MICRORISK

Microbiological risk assessment: a scientific basis for managing drinking water safety from source to tap

Final report

Quantitative Microbial Risk Assessment

in the Water Safety Plan

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Quantitative Microbial Risk Assessment in the Water Safety Plan

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Preface

Safe drinking water

Providing water that is safe to drink is the primary objective of water supply. History has learnt that drinking water that contains pathogenic micro-organisms can have a major impact on health. Over the last century, multiple barriers have been installed to block the transmission of infectious diseases through drinking water.

Guidelines and standards for drinking water quality, such as the EU Drinking Water Directive, state that pathogens should not be present in drinking water in levels that may cause adverse health effects in consumers. This has been translated into operational standards for faecal indicator bacteria, *E. coli* and enterococci. Since the early 1900's, the concept of faecal indicator bacteria (if no indicators of faecal contamination are present, no faecal pathogens are present) is being used and faecal indicators have been adopted in WHO's *Guidelines for Drinking Water Quality,* the EU Drinking Water Directive and all national drinking water quality standards. Faecal indicators, esp. *E. coli*, are today undoubtedly the most commonly used parameters for monitoring drinking water quality.

The use of indicator bacteria (esp. *E. coli*) has led to significant improvements in the safety of drinking water world-wide and has resulted in a high level of drinking water quality and supply infrastructure in European countries. From the consumer's perception, this approach has led to high confidence in drinking water safety.

The new risk-based approach

Over the years, several shortcomings of the indicator concept have been identified. The most important shortcomings are:

- Waterborne disease outbreaks have occurred through water supply systems that met the standard for absence of *E. coli* in 100 ml*,* particularly outbreaks of disease caused by viruses or protozoa.
- End-product testing of a very small fraction of the total volume of water and with microbiological methods that take at least one day to produce a result, amounts to a "*too little, too late*" approach. If water quality monitoring provides evidence of microbial contamination, in most cases, the water has already been distributed and consumed.
- End-product testing is not providing safety in itself; it is a verification that all systems and measures that are installed to protect drinking water are working properly.

Primary reliance on end-product testing is presently considered not to be sufficient to provide confidence in good and safe drinking water. It is reactive rather than preventive and does not allow the water supplier to demonstrate due diligence to the regulator and consumer. During the last decade, the use of a risk-based approach to pathogens in drinking water has been promoted by many researchers and institutions. WHO has played a key role in the integration of risk assessment and risk management into the Water Safety Plan, analogous to the HACCP system used in the food industry. Several documents have been and are being prepared to aid the implementation of the risk-

framework in the drinking water area (see http://www.who.int/water_santitation_health/en/).
The Water Safety Plan has been the subject of the Water Safety Conference in Berlin in April 2003 and the risk-based approach is also presented and discussed at the Drinking Water Seminar of the EU in October 2003. Both meetings showed a wide-spread endorsement of this new approach by all stakeholders in the drinking water arena.

Where does this document fit in?
This document describes how Quantitative Microbial Risk Assessment (QMRA) can be used in the Water Safety Plan. It highlights what QMRA is and how QMRA can provide important information to guide the HACCP-based risk management process. Every water supplier who is preparing a Water Safety Plan is faced with the basic question: "Is my system safe (enough)?". QMRA can provide the answer in a manner that is science-based and transparent. QMRA is also a tool to prioritise risks in a science-based, objective manner, to set performance targets for operations and design monitoring programs that are adequate for demonstrating that the water supply system meets the health targets.

MicroRisk
MicroRisk: scientific basis for managing drinking water safety from source to tap is a research project that is co-financed by the European Commission (Contract EVK1-CT-2002-00123) and the partners and their financers in this joint research effort: Kiwa Water Research (Co-ordinator, Netherlands), Institute of Infectious Disease Control (Sweden), Veolia Environnement - Anjou Recherche (France), Veolia Water Partnership (UK), WRc-NSF (UK), Bonn University (Germany), Suez Environnement - CIRSEE (France), University of East Anglia (UK), University of Delft (Netherlands), University of New South Wales (Australia) and Water Technology Centre (Germany).

What does this document contain?
This book is largely based on the research in the project MicroRisk. The objective of MicroRisk was to evaluate Quantitative Microbial Risk Assessment as a scientific basis and tool to assess the microbial safety of drinking water supplies.

After the framework for QMRA and the interaction with risk management was developed and verified with the stakeholders, 12 catchment-to-tap systems (CTS) were selected from different geographical and climatological areas of the EU (and Australia) as pilot sites for QMRA. The CTS were all medium to large water supply systems and all but one were surface water supplies, including a bank filtration site and artificial recharge site using pre-treated surface water infiltration (table P.1). For each of these CTSs quantitative information relevant to the microbiological safety of drinking water was collected from these systems, both historical data and new data that were collected within the scope of MicroRisk.
It is emphasised that within the scope of MicroRisk the CTSs provided information to evaluate the value, strengths and weaknesses of QMRA given the current state-of-the-art and available data, not to evaluate the CTSs against a health target.

Table P.1. Catchment-to-tap systems used for data collection and evaluation of QMRA.

CTS	Source water	Treatment	Distribution	
			Cl_2	Pop. size
1	River	Pre-O_3 (Cl_2 in summer) - Coa - Sed - RF - O_3 - GAC - super/deCl_2	Yes	224,000
2	Reservoir	AR - RF - O_3 - GAC – SSF	No	440,000
3	River	Pre -O_3 - Coa - Sed - RF – O_3 - GAC - Cl_2 (3 systems)	Yes	34,000
4	River	Coa – Sed - O_3 – GAC - Cl_2	Yes	18,000
5	River with controlled intake	Coa – Sed - O_3 or Cl_2 – GAC - Cl_2 (2 systems)	Yes	571,600
6	Reservoir	Reservoir - Cl_2 (summer) - Coa - Sed - GAC - Cl_2 (2 systems)	Yes	
7	River	Bank filtration – SSF - O_3 – GAC	Yes	120,000
8	Reservoirs	Coa – Sed - RF - GAC - Cl_2	Yes	50,000
9	Reservoir (+ river)	RSF - O_3 - GAC - SSF	No	440,000
10	Mountain reservoirs	Pre-Cl_2 - Coa - Sed – RF – O_3 - Cl_2	Yes	47,600
11	Protected reservoirs	DF - RF - Cl_2/ClO_2	Yes	300,000
12	Protected groundwater	RF - Cl_2	Yes	24,300

Coa =coagulation; Sed = sedimentation; RF is rapid filtration; O_3 is ozonation; Cl_2 is chlorination; ClO_2 is chlorine dioxide; GAC is granular activated carbon filtration; AR is artificial recharge; SSF is slow sand filtration.

MicroRisk was a scientific study, but we have decided to publish this book in the format of a guidance document rather than a scientific report (the science in MicroRisk is published in peer-reviewed literature). First, waterborne intestinal illness through drinking water in Europe is reviewed, in particular waterborne outbreaks and what caused them, the problem of small systems and the (limited) ability of health surveillance systems to pick up waterborne illness. Then the document introduces QMRA and its place and value for risk management of pathogens in drinking water. Then several chapters give guidance on how to collect information about:

- pathogens in source waters;
- removal of micro-organisms by water treatment processes;
- contamination during distribution;
- consumption of (cold) tap water;

with a particular emphasis on both normal performance and hazardous events in source water (peak rainfall events), treatment (moments of limited removal) and distribution (contamination events).

In the last two chapters the theoretical basis for data analysis (using statistics and sensitivity analysis to incorporate variation and uncertainty) is given and applied with the data collected by the CTSs. The value of QMRA for the risk manager in a water utility is illustrated with examples from the CTSs.

Enteric pathogens

MicroRisk has focussed on enteric pathogens that may be present in source waters and present a challenge to treatment systems. Already at the starting workshop it was decided to aim at *index pathogens*. Index pathogens represent a group of similar pathogens. An index pathogen can represent a control challenge (such as *Cryptosporidium* as challenge to chemical disinfection processes). If this is the case, controlling this index pathogen implicates that related pathogens are also controlled. Ideally, a small suite of index pathogens is selected based on the relevant control challenges. This would avoid the need to assay for a whole range of known waterborne pathogens. Index pathogens are used to obtain quantitative information for QMRA.

Which faecal pathogens?
The current faecal indicator bacteria are potentially index organisms for pathogenic bacteria like enterohemorhagic *Escherichia coli* (EHEC), *Shigella* and *Salmonella*. Because the severity of the symptoms, EHEC was determined a suitable index, and also *Campylobacter* is a pathogen of major interest.

Parasitic protozoa are also of interest because of their resistance to chemical disinfection: *Cryptosporidium* and *Giardia* can be analysed in parallel and further typing and taxonomic information will be available. Dose response models are in use for these micro-organisms as well as data on frequency of occurrence in source as well as reduction and data on outbreaks are available.

Viruses are most relevant to include in QMRA. They pose a challenge to filtration systems due to their very small size, including membrane filtration and soil passage, artificial recharge and bank filtration. Some viruses pose specific challenges to water utilities (such as adenoviruses for UV systems). Several dose-response relations are available for waterborne viruses and emerging for others (like Noroviruses). Information about culturable enteroviruses is available; data on the transferability of PCR-data to culturable/infectious viruses is limited, but emerging. There is a need for more quantitative data in source waters.

Growth of (opportunistic) pathogens such as *Legionella, Mycobacterium, Aeromonas,* and *Pseudomonas* in the distribution system and domestic installations was not part of MicroRisk. Not because they are no significant health risk through drinking water, but because the risk assessment and risk management of this class of pathogens is markedly different from risk assessment and management of enteric pathogens.

Target audience
The primary target audience for this document is the professionals involved in preparing a Water Safety Plan (generally the water supplier). They will get more detail on how a *System Assessment* can be done quantitatively and how such an assessment may help to make educated, science-based risk management decisions.

The MICRORISK-team

Glossary

Barrier: see Control Measure

Control Measure: Any action or activity that can be used to prevent or eliminate a hazard or reduce it to an acceptable level.

Control Point (CP): A step in the water supply at which contamination is prevented reduced or eliminated or minimised and which, if collectively in compliance, would ensure that water quality targets are met. CP's are points in the water supply where it is possible to set operational and/or critical limits, monitor those limits and take corrective action in response to a detected deviation before the water becomes unsafe. Often these points are control measures that are specifically designed to control a hazard.

Corrective Action: Control measure to be taken when monitoring of a control point indicates a loss of control.

Critical Limits: A criterion which measures performance of the control point to ensure that the control point will deliver water of a quality consistent that meets the water quality targets. Exceeding the Critical Limit implies that the Control point is no longer in compliance with the Water Safety Plan and there is an increased risk of water quality failing to meet the Health Target.

Dose-response assessment: The determination of the relationship between the magnitude of exposure (dose) to a microbiological agent and the severity and/or frequency of the associated adverse health effects (response).

Exposure: Concentration or amount of an infectious micro-organism that reaches the target population, or organism usually expressed in numerical terms of substance, concentration, duration and frequency.

Exposure assessment: Qualitative and/or quantitative evaluation of the likely intake of microbial hazard via all relevant sources or a specific source.

HACCP: Hazard Analysis Critical Control Point. A system that identifies evaluates and controls hazards that are significant for water safety.

Hazard: A biological agent with the potential to cause an adverse health effect.

Hazard identification: The identification of microbiological biological agents capable of causing adverse health effects and which may be present in water.

Hazardous event: An event that may lead to the presence of a hazard in drinking water.

Health effects: Changes in morphology, physiology growth, development or life span of an organism, which results in impairment of functional capacity or impairment of capacity to compensate for additional stress or increase in susceptibility to the harmful effects or other environmental influences.

Infection: Colonisation by a micro-organism.

Infectious disease: Colonisation by a pathogenic micro-organism leading to overt symptoms of disease.

Monitoring: The act of conducting a planned series of observations or measurements of operational and/or critical limits to assess whether a control point is under control.

Pathogen: A micro-organism **c**apable of causing disease.

QMRA: Quantitative Microbial Risk Assessment.

Risk: The likelihood of occurrence of an adverse health effect consequent to a hazard in drinking water.

Risk characterisation: The qualitative and quantitative estimation, including attendant uncertainties of the probability of occurrence and severity of known or potential adverse health effects in a given population based on hazard identification, hazard characterisation and exposure assessment.

Uncertainty: Imprecision and inaccuracy of an assessment or monitoring method.

Validation: Obtaining evidence that the elements of the WSP are effective.

Variability: Intrinsic heterogeneity in a process or parameter.

Verification: The application of methods, procedures, tests and other evaluations, in addition to monitoring to determine the compliance with the water quality targets.

Water quality targets:: The maximum levels of microbiological hazards in drinking water, which are considered acceptable for human consumption, preferably in a quantitative and verifiable manner as described by official state authorities.

Water Safety Plan (WSP): A management plan developed to address all aspects of water supply that are under the direct control of the water supplier focused on the control of water production, treatment and distribution to deliver drinking water.

[1]All terms are based on definitions of Codex Alimentarius and Water Safety Plans [Davison *et al.,* 2002], as well as definitions given by Benford [2001] or by Haas & Eisenberg [2001], adapted for microbial risk analysis of water.

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In a workshop in February 2003 (at Kiwa Water Research, Nieuwegein, The Netherlands), the framework for MicroRisk has been presented to and reviewed by an audience of international experts and stakeholders. A report of this workshop is available from the MicroRisk website (www.microrisk.org). The comments of this audience and the suggestions for improvement made at the workshop have been used to shape MicroRisk into its present format. The MicroRisk partners are gratefully acknowledging the contribution of all participants of the workshop in helping to improve the framework for the MicroRisk project, especially those that have presented their view and comments on the framework document: J. Bartram (WHO), P. Hecq (EU), M. Waite (DWI), C. Castell-Exner (EUREAU), J.F. Loret (Suez), P. Hartemann (INSERM), N. Ashbolt (UNSW), P. Gale (WRc), T.A. Stenström (SMI) and P. Hunter (UEA).

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1 Intestinal illness through drinking water in Europe

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1.1 INTRODUCTION

Ingestion of water has been demonstrated as a vehicle for multiple enteropathogens of bacterial, protozoan and viral origin [Hunter, 1997; Leclerc *et al.*, 2002]. Once ingested, enteropathogens typically cause gastrointestinal symptoms in the host including nausea, vomiting, and diarrhoea. Selective enteropathogens can also give rise to a number of more serious health outcomes including Haemolytic Uraemic Syndrome (HUS), Guillain-Barre syndrome, hepatitis, meningitis, dysentery, and death. Diarrhoeal disease is recognised by the World Health Organisation (WHO) as a major cause of infant mortality in developing countries, comprising around 15% of total child deaths under five [WHO, 2005]. Furthermore, it is estimated that by providing access to in-house regulated piped water and sewerage connection with partial treatment of waste waters, an average global reduction of 69% could be achieved in the number of episodes of diarrhoea [Hutton and Haller, 2004].

Yet the burden of waterborne disease is not restricted to low-middle income countries. Waterborne diseases still present a challenge to the more affluent nations. Immunocompromised individuals, the elderly, pregnant women and the very young are at greater risk of serious illness and mortality from water and foodborne enteric microorganisms as a much smaller infective dose can cause illness [Gerba *et al.*, 1996]. The ageing population and increased use of immunosuppressive drugs in industrialised nations could lead to a greater number of individuals at increased risk of disease.

Our heavy dependence on drinking water and the severe socioeconomic burden associated with waterborne disease emphasise the need to further our understanding of this topic. In light of this, the purpose of this chapter is to depict the scale of waterborne disease through analysis of endemic disease, outbreaks, and public health surveillance strategies.

1.2 ENDEMIC WATERBORNE DISEASE

A number of epidemiological tools have been used to investigate possible associations between drinking water and disease. Of these, randomised controlled trials (RCTs) represent the most robust methodological approach. Typically, households are randomly assigned to different water treatment groups.

Two studies conducted in Canada have looked prospectively at the incidence of gastrointestinal illness due to the consumption of drinking water from sewage contaminated surface waters meeting current (as defined at the time of study) water quality criteria [Payment *et al*., 1991, 1997]. In the first of these studies, people in households randomised to receive domestic reverse osmosis (RO) water filters were found to have a lower annual incidence of gastrointestinal illness (0.50 per

person/year) in comparison to tap water drinkers (0.76, $p<0.01$); estimating that 35% of the gastrointestinal illness reported by tap water drinkers was water-related. In a successive, larger trial, it was estimated that tap water was accountable for between 14-40% of gastrointestinal illness.

Although both Canadian studies used randomisation, participants were not blinded to the type of water treatment received which can improve the validity of results. Hellard *et al*. [2001] conducted a double-blinded RCT in Melbourne, Australia. The drinking water in the study area was reported to be of high quality, derived from a highly protected source treated with chlorination only. Six hundred households received either real or sham RO water treatment units (WTUs). Over a period of 68 weeks participants completed a health diary reporting gastrointestinal illness symptoms. The study found 0.80 highly credible gastroenteritis (HCG) cases per person/year and the ratio of HCG episode rates for families with real vs sham WTUs was 0.99 (95% CI: 0.85, 1.15, $p=0.85$), indicating that the RO-filters did not significantly reduce the HCGI incidence.

In the US, Colford *et al*. [2005] conducted a triple blinded RCT cross-over intervention study. The drinking water in this study area was derived from a challenged source treated with conventional chlorination and filtration methods to conform to all current US regulatory standards. Participants received either a sham or real treatment device for six months before switching to the opposite device for a further six months. The active device contained a 1 μm absolute ceramic filter and used UV-light. A total of 2366 HCG episodes were recorded for the 1296 participants over a period of 12 months (1.83 cases/person/year). The relative rate estimate of HCG (sham vs real device) was 0.98 (95% CI: 0.86, 1.10), no reduction in gastrointestinal illness was detected following use of the real treatment device.

Further studies from the Americas have shown an association between sporadic cases of illness and use of unfiltered municipal or non-municipal water [Birkhead and Vogt, 1989] and variation in drinking water turbidity [Morris *et al*. [1996], Schwartz *et al*. [2000]).

There have been no randomised controlled trials in Europe and few other studies of endemic waterborne disease. In France, Zmirou and colleagues reported two of the first prospective studies of endemic waterborne disease. In the first study [Zmirou *et al*. 1987] they demonstrated that the risk of childhood gastroenteritis was greater in alpine villages where the water did not satisfy drinking water standards (RR=1.68 95% CI 1.50-1.88). In a follow-up study they went on to show that an excess risk persisted in poor faecally contaminated sources even after chlorination [Zmirou *et al*. 1995]. Also from France, Beaudeau *et al*. [1999] demonstrated a correlation between drinking water turbidity and sales of anti-diarrhoeal medication in Le Havre.

In the UK, an association between self-reported diarrhoea and pressure loss (Adjusted OR 12.5, 95% CI 3.5-44.7) has been demonstrated [Hunter *et al*., 2005]. Whilst in Sweden, Nygard *et al*. [2004] showed a correlation between risk of campylobacteriosis and length of pipe run from the treatment works to the home.

Nevertheless, the level of endemic disease due to public drinking water systems remains difficult to quantify. The latter two studies suggest that there may be a risk of illness due to contamination of water in distribution. For further information we need look to outbreaks of waterborne disease.

1.3 PUBLIC SUPPLY OUTBREAKS IN THE EU

1.3.1 Introduction

Sporadic cases purportedly represent a greater proportion of waterborne disease than cases related to outbreaks [Nichols, 2003]. In addition, outbreaks are notoriously difficult to detect [Hunter *et al*., 2001]. Despite such paucity, much of what we know about the burden of disease in affluent nations has been generated through outbreak documentation.

What is evident from outbreaks implicating public supplies is that harmful pathogens have the potential to reach a large body of consumers resulting in substantial economic and health-related costs, which is shown by the April 1993 *Cryptosporidium* outbreak in Milwaukee [Mackenzie *et al*. ,1994]. As a result of a filtration failure at a public water supply it was estimated that around 403,000 people suffered illness, 4,400 people were hospitalised and 100 people died, though these figures have been disputed by others [Hunter and Syed 2001]. The total cost of outbreak-associated illness in the Milwaukee outbreak was estimated to be US$96.2 million [Corso *et al.*, 2003]. Furthermore, in a review of 25 studies on the economic burden associated with common water-related diseases [Bartram *et al*., 2002: 78], the cost of an outbreak reflected as a proportion of gross domestic product per person for 7 enteric outbreaks of waterborne disease ranged from 0.002 to 0.230. Whilst costs such as health care expenses, direct and indirect productivity loss, and bottled water purchase are incorporated into these estimates, the absence of macroeconomic costs (for example, reduced consumer confidence and tourism decline) means that the financial burden is underestimated.

Reviews have further discussed the characteristics of waterborne outbreaks inclusive of private supplies [Said *et al*., 2003], recreational water and non-enteric disease in Europe [WHO, 1999] and much investigation has already been accomplished on affluent nations [Hrudey, 2004] including Canada [Schuster *et al*., 2005] and the US [Craun *et al.*, 2002, Blackburn *et al*., 2004, Lee *et al*., 2002]. This section reviews outbreaks featuring enteric waterborne pathogens (*E.coli*, *Campylobacter*, *Cryptosporidium*, *Giardia*, *Shigella*, *Salmonella*, *Norovirus* and gastroenteritis of unknown aetiology) related to drinking water derived from public supplies in the European Union (EU); thus distinguishing them from non-enteric disease and recreational and private water source outbreaks.

1.3.2 Documented Public Water Supply Outbreaks

Electronic searching of databases (such as, Medline and Embase), and personal communication with members of Enter-net (an international surveillance network), led to the detection of outbreaks from scientific literature, outbreak reports, and other published materials.

Reported outbreaks were omitted if the water source (public or private), year, or country of the outbreak was not reported, or if published material documenting the outbreak was not available. These factors were considered important for the purpose of differentiating between outbreaks to avoid duplication. For example, 30 additional Swedish outbreaks were identified via personal communication with Torbjorn Lindberg. Twenty-five of these outbreaks implicated groundwater supplies and five surface water supplies. In these outbreaks, the aetiological agent involved was often

unknown (77%), in 20% of the outbreaks a viral agent was implicated, and in 3% *Campylobacter* was isolated from patients. Approximately 5,097 people suffered illness and over 44,575 were potentially exposed to the implicated supply. However, the data from these outbreaks was not incorporated as it was not possible to differentiate between small supplies which are part of a commercial/public activity and public drinking water supplies. Similarly, Bartram *et al*. [2002: 113] document that 55 of 154 European outbreaks were associated with networked public supplies between the years 1986 to 1996; these outbreaks were omitted here as they were not differentiable.

A total of 86 enteric disease outbreaks associated with EU public drinking water supplies for the years 1990 to 2004 were detected. Outbreaks were identified in 10 of the 25 countries of the EU. To facilitate synopsis of these outbreaks, extensive data extraction was performed of population, environmental, epidemiological, microbiological, and water supply characteristics. When interpreting the aggregated data it is important to be cautionary and to consider that there are many differences between countries (including drinking water source, water treatment processes, and surveillance practice) which may influence the characteristics shown.

1.3.2.1 Month and Year of Outbreak Onset

Figure 1.1 illustrates the number of outbreaks and cases by month of onset for the years 1990 to 2004. For 19% of outbreaks the month of onset was April. This seasonal trend is consistent with evidence of human, cattle and sheep incidents of cryptosporidiosis [Nichols, 2003] and could therefore be due to contaminated surface water ingress following heavy rainfall. Case numbers reflect the maximum number of cases of illness deemed attributable to the outbreak as calculated by the authors. Case numbers tend to follow the seasonal trend for outbreaks, however, for 15% of cases the month of outbreak onset was January. This figure is due to one gastroenteritis outbreak in which it was estimated that 30% of the population were affected [Chover *et al.*, 1995].

Figure 1.1: Number of Outbreaks and Cases by Month of the Year (n=84)

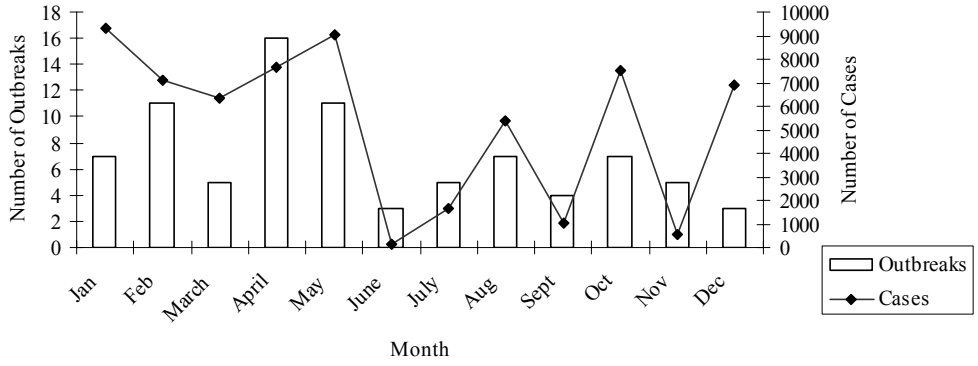
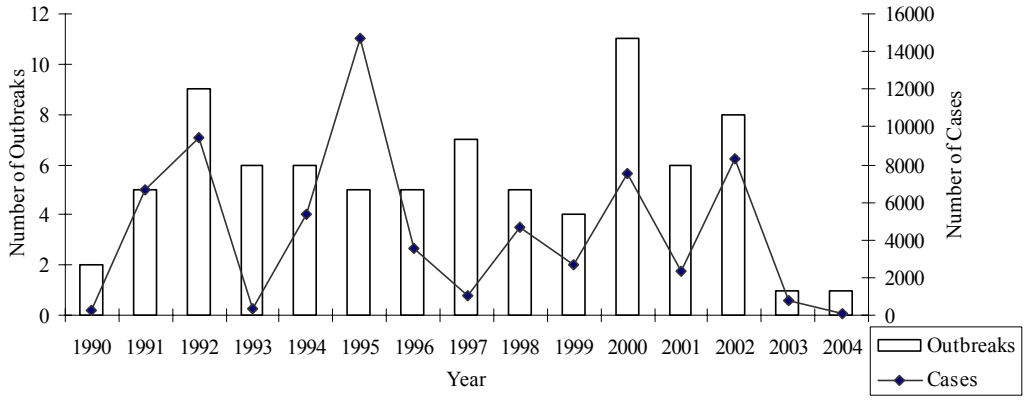


Figure 1.2 depicts the number of outbreaks and cases by year of outbreak onset. Five outbreaks were not included in this graph because they occurred in either the year 1998 or 1999 and one outbreak was not included because it did not report case numbers. The greatest number of outbreaks occurred in the year 2000 (13%) and the

least in 2003 and 2004; the decline possibly attributable to the time lag incurred through publication. The small number of cases attributed to the years 1993 and 1997 is due to the reporting of just laboratory positive cases (no population estimates or study cases) in 100% and 57% of the outbreaks respectively.

Figure 1.2: Number of Outbreaks and Cases by Year (outbreaks n=81; cases n=80)



1.3.2.2 Population, Pathogens and Water Supply

As can be seen from Table 1.1, it is possible for a large number of people (up to 1.5 million) to be in receipt of a supply implicated in a contamination event which results in high health costs.

Table 1.1: Number Receiving Supply, Hospitalisations, Fatalities and Cases Reported.

	Population Supplied (n=55)	Hospitalisations (n=32)	Fatalities (n=12)	Cases* (n=85)
Total	7,751,889	341	1	72546
Minimum	95	0	0	3
Maximum	1,500,000	91	1	10000
Mean	140,943	11	-	853
Std deviation	320353	18	-	1857
Median	7,500	5	-	150

Where n=the number of outbreaks reporting this factor.
* The maximum number of cases estimated by authors to be associated with the outbreak.

The number of outbreaks and cases associated with implicated pathogen and source of supply, for each of the ten EU countries in which outbreaks were detected can be found in Table 1.2. Most of the outbreaks were identified in England (34%), followed by Finland (14%), France (8%) and Sweden (8%). The most predominant agent isolated in the outbreaks was *Cryptosporidium* (32%) and the majority of these *Cryptosporidium* outbreaks occurred in England (61%). The bulk of the

Campylobacter and *Norovirus* outbreaks (82%) were identified in the Nordic countries, Finland and Sweden. No pathogen was isolated in 12 outbreaks and in five outbreaks a number of pathogens were involved. A further 4 outbreaks involved more than one pathogen; however, these additional outbreaks were classified elsewhere. In two of these four outbreaks, 40% or more of the cases were attributable to just one pathogen and were therefore classified under the predominant pathogens (*Cryptosporidium* and *Giardia*). Two of the 4 outbreaks were classified as 'gastroenteritis'; the number of confirmed cases from one outbreak involving multiple enteropathogens was not differentiable between pathogens and in one large outbreak (~9000 cases) only bacterial analysis was performed yielding relatively few positive results (~5 laboratory confirmed).

Although the greatest number of outbreaks implicated *Cryptosporidium*, most cases were associated with outbreaks of undetermined aetiology (gastroenteritis). In fact *Giardia* and *Cryptosporidium* had the lowest mean number of cases per outbreak (116 (std dev: 153) and 177 (std dev: 133) respectively). Viral outbreaks and *Campylobacter* had the highest mean number of cases per outbreak (1545 (std dev: 1623) and 1802 (std dev: 2140) respectively).

Overall, an equal number of surface water and groundwater supplies were implicated in the outbreaks. Sixteen outbreaks did not report the source of the water supply and 6 outbreaks had a mixed surface water and groundwater supply. The majority of groundwater outbreaks occurred in Finland (31%) and the majority of surface water outbreaks occurred in England (44%). All outbreaks in Scotland and Northern Ireland involved surface water supplies, the majority of outbreaks in Finland (83%) and France (71%) involved groundwater supplies, and a large number of outbreaks in England involved surface water supplies (48%). Groundwater supply outbreaks reported a greater number of cases of illness (60%) than surface water supplies (32%). The country-specific trends for England, France, and Finland reported here tend to reflect the predominant source of supply utilised for drinking water (as reported by Bartram *et al.,* 2002: 87).

Table 1.2: Number of Outbreaks by Country, Pathogen and Water Supply, and Maximum Cases by Pathogen and Water Supply.

Country	No. Outbreaks	Pathogen Isolated in Cases								Water Supply			
		Bacterial		**Protozoal**		**Viral**		Mixed Pathogen	Gastroenteritis	Ground-water	Surface Water	Mixed	Not Reported
		Campylobacter	*Shigella*	*Cryptosporidium*	*Giardia*	*Norovirus*	Viral (undete-rmined)						
Finland	12	4	-	-	-	6	1	-	1	10	2	-	-
France	7	-	-	2	-	-	-	3	2	5	-	-	2
Germany	2	-	-	-	1	1	-	-	-	1	-	-	1
Greece	3	-	2	-	1	-	-	-	-	2	1	-	-
Italy	1	-	-	-	-	-	-	-	1	-	-	-	1
Netherlands	1	-	-	-	-	-	-	-	1	1	-	-	-
Rep. Ireland	2	-	-	1	-	-	-	-	1	1	1	-	-
Spain	6	1	1	1	-	-	-	-	3	1	-	1	4
Sweden	7	3	-	-	-	1	-	1	2	3	3	-	1
UK (England)	29	-	-	28	-	-	-	-	1	5	14	4	6
UK (N.Ireland)	3	-	-	3	-	-	-	-	-	-	3	-	-
UK (Scotland)	6	-	-	5	-	-	-	1	-	-	6	-	-
UK (Wales)	1	1	-	-	-	-	-	-	-	-	-	-	1
UK (unspecified)	6	-	-	6	-	-	-	-	-	3	2	1	-
No. Outbreaks	86	9	3	46	2	8	1	5	12	32	32	6	16
Cases	72546	16222	531	7772	232	11408	2500	2511	31370*	43571	23047*	906	5022

* One outbreak did not report case numbers.

Of the 54 outbreaks where a pathogen could be isolated from cases and the source of the supply was known, 89% of surface water outbreaks were of protozoan origin compared to 46% of groundwater outbreaks (Table 1.3).

Table 1.3: Outbreaks by Pathogen Group and Source of Supply

Pathogen	Water Source		Total
	Groundwater Outbreaks	Surface Water Outbreaks	
Bacteria	7	2	9
Protozoa	12	25	37
Virus	7	1	8
Total	26	28	

1.3.2.3 Environmental and Epidemiological Investigation

Some form of descriptive or analytical epidemiological investigation of cases was reported in 80% of outbreaks. Seven outbreaks reported investigation of animal samples. The speciation of *Cryptosporidium* (into *C. parvum* and *C. hominis*) was well documented in reported outbreaks yet there have been no reports of the use of subtyping for *Cryptosporidium*. A total of seven outbreaks implicating Campylocater and *Norovirus* used subtyping in an attempt to match human with environmental isolates. Of these 7, only 4 outbreaks (2 implicating *Campylobacter* and 2 *Norovirus*) yielded a match between human and water isolates. Where human and environmental strains match this supports the drinking water hypothesis but the value of the negative result is unclear.

Water quality testing was reported in 88% of outbreaks. Of 62 outbreaks reporting whether or not a pathogen was present in the drinking water, 45% found a positive result (Table 1.4).

The robustness of epidemiological and environmental investigations will determine the strength of association with water [Tillet *et al*., 1998]. An outbreak is often recognised following the emergence of cases within the community, consequently environmental and epidemiological sampling is initiated after the contamination event has taken place. Hence, the associative link between cases and water can be missed.

Table 1.4: Percentage of Outbreaks with a Positive Water Quality Result

Drinking Water		Raw Water	
Pathogen (n=62)	Indicator Organism (n=32)	Pathogen (n=34)	Indicator Organism (n=24)
45%	53%	53%	71%

Where n=the number of outbreaks reporting this factor.

1.3.3 Discussion

The outbreaks listed above by no means constitute a definitive list of outbreaks in the EU. As previously noted, outbreak reports were required to meet criteria to avoid inclusion of duplicates, to be referable to the published literature and to allow data

analysis, which will undoubtedly have led to an underestimation of the number of outbreaks identified.
The decline in outbreaks reported in more recent years may in part reflect the time delay between the outbreak occurring and finally being reported in the scientific press. However, a major part of the decline has been due to the significant drop in identified outbreaks in England, possibly secondary to changes in legislation. The recent *Cryptosporidium* outbreak in North Wales has been reported in the media [BBC News, 2005] but has not been included here. It is likely to be some time before full epidemiological findings are reported in the scientific press as the investigative process can be lengthy.
Publication bias can affect the number of outbreaks or the incidence of disease documented. 'Hot topic bias', whereby articles are accepted for publication only if the subject matter conforms to current trends, could, for example, disproportionately promote a particular pathogen or specific country. In the same way, caution should be exercised when comparing outbreaks across different European countries and pathogens as different member states have very different surveillance systems. The detection and reporting of waterborne disease is a product of the adopted public health surveillance strategy. As surveillance strategies vary between countries, it is likely that outbreaks and sporadic cases of disease identified in one country would be missed in another.
No pathogen was isolated from cases in 14% of the outbreaks reviewed. Identification of pathogens in waterborne outbreaks is difficult. In an analysis of public supply outbreaks in the USA, a viral, bacterial or protozoal pathogen was identified in 41%, in 18% a chemical agent was identified, and in the remainder an aetiological agent was not determined [Craun *et al*., 2002]. A thorough and timely water sampling regime following the emergence of cases of illness within the community has the potential to make a link with water but not a definitive refutation of such a claim. Among many other factors, the sensitivity and specificity of laboratory tests must be considered.
To further understand the patterns of and interrelation between seasonality, case numbers, pathogens, countries, and water sources, it is necessary to look at the causes of these outbreaks.

1.4 OUTBREAK CAUSAL FACTORS

1.4.1 Introduction

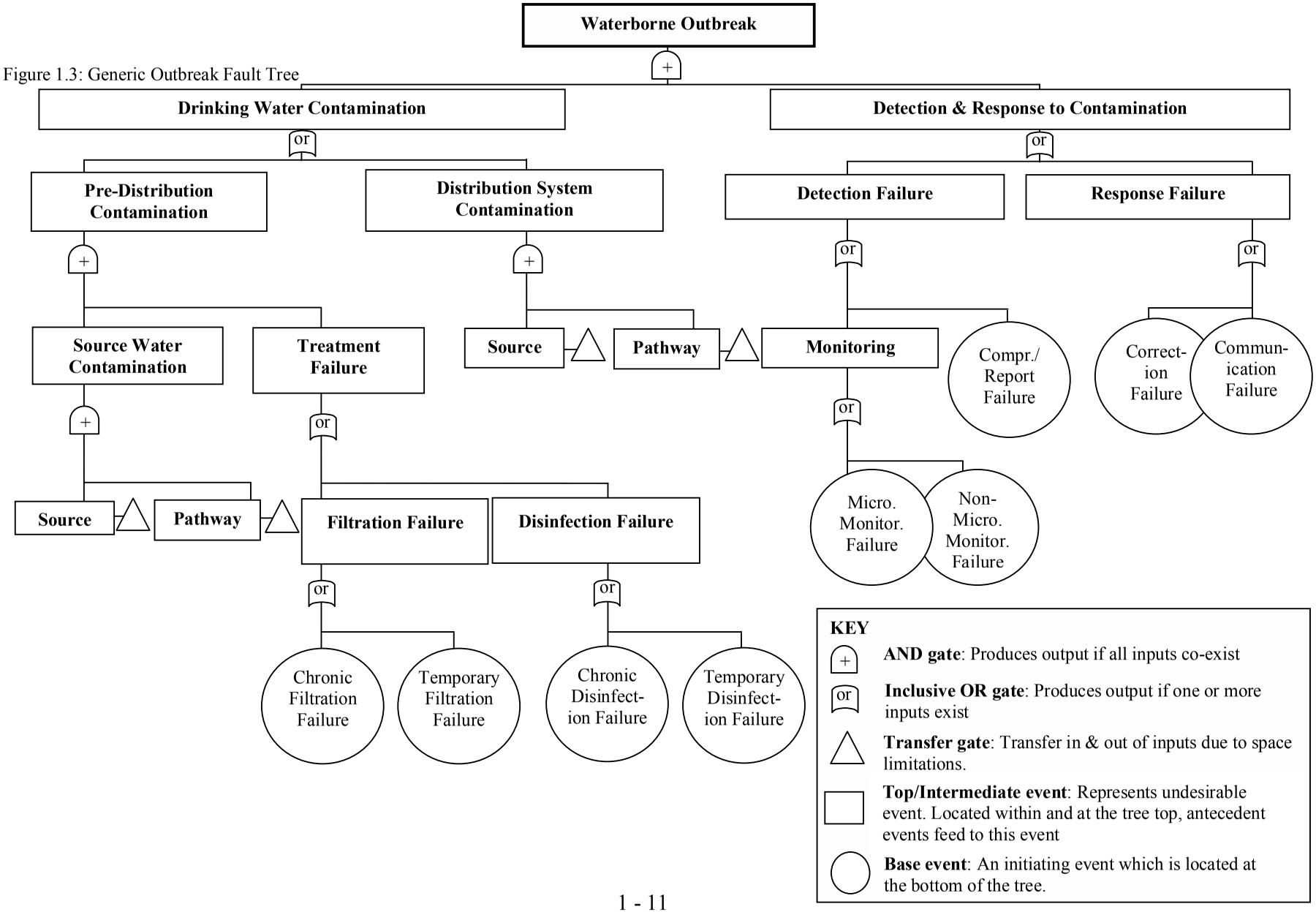
Aside from lessons for disease ecology and socioeconomic burden, outbreaks implicating public supplies also present the unique opportunity to gain a credible, realistic understanding of the contamination pathway; the large volume of consumers and the legislative and regulatory position behind public supplies can stimulate robust environmental and epidemiological investigation.
The Canadian Council of Ministers of the Environment (CCME) multi-barrier approach to safe drinking water identifies three key elements (source water, drinking water treatment plant, and distribution system) to be managed in an integrated manner using tools such as water quality management and monitoring, legislation, and guidelines [Federal-Provincial-Territorial Committee on Drinking Water, 2002]. The IWA Bonn Charter for safe drinking water [2004] also illustrates the necessity for clear roles and responsibilities and knowledge sharing between stakeholders in

achieving safe drinking water that has the trust of consumers. The flow of information between stakeholders and provision of multiple barriers is necessary to reduce the risk of contamination as outbreaks can involve failures across elements of the drinking water system. The *E.coli 0157H7* and *Campylobacter* outbreak of May 2000 in Walkerton, Ontario, occurred as a result of multiple failings including: poor operative training, inadequate monitoring, falsification of records and shortcomings in inspection programs. The contamination event culminated in more than 2,300 people suffering illness, 65 hospitalisations and 7 fatalities [O'Connor, 2002]. Among several important lessons, this outbreak highlights the need to rapidly link an outbreak to its cause to ensure implementation of confinement and correction measures (for example, a boil water advisory or flushing of the system) to reduce attack rates.
Analysis of the multi-factorial nature of outbreaks is therefore fundamental to achieving the goal of safe drinking water. This section aims to take a retrospective look at the causal pathways involved in past outbreaks to identify commonalities thus helping to direct investigations and resources to provide an effective event detection and prevention strategy.

1.4.2 Fault Tree

Sixty-one of the 86 outbreaks previously identified had sufficient information available regarding contributory failures to be utilised in the development of a generic outbreak fault tree (see Figure 1.3).
Fault tree analysis is a diagrammatical risk assessment technique to describe the sequence and interrelation of possible events leading to an undesirable outcome (in this case, an outbreak). Using a top-down approach, preconditions for the undesirable outcome (top event) are determined until the basic causes (base events) are identified. All events are joined by a series of branches and gates. An AND gate requires all input events to occur, an OR gate requires one or more input events to occur. Typically the likelihood of each event is determined and probabilities are assigned.
A fault tree for waterborne outbreaks was designed using the key elements identified by the CCME multi-barrier and IWA Bonn Charter approach to safe drinking water. Each failure considered to contribute to an outbreak was classified according to one of 32 pre-defined base events grouped under four main intermediary events (source, treatment, distribution and detection). Each base event was assigned a percentage score according to the extent of its contribution towards the outbreak. Each outbreak had a total score of 100; thus multiple failures within an outbreak would have a cumulative score of 100. The classification and scoring was performed by seven individuals from five EU countries with expertise in the field of water and health. Further details of the fault tree diagram, methodology and results are currently being prepared for publication.

Figure 1.3: Generic Outbreak Fault Tree



1.4.3 Fault Tree Analysis

A total of 198 events were scored across 30 of the 32 available base events. Table 1.5 illustrates the number of outbreaks with at least one base event failure within each of the four intermediary events and the mean % score attributed by intermediary event (mean contributory scores). The results are also broken down into water source (groundwater and surface water supplies).
Failures occurring at the 'source' of the supply and during 'treatment' occurred with similar frequency and mean contributory scores. 'Distribution' system failures occurred less often but with higher mean contributory scores. Failures associated with the 'detection' of, and response to, microbial and non-microbial pathogens occurred the least often and had the lowest mean contributory score. The pattern for groundwater and surface water outbreaks remained similar with the exception that groundwater outbreaks had a higher 'source' mean contributory score and surface water outbreaks had a higher 'treatment' mean contributory score.

1.4.3.1 Base Events

As the number of base event failures associated with each intermediary event varies it is not useful to directly compare base events across intermediary events. Base events will therefore be considered within each intermediary event.
Looking in more detail at 'source' water failures, both 'livestock activity' and 'rainfall' base events often featured in outbreaks (41% and 44% of outbreaks respectively) which is consistent with the identified seasonality of month of outbreak onset. 'Sewage discharge into the water' or 'onto surrounding land' had higher mean contributory scores (18.4 and 21.8 respectively) than 'rainfall' (17.9) and 'livestock' (14.9), but relatively low frequency of below 10%. The low mean contributory scores for rainfall and livestock are likely due to the existence of further barriers (such as treatment and detection) between source water contaminated with surface water run-off and the consumer. Direct sewage contamination of the surrounding land or water may be intense thus compromising effectiveness of further barriers such as treatment.
With regard to 'treatment' base events, 'chronic filtration failures' were the most frequently documented (38% of outbreaks), yet, 'temporary filtration failures' attained the highest mean contributory score of 58.8. Long-standing inadequate treatment of a supply occurred as a result of multiple failures (such as, poor water quality monitoring) whereas a temporary interruption to filtration was more likely to occur as a solitary event. When segregated into groundwater and surface water supplies, both types of supply suffered most often from 'chronic filtration failures' (18% and 16% of outbreaks respectively). 'Chronic disinfection failures' were deemed to have the greatest contribution to groundwater supply outbreaks (mean contributory score of 36.3) and 'temporary filtration failures' to surface water supply outbreaks (36). Some of the reports documenting groundwater supply related outbreaks noted that groundwater was considered by treatment facilities to represent a purer source than surface water. This assumption led them to apply less stringent treatment regimes resulting in chronically inadequate treatment.
For 'distribution' system base events, 'backflow/cross-connection' caused by a water company employee received a high mean contributory score (95) yet this was associated with just one outbreak. 'Backflow/cross-connection' caused by individuals outside of the water company (such as, an irrigation user) had a comparatively high

frequency (15%) and high mean contributory score (85.4). This pattern remained true when separated into groundwater and surface water outbreaks, which is likely as the distribution failures are expected to be independent of the type of source water.
Concerning 'detection' base events, 'comprehension' of the significance of existing or historical microbial or non-microbial results was the most frequently identified event (18% of outbreaks) and also marginally received the highest mean contributory score (16.7). This event occurred most frequently in outbreaks related to surface water supplies (10% of outbreaks) yet it was deemed more influential in groundwater supply-related outbreaks (mean score of 14.5). A lack of knowledge and experience concerning the significance of poor raw and treated water quality results, particularly with regard to turbidity fluctuations, contributed to this result. This finding highlights a potential knowledge gap to be addressed through additional education and training of water utility staff.

1.4.3.2 Events by Pathogen Group

Table 1.6 lists the number of outbreaks in which at least one base event failure occurred within each of the intermediary events (source, treatment, distribution and detection) and the mean contributory scores for each intermediary event by pathogen group. All pathogen groups attained the highest mean contributory score for 'distribution' system failures. Despite bacterial, protozoal, and viral outbreaks having a high mean contributory score for 'distribution' failures, this type of failure is relatively infrequent; this is in contrast to gastroenteritis outbreaks which have more 'distribution' than 'source' or 'treatment' failures, and mixed pathogen outbreaks which have the same number of 'treatment' and 'distribution' failures. 'Livestock' was more often associated with protozoal outbreaks than any other pathogen group; livestock are known risk factors of such parasites [Hunter *et al.*, 2004; Robertson *et al.*, 2002; Roy *et al.*, 2004].

Table 1.5: Intermediary Event Fault Tree Analysis Results by Water Source

Intermediary Event	No. of Outbreaks n=61	Mean % Score (std. dev)	*Water Source**			
			Groundwater		**Surface Water**	
			No. of Outbreaks n=24	Mean % Score (std dev)	No. of Outbreaks n=22	Mean % Score (std dev)
Source	41	50.5 (26.6)	19	60.3 (23.2)	17	39 (26.3)
Treatment	41	49.0 (25.6)	18	35.6 (22.3)	17	59 (23.2)
Distribution	19	87.4 (22.0)	6	84.5 (24.9)	5	80.8 (26.5)
Detection	16	22.6 (16.0)	6	17.8 (08.3)	7	18.6 (18.3)

* Fifteen outbreaks either did not report the source of the outbreak or the source constituted a mixed supply.

Table 1.6: Intermediary Event Fault Tree Analysis Results by Pathogen Group

Intermediary Event	**Pathogen**									
	Bacterial		***Protozoal***		***Viral***		***Gastroenteritis***		***Mixed***	
	No. of Outbreaks (%) n=8	Mean % (std dev)	No. of Outbreaks (%) n=31	Mean % (std dev)	No. of Outbreaks (%) n=5	Mean % (std dev)	No. of Outbreaks (%) n=12	Mean % (std dev)	No. of outbreaks (%) n=5	Mean % (std dev)
Source	5 (62.5)	57.0 (22.4)	26 (83.9)	45.3 (28.2)	3 (60)	63.7 (11.1)	5 (41.7)	61.8 (23.2)	2 (40)	53.5 (44.6)
Treatment	5 (62.5)	43.2 (16.4)	23 (74.2)	53.9 (25.8)	4 (80)	46.5 (35.8)	6 (50.0)	38.8 (9.5)	3 (60)	43.0 (49.4)
Distribution	3 (37.5)	81.0 (32.9)	5 (16.1)	86.8 (26.8)	1 (20)	100.0 (-)	7 (58.3)	88.6 (20.4)	3 (60)	88.0 (20.8)
Detection	3 (37.5)	18.7 (10.3)	10 (32.3)	25.0 (18.8)	2 (40)	11.5 (5.0)	1 (8.3)	32.0 (-)	0 (-)	-

1.4.3.3 Additional Swedish Outbreaks

A singular causal event was documented for each of the 30 additional Swedish outbreaks which were omitted from the review of outbreaks. Fifty seven percent were caused by faecally contaminated raw water passing through the waterworks, 37% were caused by faecally contaminated water entering the distribution system (after the waterworks), and in 7% of outbreaks the cause was unknown.

1.4.4 Discussion

Presenting the major causal factors involved in waterborne outbreaks of disease using the novel diagrammatic approach of fault tree analysis highlights the issues relevant to public water suppliers, consumers, and catchment users.

Results have implications for the treatment of groundwater and surface water supplies and the monitoring of metrological, microbial, and non-microbial data. Although distribution system failures were considered to have the greatest contribution to surface water outbreaks, surface water supplies suffered most often from treatment failures. Of the treatment failures, chronic filtration failures occurred most often and temporary interruption to filtration was the most influential in causing such outbreaks. This is consistent with the finding that 89% of surface water outbreaks were associated with protozoa.

Establishing and maintaining effective collaborative links with factories, farmers, and other users of the network, could help to prevent contamination of the distribution system where fewer barriers to the consumer exist. Such collaboration should also be present with catchment users. Increasing awareness about the effects of agricultural practice and sewage contamination, and communicating the importance of early warning, can help to protect the quality of source water and ensure optimal treatment.

The enteropathogenic waterborne disease outbreaks reviewed here provide valuable information concerning where and how failures can occur. It is hoped that in applying this fault tree methodology a greater understanding of the likelihood and severity of events and the complex interactions between them can be gained. This may have important policy implications for water companies in terms of targeted resource management and outbreak prevention strategies. Catchment, source water and distribution network protection, communication with stakeholders, and review of treatment and monitoring procedures have been highlighted. These are primary components of the Water Safety Plan and thus formulate the basis for hazard identification. Water Safety Plans should be developed for all water supply chains and tailored to each system. Further validation and use of this fault tree can be demonstrated through application to additional outbreaks. Fault trees could be adapted to reflect individual systems, for example, to look in more detail at the probability of human and technological failure of individual treatment processes.

1.5 WATERBORNE DISEASE IN NON-COMMUNITY SUPPLIES

1.5.1 Introduction

The regulation, legislation and outbreak documentation for public drinking water delivery system is more vigilant compared with smaller non-community supplies (private water supplies) due to the larger number of people affected by any microbiological failure. Despite non-public or private water supplies (PWS) serving just 1% of the English population [Clapham, 1993], the public health risk is high as studies have demonstrated a high level of microbiological failure [DWI, 1996] and low-level compliance with water regulations [Rutter *et al*., 2000]. The situation is much the same across rural areas of Eastern and Western Europe, with private/small community supplies receiving little or no treatment, with inadequate monitoring [DWI, 1999]. In England, the Private Water Supplies Regulations [1991] is the current legislation specifying the responsibilities of the Local Authorities over the quality of PWS drinking water. Since inception, the Water Supply (Water Quality) Regulations in England have been updated in accordance with the European Council Directive 98/83/EC [E.U., 1998], with a slant towards risk assessment. Regulatory revisions are ongoing and therefore a thorough understanding of the risk of contamination and the effect upon the health of PWS consumers is paramount.

1.5.2 Outbreaks Associated with Private Supplies

Waterborne outbreak data from England and Wales (1971 – 2005) were reviewed [Galbraith *et al*., 1987; Said *et al*., 2003]. All documented PWS associated waterborne outbreaks were summarised according to aetiological agent.
There have been 29 waterborne outbreaks related to private drinking-water supplies in England since 1971 (Table 1.7). Most of the PWS outbreaks were reported within 10 years after the introduction of enhanced surveillance in the early 1990s, suggesting that there may have been an under reporting of PWS outbreaks prior to 1990. Investigations into these 29 outbreaks have identified 2751 cases, with more than 4866 people at risk of infections. From the outbreak data, *Campylobacter* was the most commonly identified pathogen (45%), followed by unknown aetiology (17%), *Cryptosporidium* (10%); combination of *Cryptosporidium* and *Campylobacter* (7%), and *Escherichia coli 0157* (7%). *Giardia*, *Salmonella* Paratyphi *B* (PT1), *Streptobacillus moniliformis* (rats found in spring and sewer) and a combination of *Cryptosporidium* and *Escherichia coli 0157* were also identified as the causal pathogen in PWS outbreaks. The strength of association for implicating water as the vehicle or cause of the outbreaks was strong or probable in 76% of the outbreaks. Although the population at risk and the number of cases were relatively low in these outbreaks, the attack rate in each of the outbreaks ranged from 4% to 89% (mean = 42.5%). It is notable that the range of pathogens causing outbreaks associated with private supplies in England and Wales is much broader than those causing outbreaks in public supplies.

Table 1.7: PWS outbreaks from 1971-2005 (up to June 2005)

Decade	Pathogen	No. outbreaks	Population Supplied	No. cases
1971-1980	S.paratyphi B (PT1)	1	10	7
	Unknown	2	>316	172
1981-1990	*Campylobacter*	3	>767	520
	Streptobacillus moniliformis	1	700	304
	Unknown	1	?	138
1991-2000	*Campylobacter*	9	>1081	195
	Cryptosporidium	3	>664	77
	Cryptosporidium & *Campylobacter*	1	200	43
	E.coli 0157	1	16	14
	Giardia	1	260	31
	Unknown	2	752	83
2001-2005 (June)	*Campylobacter*	1	30	4
	Cryptosporidium & *Campylobacter*	1	50	2
	Cryptosporidium & *E.coli 0157*	1	16	16
	E.coli 0157	1	4	4

1.5.3 Private Water Supply Microbiological Quality

Drinking water supplied by both public and private water supplies are subjected to the same microbiological standard (i.e. the absence of E.coli and coliforms in 100ml of water sample). The water quality data from public water suppliers are nationally collated and monitored by the Drinking Water Inspectorate. In contrast, water quality data from PWS are monitored by the Local Authorities. In 1996, the Communicable Disease Surveillance Centre (CDSC) created a data collection system to collate microbial water quality results from PWS samples sent to PHLS for analyses with the aim of providing a national picture of the water quality from PWS in England and Wales [Rutter et al 2000].

The results from the first 2 years of data collection (January 1996 to December 1997) were published in Rutter et al 2000. Within this period, there were 6551 samples from 2911 supplies in the Private Water Supply Microbiological Quality Surveillance Database. The key findings from Rutter's paper indicated that over one fifth of the PWS samples failed to comply with the microbiological standards (21% contained *E.coli*, 27% contained coliforms). The quality of water from larger supplies tends to be of better quality compared with those from smaller supplies. The source (where the drinking-water derive from) is also a contributing factor to the water quality, with the gradient of contamination increases from groundwater to spring to surface water; where surface water has the highest level of *E.coli* contamination. Although there was no distinct seasonal sampling pattern during the data collection period; a seasonal trend of *E.coli* contamination was observed with an upward trend from April, culminating in a peak in August and November for both years.

The water quality of PWS can be affected by location and construction of the PWS system. Water derived from surface water and land-drain and shallow groundwater are at higher risk of microbial and chemical contamination compared with water

derived from deep boreholes. Contaminated surface water can enter into badly constructed and poorly protected water abstraction systems during rain events. Most of the PWS in England and Wales are in rural settings, delivering water to single dwellings and premises requiring less than $100m^3$ of water per day. There is a comparatively fewer number of large water supplies delivering more than $1,000m^3$ per day. The larger supplies are often maintained and monitored more frequently compared with the smaller supplies due to the public health significance of more people using these supplies. The smaller supplies tend to be of poorer quality and at higher risk of faecal contamination from wildlife and farmed animals and local sanitation systems (e.g. septic tank).

Since Rutter's publication, CDSC continued to collect PWS microbiology quality data until and between 1996 and 2003, there were over 37,000 results from more than 13,000 premises. The majority of the results were from premises where more than 1 sample was taken during the data collection period mainly due to compliance with the sampling regulations. The higher the number people using the PWS, the higher the frequency of water testing. By increasing the proportion of multiple testing per premises may increase in the probability of detecting *E.coli* in the water from larger supplies. Nonetheless, the concentration of *E.coli* detected and number of *E.coli* failure is much higher in the smaller supplies compared with the larger supplies. Further analysis on this dataset is required to assess trends between the different types of water supplies and with other external factors.

1.5.4 Endemic Disease Associated with Private Supplies

The effect of individual exposure to waterborne pathogens can differ depending on their immune status. Although certain subgroups of the population are more vulnerable to infections (e.g. the young and the old and people who are immunocompromised), repeated exposure to contaminated water may not lead to serious illness due to acquired immunity. Hence, visitors using PWS may be at higher risk of gastrointestinal illness than regular PWS owners/users due to their continuous exposure to the water. Nonetheless, the excess risk of illness associated with private water supplies in Europe is unknown. The considerable number of samples from private supplies containing *E. coli* demonstrated both here and elsewhere [the Netherlands, Schets, *et al.*, 2005] indicates that such supplies are at high risk of faecal contamination. The studies by Zmirou *et al*. [1987, 1995] reported above are almost certainly relevant to the issue of PWS. If these studies are applicable, then this would suggest that the risk of gastroenteritis illness in people with PWS, at least those subject to faecal contamination would experience a 40% increase in risk of gastroenteritis. This figure is consistent with a study of private rural systems in Canada [Strauss *et al*. 2001].

People living in rural locations often have PWS that are prone to faecal contamination. It is the possible that PWS are an important contributor to the burden of waterborne disease within Europe, although there is little published evidence for this excess illness. This is an area of water safety that is currently substantially under-researched.

1.5.5 Discussion

Reviewing cumulative water quality results and data from past outbreaks can improve understanding of the indicator organism load in PWS. These vital data can be used to examine trends; establish frequencies and consequences of system failures and identify pathogens that are frequently associated with waterborne outbreaks. The major issue in resolving the water quality problems with PWS is that every system is unique. Thus the problems associated with water quality in PWS can differ substantially from site to site. Although the population using PWS is generally less than those using public water supplies; the microbial quality of water from PWS is poorer and the water is usually consumed untreated. Consequently, the health risk to people using PWS could actually be higher than people using public water supplies.

The enhanced knowledge gained from the surveillance and outbreak data provided a view into the state of the PWS quality and the public health outcome of consuming microbiologically poor quality water. Yet there is little knowledge on the source and route of contaminations and the actual health risks association between people using PWS and the quality of their drinking water. Therefore it is necessary to conduct studies to ascertain where the contamination come from, how much and how often the consumers are exposed to the contamination and to establish the prevalence of disease associated with the consumption of PWS in the community.

Due to the lack of centralised water quality monitoring regulation and surveillance system in England, the enforcement of the PWS regulation currently lies with the Local Authorities who have limited power and resources to act. Thus the owners and users of the PWS are responsible for the quality of their water supply. However, everyone involved in PWS should be made aware of the potential hazards that can enter into their drinking water and the interventions in place to reduce the likelihood and consequence of drinking water contaminations. They should be vigilant in protecting the source water; conducting regular checks on the water supply system and water treatment facilities. Hence conducting regular assessment of the risks to PWS can be beneficial in preventing the water quality non-compliances and waterborne outbreaks.

1.6 PUBLIC HEALTH SURVEILLANCE OF WATERBORNE DISEASE

1.6.1 Introduction

National communicable disease surveillance institutes collate local, national and international intelligence to better inform public health policy. Not all countries conduct communicable disease surveillance and, of the active institutes (see Table 1.8), not all monitor enteropathogens associated with drinking water. A range of surveillance strategies have been adopted by different national institutes and researchers to monitor the incidence and prevalence of waterborne disease. The

Table 1.8: National Public Health Surveillance Centres (content derived from official institute websites).

