CHAPTER 3

Microbial hazards

A variety of microorganisms can be found in swimming pools and similar recreational water environments, which may be introduced in a number of ways.

In many cases, the risk of illness or infection has been linked to faecal contamination of the water. The faecal contamination may be due to faeces released by bathers or a contaminated source water or, in outdoor pools, may be the result of direct animal contamination (e.g. from birds and rodents). Faecal matter is introduced into the water when a person has an accidental faecal release – AFR (through the release of formed stool or diarrhoea into the water) or residual faecal material on swimmers' bodies is washed into the pool (CDC, 2001a). Many of the outbreaks related to swimming pools would have been prevented or reduced if the pool had been well managed.

Non-faecal human shedding (e.g. from vomit, mucus, saliva or skin) in the swimming pool or similar recreational water environments is a potential source of pathogenic organisms. Infected users can directly contaminate pool or hot tub waters and the surfaces of objects or materials at a facility with pathogens (notably viruses or fungi), which may lead to skin infections in other patrons who come in contact with the contaminated water or surfaces. 'Opportunistic pathogens' (notably bacteria) can also be shed from users and transmitted via surfaces and contaminated water.

Some bacteria, most notably non-faecally-derived bacteria (see Section 3.4), may accumulate in biofilms and present an infection hazard. In addition, certain free-living aquatic bacteria and amoebae can grow in pool, natural spa or hot tub waters, in pool or hot tub components or facilities (including heating, ventilation and air-conditioning [HVAC] systems) or on other wet surfaces within the facility to a point at which some of them may cause a variety of respiratory, dermal or central nervous system infections or diseases. Outdoor pools may also be subject to microorganisms derived directly from pets and wildlife.

This chapter describes illness and infection associated with microbial contamination of swimming pools, natural spas and hot tubs. The sections reflect the origin of the microbial contaminant, as illustrated in Figure 3.1. In each case, a short subsection on risk assessment and risk management is given, although general management strategies for managing air and water quality are described in detail in Chapter 5.

In most cases, monitoring for potential microbial hazards is done using indicator microorganisms (rather than specific microbial pathogens), which are easy to enumerate and would be expected to be present in greater numbers than pathogens. The traditional role of indicator parameters was to show the presence or absence of faecal pollution in water supplies. The criteria associated with microbial indicators of pollution are outlined in Box 3.1 and further discussed in WHO (2004). The use of these microorganisms in monitoring water quality is covered in Chapter 5.

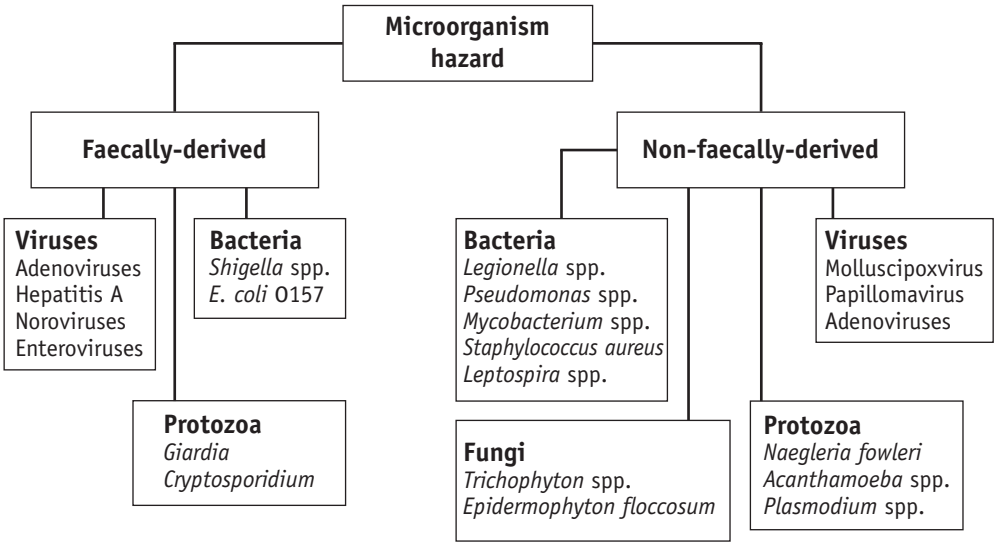

Figure 3.1. Potential microbial hazards in pools and similar environments

BOX 3.1 CRITERIA FOR INDICATOR ORGANISMS AND THEIR APPLICATION TO POOLS AND SIMILAR ENVIRONMENTS

- The indicator should be absent in unpolluted environments and present when the source of pathogenic microorganisms of concern is present (e.g. faecal material).
- The indicator should not multiply in the environment.
- The indicator should be present in greater numbers than the pathogenic microorganisms.
- The indicator should respond to natural environmental conditions and water treatment processes in a manner similar to the pathogens of concern.
- The indicator should be easy to isolate, identify and enumerate.
- Indicator tests should be inexpensive, thereby permitting numerous samples to be taken (if appropriate).

Microorganisms that are used to assess the microbial quality of swimming pool and similar environments include heterotrophic plate count – HPC (a general measure of non-specific microbial levels), faecal indicators (such as thermotolerant coliforms, *E. coli*), *Pseudomonas aeruginosa*, *Staphylococcus aureus* and *Legionella* spp. HPC, thermotolerant coliforms and *E. coli* are indicators in the strict sense of the definition.

As health risks in pools and similar environments may be faecal or non-faecal in origin, both faecal indicators and non-faecally-derived microorganisms (e.g. *P. aeruginosa*, *S. aureus* and *Legionella* spp.) should be examined. Faecal indicators are used to monitor for the possible presence of faecal contamination; HPC, *Pseudomonas aeruginosa* and *Legionella* spp. can be used to examine growth, and *Staphylococcus aureus* can be used to determine non-faecal shedding. The absence of these organisms, however, does not guarantee safety, as some pathogens are more resistant to treatment than the indicators, and there is no perfect indicator organism.

3.1 Faecally-derived viruses

3.1.1 Hazard identification

The viruses that have been linked to swimming pool outbreaks are shown in Table 3.1. Viruses cannot multiply in water, and therefore their presence must be a consequence of pollution. Some adenoviruses may also be shed from eyes and the throat and are responsible for swimming pool conjunctivitis.

Viruses of six types (rotavirus, norovirus, adenovirus, astrovirus, enterovirus and hepatitis A virus) are all shed following infection. Clinical data show that rotaviruses are by far the most prevalent cause of viral gastroenteritis in children, and noroviruses cause the most cases of viral diarrhoea in adults. However, few waterborne pool outbreaks have been associated with these agents. Although outbreaks are highlighted, it should be kept in mind that non-outbreak disease is likely to occur and that virus-associated pool or hot tub outbreaks are very uncommon. Even when outbreaks are detected, the evidence linking the outbreak to the pool is generally circumstantial. In the outbreaks summarized in Table 3.1, the etiological agents were detected in the water in only two cases (D'Angelo et al., 1979; Papapetropoulou & Vantarakis, 1998).

3.1.2 Outbreaks of viral illness associated with pools

1. Adenovirus-related outbreaks

There are over 50 types of adenoviruses (Hunter, 1997), and while some may cause enteric infections and are therefore shed in faeces, they are also associated with respiratory and ocular symptoms and non-faecally-derived transmission. Types 40 and 41 cause gastroenteritis in young children, but there is no documented association with waterborne transmission.

Foy et al. (1968) reported an outbreak of pharyngo-conjunctival fever caused by adenovirus type 3. The infection occurred in two children's swimming teams after exposure to unchlorinated swimming pool water. The attack rates in the two teams were 65% and 67%, respectively. The main symptoms were fever, pharyngitis and conjunctivitis. The virus could not be isolated from the pool water. The authors speculated that faecal contamination of the unchlorinated swimming pool water could have been the source of the contamination.

Caldwell et al. (1974) reported an outbreak of conjunctivitis associated with adenovirus type 7 in seven members of a community swimming team. The main symptoms were associated with the eyes. An investigation of the pool-related facilities suggested that the school swimming pool was the source of the infection, as both the pool chlorinator and pool filter had failed. The outbreak was brought under control by raising the pool's free residual chlorine level above 0.3 mg/l.

Adenovirus type 4 was the causative agent of a swimming pool-related outbreak of pharyngo-conjunctivitis reported by D'Angelo et al. (1979). A total of 72 cases were identified. Adenovirus type 4 was isolated from 20 of 26 swab specimens. The virus was also detected in samples of pool water. An investigation showed that inadequate levels of chlorine had been added to the pool water, resulting in no free chlorine in pool water samples. Adequate chlorination and closing the pool for the summer stopped the outbreak of illness.

