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## *Section III*

# Water-related zoonosis disease impacts — geographical prevalence

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Surveillance of waterborne infectious disease has become an important aspect of public health practice. Although not all countries do so, there is a growing recognition of the value of detecting and quantifying the occurrence of cases and outbreaks of disease. A formal surveillance programme combined with aggressive epidemiological investigations will produce data leading to the identification of emerging pathogens, sources of etiological agents, and susceptible populations. The Legionnaire's disease pneumonia in 1976 is a classic example of identifying an emerging pathogen and the follow-up tracking of the disease. Surveillance of subsequent outbreaks identified various exposure scenarios that resulted in legionellosis. Identification of sources and risks associated with this microbe has led to better risk assessments and control measures for *Legionella*. A similar history is found for the zoonotic protozoan

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pathogen, *Cryptosporidium*. This microbe was well known to veterinarians as an intestinal disease in cattle, especially calves. Epidemiological evidence collected during outbreaks clearly established the linkage between the water environment and cryptosporidiosis in humans. These emerging and re-emerging diseases, such as cholera, might not have received broad attention without active surveillance systems in place. The globalization of food markets and the potential rapid dissemination due to the ease of international travel are yet other reasons to maintain active surveillance of infectious disease.

On a local or regional basis, surveillance of infectious disease will frequently alert authorities to a breakdown in water treatment processes or to the poor design of waste management facilities. The increase in zoonotic disease outbreaks should serve to strengthen efforts to maintain a watch for infectious disease outbreaks and should encourage a closer working relationship between medical and veterinary epidemiologists to improve surveillance systems.

The first chapter in this section describes emerging and re-emerging disease in Africa, Asia, and South America. Most of the cases and outbreaks described are associated with bacteria, perhaps giving a false impression that viruses and protozoa may not be prevalent on these continents, which is well known not to be true. The authors do point out that this imbalance may be a function of the types of literature reviewed. The difficulties of addressing the collection of health data are discussed, and solutions to some of these problems are listed. The most important of these was the need to intervene with education and training programmes to enable countries to take appropriate steps to reduce waterborne disease.

Chapter 7 discusses water shortages in Mexico, Central America, and the Caribbean and the effect this has had on the health of individuals in this region. The chapter closes with a suggestion that an international network should be created, to share information between countries and to use the information to make decisions about the control of water-related zoonotic diseases.

In chapter 8, zoonotic disease outbreaks associated with drinking-water are described, as well as the deficiencies related to inadequate or interrupted treatment of surface waters. Outbreaks associated with bacteria were associated with untreated groundwater, inadequately disinfected groundwater, and distribution system contamination. It is suggested that better surveillance and outbreak investigation will lead to the reduction of waterborne risks.

The final chapter provides descriptions of zoonotic diseases, their etiological agents, and treatment.

# 6

## Tropical organisms in Asia/Africa/South America

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### 6.1 INTRODUCTION

Zoonotic infections, many transmitted by water, pose a serious threat to global health and the economy. One-third of the world's population lives in countries with some level of water stress, a number that could increase to two-thirds by the year 2025. Twenty per cent of the world's population in 30 countries faces water shortage (Tema 2001). Water use is expected to increase by 40% over the next two decades, and it is estimated that 3 billion people will face water shortages by then. The impact of waterborne and foodborne (Slifco *et al.* 2000) zoonotic pathogens on human health is expected to be significant.

In Africa, Asia, and Latin America, at least 600 million urban dwellers live in unhealthy homes or neighbourhoods. Over a billion people lack access to safe

drinking-water, increasing their vulnerability to diarrhoeal and parasitic diseases (WHO 1999). Parasitic diseases in infancy can lead to systemic immune system imbalances, increasing stunting and reducing cognizance (Berkman *et al.* 2002). Worm infestations also diminish the efficacy of certain vaccines (e.g., tuberculosis, human immunodeficiency virus [HIV], and malaria; Markus 2002). In a series of 1377 refugees and asylum seekers entering Sweden, intestinal parasites were more frequently recovered in refugees from south-east Asia, Africa, and Latin America (infection rates of 48%, 43%, and 42%, respectively) than in those from Eastern Europe (22%) and the Middle East (32%) (Benzeguir *et al.* 1999). Lack of resources can lead to a resurgence of disease in disease-free areas. According to WHO (1999), success can breed complacency, particularly when diseases have low visibility and limited impact. For waterborne zoonoses, key determinants of human health can lie outside the direct control of the health sector, being rooted in inadequate sanitation, unwholesome water supply, poorly regulated livestock and agricultural practices, environmental and climate change, education, etc. In order to execute effective surveillance, prevention, and control of waterborne zoonoses, useful background information on occurrence must be available.

This chapter highlights organisms (parasites, bacteria, and viruses) found in Asia, Africa, and South America that either are zoonotic or possess the potential to be zoonotic.

## 6.2 PARASITES

### 6.2.1 *Ascaris*

The ova of many genera of zoonotic parasites can be found in surface water and groundwater. In properly operated conventional water treatment plants, however, they are excluded from drinking-water by their size ( $>30\ \mu\text{m}$ ). Water is not regarded as a major route of transmission for *Ascaris*, although *Ascaris* ova can be found in the air and in dust and can be transferred to uncovered drinking-water sources. *Ascaris* ova are sticky and can adhere to items such as utensils, furniture, fruit, vegetables, money, door handles, and fingers (Kagei 1983). *Ascaris* infections are widespread throughout many parts of Asia, Africa, and South America. The risk of transferring other geohelminth ova, including *Trichuris* and *Taenia* spp., to uncovered drinking-water sources must also be recognized.

### 6.2.2 *Cryptosporidium* and *Giardia*

*Giardia duodenalis* cysts, *Cryptosporidium* spp. oocysts, and spores of microsporidia have also been detected in aquatic environments; however, their smaller size (range 1–17 µm) allows them to penetrate water treatment systems and cause epidemic outbreaks of waterborne disease following the consumption of treated drinking-water (Table 6.1; Smith 1998). These protozoan parasites parasitize the enterocytes, causing an imbalance in food and fluid absorption, which can lead to diarrhoea.

Table 6.1. Some protozoan parasites and the waterborne route of transmission

Organism	Disease/symptoms	Geographic distribution	Transmissive stage (size range) and route of infection
<i>Giardia duodenalis</i>	diarrhoea, malabsorption	widespread	cyst (8–12 µm), ingestion
<i>Cryptosporidium parvum</i>	diarrhoea	widespread	oocyst (4.5–5.5 µm), ingestion
<i>Cyclospora cayetanensis</i>	diarrhoea	widespread	oocyst (8–10 µm), ingestion
<i>Entamoeba histolytica</i>	dysentery, liver abscess	widespread	cyst (9–14.5 µm), ingestion
Microsporidia	diarrhoea, hepatitis, peritonitis, keratoconjunctivitis, etc.	widespread	spore (1.8–5.0 µm), ingestion/contact with eye
<i>Toxoplasma gondii</i>	lymphadenopathy, fever, congenital infections	widespread	oocyst (10–12 µm), ingestion
<i>Blastocystis hominis</i> ; <i>Blastocystis</i> sp.	diarrhoea	widespread	cyst (4–6 µm), ingestion
Free-living amoebae (e.g., <i>Naegleria fowleri</i> , <i>Acanthamoeba</i> spp.)	primary amoebic meningoencephalitis, granulomatous amoebic encephalitis, keratitis	widespread	cyst (7–18 µm for <i>Naegleria</i> ; 15–28 µm for <i>Acanthamoeba</i> ), inhalation, contact with conjunctiva

*Giardia* and *Cryptosporidium* have become significant waterborne pathogens in the developed world for three reasons. First, giardiasis and cryptosporidiosis are indigenous infections with a low infectious dose; second, densities of environmental contamination with infective cysts and oocysts [(oo)cysts] are sufficient to pollute the aquatic environment; and third, (oo)cysts are small enough to penetrate water treatment processes and are less sensitive to the disinfectants commonly used in water treatment. Undoubtedly, waterborne

outbreaks of protozoan parasitic infections following contamination from sewage, wastewater effluent, muck spreading, slurry spraying, etc., leading to the contamination of potable water, pose significant problems for both the developed and developing countries of the world. For example, two waterborne cryptosporidiosis outbreaks occurred in Japan (Smith and Rose 1998).

Potential sources of oocysts and cysts detected in raw water (adapted from Smith *et al.* 1995 with permission) are as follows:

- contribution from infected animals;
- pasturing of infected livestock;
- infected wild animals, including “on-farm” rodents;
- watering of infected animals;
- infected domestic/companion animals;
- carriage by water-roosting or aquatic birds;
- contribution from human activities;
- disposal of contaminated faeces and non-controlled effluents from farms;
- accidental spillages from poorly constructed slurry stores and middens;
- slurry spraying and muck spreading;
- intensive husbandry of livestock;
- disposal of faeces from infected animals at abattoirs;
- disposal of sewage sludge to land; and
- disposal of contaminated backwash sludge.

Surveys of occurrence in a variety of countries indicate that *Cryptosporidium* oocysts and *Giardia* cysts can occur commonly in the aquatic environment (Smith *et al.* 1995; Smith and Rose 1998; Gold and Smith 2002) (Tables 6.2 and 6.3). (Oo)cysts have been detected in wastewater, surface waters, groundwater, springs, and drinking-water samples, including those treated by disinfection alone, filtration, direct filtration, and conventional methods. Data collated by Smith and Rose (1998) and Gold and Smith (2002) indicate that *Cryptosporidium* oocysts and *Giardia* cysts occur at similar densities in various countries, with the highest oocyst densities being found in surface waters, and that (oo)cysts can be found in drinking-water in the absence of increased levels of disease among consumers.

Standardized methods, equipment, and commercial kits are available for detecting *Cryptosporidium* oocysts and *Giardia* cysts, but not for microsporidian spores. Equipment and commercial kits are expensive, particularly for developing countries; however, as for bacteriological and viral methods, standardization is paramount in order to determine the significance of occurrence and the threat to public health of waterborne (oo)cysts.

Table 6.2. Occurrence and density of *Cryptosporidium* oocysts and *Giardia* cysts in surface waters (adapted from Smith and Rose 1998; Gold and Smith 2002)

Country	Number of samples	Occurrence of <i>Cryptosporidium</i> oocysts (% samples positive)	Density of <i>Cryptosporidium</i> oocysts (oocysts/litre)	Occurrence of <i>Giardia</i> cysts (% samples positive)	Density of <i>Giardia</i> cysts (cysts/litre)	Year
USA	11	100	2–112	–	–	1987
USA	222	–	–	43	0.5–1	1989
USA	101	24	0.005–252.7	–	–	1990
UK (Scotland)	262	40.5	0.006–2.3	–	–	1990
USA	35	97.1	0.18–63.5	–	–	1991
Germany	9	78	–	–	–	1991
UK	691	52.2	0.04–3	–	–	1992
UK	375	4.4	0.07–2.75	–	–	1992
UK (Scotland)	53	–	–	33	0.01–1.05	1993
Canada	22	0	–	32	–	1993
Spain	8	50	<0.01–0.31	63	<0.01–0.21	1993
Canada	249	–	0.005–0.34	100	0.005–0.34	1996
Canada	1760	6.1	–	21	–	1996
Honduras	–	–	0.58–2.6	–	3.8–21	1998
Taiwan	31	72.2	–	77.8	–	1999
Czech Republic	–	–	0–74	–	0–4.85	2000

A particular feature of protozoan parasites is that their transmissive stages cannot be amplified readily *in vitro*, and standard methods reflect this problem. Detecting small but significant numbers of organisms in water concentrates limits the range of tests that can be performed on a defined number of organisms, particularly when attempting to extract sufficient DNA from a sample for molecular typing. Currently, the genus *Cryptosporidium* (and, to a lesser extent, *Giardia*) is being revised, which leads to much confusion. Recent genetic analyses reveal that more than one species of *Cryptosporidium* can infect susceptible immunocompromised (*C. meleagridis*, *C. felis*, and *C. muris*; Morgan *et al.* 2000; Guyot *et al.* 2001; Pedraza-Diaz *et al.* 2001a, 2001b; Cacciò *et al.* 2002; Gatei *et al.* 2002) and immunocompetent (*C. meleagridis* and *C. felis*; Katsumata *et al.* 2000; Pedraza-Diaz *et al.* 2001a, 2001b; Xiao *et*

*al.* 2001) human hosts, but *C. parvum* and *C. hominis* remain the most common species infecting humans. Additionally, the transmissive stages of differing human-infectious species possess similar morphologies and morphometries and cannot be distinguished using the standardized methods for detecting oocysts in water concentrates.

