CHAPTER 3

MICROBIOLOGICAL HAZARDS

The risk of illness or infection associated with swimming pools, spas and similar recreational-water environments has been linked to faecal contamination of the water. The faecal contamination may be due to faeces released by bathers or contaminated source water. Many of the outbreaks related to swimming pools have occurred because disinfection was poorly or not at all applied. The majority of reported swimming pool-related outbreaks have been caused by viruses; recently, however, reported outbreaks have been more frequently associated with bacteria and protozoa.

Non-faecal human shedding (e.g., from mucus, saliva, skin) in the swimming pool, spa or similar recreational-water environments is a source of potential non-enteric pathogenic organisms. Infected users can directly contaminate pool or spa waters and the surfaces of objects or materials at a facility with sufficient numbers of primary pathogens (notably viruses or fungi), which can consequently 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 contaminated water in pools or spas. In addition, certain free-living aquatic bacteria and amoebas can grow in pool or spa waters, in pool or spa 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 (opportunistic pathogens) may cause a variety of respiratory, dermal or central nervous system infections or diseases.

This chapter describes outbreaks of illness and infection associated with faecal pollution in swimming pools, spas and similar recreational-water environments, as well as those associated with microbial hazards arising from non-faecal human shedding and facilities. Risk management measures are also briefly discussed.

3.1 Faecally derived viruses

3.1.1 Hazard identification

Viruses that have been linked to swimming pool outbreaks are shown in Table 3.1. Adenoviruses have been reported most frequently; however, hepatitis A virus, Norwalk virus and echovirus have also been linked to swimming pool-associated illness. The source of the agent was unknown for most of the adenovirus outbreaks. Although the viruses could be from faecal material, another likely source is secretions from the eyes or throat. Sources for three of the other four viruses in Table 3.1 were identified. Two were associated with swimmers themselves, and the third was associated with a cross-connection to a sewer line. In most outbreaks, the chlorinator system for the pool was either inoperable or not operating properly. Only in the outbreak associated with adenovirus type 4 was the causative agent isolated from the water.

Table 3.1: Summary of viral waterborne outbreaks associated with swimming pools

Etiological agent	Source of agent	Disinfection	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	Failed chlorinator	Martone et al., 1980
Adenovirus 7a	Unknown	Malfunctioning chlorinator	Turner et al., 1987
Hepatitis A	Accidental faecal release suspected	None	Solt et al., 1994
Hepatitis A	Cross-connection to sewer line	Operating properly	Mahoney et al., 1992

Etiological agent	Source of agent	Disinfection	Reference
Norwalk virus	Unknown	Chlorinator disconnected	Kappus et al., 1982
Echovirus 30	Vomitus	Operating properly	Kee et al., 1994

3.1.2 Outbreaks of viral illness associated with swimming pools

1) Adenovirus-related outbreaks

Foy et al. (1968) reported an outbreak of pharyngo-conjunctival fever caused by adenovirus type 3 in 1968. The infection occurred in two children's swimming teams after exposure to unchlorinated 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.

In 1974, 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 indicated that the high school swimming pool was the source of the infection, because the pool chlorinator and pool filter had failed. The outbreak was put under control by raising the pool's chlorine level above 0.3 mg/litre.

Adenovirus type 4 was the causative agent of a swimming pool-related outbreak of pharyngo-conjunctivitis in the summer of 1977, 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. Less than 0.5 mg total chlorine per litre and 0 mg free chlorine per litre were detected in pool water samples. Adequate chlorination and closing the pool for the summer stopped the spread of pharyngo-conjunctival fever.

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/litre. They also pointed out that the virus was likely transmitted through water, but person-to-person transmission could not be ruled out.

In 1987, an outbreak of adenovirus type 7a infections was associated with a swimming pool (Turner et al., 1987). Seventy-seven individuals were identified with the symptoms of pharyngitis. A telephone survey indicated that persons who swam at the community swimming pool were more likely to be ill than those who did not. Furthermore, 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 cleared when proper chlorination was reinstituted.

2) Hepatitis A-related outbreaks

In September 1979, 31 children were hospitalized with hepatitis A in a small town in north-eastern Hungary (Solt et al., 1994). An epidemiological investigation of potential common sources

eliminated food and 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 that 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 behaviour contributed to the outbreak.

An outbreak of hepatitis A in several US states in 1989 that 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 spa pool 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 chlorine levels in the pools were not below appropriate concentrations.

3) Norwalk virus-related outbreak

Kappus et al. (1982) reported an outbreak of Norwalk gastroenteritis associated with a swimming pool in 1977. The outbreak affected 103 individuals. The illness typically lasted 24 h and was characterized by vomiting and cramping. Serological studies suggested that Norwalk virus 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 causative agent could not be found.

4) Echovirus-related outbreak

In 1992, an outbreak of gastroenteritis in a small village in Northern Ireland was reported (Kee et al., 1994). Forty-six people reported symptoms of vomiting, diarrhoea and headache shortly after swimming in an outdoor swimming pool. It was found that 34 swimmers had become ill and that one of the swimmers had vomited into the pool. Other cases were reported after the swimming incident. 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 as well. Proper chlorine levels had been maintained at the pool, but they were inadequate to contain the risk of infection from vomitus in the pool water.

