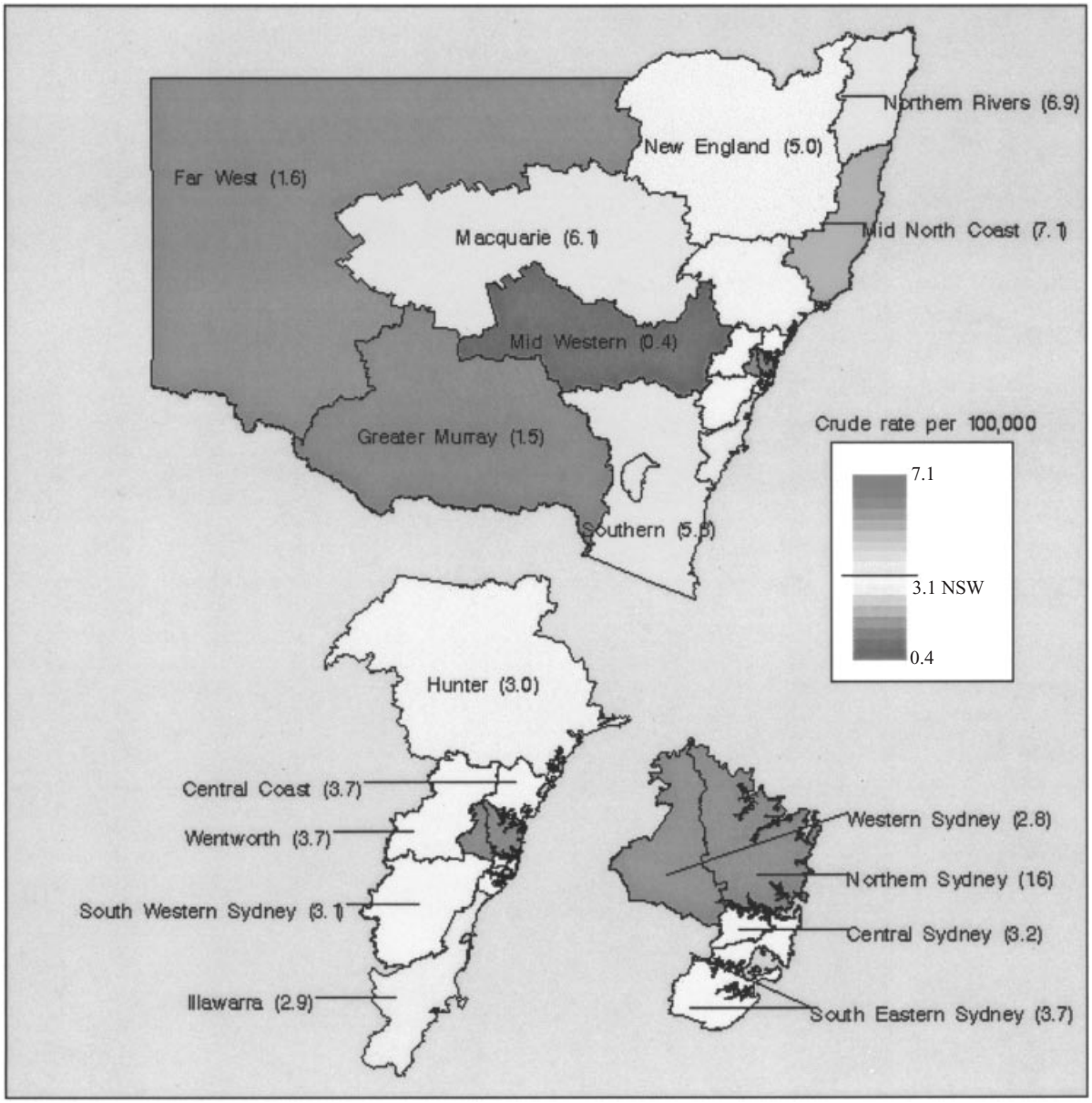

Fig. 2. NSW cryptosporidiosis outbreak (1 December 1997 to 30 April 1998). Monthly crude notification rates of laboratory-confirmed cases by Health Areas.

particular public pool, town water supply or travel destination was significantly more associated with cases than controls.