Table 6.3. Occurrence and density of *Cryptosporidium* oocysts and *Giardia* cysts in treated waters<sup>a</sup> (adapted from Smith and Rose 1998; Gold and Smith 2002)

Country	Number of samples	Occurrence of <i>Cryptosporidium</i> oocysts (% samples positive)	Density of <i>Cryptosporidium</i> oocysts (oocysts/litre)	Occurrence of <i>Giardia</i> cysts (% samples positive)	Density of <i>Giardia</i> cysts (cysts/litre)	Year
USA	36	17	0.005–0.017	0	–	1991
USA	82	26.8	–	16.9	–	1991
UK (Scotland)	15	7	0.006	–	–	1995
UK (Scotland)	106	–	–	19	0.01–1.67	1993
Spain	9	33	<0.01–0.02	22	<0.01–0.03	1993
Brazil	18	22.2	–	–	–	1993
Canada	42	5	–	17	–	1993
Canada	249	–	–	98.5	0.045–1.72	1996
Canada	1760	3.5	–	18.2	–	1996
Germany	12	66.7	0.008–1.09	83.3	0.02–1.03	1996
UK	209	37	0.007–1.36	–	–	1998
Taiwan	31	38.5	–	77	–	1999

<sup>a</sup> Waters for potable supply receive different treatments in different areas of the world; whereas some of the waters in this table received a number of treatments before being considered usable for potable supply, others may have received minimal treatment.

Clinical parasitological studies of protozoan parasites have been scarce and sporadic in many parts of Asia, Africa, and South America, and few generalized conclusions regarding transmission can be drawn from them. In Guinea-Bissau, *Cryptosporidium* was the most important risk factor for developing childhood diarrhoea (Sodemann *et al.* 1999). *Cryptosporidium* was the most common

pathogen (17%) identified in stools analysed from 75 consecutive HIV-seropositive patients with chronic diarrhoea admitted to a Nairobi hospital (Mwachari *et al.* 1998). A survey of intestinal parasites among the HIV-positive asymptomatic injecting drug users in north-east India revealed that *Cryptosporidium* sp. (94.4%) and *Isospora* sp. (10.7%) were most commonly seen (Anand *et al.* 1998). In a hospital-based study in India, 7.2% (151/2095) of stool samples were positive for *Cryptosporidium* (Nath *et al.* 1999). In Malaysia, 2% of the 237 stool specimens from children receiving cancer chemotherapy were found to be positive for *C. parvum* (Menon *et al.* 1999). Of 31 water samples collected from nine potable water treatment plants in Taiwan, 77.8% were positive for *Giardia* cysts and 72.2% for *Cryptosporidium* oocysts (Hsu *et al.* 1999).

A study in Japan showed that *Cryptosporidium* oocysts were detected in 35% (9/26) of filtered water samples (geometric mean concentration 1.2 oocysts/1000 litres) and *Giardia* cysts in 12% (3/26; geometric mean concentration 0.8 cyst/1000 litres). The estimated  $\log_{10}$  removal efficiency of *Cryptosporidium* oocysts and *Giardia* cysts by rapid sand filtration was 2.47 and 2.53, respectively (Hashimoto *et al.* 2002). The 1996 cryptosporidiosis outbreak in Ogose, Japan, forced wastewater treatment authorities to rethink the relevance of *Cryptosporidium* contamination levels in wastewater and watersheds and to develop countermeasures in wastewater treatment plants. A nationwide survey of *Cryptosporidium* occurrence in raw and treated wastewater identified relatively low densities (Suwa and Suzuki 2001).

Genetic characterization of 15 *G. duodenalis* isolates (8 from Anhui Province, China, and 7 from Seoul, Korea) revealed the same two major lineages, Assemblages A and B, described by Thompson *et al.* (2000). All Korean isolates fell into Assemblage A, whereas four Chinese isolates were in Assemblage A and four in Assemblage B. Two *G. microti* isolates and two dog-derived *Giardia* isolates fell into Assemblage B, but *G. ardeae* and *G. muris* were unique (Yong *et al.* 2000).

### 6.2.3 Toxoplasma

A Central American outbreak of toxoplasmosis, associated with the consumption of oocyst-contaminated water, has been documented. In 1979, 32 US Army soldiers demonstrated evidence of symptomatic infection with *Toxoplasma* after their return from manoeuvres in Panama. Epidemiological evidence indicated that the most likely vehicle for transmission was the ingestion of creek water contaminated with oocysts excreted by jungle cats, consumed during manoeuvres in the jungle (Benenson *et al.* 1982). Interestingly, most of the affected individuals claimed to have treated their drinking-water with iodine tablets. Primary chlorination is thought to be

unlikely to either prevent *Toxoplasma* oocysts from sporulating or kill sporulated oocysts. As for most zoonoses in Asia, Africa, and South America, this is likely to be an underestimate, because of the lack of tools for oocyst detection in the environment.

#### **6.2.4 *Blastocystis***

Although *Blastocystis* has often been incriminated as a pathogen, much debate surrounds this classification. Widely distributed, it is found in numerous animal hosts. Prevalence in animal workers is higher (44%) than in a normal population (17%) (Suresh *et al.* 2001), suggesting that close proximity with animals may facilitate transmission (Rajah Salim *et al.* 1999). It has been shown that the *in vitro* induced cysts from cultured vacuolar forms of human isolates of *Blastocystis* (Suresh *et al.* 1993) and cysts from *Blastocystis*-infected patients' stools (Moe *et al.* 1997) can cause experimental infections in laboratory-bred rats and mice, respectively, but the zoonotic potential for *B. hominis* remains undecided. Phylogenetic studies (Yoshikawa *et al.* 2003) indicate that isolates tested from humans and animals appear to be *B. hominis*. Sequence and phylogenetic analysis of partial ssu rDNA of *Blastocystis* from a human, a pig, and a horse, sharing a common subgroup, indicated that the pig and horse isolates were monophyletic and closely related (92–94% identity) to *B. hominis*, suggesting the possibility of *B. hominis* being a zoonosis (Thathaisong *et al.* 2003). Twelve *Blastocystis* isolates from primates, when analysed genetically by polymerase chain reaction (PCR) using diagnostic primers and PCR-restriction fragment length polymorphism (RFLP) of SSUrDNA, showed for the first time genetic similarity between the isolates from primates and the genotypes of *B. hominis*. However, it was unclear whether the isolates examined were zoonotic (Abe *et al.* 2003). Viable cysts have been demonstrated in sewage effluent in Pakistan (Zaman *et al.* 1994) and in Malaysian sewage and rivers (K. Suresh, T.C. Tan, and H.V. Smith, unpublished data).

#### **6.2.5 *Strongyloides***

The free-living (heterogenic) life cycle stages of *Strongyloides stercoralis* can be found in warm, moist soil and in sand filter beds of wastewater treatment works, particularly in warm climates. They have to be differentiated from the plethora of nematodes present in biofilms in these environments. While *Strongyloides* is not normally thought of as a waterborne zoonotic agent, transmission via water may be more frequent than suspected in the warmer climates of Asia, Africa, and South America.

## 6.3 BACTERIA

Many bacteria can be transmitted to humans through water. However, a number of important waterborne bacteria that cause human disease — e.g., *Vibrio cholerae*, *Salmonella typhi*, *Shigella dysenteriae*, and others — arise primarily from human wastes and thus are not considered to be zoonoses. This section presents information on waterborne zoonotic bacteria only.

### 6.3.1 *Salmonella* and *Shigella*

Between 1990 and 1991, of 3222 *Salmonella* strains identified at the National *Salmonella* and *Escherichia* Centre in Kasauli, India, 2894 were from humans, 226 from poultry, 84 from animals, and 18 from reptiles, birds, and other sources. Fifty-three serotypes were identified, including four serotypes reported for the first time in India (*S. kedougou*, *S. VP. bornheim*, *S. kisarawe*, and *S. madras*) (Mahajan *et al.* 1998).

An Indonesian surveillance study, conducted over a 2-year period among 6760 patients with debilitating diarrhoeal diseases, revealed that 587 (9%) of stools were positive for the following bacteria: *Shigella flexneri* (39%), *Salmonella* spp. (26%), *Vibrio* spp. (17%), *Shigella sonnei* (7%), *Campylobacter jejuni* (4.4%), *Salmonella typhi* (3%), and *Shigella dysenteriae* (2.3%).

From January 1983 to December 1992 in Malaysia, 20 874 *Salmonella* isolates were typed into 97 serotypes belonging to 22 Kauffmann-White groups (Yasin *et al.* 1996). The submissions represented a 100% increase over the previous 10-year period.

Studies have consistently shown that non-typhoidal *Salmonella* bacteraemia is more common during the rainy season in tropical Africa; sources and modes of transmission remain unknown, but clustering of cases in the rainy season suggests a waterborne/water-associated route. Gracey *et al.* (1979) found a high rate of carriage among healthy adults and children in Jakarta, Indonesia, where 48% of specimens of river water used for drinking contained *Salmonella* (Graham *et al.* 2000).

Of 62 faecal specimens collected from mountain gorillas (*Gorilla gorilla beringei*) in the Bwindi and Mgahinga National Parks, Uganda, in January 1999, 19% had *Campylobacter* spp., 13% *Salmonella* spp., and 6% *Shigella* spp. The prevalence of positive specimens was not related to the year of habituation of a gorilla group to humans. *Campylobacter*, *Salmonella*, and *Shigella* infections were distributed unevenly among gorilla age classes: 80% of enteropathogens and all *Shigella* (*S. sonnei*, *S. boydii*, and *S. flexneri*) were isolated from subadult and adult gorillas (age range 6.0–11.9 years). The prevalence of *Campylobacter* and *Salmonella* infections among human-habituated gorillas doubled during the last 4 years, and isolation of *Shigella* for the first time from

mountain gorillas may indicate increased anthroozoonotic transmission of these enteropathogens (Nizeyi *et al.* 2001).

### 6.3.2 *Campylobacter*

*Campylobacter jejuni* is a major cause of paediatric diarrhoea in developing countries, where free-range chickens are presumed to be a common source. Peruvian strains, from monthly surveillance and diarrhoeal cases, were compared by RFLP, rapid amplified polymorphic DNA, and Lior serotyping. RFLP analysis of 156 human and 682 avian strains demonstrated identical strains in chickens and humans in 70.7% (29/41) of families, and 35–39% of human isolates from diarrhoeal and non-diarrhoeal cases were identical to a household chicken isolate (Oberhelman *et al.* 2003).

Of a total of 620 samples collected from healthy animals and animal handlers in Calcutta, India, 128 (20.6%) were positive for *Campylobacter* spp. (*C. jejuni* 14.5%, *C. coli* 4%, and *C. lari* 2%). The isolation rate was highest in chickens and ducks (39.3% positive). The isolation rates in diarrhoeic cattle, sheep, and dogs were 22.2%, 33.3%, and 16.6%, respectively; in healthy animals, the rates were 5.3%, 1.4%, and 8.3%. In healthy pigs, the carriage rate of *Campylobacter* spp. was high (37.1%). Of the 140 human faecal samples processed, 10 (7.1%) were positive for *Campylobacter* spp., of which 9 isolates were *C. jejuni* and 1 was *C. coli* (Chattopadhyay *et al.* 2001). Macrorestriction (pulsed-field gel electrophoresis) profiles of human and porcine isolates suggest that *C. hyointestinalis* transmission from pig to human is possible (Gorkiewicz *et al.* 2002).

*Campylobacter* and *Salmonella* accounted for 40% and 24%, respectively, of 1707 diarrhoeal specimens collected over a 7-year period (1989–1996) from Oita district, Japan. Initially, *Campylobacter* was most prevalent. It then decreased, while *Salmonella* continued to increase; at the end of the study period, *Salmonella* was more prevalent than *Campylobacter*. Increases in *Salmonella* were due to the appearance of *S. Enteritidis* (Narimatsu *et al.* 1997).

### 6.3.3 *Escherichia coli*

During October 1992, a large outbreak of bloody diarrhoea affecting thousands of individuals, some of whom died, occurred in South Africa and Swaziland (Isaacson *et al.* 1993). *Escherichia coli* O157 was isolated from 22.5% of 89 stool samples, and epidemiological investigations implicated waterborne spread. In some areas, cases were mainly men who drank surface water from fields, while women and children who drank borehole water were spared. *Escherichia coli* O157 was isolated from 14.3% of 42 samples of cattle dung and 18.4% of

76 randomly collected water samples. The underlying problem seems to have been cattle carcasses and dung washed into rivers and dams by heavy rains after a period of drought.

In a semiurban slum of Varnasi, India, 53.7% of milk samples from supplementary milk feeds of 149 children were bacterially contaminated (*E. coli* [13.4%], *Klebsiella* spp. [5.4%], *Enterobacter* spp. [5.4%], *Pseudomonas aeruginosa* [4.7%], *Shigella* spp. [2.7%], and others [22.1%]), of which 16.1% were potentially enteropathogenic. The rate of contamination was significantly higher ( $P < 0.001$ ) in lower income groups (73.4%), lower castes (69.6%), and illiterate mothers (69.3%) (Ray *et al.* 2000).

#### **6.3.4 Brucella**

From 1992 to 1997, 3532 patients with pyrexia of unknown origin were tested for brucellosis, and 28 (0.8%) were seropositive. Males outnumbered females by 3:1. Seroprevalence was age-related among males, but not among females. The highest number (43%) of positive males belonged to the age group 21–30 years. The majority of patients had titres of 1:160 or 1:256; high titres of 1:512 and 1:1024 were found in 21.4% of patients (Kadri *et al.* 2000).

### **6.4 VIRUSES**

Recently, many zoonotic viruses have emerged in Australia and south-east Asian countries. Increased tourism, greater influx of migrant workers, deforestation, and current intensive farming and husbandry practices in many parts of these regions prove ideal for zoonotic transmission. Below are discussed some emerging zoonotic viral diseases that may have the potential for waterborne transmission.