3.1.3 Risk assessment

Adenoviruses are different from other viruses associated with swimming pool outbreaks of disease, in that they are double-stranded DNA viruses. They have an icosahedral shape and a diameter that ranges between 80 and 110 nm. Forty-seven serotypes of adenoviruses cause human infections. Except for adenovirus types 40 and 41, all of the adenoviruses are able to grow in commonly available cell cultures. Although adenoviruses can cause gastroenteritis, they are most frequently observed to cause pharyngo-conjunctival fever, an infection of the eyelids or the throat. The predominant symptoms of these infections are usually fever and some throat pain, headache, abdominal pain and conjunctivitis. The onset of symptoms for adenovirus infections is very rapid. An infected person may excrete virus from the respiratory tract; after a short time, however, the virus disappears from respiratory secretions and can be found in faecal specimens, sometimes for

extended periods (Fox et al., 1969). The attack rate for swimming pool outbreaks linked to adenovirus serotypes is moderately high. In a Kansas, USA, outbreak, the attack rate was 33% (Caldwell et al., 1974). In another outbreak, in Georgia, USA, the attack rate was 18% (Martone et al., 1980), and in an outbreak in Oklahoma, USA, the attack rate in swimmers who had swallowed water was 52% (Turner et al., 1987).

Hepatitis A is a single-stranded RNA virus that has an icosahedral form. The virus particle has a diameter of about 27 nm. Hepatitis A is the only species within the genus. Although hepatitis A can be cultured, its growth in culture is very slow. Molecular probes specific for this virus are able to detect it much more rapidly. Hepatitis A is transmitted by the faecal–oral route. Water and sewage are the most frequently observed means of transmission. This virus has an incubation period of 15–50 days. Anorexia, nausea and vomiting are the predominant symptoms. Jaundice is also a common symptom. Viruses can be shed before the onset of symptoms. The shedding state does not appear to be active beyond the observation period for 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).

Norwalk virus is a single-stranded RNA virus that has a round structure with a cupped surface and a diameter of about 27 nm. The symptoms of infection usually occur within one or two days of exposure and include nausea, vomiting, diarrhoea and fever. The infections usually clear spontaneously. The shedding of viruses declines very rapidly after the onset of illness. The attack rate associated with the only swimming pool outbreak reported in the literature was very high (71%) for swimmers who had swallowed water (Kappus et al., 1982).

Echoviruses belong to the group of enteroviruses. There are 34 serotypes within the subgroup echovirus. The echovirus belongs to the group of single-stranded RNA viruses. The viruses have an icosahedral structure with a diameter of 27 nm. Echoviruses are transmitted via the faecal–oral route. These viruses are known to cause several disease conditions, such as meningitis, encephalitis, pneumonitis and pleurisy, as well as respiratory-enteric disease, gastroenteritis and conjunctivitis. The predominant symptoms associated with echovirus gastroenteritis are vomiting, headache, fever and diarrhoea. The gastroenteric infections clear spontaneously.

Enteric viruses occur in very high densities in the faeces of infected individuals (Table 3.2). Hepatitis A virus has been found at densities as high as 10^{10} per gram (Coulepis et al., 1980), Norwalk viruses have been estimated to occur at densities as high as 10^{11} per gram, and echoviruses are found at densities of only 10^{6} per gram during infection. Given the high densities at which some viruses occur in infected individuals, it is not surprising that accidental faecal releases (AFRs) into swimming pools and spas can lead to very high attack rates in pools where outbreaks take place.

Table 3.2: Viral exposure factors associated with swimming pools

Agent	Density shed during infection	Duration of shedding	Infective dose	Reference
Echovirus	10^{6} per gram	1–4 weeks	–	Feachem et al., 1983; Beneson, 1990
Echovirus 12	–	–	919 pfu/ID_{50}[a]	Schiff et al., 1984
Hepatitis A	10^{10} per gram	30 days	Not known	Coulepis et al., 1980; Mao et al., 1980
Norwalk virus	10^{11} per gram[b]	–	$<6 \times 10^{3}$ PDU[c]	Moe et al., 1998

[a] pfu = plaque-forming unit; ID_{50} = infective dose for 50% of the population.
[b] Norwalk virus cannot be cultured in the laboratory. This value is an estimate.

[c] ID_{20} at 6 polymerase chain reaction (PCR) detectable units.

Infective dose data for viruses are not readily available because of the difficulties and expense of conducting human feeding studies. Some studies have, however, been conducted in spite of the difficulties (Table 3.2). Echovirus 12 was studied by Schiff et al. (1984) to determine the dose at which 50% of the challenged volunteers were infected. They calculated that the ID_{50} for echovirus 12 was about 919 plaque-forming units (pfu). The number of plaque-forming units required to infect 1% of those challenged was about 17 pfu.

