

# Guidelines for Canadian Drinking Water Quality:

# **Guideline Technical Document**

# Bacterial Waterborne Pathogens — Current and Emerging Organisms of Concern

Prepared by the Federal-Provincial-Territorial Committee on Drinking Water of the Federal-Provincial-Territorial Committee on Health and the Environment

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Other Guideline Technical Documents for the Guidelines for Canadian Drinking Water Quality can be found on the Water Quality and Health Bureau web page at http://www.healthcanada.gc.ca/waterquality.

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# Bacterial Waterborne Pathogens — Current and Emerging Organisms of Concern

# 1.0 Guideline

No maximum acceptable concentration (MAC) for current or emerging bacterial waterborne pathogens has been established. Current bacterial waterborne pathogens include those that have been previously linked to gastrointestinal illness in human populations. Emerging bacterial waterborne pathogens include, but are not limited to, Legionella, Mycobacterium avium complex, Aeromonas hydrophila, and Helicobacter pylori.

Note: Further information on the current and emerging bacterial waterborne pathogens is outlined beginning in section 3.0, Application of the guideline.

# 2.0 Executive summary for microbiological quality of drinking water

#### 2.1 Introduction

The information contained in this Executive summary applies to the microbiological quality of drinking water as a whole. It contains background information on microorganisms, their health effects, sources of exposure, and treatment. Information specific to bacteria is included as a separate paragraph. It is recommended that this document be read in conjunction with other documents on the microbiological quality of drinking water, including the guideline technical document on turbidity.

#### 2.2 Background

There are three main types of microorganisms that can be found in drinking water: bacteria, viruses, and protozoa. These can exist naturally or can occur as a result of contamination from human or animal waste. Some of these are capable of causing illness in humans. Surface water sources, such as lakes, rivers, and reservoirs, are more likely to contain microorganisms than groundwater sources, unless the groundwater sources are under the direct influence of surface water.

The main goal of drinking water treatment is to remove or kill these organisms to reduce the risk of illness. Although it is impossible to completely eliminate the risk of waterborne disease, adopting a multi-barrier, source-to-tap approach to safe drinking water will reduce the numbers of microorganisms in drinking water. This approach includes the protection of source water (where possible), the use of appropriate and effective treatment methods, well-maintained distribution systems, and routine verification of drinking water safety. All drinking water supplies should be disinfected, unless specifically exempted by the responsible authority. In addition, surface water sources and groundwater sources under the direct influence of surface water should be filtered. Drinking water taken from pristine surface water sources may be exempt from filtration requirements (Health Canada, 2003). The performance of the drinking water filtration system is usually assessed by monitoring the levels of turbidity, a measure of the relative clarity of water. Turbidity is caused by matter such as clay, silt, fine organic and inorganic matter, plankton, and other microscopic organisms, which is suspended within the water. Suspended matter can protect pathogenic microorganisms from chemical and ultraviolet (UV) light disinfection.

Currently available detection methods do not allow for the routine analysis of all microorganisms that could be present in inadequately treated drinking water. Instead, microbiological quality is determined by testing drinking water for *Escherichia coli*, a bacterium that is always present in the intestines of humans and other animals and whose presence in drinking water would indicate faecal contamination of the water. The maximum acceptable concentration (MAC) of *E. coli* in drinking water is none detectable per 100 mL.

#### 2.3 Bacteria

*E. coli* is a member of the total coliform group of bacteria and is the only member that is found exclusively in the faeces of humans and other animals. Its presence in water indicates not only recent faecal contamination of the water but also the possible presence of intestinal disease-causing bacteria, viruses, and protozoa. The detection of *E. coli* should lead to the immediate issue of a boil water advisory and to corrective actions being taken. Conversely, the absence of *E. coli* in drinking water generally indicates that the water is free of intestinal disease-causing bacteria. However, because *E. coli* is not as resistant to disinfection as intestinal viruses and protozoa, its absence does not necessarily indicate that intestinal viruses and protozoa are also absent. Although it is impossible to completely eliminate the risk of waterborne disease, adopting a multi-barrier approach to safe drinking water will minimize the presence of disease-causing microorganisms, reducing the levels in drinking water to none detectable or to levels that have not been associated with disease.

While *E. coli* is the only member of the total coliform group that is found exclusively in faeces, other members of the group are found naturally in water, soil, and vegetation, as well as in faeces. Total coliform bacteria are easily destroyed during disinfection. Their presence in water leaving a drinking water treatment plant indicates a serious treatment failure and should lead to the immediate issue of a boil water advisory and to corrective actions being taken. The presence of total coliform bacteria in water in the distribution system (but not in water leaving the treatment plant) indicates that the distribution system may be vulnerable to contamination or may simply be experiencing bacterial regrowth. The source of the problem should be determined and corrective actions taken.

In semi-public and private drinking water systems, such as rural schools and homes, total coliforms can provide clues to areas of system vulnerability, indicating source contamination as well as bacterial regrowth and/or inadequate treatment (if used). If they are detected in drinking water, the local authority responsible for drinking water may issue a boil water advisory and recommend corrective actions. It is important to note that decisions concerning boil water advisories should be made at the local level based upon site-specific knowledge and conditions.

The heterotrophic plate count (HPC) test is another method for monitoring the overall bacteriological quality of drinking water. HPC results are not an indicator of water safety and,

as such, should not be used as an indicator of adverse human health effects. Each system will have a certain baseline HPC level and range, depending on site-specific characteristics; increases in concentrations above baseline levels should be corrected.

There are naturally occurring waterborne bacteria, such as *Legionella* spp. and *Aeromonas hydrophila*, with the potential to cause illnesses. The absence of *E. coli* does not necessarily indicate the absence of these organisms, and for many of these pathogens, no suitable microbiological indicators are currently known. However, the use of a multiple-barrier approach, including adequate treatment and a well-maintained distribution system, can reduce these bacterial pathogens to non-detectable levels or to levels that have never been associated with human illness.

#### 2.4 Health effects

The health effects of exposure to disease-causing bacteria, viruses, and protozoa in drinking water are varied. The most common manifestation of waterborne illness is gastrointestinal upset (nausea, vomiting, and diarrhoea), and this is usually of short duration. However, in susceptible individuals such as infants, the elderly, and immunocompromised individuals, the effects may be more severe, chronic (e.g., kidney damage), or even fatal. Bacteria (such as *Shigella* and *Campylobacter*), viruses (such as norovirus and hepatitis A virus), and protozoa (such as *Giardia* and *Cryptosporidium*) can be responsible for severe gastrointestinal illness. Other pathogens may infect the lungs, skin, eyes, central nervous system, or liver.

If the safety of drinking water is in question to the extent that it may be a threat to public health, authorities in charge of the affected water supply should have a protocol in place for issuing, and cancelling, advice to the public about boiling their water. Surveillance for possible waterborne diseases should also be carried out. If a disease outbreak is linked to a water supply, the authorities should have a plan to quickly and effectively contain the illness.

## 2.5 Exposure

Drinking water contaminated with human or animal faecal wastes is just one route of exposure to disease-causing microorganisms. Outbreaks caused by contaminated drinking water have occurred, but they are relatively rare compared with outbreaks caused by contaminated food. Other significant routes of exposure include contaminated recreational waters (e.g., bathing beaches and swimming pools) and objects (e.g., doorknobs) or direct contact with infected humans or domestic animals (pets or livestock). Although surface waters and groundwater under the direct influence of surface water may contain quantities of microorganisms capable of causing illness, effective drinking water treatment can produce water that is virtually free of disease-causing microorganisms.

## 2.6 Treatment

The multi-barrier approach is an effective way to reduce the risk of illness from pathogens in drinking water. If possible, water supply protection programs should be the first line of defence. Microbiological water quality guidelines based on indicator organisms (e.g., *E. coli*) and treatment technologies are also part of this approach. Treatment to remove or inactivate

pathogens is the best way to reduce the number of microorganisms in drinking water and should include effective filtration and disinfection and an adequate disinfection residual. Filtration systems should be designed and operated to reduce turbidity levels as low as reasonably achievable without major fluctuations.

It is important to note that all chemical disinfectants (e.g., chlorine, ozone) used in drinking water can be expected to form disinfection by-products, which may affect human health. Current scientific data show that the benefits of disinfecting drinking water (reduced rates of infectious illness) are much greater than any health risks from disinfection by-products. While every effort should be made to reduce concentrations of disinfection by-products to as low a level as reasonably achievable, any method of control used must not compromise the effectiveness of water disinfection.

## **3.0** Application of the guideline

Routine monitoring is not recommended for either current or emerging bacterial waterborne pathogens. *E. coli* is used to indicate the presence of the current bacterial waterborne pathogens, but it does not indicate the presence of the emerging bacterial waterborne pathogens. The use of a multiple-barrier approach, including adequate treatment, a well-maintained distribution system, and source protection (in the case of enteric bacteria), can reduce both current and emerging bacterial pathogens to non-detectable levels or to levels that have not been associated with human illness.

## 4.0 Introduction

Throughout history, consumption of drinking water supplies containing enteric pathogenic bacteria has been linked to illnesses in human populations. These illnesses commonly present as gastrointestinal-related symptoms, such as diarrhoea and nausea. Faecal indicators, such as *E. coli*, are the best available surrogates for predicting the presence of such organisms. In this document, these organisms have been identified as current bacterial pathogens of concern.

However, in recent decades, there has been an increasing amount of interest in naturally occurring waterborne bacteria with the potential to cause gastrointestinal and non-gastrointestinal illnesses, particularly respiratory illnesses. These organisms have been defined within this document as emerging pathogens of concern. In most cases, although *E. coli* is able to indicate the presence of enteric pathogenic bacteria, it does not correlate with the presence of these emerging organisms. In addition, there are currently no suitable microbiological indicators for many of these bacterial pathogens.

It is not necessary to establish MACs for current and emerging waterborne pathogens at this time. The use of a multiple-barrier approach, including adequate treatment, a well-maintained distribution system, and source protection, in the case of enteric bacteria, can reduce these bacterial pathogens to non-detectable levels or to levels that have not been associated with human illness.

The following bacteria, identified as either current or emerging concerns, are those commonly recognized as the etiological agents in waterborne outbreaks or those being recognized more often as causes of other serious illnesses that have the potential for waterborne transmission. The information provided in this document focuses on emerging bacteria of

concern, as there are more unknowns associated with these organisms, and their overall significance, in many cases, still needs to be established. Additionally, the bacteria identified should not be considered a complete list of bacterial pathogens that may be present and potentially responsible for isolated cases of waterborne illness. However, they do encompass the majority that have been responsible for waterborne outbreaks. Information on protozoan and viral pathogens of concern can be found, respectively, in the protozoa and enteric viruses guideline technical documents of the *Guidelines for Canadian Drinking Water Quality* (Health Canada, 2004a, 2004b).

# 5.0 Current bacterial pathogens of concern

#### 5.1 Escherichia coli O157:H7

#### 5.1.1 Description, sources, health effects, and exposure

Escherichia coli is a bacterium found exclusively in the digestive tract of warm-blooded animals, including humans. As such, it is used in the drinking water industry as the definitive indicator of recent faecal contamination of water. While most strains of E. coli are nonpathogenic, some can cause serious diarrhoeal infections in humans. The pathogenic E. coli are divided into six groups based on serological and virulence characteristics: enterohaemorrhagic, enterotoxigenic, enteroinvasive, enteropathogenic, enteroaggregative, and diffuse adherent (APHA et al., 1998; Rice, 1999). One enterohaemorrhagic strain, E. coli O157:H7, has been implicated in many foodborne and a few waterborne outbreaks. It was first recognized in 1982, when it was associated with two foodborne outbreaks of bloody diarrhoea and abdominal cramps (Gugnani, 1999). The primary reservoir of this bacterium has been found to be healthy cattle (Jackson et al., 1998). In foodborne transmission, outbreaks are generally through the consumption of undercooked minced beef and unpasteurized juices or milk that have been contaminated with the bacteria (Gugnani, 1999). Although E. coli O157:H7 is not usually a concern in treated drinking water, outbreaks involving consumption of drinking water contaminated with human sewage or cattle faeces have been documented (Swerdlow et al., 1992; Bruce-Grey-Owen Sound Health Unit, 2000).

*E. coli* serotype O157:H7 causes abdominal pain, bloody diarrhoea, and haemolytic uraemic syndrome (HUS). This bacterium produces potent toxins (verotoxins) related to *Shigella* toxins. The incubation period is 3-4 days, and the symptoms occur for 7-10 days (Moe, 1997; Rice, 1999). It is estimated that 2-7% of *E. coli* O157:H7 infections result in HUS, in which the destruction of erythrocytes leads to acute renal failure (Moe, 1997).

Studies have shown that the dose required to produce symptoms is lower than that for most other enteric pathogenic bacteria. The probability of becoming ill depends on the number of organisms ingested, the health status of the person, and the resistance of the person to the organism or toxin (AWWA Committee Report, 1999). Children and the elderly are most susceptible to HUS complications. Evidence suggests that the incidence of *E. coli* O157:H7 infections and HUS has increased since the serotype was first recognized.

#### 5.1.2 Treatment technology

Similar to the non-pathogenic strains of *E. coli*, *E. coli* O157:H7 is susceptible to disinfection (Kaneko, 1998; Rice *et al.*, 2000). Further information on treatment technology for *E.coli* can be found in the *Escherichia coli* guideline technical document of the *Guidelines for Canadian Drinking Water Quality* (Health Canada, 2006a). In addition, a multi-barrier approach based upon source protection (where possible), effective treatment, and a well-maintained distribution system will reduce the levels of *E. coli* O157:H7 in drinking water to none detectable or to levels that have never been associated with human illness.