#### **6.4.1 Menangle virus and Tioman virus**

The new Menangle virus (Family Paramyxoviridae) causes stillbirth, mummification, occasional abortions, and deformities in pigs. The virus was isolated from lung, brain, and heart tissues of infected piglets, and serum from two workers contained high-titre, convalescent-phase neutralizing antibodies to the virus (Philbey *et al.* 1998). Tioman virus was isolated from urine of flying fox (*Pteropus* sp.) found on Tioman Island off the eastern coast of peninsular Malaysia (Chua *et al.* 2001).

#### **6.4.2 Hendra virus**

Recognized in 1994 after an explosive outbreak of severe, fatal respiratory disease, which killed 13 of 20 infected racehorses (Murray *et al.* 1995), Hendra

virus has also been responsible for a human fatality (Rogers *et al.* 1996). Black fruit bats (*Pteropus alecto*), infected subcutaneously, intranasally, or orally, contract subclinical infections. Hendra virus is widely distributed in Australian pteropid bats, with serological evidence of infection in an average of 42% of wild-caught bats, the number of seropositives varying with species (53% of 229 *P. alecto*, 47% of 195 *P. poliocephalus*, 12% of 115 *P. scapulatus*, and 41% of 99 *P. conspicillatus*) and age, but not geographic distribution (H. Field, unpublished data). Based on morphology and preliminary sequencing data of the M and F genes, Hendra virus is a member of the Paramyxoviridae (Hyatt and Selleck 1996).

#### **6.4.3 Australian bat lyssavirus**

Closely related to rabies virus, this virus was first identified in Australia in a fixed brain specimen from a young black flying fox (*Pteropus alecto*) with unusual neurological symptoms. To date, two human infections have been attributed to Australian bat lyssavirus (Hooper *et al.* 1996).

#### **6.4.4 Highly pathogenic avian influenza**

Highly pathogenic avian influenza (fowl plague) is highly infectious and contagious. Waterfowl are reservoirs and are the main source of introduction of the disease into domestic poultry flocks. The recent emergence of a new strain (H5N1) of influenza A virus, the avian flu, in Hong Kong accentuates the importance of south-east Asia as a melting pot for emerging microbial agents. H9N2 viruses were isolated from 4.7% of chickens and a smaller percentage of ducks, geese, domestic and wild pigeons, and quail and from environmental swabs in a market (Shortridge 1999).

#### **6.4.5 Nipah virus**

An outbreak of Nipah virus in Malaysia and Singapore in October 1998 highlights the epidemiological significance of changing environments and movements of animals from their natural habitats. Nipah virus takes its name from a village in peninsular Malaysia where the virus was first isolated from a human victim (Hendra virus is a close relative; Chua *et al.* 2000). The virus persists in low numbers in the island flying fox (*Pteropus hypomelanus*) (Chua *et al.* 2002), a fruit bat, and the Malayan flying fox (*P. vampyrus*). The virus replicates explosively in pigs, causing respiratory and/or neurological syndromes followed by death (Mohd. Nor *et al.* 2000), and causes severe encephalitis in humans (Mohd. Taha 1999). Of the 269 human cases of viral

encephalitis associated with Nipah virus infection reported in Malaysia in 1999, 108 were fatal (Malaysian Ministry of Health 2001). Several possible routes of transmission between farms in farming communities were suggested, including sharing boar semen and transmission by dogs and cats. It is suspected that dogs and cats were infected with urine and excreta from lorries carrying affected pigs and subsequently introduced the virus to uninfected farms.

Of great concern is the changing geographic distribution of fruit bats, which spread Nipah virus and other, as yet unknown, diseases to susceptible communities. In common with most countries in south-east Asia, peninsular Malaysia has a great diversity of bat species. At least 13 species of fruit bat (Suborder Megachiroptera), including two flying fox species and at least 60 species of insectivorous bats (Suborder Microchiroptera), have been described (Aziz *et al.* 2002).

#### **6.4.6 Haemorrhagic fevers and hantavirus**

Hantaviruses (Bunyaviridae) constitute a genus of antigenically, genetically, and epidemiologically related viruses. Laboratory outbreaks due to a causative virus called Seoul virus have been reported in Japan, the Republic of Korea, the People's Republic of China, Russia, France, Belgium, the Netherlands, and the United Kingdom (Lee 1996). Infection with Puumala or PUU virus was also found in the grey-sided vole (*Clethrionomys fufocanus*) in Hokkaido, Japan, but was not associated with human disease (Kariwa *et al.* 1995). Human infection from infected rodents is thought to occur through inhalation of excreta. In 1996, person-to-person transmission was documented in an outbreak of hantavirus pulmonary syndrome (HPS) in southern Argentina caused by the Andes virus. Following the 1993 US outbreak, many South American countries reported either isolated cases or outbreaks of HPS. In 1993, Brazil identified a cluster of cases in a rural area near São Paulo caused by a new virus (Juquitiba virus; Vasconcelos *et al.* 1997). In 1995, Paraguay reported an outbreak, and a new hantavirus (Laguna Negra virus) was isolated from the putative rodent reservoir, *Calomys laucha* (Johnson *et al.* 1997). In South America, HPS affects all ages and both sexes but has a higher prevalence among adult males from rural settings. Reported human seroprevalences vary greatly from region to region, from <2% to 40% among central Paraguayans. Chile and Argentina appear to have more paediatric cases (Pini *et al.* 1998).

#### **6.4.7 Severe acute respiratory syndrome (SARS)**

This coronavirus was reported as a new emerging disease in China in November 2002, subsequently spreading around the world to Canada within 5 months. The World Health Organization has recorded more than 83 380 cases and 770 deaths from SARS. Transmission is primarily through large droplets and aerosols, but

contact with contaminated surfaces has also been suggested. The virus can also be excreted in stools. This raises the question as to whether the SARS virus can survive wastewater and water treatment and whether it can be spread by the waterborne route.

## 6.5 CONCLUSIONS

It is evident from this brief review that, while awareness of zoonotic waterborne diseases exists in Asia, Africa, and South America, different countries have different human health priorities. Bacterial diseases seem to take precedence over viral and protozoal diseases. It is important to remember that these data are based on peer-reviewed scientific publications, which underestimate the amount of work done in the field; much is being done, but its publication is often not a priority.

Water use is expected to increase by 40% over the next two decades, and it is estimated that 3 billion people will face water shortages by then. We may know little about whether the organisms listed are transmitted by the waterborne route or their occurrence in water in Asia, Africa, and South America, but their impact on health, sanitation, and, particularly, drinking-water is potentially great. Of importance is that it is these countries that are likely to suffer greatly from potable water shortages; therefore, we must consider (and control) routes that might contaminate drinking-water with these organisms.

There have been some major diagnostic and epidemiological inroads into investigating zoonotic waterborne bacterial infections, depending on their regional importance; better detection and surveillance systems have reduced the spread of zoonotic waterborne bacterial infections. Our literature searches suggested that while there are more publications on clinical and epidemiological investigations for viral and bacterial diseases in Africa, Asia, and South America, there appear to be as many publications on environmental occurrence of the zoonotic parasitic protozoa as there are on disease occurrence.

Often environmental detection and epidemiological investigations are beyond the scope of government health departments. It is our experience that environment and health ministries do not always work together, and recognition of the potential for waterborne disease is not always conducive to increased income generation from newer industries, such as tourism. For other, less well appreciated, zoonotic waterborne diseases, only sparse, basic, clinical occurrence data are available. Hard-pressed diagnostic and epidemiological centres do not require further remits. One issue that has not been addressed in this review, but should be seriously considered, is the role of water in food production.

There is a definitive need for standardizing both diagnostic and epidemiological tools. Molecular tools are recent additions to our armoury, and, with the exception of research projects, it is impracticable to suggest that all can be adopted into health care systems in developing countries to serve infectious disease diagnosis and micro- and macro-epidemiology. The fact that clinical and epidemiological investigations are ongoing in such countries must be seen as a bonus.

The potential for reducing the economic and human cost of disease depends not only upon the identification and successful treatment of susceptible hosts, but also upon the implementation of control measures that break the transmission cycle. As for the prokaryotic zoonoses, typing and subtyping systems are available for *Cryptosporidium* and *Giardia* (Strong *et al.* 2000; Thompson *et al.* 2000; Homan and Mank 2001; Mallon *et al.* 2003). Particularly for disinfection-insensitive organisms such as *Cryptosporidium* and *Giardia*, there is a distinct need to adopt molecular tools for environmental detection and disease tracking, as microscopy is unable to determine whether those organisms detected in the environment are viable or infectious to humans. Utilizing standard methods may still lead to an underestimation of *Cryptosporidium* and *Giardia* contamination, as will confusion from detecting (oo)cysts that are not infectious to humans and that have no significance to public health. Technologies including PCR, sequencing, and microarrays have application in the environment, especially where waterborne transmission is known to occur. Development of standardized molecular biological tools for diagnostic and epidemiological purposes must be encouraged.

More effective diagnoses carried out in clinics and laboratories in the developing world will enhance the targeting of treatment and lead to a reduction in morbidity. Increased drug resistance is a continuing theme, particularly in countries where treatment programmes are deemed too expensive. People often stop taking the treatment once the symptoms disappear, increasing the risk of developing drug-resistant forms of disease. Also, self-medication can be commonplace. Even for the drug-sensitive intestinal parasitic protozoa, metronidazole resistance is on the increase.

Providing that accurate statistics are collected, the view of the importance of zoonotic waterborne diseases held by governments and international agencies will be more realistic and lead to better targeting of aid and research funds. It is evident that the prevention and control of zoonotic waterborne diseases are now more feasible than ever before.

Probably the most important intervention is to motivate, educate, and train the populations affected by these diseases to enable them to take the necessary interventions themselves to prevent the transmission of these pathogens. In addition to public information programmes and school-based health education, training courses on important zoonotic waterborne diseases should be

encouraged for primary health and day care workers, workers in diagnostic laboratories, and water treatment plant operators (Warhurst and Smith 1992; WHO 1992; Smith and Ahmad 1996).

Experience in east Africa and south-east Asia suggests that empowerment of local communities works well, following agreement with local, district, and national governments. Based on experience, we suggest the following to motivate and empower such communities:

- (1) Form smart partnerships with local nongovernmental organizations to deliver similar projects in disparate areas.
- (2) Develop multidisciplinary projects to address complete agendas (empowerment, health, environment, and education) in specified areas.
- (3) Set up similar projects in different countries with the same/similar module.
- (4) Instigate quality diagnostic testing systems for all parameters (clinical and environmental samples).
- (5) Liaise with policymakers and water providers to ensure that recommendations are translated into meaningful projects.
- (6) Identify high levels of commitment among all partners.

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# Incidence of the major zoonotic diseases transmitted by water in Mexico, Central America, and the Caribbean

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## 7.1 INTRODUCTION

Adequate water resources, both surface water and groundwater, are increasingly difficult to find due to increasing human population, per capita consumption, and impacts of human activities on the environment. The quantity and quality of water resources throughout the world have been affected. Improved and expanded use of wastewater collection systems has

decreased public health risks in urban areas; however, not all wastewaters receive adequate treatment before being discharged. The variety and densities of human pathogens present in the wastewater in a region are related to the population from which they originate, the wastewater collection and treatment system, the current diseases in the human population, and the management of wastewater by agriculture, animal production, and industry (Aguiar Prieto *et al.* 1998; Sánchez-Pérez *et al.* 2000). The need to provide universal clean water and basic environmental sanitation is the environmental health priority for Latin American countries due to the high rates of diarrhoeal and other waterborne diseases. Diarrhoea accounts for a significant burden of disease in Latin American countries and is responsible for many deaths annually. Children under 5 years of age account for 85% of all deaths attributed to diarrhoea. The majority of this disease burden falls upon poor periurban and rural households (HEMA 2002).

Almost half of the population in developing countries is at high risk of exposure to waterborne diseases, including gastroenteric diseases such as dysentery, giardiasis, hepatitis A, rotavirus, typhoid fever, and cholera. Diarrhoeal diseases are a significant cause of mortality and morbidity in developing countries (OPS 2000).

In recent years, there has been increasing concern about zoonotic diseases that can be transmitted by water. In some European countries and the USA, there have been increasing numbers of cases of diseases related to emerging pathogens, such as the protozoan *Cryptosporidium*, the bacteria *Escherichia coli* O157:H7, *Campylobacter*, and *Salmonella* Enteritidis, and hepatitis E virus (Benenson 1997; Binder *et al.* 1999; Hubálek 2003).

This chapter describes the current incidence of the most important zoonotic diseases transmitted by water in Mexico, Central America, and the Caribbean, with emphasis on emerging pathogens.

There are many differences between countries with respect to the health status of their populations. The quality of the health services, the condition and efficiency of the surveillance systems, and the national economic conditions are some of the factors that will determine the incidence of gastrointestinal diseases in the human population.

In general, each country has some form of epidemiological surveillance system that receives information from all of the different health sectors; however, many factors determine the efficacy of these surveillance systems (OPS 2000, 2002).