Attendance at childcare was not included in the multivariate analysis, as it was highly correlated with age (correlation coefficient $> 0{\cdot}5$). There was no significant biologically plausible interaction. In the final model, compared with controls, cases were significantly younger, more likely to report swimming at a public pool and in untreated water and less likely to report drinking bottled water (Table 2).

We found similar results when multivariate analyses were repeated excluding the 28 controls who reported diarrhoeal illness and 32 cases who reported exposure to another person with diarrhoea.

When the analysis was restricted to cases and controls living in urban HAs (74 cases and 148 controls), the significant risk factors in the final multivariate model were younger age [adjusted OR (95% CI) for age groups 5–9 years and 10–14 years: 0·2 (0·1–0·5) and 0·2 (0·1–0·7) respectively with age group 0–4 years as reference] and swimming at a public pool (2·6 (1·2–5·5)) while drinking bottled water had a significant protective effect (0·3 (0·1–0·7)). In the analysis restricted to rural HAs (26 cases and 52 controls), two covariates remained in the final model:

Table 1. *Case-control study – Univariate analysis**

	Cases ($n = 100$) n (%)	Controls ($n = 200$) n (%)	Crude Odds Ratio OR (95% CI)
Demographic factors			
Sex			
Male	57 (57·0)	93 (46·5)	1·5 (0·9–2·5)
Female	43 (43·0)	107 (53·5)	
Age			
0–4 years	62 (62·0)	69 (34·5)	1
5–9 years	26 (26·0)	69 (34·5)	0·4 (0·2–0·7)†
10–14 years	12 (12·0)	62 (31·0)	0·2 (0·1–0·4)†
Reported exposures			
Contact with diarrhoea case	32 (40·0)	38 (24·7)	2·0 (1·1–3·6)†
Childcare (preschool: < 5 yrs)	38 (39·6)	57 (28·9)	1·6 (1·0–2·7)
Swimming			
Anywhere	80 (80·0)	136 (68·7)	1·8 (1·0–3·2)†
At a public pool(s)	59 (59·0)	76 (38·4)	2·3 (1·4–3·8)†
At a private pool(s)	22 (22·0)	60 (30·3)	0·7 (0·4–1·1)
In untreated water‡	9 (10·1)	8 (4·2)	2·6 (1·0–7·0)†
At a beach(es)	28 (28·0)	51 (26·0)	1·1 (0·6–1·9)
At another venue§	39 (41·9)	63 (33·2)	1·5 (0·9–2·4)
Contact with animals			
Pets	75 (75·0)	143 (73·3)	1·1 (0·6–1·9)
Farm animals	4 (4·1)	28 (14·2)	0·3 (0·1–0·8)†
Zoo animals	3 (3·0)	4 (2·0)	1·5 (0·3–6·9)
Drinking water supplies			
Town water	89 (89·9)	184 (92·0)	0·8 (0·3–1·8)
Bottled water	21 (21·4)	64 (33·2)	0·6 (0·3–1·0)†
Above ground tank	8 (8·1)	22 (11·1)	0·7 (0·3–1·7)
Bore and/or well	3 (3·1)	4 (2·0)	1·5 (0·3–7·1)
Surface water‡	4 (4·1)	3 (1·5)	2·8 (0·6–12·7)
Travel (outside LGA¶ of residence)	33 (34·0)	63 (31·8)	1·1 (0·7–1·9)
Drinking water supplies when travelling outside LGA of residence‖			
Town water	23 (82·1)	44 (83·0)	0·9 (0·3–3·1)
Bottled water	2 (7·1)	8 (15·1)	0·4 (0·1–2·2)
Above ground tank	2 (7·1)	3 (5·7)	1·3 (0·2–8·2)
Bore or/and well	1 (3·6)	0 (0)	1·0 (1·0–1·1)
Surface water‡	1 (3·6)	0 (0)	1·0 (1·0–1·1)

* Missing data not in denominator.
† $P <= 0{\cdot}05$.
‡ River, lake, dam, stream.
§ Waterslide, sprinkler.
¶ Local Government Area.
‖ Apart from town water use, numbers are too small to make valid comparsions.

contact with someone suffering diarrhoea (5·5 (1·5–20·6)) and exposure to farm animals (0·1 (0·01–0·9)).