#### 5.1.3 Assessment

Studies have shown that the survival rate of *E. coli* O157:H7 approximates that of typical *E. coli* in the aquatic environment (AWWA Committee Report, 1999; Rice, 1999). Also, although routine examination methods for generic *E. coli* will not detect *E. coli* O157:H7, the former will always occur in greater concentration in faeces than the pathogenic strains, even during outbreaks. *E. coli* O157:H7 will also never occur in the absence of generic *E. coli* O157:H7.

#### 5.2 Salmonella and Shigella

#### 5.2.1 Description, sources, health effects, and exposure

*Salmonella* and *Shigella* are common etiological agents of gastrointestinal illnesses. Consequently, they are present in the faeces of colonized individuals. These organisms are also commonly present in the faeces of a variety of other animals. The presence of either of these organisms in the environment is generally the result of recent faecal contamination. Numerous outbreaks linked to contaminated drinking water have been reported (Boring *et al.*, 1971; White and Pedersen, 1976; Auger *et al.*, 1981; CDC, 1996; Angulo *et al.*, 1997; Alamanos *et al.*, 2000; R. Taylor *et al.*, 2000; Chen *et al.*, 2001). In most cases, the drinking water was not treated or was improperly treated prior to consumption.

#### 5.2.2 Treatment technology

Salmonella and Shigella survival characteristics in water and their susceptibility to disinfection have been demonstrated to be similar to those of coliform bacteria (McFeters *et al.*, 1974; Mitchell and Starzyk, 1975). Further information on treatment technology for coliforms can be found in the total coliforms guideline technical document of the *Guidelines for Canadian Drinking Water Quality* (Health Canada, 2006b). In addition, a multi-barrier approach based upon source protection, effective treatment, and a well-maintained distribution system will reduce the levels of *Salmonella* and *Shigella* in drinking water to none detectable or to levels that have never been associated with human illness.

#### 5.2.3 Assessment

The absence of *E. coli* during routine verification should be an adequate indication of the absence of *Salmonella* and *Shigella*. However, instances have been reported in which these pathogens were isolated from drinking water in the absence of coliforms (Seligmann and Reitler, 1965; Boring *et al.*, 1971). Coliform suppression by elevated HPCs and poor recovery of stressed

coliforms seem to be the most plausible explanations for these discrepancies. Total coliform and *E. coli* recoveries are not affected by elevated HPCs and environmental stress in the newer defined-substrate methods.

#### 5.3 Campylobacter and Yersinia

#### 5.3.1 Description, sources, health effects, and exposure

Waterborne outbreaks of gastroenteritis involving *Campylobacter jejuni* and *Yersinia enterocolitica* have been recorded on numerous occasions (Eden *et al.*, 1977; McNeil et al., 1981; Mentzing, 1981; Vogt *et al.*, 1982; Taylor *et al.*, 1983; Lafrance *et al.*, 1986; Sacks *et al.*, 1986; Thompson and Gravel, 1986). The most notable Canadian waterborne outbreak involving *Campylobacter* in recent history occurred in Walkerton, Ontario, in May 2000 (Clark *et al.*, 2003). This outbreak was linked to faecally contaminated well water that was not properly treated before consumption. Other reports of *Campylobacter* and *Yersinia* isolation from surface and well waters can be found in the literature (Caprioli *et al.*, 1978; Schiemann, 1978; Blaser *et al.*, 1980; OME, 1980; Taylor *et al.*, 1983; Weagant and Kaysner, 1983; El-Sherbeeny *et al.*, 1985). The survival characteristics of *C. jejuni* are similar to those of coliforms, but the frequency of isolation of *Y. enterocolitica* is higher in winter months, indicating that it can survive for extended periods and perhaps even multiply when water temperatures are low (Berger and Argaman, 1983).

#### 5.3.2 Treatment technology

The findings of Wang *et al.* (1982) indicated that conventional water treatment and chlorination will probably destroy *C. jejuni* and *Y. enterocolitica* in drinking water. In addition, a multi-barrier approach based upon source protection (where possible), effective treatment, and a well-maintained distribution system will reduce the levels of *Campylobacter* and *Yersinia* in drinking water to none detectable or to levels that have never been associated with human illness.

#### 5.3.3 Assessment

The presence of *Y. enterocolitica* has been demonstrated to be poorly correlated with levels of coliforms and HPC bacteria (Wetzler *et al.*, 1979). In addition, studies have shown no correlation between indicator organisms (e.g., *E. coli*, thermotolerant coliforms) and the presence of *Campylobacter* in raw surface water supplies (Carter *et al.*, 1987; Hörman *et al.*, 2004). Thus, coliforms may not be adequate indicators of the presence of both *C. jejuni* and *Y. enterocolitica*.

# 6.0 Emerging bacterial pathogens of concern

#### 6.1 Legionella

#### 6.1.1 Description

Legionellae were first recognized as human pathogens after a 1976 outbreak of pneumonia among veterans attending a convention in Philadelphia. Since that time, at least 42 distinct *Legionella* species have been identified. Approximately half of these species have been associated with disease in humans, with the majority of illnesses resulting from *Legionella* 

*pneumophila* infection. Other than *L. pneumophila*, human illnesses are generally the result of infection with *L. micdadei*, *L. bozemanii*, *L. longbeachae*, and *L. dumoffi*, although many other species have been implicated on occasion.

#### 6.1.2 Sources

Unlike most other common waterborne pathogens, *Legionella* species are naturally present in water environments, including surface water (Palmer *et al.*, 1993) and groundwater (Lieberman *et al.*, 1994). Their ubiquitous nature reflects their ability to survive under varied water conditions, including temperatures from 0 to 63°C and a pH range of 5.0–8.5 (Nguyen *et al.*, 1991). Their survival is, at least in part, attributed to their interactions with other members of the heterotrophic flora. For example, their ability to develop symbiotic relationships with other bacteria, such as *Flavobacterium*, *Pseudomonas*, *Alcaligenes*, and *Acinetobacter*, is thought to be important for their survival and proliferation in water (Lin *et al.*, 1998). In addition, some protozoa that are naturally occurring in water, such as *Hartmanella* sp., *Acanthamoeba castellanii*, and *Echinamoeba*, can harbour *Legionella* organisms, protecting them from environmental stresses and providing a suitable environment for their amplification (Kilvington and Price, 1990; Kramer and Ford, 1994; Fields, 1996). In general, the amount of legionellae in source waters is low compared with the concentrations that can be reached in human-made systems, as natural water conditions are not as conducive to growth.

In human-made systems, *Legionella* colonizes various locations within buildings (e.g., cooling towers, hot water tanks, shower heads, aerators) and contaminates potable water and air. Generally, the areas of a human-made system contaminated with legionellae are those where biofilm formation is most prevalent. This is because *Legionella* can thrive in biofilms. Concentrations have been found to be as much as 10 times higher in biofilms from faucets than from water collected from that faucet (Ta *et al.*, 1995). There is some evidence that pipe material can also affect colonization by legionellae. For example, studies have found that copper piping may be inhibitory for *Legionella* growth (Tiefenbrunner *et al.*, 1993; Rogers *et al.*, 1994; van der Kooij *et al.*, 2002). Water temperature is an additional factor that influences colonization, with temperatures between 20°C and 50°C being hospitable for colonization, although legionellae generally only grow to high concentrations at temperatures below 42°C. Measurable inactivation of legionellae begins at temperatures greater than 50°C (WHO, 2002). It is through human-made systems that *Legionella* is most often disseminated, causing sporadic or outbreak cases of illness.

#### 6.1.3 Health effects

There are two distinct illnesses caused by *Legionella*: Legionnaires' disease and Pontiac fever. Collectively, these illnesses are referred to as legionellosis.

Legionnaires' disease is a severe pneumonia that can be accompanied by extrapulmonary manifestations, such as renal failure, encephalopathy, and pericarditis (Oredugba *et al.*, 1980; Johnson *et al.*, 1984; Nelson *et al.*, 1985). Other common early features include confusion, disorientation, lethargy, and possible gastrointestinal symptoms, such as nausea, vomiting, and diarrhoea (U.S. EPA, 2001). The incubation period is generally 2–10 days. One problem in diagnosing Legionnaires' disease is a lack of any specific symptom that distinguishes it from other bacterial pneumonias. Early diagnosis and consequently appropriate antibiotic therapy

are important in successfully treating the disease. Overall, the mortality rate of Legionnaires' disease is approximately 15% (Oredugba *et al.*, 1980; Johnson *et al.*, 1984; Nelson *et al.*, 1985).

Pontiac fever, on the other hand, is a non-pneumonic, febrile illness with an incubation period of 24–48 hours. Unlike Legionnaires' disease, Pontiac fever has a high attack rate (Mangione *et al.*, 1985). However, this illness typically resolves without complications in 2–5 days (Glick *et al.*, 1978; Fallon *et al.*, 1993).

#### 6.1.4 Exposure

Individuals considered to be at the highest risk of contracting Legionnaires' disease are those who are immunocompromised, especially transplant patients, and those with underlying lung conditions. Outside of the high-risk category, other predisposing risk factors commonly acknowledged include being male, smoking, alcoholism, being over 40 years of age, working more than 40 hours a week, and spending nights away from home. It is therefore not surprising that children and young people are rarely affected by the disease (WHO, 1990; Straus *et al.*, 1996). An additional determinant for human infection is the concentration of *Legionella* present, as a minimum infectious dose is required to cause illness. It is not known precisely what this dose is, as infection is dependent on other factors, including the virulence of the organism and, as mentioned previously, the health status of the host. There is some evidence that replication within amoebae may contribute to enhanced virulence of legionellae (Kramer and Ford, 1994). It is speculated that infectivity may also be enhanced if amoebae containing *Legionella* cells are inhaled or aspirated, as this provides a mechanism for introducing hundreds of *Legionella* cells into the respiratory tract (Rowbotham, 1986; Berk *et al.*, 1998).

Since Legionella is a respiratory pathogen, systems that generate aerosols, such as cooling towers, whirlpool baths, and shower heads, are the more commonly implicated sources of infection, with the hot water supply system generally being the origin of the contamination (Spitalny et al., 1984; Mangione et al., 1985; Fallon and Rowbotham, 1990; Jernigan et al., 1996; Hershey et al., 1997; Brown et al., 1999; Benin et al., 2002). However, the cold water supply, when held within the range of Legionella multiplication (25°C), has also been implicated (Hoebe *et al.*, 1998). *Legionella* contamination is particularly troublesome in hospitals, where susceptible human populations are present and can be exposed to aerosols containing hazardous concentrations of Legionella spp., generally L. pneumophila (Dufour and Jakubowski, 1982). Although more prominent in hospital settings (up to 50% of nosocomial pneumonias) (Breiman and Butler, 1998), Legionella spp. have been estimated to cause 1-15% of community-acquired pneumonias (Lieberman et al., 1996; Breiman and Butler, 1998). Within the community, large buildings such as hotels, community centres, industrial buildings, and apartment buildings are most often implicated as sources of infection (Yu, 2002). Single-family dwellings have rarely been identified as the source of infection. However, studies have shown that contamination of domestic hot water systems in single-family homes with Legionella does occur (Arnow et al., 1985; Lee et al., 1988; Stout et al., 1992b; Borella et al., 2004). In a few instances, cases of Legionnaires' disease have been linked to these dwellings (Stout et al., 1992a).

The challenge to preventing *Legionella*-associated illnesses is controlling their growth in these human-made environments. Once *Legionella* becomes established in a water system (i.e., in the biofilm), it is nearly impossible to eradicate it. However, it can be kept to a minimum by implementing some control procedures. This is particularly important in health care settings.

In addition to being a waterborne illness, outbreaks of Legionnaires' disease have been associated with potting soils. In these cases, the causative agents were found to be *L. longbeachae*, *L. bozemanii*, and *L. dumoffi*, as opposed to *L. pneumophila*.

#### 6.1.5 Treatment technology

As with other bacteria, physical removal mechanisms used during drinking water treatment, such as coagulation, flocculation, sedimentation, and filtration, will reduce the number of Legionella present in finished water. Disinfection can further lower the number present. In comparison with indicator organisms commonly used in the drinking water industry, such as E. coli or total coliforms, a higher CT value (i.e., a longer contact time, a higher disinfectant concentration, or a combination of both) is necessary to achieve a comparable level of reduction in Legionella using chlorine, chlorine dioxide, and ozone. The one exception appears to be with the use of chloramine. Laboratory tests have shown that legionellae seem to be more susceptible to chloramination than E. coli (Cunliffe, 1990). As further support for this finding, it was found that hospitals with a free chlorine residual were 10 times more likely to have reported cases of Legionnaires' disease than hospitals with monochloramine residuals (Kool et al., 1999). Kool et al. (1999) also reported that when a few selected municipalities were investigated, it was found that legionellae could be isolated from systems with a free chlorine residual, but those systems with monochloramination were negative for the bacterium. UV light is also effective for inactivating Legionella, at doses commonly used in drinking water treatment (WHO, 2002). In the distribution system, current recommended disinfectant residuals are sufficient to keep the concentration of Legionella at levels that have not been associated with disease (WHO, 2002).

#### 6.1.6 Assessment

Unlike the case with gastrointestinal pathogens, where *E. coli* can be used to indicate their potential presence, no suitable indicators have been identified to signal increasing concentrations of *Legionella* spp. in a building's plumbing system. There is some evidence that increasing *Legionella* concentrations are accompanied by, or preceded by, an increase in other bacteria, resulting in an elevated HPC measurement (i.e., >100 CFU/mL) (WHO, 2002). Hence, elevated HPCs may indicate the presence of *Legionella*. However, the correlation between HPC and *Legionella* is not consistent. This may partially result from the accompanying chlorination of the water, since HPC bacteria are more readily killed than legionellae (Zacheus and Martikainen, 1996).