## 7.2 COUNTRY-SPECIFIC INFORMATION

### 7.2.1 Mexico

Children under 1 year of age continue to be those most affected by intestinal diseases, with cumulative incidence of intestinal diseases higher than 28 000 per 100 000. Mortality from these diseases in children under 5 years of age was 25 per 100 000 in 1999. The average annual incidence of bacterial infections was 34 per 100 000 in 1997–2000. Approximately 200 reported deaths per year were due to food poisoning between 1997 and 1999 (OPS 2002). In 2002, 19 305 cases of bacterial food poisoning, 6 323 520 cases of digestive infectious diseases, and 4 878 503 cases of intestinal infectious diseases caused by viruses and other organisms, including those not defined, were reported (SUVE 2002). Gutiérrez-Cogco *et al.* (2000) reported that 199 different serotypes were identified in 24 394 *Salmonella* specimens collected from 1972 to 1999 in public and private health laboratories and analysed with the Kauffmann-White method. The most frequent serotype was *S. Typhimurium* (20.4%), followed by *S. Enteritidis* (18.3%). In the past few years, the frequency of *S. Enteritidis* has been increasing, surpassing that of *S. Typhimurium* since 1991, so that *S. Enteritidis* is currently the most frequently isolated serotype. In non-human sources, *S. Derby* (13.8%) and *S. Anatum* (8.5%) are the most frequently isolated strains.

Health authorities lack accurate information on outbreaks of cryptosporidiosis, and few studies have been conducted. In studies with children and cancer patients, a prevalence of 29.6% was reported, compared with 11.4% in apparently healthy individuals (Garza Almanza and Morales Vallarta 2002). In 2002, 28 cases of leptospirosis were confirmed (OPS 2002).

Drinking-water infrastructure covered 88% of the population in 2000; 23 states had coverage higher than 85%, while 5 states had coverage lower than 70%. In 2000, 95% of the drinking-water was disinfected. Sewage disposal services covered 76% of the population in 2000; 5 states had coverage higher than 85%, 17 states had between 70% and 85% coverage, and the remaining 10 states had coverage lower than 70%. In 2000, 76% of the population had access to sewerage services and excreta disposal: 90% in urban areas and 37% in rural areas. There is an official standard for handling hospital waste, and the majority of waste is incinerated (OPS 2002).

### 7.2.2 Costa Rica

The rates for acute diarrhoeal diseases rose from 2903 per 100 000 population in 1996 to 3633 per 100 000 in 1999. In 1999, the mortality rate from acute diarrhoeal diseases was 2.8 per 100 000 population (OPS 2002). In 2002, 293 cases of leptospirosis were reported (SNVSCR 2003). Water supply service coverage was

97.5% in 1999. Coverage with sanitary sewerage and *in situ* excreta disposal reached 96.1% of the population. However, sewage disposal via sanitary sewerage lines was 26%, and only 4% of the sewage received sanitary treatment (OPS 2002).

### 7.2.3 Cuba

Cuba has reported a low incidence of gastrointestinal diseases, and a surveillance system is operating continuously to detect cases that may occur.

*Cryptosporidium* spp. are internationally distributed intestinal parasites that have been recently recognized as an important cause of diarrhoea, malabsorption, and weight loss and as a possible life-threatening factor for immunologically compromised patients, such as those suffering from acquired immunodeficiency syndrome (AIDS). In a study in which 24 patients were examined during 1995 and 1996, all the parasite-affected individuals belonged to the 4th human immunodeficiency virus (HIV) infection group. Diarrhoea and weight loss were the most frequent clinical symptoms associated with such parasitism in the 24 patients (Cassola *et al.* 1999). In another study, Martínez *et al.* (2002) reported that the protozoan *Cyclospora cayetanensis* was isolated in samples of fresh faeces in 20 patients (0.2%) selected from 7956 patients with watery diarrhoea and other clinical manifestations at the Parasitology Department of “William Soler” University Pediatric Hospital from January 2000 to July 2001.

### 7.2.4 Dominican Republic

The estimated mortality rate in children (0–4 years) was 40 per 1000 live births in 1995–2000. Under-registration of deaths in infants was estimated to be 60% in 1998. In that year, conditions arising in the perinatal period accounted for 64.5% of infant deaths, 13% of communicable diseases, and 9.4% of acute diarrhoeal diseases. In 1998, communicable diseases constituted the leading cause of death (40%) in the group aged 1–4 years, followed by external causes (24.6%). In 1999, the leading causes of morbidity in infants were acute respiratory infections (668.8 per 1000), acute diarrhoeal diseases (329.3 per 1000 live births), and parasitoses (138.5 per 1000 live births). The leading causes of morbidity in children 1–4 years of age were acute respiratory infections (221.2 per 1000 population) and acute diarrhoeal diseases (69.4 per 1000 population).

A 1999 survey showed that 65.5% of schoolchildren were infected with *Blastomyces hominis* (27%), *Entamoeba coli* (26.7%), and *Giardia lamblia* (17.7%), among others. Cases and small outbreaks of leptospirosis were confirmed, and toxoplasmosis infections were reported in pregnant women in some areas of the country (OPS 2000, 2002).

The General Bureau of Epidemiology has the normative responsibility for a decentralized surveillance system (a component for early warning and one for prevention and control). At the central level, it includes units of surveillance, health situation analysis, and computer support. Potable water and sanitation are the responsibility of the National Potable Water and Sewerage Institute. Services in the communities are the responsibility of more than 20 associations of rural water supply systems. In 2000, 71.4% of the population had drinking-water services. The coverage of excreta disposal systems was 89.5%, while coverage of sewerage services was only 20.1% (OPS 2002).

### **7.2.5 Nicaragua**

Acute diarrhoeal diseases are among the main types of notifiable diseases. Children under 5 years of age are hit hardest by these diseases, accounting for 73% of the total reported. The morbidity rate was 484 cases per 100 000 population in 1997 and 415 cases per 100 000 in 1998, with mortality rates around 7 per 100 000 in the same years (OPS 2002).

Potable water supply availability was 89.4% in 1998; the urban coverage was 89.5%, and the rural coverage 33.7%. Of the water samples collected in 1999, 4% contained over 50 faecal coliform bacteria per 100 ml. The percentage of the population without access to adequate excreta disposal service dropped to 21.1% in 1998. Only 4.7% of the urban population was still without service, compared with 31.7% of the rural population. Only 34% of collected wastewater received any type of treatment.

### **7.2.6 El Salvador**

In weeks 50–52 of 2000, an increase of diarrhoea in children was caused by rotavirus type 1. In 1999, there were 40 cases of leptospirosis reported, 0.65 per 100 000 population (OPS 2002).

### **7.2.7 Panama**

There is no information available on waterborne zoonoses in Panama.

### **7.2.8 Honduras**

According to the National Survey of Epidemiology and Family Health of 1996, the infant mortality rate was estimated at 36 per 1000 live births (53% neonatal) between 1991 and 1995. Acute respiratory infections and acute diarrhoea with dehydration were the leading causes of death in children under 5. The annual average number of cases of diarrhoea for this period was around 200 000, 85% in

children under 15. Between 1998 and 1999, a laboratory diagnostic capability was developed for leptospirosis, and in 1998, the first diagnosis was made 4 days after the occurrence of Hurricane Mitch. In 1999, 39 cases of leptospirosis were diagnosed (OPS 2002). Water and sewerage services and sanitation in general have shown limited progress in the last 5 years. Investment over the last 2 years in this sector has focused on repairing infrastructure damaged by Hurricane Mitch. In 1999, access to potable water at the national level was 80.9%; 71.1% of the population was served by some form of excreta disposal (OPS 2002).

### 7.2.9 Guatemala

In 1999, there were 385 633 reported cases of acute diarrhoeal disease (incidence: 3470 per 100 000 population) and 3244 deaths from this cause (29.2 per 100 000). In 2000, morbidity was up 21.6% from that in 1999, with 468 981 reported cases (4220 per 100 000). In 1999, children under 5 years of age were most affected, with 238 434 cases, or 61.8% of the total. In 2000, five cases of leptospirosis were documented (OPS 2002). The water supply coverage reached 92% of the population in urban areas and 54% in rural areas, while sanitation coverage was 72% and 52%, respectively. In urban areas, 47% of the population disposes of solid waste through collection services (OPS 2002).

## 7.3 SUMMARY AND CONCLUSIONS

In general, there are many differences between countries in regard to the quality, extent, completeness, and reliability of the available information. There have been very few reports of cases of some emerging zoonotic pathogens, such as *Cryptosporidium* and *Campylobacter*. This could be related to deficiencies in the diagnostic ability in most of the countries, making it very difficult to establish the presence of these pathogens. Most of the countries are able to deal with the principal agents, such as *Salmonella*; however, in the case of protozoa, viruses, and fastidious bacteria, such as *Campylobacter*, improved technical training, infrastructure, and economic resources will be required to establish a well organized surveillance system that includes human and animal information.

It is important to develop new research directions that would permit improved capabilities to detect and fight against emerging and re-emerging zoonotic diseases in Mexico and Central America. These would include the following:

- Use all the scientific and technical knowledge related to the detection, identification, treatment, and control of these diseases.
- Extend research to the study of ecological and environmental factors that influence their transmission.

- Create an international network that includes human and animal resources, in order to share information and experiences between all the member countries and to use this information to make decisions about the control of the zoonotic diseases transmitted by water.

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# 8

## Waterborne outbreaks caused by zoonotic pathogens in the USA

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### 8.1 INTRODUCTION

In 1971, the Centers for Disease Control and Prevention, the US Environmental Protection Agency (EPA), and the Council of State and Territorial Epidemiologists began a collaborative surveillance programme to collect and report data on the occurrence and causes of outbreaks associated with contaminated drinking-water and recreational water. To be defined as a waterborne outbreak, at least two persons must experience a similar illness after the ingestion of or contact with water, and epidemiological evidence must implicate water as the probable source of the illness. There are two exceptions: single-case outbreaks of chemical poisoning or dermatitis when water quality information indicates contamination by the chemical, and single-case reports of laboratory-confirmed primary amoebic meningoencephalitis. The surveillance

© World Health Organization (WHO). *Waterborne Zoonoses: Identification, Causes and Control*. Edited by J.A. Cotruvo, A. Dufour, G. Rees, J. Bartram, R. Carr, D.O. Cliver, G.F. Craun, R. Fayer, and V.P.J. Gannon. Published by IWA Publishing, London, UK. ISBN: 1 84339 058 2.

system records information about the epidemiology of the outbreak, etiologic agents, types of water system, system deficiencies, water sources, and water quality. Because the reporting of waterborne outbreaks is voluntary, the statistics do not reflect the true incidence of outbreaks or illnesses associated with the reported outbreaks. However, the information is considered useful for evaluating the relative degrees of risk associated with different types of source water and water systems and assessing the adequacy of current source water protection strategies, water treatment technologies, and drinking-water regulations (Levy *et al.* 1998; Barwick *et al.* 2000; Craun *et al.* 2002; Lee *et al.* 2002).

In this chapter, we review information available for waterborne outbreaks in the USA that were caused by zoonotic agents.

## 8.2 PRINCIPAL WATERBORNE ZOOTIC PATHOGENS

### 8.2.1 Protozoa

*Cryptosporidium parvum* is likely infectious for all species of mammals; young animals are most prone to infection and illness (Sterling and Marshall 1999). Many mammalian hosts can also carry *Giardia intestinalis* (Schaefer 1999). Because humans are significant carriers of infection for both of these protozoa, the extent to which infections in animals contribute to human infection and illness is uncertain. Recent findings emphasize the need for more information about the species and genotype of *Cryptosporidium*; various isolates may be virulent to varying degrees in humans (Okhuysen *et al.* 1999). *Cryptosporidium* and *Giardia* have been found in drinking-water and recreational water, and a significant number of outbreaks have been reported in the USA when human sewage and wild or domestic animals have contaminated surface water and groundwater sources and water distribution systems. In recreational waters, faecal contamination from bathers has been an important source of exposure.

*Blastocystis hominis* has been found in monkeys, apes, pigs, dogs, cattle, sheep, and ducks (Garcia 1999a). It has also been identified in stool specimens from ill persons in several waterborne outbreaks in the USA, but whether *B. hominis* was the cause of the reported illness is unclear, because its pathogenicity is debated. In 2000, an outbreak of undetermined etiology affected two persons using an untreated well water system in Florida; stool specimens from one person tested negative for *Giardia* and positive for *B. hominis* (Lee *et al.* 2002).

*Balantidium coli* is widely distributed in pigs in warm and temperate climates and in monkeys in the tropics (Garcia 1999b). Human infection is generally found in warmer climates but can occur sporadically in cooler areas. Waterborne transmission has been reported in areas of the Caribbean where people live in

close proximity to pigs and sanitation conditions are poor. In one outbreak, a hurricane caused widespread contamination of individual water systems. *Toxoplasma gondii* infects virtually all warm-blooded hosts, but cats and other felines are the only definitive hosts (Dubey 1999). Intermediate hosts include rodents, sheep, pigs, cattle, and birds. A waterborne outbreak was reported in US soldiers who drank from a jungle pond while on manoeuvres in Panama. In 1995, a waterborne outbreak in British Columbia, Canada, was traced to feline contamination of a surface water reservoir source for a community system.

Humans may be the only natural host of *Cyclospora cayetanensis*, but similar organisms have been observed in chimpanzees and baboons (Ortega 1999). In 1990, a waterborne outbreak affected the hospital staff at a residence dormitory in Chicago. Contamination of the building's plumbing system was traced to a storage tank at the dormitory, but the source of contamination was not identified.

