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REVIEW ARTICLE

Viruses in recreational water-borne disease outbreaks: a review

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adenovirus, coxsackievirus, echovirus, enteroviruses, hepatitis A, lake, norovirus, outbreak, pond, recreation, river, swimming pool, virus, water-borne.

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Summary

Viruses are believed to be a significant cause of recreationally associated water-borne disease. However, they have been difficult to document because of the wide variety of illnesses that they cause and the limitations in previous detection methods. Noroviruses are believed to be the single largest cause of outbreaks, which have been documented in the published literature 45% ($n = 25$), followed by adenovirus (24%), echovirus (18%), hepatitis A virus (7%) and coxsackieviruses (5%). Just under half of the outbreaks occurred in swimming pools (49%), while the second largest outbreak occurred in lakes or ponds (40%). The number of reported outbreaks associated with noroviruses has increased significantly in recent years probably because of better methods for virus detection. Inadequate disinfection was related to 69% ($n = 18$) of swimming pool outbreaks. A lack of required reporting and nonuniform water quality and chlorination/disinfection standards continues to contribute to water-borne recreational disease outbreaks.

Introduction

Water-borne disease can be acquired during water-related recreational activities such as swimming, boating or other water sports. Many epidemiological studies conducted at both marine and freshwater bathing beaches have shown that there is a significant increase in incidence of illness, including gastrointestinal, respiratory, ear and ocular and skin or wound infection among those who engage in water-based recreational activities (Cabelli *et al.* 1979, 1982; D Alessio *et al.* 1981; Seyfried *et al.* 1985a; Craun *et al.* 2005). Several viruses including coxsackieviruses, adenoviruses, echoviruses, hepatitis A virus, astroviruses and noroviruses have been shown to cause recreational water-borne disease outbreaks (Table 1). Some studies have found an association between certain bacterial water quality indicators and rates of illness among bathers (Cabelli *et al.* 1982; Corbett *et al.* 1993). Other studies have found that even water that is only marginally polluted or meets state or local water quality requirements can be the source of outbreaks of disease or can contain enteric viruses (Cabelli *et al.* 1979, 1982; Rose *et al.* 1987). There is inconsistency among the numerous epidemiological studies as to which indicator organisms best correlate with the incidence of illness (Corbett *et al.* 1993), and some studies have found illness in the absence of indicator organisms (Foy *et al.* 1968; Hauri *et al.* 2005; Papapetropoulou and Vantarakis 1998). The lack of a consistent correlation between indicator organisms and disease may be particularly troubling with respect to viral pathogens, because bacterial indicators have been found to be unreliable indicators in the presence of virus. Studies showing the presence of human enteric viruses in recreational waters and/or a positive correlation between swimming in recreational waters and increased risk of disease have been conducted at bathing venues around the world including Sydney, Australia (Corbett *et al.* 1993), Blackpool Beach, UK (Alexander *et al.* 1992), Northern Ireland (16 sites) (Hughes *et al.* 1992), Ontario, Canada (Seyfried *et al.* 1985b), Israeli coastal beaches (Fattal *et al.* 1991), Lake Pontchartrain, New Orleans (EPA 1981) and Hong Kong coastal beaches (nine sites) (Cheung *et al.* 1990).

The Centers for Disease Control (CDC), the US Environmental Protection Agency and the Council of State and Territorial Epidemiologists have collaborated to maintain a surveillance system for recreational

Table 1 Viruses shown epidemiologically to cause recreational water-borne disease outbreaks

Virus group	Family/genus	Structure	Diameter (nm)	Associated illnesses
Adenoviruses	Adenoviridae/Mastadenovirus	Double-stranded DNA	90–100	Conjunctivitis, gastroenteritis, respiratory disease, pharyngoconjunctival fever
Coxsackieviruses	Picornaviridae/Enterovirus	RNA	27–30	Meningitis, pharyngitis, conjunctivitis, encephalitis
Echoviruses	Picornaviridae/Enterovirus	RNA	24–30	Gastroenteritis, encephalitis, meningitis
Hepatitis A virus	Picornaviridae/Hepatovirus	RNA	25–30	Hepatitis
Noroviruses	Caliciviridae/Norovirus	RNA	27–38	Gastroenteritis
Astroviruses	Astroviridae	RNA	28–30	Gastroenteritis

water-borne disease outbreaks since 1978. The CDC periodically reports the data from this surveillance system in the Morbidity and Mortality Weekly Report Surveillance Summaries (MMWRSS). Reports of water-borne disease outbreaks to the CDC are voluntary on the part of the states. For this reason, many outbreaks go unreported and are not accounted for by the surveillance system. In an effort to obtain the most information possible about recreational water-borne disease outbreaks, a literature review was conducted to include information about outbreak investigations that are not reported to the CDC. Also, because many outbreaks are not published, the popular media was searched for reliable accounts of water-borne recreational disease outbreaks.

