Quantitative microbial risk assessment to estimate health risks attributable to water supply: Can the technique be applied in developing countries with limited data?

Guy Howard, Steve Pedley and Sarah Tibatemwa

ABSTRACT

In the 3rd edition of its *Guidelines for Drinking-Water Quality* (2004) (GDWQ) the World Health Organization (WHO) promotes the use of risk assessment coupled with risk management for the control of water safety in drinking water supplies. Quantitative microbial risk assessment (QMRA) provides a tool for estimating the disease-burden from pathogenic microorganisms in water using information about the distribution and occurrence of the pathogen or an appropriate surrogate. This information may then be used to inform decisions about appropriate management of the water supply system. Although QMRA has been used to estimate disease burden from water supplies in developed countries, the method has not been evaluated in developing countries where relevant data may be scarce. In this paper, we describe a simplified risk assessment procedure to calculate the disease burden from three reference pathogens – pathogenic *Escherichia coli*, *Cryptosporidium parvum* and rotavirus – in water supplies in Kampala, Uganda. The study shows how QMRA can be used in countries with limited data, and that the outcome can provide valuable information for the management of water supplies.

Key words | drinking water safety, QMRA, risk assessment, WHO Guidelines

Guy Howard (corresponding author)
Department for International Development,
United House,10 Gulshan Avenue,
Gulshan 1, Dhaka 1212, Bangladesh
E-mail: *g-howard@dfid.gov.uk*

Steve Pedley
Robens Centre for Public and Environmental Health,
University of Surrey, Guildford,
Surrey, GU2 7XH,
UK

Sarah Tibatemwa
National Water and Sewerage Corporation,
Jinja Road, Kampala,
Uganda

INTRODUCTION

For many years the water sector has relied upon compliance with end-product standards to ensure water safety. Recently, however, the water sector has begun moving towards the use of risk assessment coupled with risk management as a more effective tool for the control of water safety (Deere *et al.* 2001; Davison *et al.* 2005). This approach to water safety has been incorporated into the 3rd edition of the *Guidelines for Drinking-Water Quality* (GDWQ) (WHO 2004). In particular, the World Health Organisation (WHO) advocates the use of water safety plans (comparable to the Hazard Assessment Critical Control Point system applied in the food industry), guided by health-based targets, with independent surveillance to verify performance. The use of quantitative risk assessment is highlighted by the WHO as a valuable tool for setting health-based targets and for validation of water safety plans (WHO 2004).

doi: 10.2166/wh.2005.058

Assessing risks from water supplies is important when trying to make judgements regarding the level of safety required in the light of alternative and multiple routes of exposure (Fewtrell & Bartram 2001). These assessments are of particular value in developing countries, where microbial pathogens may be transmitted by a number of routes (Esrey *et al.* 1991). In many cases, improving the microbial quality of water may have less impact on disease than investments in sanitation, hygiene or level of water supply access (Esrey *et al.* 1985, 1991; Howard & Bartram 2003).

Quantitative microbial risk assessment (QMRA) is a technique that has been developed for calculating the burden of disease from a particular pathogen. The major tasks of a QMRA have been defined by Haas & Eisenberg (2001) as exposure assessment, dose-response analysis and risk characterisation. In order to capture and compare the

various outcomes from different pathogens, the use of disability adjusted life years (DALYs) has been recommended in risk assessment (Havelaar & Melse 2003; WHO 2004). Murray & Lopez (1996) provide data from which to calculate DALYs for health effects derived from infection by waterborne pathogens.

QMRA is typically confined to individual causative agents and specific disease symptoms rather than undifferentiated health effects (Haas *et al.* 1999). Initial work in the US developed risk models for *Giardia* and rotavirus, which provided the levels of treatment required to meet targets related to acceptable levels of risk of disease (Regli *et al.* 1991). This analysis was possible because the health data required for the analysis were readily available.

Completing a QMRA for every pathogen that may be transmitted by water would be time-consuming and the necessary information is currently not available for many pathogens. To overcome this difficulty, WHO (2004) recommended using a suite of 'reference pathogens'. A reference pathogen is an organism whose severity of impact and persistence in water is such that its control would provide confidence that health risks from pathogens of a similar nature have also been controlled (WHO 2004). The distribution and concentration of pathogens in water are highly variable, and the use of reference pathogens introduces another layer of uncertainty into the analysis. Hence, QMRA modelling often includes Monte Carlo simulation techniques to help capture the uncertainty and variability within frequency distributions. However, the complexity of these techniques and the need for proprietary and costly software, largely limits their use to the developed world. To expand the scope of QMRA, a simplified approach has been developed using point estimates (WHO 2004).

The limited data on pathogens in developing countries requires that a QMRA be based on the occurrence of indicator organisms. Despite the weaknesses of using indicator organisms, Haas *et al.* (1999) believe that many initial QMRAs will have to be performed using data on indicator organisms due to inadequate data for occurrence of pathogens. The use of indicator organisms does, however, require assumptions to be made about the relationship between pathogens and indicators that introduces an additional level of uncertainty into the risk assessment.

QMRA is an important tool for describing the potential risks from water supply and for informing water safety management strategies. There are challenges to applying the method anywhere, but these challenges are greater in developing countries with limited data and resources. If QMRA is to gain acceptance and be used in developing countries, it must be offered in a workable and simple form. This paper describes the successful use of a simplified QMRA of the piped water supply, protected springs and water stored in households in Kampala, Uganda.

Description of the water supplies in Kampala, Uganda

The National Water and Sewerage Corporation (NWSC) is responsible for the provision and quality control of domestic piped water in Kampala. During the period of study, the distribution system was managed under contract by a private operator, Ondeo Services Uganda Limited. Raw water is drawn from the Inner Murchison Bay on Lake Victoria and passed through one of two treatment works, Gaba 1 and Gaba 2. The Gaba 1 works uses rapid sand filtration followed by chlorination; the Gaba 2 works has a coagulation-flocculation-settling step before filtration and chlorination.

Gaba 1 supplies the low-level distribution system close to the plant and the Gun Hill reservoir in the centre of Kampala, which, in turn, supplies the low-level distribution system in the centre of the city. Some water from Gaba 1 is pumped into the primary transmission main from Gaba 2 that runs to the Muyenga balancing tanks. The Muyenga balancing tanks supply the high-level distribution system directly, and via three further service reservoirs. There is a total of 871 kilometres of pipeline supplying 120 Ml per day from the works.

Overall connection rates in Kampala were low during the period of study. The household connection rate was approximately 20%. Of the population without a domestic connection, previous studies have estimated that 70% use piped water for at least part of their domestic needs, but that the use of alternative sources was common (Howard *et al.* 2002). The alternative supplies in Kampala were primarily protected springs, which are found spread throughout the city in low and high density areas (Howard 2001). Many of these springs are contaminated and have been shown to be associated with diarrhoeal disease (Barrett *et al.* 2000;

Nasinyama *et al.* 2000; Howard *et al.* 2003; Howard & Bartram 2005).

Data used in the QMRA were taken from three sources: routine monitoring by NWSC, data collected during a three-year surveillance project managed by the Ministry of Health (Howard & Luyima 1999) and specific water quality assessments undertaken in 2002 and 2003 by the authors as part of a larger research project to develop water safety plans.

METHODOLOGY

Risk assessment procedure

The disease burden was calculated following the method described in the 3rd edition of the GDWQ (WHO 2004). The approach is summarised in Table 1.