National Public Health Institute (KTL), Finland Established in 1982, KTL has a number of functions including monitoring, education and training, international collaboration, research and dissemination of health information. The Department of Infectious Disease Epidemiology (INFE) at KTL is responsible for the surveillance of infectious disease and provides support to municipal authorities in outbreak/epidemic situations. The department is also the national coordinator for the EU monitoring network of infectious diseases.
The National Institute for Public Health Surveillance (InVS), France Founded in 1998, InVS is a relatively young institution. Reporting to the Ministry of Health InVS is responsible for the surveillance and monitoring of public health fulfilling a number of policy-advisory goals. Sixteen regional epidemiology units (CIRE) relay information to InVS. CIRE conduct field epidemiology, investigate epidemics and carry out quantitative risk assessment. The Department of Infectious Diseases is organised into five thematic units including 'Enteric, Food and Zoonoses Infections' which monitors mandatory notifications and conducts epidemiological investigations. The department also co-ordinates the National Reference Centre (CNR) and offers training in intervention epidemiology. The Water related risks Unit of the Environment and Health department of the InVS contributes to the surveillance and monitoring of waterborne diseases.
The Robert Koch Institute (RKI), Germany The Law for the Prevention of Infection (Infektionsschutzgesetz, IfSG) assigned the task of a federal epidemiological centre for infectious diseases to RKI. In addition to this, the new Protection against Infection Act has ensured national surveillance of a number of infectious diseases; this work is carried out by the Department of Infectious Disease Epidemiology at RKI. Division 32 (Surveillance) and 35 (Gastrointestinal Infections, Zoonoses and Tropical Infections) of this department are responsible for surveillance and the investigation of outbreaks including the development of algorithms for early outbreak recognition. A "rapid task force" is available for the investigation of regional outbreaks or epidemics if requested by federal states. National reference centres and consultant laboratories in infectious disease information are also available for certain diseases.
National Instiute of Publich Health and the Environment (RIVM), The Netherlands RIVM conducts surveillance, risk assessment and research in the areas of health, nutrition and the environment. RIVM offer guidance and support in the event of incidents and for the purpose of health protection. The Centre for Infectious Disease Control at RIVM is responsible for the prevention and control of infectious diseases through effective prevention, surveillance and rapid response. The Centre is comprised of a number of units including the Centre for Infectious Disease Epidemiology (CIE). CIE co-ordinates the Infectious Diseases Surveillance Information System (ISIS); ISIS provides a rapid visual representation of infectious diseases to health professionals. Information from laboratories and the Public Health Services (GGD) is sent electronically to CIE for this purpose. Information is assessed weekly and recent developments are relayed by the 'Reporting and Supporting' group, information is also published monthly and annually. Using mathematical modelling, RIVM provide detailed information on disease patterns and trends to support surveillance initiatives and policy-making.
The Swedish Institute for Infectious Disease Control (SMI), Sweden Of the six departments of SMI, the Department of Epidemiology is responsible for the national communicable disease surveillance. A computerised reporting system 'SmiNet' collects and analyses the surveillance data. Feedback of surveillance data is provided weekly and bimonthly as well as annually. A yearly report provides further insight on the disease patterns observed throughout the year. Sweden has collaborative links with the National Board of Health and Welfare, the National Veterinary Institute, the National Institute of Public Health, the National Food Administration, the Swedish Armed Forces, the Swedish Defence Research Agency, the Swedish Work Environment Authority, the Swedish Federation of County Councils and the Swedish Association of Local Authorities.
The Health Protection Agency (HPA), United Kingdom The HPA provides an advisory and preventative approach to environmental hazards and infectious disease. The infectious disease element of HPA is dealt with by the Centre of Infections which conducts surveillance, provides reference microbiology and advice and performs outbreak co-ordination. Information for surveillance purposes is assembled from a number of sources including the national laboratory reporting scheme, hospital episode statistics and incident and case reports gathered from physicians, laboratories, Environmental Health Officers (EHOs) and Consultants in Communicable Disease Control (CCDC). The Centre of Infections also liaises with the Water and Environmental Reference Unit and the Drinking Water Inspectorate (DWI) for the investigation of waterborne disease.

structural and organisational integrity of the strategy can not only affect the number of cases detected, but also the strength of an epidemiological association with water. Analysing reported incidence of waterborne disease in conjunction with the characteristics of adopted surveillance strategies can lead to a greater understanding of country-specific trends.

1.6.2 Laboratory and Clinician-Based Reporting

Laboratory and clinician-based reporting constitute the main body of surveillance employed by many national institutes. A host of factors can affect the accuracy and efficiency of national surveillance based on this method. The infectious intestinal disease (IID) incidence study, conducted in England between 1993 and 1996, demonstrated that one case was reported to national surveillance for every 1.4 laboratory identifications, 6.2 stools sent for laboratory investigation, 23.2 cases presenting to general practice, and 136 community cases [Food Standards Agency, 2000]. The number of cases present in the community can depend upon dose-response, acquired immunity and pathogenicity. GP utilisation may be affected by accessibility of services, stool sampling upon budgetary constraints, and positive laboratory findings upon the specificity and sensitivity of diagnostic tests and the selection of pathogens to test for.
It is possible that reporting behaviour is affected by country-specific regulatory and policy-based recommendations. A comprehensive account of the statutory position behind laboratory and clinician reporting of waterborne pathogens and diseases in Europe is given by [Poullis *et al.*, 2002]. Table 1.9 updates this information for seven enteropathogens and the six surveillance systems briefly described in Table 1.8.

Table 1.9: Enteropathogen surveillance and statutory position

Pathogen	Country					
	Finland	France	Germany	Netherlands	Sweden	UK
Campylobacter	●	o	●	o	●	o
Cryptosporidium	●	-	●	o	●	o
E.coli 0157: *H7*	●	o	●	●	●	o
Giardia	●	-	●	o	●	o
Norovirus	●	o	●	o	o	o
Salmonella	●	o	●	o	●	o
Shigella	●	o	●	●	●	o
Acute Gastroenteritis*	●	o	●	●	o	●
Outbreak	●	●	●	●	●	o

Information Source: Finland: KTL [2005] ; France: Vaillant *et al.* [2004]; Germany: IDCA [2001]; Netherlands: IDA [1999], RIVM [2005]; Sweden: CDA [2004], Lindqvist *et al*. [2001], SMI [2005]; UK: HPA [2005a; 2005b].
* Linked to either a food handler or food poisoning.
● Statutorily notifiable.
o Data collected on a voluntary reporting basis.
- No information on notification procedures were identified.

All statutorily notifiable pathogens listed in Table 1.9 require notification from the laboratory with the exception of *Shigella* in the Netherlands which is only statutorily notifiable by the attending clinician (where *Shigella* is classified as 'bacillary dysentery'). In Sweden statutorily notifiable pathogens also require notification from the attending clinician (complementary to laboratory notification).
The coverage and timeliness of surveillance varies between countries and pathogens. In France, data on *Campylobacter*, *Salmonella*, and *Shigella* are collected by National

Reference Centres (CNRs). Data on *E.coli*, *Norovirus* and gastroenteritis are based on sentinel or otherwise limited surveillance. Therefore, not all data is suitable for the purposes of rapid outbreak detection. In the Netherlands the Infectious diseases Surveillance Information System (ISIS) is an internet-based reporting system designed to describe the day-to-day changes in the frequency of communicable diseases. Statutorily notifiable diseases have national reporting coverage; voluntary reporting coverage is expected to rise to 35% by the year 2006. Coverage is higher for some pathogens, *Salmonella* coverage is estimated at 64% of the population [Widdowson *et al*., 2003]. Sweden also has a computerised reporting system (SmiNet) with approximately 18 of 21 counties connected [Jansson *et al*., 2005].

Laboratory and country specific guidelines concerning the routine screening for pathogens should also be considered. It is possible that routine screening policies or the absence of a legal requirement to report protozoal pathogens in the Netherlands contributed to the lack of protozoal outbreaks identified. However, it is also feasible that, as treatment is largely concentrated on coagulation and filtration, activated carbon filtration and ozonation or UV (rather than chlorination) and a larger proportion of drinking water is supplied by groundwater sources in the Netherlands [Bartram *et al*., 2002: 87-93], protozoa have little chance of reaching the consumer.

An awareness of such environmental and epidemiological surroundings should not be underestimated in determining the source of an illness. Many enteropathogens associated with drinking water are also associated with risk factors such as animal contact and food. Detecting an association between drinking water and infectious disease can be extremely difficult. This is especially true in the instance of sporadic illness. Therefore, in order to conduct accurate surveillance of waterborne disease, epidemiological investigation of cases must accompany microbiological detection. Information of this type is unattainable through direct laboratory-based reporting due to the absence of patient contact. Laboratory notifications need to be supplemented with case history gathered through direct patient consultation.

1.6.2.1 Reported Strengths and Weaknesses

As part of this study we were able to interview key individuals with demonstrated experience and academic record in the field of epidemiology and surveillance in a number of European states (Finland, France, Germany, the Netherlands, Sweden and the UK). Reported strengths and weaknesses of existing national surveillance strategies principally concerned methods of laboratory and clinician-based surveillance and reporting practice.

Strengths of existing waterborne disease surveillance strategies recounted by interviewees included approachability of the national centre and the ability to construct relationships with reporting individuals and institutes. Provision of feedback and accessibility of current local and national disease trend information to clinicians, laboratories, and other reporting bodies was judged to encourage continued involvement in surveillance. Statutory notification of disease with clearly defined regulatory boundaries was seen as a further means of encouraging reporting practice.

A reporting system which promotes the direct provision of information to a centralised national reporting system without delay at the intermediary or regional level of reporting was viewed as a distinct advantage. Technological process capabilities such as electronic notification and internet-based reporting were deemed to facilitate the speed and accuracy of surveillance. Standardisation of laboratory

techniques and development of common standards to improve epidemiological case investigation were also identified as means of improving the accuracy of surveillance.
The frequency of patient sampling was thought to be restricted if the financial burden for these samples was taken directly from the clinician's budget, thus limiting the number of pathogens detected and eliminating the necessity to report. In the laboratory setting, infrequent testing for parasites and a lack of detailed patient background information were believed to reduce the effectiveness of surveillance. Another reported weakness of waterborne surveillance was the difficulty encountered in linking cases to water; this was deemed to be hindered by a lack of sporadic case data, local staff lacking experience in outbreak investigation and infrequent notification of pipeline repair.

1.6.3 Additional Components of Surveillance

Alternative methods of surveillance attempt to address some of the shortcomings of national reporting based solely upon patient sampling and testing.
Laboratory sampling can become expensive and 'sentinel' practices and laboratories which have been set up for enhanced surveillance of particular diseases in known problem localities can reduce costs.
Surveillance based upon the incidence of single pathogens may not identify waterborne outbreaks caused by sewage contamination. Monitoring levels of gastrointestinal illness in the community or 'syndromic surveillance' can identify outbreaks caused by multiple enteropathogens. Studies have researched the feasibility of monitoring anti-diarrhoeal drug sales [Beaudeau *et al.*, 1999; Edge *et al.*, 2004; Sacks *et al.*, 1986], telephone help-lines [Rodman *et al.*, 1998] and emergency department visits [Heffernan *et al.*, 2004] as forms of syndromic surveillance. Guidance has been written on implementation of syndromic surveillance informed by first-hand experience of such systems [Mandi et al., 2004]. In addition to positive laboratory diagnoses, in the Netherlands records are kept of negative results. Such records could be used to inform the efficiency, sensitivity, specificity, and effectiveness of pathogen-specific testing. An increase in sampling requests could also be used to indicate an outbreak of undetermined aetiology or of mixed pathogen source.
Syndromic surveillance may identify a cluster of cases but supplementary information is required to ascertain an epidemiological link between cases and with water. 'Proper officers' such as the Consultants in Communicable Disease Control (CCDCs) in England and Wales, County Medical Officers (CMOs) in Sweden, and epidemiologists from the CIREs in France, investigate epidemiological associations between cases at the local level. Alternative risk factors can be ruled out but the link with water can be compounded by the complexity of the distribution system. Monitoring consumer complaints about the odour, colour, or taste of the drinking water can help to identify clusters and facilitate the epidemiological linkage of cases with water. Monitoring incidents affecting the water supply such as, poor water quality results, treatment deficiencies, and pipeline repair, can also be used to inform this process. Collaborative links with other organisations can promote the exchange of knowledge supplementing information from reporting and improving the accuracy of epidemiological investigation. SMI in Sweden, for example, has established links with environmental, veterinary and food organisations.
International dissemination of surveillance information is necessary to promote the linkage of purportedly sporadic cases of illness. The EU-wide surveillance network

for *Salmonella* and *VTEC 0157* infections, Enter-net, is an example of one international surveillance body which is based on a harmonised laboratory system. In using information collected from the microbiologist in charge of the national reference laboratory and the epidemiologist responsible for the national surveillance of these organisms, Enter-net has facilitated case-linkage across national boundaries [Eurosurveillance, 2002].

1.6.4 Discussion

Pathogen isolation from water and patient sampling can be an expensive and lengthy process. This is especially true where there is low reported endemicity and the nature of illness is self-limiting. In instances where testing for *Cryptosporidium* is not feasible, limiting testing to the immumocompromised and children aged fifteen years or younger has been suggested [Crook *et al.,* 2002]. Syndromic surveillance combined with water incident and consumer complaints data can provide more timely information. Although this data is less accurate, once a possible trend has been identified active surveillance using laboratory testing can be pursued [Thompson, 2003].

It is likely that national surveillance institutes employing a combination of surveillance methods coupled with a collaborative approach promoting inter-organisational information integration will generate a more accurate depiction of the burden of waterborne disease. It is important that such systems evolve continuously to embrace changing population dynamics and to incorporate technological development. Electronic reporting of notifiable diseases has been found to more than double the total number of laboratory based reports received [Effler *et al.*, 1999]. The degree and speed of notification has been noted to increase with laboratory as opposed to clinician based reporting [Rietveld *et al.*, 2005]. The feasibility of using algorithms for rapid outbreak detection has also been discussed [Buckeridge *et al.*, 2005].

In summary, reducing heterogeneity between EU surveillance systems and laboratory methods, using a combination of surveillance practices described and improving the methodological robustness of outbreak investigation should increase detection rates; making incidence patterns more representative and less a reflection of a country's surveillance system.

1.7 CONCLUSION

Levels of endemic waterborne disease are probably low in most member states. However, public supplies serve very many consumers and as such contamination, even if causing illness in a small proportion of consumers, can pose a significant threat to public health. This is most clearly seen during outbreaks of illness associated with public water supplies. Although private water supplies serve a smaller population, they are frequently prone to faecal contamination and probably pose a greater risk to people reliant on them for their primary drinking water source.

Review of outbreak data can lead to a greater understanding of the epidemiological, ecological, and environmental factors contributing to the causes of waterborne disease. Heavy rainfall and livestock activity are frequent contributory factors

involved in the occurrence of outbreaks. Although the probability of occurrence is less, the magnitude of effect is greater for distribution system incidents. Increased awareness of the public health hazard associated with illegal cross-connections and source water contamination could ameliorate these issues. Production of standardised guidelines and training may improve comprehension of existing or previous microbial and non-microbial results leading to a reduction in repetitive incidents.

The detection and investigation of outbreaks is important for the protection of public health, yet detection and reporting varies from one European member state to another making comparison across Europe difficult. A number of specialist tools and methods of surveillance have been generated from existing national surveillance systems. Studying their diversity can promote the exchange of ideas between countries and help to inform incidence data. Increased collaboration between a number of industries including water, food, veterinary, and health, can improve detection and epidemiological association. Advances in technological processes, data handling, and information integration can also improve the speed and accuracy of surveillance.

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2. QMRA: its value for risk management

Gertjan Medema and Nicholas Ashbolt.

2.1 INTRODUCTION

The EU-project MicroRisk focused on Quantitative Microbial Risk Assessment (QMRA) as a scientific basis and tool to assess the microbial safety of drinking water supplies. This chapter describes how this risk assessment fits in the overall risk and quality management and how risk assessment can aid risk management. This chapter (and indeed the whole book) focuses on the water utility in its responsibility to manage drinking water safety and how the water supplier can make use of risk assessment. That does not imply that other stakeholders have no role in this risk-based approach. Each of these stakeholders is involved in the framework; the regulator is responsible for setting health-based targets for drinking water and enforcement of these targets and health authorities are responsible for control of the transmission of disease. The involvement of other stakeholders is briefly described in this chapter.

2.2 THE SAFE WATER FRAMEWORK

An international group of experts, assembled by the World Health Organization, discussed the approach to assess and manage the health risk of pathogenic micro-organisms in drinking water, recreational water and wastewater [Fewtrell & Bartram, 2001]. This group agreed that future guidelines for safe drinking water should integrate risk assessment and risk management into a single framework, the Safe Water Framework. The simplest form of the framework is given in Figure 2.1.

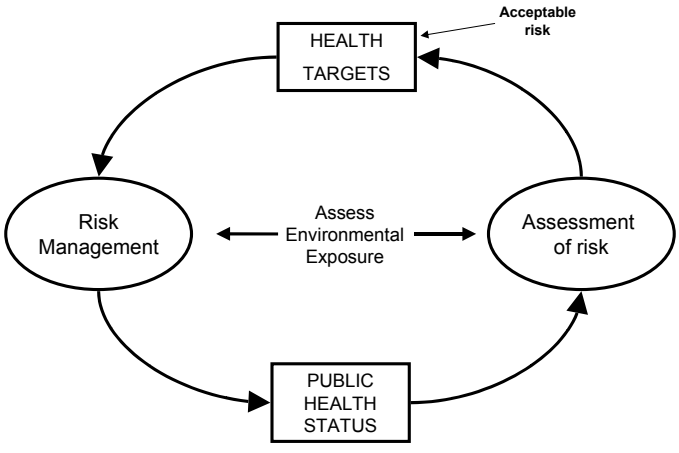

Figure 2.1. Safe Water Framework for integrated risk assessment and risk management

The risk that is assessed and managed in this approach is human health risk. Given this document is addressing pathogens in drinking waters, the risk here is that consumers of drinking water will contract an infectious disease through use of the water. It is clearly an iterative cycle in which risk assessment is a basis for decision-making in risk management. The four steps of the cycle are described in the next paragraphs.

2.2.1 Health targets

Health targets are benchmarks for the water suppliers, set by the regulator as part of their health policy. Health targets for drinking water are traditionally strict, because of the large potential impact of contaminated tap water and the basic need for safe drinking water. This leads to the question of what level of health risk through drinking water could be tolerated, given the overall health status of the consumer population and the contribution of drinking water to the overall health risk of this population in relation to other routes of exposure, such as food, person-to-person or animal contact, recreational water etc. This is a question that typically needs answering on the level of the regulator, who can translate this information into a health target for drinking water, considering other factors such as relative contribution of drinking water transmitted disease to the overall health burden and the economic climate.
The health target is the level of a tolerable risk level for drinking water, which could be expressed as the tolerable risk of infection through drinking water (i.e. risk of infection $<10^{-4}$ per person per year [Regli *et al.*, 1999]) or the tolerable amount of disease burden (i.e. $< 10^{-6}$ disability adjusted life years (DALYs) per person per year [WHO GDWQ, 2004; Havelaar *et al*., 2000]). The health target could be translated into water quality targets for pathogens (analogous to toxic chemicals). Rather than producing a standard and monitoring requirement for all pathogens that could be transmitted through drinking water, the use of a suite of "index pathogens" is advisable. Establishment of adequate control against this suite of pathogens should offer protection against the other known (and even unknown) pathogens.
It is emphasised that the health targets may differ due to health status situations. The judgement of what is a tolerable level of risk is a matter in which the society as a whole has a role to play; the decision on the cost-benefit is for each country to decide [WHO-Guidelines for Drinking Water Quality, 1993; 2004]. It is important that health-based targets, defined by the relevant health authority, are realistic under local operating conditions and are set to protect and improve public health. Health-based targets underpin development of Water Safety Plans [WHO GDWQ, 2004] and provide information with which to evaluate the adequacy of existing installations; and assist in identifying the level and type of inspection and appropriate analytical verifications. Further details on health-based targets are covered in Chapter 3 of the WHO GDWQ.

2.2.2 Risk management

In the EU, managing the safety of drinking water has been the core business of water supply companies for more than a century. Over this period, risk management has

evolved into a culture, codes and specifications of good practice. In the last decades, quality management systems have been used in the water industry to formalise this. Currently, water suppliers in several EU-countries are using a HACCP (Hazard Analysis & Critical Control Points)-based approach for management of (microbiological and other) risks. The basic principles of HACCP are to understand the system and the hazards/hazardous events that may challenge the system, provide some ranking of their (health) priority and to ensure that adequate control measures are in place and functioning. HACCP-based systems typically focus on good practice and even more specifically on ensuring that good practice is maintained at all times; it is a proactive system-wide stance, rather than one relying on 'failure' of final drinking water. It fits within existing quality management systems (i.e. ISO 9001 c.s.). HACCP is the well-established risk management tool that is used for food safety. The Codex Alimentarius (FAO/WHO code for food safety) defines HACCP as a system which identifies, evaluates and controls hazards which are significant for food safety [CODEX, 1997]. Although there are many aspects of drinking water that are similar to food, there are also differences. Based on experiences of water suppliers with HACCP, the control system has been refined and tailored for application in drinking water abstraction, treatment and distribution in WHO's Water Safety Plan. The Water Safety Plan is described in the Guidelines for Drinking Water Quality [WHO, 2004].

The principal components of the Water Safety Plan are:
System assessment to determine whether the water supply chain (from catchment through treatment to the point of consumption) as a whole can deliver water of a quality that meets the above targets.
Operational monitoring of the control measures in the supply chain which are of particular importance in securing drinking water safety.
Management plans documenting the system assessment and monitoring, and describing actions to be taken in normal operation and incident conditions, including upgrade and improvement of documentation and communication.

In the Water Safety Plan the risk assessment question: "Do we meet the health target?" is answered in the *System Assessment* and the risk management questions "How do we ensure and demonstrate that we always meet the target?" and "How do we respond to incidents?" are answered in the *Operational monitoring of control measures* and the *Management plans.*
For an overview of the Water Safety Plan and its context, the reader is referred to the WHO GDWQ and the Water Safety Plan guidance document [Davison *et al.,* 2005] that are published on the website of WHO Water, Sanitation and Health. The guidance document describes the steps of the risk management approach, illustrated with several case studies of drinking water utilities that have applied this system to their water supply systems. A Water Safety Plan Portal with information on water safety plans can be found on the same website.

2.2.3 Public Health Status

The primary objective of drinking water safety management is the adequate protection of public health. The incidence of waterborne illness in the population or the occurrence of waterborne outbreaks is a direct trigger for curative risk management. A more preventative incentive for assessing the water-related health risks and the installation of risk management is to demonstrate that the water supply is providing an adequate level of protection of public health.
The inclusion of health targets in national legislation and the risk management actions of water utilities should result in an improvement of the public health status. Without addressing this, it is impossible to see if the health targets set and risk management actions taken are effective and if money spent for improving water supply results in a relevant health gain. This step in the circle is the place where the health risk of drinking water can be compared to other routes of exposure and to other health risks. It allows comparison (harmonisation) of the effort and resources put into the provision of safe drinking water and resources allocated to manage other health risks.
The risk assessment and management framework is a process that can and should be run in an iterative manner. This tiered approach fits well with the incremental nature of health decision making, the efficient use of scarce resources and the increase of information.

2.2.4 Risk assessment

Risk assessment is used to answer the question: "is my system able to produce and deliver drinking water that meets the health targets?". The risk assessment process requires quantitative information about the exposure of drinking water consumers to pathogens. This is provided by exposure assessment, one of the components of risk assessment. Quantitative information about pathogens in water sources, their removal by treatment and protection of the distribution network and drinking water consumption is collected and translated into an estimate of the exposure of consumers to pathogens through drinking water. To complete the risk assessment, the potential effect (the risk) of pathogen exposure is estimated through known dose-response models. As will be shown later, the exposure assessment also provides valuable information to aid risk management in the prioritisation of control measures.

An important question in risk management, especially in the European setting with an already high standard of drinking water safety, is "How far do we need to go with control measures?". This is an optimisation between the safety of and the costs for the consumer of drinking water.
Quantitative microbial risk assessment (QMRA) can provide an objective and scientific basis for risk management decisions. Water utilities can use QMRA to assess if they meet the health targets with their water treatment, storage and distribution systems. QMRA should also be used to provide information for setting critical limits in the Water Safety Plans to ensure good performance. Good performance can now be based on a quantitative assessment of the contribution of the Critical Point (such as a

disinfection or filtration process) to the overall safety and the limits can be set to ensure that the multiple barrier chain of water collection, treatment and distribution as a whole does meets the desired health target.
Risk assessment and risk management should not be regarded as two separate steps in the harmonised framework. To answer the question: "Which control measures should be put in place to meet the target?", both the HACCP-based management system and quantitative risk assessment provide valuable input: for example, the hazardous events, the most important barriers in the system, the contribution of each of the barriers, target levels for control, the occurrence of weak elements in the chain, the quality of the available information etc.

2.3 THE EUROPEAN DIMENSION

The European Commissioner responsible for health and consumer safety stated “the Commission needs to find the balance between the freedom and rights of individuals, industry and organisations with the need to reduce the risk of adverse effects to human health and the environment. This balance should be science-based, proportionate, non-discriminatory, transparent and coherent and requires a structured decision-making process with detailed scientific, objective information within an overall framework of risk analysis.” [Address by D. Byrne on the Precautionary Principle in the domain of human health and food safety. The Economist conference, Nov. 9, 2000, Paris]. Promotion of such an approach has been priority for the Commission, who played an active role in the field of food safety to obtain European and international acceptance for risk analysis principles. This is illustrated by the White paper on food safety produced during 2005 by the EC and the adoption of a “modern, dynamic and effective legal framework for food safety, based on robust science”, based on risk assessment. With such an approach, the use of the Precautionary Principle (as described in the Commission Communication in 2000:1 (COM 2000)) can be based on a quantitative assessment of the risk of pathogens in drinking water to human health in EU Member States. This risk can be compared to other risks and the policy of the EC to safe drinking water can be proportionate to the level of risk, consistent with other areas of consumer safety, non-discriminatory, based on cost-benefit assessments, transparent and indicates where more scientific evidence is necessary to reduce the uncertainty in the assessment of risk.
Activities are ongoing to harmonise the different aspects of risk assessment procedures (as outlined in the Commission report *The Future of Risk Assessment in the European Union*). The EU research project MicroRisk provides guidance and scientific basis for the introduction of the risk analysis principles in the area of microbial drinking water safety. This project has provided the content of this book.

In the EU-seminar about the Drinking Water Directive (DWD) in October 2003, the risk-based approach was presented as a position paper and discussed by many different stakeholders in the drinking water industry. The main conclusions concerning the value

and acceptability of the risk-assessment & -management (RA/RM) approach are cited here:
"Incorporation of risk assessment and management strategies are of large added value for the DWD and for safeguarding the supply of safe drinking water that keeps the trust of consumers in the European Member States. The core principles given by WHO's Framework of Safe Drinking Water are in good agreement with the principles used by the EU in other areas of consumer safety, and are thus seen as a sound basis to be included in the revision of the DWD. For many water suppliers RA/RM is already common practice, but a more consistent approach formalises existing practices and makes them more rigorous and transparent."
From all stakeholders present at this seminar "there is broad support for the overall concept and the core principles of the RA/RM approach to be included in the revision of the DWD. Prioritisation of such an approach would be very helpful for accession countries. In a revision of the DWD the Water Safety Plans could be accommodated, where the EU should provide an overall framework of core principles and a knowledge base of health-based targets and Member States (in line with the Subsidiarity Principle) should implement programmes and plans that are consistent with the overall framework".

2.4 QUANTITATIVE MICROBIAL RISK ASSESSMENT

In the Water Safety Plan, a principal question in the *System assessment* is: "Does the drinking water supply system meet the health targets?". The answer to this question can be given by a quantitative microbial risk assessment (QMRA). QMRA of fecal pathogens typically quantifies the potential risks arising from pathogens in source water, the impact of the source protection and treatment system in reducing pathogen concentrations and the risks of recontamination during distribution.
QMRA gives a detailed breakdown of the contribution of each step in the chain from catchment-to-tap to the overall risk (reduction), along with the potential effects of hazardous events (such as those following heavy rainfall) and some indication of data variability and uncertainty.. The water supplier can use this information to decide where optimisation or additional control would be most effective. Hence, QMRA is also a tool to guide the risk manager to efficient control.
QMRA can be used on existing water supply systems to determine if these meet the health targets, but also on hypothetical systems to evaluate if design scenarios are potentially able to meet the health targets.

In the next paragraphs, a short description of the process of quantitative microbial risk assessment is given. For a more comprehensive description the reader is referred to Haas *et al.* [1999], Teunis *et al.* [1997], Haas & Eisenberg [2001], the ILSI framework [Benford, 2001; Teunis & Havelaar, 1999] and Medema *et al.* [2003]. Chapter 7 gives a description of the statistical methods used for QMRA in this document.

2.4.1 The process of QMRA

QMRA is derived from the chemical risk assessment paradigm, that encompasses four basic elements:

- a characterisation of the problem setting (system description), including indentification of hazards and hazardous events
- exposure assessment
- effect assessment (dose-response)
- risk characterisation

Several QMRA frameworks have been published, such as the generic ILSI framework [Benford, 2001]. In this document, most attention is given to exposure assessment and risk characterisation. We have therefore expanded the generic ILSI QMRA framework to highlight the elements that are important for exposure assessment and risk characterisation, and we have put the expanded risk assessment in the overall WHO Safe Water Framework (Figure 2.2).

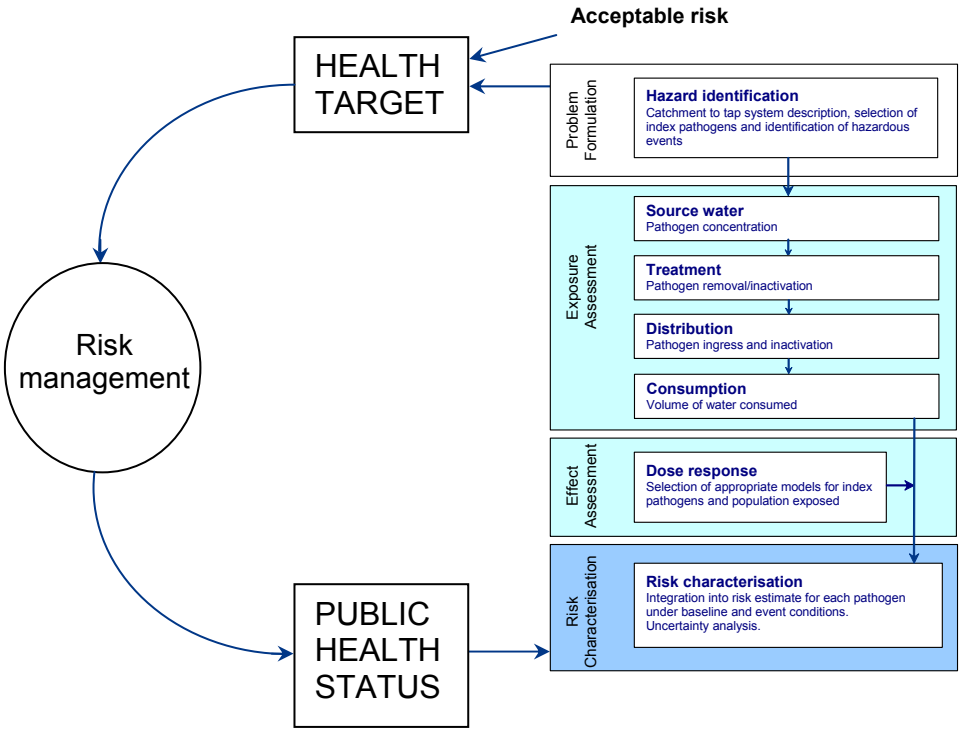

Figure 2.2. The steps of quantitative microbial risk assessment in the Safe Water Framework

2.4.2 Element 1. Problem formulation and hazard identification

This is the initialising phase of QMRA to establish which specific questions need to be addressed. The scope and the boundaries of the QMRA process are determined in this phase. This requires communication between the risk managers (regulators, public

health agencies, water utilities) and the risk assessors. The basic questions to QMRA is: “Is my system able to meet the health targets?”.

To conduct a QMRA, a good description of the system under evaluation is necessary and the hazards and hazardous events need to be identified. In this document, we use the definitions of the Water Safety Plan; a *hazard* is a biological agent with the potential to cause an adverse health effect and a *hazardous event* is an event or situation that may lead to the presence of a hazard in drinking water, such as a peak contamination of pathogens in source water, a treatment failure or a cross-connection with a sewer line in the distribution network. For a definition of terms used in this document, see the separate glossary.

Step 1. Description of the system from source to tap
The system for water treatment from catchment-to-tap is described, identifying the principal control elements and strategies.

Step 2. Hazard identification
Hazard identification is the identification of the micro-organisms within the system boundaries that could cause human illness, the processes by which each micro-organism finally reaches the customenr via drinking water and the type of illness(es) possible [Haas et al., 1999]. QMRA of drinking water systems is usually focussed on the ingestion of enteric pathogens and the potential for gastrointestinal illness.
The ideal QMRA does not focus on a single pathogen only, but on a suite of "index pathogens” that cover the range of health risks and control challenges for the particular water supply system defined. Adequate control of these index pathogens implies that the health risk of other known pathogens is also adequately controlled by the system and that the system also offers protection against unknown pathogens.

- Describe the characteristics of the pathogens, especially those related to waterborne transmission (survival in water, resistance to treatment etc.).
- Describe what is known about waterborne transmission, the causes of waterborne outbreaks and the relative significance of waterborne transmission compared to other routes.
- Describe the illness (type, duration, incubation time etc.) caused by the pathogens in the risk assessment, and when available information about sequellae. Describe what is known about protective immunity and secondary transmission.

Step 3. Description of hazardous events
In many cases, the majority of the risk is not determined during the normal (baseline) situation, but during hazardous events, such as rainfall leading to a high load of pathogens in source waters, or treatment failure or distribution network failure (or combinations thereof). It is therefore important to ensure that these hazardous events are incorporated in the QMRA, or that a separate QMRA is conducted to determine the (health) significance of the event.

2.4.3 Element 2. Exposure assessment

Exposure assessment is the quantitative assessment of the probability that drinking water consumers ingest pathogens through this drinking water. In QMRA of drinking water, this usually requires the assessment of the levels of pathogens in source water and the changes to these levels by treatment, storage and distribution, and finally the volume of water consumed.

Step 4. Assess pathogen occurrence in source water
Information about the occurrence of pathogens in source water is preferably based on a catchment survey, identifying the principal sources of contamination of the catchment and the conditions that may lead to peak events in source water, such as heavy rainfall or resuspension of sediments. Pathogen monitoring in source water can be carried out, using the information of the catchment survey, which needs to include assessment of peak events. The pathogen detection methods are ideally targeted to viable and infectious pathogens. The performance characteristics of the available detection methods for pathogens and other quality control aspects can have implications for the applicability of the data in risk assessment. These should be identified and evaluated in (the early stages of) the risk assessment process.

Step 5. Assess the elimination of pathogens during treatment
Information about the removal or inactivation of pathogens during drinking water treatment processes ideally involves data on removal of pathogens at full scale. In practice however, several other sources of data have to be used to estimate pathogen removal, such as pathogen data of pilot- or laboratory-scale systems or data on model parameters (indicator bacteria, phages, spores, particles etc.) on full-, pilot- or laboratory-scale.
The efficacy of treatment processes may vary, depending on feed water composition, operational control, temperature etc. Moments or periods of poor or suboptimal performance are hazardous events and hence most significant for risk assessment.

Step 6. Assess the changes in water quality during storage and distribution
The likelihood of recontamination of stored and distributed water (e.g. by the *E. coli* monitoring of water in these reservoirs and pipes or loss of disinfectant residual) and the significance of these contamination events needs to be assessed. In the European setting, recontamination events are rare and could be regarded as a result of a hazardous event (heavy rainfall, cross-connection, poor hygiene during repairs etc.).

Step 7. Consumption of drinking water
The last component of exposure assessment is the volume of water consumed by the population. Not only the average volume of water consumed is important, but also the person-to-person variation in consumption behaviour and especially consumption behaviour of risk groups (in terms of sensitivity to infection or high level of consumption) is relevant. The available data suggest that there is considerable difference between drinking water consumption within the population. This variation needs to be captured and incorporated in the risk assessment. Household

treatment/point-of-use devices affect the exposure. Hence, consumption data should be on consumption of drinking water without further treatment, such as heating, filters etc. Within Europe, consumption of tap water may vary from country to country. The significance of these differences for risk assessment need to be assessed.

Step 8. Dose (exposure) estimation
Dose (or exposure) is the number of pathogens consumed per unit time. The information obtained in all the previous steps of the exposure assessment are used to estimate this ingested dose. Preferably, he dose is described stochasticly, including the variability and uncertainty in all steps of the exposure assessment.

2.4.4 Element 3. Effect assessment

The effect assessment is the determination of the health outcomes associated with any (level of) exposure to waterborne pathogens.

Step 9. Dose-response data
Dose-response characterises the relationship between dose magnitude, pathogen infectivity, and quantitative health effects to an exposed population. The microbial dose-response analysis records the incidence of a particular effect against dose of the agent. In most cases, this particular effect is infection, rather than symptoms of illness. For *Cryptosporidium parvum* for instance, there is a clear relationship between ingested dose and the probability of infection, but not between dose and symptoms of intestinal illness.
Although the data-set is increasing, the number of dose-response studies with human volunteers is limited. Of most pathogens, only one or a few strains have been tested in healthy adult volunteers. Information about strain-to-strain variability and the influence of the immune response of the hosts is still limited.
There are several dose-response models available and the type of model can have a very significant impact on the response that is attributed to exposure at low doses. The models and their limitations should be well understood when applying these in QMRA. Synergistic effects between pathogens is not incorporated in the current models.

Step 10. Host Characterisation
For infectious diseases, the host susceptibility plays an important role in the health outcome of exposure. Exposure of persons with protective immunity will result in lower health outcomes than exposure of risk groups. During “Host Characterisation” the characteristics of the potentially exposed populations that are suspected for susceptibility to a particular pathogen are evaluated.

Step 11. Health outcome
Up to now, quantitative microbial risk assessment is primarily focussed on estimating the risk of infection. The relation between ingested dose and infection is relatively well-defined, while the relation between dose and other health outcomes (illness, sequellae)

is not available or less clear. This is one of the reasons why it is difficult and not recommended to establish a direct relationship between QMRA (on probability of infection) and epidemiological data (on symptoms of disease). The use of the risk (or probability) of infection is justified by the degree of conservatism in using infection as an endpoint and the inability to quantify the risk of more susceptible sub-populations [Macler & Regli, 1993]. Further, infected individuals as well as those ill can pass on pathogens to others (secondary spread).

However, waterborne diseases do differ in nature, severity and duration. A metric that takes the overall health burden of waterborne diseases is necessary. Ideally, this metric can also be used to describe the burden of the disease of chemical compounds, such as carcinogens, so all different health risks can be compared on the same scale.
In the new WHO GDWQ, the concept of Disability Adjusted Life Years (DALY) [Havelaar & Melse, 2003] is introduced as the burden of disease metric in the drinking water guidelines.
The basic principle of the DALY approach is to weigh each health effect for its severity with (usually) death as the most severe outcome, multiply this weight (a factor between 0 and 1) with the duration of the health effect ('duration' of death being the remaining group life expectancy), and with the number of people in a population affected by the particular outcome. Summarizing all the health outcomes caused by a certain agent, will result in an estimate of the burden of disease attributable to this agent.
To be able to use DALY's in the QMRA, ideally the relation between exposure (dose) and different health outcomes is known. In the absence of sufficient data (which is usually the case), the dose-response relation for infection (as the first step of the disease process) can be combined with data on the fraction of the exposed population falling ill from exposure (for instance from attack rates in waterborne outbreaks) and data on the fraction of the ill population that contract more severe health outcomes (from health surveillance data).

2.4.5 Element 4. Risk characterisation

In the process of risk characterisation, the information obtained in the exposure assessment and the effect assessment are integrated to obtain a risk estimate. This can be done as a point estimation: a point estimate of exposure can be entered into the dose-response relation to compute a point estimate of the risk of infection. The point estimate can be the 'best' estimate, to obtain a measure of central tendency of the risk. In the case of computing various risk scenarios, the computed point estimates give a quantitative estimate of the consequences of the circumstances that produce a risk scenario.
An stochastic approach that allows the incorporation of the variability and uncertainty in the steps of the risk assessment chain is promoted by Haas [1997] and Teunis *et al.* [1997]. This encompasses the characterisation of the distribution of all data used for risk assessment and to combine these distributions into a distribution of the computed risk, for instance by Monte Carlo analysis. This approach not only provides the risk manager with important information about the (un)certainty of the risk estimate, but also with the relative contribution of the uncertainty and variability in all steps of the

risk assessment. It therefore guides the risk manager to the most appropriate options for efficiently minimising the risk and the most significant research items to reduce the overall uncertainty of the risk estimate.
With high level water supply, the baseline risk is usually very low. Under such conditions, hazardous events, such as peak contamination in the source water, treatment failure and especially the combination thereof and contamination events in the distribution network, are responsible for the majority of the risk. Most waterborne outbreaks have been traced to a (combination of hazardous events (see Chapter 1) and it is likely that many events result in the presence of pathogens in tap water and hence the transmission of disease. Wherever possible, identify and evaluate these events separately in QMRA to understand the significance of these events. Analysis of events also brings forward opportunities for optimisation of the system to prevent these events from occurring or to reduce their impact on human health.

2.4.6 Tiered approach to QMRA

QMRA lends itself very well for a tiered approach and this is also commonly used in risk assessment practice, both in human health risk assessment and in ecological risk assessment. The tiered approach allows an effective interaction between risk assessment and risk management, starting with a crude risk assessment, usually based on limited information to determine the urgency of the perceived problem, to prioritise the risk of different water supply sites or scenarios and to determine the need of a more detailed study for a particular situation. This allows the effective allocation of resources to the sites or situations that give rise to the highest risk. There is no a priori definition of the tiers, only that the initial QMRA is usually generic and simple and the specificity and complexity increase in subsequent tiers.

The most basic QMRA is a screening-level study. Starting with whatever information is available, a crude first evaluation is made. Usually, the available information is not specific to the system that is studied, but has to be extrapolated from the available scientific literature.
The screening-level assessment may show that the risks are negligible, without much scientific doubt. In that case, the screening-level risk assessment can be used to demonstrate the safety of the system and obviating the need for further, more detailed assessment. Alternatively, the screening-level risk assessment may imply that the risk is unacceptably high, again without much scientific doubt. In that case, the screening-level risk assessment is used to justify the installation of additional control measures. Such a screening-level risk assessment is also very useful in comparing different scenarios for risk management, e.g. different water treatment options.

If the outcome of the screening-level risk assessment is that there may be a health risk that is not negligible, there is an incentive for a next iteration of the risk assessment, collecting site-specific data, for instance on the presence of pathogens in the source water or catchment. The QMRA is repeated with the new, site-specific information. The options for the outcome of this second-tier QMRA are the same as for the first

iteration. In general, one of the results of any risk assessment is the identification of which information is missing and the prioritisation of research needs [Gale, 2002].

The screening-level risk assessments usually work with point estimates of risk. The tendency is to use conservative or worst-case estimates, to "be on the safe side". But worst-case estimates, by nature and especially when used in combination, may severely overestimate the risk and it is not clear to the risk manager what the uncertainty of the calculated risk is, only that the uncertainty will be towards the lower risk values (the nature of a worst case assumption). More helpful for the risk manager is to provide a range of risks (interval estimate) that denote the variability and uncertainty in the risk estimate. In the case of the screening-level risk assessment this can be achieved by using an average, worst and best case, to illustrate the range of the risk that can be deduced from the available information and the level of certainty that is embedded in the QMRA.

Interval estimates require information about the variability and uncertainty. Variability is the result of intrinsic heterogeneity in the input of the risk assessment, such as the variation in *Cryptosporidium* concentration in source water over time, or the variation in the removal of particles by a filtration process over time. Variability can be characterised if sufficient data points are collected. Uncertainty is the result of unknown errors in inputs of the risk assessment, such as errors in the measurement of pathogens or the assumption that certain indicator organisms can be used to describe the removal of pathogens by treatment. Uncertainty can be reduced or characterised by specific research activities, e.g. to determine the recovery efficiency of the pathogen enumeration method or to compare the removal of the pathogen to indicator organisms by a treatment process.
When sufficient data are available, a probabilistic (stochastic) risk assessment can be performed, where the input is described by statistical distribution functions to describe the confidence interval of the input itself and of the calculated risk.

The tiered approach is used throughout this book, in Chapter 4 on treatment and Chapters 7 & 8 on risk assessment, where the reader can find several examples.

2.4.7 Risk assessment using epidemiology

The Water Safety Plan guidance document [Davison *et al.,* 2005] highlights the possibility to evaluate the performance of a water supply against health targets with an epidemiological approach. Epidemiology has a set of tools to *assess* (an estimate of) the *actual* health risk of a population and has an important role in the safe water framework, especially in the assessment of the Public Health Status and the assessment of the relative significance of the different pathogens and routes of exposure. In addition, epidemiological studies of waterborne outbreaks provide information about the events in which an outbreak may occur and are therefore very important to guide QMRA and the HACCP-based system to hazardous events. The reader is referred to

Blumenthal *et al.* [2001] and Hunter *et al.* [2002], for an overview of the use of epidemiology to assess waterborne health risks.

For assessment of the health risk of drinking water supply in Europe, epidemiological tools are less applicable in practice, as the level of safety required for drinking water exceeds the level of sensitivity of (affordable) epidemiological studies. In Australia and the USA, double-blinded, randomised case control studies have been undertaken to determine the contribution of mains drinking water to the overall incidence of gastro-intestinal illness [Hellard *et al.,* 2001; Colford *et al.,* 2005]. Both studies did not show a significant relation between tap water consumption and intestinal illness. Even though these were large studies, they could only demonstrate that tap water contributed less than approx. 10% to the overall incidence of gastro-intestinal illness.
QMRA is more sensitive, but generally requires assumptions (for instance on infectivity of pathogens in water). QMRA is therefore an appropriate tool to assess the *potential* health risk of water supply systems, but less appropriate to assess the *actual* health risk of drinking water consumers.
Comparison between epidemiology and QMRA can be done in conditions in which epidemiological studies are sensitive enough to determine the risk, such as waterborne outbreaks where infection is assessed, and sufficient information on the water system is available to perform a QMRA. In practice however, the comparison is hampered by differences in health outcome; QMRA generally uses risk of infection, while epidemiology generally uses illness type, but comparison of the level of risk estimated by both means does give insight in the validity of QMRA.

2.5 RISK MANAGEMENT

In the EU, many drinking water supplies provide adequate and safe drinking water and have introduced quality management. What is the value of the Water Safety Plan in such a context? The experience of several pioneering water suppliers and the result of the discussions on this subject at the WHO [Water Safety conference, Berlin, April 2003], EU [EU Drinking water seminar, October 2003] and at the national level indicate that formal adoption of a Water Safety Plan and associated commitment to the approach has a number of significant benefits. As stated in the current WHO Water Safety Plan: "Major benefits of developing and implementing a water safety plan for these supplies include the systematic and detailed assessment and prioritisation of hazards and the operational monitoring of barriers or control measures. In addition, it provides for an organised and structured system to minimise the chance of failure through oversight or lapse of management. This process ensures that safe water is continually supplied and that contingency plans are in place to respond to system failures or unforeseeable hazardous events."
Here, the steps of the HACCP-based risk management approach in the Water Safety Plan are briefly described.

2.5.1 Element 1. System assessment

Step 1. Assemble team and other resources

As the starting point, the management incentives are needed and a multi-disciplinary team should be assembled involving managers, engineers (operations, maintenance, design, capital investment) water quality control (microbiologists and chemists) and technical staff involved in the day to day operations with good knowledge of the system and of the safety hazards in the drinking water to be anticipated (Figure 2.3).

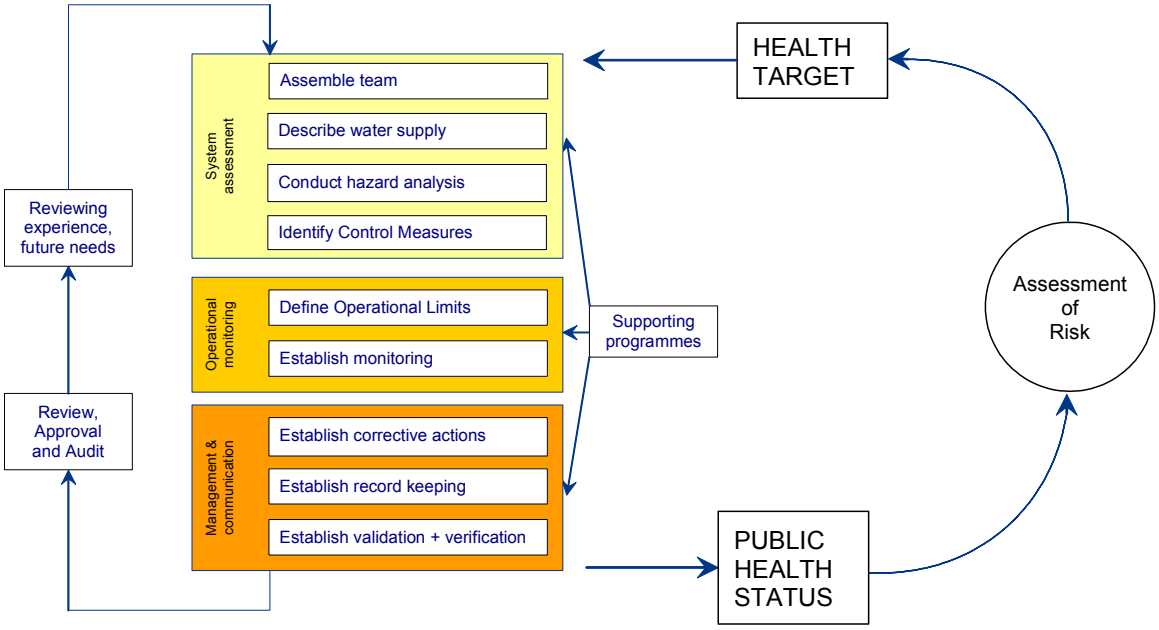

Figure 2.3. The steps of the Water Safety Plan in the Safe Water Framework

Step 2. Describe water supply

The team will start by preparing a description of the water supply system. This should include the catchment, source water reservoirs, water treatment processes, storage after treatment, water distribution and safe handling during household storage of water and treatment at point of use.

It is also important to describe how the water is going to be used and which routes of exposure to the water may occur. In the case of drinking water this is generally intended for human consumption and other household uses. Are there special considerations for vulnerable groups such as infants, elderly and immunocompromised? This information is very important because it will be used to determine the potential risk of water exposure.

To enable hazards to be clearly identified it is important to develop system-specific flow charts to describe all the processes involved at each step. The WSP team should confirm that the representation of the system in the flow diagram is accurate and complete. This is important as the flow diagram is the basis for the hazard identification and tracing its potential pathways to the consumers.

Step 3. Hazard analysis

Step 3a. Identify hazardous micro-organisms
The WSP team should consider all hazardous micro-organisms (and indeed substances, but these are no part of this document) that could be associated with the water supply system under study.

Step 3b. Identify hazardous events
Identify events that may result in the presence of a hazard (in this document an enteric pathogen) in drinking water. Biological hazards (bacteria, viruses and protozoa) generally originate from contamination of water with human or animal faeces, although opportunistic bacterial and protozoan pathogens may also develop in distributed water under specific conditions. In general, faecal contamination may be used as the primary starting point for the identification of most hazardous events.

Step 3c. Prioritise hazards for control
In any system, there may be many hazards and hazardous events and potentially large number of control measures. Priorities for control measures therefore need to be defined. Prioritisation matrixes are tools to rank control measures, to provide a focus on the most significant hazards. By using a semi-quantitative risk assessment the priority score for each identified hazard/event is calculated within the need to determine the actual risk. The *likelihood* and *severity* for each risk can be calculated and a cut-off point above which all hazards are taken into consideration is established. A QMRA provides the soundest basis for prioritisation, but requires sufficient quantitative information about the probabilities of pathogen exposure during hazardous events.

Step 4. Identify control measures
"Control measures" or "barriers" are any activity that can reduce levels of hazards within water either by reducing their entry, concentration or by reducing their proliferation. The so-called "multiple-barrier-principle" is the basis for a WSP plan. The safety of drinking water cannot be warranted by a single barrier or control measure, but only by a suite of control measures in the whole supply chain from catchment to consumer. This includes control measures in the catchment, the water collection, treatment and distribution system and the domestic installation of the consumer.
Control measures in the catchment should prevent hazards entering the water supply chain. This is in line with the European Framework Directive and draft of the Groundwater Directive. Guidelines/Codes of practice on how to define drinking water protective areas are available (DVGW W 101/102 “Protective Areas for Groundwater, reservoirs”).

For some engineered control measures (i.e. treatment processes) limits for operational acceptability can be defined, and operation can be monitored directly or indirectly (step 6). Examples are ozonation, of which the efficacy can be monitored with ozone residual, contact time and water temperature or ultrafiltration of which the efficacy can be monitored by particle counting. Other control measures cannot be monitored in a

similar fashion but are still equally important. Examples are a catchment protection programme or the Operation Procedures for maintenance of distribution networks that include hygiene considerations. Adherence to these are important control measures, and therefore part of the Water Safety Plan. These control measures can be considered in Supporting Programs (Step 6).

2.5.3 Element 2. Operational monitoring

Step 5. Define operational and critical limits
For control measures, operational and critical limits are established. Limits are set for parameters that can be monitored or aspects that can be observed and give information about the adequacy of the control measure. A Critical Limit (CL) is a performance target that, if exceeded, indicates that the ability of the supply to meet the water quality targets is compromised. This requires immediate actions to correct this.
In current water supply practice, operational limits are usually set in addition to critical limits. Operational limits are set for the same parameters as the critical limits, but the operational limits are stricter and trigger remedial actions (for example increase of the disinfectant dose when the residual disinfectant concentration is too low), before the control measure is reaching or passing its critical limit. Current knowledge and expertise (industry standards, technical data and locally derived historical data) can be used as guide to determine the limits.

Step 6. Establish monitoring system
Monitoring are all the actions of conducting a planned sequence of measurements or observations of control parameters to assess whether a control measure is operating properly. Some control measures, such as many treatment processes, allow monitoring systems for process or water quality parameters that indicate the efficacy of control (such as disinfectant residual, UV-intensity, turbidity, particle counts etc.). The use of automation in control of treatment processes is increasing rapidly in water supply companies. The use of SCADA (Supervisory Control and Data Acquisition) system uses on-line measurement systems that collect data on treatment performance very frequently. These systems have critical limits put in to guide control or raise an alarm for the operator. To date, these limits are not based on a quantitative assessment of the contribution of the treatment process to the overall safety of the system, but on rules-of-thumb/experience. QMRA has the ability to base the limits on a quantitative science-based assessment (see Chapter 8).
Other control measures require a different type of monitoring. Examples are inspection of hygienic maintenance operations of the distribution network, inspection of the integrity of infrastructure (storage reservoirs etc.).
If monitoring shows that an operational or critical limit has been exceeded then there is the potential for water to become unsafe. Monitoring should be performed according to a statistically valid sampling plan (particularly including event conditions) to prevent the supply of any potentially unsafe water.

Microbiological assays, such as for indicator organisms (*E. coli* and others), are generally still too slow and infrequent to guide for process control, hence form part of system verification (Step 9b). With the developments of molecular methods and lab-on-a-chip techniques the possibility of on-line microbial monitoring comes closer, but these methods are currently too insensitive for use in drinking water practice to monitor treatment performance or distribution integrity.

2.5.3 Element 3. Management & communication

Step 7. Establish corrective actions

Corrective actions are the actions taken when the results of monitoring indicate a loss of control. It is necessary to detect deviations through monitoring and respond through corrective action to prevent unsafe water being supplied. The corrective action will protect water safety by bringing the control point back into specifications by enhancing the control point or by implementing additional control measures. All these actions should be completed in a sufficient time frame adequate to maintain water safety.

In some cases, significant deviations occur in control measures that are outside of the scope of corrective actions. Such unpredictable incidents occur occasionally and require an incident response. The use of backup disinfection plants or spot dosing may be used to correct disinfection system failure within the water supply. By ensuring that a contingency is available and promptly applied in the event of an operational or critical limit being exceeded, safety of supply can be maintained.

Incident and emergency (natural disaster, deliberate contamination etc.) response plans are necessary to ensure the provision of safe drinking water under these conditions.

Step 8. Establish record keeping

Types of records that can be kept are support documentation for developing the WSP, records generated by the WSP system, documentation of methods and procedures used and records of employee training programs, all part of ISO reporting procedures**.**

Step 9. Establish validation and verification

Step 9a.Validation

Validation is an investigative activity to identify the effectiveness of a control measure, typically when a system is designed or rehabilitated. It is applied to ensure that the systems used in the WSP are effective and controls the hazard. Evidence to support the WSPs can come from a wide variety of sources such as scientific literature, trade association, regulation and legislation departments, historical data, professional bodies or supplier knowledge. An example is a UV system that is needed for a three log *Cryptosporidium* inactivation. The information on *Cryptosporidium* inactivation by UV is collected from the scientific literature and the dose delivery of the UV system is validated according to national standards/guidelines. Alternatively, challenge testing is applied to the full-scale barrier being validated, such as sulphite-reducing clostridia removal across a sand filter as a surrogate for *Cryptosporidium* oocyst removal.

Step 9b. Verification
Verification is the final check of the safety of the water supply system. For microbiological safety, verification is typically the monitoring for faecal indicators in treated water and in distribution. Traditionally *Escherichia coli* is used for verification monitoring.
Since *E. coli* is more sensitive to disinfection than viruses and protozoa, additional parameters, such as *Clostridium perfringens* and bacteriophages, may be needed for adequate verification.

2.5.4 Support programmes

Adequate training of personnel, involvement of all stakeholders in the provision of safe water, the development of technical standards for good operation or monitoring methods are all examples of supporting programmes that are relevant for the provision of safe water, but do not affect water quality directly. Many of such programmes are already present in water supply companies in the EU. The WSP should be composed in co-ordination with these programmes.

2.6 THE LINKS

At various steps in the HACCP-based process, questions emerge that relate to the balance between safety and costs of the water supply system. More safety can be obtained by including additional control measures, by setting very strict limits, by intensive monitoring etc. However, resources are not unlimited and drinking water is not the only transmission route for pathogens and toxic compounds that needs to be controlled. In the European setting, drinking water safety is well established and other routes (food, recreational water) of exposure are much more important for consumer health.
QMRA provides information for efficient allocation of resources to water supply. By setting health-based targets based on the contribution of drinking water to the overall health risk of the human population, it becomes clear when *safe* is *safe enough.* Links between QMRA and WSP are illustrated by the questions it answers in Figure 2.4.

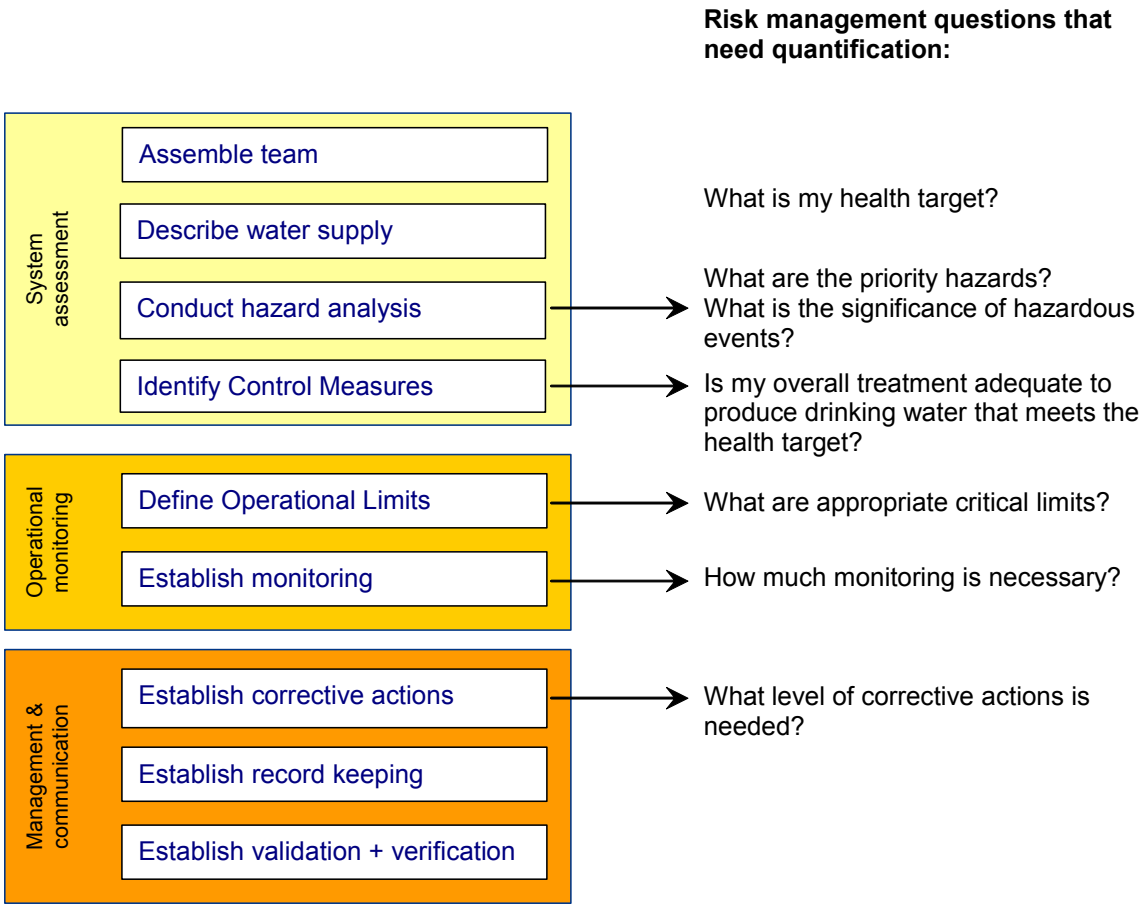

Figure 2.4. Risk management questions that relate to the balance between safety and costs that can be answered by QMRA

2.6.1 Link 1: Health targets

Setting of a health target

This link is already represented in the overall framework (Figure 2.1). The risk assessment is used to determine the risk related to drinking water. The risk estimate and the level of risk that is considered tolerable in relation to drinking water is translated into a health target. A health target is generally a tolerable disease burden (1µ DALY.y^{-1}) or annual infection risk (10^{-4}.y^{-1}), but can be translated into a water quality target or performance targets [see also WHO GDWQ, 2004]. Setting the health target is the responsibility of the regulator and the target they set for drinking water is the starting point for risk management by the water supplier. They need to design, operate, control and maintain their system in a way that ensures that the health target is met at all times.

