# 7
# Microbial aspects

The greatest risk from microbes in water is associated with consumption of drinking-water that is contaminated with human and animal excreta, although other sources and routes of exposure may also be significant.

This chapter focuses on organisms for which there is evidence, from outbreak studies or from prospective studies in non-outbreak situations, of disease being caused by ingestion of drinking-water, inhalation of droplets or contact with drinking-water; and their control.

## 7.1 Microbial hazards associated with drinking-water

Infectious diseases caused by pathogenic bacteria, viruses and parasites (e.g., protozoa and helminths) are the most common and widespread health risk associated with drinking-water. The public health burden is determined by the severity of the illness(es) associated with pathogens, their infectivity and the population exposed.

Breakdown in water supply safety may lead to large-scale contamination and potentially to detectable disease outbreaks. Other breakdowns and low-level, potentially repeated contamination may lead to significant sporadic disease, but is unlikely to be associated with the drinking-water source by public health surveillance.

Quantified risk assessment can assist in understanding and managing risks, especially those associated with sporadic disease.

### *7.1.1 Waterborne infections*

The pathogens that may be transmitted through contaminated drinking-water are diverse. Table 7.1 and Figure 7.1 provide general information on pathogens that are of relevance for drinking-water supply management. The spectrum changes in response to variables such as increases in human and animal populations, escalating use of wastewater, changes in lifestyles and medical interventions, population movement and travel and selective pressures for new pathogens and mutants or recombinations of existing pathogens. The immunity of individuals also varies considerably, whether acquired by contact with a pathogen or influenced by such factors as age, sex, state of health and living conditions.

**Table 7.1 Waterborne pathogens and their significance in water supplies**

| Pathogen | Health significance | Persistence in water supplies[a] | Resistance to chlorine[b] | Relative infectivity[c] | Important animal source |
|---|---|---|---|---|---|
| **Bacteria** | | | | | |
| *Burkholderia pseudomallei* | Low | May multiply | Low | Low | No |
| *Campylobacter jejuni, C. coli* | High | Moderate | Low | Moderate | Yes |
| *Escherichia coli* – Pathogenic[d] | High | Moderate | Low | Low | Yes |
| *E. coli* – Enterohaemorrhagic | High | Moderate | Low | High | Yes |
| *Legionella* spp. | High | Multiply | Low | Moderate | No |
| Non-tuberculous mycobacteria | Low | Multiply | High | Low | No |
| *Pseudomonas aeruginosa*[e] | Moderate | May multiply | Moderate | Low | No |
| *Salmonella typhi* | High | Moderate | Low | Low | No |
| Other salmonellae | High | May multiply | Low | Low | Yes |
| *Shigella* spp. | High | Short | Low | Moderate | No |
| *Vibrio cholerae* | High | Short | Low | Low | No |
| *Yersinia enterocolitica* | High | Long | Low | Low | Yes |
| **Viruses** | | | | | |
| Adenoviruses | High | Long | Moderate | High | No |
| Enteroviruses | High | Long | Moderate | High | No |
| Hepatitis A | High | Long | Moderate | High | No |
| Hepatitis E | High | Long | Moderate | High | Potentially |
| Noroviruses and Sapoviruses | High | Long | Moderate | High | Potentially |
| Rotavirus | High | Long | Moderate | High | No |
| **Protozoa** | | | | | No |
| *Acanthamoeba* spp. | High | Long | High | High | No |
| *Cryptosporidium parvum* | High | Long | High | High | Yes |
| *Cyclospora cayetanensis* | High | Long | High | High | No |
| *Entamoeba histolytica* | High | Moderate | High | High | No |
| *Giardia intestinalis* | High | Moderate | High | High | Yes |
| *Naegleria fowleri* | High | May multiply[f] | High | High | No |
| *Toxoplasma gondii* | High | Long | High | High | Yes |
| **Helminths** | | | | | |
| *Dracunculus medinensis* | High | Moderate | Moderate | High | No |
| *Schistosoma* spp. | High | Short | Moderate | High | Yes |

Note: Waterborne transmission of the pathogens listed has been confirmed by epidemiological studies and case histories. Part of the demonstration of pathogenicity involves reproducing the disease in suitable hosts. Experimental studies in which volunteers are exposed to known numbers of pathogens provide relative information. As most studies are done with healthy adult volunteers, such data are applicable to only a part of the exposed population, and extrapolation to more sensitive groups is an issue that remains to be studied in more detail.

[a] Detection period for infective stage in water at 20 °C: short, up to 1 week; moderate, 1 week to 1 month; long, over 1 month.

[b] When the infective stage is freely suspended in water treated at conventional doses and contact times. Resistance moderate, agent may not be completely destroyed.

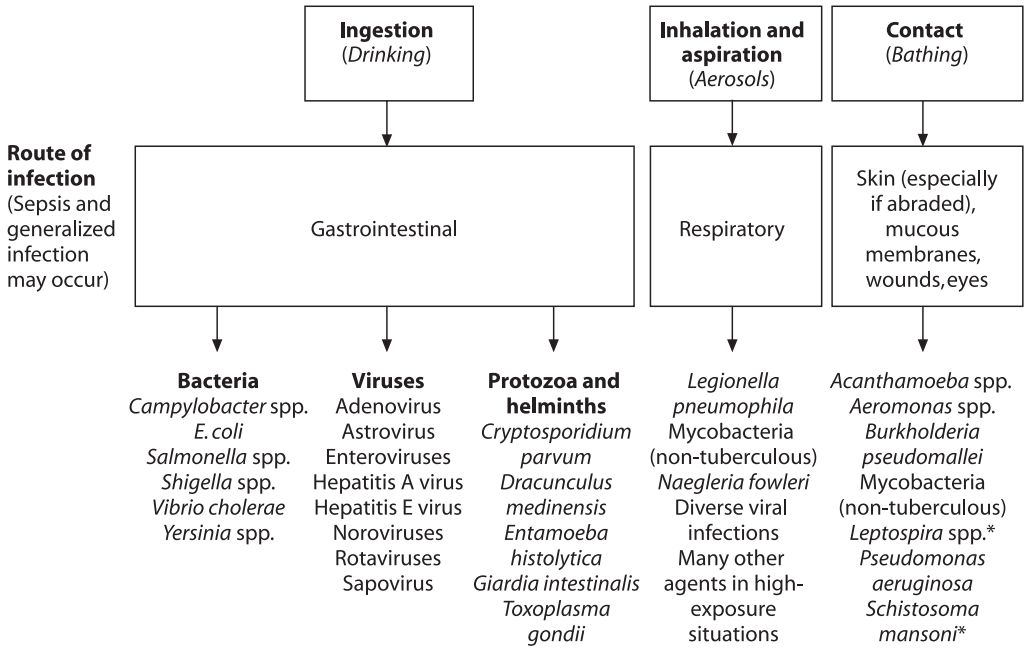
[c] From experiments with human volunteers or from epidemiological evidence.

[d] Includes enteropathogenic, enterotoxigenic and enteroinvasive.

[e] Main route of infection is by skin contact, but can infect immunosuppressed or cancer patients orally.

[f] In warm water.

For pathogens transmitted by the faecal–oral route, drinking-water is only one vehicle of transmission. Contamination of food, hands, utensils and clothing can also play a role, particularly when domestic sanitation and hygiene are poor. Improvements in the quality and availability of water, in excreta disposal and in general hygiene are all important in reducing faecal–oral disease transmission.




* Primarily from contact with highly contaminated surface waters.

