Limiting swimming pool outbreaks of cryptosporidiosis – the roles of regulations, staff, patrons and research

Una Ryan, Sheleigh Lawler and Simon Reid

ABSTRACT

Cryptosporidium is the leading cause of swimming pool outbreaks of gastroenteritis. Transmission occurs through the ingestion of oocysts that are passed in the faeces of an infected person or animal when an accidental faecal release event occurs. *Cryptosporidium* parasites present specific challenges for infection control as oocysts are highly resistant to chlorine levels used for pool disinfection, infected individuals can shed large numbers of oocysts, there is a long incubation period and shedding of oocysts occurs even after symptom resolution. The purposes of this review are to identify key barriers to limiting swimming pool-associated outbreaks of cryptosporidiosis and to outline needs for research and collaboration to advance co-ordinated management practices. We reviewed swimming pool-associated cryptosporidiosis outbreaks, disinfection teachniques, current regulations and the role of staff and patrons. Key barriers to limiting swimming pool-associated outbreaks of cryptosporidiosis are a lack of uniform national and international standards, poor adherence and understanding of regulations governing staff and patron behaviour, and low levels of public knowledge and awareness.

Key words | *Cryptosporidium*, gastroenteritis, swimming pool

Una Ryan (corresponding author)
School of Veterinary and Life Sciences,
Murdoch University,
Perth,
Australia
E-mail: *una.ryan@murdoch.edu.au*

Sheleigh Lawler
Simon Reid
The University of Queensland,
School of Public Health,
Herston,
Queensland 4006,
Australia

INTRODUCTION

Death due to diarrhoea is one of the top five leading causes of mortality in children under 5 years of age (WHO 2013; GBD Mortality Causes of Death Collaborators 2015). *Cryptosporidium* is one of the four major contributors to moderate-to-severe diarrhoeal disease during the first 5 years of life in low-to-middle income countries (Kotloff *et al.* 2013). Indeed, it was second only to rotavirus as a cause of moderate-to-severe diarrhoea in children under 2 years and associated with a two to three times higher risk of mortality among children aged 12–23 months with moderate-to-severe diarrhoea, than in controls without diarrhoea (Kotloff *et al.* 2013). The most recent Global Burden of Disease study listed *Cryptosporidium* as an important cause of diarrhoea and death in children under 5 years of age (especially under two) in sub-Saharan Africa (GBD Mortality Causes of Death Collaborators 2015).

In Australia, there are an estimated 15.9 M cases of gastroenteritis each year (Kirk *et al.* 2014), resulting in economic costs estimated at over one billion dollars per year (through visits to medical practitioners and time taken off work) (Hellard *et al.* 2003; Anon 2006; Heyworth *et al.* 2006; Kirk *et al.* 2014). There have also been reports of an association between acute gastroenteritis (AGI) and the development of irritable bowel syndrome and reactive arthritis, and the costs of these chronic sequelae account for another $75 million (Anon 2006; Ford *et al.* 2014). Important agents that cause diarrhoea include viruses (e.g., rotavirus and norovirus), bacteria (e.g., *Shigella* species, *Escherichia coli* and *Campylobacter jejuni*) and parasites (*Giardia duodenalis*,

doi: 10.2166/wh.2016.160

Entamoeba histolytica and *Cryptosporidium* species (Shah *et al.* 2009).

Waterborne parasitic protozoa such as *Cryptosporidium* that are spread through contaminated drinking water and recreational water are a major contributor to gastroenteritis as they have a worldwide distribution (Cotruva *et al.* 2004; Shields *et al.* 2008a; CDC 2011), result in more than four billion cases of diarrhoea and 1.6 million deaths annually (www.who.int) and 62.5 million disability-adjusted life years worldwide (Cotruva *et al.* 2004; Baldursson & Karanis 2011; Burnet *et al.* 2014). From 2004 to 2010, 199 outbreaks of human gastroenteritis due to the waterborne transmission of enteric parasitic protozoa were reported worldwide and, of these, *Cryptosporidium* spp. were the aetiological agent in 60.3% of the outbreaks (Baldursson & Karanis 2011). Bathing in contaminated swimming and therapeutic pools is a major mode of waterborne transmission of *Cryptosporidium* and other pathogens (Dale *et al.* 2010; Soller *et al.* 2010; Sanborn & Takaro 2013).

Given the prominence of *Cryptosporidium* as a significant source of gastroenteritis globally, this review was undertaken to identify studies that describe swimming pool-associated outbreaks of cryptosporidiosis and to identify key barriers to limiting swimming pool-associated outbreaks of cryptosporidiosis. We hoped that such information can serve as a resource to health authorities worldwide.

METHODS

To identify relevant studies, we searched PubMed (http://www.ncbi.nlm.nih.gov/pubmed/) as well as ScienceDirect (http://www.sciencedirect.com/) and Google Scholar (http://scholar.google.com/). The following search terms were included: *Cryptosporidium*, cryptosporidiosis, outbreak, diagnosis, causes, swimming pool, recreational water, disinfection, regulation, staff and patrons. A total of 1,400 potentially relevant studies were identified. Studies were considered relevant if they identified specific recreational water-associated outbreaks of cryptosporidiosis and/or causes, limits of detection, regulation and roles of staff and patrons in outbreaks, resulting in more in-depth analysis of 157 studies.

CRYPTOSPORIDIUM OUTBREAKS IN SWIMMING POOLS – TRANSMISSION AND PROBLEMS WITH DIAGNOSIS

Currently, 31 *Cryptosporidium* species have been recognized as valid and of these more than 17 species have been identified in humans (Holubová *et al.* 2016; Kváč *et al.* 2016; Zahedi *et al.* 2016). By far the most common species reported in humans worldwide are *C. parvum* and *C. hominis* (Xiao 2010; Ryan & Xiao 2014) with *C. hominis* the main cause of human cryptosporidiosis in most countries (Xiao 2010; Ryan & Power 2012). Symptoms of cryptosporidiosis include diarrhoea, dehydration, weight loss, abdominal pain, fever, nausea and vomiting. In immunocompromised people, cryptosporidiosis can become chronic and result in death (Chalmers & Davies 2010; Xiao 2010). Although hundreds of drugs have been tested for prophylaxis and treatment of cryptosporidiosis in animals and humans, only one, nitazoxanide (Alinia®), has been approved for use in humans by the US Food and Drug Administration (FDA). This drug, however, exhibits only moderate clinical efficacy in children and immunocompetent people and none in people with HIV (Abubakar *et al.* 2007; Amadi *et al.* 2009; Pankiewicz *et al.* 2015).

Drinking water-associated outbreaks of diarrhoeal illness due to *Cryptosporidium* and other pathogens are declining, particularly in high-income countries, but the number of outbreaks of cryptosporidiosis and other diarrhoeal diseases associated with swimming pools, water parks and other 'disinfected venues' has increased dramatically (Pond 2005; Beach *et al.* 2009; IOM 2009; Valderrama *et al.* 2009; Yoder & Beach 2010; Yoder *et al.* 2010; Brunkard *et al.* 2011; Hlavsa *et al.* 2011, 2015; Sanborn & Takaro 2013). Outbreaks of cryptosporidiosis have been associated with recreational water in the USA (e.g., Sorvillo *et al.* 1992; Moore *et al.* 1993; CDC 1994, 2008; McAnulty *et al.* 1994; MacKenzie *et al.* 1995a; Lim *et al.* 2004; Mathieu *et al.* 2004; Craun *et al.* 2005; Causer *et al.* 2006; Wheeler *et al.* 2007; Shields *et al.* 2008b; Boehmer *et al.* 2009; Cantey *et al.* 2012; Cope *et al.* 2015; Hlavsa *et al.* 2015; DeSilva *et al.* 2016), Canada (Bell *et al.* 1993; Louie *et al.* 2004; Ong *et al.* 2008; Hopkins *et al.* 2013), Australia (e.g., Lemmon *et al.* 1998; Hellard *et al.* 2000; Stafford *et al.* 2000; Puesch *et al.* 2001; Black &

McAnulty 2006; Paterson & Goldthorpe 2006; Dale *et al.* 2010; Mayne *et al.* 2011; Waldron *et al.* 2011; Ng-Hublin *et al.* 2015), Japan (Ichinohe *et al.* 2005; Yokoi *et al.* 2005; Takagi *et al.* 2008), Sweden (Insulander *et al.* 2005; Mattsson *et al.* 2008) and the UK (e.g., Joce *et al.* 1991; Hunt *et al.* 1994; Furtado *et al.* 1998; Chalmers 2000; McLauchlin *et al.* 2000; Smith *et al.* 2006; Coetzee *et al.* 2008; Briggs *et al.* 2014). In the USA, treated recreational water was the source for 77% of all waterborne outbreaks between 2011 and 2012 (Hlavsa *et al.* 2015). In Australia, swimming in public pools was shown to be the main risk factor associated with sporadic cryptosporidiosis (Robertson *et al.* 2002). Swimming and therapeutic pools are, therefore, considered a more important transmission route for gastrointestinal illness than drinking water in high-income countries (Pond 2005; IOM 2009; Dale *et al.* 2010; Chalmers 2012).

Cryptosporidium oocysts can survive for 3.5–10.6 days in swimming pool water where free chlorine levels are maintained at Centers for Disease Control (CDC)-recommended levels (1–3 mg/L) (Shields *et al.* 2008b). Bromine, another chemical disinfectant used in swimming pools, is also ineffective for inactivation of *Cryptosporidium* at the doses used in normal pool water treatment (Korich *et al.* 1990). Viruses and bacteria can also cause swimming pool outbreaks, however most of them are more easily controlled by the proper application of chlorine and other disinfectants (WHO 2000). For example, *Shigella* spp. are quite susceptible to low levels of chlorine disinfectants and can be inactivated at levels less than 1.0 mg/L (Kebabjian 1995). Some viruses such as norovirus are reportedly resistant to levels of chlorination used in swimming pools (Keswick *et al.* 1985), but more recent studies report that adequate chlorination of pools may be sufficient to prevent norovirus transmission (Podewils *et al.* 2007).

Cryptosporidium is particularly suited to waterborne transmission as the environmental stage, the oocyst, is highly resistant to disinfection (Korich *et al.* 1990; Chauret *et al.* 2001; Painter *et al.* 2015) and is excreted in large quantities (10^8–10^9 oocysts in a single bowel movement) (Cordell 2001; Yoder & Beach 2007; Yoder *et al.* 2012) for up to 60 days after cessation of gastrointestinal symptoms (Jokipii & Jokipii 1986; Stehr-Green *et al.* 1987). In addition, the low infectious dose (10–100 oocysts) (DuPont *et al.* 1995; Chappell *et al.* 2006), means that ingestion of a relatively small amount of contaminated water is sufficient to initiate infection in a susceptible individual. The long incubation period of cryptosporidiosis (averaging 7 days) (Chalmers & Davies 2010), delays the identification of the source and implementation of interventions to decontaminate the source thus perpetuating transmission.