Complying with the health target

At the water utility level, a QMRA can be conducted to answer the question: "Do we meet the health target?". It is the responsibility of the water utilities to meet the health-

based targets and to demonstrate to the regulators and the public that these targets are met. During the HACCP-based process the risks are approached in a semi-quantitative manner (high, medium, low etc.), based on experience, industry standards and subject to personal interpretation. In many cases, this is sufficient information for risk management; i.e. it is clear that a well-head that is not properly closed may give rise to contamination of the water from the well and the corrective action will be to close the well-head properly. In these cases, there is usually no further quantitative assessment of the risk of contamination necessary to trigger the appropriate corrective actions.
However, this does not answer the question whether the overall water supply system from source-to-tap provides safe drinking water to the consumer. A quantitative microbial risk assessment of a drinking water system can demonstrate that the health-based targets are met. In the European setting, water supply systems are usually well-developed, operated and maintained. The question is there "Are more risk management measures necessary or is the system safe enough?". QMRA can answer this question and provide justification that sufficient resources are allocated to the provision of safe drinking water to the consumers.
A QMRA (in the WSP: System assessment) is therefore the logical first step when safety of a water supply system is under consideration. The outcome of this assessment will be the basis for further development.
If the outcome indicates that overall system is adequate to provide the consumers with safe drinking water, the HACCP-based process can be used to guarantee this safety is met under all conditions.
If the outcome of the assessment indicates that the drinking water could be unsafe under some conditions, the water supply system (management) needs to be adapted. The effect of different solutions can be investigated by using the QMRA as a scenario-study tool. Feeding the alternatives into the QMRA will help to identify the most economic, sufficiently effective measure to bring the risk within the health based targets. These measures can be either physical (covering clean water reservoirs, new treatment processes), operational (new critical limits) or management measures (reducing human or domestic animal activities in catchments).

2.6.2. Link 2: Hazardous events are risk events

Hazard identification to guide QMRA to risk events
In the HACCP-based system, hazards and hazardous events are identified and prioritised. These hazardous events are significant information for risk assessment as they may comprise most of the health risk. Bartram *et al.* [2001] already identified that QMRA should not only be directed at the nominal performance of treatment systems, but also at the moments of poor source water quality and treatment performance. Knowledge about hazardous events and their probability of occurrence can be used in QMRA as risk scenarios (see Chapter 8).

QMRA to guide Hazard identification to risk events
Similarly, during exposure assessment information is collected about occurrence of pathogens in source waters, treatment efficiency and distribution system integrity. This

may yield information about peak events in source water, moments or periods of suboptimal treatment performance and distribution integrity breaches, and thus about hazardous events. This can be used in the process of hazard identification and prioritisation.

Objective risk priorities with QMRA

In the HACCP-based system, fault trees and Risk Factor Matrices are used to provide a focus on the most significant hazards and hazardous events. The priorities are set on the basis of expert judgement and historical data. Several ways to prioritise hazards in this semi-quantitative manner are described by Davison *et al.* [2002]. The estimation of occurrence and effects is subjective to personal knowledge and experience of the WSP-team members. Therefore hazards that have already occurred are likely to be weighted more heavily than yet unknown hazards. This could lead to high unnecessary investments or overseeing relevant risks.

QMRA can be used for quantitative estimates of the different routes of contamination, improvement against a major transmission route is most important as long as it is still major. Improvement of control over major routes (for instance improving surface water treatment) enhances the importance of minor routes and these minor routes need to be taken into consideration. An example is the ingress of contamination in the distribution network in an appropriately-treated water. Improving treatment may be less effective than reducing the probability of ingress in the network.

QMRA can also be used to determine the significance of "bad days" (temporal effect, periods of poor treatment performance): treatment efficacy varies, and the majority of the risk is associated with bad days, moments of poor treatment performance.

Similarly QMRA can establish the significance of "by-passes" of critical control points (spatial effect) such as one poorly performing filter in a set of parallel filters. If the performance of this one filter is severely compromised, the proper performance of the other filters does not compensate this.

HACCP can address the bad days but is less appropriate for assessing the minor routes and the by-pass. QMRA can help in addressing the important elements in the system.

Using QMRA to prioritise hazards will result in an objective, quantitative prioritisation of the hazards, provided there is sufficient quantitative information available.

QMRA can compare the risk of different hazards and hazardous events in alternative scenarios. Examples of this are:

- a surface water utility wants to focus the limited resources on monitoring of the most critical pathogen(s). A QMRA will establish the efficacy of the treatment system against the different pathogens and allow the selection of the pathogens that pose the largest control challenge, or
- the impact of a peak rainfall event in the catchment or of the failure of disinfection process can be determined quantitatively and hence objectively prioritised.

2.6.3 Link 3: Health target can be translated into target and critical limits

In the HACCP-based system, there is no direct link between the target or critical limits on system operation and the health target. The overall system needs to produce and deliver water that is safe. Safety is defined as meeting the health (or related water quality) target. Operational limits should be set at levels that ensures the treatment produces water that meets the health target. Target limits are criteria that indicate whether a control measure is functioning as designed. If monitoring shows that the target limit is exceeded, predetermined corrective actions should be put into operation to 1) ensure the system continues to meet the health target and 2) bring the control measure back into its limits. Exceedance of *critical limits* is more serious for microbiological water quality, since this means the system is not complying with the health target. This is often referred to as incident.
Setting of target and critical limits for operations may also have significant consequences for the cost of water supply; stricter limits will generally imply higher costs for catchment protection, treatment or distribution (maintenance). A sound basis for setting the limits at a level that optimises safety and costs is therefore appropriate.
Control charts are often utilised to track changes in performance against the *Target Limits*, as they provide a good visual clue to operators and assist in identifying a trend towards a potential problem before it occurs.
To reach appropriate target and critical limits for control measures, QMRA can be applied to ensure that the resulting water quality will always comply with the health targets.
Exposure assessment for QMRA provides information about the contribution of individual steps of the multiple barrier system to the overall exposure. In other words, the exposure assessment provides information about the relative contribution of the control measures to the overall risk estimate. With the health (risk) target as reference, the required contribution of individual control measures to produce and deliver drinking water that meets the health target can be assessed. This can be translated into critical limits for individual control measures.

Setting of appropriate target and critical limits is complex and may have significant impact on safety and costs. Arriving at the optimal limits will need several iterations, using practical experience and ongoing scientific insights to further improve the operation of the water system. Critical limits will depend on circumstances such as water temperature or source water turbidity. For complicated systems a real-time computer model of the water supply system (for disinfection and other water quality parameters) may be helpful in maintaining optimal water quality and choosing the most appropriate corrective measures.

2.6.4 Link 4: Designing monitoring programs

Monitoring will determine the period for which a possible failure of the water supply system may remain unnoticed. It is obvious that a longer exposure time will result in an

increased risk. However monitoring and verification will require resources and funds, and cannot be applied limitlessly. QMRA can provide validation of the monitoring plan, by determining the risk when the maximum period of (unnoticed) exposure is reached. Thus funds and resources can be divided in such a way that maximum safety for the consumers is warranted.

The monitoring results can provide information about source water quality, treatment efficacy and the integrity of the distribution system. This information is important input for the next iterations of the QMRA, as it provides information about the extend of variation in source water quality, efficacy of treatment processes and distribution system integrity. This is important to assess the level of certainty of risk estimates, but also to guide QMRA (and indeed HACCP) to hazardous events (how often does a peak contamination occur in source water and to what extend?; how variable is the efficacy of the disinfection process and under which conditions is the efficacy compromised?). In general, the first iterations will be based on expert knowledge and available data from literature and historical data on site, but as the WSP becomes implemented, more and more site-specific data will come available to improve both the HACCP-based and QMRA-process.

2.6.5. Link 5: Selecting corrective actions

Corrective actions
When target limits are exceeded, corrective actions are needed to keep the system under control. If critical limits are exceeded, urgent actions are required in order to prevent non-compliance with the health target and hence an increased health risk. Different levels of corrective actions may be undertaken. These could be restricted to the control measure that is out of bounds, but could also include other control measures that may be enhanced or even already working at a relatively high efficiency. QMRA can be used to determine to what extend exceeding the limits of the individual control measure is actually resulting in non-compliance of the system as a whole. If that is the case, QMRA can also be used to select the most appropriate corrective actions under the given conditions, as it looks at the system as a whole, rather than at individual control measures.
An example of such a situation is a groundwater system that is under the influence of surface water. Under nominal conditions, the passage of the surface water through the soil is slow and pathogens are effectively removed, indicated by the absence of indicators in the groundwater. During rainfall events, the situation is different, pathogen transport is rapid and the groundwater may become contaminated, as indicated by the presence of surrogates. UV could be installed to prevent the water of becoming unsafe under these adverse conditions. It is not possible to correct the efficacy of the soil passage during these events, but it is possible to enhance the UV as a reaction to this situation. The level of enhancement of UV can be tailored by the level of contamination found in the groundwater under such conditions.

Treatment design: comparing alternatives
During the design of a water treatment plant, or when changes to a treatment plant are required, one needs to choose between different solutions. Each (combination of) solutions needs to comply with the health based targets. A QMRA can help identifying the most economical alternative. Thus unnecessary investments can be avoided. Here, QMRA can be used as a design tool.

2.7 OUTLOOK

2.7.1 New iterative approach for safe drinking water

The Water Safety Plan is a 're-invention' of common sense that can be used by water utilities for efficient, comprehensive, transparent and documented risk management. The WSP has already been successfully piloted by water utilities in several countries outside the EU and is now being piloted within the EU. The WSP (re-)focuses the attention of the water utilities on controlling and maintaining the whole system from source-to-tap, rather than the focus on end-product monitoring. The WSP will also change the way various drinking water inspectorates and government will operate to ensure that health targets are met. Rather than looking at the monitoring data from the treated water, the inspectors/auditors can focus more and more on the success of the WSP.
Implementation of WSP will produce and document a wealth of data about the occurrence of hazards and hazardous events and the efficacy of the control measures to cope with these. The implementation should therefore be regarded as an iterative process in which more information becomes available in every cycle to improve the risk management process. Similarly, QMRA can be fed with more and more site-specific data to improve the reliability of the risk assessment.

2.7.2 The value of QMRA

Water suppliers that use the HACCP-based process are faced at several steps in this process with questions of a quantitative nature. The first question is:
- Is my system meeting the health-based targets?

This typically needs a quantitative risk assessment (System assessment (WSP)).
Other questions that require quantitative answers are:
- What is the priority of different hazards/hazardous events; so where do I focus my risk management on?
- Where do I set my operational and critical limits?
- How much monitoring is necessary?
- What level of corrective actions is necessary?

The answers to these questions are usually based on semi-quantitative expert judgements and industry or legal standards. QMRA provides more objective, science-based and quantitative information to answer these questions and hence a more precise

basis for risk management. This is particularly relevant in cases where the costs of (additional) control measures or corrective actions are high. In such cases, the high costs are an incentive to collect the quantitative information that is needed to perform a QMRA.

2.7.3 State of the art

Risk assessment allows comparison of the effort and resources put into the provision of safe drinking water against resources allocated to manage other health risks. However, given the current state-of -the-art and especially the lack of available quantitative data, QMRA of a water supply system has to rely partly on assumptions. Given the current level of uncertainty in quantitative risk assessments of drinking water supplies, the outcome should be regarded as an indication of the level of safety, rather than an absolute assessment of health risk. The outcome can be used to guide the risk management direction to pathogen control and to select the most appropriate control measures.
The benefit of risk assessment is that it gives a better understanding/breakdown of the problems and identifies what is important data. Additionally, the risk concept allows us to focus and prioritise research to the areas where important pieces of information are missing.

The large variability of pathogens in source waters and the limited availability of data (esp. in relation to peak events) and the variability in treatment efficacy are very important issues to take into consideration in QMRA. More data need to be collected and monitoring programs of water suppliers should be targeted more towards the provision of information for QMRA. The variability and limited data available will cause uncertainty in the risk assessment, but compared to chemical risk assessment with large uncertainty factors, this is not inhibitive for the implementation of microbial risk assessment, as illustrated in Chapter 8.

Pathogens to be selected for QMRA (and hence the MicroRisk project) should be detectable in source waters with reliable analytical techniques. The use of selected ‘index pathogens’, pathogens that are critical to determine if the control measures taken in water supply result in drinking water that meets the health target, are recommended. Control of these index pathogens would mean control of the other known (and even unknown) pathogens that behave in a similar way.

Most of the risk assessment in water supply is currently undertaken on large surface water supplies. The risk assessment framework should be applicable in many different situations in Europe; also in areas with high numbers of small supplies, in areas dominated by ground water sources, in tourist areas and recreational settings. Experience with the use of QMRA in these other areas is needed to evaluate the applicability under these diverse conditions.

2.7.4 Stakeholder participation

The water supplier

Water suppliers in Europe have implemented or are implementing several management systems that relate to Water Safety Plans; systems for quality management, systems for ensuring safety against deliberate contaminations, systems for ensuring continuous supply of drinking water, asset management systems, maintenance plans etc. In many of these systems, specifications are given for design of systems, operational procedures, maintenance, repair etc. When a Water Safety Plan is prepared, the links with the other systems should be established. The value of the Water Safety Plan is that the focus is on hazards/hazardous events, how these are controlled and how this control is warranted by monitoring programs and plans for response to system failures. Other management systems and current practice tend to focus on describing how things *should be* done, the Water Safety Plan focuses on monitoring that things *are* done and how they should be done.

The regulator

For implementation, the risk-based approach needs to be endorsed by the regulator of drinking water policy. The regulator needs to define the level of risk that is considered tolerable through drinking water. This is not new, water quality standards for several chemical compounds in the WHO Guidelines for Drinking Water Quality have been derived from a tolerable lifetime risk of 1 case of cancer in 100,000 people. Also the EU uses this approach in the Drinking Water Directive, only their tolerable risk level is 10 times stricter (1 cancer-case per 1 million people). However, for pathogenic micro-organisms, no such tolerable risk level is defined in the EU.

Ideally, a reference level of tolerable risk through drinking water is defined, incorporating the burden of disease, for all health risks, be it microbiological, chemical or otherwise. As stated earlier, the WHO is using the DALY's as a metric and has derived a new reference level of risk of 10^{-6} DALY's per person per year from its current tolerable risk level for carcinogens (<1 cancer-case per 100,000 people (lifetime risk)) (see WHO GDWQ, and the discussion in Chapter 7).

The need for a reference level of risk was highlighted at the EU drinking water seminar in 2003. Before the risk-approach can be implemented in the Drinking Water Directive, the EU needs to define this reference level of risk. The definition should be considered with great care and stakeholder consultation, especially the health authority, as the level of risk that is considered tolerable through drinking water has important implications for adequate health protection, consumer confidence and cost of water supply.

The regulator has a second role in the protection of the safety of drinking water. The water supplier is responsible for adequate control of the hazards and hazardous events that occur in the systems that they are controlling (abstraction, treatment, distribution). However, hazards originate from sources over which many water suppliers have no control. The discharge of treated or untreated sewage in the catchment, combined sewer overflows or agricultural practices that occur in the catchment result in the presence of pathogens at the sites where water suppliers abstract their surface water for the production of drinking water. Reduction of the pathogen load to surface water by additional sewage treatment, removal/relocation of overflows and the implementation

of protection zones around stretches of surface water that are vulnerable to surface run-off are all control measures that should be part of the multiple barrier approach in providing safe drinking water. Similarly, protection of groundwater is of primary importance. The increasing urbanisation makes it increasingly necessary to combine the water supply function of land with other functions. This combination should not compromise the safety of the water supply system.
As many outbreaks of waterborne disease have occurred due to an event in the catchment that lead to high pathogen levels at the abstraction sites (heavy rainfall, snowmelt, contamination accidents etc.), implementation of measures to control pathogen discharge into the catchment are important to reduce the risk of disease through drinking water. The Water Framework Directive does specify this in a very general manner, but more specific guidance and regulations are needed.

The consumer: risk communication
The majority of Europeans have confidence in the safety of their drinking water. The consumer expects a high level of safety from drinking water, as they do not have free choice of their drinking water. The consumer should be informed about the risk-based approach and the level of risk that is considered tolerable. Risk communication is delicate; transparent and open communication is important, as well as the choice of wording (i.e. talk about risk assessment or about safety assessment). The Water *Safety* Plan is an instrument that water suppliers can use for communicating due diligence to their consumers. It demonstrates that the water supplier has made a systematic inventory of all possible hazards/hazardous events, has control measures in place to deal with these hazards effectively and monitors whether the control measures are working all the time. Water Safety Plans will not totally eradicate waterborne outbreaks, but they will improve the standard of water supply even further.

The inspector: auditing
In the current EU Drinking Water Directive and in national legislation, water quality is primarily regulated through standards for chemical substances, physical condition and micro-organisms. The role of the inspectorate is therefore in principal to check if the water supplied meets the drinking water standards and to ask for improvements in water supply if standards are not met. In the risk-based system, the role of water quality standards and monitoring of finished water or water at the tap changes to the verification that all systems are designed and operated appropriately. In the Water Safety Plan, the water supplier documents the hazards and their control. The role of the inspectorate will shift towards an auditing process (or maybe even to auditing of the auditing done by an independent auditing agency, as is seen with the implementation of HACCP in the food industry). Science is needed to support this audit process, for instance to determine how much *E. coli* monitoring is needed to verify that the supply system is providing microbiologically safe drinking water.

The health authority: is risk management effective and efficient in terms of public health?
The WSP is a tool for the risk management process at water utilities. This is focused on the prevention of transmission of waterborne illness through drinking water. The point

of reference are the health (or related water quality) targets, but the risk management process in itself has no means to verify if the risk management actions sufficiently improve the public health status (or indeed lead to an imbalance in the allocation of resources to prevent waterborne illness, while other routes of exposure are much more significant). It is therefore important to "calibrate" the WSP with public health surveillance, taking into consideration disease outbreaks as well as sporadic cases of illness in the population who may be exposed to pathogenic microorganisms from a range of sources, not just drinking water. Health authorities may also undertake research to evaluate the role of water as a risk factor in disease, for example through case-control, cohort studies or intervention studies. In the case of an outbreak of illness that could be waterborne (see Chapter 1), the health authority will approach the water supplier to check whether water supply could be the source, for instance because the supply system is compromised. With a Water Safety Plan implemented, the water supplier can clearly demonstrate whether this was the case or not.

2.7.5 Future outlook: monitoring drinking water safety on-line

The future possibility of this approach is to design an on-line system for establishing and maintaining drinking water safety. The current monitoring for *E. coli* in treated water is valuable as a verification tool that the system has produced drinking water of good quality. The aim of this approach is to be able to determine this on-line and provide the treatment plant operator with information and tools to maintain the safety of drinking water instantaneously.

The efficacy of the total treatment that is required to produce safe drinking water from the given source water quality can be regarded as the Critical Limit of the overall treatment; if this limit is exceeded, the required treatment efficacy is not met and this may result in a health risk from drinking water that is above the target.

The monitoring program of the Control Points in the WSP monitors the performance of the individual treatment processes. This could be combined into an on-line assessment of the overall treatment efficacy, with the contribution of the individual processes. This way, the plant operator can see the efficacy of the treatment system on-line and can compare this against the Target and Critical Limit, the required treatment efficacy. If limits are not met, corrective actions need to be taken. This system allows not only to monitor the enhancement of the treatment performance on-line, but also to use different types of control measures to return to safe drinking water as rapidly and efficiently as possible.

Such an on-line control system can range from a simple assessment of key parameters at the relevant Control Points to an advanced model for treatment efficacy that uses the data from the WSP monitoring as input. A simple version (that is in operation at present) is the use of the AWWA/EPA Guidance manual for obtaining log-credits for treatment processes. At a treatment plant with coagulation/filtration and ozone, on-line information about temperature, pH, coagulant dose, turbidity, ozone residual and water flow was collected. For the ozonation, this was transformed (using the tables on the ozone efficacy in the guidance manual) to a log-removal of viruses, *Giardia* and *Cryptosporidium*. The efficacy of the coagulation/filtration was set at 2 logs for as long

as the coagulation operated within the operational limits. The operator received this information as a line on his monitor of the efficacy of the total treatment system and the contribution of the two processes. The critical limit of the overall efficacy was the treatment efficacy required under the Surface Water Treatment Rule.
More and more advanced tools for on-line monitoring, data handling and process control become available. As stated above, the implementation of the WSP with QMRA will produce a wealth of data that can be used to improve on-line process control. As more and more data and tools become available, the implementation of on-line process control systems that are directly linked to the safety of the drinking water is within reach. A point of caution, however, is that such instrumentation requires careful calibration and checking, as it is easy to accept numbers without questioning them from a machine.

It should be clear that on-line monitoring can typically be applied to monitor source water quality and the performance of treatment processes. It is less easily applied on control measures such as hygienic procedures for mains repair or inspection of well-heads or service reservoirs for leakage. This latter inspection-type monitoring is equally important for ensuring the safety of drinking water. In this form of monitoring, constant vigilance is needed to prevent contamination events. Reduced monitoring frequency causes slow deterioration of the water supply system and operational procedures and may ultimately lead to contamination of drinking water with waterborne pathogens and disease cases.

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3 Source water quality

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Assessment of source water pathogen contamination in baseline and peak conditions is the first step to quantitative microbial risk assessment of drinking water. In many cases, outbreaks of disease through drinking water have occurred as a result of hazardous events, such as heavy rainfall, which lead to high loads of pathogens in the source water. It is therefore important to incorporate hazardous events along with the variable baseline contamination in the QMRA. Furthermore, understanding of the contributing factors within the catchment is essential to assess and manage these risks. It should be based on:

- Knowledge of the different sources of contamination in the catchment and of their contribution to the contamination of the source water;
- Identification of hazardous (peak) events;
- Assessment of the levels of baseline and peak pathogen contamination of water sources.

After a review of pathogens in sources waters, this chapter proposes a framework for performing a catchment survey and designing an effective monitoring program for baseline and peak event contamination assessment. Finally, results from the Microrisk project are presented and discussed in a risk assessment context.

3.1 RATIONALE

Sources: [Medema *et al.*, 2003; Pond *et al.*, 2004]

3.1.1 Pathogens in source waters

During the last 20 years, the reliability of the faecal indicators as a mean to assure the safety of water has been increasingly challenged by water quality and public health microbiologists. In support of this contention, many publications report the limited correlation between the presence and concentration of faecal indicators and the presence and concentration of waterborne pathogens. They demonstrate in particular that faecal indicator bacteria such as *E. coli* are poor surrogates for protozoa and viral pathogens. Furthermore, several authors have shown that outbreaks of waterborne disease have occurred despite the absence of faecal indicators in source water [Barrell *et al.,* 2000]. These limitations have led several groups of workers to advocate the routine testing of water for specific pathogens. Indeed, during the recent revision of the WHO Guidelines for Drinking Water Quality, the WHO working committees suggested a list of reference pathogens that could be used as part of a water quality monitoring and assessment program.

This review is focussed on a selection of pathogens considered to be of high risk to human health and which are considered to be of concern in source water used for drinking water supply. These are (see Table 3.1):

- Protozoa: Cryptosporidium and Giardia

- Bacteria: Campylobacter and E. coli 0157:H7
- Viruses: Enterovirus and Norovirus

Table 3.1: Waterborne pathogens and their significance in water supplies after [WHO, 2004]

Pathogen	Infectious dose*	Persistence in water supplies	Resistance to chlorine	Relative infectivity	Important animal source
Campylobacter jejuni, C. coli	Low	Moderate	Low	Moderate	Yes
E. coli 0157:H7	Low	Moderate	Low	High	Yes
Enterovirus	Low	Long	Moderate	High	No
Cryptosporidium	Low	Long	High	High	Yes
Giardia intestinalis	Low	Moderate	High	High	Yes
Norovirus	Low	Long	Moderate	High	Potentially

* A detailed description of the dose-response relationship is given Chapter 7.

Protozoa

Cryptosporidium and *Giardia*

Cryptosporidium is a significant cause of waterborne outbreaks of diarrhoeal diseases. *Giardia* has been reported as the most common cause of protozoan diarrhoeal illness worldwide [Farthing, 1989; Adam, 1991]. Between 1971 and 1994, more than 25,000 cases of giardiasis were recorded in the USA [Craun, 1986; Anon, 1993, 1996]. The Centre for Disease Control and Prevention in Atlanta, USA, attributed 71% of waterborne disease outbreaks in 1993 and 1994 to *Cryptosporidium parvum* and *Giardia lamblia*, which respectively cause cryptosporidiosis and giardiasis [Gostin *et al.,* 2000]. Attack rates of cryptosporidiosis in these outbreaks are about 40% for the population at risk, as compared to 5-10% for giardiasis [Smith and Rose, 1990].

Bacteria

Campylobacter

Campylobacter is considered the most important bacterial agent in waterborne diseases in many European countries [Stenström *et al.,* 1994; Furtado *et al.,* 1998]. A large number of outbreaks of Campylobacter have been reported in Sweden for example, involving over 6,000 individuals [Furtado *et al.,* 1998].

E. coli 0157:H7

E. coli is an enteric organism and comprises the majority of the normal flora of the gut. E. coli 0157:H7 is the most widely recognised verocytotoxin-producing E. coli (VTEC) serotype and is now recognised as an important cause of food and waterborne illness in developed and some developing countries. High incidence of VTEC infections has been reported from regions of Canada, Scotland, and Argentina. In most European countries, the annual incidence may range from 1 to 4 infections per 100,000 inhabitants.

Virus

Enterovirus

Enteroviruses are one of the most common causes of human infections. They are ubiquitous, enterically transmitted viruses that have been estimated to cause about 30 million infections in the USA each year [WHO, 2004].

Norovirus

Noroviruses are a group of related, single-stranded RNA, non-enveloped viruses. Noroviruses are considered the most common viral etiologic agent of epidemic waterborne viral gastroenteritis [Brugha *et al.,* 1999].

A number of studies has been undertaken to investigate the occurrence of Campylobacter, *Cryptosporidium* and *Giardia* in source waters (Table 3.2). Fewer studies have been published on the levels of viruses and E. coli 0157:H7. In all cases presented below, it should be kept in mind that the sampling and testing methods varied and such variations can influence the numbers of pathogens detected. Methods differ in their sensitivity and selectivity, and in vitro culturing techniques do not isolate all the organisms present in samples due to the differences in metabolic condition of individual cells.

Table 3.2: Summary of concentrations of selected pathogens in water bodies

Pathogen	Water body	Concentrations	Country	Reference
Cryptosporidium	Surface water	0.006-2.5 oocysts/L	UK	Badenoch,1995
	Surface water	0-252.7 oocysts/L	11 countries	Smith & Grimason, 2003
	River water	4.1-12 oocysts/L	The Netherlands	Medema *et al.*, 1996
	Spring fed lake	0.24 oocysts/L	Ireland	Garvey *et al.*, 2002
	Surface water	3.8-21oocysts/L	Honduras	Solo-Gabriele *et al.,* 1998
	River	<5 oocysts/L	France	Rouquet *et al.,* 2000
Giardia	River	2.3 cysts/L	Canada	Ong *et al.,* 1996
	Surface water	5 cysts/L	8 countries	Smith & Grimason, 2003
	River	10-100/L	The Netherlands	Medema *et al.,* 1996
	Streams	0.1-5.2 cysts/L	USA	Ongerth *et al.,* 1989
	Surface water	0.02 cysts/L	Russian region	Ergov *et al.,* 2002
Campylobacter	Surface water	109,000 MPN/L	Germany	Feuerpfiel *et al.,* 1997
	River water	100-360/L	UK	Bolton *et al.,* 1982
	River	<100-2400 CFU/L		Stelzer *et al.,* 1989
	River	<2-93 MPN/L	Australia	Ashbolt *et al.,* 2002
	River	<1.2-110 MPN/L	Australia	Savill *et al.,* 2001
E. coli 0157	River and lake	>2000/L	Germany	Schindler, 2001
Enterovirus	Drinking WTT	0.0006 MPN/L	USA	Payment *et al.,* 1985
	River	0.3-4/L up to 13/L	The Netherlands	Theunissen *et al.*, 1998
	Dune filtrate	<0.003-13/L	The Netherlands	Theunissen *et al.*, 1998
	River	0.0033-0.46 PFU/L	Germany	DeRoda Husman *et al.*, 2004
	River	0.66-29/L	Worldwide	Gerba *et al.*, 1996
	Surface water	0.0033-0.46 PFU/L	Finland	Horman *et al.*, 2004

There are a number of limitations and sources of uncertainty in these data due to the sensitivity of analytical techniques, particularly for viruses and protozoa, and to the lack of

knowledge about the viability and human infectivity of *Cryptosporidium* oocysts, *Giardia* cysts and viruses detected in the different studies.

Concentrations in Table 3.2 vary greatly (zeros are not included):

- *Cryptosporidium* 0.006-250 oocysts / L
- *Giardia* 0.02-100 cysts / L
- Campylobacter 1.2-109,000 MPN / L
- Enterovirus 0.003-29 / L

These variations are greatly dependant on the sampling conditions and principally on the local context and hydrology. Wet weather conditions may provoke peak events with extreme values of concentrations. Monitoring is a valuable tool for identifying baseline and peak event contamination in local contexts.

3.1.2 Sources and routes of contamination

The relative significance of the different pathogens sources at a specific water site is determined by a combination of factors: (1) the contamination level of these sources, (2) the magnitude of these sources, (3) the persistence of the pathogen, (4) their transport behaviour from the source to the specific site and finally, (5) their resistance against treatment processes. Knowledge of these characteristics and about the health outcome after infection allows the appraisal of the health significance of the pathogen. The pathogens of particular interest in this project have been selected because they are considered of high health significance.

3.1.2.1 Overview on the potential sources of contamination

Source waters are vulnerable to contamination from many origins. Humans, livestock and wild animals are all sources of faecal contamination. It has been shown that many rivers in Europe are significantly contaminated with microbes arising from municipal wastewater and/or livestock [EEA, 2003]. Furthermore, source waters, and particularly surface waters, are often used for purposes such as irrigation, recreation, transport which may also affect water quality. Groundwater contamination may be induced by different practices in management of domestic wastewater and livestock manure. Precipitation events can lead to higher pathogen loads in source waters.

Waste water treatment plants are an obvious high risk source of pathogens both in terms of number and strain of pathogens (see Table 3.3). During periods of high rainfall or plant failure, WWTP may release significant amounts of poorly treated effluent. Moreover, pathogens may be dispersed in the environment through the use of sewage sludge as fertiliser.

Table 3.3: Typical concentrations of pathogens in raw and treated domestic wastewater [Medema *et al.*, 2003]

	Raw waste water	Secondary effluent
Cryptosporidium	1,000-10,000 n/L	10-1,000 n/L
Giardia	5,000-50,000 n/L	50-500 n/L
Enterovirus	10-100 n/L	1-100 n/L

Agricultural practices are an important source of contamination especially from *Cryptosporidium* oocysts, *Giardia* cysts, and Campylobacter [Carey *et al.,* 2004; Lack, 1999; Monis and Thompson, 2003]. As well as direct runoff into surface waters, animal waste is often collected in impoundments from which effluent may infiltrate groundwater.

Other sources of faecal contamination that may be a threat to water sources are stormwater discharges, accumulation of pathogens in sediment, swimming pool water?, water treatment plant discharges and wild animals.

Advances in source tracking techniques (for review of techniques see [Meays et al., 2004; Pond et al., 2004]) which differentiate animal and human sources of faecal pollution will allow more precise information on the contamination sources and will assist managers in developing strategies to protect source waters. More information on the sources and health implications of the pathogens selected in this study can be found in [Pond *et al.*, 2004].

3.1.2.2 Persistence of pathogens in the environment

After leaving the body of their host, most pathogens gradually lose viability and the ability to infect new hosts. The waterborne pathogens and parasites of greatest concern are those that have high infectivity and that can either proliferate in water or possess high resistance to decay outside the body.

The ability of pathogens to survive in surface water is variable. In general, survival is prolonged when water temperature is low. Other factors that influence survival include sunlight intensity and the presence of aquatic microorganisms that may use the pathogens as a food source or cause pathogen disintegration. Adsorption to particles facilitates survival. A summary of the major influencing factors on pathogen survivals are listed in Table 3.4. Table 3.5 outlines the disappearance rate and time for a 50% reduction in concentration of pathogens in surface water using examples of published data.

Table 3.4: Major factors influencing pathogen inactivation in surface water [Pond *et al.*, 2004]

	Solar radiation	Temperature	Salinity	Predation
Cryptosporidium	Medium (+)	High (+)	Medium (+)	Low (+)
Giardia	Medium (+)	High (+)	Medium (+)	Low (+)
Campylobacter	High (+)	High (+)	Medium (+)	Low (+)
E. coli 0157:H7	High (+)	High (none)	Medium (+)	Low (+)
Enterovirus	High (+)	High (+)	Medium (+)	Low (+)
Norovirus	Likely High (+)	Likely High (+)	Unknown – likely Medium (+)	Low(+)

It is possible that in nutrient rich sediments, micro-organisms survive for extended periods of time [Davies *et al.,* 1995]. In the case of oocysts, it has been shown that they may remain infective up to 12 weeks in water at 25°C and survive for several months in water at 4°C [Carey *et al*., 2004].

Table 3.5: Disappearance of selected pathogens in surface water [Medema *et al.*, 2003]

	Disappearance rate (per day)	Time for 50% reduction of concentration (days)
Cryptosporidium	$5.7 \cdot 10^{-3}$-$4.6 \cdot 10^{-2}$	15-150
Giardia	0.023-0.23	3-30
Enterovirus	0.01-0.2	3-70

Disappearance rates are lower in groundwater than in surface water. Pathogens may be removed during soil transfer by adsorption and inactivation. Inactivation is influenced by many factors such as soil temperature, moisture, pH, microflora and organic carbon content. International literature reveals that viruses survive longer than faecal bacteria. No data on the survival of protozoa in groundwater are available yet, but it can be assumed that these pathogens are able to survive longer than viruses [Medema *et al.*, 2003].

3.1.2.3 Transport of pathogens

Most pathogens have no means of transport in the aquatic environment other than being transported with the water flow. Pathogens can therefore be regarded as biological particles that are transported by advection. Sedimentation of viruses and parasites is very slow and probably not significant. However, many pathogens readily attach to particles in water [Gerba *et al.*, 1984] which largely determine the transport characteristics. Sedimentation may then become significant.

Sediments may contain important numbers of faecal indicators and pathogens. For example, virus levels are generally 10-fold higher in sediments than in overlying waters. Since pathogens remain viable in the sediments for variable lengths of time, it is important to consider the importance of their resuspension and subsequent redistribution. Rain events and activities such as shipping or dredging may give rise to resuspension.

Several factors affect the hydrodynamic distribution of pathogens in lakes and reservoirs. In temperate climates, lakes may be stratified in summer, with warm water at the top and colder contaminated water at the bottom of the lake. Destratification (due to temperature decrease or storms) will cause water layers to mix and particles to return to the surface layer. Inflow characteristics are also important factors: inflow speed, entrainment of lake water and resulting dilution, insertion depth [Brookes *et al.,* 2004; Hipsey *et al.*, 2005].

Rain events not only affect water quality because of runoff and stormwater discharges but also because of water flow increase. This may result in faster transport of pathogens from the contamination source to the abstraction site. Concentration of *Giardia* oocysts has been shown to be positively correlated to water flow and turbidity levels [Atherholt *et al.*, 1998].

The most important factors in the transport of microorganisms through the subsurface are water flow (the driving force) and attachment [Schijven *et al.*, 2000]. Adsorption is affected both by the characteristics of soil (texture, pH, composition) and pathogens. Bacteria and parasites are more readily removed than viruses because of their size (1-20 µm versus 20-80 nm); differences in isoelectric points and surface composition determine the pathogen adsorption rates. The unsaturated flow zone can play an important role in retarding or even

eliminating pathogens and must be considered when assessing aquifer vulnerability. Increased water flow may remobilize adsorbed microorganisms.

NB: Highly fractured aquifers
Highly fractured and karstic aquifers represent a particular problem. Groundwater flow through fractured systems may be very rapid. The potential for microorganisms to be attenuated by interaction with the aquifer matrix is reduced but not entirely absent.

3.1.3 Conclusions

The six pathogens reviewed in this document all have high health significance. It is clear that source waters are contaminated to varying degrees with these pathogens. Their presence and persistence in water is due to a number of different factors such as survival, transport, type of water source or aquifer characteristics in the case of groundwater. There sometimes is a strong seasonal effect in the occurrence of these pathogens in surface waters with periods of rainfall contributing to higher source water contamination.

To understand the dynamics of source water pathogen contamination, it is important to determine the sources of pathogens in a catchment and to quantify their environmental loadings, especially under conditions that may favor high pathogen concentrations (hazardous events). The natural variability of potentially pathogenic microorganisms from anthropogenic, natural, and livestock sources is large and difficult to quantify. It is complex to rank the various sources and transport routes in terms of relative importance to human disease. Risks depend much on the specific case and need to be considered in the local context. This is of course a big challenge for water and/or health managers.

If a monitoring program is to be planned, it is essential to identify the main sources of contamination and potential causes of peak events in the local context.

The following should be considered:

- Magnitude of contamination;
- Frequency of the discharge (continuous versus event related);
- Type of contamination (animal or human);
- Distance from the water source and travel time during events;
- Transport and survival properties of potential pathogens.
-

In the following paragraphs, a protocol for assessing contamination sources and events in a catchment is given (3.2). How this can be used to guide pathogen monitoring is developed further (3.3).

3.2 CATCHMENT SURVEY

The purpose of this step is to develop a broad overview and basic understanding of the catchment. It is not intended to be an extensive data collection exercise but rather the characterisation of the system at an appropriate level of detail to provide useful information [Nadebaum *et al.*, 2004]. The following conclusions should be drawn from this survey:

- Vulnerability of the source water;

- Importance and location of pathogen sources;
- Peak events leading to high contamination risks (type, intensity, frequency, duration).

This type of survey has been conducted on 12 different Catchment to Tap Systems (CTSs) throughout Europe plus one in Australia (see Table 3.6). They vary in size, occupation, protection, climate, etc.

Table 3.6: List of the 12 Catchment to Tap Systems (CTSs)

CT	Country	Source water	Protectio	Climate	Catchment
1	United Kingdom	River	No	Humid oceanic	12,917
2	The Netherlands	River	No	Humid oceanic	198,735
3	France	River	No	Humid oceanic	10,050
4	France	River	No	Mediterranean	522
5	Sweden	River with controlled input	No	Sub-arctic	50,180
6	Sweden	Reservoir	No	Sub-arctic	50,180
7	Germany	Groundwater & river bank filtrate	No	Humid oceanic	145
8	Australia	Reservoir	No	Mediterranean	140
9	The Netherlands	Reservoir	No	Humid oceanic	198,735
10	France	Reservoir	No	Humid oceanic	30
11	Germany	Reservoir	Yes	Humid oceanic	300
12	France	Aquifer	Yes	Humid oceanic	100

In this project, source water quality is assessed at the intake of the treatment plant. This implies that reservoir and river bank water filtrate are regarded as source waters. Another point of view may be to consider reservoirs and river bank filtration as the first treatment step and therefore sample source water upstream.

3.2.1 Guidelines for performing catchment survey

The proposed outline for performing a catchment survey is detailed in Table 3.7. Recommendations include description of the water abstraction, key catchment characteristics (morphology, hydrology, hydrogeology and climate) plus description and location of potential sources of faecal contamination.

Table 3.7: Outline for catchment survey

SURFACE WATER	GROUNDWATER
Description of water abstraction	
Water intake description	• Number of wells • Depth • Wellhead
Type of source • River • River with reservoirs upstream • Artificial reservoir (dam) • Natural reservoir (lake)	Type of source • River-aquifer connection (e.g. karstic aquifer) • Shallow hole • Lowland river gravel abstraction • Shallow water table • Confined aquifer
Catchment description	
Size of the catchment, length of river, main tributaries, maximum and minimum height, dimension of reservoir	• Total catchment • 50-days catchment • Surface water catchment (if connected)
Uses of water • Agriculture • Urban • Industry • Other	Uses of water • Agriculture • Urban • Industry • Other
Hydrology & Hydrogeology	
• Average flow • Monthly average flow • Sorted Flows • High flows (1-year, 10-year, 50-year) • Main soils • Slopes	• Description of catchment geology and hydrogeology • Average water pumped (yearly and monthly) • Maximum water pumped (yearly and monthly)
Climate	
Description of the climate including • Temperature (monthly average, minimum and maximum) • Rainfall (monthly average, minimum and maximum) • Snowmelt	
Location and description of potential sources of faecal contamination	
• Human – Waste Water Treatment Plants – Combined Sewers Overflows – Biosolids (storage and use in agriculture) • Animal – Animal breeding (manure storage, manure used as fertiliser, grazing) – Roosting birds – Slaughterhouses or livestock markets – Wildlife	• Human – Septic tanks – Biosolids (storage and use in agriculture) • Animal – Animal breeding (manure storage, manure used as fertiliser, grazing) • Other – Wellhead or borehole liable to flooding • If connected to surface water – See potential sources for surface water

3.2.2 Example of CTS surveys

Figure 3.2 and Figure 3.1 show map examples of potential contamination sources for two CTSs. Table 3.8 summarises a fulfilled generic catchment description.

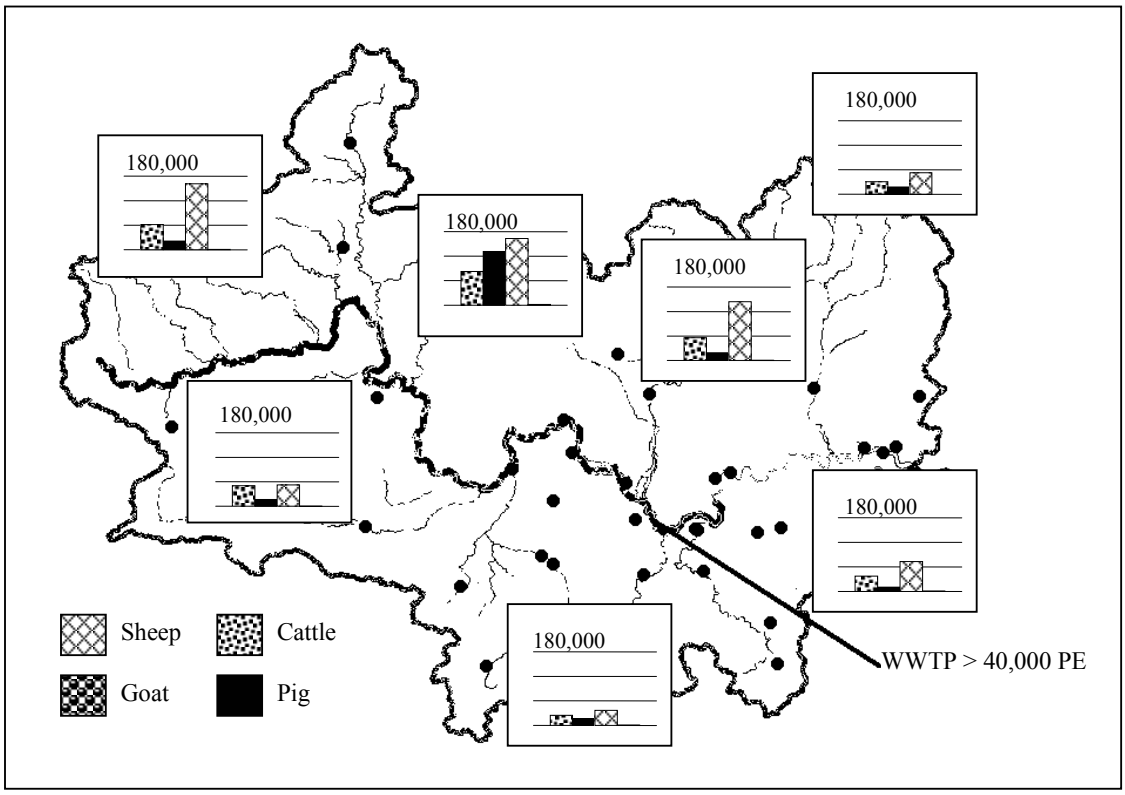

Figure 3.1: Animal breeding & waste water treatment plants in CTS 1 (UK)

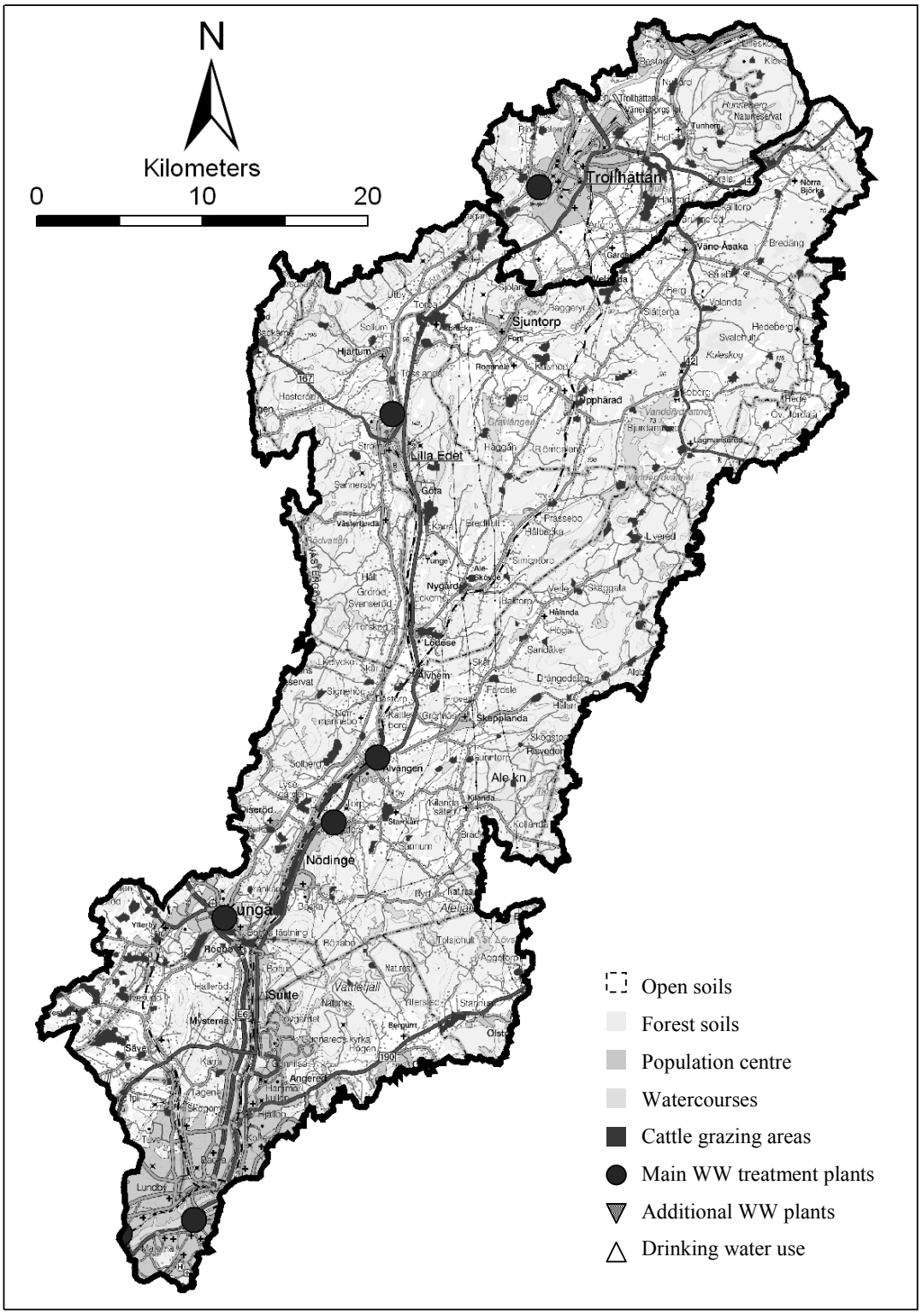

Figure 3.2: Lower catchment area of CTS 5 & 6 (Sweden)

Table 3.8: Catchment survey for CTS 8 (Australia)

CTS Name and number	**xxxxxxx**	**8**
Country	Australia	
Catchment size (km^2)	Surface water Population supplied = 50,000 Catchment area = 140 km^2	Groundwater Total: 50-days: Surface water catchment (if connected):
Potential sources of faecal contamination	Human ☒ Waste Water Treatment Plants ☐ Combined Sewers Overflows ☒ Biosolids (storage and use in agriculture) Animal ☒ Animal breeding ☒ Roosting birds ☐ Slaughterhouses or livestock markets	
Type of source (location of intake)	Surface water ☐ River ☐ River with reservoirs upstream (farm dams) ☒ Artificial reservoir (dam) ☐ Natural reservoir (lake) Groundwater ☐ River-aquifer connection ☐ Swallow hole ☐ Lowland river gravel abstraction ☐ Shallow water table ☐ Confined aquifer	

3.2.3 Hazard identification and peak events

Understanding the reasons for variations in source water quality is important, as it will influence the requirements for treatment, treatment efficiency and the resulting health risk associated with the finished water. Raw water quality is influenced by both natural and human use factors. Human use factors include point sources (municipal wastewater discharges) and non-point sources (urban and agricultural runoff).

Whether water is drawn from surface or underground sources, it is important that the characteristics of the local catchment or aquifer are understood and that the scenarios that could lead to water pollution are identified and managed. Groundwater from deep and confined aquifers is usually microbiologically safe; however, shallow or unconfined aquifers can be subject to contamination from discharges or seepages, on-site sanitation and sewerage. Hazardous, peak events that may have an impact on the catchments and that should be taken into consideration as part of a hazardous events assessment include:

- Upstream events (waste water and stormwater discharges, waste disposal sites);
- Human access (recreational activity);
- Cleaning of the river course;
- Land use (animal husbandry, agriculture, forestry) and changes in land use;

- Unconfined and shallow aquifer, including groundwater under influence of surface water and karstic aquifers;
- Inadequate wellhead protection and unhygienic practices;
- Climatic and seasonal variations (rainfall, thaw, snowmelt, droughts) [WHO, 2004].

Other situations may be important to consider locally, such as:

- Farming practices, such as in CTS 9;
- Different farming practices may yield peak events. For example, in the late winter and spring, farm animals and their young are put back on the fields. CTS 9's catchment survey identified that young animals may shed higher concentrations of pathogens.

Example: high bird loads in CTS 2
River water is abstracted, pre-treated and transported to the dunes along the North Sea coast where it remains from 60 to 400 days. It is then abstracted in an open canal system, collected in a reservoir and treated once more before distribution. During frost periods, water in the reservoir usually remains unfrozen longer than in the surrounding water bodies due to the constant temperature of the abstracted water and the flow in the basin. As a consequence, geese, ducks and swans tend to assemble on the reservoir, leading to very high bird loads. This causes high loads of pathogenic microorganisms, especially Campylobacter.

NB: The normal presence of birds on the reservoir is not necessarily a peak event; it can be considered as a baseline situation for this particular source water.

3.2.4 Historical data analysis

Historical data analysis is an essential first step for proper identification of local peak events. This analysis is necessary to define appropriate peak event sampling strategies adapted to the local context (type, propitious periods of the year, availability of real time data...).

Heavy rainfall remains the major cause of peak events and most CTSs focused on sampling this type of peak event. They are associated with high surface runoff and discharge of untreated wastewater. The difficulty lies in starting the sampling program as soon as possible after the beginning of an event. Some examples of sampling strategies are given hereafter and investigation on other potential peaks is given in 3.2.4.2.

3.2.4.1 Historical analysis of rain events

Unprotected surface water reservoir: CTS 8 (Australia)
The outcomes of CTS 8 historical data analysis are as follows:

- Event size and complexity are highly variable and hard to predict;
- Rainfall is not a good predictor of event occurrence by itself but it can be considered as a precursor when the hydraulic characteristics of the catchment are known;
- Events are recognisable by a rapid rise in river level;
- Response time to runoff is of the order of 4 to 6 hours after rainfall;
- The hydrographs evolve (rising limb > peak > falling limb) over a similar time frame which could be used to develop sampling protocols;
- Initial peak rise and fall lasts approximately 24 hours.

This led to the development of the following event based sampling strategy:

- Use of automated samplers to ensure capture of rising limb samples;
- Activation of samplers on warning of storm from weather forecasts and radar checks;
- Triggering of collection based on rate of change and magnitude of water level rise;
- Collection of excess samples to ensure 3 main stages of the hydrograph;
- Collection at increasing intervals to allow for the hydrograph skew;
- Where resources are limited, collection of first peak runoff as a priority.

Detailed pathogen data were collected for 3 small events (Figure 3.3).

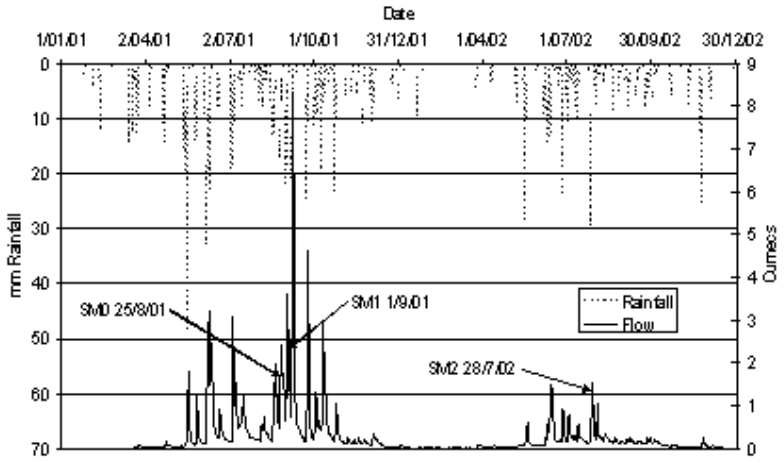

Figure 3.3: CTS 8 - Daily flow and rainfall in 2001-2002; 3 events (SM0-SM2)

Unprotected river water: CTS 3 (France)

Sampling was focused on rare, significant events. The following definition was set: a rainfall peak is a reasonably rare event and it should thus have "rare" turbidity and flow rise. Comparison of the time series for a 2-year period gives a linear relation between flow and turbidity: $r^2 = 0.73$ for daily data and $r^2 = 0.89$ for monthly averages. To avoid small variations due to minor runoff events, the sampling strategy is based on threshold values both for flow and turbidity. They are derived from the analysis of the sorted flow and turbidity curves (Figure 3.4).

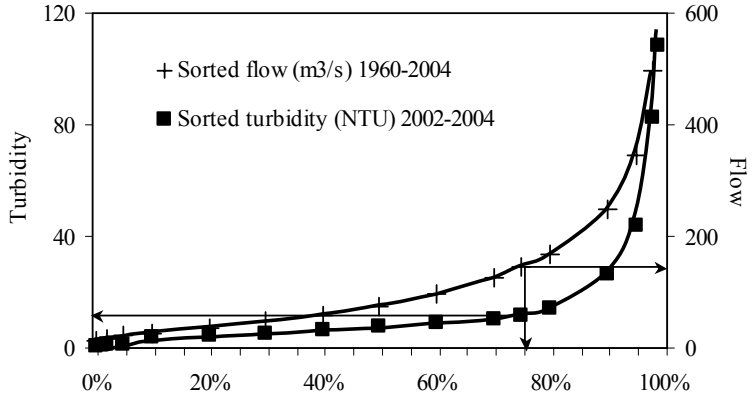

Figure 3.4: CTS 3 – Sorted flow and turbidity curves

The 75% occurrence is selected from the shape of the sorted turbidity curve. This yields thresholds of 150 m^3/s and 12 NTU. A peak starts with an increase of flow and turbidity. Unfortunately, only turbidity was measured in real time at the water treatment plant. Sampling was based on this parameter alone but peak relevance was confirmed later with flow data.

Based on historical data, rainfall peaks have the following characteristics for CTS 3:

- 9 peaks per year;
- Average duration of 8 days (minimum 2, maximum 14);
- Peak maximum reached after 3,3 days (minimum 1, maximum 9);
- Months when peaks are most frequent are November through January.

Unprotected groundwater: CTS 7 (Germany)

The CTS 7 water treatment plant uses bank filtration as treatment. About 65% of the source water is abstracted from the river after bank filtration and the rest comes from groundwater.

The events leading to high risk of contamination of the wells are fast rising water levels in the river up to very high water levels. There are several aspects to this. Firstly, fast rising water levels (3 meters or more within five days) after long dry periods cause much faster groundwater flow in the direction of the wells, thus reducing bank filtration efficiency for removing pathogens. Secondly, high river water levels lead to increased groundwater levels. Distance between the soil surface and the groundwater level becomes very small and removal of pathogens in the unsaturated zone is reduced. With falling water levels in the river, contaminated groundwater will reach the wells and lead to contamination when contact of groundwater with faecal contaminants is made possible by removal of the protecting soil layers, manure storage in garden plots, etc.

Figure 3.5 shows the changes in river water level within five days over a period of 50 years, information that can be used to determine criteria for peak events. Increase of water level of 3 meters or more within five days happened in 1.1% of time or 3.9 days per year on average. However, in the last decade, average is of 4.6 days per year.

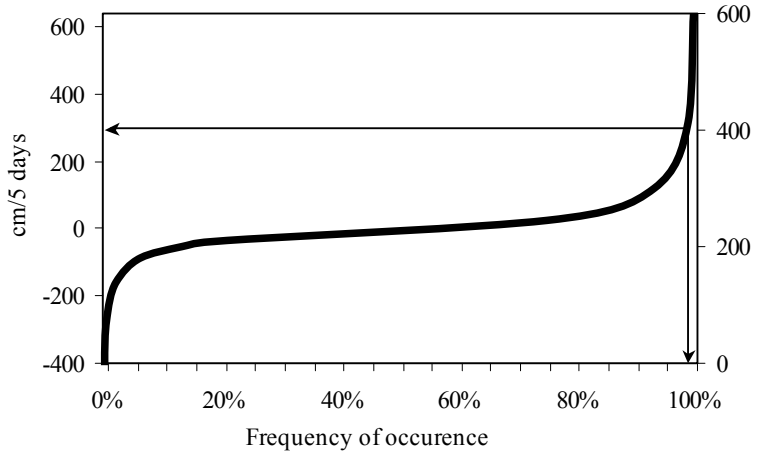

Figure 3.5: CTS 7 – Changes of river water level within 5 days (1953 –2003)

3.2.4.2 Other peak events

The following examples illustrate other types of peak events, analysed with historical data or water quality monitoring.

Historical data analysis: CTS 5 (Sweden)
Incidents leading to peak contaminations and source water intake closures are identified by microbiological source water monitoring at the intake, upstream monitoring stations and incident reports (telephone and fax). During the 2001-2005 period, 260 closures occurred. Incidents registered at the source water intake were most of the time related to high bacteria counts. In 2003 and 2004, discharge of untreated wastewater was the most common microbial incident and happened mainly in connection to heavy rain/snow. The high bacteria counts were also related to technical failures, such as the breakage of a high-pressure sewage pipe.

Water quality monitoring: CTS 9 (The Netherlands)
Water quality monitoring can demonstrate the occurrence of peak events and give information about their frequency, magnitude and duration.

In CTS 9, water is abstracted from a polder, flows through an open transport canal to a flocculation pretreatment and remains in an open lake reservoir for 89 days. Under conditions of high demand, water can also be abstracted from the nearby canal. Water from the lake is filtered and sent to the treatment plant or into an open buffer reservoir (closed in 2003 due to waterfowl contamination, as in CTS 2).

Multiyear E. coli and Coli 44 data show that peak contaminations do occur in the canal, polder, after flocculation and at the reservoir intake. A short, high peak occurred in winter 1995-1996 and a broader peak occurred in summer 1999. Several smaller peaks are visible in 1998. The peaks observed at the reservoir intake in 1998 and in summer 1999 coincide with peak E. coli concentrations from polder water. This suggests that peak contamination in the polder may travel to the water treatment plant intake much faster than the average residence time of the reservoir would suggest. None of these peaks corresponded to periods of heavy rainfall.

3.3 PATHOGEN MONITORING

The purpose of the pathogen monitoring programs is to evaluate the levels of pathogen contamination for specific sites, in baseline and peak conditions, so as to provide a strong, quantitative basis for risk assessment and QMRA. In the MicroRisk context, the monitoring programs were also valuable for assessing the levels of pathogen contamination in a representative set of EU catchment situations.