Selection of reference pathogens and indicator organisms

Three reference pathogens were selected for this assessment: pathogenic *Escherichia coli*, *Cryptosporidium parvum* and rotavirus. However, as pathogen data from water supplies in Uganda was scarce, indicator organisms were used as surrogates. The surrogate organisms were: thermotolerant coliforms (*E. coli* was confirmed in some of the studies) to indicate the presence of pathogenic *E. coli*; sulphite-reducing clostridia as a model for protozoa removal through water treatment processes; and somatic coliphages as a model for human enteric virus removal through the treatment processes. Risk assessments were performed for all three reference pathogens for water leaving the treatment works, but only for pathogenic *E. coli* in the distribution systems, protected springs and household water as data were only available for thermotolerant coliforms.

Reference pathogens

Several pathogenic strains of *E. coli* have been identified (Haas *et al.* 1999). Enterotoxigenic *E. coli* (O157:H7) is a well-known waterborne pathogen in developed countries and has been the cause of high-profile outbreaks of disease, such as that at Walkerton, Ontario (Health Canada 2000;

Table 1 | Simplified procedure for calculating disease burden (adapted from WHO 2004)

Raw water quality, organisms per litre (C_R)	Will probably be calculated from concentrations in standard volumes (e.g. 100 ml) and may not be directly for pathogens
Treatment effect (PT)	Estimated or calculated removal of pathogens
Drinking water quality (C_D)	$C_R \times (1\text{-PT})$
Consumption of unheated drinking water (V)	Estimated or calculated
Exposure by drinking water, organisms per litre (E)	$C_D \times V$
Dose-response (r)	From literature
Risk of infection per day ($P_{inf,d}$)	$E \times r$
Risk of infection per years ($P_{inf,y}$)	$1\text{-}(1\text{-}P_{inf,d})^{365}$ (Haas *et al.* 1999)
Risk of diarrhoeal disease given infection ($P_{ill\|inf}$)	From literature
Risk of diarrhoeal disease (P_{ill})	$P_{inf,y} \times P_{ill\|inf}$
Maximum disease burden (mdb)	Calculated from available data and from data reported in literature
Susceptible fraction (fs)	From literature
Disease burden (DB)	$P_{ill} \times mdb \times fs$

Hunter 2003). Animals are the major reservoir of *E. coli* O157:H7, with cattle being the principal reservoir (Haas *et al.* 1999; WHO 2004). This is particularly important in Uganda, where previous studies have shown that a significant proportion of microbial contamination is derived from animal faeces (Barrett *et al.* 2000). In undertaking the QMRA, the risk from all pathogenic *E. coli* was calculated based on the impact expected from *E. coli* O157:H7.

C. parvum was selected as a reference pathogen for protozoan pathogens found in drinking water. *Cryptosporidium* is a significant pathogen among adults who are HIV positive or suffering from AIDS and it is a cause of persistent diarrhoea among young children (Harries 1991; Tarimo *et al.* 1996; Mølbak *et al.* 1997; Mwachari *et al.* 1998; Sodemann *et al.* 1999). Water is a significant route of infection in developing countries (Kelley *et al.* 1997).

Rotavirus is believed to account for a significant proportion of diarrhoeal disease in developing countries (WHO 1996, 2004). Havelaar & Melse (2003) report that studies in the 1980s estimated the number of cases to be 125 million, of which 18 million (14.4%) were severe with 873,000 deaths (a case-fatality ratio of 0.7%). These authors note that 45% of children below two years of age in developing countries carry rotavirus and that 20–40% of severe diarrhoea cases were caused by the virus. Rotavirus transmission occurs through a number of routes and personal hygiene may be more important than drinking water (WHO 2004).

Estimating pathogen numbers based on indicator organisms

Thermotolerant coliforms were used as a surrogate for pathogenic *E. coli*. We have assumed that 95% of thermotolerant coliforms were *E. coli* (WHO 1996) and that 8% of *E. coli* were pathogenic (Haas *et al.* 1999). Whereas some studies in tropical environments have suggested that a lower proportion of thermotolerant coliforms may be *E. coli* (Hazen & Torranos 1990), previous studies in Uganda have shown that when faecal pollution is likely, based on observation and sanitary surveys, the majority of thermotolerant coliforms are *E. coli* (Howard *et al.* 2003).

Ashbolt *et al.* (2001) have noted that the spores of sulphite-reducing clostridia can be used as an index for *Cryptosporidium*, in particular in assessing removal through treatment given a similar resistance to chlorination. In this study we have used sulphite-reducing clostridia to indicate the risk from *C. parvum* in the water. A similar relationship between coliphage and enteric viruses has been reported by Grabow (2001). In this study we have used coliphage as a surrogate for rotavirus. The concentrations of sulphite-reducing clostridia and somatic coliphages in raw water were taken to represent worse case scenarios for the concentrations of the respective pathogens.

Establishing disease burdens for inclusion in risk models

In order to maintain a consistent comparison across the three pathogens, the health burden for each pathogen was related solely to that associated with diarrhoeal disease and death. The sequelae from *E. coli* O157:H7, including haemolytic uraemia syndrome (HUS) and end-stage renal disease add significantly to the overall disease burden. Although there is sufficient data for HUS in Africa to incorporate this outcome into the risk model, there is far less data regarding the sequelae from *C. parvum* and rotavirus. We considered that including sequelae data (other than death) for pathogenic *E. coli* but not for *C. parvum* or rotavirus would potentially bias the results of the risk comparisons.

The estimate of the years of life lost from premature death (the mortality fraction) and years of life impaired (the morbidity fraction) for each pathogen was calculated using the average life expectancy at birth for Uganda, 46.4 years (WHO 2002), rather than the global life expectancy that has been used in other assessments (Murray & Lopez 1996; Havelaar & Melse 2003). The use of the local life expectancy was felt to more realistically reflect the impact of diseases in Uganda and would avoid all diseases having a very large impact. Ideally in a QMRA, the years of life lost should be based on a weighted average of age of death by age group (Havelaar & Melse 2003); however, for this simplified risk assessment only a single average expected age of death was used. Similarly, no separate risk was calculated for high-risk groups, such as those with HIV/AIDS.

The use of national life expectancy does introduce a potential problem, as it distorts the size of disease burdens

towards morbidity and mortality of the very young. However, as with many of the assumptions to which QMRA model predictions are sensitive, such imperfections are only significant when the aim is to compare systems in which very different input data and assumptions might be used. When the aim is to provide a consistent internal QMRA estimate the bias is likely to be close to equivalent for the different scenarios under consideration, neutralising the effect of that bias.

Pathogenic *E. coli*

The disease burden for pathogenic *E. coli* was based on the strain with most severe outcomes, *E. coli* O157:H7; however, there is only limited dose-response data for *E. coli* O157:H7. Hunter (2003) notes that the ID_{50} (the number of organisms required to infect 50% of people exposed) ranges between 10^2 and 10^6. Haas *et al.* (2000) and Strachan *et al.* (2000) have reported disease outbreaks resulting from doses estimated to have been around 10^2 organisms. Teunis *et al.* (2004) have recently published a dose-response for *E. coli* O157:H7 based on a food-borne outbreak in Japan.