A quantitative risk assessment has estimated that ingestion of a single oocyst of the *C. parvum* IOWA isolate will result in clinical disease in 2.79% of immunologically normal persons (Pouillot *et al.* 2004). Given that the 50% infective dose (ID_{50}) for *C. hominis* is less than one-tenth that of the *C. parvum* IOWA isolate (DuPont *et al.* 1995), ingestion of only one oocyst of a more infectious isolate may lead to a higher incidence of infection in the general immunocompetent population. The following example shows how a single accidental faecal release (AFR) from a person infected with *Cryptosporidium* in a swimming pool can result in an infective dose for a human child or adult. An infected individual can excrete up to one billion oocysts during an infectious period and 1 mL of faeces can contain as many as 5×10^7 *Cryptosporidium* oocysts (Polgreen *et al.* 2012). If a child has a loose bowel movement of 150 mL into a typical 25 m × 12 m municipal pool containing about 450 m^3 of water, then there would be an average concentration of about 20,000 oocysts/litre (20/mL) (Gregory 2002). Oocysts would be dispersed throughout the pool due to the currents created by other swimmers, especially in a heavily used venue. The average swimmer ingests ~10–150 mL per hour of pool water (Dufour *et al.* 2006; Soller *et al.* 2010; Schets *et al.* 2011), therefore, a swimmer swallowing just 10 mL of water would ingest an average of 200 oocysts (Kebabjian 1995), which is well above a dose capable of causing infection (<100 oocysts) (DuPont *et al.* 1995; Chappell *et al.* 2006). A recent US study reported the risk of *Cryptosporidium* infection in all swimmers at 25 infections per 1,000 swimmers/year; 22 infections per 1,000 adult swimmers/year, and 29 infections per 1,000 child swimmers/year (Suppes *et al.* 2016).

Studies worldwide have shown that there is at least one AFR every week in most swimming pools throughout summer, with many pools reporting daily contaminations, especially hydrotherapy pools or those highly utilized by non-toilet-trained infants and toddlers (CDC 2001; Castor & Beach 2004; Schets *et al.* 2004; McManus *et al.* 2009;

Chalmers *et al.* 2011). Evidence also indicates that commercially available swim diapers are of limited value in preventing the release of *Cryptosporidium* into pool water (Maas *et al.* 2004; Amburgey & Anderson 2011).

Despite this, the majority of gastroenteritis outbreaks due to cryptosporidiosis and other enteric diseases are never identified, as: (1) it is estimated that less than 10% of individuals with gastroenteritis visit their local doctor, and of these, less than 10% have a faecal specimen collected (Garthright *et al.* 1988; Hellard & Fairley 1997; Majowicz *et al.* 2005; Tam *et al.* 2012); (2) not all individuals presenting with gastroenteritis will have faecal samples tested for microorganisms (Majowicz *et al.* 2005; Tam *et al.* 2012); (3) identification of *Cryptosporidium* and other pathogens via microscopy lacks specificity and sensitivity (Checkley *et al.* 2015); (4) microbiological confirmation of a swimming pool contamination is limited by the often transient nature of the contamination; and (5) current outbreak detection methods lack sensitivity, specificity and timeliness, i.e., there is a considerable lapse of time before an outbreak is recognized (Hellard *et al.* 2000; Dale *et al.* 2010). For example, the largest *Cryptosporidium* outbreak occurred in Milwaukee in 1993, where an estimated 403,000 individuals contracted cryptosporidiosis via contaminated drinking water (MacKenzie *et al.* 1995b) with an estimated illness-associated cost of $96.2 M and 100 deaths (Corso *et al.* 2003). Despite the high number of infected individuals, only a small number of cases were investigated prior to the identification of the outbreak and confirmation of its cause. Another outbreak of cryptosporidiosis in California involving more than 250 people, whose common exposure was a waterpark, went unreported to the local public health department for weeks, despite ~75% of the waterpark's 26 employees developing a gastrointestinal illness (Wheeler *et al.* 2007). In fact, most incident cases of cryptosporidiosis are not confirmed to be due to *Cryptosporidium* (Chalmers 2008; Smith *et al.* 2010; Tam *et al.* 2012; Briggs *et al.* 2014), and many apparently sporadic cases may be part of small outbreaks where the remainder of the cases go undiagnosed.

Cryptosporidiosis is an important disease in Oceania with an annual incidence of 12.8 illnesses per 100,000 and 22 per 100,000 in Australia (Lal *et al.* 2015a) and New Zealand (Snel *et al.* 2009), respectively. These rates are significantly higher than the USA (2.9 per 100,000), Canada (2.7 per 100,000) and in England and Wales (8 per 100,000) (Snel *et al.* 2009). In Australia, swimming pools were identified as the source of the majority of outbreaks of cryptosporidiosis in urban areas with cases mostly reported in children under 5 years of age and adult females (Lal *et al.* 2015a). This trend is also observed worldwide with the number of cryptosporidiosis outbreaks associated with disinfected recreational water venues increasing over the past several decades. For example, outbreaks linked to swimming pools have increased in the UK, with the majority of cases reported in the last 10 years (Chalmers 2012). In the USA, the number of recreational water-associated outbreaks reported annually has increased substantially since reporting to CDC began in 1978 (Hlavsa *et al.* 2011). Between 1995 and 2012, there were >1,000 recreational water-associated outbreaks of cryptosporidiosis in the USA (Painter *et al.* 2015), and the percentage of outbreaks of AGI caused by *Cryptosporidium* and associated with treated recreational water was 82.9% for both 2007–2008 (58/70) and 2005–2006 (29/35) (Yoder *et al.* 2008). In contrast, it was 68.2% (15/22), 50.0% (9/18) and 55.6% (10/18) in 1999–2000, 2001–2002 and 2003–2004, respectively (Lee *et al.* 2002; Yoder *et al.* 2004; Dziuban *et al.* 2006; Hlavsa *et al.* 2011).

There is a distinct seasonal pattern in the incidence of cryptosporidiosis, with the peak risk period coinciding with the summer recreational water season, reflecting increased use of communal swimming venues (Lal *et al.* 2015b; Painter *et al.* 2015). The recent increase in the number of reported outbreaks of recreational water-associated cryptosporidiosis is also associated with changes to reporting requirements in the USA and improvements in detection, investigation and reporting of waterborne disease outbreaks (Hlavsa *et al.* 2011).

REGULATION

In contrast to drinking water, there is a lack of uniform national and international standards for public treated recreational water venues (Chalmers 2012; Lewis *et al.* 2015; Painter *et al.* 2015). This is a major barrier to the prevention and control of illness associated with swimming pools as many swimming pool outbreaks are due to failures in

policies and procedures, poorly designed sanitation systems and inadequate maintenance (Chalmers 2012; Lewis *et al.* 2015; Painter *et al.* 2015).

The World Health Organization published guidelines for swimming pools in 2006 (WHO 2006), which addresses a wide range of hazards, including water quality, physical hazards (leading to drowning and injury), contamination of associated facilities and air quality. It includes guidelines for the assessment of the health hazards associated with swimming pools, their monitoring and assessment and maximum permissible microbiological concentrations derived from the findings of numerous studies into the infective doses of *Cryptosporidium* and other pathogens (WHO 2006). This guidance was included in the revised guidelines published by the UK Pool Water Treatment Advisory Group (PWTAG) in 2009 (http://pwtag.org). However, neither of these documents is legally enforceable and therefore adherence to the guidance varies across the industry (Lewis *et al.* 2015). In most pools, monitoring for potential microbial hazards is done using faecal indicator microorganisms such as coliform bacteria and especially *Escherichia coli* (*E. coli*) (rather than specific pathogens such as *Cryptosporidium*), which are easier and more cost-effective to detect and enumerate but are not informative on the presence of *Cryptosporidium* (Harwood *et al.* 2005; Coupe *et al.* 2006) and have much shorter survival times when exposed to chlorine than viruses and protozoa (Krusé 1969; Dahling *et al.* 1972; Steenland & Savitz 1997). Furthermore, many pools have been shown to exceed national legal limits for microbiological quality (Tesauro *et al.* 2010; Blougoura *et al.* 2011) and, in some facilities such as splash parks, no consistent requirements for water treatment exist (de Man *et al.* 2014).

In the UK, key aspects of *Cryptosporidium* (AFR) management are covered by the general requirements of current UK legislation such as the Health and Safety at Work Act 1974 (HMSO 1974). However, there are no specific regulations designed specifically for the swimming pool industry. In the USA, treated recreational water and pool regulations are enacted, implemented and enforced by state or local governments (Painter *et al.* 2015). In 2014, the CDC published a new set of guidelines – the Model Aquatic Health Code (MAHC) (http://www.cdc.gov/mahc, accessed 10 May 2016), which identifies *Cryptosporidium* as the leading cause of pool-associated outbreaks in the USA. These guidelines provide support to state and local health departments looking to reduce the risk of recreational water-associated illness. However as with the UK, they are voluntary guidelines and are currently not enforceable. Similarly, in Australia, swimming pools are managed at a local government level and while there are guidelines designed to regulate and guide the operation of swimming pools, including the Australian Standards (AS) and the Guidelines for Safe Pool Operation (GSPO), they are a voluntary guide for best practice only. In stark contrast, there is an apparent absence of swimming pool-associated outbreaks of cryptosporidiosis in Japan (Nakamura-Uchiyama *et al.* 2003; Karanis *et al.* 2007), where, anecdotally, swimming pool regulation is tightly enforced including compulsory showering prior to bathing and bans on young swimmers (<3 years old) and swimmers with signs of respiratory infections. While it is not possible to ascribe cause and effect in this situation it is tempting to consider the impact that more proactive regulation could have.

DISINFECTION

In swimming pools, chlorine is the most commonly used disinfectant. Other disinfectants, such as chloramine, chlorine dioxide, ozone and UV irradiation are also applied for water disinfection (Chowdhury *et al.* 2014). The filtration and disinfection of water in a swimming pool typically occurs as the water is recirculated through a treatment area at regular intervals that could be up to 4–8 hr on average (via a system of interconnected inlets and outlets spaced around the pool). A target pH (e.g., 7.2 to 7.6) and free chlorine level (e.g., 2 to 4 mg/L) are typically maintained in pool water (often by an automatic controller) to achieve some level of residual disinfection between treatment cycles. However, as discussed above, *Cryptosporidium* can survive for up to 11 days in properly chlorinated water (Amburgey *et al.* 2012). In addition, when chlorine reacts with organic matter in water (e.g., constituents of sweat and urine, skin particles, hair, microorganisms, cosmetics and other personal care products), a variety of disinfection by-products (DBPs) can be formed. Among these, trihalomethanes (THMs), haloacetic acids (HAAs), haloacetonitriles

(HANs) and chloral hydrate (CH) are the most prevalent chlorinated by-products (Chowdhury *et al.* 2014). Exposure to those DBPs is inevitable for any bather or trainer and can have elevated risks to human health as THMs and other DBPs are classified as probable carcinogens in humans (Chowdhury *et al.* 2014).