3.1.4 Risk management

The control of viruses in swimming pool water is usually accomplished by the proper application of chlorine and other disinfectants — for example, by maintaining a free chlorine residual of about 0.4 mg/litre in the pool water. Although this level of free chlorine in pool water is normally effective, conditions where high concentrations of organic material (e.g., high numbers of swimmers shedding skin into the pool water) exist could create a significant chlorine demand. This demand reduces the total (free and combined) chlorine in the pool, lowering or eliminating its disinfectant action.

Episodes of gross contamination of a swimming pool due to an AFR or vomitus also cannot be effectively controlled by normal chlorine levels. Where pools or spas are not disinfected, AFRs and vomitus present an even greater problem. The only approach to maintaining health safety under conditions of an AFR or vomitus is to prohibit the use of the pool until the contaminants are inactivated (see chapter 5).

The education of parents of small children and other recreationists with regard to 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 AFRs. These groups should also be cautioned about swimming in pools if they are suffering from gastroenteritis or other illnesses where viral pathogens might be transmitted from swimmer to swimmer via pool water.

3.2 Faecally derived bacteria

3.2.1 Hazard identification

Shigella species and *Escherichia coli* O157 are two closely related organisms that have been linked to outbreaks of illness associated with swimming in pools. *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 been two outbreaks reported where the focal point was a children's paddling pool. The information describing outbreaks associated with artificial lakes and ponds will be used to establish the potential risk that might be experienced in swimming pools under similar conditions.

Table 3.3 summarizes bacterial outbreaks of disease associated with swimming pools.

Table 3.3: Summary of bacterial outbreaks of disease associated with swimming pools

Etiological agent	Source of agent	Disinfection	Reference
Shigella spp.	AFR	None	Sorvillo et al., 1988
	Not known	None	Makintubee et al., 1987
	AFR	None	Blostein, 1991
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	MMWR, 1996
	Not known	None	Cransberg et al., 1996

3.2.2 Outbreaks of bacterial illness associated with swimming pools

1) Shigella-*related outbreaks*

Three outbreaks of shigellosis in the USA have been associated with swimming in freshwater lakes and ponds. In each outbreak, patients were most likely to be children. In two of the three outbreaks, infected patients, including toddlers in diapers, were identified as the source of the causative organism.

An outbreak of shigellosis associated with swimming at an artificial lake occurred in Los Angeles County, USA, in 1985 (Sorvillo et al., 1988). Sixty-eight persons were identified as having diarrhoeal illness. Thirty-three of the cases were culture confirmed as being caused by *Shigella sonnei* (29 isolates) or *S. boydii* (four isolates). Water samples from the swimming area had high faecal coliform counts. No evidence of sewage contamination was identified. It was suggested that direct bather contamination might have occurred.

In 1987, 62 cases of shigellosis associated with swimming in a natural reservoir were reported by Makintubee et al. (1987). Although *Shigella* was not isolated from the reservoir during the outbreak, the water did contain excessive levels of faecal coliforms and faecal streptococci.

In 1989, an outbreak of shigellosis occurred among visitors to a recreational park (Blostein, 1991). Sixty-five cases were linked to swimming in a pond at the park. *Shigella* was not recovered from samples of park pond water, and faecal coliform counts from samples taken shortly after the outbreak were satisfactory. The implied source of the causative agent was the swimmers themselves.

Although this is a small number of outbreaks, a discernible pattern is quite obvious. In small bodies of water, where there is little water movement, AFRs from an infected person can be the source of an outbreak. Because there is minimal dilution of the agent, an infectious dose is readily available to nearby swimmers. These agents are of particular importance in spas in which disinfection cannot be effectively practised and, hence, disinfectant is absent.

2) E. coli *O157-related outbreaks*

In the summer of 1991, an outbreak of bloody diarrhoea (haemorrhagic colitis) and haemolytic uraemic syndrome (HUS) was linked to swimming in a small lake at a public park (Keene et al., 1994). A case–control study identified 21 children with park-associated *E. coli* O157. All had played or swum in the lake during a 24-day period. An environmental health assessment revealed that contamination by swimmers, including many toddlers in diapers, was the most likely source of the

pathogen. Although *E. coli* O157 was not recovered from the lake water, enterococci were detected in the swimming area during part of the 24-day outbreak period. The geometric mean enterococcus density in lake samples was greater than 50 per 100 ml.

In 1992, an outbreak of *E. coli* O157 infections was epidemiologically and clinically linked to a collapsible children's paddling pool (Brewster et al., 1994). Six cases of diarrhoea, including one case of HUS, 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 HUS, were epidemiologically linked to a public paddling pool (Hildebrand et al., 1996). *E. coli* O157 phage 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/litre at the time of sampling.

In late June 1995, 12 cases of bloody diarrhoea in children, including three cases of HUS, were epidemiologically linked to swimming at a lake in a state park (MMWR, 1996). Water samples collected before and two weeks following the outbreak contained unacceptable levels of *E. coli*. Although *E. coli* O157 was identified as the etiological agent, the organism was not isolated from samples collected following the outbreak.