Table 3.1. Summary of waterborne disease outbreaks associated with pools due to faecally-excreted viruses

Etiological agent	Source of agent	Disinfection/ treatment	Reference
Adenovirus 3	Possible faecal contamination	None	Foy et al., 1968
Adenovirus 7	Unknown	Improper chlorination	Caldwell et al., 1974
Adenovirus 4	Unknown	Inadequate chlorine level	D'Angelo et al., 1979
Adenovirus 3	Unknown	Pool filter system defect, failed chlorinator	Martone et al., 1980
Adenovirus 7a	Unknown	Malfunctioning chlorinator	Turner et al., 1987
Adenoviruses	Unknown	Inadequate chlorination	Papapetropoulou & Vantarakis, 1998
Adenovirus 3	Unknown	Inadequate chlorination and pool maintenance	Harley et al., 2001
Hepatitis A	Accidental faecal release suspected	None	Solt et al., 1994
	Cross-connection to sewer line	Operating properly	Mahoney et al., 1992
Norovirus	Unknown	Chlorinator disconnected	Kappus et al., 1982
	Probably via public toilets	Manual chlorination three times a week	Maunula et al., 2004
	No details available	No details available	Yoder et al., 2004
	No details available	No details available	Yoder et al., 2004
	Possible faecal contamination	Chlorination failure	CDC, 2004
Echovirus 30	Vomit	Operating properly	Kee et al., 1994

A second outbreak in the same locality and year was linked to adenovirus type 3 and swimming activity (Martone et al., 1980). Based on surveys, at least 105 cases were identified. The illness was characterized by sore throat, fever, headache and anorexia. Conjunctivitis affected only 34 of the individuals. Use of a swimming pool was linked to the illness. The outbreak coincided with a temporary defect in the pool filter system and probably improper maintenance of chlorine levels. The authors suspected that the level of free chlorine in the pool water was less than 0.4 mg/l. They also pointed out that while the virus was probably transmitted through water, person-to-person transmission could not be ruled out.

In 1987, an outbreak of adenovirus type 7a infection was associated with a swimming pool (Turner et al., 1987). Seventy-seven individuals were identified with the symptoms of pharyngitis (inflammation of the pharynx). A telephone survey indicated that persons who swam at the community swimming pool were more likely to be ill than those who did not. Swimmers who reported swallowing water were more

likely to be ill than those who did not. Further investigation showed that the pool chlorinator had reportedly malfunctioned during the period when the outbreak occurred. The outbreak ceased when proper chlorination was reinstated.

An outbreak of pharyngo-conjunctivitis caused by adenoviruses occurred among swimmers participating in a competition. Over 80 people were found to be suffering from symptoms. Adenoviruses were identified in swimming pool samples using nested polymerase chain reaction, and poor chlorination (residual chlorine levels <0.2 mg/l) was considered to have contributed to the outbreak (Papapetropoulou & Vantarakis, 1998).

In 2000, an outbreak of illness related to adenovirus type 3 was detected. It was found that there was a strong association between the presence of symptoms and swimming at a school camp. Although adenoviruses were not isolated from the pool water, inspection of the pool revealed that it was poorly maintained and inadequately chlorinated (Harley et al., 2001).

2. Hepatitis A-related outbreaks

Solt et al. (1994) reported an outbreak in Hungary in which 31 children were hospitalized following hepatitis A infection. Investigation of potential common sources eliminated food, drink and person-to-person transmission. All of the patients had reported swimming at a summer camp swimming pool. Further investigation discovered 25 additional cases. All of the cases were males between the ages of 5 and 17 years. The pool, which was not chlorinated, was half full of water for a period and was used by younger children. The pool was generally overcrowded during the month of August. It was concluded that the crowded conditions and generally poor hygienic conditions contributed to the outbreak.

An outbreak of hepatitis A in several states in the USA during 1989, which may have been associated with a public swimming pool, was reported by Mahoney et al. (1992). Twenty of 822 campers developed hepatitis A infections. Case–control studies indicated that swimmers or those who used a specific hot tub were more likely than controls to become ill. It was hypothesized that a cross-connection between a sewage line and the pool water intake line may have been responsible for the outbreak or that one of the swimmers may have contaminated the water. The disinfectant levels in the pools met local standards.

3. Norovirus-related outbreaks

Few outbreaks of norovirus-related disease (previously known as Norwalk virus or Norwalk-like viruses) associated with swimming pools have been reported. Kappus et al. (1982) reported an outbreak of norovirus gastroenteritis associated with a swimming pool that affected 103 individuals. The illness typically lasted 24 h and was characterized by vomiting and cramping. Serological studies suggested that norovirus was the cause of the gastroenteritis among the swimmers. Case–control studies indicated that swimmers were more likely than non-swimmers to become ill. Similarly, the attack rate was significantly higher in swimmers who had swallowed water than in those who had not. The pool chlorinator had not been reconnected before the outbreak, which occurred at the beginning of the swimming season. The source of the virus was not found.

Maunula et al. (2004) reported an outbreak of gastroenteritis associated with norovirus contracted from a wading pool in Helsinki, Finland. Norovirus and astrovirus were isolated from water samples taken from the pool. The pool was heavily used

during the summer months (with as many as 500 bathers a day) and was manually chlorinated three times a week. There was no routine monitoring of free chlorine. It is believed that the pool had been heavily contaminated with human faecal material, with the contamination apparently being carried from the public toilets, situated very close to the pool, which were found to be grossly contaminated (although a number of nappies were also found at the bottom of the pool during the cleaning operation). The pool was emptied and cleaned and subsequently fitted with continuous filtration and chlorination.

Yoder et al. (2004) reported two outbreaks of norovirus infection that were associated with swimming pools in the USA between 2000 and 2002, one of which was associated with a hotel pool and hot tub, but gave no other details.

CDC (2004) reported an outbreak of gastroenteritis in children, whose only common exposure was attendance at a swimming club the previous weekend. Fifty-three people reported illness, and norovirus was isolated from a number of cases. An undetected accidental faecal release was suspected, and poor pool water quality monitoring and maintenance contributed to the outbreak.

4. Enterovirus-related outbreaks

Enteroviruses include polioviruses, echoviruses and coxsackieviruses types A and B. The only documented case of enterovirus infection following pool exposure was associated with echovirus, as reported by Kee et al. (1994). Thirty-three bathers had symptoms of vomiting, diarrhoea and headache shortly after swimming in an outdoor swimming pool. The outbreak is believed to have been caused by a bather who swam while ill and vomited into the pool. Individuals who had swallowed water were more likely to become ill than those who had not. Echovirus 30 was isolated from the case who had vomited and from six other cases. Proper disinfectant levels had been maintained at the pool, but they were inadequate to contain the risk of infection from vomit in the pool water.

3.1.3 Risk assessment

Determination of polluted pool water as the unequivocal cause of a viral disease outbreak requires the detection of the virus in a water sample. This is clearly not a routine procedure, but is something that is done in response to a suspected disease outbreak. Concentration techniques for viruses in water are available (e.g. SCA, 1995 and reviewed by Wyn-Jones & Sellwood, 2001), which may be adapted to pool water samples. Some agents (e.g. enteroviruses) may be detected in cell culture, but most (e.g. adenoviruses 40 and 41 and noroviruses) require molecular detection methods. If the virus has remained in contact with water containing free disinfectant for some time, then detection of infectious virus may not be possible.

Enteric viruses occur in high numbers in the faeces of infected individuals. Hepatitis A virus has been found at densities of 10^{10} per gram (Coulepis et al., 1980), and noroviruses have been estimated at 10^{11} per gram, although echoviruses may reach only 10^{6} per gram. Given the high densities at which some viruses can be shed by infected individuals, it is not surprising that accidental faecal releases into swimming pools and hot tubs can lead to high attack rates in pools where outbreaks occur, especially if the faecal release is undetected or detected but not responded to adequately.

1. *Adenoviruses*

   Most adenoviruses can be grown in commonly available cell cultures, with the exception of types 40 and 41, which may be detected by molecular biological techniques, principally by the polymerase chain reaction – PCR (Kidd et al., 1996). Types 40 and 41 are those usually associated with gastroenteritis. Other types, though more usually associated with infections of the eyelids and/or throat (pharyngo-conjunctival fever), may also be shed in the faeces for extended periods (Fox et al., 1969). The attack rate for swimming pool outbreaks linked to adenovirus serotypes is moderately high, ranging from 18% to 52% (Martone et al., 1980; Turner et al., 1987).

2. *Hepatitis A virus*

   Culture of hepatitis A virus is generally impractical, and detection relies on molecular methods (reverse transcriptase polymerase chain reaction – RT-PCR). The virus is transmitted by the faecal–oral route, with water and sewage being a frequent source of infection. The disease has an incubation period of 15–50 days, anorexia, nausea, vomiting and often jaundice being the common symptoms. Virus is shed before the onset of symptoms. The attack rate in one outbreak of illness associated with a swimming pool ranged from 1.2% to 6.1% in swimmers less than 18 years of age (Mahoney et al., 1992).

3. *Noroviruses*

   Environmental detection of these agents is restricted to RT-PCR since there is no cell culture system available. Symptoms occur within 48 h of exposure and include diarrhoea, vomiting, nausea and fever. Virus shedding, as detected by electron microscopy, stops soon after onset of symptoms, but is detectable by RT-PCR for up to five days. Attack rates are generally very high; Kappus et al. (1982), for example, reported an attack rate of 71% for those swimmers who had swallowed water.

4. *Enteroviruses*

   Coxsackieviruses are frequently found in polluted waters, and vaccine poliovirus is also found where there is a high percentage of individuals immunized (although no investigations have been reported where this has been found in pool water). Echoviruses are found less often. None of the enteroviruses commonly cause gastroenteritis in the absence of other disease, and, although they are associated with a wide variety of symptoms, most infections are asymptomatic.

3.1.4 Risk management

The control of viruses in swimming pool water and similar environments is usually accomplished by proper treatment, including the application of disinfectants. Episodes of gross contamination of a swimming pool due to an accidental faecal release or vomit from an infected person cannot be effectively controlled by normal disinfectant levels. The only approach to maintaining public health protection under conditions of an accidental faecal release or vomit is to prevent the use of the pool until the contaminants are inactivated (see Chapter 5). The education of parents/caregivers of small children and other water users about good hygienic behaviour at swimming pools is another approach that may prove to be useful for improving health safety at

swimming pools and the reduction of accidental faecal releases. It is recommended that people with gastroenteritis should not use public or semi-public pools and hot tubs while ill or for at least a week after their illness, in order to avoid transmitting the illness to other pool or hot tub users.