Outbreaks of a clinical syndrome consistent with schistosome cercarial dermatitis, commonly called swimmer's itch, have been reported in several states. The parasites belong to the family Schistosomatidae, and the disease is associated with non-human schistosomes that infect birds as the final or definitive host. In the Midwest, as many as 20 species of non-human schistosomes can cause swimmer's itch (Blankespoor 1999). Final or definite hosts include ducks, geese, gulls, starlings, and rodents. Most outbreaks reported in the USA have affected bathers in lakes and ponds. One outbreak was associated with swimming in ocean water where local snails were found to contain cercariae of *Austrobilharzia variglandis*, an avian schistosome.

Although roundworms, whipworms, and tapeworms may be transmitted by contaminated, untreated drinking-water, this is not their usual mode of transmission (Fredericksen 1999; Little 1999a, 1999b; Smith *et al.* 1999a, 1999b). *Ascaris lumbricoides* is a large roundworm found in humans. *Trichuris trichiura* is a nematode that infects the large intestine; it is commonly known as whipworm. Humans are the reservoir for both of these helminths, but pigs, dogs, cats, and chickens that feed on human faeces can also act as transport hosts, redistributing ova to other sites. *Ascaris* and *Trichuris* ova have been found in surface water and groundwater and may be a source of waterborne exposure for persons who consume untreated water in areas where sanitation is especially poor. Humans and pigs are believed to be the only reservoirs for the tapeworm *Taenia solium*. Water contaminated with faeces from either source poses a threat of infection. *Spirometra mansonioides* is a tapeworm that lives in the intestines of dogs and wild and domestic cats in the USA. Persons may become infected by drinking water contaminated by copepods or eating raw or inadequately cooked flesh of an animal containing the sparganum stage of the worm. The worm does not develop to the adult stage in humans, but a larval stage can invade cutaneous tissues and the brain. Most reported cases have occurred in the south-eastern states. No waterborne outbreaks of *A. lumbricoides*, *T. solium*, *S.*

*mansonoides*, or *T. trichiura* have been reported in the USA. *Gnathostoma spinigerum*, a roundworm commonly found in wild animals and humans in the Far East, is not known to occur in the USA. Persons may be infected by drinking water contaminated by copepods or eating raw or inadequately cooked fish.

Microsporidia produce a spore stage that survives in the environment. These protozoa are relatively ubiquitous in the environment and have been found in bird and mammal hosts, including dogs and cats (Cali 1999). Several species can infect humans. The disease and symptoms vary considerably. Although microsporidia may be transmitted via water, no waterborne outbreaks have been documented in the USA.

### 8.2.2 Bacteria

A significant number of waterborne outbreaks in the USA have been caused by *Escherichia coli* O157:H7. This pathogen is widely distributed in cows and other ruminants, where it may not cause animal disease, but can readily be spread to humans through contaminated food and water (Pruimboom-Brees *et al.* 2000). Drinking-water outbreaks in the USA have primarily been associated with inadequately disinfected or untreated groundwater and distribution system contamination. *Escherichia coli* O6:H16 and *E. coli* O121:H19 have also been implicated in waterborne outbreaks.

A wide range of domestic and wild animals, including poultry, pigs, cattle, sheep, dogs, cats, and rodents, can serve as reservoirs for *Campylobacter jejuni*, an important cause of diarrhoea throughout the world (Fricker 1999a). Although *Campylobacter* are susceptible to water disinfection, outbreaks can occur when water sources are inadequately disinfected. For example, in a 1978 outbreak in Vermont, 3000 persons became ill when an unfiltered surface water source was inadequately chlorinated; in a 1983 outbreak in Florida, 871 persons became ill when the disinfection of a groundwater source was interrupted. Outbreaks in the USA have also been attributed to the contamination of untreated groundwater sources, distribution system mains, and storage reservoirs.

Humans and a wide range of domestic and wild animals, including poultry, cattle, birds, dogs, cats, rodents, and turtles, can serve as reservoirs for *Salmonella* (Covert 1999). In the USA, waterborne outbreaks have been caused by various subspecies of *Salmonella*, including Typhimurium, Enteritidis, Bareilly, Javiana, Newport, and Weltevreden. *Salmonella* outbreaks have also been traced to contaminated wells and water storage reservoirs. In 1993, an outbreak of *S. Typhimurium* resulted in 650 illnesses and 7 deaths in Missouri; contamination was traced to a water storage tank that allowed access by birds.

Animals are the principal reservoir for *Yersinia*, and humans and pigs are important reservoirs for *Y. enterocolitica* (Fricker 1999b). Only two waterborne outbreaks of *Y. enterocolitica* have been reported in the USA. Both outbreaks

were traced to contaminated, untreated groundwater. Contaminated farm wells are suspected as the cause of sporadic, individual cases that are not reported as outbreaks.

Reservoirs for *Leptospira interrogans* include many wild and domestic animals, including rats, dogs, racoons, swine, and cattle (Benenson 1995). Contact of mucous membranes and the skin, especially if abraded, with contaminated water is one mode of transmission. Waterborne outbreaks have been associated with recreational activities in the USA. In 1998, a large outbreak of leptospirosis occurred among participants in a triathlon held in Illinois; 375 persons became ill, and 28 persons were hospitalized (Barwick *et al.* 2000). The illness was epidemiologically associated with swimming in a lake that received runoff from residential areas, agricultural land with cattle and pigs, and a wildlife refuge. Animal testing, however, did not reveal a specific animal source.

Numerous wild animals, especially rabbits, muskrats, beavers, and some domestic animals, can serve as reservoirs for *Francisella tularensis* (Benenson 1995). Tularaemia is not usually spread by the waterborne route, but two small waterborne outbreaks were reported in the USA during the period 1946–1960.

### 8.2.3 Viruses

Some human enteric viruses can infect other animals, and animal reservoirs may be important. The three serotypes of reoviruses found in humans and other mammals are indistinguishable, and human reoviruses are pathogenic in newborn mice (Sattar and Springthorpe 1999). Groups A, B, and C rotaviruses are found in humans and animals, and the interspecies transmission of rotavirus, including human infection by a bovine strain, has been reported (Abbaszadegan 1999). One waterborne outbreak of rotavirus was reported in the USA; sewage from a septic tank contaminated a well. Hepatitis E viruses (HEV) of pigs and rats are very similar to human HEV, and HEV may be zoonotic (US EPA 1999). Human strains of HEV have experimentally infected pigs, and porcine strains have experimentally infected primates (Benenson 1995; Craun *et al.* 2003). In developing countries, the seroprevalence of HEV infection can be as high as 60%. Pregnant women are at high risk of severe illness and death; the mortality can be as high as 20%. In developed countries, the seroprevalence is less than 5%, and very few cases of disease are reported. In a study of sporadic HEV cases reported in the USA, almost half of the ill persons had travelled to endemic areas or received blood transfusions (US EPA 1999).

Noroviruses and hepatitis A virus have caused a number of waterborne outbreaks in the USA. These viruses are not considered zoonotic. Although human sources of faecal contamination are the cause of most, if not all, waterborne viral outbreaks in the USA, it is important to recognize that viruses

are diverse and complex and have the ability to infect different hosts by genetic changes and expression of different phenotypic properties (Craun *et al.* 2003).

#### **8.2.4 Acute gastroenteritis of unknown origin**

For reporting purposes, outbreaks of unknown or undetermined etiology that have a symptom profile consistent with gastrointestinal disease are categorized as acute gastrointestinal illness of unknown origin (AGI) outbreaks. These AGI outbreaks may be caused by commonly identified and well known etiologic agents or less frequently identified and unrecognized waterborne agents, including zoonotic agents. In many AGI outbreaks, an agent could not be identified because the search was limited to those organisms easily cultured or clinical specimens were not collected in a timely manner. In some outbreaks, the etiologic agent was not isolated because the appropriate laboratory analysis was not available. For example, investigators may have suspected a viral etiology but did not collect clinical specimens because laboratory facilities were not available to conduct viral analyses. In several recent outbreaks, however, an etiology could not be established even though there was extensive laboratory analysis of both human specimens and water samples, including appropriate tests for newly recognized bacterial, viral, and parasitic pathogens. This serves as a reminder that although several newly recognized waterborne agents have been uncovered in recent years, additional agents are yet to be determined.

### **8.3 WATERBORNE OUTBREAKS REPORTED IN THE USA**

During 1971–2000, 648 outbreaks were reported in public water systems, and 103 outbreaks were reported in individual water systems (Table 8.1). Almost 600 000 persons were reported ill in the 1010 reported outbreaks. Public water systems are classified as community or non-community systems. A community system serves year-round residents (15 or more service connections or an average of 25 or more residents). A non-community system is used by the general public for 60 or more days per year and has at least 15 service connections or serves an average of 25 or more persons (e.g., factories, schools, restaurants, parks with their own water systems). Of the 751 reported outbreaks in drinking-water systems, 665 (89%) outbreaks were of a known or suspected infectious etiology; 86 (11%) outbreaks resulted in an acute illness, primarily nausea, vomiting, and abdominal pain, after ingestion of a chemical agent in water.

Table 8.1. Waterborne outbreaks and illness by type of system, all causes, 1971–2000

Water system type	Outbreaks	Cases of illness	Emergency visits and hospitalizations	Deaths
Non-community	340	54 893	984	4
Community	308	517 944	5928	65
Untreated recreational water	143	13 898	192	28
Treated recreational water	116	7 842	50	0
Individual	103	1 600	98	3
All water systems	1010	596 177	7252	100

During 1971–2000, an additional 259 outbreaks were associated with recreational activities in various water venues. One hundred and forty-four outbreaks (56%) were associated with recreation in untreated water (e.g., lakes, streams, springs), and 112 (43%) occurred in locations where water was treated (e.g., swimming and wading pools, interactive fountains). Three (1%) outbreaks were associated with both treated and untreated recreational water.

An etiologic agent was identified in 60% of the reported outbreaks (Table 8.2). Almost half (49%) of the bacterial outbreaks were associated with contaminated recreational water. Most (80%) protozoan outbreaks were reported in recreational water and community water systems. Protozoa were more frequently identified than bacteria in outbreaks reported in community systems, but bacteria were identified more frequently than protozoa in non-community system outbreaks.

Table 8.2. Number of waterborne outbreaks by type of water system and etiology, 1971–2000

Water system type	Unidentified agents				
	Protozoa	Viruses	Bacteria	Chemicals	
Non-community	228	31	27	43	11
Community	98	96	20	40	54
Treated and untreated recreational water <sup>a</sup>	40	98	18	97	5
Individual	39	16	9	18	21
All water systems	405	241	74	198	91

<sup>a</sup> An outbreak attributed to algal toxins is not included. An outbreak of both *Shigella* and *Cryptosporidium* is included in the protozoa category.

### 8.3.1 Waterborne outbreaks caused by zoonotic agents

A significant number of outbreaks and illnesses were caused by zoonotic agents (Tables 8.3 and 8.4). Zoonotic agents can be found in human sewage as well as domestic and wild animal faeces. Because the source of the faecal contamination was either not investigated or not identified in many of the zoonotic outbreaks, it was not possible to evaluate the importance of animal versus human sources for the agents. Reported statistics should be evaluated with this limitation in mind.

Table 8.3. Waterborne outbreaks caused by zoonotic agents by type of system, 1971–2000

Water system type	Outbreaks of zoonotic agents	% of all reported outbreaks <sup>a</sup>	% of outbreaks of identified etiology
Non-community	46	14	41
Community	118	38	56
Untreated recreational water	43	30	79
Individual	25	24	28

<sup>a</sup> Includes outbreaks of unidentified etiology.

Table 8.4. Severity of outbreaks by type of system, zoonotic agents and all causes, 1971–2000

Water system type	Cases of illness	Emergency visits and hospitalizations	Deaths
Non-community	6 033	117	2
Community	454 704	4 865	61
Untreated recreational water	1 799	69	0
Individual	383	29	0
Total	462 899	5 080	63

Zoonotic agents caused 118 outbreaks in community systems. These outbreaks represent 38% of the 308 outbreaks reported in community systems or 56% of the 210 community system outbreaks where an etiology was identified. In outbreaks associated with untreated recreational waters, zoonotic agents caused 30% of the outbreaks or 79% of the outbreaks of identified etiology. Zoonotic agents were responsible for most of the illnesses (79%), hospitalizations (71%), and deaths (88%) that were reported in outbreaks caused by contaminated drinking-water and untreated recreational water. Two-thirds of the illnesses (403 000 cases), 50 deaths, and 4400 hospitalizations were reported in a single drinking-water outbreak of cryptosporidiosis in Milwaukee in 1993.

### 8.3.1.1 Drinking-water outbreaks

*Giardia*, *Campylobacter*, *Cryptosporidium*, *Salmonella*, and *E. coli* were the zoonotic agents most frequently identified in outbreaks caused by contaminated drinking-water (Table 8.5). *Giardia* was identified in 66% of all drinking-water zoonotic outbreaks and in 70%, 62%, and 56% of the zoonotic outbreaks in community, non-community, and individual systems, respectively. *Cryptosporidium* was identified in only 8% of the zoonotic outbreaks and in 9%, 4%, and 8% of the zoonotic outbreaks in community, non-community, and individual systems, respectively. *Campylobacter* was identified in 10% of the zoonotic outbreaks. Non-typhoid *Salmonella* caused 8% of the zoonotic outbreaks, and *E. coli* O157:H7 caused 6% of the outbreaks.