**Figure 7.1 Transmission pathways for and examples of water-related pathogens**

Infectious diseases caused by pathogenic bacteria, viruses, protozoa and helminths are the most common and widespread health risk associated with drinking-water.

Drinking-water safety is not related only to faecal contamination. Some organisms grow in piped water distribution systems (e.g., *Legionella*), whereas others occur in source waters (guinea worm *Dracunculus medinensis*) and may cause outbreaks and individual cases. Some other microbes (e.g., toxic cyanobacteria) require specific management approaches, which are covered elsewhere in these Guidelines (see section 11.5).

Certain serious illnesses result from inhalation of water droplets (aerosols) in which the causative organisms have multiplied because of warm temperatures and the presence of nutrients. These include legionellosis and Legionnaires' disease, caused by *Legionella* spp., and those caused by the amoebae *Naegleria fowleri* (primary amoebic meningoencephalitis [PAM]) and *Acanthamoeba* spp. (amoebic meningitis, pulmonary infections).

Schistosomiasis (bilharziasis) is a major parasitic disease of tropical and subtropical regions that is transmitted when the larval stage (cercariae), which is released by infected aquatic snails, penetrates the skin. It is primarily spread by contact with water. Ready availability of safe drinking-water contributes to disease prevention by reducing the need for contact with contaminated water resources – for example, when collecting water to carry to the home or when using water for bathing or laundry.

It is conceivable that unsafe drinking-water contaminated with soil or faeces could act as a carrier of other parasitic infections, such as balantidiasis (*Balantidium coli*) and certain helminths (species of *Fasciola*, *Fasciolopsis*, *Echinococcus*, *Spirometra*, *Ascaris*, *Trichuris*, *Toxocara*, *Necator*, *Ancylostoma*, *Strongyloides* and *Taenia solium*). However, in most of these, the normal mode of transmission is ingestion of the eggs in food contaminated with faeces or faecally contaminated soil (in the case of *Taenia solium*, ingestion of the larval cysticercus stage in uncooked pork) rather than ingestion of contaminated drinking-water.

Other pathogens that may be naturally present in the environment may be able to cause disease in people with impaired local or general immune defence mechanisms, such as the elderly or the very young, patients with burns or extensive wounds, those undergoing immunosuppressive therapy or those with acquired immunodeficiency syndrome (AIDS). If water used by such persons for drinking or bathing contains sufficient numbers of these organisms, they can produce various infections of the skin and the mucous membranes of the eye, ear, nose and throat. Examples of such agents are *Pseudomonas aeruginosa* and species of *Flavobacterium*, *Acinetobacter*, *Klebsiella*, *Serratia*, *Aeromonas* and certain "slow-growing" (non-tuberculous) mycobacteria (see the supporting document *Pathogenic Mycobacteria in Water*; section 1.3).

Most of the human pathogens listed in Table 7.1 (which are described in more detail in chapter 11) are distributed worldwide; some, however, such as those causing outbreaks of cholera or guinea worm disease, are regional. Eradication of *D. medinensis* is a recognized target of the World Health Assembly (1991).

It is likely that there are pathogens not shown in Table 7.1 that are also transmitted by water. This is because the number of known pathogens for which water is a transmission route continues to increase as new or previously unrecognized pathogens continue to be discovered (see WHO, 2003a).

### *7.1.2 Persistence and growth in water*

While typical waterborne pathogens are able to persist in drinking-water, most do not grow or proliferate in water. Microorganisms like *E. coli* and *Campylobacter* can accumulate in sediments and are mobilized when water flow increases.

After leaving the body of their host, most pathogens gradually lose viability and the ability to infect. The rate of decay is usually exponential, and a pathogen will become undetectable after a certain period. Pathogens with low persistence must rapidly find new hosts and are more likely to be spread by person-to-person contact or poor personal hygiene than by drinking-water. Persistence is affected by several factors, of which temperature is the most important. Decay is usually faster at higher temperatures and may be mediated by the lethal effects of UV radiation in sunlight acting near the water surface.

The most common waterborne pathogens and parasites are those that have high infectivity and either can proliferate in water or possess high resistance to decay outside the body.

Viruses and the resting stages of parasites (cysts, oocysts, ova) are unable to multiply in water. Conversely, relatively high amounts of biodegradable organic carbon, together with warm temperatures and low residual concentrations of chlorine, can permit growth of *Legionella*, *V. cholerae*, *Naegleria fowleri*, *Acanthamoeba* and nuisance organisms in some surface waters and during water distribution (see also the supporting document *Heterotrophic Plate Counts and Drinking-water Safety*; section 1.3).

Microbial water quality may vary rapidly and widely. Short-term peaks in pathogen concentration may increase disease risks considerably and may also trigger outbreaks of waterborne disease. Results of water quality testing for microbes are not normally available in time to inform management action and prevent the supply of unsafe water.

### *7.1.3 Public health aspects*

Outbreaks of waterborne disease may affect large numbers of persons, and the first priority in developing and applying controls on drinking-water quality should be the control of such outbreaks. Available evidence also suggests that drinking-water can contribute to background rates of disease in non-outbreak situations, and control of drinking-water quality should therefore also address waterborne disease in the general community.

Experience has shown that systems for the detection of waterborne disease outbreaks are typically inefficient in countries at all levels of socioeconomic development, and failure to detect outbreaks is not a guarantee that they do not occur; nor does it suggest that drinking-water should necessarily be considered safe.

Some of the pathogens that are known to be transmitted through contaminated drinking-water lead to severe and sometimes life-threatening disease. Examples include typhoid, cholera, infectious hepatitis (caused by hepatitis A virus [HAV] or HEV) and disease caused by *Shigella* spp. and *E. coli* O157. Others are typically associated with less severe outcomes, such as self-limiting diarrhoeal disease (e.g., Norovirus, *Cryptosporidium*).

The effects of exposure to pathogens are not the same for all individuals or, as a consequence, for all populations. Repeated exposure to a pathogen may be associated with a lower probability or severity of illness because of the effects of acquired immunity. For some pathogens (e.g., HAV), immunity is lifelong, whereas for others (e.g., *Campylobacter*), the protective effects may be restricted to a few months to years. On the other hand, sensitive subgroups (e.g., the young, the elderly, pregnant women and the immunocompromised) in the population may have a greater probability of illness or the illness may be more severe, including mortality. Not all pathogens have greater effects in all sensitive subgroups.

Not all infected individuals will develop symptomatic disease. The proportion of the infected population that is asymptomatic (including carriers) differs between pathogens and also depends on population characteristics, such as prevalence of

immunity. Carriers and those with asymptomatic infections as well as individuals developing symptoms may all contribute to secondary spread of pathogens.

## 7.2 Health-based target setting

### *7.2.1 Health-based targets applied to microbial hazards*

General approaches to health-based target setting are described in section 2.1.1 and chapter 3.

Sources of information on health risks may be from both epidemiology and risk assessment, and typically both are employed as complementary sources.

Health-based targets may also be set using a health outcome approach, where the waterborne disease burden is believed to be sufficiently high to allow measurement of the impact of interventions – i.e., to measure reductions in disease that can be attributed to drinking-water.

Risk assessment is especially valuable where the fraction of disease that can be attributed to drinking-water is low or difficult to measure directly through public health surveillance or analytical epidemiological studies.

Data – from both epidemiology and risk assessment – with which to develop health-based targets for many pathogens are limited, but are increasingly being produced. Locally generated data will always be of great value in setting national targets.

For the control of microbial hazards, the most frequent form of health-based target applied is performance targets (see section 3.2.2), which are anchored to a tolerable burden of disease. WQTs (see section 3.2.3) are typically not developed for pathogens, because monitoring finished water for pathogens is not considered a feasible or cost-effective option.