3.2 Faecally-derived bacteria

3.2.1 Hazard identification

Shigella species and *Escherichia coli* O157 are two related bacteria that have been linked to outbreaks of illness associated with swimming in pools or similar environments. *Shigella* has been responsible for outbreaks related to artificial ponds and other small bodies of water where water movement has been very limited. The lack of water movement means that these water bodies behave very much as if they were swimming pools, except that chlorination or other forms of disinfection are not being used. Similar non-pool outbreaks have been described for *E. coli* O157, although there have also been two outbreaks reported where the source was a children's paddling pool. These outbreaks are summarized in Table 3.2, as they illustrate the potential risk that might be experienced in swimming pools under similar conditions, although only the pool specific outbreaks are covered in detail.

3.2.2 Outbreaks of bacterial illness associated with pools

1. *Shigella-related outbreaks*

An outbreak of shigellosis associated with a fill-and-drain wading pool (filled on a daily basis with potable water) was reported from Iowa, USA (CDC, 2001b). The pool, which had a maximum depth of 35 cm, was frequented by very young and non-toilet-trained children. The pool had neither recirculation nor disinfection. One pool sample was found to contain thermotolerant coliforms and *E. coli*. Sixty-nine people were considered to be infected with shigellosis, of which 26 cases were laboratory confirmed as *S. sonnei*. It is thought that the transmission of shigellosis over several days may have been a result of residual contaminated water present after draining and people with diarrhoea visiting the pool on subsequent days.

2. *E. coli O157-related outbreaks*

In 1992, an outbreak of *E. coli* O157 infection was epidemiologically and clinically linked to a collapsible children's paddling pool (Brewster et al., 1994). Six cases of diarrhoea, including one case of haemolytic uraemic syndrome, and one asymptomatic case were identified. *E. coli* O157 phage type 59 was isolated from the six cases. The pool had not been drained or disinfected over the three-day period surrounding the outbreak. It was believed that the pool had been initially contaminated by a child known to have diarrhoea.

In 1993, six children with haemorrhagic colitis, three of whom developed haemolytic uraemic syndrome, were epidemiologically linked to a disinfected public paddling pool (Hildebrand et al., 1996). *E. coli* O157 phage type 2 was isolated from faecal specimens of five cases. *E. coli* (but not *E. coli* O157) was detected in the pool during the investigation. Free chlorine levels in the pool were less than 1 mg/l at the time of sampling.

Table 3.2. Summary of outbreaks of disease associated with pools due to faecally-excreted bacteria

Etiological agent	Source of agent	Disinfection/ treatment	Reference
Shigella spp.	AFR	None	Sorvillo et al., 1988
	Not known	None	Makintubee et al., 1987
	AFR	None	Blostein, 1991
	Likely AFR	None	CDC, 2001b
E. coli O157	AFR	None	Keene et al., 1994
	AFR	Not known	Brewster et al., 1994
	AFR	Inadequate treatment	Hildebrand et al., 1996
	Not known	None	CDC, 1996
	Not known	None	Cransberg et al., 1996

AFR – Accidental faecal release

3.2.3 Risk assessment

Shigella species are small, non-motile, Gram-negative, facultatively anaerobic rods. They ferment glucose but not lactose, with the production of acid but not gas. Symptoms associated with shigellosis include diarrhoea, fever and nausea. The incubation period for shigellosis is 1–3 days. The infection usually lasts for 4–7 days and is self-limiting.

E. coli O157 are small, motile, non-spore-forming, Gram-negative, facultatively anaerobic rods. They ferment glucose and lactose. Unlike most *E. coli*, *E. coli* O157 does not produce glucuronidase, nor does it grow well at 44.5 °C. *E. coli* O157 causes non-bloody diarrhoea, which can progress to bloody diarrhoea and haemolytic uraemic syndrome. Other symptoms include vomiting and fever in more severe cases. The usual incubation period is 3–4 days, but longer periods are not uncommon. In most instances, the illness typically resolves itself in about a week. About 5–10% of individuals develop haemolytic uraemic syndrome following an *E. coli* O157 infection. Haemolytic uraemic syndrome, characterized by haemolytic anaemia and acute renal failure, occurs most frequently in infants, young children and elderly people.

Individuals infected with *E. coli* O157 shed these bacteria at similar or slightly higher densities than the non-enterohaemorrhagic *Shigella*. Literature reports indicate that *E. coli* O157 is known to be shed at densities as high as 10^8 per gram. *Shigella* species are shed at similar but somewhat lower levels by individuals who have contracted shigellosis (Table 3.3).

Table 3.3. Bacterial exposure factors

Agent	Density shed during infection	Duration of shedding	Infective dose	Reference
Shigella	10^6 per gram	30 days	$<5 \times 10^2/ID_{50}$	Makintubee et al., 1987; DuPont, 1988
Escherichia coli O157	10^8 per gram	7–13 days	Not known[a]	Pai et al., 1984

ID_{50} – dose of microorganisms required to infect 50% of individuals exposed
[a] Probably similar to *Shigella*

The infective dose for *Shigella* species is usually between 10 and 100 organisms (Table 3.3). Lower doses, however, may cause illness in infants, the elderly or immunocompromised individuals. The infective dose for *E. coli* O157 is unknown but is likely to be similar to that for *Shigella* species. Keene et al. (1994) suggested that the infective dose is very low, based on experience in an outbreak.

3.2.4 Risk management

One of the primary risk management interventions is to reduce accidental faecal release occurrence in the first place – for example, by educating pool users. *E. coli* O157 and *Shigella* species are readily controlled by chlorine and other disinfectants under ideal conditions. However, if an accidental faecal release has occurred in a swimming pool or hot tub, it is likely that these organisms will not be instantly eliminated, and other steps will have to be taken to provide time for disinfectant effect, such as evacuation of the pool (see Chapter 5).

3.3 Faecally-derived protozoa

3.3.1 Hazard identification

Giardia and particularly *Cryptosporidium* spp. are faecally-derived protozoa that have been linked to outbreaks of illness in swimming pools and similar environments. These two organisms are similar in a number of respects. They have a cyst or oocyst form that is highly resistant to both environmental stress and disinfectants, they have a low infective dose and they are shed in high densities by infected individuals. There have been a number of outbreaks of disease attributed to these pathogens, as summarized in Table 3.4.

3.3.2 Outbreaks of protozoan illness associated with pools

1. *Giardia*-related outbreaks

Giardiasis has been associated with swimming pools and water slides. In 1994, a case–control study was conducted in the United Kingdom to determine the risk factors for giardiasis. Giardiasis cases were identified from disease surveillance reports over a one-year period (Gray et al., 1994). Seventy-four cases and 108 matched controls were identified. Analysis of the data indicated that swimming appeared to be an independent risk factor for giardiasis. Other recreational exposures and ingestion of potentially contaminated water were found to be not significantly related to giardiasis.

In 1984, a case of giardiasis was reported in a child who had participated in an infant and toddler swim class in Washington State, USA (Harter et al., 1984). The identification of this case of giardiasis led to a stool survey of 70 of the class participants. The stool survey revealed a 61% prevalence of *Giardia* infection. None of the non-swimming playmates was positive. Eight of 23 children (35%) exposed only at a better maintained pool to which the classes had been moved four weeks prior to the survey were positive. The investigators did not find any evidence of transmission to non-swim-class pool users. Adequate chlorine levels were maintained in the pool. Contamination of the pool was thought to be due to an undetected accidental faecal release.

Table 3.4. Summary of disease outbreaks associated with pools due to faecally-derived protozoa

Etiological agent	Source of agent	Disinfection/ treatment	Reference
Giardia	AFR	Inadequate treatment	Harter et al., 1984
	AFR	Inadequate treatment	Porter et al., 1988
	AFR	Adequate treatment	Greensmith et al., 1988
Cryptosporidium	AFR	Adequate treatment	CDC, 1990
	Sewage intrusion	Plumbing defects	Joce et al., 1991
	AFR	Not known	Bell et al., 1993
	Sewage intrusion	Not known	McAnulty et al., 1994
	Not known	Not known	CDC, 1994
	AFR	Adequate treatment	Hunt et al., 1994
	AFR	Adequate treatment	CDSC, 1995
	Likely AFR	Adequate treatment	Sundkist et al., 1997
	AFR	Faulty ozone generator	CDSC, 1997
	Not known	Plumbing and treatment defects	CDSC, 1998
	Not known	Adequate treatment	CDSC, 1999
	Likely AFR	Treatment problems	CDSC, 1999
	Suspected AFR	Adequate treatment	CDSC, 2000
	Likely AFR	Inadequate treatment	CDSC, 2000
	Not known	Adequate treatment	CDSC, 2000
	Not known	Adequate treatment	CDSC, 2000
	Not known	Not known	CDSC, 2000
	Not known	Ozonation problems	CDSC, 2000
	AFR	Not known	CDC, 2001c
	Not known	Not known	Galmes et al., 2003

AFR – accidental faecal release
Adequate treatment – in terms of indicator bacteria monitoring results

In the autumn of 1985, an outbreak of giardiasis occurred among several swimming groups at an indoor pool in north-east New Jersey, USA (Porter et al., 1988). Nine clinical cases were identified, eight of whom had *Giardia*-positive stool specimens. All were female, seven were adults (>18 years), and two were children. A 39% attack rate was observed for the group of women who had exposure on one day. These cases had no direct contact with children or other risk factors for acquiring *Giardia*. Infection most likely occurred following ingestion of swimming pool water contaminated with *Giardia* cysts. The source of *Giardia* contamination was a child who had a faecal accident in the pool, who was a member of the group that swam the same day as the women's swimming group. A stool survey of the child's group showed that of 20 people tested, 8 others were positive for *Giardia*. Pool records showed that no chlorine measurements had been taken on the day of the accidental faecal release and that no free chlorine level was detectable on the following day.