3.3.1 Design of monitoring program

The monitoring program includes the selected pathogens (*Cryptosporidium*, *Giardia*, Campylobacter, E. coli 0157:H7, Enterovirus and Norovirus) as well as faecal indicators (E. coli, Clostridia, Total Coliforms, Enterococci) and physico-chemical characteristics of the source water (turbidity, conductivity, temperature, pH). When possible, water flow is also evaluated in order to distinguish baseline from rain event contamination.

Standard methods of sampling, sample processing and analysis are recommended to ensure comparable results. Since source water pathogen concentrations may be very low, concentration/enrichment of large volumes of water, sometimes thousands of litres, may be necessary for detection. Consequently, it is important to collect proper sample volumes.

MicroRisk samples are collected at the intake of the water treatment plant. This implies that reservoir or bank filtration is not considered as a treatment but as a water source. The reader is of course free to reconsider this in his local context.

3.3.1.1 Baseline contamination

Baseline contamination assessment requires a full year of monthly samples. Samples should be collected:

- During dry weather conditions as rain events may lead to peak events;
- Each month so as to have an idea of the seasonal variations due to hydrological or hydrogeological conditions and/or to abstraction of groundwater due to increasing (seasonal) demand.

3.3.1.2 Peak contamination due to rain events

The objective is to sample at least two peak rain events during the year of sampling. Forecasting the rain event period is necessary in order to be ready for sampling and analysis. The proposed approach distinguishes surface water, protected groundwater and groundwater influenced by surface water.

- Surface water

Rain event indicators usually available in real time are turbidity and water flow (or water level). Turbidity and/or water flow increase indicate that runoff is ongoing. Historical data analysis is valuable for estimating which values of turbidity and water flow correspond to averages and which correspond to rain events. Rain event thresholds can be fixed locally and used to set simple rules for starting the sampling period.

Example of a simple sampling strategy

Turbidity and/or flow are increasing. This indicates that water is running off. When flow reaches twice the yearly average flow, we can consider it is a rain event (Figure 3.6). Start sampling once a day for five days and continue if the peak flow is not reached (flow did not start to decrease).

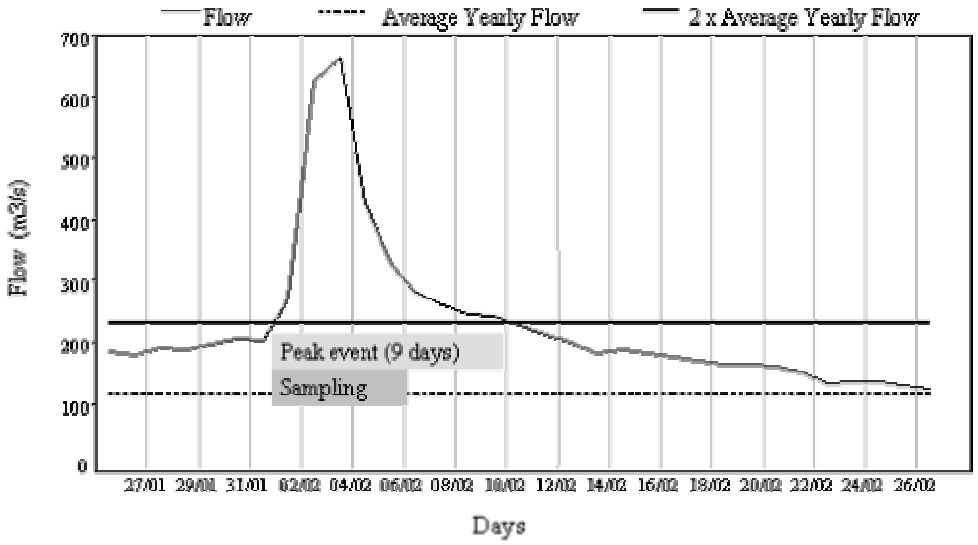

Figure 3.6: Example for water flow threshold in rain event conditions (CTS 3)

- Groundwater influenced by surface water

Such events are dependant on hydraulic conditions of surface water (see Figure 3.5) and abstraction rates. Historical data analysis is necessary to understand when influence from surface water is the highest (e.g. high abstraction rate, high surface water level). The same methodology as for surface water may be applied.

- Protected groundwater

By definition, contamination due to rain events should not occur, unless there are specific local conditions.

3.3.2 Detection methods

Assessment of the risk of infection from waterborne pathogens requires accurate determinations of microbial occurrence, concentration, viability, infectivity and human dose response data [LeChevallier *et al.,* 2003]. Existing methods have limitations in one or more of the criteria; for example, nucleic acid and antibody-based methods do not readily provide information about the concentration, viability and infectivity of the pathogen, whereas culture methods can be used only for the relatively small group of pathogens that are capable of growth in culture. Furthermore, the recovery rates of many culture methods may be very low, leading to a significant underestimate of pathogen numbers. It is important when selecting the method of analysis to balance the advantages and disadvantages of each in terms of the required output. [Source: Pond *et al.*, 2004]

An important consideration for any project is that the methods of analysis are sufficiently detailed in their scope to ensure comparable results. Therefore, whenever possible,

international standard methods of analysis should be used. Standard methods are published by several organisations (for example, ISO, CEN, APHA) and there are many supporting standards for the validation of methods and monitoring of laboratory performance. Laboratories should provide their Quality Assurance/Quality Control data on the method performance characteristics so it can be included in (statistical) interpretation of the results.

There are different ways to evaluate analytical performance and it is common for each laboratory to apply its own methodology. Recovery is evaluated from source water and/or ultra-pure water samples. It can be calculated for each sample or for a whole data set, using an average value. Controls were only available for *Cryptosporidium* and *Giardia* (Table 3.9).

Table 3.9: Example of Quality Assurance/Quality Control data for *Cryptosporidium* and *Giardia*

	Cryptosporidium		*Giardia*		Design of recovery experiments
	Mean recovery	σ	Mean recovery	σ	
CTS 2	12%	16%	6%	5.4%	Determination from 3 source water samples
CTS 3 & 4	30-40%		30-40%		Recovery is tested 6 times a year on spiked ultra-pure water samples
CTS 5	12%	7%	8%	7%	Determination from 4 source water samples
CTS 7	19.2%	5.7%	14.9%	4.5%	500 L water spiked with oocysts/cysts in concentrations of 10^2 to 10^4 / 500 L
CTS 8	50%	13%	47%	17%	Determination for each source water sample
CTS 10	26%	21%	30%	29%	Results are issued from spiked source water samples + spiked ultra-pure water samples
CTS 11	12%	3.1%	10.7%	7.3%	500 L water spiked with oocysts/cysts in concentrations of 10^2 to 10^4 / 500 L; 5 mL *Cryptosporidium*/*Giardia*-free sediment were added to simulate source water

Some laboratories encountered detection problems with standard methods of analysis:

Example: CTS 10 (France)
High values of turbidity were found to interfere with *Cryptosporidium* and *Giardia* analysis. No oocysts/cysts could be recovered from spiked samples for turbidity values higher than 8 NTU in the case of *Cryptosporidium*, or 3.5 NTU in the case of *Giardia*.

Laboratory performance in analysing pathogens is still highly variable and the quality of data produced by a laboratory cannot be taken for granted. Pathogen concentrations may otherwise be greatly underestimated. Quality control data and details of the confirmation methods should be provided along with the count results [Roser *et al*., 2002.]

3.3.3 Lacks in data

Despite all precautions, lacks in data always seem to emerge once datasets are acquired. The reader's attention is brought to the following possible deficiencies:

- Quality Assurance/Quality Control data

Laboratories do not easily provide relevant quality data or full methodology (number of samples, number of spikes). The MicroRisk project could only gather very heterogeneous recovery data for *Cryptosporidium* and *Giardia* (Table 3.9). Campylobacter, E. coli 0157:H7, Enterovirus and Norovirus quality data was not available.

- Turbidity and water flow

These two parameters represent valuable characteristics of the sampling conditions. They are particularly important for a proper assessment of peak events. Daily water flow measurements are interesting for situating samples in the course of a hydrological event.

- Precipitation data

Heavy rainfall is generally the most common peak event. Precipitation data may be useful to quantify the significance of such events.

3.4 DATA ANALYSIS

The MicroRisk partners monitored source water quality for nine European water sources and one Australian. The monitoring programs provide information on source water baseline and peak contamination in pathogens (*Cryptosporidium*, *Giardia*, Campylobacter, E. coli O157, Enterovirus and Norovirus) and faecal indicators. The objectives are to:

- Draw a picture of source water pathogen contamination in European countries,
- Assess significance of peak event contamination,
- Analyse correlation between commonly monitored faecal indicators and/or turbidity and pathogens.

NB: Laboratory determination of QA/QC[1] data not being consistent for all CTSs and all parameters, raw results are presented directly.

3.4.1 Full results per CTS

Baseline contamination results are given in Table 3.10 and in Table 3.11 for rain events. Total number of samples, number of positive samples and average, minimum and maximum calculated on the positive samples are given. If results come as a range of values, for example 10-100, they are given as 10-100(3). This means that the 10-100 range was encountered in 3 samples.

[1] Quality Assurance/Quality Control

3.4.1.1 Baseline contamination

Table 3.10: Baseline contamination in the CTSs

CTS 1 - UK	***River***			***Catchment:***	***46,830***	***km²***
Parameter	Unit	Samples	Positive samples	Average	Min	Max
Cryptosporidium	n/L	11	2	0.35	0.3	0.4
Giardia	n/L	11	0	-	-	-
Campylobacter	CFU/L	11	0	-	-	-
E. coli O157:H7	CFU/L	11	0	-	-	-
Enterovirus	PFU/L	11	4	1.55	0.4	3.4
E. coli	MPN/L	11	11	14,191	5,300	22,000
Clostridia	CFU/L	11	11	2,871	80	8,000
Total Coliforms	MPN/L	11	11	63,927	20,500	112,000
Enterococci	CFU/L	11	10	1,710	100	6,000

CTS 2 - The Netherlands	***River***			***Catchment:***	***198,735***	***km²***
Parameter	Unit	Samples	Positive samples	Average	Min	Max
Cryptosporidium	n/L	11	5	0.093	0.05	0.2
Giardia	n/L	11	3	0.015	0.003	0.023
Campylobacter	MPN/L	69	57	1,703	0.4	15,000
Enterovirus	PFU/L	3	2	0.015	0.005	0.024

CTS 3 - France	***River***			***Catchment:***	***10,050***	***km²***
Parameter	Unit	Samples	Positive samples	Average	Min	Max
Cryptosporidium	n/L	11	5	0.09	0.05	0.2
Giardia	n/L	11	10	1.16	0.05	4.7
Campylobacter	n/L	11	0	-	-	-
E. coli O157:H7	CFU/L	11	10	10-100 (9)	>1000 (1)	-
Enterovirus	PFU/L	11	0	-	-	-
E. coli	n/L	11	10	1,900	700	7,000
Clostridia	n/L	11	11	2,909	500	4,350
Total Coliforms	n/L	10	10	5,692	750	20,000
Enterococci	n/L	10	10	617	50	2,800

CTS 4 - France	***River***			***Catchment:***	***522***	***km²***
Parameter	Unit	Samples	Positive samples	Average	Min	Max
Cryptosporidium	n/L	12	3	0.12	0.05	0.2
Giardia	n/L	12	11	0.36	0.05	0.75
Campylobacter	n/L	11	0	-	-	-
E. coli O157:H7	CFU/L	12	8	10-100 (3)	100-1,000 (2)	>1,000 (3)
Enterovirus	FPU/L	12	0	-	-	-
E. coli	n/L	12	12	8,551	10	80,000
Clostridia	n/L	12	12	3,392	800	17,500
Total Coliforms	n/L	12	12	34,660	220	270,000
Enterococci	n/L	12	11	1,503	30	6,600

CTS 5 - Sweden	***River & lake***			***Catchment:***	***50,180***	***km²***
Parameter	Unit	Samples	Positive samples	Average	Min	Max
Cryptosporidium[1]	n/L	13	3	0.09	0.08	0.1
Giardia[1]	n/L	12	2	0.09	0.016	0.16
Campylobacter	n/L	13	1	10	-	-
E. coli O157:H7	n/L	13	0	-	-	-
Enterovirus[1]	n/L	12	0	-	-	-
Norovirus[1]	n/L	12	0	-	-	-
E. coli	n/L	14	14	927	310	2,200
Clostridia	n/L	15	15	157	60	350
Total Coliforms	n/L	14	14	22,650	2,600	82,000
Enterococci	n/L	15	15	519	70	1,800

CTS 7- Germany	***Groundwater and river bank filtration***			***Catchment:***	***145***	***km²***
Parameter	Unit	Samples	Positive samples	Average	Min	Max
Cryptosporidium	n/L	11	0	-	-	-
Giardia	n/L	11	0	-	-	-
E. coli	MPN/L	11	1	10	-	-
Clostridia	CFU/L	11	0	-	-	-
Total Coliforms	MPN/L	11	6	29	10	42
Enterococci	CFU/L	11	0	-	-	-

CTS 8 - Australia	***Reservoir***			***Catchment:***	***140***	***km²***
Parameter	Unit	Samples	Positive samples	Average	Min	Max
Cryptosporidium	n/L	51	2	0.1	0.1	0.1
Giardia	n/L	51	1	0.1	-	-
E. coli	n/L	78	55	125	10	1,200
Total Coliforms	n/L	124	118	2,620	10	24,000

CTS 9 - The Netherlands	***Reservoir***			***Catchment:***	***198,735***	***km²***
Parameter	Unit	Samples	Positive samples	Average	Min	Max
Cryptosporidium	n/L	25	25	0.33	0.01	4.6
Giardia	n/L	25	25	2.94	0.01	41.3
Campylobacter	n/L	37	32	72.3	0.4	500
Enterovirus	PFU/L	12	0	-	-	-

[1] On concentrate

CTS 10 - France	***Reservoir***			***Catchment:***	***30***	***km²***
Parameter	Unit	Samples	Positive samples	Average	Min	Max
Cryptosporidium	n/L	9	5	0.54	0.1	1
Giardia	n/L	9	6	0.73	0.1	3
Campylobacter	MPN/L	9	2	10-100 (2)	-	-
E. coli O157:H7	MPN/L	9	3	10-100 (3)	-	-
Enterovirus	PFU/L	9	0	-	-	-
E. coli	MPN/L	9	9	340	60	1,080
Total Coliforms	MPN/L	9	9	2,200	1,180	4,350
Enterococci	MPN/L	9	8	246	10	1,300

CTS 11 - Germany	***Reservoir***			***Catchment:***	***300***	***km²***
Parameter	Unit	Samples	Positive samples	Average	Min	Max
Cryptosporidium	n/L	11	11	0.039	0.019	0.06
Giardia	n/L	11	1	0.004	-	-
Campylobacter	CFU/L	9	0	-	-	-
E. coli	MPN/L	11	8	25.6	10	53
Clostridia	CFU/L	11	5	48	20	80
Total Coliforms	MPN/L	11	11	124	20	504
Enterococci	CFU/L	11	4	12.5	10	20

CTS 12 - France	***Groundwater***			***Catchment:***	***100***	***km²***
Parameter	Unit	Samples	Positive samples	Average	Min	Max
Cryptosporidium	n/L	10	0	-	-	-
Giardia	n/L	10	0	-	-	-
Campylobacter	MPN/L	10	0	-	-	-
E. coli O157:H7	MPN/L	10	0	-	-	-
Enterovirus	PFU/L	10	0	-	-	-
E. coli	MPN/mL	10	0	-	-	-
Total Coliforms	MPN/mL	10	1	10	-	-
Enterococci	MPN/mL	10	0	-	-	-

3.4.1.2 Rain event contamination

Rain events should be the object of a second sampling program. However, some may be sampled inadvertently during the baseline contamination program. Turbidity and/or water flow should always be checked to determine the sampling conditions.

Figure 3.7 shows an example taken from the CTS 3 baseline contamination program. The January sample was collected during a rain event: water flow is 425 m^3/s (2004 average is 98 m^3/s) and turbidity is 25 NTU (2004 average is 8.5 NTU). Such samples should be added to rain event results.

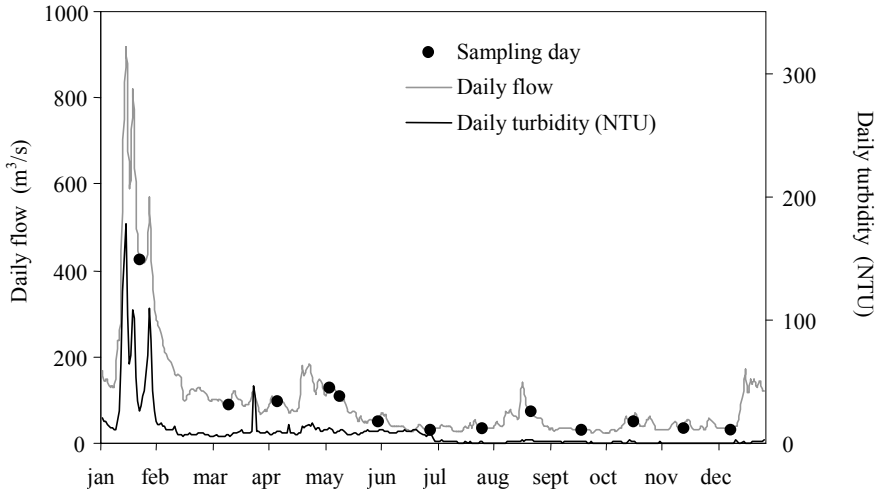

Figure 3.7: Rain event sample during the 2004 baseline contamination program (CTS 3)

Other samples transferred from the baseline contamination program are

- CTS 1: turbidity of the January sample is 36 NTU (year 2004 average is 2 NTU)
- CTS 11: turbidity of the February sample is 5 NTU (year 2004 average is 0.2 NTU)

Table 3.11: Rainfall contamination in the CTSs

CTS 1 - UK	***River***		***Catchment:***	***46,830***	***km²***
Parameter	Unit	Samples	Positive samples	Concentration	
Cryptosporidium	n/L	1	1	0.4	
Giardia	n/L	1	0	-	
Campylobacter	CFU/L	1	0	-	
E. coli O157:H7	CFU/L	1	0	-	
Enterovirus	PFU/L	1	0	-	
E. coli	MPN/L	1	1	111,000	
Clostridia	CFU/L	1	1	>10,000	
Total Coliforms	MPN/L	1	1	517,000	
Enterococci	CFU/L	1	1	35,000	

CTS 3 - France	***River***			***Catchment:***	***10,050***	***km²***
Parameter	Unit	Samples	Positive samples	Average	Min	Max
Cryptosporidium	n/L	2	1	0.5	-	-
Giardia	n/L	2	2	3.05	1.6	4.5
E. coli O157:H7	CFU/L	2	2	10-100 (1)	>1,000 (1)	-
Enterovirus	PFU/L	1	0	-	-	-
E. coli	n/L	1	1	300	-	-
Clostridia	n/L	2	2	5,750	5,500	6,000
Total Coliforms	n/L	2	2	66,000	22,000	110,000
Enterococci	n/L	2	2	4,700	300	9,100

CTS 5 - Sweden	***River & lake***			***Catchment:***	***50,180***	***km²***
Parameter	Unit	Samples	Positive samples	Average	Min	Max
Cryptosporidium[1]	n/L	10	5	0.16	0.1	0.2
Giardia[1]	n/L	10	4	0.18	0.1	0.3
Campylobacter	n/L	10	0	-	-	-
E. coli O157:H7	n/L	10	0	-	-	-
Enterovirus[1]	n/L	7	3	330	250	370
Norovirus[1]	n/L	7	3	148	111	167
E. coli	n/L	13	13	2,635	20	8,300
Clostridia	n/L	12	12	280	80	500
Total Coliforms	n/L	13	13	34,131	3,200	130,000
Enterococci	n/L	13	13	1,318	60	4,300

CTS 7 - Germany	***Groundwater and river bank filtration***			***Catchment:***	***145***	***km²***
Parameter	Unit	Samples	Positive samples	Average	Min	Max
Cryptosporidium	n/L	10	0	-	-	-
Giardia	n/L	10	0	-	-	-
E. coli	MPN/L	10	7	34.7	10	87
Clostridia	CFU/L	9	0	-	-	-
Total Coliforms	MPN/L	10	10	126	10	406
Enterococci	CFU/L	10	3	10	10	10

CTS 10 - France	***Reservoir***			***Catchment:***	***30***	***km²***
Parameter	Unit	Samples	Positive samples	Average	Min	Max
Cryptosporidium	n/L	4	1	1.9	-	-
Giardia	n/L	4	3	0.37	0.2	0.6
Campylobacter	MPN/L	4	1	10-100	-	-
E. coli O157:H7	MPN/L	4	4	10-100 (2)	>1,000 (2)	-
Enterovirus	PFU/L	2	0	-	-	-
E. coli	MPN/L	4	4	19,160	550	54,800
Total Coliforms	MPN/L	4	4	83,838	4,790	242,000
Enterococci	MPN/L	4	4	5,028	100	15,800

CTS 11 - Germany	***Reservoir***			***Catchment:***	***300***	***km²***
Parameter	Unit	Samples	Positive samples	Average	Min	Max
Cryptosporidium	n/L	10	10	0.053	0.031	0.132
Giardia	n/L	10	2	0.006	0.004	0.008
E. coli	MPN/L	9	9	134	42	254
Clostridia	CFU/L	9	9	113	60	210
Total Coliforms	MPN/L	9	9	357	178	504
Enterococci	CFU/L	9	7	28.6	20	60

[1] On concentrate

3.4.2 Results per pathogen

3.4.2.1 Protozoa

Table 3.12 and Table 3.13 show average, minimum and maximum concentrations for *Cryptosporidium* and *Giardia*. These two pathogens are frequently detected at relatively low concentrations. MicroRisk levels vary around:

- *Cryptosporidium*: 0.01-0.5 n/L and up to 4.6 n/L (literature review 0.006-250 n/L),
- *Giardia*: 0.01-1 n/L and over 40 n/L in one case (literature review 0.2-100 n/L).

CTS 1, CTS 9 and CTS 10 have the highest concentrations of *Cryptosporidium*. CTS 3, CTS 9 and CTS 10 have the highest concentrations of *Giardia*. Results are variable and concentrations are not clearly higher during runoff events. However, one must keep in mind that rain events were scarce and that there are many more baseline concentrations available than rain event concentrations. The rain event population may not be fully representative of such concentrations.

Table 3.12: Baseline and rainfall contamination in *Cryptosporidium*

CTS	Event	Unit	Samples	Positive samples	Average	Min	Max	Source
CTS 1	Baseline	n/L	11	2	0.35	0.3	0.4	River
CTS 1	Rain	n/L	1	1	0.4	-	-	River
CTS 2	Baseline	n/L	11	3	0.005	0.001	0.012	River
CTS 3	Baseline	n/L	11	5	0.09	0.05	0.2	River
CTS 3	Rain	n/L	2	1	0.5	-	-	River
CTS 4	Baseline	n/L	12	3	0.12	0.05	0.2	River
CTS 5[1]	Baseline	n/L	13	3	0.09	0.08	0.1	River & lake
CTS 5[1]	Rain	n/L	10	5	0.16	0.1	0.2	River & lake
CTS 7	Baseline	n/L	11	0	-	-	-	Groundwater & river bank filtration
CTS 7	Rain	n/L	10	0	-	-	-	Groundwater & river bank filtration
CTS 8	Baseline	n/L	51	2	0.1	0.1	0.1	Reservoir
CTS 9	Baseline	n/L	25	25	0.33	0.01	4.6	Reservoir
CTS 10	Baseline	n/L	9	5	0.54	0.1	1	Reservoir
CTS 10	Rain	n/L	4	1	1.9	-	-	Reservoir
CTS 11	Baseline	n/L	11	11	0.039	0.019	0.06	Reservoir
CTS 11	Rain	n/L	10	10	0.053	0.031	0.132	Reservoir
CTS 12	Baseline	n/L	10	0	-	-	-	Groundwater

[1] On concentrate

Table 3.13: Baseline and rainfall contamination in *Giardia*

CTS	Event	Unit	Samples	Positive samples	Average	Min	Max	Source
CTS 1	Baseline	n/L	11	0	-	-	-	River
CTS 1	Rain	n/L	1	0	-	-	-	River
CTS 2	Baseline	n/L	11	3	0.02	0.003	0.023	River
CTS 3	Baseline	n/L	11	10	1.16	0.05	4.7	River
CTS 3	Rain	n/L	2	2	3.05	1.6	4.5	River
CTS 4	Baseline	n/L	12	11	0.36	0.05	0.75	River
CTS 5[1]	Baseline	n/L	12	2	0.09	0.016	0.16	River & lake
CTS 5[1]	Rain	n/L	10	4	0.18	0.1	0.3	River & lake
CTS 7	Baseline	n/L	11	0	-	-	-	Groundwater & river bank filtration
CTS 7	Rain	n/L	10	0	-	-	-	Groundwater & river bank filtration
CTS 8	Baseline	n/L	51	1	0.1	-	-	Reservoir
CTS 9	Baseline	n/L	25	25	2.94	0.01	41.3	Reservoir
CTS 10	Baseline	n/L	9	6	0.73	0.1	3	Reservoir
CTS 10	Rain	n/L	4	3	0.37	0.2	0.6	Reservoir
CTS 11	Baseline	n/L	11	1	0.004	-	-	Reservoir
CTS 11	Rain	n/L	10	2	0.006	0.004	0.008	Reservoir
CTS 12	Baseline	n/L	10	0	-	-	-	Groundwater

3.4.2.2 Bacteria

Campylobacter is not always detected in source water. It was found in 4 out of 9 CTSs (Table 3.14). The sample volumes may have been too small. Concentrations sometimes do reach high levels (15,000 MPN/L in CTS 2). Rain event concentrations are not necessarily higher. Literature review referenced 1-109,000 MPN/L.

E. coli 0157:H7 is more commonly encountered but usually at low concentrations. However, CTS 3, CTS 4 and CTS 10 show higher concentrations in some cases and particularly during rain events (Table 3.15).

Table 3.14: Baseline and rainfall contamination in Campylobacter

CTS	Event	Unit	Samples	Positive samples	Average	Min	Max	Source
CTS 1	Baseline	CFU/L	11	0	-	-	-	River
CTS 1	Rain	CFU/L	1	0	-	-	-	River
CTS 11	Baseline	CFU/L	9	0	-	-	-	Reservoir
CTS 2	Baseline	MPN/L	69	57	1,703	0.4	15,000	River
CTS 10	Baseline	MPN/L	9	2	10-100 (2)	-	-	Reservoir
CTS 10	Rain	MPN/L	4	1	10-100	-	-	Reservoir
CTS 12	Baseline	MPN/L	10	0	-	-	-	Groundwater
CTS 3	Baseline	n/L	11	0	-	-	-	River
CTS 4	Baseline	n/L	11	0	-	-	-	River
CTS 5	Baseline	n/L	13	1	10	-	-	River & lake
CTS 5	Rain	n/L	10	0	-	-	-	River & lake
CTS 9	Baseline	n/L	37	32	72.3	0.4	500	Reservoir

[1] On concentrate

Table 3.15: Baseline and rainfall contamination in E. coli 0157:H7

CTS	Event	Unit	Samples	Positive samples	Average	Min	Max	Source
CTS 1	Baseline	CFU/L	11	0	-	-	-	River
CTS 1	Rain	CFU/L	1	0	-	-	-	River
CTS 3	Baseline	CFU/L	11	10	10-100 (9)	>1000 (1)	-	River
CTS 3	Rain	CFU/L	2	2	10-100 (1)	>1,000 (1)	-	River
CTS 4	Baseline	CFU/L	12	8	10-100 (3)	100-1,000 (2)	>1,000 (3)	River
CTS 10	Baseline	MPN/L	9	3	10-100 (3)	-	-	Reservoir
CTS 10	Rain	MPN/L	4	4	10-100 (2)	>1,000 (2)	-	Reservoir
CTS 12	Baseline	MPN/L	10	0	-	-	-	Groundwater
CTS 5	Baseline	n/L	13	0	-	-	-	River & lake
CTS 5	Rain	n/L	10	0	-	-	-	River & lake

3.4.2.3 Virus

Enteroviruses are rarely detected (Table 3.16). In CTS 5, concentrations go up as high as 370 n/L during rain events while they are undetected in baseline conditions. Literature review referenced 0.003-29 n/L.

Noroviruses were investigated in CTS 5 only. Once again, concentrations are clearly higher during rain events (Table 3.17).

Table 3.16: Baseline and rainfall contamination in Enterovirus

CTS	Event	Unit	Samples	Positive samples	Average	Min	Max	Source
CTS 5[1]	Baseline	n/L	12	0	-	-	-	River & lake
CTS 5[1]	Rain	n/L	7	3	330	250	370	River & lake
CTS 1	Baseline	PFU/L	11	4	1.55	0.4	3.4	River
CTS 1	Rain	PFU/L	1	0	-	-	-	River
CTS 2	Baseline	PFU/L	3	2	0.015	0.005	0.024	River
CTS 3	Baseline	PFU/L	11	0	-	-	-	River
CTS 3	Rain	PFU/L	1	0	-	-	-	River
CTS 4	Baseline	PFU/L	12	0	-	-	-	River
CTS 9	Baseline	PFU/L	12	0	-	-	-	Reservoir
CTS 10	Baseline	PFU/L	9	0	-	-	-	Reservoir
CTS 10	Rain	PFU/L	2	0	-	-	-	Reservoir
CTS 12	Baseline	PFU/L	10	0	-	-	-	Groundwater

Table 3.17: Baseline and rainfall contamination in Norovirus

CTS	Event	Unit	Samples	Positive samples	Average	Min	Max	Source
CTS 5[1]	Baseline	n/L	12	0	-	-	-	River & lake
CTS 5[1]	Rain	n/L	7	3	148	111	167	River & lake

[1] On concentrate

3.4.3 Source water quality

3.4.3.1 Levels of contamination

Levels of contamination for baseline and rain events are given in Table 3.18. They represent surface water quality (river and reservoir). Groundwater[1] concentrations are usually very low or below detection limits and are not included.

Table 3.18: Summary of faecal indicators and pathogen concentrations in surface water

	Baseline contamination	Rain event contamination
Faecal indicators		
E. coli	10^2-10^4 MPN/L	10^3-10^4 MPN/L and up to 50,000 MPN/L
Clostridia	≈ 3000 n/L and up to 17,500 n/L	5,000-6,000 n/L
Enterococci	10^2-10^3 n/L	> 10^3 n/L
Total Coliforms	10^3-10^5 MPN/L	30,000-130,000 MPN/L
Pathogens		
Cryptosporidium	0.05-0.5 n/L and up to 4.6 n/L	Concentrations not clearly higher
Giardia	0.01-1 n/L and over 40 n/L in one case	Concentrations not clearly higher
Campylobacter	0-100 MPN/L but up to 15,000 in one case	Concentrations not clearly higher
E. coli 0157:H7	10-100 CFU/L and up to >1,000 CFU/L	Concentrations not clearly higher
Enterovirus	Rarely detected	≈ 300 n/L in one CTS
Norovirus	Not detected (one CTS tested)	150 n/L in one CTS

These results do not account for the recovery of analytical methods. This means that pathogen contamination may be underestimated. Corrections are discussed and developed in Chapter 8.

Rain events undoubtedly yield higher faecal indicators concentrations. However, results are not as clear for pathogens. Three reasons are considered:

- Scarcity of hydrological events for most CTSs; there were many more baseline concentrations available than rain event concentrations. The rain event population may not be fully representative.
- Higher turbidity during rain events; as seen in Paragraph 3.3.2, this may affect the performance of analytical methods and concentrations may be underestimated.
- Dilution effect of rain events on concentrations but not on pollution loads.

NB: Although the MicroRisk dataset does not provide clear evidence of higher pathogen concentrations and loads during peak events, this has been largely shown in the international literature [Stelzer and Jacob, 1991; Atherholt *et al.*, 1998; O'Connor, 2002; Signor *et al.*, 2005].

Reservoir[2] water quality is often better than river[3] water quality. Concentrations are in the low range of Table 3.18. For example, in the case of E. coli, reservoir water concentrations vary

[1] CTS 7 & CTS 12
[2] CTSs 8, 9, 10 & 11
[3] CTSs 1, 2, 3, 4 & 5

around 100 MPN/L in baseline conditions. But it is not always the case. *Giardia* was an exception with highest concentrations encountered in a reservoir during baseline conditions. In rain event conditions, reservoir and river water microbial quality are generally of the same order.

3.4.3.2 Improvement of water quality

River bank filtration, selective intake, dilution and storage are ways to improve river water quality. The MicroRisk project confirmed the performance of these methods.

River bank filtration: CTS 7 (Germany)
CTS 7 uses source water from river bank filtrate (65%) and groundwater (35%). Filtration, sorption and biological processes in the river banks plus dilution with groundwater greatly improve water quality, in baseline or rain event conditions. Concentrations before and after bank filtration are presented Table 3.19 in baseline conditions.

Table 3.19: Microbial concentrations before and after bank filtration in baseline conditions (CTS 7)

Parameter	Unit	River average	River max	Groundwater average	Groundwater max
Cryptosporidium	n/L	0.051	0.112	<0.001	<0.001
Giardia	n/L	0.014	0.024	<0.001	<0.001
E. coli	MPN/L	7,300	22,200	<10	10
Clostridia	CFU/L	645	1300	<10	<10
Total Coliforms	MPN/L	26,100	78,200	29	42
Enterococci	CFU/L	1,700	4800	<10	<10

Reservoir: CTS 8 (Australia)
Source water is pumped from a large surface reservoir. Incoming river quality is greatly improved by dilution, particle settling and physico-chemical and biological processes in the reservoir (Table 3.20).

Table 3.20: Microbial concentrations before and in a water reservoir (CTS 8)

Parameter	Unit	River average	River max	Reservoir average	Reservoir max
Cryptosporidium	n/L	0.78	5.4	<0.1	0.1
Giardia	n/L	0.22	1.7	<0.1	0.1
E. coli	MPN/L	107,000	560,000	125	1,200

3.4.3.3 Data variability

Current QMRA techniques are reliant on the understanding of the overall tendencies and variations in microbiological quality of the source water [Teunis & Havelaar 1999]. Possible variations are due to the specificities of the catchment, seasons, peak events etc. If a parameter, such as pathogen concentration, is known to be a variable and not a constant, it can be quantified in different ways.

A first approach is presented here with the triangular distribution. The triangular distribution is defined by a minimum, average and maximum value. This is of course a starting point

because source water quality cannot be expected to be triangularly distributed, but it is a useful representation of a parameter's variation. It also can be used to assess the sensitivity of the experimental data.

More generally, variability in QMRA is accounted for by describing parameters using a Probability Density Function (PDF). When described by a PDF, the variable may take a range of values, each with a known probability of occurrence. Monte Carlo simulations are then used for risk assessment (see Chapter 7).

3.4.4 Correlation analysis

The following figures illustrate the presence or absence of correlations in the MicroRisk dataset.

Figure 3.8 represents Total Coliforms, Clostridia, Enterococci concentrations and turbidity as a function of E. coli concentrations for the complete dataset. It shows that faecal indicators are generally well correlated together and with turbidity to a lesser extent.

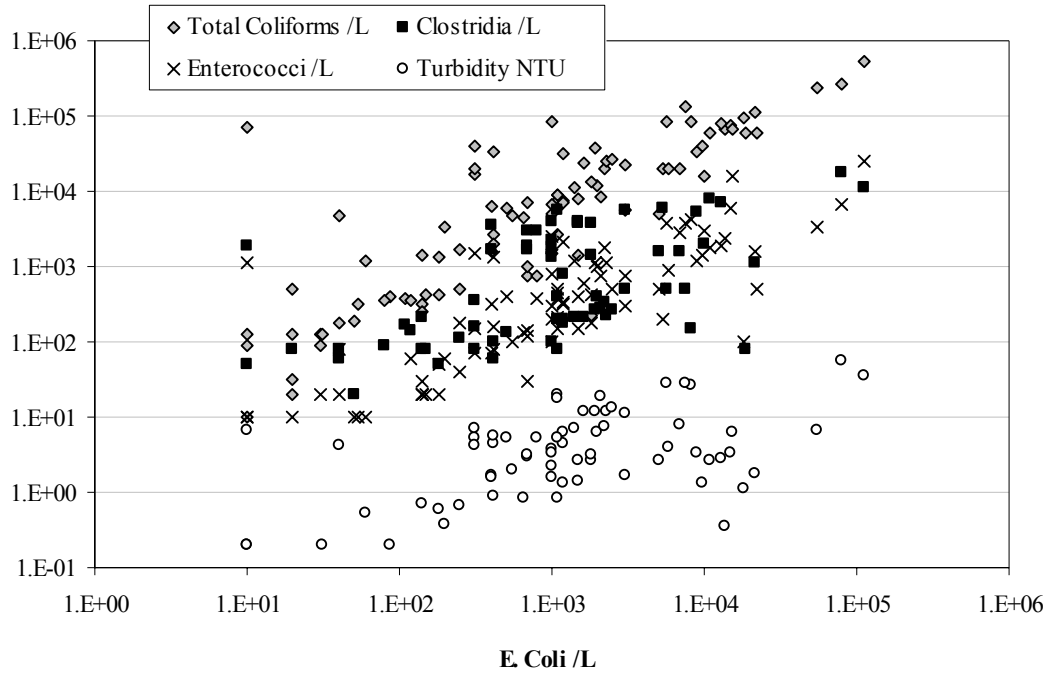

Figure 3.8: Faecal indicators concentrations and turbidity versus E. coli concentrations for all CTSs

When it comes to faecal indicator and pathogen correlations, results are not as clear. For example, E. coli and Total Coliforms concentrations vary together in CTS 11 but *Cryptosporidium* concentrations remain in the same range of values (2-7 n/100 L), independently of faecal indicators (Figure 3.9). Samples with E. coli concentrations over 10 MPNL/100 mL were all collected during rain events. They correlate with Total Coliforms concentrations but not to *Cryptosporidium* concentrations.

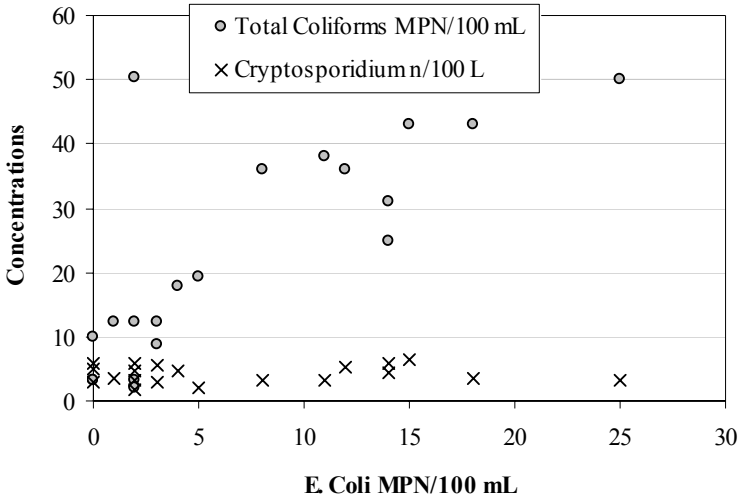

Figure 3.9: Total Coliforms and *Cryptosporidium* versus E. coli concentrations in CTS 11 (Germany)

Cryptosporidium and *Giardia* concentrations are respectively represented versus E. coli concentrations in the case of CTS 5 (Figure 3.10) and CTS 10 (Figure 3.11). These figures show that protozoa and E. coli concentrations are not correlated in these two cases. Rain event concentrations are not necessarily higher than baseline concentrations although high E. coli concentrations during the 24-25/10/2004 rain event are associated with higher *Cryptosporidium* concentrations.

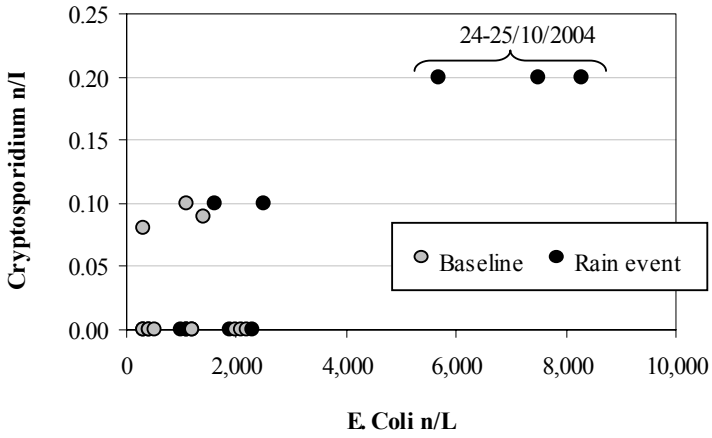

Figure 3.10: *Cryptosporidium* versus E. coli concentrations in CTS 5 (Sweden)

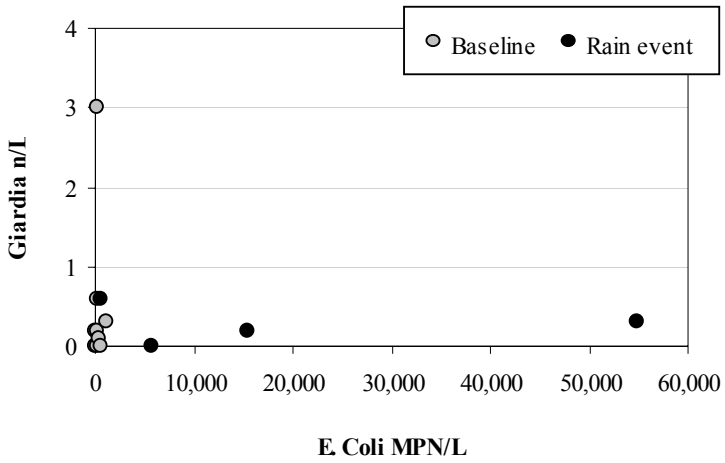

Figure 3.11: *Giardia* versus E. coli concentrations in CTS 10 (France)

Pathogen correlation is different in each case and generalisation is impossible. There is no recurring evidence of pathogens correlated together, correlated with faecal indicators and/or correlated with turbidity. Each CTS has its own behaviour, thus showing that source water quality and links between microbial parameters are site specific.

NB: The link between turbidity and analytical performance was previously discussed (see CTS 10 example in 3.3.2.). A logarithmic relationship between turbidity and recovery of protozoa analytical methods was established in CTS 10. This shows that data adjustment may be necessary to improve correlation investigation.

3.5 CONCLUSION

The MicroRisk project focused on a selection of pathogens of high risk to human health and of concern in source water used for drinking water supply:

- Protozoa: Cryptosporidium and Giardia
- Bacteria: Campylobacter and E. coli 0157:H7
- Viruses: Enterovirus and Norovirus

Source waters are contaminated to varying degrees with these pathogens. Their presence and persistence in water is due to different factors such as survival, transport and control of inputs, depending on the type of surface water and/or aquifer characteristics. Periods of rainfall usually contribute to higher source contamination. The natural variability of potentially pathogenic microorganisms in the environment from anthropogenic, natural, and livestock sources is large and difficult to quantify. It is complex to rank the various sources and transport routes in terms of relative importance to human disease. Risks depend much on the specific case and need to be considered in the local context. This is of course a considerable challenge for water and/or health managers although more and more water utilities do have pathogen data available.

As part of the MicroRisk project, a framework was set to review possible sources of pathogens in catchment areas and to assess of baseline and peak contamination in source

waters. It includes catchment survey and monitoring programs in baseline and peak hydrological conditions. This methodology was applied to nine European and one Australian source waters.

3.5.1.1 Main outcomes

Levels of pathogen contamination

The following table gives the levels of pathogen contamination encountered in the MicroRisk surface source waters. The results are consistent with those found in the literature (Table 3.2); in addition, they differentiate baseline and rain event contamination.

	Baseline contamination	Rain event contamination
Cryptosporidium	0.05-0.5 n/L and up to 4.6 n/L	Concentrations not clearly higher
Giardia	0.01-1 n/L and over 40 n/L in one case	Concentrations not clearly higher
Campylobacter	0-100 MPN/L but up to 15,000 in one case	Concentrations not clearly higher
E. coli 0157:H7	10-100 CFU/L and up to >1,000 CFU/L	Concentrations not clearly higher
Enterovirus	Rarely detected	≈ 300 n/L in one CTS
Norovirus	Not detected (one CTS tested)	150 n/L in one CTS

Surface reservoir water quality is often better than river water quality. Reservoir concentrations are usually in the low range of the above values in baseline conditions. Groundwater concentrations are either very low and/or below detection limits.

Significance of rain events

Hydrological peak events yield higher faecal indicators concentrations in surface waters. Groundwater seems unaffected. Results are not as clear for pathogens. Three reasons are suggested: non-representative rain event population, performance of analytical methods hindered by high turbidity or the dilution effect of a hydrological peak event. Anyhow, even if concentrations do not appear greater in rain event conditions, pollution flows certainly are.

On the question of faecal indicators and pathogens correlation

In most cases, faecal indicators are well correlated among them and with turbidity. However, pathogen correlation is different. There is no recurring evidence of pathogen correlated together, correlated with faecal indicators and/or turbidity. Faecal indicators and turbidity are generally poor surrogates for pathogens presence and concentrations, as reported in the international literature (see 3.1.1). Links between microbial parameters appear to be site specific. All this shows that for a proper assessment of pathogen contamination, baseline and peak event concentrations need to be evaluated in a local context with a specific monitoring program.

Analytical methods

At present, pathogen detection methods are not optimal. There are a number of limitations and sources of uncertainty due to the sensitivity of analytical techniques, particularly for viruses and protozoa, and to the lack of knowledge about the viability and human infectivity of *Cryptosporidium* oocysts, *Giardia* cysts and viruses. Recovery rates of analytical methods may be very low, as seen in the case of *Cryptosporidium* and *Giardia*, and are not always available. Conditions of high turbidity seem to interfere with detection, making it more difficult to assess peak event concentrations. All this may lead to significant underestimation

of pathogen loads. Concentrations should be corrected in regard to recovery rates and turbidity/analytical performance relationships should be investigated.

3.5.1.2 Recommendations for QMRA

Determination of the occurrence of pathogens in source water should be based on:

Catchment survey
The purpose of this step is to develop a broad overview and basic understanding of the catchment, i.e. source water vulnerability, importance and location of pathogen sources, peak events leading to high contamination risks (type, intensity, frequency, duration).

Levels of contamination
Pathogen monitoring of source water should be carried out using the information of the catchment survey. It is particularly important to assess peak event contamination as it usually yields the highest risks. Specific sampling strategies should be designed for baseline and peak event contamination.

Quality of the data
The pathogen detection methods are ideally targeted to viable and infectious pathogens. The performance of the detection methods can have implications for the applicability of the data in risk assessment. These should be identified and evaluated in the early stages of the process.

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4 Efficacy of water treatment processes

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4.1 RATIONALE

Drinking water treatment of surface water was originally started to improve the aesthetic properties of drinking water. By the time of the Egyptians (15^{th}-13^{th} century BC) and Romans (300 BC-200 AC) settling was applied to reduce turbidity and in the 5^{th} century B.C. Hippocrates, the Father of Medicine, invented the "Hippocrates Sleeve", a cloth bag to strain rainwater. Supply of settled and filtered water in modern times started in 1804 (Scotland) and 1806 (Paris). Initially slow sand filters were used to provide a more aesthetic product and soon filtration was recognised to reduce outbreaks of typhoid and cholera. In the 1870's Robert Koch demonstrated that bacteria existing in water supplies could cause disease and he studied water filtration systems that were effective in removal of bacteria after the Hamburg cholera outbreak of 1892. In his biography of Koch's work, Brock [1988] states that "water filtration has probably saved more lives than immunization and chemotherapy combined". In 1906 the first ozonation plant for disinfection was started in France, and chlorination became common practice around the same time, although promoted by John Snow after his pioneering epidemiologic studies during London's cholera outbreaks of the 1850's. From 1920 the combination of sedimentation, filtration and chlorination virtually eliminated epidemics of classical waterborne diseases, such as cholera and typhoid, in areas so supplied [AWWA, 2006]**.**

However, outbreaks of waterborne disease due to poor drinking water quality still occur today, even when treatment is in place. Chapter 1 provides an overview of 84 outbreaks in Europe between 1990-2004. Further fault tree analysis indicated 18/24 groundwater and 17/22 surface water supply outbreaks were, at least partially, caused by insufficient treatment or treatment failure. Both chronic and temporary filtration failures were identified as major contributors in the fault tree analysis. When contamination of groundwater supplies goes unnoticed, the need for additional treatment is not recognised and treatment can be chronically insufficient. Within an additional 30 outbreaks, studied in Sweden, 57% were due to faecally contaminated raw water receiving insufficient treatment.

From 1974 to 2002, 26 out of 35 outbreaks in the USA and Canada, as reported by Hrudey and Hrudey [2004], were due to surface water treatment failure or inadequate treatment to deal with sudden peak increases of pathogen concentrations in source water. Some major outbreaks like that of cryptosporidiosis in Milwaukee where treatment efficiency was compromised, would have been prevented or the impact on human health reduced, by adequate treatment. Some outbreaks resulted from a

combination of increased source contamination (mostly due to rainfall) and treatment failure. This illustrates that treatment needs to be able to deal with peak events in source water that are not prevented by source protection. The outbreaks due to peak events also showed that recognising events and taking corrective actions is essential to prevent outbreaks. Treatment is often the critical control step in the chain of barriers where sufficient (real-time) monitoring can recognise events and where corrective action can take place. Rapid changes in water quality should always be considered as indicators of events.

Although counteracting peak events is necessary to prevent outbreaks, sufficient treatment during baseline (normal) conditions is also required to safeguard public health. In specific situations the sporadic cases (during baseline conditions) appear to represent a greater proportion of waterborne disease than outbreaks [Nichols, 2003]. This was also a conclusion reached for a water supply system in Gothenburg, based on failure reporting and quantitative risk assessment [Westrell *et al.*, 2003]. Pathogens are likely to be present in most surface waters (possibly below detection limits) and sufficient drinking water treatment is required to eliminate them. The required treatment depends on the level of source water contamination and relates to the health-based target for the population that drinking water is provided for. WHO [2004] states "Performance targets are most frequently applied to treatment performance – i.e., to determine the microbial reduction necessary to ensure water safety". The health based target suggested by WHO is 10^{-6} DALY per person per year. For a given source water quality, the treatment performance target (log-removal) can be calculated using the health target and translating this back, using dose-response data to maximum exposure levels of pathogens through drinking water, as is illustrated in Figure 4.1, taken from the WHO "Drinking-water Guidelines" [WHO, 2004].

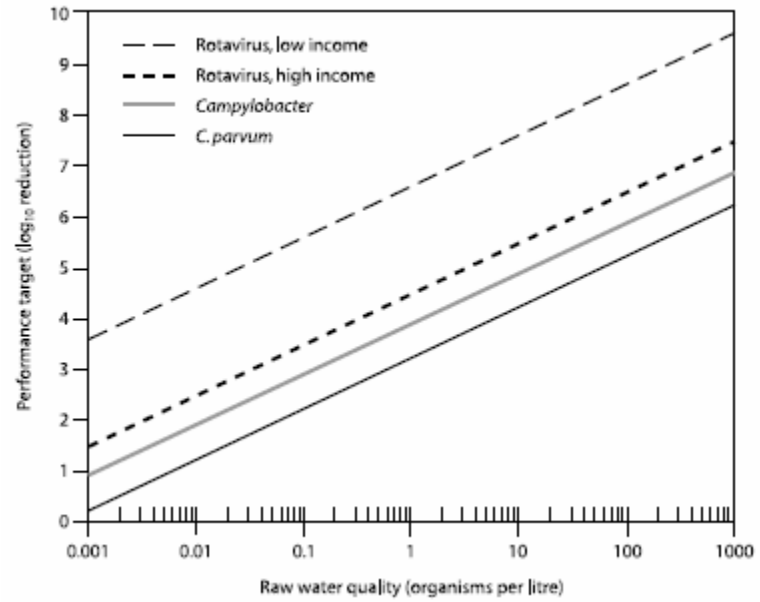


Figure 4.1 Performance targets for selected bacterial, viral and protozoan pathogens in relation to source water pathogen concentration (to achieve 10^{-6} DALYs per person per year) [WHO, 2004].

As an example a river water in The Netherlands used as source that contains up to 10 *Cryptosporidium* oocysts per litre and needs to be treated to not exceed the maximum tolerable oocysts concentration in drinking water of $6.3*10^{-4}$ oocysts per litre. Thus, such water requires treatment to provide 4.2 log reduction of *Cryptosporidium*. Similarly required reduction can be calculated for the other chosen pathogens: *Giardia*, *Campylobacter*, *E. coli* O157, Enterovirus and *Norovirus* in the MicroRisk CTSs. The example uses point estimates but as discussed in Chapters 7 & 8 the assessments attempt to account for variation and uncertainty.

All current drinking water treatments processes show variations in treatment efficacy. Source water quality changes with time resulting in different performance of the treatment process. Temperatures influence the efficacy of disinfection processes if dosing regimes are not adapted accordingly. Varying amounts of suspended solids can impact on sedimentation and filtration as well as disinfection. Furthermore treatment efficacy varies due to production flows, filtration backwash cycles and chemical dosing control loops. These known regular variations should always be accounted for in design and operation. In addition to these regular variations, hazardous events can occur. Pathogen concentration peaks in source water due to heavy rainfall have been recognised as causes of outbreaks [Hrudey and Hrudey, 2004], in addition to equipment failures, power failures or human errors. The resulting short periods of reduced efficacy can have a large impact by leading to a local outbreak, but may also affect the total yearly average health risk. For example: if a treatment normally provides 4 log inactivation but during 1 day in a year the removal is reduced to 1 log; this results in an increase of the yearly average number of organisms in the treated water, which becomes 4 times higher. Therefore an assessment of the frequency, duration and magnitude of hazardous events are all essential for properly undertaking a quantitative risk assessment. Special attention needs to be paid to simultaneous effects of hazardous events on source water concentrations and the different treatment processes.

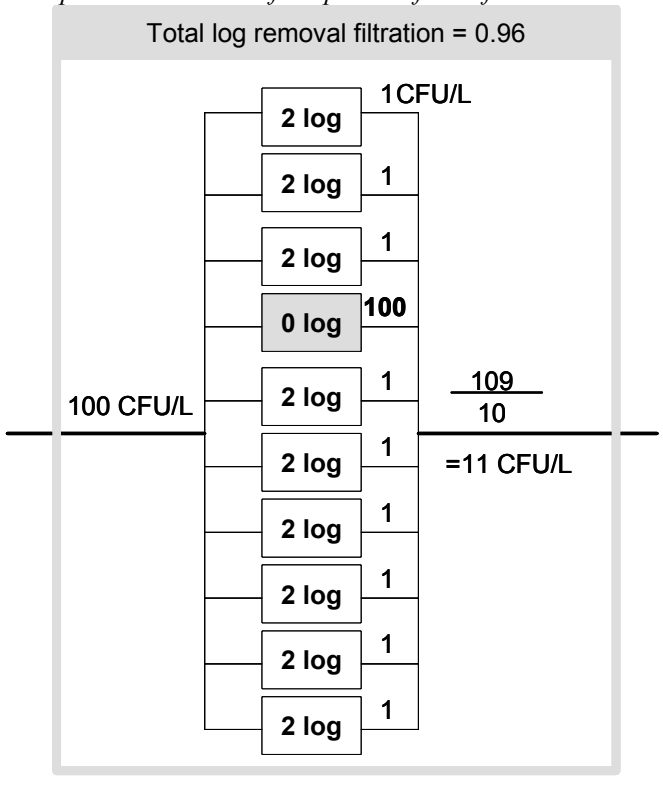
Hence, data collected for treatment assessment, as well as other parts of the QMRA model (Chapter 7), needs to reflect normal process variations and hazardous events. Treatment assessment relies on microbial as well as operational data. Monitoring data for pathogens is generally unavailable and indicators are seldom monitored throughout treatment and often at best weekly. Short-term events are therefore unlikely to be picked up. Statistical models based on operational data, therefore, generally provide the best estimate of regular variations in treatment efficacy and can be applied to extrapolate the occurrence of rare events. Hazardous events are however not likely to be part of regular variations and might require different monitoring approaches. A statistical assessment normally relies on monitoring and most treatment plants only perform monitoring of raw and treated water in order to comply with legislation. Monitoring after treatment will generally result in large datasets of non-detect samples. Probabilistic assessment can then provide the likelihood that treatment reduction was above a certain limit, but seldom provides the most likely reduction. One cause of uncertainty is that the concentrations in raw water vary with time and pre- and post-

treatment (step) samples are seldom representative of the total volume being treated. In addition, the treatment can vary between parallel units and in time, adding to sample variability. Microbiological analysis also includes a number of uncertainties of which variable recovery of pathogens is well known (Chapter 7). For example, culture-based techniques do not pick up injured but still infectious pathogens [Villarino *et al.*, 2003] and such disinfected organisms may undergo repair mechanisms [von Sonntag *et al.*, 2004]. When data on process conditions is used to model pathogen reduction, errors in the process models further add to uncertainty. Model parameters such as disinfection kinetics or log reduction for specific process conditions are often inconclusive or simply not available for some pathogens. Also model assumptions on hydraulic characteristics or other affecting mechanisms can cause considerable uncertainties. So despite extensive monitoring, the present approach with grab samples cannot fully account for all the produced water variabilities and uncertainties. Nonetheless, using the probabilistic methods presented here and in Chapter 7, factors related to variability and uncertainty are partly accounted for and are propagated throughout the risk assessment.

In risk assessment a full-scale system is represented by a model. Since a model is a simplification of reality, the amount of detail applied needs to match the purpose of the assessment (Chapter 7). In the exposure assessment (Figure 7.1) treatment is represented as a single barrier, but the multiple processes in most treatment systems, sometimes referred to as barriers, make the treatment a multiple barrier system in itself [WHO, 2003]. By combining different types of processes in series, failure of one barrier can be partially compensated by others. In practice each treatment barrier consists of several parallel process units, like filters or sedimentation tanks. The poorest performing unit then determines the total efficacy of the barrier [Gale, 2002]. This is illustrated by an example for filtration, which normally provided two log *Cryptosporidium* removal (see Box I). When one out of ten parallel filters failed completely (no removal) the total efficacy of the filtration step was reduced to 1 log removal. The barrier efficacy is thus

BOX I: Weakest link

When a treatment step consists of several parallel treatment units, the poorest performing unit will dominate the pathogen removal. This is illustrated in the following example where one out of ten parallel filters fails:



determined by its 'weakest link'. In this chapter a further focus is on how units within one process work together to safeguard against pathogens in source water.

So why model reduction by treatment instead of monitoring the produced drinking water? Chapter 3 showed that the level of presence (or absence) of pathogens in faecal polluted source water can be determined by water quality monitoring with reasonable effort. Chapter 5 showed that intensive monitoring of faecal indicators in finished water still left a major uncertainty about the presence of pathogens in the treated water since the ratio between indicators and pathogens is unknown. Table 5.10 in Chapter 5 also shows that the percentage of positive indicator samples leaving the plant is of the same order of magnitude as the percentage of positives in the distribution system. Although this does not indicate that no contamination takes place during distribution, it does show that the water quality leaving the plant is relevant for risk assessments. The WHO guidelines for drinking-water quality state "Water Quality Targets are typically not developed for pathogens, because monitoring finished water for pathogens is not considered a feasible or cost effective option" and prefer the application of performance targets (Figure 4.1). The statutory *Cryptosporidium* monitoring in the UK exemplify a dataset of finished water pathogen monitoring. Analysis in Paragraph 4.7.4 illustrates the limitations of this data for quantitative treatment assessment. Hrudey and Hrudey [2004] showed that the occurrence of false positives makes it virtually impossible to estimate indicator bacteria concentrations in drinking water by monitoring at the observed low level. By quantifying the pathogen reductions in treatment, indicator and pathogen concentrations entering the distribution network can be estimated even at levels far below current detection limits. By applying probabilistic methods the probability of pathogens occurring in a certain volume of produced drinking water can be assessed. This probability can then be used in the total QMRA chain [Haas *et al.*, 1999; Chapter 7]. Several studies on quantitative risk assessment have pointed out that the main uncertainty lies in estimating pathogen concentrations in drinking water leaving the treatment plant [Teunis *et al.*, 1997; Haas and Eisenberg, 2001]. This chapter aims towards providing data and approaches to reduce that uncertainty.

Apart from providing an estimate of pathogens in drinking water over the past period, the treatment assessment also provides valuable information for preventing future risks. Firstly it increases the knowledge of the treatment system where the approach to quantify treatment efficacy raises new questions. What is the hydrology in my disinfection contact tank or (how) do we adjust the addition of coagulants when the water temperature changes? What is the impact of a break-through in one of a series of filters? Sadly a huge amount of (operational) data is collected at most treatment plants, without being used. Analysing some of these datasets can reveal the actual performance of the plant, the challenges it faces and the way operators react to these challenges. So the treatment assessment helps you to 'know your system', a key component to risk assessment mentioned in Chapter 2. By using operational data and monitoring outcomes the evaluation of pathogen reduction and the effect of changes on the quantified risk end-point will be refined. Based on these outcomes critical limits can be set on monitoring in relation to operational parameters to keep the risk at the baseline level and prevent events. When treatment moves beyond critical limits at one point and

cannot be corrected at its source, corrective action needs to be taken at another point in the treatment chain in order to maintain the efficiency of the overall treatment barrier. Undertaking a treatment assessment also provides information for where monitoring and additional data collection will reduce treatment data uncertainty. Thus resource use can be focussed at the most appropriate points.