Haas *et al.* (1999) have argued that a reasonable estimate of disease burden for *E. coli* O157:H7 can be made using the widely published dose-response data for *Shigella* infections. We have used dose-response estimates for *Shigella* to provide a generic assessment of risk from waterborne bacteria. For watery diarrhoea and bloody diarrhoea, the proportion of symptomatic cases was 53% and 47%, respectively (Havelaar & Melse 2003). Kotloff *et al.* (1999) have reported a mortality rate for *Shigella* infection of 0.7% in developing countries. In contrast, Havelaar & Melse (2003) have estimated a mortality rate in the Netherlands of 0.2% for infection with *E. coli* O157:H7. In this analysis we have used the mortality rate quoted by Kotloff *et al.* (1999) since it is more likely to reflect the higher mortality among children in developing countries. The mortality burden was based on an average age of death of 12 months.

For this assessment the severity weights for the different outcomes were taken from Havelaar & Melse (2003). The duration of watery and bloody diarrhoea was 3.4 and 5.6 days, respectively.

Cryptosporidium parvum

The most common outcome of *C. parvum* infection is watery diarrhoea. Death can occur, mainly amongst immuno-compromised individuals. In this QMRA, a mean duration of watery diarrhoea of 7.2 days was used (Havelaar & Melse 2003). Disease burden studies in the Netherlands have produced estimates of 1.47 DALYs per 1,000 infections (Havelaar & Melse 2003). However, these authors note that immune status is an issue of much greater importance when undertaking disease burden assessments for developing countries and in regions where HIV/AIDS prevalence is high. The prevalence rate for HIV/AIDS in Uganda has been estimated as 8% (UNAIDS 2003, www.unaids.org). The mortality rate from cryptosporidiosis amongst this group in Uganda is difficult to estimate. In the absence of any conflicting data an estimate of 10% mortality among the HIV population (equivalent to 0.8% of the total population) was used (Havelaar & Melse 2003). The years-of-life-lost amongst the HIV/AIDS group was calculated using data from UNAIDS (www.unaids.org). The data shows that 85% of HIV/AIDS infections in Uganda occur in the age group 15–49 years, with a median age of death of 30.7 years. The remaining 15% was reported to occur in children under 15 years. For this group a median age of death of 7.5 years was used. The final estimate of years-of-life-lost due to cryptosporidiosis was based on a weighted average using the approach shown in Equations (1) to (3):

$$(46.4 - 30.7) \times 85\% = 13.345 \quad (1)$$

$$(46.4 - 7.5) \times 15\% = 5.835 \quad (2)$$

$$(13.345 + 5.853)/100 = 19.18, \text{rounded up to } 19.2 \text{ years lost} \quad (3)$$

Rotavirus

The severity weighting and duration of disease data for rotavirus were taken from Havelaar & Melse (2003), with the years-of-life-lost based on age of death at 12 months. The higher severity weights allocated for rotavirus reflect the distribution of mild and severe cases. The burden of disease study weightings for diarrhoea for under 5 s

(0.119) and for older age groups (0.086–0.094) were used (Havelaar & Melse 2003).

Havelaar & Melse (2003) noted that the results of several studies indicate a case-fatality rate for rotavirus of 0.7%. Nevertheless, in their calculations they used a case-fatality rate of 0.6% for developing countries to reflect what they presumed to be an improvement in treatment since the 1980s. For this study a figure of 0.7% has been used because we felt that the assumption of improvement would be offset by potentially greater numbers of severe diarrhoea and the increase in likelihood of severe end-points due to HIV infection. The dose-response estimate was taken from WHO (2004), the risk of developing illness from Havelaar & Melse (2003).

The disease burdens for each of the three pathogens are summarised in Tables 2 and 3.

Calculating susceptible populations

The susceptible population was calculated from known rates of access to piped water supply in Kampala. Previous studies have shown that 20% of the population use a direct household connection and 80% rely on communal sources of water (Howard *et al.* 2002). Data from a water usage study showed the following patterns of use (Howard *et al.* 2002):

- 25.2% of the unserved population use a single tap source
- 3.6% of the unserved population use two tap sources and no other type of water source
- 30.8% of the unserved population use a tap as a first source and another type of water as a second source
- 8.2% of the unserved population use a tap as a second source and another type of water as the first source

One of the major issues facing the use of risk assessment models in developing countries is how to assess exposure among a population that uses more than one type of drinking water source. In this study, two possible approaches were considered:

1. The total exposure from drinking water is allocated to each source. In reality, this approach may significantly overstate the allocation of exposure to one source if this source is of significantly higher quality. This is certainly the case in Kampala where piped water is of much better microbial quality than alternative sources (Howard 2001).
2. Exposure is allocated between the sources with a 'discounting' factor for exposure allocated to each source. This approach has the advantage of more reliably reflecting real exposures; however, it presents difficulties in determining what form of 'discounting' to employ.

In this study we have used the second approach. A discounting factor was calculated from the estimated

Table 2 | Severity, duration and disease burden for pathogens included in study

Pathogen	Outcomes	Severity	Duration	Disease burden (DALYs)
E. coli O157:H7	Watery diarrhoea	0.067	3.4 days	0.0006
	Bloody diarrhoea	0.39	5.6 days	0.0060
	Death from diarrhoea	1	45.4 years	45.4
Cryptosporidium parvum	Watery diarrhoea	0.067	7 days	0.0013
	Death	1	19.2 years	19.2
Rotavirus	Mild diarrhoea	0.10	7 days	0.002
	Severe diarrhoea	0.23	7 days	0.004
	Death	1	45.4 years	45.4

Table 3 | Disease burden for pathogens included in study

Pathogen	Outcomes	Disease burden per 1000 symptomatic cases of gastroenteritis	Disease burden per case
E. coli O157:H7	Watery diarrhoea	1000 × 53% (watery diarrhoea) × 0.067 × 0.009 = 0.3	
	Bloody diarrhoea	1000 × 47% (bloody diarrhoea) × 0.39 × 0.015 = 2.8	
	Death from diarrhoea	1000 × 0.7% (death) × 45.4 = 317.8	
	Total diarrhoea only	320.9	0.32
Cryptosporidium parvum	Watery diarrhoea	1000 × 0.067 × 0.02 = 1.34	
	Death	1000 × 0.8% (death) × 19.2 = 153.6	
	Total	154.94	0.15
Rotavirus	Mild diarrhoea	1000 × 85.6% × 0.10 × 0.02 = 1.71	
	Severe diarrhoea	1000 × 14.4% × 0.23 × 0.02 = 0.66	
	Death	1000 × 0.7% (death) × 45.4 = 317.8	
	Total	320.17	0.32

proportion of users taking water from each source. The calculation assumes that exposure is solely a function of the proportion of water collected from each source, rather than contaminant loads within each source, and that all sources of water have the same potential to be a route of exposure. Discounting was calculated by allocating two-thirds of the population to the first source and one-third to the second. Hence, the population using a tap as first source was multiplied by 0.67 to allocate a third of exposure to the alternative source. Where taps were the second source, the figure was multiplied by 0.33 to allocate two-thirds of the exposure to other sources. A final figure was obtained by converting the total percentage of the exposed, unserved population into a percentage of the overall population by multiplying by 0.8 and adding this to the 20% of the population that received water through a household connection.