In 1988, an outbreak of giardiasis was associated with a hotel's new water slide pool (Greensmith et al., 1988). Among 107 hotel guests and visitors surveyed, 29 probable and 30 laboratory-confirmed cases of *Giardia* infection were found. Cases ranged

from 3 to 58 years of age. Symptoms in the 59 cases included diarrhoea, cramps, foul-smelling stools, loss of appetite, fatigue, vomiting and weight loss. Significant associations were found between illness and staying at the hotel, using the water slide pool and swallowing pool water. A possible contributing factor was the proximity of a toddlers' pool, a potential source of faecal material, to the water slide pool. Water in the slide pool was treated by sand filtration and bromine disinfection.

2. *Cryptosporidium*-related outbreaks

A number of outbreaks of cryptosporidiosis have been linked to swimming pools. The sources of *Cryptosporidium* contaminating the pools were believed to be either sewage or the swimmers themselves. A number of outbreaks are reviewed below.

In 1988, an outbreak of 60 cases of cryptosporidiosis was reported in Los Angeles County, USA (CDC, 1990). Swimmers were exposed to pool water in which there had been a single accidental faecal release. The attack rate was about 73%. The common factor linking infected individuals was use of the swimming pool.

In August 1988, the first outbreak of cryptosporidiosis associated with a swimming pool in the United Kingdom was recognized following an increase in the number of cases of cryptosporidiosis that had been identified by the Doncaster Royal Infirmary microbiology laboratory (Joce et al., 1991). By October of that year, 67 cases had been reported. An investigation implicated one of two pools at a local sports centre. Oocysts were identified in the pool water. Inspection of the pool pipework revealed significant plumbing defects, which had allowed ingress of sewage from the main sewer into the circulating pool water. The epidemiological investigation confirmed an association between head immersion and illness. The concentration of oocysts detected in the pool water samples that were tested was 50 oocysts per litre. Difficulty had been experienced in controlling the level of free chlorine residual, which implied that disinfection was probably not maintained at an appropriate level.

An outbreak of cryptosporidiosis occurred in British Columbia, Canada, in 1990 (Bell et al., 1993). A case–control study and illness survey indicated that the transmission occurred in a public children's pool at the local recreation centre. Analysis using laboratory-confirmed cases showed that the illnesses were associated with swimming in the children's pool within two weeks before onset of illness. Attack rates ranged from 8% to 78% for various groups of children's pool users. Several accidental faecal releases, including diarrhoea, had occurred in the pool before and during the outbreak.

In 1992, public health officials in Oregon, USA, noted a large increase in the number of stool specimens submitted for parasitic examination that were positive for *Cryptosporidium* (McAnulty et al., 1994). They identified 55 patients with cryptosporidiosis, including 37 who were the first individuals ill in their households. A case–control study involving the first 18 case patients showed no association between illness and day-care attendance, drinking municipal drinking-water or drinking untreated surface waters. However, 9 of 18 case patients reported swimming at the local wave pool, whereas none of the controls indicated this activity. Seventeen case patients were finally identified as swimming in the same pool. The investigators concluded that the outbreak of cryptosporidiosis was probably caused by exposure to faecally contaminated pool water.

In August 1993, a parent informed the Department of Public Health of Madison, Wisconsin, USA, that her daughter was ill with a laboratory-confirmed *Cryptosporidium* infection and that members of her daughter's swim team had severe diarrhoea

(CDC, 1994). Fifty-five per cent of 31 pool users interviewed reported having had watery diarrhoea for two or more days. Forty-seven per cent of the 17 cases had had watery diarrhoea for more than five days. A second cluster of nine cases was identified later in the month. Seven of the nine reported swimming at a large outdoor pool. Public health authorities cleaned the pool, shock dosed with chlorine and prohibited people with diarrhoea from swimming in the pool.

In the UK, 18 outbreaks of cryptosporidiosis were associated with pools between 1989 and 1999. Recognized accidental faecal releases at the pool occurred in four of the outbreaks, although faecal contamination was known or suspected in a further five outbreaks. Outbreaks were associated with pools disinfected with chlorine and with ozone and with both well and poorly managed pools (PHLS, 2000).

Two protracted outbreaks of cryptosporidiosis associated with swimming pools were reported from Ohio and Nebraska, USA (CDC, 2001c). In both cases, accidental faecal releases (on more than one occasion) were observed. In the Nebraska outbreak, 32% of cases reported swimming during their illness or shortly afterwards.

In Australia, a statewide outbreak of cryptosporidiosis in New South Wales was associated with swimming at public pools (Puech et al., 2001). The association was reported to be stronger for cases from urban areas. The authors noted that *Cryptosporidium* oocysts were more commonly detected from pools where at least two notified cases had swum, and that outbreaks could involve multiple pools.

A large outbreak of cryptosporidiosis has been associated with a hotel in Majorca, Spain, used by British tourists. The outbreak was detected in Scotland, following the detection of cryptosporidiosis in tourists returning from Majorca. Almost 400 cases were identified, and the outbreak was thought to be associated with the hotel swimming pool, with oocysts being detected in samples of the pool water (Galmes et al., 2003). This outbreak resulted in guidelines on cryptosporidiosis prevention being produced for the Spanish hoteliers association (Confederation Española de Hoteles y Apartamentos Turisticos) and the UK Federation of Tour Operators (R. Cartwright, pers. comm.).

In the USA, an analysis of recreationally-associated waterborne outbreaks of illness between 2001 and 2002 was conducted (Yoder et al., 2004). *Cryptosporidium* species were the most common cause of gastrointestinal outbreaks of illness associated with treated swimming pool water.

3.3.3 Risk assessment

Giardia cysts are 4–12 µm in diameter. Viable cysts that are ingested by humans have an incubation period of about 7–12 days. The resulting gastroenteritis is characterized by diarrhoea with accompanying abdominal cramps. The illness lasts for about 7–10 days. *Cryptosporidium* oocysts are 4–6 µm in diameter and are much more resistant to chlorine than *Giardia* cysts. If viable oocysts are ingested, there is an incubation period of 4–9 days before symptoms appear. The illness lasts about 10–14 days, with symptoms typically including diarrhoea, vomiting and abdominal cramps. In patients with severely weakened immune systems, such as those with HIV infection and cancer and transplant patients taking certain immune system-suppressing drugs, cryptosporidiosis is generally chronic and more severe than in immunocompetent people and causes diarrhoea that can last long enough to be life threatening (Petersen, 1992).

The *Cryptosporidium* infective dose that affects 50% of the challenged population of humans is about 132 oocysts (DuPont et al., 1995), although this does depend upon the strain (Okhuysen et al., 1999), and for some strains fewer than 100 oocysts can lead to infection. The duration of shedding of these oocysts after infection is 1–2 weeks. The infection is self-limiting in most individuals, lasting 1–3 weeks. *Cryptosporidium* oocysts discharged by ill individuals are usually observed at densities of 10^6–10^7 per gram. The infective dose of *Giardia* that will cause gastroenteritis in 25% of an exposed population is 25 cysts. *Giardia* cysts discharged in the faeces of infected individuals are usually at densities of 3×10^6 per gram. The shedding of cysts can persist for up to six months (Table 3.5).

Table 3.5. Protozoan exposure factors

Agent	Density shed during infection[a]	Duration of shedding	Infective dose	Reference
Cryptosporidium	10^6–10^7 per gram	1–2 weeks	$132/ID_{50}$	Casemore, 1990; DuPont et al., 1995
Giardia	3×10^6 per gram	6 months	$25/ID_{25}$	Rendtorff, 1954; Feachem et al., 1983

ID_{50} (ID_{25}) – dose of microorganisms required to infect 50% (25%) of individuals exposed
[a] Figures represent the peak and are not representative of the whole of the infection period

3.3.4 Risk management

Giardia cysts and *Cryptosporidium* oocysts are very resistant to many disinfectants, including chlorine (Lykins et al., 1990). *Cryptosporidium*, for example (the more chlorine resistant of the two protozoa), requires chlorine concentrations of 30 mg/l for 240 min (at pH 7 and a temperature of 25 °C) for a 99% reduction to be achieved (i.e. an impractical level). Inactivation of oocysts with chlorine is greater when ozone, chlorine dioxide or UV irradiation is also used (Gregory, 2002). Ozone is a more effective disinfectant (compared with chlorine) for the inactivation of *Giardia* cysts and *Cryptosporidium* oocysts. *Cryptosporidium* oocysts are sensitive to 5 mg of ozone per litre. Almost all (99.9%) of the oocysts are killed after 1 min (at pH 7 and a temperature of 25 °C). Giardia cysts are sensitive to 0.6 mg of ozone per litre. Ninety per cent of the cysts are inactivated after 1 min (at pH 7 and a temperature of 5 °C). As ozone is not a residual disinfectant (i.e. it is not applied so as to persist in pool water in use), sufficient concentration and time for inactivation must be ensured during treatment before residual ozone removal and return to the pool.

It should be noted, however, that the figures above represent removal under laboratory (i.e. ideal) conditions. Additionally, studies have generally used oxidant demand-free water (i.e. they were not performed in simulated recreational water where additional organic material is present). Carpenter et al. (1999) found that the presence of faecal material increased the Ct value (disinfectant concentration in mg/l multiplied by time in minutes) needed to disinfect swimming pools.