The ubiquitous nature of legionellae in water ensures that water supplies, regardless of their source, may contain *Legionella* spp. in low quantities. On a daily basis, the population at large is exposed to these low levels with no reaction or with asymptomatic production of antibodies. In Canada, *Legionella pneumophila* and other *Legionella* species have been recovered in low concentrations from the drinking water (Dutka *et al.*, 1984; Tobin *et al.*, 1986). However, no illnesses have ever been linked to these low concentrations. For these reasons, the presence of the organism is not sufficient evidence to warrant remedial action in the absence of disease cases (Dufour and Jakubowski, 1982; Tobin *et al.*, 1986).

#### 6.2 *Mycobacterium avium* complex (Mac)

#### 6.2.1 Description

The *Mycobacterium avium* complex (Mac) consists of 28 serovars of two distinct species: *Mycobacterium avium* and *Mycobacterium intracellulare*. Based on phenotypic and genetic characteristics, three subspecies of *M. avium*, including *M. avium* subsp. *avium*, *M. avium* subsp. *paratuberculosis*, and *M. avium* subsp. *silvaticum*, have been identified (Nichols *et al.*, 2004). . Mac organisms, along with many other environmental mycobacteria species, comprise the nontuberculous mycobacterium (NTM) group. These organisms are designated as NTM to distinguish them from *Mycobacterium tuberculosis* and *Mycobacterium leprae*, the infectious agents of tuberculosis and leprosy. Unlike their NTM counterparts, neither of the latter organisms is present in the environment, and, consequently, they are not a concern in drinking water.

#### 6.2.2 Sources

Mac organisms have been identified in a broad range of environmental sources, including marine waters, rivers, lakes, streams, ponds, springs, soil, piped water supplies, plants, and house dust (Ichiyama *et al.*, 1988; Covert *et al.*, 1999; Falkinham *et al.*, 2001). Falkinham *et al.* (2001) did note, however, that both *M. avium* and *M. intracellulare* were seldom recovered from well water. In addition to these sources, Wendt *et al.* (1980) reported the isolation of NTM (mostly *M. intracellulare*) from aerosol samples taken near a river. It should be noted that although water is the focus of this document, *M. avium* levels can be hundreds or thousands of times higher in soils than in treated drinking water (LeChevallier, 1999).

The ubiquitous nature of Mac organisms results from their ability to survive and grow under varied conditions. For example, Mac organisms can proliferate in water at temperatures up to 51°C (Sniadack *et al.*, 1992). In one instance, it was found that temperatures between 52°C and 57°C encouraged proliferation of *M. avium* in hospital water supplies (du Moulin *et al.*, 1988). Mac organisms have also been shown to grow in natural waters over a wide pH range (Kirschner *et al.*, 1999). As with most organisms, some conditions will favour their growth. For example, humic and fulvic acids stimulate the growth of *M. avium* (Kirschner *et al.*, 1999). As well, natural water with zinc concentrations greater than 0.75 mg/L (Kirschner *et al.*, 1992) and waters with a low pH and a high organic content (Iivanainen *et al.*, 1993) are more likely to contain Mac organisms. The survival of Mac organisms can also be enhanced by their ability to invade and survive in some species of amoeba (Plum and Clark-Curtiss, 1994; Bermudez *et al.*, 1997; Cirillo *et al.*, 1997), such as *Acanthamoeba polyphaga* or *A. castellanii*, as well as to grow as free-living saprophytes on products secreted by these organisms (Steinert *et al.*, 1998).

Similar to *Legionella*, Mac organisms survive and persist in biofilms. In one study of 50 biofilm samples from water treatment plants, domestic water supply systems, and aquaria, 90% were positive for mycobacteria species, with concentrations up to  $5.6 \times 10^6$  CFU/cm<sup>2</sup> (Schulze-Röbbecke *et al.*, 1992). Although this study did not identify the percentage of Mac organisms within the mycobacteria species isolated, a separate study found that 131 of 267 biofilm mycobacteria isolates were *M. intracellulare* (average 600 CFU/cm<sup>2</sup>), and 4 were *M. avium* (<0.5 CFU/cm<sup>2</sup>). This confirms that Mac organisms are present in biofilm matrices. An additional

study into several types of commonly used plumbing materials concluded that the frequency of recovery of Mac organisms from biofilm was not dependent on the material type (Falkinham *et al.*, 2001).

#### 6.2.3 Health effects

The clinical presentation of Mac infections can include a productive cough, fatigue, fever, weight loss, and night sweats. It is also a leading cause of mycobacterial lymphadenitis in children less than 12 years of age. Current research suggests a possible role for Mac organisms in the development of Crohn's disease, an inflammatory bowel disease similar to Johne's disease in sheep, cattle, and goats. Johne's disease is caused by *M. avium* subsp. *paratuberculosis*. Strains of *M. avium* subsp. *paratuberculosis* have been isolated from some Crohn's patients. Although the evidence is still inconclusive, due mainly to difficulties in reliably detecting the pathogen, improvements in detection methodologies are providing better evidence linking the pathogen to Crohn's disease (Reynolds, 2001; Hermon-Taylor and El-Zaatari, 2004). Diagnosis of Mac infections is difficult and time-consuming. Therefore, treatment is usually initiated before confirmation is made as to the cause of the infection. The treatment regimen for Mac infections may include high doses of antimicrobials. These drugs can have a variety of side effects, including nausea, vomiting, diarrhoea, rashes, abdominal pain, hearing loss, eye inflammation, and damage to blood vessels or the liver (Reynolds, 2001).

The symptoms encountered with Mac infections result from colonization of either the respiratory or the gastrointestinal tract, with possible dissemination to other locations in the body. Unlike Mycobacterium tuberculosis (the infectious agent of tuberculosis), Mac organisms have low pathogenicity, so individuals can become colonized with the organisms without any adverse health effects. Individuals who are immunocompetent without underlying disease conditions have a very low risk of becoming symptomatic with a Mac infection. Recently, reports have shown an increasing recognition of Mac in individuals, especially women, with apparently no predisposing disorders of the lungs or immune system. Although recognition of this disease in immunocompetent individuals is increasing, the risks of becoming ill are still very low. Whereas the majority of healthy individuals who contract Mac infections have localized infection, disseminated Mac infections occur in a large proportion of AIDS patients (80% of those patient that are colonized), as well as in other immunosuppressed populations, such as those with severe combined immunodeficiency syndrome, transplant recipients, and patients treated with corticosteroids or cytotoxic drugs (von Reyn et al., 1993a,b). The true prevalence of Mac infections is not known, as it is not a reportable illness in Canada or the United States. It has been suggested, based on studies in Houston and Atlanta, that the rate of illness is 1 in 100 000 persons per year (Reynolds, 2001).

#### 6.2.4 Exposure

Exposure to Mac organisms may occur through the consumption of contaminated food, the inhalation of air with contaminated soil particles, or contact with or ingestion, aspiration, or aerosolization of potable water containing the organisms. Person-to-person contact is thought to be possible but has not yet been observed (Reynolds, 2001; Le Dantec *et al.*, 2002).

With respect to water supplies, infection with *M. avium* and *M. intracellulare* has been well documented (Wendt *et al.*, 1980; Grange, 1991; von Reyn *et al.*, 1993a, 1994; Glover *et al.*,

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1994; Montecalvo *et al.*, 1994; Kahana *et al.*, 1997; Aronson *et al.*, 1999; Mangione *et al.*, 2001) with *M. avium* being the leading cause of NTM infections. The route of exposure, in most cases, is inhalation of contaminated aerosols, particularly through contaminated hot tubs. Some research has shown that one *M. avium* strain in particular (Mav-B sequevar) is responsible for the majority of cases. This may be the result of a higher virulence of this strain or an increased prevalence of this strain in the environment (Hazra *et al.*, 2000). The proportion of infections caused by *M. avium* and *M. intracellulare* has been shown to vary between populations. In one study, AIDS patients were more often infected with *M. avium* (98% of 45 patients) than with *M. intracelluare* when compared with non-AIDS patients, in whom 60% of the infections were shown to be caused by *M. avium* and the remaining 40% were the result of M. *intracellulare* (Guthertz *et al.*, 1989). The infectious dose appears to range from 10<sup>4</sup> to 10<sup>7</sup> organisms, but this number depends on numerous factors, including, but not limited to, the virulence of the organism and the immune status of the host.

#### 6.2.5 Treatment technology

Water treatment technologies commonly used, including chemical disinfection and physical removal methods, have been tested for their ability to inactivate or remove mycobacteria from water supplies. Of these technologies, the most effective has been physical removal using sand filtration and coagulation-sedimentation techniques. For example, it was shown, using a surface water source, that mycobacterial numbers were reduced by almost 2 log, with the majority of the 2 log removal attributed to removal by filtration (Falkinham et al., 2001). The disinfection employed contributed only slightly to the overall log removal. In comparison with conventional indicators, Mac organisms are more resistant to the commonly used disinfectants . For example, the CT values necessary for inactivation using free chlorine (pH 7, 23°C) are 2–3 orders of magnitude higher for *M. avium* than for *E. coli*. Therefore, in a typical drinking water system, the chlorine dose added will unlikely be effective in controlling the Mac organisms (AWWA Committee Report, 1999). Similar results have been found with other commonly used disinfectants in the drinking water industry (Yu-Sen et al., 1998; R.H. Taylor et al., 2000). Nonchemical treatment methods should be effective for Mac removal and/or inactivation. Even with good removal of organisms from the source water, the number of Mac organisms may increase in the distribution system (Falkinham et al., 2001). Conditions identified to encourage growth in the distribution system include old pipes, long storage times, and high assimilable organic carbon levels (Falkinham et al., 2001).

#### 6.2.6 Assessment

Unlike gastrointestinal pathogens, where *E. coli* can be used to indicate their potential presence, no suitable indicators have been identified to signal increasing concentrations of Mac organisms in water systems. For example, studies have found no relationship between the numbers of NTM recovered from reservoir water and coliform counts, HPCs, and total and free chlorine levels (Glover *et al.*, 1994; Aronson *et al.*, 1999). There is some evidence that *M. avium* presence is associated with turbidity in raw waters (Falkinham *et al.*, 2001), but further exploration of this issue is needed.

Currently, the presence of mycobacteria in water is not regulated by any countries or international organizations, including Canada. The U.S. Environmental Protection Agency (EPA)

has identified *M. avium* and *M. intracellulare* as waterborne health-related microbes that need additional research on their health effects, their occurrence in water, and their susceptibility to treatment methods (Reynolds, 2001). These organisms have also been included in a list of candidate contaminants for possible regulation by the U.S. EPA (LeChevallier, 1999). At the present time, there is not sufficient information to warrant actions based on the presence of the organisms in the absence of disease.

#### 6.3 Aeromonas hydrophila

#### 6.3.1 Description

*Aeromonas hydrophila* are Gram-negative, non-spore-forming, rod-shaped, facultative anaerobic bacilli belonging to the family Aeromonadaceae. Although *A. hydrophila* is the focus of this section, other aeromonads, such as *A. caviae* and *A. sobria*, have also been isolated from human faeces and from water sources (Havelaar *et al.*, 1992; Janda and Abbott, 1998; Villari *et al.*, 2003). Morphologically, aeromonads are indistinguishable from members of the Enterobacteriaceae family, such as *E. coli*. They also share many biochemical characteristics, with the differentiation being that aeromonads are oxidase positive and Enterobacteriaceae are oxidase negative.

#### 6.3.2 Sources

Previous work has firmly established that *Aeromonas* species, including *A. hydrophila*, are ubiquitous in the environment. These organisms have been found in lakes, rivers, marine waters, sewage effluents, and drinking waters, among other places (Allen et al., 1983; Nakano et al., 1990; Poffe and Op de Beeck, 1991; Payment et al., 1993; Ashbolt et al., 1995; Bernagozzi et al., 1995; Chauret et al., 2001; El-Taweel and Shaban, 2001). The concentration of Aeromonas species varies with the environment being investigated. In clean rivers, lakes, and storage reservoirs, concentrations of *Aeromonas* spp. have been found to typically be around  $10^2$ CFU/mL. Groundwaters generally contain less, with fewer than 1 CFU/mL. Additionally, drinking water immediately leaving the treatment plant has been found to contain between 0 and  $10^2$  CFU/mL, with potentially higher concentrations in drinking water distribution systems, attributed to growth in biofilms (Payment et al., 1988; U.S. EPA, 2000; Chauret et al., 2001). Depending on the study, A. hydrophila comprised 20-60% of the aeromonads isolated (Millership et al., 1986; Notermans et al., 1986; Kühn et al., 1997). Aeromonas spp. have been found to grow between 5°C and 45°C (U.S. EPA, 2000). Water temperature is a significant factor for Aeromonas growth (Sautour et al., 2003). Coinciding with the optimal growth range of Aeromonas, seasonal variation has been reported for public water systems, with Aeromonas more often recovered during the warmer months (Gavriel et al., 1998). The same trend has been observed with stool samples (Burke et al., 1984; Moyer, 1987).

#### 6.3.3 Health effects

In recent years, *A. hydrophila* has gained public health recognition as an opportunistic pathogen. It has been implicated as a potential agent of gastroenteritis, septicaemia, cellulitis, colitis, and meningitis, and is frequently isolated from wound infections sustained in aquatic environments (Krovacek *et al.*, 1992; Gavriel *et al.*, 1998). It has also recently been implicated

in respiratory infections (Janda and Abbott, 1998). Treatment for infection with *Aeromonas* is generally not necessary for gastrointestinal illness. However, for other presentations of infection, antibiotic therapy is usually implemented. Individuals at the greatest risk of infection are children, the elderly, and the immunocompromised (Merino *et al.*, 1995).