Currently AFRs are managed opportunistically, with high doses of chlorine added to the contamination area. In some instances, pools are closed to the public, then either emptied, disinfected and then refilled or the entire water body of the complex is superchlorinated (usually requiring a 12–24 hr closure). These procedures are a significant cost to the industry (~US$12,000/pool in lost revenue, cleaning expenses and labour). However, the recommendations for responding to faecal accidents may not be effective. For example, 20 mg chlorine/L for 12.75 hr is required to achieve a Ct [chlorine concentration in mg/L × time in min] value of 15,300 (=3 log reduction in oocyst viability and infectivity) (Shields *et al.* 2009). Furthermore, a study that simulated an AFR in a swimming pool to test the effectiveness of superchlorination on faeces-associated oocyst infectivity reported that oocysts were still infectious for mice after 3 days of exposure, possibly because the organic material protected oocysts from inactivation (Carpenter *et al.* 1999). There is also a risk that oocysts that form aggregates could be protected from inactivation (Carpenter *et al.* 1999; Gregory 2002). In addition, cyanuric acid, a chlorine-stabilizing compound used in pools throughout the world, has been shown to decrease the inactivation rate of *Cryptosporidium* (Shields *et al.* 2009; Murphy *et al.* 2015). The pH of the water, age of the oocysts and geographical origin (i.e., isolate variation) have also been shown to result in large differences in the Ct values required for a 3 log reduction of oocysts (Shields *et al.* 2008b).

As chlorine is largely ineffective at inactivating *Cryptosporidium*, the burden of protecting the public from cryptosporidiosis typically falls on the filters. Most swimming pool filters are sand filters that are not designed to remove *Cryptosporidium* (Amburgey *et al.* 2012). Research has shown that even 'high-rate' (i.e., 25–49 m/hr filter loading rate) swimming pool sand filters can only consistently deliver 0.11 to 0.28 log (22 to 48%) removal of *Cryptosporidium* oocysts and/or a microsphere surrogate without coagulation (Croll *et al.* 2007; Amburgey *et al.* 2012), which is inadequate to prevent outbreaks of cryptosporidiosis. In addition, modelling has shown that 'dead spots' exist at either end of the pool where *Cryptosporidium* could remain, even with filtration (Lewis *et al.* 2015). Precoat (i.e., diatomaceous earth and perlite) filters have been reported to remove 2.3–4.4 log (99.5–99.996%) spiked *Cryptosporidium* from swimming pool water at a filtration rate of 6.1 m/hr (swimming pool volume was less than 1,000 L) (Amburgey *et al.* 2009a, 2009b, 2012). However the ability of precoat filters to remove *Cryptosporidium* in full-scale swimming pools has yet to be extensively researched.

Chemical coagulation (creating clumps of oocysts) prior to filtration, greatly enhances the ability of the filters to remove *Cryptosporidium* oocysts, which normally pass through the filter (by reducing the natural electrostatic repulsion that exists between negatively charged oocysts and negatively charged filter media in water (Hall *et al.* 1994; Hendricks 2006; Amburgey *et al.* 2012). For example, addition of the coagulant polyaluminium chloride (PAC) consistently removed more than 90% of *Cryptosporidium*-sized microspheres at 30 m/hr filtration rate while the removals dropped to approximately 50% at a filtration rate of 37 m/hr with a 5.5 m^3 pilot-scale swimming pool (Lu & Amburgey 2016). Coagulant efficiency is pH dependent (should be <7.5) and even with coagulation under the correct pH, filters can allow breakthrough of *Cryptosporidium* oocysts if they are not backwashed frequently enough (Hall *et al.* 1994). Filter backwashing is a cleaning process by which the water flow through the filter is reversed so that accumulated debris trapped in the filter is dislodged and directed to waste. However, filter backwashing can disrupt filter integrity and allow *Cryptosporidium* oocysts to re-contaminate the pool water and infect swimmers (Greinert *et al.* 2004; Schets *et al.* 2004; Chalmers *et al.* 2011; Ehsan *et al.* 2015). Other factors that affect the ability of filters to remove *Cryptosporidium* include filter loading rate, depth of the filter media, coagulant type, coagulant dosage and, potentially, even how and where the coagulant is dosed (Amburgey *et al.* 2007, 2012; Croll *et al.* 2007).

Other disinfection methods such as ozone followed by chlorine as a secondary disinfectant (ozone/chlorine), and electrochemically generated mixed oxidants are increasingly being substituted for chlorine to minimize the formation of DBPs. Ozone is a high potential oxidant and

disinfectant but is usually followed by sequential disinfection with chlorine (ozone/chlorine) because of its instability and the relatively high doses required (Kleiser & Frimmel 2000). UV disinfection can reduce the overall chlorine consumption by 50%, thus treatments with UV light and/or ozone can increase the efficacy of chemical disinfectants for *Cryptosporidium* (Chalmers 2012; Donofrio *et al.* 2013; Lu *et al.* 2013) if the dose and contact time (CT) are sufficient (Driedger *et al.* 2000; Rennecker *et al.* 2000; Chalmers *et al.* 2011). However, as with ozone, UV only renders the water bactericidal at the point of use and therefore additional (residual) disinfection is required in swimming pools to deal with the effective inactivation of *Cryptosporidium*. In addition, bathers in the vicinity of an AFR from someone infected with *Cryptosporidium* may still be at risk if they ingest pool water because outbreaks have been documented even in pools disinfected with chlorine and ozone (Moore *et al.* 1993; McAnulty *et al.* 1994; Chalmers 2000; Boehmer *et al.* 2009; Chalmers *et al.* 2011). In addition, ozone is depleted more rapidly in the presence of organic materials and its efficacy is highly temperature dependent (i.e., decreasing at lower temperature) (Chalmers *et al.* 2011). Although widely used in the drinking water industry, there are relatively few data relating directly to UV light disinfection of *Cryptosporidium* in swimming pool water, but bench-scale studies from drinking water have shown that *Cryptosporidium* oocysts are irreversibly inactivated by UV treatment (at doses of ~10 mJ/cm^2) (Clancy *et al.* 2004; Rochelle *et al.* 2005; Hayes *et al.* 2013). Typically, doses of >60 mJ/cm^2 are used in swimming pools, which should be effective (Chalmers *et al.* 2011). While UV is effective at killing oocysts, such a barrier will only be effective if it is maintained and operated correctly. UV irradiation has been suspected to result in enhancement of THM concentrations (Judd *et al.* 1998); however, another study reported an absence of THM formation by UV and UV combined with chlorine (Spiliotopoulou *et al.* 2015). In addition, most of the research on ozone and UV disinfection of *Cryptosporidium* has involved small-scale laboratory bench studies, using very limited numbers of *Cryptosporidium* species. This is important because the robustness of the oocysts can vary substantially between sources and batches (Gregory 2002). More evidence of the efficacy of combined disinfection methods is needed under real-world conditions in continuous flow swimming pool waters.

Membrane filtration processes where water is forced by pressure (positive or vacuum) across a surface, excluding or rejecting particles greater than the effective pore size of the membrane material, such as microfiltration, ultrafiltration (UF), nanofiltration (NF), and reverse osmosis (RO) have been applied in swimming pools to remove DBPs (Barbot & Moulin 2008; Klüpfel *et al.* 2011) and *Cryptosporidium*-sized particles have been removed by NF membranes from a public drinking water source (Patterson *et al.* 2012). Largely because of the costs involved, this technology has yet to be routinely applied to swimming pools but full-scale application of a combined UF and RO treatment significantly reduced particulate matters from a swimming pool (Reißmann *et al.* 2005) and would prove useful in removing *Cryptosporidium* oocysts in response to an AFR.

ROLE OF STAFF

Staff at recreational facilities play a key role in controlling cryptosporidiosis outbreaks via an informed and timely response to AFRs and by adhering to strict hygiene standards themselves. Illness among lifeguards has been noted previously in reports of cryptosporidiosis outbreaks associated with recreational water. In the first reported outbreak of swimming pool-associated cryptosporidiosis in the USA, the authors noted that 75% of the lifeguards were ill (Sorvillo *et al.* 1992). Likewise, a swimming pool-associated cryptosporidiosis outbreak in Canada reported that 27% of the lifeguards had been ill (Bell *et al.* 1993). However, neither of these reports included the illness onset times of lifeguards relative to patrons, nor whether the lifeguards could have contributed to illness among patrons. In a Canadian swimming pool-associated cryptosporidiosis outbreak in 2004, a pool employee had a positive stool test for *Cryptosporidium* one month before the outbreak had been detected, and this person and other employees had been in the pools during their symptomatic periods (Louie *et al.* 2004). In 2012, in Western Australia, a swimming pool outbreak of cryptosporidiosis was linked to a swimming instructor, diagnosed with cryptosporidiosis, who continued to swim at the local pool,

contrary to the doctor's advice (Ng-Hublin *et al.* 2015). Thus lifeguards and swimming instructors may, in some cases, initiate, amplify or prolong an outbreak by going into the water while ill. A clear policy to keep lifeguards and bathers with diarrhoea out of pools and waterparks should be standardized, implemented and enforced.

A rapid and adequate response by staff to known and suspected faecal contamination events is also essential to prevent outbreaks. Management of faecal and diarrhoeal episodes should adhere to recommended guidelines and should include hyperchlorination for a time period that equates to a CT inactivation value of 15,300 (e.g., free chlorine levels of 20 ppm for 12.5 hr) (CDC 2016). Pool management and staff should ensure the facility complies with current codes and standards of operation and insist on patrons' adherence with regulations and practice of good hygiene when using the pool facilities. However, a recent survey of AFR responses in Australian pools revealed that many pools were not super-chlorinating adequately following an AFR (i.e., the majority of pools (>80%) were using less than half the recommended chlorine concentration and for only a fraction of time (10 min)). Many operators were also unclear as to how *Cryptosporidium* is transmitted. This finding is consistent with similar surveys conducted in the USA (CDC 2010). Studies of swimming pool-associated waterborne outbreaks have reported that a lack of coherent and standardized pool regulations are a major reason for outbreaks (Wilberschied 1995; Joce *et al.* 1991; Wheeler *et al.* 2007), which highlights the need for better education of staff and more effective and standardized management practices.