A small, shallow, semi-natural lake used for swimming in the Netherlands was determined to be the most likely source of an outbreak of four cases of HUS (Cransberg et al., 1996). Diarrhoea was reported in several family members who had also swum in the lake. All four children, ages 1.5–3.5 years, who had bathed in the lake within a five-day period showed a positive serology for *E. coli* O157. *E. coli* O157 was identified in the laboratory and was shown to produce verotoxin types I and II. Lake water samples collected 16 days after the last patient could have been infected did not contain *E. coli* O157.

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 HUS. 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 one week. About 5–10% of individuals develop HUS following an *E. coli* O157 infection. HUS, 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 3.4).

Table 3.4: Bacterial exposure factors associated with swimming pools

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

[a] Probably similar to *Shigella*.

The infective dose for *Shigella* species is usually between 10 and 100 organisms (Table 3.4). Lower doses, however, may cause illness in infants, the elderly or immunocompromised individuals. The infectious dose for *E. coli* O157 is unknown but is likely similar to that of *Shigella* species. Keene et al. (1994) have suggested that the infective dose is very low, based on experience in an outbreak situation.

3.2.4 Risk management

E. coli O157 and *Shigella* species are readily controlled by chlorine and other disinfectants under ideal conditions. One of the primary risk management interventions is to reduce AFR occurrence in the first place — for example, by educating pool users. However, if an AFR has occurred in a swimming pool, 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. Similarly, an AFR will present a greater problem if it occurs in an undisinfected spa or pool. Maintaining health safety under these conditions involves prohibition from using the pool and education of recreationists.

3.3 Faecally derived pathogenic protozoa

3.3.1 Hazard identification

Risk of illness in swimming pools that has been linked to pathogenic protozoa mainly involves two parasites: *Giardia* and *Cryptosporidium*. These two organisms are similar in a number of respects. They have a cyst or oocyst form that is highly resistant to environmental stress and highly resistant to disinfectants, they have a low infective dose and they are shed in high densities by individuals who have giardiasis or cryptosporidiosis. There have been a number of outbreaks of disease attributed to these pathogens, as summarized in Table 3.5.

Table 3.5: Summary of protozoan waterborne outbreaks associated with swimming pools

Etiological agent	Source of agent	Disinfection	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	MMWR, 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	MMWR, 1994

3.3.2 Outbreaks of protozoan illness associated with swimming pools

1) **Giardia-*related outbreaks***

Giardiasis has been associated with swimming pools and water slides. In 1984, a case of giardiasis was reported in a child who was a participant in an infant and toddler swim class in Washington State, USA (Harter et al., 1984). The identification of this case of giardiasis led to the conduct of a stool survey of 70 child participants in the class. The stool survey revealed a 61% prevalence of *Giardia* infection. None of the non-swimming playmates was positive. Thirty-five per cent of 23 children 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 AFR.

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 handicapped child who had a faecal accident in the pool. He was a member of the group that swam the same day as the women's swimming group. A stool survey of the handicapped child's group showed that of 20 persons tested, nine were positive for *Giardia*, including the child mentioned above. Pool records showed that no chlorine measurements had been taken on the day of the AFR and that the chlorine level was zero on the following day.

In 1988, an outbreak of giardiasis was associated with a hotel's new water slide pool in Manitoba, Canada (Greensmith et al., 1988). Among 107 hotel guests and their 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 for staying at the hotel, using the water slide pool and swallowing pool water. A possible contributing factor was the proximity of a toddlers' wading pool, a potential source of faecal material, to the water slide pool. Water in the slide pool was treated by sand filtration, and disinfection was accomplished through the use of bromine.

In 1994, a case–control study was done in the United Kingdom to determine the risk factors for giardiasis. Giardiasis cases were identified from reports by the consultants for Communicable Disease Control over a duration of one year (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. Travel and type of travel were also significant risk factors. Other recreational exposures and ingestion of potentially contaminated water were found to be not significantly related to giardiasis.

2) Cryptosporidium-*related outbreaks*

Outbreaks of cryptosporidiosis have been linked to swimming pools and a wave pool. The sources of *Cryptosporidium* contaminating the pools were 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 (MMWR, 1990). Swimmers were exposed to pool water in which there had been a single faecal accident. The attack rate was about 73%. The common factor linking infected individuals was use of the swimming pool.

In August 1988, an increase was noted in the number of cases of cryptosporidiosis that had been identified by the Doncaster Royal Infirmary (United Kingdom) 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 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 was experienced in controlling the level of free chlorine residual, which implied that disinfection was probably not maintained at appropriate concentrations.

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 prior to onset of illness. Attack rates ranged from 8% to 78% for various groups of children's pool users. Unusually frequent defecations, including liquid stools, had occurred 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 attendance at day care, 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 wave pool. The investigators concluded that the outbreak of cryptosporidiosis was likely caused by exposure to faecally contaminated wave 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 (MMWR, 1994). Fifty-five per cent of 31 pool patrons 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, hyperchlorinated the water and prohibited persons with diarrhoea from swimming in the pool.