#### 6.3.4 Exposure

The common routes of infection suggested for *Aeromonas* are the ingestion of contaminated water or food or contact of the organism with a break in the skin (Schubert, 1991). No person-to-person transmission has been reported. It should be noted that although *A. hydrophila* is water based, waterborne outbreaks have not been reported, and waterborne transmission has not been well established. For example, various studies have been unsuccessful in linking patient isolates of *A. hydrophila* with isolates recovered from the water supply (Havelaar *et al.*, 1992; Moyer *et al.*, 1992; Hänninen and Siitonen, 1995; WHO, 2002; Borchardt *et al.*, 2003). As mentioned above, the growth of *A. hydrophila* is temperature dependent. Therefore, the risk of infection is highest in the summer months, when these microorganisms are multiplying more rapidly (Holmes and Nicolls, 1995).

The dose necessary to cause infections in humans has not been established. In the limited number of studies done, the dose was quite high, and only a limited number of participants were infected (Morgan et al., 1985; Janda and Abbott, 1998; WHO, 2002). The virulence of the strain is one factor that can influence the infectious dose needed. For A. hydrophila, the virulence of the organism is, at least in part, thought to result from the production of specific enterotoxins (Schubert, 1991). The primary toxins are haemolysins(Janda, 1991). In addition, some aeromonads produce a range of cell surface and secreted proteases that may enhance their virulence (Janda, 1991; Gosling, 1996). It has been demonstrated that a significant proportion of the A. hydrophila isolated from water (chlorinated and unchlorinated supplies) contained genes responsible for enterotoxigenic or cytotoxic activity (Ormen and Ostensvik, 2001). Expression of virulence factors has been shown to be influenced by environmental temperature. A. hvdrophila isolated from the environment produced significantly less enterotoxins when grown at 37°C compared with 28°C, whereas the clinical isolates tested produced more enterotoxins at 37°C than at 28°C (Mateos et al., 1993). The temperature of the human body is approximately 37°C; therefore, strains that produce virulence factors at this temperature are likely to be more important as pathogens.

#### 6.3.5 Treatment technology

As mentioned previously, aeromonads are ubiquitous in many water environments. Consequently, they will be present in most source waters used for drinking water production. The methods currently used for treatment and disinfection are effective in minimizing the level of aeromonads in the finished drinking water. For example, it has been shown that *A. hydrophila* is generally more susceptible to chlorine and monochloramine than coliforms (Knøchel, 1991; Sisti *et al.*, 1998). Chlorine dioxide has also been shown to be an effective disinfectant (Medema *et al.*, 1991). In the distribution system, there is the potential for *Aeromonas* to regrow. Maintaining chlorine at or above 0.2 mg/L should provide adequate control of *A. hydrophila* in the water (Holmes and Nicolls, 1995). However, it is difficult to control its growth in biofilms (Gavriel *et al.*, 1998; Chauret *et al.*, 2001; WHO, 2002). The most effective approach for controlling *Aeromonas* growth is to limit the *Aeromonas* spp. entering the distribution system through effective treatment and maintenance, to maintain temperatures below 14°C, to provide free chlorine residuals above 0.1–0.2 mg/L, and to limit the levels of organic carbon compounds (WHO, 2002). If there are significant increases in *Aeromonas* concentrations in a drinking water supply, this indicates a general deterioration of bacteriological quality.

#### 6.3.6 Assessment

Some studies have been undertaken to determine if the indicators currently used in the drinking water industry, including *E. coli*, total coliforms, and HPC, can be used as surrogates for the presence of *Aeromonas*. Several studies, including a large study in England, showed no relationship between *Aeromonas* incidence and coliforms, *E. coli*, or HPCs (Holmes *et al.*, 1996; Gavriel *et al.*, 1998; Fernandez *et al.*, 2000). Although all the studies had similar findings, not all could draw definite conclusions, because of limited sample sizes, minimal occurrences of coliforms, and/or the absence of *E. coli* in the water.

When looking at the overall public health significance of *A. hydrophila* in drinking water, further epidemiological studies are needed to ascertain the relationship between *Aeromonas* illness and the presence of these organisms in drinking water (WHO, 2002). The European Community has established a drinking water standard for *A. hydrophila* of no more than 20 CFU/100 mL in water leaving the treatment plant and 200 CFU/100 mL in distribution system water (van der Kooj, 1993; Moyer, 1999). These values are based on an assessment of achievability, motivated by a precautionary approach, and not on the public health significance of their occurrence in drinking-water (WHO, 2002). Based on what is currently known, treated drinking water probably represents a very low risk. However, it is advisable to minimize the concentration of *A. hydrophila*, as well as other aeromonads, in drinking water supplies until their public health significance has been fully investigated.

#### 6.4 Helicobacter pylori

#### 6.4.1 Description

*Helicobacter pylori*, formerly known as *Campylobacter pylori*, was first recognized as a human pathogen in 1983 (Postius, 2001) and was subsequently identified as a human carcinogen by the International Agency for Research on Cancer (IARC, 1994).

Two morphologically distinct forms of *H. pylori*, a spiral shape and a coccoid form, have been identified (van Duynhoven and de Jonge, 2001). The spiral shape is cultured routinely from clinical samples. To date, the coccoid form has been found to be non-culturable. Transformation from the spiral-shaped bacterium grown in culture to the non-culturable coccoid form is thought to result from variations in the environment, such as oxygen stress, temperature changes, the presence of antibiotics, and other stress-inducing conditions (Engstrand, 2001). At present, it is still unclear whether the coccoid form is viable but non-culturable (VBNC), similar to VBNC states found with *Salmonella, Campylobacter*, and *Vibrio* spp. (Byrd *et al.*, 1991), and therefore able to infect humans, or if it is simply non-viable (van Duynhoven and de Jonge, 2001). Attempts to revert the coccoid form to the spiral form using nutrient supplementation (Sörberg *et* 

*al.*, 1996) have been unsuccessful. Reversion has been successful in only one report, using mice (Wang *et al.*, 1995). Attempts to use the same procedure in pigs resulted in contradictory results (Eaton *et al.*, 1995).

#### 6.4.2 Sources

*H. pylori* has not yet been isolated from environmental sources, including water. However, other methods have been able to detect *H. pylori*. For example, it has been found microscopically, using a fluorescent antibody, in surface waters and shallow groundwaters (Hegarty *et al.*, 1999). Molecular methods, such as polymerase chain reaction, have also been used to detect the presence of *H. pylori* DNA in water (Enroth and Engstrand, 1995). Under laboratory conditions, *H. pylori* has been shown to survive for days, up to weeks, in sterile river water, saline solution, and distilled water at a wide variety of pH levels and in temperatures ranging from 4°C to  $15^{\circ}$ C (West *et al.*, 1992; Shahamat *et al.*, 1993). These results indicate that water may be a potential source of transmission for *H. pylori*. Currently, the only substantial reservoir of *H. pylori* has been found to be the human stomach (Dunn *et al.*, 1997). Domestic cats have been found to harbour the organisms, but studies conducted have been unsuccessful at linking pet ownership with *H. pylori* seropositivity (Webb *et al.*, 1996b; Bode *et al.*, 1998). The bacterium has also been isolated from primates, but, due to rare contact, primates are unlikely to be important reservoirs.

#### 6.4.3 Health effects

Human infection with *H. pylori* has been linked to gastritis, duodenal ulcers, and an increased risk of gastric adenocarcinoma (Jekel, 1993; Hunter, 1997; Engstrand, 2001). These health effects reflect the ability of H. pylori to colonize the human stomach and establish a chronic infection associated with an inflammatory response. In addition to gastrointestinal disorders, some studies have shown an association between H. pylori infection and anaemia (i.e., decreased serum ferritin levels) (Milman et al., 1998; Peach et al., 1998; CDC, 1999), although there are also studies to the contrary (Haruma et al., 1995; Perez-Perez, 1997). The prevalence of *H. pylori* infection in the world is assumed to be 50%, with higher prevalence in developing countries (90%). Both immunocompromised and immunocompetent individuals can become infected with *H. pylori*, and both groups can develop associated illnesses (Battan et al., 1990; Edwards et al., 1991; Vaira et al., 1995). In children, H. pylori can cause antral gastritis and duodenal ulcer disease, although most infections in children are asymptomatic (Rowland and Drumm, 1998). It has been well established that infections with H. pvlori are generally acquired during childhood, with a lower frequency of infection in adults (Feldman et al., 1998; Allaker et al., 2002). The infectious dose necessary for colonization of humans is not known. It is assumed to be low because of the high percentage of infected individuals; in human testing, however, it was shown that the minimum required dose was  $3 \times 10^5$  CFU when given in combination with an acid suppressant (Morris and Nicholson, 1987). Incidences of accidental infection, such as ingestion resulting from laboratory work (Matysiak-Budnik et al., 1995) and use of improperly maintained endoscopes, suggest that the dose could be much lower. Of those individuals who become infected, only a subpopulation (6–20%) will develop gastroduodenal disease (Go, 1997; Parsonnet, 1998; Patchett, 1998; Engstrand, 2001), with approximately 1% of all infections progressing ultimately to gastric cancer. Gastric cancer is the second most common cause of

cancer, and 40–50% of these cases are related to *H. pylori* (Parsonnet, 1998; Parkin *et al.*, 1999). Infection with *H. pylori* is treatable using a combination of bismuth and antibiotics or a combination of a proton-pump inhibitor and antibiotics (Scott *et al.*, 1998). This translates into a significant number of cases of disease due to *H. pylori* that are preventable.

#### 6.4.4 Exposure

How the organism is transmitted is still not fully understood; however, the fact that it has been recovered from saliva, dental plaques, the stomach, and faecal samples strongly indicates oral-oral or faecal-oral transmission (Ferguson et al., 1993; Jekel, 1993; Nguyen et al., 1993; Goodman et al., 1996; Dunn et al., 1997; Feldman et al., 1998). Associations between the seroprevalence of hepatitis A, which is known to be transmitted by the faecal-oral route, and H. pylori shows the potential for faecal-oral transmission (Hazell et al., 1994; Rudi et al., 1997). As well, consumption of uncooked vegetables irrigated with untreated sewage has been suggested as a risk factor for H. pylori (Hopkins et al., 1993). On the other hand, there have been studies using hepatitis A showing no association with H. pylori infection and consequently no link to faecal-oral transmission (Webb et al., 1996a; Furuta et al., 1997). Additional studies examining H. pylori seropositivity in sewage workers (Friis et al., 1996) and in travellers recently returned from areas of the world with a high prevalence of H. pylori (Lindkvist et al., 1995) also found no connection to faecal-oral transmission. It has been suggested that the link to faecal samples is better when looking at transmission routes for children than for adults (Thomas et al., 1992; Mapstone et al., 1993). Studies in some developing countries found that transmission of H. *pylori* was due to environmental conditions, such as poor hygiene or the consumption of contaminated water (Klein et al., 1991; Hopkins et al., 1993). Evidence that waterborne transmission may be important in areas of the world with less than adequate water quality comes from studies conducted worldwide, including in Inuit communities in Canada (Klein et al., 1991; Goodman et al., 1996; Hulten et al., 1996; McKeown et al., 1999). Epidemiological studies conducted in developed countries have found no association between environment and infection (Hultén et al., 1998). In the latter studies, clustering of infections within families was prevalent, supporting the oral-oral route (Brenner et al., 1999; Allaker et al., 2002), with infected mothers playing a key role in transmission (Rothenbacher et al., 1999). In contrast, it was found that oral-oral transmission between spouses was unlikely to be an important mode of transmission (Luman et al., 2002).

Overall, the predominant transmission route for *H. pylori* seems to be situation dependent, with person-to-person transmission playing a key role in many circumstances. Water and food appear to be of lesser direct importance, but they can still play a significant role in situations with improper sanitation and lax hygiene. Further investigation into the role of water is needed.

#### 6.4.5 Treatment technology

Some work has been carried out on the relative sensitivities of *H. pylori* and *E. coli* to currently used drinking water treatment methods. Further information on the role of *E. coli* in drinking water can be found in *The Guideline for Canadian Drinking Water Quality: Guideline Technical Document* — *E. coli* (Health Canada, 2006a). Similar to other bacteria, a proportion of the *H. pylori* present in the source water will be removed using physical methods, such as

coagulation, sedimentation, and filtration. This organism is also susceptible to disinfectants commonly used in drinking water treatment. In laboratory disinfectant testing, *E.coli* proved to be more sensitive to chlorine and ozone than *H. pylori* (Johnson *et al.*, 1997; Baker *et al.*, 2002); however, there was little difference between the effectiveness when monochloramine was used (Baker *et al.*, 2002). Although *E. coli* is easier to inactivate than *H. pylori* with some disinfectants, the CT provided by a typical water treatment plant is sufficient to inactivate *H. pylori* in the finished water (Peeters *et al.*, 1989; Johnson *et al.*, 1997). However, if *H. pylori* does enter the distribution system, potentially through a break in treatment or infiltration into the system, the disinfectant residuals found in the distribution system are probably not sufficient for inactivation (Baker *et al.*, 2002).

#### 6.4.6 Assessment

Currently, there are no regulations governing the presence of *H. pylori* in drinking water, either nationally or internationally. The U.S. EPA has included it on their list of candidate contaminants for possible regulation in drinking water. Further studies are needed to confirm that *H. pylori* are present in drinking water in a viable state and that they can be transmitted by this medium.