ROLE OF PATRONS

Swimming pool patrons also have an important role to play in preventing *Cryptosporidium* transmission. Prevention is a two-fold approach where a patron needs to prevent self-exposure, but also contaminaton of others (McClain *et al.* 2005). Current recommendations to prevent introducing *Cryptospodirium* into a pool are that swimmers refrain from entering a pool while ill with diarrhoea and, if diagnosed with cryptosporidiosis, for at least 2 weeks following complete symptom resolution (Painter *et al.* 2015). Yet, a recent US survey examining caregivers' awareness of cryptosporidiosis in children found just over a quarter of respondents (26.9%) were aware of abstaining from swimming until at least 2 weeks following complete symptom resolution (Munoz *et al.* 2016).

High risk swimming activities or behaviours (e.g., pool water ingestion, frequency of splashes to the face, leisurely swimming) (Suppes *et al.* 2014) should be avoided; however, one study reported that only 15% of respondents were aware that swallowing recreational water while swimming can lead to *Cryptosporidium* transmission (Munoz *et al.* 2016). Outbreak investigations of cryptosporidiosis related to exposures in swimming pools show that questionnaires are typically administered 2–3 weeks after exposures occur due to illness latency and case identification complications (Hellard *et al.* 2000; Insulander *et al.* 2005; Causer *et al.* 2006; Boehmer *et al.* 2009). Extended time periods between exposure and questionnaire administration increases the likelihood that recall bias of swimming activities associated with outbreaks will occur, with a recent study reporting up to 28% difference in recall bias when a questionnaire was administered immediately and 1 week after study participants swam in pool water (Suppes & Reynolds 2014). The uncertainties associated with swimmer recall have important implications for statistical associations between outbreaks and patron swimming activities, and needs to be factored into conclusions formulated by swimming pool outbreak investigators.

Recreational water can amplify smaller outbreaks into community-wide transmission when persons who are ill introduce the parasite into multiple recreational water venues or other settings (e.g., childcare facilities) (Cordell 2001; Turabelidze *et al.* 2007). Thus, management of an individual cluster of cases of cryptosporidiosis associated with a particular venue may be confounded by the simultaneous transmission at other venues (Painter *et al.* 2015). For example, an investigation of a community-wide cryptosporidiosis outbreak in Utah indicated that 20% of case-patients swam while ill with diarrhoea and identified approximately 450 potentially contaminated recreational water venues (CDC 2008). Educational and health promotion efforts to improve awareness and change swimmer behaviour is therefore a critical component of outbreak prevention.

CONCLUSIONS

Cryptosporidium is a major cause of swimming pool-associated outbreaks and developing a safer swimming environment is essential to protect patrons against contracting cryptosporidiosis from pools. Further research is required to better understand the knowledge, attitudes and practices (KAP) of swimming pool operators with regards to the public health risks associated with AFR and their management in order to inform swimming pool regulations. In addition, a better understanding of the KAP of swimming pool patrons, especially parents of young children, would enable the development of evidence-based education programmes to reduce the likelihood of AFR and reduce the likelihood of an infected person swimming. As child swimmers have the highest risk of infection due to their immature immunity, they should be targeted by public awareness messages and in education campaigns.

Lack of uniform national and international standards for swimming pools is a major issue, and state and local governments need to establish and enforce regulations for protecting swimming pools from *Cryptosporidium* contamination, including legislation on minimum design standards, operation, disinfection, filtration and AFR management. In the absence of this, communication bridges between pool operators, local health departments and other aquatic facilities should be encouraged. Pool operators need to be better educated on the risks of *Cryptosporidium* contamination and best practice management procedures, including a written AFR response policy and keeping records of all faecal accidents, chlorine and pH level measurements, and any major equipment repairs or changes. A number of factors can contribute to inadequate pool treatment: poor operating procedures (including disinfection), inadequate or inappropriate filtration equipment, excessive numbers of bathers, a high contamination load or a combination of these factors (Chalmers 2000). Most swimming pool filters have have limited effectiveness for *Cryptosporidium* removal and therefore continuous application of coagulants such as polyaluminum chloride (PAC) to reduce oocyst concentrations that may be present from unreported AFRs is recommended. The Pool Water Treatment Advisory Group recommends dosing pool water with PAC continuously by peristaltic pump at 0.1 mL/m^3 of the circulation flow in the UK and other European countries (Pool Water Treatment Advisory Group 2015). Very little is known about filter monitoring practices in most swimming pools but monitoring filter performance using parameters such as turbidity might provide an indication of filter performance, especially when coagulation is used. Pool operators should also be encouraged to separate filtration systems between childrens' paddling pools and adult pools, as if they are connected, faecal contamination can be dispersed from the children's paddling pool to the other pools. Provision of clean toilets, stocked with toilet paper, with ample soap for hand washing as well as clean shower areas and child changing rooms will also promote better hygiene practices by patrons.

Understanding the efficacy of existing pool management practices is essential and requires sensitive molecular detection and typing of *Cryptosporidium* from pool filters and AFRs (in addition to standard microbial testing) to determine which pools have the highest prevalence (and if specific strains/subtypes are being spread to other pools), coupled with patron and staff questionnaires to analyse factors linked to high prevalence levels and transmission of the parasite. Molecular detection should also be coupled with trialling of improved accidental faecal release management guidelines (AFRM) to determine if their implementation will reduce the public health risk associated with *Cryptosporidium* contamination of public swimming pools. These would include development and distribution of healthy swimming education materials to season pass holders, daily patrons and children at schools, prior to the 'pool season', that focus on avoiding swimming for 2 weeks after experiencing diarrhoea, intentional pool water ingestion, and splashing others in the face, etc.

Outbreaks of cryptosporidiosis are difficult to detect because surveillance systems are relatively insensitive (Hellard *et al.* 2000). However, timely intervention in the event of a rapid increase in notifications of disease linked to swimming pools has been shown to reduce the probability and scale of a community-wide outbreak (Coetzee *et al.* 2008). The interventions are broad in nature and generally include communication with local medical services (community and acute care) to increase vigilance and rate of confirmatory

testing and to give patients appropriate hygiene and exclusion advice (Coetzee *et al.* 2008). In addition, swimming pools can be encouraged to increase chlorination rates and enhance signage regarding exclusion after illness (Hellard *et al.* 2000).

Lastly, most disinfection studies have been conducted on only a few isolates of *C. parvum* and only one study has been conducted on UV inactivation of *C. hominis* (Johnson *et al.* 2005), despite this species being responsible for 70–90% of human cases of cryptosporidiosis (Xiao 2010). Although morphologically indistinguishable, there are distinct biological differences between the two species. *Cryptospordium hominis* is more virulent than *C. parvum* and has been associated with increased oocyst shedding (intensity and duration) and non-intestinal symptoms such as joint pain, eye pains, recurrent headache and fatigue whereas *C. parvum* is only associated with diarrhoea (Chalmers & Davies 2010). Therefore, full-scale swimming pool experiments to estimate the effectiveness of disinfection methods including superchlorination, filtration, ozone, UV and membrane filtration on a range of *C. parvum* and *C. hominis* isolates with and without the presence of faecal material need to be conducted.

REFERENCES

Abubakar, I., Aliyu, S. H., Arumugam, C., Usman, N. K. & Hunter, P. R. 2007 Treatment of cryptosporidiosis in immunocompromised individuals: systematic review and meta analysis. *Br. J. Clin. Pharmacol.* **63**, 387–393.

Amadi, B., Mwiya, M., Sianongo, S., Payne, L., Watuka, A., Katubulushi, M. & Kelly, P. 2009 High dose prolonged treatment with nitazoxanide is not effective for cryptosporidiosis in HIV positive Zambian children: a randomised controlled trial. *Infect. Dis.* **9**, 195.

Amburgey, J. E. & Anderson, J. B. 2011 Disposable swim diaper retention of *Cryptosporidium*-sized particles on human subjects in a recreational water setting. *J. Water Health* **9**, 653–658.

Amburgey, J. E., Fielding, R. R. & Arrowood, M. J. 2007 Removing *Cryptosporidium* oocysts from swimming pools with sand filters. In: *Proceedings National Swimming Pool Foundation (NSPF) 2007 World Aquatic Health™ Conference (WAHC), Cincinnati, OH, USA*.

Amburgey, J. E., Fielding, R. R. & Arrowood, M. J. 2009a Filtration removals and swim diaper retention of *Cryptosporidium* in swimming pools. In: *Swimming Pool and Spa International Conference, London, UK*.

Amburgey, J. E., Fielding, R. R. & Arrowood, M. J. 2009b Latest development in *Cryptosporidium* removal by swimming pool filters. In: *2009 World Aquatic Health Conference, Atlanta, GA, USA*.

Amburgey, J. E., Walsh, K. J., Fielding, R. R. & Arrowood, M. J. 2012 Removal of *Cryptosporidium* and polystyrene microspheres from swimming pool water with sand, cartridge, and precoat filters. *J. Water Health* **10** (1), 31–42.

Anon 2006 The annual cost of foodborne illness in Australia. www.ozfoodnet.gov.au/internet/ozfoodnet/…/cost-foodborne.pdf.

Baldursson, S. & Karanis, P. 2011 Waterborne transmission of protozoan parasites: review of worldwide outbreaks – an update 2004–2010. *Water Res.* **45**, 6603–6614.

Barbot, E. & Moulin, P. 2008 Swimming pool water treatment by ultrafiltration-adsorption process. *J. Membr. Sci.* **314**, 50–57.

Beach, M. J., Roy, S., Brunkard, J., Yoder, J. & Hlavsa, M. C. 2009 The changing epidemiology of waterborne disease outbreaks in the United States: implications for system infrastructure and future planning. In: *IOM (Institute of Medicine), Global Issues in Water, Sanitation, and Health*, The National Academies Press, Washington, DC, USA, pp. 156–169.

Bell, A., Guasparini, R., Meeds, D., Mathias, R. G. & Farley, J. D. 1993 A swimming pool-associated outbreak of cryptosporidiosis in British Columbia. *Can. J. Public Health* **84**, 334–337.

Black, M. & McAnulty, J. 2006 The investigation of an outbreak of cryptosporidiosis in NSW in 2005. *N. S. W. Public Health Bull.* **17**, 76–79.

Blougoura, A., Smeti, E. & Mavridou, A. 2011 *Sanitary Surveillance of Greek Swimming Pools. Hellenic Centre for Disease Control and Prevention Newsletter*, June, pp. 6–12 (edition in English).

Boehmer, T. K., Alden, N. B., Ghosh, T. S. & Vogt, R. L. 2009 Cryptosporidiosis from a community swimming pool: outbreak investigation and follow-up study. *Epidemiol. Infect.* **137** (11), 1651–1654.

Briggs, A. D., Boxall, N. S., Van Santen, D., Chalmers, R. M. & McCarthy, N. D. 2014 Approaches to the detection of very small, common, and easily missed outbreaks that together contribute substantially to human *Cryptosporidium* infection. *Epidemiol. Infect.* **142** (9), 1869–1876.