3.3.3 Risk assessment

Protozoan pathogens such as *Giardia* have a stage found in the intestine of humans and some animals called the trophozoite stage. As these organisms are discharged to natural environments, such as water, they assume a cyst stage, which is very resistant to natural environmental stresses and disinfectants. The cysts are 4–12 µm in diameter. 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* has a stage that is related to gastroenteritis and an oocyst stage that occurs outside of the body. *Cryptosporidium* oocysts are 4–6 µm in diameter and are much more resistant to chlorine than *Giardia* cysts. If oocysts are ingested, there is a 7-day incubation period before symptoms appear. The illness lasts about 10–14 days, with symptoms of diarrhoea, vomiting, fever and abdominal cramps. In patients infected with human immunodeficiency virus (HIV), cryptosporidiosis is generally chronic and more severe than in immunocompetent persons (Petersen, 1992).

The *Cryptosporidium* infectious dose that affects 50% of the challenged population of humans is about 132 oocysts. 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 infectious 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 6 months (Table 3.6).

Table 3.6: Protozoan exposure factors associated with swimming pools

Agent	Density shed during infection	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

3.3.4 Risk management

Giardia cysts and *Cryptosporidium* oocysts are very resistant to chlorine (Lykins et al., 1990). *Giardia* must be exposed to a chlorine concentration of 5 mg/litre for 30 min (pH 7, 5 °C) for a 99% reduction to be achieved. *Cryptosporidium*, on the other hand, requires chlorine concentrations of 30 mg/litre for 240 min (pH 7, 25 °C) for a 99% reduction to be achieved.

Ozone is more effective for inactivating *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 (pH 7, 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 (pH 7, 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 secured during treatment before residual ozone removal and return to the pool.

Another approach to eliminating cysts and oocysts is through the use of filtration. *Cryptosporidium* oocysts are effectively 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. Statistics on removal efficiency during filtration should be interpreted with caution. Removal and

inactivation of cysts 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.

The outbreaks of giardiasis and cryptosporidiosis among pool swimmers have been linked to pools contaminated by sewage or AFRs. Pool maintenance and appropriate disinfection levels are easily overwhelmed by AFRs or sewage intrusion; therefore, the only possible response to this condition, once it has occurred, is to prohibit 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. However, the best intervention is to prevent AFRs 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, spas and similar recreational-water environments are summarized in Table 3.7.

Table 3.7: Bacteria and associated infections/diseases found in swimming pools and spas

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

3.4.1 Legionella *spp.*

1) Risk assessment

Legionella are Gram-negative, non-spore-forming, motile, aerobic bacilli. Under natural conditions, they are approximately 0.3–0.9 µm × 2–20 µm or more in size. *Legionella* are natural inhabitants of fresh water. Sources of inoculum may include water, HVAC equipment serving the room in which the spa is located and possibly bathers. Inhalation of contaminated aerosols appears to be the sole route of exposure. There is also evidence to suggest that aerosolized amoebas colonized with *Legionella* can provide a concentrated inoculum and therefore be a more significant source of infection than the direct inhalation of aerosolized *Legionella*.

Legionella spp. can cause two distinct syndromes — Legionnaire's disease and Pontiac fever, collectively referred to as legionellosis (APHA, 1989). Legionnaire's disease is characterized as a form of pneumonia. General risk factors for the illness include males 50 years of age or older,

chronic lung disease, cigarette smoking and excess consumption of alcohol. Specific risk factors include frequency of spa use and length of time spent in or around spas. Although the attack rate is less than 1%, mortality among hospitalized cases can be as high as 15%. Pontiac fever is a non-pneumonic, non-infectious, 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. Ninety per cent of cases of legionellosis are caused by *L. pneumophila*.

Most of the fraction of legionellosis associated with recreational-water use appears to be associated with spas (Groothuis et al., 1985; Althaus, 1986). Outbreaks in swimming pools and ambient recreational waters have never been reported (Marston et al., 1994). Spa waters and associated equipment create an ideal habitat (warm, nutrient-containing, aerobic water) for the selection and proliferation of *Legionella*. Dose–response experiments using animals suggest that extremely high doses (~10^7) are required to initiate infection and disease (O'Brien & Bhopal, 1993).

2) Risk management

It is extremely difficult to control the growth of *Legionella* in spas. Free chlorine residuals must be at least 1.0 mg/litre at all times. Filters should be backwashed frequently. Novel features such as water sprays, etc., in pool facilities should be periodically cleaned and flushed with a solution of 5–10 mg of hypochlorite per litre (PWTAG, 1999). HVAC systems serving the room in which the spa is located should be cleaned and disinfected regularly. Rooms housing spas should be well ventilated to avoid an accumulation of *Legionella* in the indoor air. In facilities where disinfection is undesirable or impossible (e.g., hot springs), the spa and ancillary equipment should be drained regularly and thoroughly cleaned. Patrons should be warned that the risk of illness increases with bather density and time spent in the spa and 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. Shower water should preferably be stored at 60 °C to reduce the growth of *Legionella* and other thermophilic microorganisms. High-risk individuals should be cautioned about the risks of exposure to *Legionella* in or around pools and spas.