# 7.0 Conclusions and recommendations

The organisms identified as current bacterial waterborne pathogens of concern within this document are those that have a well-established history of being responsible for bacterial waterborne outbreaks, presenting as gastrointestinal illnesses. Drinking water is not tested for these organisms directly; instead, *E. coli* is used as an indicator of their presence. The guideline value of no *E. coli* in 100 mL of drinking water is set to protect human health from these organisms.

The emerging pathogenic bacteria of concern outlined here also have the potential to be spread through drinking water, but they do not correlate with the presence of *E. coli* or with other commonly used drinking water quality indicators, such as total coliforms and HPC bacteria. In most cases, there are no satisfactory microbiological indicators of their presence. Although surrogate organisms are not known, it is not practical to routinely monitor the drinking water for the pathogens themselves. The use of a multiple-barrier approach, including source water protection (where possible), adequate treatment, and a well-maintained distribution system, can reduce these pathogens to non-detectable levels or to levels that have never been associated with human illness.

# 8.0 References

Alamanos, Y., Maipa, V., Levidiotou, S., and Gessouli, E. (2000) A community waterborne outbreak of gastro-enteritis attributed to *Shigella sonnei*. Epidemiol. Infect., 125: 499–503.

Allaker, R.P., Young, K.A., Hardie, J.M., Domizio, P., and Meadows, N.J. (2002) Prevalence of *Helicobacter pylori* at oral and gastrointestinal sites in children: evidence for possible oral-to-oral transmission. J. Med. Microbiol., 51: 312–317.

Allen, D.A., Austin, B., and Colwell, R.R. (1983) *Aeromonas media*, a new species isolated from river water. Int. J. Syst. Bacteriol., 33: 599–604.

Angulo, F.J., Tippen, S., Sharp, D.J., Payne, B.J., Collier, C., Hill, J.E., Barrett, T.J., Clark, R.M., Geldreich, E.E., Donnell, H.D., Jr., and Swerdlow, D.L. (1997) A community waterborne outbreak of salmonellosis and the effectiveness of a boil water order. Am. J. Public Health, 87: 580–584.

APHA (American Public Health Association) / AWWA (American Water Works Association / WEF (Water Environment Federation) (1998) Standard methods for the examination of water and wastewater. 20th edition. Washington, DC.

Arnow, P.M., Weil, D., and Para, M.F. (1985) Prevalence and significance of *Legionella pneumophila* contamination of residential hot-tap water systems. J. Infect. Dis., 152(1): 145–151.

Aronson, T., Holtzman, A., Glover, N., Boian, M., Froman, S., Berlin, O.G.W., Hill, H., and Stelma, G., Jr. (1999) Comparison of large restriction fragments of *Mycobacterium avium* isolates recovered from AIDS and non-AIDS patients with those of isolates from potable water. J. Clin. Microbiol., 37(4): 1008–1012.

Ashbolt, N.J., Ball, A., Dorsch, M., Turner, C., Cox, P., Chapman, A., and Kirov, S.M. (1995) The identification of human health significance of environmental aeromonads. Water Sci. Technol., 31: 263–269.

Auger, P., Pouliot, B., De Grace, M., Milot, C., Lafortune, M., and Bergeron, Z. (1981) Epidemic of bacillary dysentery. Can. Med. Assoc. J., 125: 733–736.

AWWA Committee Report (1999) Emerging pathogens — bacteria. J. Am. Water Works Assoc., 91(9): 101-109.

Baker, K.H., Hegarty, J.P., Redmond, B., Reed, N.A., and Herson, D.S. (2002) Effect of oxidizing disinfectants (chlorine, monochloramine, and ozone) on *Helicobacter pylori*. Appl. Environ. Microbiol., 68: 981–984.

Batton, R., Raviglione, M.C., Palagiano, A., Boyle, J.F., Sabatini, M.T., Sayad, K., and Ottaviano, L.J. (1990) *Helicobacter pylori* infection in patients with acquired immune deficiency syndrome. Am. J. Gastroenterol., 85: 1576–1579.

Benin, A.L., Benson, R.F., Arnold, K.E., Fiore, A.E., Cook, P.G., Williams, L.K., Fields, B., and Besser, R.E. (2002) An outbreak of travel-associated Legionnaires' disease and Pontiac fever: the need for enhanced surveillance of travel-associated legionellosis in the United States. J. Infect. Dis., 185: 237–243.

Berger, P.S. and Argaman, Y. (eds.). (1983) Assessment of microbiology and turbidity standards for drinking water. U.S. Environmental Protection Agency, Washington, DC (EPA 570-9-83-001).

Berk, S.G., Ting, R.S., Turner, G.W., and Ashburn, R.J. (1998) Production of respirable vesicles containing live *Legionella pneumophila* cells by two *Acanthamoeba* spp. Appl. Environ. Microbiol., 64: 279–286.

Bermudez, L.E., Parker, A., and Goodman, J.R. (1997) Growth within macrophages increases the efficiency of *Mycobacterium avium* in invading other macrophages by a complement receptor-independent pathway. Infect. Immun., 65: 1916–1925.

Bernagozzi, M., Bianucci, F., and Sacchetti, R. (1995) Prevalence of *Aeromonas* spp. in surface waters. Water Environ. Res., 67(7): 1060–1064.

Blaser, M.J., Wells, J.H., Powers, B., and Wang, W.L. (1980) Survival of *Campylobacter fetus* subsp. *jejuni* in biological milieus. J. Clin. Microbiol., 11: 309–313.

Bode, G., Rothenbacher, D., Brenner, H., and Adler, G. (1998) Pets are not a risk factor for *Helicobacter pylori* infection in young children: results of a population-based study in southern Germany. Pediatr. Infect. Dis. J., 17: 909–912.

Borchardt, M.A., Stemper, M.E., and Standridge, J.H. (2003) *Aeromonas* isolates from human diarrheic stool and groundwater compared by pulse-field gel electrophoresis. Emerg. Infect. Dis., 9: 224–228.

Borella, P., Montagna, M.T., Romano-Spica, V., Stampi, S., Stancanelli, G., Triassi, M., Neglia, R., Marchesi, I., Fantuzzi, G., Tatò, D., Napoli, C., Quaranta, G., Laurenti, P., Leoni, E., De Luca, G., Ossi, C., Moro, M., and Ribera D'Alcalà, G. (2004) *Legionella* infection risk from domestic hot water. Emerg. Infect. Dis., 10(3): 457–464.

Boring, J.R., III, Martin, W.T., and Elliott, L.M. (1971) Isolation of *Salmonella typhimurium* from municipal water, Riverside, California, 1965. Am. J. Epidemiol., 93: 49–54.

Breiman, R.F. and Butler, J.C. (1998) Legionnaires' disease: clinical, epidemiological, and public health perspectives. Semin. Respir. Infect., 13: 84–89.

Brenner, H., Rothenbacher, D., Bode, G., Dieudonné, P., and Adler, G. (1999) Active infection with *Helicobacter pylori* in healthy couples. Epidemiol. Infect., 122: 91–95.

Brown, C.M., Nuorti, P.J., Breiman, R.F., Hathcock, A.L., Fields, B.A., Lipman, H.B., Llewellyn, G.C., Hofmann, J., and Cetron, M. (1999) A community outbreak of Legionnaires' disease linked to hospital cooling towers: an epidemiological method to calculate dose of exposure. Int. J. Epidemiol., 28: 353–359.

Bruce-Grey-Owen Sound Health Unit (2000) The investigative report on the Walkerton outbreak of waterborne gastroenteritis. May–June, Owen Sound (http://www.publichealthgreybruce.on.ca/\_private/Report/SPReport.htm).

Burke, V., Robinson, J., Gracey, M., Peterson, D., and Partridge, K. (1984) Isolation of *Aeromonas hydrophila* from a metropolitan water supply: seasonal correlation with clinical isolation. Appl. Environ. Microbiol., 48(2): 361–366.

Byrd, J.J., Xu, H.S., and Colwell, R.R. (1991) Viable but nonculturable bacteria in drinking water. Appl. Environ. Microbiol., 57: 875–878.

Caprioli, T., Drapeau, A.J., and Kasatiya, S. (1978) *Yersinia enterocolitica*: serotypes and biotypes isolated from humans and the environment in Quebec, Canada. Appl. Environ. Microbiol., 8: 7–11.

Carter, A.M., Pacha, R.E., Clark, G.W., and Williams, E.A. (1987) Seasonal occurrence of *Campylobacter* spp. in surface waters and their correlation with standard indicator bacteria. Appl. Environ. Microbiol., 53: 523–526.

CDC (Centers for Disease Control and Prevention) (1996) *Shigella sonnei* outbreak associated with contaminated drinking water — Island Park, Idaho, August 1995. J. Am. Med. Assoc., 275: 1071.

CDC (Centers for Disease Control and Prevention) (1999) Iron deficiency anemia in Alaska native children — Hooper Bay, Alaska, 1999. Morb. Mortal. Wkly. Rep., 48: 714–716.

Chauret, C., Volk, C., Creason, R., Jarosh, J., Robinson, J., and Warnes, C. (2001) Detection of *Aeromonas hydrophila* in a drinking-water distribution system: a field and pilot study. Can. J. Microbiol., 47: 782–786.

Chen, K.T., Chen, C.J., and Chiu, J.P. (2001) A school waterborne outbreak involving both *Shigella sonnei* and *Entamoeba histolytica*. J. Environ. Health, 64: 9–13.

Cirillo, J.D., Falkow, S., Tompkins, L.S., and Bermudez, L.E. (1997) Interaction of *Mycobacterium avium* with environmental amoebae enhances virulence. Infect. Immun., 65(9): 3759–3767.

*Guidelines for Canadian Drinking Water Quality: Guideline Technical Document* 

Clark, C.G., Price, L., Ahmed, R., Woodward, D.L., Melito, P.L., Rodgers, F.G., Jamieson, F., Ciebin, B., Li, A., and Ellis, A. (2003) Characterization of waterborne outbreak-associated *Campylobacter jejuni*, Walkerton, Ontario. Emerg. Infect. Dis., 9: 1232–1241.

Covert, T.C., Rodgers, M.R., Reyes, A.L., and Stelma, G.N., Jr. (1999) Occurrence of nontuberculous mycobacteria in environmental samples. Appl. Environ. Microbiol., 65(6): 2492–2496.

Cunliffe, D.A. (1990) Inactivation of Legionella pneumophila by monochloramine. J. Appl. Bacteriol., 68: 453-459.

Dufour, A.P. and Jakubowski, W. (1982) Drinking water and Legionnaires' disease. J. Am. Water Works Assoc., 74: 631–637.

du Moulin, G.C., Stottmeier, K.D., Pelletier, P.A., Tsang, A.Y., and Hedley-White, J. (1988) Concentration of *Mycobacterium avium* by hospital hot water systems. J. Am. Med. Assoc., 260: 1599–1601.

Dunn, B.E., Cohen, H., and Blaser, M.J. (1997) Helicobacter pylori. Clin. Microbiol. Rev., 10: 720-741.

Dutka, B.J., Walsh, K., Ewan, P., El-Shaarawi, A., and Tobin, R.S. (1984) Incidence of *Legionella* organisms in selected Ontario (Canada) cities. Sci. Total Environ., 39: 237–249.

Eaton, K.A., Catrenich, C.E., Makin, K.M., and Krakowka, S. (1995) Virulence of coccoid and bacillary forms of *Helicobacter pylori* in gnotobiotic piglets. J. Infect. Dis., 171: 459–462.

Eden, K.V., Rosenburg, M.L., Stoopler, M., Wood, B.T., Highsmith, A.K., Skaliy, P., Wells, J.G., and Feeley, J.C. (1977) Waterborne gastrointestinal illness at a ski resort. Public Health Rep., 92: 245–250.

Edwards, P.D., Carrick, J., Turner, J., Lee, A., Mitchell, H., and Cooper, D.A. (1991) *Helicobacter pylori*-associated gastritis is rare in AIDS: antibiotic effect or a consequence of immunodeficiency? Am. J. Gastroenterol, 86: 1761–1764.

El-Sherbeeny, M.R., Bopp, C., Wells, J.G., and Morris, G.K. (1985) Comparison of gauze swabs and membrane filters for isolation of *Campylobacter* spp. from surface water. Appl. Environ. Microbiol., 50: 611–614.

El-Taweel, G.E. and Shaban, A.M. (2001) Microbiological quality of drinking water at eight water treatment plants. Int. J. Environ. Health Res., 11: 285–290.

Engstrand, L. (2001) Helicobacter in water and waterborne routes of transmission. J. Appl. Microbiol., 90: 80S-84S.

Enroth, H. and Engstrand, L. (1995) Immunomagnetic separation and PCR for detection of *Helicobacter pylori* in water and stool specimens. J. Clin. Microbiol., 33: 2162–2165.

Falkinham, J.O., III, Norton, C.D., and LeChevallier, M.W. (2001) Factors influencing numbers of *Mycobacterium avium*, *Mycobacterium intracellulare*, and other mycobacteria in drinking water distribution systems. Appl. Environ. Microbiol., 67(3): 1225–1231.

Fallon, R.J. and Rowbotham, T.J. (1990) Microbiological investigations into an outbreak of Pontiac fever due to *Legionella micdadei* associated with use of a whirlpool. J. Clin. Pathol., 43: 479–483.

Fallon, R.J., Goldberg, D.J., Wrench, J.G., Green, S.T., and Emslie, J.A.M. (1993) Pontiac fever in children. In: *Legionella*: Current status and emerging perspectives. J.M. Barbaree, R.F. Breiman, and A.P. Dufour (eds.). American Society for Microbiology, Washington, DC. pp. 50–51.