Inclusion criteria for data set creation

Construction of the set of outbreaks was accomplished by searching Google, Medline, Pubmed, the CDC MMWRSS, Science Direct and LexisNexis databases for published journal articles and reports in the popular press. The following virus names norovirus, norwalk virus, adenovirus, echovirus, hepatitis A, hepatitis E, coxsackievirus and enterovirus were used individually in combination with the terms water, recreation, outbreak, swimming, pool, lake and river.

Inclusion criteria for outbreaks were as follows: viral disease outbreaks originating from water recreation and not linked to transmission via drinking water or contaminated food. A total of 55 outbreaks were identified for inclusion, occurring between 1951 and 2006, using these criteria. This collection of outbreaks does not represent a random sample of outbreaks, but represents only the available small fraction of outbreaks published in journals, the popular press or reported to the CDC MMWRSS. Published outbreaks represent those outbreaks that were detectable and those outbreaks where both sufficient expertize and sufficient funds to investigate were available. Consequently, outbreaks in US counties or countries with fewer resources will be underrepresented.

This paper uses the CDC MMWRSS surveillance classification scheme to rank the strength of evidence for Water Borne Disease Outbreak reports. Epidemiological data are weighed more than water quality data, where a classification I indicates that adequate epidemiological and water quality data were reported. A ranking of II indicates adequate epidemiological data and absent or inadequate water quality data. The ranking of III indicates limited epidemiological data and adequate water quality data, while IV indicates limited epidemiological data and absent or inadequate water quality data (Yoder *et al.* 2004). This system classifies water quality data as either laboratory data from the field (i.e. positive pool samples for norovirus) or adequate historical data from the pool indicating a failure in the pool's or lake's sanitation/disinfection control. Most papers reviewed for this study included historical data on the pool's sanitation/disinfection or bacterial indicators, while only two papers included information on the actual pathogen detected in the suspect recreational water source. For this reason, many of the papers reviewed are ranked as 'I', even if they lack the bacterial water quality indicators (Table 1–5).

Adenovirus outbreaks

Adenoviruses are members of the family Adenoviridae and the genus Mastadenovirus, which contains all human serotypes. They are icosahedral in shape, 80–90 nm in size and contain double-stranded DNA. Adenoviruses can cause a myriad of clinical illnesses including respiratory infections, ocular infections, enteric infections (gastroenteritis), encephalitis, pneumonia and even genitourinary infections.

Adenoviruses 40 and 41 have been shown to cause gastroenteritis specifically and are spread through the faecal–oral route. Respiratory spread via droplets or contact occurs in the case of types 3, 4 and 7, and these are the serotypes responsible for swimming pool outbreaks of conjunctivitis or pharyngoconjunctival fever (White and Fenner 1994).

The present review identified 13 published adenovirus outbreaks spanning six decades (Table 2). The first outbreak report appeared in 1953 (Cockburn 1953) and

Table 2 Adenovirus outbreaks (n = 13)

Year	Location	Source	Number Ill	Type	Rank	Reference
1951	Colorado	Pool	206	'APC'	IV	Cockburn (1953)
1954	Canada	Pool	112	'APC'	IV	Ormsby H.L. *et al.* (1955)
1955	Sweden	Lake	125	3	IV	Kjellen *et al.* (1957)
1959	Japan	Pool	124	3	IV	Kaji M. *et al.* (1961)
1960	Japan	Pool	48	3	IV	Kaji M. *et al.* (1961)
1966	Washington	Pool	26	3	IV	Foy *et al.* (1968)
1973	Kansas	Pool	44	7	III	Caldwell *et al.* (1974)
1977	Georgia	Pool	105	3	III	Martone *et al.* (1980)
1977	Georgia	Pool	72	4	III	D'Angelo L.J. *et al.* (1979)
1982	Oklahoma	Pool	77	7a	I	Turner *et al.* (1987)
1991	North Carolina	Pond	595	3	I	Moore *et al.* (1993)
1995	Greece	Pool	80		I	Papapetropoulou and Vantarakis (1998)
2000	Australia	Pool	34	3	?	Harley *et al.* (2001)

Table 3 Coxsackievirus outbreaks (n = 3) and echovirus outbreaks (n = 10)

Year	Location	Source	Number Ill	Type	Rank	Reference
Coxsackievirus outbreaks						
1972	Vermont	Lake	21	B5	IV	Hawley *et al.* (1973)
1974	France	Lake	5	A16	IV	Denis *et al.* (1974)
2004	Mexico	Ocean	21	A1	II	Begier *et al.* (2008)
Echovirus outbreaks						
1992	Ireland	Pool	46	30	I	Kee *et al.* (1994)
1997	Italy	Pool	68	30	I	Faustini A *et al.* (2006)
1998	Italy	Pool	?	30	?	Manzara S *et al.* (2002)
1999	Romania	Water body	5000	4,7 and 30	?	CDC (2000)
2000	Italy	Pool	?	30	?	Manzara S. *et al.* (2002)
2001	South Africa	Pool	90	3	I	Yeats J. *et al.* (2005)
2001	Germany	Pool	215	13 and 30	I	Hauri *et al.* (2005)
2003	Connecticut	Pool	36	9	I	Yoder *et al.* (2004)
2004	Siberia	Lake	294		II	Manenkov (2004)
2004	Mexico	Ocean	21	30	IV	Begier *et al.* (2008)