### *7.2.2 Risk assessment approach*

In many circumstances, estimating the effects of improved drinking-water quality on health risks in the population is possible through constructing and applying risk assessment models.

QMRA is a rapidly evolving field that systematically combines available information on exposure and dose–response to produce estimates of the disease burden associated with exposure to pathogens. Mathematical modelling is used to estimate the effects of low doses of pathogens in drinking-water on populations and subpopulations.

Interpreting and applying information from analytical epidemiological studies to derive health-based targets for application at a national or local level require consideration of a number of factors, including the following:

- Are specific estimates of disease reduction or indicative ranges of expected reductions to be provided?
- How representative of the target population was the study sample in order to ensure confidence in the reliability of the results across a wider group?

- To what extent will minor differences in demographic or socioeconomic conditions affect expected outcomes?

Risk assessment commences with problem formulation to identify all possible hazards and their pathways from source(s) to recipient(s). Human exposure to the pathogens (environmental concentrations and volumes ingested) and dose–responses of these selected organisms are then combined to characterize the risks. With the use of additional information (social, cultural, political, economic, environmental, etc.), management options can be prioritized. To encourage stakeholder support and participation, a transparent procedure and active risk communication at each stage of the process are important. An example of a risk assessment approach is described in Table 7.2 and outlined below.

### Problem formulation and hazard identification

All potential hazards, sources and events that can lead to the presence of these hazards (i.e., what can happen and how) should be identified and documented for each component of the drinking-water system, regardless of whether or not the component is under the direct control of the drinking-water supplier. This includes point sources of pollution (e.g., human and industrial waste discharge) as well as diffuse sources (e.g., those arising from agricultural and animal husbandry activities). Continuous, intermittent or seasonal pollution patterns should also be considered, as well as extreme and infrequent events, such as droughts and floods.

The broader sense of hazards focuses on hazardous scenarios, which are events that may lead to exposure of consumers to specific pathogenic microorganisms. In this, the hazardous event (e.g., peak contamination of source water with domestic wastewater) may be referred to as the hazard.

Representative organisms are selected that, if controlled, would ensure control of all pathogens of concern. Typically, this implies inclusion of at least one bacterial pathogen, virus and protozoan.

**Table 7.2 Risk assessment paradigm for pathogen health risks**

| Step | Aim |
|---|---|
| 1. Problem formulation and hazard identification | To identify all possible hazards associated with drinking-water that would have an adverse public health consequence, as well as their pathways from source(s) to consumer(s) |
| 2. Exposure assessment | To determine the size and nature of the population exposed and the route, amount and duration of the exposure |
| 3. Dose–response assessment | To characterize the relationship between exposure and the incidence of the health effect |
| 4. Risk characterization | To integrate the information from exposure, dose–response and health interventions in order to estimate the magnitude of the public health problem and to evaluate variability and uncertainty |

Source: Adapted from Haas *et al.* (1999).

### Exposure assessment

Exposure assessment involves estimation of the number of pathogenic microbes to which an individual is exposed, principally through ingestion. Exposure assessment is a predictive activity that often involves subjective judgement. It inevitably contains uncertainty and must account for variability of factors such as concentrations of microorganisms over time, volumes ingested, etc.

Exposure can be considered as a single dose of pathogens that a consumer ingests at a certain point of time or the total amount over several exposures (e.g., over a year). Exposure is determined by the concentration of microbes in drinking-water and the volume of water consumed.

It is rarely possible or appropriate to directly measure pathogens in drinking-water on a regular basis. More often, concentrations in source waters are assumed or measured, and estimated reductions – for example, through treatment – are applied to estimate the concentration in the water consumed. Pathogen measurement, when performed, is generally best carried out at the location where the pathogens are at highest concentration (generally source waters). Estimation of their removal by sequential control measures is generally achieved by the use of surrogates (such as *E. coli* for enteric bacterial pathogens).

The other component of exposure assessment, which is common to all pathogens, is the volume of unboiled water consumed by the population, including person-to-person variation in consumption behaviour and especially consumption behaviour of at-risk groups. For microbial hazards, it is important that the unboiled volume of drinking-water, both consumed directly and used in food preparation, is used in the risk assessment, as heating will rapidly inactivate pathogens. This amount is lower than that used for deriving chemical guideline values and WQTs.

The daily exposure of a consumer can be assessed by multiplying the concentration of pathogens in drinking-water by the volume of drinking-water consumed. For the purposes of the Guidelines, unboiled drinking-water consumption is assumed to be 1 litre of water per day.

### Dose–response assessment

The probability of an adverse health effect following exposure to one or more pathogenic organisms is derived from a dose–response model. Available dose–response data have been obtained mainly from studies using healthy adult volunteers. Several subgroups in the population, such as children, the elderly and immunocompromised persons, are more sensitive to infectious disease; currently, however, adequate data are lacking to account for this.

The conceptual basis for the infection model is the observation that exposure to the described dose leads to the probability of infection as a conditional event. For infection to occur, one or more viable pathogens must have been ingested. Furthermore, one or more of these ingested pathogens must have survived in the host's body. An important concept is the single-hit principle (i.e., that even a single organism may

be able to cause infection and disease, possibly with a low probability). This concept supersedes the concept of (minimum) infectious dose that is frequently used in older literature (see the supporting document *Hazard Characterization for Pathogens in Food and Water*; section 1.3).

In general, well dispersed pathogens in water are considered to be Poisson distributed. When the individual probability of any organism to survive and start infection is the same, the dose–response relation simplifies to an exponential function. If, however, there is heterogeneity in this individual probability, this leads to the beta-Poisson dose–response relation, where the "beta" stands for the distribution of the individual probabilities among pathogens (and hosts). At low exposures, such as would typically occur in drinking-water, the dose–response model is approximately linear and can be represented simply as the probability of infection resulting from exposure to a single organism (see the supporting document *Hazard Characterization for Pathogens in Food and Water*; section 1.3).

### Risk characterization

Risk characterization brings together the data collected on pathogen exposure, dose–response, severity and disease burden.

The probability of infection can be estimated as the product of the exposure by drinking-water and the probability that exposure to one organism would result in infection. The probability of infection per day is multiplied by 365 to calculate the probability of infection per year. In doing so, it is assumed that different exposure events are independent, in that no protective immunity is built up. This simplification is justified for low risks only.

Not all infected individuals will develop clinical illness; asymptomatic infection is common for most pathogens. The percentage of infected persons that will develop clinical illness depends on the pathogen, but also on other factors, such as the immune status of the host. Risk of illness per year is obtained by multiplying the probability of infection by the probability of illness given infection.

The low numbers in Table 7.3 can be interpreted to represent the probability that a single individual will develop illness in a given year. For example, a risk of illness for *Campylobacter* of $2.5 \times 10^{-4}$ per year indicates that, on average, 1 out of 4000 consumers would contract campylobacteriosis from drinking-water.