UV is also effective at inactivating *Giardia* cysts and *Cryptosporidium* oocysts. A near complete inactivation (99.9%) of *Cryptosporidium* occurs at UV exposures of 10 mJ/cm^2

(WHO, 2004). Inactivation of *Giardia* cysts (99%) occurs at lower UV intensities of 5 mJ/cm^2 (WHO, 2004). The efficacy of UV is impacted by particulate matter and the growth of biofilms. Thus, turbidity should be low, and UV lamps need to be cleaned periodically to remove biofilms or other substances that interfere with UV light emission. Like ozone, UV leaves no disinfectant residual and thus should be combined with chlorine or another disinfectant that remains in the water after treatment (WHO, 2004).

At present, the most practical approach to eliminating cysts and oocysts is through the use of filtration. *Cryptosporidium* oocysts are removed by filtration where the porosity of the filter is less than 4 µm. *Giardia* cysts are somewhat larger and are removed by filters with a porosity of 7 µm or less, although statistics on removal efficiency during filtration should be interpreted with caution. Removal and inactivation of cysts and oocysts occur only in the fraction of water passing through treatment. Since a pool is a mixed and not a plug flow system, the rate of reduction in concentration in the pool volume is slow.

Most outbreaks of giardiasis and cryptosporidiosis among pool swimmers have been linked to pools contaminated by sewage, accidental faecal releases or suspected accidental faecal releases. A study conducted in six pools in France, in the absence of detected faecal releases, found only a single instance when *Cryptosporidium* oocysts were detected (Fournier et al., 2002). An Italian investigation of 10 chlorinated swimming pools found *Cryptosporidium* and *Giardia* in 3% of pool water samples despite otherwise good water quality (according to microbial monitoring results) and free chlorine levels of approximately 1 mg/l. In addition, both *Cryptosporidium* and *Giardia* were always detected in the filter backwash water (Bonadonna et al., 2004). Pool maintenance and appropriate disinfection levels are easily overwhelmed by accidental faecal releases or sewage intrusion; therefore, the only possible response to this condition, once it has occurred, is to prevent use of the pool and physically remove the oocysts by draining or by applying a long period of filtration, as inactivation in the water volume (i.e. disinfection) is impossible (see Chapter 5). However, the best intervention is to prevent accidental faecal releases from occurring in the first place, through education of pool users about appropriate hygienic behaviour. Immunocompromised individuals should be aware that they are at increased risk of illness from exposure to pathogenic protozoa.

3.4 Non-faecally-derived bacteria

Infections and diseases associated with non-enteric pathogenic bacteria found in swimming pools and similar recreational water environments are summarized in Table 3.6. A number of these bacteria may be shed by bathers or may be present in biofilms (assemblages of surface-associated microbial cells enclosed in an extracellular matrix – Donlan, 2002). Biofilms may form on the lining of pipes (for example) in contact with water and may serve to protect the bacteria from disinfectants.

3.4.1 Legionella spp.

1. Risk assessment

Legionella are Gram-negative, non-spore-forming, motile, aerobic bacilli, which may be free-living or living within amoebae and other protozoa or within biofilms. *Legionella* spp. are heterotrophic bacteria found in a wide range of water environments and can proliferate at temperatures above 25 °C. They may be present in high numbers in natural spas using thermal spring water, and they can also grow in poorly main-

Table 3.6. Non-faecally-derived bacteria found in swimming pools and similar environments and their associated infections

Organism	Infection/disease	Source
Legionella spp.	Legionellosis (Pontiac fever and Legionnaires' disease)	Aerosols from natural spas, hot tubs and HVAC systems Poorly maintained showers or heated water systems
Pseudomonas aeruginosa	Folliculitis (hot tubs) Swimmer's ear (pools)	Bather shedding in pool and hot tub waters and on wet surfaces around pools and hot tubs
Mycobacterium spp.	Swimming pool granuloma Hypersensitivity pneumonitis	Bather shedding on wet surfaces around pools and hot tubs Aerosols from hot tubs and HVAC systems
Staphylococcus aureus	Skin, wound and ear infections	Bather shedding in pool water
Leptospira spp.	Haemorrhagic jaundice Aseptic meningitis	Pool water contaminated with urine from infected animals

HVAC – heating, ventilation and air conditioning

tained hot tubs, associated equipment and HVAC systems. *Legionella* spp. can also multiply on filter materials, namely granular activated carbon. However, exposure to *Legionella* is preventable through the implementation of basic management measures, including filtration, maintaining a continuous disinfectant residual in hot tubs (where disinfectants are not used, there must be a high dilution rate with fresh water) and the maintenance and physical cleaning of all natural spa, hot tub and pool equipment, including associated pipes and air-conditioning units.

The risk of infection following exposure to *Legionella* is difficult to assess and remains a matter of some debate (Atlas, 1999). Due to its prevalence in both natural and artificial environments, it must be considered that people are frequently exposed (at least to low numbers). Generally, there is no reaction to such exposure, asymptomatic production of antibodies or development of a mild flu-like illness, which may not be attributed to *Legionella* infection.

Legionella spp. can cause legionellosis, a range of pneumonic and non-pneumonic disease (WHO, 2005). Ninety per cent of cases of legionellosis are caused by *L. pneumophila*. Legionnaires' disease is characterized as a form of pneumonia. General risk factors for the illness include gender (males are roughly three times more likely than females to contract Legionnaires' disease), age (50 or older), chronic lung disease, cigarette smoking and excess consumption of alcohol. Specific risk factors, in relation to pools and hot tubs, include frequency of hot tub use and length of time spent in or around hot tubs. Although the attack rate is often less than 1%, mortality among hospitalized cases ranges widely up to 50%. Pontiac fever is a non-pneumonic, non-transmissible, non-fatal, influenza-like illness. The attack rate can be as high as 95% in the total exposed population. Patients with no underlying illness or condition recover in 2–5 days without treatment.

Risk of legionellosis from pools and similar environments is associated with proliferation of *Legionella* in spas or hot tubs, associated equipment and HVAC systems. The inference to be drawn from reported outbreaks and documented single cases is that inhalation of bacteria, or aspiration following ingestion, during natural spa or hot tub use may lead to disease, although Leoni et al. (2001) concluded that showers may present a greater risk of legionellosis than pool water. Thermal spring waters, especially, may be a source of high numbers of *Legionella* spp. (Bornstein et al., 1989; Martinelli et al., 2001), and they have been implicated in cases of legionnaires' disease (Bornstein et al., 1989; Mashiba et al., 1993).

Piped drinking-water distribution systems, household hot and cold water maintained between 25 °C and 50 °C, cooling towers, evaporative condensers of air-conditioning devices, water fountains and mist-generating machines are also potential sources of exposure to *Legionella*.

2. Risk management

Control of *Legionella* follows similar general principles to water safety plans applied to drinking-water supplies (WHO, 2004), although, in this instance, the principal responsibility will not lie with the water supplier. Authorities responsible for regulation of recreational facilities should ensure the implementation of safety plans, and such plans should address not only pools and hot tubs but also other water systems, including cooling towers and evaporative condensers operating at these facilities. As safety plans are limited to the recreational facility and the dose response is not easily described, adequate control measures should be defined in terms of practices that have been shown to be effective. Important control measures include appropriate design, to minimize the available surface area within the pool and hot tub system and associated pipework to reduce the area for possible bacterial colonization, ensuring an adequate disinfection residual in pools and hot tubs, proper maintenance and cleaning of equipment, and adequate ventilation.

Most of the reported legionellosis associated with recreational water use has been associated with hot tubs and natural spas (Groothuis et al., 1985; Althaus, 1986; Bornstein et al., 1989; Mashiba et al., 1993). Natural spa waters (especially thermal water) and associated equipment create an ideal habitat (warm, nutrient-containing aerobic water) for the selection and proliferation of *Legionella*. Hot tubs used for display in retail/wholesale outlets are also potential sources of infection (McEvoy et al., 2000). Outbreaks as a result of using swimming pools have not been reported (Marston et al., 1994), although *Legionella* spp. have been isolated from pool water and filter samples (Jeppesen et al., 2000; Leoni et al., 2001). Hot tubs integrated into larger swimming pool complexes appear to be less of a source of *Legionella* infection where shared water treatment facilities exist due to dilution of hot tub water into larger volumes of water for treatment.

Increased risk of *Legionella* in drinking-water has been associated with systems operating within the temperature range 25–50 °C. In hot tub facilities it is impractical to maintain a water temperature outside this range. Therefore, it is necessary to implement a range of other management strategies, which may include:

- ensuring a constant circulation of water in the hot tub;
- programming 'rest periods' during hot tub operation, in order to discourage excessive use and also to allow disinfectant levels to 'recover';
- frequent inspection and cleaning of all filters, including backwash filters (e.g. at least daily and when triggered by a pressure drop);

- cleaning pool surroundings, inspection of the physical conditions of the hot tub (e.g. daily);
- replacing at least half the water in each hot tub (e.g. daily);
- completely draining hot tubs and thoroughly cleaning all surfaces and all pipework (e.g. weekly);
- maintaining and physically cleaning heating, ventilation and air-conditioning systems serving the room in which hot tubs are located (e.g. weekly to monthly);
- inspection of the sand filter (e.g. quarterly); and
- ensuring staff are appropriately qualified and competent to operate the recreational facility.