Table 4 Hepatitis A outbreaks (n = 4)

Year	Location	Source	Number Ill	Rank	Reference
1969	South Carolina	Lake	14	IV	Bryan *et al.* (1974)
1979	Hungary	Thermal pool/spa	56	IV	Solt (1994)
1989	Louisiana	Pool	20	I	Mahoney *et al.* (1992)
1997	Australia	Pool	6	IV	Tallis and Gregory (1997)

represented the first time spread of viral pharyngoconjunctival fever that was reported to have occurred from swimming. The most recent outbreak occurred in Australia in 2000 and was a primary school outbreak of pharyngoconjunctival fever caused by adenovirus type 3 (Harley *et al.* 2001) in which transmission occurred via an inadequately chlorinated swimming pool at a school camp. The other outbreaks occurred in five different countries and five different states in the United States. Most of these outbreaks occurred in swimming pools (n = 11), while two occurred in a lake or pond (Kjellén *et al.* 1957; Moore *et al.* 1993). Just over half (54%) of the outbreaks were caused by adenovirus type 3 (n = 7). The incidence of adenovirus outbreak reports has declined over the decades examined in this review. Nine of the outbreaks (69%) occurred prior to 1980, and there have only been four reported outbreaks in the decades since 1980. The largest outbreak, however, did take place in 1991 in North Carolina (Moore *et al.* 1993). This outbreak caused a reported 595 cases of adenovirus type 3 infections. The overall reported morbidity caused by all adenovirus outbreaks was 1648.

Table 5 Norovirus outbreaks (n = 25)

Year	Location	Source	Number Ill	Rank	Reference
1977	Ohio	Pool	229	III	Kappus *et al.* (1982)
1979	Michigan	Lake	49	IV	Koopman *et al.* (1982)
1987	North Dakota	Pool	48	?	Levine *et al.* (1990)
1994	England	Lake	7	I	Gray *et al.* (1997)
1996	Idaho	Lake	55	?	Levy *et al.* (1998)
1998	Ohio	Lake	30	III	Barwick *et al.* (2000)
1998	Minnesota	Lake	15	?	Yoder *et al.* (2004)
1998	Wisconsin	Lake	18	I	Barwick *et al.* (2000)
1999	Idaho	Hot springs	25	IV	Yoder *et al.* (2004)
1999	New York	Lake	168	III	Yoder *et al.* (2004)
2001	Finland	Pool	242	I	Maunula *et al.* (2004)*
2001	Minnesota	Lake	40	IV	Yoder *et al.* (2004)
2002	the Netherlands	Fountain	100	I	Hoebe *et al.* (2004)
2002	Arizona	River rafting	130	IV	Jones *et al.* (2009)
2002	Minnesota	Pool	36	II	Yoder *et al.* (2004)
2002	Minnesota	Lake	11	IV	Yoder *et al.* (2004)
2002	Wisconsin	Pool	15	IV	Yoder *et al.* (2004)
2002	Wisconsin	Lake	44	III	Yoder *et al.* (2004)
2003	Arizona	River rafting	22	IV	Jones *et al.* (2009)
2004	Oregon	Lake	150	IV	Tomlinson (2004)
2004	Vermont	Pool	53	II	Podewils *et al.* (2007)
2004	Sweden	Lake	163	I	Sartorius *et al.* (2007)
2005	Minnesota	Lake	8	IV	Yoder *et al.* (2008)
2006	Wisconsin	Pool	18	I	Yoder *et al.* (2008)
2006	Florida	Lake	50	II	Yoder *et al.* (2008)

*Astroviruses also isolated during this outbreak.

Coxsackievirus and echovirus outbreaks

Coxsackieviruses and echoviruses belong to the genus Enterovirus, which is in the family Picornaviridae. These viruses are small in size (30 nm diameter) and have a nonenveloped capsid that is icosahedral in shape. They are comprised of a linear, plus sense single stranded-RNA genome. Together with polioviruses, coxsackieviruses and echoviruses are commonly referred to collectively as, enteroviruses. Enteroviruses enter the body via ingestion and grow both in the throat and in the intestinal tract and are subsequently shed in the faeces. They can cause conjunctival, respiratory or gastrointestinal illness, but can also cause more serious diseases such as meningitis, paralysis, myocarditis or hand–foot-and-mouth diseases (White and Fenner 1994).