Table 8.5. Drinking-water-borne outbreaks of zoonotic agents, 1971–2000

Etiologic agent	Total	Type of water system <sup>a</sup>			Water source <sup>b</sup>		
		C	NC	I	GW	SW	M/U
<i>Giardia</i>	126	83	29	14	31	90	5
<i>Campylobacter</i>	19	9	7	3	12	3	4
<i>Cryptosporidium</i>	15	11	2	2	8	5	2
<i>Salmonella</i>	15	11	2	2	11	2	2
<i>E. coli</i> O157:H7	11	4	4	3	8	2	1
<i>Yersinia</i>	2	–	1	1	2	–	–
<i>E. coli</i> O6:H16	1	–	1	–	1	–	–
<i>E. coli</i> O157:H7 and <i>Campylobacter</i>	1	–	1	–	1	–	–
Total	190	118	47	25	74	102	14

<sup>a</sup> C = community; NC = non-community; I = individual.

<sup>b</sup> GW = groundwater; SW = surface water; M/U = mixed or unknown.

Most (71%) outbreaks of giardiasis occurred in systems using surface water, whereas most (53%) outbreaks of cryptosporidiosis occurred in groundwater systems. Bacterial pathogens were identified in 49 (26%) of the zoonotic outbreaks and 20%, 34%, and 36% of the zoonotic outbreaks in community, non-community, and individual systems, respectively. Most (71%) outbreaks of zoonotic bacteria were reported in groundwater systems.

Outbreaks caused by protozoan and bacterial zoonotic agents were evaluated to determine the water system deficiencies that were responsible for the outbreak (Table 8.6). Inadequate disinfection as the only treatment of surface water and inadequate or interrupted treatment of surface water caused over half (52%) of the outbreaks of giardiasis and cryptosporidiosis. Nineteen

per cent of the outbreaks of giardiasis and cryptosporidiosis were due to contaminated, untreated, or inadequately treated groundwater; 11% were associated with contamination entering the distribution system. Although untreated surface water was responsible for 10% of the outbreaks of giardiasis and cryptosporidiosis, almost all of these outbreaks occurred in the early 1970s before EPA regulations required treatment.

Table 8.6. Number of waterborne outbreaks by deficiencies in drinking-water systems, 1971–2000

Type of contamination	<i>Giardia</i> , <i>Cryptosporidium</i>	<i>Campylobacter</i> , <i>E. coli</i> , <i>Salmonella</i> , <i>Yersinia</i>
Distribution system contamination	16	11
Inadequate disinfection; only treatment, surface water <sup>a</sup>	52	3
Inadequate, interrupted, or bypass of filtration; surface water	22	–
Untreated groundwater	14	14
Untreated surface water	14	2
Inadequate or interrupted disinfection; groundwater <sup>b</sup>	13	11
Water not intended for drinking; contaminated faucet or ice; unknown	10	8
Total	141	49

<sup>a</sup> Includes two outbreaks with surface water and groundwater sources.

<sup>b</sup> Includes three outbreaks where groundwater was filtered.

The three most important deficiencies identified for outbreaks of *Campylobacter*, *Salmonella*, *E. coli*, and *Yersinia enterocolitica* were use of contaminated, untreated groundwater (29%), distribution system contamination (22%), and inadequate treatment of contaminated groundwater (22%). Few bacterial outbreaks were attributed to untreated or inadequately treated surface water.

### 8.3.1.2 Outbreaks associated with untreated recreational water

Schistosomatidae caused 30% of the outbreaks of zoonotic etiology reported in untreated recreational waters (Table 8.7). *Escherichia coli* and *Leptospira* each caused 30% and 16% of the outbreaks; 23% of the outbreaks were caused by either *Giardia* or *Cryptosporidium*. Most (84%) outbreaks were associated with recreational activities in lakes or ponds.

Table 8.7. Outbreaks and illnesses, untreated recreational water, by zoonotic agent and water venue, 1971–2000

Etiologic agent	Total number of		River, springs, and other
	outbreaks	Lakes or ponds	
Schistosomatidae	13	12	1
<i>E. coli</i> O157:H7	12	11	1
<i>Leptospira</i>	7	4	3
<i>Giardia</i>	6	4	2
<i>Cryptosporidium</i>	4	4	–
<i>E. coli</i> O121:H19	1	1	–
Total	43	36	7

The source of contamination was identified in 24 recreational water outbreaks (Table 8.8). Faecal contamination by bathers was identified in 11 of the *E. coli* outbreaks. An avian source was identified in seven outbreaks of schistosome dermatitis and suspected in six outbreaks. Animals, including dogs, cattle, and water buffalo, were suspected sources of *Leptospira*, but could be identified in only one outbreak.

Table 8.8. Identified causes of outbreaks of zoonotic agents associated with untreated recreational water

Source of contamination or deficiency	<i>Cryptosporidium</i>	Schistosomatidae	<i>E. coli</i>	<i>Leptospira</i>
	and <i>Giardia</i>			
Animals, birds	2	7		1
Faecal accident, ill bathers	1		5	
Children in diapers	1		3	
Bather overload or crowding			3	
Seepage or overflow of sewage	1			
Total	5	7	11	1

#### 8.4 SUMMARY, CONCLUSIONS, AND RECOMMENDATIONS

Protozoan and bacterial pathogens are important causes of waterborne outbreaks in the USA. A protozoan pathogen was identified in 24% of all reported waterborne outbreaks, and a bacterial pathogen was identified in 20% of the outbreaks. No etiologic agent was identified in 40% of all outbreaks, and some of these outbreaks may have been protozoan or bacterial. Zoonotic protozoa were more frequently identified as the cause of outbreaks in drinking-water

systems (74%) and untreated recreational water (53%) than zoonotic bacteria. Most drinking-water outbreaks of zoonotic bacteria (71%) and cryptosporidiosis (53%) occurred in groundwater systems, but most drinking-water outbreaks of giardiasis (71%) occurred in surface water systems.

Reported outbreaks of zoonotic agents associated with recreational waters increased during 1991–2000, but drinking-water outbreaks of zoonotic agents did not. From 1991–1995 to 1996–2000, there was an almost 3-fold increase in the reporting of recreational water outbreaks of cryptosporidiosis. During the same period, the number of reported drinking-water outbreaks of cryptosporidiosis decreased by more than one-half.

#### 8.4.1 Waterborne risks of zoonotic protozoa

Either *Giardia* or *Cryptosporidium* caused 60% of the outbreaks of identified etiology in community systems, but only 31% and 37% of the outbreaks of identified etiology in non-community and individual systems. Almost half of the drinking-water outbreaks of giardiasis and cryptosporidiosis occurred in the summer.

Current water filtration and disinfection practices and EPA regulations have reduced the risk of outbreaks associated with *Giardia* and *Cryptosporidium* in surface water systems. However, recent serological-epidemiological evidence suggests that the role of protective immunity is important to consider when assessing *Cryptosporidium* waterborne disease risks (Frost *et al.* 1997, 2000a; Craun *et al.* 1998). The severity and persistence of symptoms of cryptosporidiosis are related to both the immunocompetence of the host and previous infection (Okhuysen *et al.* 1998). Surface water sources may be a significant source of frequent, low-level exposure to *Cryptosporidium* oocysts, and the risk of symptomatic or severe illness among persons consuming water from these systems may be reduced because of protective immunity. Serological studies have found elevated levels of *Cryptosporidium* infection without an apparent increase in disease risk in populations with surface water systems that meet current water quality standards and regulations (Frost *et al.* 2002). Outbreak investigations also provide evidence of the importance of protective immunity for cryptosporidiosis (Frost *et al.* 2000b). Outbreak surveillance generally focuses on the occurrence of clinically detected disease, and waterborne outbreaks are usually detected only when water treatment deficiencies or distribution system contamination contribute to increased levels of exposure and cause increased symptomatic illness. A significant number of outbreaks of cryptosporidiosis occurred after groundwater systems were contaminated by surface water or sewage, resulting in a high incidence of clinical illness. Populations using groundwater sources have lower *Cryptosporidium* infection levels than populations using surface water sources,

and the high incidence of clinically detected cryptosporidiosis may be due to a low level of protective immunity in these populations. Additional evidence for protective immunity is provided by several waterborne outbreaks that were recognized in populations using surface water systems only because illness was reported by visitors; disease surveillance activities detected no increased illness among the residents (Frost *et al.* 1998, 2000a).

*Naegleri fowleri*, a naturally occurring environmental protozoan, caused most of the outbreaks of identified etiology in untreated recreational waters. Schistosomatidae, *Giardia*, and *Cryptosporidium* were the zoonotic protozoa that were identified in untreated recreational water outbreaks. These three protozoa caused 42% of the outbreaks associated with untreated recreational water. Birds such as ducks and geese are important reservoirs for the transmission of Schistosomatidae. Although wild and domestic animals can be reservoirs for *Giardia* and *Cryptosporidium*, important sources of contamination identified for untreated recreational waters included faecal contamination from bathers, septic tanks, and other sources.

#### **8.4.2 Waterborne risks of zoonotic bacteria**

*Campylobacter*, non-typhoid *Salmonella*, *E. coli*, and *Yersinia* were the zoonotic bacteria that caused outbreaks in public and individual water systems. Zoonotic bacteria caused fewer outbreaks than zoonotic protozoa. Zoonotic bacteria caused 15%, 16%, and 21% of the outbreaks of identified etiology in community, non-community, and individual water systems, respectively. Most of these outbreaks occurred in systems that use groundwater. A significant number of outbreaks were associated with groundwater contaminated by surface water or sewage. Outbreaks occurred because the source water was inadequately protected, treatment was not provided for contaminated sources, or disinfection was inadequate or interrupted. Bacterial pathogens are susceptible to water disinfection, but only if adequate disinfectant concentrations and contact times are maintained. Outbreaks of bacterial etiology were also associated with the contamination of distribution systems through cross-connections, back-siphonage, main breaks, main repairs, inadequately protected storage tanks, or uncovered reservoirs.

Bacterial zoonotic agents (*E. coli* O157:H7 and O121:H19 and *Leptospira*) caused 38% of the outbreaks associated with untreated recreational water. Animals are important reservoirs for the transmission of *Leptospira*. Although animals are also reservoirs for *E. coli* O157:H7 and O121:H19, faecal contamination from bathers, septic tanks, and other sources was identified as the important cause of outbreaks in untreated recreational waters.

### 8.4.3 Recommendations

The contamination of groundwater by *Cryptosporidium* or bacterial zoonotic agents is a frequent cause of outbreaks, and caution is urged for populations that use groundwater sources that may be subject to surface water runoff or sewage contamination. Sources of faecal contamination should be identified and action taken to prevent surface water and sewage from entering the well or spring. If contamination cannot be prevented, the well or spring should be treated to effectively remove and inactivate *Giardia* and *Cryptosporidium*. When groundwater is disinfected, the disinfection must be continuous and applied in concentrations and at contact times sufficient for anticipated contamination levels.

The importance of outbreaks associated with distribution system contamination has increased in recent years, and zoonotic agents have caused many of these outbreaks. Distribution systems become contaminated through cross-connections, main breaks and repairs, and poorly covered or uncovered storage tanks. More attention should be given to protecting the distribution system from contamination. It is especially important to prevent storage tanks and reservoirs from being contaminated by birds and rodents and water mains from being contaminated by runoff from animal feedlots, grazing lands, and food processing plants. Zoonotic agents can enter water mains that are leaking or under repair. Back-siphonage and intrusion of zoonotic agents can also occur in areas of low water pressure and through cross-connections, especially in rural and agricultural areas, at agricultural fairs, and in food processing plants. Hydraulic modelling of the system can help identify vulnerable areas of the system, and monitoring water pressure and loss of chlorine residuals in the system can help detect the possible entry of microbial contaminants into the system.

Important sources of contamination of untreated recreational waters include poor hygiene practices of bathers, septic tank and other sewage discharges, and contamination by wild and domestic animals. Additional public education is needed to prevent faecal contamination of recreational waters by bathers. Bathers should be warned about the risks of swimmer's itch, gastroenteritis, leptospirosis, and primary amoebic meningoencephalitis, how these diseases are transmitted, and the potential sources of contamination. Facilities for changing diapers should be available at all bathing sites. Prohibited activities while in the water should include the changing of diapers, rinsing diapers, and cleaning infants after bowel movements. Infants, children, and adults with symptoms of vomiting or diarrhoea should refrain from bathing activities while ill. Contamination from sewage discharges and surface water runoff should be reduced, and water quality monitoring can assist public health officials in closing beaches when needed.

Better surveillance to detect possible waterborne outbreaks, more complete investigation of sources of contamination, and improved laboratory capabilities should be provided to help identify additional zoonotic agents that may be transmitted by contaminated drinking-water and recreational water. Serological-epidemiological studies of *Cryptosporidium* infection should continue to be conducted to provide additional information about protective immunity, illness severity, and outbreak risks.

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# 9

## Symptoms, treatments, and health consequences of waterborne zoonotic diseases

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*S. Kanarat*

### 9.1 INTRODUCTION

Zoonotic agents can be transmitted from animals to humans either directly or indirectly. Indirect transmission means that the agents are passed from animals to humans via food, water, environment, vectors, etc.