Brunkard, J. M., Ailes, E., Roberts, V. A., Hill, V., Hilborn, E. D., Craun, G. F., Rajasingham, A., Kahler, A., Garrison, L., Hicks, L., Carpenter, J., Wade, T. J., Beach, M. J. & Yoder, J. S. 2011 Surveillance for waterborne disease outbreaks associated with drinking water – United States, 2007-2008. *MMWR Surveill. Summ.* **60** (12), 38–68.

Burnet, J. B., Penny, C., Ogorzaly, L. & Cauchie, H. M. 2014 Spatial and temporal distribution of *Cryptosporidium* and *Giardia* in a drinking water resource: Implications for monitoring and risk assessment. *Sci. Total Environ.* **47**, 1023–1035.

Cantey, P. T., Kurian, A. K., Jefferson, D., Moerbe, M. M., Marshall, K., Blankenship, W. R., Rothbarth, G. R., Hwang, J., Hall, R.,

Yoder, J., Brunkard, J., Johnston, S., Xiao, L., Hill, V. R., Sarisky, J., Zarate-Bermudez, M. A., Otto, C. & Hlavsa, M. C. 2012 Outbreak of cryptosporidiosis associated with a man-made chlorinated lake – Tarrant County, Texas, 2008. *J. Environ. Health* **75** (4), 14–19.

Carpenter, C., Fayer, R., Trout, J. & Beach, M. J. 1999 Chorine disinfection of recreational water for *Cryptosporidium parvum*. *Emerg. Infect. Dis.* **5** (4), 1–10.

Castor, M. L. & Beach, M. J. 2004 Reducing illness transmission from disinfected recreational water venues: swimming, diarrhea and the emergence of a new public health concern. *Pediatr. Infect. Dis. J.* **23** (9), 866–870.

Causer, L. M., Handzel, T., Welch, P., Carr, M., Culp, D., Lucht, R. & Mudahar, K. 2006 An outbreak of *Cryptosporidium hominis* infection at an Illinois recreational waterpark. *Epidemiol. Infect.* **134** (1), 147–156.

Centers for Disease Control and Prevention (CDC) 1994 *Cryptosporidium* infections associated with swimming pools – Dane County, Wisconsin, 1993. *MMWR Morb. Mortal. Wkly Rep.* **43** (31), 561–563.

Centers for Disease Control and Prevention (CDC) 2001 Responding to fecal accidents in disinfected swimming venues. *MMWR Morb. Mortal. Wkly Rep.* 50 (20), 416–417.

Centers for Disease Control and Prevention (CDC) 2008 Communitywide cryptosporidiosis outbreak – Utah, 2007. *MMWR Morb. Mortal. Wkly Rep.* 57 (36), 989–993.

Centers for Disease Control and Prevention (CDC) 2010 Violations identified from routine swimming pool inspections – selected states and counties, United States, 2008. *J. Am. Med. Assoc.* **303**, 2468–2470.

Centers for Disease Control and Prevention 2011 Surveillance for waterborne disease outbreaks and other health events associated with recreational water United States, 2007–2008 and surveillance for waterborne disease outbreaks associated with drinking water, United States, 2007–2008. *MMWR* **60** (RR-12), 1–75.

Centers for Disease Control and Prevention 2016 http://www.cdc.gov/healthywater/swimming/pdf/fecal-incident-response-guidelines.pdf.

Chalmers, R. 2000 *Review of Outbreaks of Cryptosporidiosis in Swimming Pools.* Final report to Drinking Water Inspectorate. http://dwi.defra.gov.uk/research/completed-research/reports/DWI70_2_131_swimming%20pools.pdf.

Chalmers, R. 2008 *Cryptosporidium*: from laboratory diagnosis to surveillance and outbreaks. *Parasite* **15**, 372–378.

Chalmers, R. M. 2012 Waterborne outbreaks of cryptosporidiosis. *Ann. Ist. Super Sanita.* **48** (4), 429–446.

Chalmers, R. M. & Davies, A. P. 2010 Minireview: clinical cryptosporidiosis. *Exp. Parasitol.* **124**, 138–146.

Chalmers, R., McCann, R., Lowe, G., Modi, A., Stiff, R., Quigley, C., Cleary, P., Oyinloye, A., Lamb, P., Sewell, C., Jones, S., Elliott, M., Barratt, J., Calvert, J., Riley, R., Nichols, G. & Elliott, C. 2011 *Guidance for the Investigation of* Cryptosporidium *Linked to Swimming Pools. Public Health Wales*. www.cieh.org/WorkArea/DownloadAsset.aspx?id=41176.

Chappell, C. L., Okhuysen, P. C., Langer-Curry, R., Widmer, G., Akiyoshi, D. E., Tanriverdi, S. & Tzipori, S. 2006 *Cryptosporidium hominis*: experimental challenge of healthy adults. *Am. J. Trop. Med. Hyg.* **75**, 851–857.

Chauret, C. P., Radziminski, C. Z., Lepuil, M., Creason, R. & Andrews, R. C. 2001 Chlorine dioxide inactivation of *Cryptosporidium parvum* oocysts and bacterial spore indicators. *Appl. Environ. Microbiol.* **67**, 2993–3001.

Checkley, W., White Jr, A. C., Jaganath, D., Arrowood, M. J., Chalmers, R. M., Chen, X. M., Fayer, R., Griffiths, J. K., Guerrant, R. L., Hedstrom, L., Huston, C. D., Kotloff, K. L., Kang, G., Mead, J. R., Miller, M., Petri Jr, W. A., Priest, J. W., Roos, D. S., Striepen, B., Thompson, R. C., Ward, H. D., Van Voorhis, W. A., Xiao, L., Zhu, G. & Houpt, E. R. 2015 A review of the global burden, novel diagnostics, therapeutics, and vaccine targets for *Cryptosporidium*. *Lancet Infect. Dis.* **15**, 85–94.

Chowdhury, S., Alhooshani, K. & Karanfil, T. 2014 Disinfection byproducts in swimming pool: Occurrences, implications and future needs. *Water Res.* **53**, 68–109.

Clancy, J. L., Marshall, M. M., Hargy, T. M. & Dick, G. 2004 Susceptibility of five strains of *Cryptosporidium parvum* oocysts to UV light. *J. Am. Water Work Assoc.* **96**, 84–93.

Coetzee, N., Edeghere, O., Orendi, J., Chalmers, R. & Morgan, L. 2008 A swimming pool-associated outbreak of cryptosporidiosis in Staffordshire, England, October to December 2007. *Eurosurveill.* **13** (45), pii: 19028.

Cope, J. R., Prosser, A., Nowicki, S., Roberts, M. W., Roberts, J. M., Scheer, D., Anderson, C., Longsworth, A., Parsons, C., Goldschmidt, D., Johnston, S., Bishop, H., Xiao, L., Hill, V., Beach, M. & Hlavsa, M. C. 2015 Preventing community-wide transmission of *Cryptosporidium*: a proactive public health response to a swimming pool-associated outbreak – Auglaize County, Ohio, USA. *Epidemiol. Infect.* **143** (16), 3459–3467.

Cordell, R. L. 2001 The risk of infectious diseases among childcare providers. *J. Am. Med. Wom. Assoc.* **56**, 109–112.

Corso, P. S., Kramer, M. H., Blair, K. A., Addiss, D. G., Davis, J. P. & Haddix, A. C. 2003 Cost of illness in the 1993 waterborne *Cryptosporidium* outbreak, Milwaukee, Wisconsin. *Emerg. Infect. Dis.* **9** (4), 426–431.

Cotruva, J. A., Dufour, A., Rees, G., Bartram, J., Carr, R., Cliver, D. O., Craun, G. F., Fayer, R. & Gannon, V. P. J. 2004 *Waterborne Zoonoses: Identification Causes and Control*. World Health Organization, IWA Publishing, London, UK.

Coupe, S., Delabre, K., Pouillot, R., Houdart, S., Santillana-Hayat, M. & Derouin, F. 2006 Detection of *Cryptosporidium, Giardia* and *Enterocytozoon bieneusi* in surface water, including recreational areas: a one-year prospective study. *FEMS Immunol. Med. Microbiol.* **47** (3), 351–359.

Craun, G. F., Calderon, R. L. & Craun, M. F. 2005 Outbreaks associated with recreational water in the United States. *Int. J. Environ. Health Res.* **15** (4), 243–262.

Croll, B. T., Hayes, C. R. & Moss, S. 2007 Simulated *Cryptosporidium* removal under swimming pool filtration conditions. *Water Environ. J.* **21**, 149–156.

Dahling, D., Scarpino, P. V., Lucas, M., Berg, G. & Chang, S. L. 1972 *Destruction of Viruses and Bacteria in Water by Chlorine*. Abstr. Annual Meeting of the American Society for Microbiology, Washington, DC. Abstr. No. E152, p. 26.

Dale, K., Kirk, M., Sinclair, M., Hall, R. & Leder, K. 2010 Reported waterborne outbreaks of gastrointestinal disease in Australia are predominantly associated with recreational exposure. *Aust. N Z. J. Public Health* **34** (5), 527–530.

De Man, H., Leenen, E. J. T. M., van Knapen, F. & Roda Husman, A. M. 2014 Risk factors and monitoring for water quality to determine best management practices for splash parks. *J. Water Health* **12** (3), 399–403.

DeSilva, M. B., Schafer, S., Kendall Scott, M., Robinson, B., Hills, A., Buser, G. L., Salis, K., Gargano, J., Yoder, J., Hill, V., Xiao, L., Roellig, D. & Hedberg, K. 2016 Community-wide cryptosporidiosis outbreak associated with a surface water-supplied municipal water system – Baker City, Oregon, 2013. *Epidemiol. Infect.* **144** (2), 274–284.

Donofrio, R. S., Aridi, S., Saha, R., Bechanko, R., Schaefer, K., Bestervelt, L. L. & Hamil, B. 2013 Laboratory validation of an ozone device for recreational water treatment. *J. Water Health* **11** (2), 267–276.

Driedger, A. M., Rennecker, J. L. & Marinas, B. J. 2000 Sequential inactivation of *Cryptosporidium parvum* oocysts with ozone and free chlorine. *Water Res.* **34** (14), 3591–3597.

Dufour, A. P., Evans, O., Behymer, T. D. & Cantu, R. 2006 Water ingestion during swimming activities in a pool: a pilot study. *J. Water Health* **4**, 425–430.

DuPont, H. L., Chappell, C. L., Sterling, C. R., Okhuysen, P. C., Rose, J. B. & Jakubowski, W. 1995 The infectivity of *Cryptosporidium parvum* in healthy volunteers. *N. Engl. J. Med.* **332**, 855–859.