Three outbreaks of recreational water-borne coxsackievirus were identified (Table 3). Two of the outbreaks took place over 30 years ago and one in 2004. The first occurred in 1972 in Vermont at a boys' summer camp (Hawley *et al.* 1973) and the second occurred in France in 1974 (Denis *et al.* 1974). Both outbreaks were caused by polluted freshwater lakes where children swam. A recent outbreak was reported from 21 persons returning from a school-organized trip to Mexico. The clustered illness onset suggested a point source exposure likely from swimming in sewage-contaminated seawater (Begier *et al.* 2008).

Nine echovirus outbreaks were identified (Table 3) with the first reported outbreak in Ireland during 1992 (Kee *et al.* 1994) and the most recent associated with the above school-organized trip to Mexico (Begier *et al.* 2008). Six of the ten outbreaks (60%) were the result of contaminated swimming pools. The other four outbreaks were the result of contact with other water bodies of water, such as lake, pond, reservoir and ocean (CDC 2000; Manenkov 2004; Hauri *et al.* 2005; Begier *et al.* 2008). All nine outbreaks have occurred in the last decade and the first half of the present decade. Whether this represents more diligent reporting or improved surveillance and detection or whether it represents an actual increase in incidence is unclear, but merits investigation. The total number of reported cases associated with all coxsackievirus outbreaks is only 47; the total number of cases from all echovirus outbreaks is unknown, but is likely much >5000.

Hepatitis A and E virus outbreaks

Hepatitis A virus belongs to the family Picornaviridae and has its own genus Hepatovirus. Hepatitis A virus is a 27-nm icosahedral and single-stranded RNA virus. It

enters the body via ingestion, multiplies in the intestine and then spreads to the liver through the blood stream. The incubation period for hepatitis A virus is long – about 4 weeks with a range from 2 to 6 weeks. This makes it more difficult to isolate virus from patients when they present for treatment (White and Fenner 1994).

Four outbreaks of recreational water-borne hepatitis A were identified (Table 4). Outbreak reports have occurred at a rate of one per decade since the 1960s. The earliest report was from an outbreak in 1969 in South Carolina affecting 14 people (Bryan *et al.* 1974), and the most recent outbreak took place in Australia in 1997 and affected six people (Tallis and Gregory 1997). The relative morbidity contributed by these four outbreaks is low (96 cases) when compared to outbreaks of other aetiologies.

Although hepatitis E viruses (HEV) excreted in faeces and urine constitute a significant proportion of pathogens present in sewage (Kopecka *et al.* 1993), no reports of HEV linked to recreational water were found in the published literature. Two cases of HEV were reported in persons who swam in the River Ganges, but they also drank unboiled or unfiltered water while in India (Anon 1993; Pond 2005).

Norovirus outbreaks

Noroviruses are the most common aetiological agent of gastroenteritis in US, causing an estimated 23 million cases per year (Mead *et al.* 1999). Noroviruses are members of the family Caliciviridae and the genus Norovirus. They are 27–32 nm, single-stranded, nonenveloped RNA viruses (Heymann 2004). Noroviruses cause acute onset of projectile vomiting and diarrhoea, sometimes with low grade fever, headache and malaise (Dolin *et al.* 1972). Symptoms are usually self-limited, lasting for 24–72 h. The incubation period is usually 24–48 h, but onset of symptoms as soon as 10 h after exposure has been reported (Heymann 2004). Noroviruses are extremely contagious. Norovirus gastroenteritis causes rapid dehydration, which is of particular concern among the elderly and very young, as well as those who are engaged in physically demanding activities, which hasten dehydration during outbreaks among soldiers during deployment (Matson 2005). There are no known nonhuman reservoirs for human norovirus and no long-term immunity is gained from infection (Parrino *et al.* 1977). It is not yet possible to grow noroviruses in cell culture. Reverse transcriptase polymerase chain reaction (RT-PCR) identification of norovirus has only been possible since the early 1990s, and the test has become widely available only in the last decade.

Twenty-five recreational water-borne norovirus outbreaks were identified, occurring between 1977 and 2006 (Table 5). Outbreaks occurred in a wide variety of types of recreational water settings. Seven outbreaks (28%) were associated with swimming pools, 14 outbreaks (56%) resulted from lakes, two from rivers (8%) and one each from a recreational fountain and a hot spring. Many other water-borne or fomite-borne norovirus outbreaks occur as a result of poor sanitation and drinking water disinfection. In addition to recreational water, these outbreaks commonly occur among crowded facilities such as cruise ships (Isakbaeva *et al.* 2005), emergency shelters (Yee *et al.* 2007) or other crowded and unhygienic areas.

Because there are no cell culture techniques available, outbreak investigators used clinical diagnosis with nucleic acid amplification techniques for norovirus, or other similar viruses. In 1993, the United States the Centers for Disease Control adopted RT-PCR for routine testing. Since then, public health laboratories around the United States have adopted this testing method. Consequently, norovirus outbreaks were not frequently reported until the late 1990s, although many outbreaks may have probably occurred and gone undetected prior to the availability of the rapid, easy and specific test. At the same time, there has been a marked increase in the incidence of norovirus outbreaks in recent years (CDC 2003), and this may be an actual increase not entirely explained by increased testing (Fig. 3).