In order to control the growth of *Legionella* in hot tubs and natural spas, physical cleaning of surfaces is critical, and high residual disinfectant concentrations may be required – e.g. free chlorine, where used, must be at least 1 mg/l at all times. Features such as water sprays, etc., in pool facilities should be periodically cleaned and flushed with a level of disinfectant adequate to eliminate *Legionella* spp. (e.g. by use of a solution of at least 5 mg of hypochlorite per litre).

Bathers should be encouraged to shower before entering the water. This will remove pollutants such as perspiration, cosmetics and organic debris that can act as a source of nutrients for bacterial growth and neutralize oxidizing biocides. Bather density and duration spent in hot tubs should also be controlled. Public and semi-public spa facilities should have programmed rest periods during the day. High-risk individuals (such as those with chronic lung disease) should be cautioned about the risks of exposure to *Legionella* in or around pools and hot tubs.

Operators of hot tub facilities should undertake a programme of verification of control measures, including:

- checking and adjusting residual disinfectant levels and pH (several times a day);
- inspection and maintenance of cleaning operations (daily to weekly); and
- where microbial testing for *Legionella* is undertaken, ensuring that *Legionella* levels are <1/100 ml.

3.4.2 *Pseudomonas aeruginosa*

1. *Risk assessment*

Pseudomonas aeruginosa is an aerobic, non-spore-forming, motile, Gram-negative, straight or slightly curved rod with dimensions 0.5–1 µm × 1.5–4 µm. It can metabolize a variety of organic compounds and is resistant to a wide range of antibiotics and disinfectants.

P. aeruginosa is ubiquitous in water, vegetation and soil. Although shedding from infected humans is the predominant source of *P. aeruginosa* in pools and hot tubs (Jacobson, 1985), the surrounding environment can be a source of contamination. The warm, moist environment on decks, drains, benches and floors provided by pools and similar environments is ideal for the growth of *Pseudomonas*, and it can grow well up to temperatures of 41 °C (Price & Ahearn, 1988). *Pseudomonas* tends to accumulate in biofilms in filters that are poorly maintained and in areas where pool hydraulics are poor (under moveable floors, for example). It is also likely that bathers pick up the organisms on their feet and hands and transfer them to the water. It has been proposed that the high water temperatures and turbulence in aerated hot tubs promote perspiration and desquamation (removal of skin cells). These materials protect

organisms from exposure to disinfectants and contribute to the organic load, which, in turn, reduces the residual disinfectant level; they also act as a source of nutrients for the growth of *P. aeruginosa* (Kush & Hoadley, 1980; Ratnam et al., 1986; Price & Ahearn, 1988).

In one study, *P. aeruginosa* was isolated from all nine hot tubs examined (seven of which were commercial facilities and two domestic – Price & Ahearn, 1988). In the majority of hot tubs, concentrations ranged from 10^2 to 10^5 per ml. Locally recommended disinfection levels (of between 3 and 5 mg/l chlorine or bromine) were not maintained in any of the commercial hot tubs examined. In the same study, the two domestic hot tubs developed *P. aeruginosa* densities of 10^4–10^6 per ml within 24–48 h following stoppage of disinfection. In Northern Ireland, UK, Moore et al. (2002) found *P. aeruginosa* in 72% of hot tubs and 38% of swimming pools examined.

In hot tubs, the primary health effect associated with the presence of *P. aeruginosa* is folliculitis. Otitis externa and infections of the urinary tract, respiratory tract, wounds and cornea caused by *P. aeruginosa* have also been linked to hot tub use. Infection of hair follicles in the skin with *P. aeruginosa* produces a pustular rash, which may appear under surfaces covered with swimwear or may be more intense in these areas (Ratnam et al., 1986). The rash appears 48 h (range 8 h to 5 days) after exposure and usually resolves spontaneously within 5 days. It has been suggested that warm water supersaturates the epidermis, dilates dermal pores and facilitates their invasion by *P. aeruginosa* (Ratnam et al., 1986). There are some indications that extracellular enzymes produced by *P. aeruginosa* may damage skin and contribute to the bacteria's colonization (Highsmith et al., 1985). Other symptoms, such as headache, muscular aches, burning eyes and fever, have been reported. Some of these secondary symptoms resemble humidifier fever (Weissman & Schuyler, 1991) and therefore could be caused by the inhalation of *P. aeruginosa* endotoxins. Investigations have indicated that duration or frequency of exposure, bather loads, bather age and using the facility later in the day can be significant risk factors for folliculitis (Hudson et al., 1985; Ratnam et al., 1986; CDC, 2000). The sex of bathers does not appear to be a significant risk factor, but Hudson et al. (1985) suggest that women wearing one-piece bathing suits may be more susceptible to infection, presumably because one-piece suits trap more *P. aeruginosa*-contaminated water next to the skin. It has been suggested that the infective dose for healthy individuals is greater than 1000 organisms per ml (Price & Ahearn, 1988; Dadswell, 1997).

In swimming pools, the primary health effect associated with *P. aeruginosa* is otitis externa or swimmer's ear, although folliculitis has also been reported (Ratnam et al., 1986). Otitis externa is characterized by inflammation, swelling, redness and pain in the external auditory canal. Risk factors reported to increase the occurrence of otitis externa related to water exposure include amount of time spent in the water prior to the infection, less than 19 years of age and a history of previous ear infections (Seyfried & Cook, 1984; van Asperen et al., 1995). Repeated exposure to water is thought to remove the protective wax coating of the external ear canal, predisposing it to infection.

An indoor swimming pool with a system of water sprays has been implicated as the source of two sequential outbreaks of granulomatous pneumonitis among lifeguards (Rose et al., 1998). Inadequate chlorination led to the colonization of the spray circuits and pumps with Gram-negative bacteria, predominantly *P. aeruginosa*.

The bacteria and associated endotoxins were aerosolized and respired by the lifeguards when the sprays were activated. When the water spray circuits were replaced and supplied with an ozonation and chlorination system, there were no further occurrences of disease among personnel.

An outbreak of pseudomonas hot-foot syndrome, erythematous plantar nodules, has been reported as a result of exposure to a community wading pool. The floor of the pool was coated in abrasive grit, and the water contained high concentrations of *P. aeruginosa* (Fiorillo et al., 2001). Another outbreak occurred in Germany due to high concentrations of *P. aeruginosa* on the stairs to a water slide and resulted in some of the children being admitted to hospital (A. Wiedenmann, pers. comm.).

The true incidence of illnesses associated with *P. aeruginosa* in pools and similar environments is difficult to determine. Since the symptoms are primarily mild and self-limiting, most patients do not seek medical attention. In the USA, Yoder et al. (2004) reported 20 outbreaks of dermatitis between 2000 and 2001 associated with pools and hot tubs. In eight of these outbreaks *P. aeruginosa* was identified from water or filter samples; in the other 12 outbreaks *Pseudomonas* was suspected to be the cause. It was noted that contributing factors to these outbreaks included inadequate pool and hot tub maintenance and exceeding the bather load limit.

2. *Risk management*

Maintaining adequate residual disinfectant levels and routine cleaning are the key elements to controlling *P. aeruginosa* in swimming pools and similar recreational environments (see Chapter 5). While maintaining residual disinfectant levels in pools is relatively easy, the design and operation of some hot tubs make it difficult to achieve adequate disinfectant levels in these facilities. Under normal operating conditions, disinfectants can quickly dissipate (Highsmith et al., 1985; Price & Ahearn, 1988). In all facilities, frequent monitoring and adjustment of pH and disinfectant levels are essential. Most hot tubs use either chlorine- or bromine-based disinfectants. Shaw (1984) showed that chlorination was superior to bromine in controlling *P. aeruginosa*. He reported that during an outbreak investigation, *P. aeruginosa* could be isolated from water despite having a total bromine level of 5 mg/l and a pH of 7.5. Even in hot tubs with heterotrophic plate counts of <1 cfu/ml, *P. aeruginosa* was isolated from 5% of bromine-disinfected pools compared with only 0.8% of chlorine-disinfected pools (Shaw, 1984).

Routine, thorough cleaning of surrounding surfaces will help to reduce infections with *P. aeruginosa*. In addition, swimming pool, hot tub and natural spa operators should strongly encourage users to shower before entering the water and, where possible, control the number of bathers and their duration of hot tub exposure (Public Health Laboratory Service Spa Pools Working Group, 1994).

3.4.3 *Mycobacterium spp.*

1. *Risk assessment*

Mycobacterium spp. are rod-shaped bacteria that are 0.2–0.6 µm × 1.0–10 µm in size and have cell walls with a high lipid content. This feature means that they retain dyes in staining procedures that employ an acid wash; hence, they are often referred to as acid-fast bacteria. Atypical mycobacteria (i.e. other than strictly pathogenic species, such as *M. tuberculosis*) are ubiquitous in the aqueous environment and proliferate in and around swimming pools and similar environments (Leoni et al., 1999).

In pool environments, *M. marinum* is responsible for skin and soft tissue infections in normally healthy people. Infections frequently occur on abraded elbows and knees and result in localized lesions, often referred to as swimming pool granuloma. The organism is probably picked up from the pool edge by bathers as they climb in and out of the pool (Collins et al., 1984).

Respiratory illnesses associated with hot tub use in normally healthy individuals have been linked to other atypical mycobacteria (Embil et al., 1997; Kahana et al., 1997; Grimes et al., 2001; Khoor et al., 2001; Mangione et al., 2001; Cappelluti et al., 2003; Lumb et al., 2004). For example, *M. avium* in hot tub water has been linked to hypersensitivity pneumonitis and possibly pneumonia (Embil et al., 1997). Symptoms were flu-like and included cough, fever, chills, malaise and headaches. The illness followed the inhalation of heavily contaminated aerosols generated by the hot tub. The reported cases relate to domestic hot tubs, many of which were located outdoors. In most instances the frequency of hot tub use was high, as was the duration of exposure (an extreme example being use for 1–2 h each day), and maintenance of disinfection and cleaning were not ideal. It is likely that detected cases are only a small fraction of the total number of cases. Amoebae may also play a role in the transmission of *Mycobacterium* spp. (Cirillo et al., 1997).