This chapter discusses the common drinking water treatment processes in relation to quantitative risk assessment for the MicroRisk project. A short overview of treatment principals and its capacity to eliminate pathogens is provided. Next, the chapter introduces the twelve systems that have been assessed within the MicroRisk project. A framework for interpreting the data collected follows and is then applied to the systems. Rather than presenting all assessments in full, examples from these are used to illustrate the treatment assessment approaches that ranged from simple to complex. Also a full assessment of one system is presented. Finally the experiences from performing the assessments are discussed and conclusions drawn. Chapter 7 provides more background on the applied statistical approaches, and integration of the treatment data into each catchment-to-customer (CTS) QMRA is addressed in Chapter 8.

4.2 TREATMENT PROCESSES THAT ELIMINATE PATHOGENS

4.2.1 Pathogen reduction in drinking water treatment

Drinking water treatment strives to provide safe and aesthetic drinking water in sufficient quantities. Chemical flocculation and filtration reduce pathogens as well as turbidity and dissolved organic carbon (DOC). Oxidation processes like ozonation disinfect the water, reduce colour, taste and odour, break down micro-pollutants and can have a positive effect on subsequent clarification processes. Disinfection can also have adverse health effects by producing disinfection by-products (DBPs) [USEPA, 2003].

Outbreak surveys [Hrudey and Hrudey, 2004; Chapter 1] show that contaminated groundwater is a major source. The treatment of groundwater is generally less extensive than for surface water and is not primarily aimed to eliminate faecal pathogens. In some cases disinfection will take place partly when the groundwater is known to be of variable quality and partly to reduce bacterial regrowth potential in the distribution system. When contamination of a groundwater source is suspected, the approaches for surface water treatment presented in this chapter can also be applied to groundwater treatment.

When assessing pathogen reduction by treatment, all treatment processes that are known to affect pathogens are addressed, even if they were not originally aimed at reducing pathogens, such as pre-oxidation. The assessment strives to provide a best estimate of pathogen reductions, which includes an assessment of variability and

uncertainty. Minimal reduction is typically the only data considered when supplied information consist of 'removal greater than …', which is often the case when monitoring results in only non-detects. Potential reduction is the reduction that might be expected when data is insufficient to set an upper limit ('removal smaller than …'). For the assessment itself a conservative safe approach is taken, with minimal and potential reductions estimated or derived from expert judgement. Sensitivity analysis utilising these estimates, as discussed in Chapter 7 can provide insight into the uncertainty and importance of these treatment reduction estimates. It also provides a rationale to support decisions for further data collection or treatment adaptation.

Treatment can be divided in two reduction principles; particle removal and disinfection. In order to compile current knowledge on pathogen reduction by particle removal processes, a literature research was conducted based on lab-scale, pilot-scale and full-scale conditions [Hijnen *et al.*, 2005a]. The resulting dataset of microbial reduction provides reference values for full-scale treatment assessment, referred to as Mean Elimination Capacity (MEC[a]). The full-scale conditions were given higher weight-of-evidence in the MEC estimation than laboratory assessments. To illustrate the variation observed in literature, minimum and maximum reported reductions were also presented. Effects of specific design parameters, operation and water quality are discussed separately and only quantified in the range of "better" or "worse" than average.

Chemical disinfection is typically related to process conditions like disinfectant concentration, contact time, temperature and other associated water quality characteristics. Disinfection tables or kinetic parameters as reported in guidelines [USEPA, 2003] were used for some of the MicroRisk index pathogens. For the remaining index pathogens, disinfection kinetic parameters were obtained from a limited literature review.

Several sources in literature and guidelines provide an overview of reported removal or inactivation of typical treatment processes for a range of organism [Sobsey, 1989; LeChevallier and Au, 2004; USEPA, 2003; Hijnen *et al.*, 2005a, b]. The sections below on process characteristics and reduction principles are not intended as a handbook but rather to provide 'ballpark figures' from literature and are illustrated in the examples in subsequent sections of the chapter. The information provided in Sections 4.2.2 to 4.2.6 is based on [Hijnen *et al.*, 2005a, b] except when stated otherwise.

[a] Each experiment in the review was awarded a weight according to how closely it resembled reality. Experiments conducted in the laboratory with cultured micro-organisms resulted in a weight of 1, an experiment conducted at full-scale using environmental micro-organisms already present in the water resulted in the maximum weight of 5. The MEC was calculated as the weighted average of all experiments for one type of treatment process and micro-organism (sum of log removal*weight per experiment, divided by the total sum of weights). The FS-score is the average of all weights used to calculate the MEC. A FS-index of 5 means all studies were performed at full scale with environmental micro-organisms.

4.2.2 Coagulation-flocculation, sedimentation, flotation

Principle
Chemical coagulants added to water result in floc formation. These flocs partly capture dissolved and colloidal matter, and particulate materials, and are subsequently removed by clarification processes. The temperature, dose of coagulants, pH and the characteristics of the organic matter govern the overall efficiency of chemical treatments. Limitations may be that small flocs partly remain in the water after clarification, protecting micro-organisms within the floc structure. Because of their size (0.02-10 μm) free suspended micro-organisms can be regarded as colloids, which under normal conditions do not settle at a significant rate. Settling is enhanced by coagulation-flocculation. The negative surface charges of the colloids are neutralized, enhancing the formation of larger particle aggregates during mixing. Dosed polymer aids may be used to further enhance floc formation. Processes like, settling, floatation, clarification or filtration will remove the flocs. For all floc removal processes, stable conditions are essential, hence sudden flow changes should be regarded as hazardous events and minimised. Design, operation and water quality all influence the efficacy of flocculation. To assess the local operational conditions, regular (jar)tests are required to optimize the system and adapt it to changing conditions. Factors influencing pathogen removal are briefly discussed.

Clarifier types are chosen based on local conditions including, costs, maintenance and local suitability. Hijnen [2005a] however reported that variation in pathogen removal within one type of clarifier was similar to variation between different types. Most systems are able to provide adequate removal if properly optimised and operated for the local situation.

The coagulant and polymer type need to be chosen in relation to the water quality and process design. Disease outbreaks following conventional treatment (Chapter 1) illustrate the need for frequent optimisation. For example, preceding the Milwaukee outbreak, the coagulant was changed from alum to alum chloride resulting in smaller flocs and thus, less entrapment of smaller particles like *Cryptosporidium* oocysts (4-5 μm dia), which eventually contributed to the outbreak [Hrudey and Hrudey, 2004].

Coagulant and coagulant aids (polymer) dose and mixing energy are the main operational parameters for conventional treatment. The dose of coagulant and polymer needs to be adjusted to the amount and type of particles in the water. For example, algal blooms are known to have an adverse effect on clarification. The coagulant is rapidly mixed during dosing, followed by gentle mixing in order to facilitate the flocculation, without short-circuiting. Sometimes pre-oxidation is applied before or in combination with coagulation to enhance process efficiency. A optimisation of dosing and settling can improve *Cryptosporidium* removal from less than two to almost five log units while total particle removal can be improved from 2 to 3 log [Dugan *et al.*, 2001].

Water quality: A pH between 5.8 and 8.5 is necessary when using alum as coagulant, otherwise poor coagulation is likely. Sufficient alkalinity is also required when using

Alum. Cold water temperature will interfere with coagulation and sedimentation by decreasing the settling rate of the flocs. At low turbidity (< 1-5 NTU), insufficient material can be present to form suitable flocs. Thus co-entrapment of pathogens with flocs, whose presence in not always related to turbidity, is also reduced. High turbidity may require an increase of coagulant dose to effectively flocculate particulates and pathogens.

Operation
Settling is generally operated as a continuous process, which requires skilled operation, backed up with regular jar-tests to provide optimal treatment under all conditions. The operation includes flow control, chemical dosing (coagulant, coagulant aid, pre-oxidation, pH adjustment), mixing conditions, sludge blanket height, sludge removal rate, air flow rate (if flotation used) and other conditions, depending on the process type. All these can have a significant impact on pathogen removal.

Indicators
Surface properties of micro-organisms may impact on their removal, however, that is largely reduced by the presence of sufficient coagulant. Inactivation can play a role when pre-oxidation is practiced, especially for bacteria and viruses. This is discussed in more detail in Section 4.2.5 (disinfection). Other studies have shown that some process indicator micro-organisms like MS2 phage can be very sensitive to chemical dosing (see experiments in Paragraph 4.7). Hence the zone of surrogate micro-organisms introduction (before or after chemical dosing) may have significant impact on the assessed removal in challenge tests. Experimental studies have shown that removal of most micro-organism types is quite similar (Table 4.1).

Surrogates
Turbidity is traditionally used to monitor clarification and in some cases particle removal is used. The latter cannot be used as an index of pathogen removal in the chemical treatment, since new particles are formed during the process. Auto fluorescent micro-algae from surface waters, in the size fractions between 1-20 μm, however, seem to account for this and may potentially be used as an in-line surrogate for particle removal. Stable low turbidity does indicate good performance, but there is no direct relationship with the removal of pathogens. Sudden peaks in turbidity can indicate events of poor performance, but conversely, peaks in pathogens may not be associated with turbidity peaks.

Critical limits
Critical limits can be set to ensure proper functioning. High turbidity or low particle removal is an obvious trigger for corrective actions. Use of streaming current detectors and zeta potential monitors to measure net surface charge of particles is currently studied and could lead to operational guidelines and limits based on local jar tests.

Table 4.1 The MEC (mean elimination capacity) of micro-organisms by coagulation/floc-removal

Organisms	Data characteristics			MEC		
	Studies	Data	FS-index[a]	MEC	50%ile	Range
Viruses	5	12	3.5	1.8	1.7	0.2 – 4.3
Bacteria[b]	6	9	4.7	1.5	1.4	0.6 – 3.7
Bacterial spores	6	11	4.7	1.4	1.2	0.8 – 3.2
Cryptosporidium	8	13	3.9	1.9	1.8	0.4 – 3.8
Giardia	8	12	4.3	1.6	1.3	0.0 – 2.9

[a] the higher the number, the more experiments resembled full scale and environmental organisms
indicator bacteria (*E. coli,* coliforms, faecal streptococci)

Other types of particle formation and removal processes in addition to conventional treatment (see further 4.2.4), includes direct filtration and natural sedimentation. Natural sedimentation, without addition of chemicals, generally takes place in (storage) reservoirs before treatment and is not controlled.

4.2.3 Granular media filtration

Principle

The reduction mechanisms of micro-organisms during filtration through porous media are a combination of size exclusion, physical/chemical adsorption/desorption and biological competition/predation. In the process, water is directed through a bed of granular media, generally a range of fine sands. Particles are entrapped between or attach to the filter grains. During backwash particles are partly rinsed off and disposed of with the backwash water. Micro-organisms that remain attached either die-off or detach and thereby penetrate the barrier. The attachment processes of small particles are generally described by colloid filtration theory [Yao *et al.*, 1971]. The strength of attachment to the filter grain surface is determined by particle and grain surface characteristics (electric charge, pre-coating, hydrophobic/hydrophilic interactions) and water characteristics (salt content, pH, DOC). The adsorption-efficiency varies substantially between different types of organisms and even between different strains of the same species [Gerba *et al.*, 1980]. Efforts to predict filtration efficiency incorporating these effects have not yet lead to satisfactory results due to the large number of variables to account for, but generally the following relations have been observed:

Bi- and trivalent cations increase the adsorption efficiency by reducing electrostatic repulsion. At pH 5-7 the adsorption efficiency is higher than at higher pH, although the effect depends on the iso-electric points of the particles and the filter grains. Organic compounds compete for attachment sites to filter grains, but since attachment does not appear to be site-limited, DOC may have a minor effect [Lo and Sproul, 1977; Gerba, 1984; Dizer, 1988]. Surface structure and ionic exchange capabilities of filter material can increase adsorption, although this is less well quantified [Elimelech and O'Melia, 1990]. Changes in flow can cause attached organisms to detach by sheer forces. Detachment is a goal during backwash, but sudden changes in filtration velocity can also lead to increased turbidity or particle counts post filtration. Apart from the initial effect of straining, the log removal of micro-organisms is expected to relate to filter bed

depth. In theory removal of micro-organisms is higher at lower filtration rates. Pilot studies have supported this relationship, although not as strongly as predicted. Table 4.2 illustrates that the observed removal of different types of micro-organisms in experiments is not as large as theoretically expected.

Operation
Filters are operated in cycles (between backwashing) of generally one to several days. After backwash turbidity is generally higher, but normally stabilises at low levels in the filtrate after 15 to 45 minutes. The filter resistance (pressure drop across the filter) slowly increases during filtration when the removed particles are building up in the filter. By the end of the filter cycle an increase of turbidity can sometimes be observed again, indicating breakthrough of particles. Huck *et al.* [2002] showed that removal of *Cryptosporidium* can be severely impaired at this stage, while Emelko *et al.* [1999] observed breakthrough already before turbidity increase was observed. Other studies have shown a gradual reduction in removal efficiency down to 50% of the initial log removal during the filter cycle. The filter is backwashed by reversing the flow at an increased velocity, often combined with air bubbles. The accumulated matter is disposed of with the backwash water and the filter cycle starts again. Many variations of filter operations are possible and filter operation requires skilled personnel. More detail on the operation and maintenance of filters can be found in [Hrudey and Hrudey, 2004]. Significant to pathogens is that, settled backwash water is in some waterworks returned to the head of the plant to reduce water losses. From a risk perspective this is not recommended, since this increases the potential pathogen concentration at the head of the plant, combined with a period of less efficient filter performance during the new cycle, and has though to be responsible for the prolonged plant challenge during the Swindon cryptosporidiosis outbreak [Badenoch *et al.*, 1995]. Filter cycle management in relation to pathogen reduction is further discussed under critical limits.

Pathogen indicators or model micro-organisms
Since filtration relates to the size of particles, the size of the pathogen indicator (called a model micro-organism [Ashbolt *et al.*, 2001]) needs to be the same as the size of the pathogen. Phages can be used as models for viruses, bacteria for bacterial pathogens and bacterial spores model protozoan oo/cyst removal. Surface properties differ between strains and also within a population of the same strain. Hijnen [2005b] reported that the surface properties of *Cryptosporidium* change with age. While there is no perfect model for any pathogen, as even pathogens of the one group vary greatly, for risk assessment the above proposed model micro-organisms were applied from a practical point of view (it's the best we got).

Surrogates
Traditionally turbidity has been used as a surrogate for filter performance. A direct relation between turbidity and micro-organism removal does not exist. However a stable and low turbidity of the filtrate does indicate that a filter is working properly, and based on this it can be assumed that the filtration removal is efficient as a barrier step. The USEPA "recognizes that turbidity reduction is not a direct indication of pathogen removal, but it is an effective indicator of process control" [USEPA, 2003]. Rather, it is

the variation in drinking water turbidity that should be targeted and this has also been shown as an association to sporadic disease (see Chapter 1). Particle counting at different size fractions is a more sensitive technique than turbidity, yet more costly. Reduction of different sized particles enables to differentiate between different groups of pathogens, although it is still not a direct measurement of pathogen reduction. This is exemplified in Section 4.7 where reduced particle removal at the start of a filter cycle, did not correspond with the *E. coli* reduction.

Critical limits

Once a filter has been put into operation, the effect relies on the continuous operation and avoiding changes in flow rate or water quality. Periods of poor removal can be circumvented by applying filtrate to waste after backwashing and backwashing before breakthrough occurs, based on appropriate operational limits. This is currently assessed through turbidity measurement or particle counting after filtration with a high-enough resolution to account for short-time variations. Prior to the Milwaukee outbreak treated water turbidity increased from 0.1 to 1.5 NTU, and similar turbidity peaks were observed at other outbreaks [Hrudey and Hrudey, 2004]. As a control measure, the USEPA [2003] states that 95% of time 0.3 NTU must not be exceeded in filtered water, and turbidity must never exceed 1 NTU when monitoring each individual filter with a frequency of 1/15 minute or higher in order to apply 0.5 log removal credit for *Cryptosporidium*. When 0.1 NTU is maintained a 1 log removal filtration credit can be applied. Remarkably these restrictions are not required during the 15 minute period after backwash when the highest turbidity is expected. The USEPA [2003] applies 0.5 log removal credits for a second stage filtration for *Cryptosporidium.* WHO [2004] indicates < 1 log removal for most micro-organisms under baseline conditions and a maximum of 2-4 logs.

Other sudden changes in water quality (pH, electrical conductivity) are also precursors of events and, based on their operational identification, this is corrected either upstream (improve clarification) or downstream (increase disinfection dose). When a single filter in an array of parallel filters fails, this filter may be taken out of production, since it will have a large impact on the overall performance (see also example in Box I).

Table 4.2 The MEC (mean elimination capacity) for micro-organisms by rapid sand filtration

Organisms	**Data characteristics**			**MEC**		
	Studies	Data	FS-index[a]	MEC	50%ile	Range
Viruses	12	63	3.2	0.8	0.6	0.1 – 3.8
Bacteria[b]	12	109	3.2	0.6	0.6	0.1 – 1.5
Bacterial spores	11	102	4.2	1.3	1.4	0.0 – 2.9
Cryptosporidium	15	151	3.3	2.0	1.8	0.0 – 3.1
Giardia	10	124	3.3	1.7	1.6	0.0 – 6.5

[a] the higher the number (1-5), the more experiments resembled full scale and environmental organisms; [b] indicator bacteria (*E. coli,* coliforms, faecal streptococci)

Other types of granular media filtration

Granular Activated Carbon (GAC) filtration

GAC filtration uses granular activated carbon as filter material and is generally applied as second stage filtration to remove taste and soluble micro-pollutants but will also account for particle removal (Table 4.3). GAC filters are sometimes operated up to several months without backwashing when the inflow contains little suspended material. The filter material is relatively coarse, but its surface characteristics can improve attachment. Relatively few well-performed studies on GAC filtration are available, with partly contradictory results, both indicating better and worse removal of micro-organisms as compared to rapid sand filters [Hijnen *et al.*, 2005b].

Table 4.3 The MEC (mean elimination capacity) for micro-organisms by granular activated carbon (GAC) filtration

Organisms	**Data characteristics**			**MEC**		
	Studies	Data	FS-index[a]	MEC	50%ile	Range
Viruses	2	10	2	0.4	-	0.2 – 0.7
Bacteria[b]	3	16	3	1.4	-	0.9 – 2.9
Bacterial spores	4	8	5	0.8	-	0.4 – 1.2
Cryptosporidium	1	12	3	0.9	-	0.7 – 1.1
Giardia	2	16	3	1.7	-	0.4 – 3.3

[a] the higher the number (1-5), the more experiments resembled full scale and environmental organisms; [b] indicator bacteria (*E. coli,* coliforms, faecal streptococci)

Slow sand filtration

Slow sand filters are operated at low filtration rates (0.1-1.0 $m.h^{-1}$) and contain fine sand (0.2-1.0 mm) with a filter bed depth of 0.5 to 1 m. Headloss slowly increases due to filter ripening and build up of the 'smutzdecke' in the top layer, where the main reduction of micro-organisms appears to occur, partly due to biological processes like predation and partly due to adsorption and size exclusion in the pore matrix. After the filter bed surface is scraped off (months –year interval) when headloss becomes too great, filter to waste is applied and several weeks need to be allowed for filter ripening before it goes back in production. The fine filter material, low filtration rate, no backwash and biological activity in the filter make it a potentially effective barrier against pathogens and will level out peaks from the inlet water (Table 4.4). Due to retardation (attachment and detachment) a long-term, gradual increase of micro-organisms breakthrough may be observed over time.

Table 4.4 The MEC (mean elimination capacity) for micro-organisms by slow sand filtration

Organisms	**Data characteristics**			**MEC**		
	Studies	Data	FS-index[a]	MEC	50%ile	Range
Viruses	10	13	3.3	2.2	2.1	0.6 – 4.0
Bacteria	9	17	3.4	2.7	2.4	1.2 – 4.8
Bacterial spores	5	9	4.4	1.5	1.3	0.0 – 4.0
Cryptosporidium	6	8	3.4	3.8	nd	0.3 – >6.5
Giardia	3	3	3.7	3.3	nd	1.2 – 6

[a] the higher the number (1-5), the more experiments resembled full scale and environmental organisms;

Bank filtration and artificial recharge
The processes of bank filtration and artificial recharge are similar to slow sand filtration. Since filter bed composition is less well controlled, there is a potential for the presence of macro-pores reducing efficiency. Due to the natural processes in the soil, the water characteristics can change (redox potential, salt content) which in turn influences pathogen attachment efficacy. Bank filtration is particularly vulnerable to high water levels, which reduce the travel distance between the surface water and the abstraction well. Ultimately the abstraction well can be submerged during floods. Adequate sealing of the well-head is then required. The LT2ESWTR [USEPA, 2003] gives only a 0.5 to 1 log *Cryptosporidium* removal credit for bank infiltration, depending on the distance between well and surface water. WHO [2004] suggests a 4 log removal after 4 m transport for all pathogens, which appears too high compared to some studies [Schijven et. al, 1998]. For MicroRisk the treatment assessment values for slow sand filtration in Table 4.4 were used for bank filtration.

Other granular media filtration
A whole range of different filter materials are used in drinking water treatment, where the removal of micro-organisms are poorly documented. Precoat types of filtration, including diatomaceous earth and perlite are mentioned in an overview of treatment reduction efficacy [WHO, 2004]; where they were accredited a baseline removal of 50%, 90% and 99.9% for bacteria, viruses and protozoa respectively. For all filter materials, the use of sand filtration reference values in Table 4.2 are suggested when they are supported by actual measured turbidity, particle or indicator removal (see Section 4.5.1).

4.2.4 Conventional treatment

Principle
The term ‘Conventional treatment’ is used here to address the combination of coagulation-flocculation-floc removal including granular media filtration. This combination of processes is very common for surface water treatment and the total reduction by the combined processes has been studied extensively. The results are summarized in the Table 4.5. The principle, operation, indicators and the control points have been discussed in the former ‘coagulation’ and ‘filtration’ Sections 4.2.2 and 4.2.3.

Surrogates
Log removal of turbidity has been recognised as a generally conservative surrogate for *Cryptosporidium* oocyst removal by conventional treatment (in contrast to the individual processes). Particles (in the size fractions of 7-11 µm and 4-7 µm respectively) were found to be a more accurate and still conservative surrogate for *Cryptosporidium* and *Giardia* oo/cysts..

Table 4.5 The MEC (mean elimination capacity) for micro-organisms by conventional treatment (coagulation-flocculation-sedimentation-filtration)

Organisms	Data characteristics	MEC

	Studies	Data	FS-index[a]	MEC	50%ile	Range
Viruses	7	69	3.6	3.0	2.5	1.2 – 5.3
Bacteria	7	54	3.1	2.1	2.1	1.0 – 3.4
Bacterial spores	11	62	4.7	2.4	2.1	1.4 – 4.7
Cryptosporidium	15	162	3.7	3.2	2.9	1.4 – 5.5
Giardia	8	67	4.3	3.4	3.3	2.1 – 5.1

[a] the higher the number (1-5), the more experiments resembled full scale and environmental organisms; [b] indicator bacteria (*E. coli,* coliforms, faecal streptococci)

Direct filtration

Direct-filtration or in-line filtration is when coagulation takes place directly before granular media filtration (no separate floc removal stage). Depending on the design of the system, some sedimentation will take place on top of the filter, while the main reduction occurs within the filter, increasing the filter efficacy and resistance (Table 4.6).

Table 4.6 The MEC (mean elimination capacity) for micro-organisms by direct filtration

Organisms	**Data characteristics**			**MEC**		
	Studies	Data	FS-index[a]	MEC	50%ile	Range
Viruses	7	48	2.9	0.9	0.5	0.1 – 3.9
Bacteria[b]	4	35	3.0	1.4	1.5	0.8 – 3.3
Bacterial spores	5	31	2.6	2.2	2.2	1.5 – 3.9
Cryptosporidium	11	244	2.6	3.0	2.9	0.8 – 5.4
Giardia	9	115	3.1	2.5	2.8	0.8 – 3.9

[a] the higher the number (1-5), the more experiments resembled full scale and environmental organisms; [b] indicator bacteria (*E. coli,* coliforms, faecal streptococci)

4.2.5 Chemical disinfection

Principle

A disinfectant (a strong oxidant) is added to the water and through chemical changes yields non-infectious pathogens. Commonly used oxidants in drinking water production and distribution are (in increasing oxidant strength) chloramines, chlorine, chlorine dioxide and ozone. The disinfectants also form a range of reactive decomposition products, depending on water composition. At higher pH chlorine and ozone are less effective because the decomposition equilibrium shifts from highly reactive to less reactive compounds. Protozoa are most resistant against oxidants, followed by viruses and bacteria. The level of inactivation depends on the concentration (C) of the disinfectant and the contact time (t), expressed as Ct (concentration in $mg.L^{-1}$ x contact time in min.). Special contact tanks or clear water tanks are used to provide sufficient contact time. The concentration of oxidants decreases due to reaction with organic compounds, organisms and auto-decomposition. At low temperatures both oxidant decay and inactivation of micro-organisms slows down. After sub-optimal exposure to oxidants some pathogens/indicators may become non-culturable but still infectious (after time for repair). The oxidants also form Disinfection-By-Products (DBP's) which may have adverse health effects. This limits the level of oxidation that can be applied

and requires a balance between level of disinfection and formation of DBP's [Havelaar *et al*., 2000].
The presence of particulate matter will also affect the disinfection efficacy. Micro-organisms attached to particles are more likely to be resistant to oxidation than when freely suspended [Marin *et al.*, 1996]. The choice of disinfectant depends on several factors including:

- Efficacy against pathogens (bacteria, viruses and protozoa);
- Ability for accurate monitoring and control of the process;
- Ability to maintain a residual in the distribution system;
- Water composition and formation of DBPs; and
- They should not comprise the aesthetic quality of the drinking water.

Operation
The oxidant can be dosed as a liquid (chlorine, chlorine dioxide) or as a gas (chlorine, ozone). Sequential dosing of chlorine and ammonium compounds forms chloramines. The level of dosing is generally proportional to flow, without adjustment to temperature or contact time.

Indicators
Each species and strains of micro-organism can differ in their susceptibility to oxidants, but this is often generalised based on the inactivation of bacterial indicators. Even the state of the organisms can result in different susceptibilities. An indicator with an inactivation rate constant close to that of the pathogen should be selected to reduce uncertainties.

Surrogates
A surrogate needs to reflect the amount of oxidant exposure during a process. Some suggested DBPs as a surrogate [Gunten *et al*., 2001], however, generally a very low level of DBPs is strived for, making it a relatively insensitive measure. Monitoring for DBPs is generally expensive. Further, operators will strive to reduce DBPs while maintaining disinfection, thus breaking the link between DBPs and pathogens. Overall, DBPs are not considered a suitable surrogate for chemical disinfection, and modelling Ct from *in situ* sensors provides the preferred surrogate measure.

Critical limits
Disinfection is an actively controlled process and operational and critical limits can be directly related to the functioning of equipment (dosing pump operation, dosing flow, chemical concentration) or measured process condition (water flow, temperature, measured residual). In the future a critical limit could be set for the inactivation of the target organism(s) by applying 'soft-sensors', which combine signals and translate these into disinfection efficacy by modelling. With the disinfection model proposed below, the measured flow, temperature, and oxidant residual can be combined into a 'disinfection soft-sensor'.

Chemical disinfection calculations:
Inactivation is usually determined in laboratory systems with high numbers of cultured micro-organisms spiked into the system under well-controlled Ct conditions. In practice several factors cause a deviation from the laboratory conditions.

Water will pass a full-scale reservoir with varying contact times since it is not a plug-flow or batch reactor. A small percentage of the total flow will receive little disinfection, thus having a large impact on the average concentration after disinfection, similar to the weakest link example of filtration in Box 1. In disinfection modelling the continuously stirred tank reactor (CSTR) model is applied to account for hydraulics [USEPA, 2003], assuming that a disinfection contactor consists of a number of CSTRs in series. The applicable number for a full-scale system can be determined with tracer tests or CFD modelling. Do Quang *et al.* [2000] showed how the number of CSTRs can be estimated from the contactor geometry. Basically a reservoir without baffles acts as a single CSTR and (ozone) contactors with baffles can be modelled by one CSTR per contact chamber. These assumptions are applied in treatment assessment unless full-scale tracer tests show different hydraulics. Inactivation in a single CSTR is calculated as:

$$\frac{N}{N_0} = \frac{1}{1 + k_e \bullet c \ \bullet t_h}$$

where N_0 and N (micro-organisms L^{-1}) are the concentrations before and after the CSTR respectively, k_e ($L.mg^{-1}.min^{-1}$) is the natural logarithm based inactivation rate constant, c ($mg.L^{-1}$) is the disinfectant concentration at the outlet of the CSTR and t_h (min) is the hydraulic residence time in the CSTR. The total inactivation by a number of CSTRs is calculated as the product of the inactivation in each of the CSTRs. Since full-scale installations are characterized by a few CSTRs, the achievable inactivation is strongly reduced. Do Quang *et al.* [2000b] provide more advanced CSTR models to include different types of baffles and disinfectant decay.
An alternative to CSTR modelling is the Ct10 calculation [USEPA, 2003] where the Ct in reservoirs is corrected by applying a baffling factor to account for short-circuiting. A baffling factor of 0.1 is applied to an unbaffled reservoir (which corresponds to the T10 of a single CSTR). Figure 4.2 shows that the models can provide different inactivation results when they use the same measurements of C and t. At low Ct values the CSTR model predicts higher inactivation that the Ct10 method. At higher Ct values, the Ct10 approach predicts high levels of inactivation, whereas the CSTR approach strongly reduces the effect of high Ct values. Although it is unlikely that full-scale contactors really are completely mixed, the CSTR seems to provide a more realistic estimate of inactivation in full-scale reactors than the Ct10 method. This was partly verified by Smeets *et al.* [2006] when large volume samples of ozonated water at full scale still showed the presence of *E. coli*, limiting the inactivation to an estimated 2.6 logs; while the Ct10 models for this situation predicted over 40 log inactivation versus 5 log by the CSTR model. The CSTR model is recommended for treatment assessment in QMRA to prevent unrealisticly high estimates of inactivation. Based on such modelling,

guidelines for disinfectant concentration in relation to flow and temperature can be set for operational purposes. The example also illustrates that although inactivation models are helpful for treatment assessment, their results still need to be critically judged.

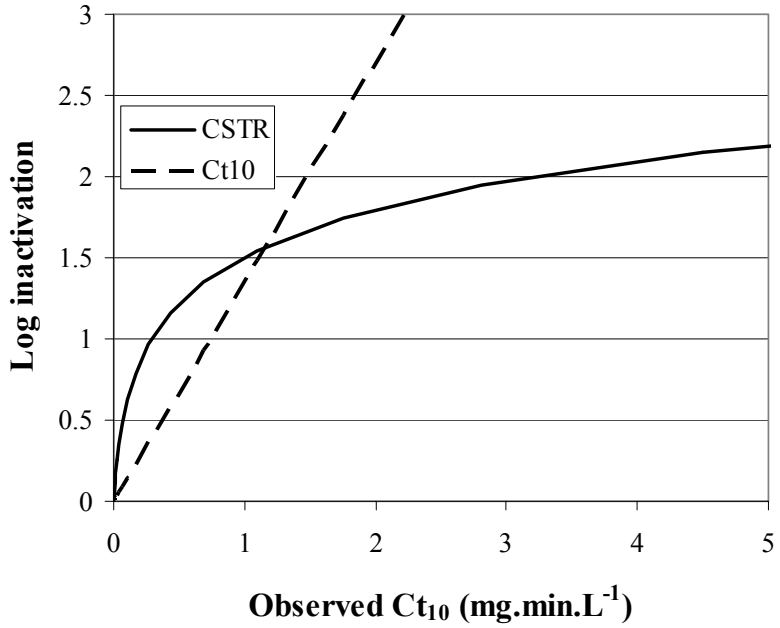


Figure 4.2 *E. coli* inactivation by chlorine in a reservoir (one CSTR, baffling factor 0.1) at 5°C modelled by CSTR and Ct10 methods

The inactivation rate constant k_e is specific for the assessed micro-organism and disinfectant conditions used. Values reported here for viruses and *Giardia* were taken from the SWTR Ct tables [AWWA, 1991], for *Cryptosporidium* from [USEPA, 2003] and for *E. coli* from experiments described in USEPA [2003]. Studies can show considerable differences in disinfection kinetics under similar conditions. Most kinetics have been determined with cultured micro-organisms in artificial, disinfectant demand free water, which does not reflect the reduced inactivation in natural waters [Sobsey, 1989; Thurston Enriquez *et al.,* 2003; Hijnen *et al.*, 2004; Smeets *et al.*, 2005]. Possible explanations are the heterogeneous susceptibility of environmental *E. coli* [Hom, 1972], starvation status of bacteria [Lisle *et al.*, 1998] or protection by encapsulation in aggregates of micro-organisms or particles [Sobsey, 1989; Hijnen *et al.*, 2004]. Haas and Kaymak [2003] suggest that high initial microbiological densities in disinfection experiments result in higher inactivation rate constants. These findings stress the need to determine inactivation rates under full-scale conditions with environmental and not laboratory organisms. Conservative k_e values from literature were used in treatment assessments (Tables 4.7, 4.8, 4.9). Sensitivity analysis showed that uncertainty about full-scale hydraulics has a much stronger effect on the assessed inactivation than uncertainty about k_e.

Inactivation is temperature dependant, so k_e is temperature specific. The Arrhenius equation was used to determine k_e at different temperatures:

$$k_e = A * e^{\left(\frac{-E_a}{RT}\right)}$$

A is the frequency factor in $l.mg^{-1}.min^{-1}$, E_a is the activation energy ($J.mol^{-1}$), R=8.314 ($J.mol^{-1}.K^{-1}$) is the ideal gas constant and T is the absolute temperature (K). In general the inactivation rate doubles with each 10 °C increase in temperature. The effect of pH was only taken into account for chlorine disinfection of *Giardia*. Information was insufficient to account for pH effects with other micro-organisms.

When exposure increases the inactivation rate decreases at a certain point, which is referred to as tailing, due to reduced susceptibility of environmental organisms. Also a 'threshold' exposure is sometimes observed below which no inactivation takes place, referred to as 'shoulder'. Several alternative models have been suggested to account for these effects [Finch *et al.*, 2001]. However these models require a number of additional parameters, which are generally not available since most inactivation experiments have less than 7 log dynamic range, hence the maximum model output was set to 7 log units.

Tables 4.7, 4.8 and 4.9 provide an overview of the disinfection kinetic rate constants used as 'ballpark figures' for different groups of micro-organisms. The values for *Cryptosporidium*, *Giardia* and (entero)viruses were based on USEPA Ct tables [USEPA, 2003]. These only included pH dependency for *Giardia* and chlorine, while virus figures are valid for pH 6-9. *E. coli* inactivation by chlorine and chlorine dioxide was based on literature values from [Narkis *et al.*, 1995; Rice *et al.*, 1999], while *E. coli* and *Campylobacter* inactivation by ozone was a combination of literature values and experimental results (see Section 4.7 and Smeets *et al.* [2006]). Smeets *et al.* [2006] demonstrated that environmental bacteria were less susceptible to ozone than cultured bacteria, hence both inactivation rate constants are presented here. Inactivation of *Campylobacter* and *E. coli* O157 were considered to be similar to the *E. coli* inactivation reported for environmental organisms (ozone).

Table 4.7 Chlorine inactivation rate constants by micro-organisms

Chlorine inactivation rate constants				Calculated log inactivation for observed Ct of 1 $mg.min.L^{-1}$	
Micro-organism	Temperature dependency (Arrhenius)		k_e	plug flow	single
	E $J.mol^{-1}$ $*10^3$	A $l.mg^{-1}.min^{-1}$	$L.mg^{-1}$ $.min^{-1}$ 10°C		CSTR
Cryptosporidium	-	-	-	-	-
Giardia pH 7	47	7.5	0.062	0.027	0.026
Virus	50	9.8	4.58	2.0	0.75
Ct<0.5 *E. coli*	46	9.8	19.6	[a]	[a]
Ct>0.5 *E. coli*	49	9.8	6.67	2.9	0.88

[a] Not valid under these conditions

Table 4.8 Chlorine dioxide inactivation rate constants by micro-organisms

	Chlorine dioxide inactivation rate constants			Calculated log inactivation for observed Ct of 1 mg.min.L^{-1}	
Micro-organism	Temperature dependency (Arrhenius) E $J.mol^{-1}$ $*10^3$	A $l.mg^{-1}.min^{-1}$	k_e $L.mg^{-1}.min^{-1}$ 10°C	plug flow	single CSTR
Cryptosporidium	59	8.8	0.0054	0.0023	0.0023
Giardia <5°	138	25.3	[a]	[a]	[a]
Giardia >5°	30	5.0	0.24	0.10	0.093
Virus	48	8.8	0.47	0.20	0.17
E. coli	23	5.7	16.4	7.1	1.24

[a] Not valid under these conditions

Table 4.9 Ozone inactivation rate constants by micro-organisms

	Ozone inactivation rate constants			Calculated log inactivation for observed Ct of 1 mg.min.L^{-1}	
Micro-organism	Temperature dependency (Arrhenius) E $J.mol^{-1}$ $*10^3$	A $l.mg^{-1}.min^{-1}$	k_e $L.mg^{-1}.min^{-1}$ 10°C	plug flow	single CSTR
Cryptosporidium	63	11.0	0.24	0.10	0.093
Giardia	49	9.8	4.9	2.14	0.77
Virus	47	9.7	10.0	4.35	1.04
E. coli lab	48	11.6	[a] 576	[a] 250	[a] 2.76
E. coli environ	48	11.1	174	75.6	2.24

[a] Included to illustrate the difference between cultured and environmental *E. coli*, use of inactivation rate constants for environmental *E. coli* is preferred when assessing full scale treatment.

4.2.5 UV disinfection

Principle

Ultra violet (UV) disinfection relies on UV transmission through water. Lamps are placed in a flow-through contact compartment that is continuously operated. The inactivation of pathogens is related to the UV fluence, which is the product of UV light intensity and contact time. Contact time depends on rector volume and flow and the number of lamps, and the lamp power determines the intensity. The intensity decreases with the distance from the lamp surface due to radial expansion and the UV absorption of water and dissolved organic substances therein. Therefore the flow pattern inside the UV reactor has a high impact on reactor efficacy and considerable effort has been put in optimising reactor configurations for disinfection. In order to test this, dosimeter experiments are carried out which result in a Reduction Equivalent Fluence (REF). High concentrations of micro-organisms are treated, and simultaneously the UV intensity is measured inside the reactor. The measured inactivation is then related to the measured intensity providing guidelines for operation [Sommer *et al.*, 2000].

Low-pressure or medium pressure mercury lamps are generally used. Low-pressure lamps emit a major peak at a narrow spectrum around 256 nm (monochromatic light), at which frequency thymine dimers are formed within microbial DNA or RNA, hampering reproduction. Medium pressure lamps emit a much broader spectrum of UV light (polychromatic UV), which also affects proteins and membrane transport. Polychromatic light can be twice as effective as monochromatic light at the same fluence. In some cases repair mechanisms of bacteria either in light or dark conditions can make an inactivated organism infectious again and several viruses have been shown to use repair enzymes in the host cell [von Sonntag *et al.*, 2004]. Although protozoa repair was demonstrated they appear not to regain infectivity. Turbidity will have a negative effect on UV disinfection by shielding pathogens from UV light. Temperature, pH, electric conductivity and alkalinity seem to have no effect on inactivation. Hijnen *et al.* [2006] has recently published a detailed literature review of UV disinfection including these observations.

Operation
Operation consists of maintaining flow and lamp intensity within given specifications. Operating a number of reactors in parallel and bringing units on- or off-line controls flow and in some units lamp power can be varied. During stable operation, UV intensity decreases due to fouling of the lamp sleeve and ageing of the lamp. When UV intensity drops below a threshold value, the sleeve is cleaned, the lamp power increased or the lamps are replaced. UV disinfection requires regular maintenance and testing of equipment.

Indicators
Indicators can be used to determine fluence, and predict pathogen inactivation. Table 4.10 shows the susceptibility of pathogens and potential indicator organisms. MS2 is a conservative modelr for most viruses although a single study on *Norovirus* inactivation suggests that it may be relatively UV resistant. *Clostridium perfringens* and the aerobic spore-former *Bacillus subtilis* are conservative indicators for bacteria. Some studies show that environmental populations of bacteria are less susceptible to UV than cultured organisms. In contrast, cultured Adenovirus were more UV resistant [Hijnen *et al.* 2006].

Surrogates
A surrogate needs to represent the fluence that pathogens are exposed to with sufficient sensitivity to identify short-circuiting. Currently several substances are tested for suitability, but so far no definite applicable surrogate has been reported [Kiwa, 2006].

Critical limits
As described under operations, critical limits are set to the desired level of inactivation of target pathogens. Through the REF, this is translated to the measured UV intensity. Selecting the appropriate number of reactors, the number of UV lamps and their intensity/power sets the required dose.

Table 4.10 UV inactivation dose (fluence) for a required log inactivation by micro-organism*

	Required fluence ($mJ.cm^{-2}$)			
MIC required (log):	1	2	3	4
Adenovirus type 2,15,40,41	42	83	125	167
Adenovirus type 40	56	111	167	-[d]
Adenovirus[a] (not type 40)	25	50	-[d]	-[d]
Calicivirus canine	10	21	31	41
Calicivirus bovine	5	11	16	21
Calicivirus feline	9	19	28	38
Coxsackie virus B5	8	17	25	34
Hepatitis A	6	11	17	22
Poliovirus type 1	7	15	22	30
Rotavirus SA-11	10	20	29	39
Bacillus subtilis[a]	56	111	167	222
Campylobacter jejuni[b]	3	7	10	14
Clostridium perfringens[a]	45	95	145	-[d]
E. coli O157[b]	5	9	14	19
E. coli[a]	5	9	14	18
Legionella pneumophila[b]	8	15	23	30
Salmonella typhi[a]	6	12	17	51
Enterococcus faecalis[a]	9	16	23	30
Shigella dysenteriae[b]	3	5	8	11
Shigella sonnei[b]	6	13	19	26
Vibrio cholerae[b]	2	4	7	9
Yersinia enterocolitica[b]	3	7	10	13
Cryptosporidium USEPA[c]	3	6	12	-[e]
Giardia USEPA[c]	2	5	11	-[e]
Acanthamoeba[c]	40	71	119	167

*UV fluence ($mJ.cm^{-2}$) requirements for a MIC (Mean Inactivation Capacity) of 1 up to 4 log by monochromatic UV radiation for viruses, bacteria, bacterial spores and protozoan (oo)-cysts based on the k-values with or without correction for environmental species; for bacteria in wastewater a higher correction for environmental species is needed and further research has to clarify the need for a higher fluence to account for photoreactivation; for *Giardia* increased fluence requirement because of dark repair is a factor for further research. [a] environmental spp. ; [b] corrected for environmental spp. ; [c] no correction for environmental spp. (research needed) ; [d] MICmax < 4 log ; [e] no value due to tailing [Hijnen *et al.*, 2006].

4.2.6 Membrane filtration

Membrane filtration has become a cost-effective alternative to conventional separation processes. Common applications include low-pressure membranes, microfiltration and

ultrafiltration as particle barriers, and high-pressure membranes, nanofiltration and reverse osmosis (RO) to remove colloidal and dissolved compounds.

Principle

Membranes are used to remove particulate matter by size exclusion (microfiltration [MF] and ultrafiltration [UF]) or dissolved substances by a combination of size exclusion and more complex process (Nanofiltration [NF] and reverse osmosis [RO]). Although these terms are used to indicate the size range that is removed by these membranes, there actually is a gradual change in pore size and molecular weight cut-off due to the large choice of available membranes. Microfiltration removes protozoan oo/cysts and most bacterial pathogens. Ultrafiltration removes bacteria and most viruses, depending on the chosen membrane type. Nanofiltration and reverse osmosis theoretically remove all organisms; since their pores are small enough to also account for dissolved molecules and salt. Jacangelo [2005] showed that the molecular weight cut-off for membranes is not always a good predictor of virus removal, since a few large pores have a limited effect on removal of dissolved molecules (typically less than 1 log) but a large effect on removal of pathogens (over 6 log units). Removal of pathogens then not only relies on pore size, but also on surface properties of the organisms in relation to the membrane material, similar to granular filtration, as well as loss of membrane integrity with age. Three membrane forms are most common; spiral wound (RO and NF), tubular (NF, UF, MF), ceramic (all, but expensive, so hardly applied in drinking water).

Membranes can be operated as ‘dead end’, where all the water goes through or with cross flow operation, which drains water from the feed-side of the membrane to keep a sufficiently high flow at the membrane surface to reduce fouling. Sometimes multiple membrane ‘inserts’ are placed in a pressure vessel which are combined in a ‘stack’. This is the operational unit that is generally backwashed or cleaned. Several stacks are operated in parallel so one or two can be cleaned while the others maintain production.

Operation

The water production increases with increasing pressure across the membrane. At high pressure less membranes are needed, but energy costs and fowling of membranes increases. Therefore the pressure (and flow or flux) across the membrane is kept between strict operational limits. Membranes are operated in cycles, micro- and ultra filters are backwashed frequently to remove solids accumulated at the feed side of the membrane. Backwashing is not possible with nanofiltration and reverse osmosis. All membranes require regular chemical cleaning during which they are taken off-line. Monitoring particle removal when sufficient particles are present in the feed water tests membrane integrity. Additionally bubble or acoustic testing can be used *in situ.* NF and RO are typically applied to waters with low particle content to prevent fouling, and particle removal can only provide limited verification of membrane integrity. Therefore other substances, like sulphate are monitored to verify integrity of RO membranes. Membrane integrity can be compromised by rupture due to physical stress, damaging by sharp particles, membrane decomposition or leakage of seals and fittings. In practice loss of membrane integrity occurs frequently and this is accepted up to a point out of practical considerations. When integrity monitoring reaches the intervention level

integrity is restored by repair or replacement of membranes. The varying removal of pathogens in practice is illustrated in Figure 4.3. Table 4.11 provides some information on the potential removal by an intact membrane and the practical removal due to monitoring and leak repair limitations.

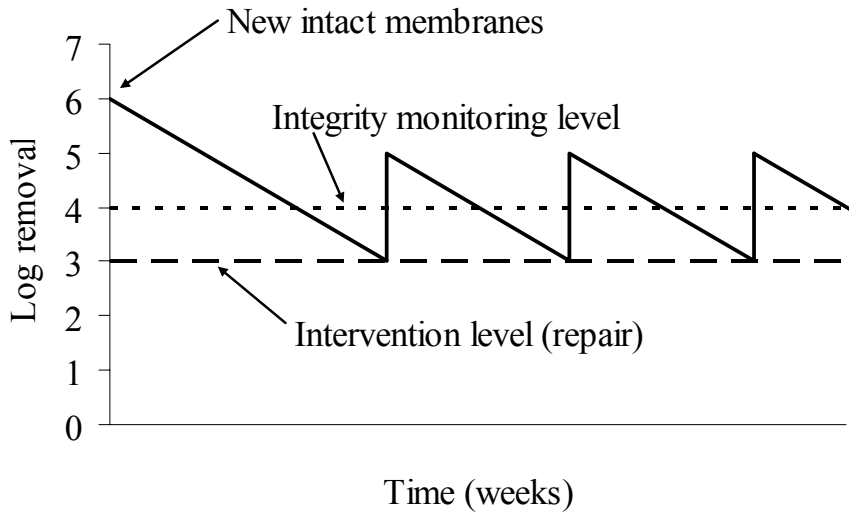


Figure 4.3 Theoretical variation of pathogen removal by membranes at full-scale due to integrity failure and repair in relation to monitoring

Indicators
The size of the indicator needs to represent pathogen size. Therefore phages and indicator bacteria are used for viruses and bacteria respectively.

Surrogates
Particle counting is generally used for MF and UF integrity monitoring since it is more sensitive than turbidity monitoring. It also provides information on the cut-off of different sizes of the particles, which can then be related to groups of pathogens. However particle counting does not account for surface characteristics. Kruithof *et al.* [2001] used powdered activated carbon as challenge particles to verify membrane integrity for up to 5.8 log units of virus removal. Conductivity or sulphate reduction is used for integrity testing of RO membranes up to 2 and 3 logs respectively, representing the measured removal of MS2 phages in challenge tests. The assumed (linear) relationship between sulphate removal and pathogen removal was not investigated [Kruithof *et al.*, 2001].

Critical limits
The integrity monitoring (by particle counting or sulphate measurements) is the most important in verifying sufficient removal. By monitoring individual membranes, a smaller leak can be detected than at the level of total flow. However, this is seldom practical to perform.

Table 4.11 MEC values for pathogen removal by intact membranes and monitored removal in practice due to leakage and monitoring limits. (ranges found in [Kruithf et al., 2001; Jacangelo, 2005])

	pore size µm	Log removal Viruses	Log removal Bacteria	Log removal Protozoa	Operational level (log)[c]
Microfiltration	0.1-1	0 – 3.7	0-4.3	2.3 - >7	
Ultrafiltration	0.01-0.1	>6.5	>7	>7	4
Nanofiltration[a]	0.001-0.01			2.2	
Nanofiltration[b]	0.001-0.01			5.5	
Reverse Osmosis	0.0001-0.001	2.7-6.5			2-3

[a] Cellulose acetate membrane, [b] composite thin film membrane

[c] Operational removal level is limited by source water particle or sulphate concentration and the intensity of product water monitoring (total flow or per stack) based on practical examples.

4.3 TREATMENT ASSESSMENT FRAMEWORK

This paragraph introduces the method that was used to assess the reduction of pathogens by a treatment system. The treatment schemes of the twelve Catchment-to-Tap Systems (CTS) that were assessed in the MicroRisk project (Chapter 3) are described and the available data at these sites is discussed. A framework is presented to combine this data for input into a QMRA model.

4.3.1 Treatment assessment approach

The goal of the treatment assessment is to describe pathogen reduction performance for input into a QMRA model. By combining this information with the pathogen concentration in the source water, the number of pathogens in the treated water can be estimated. Chapters 7 & 8 describes how this is combined with consumption and distribution data to calculate the health risk in a QMRA. Treatment performance for the twelve CTS are presented in Section 4.3.2 as case studies. Examples from these case studies are used in Sections 4.4, 4.5 and 4.6 to illustrate the methodology.

Different pathogens pose varying challenges to water treatment. Bacteria are less well removed by filtration than other micro-organisms but are readily inactivated by disinfection. Protozoa are relatively insensitive to chlorine disinfection but better removed by filtration than bacteria and readily inactivated by UV, whereas viruses are somewhere in between for both types of processes. By assessing the reduction of the selected suite of pathogens and indicators, the challenges posed by other (unknown) pathogens is probably covered. The treatment assessment does not differentiate between different species of micro-organisms unless specifically discussed in Section 4.2.

In order to assess a treatment, specific data that provides information on the performance of that system is needed. Frequent monitoring of pathogens at different stages of treatment would provide the ideal dataset to determine the barrier efficiency, but pathogen concentrations are often too low for ready diction and especially monitoring after the first treatment steps results in mostly non-detects. Various indicators, surrogates or process conditions that can be used to estimate pathogen treatment efficacy were described in Section 4.2. Many water utilities already collect some of this data either for compliance to drinking water laws (indicator sampling) or for operational purposes (residual chlorine measurement to control chlorine dosing). The available data was therefore compiled from the Microrisk CTSs to provide treatment performance data.

Reduction at a single site varies in time and this variation was accounted for by collecting data over a period of several years. In order to do risk calculations these variations were expressed as Probability Density Function (PDF) (see Chapter 7). The quality of the data varied between the CTSs, and between treatment processes within a single CTS. This chapter describes how reduction by each treatment barrier can be estimated based on commonly available data, as illustrated in Figure 4.4.

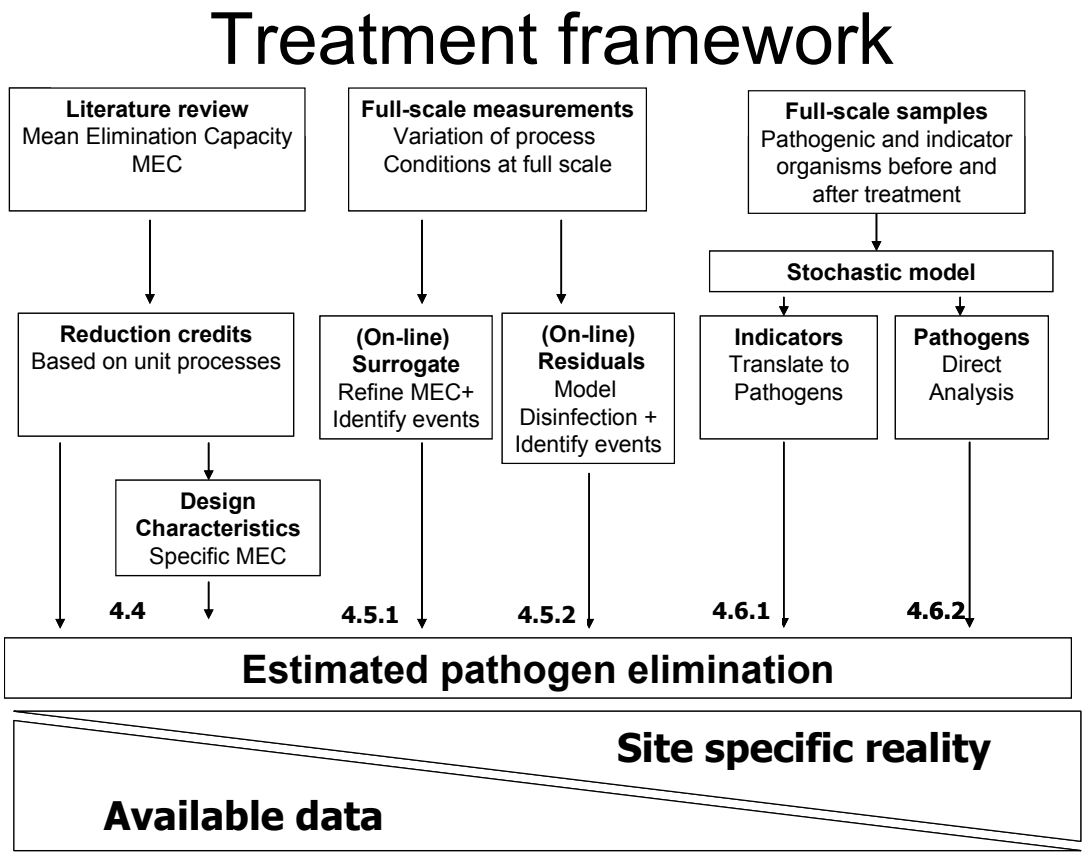


Figure 4.4 MicroRisk treatment performance data framework. The numbers correspond to the paragraphs that discuss the use of this type of data

Other types of data that can be used for treatment performance assessment include results from (site specific) pilot test and failure reports. Such tests are more applicable to the local situation than general literature values. Other data like operational diaries or failure reports can provide information about the frequency and duration of events in treatment.

4.3.2 Treatments assessed in the MicroRisk project

Within the MicroRisk project the twelve systems (as in Chapters 3, 5, 7 and 8) were used. These treatment schemes are briefly outlined in Table 4.12.

Table 4.12 Treatment schemes of the catchment-to-tap systems (CTSs) in the MicroRisk project

CTS	Treatment scheme [1]
1	PreO_3 (Cl_2 in summer) - Coa - Sed - RF - O_3 - GAC - super Cl_2-deCl_2
2	Coa - Sed - RF - infiltration - reservoir - RF - O_3 - GAC - SSF
3	PreO_3 - Coa - Sed - RF - O_3 - GAC - Cl_2 (ClO_2 until July 2003)
4	Coa - Sed - O_3 -GAC - ClO_2
5	Coa - Sed - O_3 or Cl_2 - GAC - Cl_2
6	Reservoir - Cl_2 (summer) - Coa - Sed - GAC - Cl_2
7	Bank filtration - O_3 - GAC
8	Pre-Cl_2-Coa - DAF - RF - GAC - Cl_2
9	RSF - O_3 - GAC - SSF
10	PreCl_2 - Coa - Sed - RF - O_3 - Cl_2
11	DF - RF - Cl_2 + ClO_2
12	RF - Cl_2

[1]Coa = coagulation, Sed = sedimentation, DAF = dissolved air flotation, RF = rapid (granular) filtration, DF = direct filtration, O_3 = ozonation, GAC = granulated activated carbon filtration, Cl_2 = chlorination, ClO_2 = chlorine dioxide dosing, SSF = slow sand filtration.

Collected data included pathogen and indicator monitoring results, measured surrogates like turbidity and particle counting, process conditions like temperature, pH, DOC concentrations, disinfectant residual measurement and operational data like flows, disinfectant dose failure reports and supporting pilot information as exemplified here.

Pathogen monitoring

Most systems only monitored pathogens in the source water (Chapter 3). CTS 1 monitored Enterovirus, *Campylobacter*, *E. coli* O157, *Giardia* and *Cryptosporidium* monthly after each treatment step for a year. The majority of these samples resulted in non-detects (see Section 4.9). CTS 2 and 9 provided approximately 25 results of *Campylobacter* monitoring both after filtration and ozonation with a certain percentage of positives due to the large analysed volume. CTS 10 monitored *Giardia* both in raw and treated water resulting in approximately 50% positive samples (see analysis in Chapter 7).

Indicator monitoring

All systems provided their results of indicator motoring. Raw water was typically monitored daily to weekly and treated water was monitored daily for the presence of *E. coli* and/or different types of coliforms. Some sites also monitored *Enterococci*/ *faecal streptococci* or other faecal indicators. Spores of sulphite-reducing *clostridia* (SSRC) were monitored daily to monthly in treated water at several sites (1, 2, 3, 4, 9,

10, 12) but only four had corresponding source water data. Some large treatment plants also monitored *E. coli* at several points during the process (CTS 1, 2, 5, 6, 9) and sometimes also SSRC (CTS 1, 2, 9).

Surrogates
Turbidity of the treated water was monitored at most sites although the frequency varies between once per week to every twenty seconds. Most CTSs also monitored turbidity after filtration, generally on the combined flow of all filters. Some sites reported that turbidity was monitored on-line but data were unavailable and only used for alarm purposes. Particle counting was only applied over a longer period at CTS 5 and 6.

Process conditions
General process conditions like temperature and pH were provided for all CTSs in sufficient numbers to describe variation over the year. Specific process conditions like disinfectant residuals showed some shortcomings. Some CTSs (2, 9) only measure ozone residual once per week. Since this process is very variable, such a sampling strategy will miss short periods of poor removal. The on-line ozone measurements at these sites were deemed unreliable, and were placed at a point where generally no ozone residual was present. Others were unsure of the point of residual measurement in a contactor or the contactor volume.

Operational data
Most operational data provided was from on-line measurements of water treatment equipment, like dosing pump flows. This helped to estimate frequency of equipment failure, although failure could not always be separated from intentional shutdown of equipment for maintenance. This required diary reports or failure reports, which most CTSs couldn't provide in computer format. Only CTS 5 provided detailed diary and failure reports which are analysed in Chapter 8.
Collection of flow data was essential for data analysis and process modelling, since it provided information whether a line was actually in operation during a measurement. CTS 3 provided on-line disinfectant residual data which showed regular periods of low residual. Presumably this occurred during periodic shut-down of the plant (daily). However, no data was available to indicate when the plant was actually producing water.

Conclusions data collection
The data collection made clear that most data was not collected with the aim to assess treatment efficacy. As a result, no 'ideal' CTS was identified, which had high quality data for all treatment steps. In general the large surface water treatment plants monitor more rigorously than the smaller plants. Still, a lot of potentially useful data could not be used because some information, which could easily be recorded or monitored, was missing. The use and shortcomings of the data is illustrated in the examples in this chapter. Based on the first iteration of the treatment assessment of the CTSs, general guidelines on additional data collection are given in Section 4.10 to address the shortcomings.

4.4 POINT ESTIMATES

4.4.1 Point estimates

The first step in assessing treatment efficacy was the collection of relevant literature and local data. Basic information about the local treatment design is required; the unit processes, how these are connected and their design specifications. Relevant information like design Ct values for chemical disinfection processes or UV dose are generally available for a given treatment plant. For physical removal processes like coagulation-sedimentation and filtration basic information like the application of coagulants, filtration rates and filter material and size provide additional information to better estimate the efficacy.