Viral outbreaks *vs* bacterial/protozoan outbreaks

The published literature would suggest that the majority of disease burden from recreational water-borne disease results from infections that are bacterial or protozoan. Several of the protozoan pathogens responsible for the highest disease burden are quite resistant to disinfection by chlorination. The relative numbers of viral *vs* bacterial/parasitic recreational water-borne gastrointestinal disease outbreaks were reported to CDC, and the per cent of these outbreaks that are of viral aetiology for the years from 1989 to 2002 are shown in Table 6. From 1998 to 2002 (the most recent year for which data are available), viral outbreaks have been reported every year. Also, the highest contribution (44%) of viral outbreaks occurred in 2002. It seems that viral outbreaks are beginning to comprise more of the total recreational water-borne outbreaks reported to the CDC, which is most likely the result of simplification of testing methods, such as the advent of the use of PCR for detection of environmental pathogens, but may also represent, at least partially, a true increase in incidence because of changes in viral epidemiology or changing host behavioural patterns, such as increased participation in highdensity recreations like travel on cruise ships, participation in package vacations at all-inclusive resorts and the increasing popularity of water parks.

Table 6 Relative numbers of viral *vs* bacterial/parasitic recreational water-borne gastrointestinal disease* outbreaks reported to CDC, 1989–2002

Year	Viral gastroenteritis	Bacterial/protozoal gastroenteritis†	Viral (%)
1989	1	9	11
1990	0	12	0
1991	1	9	11
1992	0	2	0
1993	0	8	0
1994	0	6	0
1995	0	9	0
1996	1	8	12
1997	0	3	0
1998	2	10	20
1999	2	11	18
2000	1	16	6
2001	1	9	11
2002	4	9	44

*Excludes acute gastrointestinal illness of unknown aetiology.
†Nondermatologic and excluding *Naegleria fowleri*, Pontiac fever and Legionnaire's disease.

Outbreaks by aetiologic agent

Forty-six per cent ($n = 25$) of all outbreaks were caused by noroviruses (Fig. 1). This represents the largest proportion of outbreaks by aetiology. The second most prevalent type of infection was adenovirus, which caused 24% (13). Ten outbreaks, or 18%, were caused by echoviruses. Hepatitis A virus was responsible for four outbreaks (7%), and coxsackieviruses caused three (5%).

Outbreaks by type of recreational water

Half of the outbreaks occurred in swimming pools ($n = 27$), while the second largest percentage, 40%, occurred in lakes or ponds ($n = 22$). Two outbreaks were reported following exposure to river water, and two occurred after exposure to hot springs. Two additional outbreaks were the result of contaminated recreational fountains, designed for children to play in (Fig. 2). Outbreaks in swimming pools are more likely to be reported, because they represent a limited defined population; these outbreaks may also be more likely recognized among groups such as swim teams where members involved know each other. Ponds and lakes may be larger in volume and are not disinfected. Because most of the outbreaks occurred in countries above the 40° latitude, most investigations in this review can be indicated as summer outbreaks. Individuals in cold climates are less likely to swim in pools, and especially outdoor lakes during the wintertime. White *et al.* (1994) present a dataset showing the occurrence of enteroviruses and adenoviruses unique to the summer or spring season. All other viruses discussed are either not mentioned or not specific to any season.

The role of inadequate disinfection

For the purposes of this review, inadequate disinfection is defined as absent or insufficient free chlorine (<1 ppm), chlorination or disinfection equipment failure, or inadequate method of disinfection such as improper use of hydrogen peroxide and/or UV light used in swimming pools. Only outbreaks taking place in swimming pools or other typically disinfected venues were included in this

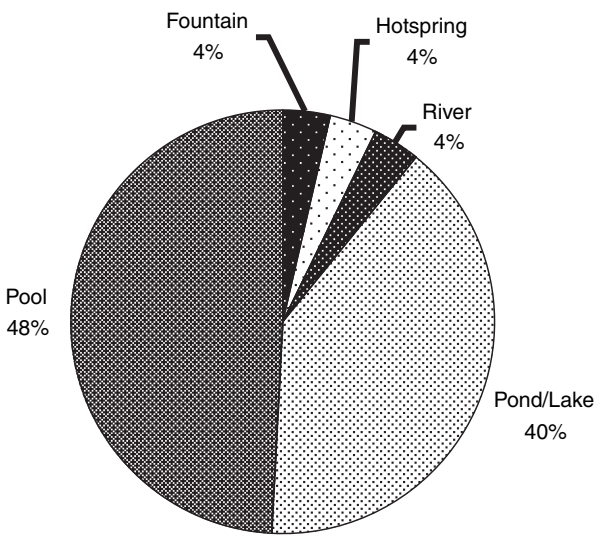


Figure 1 Outbreaks by an aetiologic agent, $n = 55$.