To translate the risk of developing a specific illness to disease burden per case, the metric DALYs is used. This should reflect not only the effects of acute end-points (e.g., diarrhoeal illness) but also mortality and the effects of more serious end-points (e.g., Guillain-Barré syndrome associated with *Campylobacter*). Disease burden per case varies widely. For example, the disease burden per 1000 cases of rotavirus diarrhoea is 480 DALYs in low-income regions, where child mortality frequently occurs. However, it is only 14 DALYs per 1000 cases in high-income regions, where hospital facilities are accessible to the great majority of the population (see the supporting document *Quantifying Public Health Risk in the WHO Guidelines for Drinking-water*

**Table 7.3 Linking tolerable disease burden and source water quality for reference pathogens: example calculation**

| **River water (human and animal pollution)** | | ***Cryptosporidium*** | ***Campylobacter*** | **Rotavirus**[a] |
|---|---|---|---|---|
| Raw water quality ($C_R$) | Organisms per litre | 10 | 100 | 10 |
| *Treatment effect needed to reach tolerable risk (PT)* | *Percent reduction* | *99.994%* | *99.99987%* | *99.99968%* |
| Drinking-water quality ($C_D$) | Organisms per litre | $6.3 \times 10^{-4}$ | $1.3 \times 10^{-4}$ | $3.2 \times 10^{-5}$ |
| Consumption of unheated drinking-water (V) | Litres per day | 1 | 1 | 1 |
| Exposure by drinking-water (E) | Organisms per day | $6.3 \times 10^{-4}$ | $1.3 \times 10^{-4}$ | $3.2 \times 10^{-5}$ |
| Dose–response (r) | Probability of infection per organism | $4.0 \times 10^{-3}$ | $1.8 \times 10^{-2}$ | $2.7 \times 10^{-1}$ |
| Risk of infection ($P_{inf,d}$) | Per day | $2.5 \times 10^{-6}$ | $2.3 \times 10^{-6}$ | $8.5 \times 10^{-6}$ |
| Risk of infection ($P_{inf,y}$) | Per year | $9.2 \times 10^{-4}$ | $8.3 \times 10^{-4}$ | $3.1 \times 10^{-3}$ |
| Risk of (diarrhoeal) illness given infection ($P_{ill\|inf}$) | | 0.7 | 0.3 | 0.5 |
| Risk of (diarrhoeal) illness ($P_{ill}$) | Per year | $6.4 \times 10^{-4}$ | $2.5 \times 10^{-4}$ | $1.6 \times 10^{-3}$ |
| Disease burden (db) | DALYs per case | $1.5 \times 10^{-3}$ | $4.6 \times 10^{-3}$ | $1.4 \times 10^{-2}$ |
| Susceptible fraction ($f_s$) | Percentage of population | 100% | 100% | 6% |
| Disease burden (DB) | DALYs per year | $1 \times 10^{-6}$ | $1 \times 10^{-6}$ | $1 \times 10^{-6}$ |
| ***Formulas:*** | $C_D = C_R \times (1 - PT)$<br>$E = C_D \times V$<br>$P_{inf,d} = E \times r$ | | | |

[a] Data from high-income regions. In low-income regions, severity is typically higher, but drinking-water transmission is unlikely to dominate.

*Quality*; section 1.3). This considerable difference in disease burden results in far stricter treatment requirements in low-income regions for the same source water quality in order to obtain the same risk (expressed as DALYs per year). Ideally, the default disease burden estimates in Table 7.3 should be adapted to specific national situations. In Table 7.3, no accounting is made for effects on immunocompromised persons (e.g., cryptosporidiosis in HIV/AIDS patients), which is significant in some countries. Section 3.3.3 gives more information on the DALY metric and how it is applied to derive a reference level of risk.

Only a proportion of the population may be susceptible to some pathogens, because immunity developed after an initial episode of infection or illness may provide lifelong protection. Examples include HAV and rotaviruses. It is estimated that in developing countries, all children above the age of 5 years are immune to rotaviruses because of repeated exposure in the first years of life. This translates to an

average of 17% of the population being susceptible to rotavirus illness. In developed countries, rotavirus infection is also common in the first years of life, and the illness is diagnosed mainly in young children, but the percentage of young children as part of the total population is lower. This translates to an average of 6% of the population in developed countries being susceptible.

The uncertainty of the risk estimate is the result of the uncertainty and variability of the data collected in the various steps of the risk assessment. Risk assessment models should ideally account for this variability and uncertainty, although here we present only point estimates (see below).

It is important to choose the most appropriate point estimate for each of the variables. Theoretical considerations show that risks are directly proportional to the arithmetic mean of the ingested dose. Hence, arithmetic means of variables such as concentration in raw water, removal by treatment and consumption of drinking-water are recommended. This recommendation is different from the usual practice among microbiologists and engineers of converting concentrations and treatment effects to log-values and making calculations or specifications on the log-scale. Such calculations result in estimates of the geometric mean rather than the arithmetic mean, and these may significantly underestimate risk. Analysing site-specific data may therefore require going back to the raw data rather than relying on reported log-transformed values.

### *7.2.3 Risk-based performance target setting*

The process outlined above enables estimation of risk on a population level, taking account of source water quality and impact of control. This can be compared with the reference level of risk (see section 3.3.2) or a locally developed tolerable risk. The calculations enable quantification of the degree of source protection or treatment that is needed to achieve a specified level of acceptable risk and analysis of the estimated impact of changes in control measures.

Performance targets are most frequently applied to treatment performance – i.e., to determine the microbial reduction necessary to ensure water safety. A performance target may be applied to a specific system (i.e., allow account to be taken of specific source water characteristics) or generalized (e.g., impose source water quality assumptions on all systems of a certain type or abstracting water from a certain type of source).

Figure 7.2 illustrates the targets for treatment performance for a range of pathogens occurring in the raw water. For example, 10 microorganisms per litre of source water will lead to a performance target of 4.2 logs (or 99.994%) for *Cryptosporidium* or of 5.5 logs (99.99968%) for rotavirus in high-income regions (see also Table 7.4 below). The difference in performance targets for rotavirus in high- and low-income countries (5.5 and 7.6 logs; Figure 7.2) is related to the difference in disease severity by this organism. In low-income countries, the child case fatality rate is relatively high, and, as a consequence, the disease burden is higher. Also, a larger proportion of the

**Figure 7.2 Performance targets for selected bacterial, viral and protozoan pathogens in relation to raw water quality (to achieve $10^{-6}$ DALYs per person per year)**