Environmental investigations

Ten case and 13 control pools were sampled. *Cryptosporidium* oocysts were identified more commonly in water from case pools ($n = 6$; 60%) than control pools ($n = 2$; 15%) (Fisher Exact test $P = 0{\cdot}04$).

Control measures

In mid-February 1998, we initiated several control measures, including media statements and advice to swimming pool operators that: (1) people with diarrhoea in the previous week should not swim in public pools; (2) users should shower before entering pools; (3) non-toilet trained toddlers should be restricted to toddler pools and wearing waterproof pants; (4) faecal material should be promptly removed from pools when identified; and (5) pool filtration and

Table 2. *Case-control study: adjusted analysis, final model (100 cases and 200 controls)*

Covariate	Adjusted odds ratio (95% CI)
Age group	
0–4 years (reference)	1
5–9 years	0·3 (0·2–0·7)
10–14 years	0·2 (0·1–0·6)
	-2 Log l = 17·2; 2 D.F.; $P < 0{\cdot}001$
Contact with diarrhoea case	1·7 (0·9–3·4)
	Wald $\chi^2 = 2{\cdot}6$; $P = 0{\cdot}1$
Swimming at a public pool	2·7 (1·4–5·1)
	Wald $\chi^2 = 9{\cdot}2$; $P = 0{\cdot}002$
Swimming in untreated water (i.e. dam, river or lake)	4·8 (1·1–20·3)
	Wald $\chi^2 = 4{\cdot}6$; $P = 0{\cdot}03$
Drinking bottled water	0·4 (0·2–0·9)
	Wald $\chi^2 = 5{\cdot}5$; $P = 0{\cdot}02$

disinfectants levels should be properly maintained, supplemented by fortnightly overnight superchlorination [6, 26–27]. Information and warnings for the public were displayed on notice boards at pool sites. Pools where *Cryptosporidium* oocysts were identified in water samples, were temporarily closed and superchlorinated.

Notifications peaked mid March and then rapidly decreased, as a consequence most probably of cooler weather and the associated decrease in pool attendance as well as control measures (Fig. 1).

Longer-term initiatives included the development by NSW Health of facts-sheets about cryptosporidiosis for the general public [28] and the immunocompromised [29] and a risk minimization protocol specific to *Cryptosporidium* contamination of pools and spas [30].

DISCUSSION

This outbreak included over 1000 laboratory-reported cases of cryptosporidiosis. It is likely that many thousands more were infected as a many infections are asymptomatic [7, 17], many symptomatic persons do not seek medical attention, and those who do uncommonly have their stools examined for *Cryptosporidium* [31].

Differences in notification rates among the HAs may be partly explained by differences in faecal screening practises, as well as by differences in disease occurrence. Plotting of monthly notifications rates by Health Area suggests an initial rural onset later extending to urban areas. The case control study subanalyses are consistent with person-to-person contact being the major mode of spread in rural areas and contaminated swimming pools the most important factor in the urban spread of the outbreak.

The predominance of notifications among young children is consistent with other swimming-related outbreaks [10, 14–16] as young children are more likely to swallow pool water whilst swimming. By contrast, a wider age range of cases is reported in outbreaks linked to contaminated drinking water supplies [7, 8]. However, the young age of the notifications might partly be explained by ascertainment bias as young children are more likely than older children and adults to have stool examination for gastro-intestinal illness [32].

This outbreak was linked to multiple pools, in contrast with other reported swimming-related outbreaks [10–16]. Pool contamination by *Cryptosporidium* is a particular challenge to control not only because of the oocysts' resistance to chlorine [6] but also because of their small size as they may not be adequately removed by the usual pool filtration systems [15, 26]. In addition, shared water filtration systems may allow spread of oocysts between pools within a swimming complex. Once introduced into pools, *Cryptosporidium* oocysts may survive many days or weeks infecting other swimmers.