This gives a total percentage of the unserved population that is susceptible as:

Percentage of population using other water sources who are also exposed from piped water = (30.8 * 0.67) + (8.2 * 0.33) = 23.3%

Total percentage of unserved population exposed = (25.2 + 3.6 + 23.3) = 52.1%

Total percentage of unserved population exposed to piped water as proportion of total unserved population = (52.1 * 0.8) = 41.7%

Total population exposed = 20% + 41.7% = 61.7%

For ease of calculation this was rounded to 62%. We have assumed that each of the two treatment works serves an equal number of people: 31% of the total population.

Further adjustments were made to the exposed and susceptible populations figures to account for specific properties linked to the pathogen or water system:

- For the QMRA of pathogenic *E. coli*, we estimated that 10% of the population served by the piped water system would be exposed since compliance failures occurred only at the end of distribution lines (Howard, unpublished observation).
- For the risk assessment of the protected springs, a susceptible fraction of 28% was used (100% minus the proportion allocated to piped water).

- For the risk assessment of recontamination of piped water stored in homes, a susceptible fraction of 42% (rounded up from the 41.7% calculated above) was used, as only those unserved households exposed to piped water were included.
- We have assumed that all the population exposed would be susceptible to infection by pathogenic *E. coli* and *C. parvum*. Although some immunity to these pathogens may be acquired, it was assumed that this is relatively short-lived.
- The fraction of the population susceptible to rotavirus infection is influenced by the vulnerability of young children and the development of immunity in older children and adults. Based on estimates published in the GDWQ (WHO 2004), 17% of the total population exposed was considered susceptible.

Water quality data

Table 4 summarises the sources of data used for the risk assessment. Each assessment of the treatment works was carried out using paired samples: a raw water sample and a sample from the outlet of each treatment process. Samples taken from the distribution system in the surveillance project were based on stratified random sampling (Howard & Luyima 1999). Data on protected springs were taken from a longitudinal study of 63 springs in high, medium and low-density areas of Kampala, with samples taken between April 1998 and March 1999 (Howard & Luyima 1999). The springs varied in their condition; high-density areas were generally less well maintained and had higher sanitary risk scores (Howard *et al.* 2003). Water quality data for household storage was taken from Howard & Luyima (1999). Only those data from samples that could be matched with a source water from an NWSC supply were included.

The figures used in the QMRA are expressed in infectious pathogens per litre.

Analytical methods

Somatic coliphages

Somatic coliphages were isolated and enumerated using the method of Borrego *et al.* (1987). A 1 ml sample of the water under test was mixed with 0.2 ml of *E. coli* C (ATCC 13706), grown to mid-log phase, and 3 ml of molten soft Luria agar. The mixture was poured onto pre-prepared petri dishes containing a solid Luria agar base. The agar was allowed to solidify at room temperature before the plates were incubated at 37°C for 18 hours. The results were recorded as plaque forming units per millilitre (pfu ml^{-1}).

Escherichia coli

In the assessment performed by the authors in 2002 and 2003, *E. coli* was isolated and enumerated using the membrane filtration technique and membrane lauryl sulphate broth (MLSB) (Oxoid, UK) as the selective medium (Anon 2002*a*). An appropriate volume of the water sample (normally 100 ml) was filtered though a cellulose acetate filter with a pore size of 0.45 μm. The filter was layered onto a filter pad saturated with MLSB and pre-incubated at room temperature (approximately 25°C) for 4 hours followed by incubation at 44.5 + /− 0.5°C for 14 hours. *E. coli* was confirmed from all presumptive positive colonies by inoculating a sample of the colonies into separate 0.5 ml volumes of IDEXX Colilert®-18 reagent and incubating at 37°C for between 8 and 14 hours. A yellow colour in the IDEXX Colilert®-18 reagent with fluorescence under UV light was taken as confirmation of *E. coli*. All results were reported as colony forming units per 100 ml (cfu 100 ml^{-1}).

The analytical method used by NWSC was identical to the method described above, except that the incubation at 44.5 + /− 0.5°C was continued for 20 hours and *E. coli* was confirmed from all presumptive positive colonies using the Enterotube identification system (Beckton Dickinson, UK).

The assessment programmes carried out by the Ministry of Health (Howard & Luyima 1999) were performed using portable water test kits (Oxfam-Delagua water test kit, RCPEH, University of Surrey, UK). The analyses were carried out using the membrane filtration technique described above (RCPEH 2004). No confirmatory *E. coli* analysis was undertaken.

Sulphite-reducing clostridia

Clostridium perfringens spores were isolated and enumerated using membrane filtration technique and growth on

Table 4 | Summary of water quality data used in the QMRAs

Assessment and date (source of data)	No. samples	Median no. organisms raw water	Calc. pathogenic *E. coli* raw water	Median no. organisms final water	Calc. pathogenic *E. coli* final water	Treatment effect (excluding chlorination)
E. coli through Gaba 1, 2002 (by authors)	36	42 100 ml^{-1} (range <1-TNTC)	32 l^{-1}	<1 100 ml^{-1}	<0.9 l^{-1}	3 $\log_{10}$ (min)
E. coli through Gaba 2, 2002 (by authors)	19	20 100 ml^{-1} (range 2–120)	18 l^{-1}	<1 100 ml^{-1}	<0.9 l^{-1}	3 $\log_{10}$ (min)
Thermotolerant coliforms through Gaba 1, 1994–1998 (NWSC data)	200	15 100 ml^{-1}	11.5 l^{-1}	<1 100 ml^{-1}	<0.9 l^{-1}	3 $\log_{10}$ (min)
Thermotolerant coliforms through Gaba 2, 1994–1998 (NWSC data)	200	10 100 ml^{-1}	7.5 l^{-1}	<1 100 ml^{-1}	<0.9 l^{-1}	3 $\log_{10}$ (min)
Thermotolerant coliforms: distribution 1998 (Ministry of Health data)	713	Not applicable	Not applicable	0.23 100 ml^{-1} (range <1–17 100 ml^{-1})	0.18 l^{-1}	Not applicable
Thermotolerant coliforms: distribution 1999 (Ministry of Health data)	913	Not applicable	Not applicable	0.13 100 ml^{-1} (range <1–30 100 ml^{-1})	0.10 l^{-1}	Not applicable
Thermotolerant coliforms: protected springs 1998–1999 (Ministry of Health data)	609	Not applicable	Not applicable	14 100 ml^{-1} (range <1–TNTC)	10.6 l^{-1}	Not applicable
Thermotolerant coliforms: household water 1999 (Ministry of Health data)	97	Not applicable	Not applicable	3 100 ml^{-1} (range <1–TNTC)	2.3 l^{-1}	Not applicable
Sulphite-reducing clostridia through Gaba 1, 2002 & 2003 (by authors)	36	3 100 ml^{-1} (range <1–TNTC)	Not applicable	<1 100 ml^{-1}	Not applicable	4 $\log_{10}$
Sulphite-reducing clostridia through Gaba 2, 2002 & 2003 (by authors)	19	5 100 ml^{-1} (range 1–5)	Not applicable	<1 100 ml^{-1} (range <1–20)	Not applicable	4 $\log_{10}$
Somatic coliphage through Gaba 1, 2003 (by authors)	36	<1 1 ml^{-1}	Not applicable	<1 1 ml^{-1}	Not applicable	Not done
Somatic coliphage through Gaba 2, 2003 (by authors)	19	1 1 ml^{-1}	Not applicable	<1 1 ml^{-1}	Not applicable	Not done

membrane *Clostridium perfringens* agar (Oxoid, UK) in an anaerobic atmosphere (Anon 2002*b*). A 100 ml volume of the water sample was heated to 60°C for 15 minutes to kill the vegetative cells. The sample was cooled to room temperature and filtered through a 0.45 μm cellulose acetate membrane. The membrane was layered onto *C. perfringens* agar and the petri dishes incubated in an anaerobic atmosphere at 37°C for 24 hours. All black colonies were counted and the results were recorded as cfu per 100 ml sulphite-reducing clostridia.