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 spas (Jacobson, 1985), the surrounding environment can be a source of contamination. The warm, moist environment on decks, drains, benches and floors provided by spas provides an ideal environment for the growth of *Pseudomonas*. It is also likely that bathers pick up the organisms on their feet and hands and transfer them to the water. In water, bathers can be a source of nutrients required for the growth of *P. aeruginosa.* It has been proposed that the high water temperatures and turbulence in whirlpools promote perspiration and desquamation. These materials protect organisms from exposure to disinfectants and contribute to the organic load, which in turn reduces the disinfection residual and acts as a source of nutrients for the growth of *P. aeruginosa*. *P. aeruginosa* can grow well up to temperatures of 41 °C (Price & Ahearn, 1988).

P. aeruginosa is frequently present in whirlpool spas, as it is able to withstand high temperatures and disinfectants and to grow rapidly in nutrient-containing waters (Ratnam et al., 1986; Price & Ahearn, 1988). In one study, *P. aeruginosa* was isolated from seven commercial whirlpools (Price & Ahearn, 1988). In the majority of whirlpools, concentrations ranged from 10^2 to 10^5 per ml. Recommended disinfection levels were not maintained in any of the pools. In the same study, two residential whirlpools developed densities of 10^4–10^6 per ml within 24–48 h following stoppage of disinfection.

In spas, 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 have been linked to spas. Infection of the hair follicles of the skin with *P. aeruginosa* produces a pustular rash, which generally appears under surfaces covered with swim wear (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, dilutes 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 in spas 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 (Hudson et al., 1985; Ratnam et al., 1986). 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 infectious dose for healthy individuals is greater than 1000 organisms per ml (Price & Ahearn, 1988; Dadswell, 1997).

In swimming pools, the primary health effect is otitis externa or swimmer's ear, although folliculitis has been reported (Ratnam et al., 1986). Otitis externa is characterized by inflammation, swelling, redness and pain in the external auditory canal. Risk factors reported by Seyfried & Cook (1984) and van Asperen et al. (1995) to increase the occurrence of otitis externa in ambient waters 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. 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.

The true incidence of illnesses associated with *P. aeruginosa* in pools and spas is difficult to determine. Since the symptoms are mild and self-limiting, most patients do not seek medical attention. Nevertheless, outbreaks do appear to be widespread. Most outbreaks linked to pools and

spas appear to occur in temperate climates during the winter months. This probably reflects the increased tendency to use these facilities during the winter (Ratnam et al., 1986).

2) Risk management

Adequate disinfectant residuals and routine maintenance are the key elements to controlling *P. aeruginosa* in swimming pools, spas and whirlpools (see chapter 5). While maintaining residuals in pools is relatively easy, the design and operation of spas make it difficult to achieve adequate residuals in these facilities. Under normal operating conditions, recommended residuals can quickly dissipate (Highsmith et al., 1985; Price & Ahearn, 1988). In all facilities, frequent monitoring and adjustment of pH and disinfectant levels are essential. The adequacy of disinfection should be verified routinely using heterotrophic plate counts (in disinfected pools and spas) and faecal coliform (or *E. coli*) tests (in all pools and spas), as well as tests for specific organisms such as *P. aeruginosa* (Crandall & Mackenzie, 1984). Faecal coliform and *E. coli* concentrations of less than 1 per 100 ml should be readily achievable through good management practices. *P. aeruginosa* concentrations of less than 1 per 100 ml should be readily achievable in continuously disinfected pools or spas, whereas a guideline concentration of less than 10 per 100 ml is more realistic for pools and spas without residual disinfectant. Routine, thorough cleaning of surrounding surfaces that could harbour pathogens will also help to reduce the spread of *P. aeruginosa*. In addition, swimming pool and spa operators should require users to shower before entering the water and, where possible, control the number of bathers and their duration of 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 that have cell walls with a high lipid content. This feature enables them to 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 spas. 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 spa use in normally healthy individuals have been linked to other atypical mycobacteria. For example, *M. avium* in spa 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 follows the inhalation of heavily contaminated aerosols generated by the spa. It is likely that detected cases are only a small fraction of the total number of cases. Amoebas 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, it is essential that recommended disinfection

residuals in spas and pools be maintained at all times in order to reduce the risks of acquiring swimming pool granuloma or respiratory illness caused by atypical *Mycobacterium* spp. Thorough cleaning of surfaces and materials around pools and spas where the organism may persist is also necessary. In addition, occasional shock chlorination may be required to eradicate mycobacteria accumulated in biofilms within pool or spa components (Aubuchon et al., 1986). In spas where the use of disinfectants is undesirable or where it is difficult to maintain an adequate disinfectant residual, superheating spa water to 70 °C on a daily basis during periods of non-use may help to control *M. marinum* (Embil et al., 1997). Bathers should be encouraged to use handrails when entering and exiting pools and spas to avoid scrapes and scratches, which can act as an entry point for infectious *Mycobacterium* spp. Immunocompromised individuals should be cautioned about the risks of exposure to atypical mycobacteria in and around pools and spas.

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. The latter two are considered to be, at most, only low-grade pathogens (Duerden et al., 1990).