Dziuban, E. J., Liang, J. L., Craun, G. F., Hill, V., Yu, P. A., Painter, J., Moore, M. R., Calderon, R. L., Roy, S. L., Beach, M. J.; Centers for Disease Control and Prevention (CDC) 2006 Surveillance for waterborne disease and outbreaks associated with recreational water – United States, 2003-2004. *MMWR Surveill. Summ.* **55** (12), 1–30.

Ehsan, M. A., Casaert, S., Levecke, B., Van Rooy, L., Pelicaen, J., Smis, A., De Backer, J., Vervaeke, B., De Smedt, S., Schoonbaert, F., Lammens, S., Warmoes, T., Geurden, T. & Claerebout, E. 2015 *Cryptosporidium* and *Giardia* in recreational water in Belgium. *J. Water Health* **13** (3), 870–878.

Ford, L., Kirk, M., Glass, K. & Hall, G. 2014 Sequelae of foodborne illness caused by 5 pathogens, Australia, circa 2010. *Emerg. Infect. Dis.* **20** (11), 1865–1871.

Furtado, C., Adak, G. K., Stuart, J. M., Wall, P. G., Evans, H. S. & Casemore, D. P. 1998 Outbreaks of waterborne infectious intestinal disease in England and Wales, 1992-5. *Epidemiol. Infect.* **121** (1), 109–119.

Garthright, W. E., Arche, D. L. & Kvenberg, J. E. 1988 Estimates of incidence and costs of intestinal infectious diseases in the United States. *Public Health Rep.* **103** (2), 107–115.

GBD mortality causes of death collaborators 2015 Global, regional, and national age-sex specific all-cause and cause-specific mortality for 240 causes of death, 1990–2013: a systematic analysis for the Global Burden of Disease Study 2013. *Lancet* **385**, 117–171.

Gregory, T. 2002 Bench-marking pool water treatment for coping with *Cryptosporidium*. *J. Environ. Health Res.* **1** (1), 11–18.

Greinert, J., Furtado, D., Smith, J., Monte Barardi, C. & Simões, C. 2004 Detection of *Cryptosporidium* oocysts and *Giardia* cysts in swimming pool filter backwash water concentrates by flocculation and immunomagnetic separation. *Int. J. Environ. Health Res.* **14** (6), 395–404.

Hall, T., Pressdee, J. & Carrington, E. G. 1994 *Removal of* Cryptosporidium *oocysts by Water Treatment Processes*, Report FR 0457, Foundation for Water Research.

Harwood, V. J., Levine, A. D., Scott, T. M., Chivukula, V., Lukasik, J., Farrah, S. R. & Rose, J. B. 2005 Validity of the indicator paradigm for waterborne pathogen reduction in reclaimed water and public health protection. *Appl. Environ. Microbiol.* **71**, 3163–3170.

Hayes, J., Kirf, D., Garvey, M. & Rowan, N. 2013 Disinfection and toxicological assessments of pulsed UV and pulsed-plasma gas-discharge treated-water containing the waterborne protozoan enteroparasite *Cryptosporidium parvum*. *J. Microbiol Methods* **94** (3), 325–337.

Hellard, M. E. & Fairley, C. K. 1997 Gastroenteritis in Australia: who, what, where, and how much? *Aust. NZ J. Med.* **27** (2), 147–149.

Hellard, M. E., Sinclair, M. I., Fairley, C. K., Andrews, R. M., Bailey, M., Black, J., Dharmage, S. C. & Kirk, M. D. 2000 An outbreak of cryptosporidiosis in an urban swimming pool: why are such outbreaks difficult to detect? *Aust. N Z J Public Health* **24** (3), 272–275.

Hellard, M. E., Sinclair, M. I., Harris, A. H., Kirk, M. & Fairley, C. K. 2003 Cost of community gastroenteritis. *J. Gastroenterol. Hepatol.* **18** (3), 322–328.

Hendricks, D. W. 2006 *Water Treatment Unit Processes: Physical and Chemical*. CRC Press (Taylor and Francis Group), Boca Raton, FL, USA.

Heyworth, J. S., Jardine, A., Glonek, G. & Maynard, E. J. 2006 Incidence, impact on the family and cost of gastroenteritis among 4 to 6-year-old children in South Australia. *J. Gastroenterol. Hepatol.* **21** (8), 1320–1325.

Hlavsa, M. C., Roberts, V. A., Anderson, A. R., Hill, V. R., Kahler, A. M., Orr, M., Garrison, L. E., Hicks, L. A., Newton, A., Hilborn, E. D., Wade, T. J., Beach, M. J., Yoder, J. S.; CDC 2011 Surveillance for waterborne disease outbreaks and other health events associated with recreational water – United States, 2007–2008. *MMWR Surveill. Summ.* **60** (12), 1–32.

Hlavsa, M. C., Roberts, V. A., Kahler, A. M., Hilborn, E. D., Mecher, T. R., Beach, M. J., Wade, T. J., Yoder, J. S.; Centers for Disease Control and Prevention (CDC) 2015 Outbreaks of illness associated with recreational water – United States, 2011–2012. *MMWR Morb. Mortal. Wkly Rep.* **64** (24), 668–672.

HMSO 1974 Health and Safety at Work etc Act 1974, Chapter 37. http://www.legislation.gov.uk/ukpga/1974/37 (accessed 1 June 2016).

Holubová, N., Sak, B., Horčičková, M., Hlásková, L., Květoňová, D., Menchaca, S., McEvoy, J. & Kváč, M. 2016 *Cryptosporidium* avium n. sp. (Apicomplexa: Cryptosporidiidae) in birds. *Parasitol. Res.* doi:10.1007/s00436-016-4967-8.

Hopkins, J., Hague, H., Hudgin, G., Ross, L. & Moore, D. 2013 An outbreak of *Cryptosporidium* at a recreational water park in Niagara Region, Canada. *J. Environ. Health* **75** (9), 28–33.

Hunt, D. A., Sebugwawo, S., Edmondson, S. G. & Casemore, D. P. 1994 Cryptosporidiosis associated with a swimming pool complex. *Commun. Dis. Rep. CDR Rev.* **4** (2), R20–R22.

Ichinohe, S., Fukushima, T., Kishida, K., Sanbe, K., Saika, S. & Ogura, M. 2005 Secondary transmission of cryptosporidiosis associated with swimming pool use. *Jpn. J. Infect. Dis.* **58** (6), 400–401.

Insulander, M., Lebbad, M., Stenstrom, T. A. & Svenungsson, B. 2005 An outbreak of cryptosporidiosis associated with exposure to swimming pool water. *Scand. J. Infect. Dis.* **37** (5), 354–360.

IOM (Institute of Medicine) 2009 *Global Issues in Water, Sanitation, and Health*. The National Academies Press, Washington, DC, USA.

Joce, R. E., Bruce, J., Kiely, D., Noah, N. D., Dempster, W. B., Stalker, R., Gumsley, P., Chapman, P. A., Norman, P., Watkins, J., Smith, H. V., Price, T. J. & Watts, D. 1991 An outbreak of cryptosporidiosis associated with a swimming pool. *Epidemiol Infect.* **107** (3), 497–508.

Johnson, A. M., Linden, K., Ciociola, K. M., De Leon, R., Widmer, G. & Rochelle, P. A. 2005 UV inactivation of *Cryptosporidium hominis* as measured in cell culture. *Appl. Environ. Microbiol.* **71** (5), 2800–2802.

Jokipii, L. & Jokipii, A. M. 1986 Timing of symptoms and oocyst excretion in human cryptosporidiosis. *New Engl. J. Med.* **315**, 1643–1647.

Judd, S. J., Black, S. H. & Darkel, K. 1998 Disinfection by-product formation in swimming pool waters. In: *Swimming Pool Water Disinfection II Conference*, School of Water Sciences & PWTAG, Cranfield, UK.

Karanis, P., Kourenti, C. & Smith, H. 2007 Waterborne transmission of protozoan parasites: a worldwide review of outbreaks and lessons learnt. *J. Water Health* **5** (1), 1–38.

Kebabjian, R. S. 1995 Disinfection of public pools and management of fecal accidents. *J. Environ. Health* **58**, 8–12.

Keswick, B. H., Satterwhite, T. K., Johnson, P. C., DuPont, H. L., Secor, S. L., Bitsura, J. A., Gary, G. W. & Hoff, J. C. 1985 Inactivation of Norwalk virus in drinking water by chlorine. *Appl. Environ. Microbiol.* **50** (2), 261–264.

Kirk, M., Ford, L., Glass, K. & Hall, G. 2014 Foodborne illness, Australia, circa 2000 and circa 2010. *Emerg. Infect. Dis.* **20** (11), 1857–1864.

Kleiser, G. & Frimmel, F. H. 2000 Removal of precursors for disinfection by-products (DBPs) – differences between ozone- and OH-radical-include oxidation. *Sci. Total Environ.* **256**, 1–9.

Klüpfel, A. M., Glauner, T., Zwiener, C. & Frimmel, F. H. 2011 Nanofiltration for enhanced removal of disinfection byproduct (DBP) precursors in swimming pool water-retention and water quality estimation. *Water Sci. Technol.* **63** (8), 1716–1725.

Korich, D. G., Mead, J. R., Madore, M. S., Sinclair, N. A. & Sterling, C. R. 1990 Effects of ozone, chlorine dioxide, chlorine and monochloramine on *Cryptosporidium parvum* viability. *Appl. Environ. Microbiol.* **56**, 1423–1428.

Kotloff, K. L., Nataro, J. P., Blackwelder, W. C., Nasrin, D., Farag, T. H., Panchalingam, S., Wu, Y., Sow, S. O., Sur, D., Breiman, R. F., Faruque, A. S., Zaidi, A. K., Saha, D., Alonso, P. L., Tamboura, B., Sanogo, D., Onwuchekwa, U., Manna, B., Ramamurthy, T., Kanungo, S., Ochieng, J. B., Omore, R., Oundo, J. O., Hossain, A., Das, S. K., Ahmed, S., Qureshi, S., Quadri, F., Adegbola, R. A., Antonio, M., Hossain, M. J., Akinsola, A., Mandomando, I., Nhampossa, T., Acacio, S., Biswas, K., O'Reilly, C. E., Mintz, E. D., Berkeley, L. Y., Muhsen, K., Sommerfelt, H., Robins-Browne, R. M. & Levine, M. M. 2013 Burden and aetiology of diarrhoeal disease in infants and young children in developing countries (the Global Enteric Multicenter Study, GEMS): a prospective, case-control study. *Lancet* **382**, 209–222.

Krusé, C. W. 1969 *Mode of Action of Halogens on Bacteria and Viruses and Protozoa in Water Systems*. Final report to the Commission on Environmental Hygiene of the Armed Forces Epidemiological Board, US Army Med. Res. Dev. Command. Contract No. DA-49-193-MD-2314.