RESULTS

Risk assessment findings

Pathogenic *E. coli*

In addition to the median three log reductions in the number of *E. coli* (Table 4), a further two log reduction was added to account for disinfection performance. The final log reduction of 10^5 is consistent with published data for other conventional treatment systems (WHO 2004). Table 5 summarises the risk assessment for pathogenic *E. coli*. WHO (2004) suggests that a reference level of risk of 10E-06 is tolerable. The results of the QMRA performed using the 2002 assessment data indicate that the disease burden slightly exceeds the reference level of risk. The QMRA carried out using the 1999 monitoring data shows that the levels of risk are lower. Gaba 1 slightly exceeds the WHO Guidelines for health-based targets and Gaba 2 is below the level of risk suggested by WHO as tolerable.

The QMRA for pathogenic *E. coli* in the distribution system is shown in Table 6. Using the data from monitoring programmes carried out in 1998 and 1999 (Table 4), the calculated risks were much higher (5.26E-04 for 1998 and 2.92E-04 for 1999) than they were for the water leaving the treatment works (Table 5). Re-running the QMRA for single

Table 5 | Risk assessment for pathogenic *E. coli* following treatment works

	2002 Assessment data		1999 Monitoring data	
	Gaba 1	Gaba 2	Gaba 1	Gaba 2
Raw water quality (C_R)	32	18	11.5	7.5
Treatment effect (PT)	0.99999	0.99999	0.99999	0.99999
Drinking water quality (C_D)	3.20E-04	1.80E-04	1.15E-04	7.50E-05
Consumption of unheated drinking water (V)	1	1	1	1
Exposure by drinking water, organisms per litre (E)	3.20E-04	1.80E-04	1.15E-04	7.50E-05
Dose-response (r)	1.00E-03	1.00E-03	1.00E-3	1.00E-03
Risk of infection per day ($P_{inf,d}$)	3.20E-07	1.80E-07	1.15E-07	7.50E-08
Risk of infection per years ($P_{inf,y}$)	1.17E-04	6.57E-05	4.20E-06	2.74E-05
Risk of diarrhoeal disease given infection ($P_{ill|inf}$)	0.25	0.25	0.25	0.25
Risk of diarrhoeal disease (P_{ill})	2.92E-05	1.64E-05	1.05E-05	6.84E-06
Maximum disease burden (mdb)	3.20E-01	3.20E-01	3.20E-01	3.20E-01
Susceptible fraction (fs)	0.31	0.31	0.31	0.31
Disease burden (DB)	2.90E-06	1.63E-06	1.04E-06	6.79E-07

Table 6 | Annualised risk assessment for pathogenic *E. coli* for the distribution system

	1998	1999
Drinking water quality (C_D)	0.18	0.10
Consumption of unheated drinking water (V)	1	1
Exposure by drinking water, organisms per litre (E)	1.80E-01	1.00E-01
Dose-response (r)	1.00E-3	1.00E-3
Risk of infection per day ($P_{inf,d}$)	1.80E-04	1.00E-04
Risk of infection per years ($P_{inf,y}$)	6.57E-02	3.65E-02
Risk of diarrhoeal disease given infection ($P_{ill|inf}$)	0.25	0.25
Risk of diarrhoeal disease (P_{ill})	1.64E-02	9.13E-03
Maximum disease burden (mdb)	3.20E-01	3.20E-01
Susceptible fraction (fs)	0.1	0.1
Disease burden (DB)	5.26E-04	2.92E-04

contamination events gives an upper-bound estimate. For 1998 this is based on 17 cfu 100 ml- and for 1999 30 cfu 100 ml^{-1} (Table 4). The calculated upper-bound potential burdens are 3.77E-02 for 1998 and 6.66E-02 for 1999. This analysis suggests that post-works contamination is a much greater problem than treatment failure.

Table 7 summarises the risk assessment of stored household water, using the median level of contamination (Table 4). A final disease burden of 2.82E-02 DALYs was obtained. To provide a point of comparison, the QMRA was re-run using the highest countable result of 562 cfu 100 ml^{-1}. This provides a disease burden estimate of 5.24E + 00. The risk assessment for protected springs is also summarised in Table 7 using the median level of contamination (Table 4). An annualised disease burden was calculated as 8.67E-02 DALYs. Using the highest countable result of 770 cfu 100 ml^{-1}, the disease burden was 4.78E + 00.

C. parvum

Table 4 shows that a minimum of 10^4 removal of *C. perfringens* was achieved in Gaba 1 and a similar figure was assigned to Gaba 2. The risk estimates for *C. parvum* are shown in Table 8. The estimated disease burden for Gaba 1 was low, but exceeds the WHO reference level of risk. The risk from Gaba 2 under normal conditions was significantly higher. This is likely to be a consequence of limited data being available and the high mortality burden among people with HIV/AIDS. There was one failure at Gaba 2 and performing a risk assessment on this event demonstrated a significant increase in the disease burden (4.28E + 00).

Rotavirus

The risk estimates for rotavirus are shown in Table 8. The final burden of disease was high for both the Gaba 1 and Gaba 2 treatment works (7.88E-03 and 7.10E-03, respectively). Since coliphage were not detected in the final water (Table 4), we have assumed at least a 10^4 reduction. This was increased to 10^5 based on potential removal through conventional treatment (WHO 2004).

DISCUSSION

The simplified methodology for QMRA described here provided a first estimate of the health burden from microbial contaminants in water sources in Uganda. These estimates will be improved as more data become available. There are, however, some limitations to the QMRA carried out in this study.

The use of indicator and index organisms to provide estimates of pathogen concentrations in raw water and to measure treatment efficiency, introduces uncertainties into the final estimates, since their numbers may not reflect pathogen loads in the source water or pathogen recontamination during distribution. Furthermore, the use of point estimates does not capture the full range of disease outcomes. Approaches that use statistical distributions of health effects can provide more useful information, but only if there is sufficient data to create such distributions (Teunis *et al.* 1997; Havelaar & Melse 2003; WHO 2004).