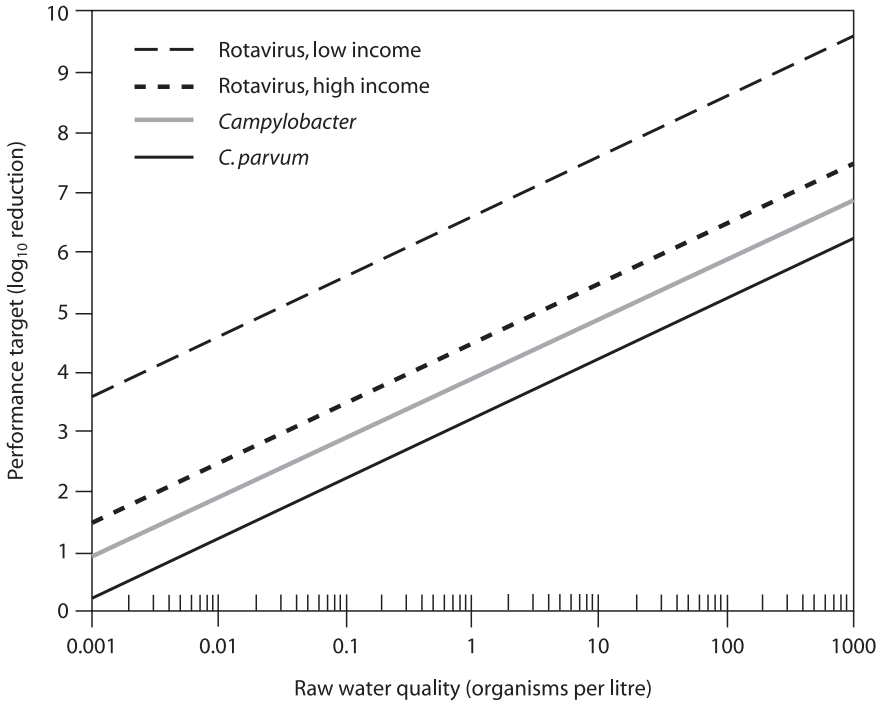



**Table 7.4 Health-based targets derived from example calculation in Table 7.3**

| | Cryptosporidium | Campylobacter | Rotavirus[a] |
|---|---|---|---|
| Organisms per litre in source water | 10 | 100 | 10 |
| Health outcome target | $10^{-6}$ DALYs per person per year | $10^{-6}$ DALYs per person per year | $10^{-6}$ DALYs per person per year |
| Risk of diarrhoeal illness[b] | 1 per 1600 per year | 1 per 4000 per year | 1 per 11 000 per year |
| Drinking-water quality | 1 per 1600 litres | 1 per 8000 litres | 1 per 32 000 litres |
| Performance target[c] | 4.2 $\log_{10}$ units | 5.9 $\log_{10}$ units | 5.5 $\log_{10}$ units |

[a] Data from high-income regions. In low-income regions, severity is typically higher, but drinking-water transmission is unlikely to dominate.
[b] For the susceptible population.
[c] Performance target is a measure of log reduction of pathogens based on source water quality.

population in low-income countries is under the age of 5 and at risk for rotavirus infection.

The derivation of these performance targets is described in Table 7.4, which provides an example of the data and calculations that would normally be used to construct a risk assessment model for waterborne pathogens. The table presents data for representatives of the three major groups of pathogens (bacteria, viruses and protozoa) from a range of sources. These example calculations aim at achieving the reference level of risk of $10^{-6}$ DALYs per person per year, as described in section 3.3.3. The

data in the table illustrate the calculations needed to arrive at a risk estimate and are not guideline values.

### 7.2.4 Presenting the outcome of performance target development

Table 7.4 presents some data from Table 7.3 in a format that is more meaningful to risk managers. The average concentration of pathogens in drinking-water is included for information. It is not a WQT, nor is it intended to encourage pathogen monitoring in finished water. As an example, a concentration of $6.3 \times 10^{-4}$ *Cryptosporidium* per litre (see Table 7.3) corresponds to 1 oocyst per 1600 litres (see Table 7.4). The performance target (in the row "Treatment effect" in Table 7.3), expressed as a percent reduction, is the most important management information in the risk assessment table. It can also be expressed as a log-reduction value. For example, 99.99968% reduction for rotavirus corresponds to $5.5 \log_{10}$ units.

### 7.2.5 Issues in adapting risk-based performance target setting to national/local circumstances

The choice of pathogens in Table 7.4 was based mainly on availability of data on resistance to water treatment, infectivity and disease burden. The pathogens illustrated may not be priority pathogens in all regions of the world, although amending pathogen selection would normally have a small impact on the overall conclusions derived from applying the model.

Wherever possible, country- or site-specific information should be used in assessments of this type. If no specific data are available, an approximate risk estimate can be based on default values (see Table 7.5 below).

Table 7.4 accounts only for changes in water quality derived from treatment and not source protection measures, which are often important contributors to overall safety, impacting on pathogen concentration and/or variability. The risk estimates presented in Table 7.3 also assume that there is no degradation of water quality in the distribution network. These may not be realistic assumptions under all circumstances, and it is advisable to take these factors into account wherever possible.

Table 7.4 presents point estimates only and does not account for variability and uncertainty. Full risk assessment models would incorporate such factors by representing the input variables by statistical distributions rather than by point estimates. However, such models are currently beyond the means of many countries, and data to define such distributions are scarce. Producing such data may involve considerable efforts in terms of time and resources, but will lead to much improved insight into the actual source water quality and treatment performance.

The necessary degree of treatment also depends on the values assumed for variables (e.g., drinking-water consumption, fraction of the population that is susceptible) that can be taken into account in the risk assessment model. Figure 7.3 shows the effect of variation in the consumption of unboiled drinking-water on the performance targets for *Cryptosporidium parvum*. For example, if the raw water concentration

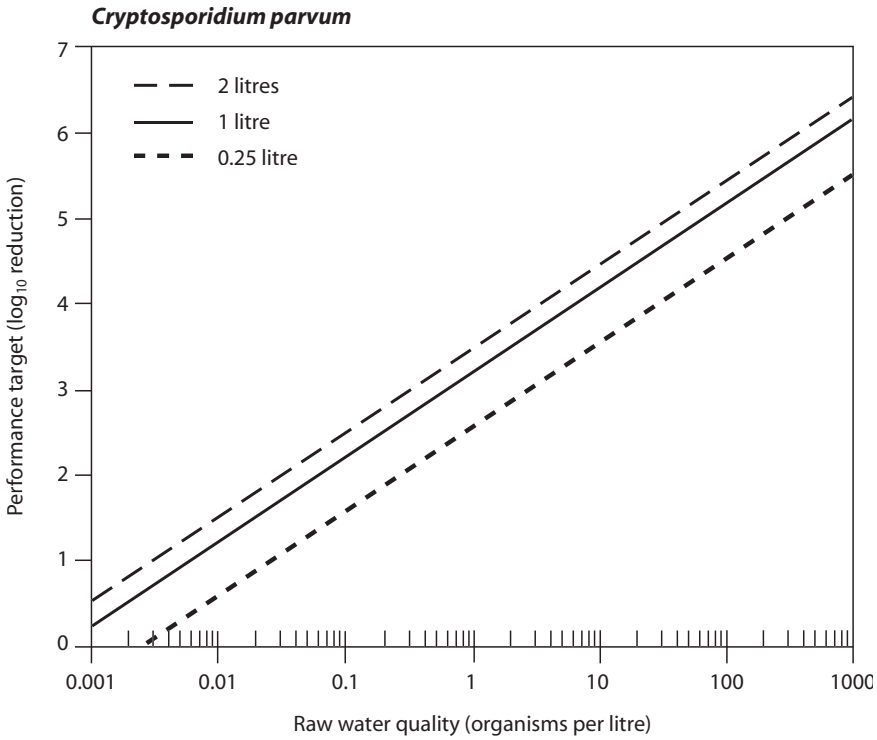



**Figure 7.3 Performance targets for *Cryptosporidium parvum* in relation to the daily consumption of unboiled drinking-water (to achieve $10^{-6}$ DALYs per person per year)**

is 1 oocyst per litre, the performance target varies between 2.6 and 3.5 $\log_{10}$ units if consumption values vary between 0.25 and 2 litres per day. Some outbreak data suggest that in developed countries, a significant proportion of the population above 5 years of age may not be immune to rotavirus illness. Figure 7.4 shows the effect of variation in the susceptible fraction of the population. For example, if the raw water concentration is 10 virus particles per litre, the performance target increases from 5.5 to 6.7 if the susceptible fraction increases from 6 to 100%.

### *7.2.6 Health outcome targets*

Health outcome targets that identify disease reductions in a community may be applied to the WSPs developed for specified water quality interventions at community and household levels. These targets would identify expected disease reductions in communities receiving the interventions.