Humans are the only known reservoir of *S. aureus*. About 15% of normal healthy individuals carry it in their noses and throats (Sheagren, 1984). It can be found on the anterior nasal mucosa of 40–50% of healthy adults and also in the throats of many of them, in the faeces of approximately 20% and on the skin of between 5% and 10%. Robinton & Mood (1966) found that *S. aureus* organisms were shed by bathers under all conditions of swimming. Coagulase-positive *Staphylococcus* strains of normal human flora have been found in chlorinated swimming pools and marine waters with coliform counts that pass bathing water standards (Rocheleau et al., 1986). Although *S. aureus* is resistant to many environmental stresses and able to survive for long periods of time in water, it is not usually able to grow in water. Diaper & Edwards (1994) demonstrated that *S. aureus* survived for several days when inoculated in filtered autoclaved fresh water.

The presence of *S. aureus* in swimming pools has been linked to skin rashes, wound infections, urinary tract infections, eye infections, otitis externa, impetigo and other infections (Calvert & Storey, 1988; Rivera & Adera, 1991). Infections are acquired through contact with bathers with purulent discharge from lesions or who are asymptomatic carriers of *S. aureus*. 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*.

2) Risk management

Adequate inactivation of potentially pathogenic *S. aureus* in swimming pools can be attained by maintaining free residual chlorine levels greater than 1.0 mg/litre (Keirn & Putnam, 1968; Rivera &

Adera, 1991) or equivalent disinfection efficiency. Filters not subjected to thorough chlorine disinfection should be regularly dosed with a high concentration of chlorine during backwashing and checked regularly for microbiological contamination (Calvert & Storey, 1988). There is presumptive evidence that showering before pool entry can reduce the shedding of staphylococci from the skin into the pool (Robinton & Mood, 1966). 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. Favero et al. (1964) have recommended a maximum allowable concentration of total staphylococci in recreational waters of less than 100 per 100 ml. In studies by Keirn & Putnam (1968), iodine and free residual chlorine concentrations greater than 1.0 mg/litre reduced the staphylococci counts to less than 10 organisms per 100 ml during periods of use. It was therefore suggested by the authors that a more realistic limit might be 30 staphylococci per 100 ml in no more than 15% of the samples taken during normal operation.

3.4.5 Leptospira interrogans

1) Risk assessment

Leptospira are aerobic, motile, helicoidal, flexible spirochaetes, usually 6–20 µm long and approximately 0.1 µm in diameter, with semicircular hooked ends. There are two recognized species: the pathogenic species *Leptospira interrogans*, which includes about 180 serotypes, and the free-living non-pathogenic species *L. biflexa*.

Leptospira contaminate water via the urine of domestic animals, such as cattle, pigs and dogs, and wild animals, such as rats. Humans are dead-end hosts in the chain of transmission, with possible incidents of person-to-person transmission rarely being reported. Pathogenic leptospires can survive for days to months or longer in neutral or slightly alkaline waters. They do not survive well in salt water or in environments with low pH or low humidity (Weyant et al., 1999).

Leptospirosis (Weil's disease or haemorrhagic jaundice) is usually characterized by sudden onset of fever and chills, severe headache, muscular pain, abdominal pain, nausea and conjunctivitis. Other symptoms may include aseptic meningitis, conjunctival haemorrhage, rash, jaundice and cough with blood-stained sputum. The organism enters the body either through abraded skin or by contact with mucous membranes. The incubation period is from 10 to 12 days (range 3–30 days), and symptoms persist for approximately one week. Prolonged mental health symptoms may occur after leptospirosis, but the relationship is not well documented.

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; Dufour, 1986; de Lima et al., 1990). Domestic or wild animals with access to the implicated waters were the probable sources of *Leptospira* responsible for most of the outbreaks.

2) Risk management

The risk of leptospirosis can be reduced by preventing direct animal access to swimming pools and informing users about the hazards of swimming in water that is accessible to domestic and wild animals. Outbreaks are not common; thus, it appears that the risk of leptospirosis associated with swimming pools and spas is low. Normal disinfection of pools is sufficient to inactivate *Leptospira* spp.

3.5 Non-faecally derived viruses

Infections associated with non-enteric viruses found in swimming pools and spas are summarized in Table 3.8.

Table 3.8: Non-enteric viruses and associated infections found in swimming pools and spas

Organism	Infection	Source
Molluscipoxvirus	Molluscum contagiosum	Bather shedding on benches, pool or spa decks, flutter boards
Human papilloma virus	Plantar wart	Bather shedding on pool and spa decks and floors in showers and change rooms

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 much less than the total number. Lesions are most often found on the arms, 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 spas and similar facilities 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 spa 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 regular cleaning and sanitation of surfaces in facilities that are prone to contamination can reduce the spread of the disease. In extreme cases, facility staff could also be trained to recognize the infection.

3.5.2 Human papilloma virus

1) Risk assessment

Human papilloma virus (HPV) is a double-stranded DNA virus in the family Papovaviridae. The virions are spherically shaped and approximately 55 nm in diameter. The virus causes benign cutaneous tumours in humans. Infections that occur on the sole (or plantar surface) of the foot are referred to as verruca plantaris or plantar warts. They are extremely resistant to freezing and desiccation and thus can remain infectious for many years. The incubation period of the virus remains unknown, yet 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 spas. 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 locker room floors contaminated with infected skin fragments (Conklin, 1990; Johnson, 1995). HPV is not transmitted via swimming pool or spa waters.