In the absence of local public health and microbiological data, the concentration of *E. coli* O157:H7 was estimated from published ratios of generic *E. coli* to

Table 7 | Risk assessment for pathogenic *E. coli* O157:H7 in protected springs and stored water in households using NWSC water

	Stored water in household using NWSC water	Protected springs
Raw water quality (C_R)	2.3	10.6
Treatment effect (PT)	0	0
Drinking water quality (C_D)	2.3	10.6
Consumption of unheated drinking water (V)	1	1
Exposure by drinking water, organisms per litre (E)	2.3E + 00	1.06E + 01
Dose-response (r)	1.00E-03	1.00E-03
Risk of infection per day ($P_{inf,d}$)	2.30E-03	1.06E-02
Risk of infection per years ($P_{inf,y}$)	8.40E-01	3.87E + 00
Risk of diarrhoeal disease given infection ($P_{ill|inf}$)	0.25	0.25
Risk of diarrhoeal disease (P_{ill})	2.10E-01	9.67E-01
Maximum disease burden (mdb)	3.20E-01	3.20E-01
Susceptible fraction (fs)	0.42	0.28
Disease burden (DB)	2.82E-02	8.67E-02

pathogenic *E. coli* (Haas *et al.* 1999). However, it is likely that the relative levels of *E. coli* and *E. coli* O157:H7 vary spatially and temporally and the use of a single estimate may significantly exaggerate or underplay the risk. For example, if the ratio of *E. coli* to strain O157:H7 is reduced by an order of magnitude, the final risk estimate would be reduced by the same amount. Nevertheless, 8% is a reasonable first estimate for this study since it highlights the differences in disease burden between water taken from different sources and between water at the outlet from the treatment works and in the distribution system. Furthermore, although the ratio may not accurately predict *E. coli* O157:H7, it should provide a reasonable estimate of the overall health burden derived from bacterial pathogens. Using the dose-response reported by Teunis *et al.* (2004) the risk estimates increase significantly for *E. coli* O157:H7. However, the use of this dose-response relationship is probably only appropriate when assessing actual levels of *E. coli* O157:H7 in source waters and not when applied as a generic reference for all bacterial pathogens.

We found that the risk assessment for *C. parvum* was limited by the absence of data from Uganda to show a direct relationship between the numbers of presumptive *C. perfringens* and *C. parvum* in source waters. For the purpose of this assessment, we have assumed that the removal rate of *C. perfringens* during treatment is indicative of the removal rate of *C. parvum*. According to WHO (2004) moderate to high concentrations of *C. parvum* in raw water range between 30 and 300 oocysts per litre. Therefore, the predicted concentration in the raw water at the Gaba 2 works based upon the concentration of *C. perfringens* may be reasonable. It is of greater concern to this assessment that only a small number of data are available from a single assessment.

The risk assessment for rotavirus probably significantly over-estimates the disease burden from water. The absence of somatic coliphage in the final water and the limited amount of data collected makes more reliable estimates difficult. Although the risk may be over-estimated, it is likely that water contributes to a significant proportion of rotavirus infections.

Table 8 | Risk assessment for *Cryptosporidium parvum* and rotavirus

	Cryposporidium parvum			Rotavirus	
	Gaba 1	**Gaba 2 (normal)**	**Gaba 2 (failure)**	**Gaba 1**	**Gaba 2**
Raw water quality (C_R)	30	50	210	1000	900*
Treatment effect (PT)	0.9999	0.9999	0	0.99999	0.99999
Drinking water quality (C_D)	3.00E-03	5.00E-03	2.10E+2	1.00E-02	9.00E-03
Consumption of unheated drinking water (V)	1	1	1	1	1
Exposure by drinking water, organisms per litre (E)	3.00E-03	5.00E-03	2.10E+2	1.00E-02	9.00E-03
Dose-response (r)	4.00E-03	4.00E-03	4.00E-03	2.70E-01	2.70E-01
Risk of infection per day ($P_{inf,d}$)	1.20E-05	2.00E-05	8.40E-01	2.70E-03	2.43E-03
Risk of infection per year ($P_{inf,y}$)	4.38E-03	7.30E-03	3.07E+02	9.85E-01	8.87E-01
Risk of diarrhoeal disease given infection ($P_{ill\|inf}$)	0.30	0.30	0.30	0.50	0.50
Risk of diarrhoeal disease (P_{ill})	1.31E-03	2.19E-03	9.20E+01	4.93E-01	4.43E-01
Maximum disease burden (mdb)	1.50E-01	1.50E-01	1.50E-01	3.20E-01	3.20E-01
Susceptible fraction (fs)	0.31	0.31	0.31	0.05	0.05
Disease burden (DB)	6.11E-05	1.02E-04	4.28E+00	7.88–03	7.10E-03

*Estimated based on non-detection in 1 ml and a default concentration of 0.9 ml^{-1}

The use of multiple sources of water is a common practice in Uganda and it is important that this is reflected in the QMRA. We have used 'discounting' factors to take into account the use of multiple sources and to allocate a reasonable proportion of total exposure to an individual source. Although this is likely to be somewhat controversial, discounting does attempt to reflect the reality of water collection patterns and, therefore, potential exposure. Monte Carlo simulation could have been used to estimate the proportion of users of taps, but this was considered to introduce a level of complexity that would have been contrary to the goal of developing a simplified risk assessment approach.

Despite the limitations of the analysis, the evidence from this study shows that QMRA provides useful quantitative data for planning and policy, in particular where investments in water supply improvement can deliver the greatest health gain. When comparing the results of the QMRA with the WHO reference level of risk, a number of key findings emerge. For pathogenic *E. coli*, there appeared to be relatively little risk from water leaving the treatment works. The analysis of the data from 2002 showed that the risk marginally exceeded the WHO reference level, but when considered against other routes of transmission, the risk was very low. This suggests that, for bacterial pathogens, there is no need to upgrade the treatment works; rather, efforts should be concentrated on improved operational management of filtration and chlorination processes.

In this study, the risk assessment demonstrated the greater potential disease burden from water in the distribution system compared with water leaving the treatment works, which highlights the need to direct investment to improved operation and maintenance. It also showed that

increasing access to piped supplies is a more important public health objective than improvement in the piped supply because the potential disease burden from the use of alternative supplies was much higher than from piped water. Furthermore, the analysis shows that there was a greater potential disease burden from the recontamination of stored water in the home. The QMRAs were used solely to provide water suppliers with an understanding of the potential public health risks from the piped water supply, and the information they need to make decisions about appropriate levels of investment. As discussed below, QMRA was successful in achieving this goal, and it is this application that appears to be most promising in developing countries.

The QMRA shows that the risk was significantly increased within the distribution system. This result suggests that investment in the operation, maintenance and upgrading of the distribution system should be a priority. Tackling issues such as leakage control is likely to be essential in this process and is not unexpected. Clark *et al.* (1993) and Geldreich (1996) have shown that water quality problems are typically more associated with poor distribution systems than failures in treatment works in developing countries and that poor distribution systems have led to disease outbreaks in developed countries.

The *C. parvum* risk was higher than for *E. coli* at the treatment works, although, as noted above, these results are only provisional. Under normal operating conditions, Gaba 2 has a high risk of *C. parvum* breakthrough. It is difficult to characterise the level of risk from *C. parvum* in the water supply, particularly in comparison to other transmission routes; however, it is possible that a high level of risk will be posed by *C. parvum*. Host animal species are present in the catchment area of the Inner Murchison Bay and it is likely that contamination of the bay occurs from time to time. The water treatment system would not originally have been designed for *Cryptosporidium* removal and, therefore, may not be capable of removing the oocysts to a satisfactory degree. It would seem that investment in treatment works would be of benefit for *C. parvum* removal, although this would be best achieved through improving the coagulation-flocculation-settling and rapid sand filtration steps. The provision of ozonation, as an alternative disinfectant to chlorine, is likely to be prohibitively expensive.

The risks associated with rotavirus were also high. However, perhaps more than the other pathogens, the accuracy of the risk estimate is questionable, particularly because the role of drinking water in transmission may be much more limited in comparison with other transmission routes. Although WHO recommends rotavirus as the reference pathogen for viruses because of its virulence and widespread occurrence, future QMRAs could consider other waterborne viruses, such as hepatitis A & E viruses and coxsackievirus as the reference pathogen to calculate a disease burden.