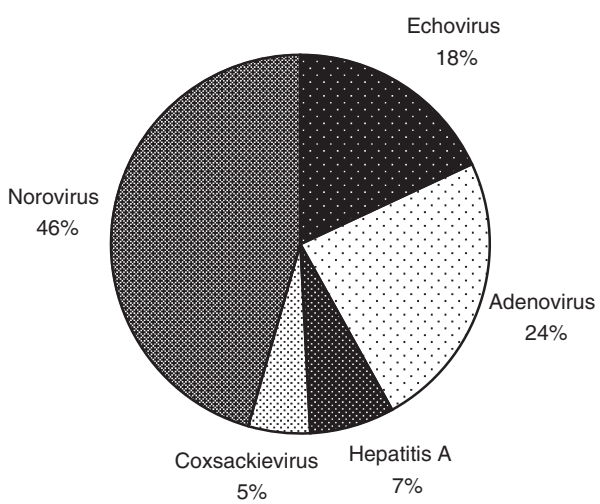


Figure 2 Outbreaks by type of recreational water, $n = 55$.

portion of the analysis; lakes, rivers and ponds were excluded. Inadequate disinfection was found in 69% ($n = 18$) of outbreak investigations, indicating that inadequate disinfection is still a major factor in swimming pool outbreaks. Either seven outbreak reports (27%) did not include information about the level of disinfection of the water or this information was unknown to the investigators. Only one report could be found in which an outbreak occurred despite adequate disinfection. This was a case of an overwhelming volume of inoculum entering a swimming pool (Kee *et al.* 1994). An outbreak of echovirus type 30 began when two children vomited into a crowded swimming pool. Despite hourly chlorine level readings all within the recommended limits, an outbreak ensued after the vomiting incidents. These findings reiterate that proper disinfection is important in controlling viral outbreaks in swimming pools, although many swimming pool outbreaks are caused by other organisms that may be more disinfection resistant, such as *Cryptosporidium* spp. There is evidence that norovirus may be more resistant to chlorine disinfection than other enteric viruses (Barker *et al.* 2004). Of the seven outbreaks of norovirus resulting from swimming pool exposure, five reports (71%) indicated inadequate chlorination (Kappus *et al.* 1982; Levine *et al.* 1990; Maunula *et al.* 2004; Blevins *et al.* 2004), and two reports did not contain information regarding disinfection. Thus, adequate chlorination can be inferred to be an important component in the prevention of norovirus outbreaks originating from swimming pools.

In 2003, the CDC reported surveillance data from swimming pool inspections in five different states, Pennsylvania, Florida, California, Minnesota, and Wyoming. Local environmental health programmes in these areas inspect public and semipublic pools periodically to determine compliance with health regulations. A total of 22 131 pool inspections from these locations were combined to form one data set containing information about code violations. From these inspections, 21 561 violations of pool codes were noted. Water chemistry accounted for 38·7% of violations, and filtration and recirculation system violations represented 38·6% of the total. It was found that the highest percentage of total violations attributable to pH infractions occurred in child wading pools where 8% had coincident free chlorine and pH violations (CDC 2003). Wading pools may host babies in diapers and because of the small volume of water in wading pools there is a smaller ratio of chlorinated water to occupant mass. It has been demonstrated that swimmers themselves have a negative influence on the bacterial and chemical quality of bathing waters (Hanes and Fossa 1970; Gerba 2000), and that occupant load is a factor level of water quality where the water supply is finite or has limited opportunity for exchange (Fattal *et al.* 1991).

Children and recreational water-borne disease

Higher rates of illness among children swimmers than adult swimmers have been evidenced repeatedly (Seyfried *et al.* 1985; Fattal *et al.* 1991). In this review, 51% of outbreaks were reported to primarily affect children ($n = 28$) *vs* 24% of outbreaks that affected all ages ($n = 13$) and 25% where the age distribution was unknown or not reported ($n = 14$). Children are disproportionately affected by water-borne recreational outbreaks. This is likely because of immunological, physiological and behavioural factors (Parkin *et al.* 2002). Children are more susceptible to infection because of their naive immune systems. For the same reason, they also experience more severe symptoms than adults. Physiological differences between children and adults also leave children more vulnerable to some forms of infection (Parkin *et al.* 2002). Also because of their small size, children are more vulnerable to rapid dehydration caused by vomiting and diarrhoea, making them more likely to have poor prognosis gastroenteritis infections. Behavioural aspects also put children at increased risk of infection (White and Fenner 1994). Children may also be less likely than adults to bathe after swimming or even to wash their hands between swimming and eating. Children are also more likely to put their heads underwater or ingest recreational water (Alexander *et al.* 1992).