2. *Risk management*

Mycobacteria are more resistant to disinfection than most bacteria due to the high lipid content of their cell wall (Engelbrecht et al., 1977). Therefore, thorough cleaning of surfaces and materials around pools and hot tubs where the organism may persist is necessary, supplemented by the maintenance of disinfection at appropriate levels. In addition, occasional shock dosing of chlorine (see Chapter 5) may be required to eradicate mycobacteria accumulated in biofilms within pool or hot tub components (Aubuchon et al., 1986). In natural spas where the use of disinfectants is undesirable or where it is difficult to maintain adequate disinfectant levels, superheating the water to 70 °C on a daily basis during periods of non-use may help to control *M. marinum* (Embil et al., 1997). Immunocompromised individuals should be cautioned about the risks of exposure to atypical mycobacteria in and around pools and hot tubs.

3.4.4 *Staphylococcus aureus*

1. *Risk assessment*

The genus *Staphylococcus* comprises non-motile, non-spore-forming and non-encapsulated Gram-positive cocci (0.5–1.5 µm in diameter) that ferment glucose and grow aerobically and anaerobically. They are usually catalase positive and occur singly and in pairs, tetrads, short chains and irregular grape-like clusters. In humans, there are three clinically important species – *Staphylococcus aureus, S. epidermidis* and *S. saprophyticus*. *S. aureus* is the only coagulase-positive species and is clinically the most important (Duerden et al., 1990).

Humans are the only known reservoir of *S. aureus*, and it is found on the anterior nasal mucosa and skin as well as in the faeces of a substantial portion of healthy individuals. Robinton & Mood (1966) found that *S. aureus* was shed by bathers under all conditions of swimming, and the bacteria can be found in surface films in pool water. Coagulase-positive *Staphylococcus* strains of normal human flora have been found in chlorinated swimming pools (Rocheleau et al., 1986).

The presence of *S. aureus* in swimming pools is believed to have resulted in skin rashes, wound infections, urinary tract infections, eye infections, otitis externa, impetigo and other infections (Calvert & Storey, 1988; Rivera & Adera, 1991). Infections of *S. aureus* acquired from recreational waters may not become apparent until 48 h after contact. De Araujo et al. (1990) have suggested that recreational waters with a high density of bathers present a risk of staphylococcal infection that is comparable to the risk of gastrointestinal illness involved in bathing in water considered unsafe because of faecal pollution. According to Favero et al. (1964) and Crone & Tee (1974), 50% or more of the total staphylococci isolated from swimming pool water samples are *S. aureus*. In Italy, however, in a study on chlorinated pools where the free chlorine level varied between 0.8 and 1.2 mg/l, *S. aureus* was not recovered from water samples (Bonadonna et al., 2004).

2. *Risk management*

Adequate inactivation of potentially pathogenic *S. aureus* in swimming pools can be attained by maintaining free chlorine levels greater than 1 mg/l (Keirn & Putnam, 1968; Rivera & Adera, 1991) or equivalent disinfection efficiency. There is evidence that showering before pool entry can reduce the shedding of staphylococci from the skin into the pool (Robinton & Mood, 1966). Continuous circulation of surface water through the treatment process helps to control the build-up of *S. aureus*. Pool contamination can also be reduced if the floors surrounding the pool and in the changing areas are kept at a high standard of cleanliness. Although it is not recommended that water samples be routinely monitored for *S. aureus*, where samples are taken, levels should be less than 100/100 ml.

3.4.5 *Leptospira interrogans sensu lato*

1. *Risk assessment*

Leptospires are motile spirochaete (helically coiled) bacteria. Traditionally, the genus *Leptospira* consists of two species, the pathogenic *L. interrogans* sensu lato and the saprophytic *L. biflexa* sensu lato. Serological tests within each species revealed many antigenic variations, and, on this basis, leptospires are classified as serovars. In addition, a classification system based on DNA relatedness is used (Brenner et al., 1999). The current species determination is based on this principle. The serological and genetic taxonomies are two different systems with only little correlation (Brenner et al., 1999). Free-living strains (*L. biflexa* sensu lato) are ubiquitous in the environment (Faine et al., 1999); the pathogenic strains (*L. interrogans* sensu lato), however, live in the kidneys of animal hosts.

Pathogenic leptospires live in the proximal renal tubules of the kidneys of carrier animals (including rats, cows and pigs) and are excreted in the urine, which can then contaminate surface waters. Humans and animals (humans are always incidental hosts) become infected either directly through contact with infected urine or indirectly via contact with contaminated water. Leptospires gain entry to the body through cuts and abrasions of the skin and through the mucosal surfaces of the mouth, nose and conjunctiva.

Diseases caused by *Leptospira interrogans* sensu lato have been given a variety of names, including swineherd's disease, Stuttgart disease and Weil's syndrome, but collectively all of these infections are termed leptospirosis. The clinical manifestations of leptospirosis vary considerably in form and intensity, ranging from a mild flu-like

illness to a severe and potentially fatal form of the disease, characterized by liver and kidney failure and haemorrhages (Weil's syndrome). Severity is related to the infecting serovar as well as host characteristics, such as age and underlying health and nutritional status. Specific serovars are often associated with certain hosts.

Compared with many other pathogens, leptospires have a comparatively low resistance to adverse chemical and physical conditions, including disinfectants. They are seldom found in water of below pH 6.8, and they cannot tolerate drying or exposure to direct sunlight (Noguchi, 1918; Alston & Broom, 1958; Weyant et al., 1999).

The majority of reported outbreaks of waterborne leptospirosis have involved fresh recreational waters, but two outbreaks have been associated with non-chlorinated swimming pools (Cockburn et al., 1954; de Lima et al., 1990). Domestic or wild animals with access to the implicated waters were the probable sources of *Leptospira*.

2. *Risk management*

The risk of leptospirosis can be reduced by preventing direct animal access to swimming pools and maintaining adequate disinfectant concentrations. Informing users about the hazards of swimming in water that is accessible to domestic and wild animals may also help to prevent infections. Outbreaks are not common; thus, it appears that the risk of leptospirosis associated with swimming pools and hot tubs is low. Normal disinfection of pools is sufficient to inactivate *Leptospira* spp.

3.5 Non-faecally-derived viruses

Infections associated with non-faecally-derived viruses found in swimming pools and similar environments are summarized in Table 3.7.

Table 3.7. Non-faecally-derived viruses found in swimming pools and similar environments and their associated infections

Organism	Infection	Source
Adenoviruses[a]	Pharyngo-conjunctivitis (swimming pool conjunctivitis)	Other infected bathers
Molluscipoxvirus	Molluscum contagiosum	Bather shedding on benches, pool or hot tub decks, swimming aids
Papillomavirus	Plantar wart	Bather shedding on pool and hot tub decks and floors in showers and changing rooms

[a] Covered in Section 3.1.2

3.5.1 Molluscipoxvirus

1. *Risk assessment*

Molluscipoxvirus is a double-stranded DNA virus in the Poxviridae family. Virions are brick-shaped, about 320 nm × 250 nm × 200 nm. The virus causes molluscum contagiosum, an innocuous cutaneous disease limited to humans. It is spread by direct person-to-person contact or indirectly through physical contact with contaminated

surfaces. The infection appears as small, round, firm papules or lesions, which grow to about 3–5 mm in diameter. The incubation period is 2–6 weeks or longer. Individual lesions persist for 2–4 months, and cases resolve spontaneously in 0.5–2 years. Swimming pool-related cases occur more frequently in children than in adults. The total number of annual cases is unknown. Since the infection is relatively innocuous, the reported number of cases is likely to be much less than the total number. Lesions are most often found on the arms, back of the legs and back, suggesting transmission through physical contact with the edge of the pool, benches around the pool, swimming aids carried into the pool or shared towels (Castilla et al., 1995). Indirect transmission via water in swimming pools is not likely. Although cases associated with hot tubs have not been reported, they should not be ruled out as a route of exposure.

2. *Risk management*

The only source of molluscipoxvirus in swimming pool and similar facilities is infected bathers (Oren & Wende, 1991). Hence, the most important means of controlling the spread of the infection is to educate the public about the disease, the importance of limiting contact between infected and non-infected people and medical treatment. Thorough frequent cleaning of surfaces in facilities that are prone to contamination can reduce the spread of the disease.

3.5.2 Papillomavirus

1. *Risk assessment*

Papillomavirus is a double-stranded DNA virus in the family Papovaviridae. The virions are spherical and approximately 55 nm in diameter. The virus causes benign cutaneous tumours in humans. An infection that occurs on the sole (or plantar surface) of the foot is referred to as a verruca plantaris or plantar wart. Papillomaviruses are extremely resistant to desiccation and thus can remain infectious for many years. The incubation period of the virus remains unknown, but it is estimated to be 1–20 weeks. The infection is extremely common among children and young adults between the ages of 12 and 16 who frequent public pools and hot tubs. It is less common among adults, suggesting that they acquire immunity to the infection. At facilities such as public swimming pools, plantar warts are usually acquired via direct physical contact with shower and changing room floors contaminated with infected skin fragments (Conklin, 1990; Johnson, 1995). Papillomavirus is not transmitted via pool or hot tub waters.