Kváč, M., Havrdová, N., Hlásková, L., Daňková, T., Kanděra, J., Ježková, J., Vítovec, J., Sak, B., Ortega, Y., Xiao, L., Modrý, D., Chelladurai, J. R., Prantlová, V. & McEvoy, J. 2016 *Cryptosporidium proliferans* n. sp. (Apicomplexa: Cryptosporidiidae): Molecular and biological evidence of cryptic species within gastric *Cryptosporidium* of mammals. *PLoS One.* **11** (1), e0147090.

Lal, A., Cornish, L. M., Fearnley, E., Glass, K. & Kirk, M. 2015a Cryptosporidiosis: A disease of tropical and remote areas in Australia. *PLoS Negl Trop Dis.* **9**, e0004078.

Lal, A., Fearnley, E. & Kirk, M. 2015b The risk of reported Cryptosporidiosis in children aged <5 years in Australia is highest in very remote regions. *Int. J. Environ. Res. Public Health* **12**, 11815–11828.

Lee, S. H., Levy, D. A., Craun, G. F., Beach, M. J. & Calderon, R. L. 2002 Surveillance for waterborne-disease outbreaks-United States, 1999–2000. *MMWR Surveill. Summ.* **51** (8), 1–47.

Lemmon, J. M., McAnulty, J. M. & Bawden-Smith, J. 1998 Outbreak of cryptosporidiosis linked to an indoor swimming pool. *Med. J. Australia* **165** (11–12), 296–297.

Lewis, L., Chew, J., Woodley, I., Colbourne, J. & Pond, K. 2015 The application of computational fluid dynamics and small-scale physical models to assess the effects of operational practices on the risk to public health within large indoor swimming pools. *J. Water Health* **13** (4), 939–952.

Lim, L. S., Varkey, P., Giesen, P. & Edmonson, L. 2004 Cryptosporidiosis outbreak in a recreational swimming pool in Minnesota. *J. Environ. Health* **67** (1), 16–20, 28, 27; quiz 31–32.

Louie, K., Gustafson, L., Fyfe, M., Gill, I., MacDougall, L., Tom, L., Wong, Q. & Isaac-Renton, J. 2004 An outbreak of *Cryptosporidium parvum* in a Surrey pool with detection in pool water sampling. *Can. Commun. Dis. Rep.* **30** (7), 61–66.

Lu, P. & Amburgey, J. E. 2016 A pilot-scale study of *Cryptosporidium*-sized microsphere removals from swimming pools via sand filtration. *J. Water Health* **14** (1), 109–120.

Lu, P., Yuan, T., Feng, Q., Xu, A. & Li, J. 2013 Review of swimming-associated cryptosporidiosis and *Cryptosporidium* oocyst removals from swimming pools. *Water Quality Res. J. Canada* **48**, 30–39.

Maas, R. P., Patch, S. C., Berkowitz, J. F. & Johnson, H. D. 2004 Determination of the solids retainment effectiveness of disposable swim diapers. *J. Environ. Health* **66** (10), 16–20, 28.

MacKenzie, W. R., Kazmierczak, J. J. & Davis, J. P. 1995a An outbreak of cryptosporidiosis associated with a resort swimming pool. *Epidemiol. Infect.* **115** (3), 545–553.

MacKenzie, W. R., Schell, W. L., Blair, K. A., Addiss, D. G., Peterson, D. E., Hoxie, N. J., Kazmierczak, J. J. & Davis, J. P. 1995b Massive outbreak of waterborne *Cryptosporidium* infection in Milwaukee, Wisconsin: recurrence of illness and risk of secondary transmission. *Clin. Infect. Dis.* **21** (1), 57–62.

Majowicz, S. E., Edge, V. L., Fazil, A., McNab, W. B., Doré, K. A., Sockett, P. N., Flint, J. A., Middleton, D., McEwen, S. A. & Wilson, J. B. 2005 Estimating the under-reporting rate for infectious gastrointestinal illness in Ontario. *Can. J. Public Health* **96** (3), 178–181.

Mathieu, E., Levy, D. A., Veverka, F., Parrish, M. K., Sarisky, J., Shapiro, N., Johnston, S., Handzel, T., Hightower, A., Xiao, L., Lee, Y. M., York, S., Arrowood, M., Lee, R. & Jones, J. L. 2004 Epidemiologic and environmental investigation of a recreational water outbreak caused by two genotypes of *Cryptosporidium parvum* in Ohio in 2000. *Am. J. Trop. Med. Hyg.* **71** (5), 582–589.

Mattsson, J. G., Insulander, M., Lebbad, M., Bjorkman, C. & Svenungsson, B. 2008 Molecular typing of *Cryptosporidium parvum* associated with a diarrhoea outbreak identifies two sources of exposure. *Epidemiol. Infect.* **136** (8), 1147–1152.

Mayne, D. J., Ressler, K. A., Smith, D., Hockey, G., Botham, S. J. & Ferson, M. J. 2011 A community outbreak of cryptosporidiosis in Sydney associated with a public swimming facility: a case-control study. *Interdiscip. Perspect. Infect. Dis.* **2011**, 341065.

McAnulty, J. M., Fleming, D. W. & Gonzalez, A. H. 1994 A community-wide outbreak of cryptosporidiosis associated with swimming at a wave pool. *JAMA* **272** (20), 1597–1600.

McClain, J., Bernhardt, J. M. & Beach, M. J. 2005 Assessing parents' perception of children's risk for recreational water illnesses. *Emerg Infect Dis.* **11** (5), 670–676.

McLauchlin, J., Amar, C., Pedraza-Díaz, S. & Nichols, G. L. 2000 Molecular epidemiological analysis of *Cryptosporidium* spp. in the United Kingdom: results of genotyping *Cryptosporidium* spp. in 1,705 fecal samples from humans and 105 fecal samples from livestock animals. *J. Clin Microbiol.* **38** (11), 3984–3990.

McManus, A., Bertolatti, D. & Devine, B. 2009 Management of waterborne pathogens in public swimming pools – evidence needed. *Australasian. Med. J.* **1** (2), 1–4.

Moore, A. C., Herwaldt, B. L., Craun, G. F., Calderon, R. L., Highsmith, A. K. & Juranek, D. D. 1993 Surveillance for waterborne disease outbreaks – United States, 1991–1992. *MMWR CDC Surveill. Summ.* **42** (5), 1–22.

Munoz, F. M., Attias, E., Czinn, S. J., Black, J.; Persistent Diarrhea Working Group 2016 Paediatrician and caregiver awareness of Cryptosporidiosis and Giardiasis in children: US survey responses. *Zoonoses Public Health* **63**, 410–419.

Murphy, J. L., Arrowood, M. J., Lu, X., Hlavsa, M. C., Beach, M. J. & Hill, V. R. 2015 Effect of cyanuric acid on the inactivation of *Cryptosporidium parvum* under hyperchlorination conditions. *Environ. Sci. Technol.* **49** (12), 7348–7355.

Nakamura-Uchiyama, F., Hiromatsu, K., Ishiwata, K., Sakamoto, Y. & Nawa, Y. 2003 The current status of parasitic diseases in Japan. *Intern. Med.* **42** (3), 222–236.

Ng-Hublin, J. S., Hargrave, D., Combs, B. & Ryan, U. 2015 Investigation of a swimming pool-associated cryptosporidiosis outbreak in the Kimberley region of Western Australia. *Epidemiol. Infect.* **143** (5), 1037–1041.

Ong, C. S., Chow, S., Gustafson, R., Plohman, C., Parker, R., Isaac-Renton, J. L. & Fyfe, M. W. 2008 Rare *Cryptosporidium hominis* subtype associated with aquatic center use. *Emerg. Infect. Dis.* **14** (8), 1323–1325.

Painter, J. E., Hlavsa, M. C., Collier, S. A., Xiao, L., Yoder, J. S.; Centers for Disease Control and Prevention 2015 Cryptosporidiosis surveillance – United States, 2011–2012. *MMWR Surveill. Summ.* **64** (Suppl. 3), 1–14.

Pankiewicz, K. W., Petrelli, R., Singh, R. & Felczak, K. 2015 Nicotinamide adenine dinucleotide based therapeutics, Update. *Curr. Med. Chem.* **2** (34), 3991–4028.

Paterson, J. & Goldthorpe, I. 2006 Managing a cluster of cryptosporidiosis associated with a public swimming pool. *N S W Public Health Bull.* **17** (5–6), 80.

Patterson, C., Anderson, A., Sinha, R., Muhammad, N. & Pearson, D. 2012 Nanofiltration membranes for removal of color and pathogens in small public drinking water sources. *J. Environ. Eng.* **138** (1), 48–57.

Podewils, L. J., Zanardi Blevins, L., Hagenbuch, M., Itani, D., Burns, A., Otto, C., Blanton, L., Adams, S., Monroe, S. S., Beach, M. J. & Widdowson, M. 2007 Outbreak of norovirus illness associated with a swimming pool. *Epidemiol. Infect.* **135** (5), 827–833.

Polgreen, P. M., Sparks, J. D., Polgreen, L. A., Yang, M., Harris, M. L., Pentella, M. A. & Cavanaugh, J. E. 2012 A statewide outbreak of *Cryptosporidium* and its association with the distribution of public swimming pools. *Epidemiol. Infect.* **140** (8), 1439–1445.

Pond, K. 2005 *Water Recreation and Disease. Plausibility of Associated Infections: Acute Effects, Sequelae and Mortality*. IWA Publishing, London, UK, on behalf of the World Health Organization.

Pool Water Treatment Advisory Group 2015 Polyaluminum chlorine dosing. Available at: http://pwtag.org/technicalnotes/polyaluminium-chloride-pac-dosing/ (accessed 6 August 2016).

Pouillot, R., Beaudeau, P., Denis, J. B., Derouin, F.; AFSSA *Cryptosporidium* Study Group 2004 A quantitative risk assessment of waterborne cryptosporidiosis in France using second-order Monte Carlo simulation. *Risk Anal.* **24** (1), 1–17.

Puesch, M. C., McAnulty, J. M., Lesjak, M., Shaw, N., Heron, L. & Watson, J. M. 2001 A statewide outbreak of cryptosporidiosis in New South Wales associated with swimming at public swimming pools. *Epidemiol. Infect. Dis.* **126** (3), 385–396.

Reißmann, F. G., Schulze, E. & Albrecht, V. 2005 Application of a combined UF/RO system for the reuse of filter backwash water from treated swimming pool water. *Desalination* **178**, 41–49.

Rennecker, J. L., Driedger, A. M., Rubin, S. A. & Marinas, B. J. 2000 Synergy in sequential inactivation of *Cryptosporidium parvum* with ozone/free chlorine and ozone/monochloramine. *Water Research.* **34** (17), 4121–4130.