Feldman, R.A., Eccersley, A.J.P., and Hardie, J.M. (1998) Epidemiology of *Helicobacter pylori*: acquisition, transmission, population prevalence and disease-to-infection ratio. Br. Med. Bull., 54: 39–53.

Ferguson, D.A., Jr., Li, C., Patel, N.R., Mayberry, W.R., Chi, D.S., and Thomas, E. (1993) Isolation of *Helicobacter pylori* from saliva. J. Clin. Microbiol., 31: 2802–2804.

Fernandez, M.C., Giampaolo, B.N., Ibanez, S.B., Guagliardo, M.V., Esnaola, M.M., Conca, L., Valdivia, P., Stagnaro, S.M., Chiale, C., and Frade, H. (2000) *Aeromonas hydrophila* and its relation with drinking water indicators of microbiological quality in Argentine. Genetica, 108: 35–40.

Fields, B.S. (1996) The molecular ecology of Legionellae. Trends Microbiol., 4: 286-290.

Friis, L., Engstrand, L., and Edling, C. (1996) Prevalence of *Helicobacter pylori* infection among sewage workers. Scand. J. Work Environ. Health, 22: 364–368.

Furuta, T., Kamata, T., Takashima, M., Futami, H., Arai, H., Hanai, H., and Kaneko, E. (1997) Study of transmission routes of *Helicobacter pylori* in relation to seroprevalence of hepatitis A virus. J. Clin. Microbiol., 35: 1891–1893.

Gavriel, A.A., Landre, J.P.B., and Lamb, A.J. (1998) Incidence of mesophilic *Aeromonas* within a public drinking water supply in north-east Scotland. J. Appl. Microbiol., 84: 383–392.

Glick, T.H., Gregg, M.B., Berman, B., Mallison, G., Rhodes, W.W., Jr., and Kassanoff, I. (1978) Pontiac fever: an epidemic of unknown etiology in a health department. I: Clinical and epidemiologic aspects. Am. J. Epidemiol., 107: 149–160.

Glover, N., Holtzman, A., Aronson, T., Froman, S., Berlin, O.G.W., Dominguez, P., Kunkel, K.A., Overturf, G., Steima, G., Jr., Smith, C., and Yakrus, M. (1994) The isolation and identification of *Mycobacterium avium* complex (MAC) recovered from Los Angeles potable water, a possible source of infection in AIDS patients. Int. J. Environ. Health Res., 4: 63–72.

Go, M.F. (1997) What are the host factors that place an individual at risk for *Helicobacter pylori*-associated disease? Gastroenterology, 113(6): S15–20.

Goodman, K.J., Correa, P., Tengana, A.J., Ramírez, H., DeLany, J.P., Pepinosa, O.G., Quiñones, M.L., and Parra, T.C. (1996) *Helicobacter pylori* infection in the Colombian Andes: a population-based study of transmission pathways. Am. J. Epidemiol., 144: 290–299.

Gosling, P.J. (1996) Pathogenic mechanisms. In: The genus *Aeromonas*. B. Austin, M. Altwegg, P. Gosling, and S. Joseph (eds.). John Wiley and Sons, London.

Grange, J.M. (1991) Environmental mycobacteria and human disease. Lepr. Rev., 62: 353-361.

Gugnani, H.C. (1999) Some emerging food and water borne pathogens. J. Commun. Dis., 31(2): 65-72.

Guthertz, L.S., Damsker, B., Bottone, E.J., Ford, E.G., Midura, T.D., and Janda, J.M. (1989) *Mycobacterium avium* and *Mycobacterium intracellulare* infections in patients with and without AIDS. J. Infect. Dis., 160: 1037–1041.

Hänninen, M.L. and Siitonen, A. (1995) Distribution of *Aeromonas* phenospecies and genospecies among strains isolated from water, foods or from human clinical samples. Epidemiol. Infect., 115: 39–50.

Haruma, K., Komoto, K., Kawaguchi, H., Okamoto, S., Yoshihara, M., Summii, K., and Kajiyama, G. (1995) Pernicious anemia and *Helicobacter pylori* infection in Japan: evaluation in a country with a high prevalence of infection. Am. J. Gastroenterol., 90: 1107–1110.

Havelaar, A.J., Schets, F.M., van Silfhout, A., Jansen, W.H., Wieten, G., and van der Kooij, D. (1992) Typing of *Aeromonas* strains from patients with diarrhoea and from drinking water. J. Appl. Bacteriol., 72: 435–444.

Hazell, S.L., Mitchell, H.M., Hedges, M., Shi, X., Hu, P.J., Li, Y.Y., Lee, A., and Reiss-Levy, E. (1994) Hepatitis A and evidence against the community dissemination of *Helicobacter pylori* in feces. J. Infect. Dis., 170: 686–689.

Hazra, R., Lee, S.H., Maslow, J.N., and Husson, R.N. (2000) Related strains of *Mycobacterium avium* cause disease in children with AIDS and in children with lymphadenitis. J. Infect. Dis., 181: 1298–1303.

Health Canada (2003) Guidelines for Canadian drinking water quality: Supporting documentation — Turbidity: Water Quality and Health Bureau, Healthy Environments and Consumer Safety Branch, Health Canada, Ottawa, Ontario.

Health Canada (2004a) Guidelines for Canadian drinking water quality: Supporting documentation — Protozoa: *Giardia* and *Cryptosporidium*. Water Quality and Health Bureau, Healthy Environments and Consumer Safety Branch, Health Canada, Ottawa, Ontario.

Health Canada (2004b) Guidelines for Canadian drinking water quality: Supporting documentation — Enteric viruses. Water Quality and Health Bureau, Healthy Environments and Consumer Safety Branch, Health Canada, Ottawa, Ontario.

Health Canada (2006a) Guidelines for Canadian drinking water quality: Technical Guideline Document— *Escherichia coli*. Water Quality and Health Bureau, Healthy Environments and Consumer Safety Branch, Health Canada, Ottawa, Ontario.

Health Canada (2006b) Guidelines for Canadian drinking water quality: Technical Guideline Document — Total coliforms. Water Quality and Health Bureau, Healthy Environments and Consumer Safety Branch, Health Canada, Ottawa, Ontario.

Hegarty, J.P., Dowd, M., and Baker, K.H. (1999) Occurrence of *Helicobacter pylori* in surface water in the United States. J. Appl. Microbiol., 87: 697–701.

Hermon-Taylor, J. and El-Zaatari, F.A.K. (2004) The *Mycobacterium avium* subspecies *paratuberculosis* problem and its relation to the causation of Crohn disease. In: Pathogenic mycobacteria in water, a guide to public health consequences, monitoring and management. S. Pedley, J. Bartram, G. Rees, A. Dufour, and J.A. Cotruvo (eds.). Published by IWA Publishing, London, on behalf of the World Health Organization, Geneva. pp. 74–94.

Hershey, J., Burrus, B., Marcussen, V., Notter, J., Watson, K., Wolford, R., Shaffner, R.E., III, Barrett, E., Woolard, D., Branch, L., Hackler, R., Rouse, B., Gibson, L., Jenkins, S., Rullan, J., Miller, G., Jr., and Curran, S. (1997) Legionnaires disease associated with a whirlpool spa display — Virginia, September–October, 1996. Morb. Mortal. Wkly. Rep., 46: 83–86.

Hoebe, C.J.P., Cluitmans, J.J.M., and Wagenvoort, J.H.T. (1998) Two fatal cases of nosocomial *Legionella pneumophila* pneumonia associated with a contaminated cold water supply. Eur. J. Clin. Micriobiol. Infect. Dis., 17: 740–749.

Holmes, P., and Nicolls, L.M. (1995) Aeromonads in drinking water supplies — their occurrence and significance. J. Chartered Inst. Water Environ. Manage., 5: 464–469.

Holmes, P., Nicolls, L.M., and Sartory, D.P. (1996) The ecology of mesophilic *Aeromonas* in the aquatic environment. In: The genus *Aeromonas*. B. Austin, M. Altwegg, P. Gosling, and S.W. Joseph (eds.). John Wiley & Sons, New York, NY. p. 127.

Hopkins, R.J., Vial, P.A., Ferreccio, C., Ovalle, J., Prado, P., Soromayor, V., Russell, R.G., Wassermann, S.S., and Morris, J.G., Jr. (1993) Seroprevalence of *Helicobacter pylori* in Chile: vegetables may serve as one route of transmission. J. Infect. Dis., 168: 222–226.

Hörman, A., Rimhanen-Finne, R., Maunula, L., von Bonsdorff, C., Torvela, N., Heikinheimo, A., and Hänninen, M. (2004) *Campylobacter* spp., *Giardia* spp., *Cryptosporidium* spp., Noroviruses, and indicator organisms in surface water in southwestern Finland, 2000–2001. Appl. Environ. Microbiol., 70(1): 87–95.

Hultén, K., Han, S.W., Enroth, H., Klein, P.D., Opekun, A.R., Gilman, R.H., Evans, D.G., Engstrand, L., Graham, D.Y., and El-Zaatari, F. (1996) *Helicobacter pylori* in the drinking water in Peru. Gastroenterology, 110: 1031–1035.

Hultén, K., Enroth, H., Nyström, T., and Engstrand, L. (1998) Presence of *Helicobacter* species DNA in Swedish water. J. Appl. Microbiol., 85: 282–286.

Hunter, P.J. (1997) Mycobacterial disease. In: Waterborne disease epidemiology and ecology. John Wiley and Sons, Chichester, England. pp. 189–198.

IARC (International Agency for Research on Cancer) (1994) Schistosomes, liver flukes and *Helicobacter pylori*. IARC Monogr. Eval. Carcinog. Risks Hum., 61: 1–241.

Ichiyama, S., Shimokata, K., and Tsukamura, M. (1988) The isolation of *Mycobacterium avium* complex from soil, water, and dusts. Microbiol. Immunol., 32(7): 733–739.

Iivanainen, E.K., Martikainen, P.J., Vaananen, P.K., and Katila, M.L. (1993) Environmental factors affecting the occurrence of mycobacteria in brook waters. Appl. Environ. Microbiol., 59: 398–404.

Jackson, S.G., Goodbrand, R.B., Johnson, R.P., Odorico, V.G., Alves, D., Rahn, K., Wilson, J.B., Welch, M.K., and Khakhria, R. (1998) *Escherichia coli* O157:H7 diarrhoea associated with well water and infected cattle on an Ontario farm. Epidemiol. Infect., 120: 17–20.

Janda, J.M. (1991) Recent advances in the study of taxonomy, pathogenicity, and infectious syndromes associated with the genus *Aeromonas*. Clin. Microbiol. Rev., 4(4): 397–410.

Janda, J.M. and Abbott, S.L. (1998) Evolving concepts regarding the genus *Aeromonas*: an expanding panorama of species, disease presentations, and unanswered questions. Clin. Infect. Dis., 27: 332–344.

Jekel, P. (1993) Mode of transmission of Helicobacter pylori. J. Can. Inst. Public Health Inspect., 37(1): 22, 27.

Jernigan, D.B., Hofmann, J., Cetron, M.S., Genese, C.A., Nuorti, J.P., Fields, B.S., Benson, R.F., Carter, R.J., Edelstein, P.H., Guerrero, I.C., Paul, S.M., Lipman, H.B., and Breiman, R.F. (1996) Outbreak of Legionnaires' disease among cruise ship passengers exposed to a contaminated whirlpool spa. Lancet, 347: 494–499.

Johnson, C., Rice, E., and Reasoner, D. (1997) Inactivation of *Helicobacter pylori* by chlorination. Appl. Environ. Microbiol., 63: 4969–4970.

Johnson, J.D., Raff, M.J., and Van-Arsdall, J.A. (1984) Neurological manifestations of Legionnaires' disease. Medicine, 63: 303–310.

Guidelines for Canadian Drinking Water Quality: Guideline Technical Document

Kahana, L.M., Kay, J.M., Yakrus, M.A., and Waserman, S. (1997) *Mycobacterium avium* complex infection in an immunocompetent young adult related to hot tub exposure. Chest, 111: 242–245.

Kaneko, M. (1998) Chlorination of pathogenic E. coli O157. Water Sci. Technol., 38(12): 141-144.

Kilvington, S. and Price, J. (1990) Survival of *Legionella pneumophila* within cysts of *Acanthamoeba polyphaga* following chlorine exposure. J. Appl. Bacteriol., 68: 519–525.

Kirschner, R.A., Parker, B.C., and Falkinham, J.O., III (1992) Epidemiology of infection by nontuberculous mycobacteria: *Mycobacterium avium*, *Mycobacterium intracellulare*, and *Mycobacterium scrofulaceum* in acid, brown-water swamps of the southeastern United States and their association with environmental variables. Am. Rev. Respir. Dis., 145: 271–275.

Kirschner, R.A., Jr., Parker, B.C., and Falkinham, J.O., III (1999) Humic and fulvic acids stimulate the growth of *Mycobacterium avium*. FEMS Microbiol. Ecol., 30: 327–332.

Klein, P.D., Graham, D.Y., Gaillour, A., Opckun, A.R., and Smith, E.O. (1991) Water source as risk factor for *Helicobacter pylori* infection in Peruvian children. Lancet, 337: 1503–1506.

Knøchel, S. (1991) Chlorine resistance of motile Aeromonas spp. Water Sci. Technol., 24: 327-330.

Kool, J.L., Carpenter, J.C., and Fields, B.S. (1999) Effect of monochloramine disinfection of municipal drinking water on risk of nosocomial Legionnaires' disease. Lancet, 353: 272–276.