The role of bacterial indicators of water quality

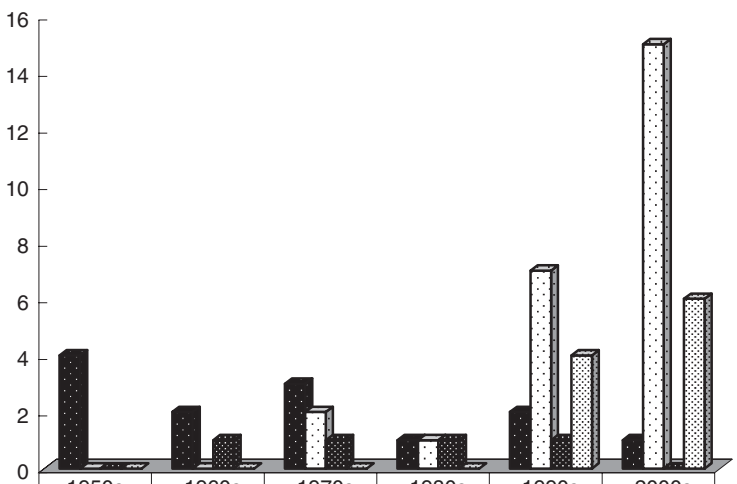
Of the 55 outbreak reports included in this review, 57% ($n = 31$) did not contain information about bacterial indicators of faecal contamination in the source water. Of the remaining outbreaks, 33% ($n = 18$) were positive for bacterial indicators of faecal contamination in the source water, while the rest (10%, $n = 6$) were negative. However, in three outbreaks, disease occurred even in the absence of bacterial indicators of faecal contamination. Foy *et al.* (1968) investigated an outbreak of adenovirus type 3 associated with intermittent chlorination of a swimming pool in 1968. No coliform bacteria could be detected in the pool water in tests performed on three consecutive days, despite the absence of detectable chlorine residual. During the investigation of an adenovirus outbreak at a municipal swimming pool in Greece in 1995, Papapetropoulou and Vantarakis (1998) found that they could detect adenovirus in the pool water, using PCR with primers specific for the detection of adenoviruses, but all bacteriological indicators tested, total coliforms, faecal coliforms and faecal streptococci were

negative. During a community-wide outbreak of echovirus in the city of Kassel, Germany in 2001, investigators found that bathing in a particular pond, pond A, was associated with illness. However, weekly testing of pond water for total coliforms, faecal coliforms, enterococci and *Staphylococcus aureus* never indicated levels above the limit suggested by the European Union bathing water guideline (Hauri *et al.* 2005). Rose *et al.* (1987) reported that enterovirus and rotavirus could be readily isolated from the recreational waters of Oak Creek, Arizona, despite the faecal indicators within the range of regulatory limits (200 faecal coliform per 100 ml). The authors demonstrated that 18 of 41 recreational water samples were positive for either enterovirus or rotavirus. This study illustrates that faecal coliforms do not always adequately reflect viral pollution of recreational water.

One outbreak of echovirus infection associated with an outdoor swimming pool in Ireland (Kee *et al.* 1994) was excluded from this analysis, because the source of the outbreak was known to be nonfaecal as two children were observed vomiting into the pool at the advent of the outbreak. Although bacteriological indicators of faecal contamination do not always correlate with viral contamination, they seem to be an important indicator of water quality in bathing venues and were only absent in the presence of viral outbreaks in three of 18 (16·7%) outbreaks where testing was conducted. Over half of outbreak reports did not include information about bacterial indicators. Whether this is because bacterial indicators were not tested for or because the information was not included in the report is unknown.

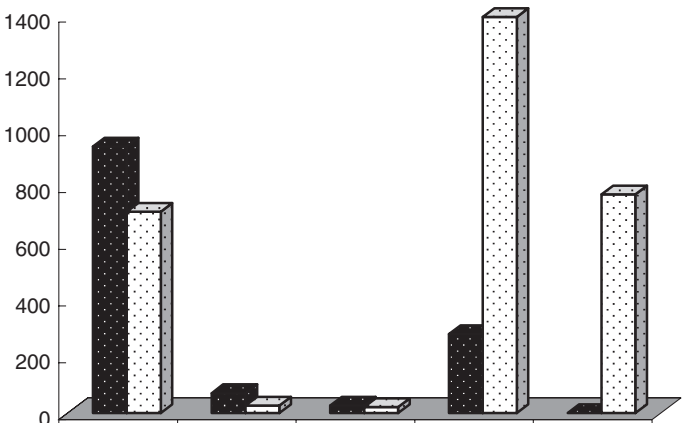
Trends in reported viral recreational water-borne disease outbreaks