Robertson, B., Sinclair, M. I., Forbes, A. B., Veitch, M., Kirk, M., Cunliffe, D., Willis, J. & Fairley, C. K. 2002 Case-control studies of sporadic cryptosporidiosis in Melbourne and Adelaide, Australia. *Epidemiol. Infect.* **128** (3), 419–431.

Rochelle, P. A., Upton, S. J., Montelone, B. A. & Woods, K. 2005 The response of *Cryptosporidium parvum* to UV light. *Trends Parasitol.* **21** (2), 81–87.

Ryan, U. & Power, M. 2012 *Cryptosporidium* species in Australian wildlife and domestic animals. *Parasitol.* **139** (13), 1673–1688.

Ryan, U. & Xiao, L. 2014 Taxonomy and molecular taxonomy. In: *Cryptosporidium: Parasite and Disease* (S. M. Caccio & G. Widmer, eds). Springer, New York, USA, p. 28.

Sanborn, M. & Takaro, T. 2013 Recreational water-related illness: office management and prevention. *Can. Fam. Physician.* **59** (5), 491–495.

Schets, F. M., Engels, G. B. & Evers, E. G. 2004 *Cryptosporidium* and *Giardia* in swimming pools in the Netherlands. *J. Water Health* **2** (3), 191–200.

Schets, F. M., Schijven, J. F. & de Roda Husman, A. M. 2011 Exposure assessment for swimmers in bathing waters and swimming pools. *Water Res.* **45** (7), 2392–2400.

Shah, N., Dupont, H. L. & Ramsey, D. J. 2009 Global etiology of travellers diarrhea: systematic review from 1973 to the present. *Am. J. Med. Hyg.* **80**, 609–614.

Shields, J. M., Gleim, E. R. & Beach, M. J. 2008a Prevalence of *Cryptosporidium* spp. and *Giardia intestinalis* in swimming pools, Atlanta, Georgia. *Emerg. Infect. Dis.* **14** (6), 948–950.

Shields, J. M., Hill, V. R., Arrowood, M. J. & Beach, M. J. 2008b Inactivation of *C. parvum* under chlorinated recreational water conditions. *J. Water Health* **6**, 513–520.

Shields, J. M., Arrowood, M. J., Hill, V. R. & Beach, M. J. 2009 The effect of cyanuric acid on the disinfection rate of *Cryptosporidium parvum* in 20-ppm free chlorine. *J. Water Health* **7**, 109–114.

Smith, A., Reacher, M., Smerdon, W., Adak, G. K., Nichols, G. & Chalmers, R. M. 2006 Outbreaks of waterborne infectious intestinal disease in England and Wales, 1992–2003. *Epidemiol. Infect.* **134** (6), 1141–1149.

Smith, S., Elliot, A. J., Mallaghan, C., Modha, D., Hippisley-Cox, J., Large, S., Regan, M. & Smith, G. E. 2010 Value of syndromic surveillance in monitoring a focal waterborne outbreak due to an unusual *Cryptosporidium* genotype in Northamptonshire, United Kingdom, June–July 2008. *Euro Surveill.* **15** (33), 19643.

Snel, S. J., Baker, M. G., Kamalesh, V., French, N. & Learmonth, J. 2009 A tale of two parasites: the comparative epidemiology of cryptosporidiosis and giardiasis. *Epidemiol Infect.* **137**, 1641–1650.

Soller, J. A., Bartrand, T., Ashbolt, N. J., Ravenscroft, J. & Wade, T. J. 2010 Estimating the primary etiologic agents in recreational freshwaters impacted by human sources of faecal contamination. *Water Res.* **44**, 4736–4747.

Sorvillo, F. J., Fujioka, K., Nahlen, B., Tormey, M. P., Kebabjian, R. & Mascola, L. 1992 Swimming-associated cryptosporidiosis. *Am. J. Public Health* **82** (5), 742–744.

Spiliotopoulou, A., Hansen, K. M. & Andersen, H. R. 2015 Secondary formation of disinfection by-products by UV treatment of swimming pool water. *Sci. Total Environ.* **520**, 96–105.

Stafford, R., Neville, G., Towner, C. & McCall, B. 2000 A community outbreak of *Cryptosporidium* infection associated with a swimming pool complex. *Commun. Dis. Intell.* **24** (8), 236–239.

Steenland, K. & Savitz, D. A. 1997 *Topics in Environmental Epidemiology*. Oxford University Press, Oxford, UK.

Stehr-Green, J. K., McCaig, L., Remsen, H. M., Rains, C. S., Fox, M. & Juranek, D. D. 1987 Shedding of oocysts in immunocompetent individuals infected with *Cryptosporidium*. *Am. J. Trop. Med. Hyg.* **36** (2), 338–342.

Suppes, L. M. & Reynolds, K. A. 2014 Swimmer recall on a Recreational Waterborne Illness outbreak questionnaire. *Microchemical J.* **112**, 150–152.

Suppes, L. M., Abrell, L., Dufour, A. P. & Reynolds, K. A. 2014 Assessment of swimmer behaviors on pool water ingestion. *J. Water Health* **112**, 150–152.

Suppes, L. M., Canales, R. A., Gerba, C. P. & Reynolds, K. A. 2016 *Cryptosporidium* risk from swimming pool exposures. *Int. J. Hyg. Environ. Health* **219** (8), 915–919.

Takagi, M., Toriumi, H., Endo, T., Yamamoto, N. & Kuroki, T. 2008 An outbreak of cryptosporidiosis associated with swimming pools. *Kansenshogaku Zasshi.* **82** (1), 14–19.

Tam, C. C., Rodrigues, L. C., Viviani, L., Dodds, J. P., Evans, M. R., Hunter, P. R., Gray, J. J., Letley, L. H., Rait, G., Tompkins, D. S., O'Brien, S. J.; IID2 Study Executive Committee 2012 Longitudinal study of infectious intestinal disease in the UK (IID2 study): incidence in the community and presenting to general practice. *Gut* **61** (1), 69–77.

Tesauro, M., Bianchi, A., Consonni, M., Bollani, M., Cesaria, M., Trolli, F. R. & Galli, M. G. 2010 Hygienic profile of the water in Milan swimming pools: a three-year comparative study. *Ann. Ig.* **22**, 345–355.

Turabelidze, G., Lin, M., Weiser, T. & Zhu, B. P. 2007 Communitywide outbreak of cryptosporidiosis in rural Missouri associated with attendance at child care centers. *Arch. Pediatr. Adolesc. Med.* **161**, 878–883.

Valderrama, A. L., Hlavsa, M. C., Cronquist, A., Cosgrove, S., Johnston, S. P., Roberts, J. M., Stock, M. L., Xiao, L., Xavier, K. & Beach, M. J. 2009 Multiple risk factors associated with a large statewide increase in cryptosporidiosis. *Epidemiol. Infect.* **137** (12), 1781–1788.

Waldron, L. S., Ferrari, B. C., Cheung-Kwok-Sang, C., Beggs, P. J., Stephens, N. & Power, M. L. 2011 Molecular epidemiology and spatial distribution of a waterborne cryptosporidiosis outbreak in Australia. *Appl. Environ. Microbiol.* **77** (21), 7766–7771.

Wheeler, C., Vugia, D. J., Thomas, G., Beach, M. J., Carnes, S., Maier, T., Gorman, J., Xiao, L., Arrowood, M. J., Gilliss, D. & Werner, S. B. 2007 Outbreak of cryptosporidiosis at a California waterpark: employee and patron roles and the long road towards prevention. *Epidemiol. Infect.* **135** (2), 302–310.

WHO 2000 *Guidelines for Safe Recreational-Water Environments*, Vol. 2, Swimming Pools, Spas and Similar Recreational-Water Environments. World Health Organization, Geneva, Switzerland. www.disaster-info.net/infovolcanes/pdf/eng/…/doc14599-0.pdf.

WHO 2006 *Guidelines for Safe Recreational Water Environments*, Vol. 2, Swimming Pools and Similar Recreational Water Environments. World Health Organization, Geneva, Switzerland.

WHO 2013 *Diarrhoeal Disease*. World Health Organization, Geneva, Switzerland. http://www.who.int/mediacentre/factsheets/fs330/en/.

Wilberschied, L. 1995 A swimming-pool-associated outbreak of cryptosporidiosis. *Kans. Med.* **96** (2), 67–68.

Xiao, L. 2010 Molecular epidemiology of cryptosporidiosis: an update. *Exp. Parasitol.* **124**, 80–89.

Yoder, J. S. & Beach, M. J. 2010 *Cryptosporidium* surveillance and risk factors in the United States. *Exp. Parasitol.* **124**, 31–39.

Yoder, J. S., Blackburn, B. G., Craun, G. F., Hill, V., Levy, D. A., Chen, N., Lee, S. H., Calderon, R. L. & Beach, M. J. 2004 Surveillance for waterborne-disease outbreaks associated with recreational water – United States, 2001–2002. *MMWR Surveill. Summ.* **53** (8), 1–22.

Yoder, J. S., Beach, M. J.; Centers for Disease Control and Prevention (CDC) 2007 Cryptosporidiosis surveillance – United States, 2003–2005. *MMWR Surveill. Summ.* **56** (7), 1–10.

Yoder, J., Roberts, V., Craun, G. F., Hill, V., Hicks, L. A., Alexander, N. T., Radke, V., Calderon, R. L., Hlavsa, M. C., Beach, M. J., Roy, S. L.; Centers for Disease Control and Prevention (CDC) 2008 Surveillance for waterborne disease and outbreaks associated with recreational water use and other aquatic facility-associated health events – United States, 2005–2006. *MMWR Surveill. Summ.* **57** (9), 39–62.

Yoder, J. S., Harral, C., Beach, M. J.; Centers for Disease Control and Prevention (CDC) 2010 Cryptosporidiosis surveillance – United States, 2006–2008. *MMWR Surveill. Summ.* **59** (6), 1–14.

Yoder, J. S., Wallace, R. M., Collier, S. A., Beach, M. J., Hlavsa, M. C.; Centers for Disease Control and Prevention (CDC) 2012 Cryptosporidiosis surveillance – United States, 2009–2010. *MMWR Surveill. Summ.* **61** (5), 1–12.

Yokoi, H., Tsuruta, M., Tanaka, T., Tsutake, M., Akiba, Y., Kimura, T., Tokita, Y., Akimoto, T., Mitsui, Y., Ogasawara, Y. & Ikegami, H. 2005 *Cryptosporidium* outbreak in a sports center. *Jpn. J. Infect. Dis.* **58** (5), 331–332.

Zahedi, A., Paparini, A., Jian, F., Robertson, I. & Ryan, U. 2016 Public health significance of zoonotic *Cryptosporidium* species in wildlife: critical insights into better drinking water management. *Int. J. Parasitol: Parasites and Wildlife.* **5**, 88–109.

First received 7 June 2016; accepted in revised form 26 September 2016. Available online 10 November 2016