Kramer, M.H.J. and Ford, T.E. (1994) Legionellosis: Ecological factors of an environmentally "new" disease. Zentralbl. Hyg., 195: 470–482.

Krovacek, K., Faris, A., Baloda, S.J., Lindberg, T., Peterz, M., and Mansson, I. (1992) Isolation and virulence profiles of *Aeromonas* spp. from different municipal drinking water supplies in Sweden. J. Food Microbiol., 9(3): 215–222.

Kühn, I., Allestam, G., Huys, G., Janssen, P., Kersters, K., Krovacek, K., and Stenstrom, T. (1997) Diversity, persistence, and virulence of *Aeromonas* strains isolated from drinking water distribution systems in Sweden. Appl. Environ. Microbiol., 63: 2708–2715.

Lafrance, G., Lafrance, R., Roy, G.L., Ouellete, D., and Bourdeau, R. (1986) Outbreak of enteric infection following a field trip — Ontario. Can. Dis. Wkly. Rep., 12: 171–172.

LeChevallier, M.W. (1999) *Mycobacterium avium* complex. In: Manual of water supply practices, waterborne pathogens. American Water Works Association, Denver, CO. pp. 99–102.

Le Dantec, C., Duguet, J., Montiel, A., Dumoutier, N., Dubrou, S., and Vincent, V. (2002) Chlorine disinfection of atypical mycobacteria isolated from a water distribution system. Appl. Environ. Microbiol., 68(3): 1025–1032.

Lee, T.C., Stout, J.E., and Yu, V.L. (1988) Factors predisposing to *Legionella pneumophila* colonization in residential water systems. Arch. Environ. Health, 43(1): 59–62.

Lieberman, R.J., Shadix, L.C., Newport, B.S., Crout, S.R., Buescher, S.E., Safferman, R.S., Stetler, R.E., Lye, D., Shay Fout, D., and Dahling, D.R. (1994) Source water microbial quality of some vulnerable public ground water supplies. In: Proceedings of the 1994 Water Quality Technology Conference, Part II. pp. 1425–1436. American Water Works Association, Denver, CO.

Lieberman, D., Porath, A., Schlaeffer, F., Lieberman, D., and Boldur, I. (1996) *Legionella* species community-acquired pneumonia. A review of 56 hospitalized adult patients. Chest, 109: 1243–1249.

Lin, Y.E., Stout, J.E., Yu, Y.L., and Vidic, R.D. (1998) Disinfection of water distribution systems for *Legionella*. Semin. Respir. Infect., 13: 147–159.

Lindkvist, P., Wadström, T., and Giesecke, J. (1995) *Helicobacter pylori* and foreign travel. J. Infect. Dis., 172: 1135–1136.

Luman, W., Zhao, Y., Ng, H.S., and Ling, K.L. (2002) *Helicobacter pylori* infection is unlikely to be transmitted between partners: evidence from genotypic study in partners of infected patients. Eur. J. Gastroenterol. Hepatol., 14: 521–528.

Mangione, E.J., Remis, R.S., Tait, K.A., McGee, H.B., Gorman, G.W., Wentworth, B.B., Baron, P.A., Hightower, A.W., Barbaree, J.M., and Broome, C.V. (1985) An outbreak of Pontiac fever related to whirlpool use, Michigan 1982. J. Am. Med. Assoc., 253: 535–539.

Mangione, E.J., Huitt, G., Lenaway, D., Beebe, D., Bailey, A., Figoski, M., Rau, M.P., Albrecht, K.D., and Yakrus, M.A. (2001) Nontuberculous mycobacterial disease following hot tub exposure. Emerg. Infect. Dis., 7(6): 1039–1042.

Mapstone, N.P., Lynch, D.A., Lewis, F.A., Axon, A.T., Tompkins, D.S., Dixon, M.F., and Quirke, P. (1993) PCR identification of *Helicobacter pylori* in faces from gastritis patients [Letter]. Lancet, 341: 447.

Mateos, D., Anguita, J., Naharro, G., and Paniagua, C. (1993) Influence of growth temperature on the production of extracellular virulence factors and pathogenicity of environmental and human strains of *Aeromonas hydrophila*. J. Appl. Bacteriol., 74: 111–118.

Matysiak-Budnik, T., Briet, F., Heyman, M., and Megraud, F. (1995) Laboratory-acquired *Helicobacter pylori* infection [letter]. Lancet, 346: 1489–1490.

McFeters, G.A., Bissonnette, G.K., Jezeski, J.J., Thomson, C.A., and Stuart, D.G. (1974) Comparative survival of indicator bacteria and enteric pathogens in well water. Appl. Microbiol., 27: 823–829.

McKeown, I., Orr, P., MacDonald, S., Kabani, A., Brown, R., Coghlan, G., Dawood, M., Embil, J., Sargent, M., Smart, G., and Bernstein, C.N. (1999) *Helicobacter pylori* in the Canadian Arctic: seroprevalence and detection in community water samples. Am. J. Gastroenterol., 94: 1823–1829.

McNeil, C.A., Out, K., Pagan, R.T., McMyre, P., Black, W.A., and Mathias, R.G. (1981) Possible waterborne *Campylobacter* outbreak — British Columbia. Can. Dis. Wkly. Rep., 7: 223–227.

Medema, G.J., Wondergem, E., van Dijk-Looyaard, A.M., and Havelaar, A.H. (1991) Effectivity of chlorine dioxide to control *Aeromonas* in drinking water distribution systems. Water Sci. Technol., 24: 325–326.

Mentzing, L.O. (1981) Waterborne outbreaks of Campylobacter enteritis in central Sweden. Lancet, ii: 352-354.

Merino, S., Rubires, X., Knochel, S., and Tomas, J.M. (1995) Emerging pathogens: *Aeromonas* spp. Int. J. Food Microbiol., 28: 157–168.

Millership, S.E., Barer, M.R., and Tabaqchali, S. (1986) Toxin production by *Aeromonas* spp. from different sources. J. Med. Microbiol., 22: 311–314.

Milman, N., Rosenstock, S., Anderson, L., Jorgensen T., and Bonnevie, O. (1998) Serum ferritin, hemoglobin, and *Helicobacter pylori* infection: a seroepidemiologic survey comprising 2794 Danish adults. Gastroenterology, 115: 268–274.

Mitchell, D.O. and Starzyk, M.J. (1975) Survival of *Salmonella* and other indicator microorganisms. Can. J. Microbiol., 21: 1420–1421.

Moe, C.L. (1997) Waterborne transmission of infectious agents. In: Manual of environmental microbiology. C.J. Hurst, G.R. Knudsen, M.J. McInerney, L.D. Stetzenbach, and M.V. Walter (eds.). ASM Press, Washington, DC.

Montecalvo, M.A., Forester, G., Tsang, A.Y., du Moulin, G., and Wormser, G.P. (1994) Colonisation of potable water with *Mycobacterium avium* complex in homes of HIV-infected patients [letter]. Lancet, 343: 1639.

Morgan, D.R., Johnson, P.C., DuPont, H.L., Satterwhite, T.K., and Wood, L.V. (1985) Lack of correlation between known virulence properties of *Aeromonas hydrophila* and enteropathogenicity for humans. Infect. Immun., 50: 62–65.

Morris, A., and Nicholson, G. (1987) Ingestion of *Campylobacter pyloris* causes gastritis and raised fasting gastric pH. Am. J. Gastroenterol., 82: 192–199.

Moyer, N.P. (1987) Clinical significance of *Aeromonas* species isolated from patients with diarrhea. J. Clin. Microbiol., 25: 2044–2048.

Moyer, N.P. (1999) *Aeromonas*. In: AWWA manual M48: Waterborne pathogens. American Water Works Association, Denver, CO. pp. 63–66.

Moyer, N.P., Luccini, G.M., Holcomb, L.A., Hall, N.H., and Altwegg, M. (1992) Application of ribotyping for differentiating aeromonads isolated from clinical environmental sources. Appl. Environ. Microbiol., 58: 1940–1944.

Nakano, J., Kameyama, T., Venkateswaran, K., Kawakami, J., and Hashimoto, J. (1990) Distribution and characterisation of hemolytic, and enteropathogenic motile *Aeromonas* in aquatic environment. Microbial Immunol., 34: 447–458.

Nelson, D.P., Rensimer, E.R., and Raffin, T.A. (1985) *Legionella pneumophila* pericarditis without pneumonia. Arch. Intern. Med., 145: 926.

Nguyen, M.H., Stout, J.E., and Yu, V.L. (1991) Legionellosis. Infect. Dis. Clin. North Am., 5: 561–584.

Nguyen, A.M.H., Engstrand, L., Genta, R.M., Graham, D.Y., and El-Zaatari, F.A.K. (1993) Detection of *Helicobacter pylori* in dental plaque by reverse transcription–polymerase chain reaction. J. Clin. Microbiol., 31: 783–787.

Nichols, G., Ford, T., Bartram, J., Dufour, A., and Portaels, F. (2004) Introduction. In: Pathogenic mycobacteria in water, a guide to public health consequences, monitoring and management. S. Pedley, J. Bartram, G. Rees, A. Dufour, and J.A. Cotruvo. Published by IWA Publishing, London, on behalf of the World Health Organization, Geneva. pp 1–14.

Notermans, S., Havelaar, A., Jansen, W., Kozaki, S., and Guinee, P. (1986) Production of "Asao toxin" by *Aeromonas* strains isolated from feces and drinking water. J. Clin. Microbiol., 23: 1140–1142.

OME (Ontario Ministry of the Environment) (1980) Yersinia enterocolitica in recreational lakes and sewage systems. Lakeshore Capacity Study, Laboratory Services Branch, Ontario Ministry of the Environment, Toronto, Ontario.

*Guidelines for Canadian Drinking Water Quality: Guideline Technical Document* 

Oredugba, O., Mazumdar, D.C., Smoller, M.B., Meyer, J., and Lubowitz, H. (1980) Acute renal failure in Legionnaires' disease. Clin. Nephrol., 13: 142–145.

Ormen, O. and Ostensvik, O. (2001) The occurrence of aerolysin-positive *Aeromonas* spp. and their cytotoxicity in Norwegian water sources. J. Appl. Microbiol., 90: 797–802.

Palmer, C.J., Tsai, Y-L., Paszko-Kolva, C., Mayer, C., and Sangermano, L.R. (1993) Detection of *Legionella* species in sewage and ocean water by polymerase chain reaction, direct fluorescent-antibody, and plate culture methods. Appl. Environ. Microbiol., 59: 3618–3624.

Parkin, D.M., Pisani, P., and Ferlay, J. (1999) Global cancer statistics. CA Cancer J. Clin., 49(1): 33-64.

Parsonnet, J. (1998) Helicobacter pylori. Infect. Dis. Clin. North Am., 12: 185–197.

Patchett, S.E. (1998) Helicobacter pylori eradication cost-benefit: the case against. Br. Med. Bull., 54: 251-257.

Payment, P., Gamache, F., and Paquette, G. (1988) Microbiological and virological analysis of water from two water filtration plants and their distribution systems. Can. J. Microbiol., 34: 1304–1309.

Payment, P., Franco, E., and Siemiatycki, J. (1993) Absence of relationship between health effects due to tap water consumption and drinking water quality parameters. Water Sci. Technol., 27: 137–143.

Peach, H.G., Bath, N.E., and Farish, S.J. (1998) *Helicobacter pylori* infection: an added stressor on iron status of women in the community. Med. J. Aust., 169: 188–190.

Peeters, J.E., Mazas, E.A., Masschelein, W.J., Martinez de Maturana, I.V., and Debacker, E. (1989) Effect of disinfection of drinking water with ozone or chlorine dioxide on survival of *Cryptosporidium parvum* oocysts. Appl. Environ. Microbiol., 55: 1519–1522.

Perez-Perez, G.I. (1997) Role of *Helicobacter pylori* infection in the development of pernicious anemia. Clin. Infect. Dis., 25: 1020–1022.

Plum, G. and Clark-Curtiss, J.E. (1994) Induction of *Mycobacterium avium* gene expression following phagocytosis by human macrophages. Infect. Immun., 62: 476–483.

Poffe, R. and Op de Beeck, E. (1991) Enumeration of *Aeromonas hydrophila* from domestic wastewater treatment plants and surface waters. J. Appl. Bacteriol., 71: 366–370.

Postius, S. (2001) Helicobacter pylori. Contrib. Microbiol., 8: 35-50.

Reynolds, K.A. (2001) Return of the MAC: Risks of waterborne *Mycobacterium avium*. Water Conditioning and Purification Magazine, WC&P International, June. pp. 90–93.

Rice, E.W. (1999) *Escherichia coli*. In: AWWA manual M48: Waterborne pathogens. American Water Works Association, Denver, CO. pp. 75–78.

Rice, E.W., Clark, R.M., and Johnson, C.H. (2000) Chlorine inactivation of *Escherichia coli* O157:H7. Emerg. Infect. Dis., 5(3): 461–463.

Rogers, J., Dowsett, A.B., Dennis, P.J., Lee, J.V., and Keevil, C.W. (1994) Influence of temperature and plumbing material selection on biofilm formation and growth of *Legionella pneumophila* in a model potable water system containing complex microbial flora. Appl. Environ. Microbiol., 60(5): 1585–1592.

Rothenbacher, D., Bode, G., Berg, G., Knayer, U., Gonser, T., Adler, G., and Brenner, H. (1999) *Helicobacter pylori* among preschool children and their parents: evidence of parent–child transmission. J. Infect. Dis., 179: 398–402.

Rowbotham, T.J. (1986) Current views on the relationships between amoebae, legionellae and man. Isr. J. Med. Sci., 22: 678–689.