The prioritization of water quality interventions should focus on those aspects that are estimated to contribute more than e.g. 5% of the burden of a given disease (e.g., 5% of total diarrhoea). In many parts of the world, the implementation of a water quality intervention that results in an estimated health gain of more than 5% would

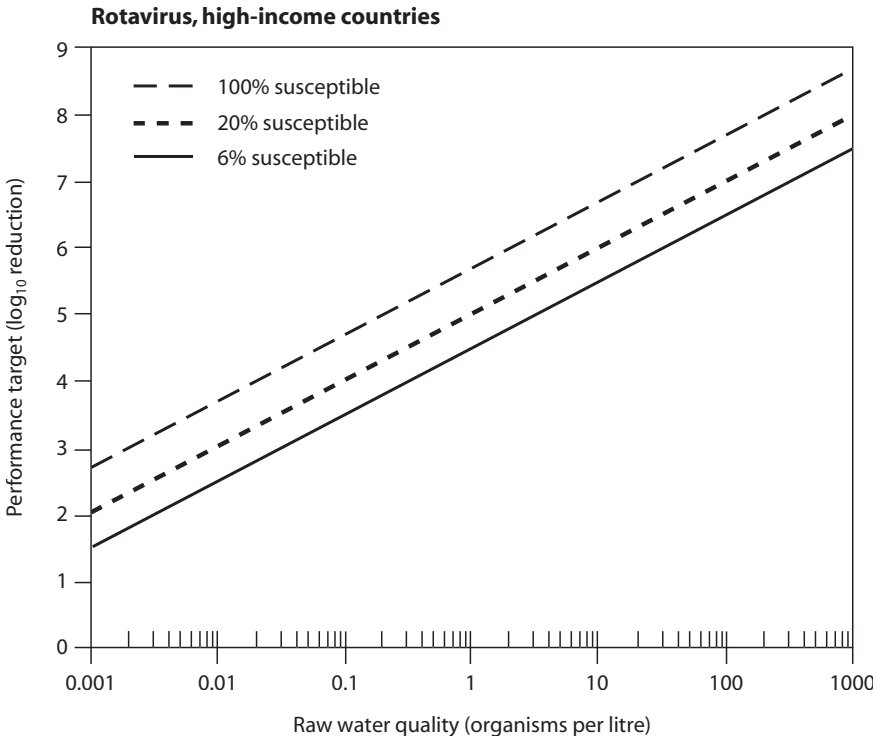



**Figure 7.4 Performance targets for rotavirus in relation to the fraction of the population that is susceptible to illness (to achieve $10^{-6}$ DALYs per person per year)**

be considered extremely worthwhile. Directly demonstrating the health gains arising from improving water quality – as assessed, for example, by reduced *E. coli* counts at the point of consumption – may be possible where disease burden is high and effective interventions are applied and can be a powerful tool to demonstrate a first step in incremental water safety improvement.

Where a specified quantified disease reduction is identified as a health outcome target, it may be advisable to undertake ongoing proactive public health surveillance among representative communities rather than through passive surveillance.

## 7.3 Occurrence and treatment of pathogens

As discussed in section 4.1, system assessment involves determining whether the drinking-water supply chain as a whole can deliver drinking-water quality that meets identified targets. This requires an understanding of the quality of source water and the efficacy of control measures.

An understanding of pathogen occurrence in source waters is essential, because it facilitates selection of the highest-quality source for drinking-water supply, determines pathogen loads and concentrations in source waters and provides a basis for establishing treatment requirements to meet health-based targets within a WSP.

Understanding the efficacy of control measures includes validation (see sections 2.1.2 and 4.1.7). Validation is important both in ensuring that treatment will achieve the desired goals (performance targets) and in assessing areas in which efficacy may be improved (e.g., by comparing performance achieved with that shown to be achievable through well run processes).

### *7.3.1 Occurrence*

The occurrence of pathogens and indicator organisms in groundwater and surface water sources depends on a number of factors, including intrinsic physical and chemical characteristics of the catchment area and the magnitude and range of human activities and animal sources that release pathogens to the environment.

In surface waters, potential pathogen sources include point sources, such as municipal sewerage and urban stormwater overflows, as well as non-point sources, such as contaminated runoff from agricultural areas and areas with sanitation through on-site septic systems and latrines. Other sources are wildlife and direct access of livestock to surface water bodies. Many pathogens in surface water bodies will reduce in concentration due to dilution, settling and die-off due to environmental effects (thermal, sunlight, predation, etc.).

Groundwater is often less vulnerable to the immediate influence of contamination sources due to the barrier effects provided by the overlying soil and its unsaturated zone. Groundwater contamination is more frequent where these protective barriers are breached, allowing direct contamination. This may occur through contaminated or abandoned wells or underground pollution sources, such as latrines and sewer lines. However, a number of studies have demonstrated pathogens and indicator organisms in groundwater, even at depth in the absence of such hazardous circumstances, especially where surface contamination is intense, as with land application of manures or other faecal impacts from intensive animal husbandry (e.g., feedlots). Impacts of these contamination sources can be greatly reduced by, for example, aquifer protection measures and proper well design and construction.

For more detailed discussion on both pathogen sources and key factors determining their fate, refer to the supporting documents *Protecting Surface Waters for Health* and *Protecting Groundwaters for Health* (section 1.3).

Table 7.5 presents estimates of high concentrations of enteric pathogens and microbial indicators in different types of surface waters and groundwaters, derived primarily from a review of published data. High values have been presented because they represent higher-risk situations and, therefore, greater degrees of vulnerability. The table includes two categories of data for rivers and streams: one for impacted sources and one for less impacted sources. More detailed information about these data is published in a variety of references, including several papers cited in Dangendorf et al. (2003).

The data in Table 7.5 provide a useful guide to the concentrations of enteric pathogens and indicator microorganisms in a variety of sources. However, there are a number of limitations and sources of uncertainty in these data, including:

**Table 7.5 Examples of high detectable concentrations (per litre) of enteric pathogens and faecal indicators in different types of source waters from the scientific literature**

| Pathogen or indicator group | Lakes and reservoirs | Impacted rivers and streams | Wilderness rivers and streams | Groundwater |
|---|---|---|---|---|
| *Campylobacter* | 20–500 | 90–2500 | 0–1100 | 0–10 |
| *Salmonella* | — | 3–58 000 (3–1000)[a] | 1–4 | — |
| *E. coli* (generic) | 10 000–1 000 000 | 30 000–1 000 000 | 6000–30 000 | 0–1000 |
| Viruses | 1–10 | 30–60 | 0–3 | 0–2 |
| *Cryptosporidium* | 4–290 | 2–480 | 2–240 | 0–1 |
| *Giardia* | 2–30 | 1–470 | 1–2 | 0–1 |

[a] Lower range is a more recent measurement.

— the lack of knowledge on sampling locations in relation to pollution sources;
— concerns about the sensitivity of analytical techniques, particularly for viruses and protozoa; and
— the lack of knowledge about the viability and human infectivity of *Cryptosporidium* oocysts, *Giardia* cysts and viruses detected in the different studies, because the various methods used are based upon non-culture methods (e.g., microscopy or molecular/nucleic acid analysis).

While the table provides an indication of concentrations that might be present in water sources, by far the most accurate way of determining pathogen loads and concentrations in specific catchments and other water sources is by analysing water quality over a period of time, taking care to include consideration of seasonal variation and peak events such as storms. Direct measurement of pathogens and indicators in the specific source waters for which a WSP and its target pathogens are being established is recommended wherever possible, because this provides the best estimates of microbial concentrations and loads.

### *7.3.2 Treatment*

Waters of very high quality – for example, groundwater from confined aquifers – may rely on source water and distribution system protection as the principal control measures for provision of safe water. More typically, water treatment is required to remove or destroy pathogenic microorganisms. In many cases (e.g., poor-quality surface water), multiple treatment stages are required, including, for example, coagulation, flocculation, sedimentation, filtration and disinfection. Table 7.6 provides a summary of treatment processes that are commonly used individually or in combination to achieve microbial reductions.