Waterborne zoonotic agents include bacteria, protozoa, viruses, and helminths, but bacteria and protozoa are the zoonotic agents that are most often implicated in waterborne disease outbreaks. From 1986 through 1990, 20 waterborne outbreaks due to intestinal protozoa were reported in the USA

© World Health Organization (WHO). *Waterborne Zoonoses: Identification, Causes and Control*. Edited by J.A. Cotruvo, A. Dufour, G. Rees, J. Bartram, R. Carr, D.O. Cliver, G.F. Craun, R. Fayer, and V.P.J. Gannon. Published by IWA Publishing, London, UK. ISBN: 1 84339 058 2.

(Burke 1993). Surveillance carried out by Levy *et al.* (1998) showed that *Cryptosporidium parvum*, *Giardia lamblia*, and *Escherichia coli* O157:H7 were the zoonotic agents found to be frequent causes of waterborne disease outbreaks in the USA. In developing countries, waterborne disease is a major problem.

In this chapter, the symptoms, treatments, and health consequences of major waterborne zoonotic diseases caused by bacteria — campylobacteriosis, *E. coli* (gastroenteritis and infective), salmonellosis, and leptospirosis — and by protozoa — cryptosporidiosis, giardiasis, and toxoplasmosis — are reviewed and discussed. These diseases are gastrointestinal tract infections, except for leptospirosis and toxoplasmosis, which are systemic infections. Therefore, diarrhoea is the common symptom of these diseases except for leptospirosis and toxoplasmosis. Mild cases of bacterial gastrointestinal infections are self-limiting, and antibiotic treatment is not recommended, especially for enterohaemorrhagic *E. coli* infection. Most of the diseases are effectively curable. The severity of the symptoms depends on the infective organisms as well as the health status of the infected person. Patients with acquired immunodeficiency syndrome (AIDS), immunocompromised persons, the old, and the young experience more severe symptoms. Most of the diseases give rise to health consequences.

## 9.2 BACTERIAL WATERBORNE ZONOTIC DISEASES: SUMMARIES

### 9.2.1 Campylobacteriosis

#### 9.2.1.1 Causative agent

Cases are usually caused by *Campylobacter jejuni* or, to a lesser extent, *C. coli* (Reynolds *et al.* 1993; IFST 1995; Nielsen *et al.* 1997; Wooldridge and Ketley 1997; Anonymous 1999; Myint *et al.* 1999a; Nadeau *et al.* 2002).

#### 9.2.1.2 Symptoms

The incubation period is 2–5 days, with a range of 1–10 days. The illness normally lasts for 2–3 days, but severe cases can persist for up to 3 weeks. The infectious dose may be very low — i.e., only a few hundred cells. Symptoms include diarrhoea, which is the most consistent and prominent manifestation of campylobacteriosis, and the stool is often bloody. Other symptoms may include fever, nausea, vomiting, abdominal pain, and headache. Normally, hospitalization is not required. Mild cases are self-limiting; however, the disease can be severe and life threatening. The most severe infections occur in the very

young, the elderly, and the malnourished. Death is more common when there are underlying diseases (e.g., cancer, liver disease, and immunodeficiency disease) (Reynolds 1993; IFST 1995; Myint *et al.* 1999a, 1999b; <http://www.dhs.sa.gov.au/pehs/Youve-got-what/specific-conditions/campylobacter.htm>; <http://www.about-campylobacter.com/page3.htm>).

#### 9.2.1.3 Treatment

Antibiotic treatment is not recommended, unless the infected person is severely ill. Erythromycin is the drug of choice, with ciprofloxacin as an alternative in adults. In case of septicaemia, gentamicin is used, but erythromycin, chloramphenicol, and tetracycline may also be used (IFST 1995).

#### 9.2.1.4 Consequences

Long-term consequences from a *Campylobacter* infection can sometimes occur. Some people may develop a rare disease called Guillain-Barré syndrome, which affects the nerves of the body. Although rare, it is the most common cause of acute generalized paralysis in the western world. It begins several weeks after the diarrhoeal illness in a small minority of *Campylobacter* victims, and it occurs when a person's immune system produces antibodies against components of *Campylobacter* and these antibodies attack the body. Guillain-Barré syndrome begins in the feet and spreads up the body. Prickling sensations give way to weakness, which may lead to paralysis. The disease lasts for weeks to months and often requires intensive care. Full recovery is common; however, some victims may be left with severe neurological damage. Approximately 15% of Guillain-Barré victims remain bedridden or wheelchair bound at the end of 1 year. Two therapies, intravenous immunoglobulin infusions and plasma exchange, may improve the rate of recovery.

It is estimated that approximately 1 in every 1000 reported campylobacteriosis cases leads to Guillain-Barré syndrome. As many as 40% of Guillain-Barré syndrome cases reported in the USA occur following campylobacteriosis. Miller Fisher syndrome is another related neurological syndrome following campylobacteriosis, and it is also caused by immunological mimicry. In Miller Fisher syndrome, the nerves of the head are affected more than the nerves of the body.

Another potential associated chronic condition is an arthritis called Reiter's syndrome. This reactive arthritis most commonly affects large weight-bearing joints such as the knees and the lower back. It is strongly associated with a particular genetic make-up; persons with the human lymphocyte antigen B27 (HLA-B27) are most susceptible.

*Campylobacter* may also cause appendicitis or infect the abdominal cavity (peritonitis), the heart (carditis), the central nervous system (meningitis), the gall-bladder (cholecystitis), the urinary tract, and the bloodstream (<http://www.about-campylobacter.com/page3.htm>).

## **9.2.2 *E. coli* (gastroenteritis and infective)**

### *9.2.2.1 Causative agents*

Causative agents include:

- (1) enterotoxigenic *E. coli* (ETEC);
- (2) enteroinvasive *E. coli* (EIEC);
- (3) enteropathogenic *E. coli* (EPEC); and
- (4) enterohaemorrhagic *E. coli* (EHEC).

### *9.2.2.2 Symptoms*

The incubation period ranges from 1 to 5 days, and the duration of the illness is 3–5 days (Myint *et al.* 1999c). Symptoms vary from mild to severe, depending on the strain and the underlying health of the host. Symptoms include diarrhoea, vomiting, stomach-ache, and fever. EHEC infection often causes severe bloody diarrhoea and abdominal cramps; especially in children under 5 years of age and the elderly, a complication called haemolytic uraemic syndrome (HUS) may occur in about 2–7% of the EHEC infections. Persons who have only diarrhoea usually recover completely (Mims *et al.* 1993; Myint *et al.* 1999a, 1999b; Shanson 1999a; [http://www.cdc.gov/ncidod/dbmd/diseaseinfo/escherichiacoli\\_g.htm](http://www.cdc.gov/ncidod/dbmd/diseaseinfo/escherichiacoli_g.htm)).

### *9.2.2.3 Treatments*

Antibiotics are not recommended for gastrointestinal infection. Fluid replacement may be necessary, especially in young children. When septicaemia is suspected, systemic antibiotics are indicated, and the rational choice depends on the results of the sensitivity of the epidemic strains. For infection with EHEC, antibiotic treatment is not recommended, since treatment with antibiotics may precipitate kidney complications. Antidiarrhoeal agents should also be avoided. Treatment of HUS is urgent and may require dialysis (Mims *et al.* 1993; Shanson 1999a; [http://www.cdc.gov/ncidod/dbmd/diseaseinfo/escherichiacoli\\_g.htm](http://www.cdc.gov/ncidod/dbmd/diseaseinfo/escherichiacoli_g.htm)).

#### 9.2.2.4 Consequences

Following an attack of *E. coli* gastroenteritis, some infants develop a disaccharidase and lactose intolerance, which may become clinically manifested as chronic diarrhoea (Shanson 1999a). About one-third of persons with HUS have abnormal kidney function many years later, and a few require long-term dialysis. Another 8% may have other lifelong complications, such as high blood pressure, seizures, blindness, paralysis, and the consequences of having part of their bowel removed ([http://www.cdc.gov/ncidod/dbmd/diseaseinfo/escherichiacoli\\_g.htm](http://www.cdc.gov/ncidod/dbmd/diseaseinfo/escherichiacoli_g.htm)).

### 9.2.3 Salmonellosis

#### 9.2.3.1 Causative agent

The causative agent for salmonellosis is non-typhoidal salmonellae.

#### 9.2.3.2 Symptoms

The incubation period of intestinal salmonellosis is 8–72 h, and the duration of the illness is 2–7 days (Brooks *et al.* 1991a; Mahon and Manuselis 1995a; Myint *et al.* 1999a, 1999c; Shanson 1999a; [http://www.cdc.gov/ncidod/dbmd/diseaseinfo/salmonellosis\\_g.htm](http://www.cdc.gov/ncidod/dbmd/diseaseinfo/salmonellosis_g.htm)). Symptoms are characterized by an abrupt onset of diarrhoea, abdominal pain, prostration, chills, fever, and vomiting. The vast majority of cases are self-limiting, but in the young, the elderly, and those with impaired immune systems and underlying diseases, symptoms may be severe. Bacteraemia and septicaemia may occur if the strain is invasive. Vomiting is rare, and fever is usually a sign of invasive disease (Brooks *et al.* 1991a; Mims *et al.* 1993; Reynolds *et al.* 1993; Myint *et al.* 1999a; Murray *et al.* 2002a; <http://netvet.wustl.edu/species/primates/primzoon.txt>; [http://www.cdc.gov/ncidod/dbmd/diseaseinfo/salmonellosis\\_g.htm](http://www.cdc.gov/ncidod/dbmd/diseaseinfo/salmonellosis_g.htm); <http://www.tc.umn.edu/~devo0028/zoonos.htm>).

#### 9.2.3.3 Treatments

Diarrhoea is usually self-limiting and resolves without treatment. Fluid and electrolyte replacement may be required in the very young and the elderly. Antibiotics should not be used, except where there is evidence of invasion and septicaemia, as they do not reduce the symptoms or shorten the illness, but may prolong excretion of *Salmonella* in the faeces. Antidiarrhoeal agents are also restricted, as they encourage adherence and further invasion (Brooks *et al.* 1991a; Mims *et al.* 1993; Mahon and Manuselis 1995a; Murray *et al.* 2002a). In case of bacteraemia, the disease should be treated with an effective antibiotic

selected by susceptibility tests. Fluoroquinolones (e.g., ciprofloxacin), chloramphenicol, trimethoprim/sulfamethoxazole, or broad-spectrum cephalosporin can be used (Brooks *et al.* 1991a; Murray *et al.* 2002a).

#### 9.2.3.4 Consequences

Species of *Salmonella* that normally cause diarrhoea may become invasive in patients with particular predispositions. The organisms are not contained in the gastrointestinal tract but invade the body to cause septicaemia; consequently, many organs become seeded with salmonellae, sometimes leading to necrotizing cholecystitis, haemorrhage, osteomyelitis, pneumonia, thrombophlebitis, endocarditis, abscesses, or meningitis (Mims *et al.* 1993; Mahon and Manuselis 1995a).

### 9.2.4 Leptospirosis

#### 9.2.4.1 Causative agent

There are three main serogroups of *Leptospira*: *L. canicola* (dogs are hosts; pigs are reservoirs), *L. icterohaemorrhagiae* (rats are reservoirs), and *L. hebdomadis* (cattle, mice, and voles are reservoirs) (Shanson 1999b). Any of the pathogenic serovars may cause benign leptospirosis, but Weil's disease, the severe form, is usually due to the serovar *icterohaemorrhagiae*.

#### 9.2.4.2 Symptoms

The incubation period of leptospirosis is usually from 10 to 12 days, but may range from 3 to 30 days after inoculation. Duration of the illness varies from less than 1 week to 3 weeks. The severity of leptospirosis depends on many factors, such as strains and the general health of the host. The initial clinical sign is similar to an influenza-like illness, with fever and myalgias. During this phase, the leptospire are present in the bloodstream, and the organisms can frequently be isolated in cerebrospinal fluid. The fever and myalgia may remit after 1 week, or the patient may develop a more advanced disease, including aseptic meningitis or generalized illness with headache, rash, vascular collapse, thrombocytopenia, haemorrhage, and hepatic and renal dysfunction resulting in jaundice and nitrogen retention. Uncomplicated cases have a very low mortality rate. The icteric form is more severe and is associated with mortality approaching 10% (Gillespie 1994a; Mahon and Manuselis 1995a; Murray *et al.* 2002b).

### 9.2.4.3 Treatments

Leptospirosis usually responds to treatment with antibiotics if they are administered in large enough doses early in the infection. For severe infection, penicillin or ampicillin is given intravenously for up to 7 days. For mild infection, ampicillin, amoxicillin, or doxycycline is taken orally. Patients allergic to penicillin may be treated with streptomycin, tetracycline, or erythromycin. Leptospirosis is usually not fatal, particularly in the absence of icteric disease. When there is impairment of kidney function, as sometimes happens in Weil's disease, renal dialysis may be required to counteract the uraemia, which is the main cause of death. Tetracycline should not be used if there is evidence of renal failure (Brooks *et al.* 1991b; Mahon and Manuselis 1995a; Greenwood *et al.* 1997a; Shanson 1999b; Murray *et al.* 2002b).