The following example illustrates a basic assessment from unit processes for CTS 11. Treatment consisted of direct filtration (in-line coagulant dosing before the rapid sand filter), a second rapid sand filtration step and disinfection with chlorine and chlorine-dioxide. Filter specifications like bed height, filtration rate, filter material and size at CTS 11 corresponded to the studied filters described in Section 4.2, so the removal efficacies presented in Tables 4.2 and 4.6 were used here. At CTS 11 the Ct for chlorine and chlorine dioxide were 7.5 and 4.5 $mg.min.l^{-1}$ respectively based on design flow, reservoir size and average disinfectant residual reported by the water company. Inactivation by disinfection was calculated with the single CSTR model (Section 4.2.5). Table 4.13 illustrates the estimated pathogen reductions for the unit processes based on the MEC values reported in Section 4.2.

Table 4.13 Log inactivation at CTS 11 based on unit processes

	Direct filtration	**Rapid filtration**	**Disinfection Chlorine**	**Disinfection Chlorine Dioxide**	**Total treatment**
Virus	0.9	0.9	1.5	0.6	3.8
Bacteria	1.4	0.6	1.7	1.9	5.6
Cryptosporidium	3.0	2.0	0.0	0.0	5
Giardia	2.5	1.7	0.1	0.4	4.7

From Table 4.13 it is clear that direct filtration performs better than rapid filtration only. Filtration is an important barrier for the protozoa and disinfection for the bacteria and viruses. The inactivation reported in Table 4.13 is a mean estimate of the potential inactivation at full-scale. Since conditions can vary substantially, a more sophisticated estimate of inactivation is needed and is discussed in the next paragraph.

4.4.2 Uncertainty of point estimates

The basic assessment described in Table 4.13 for pathogen removals at CTS 11 lack any interpretation of the expected variations described in Section 4.2. Table 4.14 shows the best estimate again, but now with the minimum and maximum removal reported for the processes at CTS 11. A worst case assumption is that minimum disinfection could be 0 log due to periodic under-dosing of disinfectant. Maximum possible inactivation is

calculated assuming ideal plug flow (Ct calculation), highest estimate of disinfectant concentration and temperature. Inactivation is unlikely to exceed this maximum. Thus the range of all possible inactivation outcomes in Table 4.14 was established for CTS 11.

Table 4.14 Log reduction at CTS 11 based on unit processes, including maximum likely uncertainty

	Viruses min-MEC-max	**Bacteria min-MEC-max**	***Cryptosporidium* min-MEC-max**	***Giardia* min-MEC-max**
Direct Filtration	0.1-0.9-3.9	0.8-1.4-3.3	0.8-3-5.4	0.8-2.5-3.9
Rapid Filtration	0.1-0.8-3.8	0.1-0.6-1.5	0.0-2-3.1	0.0-1.7-6.5
Disinfection Chlorine	0-1.5- >7	0-1.7- >7	0-0-0	0-0.1-0.3
Disinfection Chlorine-Dioxide	0-0.6-3.5	0-1.9- >7	0-0-0	0-0.4-1
Minimal total reduction	0.2	0.9	0.8	0.8
Best estimate reduction	**3.8**	**5.6**	**5.0**	**4.7**
Maximum total reduction	>18.2	>18.8	8.6	11.7

Table 4.14 and the case study in Table 4.23 illustrate that without site-specific verification of the performance, the range in possible treatment performance is large, and needs to be narrowed down to better describe what may actually be occuring.

4.4.3 Modelling variation in point estimates

The minimal estimate of reduction by the total treatment in Table 4.14 was a 'worst case' assumption in which every treatment step performs at its worst. Most systems are expected to have some treatment steps perform better. The way variation in the data described in Table 4.14 has been modelled in the MicroRisk project is to describe each treatment step by a triangular PDF with the parameters minimum, MEC and maximum to represent 'expert knowledge' as discussed in Chapter 7. The best estimate is now not necessarily the sum of the best estimates of each unit processes; rather, each triangular distribution provides insight into which step and pathogen has the most uncertainty/variability associated with it (see Figure 4.5 for direct filtration).

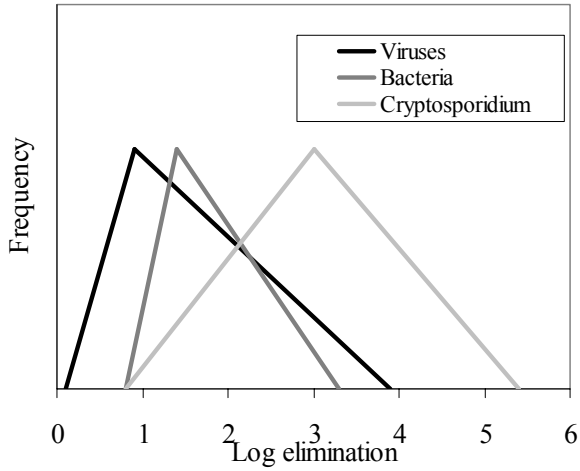


Figure 4.5 Triangular PDF (Probability Density Function) of virus, bacteria and *Cryptosporidium* removal by direct filtration

The triangular distribution is used as a first pass representation of process variability and uncertainty. This is especially useful when assessing several treatment steps in series to provide an estimate of the combined reduction. When one process is performing poorly, the other processes are still providing reduction. By combining the triangular distributions of the individual processes in a Monte Carlo analysis, the total reduction with its variability can be simulated. The result is a description of how the total treatment efficacy is likely to vary. This is explained in more detail in Chapter 7. The overall reduction represents the pathogen removal over the assessed period (see Box II).

BOX II Over-all treatment performance

Pathogen reduction by treatment varies over time. The total effect of treatment over a given period is expressed by the 'over-all' log reduction. The average log removal does not represent the actual fraction of pathogens that pass treatment. The average fraction over the period is therefore used to calculate over-all reduction (over-all reduction = -log (average fraction)). This is illustrated by the following example for a period of three days. The average log reduction was 2.3 but the over-all reduction was 1.5 log, so the number of pathogens (load) after treatment is 1.5 log lower than in source water.

	day 1	***day 2***	***day 3***	***average***	***over-all reduction***
log reduction	*3*	*1*	*3*	*2.3*	
fraction	*0.001*	*0.1*	*0.001*	*0.034*	***1.5***

4.4.4 Results for MicroRisk Systems

Initial treatment performance assessments for all systems (CTS) in MicroRisk are summarised in Tables 4.15 to 4.18. The first observation for all systems and organisms is that the minimum removal is generally low, whereas the potential (maximum) removal can be extremely high. This shows that although point estimates are often used to estimate removal, their uncertainty generally ranges between zero and >10 log removal. This large uncertainty is partly caused by lack of information on system operation, especially disinfection processes can have a large impact.

The point estimate of the reduction is generally slightly higher than the estimated over-all reduction based on triangular distributions. The 2.5% percentile of the estimated variation is generally substantially higher than the minimum estimate. The extremely low minimum estimates are unlikely to occur due to the multiple barriers in the treatment systems.

Table 4.15 Model process schemes used for initial assessment of over-all virus reduction by treatment of the catchment-to-tap systems (CTS) in the MicroRisk project

CTS	Viruses Treatment model	Point estimates Best estimate	Min	Max	Triangular distributions Best estimate	2.5%	97.5%
1	O_3 - Coa/Sed - RF - O_3 - GAC - Cl_2 - Cl_2	12.2	1.4	> 34	8.1	10.1	22.0
2	RF - O_3 - GAC – SSF	4.7	0.9	> 15.5	4.7	3.8	11.0
3	O_3 - Conv - O_3 - GAC - Cl_2	6.5	1.4	> 22.1	6.4	5.8	14.8
4	Coa/Sed - O_3 - GAC – ClO_2	4.3	0.4	> 18.8	4.7	3.9	12.9
5	Coa/Sed - O_3 - GAC - Cl_2	2.2	0.4	5	1.9	1.0	4.3
6	Coa/Sed - GAC - Cl_2	2.2	0.4	5	1.9	1.1	4.3
7	SSF - O_3 – GAC	3.8	0.8	> 11.7	3.6	2.7	8.9
8	Cl_2 - Conv - GAC - Cl_2	6.9	1.4	> 13	6.5	6.1	14.6
9	RSF - O_3 - GAC – SSF	4.5	0.9	> 15.5	4.6	3.7	10.8
10	Cl_2 – Conv - O_3 - Cl_2	6.9	1.2	> 26.3	6.0	5.6	14.5
11	DF - RF - Cl_2 - ClO_2	3.8	0.2	> 18.2	5.2	4.6	14.0
12	RF - Cl_2	2.2	0.1	> 10.8	2.5	1.5	8.0

Table 4.16 Model process schemes used for initial assessment of over-all reduction of bacteria by treatment of the catchment-to-tap systems (CTS) in the MicroRisk project

CTS	Bacteria Treatment model	Point estimates Best estimate	Min	Max	Triangular distributions Best estimate	2.5%	97.5%
1	O_3 - Coa/Sed - RF - O_3 - GAC - Cl_2 - Cl_2	15.1	1.9	> 34.3	10.2	11.7	23.2
2	RF - O_3 - GAC - SSF	7.3	2.2	> 16.2	6.3	5.5	12.0
3	O_3 - Conv - O_3 - GAC - Cl_2	9.0	1.9	> 27.3	8.0	8.1	18.4
4	Coa/Sed - O_3 - GAC – ClO_2	7.7	1.5	> 20.6	6.5	6.0	14.5
5	Coa/Sed - O_3 - GAC - Cl_2	2.9	1.5	6.6	3.1	2.3	5.4
6	Coa/Sed - GAC - Cl_2	2.9	1.5	6.6	3.2	2.3	5.5
7	SSF - O_3 – GAC	6.5	2.1	> 14.7	5.6	4.8	11.2
8	Cl_2 - Conv - GAC - Cl_2	7.1	1.9	> 13.3	7.2	6.7	15.1
9	RSF - O_3 - GAC – SSF	7.0	2.2	> 16.2	6.3	5.4	11.9
10	Cl_2 – Conv - O_3 - Cl_2	7.1	1.0	> 24.4	6.2	6.3	16.6
11	DF - RF - Cl_2 - ClO_2	5.6	0.9	> 18.8	5.6	4.9	13.2
12	RF - Cl_2	2.1	0.1	> 8.5	2.1	1.2	6.8

Table 4.17 Model process schemes used for the initial assessment of over-all *Cryptosporidium* reduction by the treatment of catchment-to tap-systems (CTS) in the MicroRisk project

CTS	*Cryptosporidium* Treatment model	Point estimates Best estimate	Min	Max	Triangular distributions Best estimate	2.5%	97.5%
1	O_3 - Coa/Sed - RF - O_3 - GAC - Cl_2 - Cl_2	4.8	2.1	10.8	4.8	3.9	8.1
2	RF - O_3 - GAC - SSF	6.9	1.0	> 11.4	4.3	3.6	9.0
3	O_3 - Conv - O_3 - GAC - Cl_2	4.2	2.1	7.7	4.0	3.1	6.4
4	Coa/Sed - O_3 - GAC – ClO_2	3.0	1.1	> 6.4	2.6	1.8	4.5
5	Coa/Sed - O_3 - GAC - Cl_2	2.8	1.1	4.9	2.4	1.6	4.2
6	Coa/Sed - GAC - Cl_2	2.8	1.1	4.9	2.4	1.6	4.2
7	SSF - O_3 – GAC	4.8	1.0	> 7.9	3.1	2.1	6.9
8	Cl_2 - Conv - GAC - Cl_2	4.1	2.1	6.6	3.6	2.7	5.9
9	RSF - O_3 - GAC – SSF	6.8	1.0	> 11.3	4.4	3.5	9.0
10	Cl_2 – Conv - O_3 - Cl_2	3.9	1.4	8.5	3.7	2.9	6.5
11	DF - RF - Cl_2 - ClO_2	5.0	0.8	8.6	4.0	3.1	7.7
12	RF - Cl_2	2.0	0.0	3.1	1.2	0.4	2.8

Table 4.18 Model process schemes used for the initial assessment of over-all *Giardia* reduction by the catchment-to-tap systems (CTS) in the MicroRisk project

CTS	*Giardia* Treatment model	Point estimates			Triangular distributions		
		Best estimate	Min	Max	Best estimate	2.5%	97.5%
1	O_3 - Coa/Sed - RF - O_3 - GAC - Cl_2 - Cl_2	9.6	2.5	> 31.2	9.1	9.4	20.1
2	RF - O_3 - GAC - SSF	7.8	1.6	> 22.8	7.0	6.5	15.6
3	O_3 - Conv - O_3 - GAC - Cl_2	6.4	2.5	> 17.5	7.4	6.6	15.9
4	Coa/Sed - O_3 - GAC – ClO_2	4.9	0.7	> 15.2	5.1	4.4	13.6
5	Coa/Sed - O_3 - GAC - Cl_2	3.3	0.7	6.2	2.6	1.7	5.1
6	Coa/Sed - GAC - Cl_2	3.3	0.7	6.2	2.6	1.7	5.1
7	SSF - O_3 – GAC	5.9	1.6	15.3	5.3	4.5	11.2
8	Cl_2 - Conv - GAC - Cl_2	6.6	2.5	> 15.4	7.1	6.5	15.6
9	RSF - O_3 - GAC – SSF	7.5	1.6	> 22.8	7.0	6.4	15.5
10	Cl_2 – Conv - O_3 - Cl_2	5.5	2.1	> 19.5	5.9	5.1	13.9
11	DF - RF - Cl_2 - ClO_2	4.7	0.8	> 11.7	5.1	4.3	12.5
12	RF - Cl_2	1.8	0.0	> 8.5	2.0	1.0	6.4

4.5 USE OF SURROGATES AND PROCESS MODELLING

4.5.1 Validating physical processes with measured surrogates

The large ranges in estimated log removals described in the previous section indicated the need to reduce the uncertainty at specific sites. Many sites apply (on-line) turbidity measurement to monitor filter performance. As stated in Section 4.2, a universal relation between turbidity and pathogen reduction does not exist, yet a good working filter is able to produce water with a turbidity constantly below 0.1 NTU.

Site 10 provides an example where turbidity was measured before and after conventional treatment (coagulation-sedimentation and filtration). The filtrate turbidity varies between 0.1 and 1 NTU, indicating that filtration does not work effectively. The triangular PDF for a poor performing filter applied the MEC as maximum and the mean of the minimum and the MEC as most likely removal (see Figure 4.6).

Turbidity at site 11 is recorded daily before and after direct filtration. Turbidity is consistently reduced from >1 NTU to <0.06 NTU thus verifying that the filter is working well. The triangular PDF for a well performing filter applied the MEC as minimum and the mean of the MEC and the maximum as most likely removal. Figure 4.6 illustrates how the triangular PDF is adapted for these CTSs based on the recorded turbidity.

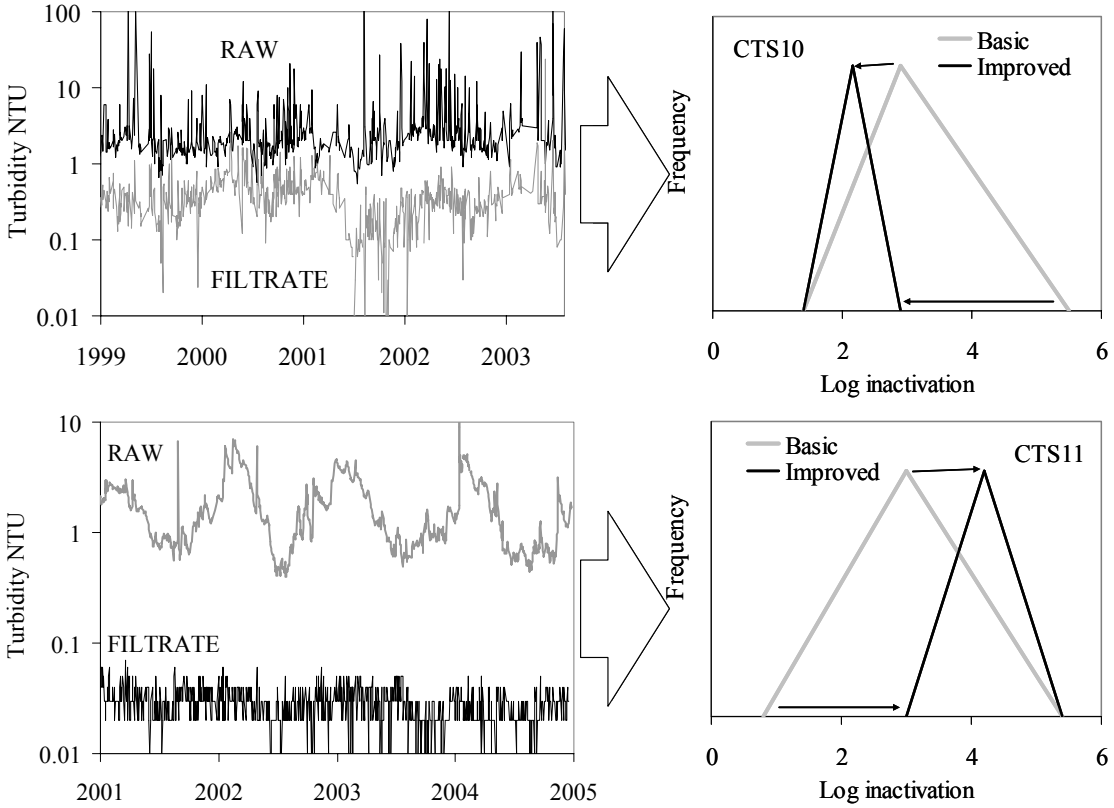


Figure 4.6 Turbidity before and after conventional treatment (CTS 10) and direct filtration (CTS 11) is used to refine the triangular PDF from point estimates to use of turbidity as a surrogate for "good" or "poor" performing filters

Particle removal based on particle counts before and after treatment has a better relationship with micro-organism removal than turbidity removal [Hijnen *et al.*, 2005a]. Changing process monitoring from turbidity to particle counting should lead to a more accurate assessment of treatment efficacy.

4.5.2 Modelling disinfection processes

Initial point estimates for disinfection were modelled based on general operational information, which generally resulted in a very wide range of possible inactivation. By including all available information about the variation of the conditions during disinfection, uncertainty can be reduced. Disinfectant concentration monitoring at the outlet of a contactor, or at several points when the contactor is baffled verifies the presence of disinfectant which is the primary requirement for disinfection. Continuous monitoring (recorded every 1 to15 minutes) is preferred to account for short failure of dosing which results in no inactivation. Flow monitoring verifies the contact time in the contactor and should be recorded at least hourly, since flow variations are generally gradual., Daily recording of temperature is sufficient since it varies gradually.
The effect of variation in disinfection conditions can be verified by modelling based on these input data (see 4.2.5). Figure 4.7 illustrates the result of this approach to better

model *E. coli* O157 inactivation at site 10. Flow variations were not recorded, so design flow was applied in the calculations. The clear water reservoirs (no baffles) were used as contactors, therefore a single CSTR model was applied assuming 30% filling of the reservoir resulting in a conservative estimate of the inactivation.

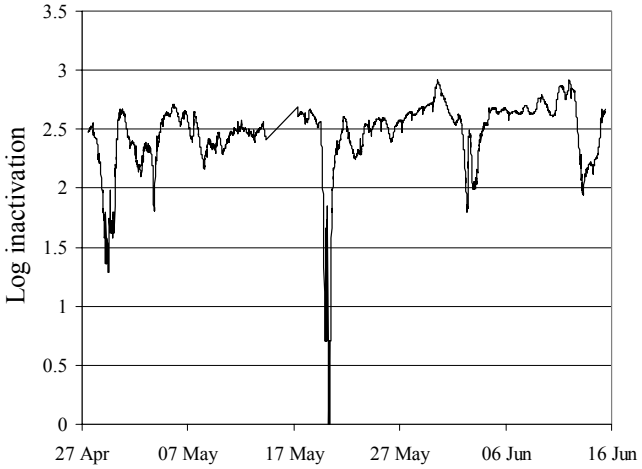


Figure 4.7 Modelled variation of *E. coli* O157 inactivation by chlorination at CTS 10

The estimated mean inactivation of *E. coli* was approximately 2.5 log. On four occasions, however, the chlorine concentration was reduced for a period of two days (not specifically weekends), resulting in 1.5 to 2 log inactivation. The event on 20/5/04 lasted for 12 hours with practically complete failure of disinfection (no chlorine residual). Since no records of flow, operational diary or failure report was available for this site, it was assumed that the treatment was operating and supplying water during this 'event'. As a result the over-all inactivation of *E. coli* by chlorine disinfection in this period was reduced from 2.5 to 2.1 log by this one event. The initial point estimates for disinfection at CTS 10 resulted in a triangular PDF with parameters 0, 3 and 7. Modelling disinfection verified that inactivation appears to vary between 2 and 3 log 95% of the time and also showed that some events of less inactivation occurred. A beta distribution was fitted to the calculated inactivation (expressed as fraction, see Chapter 7) to provide more information in the QMRA. The same approach was applied to determine the PDF of virus and *Giardia* inactivation. For *Cryptosporidium* no further assessment was necessary as the point estimates described previous had showed that even under ideal conditions at CTS 10, no inactivation of *Cryptosporidium* by chlorine is expected.

The largest uncertainty in this more detailed modelling was in the hydraulic characteristics of the clear water reservoirs for different fill levels. If a 2 or 10 CSTRs in series hydraulic model was verified by tracer tests, calculated inactivation of *E. coli* would be 4 or >7 log respectively. Additional monitoring of flow and the variation of the reservoir levels could improve the assessment of inactivation by chlorine disinfection at CTS 10. Temperature should be monitored daily instead of monthly.

Disinfection assessments at other CTSs indicated some general shortcomings of available data to model disinfection. Only CTS 2 provided results of tracer tests of the ozone contactors. Despite baffling, the hydraulics were characterised by only 4 CSTRs. For the other CTSs a single CSTR model was used for modelling. Some sites only monitored disinfectant dose so disinfectant decay was not included in the models, thus conservative estimates of disinfectant concentrations were applied for modelling disinfection.

Further uncertainties arise from the limited knowledge of inactivation kinetics for (environmental) pathogens (Section 4.2.5). For CTS 2 special pilot tests (see 4.7.1) were performed to determine kinetics of *Campylobacter* inactivation by ozone, since information was not available in the literature.

The measurement of residuals at low concentrations can cause analytical errors due to the cross sensitivity to disinfection by-products [USEPA, 1999] and can result in an over-estimation of the achieved inactivation.

Most CTSs operate their disinfection processes at a fixed set-point for disinfectant residual. Thus seasonal variation of inactivation due to temperature fluctuations and hourly variation due to flow fluctuation occured. This would generally be expected to result in reduced inactivation at lower winter temperatures, when the source water contains the highest number of pathogens (Chapter 3). This provides opportunities for improved operation by adapting operation to changing conditions.

4.6 INDICATOR AND PATHOGEN MONITORING

4.6.1 Indicator monitoring

Compliance with drinking water legislation is partly based on monitoring for indicators like *E. coli* and sulphite-reducing clostridia. In general, most samples were non-detects, which widens the uncertainty in estimating their actual values. Conclusions from mainly non-detect samples results in an estimate of an “upper limit”, for example the average concentration in treated water is below X organisms per litre with a 95% confidence. The uncertainties when translated to pathogen concentrations are very large (see 5.4.2).

If indicators are also monitored in the raw water, their reduction by treatment can be assessed. *E. coli* removal can than be related to removal of bacterial pathogens with similar characteristics, which supports their expected removal but the non-detects again increase uncertainties.
When indicators are present and monitored before and after a single process, its efficacy can be assessed and described by a PDF. The number of positive samples may decrease in the subsequent treatment steps and will be of less value for the QMRA unless a reasonable percentage of the samples remain positive (say 50%).

When possible, 'paired' measurements before and after treatment on the same day should be used to calculate log removals. Differences in measured concentrations will however occur due to the variations in time and "space" (see Chapter 7). Indicators are generally monitored 12 to 52 times per year. It is unlikely that rare events would be captured in such a dataset. Despite these shortcomings, the removal of indicators can support the risk assessment and reduce the uncertainty on the overall (baseline) reduction of pathogens, i.e. for non-event periods. Further advise on appropriate statistical methods are described in Chapter 7.

Section 4.9.3.1 provides an example of how *E. coli* monitoring before and after pre-ozonation at CTS 1 was used to determine the PDF for inactivation of the bacterial pathogens *E. coli* O157 and *Campylobacter*.

The following example illustrates how analysing sufficient sampling volume can be used to refine the assessment of log removals. At CTS 2 *E. coli* is monitored in the raw water, filtered water and ozonated water. In raw water 100 mL samples were sufficient to monitor *E. coli* concentrations, in the range of 10-10,000 $CFU.L^{-1}$, while one-litre samples after filtration gave some 85% positives at concentrations between 1 and 100 $CFU.L^{-1}$. A PDF of bacterial removal was based on the 400 data pairs with the method described in Section 4.9.3.1.

After ozonation only 3% of the 1-L samples were positive and thus provided limited information on variation of *E. coli* inactivation. The inactivation was "larger than 1.2 log units" although several log units of inactivation by ozonation were expected. By increasing the sample volume to 25-100 L after ozonation, 44% were positive for *E. coli*. The concentrations ranged from 0.02 to 0.15 $CFU.L^{-1}$, indicating 2.2 to 3 log inactivation. In this case the 3% positive 1 L samples apparently represented the occasional positives of a low baseline concentration rather than events.

In these examples, *E. coli* reduction was used as an indicator of *E. coli* 0157 and *Campylobacter*. Alternative model micro-organisms are discussed in Section 4.2, and include the use of spores of sulphite-reducing clostridia or aerobic spores as indicators for protozoa reduction and phages for enteric viruses. Monitoring of these indicators is less common and their applicability needs to be considered based on the local situation before starting a monitoring programme.

Figure 4.8 shows that under certain conditions indicator organisms can be measured all the way through a multiple barrier treatment system such as CTS 1. In this case aerobic spores are present in very high numbers in the raw water (10,000 $CFU.100\ mL^{-1}$). Since aerobic spores have a high ozone resistance, both pre-ozonation and intermediate-ozonation have little effect on their concentrations. Sedimentation, rapid gravity filtration and granular activated carbon filtration will more effectively remove spores. The final disinfection by super-chlorination and de-chlorination achieves substantial reduction of aerobic spores. Remarkably 5 out of 15 samples of the raw water were negative for aerobic spores (<100 $cfu.100\ mL^{-1}$). This either indicates high variability of the aerobic spore concentration or problems at the laboratory.

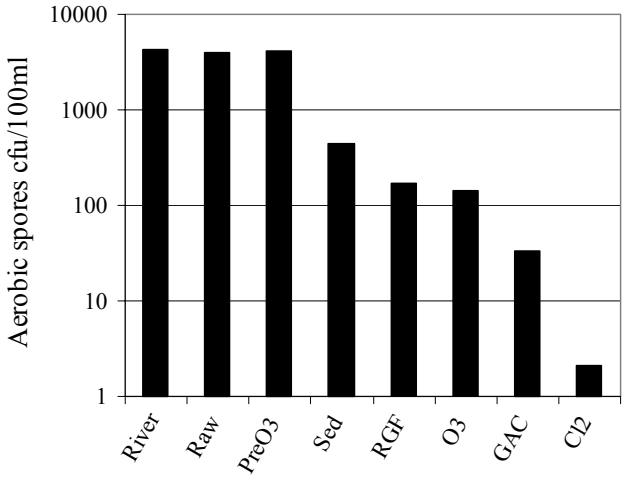


Figure 4.8 Average aerobic spore concentrations at a multiple barrier treatment plant (CTS 1)

Other organisms monitored at CTS 1 showed that removal of *E. coli,* Enterococci and Total Coliforms mainly occurred at the pre-oxidation stage, while Aerobic Spores, Sulphite-reducing clostridia and *Staphylococcus aureus* were mainly removed during the coagulation-clarification step.

4.6.2 Pathogen monitoring

The most direct determination of the pathogen risks and treatment barrier efficacy is by sampling for pathogens at full-scale, but is costly and long-term investigation with frequent sampling is seldom feasible.

At CTS 1, *Campylobacter*, *E. coli* O157, Enterovirus, *Giardia*, *Cryptosporidium* and *Pseudomonas aeruginosa* were monitored monthly in the raw water and after each treatment step for one year. However, only *Pseudomonas* and Enterovirus were detected in the raw water. The former was detected three times after pre-ozonation and once after sedimentation, while infectious Enterovirus was reduced strongly by pre-ozonation but still detected twice after inter-ozonation. Since many pathogens were absent in the highly polluted source water, the monitoring within treatment provided high uncertainty on the efficacy of pathogen removal at CTS 1.

At CTS 2 *Campylobacter* was monitored in the raw water, after filtration and after ozonation. All raw water samples were positive, in concentrations of 10 to 1,100 $MPN.L^{-1}$ (mean 211 $MPN.L^{-1}$). The corresponding values after filtration were 0.4 and 110 $MPN.L^{-1}$ (mean 11 $MPN.L^{-1}$) and after ozonation 39% were positive, between 0.04 and 0.4 $MPN.L^{-1}$ (mean 0.05 $MPN.L^{-1}$). Thus, filtration provided 1.3 log removal, which confirms 'good' performance of the filter compared to the initial estimate of 0.1 to 1.5 log removal and a MEC of 0.6. Ozonation provided 2.3 log inactivation which is in the same range as the initial conservative estimated inactivation of 2.6 (as part of calculations that resulted in table 4.16).

When samples before and after filtration were paired, the inactivation seemed to vary between 0 and 2.5. This large observed variation may be caused by the MPN method used (three dilutions in tripled), which resulted in a most probable number with a 95% confidence interval of 0.4 to 1 log depending on the number of positive tubes. When pairing MPN numbers before and after filtration the calculated reduction had a confidence interval of approximately 1.6 log. Advanced statistical methods as described in Chapter 7 are required to estimate the PDF parameters directly from the presence-absence results of the MPN method.

4.7 COLLECTING ADDITIONAL INFORMATION

4.7.1 Pilot-scale experiments

Pilot-scale experiments may supply additional information where the monitoring or operational full-scale installations give insufficient information about treatment performance for different organisms. This may be due to the absence or low numbers of organisms in the final stages of treatment or limitations of monitoring to assess the reduction efficiency for certain groups, like viruses. Pilot experiments can help determine the applicable (local) reduction values from the wide range found in the literature When additional treatment is considered on-site pilot testing will provide site specific performance indication of the process under consideration.

Pilot challenge tests under full-scale conditions: SSF
Slow Sand Filtration (SSF) was one of the major barriers against pathogens at CTS 2, and data indicated that *Campylobacter* reduction might be insufficient to reach health based targets. As described in Section 4.2 SSF can reduce bacteria by 1.2 to 4.8 log. This could not be verified by indicator monitoring at full-scale since most samples before and all samples after SSF were negative for indicator organisms. Long term pilot tests were conducted at the treatment site where the pilot filters were run parallel to the full-scale filters under the same conditions but with additional indicator micro-organisms dosed directly before the SSF pilot filter. The results indicated that over 2 log removal occurred shortly after scraping off the “schmutzdecke” up to 4 log removal for a fully ripened filter. At CTS 2 only 2 out of 26 filters are scraped at the same time, and filter to waste is applied for several weeks. Hence, the point estimate for the SSF treatment step of 3 logs removal was applied [Hijnen *et al.,* 2004; Dullemont *et al.*, 2006].

Bench-scale ozone disinfection tests with natural water
At CTS 2 *E. coli* was occasionally detected after ozonation, which overall indicated some 2.2 log inactivation. Further, *E. coli* was regarded as a potential index for *Campylobacter* inactivation by ozonation. This was suggested as the literature provided no references on *Campylobacter* inactivation by ozone and only four publications on *E. coli* inactivation were identified. Bench-scale ozone inactivation tests of laboratory-cultured and environmental *E. coli* and *Campylobacter* were performed using water from CTS 2. Results showed that inactivation of both organisms was similar and that

environmental *E. coli* and *Campylobacter* were more resistant to ozone than cultured organisms. The resulting inactivation kinetic parameters are presented in Table 4.9 and were also used for modelling ozone disinfection of bacteria at other CTSs [Smeets *et al.*, 2005].

Pilot challenge tests under full-scale conditions: Conventional treatment
The reduction efficiency of enteric viruses by chemical flocculation in drinking water treatment was assessed in a pilot-plant under similar conditions to full-scale. The relative reductions of index bacteriophages φX174 and MS-2 by different sub-treatment steps was assessed. The phages were added either before the addition of chemicals or in the first flocculation chamber and the relative cumulative recovery was measured at subsequent steps. Results are summarized in Figure 4.9, but in general φX174 was not reduced as well as MS-2 and thus φX174 was a more conservative indicator of the barrier function and the preferred model organism (surrogate) for human enteric viruses. It is evident in Figure 4.9 that the chemical addition alone provided more than 1 log reduction, and flocculation and sedimentation/filtration each an additional log reduction. The total reduction by conventional treatment was in the range of 3.8 logs. The finding that chemical addition can may lead to substantial virus inactivation needs to be demonstrated with human enteric viruses, as phages are known to be more sensitive to some chemical effects, such as pH shock, and factors other than the current Derjaguin-Landau-Verwey-Overbeek (DLVO) model of virus attachment are important and even differ between MS2 and φX174 [Song *et al.*, 2005].

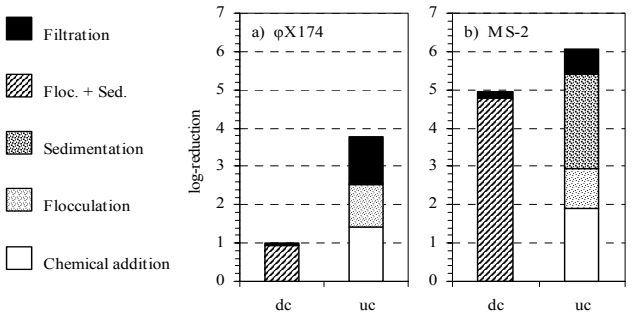


Figure 4.9 Log-reduction of a) φX174 and b) MS-2 bacteriophages by conventional treatment.
dc= downstream case, bacteriophage addition into first flocculation chamber.
uc=upstream case, bacteriophage addition upstream of chemical addition, differentiated between floccuation/sedimentation**.**

Occurrence of retardation in conventional treatment
Retardation during conventional processes is suspected. A peak concentration of pathogens appear to be removed substantially when comparing samples shortly after spiking the source water. However, the removed pathogens are slowly released over time, so a substantial part of the total pathogen load does pass treatment. This was tested by spiking the water with both phage surrogates and a salt solution. The passage of the phage spike and the conductivity increase was followed through the system after sedimentation (Figure 4.10a) and filtration (Figure 4.10b). The peak of both

bacteriophages coincides with the conductivity peak which showed that no apparent retardation through sedimentation and filtration occurred (Figure 4.10) although phages φX174 were released in low concentrations from the filter after 12h of operation [Heinicke *et al*., 2004]. This study supports that measurements before and after treatment can be paired when sufficient hydraulic residence time is accounted for.

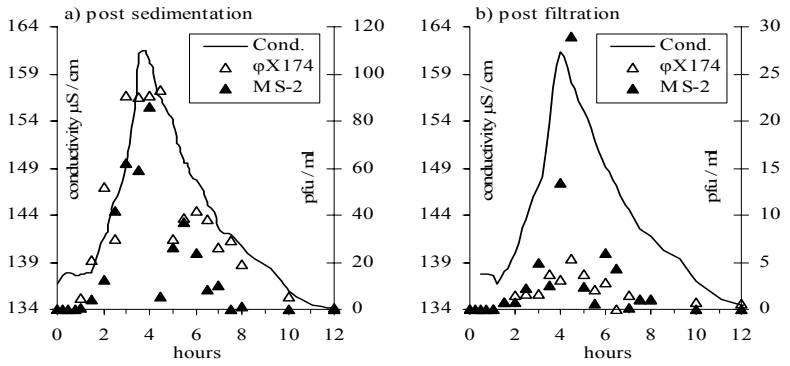


Figure 4.10 Peaks of conductivity and bacteriophages in the upstream case (uc). A) post sedimentation and B) post filtration. Note difference in scale on secondary y-axis.

4.7.2 Full-scale indicator spiking

Naturally occurring pathogens in source waters can be so low that removal at full-scale cannot be determined from ordinary sampling. In specific cases it may be possible to challenge an operational full-scale plant with model micro-organisms. The model micro-organism needs to be safe for the consumers and should not lead to a deterioration of water quality. At CTS 8 sufficient reduction of *Cryptosporidium* is essential, and performance of the full-scale treatment needed to be verified. An underlying difficulty in simply measuring naturally occurring *Cryptosporidium* densities before and after DAF (or some other) treatment to determine the treatment efficacy is that oocysts typically exist in low numbers that are difficult to detect. Dosing full-scale plants with oocysts to attain high enough densities to obtain detects is simply not an option due to associated health concerns. Chung *et al.* [2004] demonstrated that Baker's yeast (*Saccharomyces cerevisiae*) is a good model for *Cryptosporidium* removal by water treatment processes and is a substantially safer organism to work with than *Cryptosporidium*. The functioning full-scale water treatment plant in CTS 8 was challenged with *S. cerevisiae* to gain an initial assessment of the *Cryptosporidium* removal capabilities of the DAF and rapid sand filtration (RSF) water treatment plant.

Two out of three parallel DAF units studied showed a mean *S. cerevisiae* removal of 1.5 log, whereas the third line only provided 1 log removal. Combined removal by the three parallel DAF units was therefore 1.3 log. The lower saturated air flow to one unit was considered as the cause for reduced removal.

RSF provided 2.0, 2.3 and 2.6 log removals for three parallel filters respectively, providing a combined removal of 2.2 log. After six hours of operation the removal by

one filter was reduced from 2.0 to 1.4 log for approximately 10 minutes, while the other filters maintained their removal capacity. The combined removal was reduced to 1.8 log removal during this event for which no clear cause was found.

The effect of the ripening period after backwashing was also studied at one filter at CTS 8. The mean removal during the first hour after the filter was brought back on-line (2.4-$\log_{10}$) was not significantly different (5% level) to that observed during other periods. Although no filter-ripening period was observed in the microbiological data, concurrent particle count data collected during the trial did display a definite 'ripening-period' involving *Cryptosporidium* and *S. cerevisiae* sized particles. Backwash water recirculation resulted in increased numbers before DAF treatment, but had little effect on the removal efficiency of the treatment.

When using these results in QMRA to assess *Cryptosporidium* risks, the uncertainty concerning how closely *S. cerevisiae* model naturally-occurring oocysts needs to be accounted for. Regardless, such work does provide the opportunity to identify weaknesses in a system, such as poorly performing components of the treatment system, or the magnitude of inherent fluctuations in treatment performance. Combination with surrogate measurements such as particle counting demonstrates potential limitations of such surrogates for quantifying treatment efficacy for pathogen removal. It also shows which issues are irrelevant to microbiological risk, such as backwash water recirculation at this site. Such details may otherwise go unnoticed when comparing pre- and post-treatment water quality samples of pathogens or model micro-organisms that do not exist in large enough quantities to be detected in numbers sufficient to ensure relatively accurate representation of actual conditions [Signor *et al.*, submitted].

4.7.3 Failure reports

Although short periods of treatment failure can have a large impact on average risk of infection, water companies generally do not record failure events in treatment systematically. A study of collected treatment failure reports aimed to examine the impact of incidents on the annual risk of infection [Westrell *et al.*, 2003]. It accounted for the type, frequency and magnitude of failures in treatment and distribution in a microbial risk assessment that included *Cryptosporidium*, rotavirus and *Campylobacter jejuni*. The main risk incidents in water treatment were associated with sub-optimal particle removal or malfunction in disinfection. The majority of the potential annual infections were likely to be due to the pathogens passing treatment, due to its variability, and not due to the actual failures thus adding to the background endemic rate. However, the failures represent short periods of malfunctioning and may potentially be linked with outbreaks as well as to exposure of sensitive sub-groups of the population. Among the modelled pathogens, viruses appear to pose the largest risk of infection. The simulated total number of annual infections was within the equivalent range estimated from epidemiological data, accounting for the fraction of gastroenteritis attributable to tap water.

4.7.4 Direct monitoring of pathogens in drinking water: UK *Cryptosporidium* survey

Data from UK water companies on the outcome of their statutory monitoring for *Cryptosporidium* was obtained for 216 sites. For 8 sites data has also been provided on the numbers of *Cryptosporidium* in the source waters. The overall removal at these sites was calculated from the average concentration before and after sampling. Table 4.19 shows the treatment processes used at each site and the average overall *Cryptosporidium* oocyst removal. A variation of one log was observed among similar treatments.

Table 4.19 *Cryptosporidium* reduction based on UK regulatory monitoring

Treatment processes	**log removal**
A. Coagulation-Polyelectrolyte-Sedimentation-Filtration-GAC filtration-Chlorination	4.3
B. Coagulation-Polyelectrolyte-Sedimentation-Filtration-GACfiltration-Ozone-Chlorination	4.3
C. Coagulation-Polyelectrolyte-Sedimentation-Filtration-GAC filtration-Chlorination	3.4
D. Coagulation-Polyelectrolyte-Sedimentation-Filtration-GAC filtration-Chlorination	3.3
E. Impoundment-Coagulation-Polyelectrolyte-Sedimentation-Dissolved Air Flotation-Filtration-GAC filtration-Chlorination-	3.2
F. Impoundment-Coagulation-Polyelectrolyte-Sedimentation-Filtration-GAC filtration-Chlorination-	3.2
G. Coagulation-Sedimentation-GAC filtration-Ozone-Chlorination-	3.1
H. Coagulation-Sedimentation-Filtration-GAC filtration-Chlorination-	2.6

The typical minimum-mean-maximum treatment performance criteria for a *conventional treatment-GAC filtration-chlorination* plant is presented in Table 4.20. Compared to CTS 8 (Table 4.17), which applies a similar process scheme, the estimated 3.6 log over-all reduction of *Cryptosporidium* (95% confidence interval: 2.7 to 5.9 log) is similar.

Table 4.20 Minimum, Mean Elimination Capacity (MEC) and maximum log reduction of *Cryptosporidium* oocysts by processes and worst case, estimated and best case log reduction by a typical surface water treatment system

	Cryptosporidium **min-MEC-max**
Conventional treatment (Coag.-Sed.-Filtr.)	1.4-3.2-5.5
GAC filtration	0.7-0.9-1.1
Disinfection Cl_2	0-0-0
Estimated total log reduction	**2.1-4.1-6.6**

4.8 INFLUECE OF SYSTEM CONFIGURATIONS

4.8.1 Assessment of combined processes

The application of multiple barriers in treatment provides process performance stability, as failure of one barrier does not directly result in the failure of the whole treatment. However, the barriers need to be assessed for their combined effect on drinking water safety. Both positive and negative interaction between treatment steps can be observed.

Positive interaction occurs when the failure of a preceding treatment step leads to increased efficacy of the following treatment step. When coagulation-sedimentation is sub-optimal, rapid sand filters typically show an increased performance. The resulting combined removal efficacy is much more stable that the individual removal efficacies.

Negative interaction can also occur. When particle removal before a disinfection process is reduced, the efficacy of the disinfection can also be reduced due to protection of organisms in aggregates. This can occur during both chemical and UV disinfection. A failing pre-oxidation step can also result in an increase of disinfectant demand later in treatment. This leads to lower Ct values and thus to less disinfection. This is illustrated by the on-line data analysis of CTS 1 in Chapter 8 (SCADA monitoring data analysis). Failing coagulation-sedimentation can also lead to increased oxidant demand with the same result.

Both positive and negative interaction can be observed with conventional treatment. Table 4.21 shows the range of organism removal by coagulation-sedimentation and filtration separately and combined found in literature. When one treatment fails the other one stays intact or even increases removal. So the observed minimum for combined process in Table 4.21 is higher than the added minimal removal of both separate processes. On the other hand, when the first process is very effective in removing pathogens, the second step can be less effective, therefore maximum removal of the combined process is lower than the individual process maxima added.

Table 4.21 The range of *Cryptosporidium* removal found in conventional treatment experiments is smaller than when both processes are studied separately (Section 4.2), showing that positive interaction between processes has occurred.

	min	MEC	max
A Coagulation-sedimentation experiments	0.4	1.9	3.8
B Filtration experiments	0	2	3.1
A + B	0.4	3.9	6.9
Conventional treatment (coag-sed-filt.) experiments	1.4	3.2	5.5

The combination of physical removal and inactivation has proved to be effective in maintaining high removal for different organisms. Studies on removal of indicators at full-scale suggest that where prechlorination is used there would appear to be only a low risk of microbial breakthrough. Micro-organisms surviving the initial impact of prechlorination were removed efficiently by the combined effects of chlorine continuing to act and the subsequent treatment processes used. GAC filtration enhances

the survival and even proliferation of micro-organisms including those of sanitary significance such as members of the coliform group. Final disinfection would act as an effective final barrier and there is no evidence to suggest that, even in the case of GAC effluents, this is being unduly challenged because of any inadequacies of previous treatment stages. A two-stage treatment by rapid and slow sand filtration plus post chlorination removed >99.99 % of *E. coli*, faecal streptococci, *C. perfringens* and coliphages, as did treatment by pre-chlorination, coagulation, sedimentation, rapid sand filtration and post chlorination.

Careful selection of data is needed in a treatment assessment, as now illustrated. At CTS 3 turbidity was monitored in the raw water, after coagulation-sedimentation and after rapid sand filtration-softening-GAC filtration. Figure 4.11 shows how turbidity decreased after settling. This can be used to verify settling efficacy with greater detail. After the two filtration processes turbidity frequently exceeded the settled water turbidity up to the level of raw water turbidity. This turbidity increase may be caused by particles formed during softening or by particle release by the GAC filters. Comparing filtrate turbidity to raw water turbidity without considering settled water turbidity would erratically suggest poor particle removal by treatment. This example illustrates the importance of monitoring each process individually for interpretation in a risk assessment.

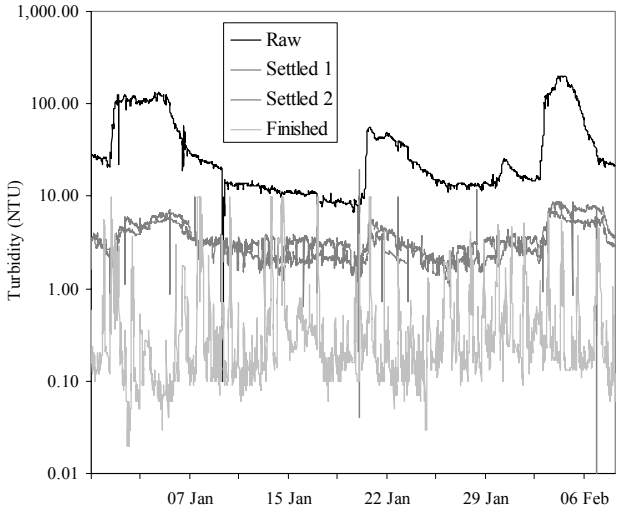


Figure 4.11 Turbidity removal at CTS 3, settled 1 and settled 2 are two parallel lines. Turbidity decreased by settling and then increased periodically after filtration due to other treatment processes.

4.8.2 Effect of treatment configuration and parallel lines

Full-scale treatments generally consist of two or more lines and multiple parallel process units for each process step. In general, other units compensate for a single failing unit. However, this is not the case for pathogen removal. When a process consisting of 10 parallel units has a typical removal of 3 log units, the failure of one unit will reduce the effect of the whole process to one log removal (see Box I – weakest link).

At CTS 2 the ozonation takes place in five parallel contactors each treating one fifth of the total flow. All lines are operated in the same way. Still ozone measurements in all contactors show differences in performance of each line. *E. coli* disinfection was modelled based on these measurements. Figure 4.12 shows the relative contribution of each line to the total amount of *E. coli* in the treated water on one day that ozone was measured. In Figure 4.2 line 3 performs relatively poorly, on other days this was observed for line 1. Depending on the period either one of these lines was responsible for 70%-90% of the total load of organisms in the treated water. Line 2 performed the best and was only responsible for a fraction of the organisms after ozonation.

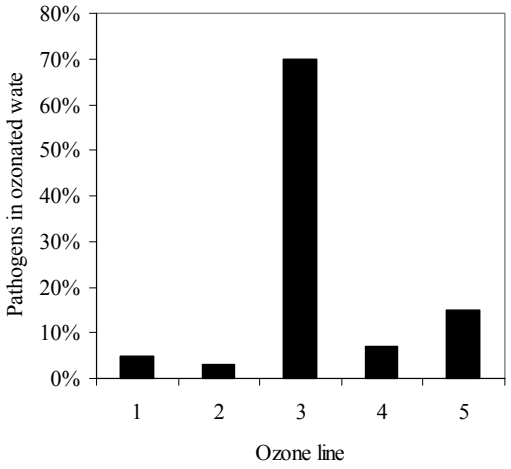


Figure 4.12 Relative contribution of each of 5 ozone lines tot the total number of bacteria after ozonation modelled based on measured ozone concentrations on one day at CTS 2

4.9 EXAMPLE OF A FULL TREATMENT ASSESSMENT

A full assessment of the treatment of CTS 1 is presented as an example. Figure 4.13 shows the treatment scheme. Background information on the reduction of pathogens by these treatment processes is given in Paragraph 4.2. To keep the example comprehensive it is focussed on bacteria. The same methods can be applied to other pathogens.

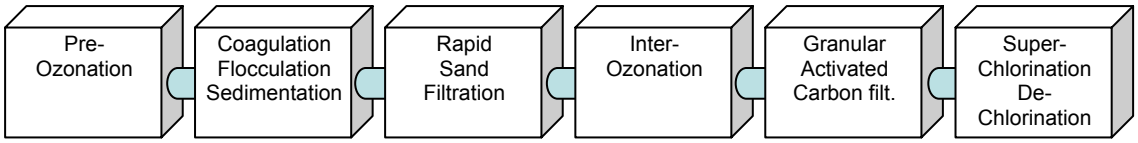


Figure 4.13 Treatment scheme at CTS 1

4.9.1 CTS 1 point estimate assessment

The efficacy of the physical treatment steps was estimated from values reported in the literature in Section 4.2. Coagulation, sedimentation and filtration at CTS 1 are considered as conventional treatment. Disinfection takes place at three stages in the plant. For a first estimate of the inactivation by disinfection, general information about the treatment processes at CTS 1 was collected (Table 4.22).

Table 4.22 Disinfection process characteristics for CTS 1

	Contact time	**Disinfectant residual**	**CT**	**Temperature**
	min	**mg.L^{-1}**	**mg.min.L^{-1}**	**°C**
Pre-oxidation Cl_2+O_3	4	0-2	0-8	5-23
Oxidation O_3	15	0.3	4.5	5-23
Disinfection Cl_2	120	2-3	240-360	5-23

Based on the specified conditions in Table 4.22, the expected range of inactivation was estimated by calculating minimal, likely and maximum inactivation respectively. Data analysis for this as well as the other CTSs showed that most systems do experience short periods of no disinfectant residual. Therefore as a first approximation minimum inactivation was set to 0, since a continuous presence of disinfectant needs to be verified by further data analysis. The likely reduction was calculated with the CSTR model, assuming 1 CSTR and 'mean' disinfectant residual and representative contact time and temperature values as a conservative estimate to calculate the assumed reduction. The more detailed analysis that follows was used to refine the calculations. The maximum inactivation was calculated based on Ct assuming plug-flow conditions (no correction for t10) during 'high' disinfectant concentration and temperature and longest contact time. Disinfection is unlikely to exceed this level. Table 4.23 summarizes the resulting reductions from these disinfection calculations in combination with the literature values of the physical processes for the treatment steps at CTS 1.

Table 4.23 Minimum, Mean Elimination capacity (MEC) and maximum log reduction of pathogens by processes and worst case, estimated and best case log reduction by total treatment at CTS 1

	Viruses min-MEC-max	**Bacteria min-MEC-max**	***Cryptosporidium* min-MEC-max**	***Giardia* min-MEC-max**
Pre-oxidation Cl+O3	0-1.6->7	0-2.9->7	0-0.3-2.7	0-1.3->7
Conventional treatment (Coag.-Sed.-Filtr.)	1.2-3.0-5.3	1.0-2.1-3.4	1.4-3.2-5.5	2.1-3.4-5.1
Oxidation O3	0-1.7->7	0-2.9->7	0-0.4-1.5	0-1.4->7
GAC filtration	0.2-0.4-0.7	0.9-1.4-2.9	0.7-0.9-1.1	0.4-1.7-3.3
Super chlorination	0-3.2->7	0-3.4->7	0	0-1.3->7
Disinfection Cl	0-2.3->7	0-2.4->7	0	0-0.5-1.8
Estimated total log reduction	**1.4-12->34**	**1.9-15->34**	**2.1-4.8-11**	**2.5-9.6->31**

Table 4.23 illustrates that the point estimates result in a broad range between the minimum and maximum removal. The estimates for the single processes represent the variability of pathogen reduction by these processes based on the variables for the disinfection conditions or by reported values in literature. The minimum estimate of the

total reduction actually represents a 'worst case' in which all treatment steps perform at their worst or fail, which has a extremely low likelihood of occurrence. Usually other processes will continue to function when one process fails. This combined effect was simulated by applying a triangular distributed PDF for each treatment step and combining the treatments steps in a Monte Carlo simulation, as described in Chapter 7. The min, MEC and max values in Table 4.23 are applied as the minimum, most likely and maximum parameters for the PDF. By randomly drawing reduction values (generally 10,000 draws or more) from each PDF of the respective treatment steps, reduction by the total treatment was calculated. The result represents the expected variation of reduction by the total treatment in time. This variation is presented by three values in Table 4.24. The over-all reduction is the total reduction of pathogens over the period (see Box II over-all reduction). The reduction varies between the 5% and 95% percentiles for 90% of the time.

Table 4.24 Pathogen log removals at CTS 1 based on a Monte Carlo assessment of treatment performance

	Viruses	**Bacteria**	***Cryptosporidium***	***Giardia***
Over-all log reduction	8.1	10.2	4.8	9.1
5th percentile	11.0	11.8	4.2	10.0
50th percentile	15.7	16.6	5.9	14.3
95th percentile	21.0	20.6	7.8	19.2
Minimal log reduction in 10 000 simulations	4.1	6.6	2.7	5.3

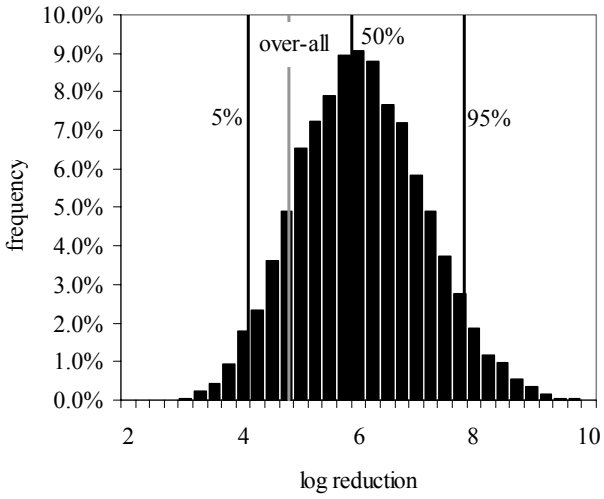


Figure 4.14 Monte Carlo frequency distribution of *Cryptosporidium* reduction at CTS 1. Over-all reduction is the resulting reduction of all the treated water according to this frequency distribution.

Figure 4.14 and Table 4.24 show that without further information, we would expect that *Cryptosporidium* reduction by CTS 1 varies between 4.2 and 7.8 log for 90% of the time resulting in over-all reduction of *Cryptosporidium* of 4.8 log.
The corresponding values for virus removal were between 11 and 21 log reduction 90% of the time, with an over-all removal of 8.1 log. The calculations illustrate that the

expected poor removal during 2.5% of the time has a high impact on the overall removal of viruses. So if the rare occasions of poor removal would not occur, the overall reduction would be much higher. From Table 4.23 it is clear that no removal may occur at the disinfection processes part of the time. Therefore disinfection is an important control point in drinking water treatment, as described in Chapter 2. Detailed data analysis using additional data is needed to determine the frequency of "no inactivation".

The catchment of CTS 1 is a highly polluted river and the expected reductions provided in Table 4.24 and Figure 4.14 are likely to be sufficient to reach health based targets as presented in Section 4.1. The minimal reduction in Table 4.23 represents performance that will not reach a health based target. The expected reduction in Table 4.24 should therefore be verified with additional data.

4.9.2 CTS 1 assessment using surrogates and process conditions

For each individual treatment step the available local surrogate and process data was investigated to reduce the uncertainty about pathogen reduction by the CTS 1 water treatment. Each treatment step is now discussed in further detail..

4.9.2.1 CTS 1 Modelling pre-oxidation with ozone

The inactivation by a disinfection process is modelled to verify inactivation based on the measured process conditions and design characteristics at full scale. At CTS 1 only the general values on process conditions in Table 4.22 were provided for pre-ozonation. No data on ozone concentrations or dosage was available to provide information on variation of inactivation in time, only the variation of water temperature was provided. To better account for variation the inactivation was calculated at minimum and maximum temperature to replace the single most likely value of the triangular PDF by 1.5 and 2.0 log (Figure 4.15).
The design of the system was next taken into account. At CTS1 the contact time was very short and no baffles were applied in the contact chamber, therefore plug-flow was unlikely. The maximum estimate of inactivation was adjusted by performing a single CSTR calculation under 'high' conditions instead of a Ct calculation, which resulted in 2.3 log inactivation (Figure 4.15).
Ozone gas flow was recorded every 20 seconds, but since the ozone in gas concentration was unknown, it could not be used to calculate ozone dose. The on-line records of ozone gas flow were only used for event analysis. Periods of zero gas flow represent the absence of ozone. During some of these periods, chlorine may have been used for pre-oxidation, but no records on that were provided. The event analysis provides an estimate of 'how bad it could be'. Ozone gas flow normally varies between 60% and 200% of the mean flow. The data shows that ozone gas flow is less than 5% of the mean flow for 1% of the time, indicating no ozonation. Therefore the frequency of no inactivation was increased to 1%. Figure 4.15 shows how the triangular PDF was refined.

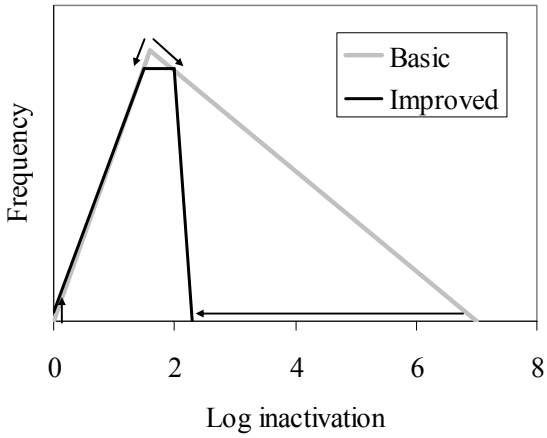


Figure 4.15 Refining the triangular PDF of log inactivation of bacteria by pre-ozonation at CTS 1 using local process data.

The PDF could be further refined by determining the residence time distribution in the contactor, monitoring disinfectant concentration and recording temperature at a higher frequency.

Two other CTSs have a pre-oxidation step. CTS 3 also used ozone and provided similar information on disinfection conditions, apart from the ozone gas flow. Therefore the same approach was applied there. CTS 10 used Cl_2 but provided no information on the disinfection conditions, therefore disinfection could not be modelled.

4.9.2.2 CTS 1: Surrogates for conventional treatment (coag.-sed.-filtr.)

On-line measurements of turbidity at CTS 1 were available for the raw water, after the flat bed clarifiers (two lines) and after a single filter (out of 20 parallel filters). Turbidity in raw water was assumed to be unaffected by pre-ozonation so it was representative of conventional treatment influent. The turbidity measurement for the single filter was regarded to be representative for all individual filters (not for the combined flow). Since conventional treatment (coagulation-sedimentation-filtration) is regarded as a combined process, only the raw water and the filtrate turbidity were analyzed.

The literature review in Section 4.2 made clear that a good working filter (as part of conventional treatment) would provide a constant turbidity below 0.1 NTU. The turbidity after the filter in Figure 4.16 was >0.1 NTU for 50% of the time reaching up to 2 NTU. Turbidity after filtration showed a clear daily variation due to the filter backwash cycle (see magnification in Figure 4.16). Apparently filter to waste was not applied, increasing the chance of pathogen breakthrough. The triangular distribution from the basic assessment was therefore adjusted to represent a poorly working filter as explained in Section 4.5.1. The maximum removal was set to the MEC value, the minimum value was not changed. The most likely reduction was arbitrarily chosen as the mean of the minimum and MEC. Table 4.25 shows the improved triangular PDF parameters based on turbidity monitoring.