Nevertheless, only 59% of cases reported swimming during their exposure periods. The remaining cases are likely to have been infected through person-to-person transmission [17–19] or through other unidentified modes.

The reason for the protective effect of drinking bottled water (even after adjustment for all other covariates including age) is unclear. It is, however,

unlikely to be an indicator of the avoidance of town water since 85% of the cases and 95% of the controls who drank bottled water also drank town water. One explanation is that people who drink bottled water are more cautious about their health.

The environmental investigations provided further evidence that multiple pool contamination was associated with the outbreak of illness. However, some degree of pool misclassification is likely as there was a lag time between reported exposures and pool samplings.

This large and prolonged state-wide outbreak of cryptosporidiosis was unusual because of its association with swimming at multiple pools. The control of swimming-pool-related cryptosporidiosis outbreaks presents challenges because of both host and organism characteristics. The cooperation of pool users and pool operators is paramount to control and prevent such outbreaks. Public health authorities also play a crucial part in: (i) raising awareness about the disease among health care providers; (ii) promoting with laboratories the importance of testing for cryptosporidiosis and reporting cases; (iii) stressing the importance of a timely, comprehensive and accurate surveillance system to allow the early detection and investigations of outbreaks; (iv) facilitating public education about cryptosporidiosis; and (v) educating pool operators about the prevention and control of *Cryptosporidium* pool water contamination [32–35].

ACKNOWLEDGEMENTS

We would like to thank: the staff from NSW Public Health Units, Dr Louisa Jorm, Director, Epidemiology and Surveillance Branch, NSW Health Department, Ms Jill Kaldor, Biostatistician, Epidemiology and Surveillance Branch, NSW Health Department, Ms Margaret Williamson, Manager, Health Survey Program Unit, and the staff of the Health Survey Program Unit, Epidemiology and Surveillance Branch, NSW Health Department.

REFERENCES

1. Guerrant RL. Cryptosporidiosis: an emerging, highly infectious threat. Emerg Infect Dis 1997; **3**: 51–7.
2. Current WL, Reese NC, Ernst JV, et al. Human cryptosporidiosis in immunocompetent and immunodeficient persons: studies of an outbreak and experimental transmission. N Engl J Med 1983; **308**: 1252–7.
3. Petersen C. Cryptosporidiosis in patients infected with the human immunodeficiency virus. Clin Infect Dis 1992; **15**: 903–9.
4. Jokipii L, Jokipii AMM. Timing of symptoms and oocyst excretion in human cryptosporidiosis. N Engl J Med 1986; **315**: 1643–7.
5. DuPont HL, Chappell CL, Sterling CR, et al. The infectivity of *Cryptosporidium parvum* in healthy volunteers. N Engl J Med 1995; **332**: 855–9.
6. Korich DG, Mead JR, Madore MS, et al. Effects of ozone, chlorine dioxide, chlorine, and monochloramine on *Cryptosporidium parvum* oocyst viability. Appl Environ Microbiol 1990; **56**: 1423–8.
7. McKenzie WR, Hoxie NJ, Proctor ME, et al. A massive outbreak in Milwaukee of *Cryptosporidium* infection transmitted through the public water supply. N Engl J Med 1994; **331**: 161–7.
8. Hayes EB, Matte TD, O'Brien TR, et al. Large community outbreak of cryptosporidiosis due to contamination of a filtered public water supply. N Engl J Med 1989; **320**: 1372–6.
9. Willocks L, Crampin A, Milne L, et al. A large outbreak of cryptosporidiosis associated with a public water supply from a deep chalk borehole. Commun Dis Public Health 1998; **1**: 239–43.
10. Lemmon JM, McAnulty JM, Bawden-Smith J. Outbreak of cryptosporidiosis linked to an indoor swimming pool. Med J Aust 1996; **165**: 613–6.
11. Sorvillo F, Fujioka K, Nahlen B, et al. Swimming-associated cryptosporidiosis. Am J Public Health 1992; **82**: 742–4.
12. Bell A, Guasparini R, Meeds D, et al. A swimming pool-associated outbreak of cryptosporidiosis in British Columbia. Can J Public Health 1993; **84**: 334–7.
13. Joce RE, Bruce J, Kiely D, et al. An outbreak of cryptosporidiosis associated with a swimming pool. Epidemiol Infect 1991; **107**: 497–508.
14. McAnulty JM, Fleming DW, Gonzales AH. A community-wide outbreak of cryptosporidiosis associated with swimming at a wave pool. JAMA 1994; **272**: 1597–600.
15. Centers for Disease Control and Prevention. *Cryptosporidium* infections associated with swimming pools – Dane County, Wisconsin, 1993. MMWR 1994; **43**: 561–3.
16. Baker M, Russell N, Roseveare C, et al. Outbreak of cryptosporidiosis linked to Hutt Valley swimming pool. New Zealand Public Health Rep 1998; **5**: 41–5.
17. Tangermann R, Gordon S, Wiesner P, et al. An outbreak of cryptosporidiosis in a day-care center in Georgia. Am J Epidemiol 1991; **133**: 471–6.
18. Cordell RL, Addiss DG. Cryptosporidiosis in childcare settings: a review of the literature and recommendations for prevention and control. Pediatr Infect Dis J 1994; **13**: 310–7.
19. Newman RD, Zu S, Wuhib T, et al. Household epidemiology of *Cryptosporidium parvum* infection in an urban community in Northeast Brazil. Ann Intern Med 1994; **120**: 500–5.