## 9.3 PROTOZOAN WATERBORNE ZOONOTIC DISEASES: SUMMARIES

### 9.3.1 Cryptosporidiosis

#### 9.3.1.1 Causative agent

Cryptosporidiosis is caused by *Cryptosporidium parvum*.

#### 9.3.1.2 Symptoms

Infection with *Cryptosporidium* organisms may result in asymptomatic carriage. In symptomatic individuals, the incubation period varies from 3 to 14 days (Myint *et al.* 1999a; Shanson 1999a). Disease in previously healthy individuals is usually mild, self-limited enterocolitis characterized by watery diarrhoea without blood. Spontaneous remission after an average of 10 days is characteristic. Patients with immunocompromised conditions (e.g., patients with human immunodeficiency virus [HIV], AIDS) are characterized by 50 or more stools per day and tremendous fluid loss, which can last for months to years. The symptoms may include abdominal cramps, weight loss, nausea, vomiting, and fever (Mims *et al.* 1993; Gillespie 1994b; Colley 1995; Greenwood *et al.* 1997b; Guerrant 1997; Carpenter *et al.* 1999; Shanson 1999a; Murray *et al.* 2002c; <http://www.cdc.gov/ncidod/dpd/parasites/cryptosporidiosis>).

#### 9.3.1.3 Treatments

Currently, there are no broadly effective therapies for cryptosporidiosis. Therapy consists primarily of supportive measures such as fluid and electrolyte replacement. Spiramycin may help control the diarrhoea in some patients in the

early stages of AIDS who have cryptosporidiosis, but is ineffective in patients who have progressed to the later stages of AIDS. Azithromycin and paromomycin appear to offer some benefit (Anonymous 1984; Gillespie 1994b; Greenwood *et al.* 1997b; Guerrant 1997; Shanson 1999a; Murray *et al.* 2002c; <http://www.cdc.gov/ncidod/dpd/parasites/cryptosporidiosis>).

### 9.3.2 Cyclosporiasis

#### 9.3.2.1 Causative agent

The causative agent of cyclosporiasis is *Cyclospora cayetanensis*.

#### 9.3.2.2 Symptoms

The incubation period of cyclosporiasis varies from 1 to 14 days, with an average of 7 days. The organisms infect the small intestine and usually cause watery diarrhoea, mild nausea, anorexia, and abdominal cramping. Other symptoms may include weight loss, myalgias, fatigue, malaise, flatulence, and bloating. In immunocompetent hosts, diarrhoea is self-limiting, but may be prolonged and last for weeks. The clinical illness is typically prolonged and severe and is associated with a high rate of recurrence in immunocompromised patients, particularly those infected with HIV. Biliary tract infection with *Cyclospora* has been reported in two patients with AIDS (Murray *et al.* 2002c).

#### 9.3.2.3 Treatments

Trimethoprim-sulfamethoxazole in combination is reported to be effective for the treatment of *Cyclospora* infection in both immunocompetent and immunocompromised patients. In HIV-infected patients, the high rate of recurrence can be attenuated by long-term suppressive therapy with trimethoprim-sulfamethoxazole. Metronidazole, norfloxacin, quinacrine, nalidixic acid, tinidazole, and diloxanide furoate have also been used in various trials, but the effectiveness of any one of these agents has not been proved (Shlim *et al.* 1991; Connor *et al.* 1999; Murray *et al.* 2002c).

#### 9.3.2.4 Consequences

Guillain-Barré syndrome, reactive arthritis, acalculous cholecystitis, and biliary infection have been reported to be sequelae of *Cyclospora* infection (Connor *et al.* 2001; Murray *et al.* 2002c).

### 9.3.3 Giardiasis

#### 9.3.3.1 Causative agent

Giardiasis is caused by *Giardia lamblia* (*G. duodenalis*).

#### 9.3.3.2 Symptoms

*Giardia lamblia* is the most reported intestinal parasite. The incubation period ranges from 1 to 4 weeks, with an average of 10 days; 50% of infected individuals are asymptomatic. Symptomatic disease ranges from mild diarrhoea to a severe malabsorption syndrome (Gillespie 1994b; Mahon and Manuselis 1995b; Myint *et al.* 1999c; Shanson 1999a; Murray *et al.* 2002c). The onset of the disease is sudden and consists of self-limiting, foul-smelling, watery diarrhoea, abdominal cramps, flatulence, and steatorrhoea. The stool may be semisolid, greasy, and bulky. These symptoms may lead to weight loss and dehydration (Brooks *et al.* 1991c; Gillespie 1994b; Murray *et al.* 2002c; [http://www.cdc.gov/ncidod/dpd/parasites/giardiasis/factsht\\_giardia.htm](http://www.cdc.gov/ncidod/dpd/parasites/giardiasis/factsht_giardia.htm)). Spontaneous recovery generally occurs after 10–14 days (Myint *et al.* 1999a, 1999b, 1999c; Murray *et al.* 2002c). Multiple relapses may develop in chronic patients. Patients with immunoglobulin A deficiency or achlorhydria seem not only to be prone to the infection but also to develop chronic infection (Mahon and Manuselis 1995b; Shanson 1999a; Murray *et al.* 2002c; <http://netvet.wustl.edu/species/primates/primzoon.txt>).

#### 9.3.3.3 Treatments

Quinacrine hydrochloride by the oral route can treat 90% of *Giardia lamblia* infections. Metronidazole and furazolidone are alternatives. Tinidazole is widely used for 1-day treatment (Brooks *et al.* 1991c; Mims *et al.* 1993; Mahon and Manuselis 1995b; Greenwood *et al.* 1997b; Murray *et al.* 2002c).

### 9.3.4 Toxoplasmosis

#### 9.3.4.1 Causative agent

*Toxoplasma gondii* is the causative agent of toxoplasmosis.

#### 9.3.4.2 Symptoms

*Toxoplasma gondii* can infect populations of all ages. It has emerged as the second most common opportunistic infection in AIDS patients, leading to a 75% mortality rate (Jalan 1998). In immunocompetent individuals, the *Toxoplasma* infection is mostly asymptomatic. It may cause mild influenza-like disease with

enlargement of lymph nodes, fever, fatigue, muscle pain, headache, malaise and anaemia (Mims *et al.* 1993; Myint *et al.* 1999c; <http://martin.parasitology.mcgill.ca/jimspage/biol/toxoplas.htm>). The initial infection is self-limiting, becomes chronic (latent), and poses no serious risk unless the host is immunocompromised, in which case it frequently involves the central nervous system and lungs and is often fatal. The central nervous system can be severely affected in patients with congenital infections, those treated with immunosuppressant drugs, and patients with AIDS. In these cases, acute focal or diffuse meningoencephalitis with extensive areas of brain necrosis and vascular involvement may be observed. *Toxoplasma* proliferates in the ependymal and subependymal regions and spreads widely. Morphological changes include lymphocytic infiltration of the meninges, destructive lesions of both brain and white matter, and focal periventricular and periaqueductal calcification. In immunodeficient adult patients, the major finding is necrotizing encephalitis; large abscesses can occur. Symptoms of toxoplasmosis in immunocompromised patients include myocarditis, chorioretinitis, meningoencephalitis, and death.

#### 9.3.4.3 Ocular toxoplasmosis

The lesions include acute chorioretinitis with severe inflammations and necrosis, necrotizing retinitis, and granulomatous chorioretinitis (Mims *et al.* 1993; Myint *et al.* 1999c; <http://martin.parasitology.mcgill.ca/jimspage/biol/toxoplas.htm>).

#### 9.3.4.4 Congenital toxoplasmosis due to acute symptomatic or asymptomatic infections of the mother during pregnancy

Up to 8% of pregnant women get acute *Toxoplasma* infection, and only 10% of them are symptomatic. The acute disease contracted during pregnancy is a serious danger to the offspring, as 40% of the infected pregnant women transmit *Toxoplasma* organisms to the placenta and fetus, leading to *Toxoplasma* infection in the newborn. There are indications from several studies that there is a greater chance of severe illness among the infants whose mothers become infected with *T. gondii* during the first two trimesters. However, infections during the third trimester are more common, but usually result in subclinical disease. The later in pregnancy the acute infection, the more likely the involvement of the placenta at the time of delivery. If treatment of the mother is disregarded and the onset of the infection is during the first trimester, the chance of infection in the newborn is 10–15%, and the consequences will be severe in two-thirds. If the onset is during the second trimester, about 30% will be infected at birth, nearly 10% of which will be severe. If the disease is contracted during the last trimester, approximately 60% of

the neonates will be infected, and virtually none will be severe (Jalan 1998). If the pregnant woman is infected before conception, there is no risk of transmission of the organism to the fetus. Maternal antibodies acquired from the infection prior to pregnancy prevent fetal infection. The effect of the illness acquired during pregnancy on the occurrence of spontaneous abortions and stillbirths is not certain.

At birth, 60–75% of infants with congenital toxoplasmosis have subclinical infections. Among the minority symptomatic infected infants, hydrocephalus or microcephalus, chorioretinitis, convulsion, and intracerebral calcification may be present at birth, but are usually not evident until the infants are a few months of age. The brain is the site of most evident abnormality, together with the retina, the area most commonly and most severely diseased. In the severe form, neonatal sepsis, fetal hydrops, and congenital nephrotic syndrome may occur; however, the disease in the early days of life may be unapparent, only to manifest itself in a few days or weeks. It may have general characteristics of neonatal sepsis, and death results in a short time (Jalan 1998).

#### *9.3.4.5 Treatments*

The drugs used for treatment of *Toxoplasma* infection are pyrimethamine, sulfadiazine, trimethoprin, and spiramycin. The standard treatment of toxoplasmosis is to use the combination of pyrimethamine and sulfonamide (sulfadiazine) in equal parts. Both pyrimethamine and sulfonamide are toxic. During treatment, examining for crystalluria and haematuria is required in order to modify the dose of sulfonamide used. In most countries, the favoured agent for the treatment of acute toxoplasmosis during pregnancy is spiramycin. Spiramycin is a relatively safe drug that concentrates in the placenta and may reduce the risk of maternal/fetal transmission by 60% without having any effect on the fetus. The benefits to the fetus and newborn of spiramycin treatment provided the mother during pregnancy appear to be significant. Based on isolates of the organism from the placenta at birth, the advantages seem to be most marked if spiramycin is begun during the first two trimesters. If the fetus is shown to be infected, the combination of pyrimethamine, sulfonamide(s), and folic acid is added for the duration of the pregnancy. The use of pyrimethamine, sulfadiazine, pyrimethamine, and clindamycin in combination for the treatment of toxoplasmosis encephalitis gives comparable results (Jalan 1998).

#### *9.3.4.6 Recommended treatments for infants*

Remington and Desmonts (1990) recommend treatment of newborn infants with congenital toxoplasmosis as follows:

- (1) Include a 21-day course of 1 mg of pyrimethamine per kilogram of body weight orally once every 1–4 days (preferably every 3–4 days) together with 25–50 mg of sulfadiazine per kilogram of body weight orally twice each day (triple sulfonamides may be substituted).
- (2) Follow by a 30- to 45-day course of 50 mg of spiramycin per kilogram of body weight orally twice each day. Periods of antimicrobial therapy should be alternated for 1 year.
- (3) Because pyrimethamine is a folic acid antagonist, 5 mg of folic acid should be provided orally twice each week while it is being used.
- (4) For those infants with high concentrations of protein in their cerebrospinal fluid (CSF) or active chorioretinitis, it is suggested that 1 mg of prednisone per kilogram of body weight be given orally twice each day until the active retinal inflammation is resolved or the elevated concentrations of CSF protein are no longer present.

Indications for treatment are as follows:

- (1) newborn infants with overt *Toxoplasma* infection;
- (2) active chorioretinitis;
- (3) elevated concentrations of CSF protein;
- (4) infants known to be infected but without clinical evidence are included among those to be treated according to the regimen described above; and
- (5) for apparently healthy infants with equivocal laboratory findings and uncertain historical information, if the mother is known to have contracted toxoplasmosis during the pregnancy and the newborn infant lacks clinical or laboratory evidence of the disease, one cycle of pyrimethamine with sulfadiazine and folic acid followed by spiramycin is suggested while further investigation is carried out (Jalan 1998).

Treatments of paediatric patients for toxoplasmosis are as follows:

- (1) Sulfadiazine (100 mg/kg of body weight per day) and pyrimethamine (1 mg/kg of body weight per day) are given twice a week.
- (2) Folic acid (5 mg) is given twice a week.
- (3) Sulfadiazine and pyrimethamine should be alternated with spiramycin (100 mg/kg of body weight per day), given in 6-week cycles.
- (4) Therapy for small children consists of sulfadiazine or trisulfapyrimidine (150 mg/kg of body weight per day in divided doses) and pyrimethamine (1 mg/kg of body weight per day in divided doses).
- (5) A double dose is used for the first 3 days.

- (6) Prednisolone (1–2 mg/kg of body weight per day) should be added to the therapy in newborns with a high protein concentration in the CSF or chorioretinitis.
- (7) Treatment should be continued for 6 months for both congenital and acquired toxoplasmosis.

#### 9.3.4.7 Consequences

In pregnant women, acute toxoplasmosis contracted during pregnancy leads to *Toxoplasma* infection of the infant, resulting in seizures, mental retardation, other manifestations of severe brain damage, or death in severe cases (Jalan 1998).

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