Using risk assessment data for investment planning

The QMRA carried out here provides an indication of the present disease burden from each reference pathogen. Having established the disease risk from the water supply, the QMRA can be used to calculate performance targets for the water treatment and supply system if the disease burden from any one of the pathogens was to be reduced. For example, if the authorities in Uganda were to set a health target of 10^{-6} DALYs, in line with the WHO reference level of risk, the QMRA matrix can be used to calculate the required reduction in pathogen numbers through the treatment works by changing the final disease burden (DB) term in the methodology outlined in Table 7 to 10^{-6}, whilst retaining the same raw water quality, dose response and disease burden per case. For *E. coli* this would require an improvement to approximately 6 logs, 7 logs for *Cryptosporidium* and up to 8 logs for rotavirus.

Whilst it is possible to reach such levels of performance, it is questionable whether this would be cost-effective, given the low levels of access to piped water in the home, the use of alternative (and more contaminated) water sources and low sanitation coverage. Improvements in access to water supply, sanitation and hygiene could be expected to reduce the disease burden, as improvements in nutrition and boosting of immune systems would be expected to reduce the number of individuals developing more severe end-points. Furthermore, ongoing efforts to reduce HIV/AIDS prevalence would also be expected to significantly reduce the mortality burden. Therefore, the actual level of investment required to meet the WHO reference level of risk in the future may be significantly lower than at the present time.

The data on re-contamination of household water indicates that the disease burden estimates from recontamination was over one order of magnitude higher than that posed directly from the piped water, and the likelihood of diarrhoea two orders of magnitude higher. It should also be noted that the use of NWSC water was found to be associated with better quality of household water than other sources of water (Howard, unpublished observation). This assessment should be treated with some caution as some of the data from household water storage were taken from studies of other towns; however, there was no significant difference in average quality of the water supply from that of Kampala. It does, however, point to the need for improved water hygiene over improvements to the quality of the supply. Risks from recontamination could be greatly reduced by using household water treatment, as a further 4 to 5 log reduction in contamination can be expected using chlorine-based treatment (Sobsey 2002). This would lead to a final disease burden estimate of 1.23E-08.

The disease burden from protected springs is over one order of magnitude higher than the risk posed by the piped water supply, and for cases of diarrhoea it exceeds the risk from piped water by about 1.5 orders of magnitude. This is consistent with the results from other studies that have shown that the use of non-piped water sources was a significant risk factor for severe childhood diarrhoea in Uganda (Nasinyama *et al.* 2000). This assessment further emphasises the value in improving access to the current NWSC supply as a means of reducing health risks.

CONCLUSIONS

A simple quantitative microbial risk assessment has been successfully applied in a developing country and the data has been shown to be a useful tool in supporting investment planning and decision-making for promoting safer water supply. Further data are required to refine the estimates made, or at least to try to assess the degree to which this current risk assessment deviates from estimates based on pathogen data. In the context of water supplies in Uganda we have shown that QMRA is a valuable tool for a water supplier in understanding the potential public health risks associated with their supplies.

Within the Uganda setting, three key findings emerge of particular interest to policy makers and water safety managers.

(1) Water quality deterioration in the distribution system represents a far greater risk than treatment performance. This finding is similar to other studies from the developed and developing world. This implies that the main need for water safety improvement in Kampala is within distribution management rather than treatment plant upgrades.
(2) For bacterial pathogens, the protected springs and recontamination of water during household storage pose a greater risk to health than the water in the piped distribution system. This suggests that increasing access to piped water closer to people's homes and promotion of household treatment of water is important to promote better health. However, this should be balanced with the need to ensure better water safety management within the distribution system, as increasing numbers of users will result in a changing risk assessment result.
(3) At the water treatment works, the risks posed by *C. parvum* are higher than for pathogenic *E. coli*. This is not unexpected, as the works were not designed with *C. parvum* removal in mind. There is great reliance on chlorination to produce safe water, which, whilst effective against bacterial and (to a lesser extent) viral pathogens, will be ineffective for protozoa. Any upgrading for water quality improvements should therefore be based on improvement of *C. parvum* removal, primarily through the use of coagulation-flocculation-settling to improve removal efficiency in rapid sand filtration. In addition, attention to the reliability of existing treatment is a priority and this should be placed within the context of a water safety plan. Further work is also required to assess the level of *C. parvum* in the source waters before any such upgrade be considered.

ACKNOWLEDGEMENT

The authors wish to thank the Department for International Development, UK (DFID) and NWSC for their support in

funding the research under project R8023 that led to this paper. The views expressed in this paper are those of the authors and do not reflect the official position of DFID or NWSC. The authors would also like to acknowledge the support of colleagues in the laboratories at NWSC and Makerere University and the Environmental Health Departments in Kampala City Council in undertaking the water quality analyses.

REFERENCES

Anon 2002 *The Microbiology of Drinking Water (2002) – Part 4 – Methods for the Isolation and Enumeration of Coliform Bacteria and Escherichia coli (including E. coli O157:H7)*, Environment Agency, www.environment-agency.gov.uk.

Anon 2002 *The Microbiology of Drinking Water (2002) – Part 6 – Methods for the Isolation and Enumeration of Sulphite-Reducing Clostridia and Clostridium perfringens by membrane technique*, Environment Agency, www.environment-agency.gov.uk.

Ashbolt, N. J., Grabow, W. O. K. & Snozzi, M. 2001 Indicators of microbial water quality. In *Water Quality: Guidelines, Standards and Health* (ed. L. Fewtrell & J. Bartram), International Water Association, London, pp. 289–315.

Barrett, M. H., Johal, K., Howard, G., Pedley, S. & Nalubega, M. 2000 Sources of faecal contamination in shallow groundwater in Kampala. In *Groundwater: Past Achievements and Future Challenges: Proceedings of the XXX IAH congress on groundwater, Cape Town, South Africa*, (ed. Sililo, *et al.*), Balkema, Netherlands, pp. 691–696.

Borrego, J. J., Morinigo, M. A., de Vicente, A., Cornax, R. & Romero, P. 1987 Coliphages as an indicator of faecal pollution in water: its relationship with indicator and pathogenic microorganisms. *Wat. Res.*, **21**, 1473–1480.

Clark, R. M., Goodrich, J. A. & Wymer, L. J. 1993 Effect of the distribution system on drinking-water quality. *J. Wat. Suppl.: Res. & Technol. – AQUA*, **42**(1), 30–38.

Davison, A., Howard, G., Stevens, M., Callan, P., Fewtrell, L., Deere, D. & Bartram, J. 2005 *Water Safety Plans*. World Health Organization, Geneva.

Deere, D., Stevens, M., Davison, A., Helm, G. & Dufour, A. 2001 Management strategies. In *Water Quality: Guidelines, Standards and Health* (L. Fewtrell & J. Bartram eds), IWA Publishing, London, pp. 257–288.

Esrey, S. A., Feacham, R. G. & Hughes, J. M. 1985 Interventions for the control of diarrhoeal diseases among young children: improving water supplies and excreta disposal facilities. *Bull. Wld. Health Orgn.* **63**(4), 757–772.

Esrey, S. A., Potash, J. B., Roberts, L. & Shiff, C. 1991 Effects of improved water supply and sanitation on ascariasis, diarrhoea, dracunculiasis, hookworm infection, schistosomiasis, and trachoma. *Bull. Wld Health Orgn*, **69**(5), 609–621.