The microbial reductions presented in Table 7.6 are for broad groups or categories of microbes: bacteria, viruses and protozoa. This is because it is generally the case that treatment efficacy for microbial reduction differs among these microbial groups due to the inherently different properties of the microbes (e.g., size, nature of protective outer layers, physicochemical surface properties, etc.). Within these microbial groups,

**Table 7.6 Reductions of bacteria, viruses and protozoa achieved by typical and enhanced water treatment processes**

| Treatment process | Enteric pathogen group | Baseline removal | Maximum removal possible |
|---|---|---|---|
| **Pretreatment** | | | |
| Roughing filters | Bacteria | 50% | Up to 95% if protected from turbidity spikes by dynamic filter or if used only when ripened |
| | Viruses | No data available | |
| | Protozoa | No data available, some removal likely | Performance for protozoan removal likely to correspond to turbidity removal |
| Microstraining | Bacteria, viruses, protozoa | Zero | Generally ineffective |
| Off-stream/ bankside storage | All | Recontamination may be significant and add to pollution levels in incoming water; growth of algae may cause deterioration in quality | Avoiding intake at periods of peak turbidity equivalent to 90% removal; compartmentalized storages provide 15–230 times rates of removal |
| | Bacteria | Zero (assumes short circuiting) | 90% removal in 10–40 days actual detention time |
| | Viruses | Zero (assumes short circuiting) | 93% removal in 100 days actual detention time |
| | Protozoa | Zero (assumes short circuiting) | 99% removal in 3 weeks actual detention time |
| Bankside infiltration | Bacteria | 99.9% after 2 m<br>99.99% after 4 m (minimum based on virus removal) | |
| | Viruses | 99.9% after 2 m<br>99.99% after 4 m | |
| | Protozoa | 99.99% | |
| **Coagulation/flocculation/sedimentation** | | | |
| Conventional clarification | Bacteria | 30% | 90% (depending on the coagulant, pH, temperature, alkalinity, turbidity) |
| | Viruses | 30% | 70% (as above) |
| | Protozoa | 30% | 90% (as above) |
| High-rate clarification | Bacteria | At least 30% | |
| | Viruses | At least 30% | |
| | Protozoa | 95% | 99.99% (depending on use of appropriate blanket polymer) |
| Dissolved air flotation | Bacteria | No data available | |
| | Viruses | No data available | |
| | Protozoa | 95% | 99.9% (depending on pH, coagulant dose, flocculation time, recycle ratio) |

**Table 7.6** ***Continued***

| Treatment process | Enteric pathogen group | Baseline removal | Maximum removal possible |
|---|---|---|---|
| Lime softening | Bacteria | 20% at pH 9.5 for 6 h at 2–8 °C | 99% at pH 11.5 for 6 h at 2–8 °C |
| | Viruses | 90% at pH < 11 for 6 h | 99.99% at pH > 11, depending on the virus and on settling time |
| | Protozoa | Low inactivation | 99% through precipitative sedimentation and inactivation at pH 11.5 |
| **Ion exchange** | | | |
| | Bacteria | Zero | |
| | Viruses | Zero | |
| | Protozoa | Zero | |
| **Filtration** | | | |
| Granular high-rate filtration | Bacteria | No data available | 99% under optimum coagulation conditions |
| | Viruses | No data available | 99.9% under optimum coagulation conditions |
| | Protozoa | 70% | 99.9% under optimum coagulation conditions |
| Slow sand filtration | Bacteria | 50% | 99.5% under optimum ripening, cleaning and refilling and in the absence of short circuiting |
| | Viruses | 20% | 99.99% under optimum ripening, cleaning and refilling and in the absence of short circuiting |
| | Protozoa | 50% | 99% under optimum ripening, cleaning and refilling and in the absence of short circuiting |
| Precoat filtration, | Bacteria | 30–50% | 96–99.9% using chemical pretreatment with coagulants polymers |
| diatomaceous earth and perlite | Viruses | 90% | 98% using chemical pretreatment with coagulants or polymers |
| | Protozoa | 99.9% | 99.99%, depending on media grade and filtration rate |
| Membrane filtration – microfiltration | Bacteria | 99.9–99.99%, providing adequate pretreatment and membrane integrity conserved | |
| | Viruses | <90% | |
| | Protozoa | 99.9–99.99%, providing adequate pretreatment and membrane integrity conserved | |
| Membrane filtration – ultrafiltration, | Bacteria | Complete removal, providing adequate pretreatment and membrane integrity conserved | |

*continued*

**Table 7.6** ***Continued***

| Treatment process | Enteric pathogen group | Baseline removal | Maximum removal possible |
|---|---|---|---|
| nanofiltration and reverse osmosis | Viruses | Complete removal with nanofilters, with reverse osmosis and at lower pore sizes of ultrafilters, providing adequate pretreatment and membrane integrity conserved | |
| | Protozoa | Complete removal, providing adequate pretreatment and membrane integrity conserved | |
| **Disinfection** | | | |
| Chlorine | Bacteria | $Ct_{99}$: 0.08 mg·min/litre at 1–2 °C, pH 7; 3.3 mg·min/litre at 1–2 °C, pH 8.5 | |
| | Viruses | $Ct_{99}$: 12 mg·min/litre at 0–5 °C; 8 mg·min/litre at 10 °C; both at pH 7–7.5 | |
| | Protozoa | *Giardia*<br>$Ct_{99}$: 230 mg·min/litre at 0.5 °C; 100 mg·min/litre at 10 °C; 41 mg·min/litre at 25 °C; all at pH 7–7.5<br>*Cryptosporidium* not killed | |
| Monochloramine | Bacteria | $Ct_{99}$: 94 mg·min/litre at 1–2 °C, pH 7; 278 mg·min/litre at 1–2 °C, pH 8.5 | |
| | Viruses | $Ct_{99}$: 1240 mg·min/litre at 1 °C; 430 mg·min/litre at 15 °C; both at pH 6–9 | |
| | Protozoa | *Giardia*<br>$Ct_{99}$: 2550 mg·min/litre at 1 °C; 1000 mg·min/litre at 15 °C; both at pH 6–9<br>*Cryptosporidium* not inactivated | |
| Chlorine dioxide | Bacteria | $Ct_{99}$: 0.13 mg·min/litre at 1–2 °C, pH 7; 0.19 mg·min/litre at 1–2 °C, pH 8.5 | |
| | Viruses | $Ct_{99}$: 8.4 mg·min/litre at 1 °C; 2.8 mg·min/litre at 15 °C; both at pH 6–9 | |
| | Protozoa | *Giardia*<br>$Ct_{99}$: 42 mg·min/litre at 1 °C; 15 mg·min/litre at 10 °C; 7.3 mg·min/litre at 25 °C; all at pH 6–9<br>*Cryptosporidium*<br>$Ct_{99}$: 40 mg·min/litre at 22 °C, pH 8 | |

**Table 7.6** ***Continued***

| Treatment process | Enteric pathogen group | Baseline removal | Maximum removal possible |
|---|---|---|---|
| Ozone | Bacteria | $Ct_{99}$: 0.02 mg·min/litre at 5 °C, pH 6–7 | |
| | Viruses | $Ct_{99}$: 0.9 mg·min/litre at 1 °C, 0.3 mg·min/litre at 15 °C | |
| | Protozoa | *Giardia*<br>$Ct_{99}$: 1.9 mg·min/litre at 1 °C; 0.63 mg·min/litre at 15 °C, pH 6–9<br>*Cryptosporidium*<br>$Ct_{99}$: 40 mg·min/litre at 1 °C; 4.4 mg·min/litre at 22 °C | |
| UV irradiation | Bacteria | 99% inactivation: 7 mJ/cm$^2$ | |
| | Viruses | 99% inactivation: 59 mJ/cm$^2$ | |
| | Protozoa | *Giardia*<br>99% inactivation: 5 mJ/cm$^2$<br>*Cryptosporidium*<br>99.9% inactivation: 10 mJ/cm$^2$ | |

Note: Ct and UV apply to microorganisms in suspension, not embedded in particles or in biofilm.

differences in treatment process efficiencies are smaller among the specific species, types or strains of microbes. Such differences do occur, however, and the table presents conservative estimates of microbial reductions based on the more resistant or persistent pathogenic members of that microbial group. Where differences in removal by treatment between specific members of a microbial group are great, the results for the individual microbes are presented separately in the table.