Table 4.25 Improved parameter values using triangular distributions to describe pathogen removal by conventional treatment at CTS 1

Organisms	**PDF parameters**		
	Min	Most likely	Max
Viruses	1.2	2.1	3.0
Bacteria	1.0	1.8	2.1
Cryptosporidium	1.4	2.3	3.2
Giardia	2.1	2.7	3.4

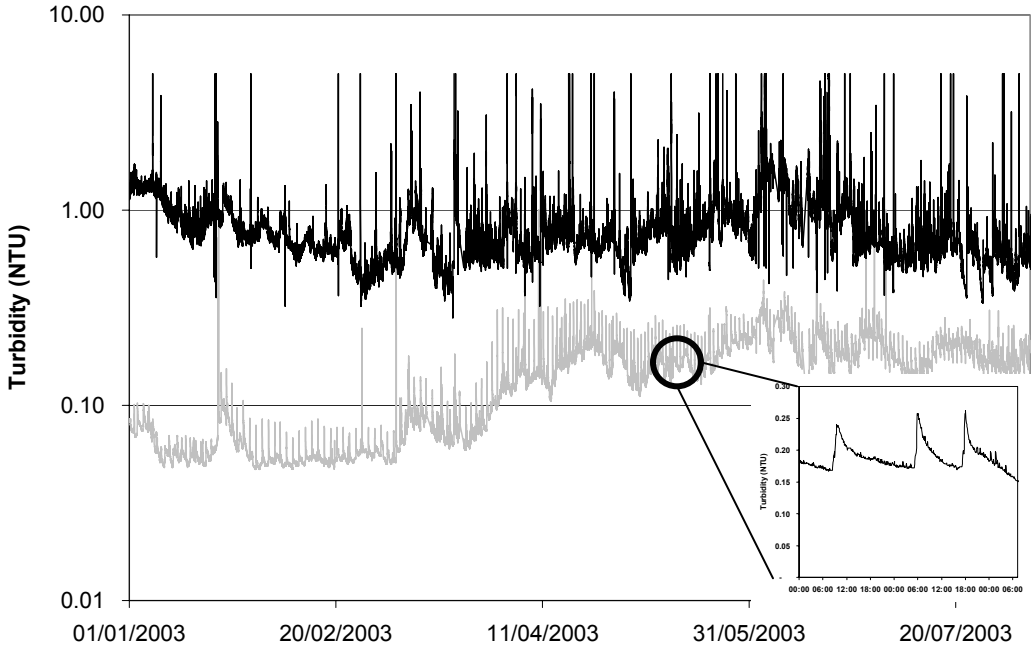


Figure 4.16 Turbidity of raw water (black) and after filter 9 (grey), on-line measurement recorded every 5 minutes and magnification of 3 filter cycles with increased turbidity after backwash.

Including turbidity monitoring of all filters could refine the assessment. Examples of using surrogate monitoring at CTS 10 and 11 were given in Section 4.5.1.

4.9.2.3 CTS 1: Modelling oxidation with ozone

Ozone concentration during main disinfection was recorded every 25 seconds at two parallel lines at CTS 1. Temperature was recorded 49 times in two and a half years, flow was recorded every 5 minutes. Apart from volume no information about the hydraulic characteristics of the ozone contactor was provided.

MEC and over-all inactivation

A single CSTR model was used to calculate a conservative estimate of inactivation. The average measured ozone concentration was 0.48 $mg.L^{-1}$ which is higher than the set point of 0.3 $mg.L^{-1}$. At 0.48 $mg.L^{-1}$ and 10°C a MEC of 3.1 log inactivation of bacteria was calculated, which is slightly higher than first estimated (2.9 log Section

4.9.1). Disinfection was calculated for every single ozone residual record, corresponding temperature and flow were determined by interpolation. Due to variation of the (measured) ozone concentration the overall inactivation of bacteria was slightly lower; 2.8 log for line 1 and 3.0 for line 2 and 2.9 for the combined flow.

Event analysis and PDF
Event analysis at line 1 showed 9 measurements with no ozone residual and 154 measurements that lead to less than 1 log inactivation (0.07 %). Line 2 only contained one measurement leading to less than 1 log inactivation. The results indicated that the main disinfection at CTS 1 was controlled very well, and the minimum inactivation of 0 was too pessimistic. Since the data accurately describes variation of inactivation in time, the baseline variation was described by a beta distribution (of π). The parameters of the PDF are based on the calculated inactivation by maximum likelihood estimation as described in Chapter 7. Statistical uncertainty of the PDF parameters was nil due to the high number of data points. The baseline PDF did not account for the events of less than one log removal. When 0.3 log removal was assumed 0.07% of time (representing no inactivation by one process line so π of the total flow is 0.5) this has no impact on the over-all inactivation by ozonation. Therefore the baseline PDF was applied.

Determining residence time distribution in the contactor and monitoring temperature more frequently (daily) at CTS 1 could further reduce the uncertainty. To reduce remaining uncertainty about inactivation kinetics of environmental pathogens pilot research is needed.

Other CTSs applied only intermediate disinfection (2, 7, 9), only final disinfection (8, 11, 12) or both (3, 4, 5, 6, 10) either with ozone or chlorine. Many sites provided on-line monitoring data but CTS 1 provided the most detailed data on disinfectant concentration and flow. Often a clear water reservoir was used for the final disinfection, so the water level was likely to vary during the day, thus impacting on residence time (distribution). None of the CTSs provided data on the water level and most did not provide the flow data, leaving unnecessary uncertainty in the disinfection calculations.

4.9.2.3 CTS 1: Surrogates for GAC filtration

Turbidity was monitored after rapid sand filtration. The ozonation has no effect on turbidity so this turbidity was considered to be representative for GAC filtration influent. GAC was followed by super chlorination and de-chlorination, which have no effect on turbidity. The monitored turbidity of the treated water was considered representative for GAC filtration effluent. Comparing these monitoring data shows that the GAC filtration has very little effect on the turbidity. Since the GAC filters were not effective in removing turbidity, the triangular PDF was adjusted for a poorly performing filter as described in Section 4.5.1. The MEC was applied as the maximum, while the minimum was maintained. The most likely reduction was arbitrarily chosen as the mean of the MEC and the maximum. Table 4.26 shows the adjusted parameters of the triangular PDF for GAC filtration.

Table 4.26 Improved parameter estimates using a triangular distribution to describe pathogen removal by GAC filtration at CTS 1

Organisms	**PDF parameters**		
	Min	Most likely	Max
Viruses	0.2	0.3	0.4
Bacteria	0.9	1.1	1.4
Cryptosporidium	0.7	0.8	0.9
Giardia	0.4	1.0	1.7

4.9.2.4 CTS 1: Super-chlorination

Chlorine residuals were measured on-line at several points:

1 post super-chlorination
2 pre de-chlorination
3 post de-chlorination
4 post reservoir (drinking water)

The inactivation of *E. coli* by super-chlorination was modelled based on on-line measurements (one per minute) of the residual chlorine before de-chlorination, monitored flow (every 5 minutes) and records of river water temperature. No hydraulic characteristics of the contactor were provided, therefore it was modelled conservatively as a single CSTR.

This resulted in 2.7 log inactivation of bacteria in winter up to 2.9 log in summer. In 2003 only 440 out of 525600 measurements (0.08%) indicated less than 1 log inactivation of bacteria. These short periods of recorded low residual concentrations might be due to failure of chlorine measurement or signal processing since they abruptly drop from normal level to 0 while a gradual decrease of concentration is expected when dosing is interrupted. The modelling predicted that sufficient residual was maintained 99.92% of the time and that the moments of possible loss of residual have had no significant impact on the over-all inactivation by super-chlorination. Since the data accurately describes variation of inactivation in time, the baseline variation was described by a beta distribution (of π). The parameters of the PDF were based on the calculated inactivation by maximum likelihood estimation as described in Chapter 7. Statistical uncertainty of the PDF parameters was nil due to the high number of data points.

By determining residence time distribution in the contactor and monitoring temperature more frequently (daily) at CTS 1 one could further reduce the uncertainty. To reduce remaining uncertainty about inactivation kinetics of environmental pathogens pilot research is needed. CTS 1 was the only system where super-chlorination was applied.

4.9.2.5 CTS 1: Modelling Post-chlorination

After de-chlorination approximately 0.5 $mg.L^{-1}$ chlorine residual is present in the water, which is monitored every 5 minutes. The residence time in the completely filled 5 000 m^3 treated water reservoir is approximately 75 minutes. However, the reservoir was not

always 100% full, especially during peak demand the level dropped. Therefore a residence time of 30 minutes was applied in the CSTR model (40% full). Disinfection ranged from 1.7 log in winter to 2.5 log in summer. Approximately 0.1% of the measurements resulted in less than 1 log disinfection of bacteria.

Determining residence time distribution in the reservoir at different fill levels and monitoring reservoir levels could further reduce the uncertainty. Again monitoring temperature more frequently (daily) at CTS 1 would also reduce uncertainty. To reduce remaining uncertainty about inactivation kinetics of environmental pathogens pilot research is needed.

Most CTSs applied post-disinfection (CTS 1, 3, 4, 5, 6, 8, 10, 11, 12) and these all provided data on disinfectant concentration monitored daily to every minute. None of the sites provided records of reservoir levels, thus leaving uncertainty in the assessment.

Apart from the contact time in the reservoir, a significant amount of disinfection can take place during transport and distribution. This was regarded part of distribution and was not assessed.

4.9.2.5 CTS 1 Improved treatment assessment results

Combining the described PDFs for each treatment step in a Monte Carlo analysis assessed the total treatment. Table 4.27 show the results of this improved assessment for bacteria, compared to the point estimate results. The estimated over-all log reduction is very similar for both approaches. The 90% confidence interval is much smaller using local data. The largest difference is in the minimum log reduction in 10.000 simulations. In the basic assessment, relatively low removal of bacteria of 6.6 log could still occur, although not very frequent. The local information actually indicated that this very low reduction is very unlikely, and that 9.5 log reduction is always maintained, thus reaching the health targets. Analyzing micro-biological data would then not be necessary but has been included for completeness below.

Table 4.27 Results of the improved treatment assessment of bacteria reduction at CTS 1 using surrogate monitoring and process modelling.

	Triangular PDF from literature	**Improved PDF surrogates/modelling**
Over-all log reduction	10.2	11.8
p5	11.8	11.3
p50	16.6	13.2
p95	20.6	15.0
Minimal log reduction in 10.000 simulations	6.6	9.5

4.9.3 CTS 1: Utilization of micro-biological data

The results of microbiological samples represent the actual reduction by treatment. Since they are generally monitored less frequently than surrogates or process conditions, they provide less detailed information on variation. Issues concerning

recovery of methods, (human) infectivity and the translation of indicators reduction to pathogens require more advanced statistical approaches to construct PDFs from this data. The statistical methods are described Chapter 7. This paragraph describes how the micro biological data from CTS 1 was used to improve the treatment assessment. Individual processes were assessed based on available microbiological data.

4.9.3.1 CTS 1: Indicator inactivation by pre-oxidation

The indicator organisms *E. coli* was measured (twice) monthly before and after pre-oxidation resulting in 122 and 59 records respectively. Results for *E. coli* are presented in Figure 4.17. *E. coli* concentrations in raw water varied between 100 and 10,000 MPN.100mL^{-1}. After pre-oxidation the concentration varied between 1 and 100 MPN.100 mL^{-1}, so approximately 2 log inactivation of *E. coli* was achieved. Breakthrough occured on two dates in January and February 2004 when the post pre-ozonation *E. coli* concentration showed peaks up to raw water concentrations. This illustrated that the process was susceptible to breakthrough. The main goal of pre-oxidation at CTS 1 was improvement of coagulation and not directed at inactivation. Nonetheless, the analysis shows that pre-oxidation does have potential to provide direct inactivation. Section 4.9.4.3 discusses the need to use this potential to reach health based targets.

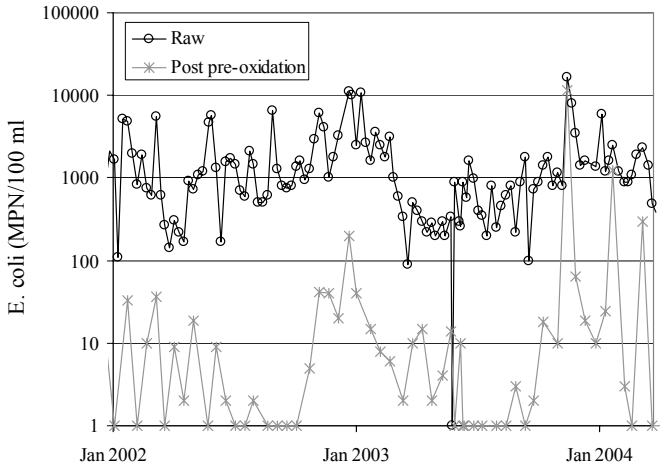


Figure 4.17 *E. coli* monitoring before and after pre-ozonation generally show 2 log inactivation of *E. coli*, but some events of no inactivation occur.

When measurements before and after pre-oxidation were paired by date the resulting dataset of *E. coli* inactivation, presented in Figure 4.18, shows how frequently a level of inactivation is reached. Inactivation of *E. coli* was generally 2 to 3.5 log. This is slightly higher than the inactivation assessed by modelling (1.5-2 log, Section 4.9.2.1). This suggests that the model may be too conservative.
The inactivation of bacteria was described by a beta distributed PDF. The parameters of the beta distribution were estimated from the measured *E. coli* concentrations using the Bayesian MCMC framework described in Chapter 7.

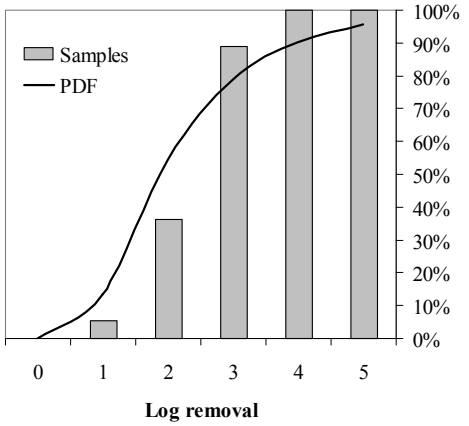


Figure 4.18 Cumulative frequency distribution and CDF (Cumulative probability Density Function) of *E. coli* inactivation by pre-oxidation at CTS 1

4.9.3.2 CTS 1: Indicator removal by conventional treatment

Indicator organisms were measured historically (twice) monthly before and after coagulation-sedimentation and filtration and monthly during the monitoring program. Four of the *E. coli* samples after filtration (12%) were positive showing 1-1.3 log removal. Seven of the negative samples after filtration indicated 1.3 log up to 3 log removal. Both surrogate and indicator monitoring indicate the same level of removal of bacteria (1-3 log). The 11 removal values do not provide additional information about treatment variation, therefore the triangular distribution determined from surrogates (Section 4.9.2.2) is maintained for removal of bacteria. In order to refine this PDF, substantial monitoring of filtered water would be required, preferably in larger sample volumes.

Some 38% of the samples after conventional treatment were positive for *Clostridium perfringens* at CTS 1. Only 4 positive samples and 13 negative samples out of 29 could be paired with samples before conventional treatment. All 13 negative samples indicated more removal than the positive samples. Figure 4.19 shows the frequency distribution. Removal of *Clostridium perfringens* was regarded a model for protozoan (*Cryptosporidium* and *Giardia*) removal, and resulted in 1.4-3.2 and 2.1-3.4 log removal estimates for *Cryptosporidium* and *Giardia* respectively (Section 4.9.2.2). The only exception (or possible event) was a single record of over 100 CFU.100 mL^{-1} of *Clostridium perfringens* after conventional treatment. This indicated that conventional treatment may be susceptible to events but the data is insufficient to estimate frequency, duration and magnitude of such an event. The triangular PDF of protozoan removal from surrogate monitoring (Table 4.25) was used in the treatment assessment.

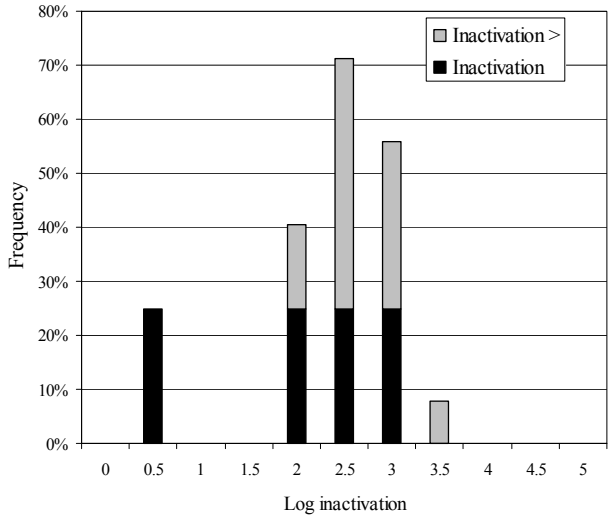


Figure 4.19 Frequency distribution of *Clostridium perfringens* removal by conventional treatment

More intensive monitoring is required to better assess the possibility of breakthrough of the conventional treatment, since the applied PDF may be too optimistic. Other CTSs generally did not monitor indicators after the first clarification steps, apart from CTS 2, which is discussed in Chapter 7.

4.9.3.3 CTS 1: Indicator inactivation by inter-ozonation

Indicator organisms were measured historically (twice) monthly before and after ozonation. No sample after ozonation was positive for *E. coli*, indicating that over-all inactivation of *E. coli* was approximately 2.7 logs. About 38% of the samples before and 24% of the samples after ozonation were positive for *Clostridium perfringens*. Most positive samples could not be paired so the dataset was not analysed as described above, but could have been modelled based on the fraction removed (Chapter 7). Due to a single 100 $CFU.L^{-1}$ result, an over-all inactivation of *C. perfringens* spores of 0.9 log was indicated. These observations support estimates of inactivation efficacy based on modelling (Section 4.9.2.3), but provide no additional information on the level and variation of the inactivation by ozonation.

4.9.3.4 CTS 1: Indicator removal by GAC filtration

Indicator organisms were measured historically (twice) monthly before and after GAC filtration. No *E. coli* was found before or after GAC. One sample after GAC (6.25%) was positive for *Clostridium perfringens*. So the number of data that could be paired was insufficient to provide such a PDF of removal. Approximately 0.9 log over-all removal of *C. perfringens* spores by GAC was shown by the data. The *Clostridium* removal was similar to the MEC reported in Table 4.3. This spore reduction was more optimistic than the turbidity assessment of 'poor' removal of *Cryptosporidium* and *Giardia*. However, the data was so scarce it provided too little information on the variation of removal, so the triangular PDF based on turbidity (Table 4.26) was applied in the treatment assessment.

4.9.3.5 CTS1: Indicator inactivation by Super-chlorination and de-chlorination

All indicator monitoring was negative at this point in treatment, therefore no assessment of indicator inactivation is possible for these treatment steps.

4.9.4 CTS 1: Total treatment assessment results

4.9.4.1 Results treatment assessment

At CTS 1 the micro-biological data that was available had little influence on the PDFs of the improved assessment. Only the PDF for the first treatment step of pre-ozonation was changed based on the *E. coli* monitoring data. The results of the total assessment including all available data in Table 4.28 are very similar to the improved assessment.

Table 4.28 Results of the total treatment assessment of bacteria reduction at CTS 1 using all available data.

	Triangular PDF from literature	**Improved PDF surrogates/modelling**	**Best PDF all data**
Over-all log reduction	10.2	11.8	11.8
p5	11.8	11.3	11.2
p50	16.6	13.2	12.9
p95	20.6	15.0	16.1
Minimal log reduction in 10.000 simulations	6.6	9.5	9.7

4.9.4.2 Comparing results to health basted performance targets

The source water for CTS 1 was a heavily polluted river, for which *Campylobacter* concentrations of 100 $CFU.L^{-1}$ were likely (see Chapter 3). Following the WHO example of a health based target at 10^{-6} DALY (see Section 4.1) a *Campylobacter* removal performance target of 5.9 log inactivation was required. The results in Table 4.28 verify that the treatment potentially achieved over 9.7 log reduction of bacteria in the assessed period. Since sufficient reduction was verified already using surrogates and process monitoring, the extra monitoring of micro-organisms that was performed for this assessment was not required to verify *Campylobacter* reduction. Using only triangular distributions based on literature, rare occasions of non-compliance to the health based performance target were already unlikely to occur (6.6 log reduction during 1/10,000 of time). The surrogate and process monitoring was valuable to verify that the minimum reduction actually was much higher than required by the health based performance target. Some suggestions on how to further improve the surrogate and process monitoring have been provided.

Only a basic assessment using point estimates and triangular distributions was presented for other pathogens. For *Cryptosporidium* with an assumed source water concentration of 10 oocysts per litre a performance target of 4.2 log units can be derived from Figure 4.1. The over-all reduction of 4.8 log (Table 4.28) was just sufficient to reach the yearly target. Reduction was sufficient 95% of the time (p5 = 4.2) but on rare events (1/10,000) it may be as low as 2.7 log. An improved assessment for *Cryptosporidium* would result in less removal by conventional treatment and GAC filtration, same as for bacteria. The disinfection steps were unlikely to result in significant *Cryptosporidium* reduction. As a result *Cryptosporidium* reduction might be

critical at CTS 1. Depending on the interpretation of the assessment results by the health inspectorate, additional data collection, improving operation of current treatment or even additional treatment could be necessary. When the hydraulic characteristics of the ozonation processes actually better resemble plug-flow, this would lead to a substantially higher level of *Cryptosporidium* inactivation. Applying a higher ozone dose would also lead to more inactivation. Monitoring conventional treatment with frequent (daily to weekly) clostridial spore analysis in sufficiently large volumes (1 L) could verify that the treatment is currently providing more removal than expected from the turbidity measurements. Improving the performance of conventional treatment could improve the actual *Cryptosporidium* reduction. Optimization could be studied in a pilot plant.

A (Rota)virus reduction in the order of 5.5 log units may be required based on the example in Figure 4.1 at a source water concentration of 10 viral particles.L^{-1}. Over-all virus removal appears to be sufficient based on the basic assessment, although rare occasions (1/10,000) of low reduction (4.4 log) might occur. Viruses, like bacteria, are susceptible to disinfection processes. Modelling these processes for bacteria showed that they perform consistently well, and that the basic assessment is too conservative. It is expected that an improved assessment for viruses will verify that current treatment performance is sufficient to reach health based targets.

4.9.4.3 Setting of critical limits and corrective actions

Each treatment process at CTS 1 contributes to the reduction of one or more pathogens, and thus can be considered as a control point. For each treatment process, critical limits can be defined to either set the proper conditions for the process and/or to verify that a process is working properly.

The goal of the pre-ozonation is improvement of the conventional treatment, and is primarily operated for that cause. The rest of the treatment sufficiently reduces bacteria and viruses under base-line conditions but *Cryptosporidium* inactivation by pre-ozonation could be enhanced. During events, like failure of the other disinfection steps, the ozone dose at pre-ozonation can be increased as a corrective action to maintain sufficient disinfection.

Conventional treatment could potentially provide more *Cryptosporidium* removal. Optimizing the coagulation-sedimentation process with jar tests and applying filter to waste could be considered. These would lead to new set points for operation and critical limits for turbidity.

The disinfection processes at CTS 1 (inter-ozonation, super-chlorination and de-chlorination) are currently operated at a fixed disinfectant concentration. By measuring this concentration at the contactor outlet, proper functioning of the dosing equipment and control loops are currently verified. This could be improved by varying the set-point for the disinfectant concentration with changing process conditions, like temperature and flow, to provide sufficient inactivation of target pathogens. When the source water is sufficiently characterized, the target level of disinfection could be

adapted to address the variations in source water. Thus process operation can react to seasonality and peak events with sufficient disinfection and without substantial over-dosing.

4.9.4.4 Monitoring plan

Some possible improvements of monitoring were suggested in Sections 4.9.2 and 4.9.3. In general monitoring of indicator organisms is only useful for risk assessment after the first treatment steps when most samples are still positive. Each step in the assessment needs to be monitored separately (inflow and outflow) in order to translate the indicator reduction to pathogens. Indicator monitoring needs to be applied frequently (daily to weekly) to provide sufficient information on variation and limit statistical uncertainty. In the example the on-line monitoring of surrogates and process conditions proved to be more effective to verify treatment efficacy than micro-biological monitoring. Some additional monitoring of reservoir levels and flow would further refine this assessment. Determining flow conditions for the disinfection processes would greatly improve the interpretation of the monitored process conditions.

4.10 DISCUSSION AND CONCLUSIONS

4.10.1 General findings in CTS treatment assessments

Within the MicroRisk project twelve treatment system throughout Europe and including Australia were assessed as a pilot for QMRA. Some general lessons were learned from this. Microbiological data that was collected according to current practice proved to be insufficient to quantify treatment efficacy without large uncertainty. End-product testing generally resulted in close to 100% non-detects. This approach only provided an estimate of minimal pathogen reduction by treatment. The reduction that could thus be indicated was generally insufficient to provide safe drinking water. Microbiological samples after the first treatment steps provided additional information on the efficacy of these steps. These were almost in the same order of magnitude as those found for the total treatment. Additional treatment steps, such as disinfection, were quantified by various indirect means. Process models used measured disinfectant residuals, temperature, hydraulic characteristics of contact chambers and inactivation kinetics from literature to calculate inactivation of different pathogens at full-scale. Physical removal processes were quantified based on reported efficacy in literature and monitored removal of surrogates such as turbidity or particles. Combining the results of the different treatment steps in a stochastic model led to a decrease in uncertainty related to pathogen reduction by water treatment. Further, the results led to a change in focus on what types of data need to be collected for quantitative assessment of pathogen reduction by treatment. Full-scale hydraulics plays a major role in disinfection and is the main cause of uncertainty in disinfection processes. Recommendations for improvement of treatment configuration or operation could be made while providing a quantified estimate of the effect of such an improvement on microbiological safety.

4.10.2 Conclusions and recommendations

Despite the level of data available on treatment, there is always a degree of uncertainty on the efficacy of pathogen reduction. The amount of relevant data available for a treatment system will have a large impact on the certainty that can be reached. Still some uncertainty will remain in the outcome of the treatment assessment. The impact of the uncertainty on the performance of the treatment component of a full risk assessment model (Chapter 7) therefore also needs to be assessed. In the end a best estimate with confidence intervals is required to apply legislation, and to focus attention on where to best manage water treatment.

The intention of the treatment work package in the MicroRisk project was to provide a protocol on how to assess treatment. Working through the CTSs it became clear that each system is so specific that a generally applicable protocol is not possible at this stage. Therefore the presented approach was applied to provide guidelines on how to assess a treatment in a systematic way. When going through this in cycles of data collection, treatment assessment and adapted monitoring the assessment will reach a satisfactory level of verified water safety. A guideline for the first steps is:

1 Collect all relevant data that is available for the treatment studied and assess the treatment as presented in this chapter (first iteration).
2 Collect additional data by monitoring or pilot experiments. From the CTSs in MicroRisk the following prioritisation was suggested:
   - perform additional pathogen sampling to determine base-line and peak (event) pathogen concentrations in the source to better establish health based performance targets;
   - monitor relevant indicators frequently (daily to weekly) after the first (physical) treatment steps at the point where more than 50% of the sample are positive;
   - use on-line measurements, such as turbidity or particle monitoring for physical processes to verify that events are rare (and their duration);
   - monitor disinfectant residual, reservoir levels, flow and temperature on-line to model disinfection processes; and
   - improve hydraulic characterization of these disinfection processes by tracer test or CFD modelling to refine these models.
3 When uncertainty remains too large, more drastic measures like large volume sampling, pilot studies or full-scale studies with model micro-organisms should be considered.

Recommendations for scientific research include:

- Setting up of a common database for treatment efficacy (along the lines of Hijnen *et al.* [2005a]) to keep up to date removal credits and inactivation rate constants accessible;
- Improved disinfection modelling to verify performance of disinfection processes since micro-biological sampling at the last stages of treatment provides minimal quantitative data. Knowledge about disinfection kinetics of environmental pathogens is still limited and more research on topics like kinetic parameters and tailing is required;

- The applicability of surrogates and indicator organisms is still inconclusive and requires more research under full-scale conditions; and
- Improve detection and recovery techniques for pathogens.

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5. Contamination during distribution

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The integrity of the reservoirs and mains in the distribution network is critical for the safety of the drinking-water, as is hygiene during invasive operations. Especially in the case of the buried mains, it is difficult for water companies, inspectorates and regulators to verify whether the efforts of safeguarding water safety are sufficient. Although the health impact of reported waterborne outbreaks usually is well-known, the *potential* health impact of the more frequently occurring non-outbreak contamination events is not. The purpose of this chapter is to provide and demonstrate a method of estimating the probability of faecal contamination of distributed water and the pathogen concentrations in faecally contaminated water. The method is a part of the total set of instruments of Quantitative Microbial Risk Assessment (in line with Haas et al, 1999a) that can be used to assess the health risk of microbial contamination of drinking-water. The method is applicable to all secondary faecal contaminations, i.e. faecal contaminations that are not originating from the source water (Chapter 3) and insufficient elimination of pathogens therein (Chapter 4).
Quantitative assessment of the probability and severity of exposure of tap water consumers to pathogens that entered during secondary faecal contaminations requires information about:

1. The probability (= incidence or frequency of occurrence) of contamination events.
2. The duration of these events.
3. The severity of the contamination: the resulting pathogen concentrations in tap water.

Paragraphs 5.1 through 5.2 describe the experienced health risks of outbreaks and possible health risks of non-outbreak contamination events, respectively. Paragraph 5.3 gives an overview of variables that could be monitored to detect contaminations. Paragraph 5.4 explains why the detection and concentration of the bacterium *Escherichia coli (E. coli)*[1] in distributed water was chosen as a basis for estimating the probability and severity of faecal contamination events. It further describes how the detected *E. coli* concentrations can be used to estimate pathogen concentrations by assuming pathogen to *E. coli* ratios that were found in possible contamination sources. In paragraph 5.5 and 5.6 the method is demonstrated in estimating pathogen concentrations during outbreaks and non-outbreak events respectively. Paragraph 5.7 demonstrates that most occasional detections of *E. coli* in drinking-water are likely to be indicating contamination events, as opposed to representing the upper levels of baseline concentrations. In paragraph 5.8 this conclusion is a rationale for evaluating faecal contamination events represented by detectable *E. coli* concentrations determined during periodical monitoring of drinking-water and monitoring after operations. Paragraph 5.9 discusses the high sensitivity of the method of estimating pathogen concentrations described in this chapter, to the variation of available data and the uncertainties leading to inaccuracy

[1] This chapter describes a method for evaluating health risks based on past and future *E. coli* data. Until the beginning of the 21st century however, many water companies in Europe and abroad used an analysis method that determines the presence of *E. coli* as well as other thermotolerant coliforms (abbreviated as coli44, also named faecal coliforms). Many of the data used in demonstrating the method are concentrations of thermotolerant coliforms, possibly inclusing other bacteria, such as *Klebsiella* spp. As the *E. coli* method is the new European standard, *E. coli* occurrence and concentrations are the standard data source in this chapter.

(systematic errors). Conclusions regarding the method and recommendations for water companies are provided in paragraph 5.10.

5.1 OUTBREAKS ASSOCIATED WITH CONTAMINATION EVENTS IN DISTRIBUTION SYSTEMS

There have been many reports of waterborne outbreaks through drinking-water that is contaminated within the distribution system.

In chapter 1, outbreaks through public water supplies in Europe, from 1990-2004, are reviewed and analysed in the fault tree analysis. 86 outbreaks were reported, with a total of 72,546 cases, of which 341 were hospitalised and 1 died. In 33% of these outbreaks, contamination during distribution was the dominant cause of the outbreak.
The outbreaks have provided information about the problems and events leading to contamination of drinking-water in the distribution system. The fault tree analysis showed that events that have contributed to outbreaks through contamination of distributed water were:

- cross connections/backflow
- construction or repair
- damaged/old mains
- low pressure
- cleaning
- reservoir contamination

Bartram *et al.* (2002) evaluated waterborne outbreaks in Europe from 1986-1996. They found that 55 of these were related to networked public supplies and 36 to private or standpipe supplies. The contribution of contamination within the distribution network was not reported. Hunter (1997) reported that 15 of the 57 outbreaks in public water supplies in the UK between 1911 and 1995 were associated with contamination within the distribution. In the Nordic countries, 18-20% of the outbreaks through drinking-water between 1975 and 1991 were associated with cross connections, both in community and private supplies (Stenström, 1994).

In the USA, 18% of 619 outbreaks reported in public water systems from 1971 to 1998 were caused by chemical or microbial contaminants entering the distribution system or water that was corrosive to plumbing systems within premises (Craun & Calderon, 2001). From 1991-2002, 23 of the 58 outbreaks through drinking-water in community supplies were related to a contamination in the distribution system or in household plumbing. When related to all drinking-waterborne outbreaks (including non-community and private systems), contamination during distribution accounted for 22% (15-32%) of the outbreaks. In the period 1920-1990, this was 11-18% (Craun, 1986, Craun & Calderon, 1999).

From the review of outbreaks through drinking-water of Hrudey & Hrudey (2004) it is clear that in many distribution-related outbreaks, lack of or non-compliance to adequate hygiene procedures to maintain the integrity of the network or to ensure safety during and after breaks and repairs have led to gross contamination of mains water which resulted in people falling ill and even to fatalities, such as in the Cabool and Gideon outbreaks in Missouri, USA.

Hrudey & Hrudey state: “many of the most troubling cases have revealed no effort whatsoever at assuring distribution system integrity”. In other cases, outbreaks resulted from cross-connections or open connections with contamination sources. The outbreak review of Hrudey and Hrudey contains very illustrative information about errors and events that have lead to outbreaks and the reader is referred to this review.

5.2 NON-OUTBREAK CONTAMINATION EVENTS

Outbreaks show the tip of the iceberg. Many smaller contamination events are likely to occur. These events may even lead to illness in the community supplied, without a link being made to the water system. Evidence that contamination events occur much more frequently than outbreaks is provided by the statutory monitoring of drinking-water for *E. coli* (formerly also determined as thermotolerant coliforms). Bartram *et al.* (2002) evaluated the results of monitoring of thermotolerant coliforms in drinking-water samples in European countries and suggested that, on average, the percentage of samples showing the presence of thermotolerant coliforms in drinking-water from public systems is around 1-2% (range 0-12%). Although these levels appear high compared to other studies (Van der Kooij *et al.*, 2003, Mendez *et al.,* 2004, Van Lieverloo *et al,* 2006, this study), most studies show that faecal contamination (as suggested by *E. coli* detection) is more frequent than outbreaks would suggest. Mendez et al. (2004) showed that other indicators of faecal contamination (*Clostridium* spores, somatic coliphages, F-RNA phages and *Bacteroides fragilis* phages) may be present in (chlorinated) tap water in which no *E. coli* is detected. Several outbreaks of viral and protozoal illness occurred through water that met the *E. coli* standard of absence in 100 ml (Craun & Calderon*,* 2001; Anderson & Bohan, 2001). *E. coli* is more sensitive to chlorine than viral and protozoal pathogens (Chapter 4, paragraph 4.2.5). So especially in chlorinated tap water, the frequency of *E. coli* detection is likely to underestimate the frequency of faecal contamination.

Event reports of water companies also show that contamination events are not as rare as outbreaks. Van Lieverloo *et al.* (2003) evaluated contamination events reported by 8 water utilities in the Netherlands in 1995-2000. In 9 of the 27 events reported, thermotolerant coliforms or *E. coli* were detected in drinking-water on several occasions. This was considered to be a lower estimate as the water companies had not documented all events. No outbreaks were reported in the same period. Of these events, 5 were associated with a contamination in the distribution network due to cross-connection, open connection and mains breaks and 3 with a leaking reservoir.

Other studies suggest that contamination may occur during standard operating conditions. LeChevallier *et al.,* 2003 studied the impact of transient pressure events in distribution networks. Negative pressure events occur due to power failures or other sudden pump shutdowns. They have shown that i) these events occur in practice, ii) during these events, leaks provide a portal of entry for groundwater to enter the distributions system and iii) faecal indicators and human viruses may be present in the groundwater surrounding drinking-water mains. They could not determine if this contamination route may lead to significant contamination of drinking-water, because of insufficient data. Negative pressure events were usually short (less than 1 minute) and outside the transient events leaks result in an outflow of uncontaminated drinking-water of the water mains. The level of contamination of water

entering the distribution network during short negative pressure events is therefore difficult to assess.

A recent case control study on sporadic cryptosporidiosis in the UK reported an association between gastro-intestinal illness and the loss of water pressure in the distribution network (Hunter *et al.,* 2005). 28 of 423 controls reported diarrhoea in the two weeks before the questionnaire. Analysis of the risk factors showed a strong association with the loss of water pressure at the household tap. Most of these pressure-losses were associated with reported events in the distribution network, such as a burst of water mains. They suggest that failures in the distribution network could have a significant contribution (around 15%) to the overall rate of gastro-enteritis in the population.

5.3 DETECTING CONTAMINATION EVENTS

5.3.1 Indicators of faecal contamination

The presence of bacteria indicatove of faecal contamination is a very powerful indication of the possible presence of faecal pathogens and therefore of a risk to public health. Only small sample volumes (100 ml) are tested, resulting in high detection limits.
Escherichia coli (*E. coli*) and faecal enterococci (Köster et al. 2003) are embedded in the EU Drinking-water Directive (The Council of the European Union, 1998) and statutory monitoring programmes for *E. coli* in distributed water exist in all Member States. Since it is impossible to detect all contamination events, it is important to know the sensitivity of the *E. coli* monitoring program. A preliminary simulation study of contamination events of nine different mains in a small city distribution system ($15 \cdot 10^{-6}$ l/day) with 16 litre (l l/hr) of untreated sewage ($1 \cdot 10^{8}$ CFP/l *E. coli*, from Medema et al., 2001) revealed that with the current statutory *E. coli* monitoring program, the probability of detection varied between 0 and 15% with a mean of 5% (Van Lieverloo et al., in prep.). These results suggest that high percentages of contamination events may go unnoticed.

5.3.2 Indirect indicators of (possible) contaminations

Although *E. coli* and other faecal indicators give an indication of the occurrence of a contamination, changes in several other variables may be indicative of contaminations as well (table 5.1), especially when deviating values of multiple variables coincide. A sudden drop in disinfectant residual or increase in turbidity may be such an indication, as well as low pressure. Most outbreaks are detected only after the incidence of gastroenteritis has increased (Hrudey & Hrudey, 2004). Several events and outbreaks have been detected as a result of consumer complaints concerning taste (Huisman and Nobel, 1981; Fogarty et al, 1995; Fernandes et al., accepted). These complaints should always be taken seriously, as the consumers are currently the most sensitive and rapid monitoring system of contamination events.
Small contaminations are not likely to be noticed by consumers, however, neither epidemiologically (illness), nor aesthetically (taste, colour, turbidity). Furthermore, in the

absence of changes of other variables, small increases of the number of consumer complaints usually will not cause water companies to look for causes, as the frequency and nature of consumer complaints are also subject to factors other than water quality or quantity.

Table 5.1 Variable changes theoretically able to indicate (possible) contaminations. The quality of the indicators highly depends on the levels and variation during standard operating conditions and can vary from supply zone to supply zone.

Indicator	Leaks / breaks[a]	Backflow / backpressure	Cross-connection	Hygiene (operations)
Organisms[b] ↑[c]	+ / +	+ / +	+	+
Disinfectant ↓[c]	+ / +	+ / +	+	+
Turbidity ↑	- / +	+ / +	-	-
Flow ↑	- / +	- / -	-	
Pressure ↓	+ / +	+ / -	-	
Leakage ↑↓	+ / + (↑)	- / - (↓)	+ (↓)	
Complaints ↑				
- Taste/odour	+ / +	+ / +	+	+
- Turbidity/colour	- / +	- / -	-	-

[a] + = relatively good indicator; - = relatively poor indicator
[b] Particularly concentrations of species that can not multiply in drinking-water systems, e.g. *E. coli*, enterococci, *C. perfringens* and faecal bacteriophages.
[c] ↑ and ↓ = increase and decrease respectively of indicator value indicates (possible) contamination.

Contaminations may go unnoticed, since:
- It is virtual impossible to continuously monitor relatively good indicators of contamination in all parts of distribution systems.
- Indicators that can be monitored continuously are poor (variation of the value due to contamination is low compared to variation caused by other factors).
- A combined real-time monitoring of multiple variables would be required to notice changes indicative of a possible contaminations.

In the rest of this paragraph, common causes of contamination and the possibilities of detecting these contaminations are summarised. In all these cases, monitoring of failures (infrastructure, operations) that might lead to contaminations are far more effective in safeguarding drinking-water safety than monitoring the effects of contaminations. In many cases however, monitoring effects is far more cost-effective.

5.3.3 Cross-connections

The only feasible moment for visually detecting buried cross-connections is during or just after construction. Visual inspections of mains and pipes in premises however can result in detection of cross-connections. Calibration of flow and pressure models of distribution systems may also reveal their presence.

5.3.4 Backflow and backpressure

Periodic inspection of the presence and testing of the performance of backflow prevention devices is essential in preventing contaminations, especially when activities or infrastructure in the connected premises harbour risk for public health, such as slaughter-houses, farms, laboratories, hospitals etc. High-risk connections should be interrupted with a break-tank. In uninterrupted medium-risk connections, such as farms, inspection of the piping and installations (such as pumps, drains and taps) is necessary to detect risks of backpressure. To detect backflow or backpressure in uninterrupted low-risk connections (e.g. homes) however, only installing online flow detection devices (e.g. in the water meter) is feasible.

5.3.5 Backflow via overflow or drainage pipes of distribution reservoirs

Rainwater pipes of water towers often are connected to overflow or drainage pipes. When these become clogged, e.g. by leaves and twigs, faecally contaminated rainwater may flow back into the drinking-water reservoir. These constructions should either be changed or be regularly inspected for clogging.
Overflow pipes of lower distribution reservoirs often are only protected from the environment by a water seal, a rat screen and/or a hatch. Floods may cause backflow into the reservoir. Damage to the screen or the hatch may allow rats and other animals to enter and an empty water seal may allow insects to enter the reservoir.
The residence time of drinking-water in distribution reservoirs often is relatively high, especially in suppletion reservoirs. A contamination event may be detectable here, longer than in trunk mains and distribution mains (Van Lieverloo et al. in prep). Reservoirs are therefore suitable as sampling sites for the presence of *E. coli* and other faecal indicators.

5.3.5 Leaks and breaks

Mains breaks and larger leaks usually are detected when flooding of streets is reported. If water companies respond rapidly to these reports and work hygienically during repairs, contaminations can be prevented. Small leaks sometimes may pose a larger threat, since they are not easily detected. In combination with a pressure drop or loss, leaks can allow ingress of (possibly) contaminated soil and water (LeChevallier et al, 2003).
There is no overpressure in distribution reservoirs, so leaks in reservoir covers or in walls below groundwater levels may cause a (faecal) contamination. Leak detection (and repair) programs therefore are a major part of risk management.

5.3.6 Drop or loss of pressure

Infrastructural integrity is the first line of defence against secondary contamination of distribution systems. When there is a breach in this line, either by a cross-connection, missing backflow prevention devices or by leaks, overpressure is the remaining safeguard in mains. Low or negative pressure transients (LeChevallier et al, 2003) and periods of loss of pressure may cause ingress of soil and water from the surroundings of leaks. When the integrity of the mains can not be sufficiently ensured, events of low pressure or loss of pressure need to be monitored and responded to. These events may be detected by monitoring pump operation, mains pressure, but also by reporting intentional drops and losses of pressure or changes of flow due to valve and hydrant operations.

5.3.7 Drop or loss of disinfectant residual

The presence of a disinfectant residual in many countries is considered the last line of defence against contaminations (Haas, 1999b, LeChevallier, 1999). These residuals are known to be ineffective in case of larger contaminations and when resistant pathogens such as *Cryptosporidium* are present (Chapter 4, paragraph 4.2.5). Drops in disinfectant residual concentrations due to failures may cause disinfection to be inadequate to control small contamination events. Failure of disinfectant dosing should therefore be monitored, as well as the actual disinfectant residuals. A drop in disinfectant residuals can however also indicate the occurrence of a contamination.

In a number of countries in the European Union (e.g. Denmark, Germany, the Netherlands), however, both water companies as well as regulators strive for maintaining microbial safety without the need for the maintenance of a disinfectant residual. Many of these water companies achieve this goal, a.o. by maintaining relatively low leakage rates (< 5%) and following hygiene procedures during operations. This results in a high appreciation of the taste and odour by the consumer and in absence or low concentrations of disinfection by-products (Van der Kooij *et al*., 1999, Hambsch 1999, Van Lieverloo *et al*., 2006).

5.4 CALCULATING THE PATHOGEN CONCENTRATION DURING CONTAMINATION EVENTS

5.4.1 Pathogen concentration in tap water

In order to assess the effect of a contamination event on public health, ideally the concentrations of pathogens present in drinking-water should be known. After many outbreaks, drinking-water water is tested for the presence of pathogens (Hrudey & Hrudey, 2004). This is not common practice for contamination events, however, as quantification of pathogen concentrations is expensive and detection limits are high compared to acceptable concentrations. Furthermore, in most contamination events the signs of faecal contamination may quickly disappear. In most cases, *E. coli* is no longer detectable in the required repeat usually taken the following day. In the absence of quantitative data to assess the effect of

detected faecal contamination of drinking-water, pathogen concentrations must be estimated per case of contamination. Westrell *et al.* (2003) assumed effects for each recorded failure in a treatment plant and the distribution system of the city of Gothenburg, Sweden. Failures in the distribution system were derived from the incidence reports between 1980 – 2000 and personnel interviews. When cross-connections with pressurised sewage pipes were detected as the cause of the contamination, the pathogen concentrations in drinking-water were deduced from pathogen data from sewage and the dilution of sewage in drinking-water. For contamination events in the periphery of the distribution system or in reservoirs, no information of the contamination source was available. Therefore the coliform concentrations detected in drinking-water during the event were used and related to the coliform concentrations in sewage to calculate the level of sewage contamination. In addition, the pathogen concentration in sewage was used to calculate their subsequent pathogen concentrations in drinking-water during the contamination event. The events in the periphery and reservoirs were caused by leakage through cracks in concrete reservoir walls or through damage during maintenance of the network. The pathogen concentrations in the drinking-water were translated to a risk of infection to the exposed consumers, taking the size of the affected areas and the duration of the contamination event into account. The resulting annual risk of infection from contamination events in the distribution system was found to be lower than the risks resulting from normal operation of the Gothenburg system (Figure 5.1).

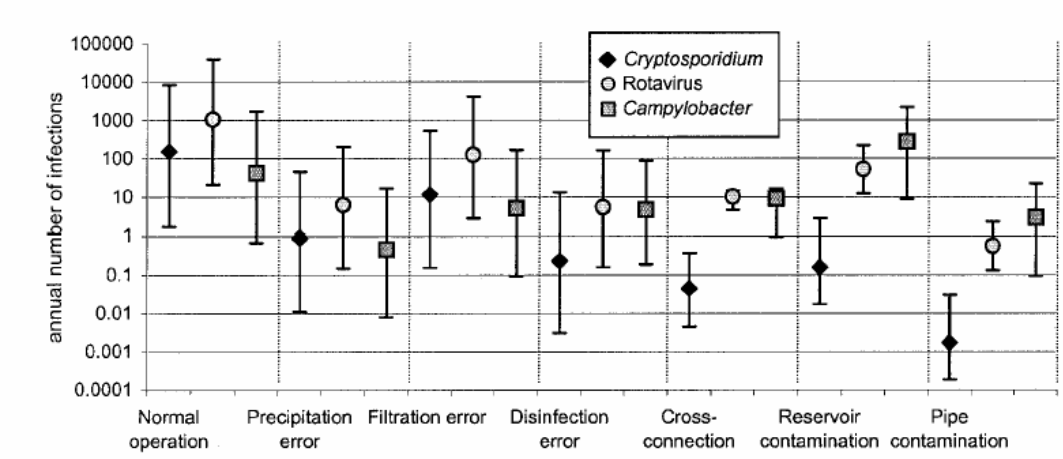


Figure 5.1 Estimated median annual infections during normal operation and during failures in treatment and distribution in a water supply area in Gothenburg (population 250.000) with 95% confidence interval (after Westrell *et al.,* 2003)

In this chapter, the approach of Westrell *et al.* (2003) is extended by using *Escherichia coli*[2] concentrations determined during standard operating conditions and after operations (mostly repairs) as an index of the severity of the contamination. The *E. coli* concentration detected in the drinking-water was translated to pathogen concentrations using the ratio of *E. coli* to pathogens in potential contamination sources (such as sewage). *E. coli* is chosen as most pathogens that have caused outbreaks are of faecal origin and multiplication of *E. coli* is almost exclusively limited to the intestines of humans and warm-blooded animals (Ashbolt et

[2] As stated earlier in this chapter, *E. coli* concentrations until the beginning of the 21st century determined together with other thermotolerant coliforms. As this guidance document is targeted at future evaluations and to improve readability, in most cases, only *E. coli* will be referred to.

al. 2001). Evidently, there are drawbacks of using *E. coli* concentrations as an index of pathogen concentrations. Due to differences in survival of *E. coli* and pathogens (especially viruses and protozoans), pathogen to *E. coli* ratios vary and though the presence of *E. coli* is an indication of faecal contamination, the absence of *E. coli* is not an indication of the absence of a faecal contamination (Ashbolt *et al.* 2001)

However, the concentrations of this variable is more likely to correlate quantitatively with pathogen concentrations than variables that have similar sensitivities to variation and uncertainty and also change as a result of occurrences other than faecal contamination (Table 5.1 in paragraph 5.3.2). For this reason, disinfectant concentrations (when present at all), pressure, leakage, turbidity or consumer complaints are less well applicable. Furthermore, concentrations of *E. coli* are most ubiquitously present in the monitoring databases of water companies in the European Union and in other parts of the world.

5.4.2 Calculating pathogen concentrations from *E. coli* concentrations

The first step in the calculation of the infection risk to consumers is to estimate the pathogen concentrations in the drinking-water during a contamination event. In the method presented in this chapter, these concentrations are estimated by multiplying the *E. coli* concentration observed in drinking-water during the contamination event with pathogen to *E. coli* ratios in the most likely source of the contamination (formula 1).

$$P([\text{pathogen}])_{water} = P([E.\ coli])_{water} * P(\text{pathogen}/E.\ coli)_{source} \quad (1)$$

where:

P	= probability of an occurrence
$[\text{pathogen}]_{water}$	= pathogen concentration in the consumed water
$[E.\ coli]_{water}$	= *E. coli* concentration in the consumed water
$(\text{pathogen}/E.\ coli)_{source}$	= pathogen to *E. coli* ratio in the actual or presumed contamination source

5.4.3 Demonstration and evaluation of the applicability of the method

The approach first is demonstrated by estimating pathogen concentrations during a waterborne outbreak, based on concentrations of thermotolerant coliforms in drinking-water samples (paragraph 5.5). Subsequently, the approach is used to estimate the pathogen concentrations during contamination events recorded by water supply companies (paragraph 5.6) and contamination events as indicated by the statutory *E. coli* monitoring of drinking-water (paragraph 5.8). The method is evaluated in paragraph 5.9.

5.5 A WATERBORNE OUTBREAK

In the Netherlands, only three outbreaks were reported since the end of World War II. The first of these occurred in 1962, when 5 cases of typhoid fever were reported in Amsterdam, probably as a result of a contamination of a drinking-water main with sewage (Anon, 1962). The second reported outbreak occurred in 1981 in Rotterdam, when sewage and wastewater from a foreign marine vessel were pumped into the distribution system via a drinking-water supply valve for marine vessels. This event led to 609 reported cases of mainly gastroenteritis. Pathogens isolated from stool samples included *Giardia* (8%), *Campylobacter* (5%), *Entamoeba histolytica* (2.3%), and *Salmonella* (1.2%) (Huisman & Nobel, 1981). The third reported outbreak occurred more recently and is evaluated for purpose of calculating the retrospectively expected exposure to pathogens, based on the concentrations of *E. coli* (determined as thermotolerant coliforms) found in tap water samples during the outbreak.

5.5.1 The outbreak

In 2001, an outbreak of waterborne gastro-enteritis occurred in the Netherlands as a result of a cross-connection between the drinking-water distribution system and a grey water distribution system in a new residential area.

Cause

1. In the new residential area water was intended to be supplied via two parallel distribution systems, one for drinking-water and one for partially treated surface water (called 'grey water') intended for toilet flushing, supplying washing-machines and watering gardens.
2. Around April 2000, the grey water system of a new part of the residential area was connected to the drinking water system of a part finished earlier. The connection was a flexible hose intended as a temporary connection. Via this hose, the new part was filled with drinking-water from May 2001 to August 30th, 2001. The valve connecting the grey water systems of both parts of the residential areas remained closed.
3. After monitoring water quality to test the integrity of the new part of the grey water system (filled with drinking-water), ***the hose accidentally was not removed***.
4. Starting September 2001, both the grey water system as well as the drinking-water system of the new part of the residential area were taken into use, both supplying drinking-water.
5. Probably on November 20th, on a site away from the 'temporary' hose cross-connecting both systems, the grey water system of the new part was connected to the existing grey water system, already supplied with grey water. At that moment, the pressure in the drinking-water system was higher than in the grey water system. This overpressure prevented water from the grey water system to enter the drinking-water system.
6. At the end of November 2001, a gradual pressure increase in the feeding trunk main of the grey water system occurred, which was a common event. Probably on December 1st, the pressure in the grey water system grew higher than the pressure in the drinking-water system, causing grey water to enter the drinking-water system via the cross-connecting hose. Approx. 1000 households received grey water for drinking-water.

Detection, protective measures and corrective measures
Two days later, on December 3rd, the first consumer complaints about drinking-water taste were received by the water company. On December 4th samples were collected at these premises. In the evening of December 6th, a boiling advisory was issued for the c. 900 premises in the southern part of the residential area, in the morning of December 5th these were issued for c. 100 premises in the northern part. On December 6th, the cross-connection was removed. From December 7th on, no thermotolerant coliforms were found in the drinking-water samples collected. On December 9th and 12th, however, spores of sulphite-reducing clostridia were found in two samples collected, both containing 1 CFP per 100 ml, indicating that remains of the contaminated water, possibly in precipitated matter, were present in the drinking-water system. On December 17th, the boiling advisory was withdrawn.

Epidemiology
On December 6th a general practitioner, triggered by the boiling advisory, informed local health authorities of an excess of patients with gastroenteritis. Reported symptoms a.o. included diarrhoea (54% vs. 24% in the non-contaminated area), vomiting (38% vs. 21%) and nausea (52% vs. 28%). A clear dose-response relation was found between the average daily consumption per individual per household and the attack rate of households (a case being defined as at least one individual having reported symptoms of diarrhoea (three or more loose stools on 24 hours)[3]. Norovirus was considered the most likely predominant agent causing the outbreak, as high norovirus concentrations were found in the grey water in the spring of 2001 and norovirus was detected in a sample collected on December 20th.
In a retrospective cohort study, 223 of c. 1000 households in the affected area reported gastro-intestinal illness, but possibly c. 500 households experienced gastro-enteritis as a result of the contamination (Fernandes et al, accepted).

5.5.2 *E. coli* concentrations in tap water

In the two drinking-water samples taken on December 4th, total coliforms were found, later identified as *E. coli* and *Enterobacter cloacae* (both from faecal origin). The samples were taken after consumer complaints from two premises in two streets in the same area. Nine out of twelve repeat samples collected the same day contained thermotolerant coliform bacteria. Table 5.2 shows the results of the total program of sampling and analytical program. The concentrations of thermotolerant coliforms on December 4th were estimated from total coliform numbers the same day (16 and 19 CFP per 100 ml) and the ratios of thermotolerant coliforms to total coliforms the following day (4:5 and 2:12) in samples from the same street and address respectively. The concentration curve of thermotolerant coliforms is presented in Figure 5.2. The decline of *E. coli* concentrations in the drinking-water samples collected from December 4th through December 6th started before the removal of the accidental cross-connection (on December 6th). This decline is also noticeable at the two sites where the taste complaints originated and the first total coliform samples were collected. Therefore, this decline might suggest a decline in faecal contamination in the grey water flowing into in the drinking-water mains before the cross-connection was removed.

[3] An unexplained, clear dose-response relationship was found in the adjacent reference area as well. Circumstances suggested a viral cause.

Table 5.2 Number of samples collected, analysed and containing indicator bacteria after two consumer complaints about the taste of the tap water in a district of a city in the Netherlands

Sample date (2001)	No. of premises (samples 100 ml)	No. of samples containing indicator bacteria of no. of samples analysed (and maximum concentration in CFP per 100 ml)			
		Coli37[a]	Coli44[a]	SSRC[a]	FS[a]
Dec. 4	2	**2** of 2 (19)	-	-	-
Dec. 5	12	**9** of 12 (13)	**8** of 12 (5)	**1** of 2 (4)	**2** of 12 (4)
Dec. 6	28	**8** of 28 (14)	**5** of 27 (9)	**8** of 22 (5)	**2** of 22 (3)
Dec. 7	19	0 of 19	0 of 19	0 of 19	0 of 19
Dec. 8	7	0 of 7	0 of 7	0 of 7	0 of 7
Dec. 9	21	0 of 21	0 of 21	**1** of 21 (1)	0 of 21
Dec. 10	23	0 of 23	0 of 23	0 of 21	0 of 23
Dec. 11	5	0 of 5	0 of 5	0 of 5	0 of 5
Dec. 12	12	0 of 12	0 of 12	**1** of 12 (1)	0 of 12
Dec. 13	6	0 of 6	0 of 6	0 of 6	0 of 6
Dec. 14	10	0 of 10	0 of 10	0 of 10	0 of 10
Dec. 15	13	0 of 13	0 of 13	0 of 13	0 of 13
Total	158	17 of 156	13 of 155	11 of 138	4 of 150

[a] Coli37: Total coliforms; Coli44: thermotolerant coliforms; SSRC = spores of sulphite-reducing clostridia; FS: faecal streptococci.

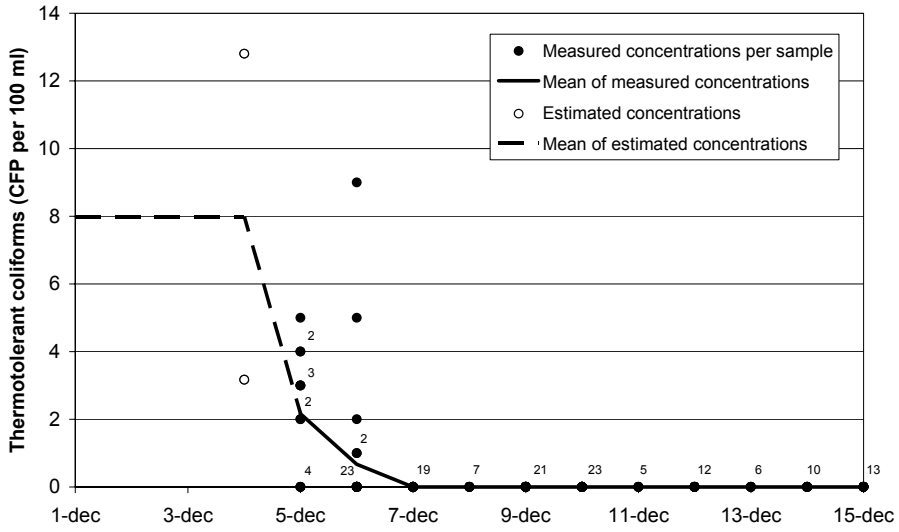


Figure 5.2 Concentrations of thermotolerant coliforms in 158 drinking-water samples collected from premises in a district of a city in the Netherlands in December 2001, after consumer complaints (taste) on December 3rd. Concentrations on December 1st through 4th were estimated from total coliform numbers on December 4th (16 and 19 CFP per 100 ml) and the ratios of thermotolerant coliforms to total coliforms on December 5th (4:5 and 2:12) in samples from the same streets. Labels near markers indicate the number of samples with identical concentrations.

However, the pressure difference between trunk mains of both systems was highest on December 2nd and declined on December 3rd. As pressures may have decreased differently from trunk mains to distribution mains in both systems, the pressure difference at the cross-connection site may have been negative only on December 3rd. This would leading to a gradual replacement of grey water in the drinking-water system by drinking-water, which might explain the decline in the concentration curve in Figure 5.2

5.5.3 Exposure period

Based on monitored pressure differences between trunk mains of both systems, exposure was assumed to start December 1st, when at the at of that day all households in the contaminated area were assumed to receive undiluted grey water. According to the results of the questionnaire, 82% of the households started boiling drinking-water before consumption after receiving the boiling advisory. In the evening of December 5th 900 premises received this advisory, the remaining 100 premises received it in the morning of December 6th. On December 6th, flushing of the mains was started and the cross-connection was discovered and closed. Presumably, exposure of the persons not complying with the boiling advisory lasted throughout December 6th before the drinking-water mains were clean. Therefore, the maximum period of exposure was from December 1st through December 6th and the minimum exposure period was from December 2nd through (the evening of) December 5th.

5.5.4 Pathogen to *E. coli* ratios in the contamination source

Index-pathogens
For each group of pathogens, one or more representatives were chosen. These so-called index-pathogens were *Cryptosporidium* and *Giardia* for protozoan parasites, *Campylobacter* for bacteria and enterovirus for viruses. Prior to the outbreak, pathogen and indicator data had been collected from the source water and after treatment. The treatment consisted of screening, coagulation, flocculation, sedimentation and rapid sand filtration of surface water from a canal that connects the river Lek (lower part of the river Rhine) and Amsterdam. This pre-treated water is also used for drinking-water production by another water company. Table 5.3 shows the pathogen to *E. coli* ratios that were calculated from the concentrations in the partially treated water. The concentrations of protozoa and *Campylobacter* were estimated from concentrations in the source water and the mean elimination capacity for pathogens and *E. coli* in the same period or in the same seasonal period of another year (as described in Chapter 4). The estimated concentrations of *E. coli* or thermotolerant coliforms matched measured concentrations in the finished water of the pretreatment plant well[4]. Enterovirus concentrations were available from the pre-treated water and were divided by *E. coli* concentrations determined in pre-treated water to calculate the ratios.