20. Miron D, Kenes J, Dagan R. Calves as a source of an outbreak of cryptosporidiosis among young children in an agricultural closed community. Pediatr Infect Dis J 1991; **10**: 438–41.
21. Millard PS, Gensheimer KF, Addiss DG, et al. An outbreak of cryptosporidiosis from fresh-pressed apple cider. JAMA 1994; **272**: 1592–6.
22. Cryptosporidiosis – The ACT outbreak. Presentation by Dr Doris Zonta, Chief Health Officer, to the ACT Legislative Assembly's Standing Committee on Health, December 1998.
23. Epidemiology and Surveillance Branch. 1997 NSW Health Survey Electronic Report: Methods. NSW Health Department 1999. http://www.health.nsw.gov.au/public-health/hs97/methods.htm
24. Fricker CR, Crabb JH. Water-borne cryptosporidiosis: Detection methods and treatment options. Adv Parasitol 1998; **40**: 241–78.
25. Centers for Disease Control and Prevention. Vessel sanitation program: Operations Manual. Atlanta, Georgia, August, 1989.
26. Juranek DD. Cryptosporidiosis: sources of infection and guidelines for prevention. Clin Infect Dis 1995; **21**: S57–61.
27. Public swimming pool and spa pool guidelines. NSW Health Department, ISBN 07310 9225 2, Sydney, NSW, 1996.
28. Cryptosporidiosis: the facts. NSW Health Department, Sydney, NSW, 1999.
29. Cryptosporidiosis: a guide for persons with HIV, AIDS, and immuno-suppressed systems. NSW Health Department, Sydney, NSW, 1999.
30. Protocol for minimising the risk of *Cryptosporidium* contamination in public swimming pools and spa pools. NSW Health Department, ISBN 07347 3125 6, Sydney, NSW, 1999.
31. Wheeler JG, Sethi D, Cowden JM, et al. Study of infectious intestinal disease in England: rates in the community, presenting to general practice, and reported to national surveillance. BMJ 1999; **318**: 1046–50.
32. Meinhardt PL, Casemore DP, Miller KB. Epidemiologic aspects of human cryptosporidiosis and the role of waterborne transmission. Epidemiol Rev 1996; **18**: 118–36.
33. Proctor ME, Blair KA, Davis JP. Surveillance data for waterborne illness detection: an assessment following a massive waterborne outbreak of *Cryptosporidium* infection. Epidemiol Infect 1998; **120**: 43–54.
34. Centers for Disease Control and Prevention. Assessing the public health threat associated with waterborne cryptosporidiosis: report of a workshop. MMWR 1995; **44**(RR-6): 1–16.
35. Rose JB. Environmental ecology of *Cryptosporidium* and public health implications. Annu Rev Public Heatlh 1997; **18**: 135–61.