2. *Risk management*

The primary source of papillomavirus in swimming pool facilities is infected bathers. Hence, the most important means of controlling the spread of the virus is to educate the public about the disease, the importance of limiting contact between infected and non-infected people and medical treatment. The use of pre-swim showering, wearing of sandals in showers and changing rooms and regular cleaning of surfaces in swimming pool facilities that are prone to contamination can reduce the spread of the virus.

3.6 Non-faecally-derived protozoa

Table 3.8 summarizes the non-faecally-derived protozoa found in or associated with swimming pools and similar environments and their associated infections.

Table 3.8. Non-faecally-derived protozoa found in swimming pools and similar environments and their associated infections

Organism	Infection	Source
Naegleria fowleri	Primary amoebic meningoencephalitis (PAM)	Pools, hot tubs and natural spas including water and components
Acanthamoeba spp.	Acanthamoeba keratitis Granulomatous amoebic encephalitis (GAE)	Aerosols from HVAC systems
Plasmodium spp.	Malaria	Seasonally used pools may provide a breeding habitat for mosquitoes carrying *Plasmodium*

HVAC – heating, ventilation and air conditioning

3.6.1 Naegleria fowleri

1. Risk assessment

Naegleria fowleri is a free-living amoeba (i.e. it does not require the infection of a host organism to complete its life cycle) present in fresh water and soil. The life cycle includes an environmentally resistant encysted form. Cysts are spherical, 8–12 µm in diameter, with smooth, single-layered walls containing one or two mucus-plugged pores through which the trophozoites (infectious stages) emerge. *N. fowleri* is thermophilic, preferring warm water and reproducing successfully at temperatures up to 46 °C.

N. fowleri causes primary amoebic meningoencephalitis (PAM). Infection is usually acquired by exposure to water in ponds, natural spas and artificial lakes (Martinez & Visvesvara, 1997; Szenasi et al., 1998). Victims are usually healthy children and young adults who have had contact with water about 7–10 days before the onset of symptoms (Visvesvara, 1999). Infection occurs when water containing the organisms is forcefully inhaled or splashed onto the olfactory epithelium, usually from diving, jumping or underwater swimming. The amoebae in the water then make their way into the brain and central nervous system. Symptoms of the infection include severe headache, high fever, stiff neck, nausea, vomiting, seizures and hallucinations. The infection is not contagious. For those infected, death occurs usually 3–10 days after onset of symptoms. Respiratory symptoms occur in some patients and may be the result of hypersensitivity or allergic reactions or may represent a subclinical infection (Martinez & Visvesvara, 1997).

Although PAM is an extremely rare disease, cases have been associated with pools and natural spas. In Usti, Czech Republic, 16 cases of PAM were associated with a public swimming pool (Cerva & Novak, 1968). The source of the contamination was traced to a cavity behind a false wall used to shorten the pool length. The pool took water from a local river, which was the likely source of the organism. One confirmed case of PAM occurred in Bath Spa, in the UK, in 1978. The victim was a young girl who swam in a public swimming pool fed with water from the historic thermal springs that rise naturally in the city (Cain et al., 1981). Subsequent analysis confirmed the thermal springs to be the source of the infection (Kilvington et al., 1991). *N. fowleri* has also been isolated from air-conditioning units (Martinez, 1993).

2. *Risk management*

Risk of infection can be reduced by minimizing the occurrence of the causative agent through appropriate choice of source water, proper cleaning, maintenance, coagulation–filtration and disinfection.

3.6.2 *Acanthamoeba spp.*

1. *Risk assessment*

Several species of free-living *Acanthamoeba* are human pathogens (*A. castellanii*, *A. culbertsoni*, *A. polyphaga*). They can be found in all aquatic environments, including disinfected swimming pools. Under adverse conditions, they form a dormant encysted stage. Cysts measure 15–28 µm, depending on the species. *Acanthamoeba* cysts are highly resistant to extremes of temperature, disinfection and desiccation. The cysts will retain viability from –20 °C to 56 °C. When favourable conditions occur, such as a ready supply of bacteria and a suitable temperature, the cysts hatch (excyst) and the trophozoites emerge to feed and replicate. All pathogenic species will grow at 36–37 °C, with an optimum of about 30 °C. Although *Acanthamoeba* is common in most environments, human contact with the organism rarely leads to infection.

Human pathogenic species of *Acanthamoeba* cause two clinically distinct diseases: granulomatous amoebic encephalitis (GAE) and inflammation of the cornea (keratitis) (Ma et al., 1990; Martinez, 1991; Kilvington & White, 1994).

GAE is a chronic disease of the immunosuppressed; GAE is either subacute or chronic but is invariably fatal. Symptoms include fever, headaches, seizures, meningitis and visual abnormalities. GAE is extremely rare, with only 60 cases reported worldwide. The route of infection in GAE is unclear, although invasion of the brain may result from the blood following a primary infection elsewhere in the body, possibly the skin or lungs (Martinez, 1985, 1991). The precise source of such infections is unknown because of the almost ubiquitous presence of *Acanthamoeba* in the environment.

Acanthamoeba keratitis affects previously healthy people and is a severe and potentially blinding infection of the cornea (Ma et al., 1990; Kilvington & White, 1994). In the untreated state, acanthamoeba keratitis can lead to permanent blindness. Although only one eye is usually affected, cases of bilateral infection have been reported. The disease is characterized by intense pain and ring-shaped infiltrates in the corneal stroma. Contact lens wearers are most at risk from the infection and account for approximately 90% of reported cases (Kilvington & White, 1994). Poor contact lens hygiene practices (notably ignoring recommended cleaning and disinfection procedures and rinsing or storing of lenses in tap water or non-sterile saline solutions) are recognized risk factors, although the wearing of contact lenses while swimming or participating in other water sports may also be a risk factor. In non-contact lens related keratitis, infection arises from trauma to the eye and contamination with environmental matter such as soil and water (Sharma et al., 1990). Samples et al. (1984) report a case of keratitis that may have been acquired from domestic hot tub use.

2. *Risk management*

Although *Acanthamoeba* cysts are resistant to chlorine- and bromine-based disinfectants, they can be removed by filtration. Thus, it is unlikely that properly operated

swimming pools and similar environments would contain sufficient numbers of cysts to cause infection in normally healthy individuals. Immunosuppressed individuals who use swimming pools, natural spas or hot tubs should be aware of the increased risk of GAE. A number of precautionary measures are available to contact lens wearers, including removal before entering the water, wearing goggles, post-swim contact lens wash using appropriate lens fluid and use of daily disposable lenses.

3.6.3 Plasmodium spp.

1. *Risk assessment*

   Swimming pools are associated not with *Plasmodium* spp. but with anopheline mosquito larvae, the insect vectors of *Plasmodium*. Mbogo et al. (submitted) found that over 70% of swimming pools sampled in urban Malindi in Kenya were positive for mosquito larvae. The problem relates to the seasonal use of the pools. Before people leave their summer houses, it is common to drain the pool; however, rainwater accumulated during the rainy season provides a suitable habitat for mosquito breeding, with the attendant risks of malaria as a result.

2. *Risk management*

   During the rains, when the pools fill with water, they should be drained every 5–7 days to avoid mosquito larvae developing into adults. The swimming pools may also be treated with appropriate larvicides when not in use for long periods.

3.7 Non-faecally-derived fungi

Infections associated with fungi found in swimming pools and similar environments are summarized in Table 3.9.

Table 3.9. Fungi found in swimming pools and similar environments and their associated infections

Organism	Infection	Source
Trichophyton spp. *Epidermophyton floccosum*	Athlete's foot (tinea pedis)	Bather shedding on floors in changing rooms, showers and pool or hot tub decks

3.7.1 Trichophyton spp. and Epidermophyton floccosum

1. *Risk assessment*

   Epidermophyton floccosum and various species of fungi in the genus *Trichophyton* cause superficial fungal infections of the hair, fingernails or skin. Infection of the skin of the foot (usually between the toes) is described as tinea pedis or, more commonly, as 'athlete's foot' (Aho & Hirn, 1981). Symptoms include maceration, cracking and scaling of the skin, with intense itching. Tinea pedis may be transmitted by direct person-to-person contact; in swimming pools, however, it may be transmitted by physical contact with surfaces, such as floors in public showers, changing rooms, etc., contaminated with infected skin fragments. In Japan, a study comparing students attending a regular swimming class with those who did not found a significantly greater level of infection in the swimmers (odds ratio of 8.5), and *Trichophyton* spp. were

isolated from the floor of a hot tub and the floor of one of the changing rooms (Kamihama et al., 1997). The fungus colonizes the stratum corneum when environmental conditions, particularly humidity, are optimal. From in vitro experiments, it has been calculated that it takes approximately 3–4 h for the fungus to initiate infection. The infection is common among lifeguards and competitive swimmers, but relatively benign; thus, the true number of cases is unknown.

2. Risk management

The sole source of these fungi in swimming pool and similar facilities is infected bathers. Hence, the most important means of controlling the spread of the fungus is to educate the public about the disease, the importance of limiting contact between infected and non-infected bathers and medical treatment. The use of pre-swim showers, wearing of sandals in showers and changing rooms and frequent cleaning of surfaces in swimming pool facilities that are prone to contamination can reduce the spread of the fungi (Al-Doory & Ramsey, 1987). People with severe athlete's foot or similar dermal infections should not frequent public swimming pools, natural spas or hot tubs. Routine disinfection appears to control the spread of these fungi in swimming pools and similar environments (Public Health Laboratory Service Spa Pools Working Group, 1994).

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