Fewtrell, L. & Bartram, J. (eds) 2001 *Water Quality: Guidelines, Standards and Health*. IWA Publishing, London.

Geldreich, E. E. 1996 *Microbiol Quality of Water Supply in Distribution Systems*. Lewis Publishers, New York.

Grabow, W. O. K. 2001 Bacteriophages: update on application as models for viruses in water. *Water SA* **27**(2), 251–268.

Haas, C. N. & Eisenberg, J. N. S. 2001 Risk assessment. In *Water Quality: Guidelines, Standards and Health* (L. Fewtrell & J. Bartram eds), IWA Publishing, London, pp. 161–183.

Haas, C. N., Rose, J. B. & Gerba, C. P. 1999 *Quantitative Microbial Risk Assessment*. John Wiley, New York.

Haas, C. N., Thayer-Madabusi, A., Rose, J. P. & Gerba, C. P. 2000 Development of a dose-response relationship for *Escherichia coli* O157:H7. *Int. J. Food Microbiol.* **1748**, 153–159.

Harries, A. 1991 Some clinical aspects of HIV infection. *Curr. Opin. Infect. Dis.* **14**(15), 567–571.

Havelaar, A. H. & Melse, J. M. 2003 *Quantifying Public Health Risks in the WHO Guidelines for Drinking-Water Quality: A Burden of Disease Approach. Report 734301022/2003*. RIVM, Bilthoven, Netherlands.

Hazen, T. C. & Torranos, G. A. 1990 Tropical source water. In *Drinking Water Microbiology: Progress and Recent Developments* (ed. G. A. McFeters), Springer-Verlag, New York. pp. 32–53.

Health Canada 2000 *Waterborne Outbreak of Gastroenteritis Associated with a Contaminated Municipal Water Supply, Walkerton, Ontario, May–June 2000* **26**(20) Canada Communicable Disease Report, 15 October 2000.

Howard, G. 2001 Challenges in increasing access to safe water in urban Uganda: economic, social and technical issues. In *Microbial Pathogens and Disinfection By-products in Drinking Water: Health Effects and Management of Risks* (ed. G. F. Cruan, F. S. Hauchman & D. E. Robinson), ILSI Press, Washington, DC, pp. 483–499.

Howard, G. & Bartram, J. 2003 *Domestic Water Quantity, Service Level and Health*. World Health Organization, Sustainable Development and Healthy Environments, Geneva, Switzerland, SDE/WSH/03.02.

Howard, G. & Bartram, J. 2005 Effective water supply surveillance in urban areas of developing countries. *J. Wat. & Health.* **3**(1), 31–43.

Howard, G. & Luyima, P. G. 1999 *Report on Water Supply Surveillance in Ten Selected Towns in Uganda*. Government of Uganda, Kampala, www.lboro.ac.uk/watermark.

Howard, G., Teuton, J., Luyima, P. & Odongo, R. 2002 Water usage patterns in low-income urban communities in Uganda: implications for surveillance Int. *J. Environ. Health Res.* **12**(1), 63–73.

Howard, G., Pedley, S., Barrett, M., Nalubega, M. & Johal, K. 2003 Risk factors contributing to microbiological contamination of shallow groundwater in Kampala, Uganda. *Wat. Res.* **37**, 3421–3429.

Hunter, P. R. 2003 Drinking water and diarrhoeal disease due to *Escherichia coli*.. *J. Wat. & Health* **1**(2), 65–72.

Kelley, P., Sri Babooi, K., Nduban, P., Nchito, M., Okeowo, N. P., Luo, N. P., Feldman, R. & Farthing, M. J. G. 1997 *Cryptosporidiosis* in adults in Lusaka, Zambia and its relationship to oocyst contamination of drinking water. *J. Infect. Dis.* **176**(4), 1120–1123.

Kotloff, K. L., Winickoff, J. P., Ivanoff, B., Clemens, J. D., Swerdlow, D. L., Sansonetti, P. J., Adak, G. K. & Levine, M. M. 1999 Global burden of *Shigella* infections: implications for vaccine development and implementation of control strategies. *Bull. Wld Health Orgn* **77**(8), 651–666.

Mølbak, K., Andersen, M., Aaby, P., Højlyng, N., Jakobsen, M., Sodemann, M. & da Silva, A. P. 1997 *Cryptosporidium* infection in infancy as a cause of malnutrition: a community study from Guinea-Bissau, west Africa. *Am. J. Clin. Nutr.* **65**(91), 149–152.

Murray, C. J. L. & Lopez, A. D. 1996 *The Global Burden of Disease*. World Health Organization, Geneva.

Mwachari, C., Batchelor, B. I., Paul, J., Waiyaki, P. G. & Gilks, C. F. 1998 Chronic diarrhoea among HIV-infected adult patients in Nairobi. *Kenya. J. Infect.* **37**(1), 48–63.

Nasinyama, G. W., McEwen, S. A., Wilson, J. B., Waltner-Towers, D., Gyles, C. L. & Opuda-Asibo, J. 2000 Risk factors for acute diarrhoea among inhabitants of Kampala District, Uganda. *SA Med. J.* **90**(9), 891–898.

RCPEH 2004 *Oxfam-DelAgua Kit Users Manual*. University of Surrey, Guildford, UK, www.rcpeh.com.

Regli, S., Rose, J. B., Haas, C. N. & Gerba, C. P. 1991 Modeling the risk from giardia and viruses in drinking water. *J. Am. Wat. Wks Assoc.* **83**(11), 76–84.

Sobsey, M. 2002 *Managing Water in the Home: Accelerated Health Gains from Improved Water Supply*. World Health Organization, Geneva.

Sodemann, M., Jakobsen, M. S., Mølbak, K., Martins, C. & Aaby, P. 1999 Episode-specific risk factors for progression of acute diarrhoea to persistent diarrhoea in west African children. *Trans. Roy. Soc. Trop. Med. Hyg.* **93**(1), 65–68.

Strachan, N. J. C., Fenlon, D. R. & Ogden, I. D. 2000 Modelling the infection pathway and infection in an environmental outbreak of *Escherichia coli* O157. *FEMS Microbiol. Lett.* **203**, 69–73.

Tarimo, D. S., Killewo, J. Z., Mingas, J. N. & Msamanga, G. I. 1996 Prevalence of intestinal parasites in adult patients with enteropathic AIDS in north-eastern Tanzania. *E. Afr. Med. J.* **73**(6), 397–399.

Teunis, P. F. M., Madema, G. J., Kruidenier, L. & Havelaar, A. H. 1997 Assessment of the risk of infection by *Cryptosporidium* or *Giardia* in drinking water from a surface water source. *Wat. Res.* **31**(6), 1333–1346.

Teunis, P., Takumi, K. & Shinagawa, K. 2004 Dose response or infection by *Escherichia coli* O157:H7 from outbreak data. *Risk Anal.* **24**(2), 401–407.

UNAIDS 2003, www.unaids.org.

WHO 1996 *Guidelines for Drinking-Water Quality: Volume 2 Health and Other Supporting Criteria*, 2nd edn. World Health Organization, Geneva.

WHO 2002 *World Health Report: Reducing Risks and Promoting Healthy Life*. World Health Organization, Geneva.

WHO 2004 *Guidelines for Drinking-Water Quality: Volume 1 Recommendations*, 3rd edn. World Health Organization, Geneva.

Available online January 2006