2) Risk management

The primary source of HPV 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 sanitizing footbaths, wearing of sandals in showers and change rooms and regular cleaning of surfaces in swimming pool facilities that are prone to contamination can reduce the spread of the virus. Facilities should be designed to control the transport of debris on footwear into change rooms.

3.6 Non-faecally derived amoebas

Table 3.9 summarizes the amoebas found in swimming pools and spas and their associated infections.

Table 3.9: Amoebas and associated infections found in swimming pools and spas

Organism	Infection	Source
Naegleria fowleri	Primary amoebic meningoencephalitis Granulomatous amoebic encephalitis	Pools and spas, including water and components
Acanthamoeba spp.	Acanthamoeba keratitis	Aerosols from HVAC systems

3.6.1 Naeglaria 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. In water, concentrations of the amoebas increase as they feed on aquatic bacteria. In temperate climates, the amoebas can overwinter as cysts in the bottom sediments of bodies of fresh water and swimming pools.

N. fowleri causes primary amoebic meningoencephalitis (PAM). Infection is acquired by exposure to polluted water in ponds, swimming pools and artificial lakes (Martinez & Visvesvara, 1997; Szenasi et al., 1998). Victims are usually healthy young children and 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 amoebas 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 exposure. 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). PAM is an extremely rare disease. Wellings (1977) has estimated that only one case of PAM occurs for every 2.6 million exposures to water containing *N. fowleri*.

2) Risk management

Risk of infection can be reduced by reducing the occurrence of the causative agent through proper cleaning, maintenance, coagulation–filtration and disinfection of swimming pools. Transmission of PAM in spas has not been reported; however, because *N. fowleri* grows at high temperatures and is resistant to disinfection, spas could be a source of exposure. Aerosols generated by spas may also contain *N. fowleri*. Users should be aware that the risk of infection increases with time spent in the pool or with immersion of the head. *N. fowleri* has also been isolated from air conditioning units. Therefore, HVAC systems serving the pool or spa facilities should be cleaned and disinfected regularly.

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 chlorinated 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 infections, affecting the brain and the cornea, respectively (Martinez & Visvesvara, 1997; Szenasi et al., 1998). *Acanthamoeba* spp. are responsible for granulomatous amoebic encephalitis (GAE), a subacute or chronic infection that can occur in people who are immunosuppressed as a result of acquired immune deficiency syndrome (AIDS), chemotherapy, or drug or alcohol abuse. GAE is invariably fatal. The route of infection in GAE is unclear, although invasion of the brain may be via the blood following a primary infection elsewhere in the body, possibly the skin or respiratory tract. GAE is extremely rare; only slightly more than 100 cases have been reported worldwide (Martinez & Visvesvara, 1997). A recent history of contact with water has not been seen in patients with GAE (Marshall et al., 1997).

Several species of *Acanthamoeba* can also produce a chronic sight-threatening ulceration of the cornea called acanthamoeba keratitis, mostly in previously healthy individuals who wear contact lenses or with minor corneal abrasions. Infection follows the colonization of the internal surface of the contact lens. The primary source of acanthamoebic infection of the cornea in contact lens wearers is thought to be tapwater that is used to clean storage cases or to prepare solutions. However, acanthamoeba keratitis may also be transferred via hot tubs, chlorinated swimming pools and air conditioning units (Marshall et al., 1997).

2) Risk management

Although *Acanthamoeba* cysts are resistant to chlorine-based disinfectants, they can be removed by filtration. Thus, it is unlikely that properly operated swimming pools and spas would contain sufficient numbers of cysts to cause infection in normally healthy individuals. Nevertheless, as a precautionary measure, persons wearing contact lenses should remove the lenses before entering the water. Immunosuppressed individuals who use swimming pools or spas should be aware of the increased risk of GAE.

3.7 Non-faecally derived fungi

Infections associated with fungi found in swimming pools and spas are summarized in Table 3.10.

Table 3.10: Fungi and associated infections found in swimming pools and spas

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

3.7.1 Trichophyton *spp. and* Epidermophyton floccosum

1) Risk assessment

Epidermophyton floccosum and various species of fungi in the genera *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 "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 is usually transmitted by physical contact with surfaces, such as floors in public showers, change rooms, etc., contaminated with infected skin fragments (PWTAG, 1999). 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 dermatophytes in swimming pool or spa 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 sanitizing foot baths, wearing of sandals in showers and change rooms and regular sanitation of surfaces in swimming pool facilities that are prone to contamination can reduce the spread of the fungi (Al-Doory & Ramsey 1987; Public Health Laboratory Service Spa Pools Working Group, 1994). People with severe athlete's foot or similar dermal infections should not frequent public swimming pools or spas. Routine disinfection appears to control the spread of these fungi in swimming pools and spas (Public Health Laboratory Service Spa Pools Working Group, 1994).

3.8 References

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