Non-piped water supplies such as roof catchments (rainwater harvesting) and water collected from wells or springs may often be contaminated with pathogens. Such sources often require treatment and protected storage to achieve safe water. Many of the processes used for water treatment in households are the same as those used for community-managed and other piped water supplies (Table 7.6). The performance of these treatment processes at the household level is likely to be similar to that for baseline removal of microbes, as shown in Table 7.6. However, there are additional water treatment technologies recommended for use in non-piped water supplies at the household level that typically are not used for piped supplies.

Further information about these water treatment processes, their operations and their performance for pathogen reduction is provided in more detail in supporting documents (for piped water supplies: *Water Treatment and Pathogen Control*; for non-piped [primarily household] water supplies: *Managing Water in the Home*; see section 1.3).

## 7.4 Verification of microbial safety and quality

Pathogenic agents have several properties that distinguish them from other drinking-water contaminants:

- Pathogens are discrete and not in solution.
- Pathogens are often clumped or adherent to suspended solids in water.
- The likelihood of a successful challenge by a pathogen, resulting in infection, depends upon the invasiveness and virulence of the pathogen, as well as upon the immunity of the individual.
- If infection is established, pathogens multiply in their host. Certain pathogenic bacteria are also able to multiply in food or beverages, thereby perpetuating or even increasing the chances of infection.
- Unlike many chemical agents, the dose–response of pathogens is not cumulative.

Faecal indicator bacteria, including *E. coli*, are important parameters for verification of microbial quality (see also section 2.2.1). Such water quality verification complements operational monitoring and assessments of contamination risks – for instance, through auditing of treatment works, evaluation of process control and sanitary inspection.

Faecal indicator bacteria should fulfil certain criteria to give meaningful results. They should be universally present in high numbers in the faeces of humans and other warm-blooded animals, should be readily detectable by simple methods and should not grow in natural water.

The indicator organism of choice for faecal pollution is *E. coli*. Thermotolerant coliforms can be used as an alternative to the test for *E. coli* in many circumstances.

Water intended for human consumption should contain no indicator organisms. In the majority of cases, monitoring for indicator bacteria provides a high degree of safety because of their large numbers in polluted waters.

Pathogens more resistant to conventional environmental conditions or treatment technologies may be present in treated drinking-water in the absence of *E. coli*. Retrospective studies of waterborne disease outbreaks and advances in the understanding of the behaviour of pathogens in water have shown that continued reliance on assumptions surrounding the absence or presence of *E. coli* does not ensure that optimal decisions are made regarding water safety.

Protozoa and some enteroviruses are more resistant to many disinfectants, including chlorine, and may remain viable (and pathogenic) in drinking-water following disinfection. Other organisms may be more appropriate indicators of persistent microbial hazards, and their selection as additional indicators should be evaluated in relation to local circumstances and scientific understanding. Therefore, verification may require analysis of a range of organisms, such as intestinal enterococci, (spores of) *Clostridium perfringens* and bacteriophages.

Table 7.7 presents guideline values for verification of microbial quality of drinking-water. Individual values should not be used directly from the tables. The

**Table 7.7 Guideline values for verification of microbial quality[a] (see also table 5.2)**

| Organisms | Guideline value |
|---|---|
| **All water directly intended for drinking** | |
| *E. coli* or thermotolerant coliform bacteria[b,c] | Must not be detectable in any 100-ml sample |
| **Treated water entering the distribution system** | |
| *E. coli* or thermotolerant coliform bacteria[b] | Must not be detectable in any 100-ml sample |
| **Treated water in the distribution system** | |
| *E. coli* or thermotolerant coliform bacteria[b] | Must not be detectable in any 100-ml sample |

[a] Immediate investigative action must be taken if *E. coli* are detected.

[b] Although *E. coli* is the more precise indicator of faecal pollution, the count of thermotolerant coliform bacteria is an acceptable alternative. If necessary, proper confirmatory tests must be carried out. Total coliform bacteria are not acceptable indicators of the sanitary quality of water supplies, particularly in tropical areas, where many bacteria of no sanitary significance occur in almost all untreated supplies.

[c] It is recognized that in the great majority of rural water supplies, especially in developing countries, faecal contamination is widespread. Especially under these conditions, medium-term targets for the progressive improvement of water supplies should be set.

guidelines values should be used and interpreted in conjunction with the information contained in these Guidelines and other supporting documentation.

A consequence of variable susceptibility to pathogens is that exposure to drinking-water of a particular quality may lead to different health effects in different populations. For guideline derivation, it is necessary to define reference populations or, in some cases, to focus on specific sensitive subgroups. National or local authorities may wish to apply specific characteristics of their populations in deriving national standards.

## 7.5 Methods of detection of faecal indicator bacteria

Analysis for faecal indicator bacteria provides a sensitive, although not the most rapid, indication of pollution of drinking-water supplies. Because the growth medium and the conditions of incubation, as well as the nature and age of the water sample, can influence the species isolated and the count, microbiological examinations may have variable accuracy. This means that the standardization of methods and of laboratory procedures is of great importance if criteria for the microbial quality of water are to be uniform in different laboratories and internationally.

International standard methods should be evaluated under local circumstances before being adopted. Established standard methods are available, such as those of the ISO (Table 7.8) or methods of equivalent efficacy and reliability. It is desirable that established standard methods be used for routine examinations. Whatever method is chosen for detection of *E. coli* or thermotolerant coliforms, the importance of "resuscitating" or recovering environmentally damaged or disinfectant-damaged strains must be considered.

**Table 7.8 International Organization for Standardization (ISO) standards for detection and enumeration of faecal indicator bacteria in water**

| ISO standard | Title (water quality) |
|---|---|
| 6461-1:1986 | Detection and enumeration of the spores of sulfite-reducing anaerobes (clostridia) — Part 1: Method by enrichment in a liquid medium |
| 6461-2:1986 | Detection and enumeration of the spores of sulfite-reducing anaerobes (clostridia) — Part 2: Method by membrane filtration |
| 7704:1985 | Evaluation of membrane filters used for microbiological analyses |
| 7899-1:1984 | Detection and enumeration of faecal streptococci – Part 1: Method by enrichment in a liquid medium |
| 7899-2:1984 | Detection and enumeration of faecal streptococci – Part 2: Method by membrane filtration |
| 9308-1:1990 | Detection and enumeration of coliform organisms, thermotolerant coliform organisms and presumptive *Escherichia coli* – Part 1: Membrane filtration method |
| 9308-2:1990 | Detection and enumeration of coliform organisms, thermotolerant coliform organisms and presumptive *Escherichia coli* – Part 2: Multiple tube (most probable number) method |