Rowland, M. and Drumm, B. (1998) Climical significance of *Helicobacter* infection in children. Br. Med. Bull., 54: 95–103.

Rudi, J., Toppe, H., Marx, N., Zuna, I., Theilmann, L., Stremmel, W., and Raedsch, R. (1997) Risk of infection with *Helicobacter pylori* and hepatitis A virus in different groups of hospital workers. Am. J. Gastroenterol., 92: 293–295.

Sacks, J.J., Spencer, L., Baldy, L., Berta, S., Patton, C.M., White, M.C., Bigler, W.J., and Witte, J.J. (1986) Epidemic campylobacteriosis associated with a community water supply. Am. J. Public Health, 76: 424–428.

Sautour, M., Mary, P., Chihib, N.E., and Hornez, J.P. (2003) The effects of temperature, water activity and pH on the growth of *Aeromonas hydrophila* and on its subsequent survival in microcosm water. J. Appl. Microbiol., 95: 807–813.

Schiemann, D.A. (1978) Isolation of *Yersinia enterocolitica* from surface and well waters in Ontario. Can. J. Microbiol., 24: 1048–1052.

Schubert, R.H.W. (1991) Aeromonads and their significance as potential pathogens in water. J. Appl. Bacteriol. Symp. Suppl., 70: 131S–135S.

Schulze-Röbbecke, R., Janning, B., and Fischeder, R. (1992) Occurrence of mycobacteria in biofilm samples. Tuber. Lung Dis., 73: 141–144.

Scott, D., Weeks, D., Melchers, K., and Sachs, G. (1998) The life and death of *Helicobacter pylori*. Gut, 43(Suppl. 1): S56–S60.

Seligmann, R. and Reitler, R. (1965) Enteropathogens in water with low *Esch. coli* titer. J. Am. Water Works Assoc., 57: 1572–1574.

Shahamat, M., Mai, U., Paszko-Kolva, C., Kessel, M., and Colwell, R.R. (1993) Use of autoradiography to assess viability of *Helicobacter pylori* in water. Appl. Environ. Microbiol., 59: 1231–1235.

Sisti, M., Albano, A., and Brandi, G. (1998) Bactericidal effect of chlorine on motile *Aeromonas* spp. in drinking water supplies and influence of temperature on disinfection efficacy. Lett. Appl. Micriobiol., 26: 347–351.

Sniadack, D.E., Ostroff, S.D., Karlix, M.A., Smithwick, R.W., Schwartz, B., Spaucer, M.A., Silcox, V.A., and Good, R.C. (1992) A nosocomial pseudo-outbreak of *Mycobacterium xenopi* due to a contaminated potable water supply: lessons in prevention. Infect. Control Hosp. Epidemiol., 14: 636–641.

Sörberg, M., Nilsson, M., Iianberger, H., and Nilsson, L.E. (1996) Morphologic conversion of *Helicobacter pylori* from bacillary to coccoid form. Eur. J. Clin. Microbiol. Infect. Dis., 15: 216–219.

Spitalny, K.C., Vogt, R.L., Orciari, L.A., Witherell, L.E., Etkind, P., and Novick, L.F. (1984) Pontiac fever associated with a whirlpool spa. Am. J. Epidemiol., 120: 809–817.

Steinert, M., Birkness, K., White, E., Fields, B., and Quinn, F. (1998) *Mycobacterium avium* bacilli grow saprozoically in coculture with *Acanthamoeba polyphaga* and survive within cyst walls. Appl. Environ. Microbiol., 64(6): 2256–2261.

Stout, J.E., Yu, V.L., Muraca, P., Joly, J., Troup, N., and Tompkins, L. (1992a) Potable water as a cause of sporadic cases of community-acquired Legionnaires' disease. N. Engl. J. Med., 326: 151–155.

Stout, J.E., Yu, V.L., Yee, Y.C., Vaccarello, S., Diven, W., and Lee, T.C. (1992b) *Legionella pneumophila* in residential water supplies: environmental surveillance with clinical assessment for Legionnaires' disease. Epidemiol. Infect., 109: 49–57.

Straus, W.L., Plouffe, J.F., File, T.M., Lipman, H.B., Hackman, B.H., Salstrom, S., Benson, R.F., and Breiman, R.F. (1996) Risk factors for domestic acquisition of Legionnaires disease. Arch. Intern. Med., 156: 1685–1692.

Swerdlow, D.L., Woodruff, B.A., Brady, R.C., Griffin, P.M., Tippen, S., Donnell, H.D., Jr., Geldreich, E., Payne, B.J., Meyer, A., Jr., Wells, J.G., Greene, K.D., Bright, M., Bean, N.H., and Blake, P.A. (1992) A waterborne outbreak in Missouri of *Escherichia coli* O157:H7 associated with bloody diarrhea and death. Ann. Intern. Med., 17(10): 812–819.

Ta, A.C., Stout, J.E., Yu, V.L., and Wagener, M.M. (1995) Comparison of culture methods for monitoring *Legionella* species in hospital potable water systems and recommendations for standardization of such methods. J. Clin. Microbiol., 33: 2118–2123.

Taylor, D.N., McDermott, K.T., Little, J.R., Wells, J.G., and Blaser, M.J. (1983) *Campylobacter* enteritis from untreated water in the Rocky Mountains. Ann. Intern. Med., 99: 38–40.

Taylor, R., Sloan, D., Cooper, T., Morton, B., and Hunter, I. (2000) A waterborne outbreak of *Salmonella saintpaul*. Commun. Dis. Intell., 24: 336–340.

Taylor, R.H., Falkinham, J.O., III, Norton, C.D., and LeChevallier, M.W. (2000) Chlorine, chloramine, chlorine dioxide, and ozone susceptibility of *Mycobacterium avium*. Appl. Environ. Microbiol., 66(4): 1702–1705.

Thomas, J.E., Gibson, G.R., Darboe, M.K., Dale, A., and Weaver, L.T. (1992) Isolation of *Helicobacter pylori* from human faeces. Lancet, 340: 1194–1195.

Thompson, J.S. and Gravel, M.J. (1986) Family outbreak of gastroenteritis due to *Yersinia enterocolitica* serotype 0:3 from well water. Can. J. Microbiol., 32: 700–701.

Tiefenbrunner, F., Arnold, A., Dierich, M.P., and Emde, K. (1993) Occurrence and distribution of *Legionella pneumophila* in water systems of central European private homes. In: *Legionella*: Current status and emerging perspectives. J.M. Barbaree, R.F. Breiman, and A.P. Dufour (eds.). American Society for Microbiology, Washington, DC. pp. 235–238.

Tobin, R.S., Ewan, P., Walsh, K., and Dutka, B. (1986) A survey of *Legionella pneumophila* in water in 12 Canadian cities. Water Res., 20: 495–501.

U.S. EPA (United States Environmental Protection Agency) (2000) *Aeromonas* criteria document. Prepared by A. Highsmith and C. Bouma, GRAM Inc., Albuquerque, NM, for the Office of Science and Technology, Washington, DC.

U.S. EPA (United States Environmental Protection Agency) (2001) *Legionella*: Drinking water health advisory. Office of Water, Washington, DC (EPA-822-B-01-005).

Vaira, D., Miglioli, M., Menegatti, M., Holton, J., Boschini, A., Verguar, A., Ricci, C., Azzarone, P., Mule, P., and Barbara, L. (1995) *Helicobacter pylori* status, endoscopic findings and serology in HIV-1-positive patients. Dig. Dis. Sci., 40: 1622–1626.

van der Kooij, D. (1993) Importance and assessment of the biological stability of drinking water in the Netherlands. In: Safety of water disinfection: Balancing the chemical and microbial risks. C. Craun (ed.). ILSI Press, Washington, DC. pp. 165–179.

van der Kooij, D., Veenendaal, H.R., Slaats, N.P.G., and Vonk, D. (2002) Biofilm formation and multiplication of *Legionella* on synthetic pipe materials in contact with treated water under static and dynamic conditions. In: *Legionella*. R. Marre, Y.A. Kwaik, C. Bartlett, N.P. Cianciotto, B.S. Fields, M. Frosch, J. Hacker, and P.C. Luck (eds.). ASM Press, Washington, DC. pp. 176–180.

van Duynhoven, Y.T.H.P. and de Jonge, R. (2001) Transmission of *Helicobacter pylori*: a role for food? Bull. World Health Organ., 79(5): 455–460.

Villari, P., Crispino, M., Montuori, P., and Boccia, S. (2003) Molecular typing of *Aeromonas* isolates in natural mineral waters. Appl. Environ. Microbiol., 69: 697–701.

Vogt, R.L., Sours, H.E., Barrett, T., Feldman, R.A., Dickson, R.J., and Witherell, L. (1982) *Campylobacter* enteritis associated with contaminated water. Ann. Intern. Med., 96: 292–296.

von Reyn, C.F., Barber, T.W., Arbeit, R.D., Sox, C.H., O'Connor, G.T., Brindle, R.J., Gilks, C.F., Hakkarainen, I.C., Ranki, A., Bartholomew, C., Edward, J., Tosteson, A.N.A., and Magnusson, M. (1993a) Evidence of previous infection with *Mycobacterium avium – Mycobacterium intracellulare* complex among healthy subjects: an international study of dominant mycobacterial skin test reactions. J. Infect. Dis., 168: 1553–1558.

von Reyn, C.F., Waddell, R.D., Eaton, T., Arbeit, R.D., Maslow, J.N., Barber, T.W., Brindle, R.J., Gilks, C.F., Lumio, J., Lahdevirta, J., Ranki, A., Dawson, D., and Falkinham, J.O., III (1993b) Isolation of *Mycobacterium avium* complex from water in the United States, Finland, Zaire, and Kenya. J. Clin. Microbiol., 31: 3227–3230.

von Reyn, C.F., Maslow, J.N., Barber, T.W., Falkinham, J.O., III, and Arbeit, R.D. (1994) Persistent colonisation of potable water as source of *Mycobacterium avium* infection in AIDS. Lancet, 343: 1137–1141.

Wang, W.L.L., Powers, B.W., Blaser, M.J., and Leuchtefeld, N.W. (1982) Laboratory studies of disinfectants against *Campylobacter jejuni*. In: Proceedings of the Annual Meeting of the American Society of Microbiology. American Society of Microbiology, Washington, DC.

Wang, X., Sturegard, E., Rupar, R., Nilsson, H.O., Alelijung, P.A., Carlén, B., Willen, R., and Wadstrom, T. (1995) Infection of BALB/cA mice by spiral and coccoid forms of *Helicobacter pylori*. J. Med. Microbiol., 46: 657–663.

Weagant, S.D. and Kaysner, C.A. (1983) Modified enrichment broth for isolation of *Yersinia enterocolitica* from non food sources. Appl. Environ. Microbiol., 45: 468–471.

Webb, P.M., Knight, T., Elder, J.B., Newell, D.G., and Forman, D. (1996a) *Helicobacter pylori* transmission: evidence from a comparison with hepatitis A virus. Eur. J. Gastroenterol. Hepatol., 8: 439–441.

Webb, P.M., Knight, T., Elder, J.B., Newell, D.G., and Forman, D. (1996b) Is *Helicobacter pylori* transmitted from cats to humans? Helicobacter, 1: 79–81.

Wendt, S.L., George, K.L., Parker, B.C., Gruft, H., and Falkinham, J.O., III (1980) Epidemiology of infection by nontuberculous mycobacteria: isolation of potentially pathogenic mycobacteria from aerosols. Am. Rev. Respir. Dis., 122: 259–263.

*Guidelines for Canadian Drinking Water Quality: Guideline Technical Document* 

West, A.P., Millar, M.R., and Tomkins, D.S. (1992) Effect of physical environment on survival of *Helicobacter pylori*. J. Clin. Pathol., 45: 228–231.

Wetzler, T.F., Rea, J.R., Ma, G.J., and Glass, M. (1979) Non-association of *Yersinia* with traditional coliform indicators. In: Proceedings of the Annual Meeting of the American Water Works Association. American Water Works Association, Denver, CO.

White, F.M. and Pedersen, A.T. (1976) Epidemic shigellosis on a worktrain in Labrador. Can. Med. Assoc. J., 115: 647–649.

WHO (World Health Organization) (1990) Epidemiology, prevention and control of legionellosis: memorandum from a WHO meeting. Bull. World Health Organ., 68: 155–164.

WHO (World Health Organization) (2002) Guidelines for drinking-water quality. 2nd edition. Addendum: Microbiological agents in drinking water. World Health Organization, Geneva.

Yu, V. (2002) *Legionella* surveillance: political and social implications — a little knowledge is a dangerous thing. J. Infect. Dis., 185: 259–261.

Yu-Sen, E., Lin, R.D.V., Stout, J.E., McCartney, C.A., and Yu, V.L. (1998) Inactivation of *Mycobacterium avium* by copper and silver ions. Water Res., 32: 1997–2000.

Zacheus, O.M. and Martikainen, P.J. (1996) Effect of heat flushing on the concentrations of *Legionella pneumophila* and other heterotrophic microbes in hot water systems of apartment buildings. Can. J. Microbiol., 42: 811–818.

# **Appendix A: List of acronyms**

AIDS	acquired immunodeficiency syndrome
CFU	colony-forming unit
СТ	product of disinfectant concentration and contact time
DNA	deoxyribonucleic acid
HPC	heterotrophic plate count
HUS	haemolytic uraemic syndrome
Mac	Mycobacterium avium complex
MAC	maximum acceptable concentration
NTM	non-tuberculous mycobacteria
U.S. EPA	United States Environmental Protection Agency
UV	ultraviolet
VBNC	viable but non-culturable