Figure 3 shows outbreaks over time by aetiological agent for 52 outbreak reports. The three coxsackievirus outbreaks were excluded from this analysis, because the number of outbreaks was too low to show a trend. There has been a change in the aetiologies of reported outbreaks over the last five decades. From the 1950s through the 1960s, the majority of outbreaks reported were adenovirus. In fact, only one other outbreak was reported to have occurred during this time period, an outbreak of hepatitis A among boy scouts (Bryan *et al.* 1974). There have only been three outbreaks of adenovirus reported since 1990, and the remaining ten outbreaks were reported prior to 1990. At the same time, reports of norovirus infection have been increasing. Since 1990, there have been 22 documented recreational water-borne norovirus outbreaks, while only three were reported prior to 1990. Testing for noroviruses became possible in the 1970s, and since then reports of norovirus outbreaks have been increasing. In the 1990s and the current decade, norovirus and echovirus outbreaks have been reported more than other aetiologies. Norovirus outbreaks were reported more than any other viral agent since 2000 (n = 15). There are probably numerous reasons for these changes, some of which reflect actual changes in disease incidence over time as new illnesses emerge, and some of which reflect reporting biases, changes and improvements in our ability to detect viruses in the environment, and trends in public health



	1950s	1960s	1970s	1980s	1990s	2000s
Adenovirus	4	2	3	1	2	1
Norovirus	0	0	2	1	7	15
HepA Virus	0	1	1	1	1	0
Echovirus	0	0	0	0	4	6

Figure 3 Outbreaks over time by an aetiologic agent, n = 52*. *The three coxsackievirus outbreaks were excluded from this analysis.



	Adenovirus	Hep A	Coxsackievirus	Norovirus	Echovirus
Morbidity >20 years ago	939	70	26	278	0
Morbidity, last 20 years	709	26	21	1395	770

Figure 4 Morbidity by an aetiology; last 20 years *vs* >20 years ago, $n = 55$*. *The large echovirus outbreak in Romania in 1999 was excluded on the basis that it was not comparable with the other outbreaks, as it was not a focal outbreak, but an ongoing statewide outbreak that became endemic in nature.

interests that influence attention to and reporting of certain diseases over others.

Changes in the total morbidity contributed by each type of virus during the last 20 years compared to >20 years ago are displayed in Fig. 4. Fifty-five outbreaks were included in this analysis. The large echovirus outbreak in Romania in 1999 was excluded on the basis that it was not comparable with the other outbreaks, as it was not a focal outbreak, but an ongoing statewide outbreak that became endemic in nature. In this analysis, morbidity was assessed as total number of cases reported for each aetiology during the designated period. Adenovirus and norovirus have contributed the most morbidity with 1648 and 1673 cases respectively reported. While adenovirus morbidity has decreased over the last 20 years, both norovirus and echovirus have contributed more morbidity in the last 20 years than in prior years.

Conclusions and research needs

Epidemiological studies have shown that swimmers have higher rates of illness than nonswimmers, especially gastrointestinal, respiratory and eye–ear–nose symptoms. Children have the highest rate of gastrointestinal illness and a greater overall increased risk of illness, when compared to adults. Some epidemiological studies have shown that there is a correlation between level of contamination of recreational water and increased rates of disease among swimmers (Cabelli *et al.* 1982). The present review has supported the observations and conclusions drawn from epidemiological studies. In the outbreaks reviewed, children were the primarily affected population in over half (54%). Also, disinfection was an important factor in outbreak causation in swimming pools; 68% of outbreak reports reviewed included mention of inadequate chlorination or other disinfection in swimming pools. Disinfection failure because of equipment malfunction was found to have caused some outbreaks (Caldwell *et al.* 1974; Martone *et al.* 1980; Turner *et al.* 1987; Papapetropoulou *et al.* 1998; Harley *et al.* 2001; Yoder *et al.* 2004). Additionally, several outbreak investigations showed that head immersion is an important factor in increased risk of illness, indicating that there is a behavioural component to disease transmission among swimmers (Seyfried *et al.* 1985; Parkin *et al.* 2002). A marked increase in the number of echovirus outbreaks reported was noted to have occurred over approximately the last 10 years. Whether this is the result of increased detection and reporting or an actual increase in incidence of echovirus is unclear and warrants further investigation. Seventy per cent of these outbreaks occurred in swimming pools, suggesting that assessment of current disinfection guidelines and their efficacy for echovirus may be needed.

Nonuniform water quality and chlorination/disinfection standards may contribute to water-borne recreational disease outbreaks, because regulations are determined by localities and may not be sufficient to control the spread of disease. The lack of required reporting for recreational water-borne disease outbreaks makes surveillance efforts more difficult, and may bias or obscure observations made from those outbreaks that are reported. Training of pool maintenance personnel may also play an important role in reducing outbreaks, because it may reduce inadequate disinfection because of operator error and equipment malfunction. Castor and Beach (2004) have recently

made several recommendations for the prevention and control of disease transmission in swimming venues. They recommend the redesign of aquatic facilities, increased governmental oversight of swimming pool maintenance and training of staff and education of the public regarding healthy swimming habits. Additionally, they recommend that high risk groups, such as the elderly and infirm and pregnant women, should be made aware of their increased risk of illness in swimming, even in apparently adequately disinfected swimming waters. This review of recreational water-borne viral disease outbreaks supports those recommendations.

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