[4] Means ± SD in finished water were (estimated vs. measured):
44 ± 29 vs. 35 ± 33 CFP coli44/l (for ratios of *Cryptosporidium* and *Giardia* to coli44);
67 ± 27 vs. 51 ± 102 (for *Campylobacter*) and 61 ± 61 vs. 40 ± 55 (for enterovirus)

Table 5.3 Pathogen to *E. coli* (or thermotolerant coliform) ratios in the grey water system of a residential area in a city in the Netherlands in the period 1997-2001 (Hijnen et al, 2003). Ratios are based on concentrations in partially treated water, estimated from protozoa and bacteria concentrations in the source water and the mean elimination capacity during treatment. Enterovirus to *E. coli* ratios were determined in the treated water.

Coli44[a, b]		*Cryptosporidium*[b]		Ratio	Coli44[a]		*Giardia*[b]		Ratio
Date	CFP/l	Date	n/l		Date	n/l	Date	n/l	
21-5-97	51	21-5-97	$5.6.10^{-2}$	$1.1.10^{-3}$	21-5-97	51	21-5-97	1.8	$3.6.10^{-2}$
16-6-97	29	16-6-97	0	0	16-6-97	29	16-6-97	$8.3.10^{-3}$	$2.9.10^{-4}$
15-7-97	22	15-7-97	0	0	15-7-97	22	15-7-97	0	0
12-8-97	5	12-8-97	0	0	12-8-97	5	12-8-97	0	0
9-9-97	33	9-9-97	0	0	9-9-97	33	9-9-97	$1.3.10^{-3}$	$4.0.10^{-5}$
13-10-97	99	13-10-97	0	0	13-10-97	99	13-10-97	$8.9.10^{-2}$	$9.0.10^{-4}$
10-11-97	84	10-11-97	0	0	10-11-97	84	10-11-97	$5.1.10^{-3}$	$6.1.10^{-5}$
8-12-97	66	8-12-97	0	0	8-12-97	66	8-12-97	$2.9.10^{-2}$	$4.4.10^{-4}$
13-1-98	66	13-1-98	$4.5.10^{-2}$	$6.8.10^{-4}$	13-1-98	66	13-1-98	$4.1.10^{-2}$	$6.2.10^{-4}$
11-2-98	20	11-2-98	$1.6.10^{-2}$	$8.0.10^{-4}$	11-2-98	20	11-2-98	$5.6.10^{-2}$	$2.8.10^{-3}$
9-3-98	38	9-3-98	$3.8.10^{-2}$	$1.0.10^{-3}$	9-3-98	38	9-3-98	$3.5.10^{-2}$	$9.4.10^{-4}$
6-4-98	17	6-4-98	0	0	6-4-98	17	6-4-98	0.11	$6.5.10^{-3}$
Mean	44	Mean	$1.3.10^{-2}$	$2.9.10^{-4}$	Mean	44	Mean	0.18	$4.2.10^{-3}$
E. coli		*Campylobacter*[b]		Ratio	*E. coli*		Enterovirus[b]		Ratio
Date	CFP/l	Date	MPN/l		Date	CFP/l	Date	PFP/l	
21-3-01	90	6-3-01	0	0	26-1-00	28	23-1-01	$7.0.10^{-2}$	$2.5.10^{-3}$
13-6-01	22	5-6-01	$8.8.10^{-2}$	$4.0.10^{-3}$	23-2-00	150	21-2-01	$3.1.10^{-2}$	$2.1.10^{-4}$
8-8-01	51	7-8-01	0	0	22-3-00	32	20-3-01	$2.2.10^{-2}$	$6.9.10^{-4}$
5-9-01	69	5-9-01	1.1	$1.6.10^{-2}$	5-4-00	12	3-4-01	$7.5.10^{-2}$	$6.9.10^{-3}$
4-10-00	93	2-10-01	0.51	$5.5.10^{-3}$	19-4-00	16	18-4-01	$3.2.10^{-2}$	$2.0.10^{-3}$
1-11-00	49	6-11-01	0.88	$1.8.10^{-2}$	16-5-01	0 [c]	15-5-01	$5.0.10^{-3}$	$1.0.10^{-2}$
4-10-00	93	4-12-01	2.0	$2.1.10^{-2}$					
Mean	67	Mean	0.65	$9.8.10^{-3}$	Mean	40	Mean	$3.9.10^{-2}$	$9.9.10^{-4}$

[a] Coli44 = Thermotolerant coliforms

[b] Mean Decimal Elimination Capacities (DEC = ^{10}logarithms (finished)/(source)) were:
1.66 (*E. coli* or coli44); 2.44 (Cryptosporidium); 2.44 (Giardia); 1.66 (Campylobacter);
based on elimation of *Clostridium perfringens* (for *Cryptosporidium* and *Giardia*) and *E. coli* or coli44 (for Campylobacter)

[c] For calculation of ratio, the *E. coli* concentration was estimated at 0.5.

5.5.5 Tier 1: basic calculation of the pathogen concentrations

All data used in the calculations are subject to variation. The variation of data can be expressed in Probability Density Functions (PDF), that can be graphically presented as histograms, but also cumulatively as a Cumulative Probability Density Functions (CDF). In calculations, either the actual measurements (empirical data) can be used or statistical models that are fitted to these measurements. As is highlighted in chapter 2, 4, 7 and 8, the calculations for Quantitative Microbial Risk Assessment can and should be performed in a tiered approach. Here, the Tier 2 type calculation is a multiplication of the triangular fits of the PDFs of *E. coli* (during this outbreak measured as thermotolerant coliforms, Figure 5.2) and pathogen to *E. coli* ratios (Table 5.3).

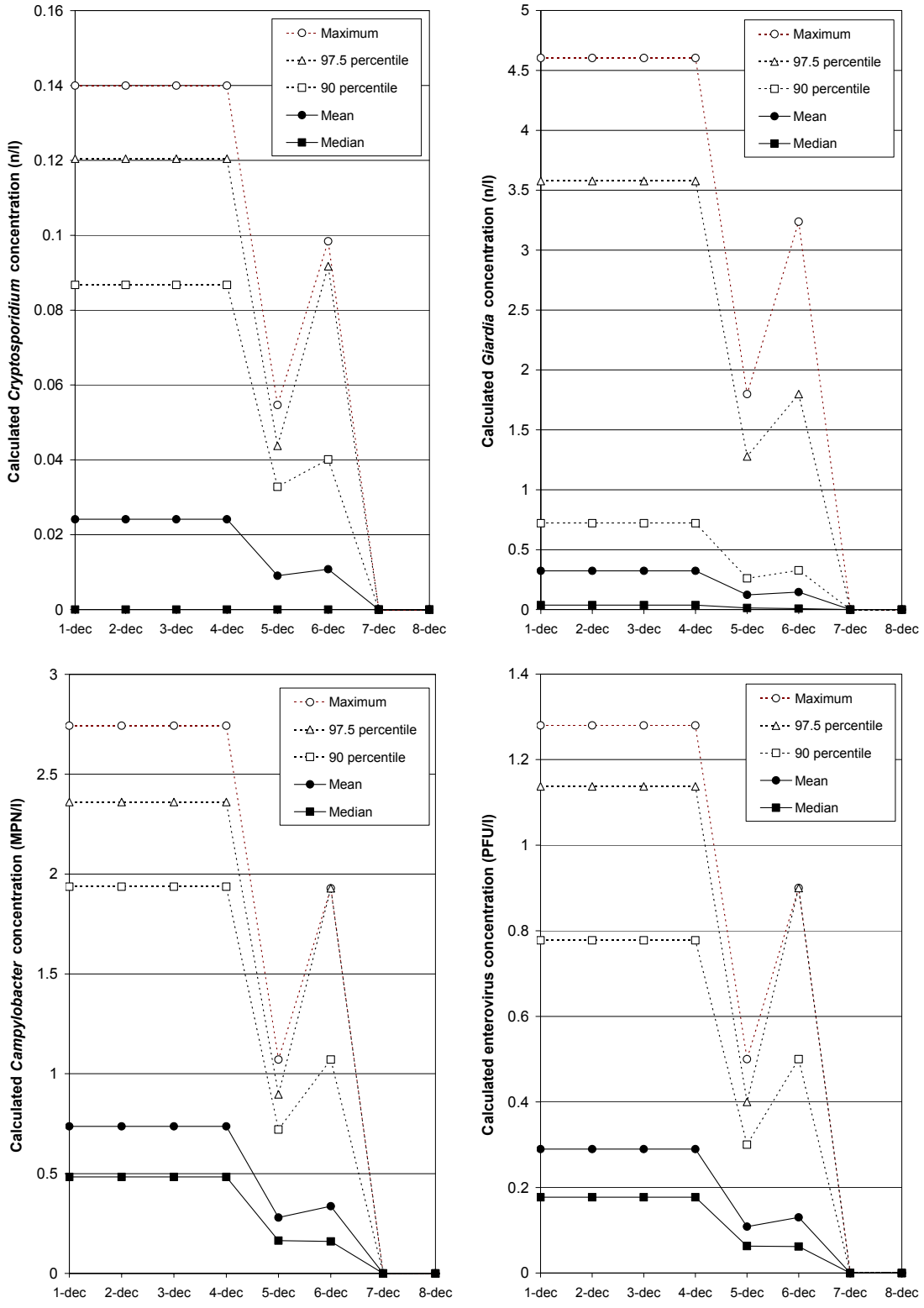


Figure 5.3 Index numbers of the probability density functions of calculated daily pathogen concentrations during the contamination event case (please note the differences in scales of pathogen concentrations).

This means that the minimum *E. coli* concentration per day is multiplied with the minimum pathogen to *E. coli* ratio, the mean with the mean and the maximum with the maximum. Subsequently, the resulting estimates of daily pathogen concentrations are summed for the exposure period. This results in the retrospectively expected exposure per person during the outbreak, consuming a standard consumption[5] of 1 litre per person per day. The mean expected exposure to *Campylobacter* and *Giardia* was highest and it is very likely that some of the inhabitants that consumed 1 litre per day ingested several *Campylobacter* (table 5.4).

5.5.6 Tier 3: including variation

A statistical fit to a more detailed function describing the variation of the data can dampen the variation in the empirical data caused by small sample size and can interpolate (and extrapolate) data. Statistical models can be induced from theory, from comparable cases or empirically when the statistical model fits the measured data well. In this paragraph and the rest of this chapter the empirical data are used. The data are not fitted, as neither induction method is possible with the limited data and knowledge available. In other words, statistical fitting would change the PDF without being able to verify whether the fitted PDF would represent real conditions better than the empirical distribution of the data.

The pathogen concentrations during the outbreak were calculated from the *E. coli* concentrations (measured as thermotolerant coliforms) using MatLab® 7.0.4. The following steps in the calculations were performed:

- The concentrations of *E. coli* on December 1st through December 3rd were assumed to be identical to those calculated for December 4th.
- For every day of the event, the empirical PDFs of *E. coli* concentrations and pathogen to *E. coli* ratios were multiplied by bootstrapping[6] (100,000-fold), resulting in a PDF of daily pathogen concentrations.
- The statistical index numbers of each daily PDF of 100,000 daily pathogen concentrations are presented in Figure 5.3.

The retrospectively expected exposure per person during the outbreak, consuming a standard consumption of 1 litre per person per day, is calculated by summing the daily pathogen concentrations. The mean expected exposures calculated using the Tier 2 method are comparable to the results of the Tier 1 method (table 5.4). Using the Tier 1 method, however, it is not possible to calculate risks other than minimum, mean and maximum risks. As mean values may differ from median (50-percentile) values due to high maximum values, it is difficult to evaluate the number of inhabitants exposed to a certain level of exposure. Using the Tier 2 method, it is possible to estimate that at least 50% of the inhabitants (median) that consumed 1 litre per day ingested 1.6 *Campylobacter* or more and that 97.5% of the inhabitants ingested 8 *Campylobacter* or less (table 5.4, minimum exposure).

Uncertainties in data and sensitivity of the Tier 2 method are discussed in paragraph 5.9.

[5] Actual consumption data from chapter 6 will be used for calculation of actual exposure and infection risks in chapter 7.

[6] During bootstrapping, also called Monte-Carlo analysis, random values are drawn from every dataset and entered into the calculation formula. This is performed repeatedly.

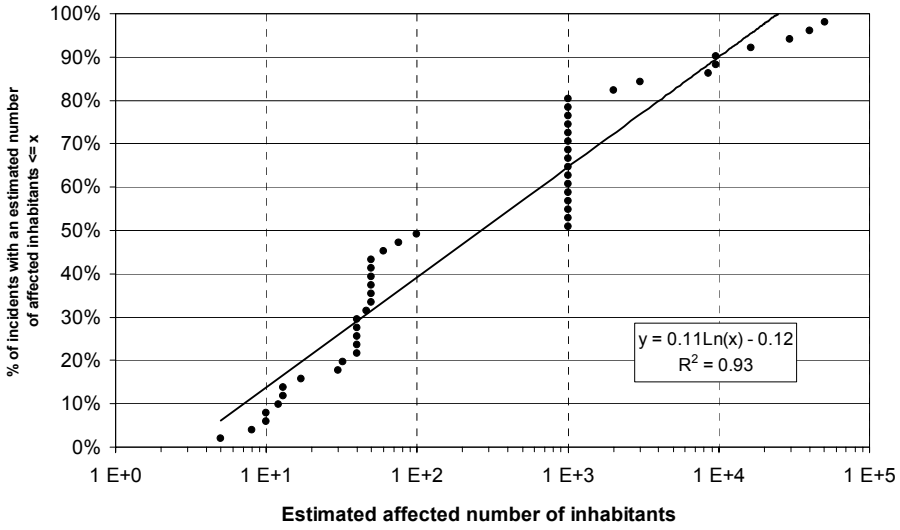
Table 5.4 Retrospectively expected total exposure to pathogens per person during the outbreak case event assuming a standard consumption of 1 litre per person per day. Numbers of 1 and over are expected numbers of pathogens ingested per person during the outbreak. Numbers below 1 are best interpreted as the probability per person of ingesting 1 pathogen during the outbreak. Variation of estimated exposure is primarily caused by variation of the pathogen to *E. coli* ratios in the source of the contamination (household water, which is pretreated river water, see table 5.3). Tier 1 calculations multiply triangular PDFs of *E. coli* (in this case measured as thermotolerant coliforms) and pathogen to *E. coli* ratios (minimum x minimum, mean x mean, maximum x maximum, see paragraph 5.5.5). Tier 2 calculations multiply empirical PDFs using bootstrapping.

Exposure period	Expected total number ingested during outbreak per person drinking 1 litre per day			
	Cryptosporidium	*Giardia*	*Campylobacter*	Enterovirus
Minimum: December 2nd through 5th				
Tier 1				
- Minimum	0	0	0	$1.9.10^{-2}$
- Mean	$7.6.10^{-2}$	1.1	2.6	0.26
- Maximum	0.48	16	9.4	4.4
Tier 2				
- Mean	$8.1.10^{-2}$	1.1	2.5	0.98
- Median	0	0.12	1.6	0.59
- 2.5 percentile	0	0	0	$1.3.10^{-2}$
- 97.5 percentile	0.41	12	8.0	3.8
Maximum: December 1st through 6th				
Tier 1				
- Minimum	0	0	0	$2.5.10^{-2}$
- Mean	0.10	1.4	3.4	0.34
- Maximum	0.72	24	14	6.6
Tier 2				
- Mean	0.12	1.6	3.6	1.4
- Median	0	0.17	2.3	0.83
- 2.5 percentile	0	0	0	$1.5.10^{-2}$
- 97.5 percentile	0.62	17	12	5.8

5.6 REPORTED NON-OUTBREAK EVENTS

5.6.1 Characteristics of 50 events

Water companies in the Netherlands were asked to supply records of events that had occurred in the period from 1994 through 2003. For this survey, events were defined as cases of water quality degradation, as determined by repeated detection of total coliforms and/or indicators of faecal contamination, during which event at least one sample contained an indicator of faecal contamination. The survey resulted in reports of 50 event from 7 water companies together supplying c. 11 million inhabitants. The estimated number of inhabitants affected by the contaminations varied from 5 to c. 50,000, with 9 events affecting over 1000 and a total number of c. 185,000. For some unknown reason, the CDF of affected inhabitants increases logarithmically ($R^2 = 0.93$, Figure 5.4). The reporting water companies stressed in their contributions that, although all events have been reported to the national inspectorate, event reports have not been archived separately and were not all retrievable. Therefore, the survey resulted in an incomplete overview of both frequency as well as impact (inhabitants in contaminated area) and circumstances (cause, source, countermeasures, etc.).
Based on these data, for the c. 11 million inhabitants of the participating water companies in the Netherlands, the probability of being affected by a contamination event would be



185,000 / 11 million / 10 years = c. $1.7 \cdot 10^{-3}$ per person per year.

Figure 5.4 Estimated number of affected inhabitants (total 185,000) during 50 faecal contamination events reported in the Netherlands from 1994 through 2003 by 7 water companies supplying c. 11 million inhabitants. Most estimates were supplied by the reporting water companies. Missing values in the reports were estimated at 1000 in case of contamination during distribution and the total number of inhabitants of the supply zone in case of contamination of the treatment plant.

Information about concentrations of *E. coli* measured during the event were reported for 50 events, the majority of the reported events (including the case of paragraph 5.5). In most cases *E. coli* was measured as thermotolerant coliforms (coli44).

Table 5.5 Characteristics of 50 faecal contamination events reported in the Netherlands from 1994 through 2003 by 7 water companies supplying 11 million inhabitants, affecting c. 185,00 inhabitants. Causes and sources are presented when likely or certain, otherwise indicated as 'unknown'. The overview is not representative of faecal contamination events due to poor recording of events and characteristics.

Cause	Phase	Source	Treatment	Before distribution	Distribution	Unknown
		1	1	1	37	10
	Construction	Well	Treatment	Reservoir	Main	Unknown
		1	0	2	34	13
	Failure	Replacement main, hydrant	Repair of mains	Damaged main: 1	Cross-connection: 1	Unknown
		18	8	Leak in reservoir: 1	Flushing: 1 Swabbing: 1	19
Source		Sewage	Surface water	Soil(water)	Roof material	Unknown
		3	2	26	0	19
Detection	Method*	1st sample	2nd sample	3rd sample	4th sample	Complaints
		25	7	1	1	3
	Sampling site	Treatment plant	Distribution reservoir	Periodical tap water sample	After operations	
		2	12	7	29	
Measures		Boiling advisory	Dosage of disinfectant	Protective measures unknown	Flushing mains	Isolation of mains section
		7	2	16	48	29

* 3 of the 50 events were detected as a result of consumer complaints, the remaining 47 as a result of testing for the presence of total coliforms. The table shows in which sample rank the first sign of faecal contamination (*E. coli*, thermotolerant coliforms, faecal streptococci) was found.

Only 3 of 50 events were reported to have occurred in wells or (groundwater) treatment plants, whereas 37 events occurred in distribution systems (Table 5.5). Over half of the reported events concerned contaminations that were detected after operations in mains (18 replacements, 8 repairs, 2 cleaning operations). In these cases, supply was commenced immediately after the operations, but not before the mains were flushed and in some cases disinfected. Standard procedure for operations is isolation of the distribution mains that were opened, until microbial safety has been verified by water quality testing. Of the unknown causes, most were not recorded well as information was limited to sampling dates and concentrations of thermotolerant coliforms in laboratory databases. It is likely that the causes of these events in most cases also were a result of mains operations. These results do not imply, however, that the probability of contamination is highest during operations, as the probability of detecting contaminations after operations is likely to be much higher than under normal operations, knowing when and where to take samples to verify the microbial safety of drinking-water.

The median duration of the events from detection to the end (defined as no further detection of *E. coli* or coliforms) is 8 days with a 95-percentile of 30 days (Figure 5.5). The real duration is longer, as events usually are not immediately detected at the onset. Where available, the difference between the real onset of the event and the detection is depicted in Figure 5.5; this is usually 2 days.

During 26 events, no boiling advisory was issued or disinfectant was dosed. Flushing was the standard response to detection of contamination events, but the contamination may still exist. In most cases however, flushing results in a rapid decrease of concentrations of faecal indicators and total coliforms (in 100 ml samples), after which the contamination is considered to have been removed.

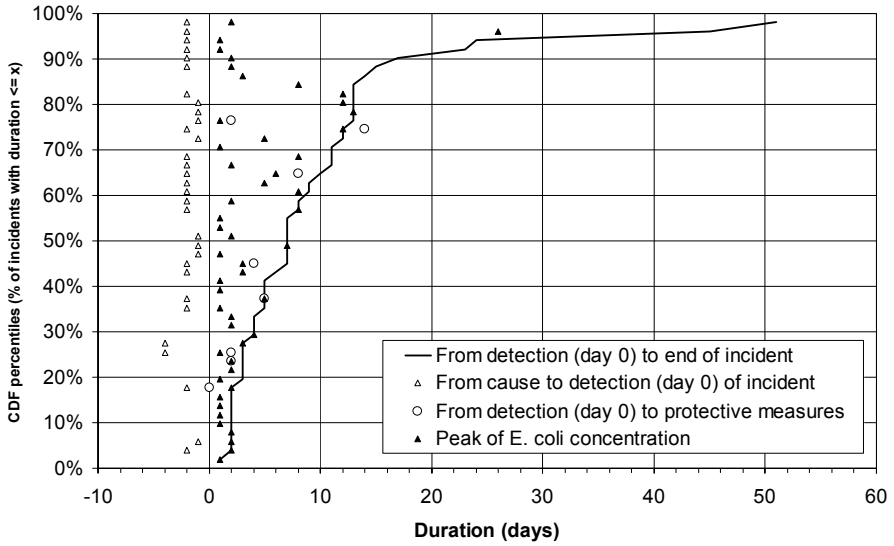


Figure 5.5 Duration of 50 faecal contamination events reported in the Netherlands from 1994 through 2003 by 7 water companies supplying c. 11 million inhabitants, affecting c. 185,000 inhabitants. The end of the event is the second day when no indicator bacteria are found in 100 ml samples. If no protective measures (boiling advisory, disinfectant dose) were taken, duration is not presented.

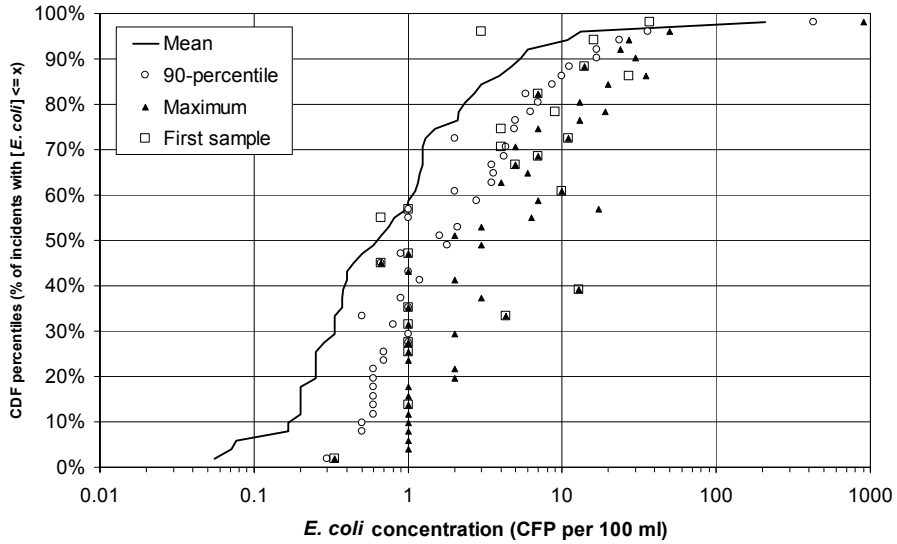


Figure 5.6 *E. coli* concentrations per 100 ml in water samples collected from finished water (300 ml) or distributed water (100 ml) during 50 faecal contamination events reported in the Netherlands from 1994 through 2003 by 7 water companies supplying c. 11 million inhabitants, affecting c. 185,000 inhabitants. Individual samples (90-percentiles, maximum or first) containing no *E. coli* are not included in the figure.

5.6.2 *E. coli* concentration in water during the events

The mean concentration of *E. coli*, often measured as thermotolerant coliforms, found in samples (mostly) collected from taps during the event, ranged from 0.055 CFP per 100 ml to 210 CFP per 100 ml (Figure 5.6). The maximum concentration of 900 CFP per 100 ml was found on the second day of the event with the highest initial (37 CFP per 100 ml) and mean concentration (210 CFP per 100 ml), lasting 10 days. During 17 events, the highest concentration of *E. coli* was measured in the first sample that was collected, so in the majority of the cases the peak concentration followed after detection.

5.6.3 Pathogen to *E. coli* ratios

In order to calculate the pathogen concentrations as accurately as possible, the pathogen to *E. coli* ratios to be used in the calculations are best determined in samples of the contamination source as soon as possible after the start of the contamination. In ideal cases, the cause of every contamination is traced and the contamination source is identified and characterised. The pathogen and *E. coli* in the (most likely) contamination sources have not been determined in any of the presented events, however. Therefore, to calculate the expected exposure, pathogen to *E. coli* ratios in three common sources of contaminations are available: sewage, surface water and soil and shallow groundwater close to distribution mains. The tables are included in detail to enable the reader to perform the same calculations with *E. coli* concentrations collected during faecal contamination incidents.

Sewage as presumed source
In 1997 and 1998 pathogen and thermotolerant coliform concentrations were determined in 11 samples collected in a period of 12 months from the untreated influent of a sewage treatment plant in the Netherlands (Medema *et al.*, 2001). *Campylobacter* as well as *E. coli* concentrations in samples of untreated sewage were found in a paper by Höller et al. (1988). The concentrations and ratios are presented in Table 5.6.

Surface water as presumed source
In 1997 and 1998 pathogen and thermotolerant coliform concentrations were determined in 26 samples collected in a period of 12 months from the river Rhine and the river Meuse at the border of the Netherlands (Medema *et al.*, 2001). The concentrations and ratios are presented in Table 5.7 and 5.8.

Soil and shallow groundwater as presumed source
Only one dataset is known for soil and shallow groundwater (Karim *et al*., 2000, 2003, LeChevallier *et al.*, 2003). The concentrations and ratios are presented in Table 5.9. The data set is used to make three sets of enterovirus to *E. coli* ratios:

1. Ratios of culturable enteric viruses vs. thermotolerant coliforms from data pairs in which thermotolerant coliforms were detectable.
2. Ratios of culturable enterovirus and enteroviruses detectable with PCR vs. detectable thermotolerant coliform concentrations.
3. Ratios of both culturable enterovirus as well as enteroviruses detectable with PCR vs. thermotolerant coliforms from all data pairs. When thermotolerant coliforms were not detectable, their concentration was estimated to be half the detection limit in order to be able to calculate a ratio.

Table 5.6 Pathogens and thermotolerant coliforms or *E. coli* in untreated sewage
A: from a sewage treatment plant in the Netherlands (from Medema *et al.,* 2001)
B: Campylobacter from sewer lines in a city in Germany (from Höller *et al.*, 1988)

A

Sampling	Coli44*	*Cryptosporidium*		*Giardia*		Enterovirus	
Date	CFP/l	n/l	Ratio**	n/l	Ratio**	PFU/l	Ratio**
30-6-97	$1.5 \cdot 10^{8}$	37	$2.5 \cdot 10^{-7}$	856	$5.8 \cdot 10^{-6}$	3	$1.9 \cdot 10^{-8}$
25-8-97	$1.6 \cdot 10^{8}$	0	0	0	0		
8-9-97	$1.6 \cdot 10^{8}$	0	0	0	0	127	$7.9 \cdot 10^{-7}$
23-9-97	$1.3 \cdot 10^{8}$	24	$1.9 \cdot 10^{-7}$	91	$7.1 \cdot 10^{-7}$	136	$1.1 \cdot 10^{-6}$
20-10-97	$1.2 \cdot 10^{8}$	28	$2.3 \cdot 10^{-7}$	921	$7.8 \cdot 10^{-6}$	36	$3.1 \cdot 10^{-7}$
17-11-97	$1.1 \cdot 10^{8}$	19	$1.7 \cdot 10^{-7}$	445	$4.1 \cdot 10^{-6}$	30	$2.8 \cdot 10^{-7}$
15-12-97	$5.2 \cdot 10^{7}$	10	$2.0 \cdot 10^{-7}$	161	$3.1 \cdot 10^{-6}$	42	$8.0 \cdot 10^{-7}$
19-1-98	$3.1 \cdot 10^{7}$	42	$1.4 \cdot 10^{-6}$	180	$5.9 \cdot 10^{-6}$	18	$6.0 \cdot 10^{-7}$
9-2-98	$6.6 \cdot 10^{7}$	155	$2.3 \cdot 10^{-6}$	2051	$3.1 \cdot 10^{-5}$	32	$4.8 \cdot 10^{-7}$
9-3-98	$6.8 \cdot 10^{7}$	25	$3.7 \cdot 10^{-7}$	357	$5.2 \cdot 10^{-6}$	9	$1.3 \cdot 10^{-7}$
4-5-98	$1.1 \cdot 10^{8}$	22	$2.0 \cdot 10^{-7}$	696	$6.2 \cdot 10^{-6}$	164	$1.5 \cdot 10^{-6}$
Mean	$1.0 \cdot 10^{+8}$	33	$4.8 \cdot 10^{-7}$	520	$6.4 \cdot 10^{-6}$	60	$5.9 \cdot 10^{-7}$
SD	$4.4 \cdot 10^{7}$	43	$7.2 \cdot 10^{-7}$	600	$8.6 \cdot 10^{-6}$	59	$4.5 \cdot 10^{-7}$
Median	$1.1 \cdot 10^{+8}$	24	$2.0 \cdot 10^{-7}$	360	$5.2 \cdot 10^{-6}$	34	$5.4 \cdot 10^{-7}$
2.5 Percentile	$3.6 \cdot 10^{7}$	0	0	0	0	42	$4.4 \cdot 10^{-8}$
97.5 Percentile	$1.6 \cdot 10^{+8}$	130	$2.1 \cdot 10^{-6}$	1800	$2.5 \cdot 10^{-5}$	160	$1.4 \cdot 10^{-6}$
Minimum	$3.1 \cdot 10^{7}$	0	0	0	0	3	$1.9 \cdot 10^{-8}$
Maximum	$1.6 \cdot 10^{+8}$	155	$2.3 \cdot 10^{-6}$	2051	$3.1 \cdot 10^{-5}$	164	$1.5 \cdot 10^{-6}$

B

Sampling	*E. coli*	*Campylobacter*		Date	*E. coli*	*Campylobacter*	
date	CFP/l	MPN/l	Ratio**		CFP/l	MPN/l	Ratio**
Sampling site: HassStrasse				*Sampling site: Möltenboe*			
18-6-1985	$3{,}7 \cdot 10^{+8}$	$2{,}6 \cdot 10^{4}$	$7{,}1 \cdot 10^{-5}$	18-6-1985	$4{,}0 \cdot 10^{+8}$	$2{,}0 \cdot 10^{3}$	$5{,}0 \cdot 10^{-6}$
15-7-1985	$1{,}1 \cdot 10^{+8}$	$7{,}9 \cdot 10^{3}$	$7{,}4 \cdot 10^{-5}$	15-7-1985	$1{,}3 \cdot 10^{+8}$	$6{,}3 \cdot 10^{4}$	$4{,}7 \cdot 10^{-4}$
16-9-1985	$1{,}5 \cdot 10^{7}$	$2{,}0 \cdot 10^{5}$	$1{,}3 \cdot 10^{-2}$	16-9-1985	$2{,}1 \cdot 10^{6}$	$1{,}3 \cdot 10^{4}$	$6{,}0 \cdot 10^{-3}$
11-11-1985	$1{,}9 \cdot 10^{7}$	$1{,}0 \cdot 10^{5}$	$5{,}4 \cdot 10^{-3}$	11-11-1985	$2{,}1 \cdot 10^{+8}$	$7{,}9 \cdot 10^{3}$	$3{,}7 \cdot 10^{-5}$
13-1-1986	$3{,}5 \cdot 10^{9}$	$7{,}9 \cdot 10^{2}$	$2{,}2 \cdot 10^{-7}$	13-1-1986	$6{,}0 \cdot 10^{+8}$	$1{,}6 \cdot 10^{3}$	$2{,}6 \cdot 10^{-6}$
3-3-1986	$2{,}7 \cdot 10^{7}$	$2{,}0 \cdot 10^{4}$	$7{,}4 \cdot 10^{-4}$	3-3-1986	$1{,}9 \cdot 10^{7}$	$7{,}9 \cdot 10^{3}$	$4{,}3 \cdot 10^{-4}$
21-4-1986	$5{,}1 \cdot 10^{+8}$	$5{,}0 \cdot 10^{3}$	$9{,}8 \cdot 10^{-6}$	21-4-1986	$3{,}1 \cdot 10^{+8}$	$5{,}0 \cdot 10^{3}$	$1{,}6 \cdot 10^{-5}$
9-6-1986	$5{,}2 \cdot 10^{+8}$	$8{,}9 \cdot 10^{5}$	$1{,}7 \cdot 10^{-3}$	9-6-1986	$8{,}3 \cdot 10^{+8}$	$1{,}6 \cdot 10^{4}$	$1{,}9 \cdot 10^{-5}$
25-8-1986	$2{,}0 \cdot 10^{+8}$	$6{,}3 \cdot 10^{4}$	$3{,}2 \cdot 10^{-4}$	25-8-1986	$4{,}8 \cdot 10^{+8}$	$5{,}0 \cdot 10^{3}$	$1{,}0 \cdot 10^{-5}$
Sampling site: Wik				*Sampling site: Pries*			
18-6-1985	$3{,}7 \cdot 10^{+8}$	$1{,}8 \cdot 10^{5}$	$4{,}8 \cdot 10^{-4}$	11-11-1985	$3{,}0 \cdot 10^{+8}$	$8{,}9 \cdot 10^{2}$	$3{,}0 \cdot 10^{-6}$
15-7-1985	$1{,}1 \cdot 10^{+8}$	$1{,}4 \cdot 10^{4}$	$1{,}3 \cdot 10^{-4}$	13-1-1986	$8{,}5 \cdot 10^{+8}$	$2{,}5 \cdot 10^{3}$	$3{,}0 \cdot 10^{-6}$
16-9-1985	$1{,}1 \cdot 10^{7}$	$5{,}0 \cdot 10^{4}$	$4{,}6 \cdot 10^{-3}$	3-3-1986	$2{,}4 \cdot 10^{7}$	$5{,}0 \cdot 10^{2}$	$2{,}1 \cdot 10^{-5}$
11-11-1985	$2{,}7 \cdot 10^{7}$	$1{,}3 \cdot 10^{4}$	$4{,}7 \cdot 10^{-4}$	21-4-1986	$1{,}6 \cdot 10^{+8}$	$7{,}9 \cdot 10^{2}$	$5{,}0 \cdot 10^{-6}$
13-1-1986	$2{,}6 \cdot 10^{9}$	$1{,}1 \cdot 10^{3}$	$4{,}3 \cdot 10^{-7}$	9-6-1986	$3{,}5 \cdot 10^{+8}$	$1{,}3 \cdot 10^{3}$	$3{,}6 \cdot 10^{-6}$
3-3-1986	$2{,}1 \cdot 10^{7}$	$4{,}5 \cdot 10^{3}$	$2{,}1 \cdot 10^{-4}$	25-8-1986	$3{,}4 \cdot 10^{7}$	$6{,}3 \cdot 10^{3}$	$1{,}9 \cdot 10^{-4}$
21-4-1986	$6{,}2 \cdot 10^{+8}$	$2{,}0 \cdot 10^{3}$	$3{,}2 \cdot 10^{-6}$				
9-6-1986	$5{,}6 \cdot 10^{+8}$	$4{,}5 \cdot 10^{4}$	$7{,}9 \cdot 10^{-5}$				
25-8-1986	$1{,}2 \cdot 10^{7}$	$2{,}5 \cdot 10^{4}$	$2{,}1 \cdot 10^{-3}$				
Mean	$4{,}4 \cdot 10^{+8}$	$5{,}4 \cdot 10^{4}$	$1{,}1 \cdot 10^{-3}$	P2.5 ***	$9{,}2 \cdot 10^{6}$	$7{,}4 \cdot 10^{2}$	$3{,}9 \cdot 10^{-7}$
SD	$7{,}4 \cdot 10^{+8}$	$1{,}6 \cdot 10^{5}$	$2{,}7 \cdot 10^{-3}$	P97.5 ***	$2{,}8 \cdot 10^{9}$	$3{,}4 \cdot 10^{5}$	$7{,}5 \cdot 10^{-3}$
Median	$2{,}1 \cdot 10^{+8}$	$7{,}9 \cdot 10^{3}$	$7{,}4 \cdot 10^{-5}$	Minimum	$2{,}1 \cdot 10^{6}$	$5{,}0 \cdot 10^{2}$	$2{,}2 \cdot 10^{-7}$
N	33			Maximum	$3{,}5 \cdot 10^{9}$	$8{,}9 \cdot 10^{5}$	$1{,}3 \cdot 10^{-2}$

*Coli44 = thermotolerant coliforms ** Ratio = pathogen to coli44 ratio *** P = Percentile

Table 5.7 Concentrations and ratios of pathogens and thermotolerant coliforms in the river Rhine and Meuse at the border of the Netherlands (Lobith and Eijsden, respectively, from Medema *et al.*, 2001)

Sample	Coli44*	Cryptosporidium		Giardia		Enterovirus	
Date	CFP per l	N per l	Ratio**	N per l	Ratio**	PFU per l	Ratio**
20-5-97	$2.5 \cdot 10^{5}$	5.8	$2.4 \cdot 10^{-5}$	16	$6.4 \cdot 10^{-5}$	1.9	$7.5 \cdot 10^{-6}$
17-6-97	$1.8 \cdot 10^{4}$	2.5	$1.4 \cdot 10^{-4}$	8.6	$4.9 \cdot 10^{-4}$	4.2	$2.4 \cdot 10^{-4}$
15-7-97	$7.7 \cdot 10^{4}$	3.1	$4.1 \cdot 10^{-5}$	13	$1.7 \cdot 10^{-4}$	2.7	$3.5 \cdot 10^{-5}$
12-8-97	$6.5 \cdot 10^{4}$	0.42	$6.4 \cdot 10^{-6}$	2.1	$3.2 \cdot 10^{-5}$	$3.2 \cdot 10^{-2}$	$4.9 \cdot 10^{-7}$
9-9-97	$3.5 \cdot 10^{4}$					1.8	$5.1 \cdot 10^{-5}$
7-10-97	$3.4 \cdot 10^{4}$	0.20	$5.8 \cdot 10^{-6}$	0.40	$1.2 \cdot 10^{-5}$	0.20	$5.9 \cdot 10^{-6}$
4-11-97	$4.5 \cdot 10^{4}$	0.90	$2.0 \cdot 10^{-5}$	5.7	$1.3 \cdot 10^{-4}$	$7.6 \cdot 10^{-2}$	$1.7 \cdot 10^{-6}$
2-12-97	$2.2 \cdot 10^{5}$	0.57	$2.6 \cdot 10^{-6}$	16	$7.4 \cdot 10^{-5}$	0.20	$9.0 \cdot 10^{-7}$
16-12-97	$1.4 \cdot 10^{5}$	1.5	$1.1 \cdot 10^{-5}$	8.6	$6.3 \cdot 10^{-5}$	0.23	$1.7 \cdot 10^{-6}$
24-2-98	$9.5 \cdot 10^{4}$	1.6	$1.7 \cdot 10^{-5}$	17	$1.7 \cdot 10^{-4}$	0.65	$6.8 \cdot 10^{-6}$
24-3-98	$9.7 \cdot 10^{4}$	0.20	$2.1 \cdot 10^{-6}$	19	$1.9 \cdot 10^{-4}$	1.0	$1.0 \cdot 10^{-5}$
21-4-98	$4.7 \cdot 10^{5}$	0.31	$6.7 \cdot 10^{-7}$	19	$4.0 \cdot 10^{-5}$	0.94	$2.0 \cdot 10^{-6}$
19-5-98	$4.2 \cdot 10^{4}$	0.86	$2.0 \cdot 10^{-5}$	12	$2.9 \cdot 10^{-4}$	$9.1 \cdot 10^{-2}$	$2.2 \cdot 10^{-6}$
21-5-97	$3.6 \cdot 10^{3}$	16	$4.4 \cdot 10^{-3}$	21	$5.8 \cdot 10^{-3}$	0.18	$5.0 \cdot 10^{-5}$
18-6-97	$6.7 \cdot 10^{3}$	5.2	$7.8 \cdot 10^{-4}$	9.8	$1.5 \cdot 10^{-3}$	$2.8 \cdot 10^{-2}$	$4.2 \cdot 10^{-6}$
16-7-97	$7.5 \cdot 10^{3}$	0.46	$6.1 \cdot 10^{-5}$	1.5	$2.0 \cdot 10^{-4}$	0.16	$2.1 \cdot 10^{-5}$
13-8-97	$5.2 \cdot 10^{3}$	0.39	$7.6 \cdot 10^{-5}$	0.98	$1.9 \cdot 10^{-4}$	$1.4 \cdot 10^{-2}$	$2.7 \cdot 10^{-6}$
10-9-97	$9.5 \cdot 10^{3}$	0.27	$2.9 \cdot 10^{-5}$	0.68	$7.1 \cdot 10^{-5}$	$6.9 \cdot 10^{-2}$	$7.3 \cdot 10^{-6}$
8-10-97	$7.3 \cdot 10^{3}$	0.26	$3.6 \cdot 10^{-5}$	0.92	$1.3 \cdot 10^{-4}$	$4.2 \cdot 10^{-2}$	$5.8 \cdot 10^{-6}$
5-11-97	$4.1 \cdot 10^{3}$	0.13	$3.0 \cdot 10^{-5}$	0.63	$1.5 \cdot 10^{-4}$	0.11	$2.7 \cdot 10^{-5}$
3-12-97	$1.8 \cdot 10^{4}$	0.12	$7.0 \cdot 10^{-6}$	3.9	$2.2 \cdot 10^{-4}$	$3.7 \cdot 10^{-2}$	$2.1 \cdot 10^{-6}$
17-12-97	$1.5 \cdot 10^{4}$	2.7	$1.8 \cdot 10^{-4}$	17	$1.2 \cdot 10^{-3}$	0.30	$2.0 \cdot 10^{-5}$
28-1-98	$8.9 \cdot 10^{3}$	2.5	$2.9 \cdot 10^{-4}$	13	$1.5 \cdot 10^{-3}$	0.11	$1.2 \cdot 10^{-5}$
25-2-98	$7.0 \cdot 10^{3}$	1.5	$2.2 \cdot 10^{-4}$	7.6	$1.1 \cdot 10^{-3}$	$5.2 \cdot 10^{-2}$	$7.4 \cdot 10^{-6}$
25-3-98	$8.4 \cdot 10^{3}$	0.55	$6.5 \cdot 10^{-5}$	3.1	$3.7 \cdot 10^{-4}$	$6.6 \cdot 10^{-2}$	$7.9 \cdot 10^{-6}$
22-4-98	$8.0 \cdot 10^{3}$	0.70	$8.8 \cdot 10^{-5}$	5.9	$7.3 \cdot 10^{-4}$	$4.7 \cdot 10^{-2}$	$5.9 \cdot 10^{-6}$
18-5-98	$1.6 \cdot 10^{3}$	1.1	$6.8 \cdot 10^{-4}$	7.9	$5.0 \cdot 10^{-3}$	$1.5 \cdot 10^{-2}$	$9.4 \cdot 10^{-6}$
Mean	$6.3 \cdot 10^{4}$	1.9	$2.8 \cdot 10^{-4}$	8.9	$7.6 \cdot 10^{-4}$	0.56	$2.0 \cdot 10^{-5}$
SD	$1.0 \cdot 10^{5}$	3.2	$8.5 \cdot 10^{-4}$	6.7	$1.4 \cdot 10^{-3}$	1.0	$4.6 \cdot 10^{-5}$
Median	$1.8 \cdot 10^{4}$	0.78	$3.3 \cdot 10^{-5}$	8.3	$1.9 \cdot 10^{-4}$	0.11	$7.3 \cdot 10^{-6}$
N	27	26	26	26	26	27	27
2.5 percentile	$2.9 \cdot 10^{3}$	0.12	$1.5 \cdot 10^{-6}$	0.54	$2.4 \cdot 10^{-5}$	$1.5 \cdot 10^{-2}$	$7.6 \cdot 10^{-7}$
97.5 percentile	$3.3 \cdot 10^{5}$	9.5	$2.1 \cdot 10^{-3}$	20	$5.3 \cdot 10^{-3}$	3.2	$1.2 \cdot 10^{-4}$
Minimum	$1.6 \cdot 10^{3}$	0.12	$6.7 \cdot 10^{-7}$	0.4	$1.2 \cdot 10^{-5}$	$1.4 \cdot 10^{-2}$	$4.9 \cdot 10^{-7}$
Maximum	$4.7 \cdot 10^{5}$	16	$4.4 \cdot 10^{-3}$	21	$5.8 \cdot 10^{-3}$	4.2	$2.4 \cdot 10^{-4}$

*Coli44 = thermotolerant coliforms ** Ratio = pathogen to coli44 ratio

Table 5.8 Concentrations and ratios of *E. coli* and Campylobacter in the river Meuse at the intake site of a water company in the Netherlands (from de Roda Husman *et al.,* 2006)

Sample date Week in 1994	*E. coli* CFP/l	*Campylobacter* MPN/l	Ratio*
7	$4.2 \cdot 10^{3}$	23	$5.5 \cdot 10^{-3}$
8	$3.2 \cdot 10^{3}$	500	0.16
9	$2.1 \cdot 10^{4}$	500	$2.4 \cdot 10^{-2}$
10	$6.8 \cdot 10^{3}$	500	$7.4 \cdot 10^{-2}$
11	$2.8 \cdot 10^{4}$	90	$3.2 \cdot 10^{-3}$
13	$4.6 \cdot 10^{3}$	1100	0.24
14	$1.5 \cdot 10^{4}$	500	$3.3 \cdot 10^{-2}$
15	$1.6 \cdot 10^{4}$	90	$5.6 \cdot 10^{-3}$
16	$2.9 \cdot 10^{3}$	40	$1.4 \cdot 10^{-2}$
17	$9.5 \cdot 10^{3}$	40	$4.2 \cdot 10^{-3}$
29	$4.8 \cdot 10^{3}$	23	$4.8 \cdot 10^{-3}$
31	$6.8 \cdot 10^{3}$	50	$7.4 \cdot 10^{-2}$
33	$6.0 \cdot 10^{3}$	40	$6.7 \cdot 10^{-3}$
35	$4.4 \cdot 10^{3}$	23	$5.2 \cdot 10^{-3}$
37	$4.7 \cdot 10^{3}$	9	$1.9 \cdot 10^{-3}$
39	$6.8 \cdot 10^{3}$	23	$3.4 \cdot 10^{-3}$
41	$3.2 \cdot 10^{3}$	500	0.16
45	$3.2 \cdot 10^{3}$	90	$2.8 \cdot 10^{-2}$
46	$3.9 \cdot 10^{3}$	40	$1.0 \cdot 10^{-2}$
48	$3.5 \cdot 10^{3}$	70	$2.0 \cdot 10^{-2}$
50	$7.5 \cdot 10^{3}$	150	$2.0 \cdot 10^{-2}$
51	$5.7 \cdot 10^{3}$	90	$1.6 \cdot 10^{-2}$
Mean	$7.8 \cdot 10^{3}$	220	$4.1 \cdot 10^{-2}$
SD	$6.5 \cdot 10^{3}$	280	$6.3 \cdot 10^{-2}$
Median	$5.3 \cdot 10^{3}$	90	$1.5 \cdot 10^{-2}$
N	22	22	22
2.5 percentile	$3.1 \cdot 10^{3}$	16	$2.6 \cdot 10^{-3}$
97.5 percentile	$2.4 \cdot 10^{4}$	780	0.20
Minimum	$2.9 \cdot 10^{3}$	9	$1.9 \cdot 10^{-3}$
Maximum	$2.8 \cdot 10^{4}$	1100	0.24

* Ratio of *Campylobacter* to *E. coli*

Table 5.9 Concentrations of enteroviruses and faecal (= thermotolerant) coliforms in material outside drinking-water mains in the United States (from Karim et al., 2000, 2003).

		Soil samples			Shallow groundwater samples		
		Faecal coliforms	PCR Enterovirus	Ratio[b]	Faecal coliforms	PCR Enterovirus	Ratio[b]
System code	Sample received	MPN/100 g[a]	N/100 g[a]		MPN/100 ml[a]	N/100 ml[a]	
B-01	27-1-99	ND	ND		ND	ND	
A-01	11-2-99	ND	0		ND	0	
B-02	11-2-99	ND	ND		ND	0	
A-02	3-3-99	0	ND[b]		0	0.3[b]	***0.3***
A-03	25-3-99	140	ND		0	0	0
A-04	26-3-99	170	ND		7	0	0
A-05	7-4-99	20	ND		0	0	0
A-06	7-4-99	0	6	***6***	0	0.3	***0.3***
B-03	21-4-99	0	0	0	0	0.3	***0.3***
A-07	6-5-99	0	0	0	30	0	0
A-08	11-5-99	0	0	0	0	0	0
A-09	11-5-99	0	ND		0	0.3	***0.3***
A-10	13-5-99	0	ND		0	0.3	***0.3***
A-11	13-5-99	0	ND		0	0	0
A-12	21-5-99	800	0[b]	0	0	0.3	***0.3***
B-04	26-5-99	0	0	0	0	0.3	***0.3***
B-05	28-7-99	0	0	0	0	0.3[b]	***0.3***
I-01	4-8-99	140	0	0	0	0.3	***0.3***
E-01	11-8-99	0	6	***6***	0	0	0
E-02	12-8-99	0	6	***6***	0	0.3	***0.3***
A-13	17-8-99	300	6	**$2.0.10^{-2}$**	2	0.3	**0.15**
A-14	17-8-99	1100	ND		70	0.3	**$4.3.10^{-3}$**
D-01	18-8-99	90	6	**$6.7.10^{-2}$**	300	0	0
D-02	19-8-99	40	ND		23	0	0
D-03	24-8-99	0	0	0	50	0	0
D-04	25-8-99	0	0	0	17	0	0
D-05	7-9-99	$1.6.10^{4}$	ND		> 1600	0	0
H-01	8-9-99	40	0	0	ND	ND	
F-01	9-9-99	120	ND		ND	ND	
E-03	14-9-99	0	0	0	2	0	0
D-06	13-9-99	40	0	0	50	0.3	**$6.0.10^{-3}$**
D-07	14-9-99	20	ND		0	0.3	***0.3***
G-01	15-9-99	500	0[b]	0	> 1600	ND[b]	
			Culturable enteric virus N/100 g[a]			Culturable enteric virus N/100 ml[a]	
A-02	3-3-99	0	6		0	0.3	
A-12	21-5-99	800	6	$7.5.10^{-3}$	0	0	
B-05	28-7-99	0	0		0	0.3	
G-01	15-9-99	500	6	$1.2.10^{-2}$	> 1600	0.3	$1.9.10^{-4}$

[a] Analysed volume: faecal coliforms: 50 g soil or 50 ml water; PCR: 6.7 g soil or 333 ml water; ND = not determined.

[b] Ratios in bold italic font: faecal coliforms absent, ratio based on 0.5 * detection limit = 1 CFP/100 ml or 1 CFP/100 g.
Cells with lined border and bold font: ratios of both PCR and fecal coliforms above detection limit.
Cells with grey background: culturable enteroviruses found in same samples.

5.6.5 Calculating the risks of exposure

The annual expected[7] exposure to pathogens can be calculated as

$P_{exp} = P_I * P_{ED}(E * D) * P_R(R)$

Where
- P_{exp} = The probability (per person per year) of an inhabitant of the Netherlands being exposed to a pathogen or (when the probability is higher than 1), the expected number of pathogens per person per year.
- P_I = The probability of being an inhabitant of a contaminated area (estimated $1.7 \cdot 10^{-3}$ per person per year). This probability determines the
- P_{ED} = The probability of being an inhabitant of a contaminated area exposed to a certain intensity of *E. coli* concentrations (duration of the event and concentrations of *E. coli*). This probability determines the location on the y-axis of the graphs in Figure 5.7.
- P_R = The probability of the event the inhabitant is being affected by is being caused by a contamination source with a certain pathogen to *E. coli* ratio (similar to ratios in surface water). This probability determines the location on the x-axis in Figure 5.7.
- E = *E. coli* concentrations during the event
- D = Duration of the event
- R = Pathogen to *E. coli* ratio during the event

As presented in paragraph 5.6.1 the risk of being an inhabitant of a contaminated area of a supply zone in the Netherlands is estimated at $1.7 \cdot 10^{-3}$ per person per year[8]. In this paragraph the risk of exposure to the index pathogens is estimated for the inhabitants that are affected by an event. The calculations are performed using the Tier 2 method described in paragraph 5.5.6, using pathogen to *E. coli* ratios found in surface water (Tables 5.7 and 5.8)

The resulting values are retrospectively expected total 'standard' pathogen exposures per person during each event, assuming a fixed consumption of 1 litre per person per day as a 'standard' consumption. For expected exposures below 1, these data are best interpreted as the probability of being exposed to 1 pathogen during the event. For expected exposures over 1, these data are best interpreted as the expected number of pathogens being exposed to during the total event.

Cumulative Probability Density Functions of the probabilities
Figure 5.7 shows the means as well as the 2.5 and 97.5 percentiles of the expected standard exposure per event. These statistical index numbers per event are sorted by the mean exposure and presented in Cumulative Probability Density Functions (CDF). The y-axis increments per event in the CDF are the percentages of affected inhabitants during that event relative to the total of 185,000 inhabitants affected. The x-value at a y-value of 50% is the highest probability of pathogen exposure for 50% of c. 185,000 = c. 93,000 inhabitants.

When affected by a contamination event, Figure 5.7 shows the expected standard exposure (or probability of being exposed to 1 pathogen) when consuming 1 litre of drinking-water per day during the event. The y-value indicates the probability of the intensity (mean exposure) of the event. The x-value indicates the probability of the exposure during the event.

[7] Assuming identical probabilities in the future as were calculated from recorded data of past events.
[8] Note the under-recording of events occurrence and details reported by the participating companies

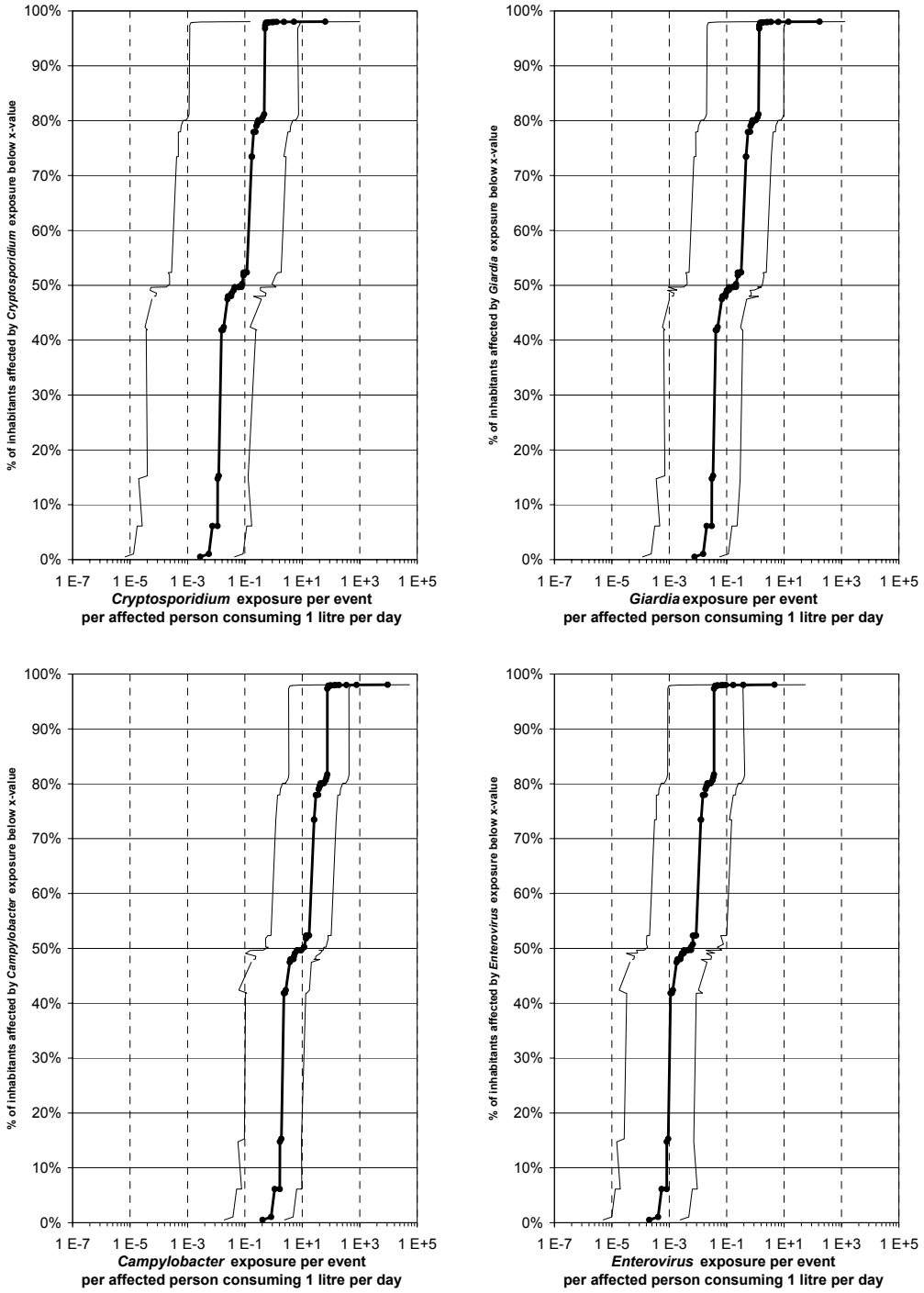


Figure 5.7 11 Retrospectively expected standard exposure to pathogens (per person per event drinking 1 litre per day) estimated from detectable *E. coli* concentrations during 50 non-outbreak faecal contamination events recorded by 7 water companies in the Netherlands from 1994 through 2003. Presented are the mean, 2.5 and 97.5 percentiles of the exposure per event, CDF percentiles (y-axis position of the event) are cumulative percentages of the total of c.185,000 inhabitants estimated to be affected on a population of c. 11 million people served by the 7 water companies.

The mean standard exposure per event is determined by the mean concentration of *E. coli* in the drinking-water during the event and the mean pathogen to *E. coli* ratio in the assumed contamination source (in this case surface water ratios from Table 5.7 and 5.8). The width of the 95% confidence interval of the pathogen exposure (indicated by the range between the 2.5 and the 97.5 percentiles) is determined by the variation of the *E. coli* concentrations in the samples collected during the event and the variation of the pathogen to *E. coli* ratios.

Expected annual exposure

Including the probability of c. $1.7 \cdot 10^{-3}$ per person per year to be affected by a contamination event, the ranges of probability of standard exposure to pathogens is presented in table 5.10. Taking into account the assumption that the pathogen to *E. coli* ratios in the contamination sources could be any of the ratios found in the limited number of surface water sources, exposure to *Campylobacter* would have been highest during all events, followed by exposure to the other index pathogens *Giardia*, *Cryptosporidium* and enteroviruses.

Table 5.10 Maximum of expected exposure per person per year to index pathogens due to events, including the probability of being an inhabitant of a contaminated area ($1.7 \cdot 10^{-3}$ per person per year). The expected exposures are presented for 12 combinations of 4 percentiles of the events (expressed in numbers of inhabitants affected) and 3 statistical index numbers of standard exposure per event. Risks are based on a standard consumption of 1 litre per person per day. The expected exposures below 1 can be interpreted as probabilities of exposure to 1 index pathogen.

Fraction of events as % of affected inhabitants	Index numbers per event	Maximum risk of exposure			
		Cryptosporidium	*Giardia*	*Campylobacter*	Enterovirus
Minimum (c. 50,000)	P2.5	$1.1 \cdot 10^{-8}$	$2.0 \cdot 10^{-7}$	$3.2 \cdot 10^{-5}$	$8.3 \cdot 10^{-9}$
	mean	$4.7 \cdot 10^{-6}$	$1.3 \cdot 10^{-5}$	$6.9 \cdot 10^{-4}$	$3.4 \cdot 10^{-7}$
	P97.5	$7.3 \cdot 10^{-5}$	$9.7 \cdot 10^{-5}$	$4.0 \cdot 10^{-3}$	$4.0 \cdot 10^{-6}$
Median (c. 93,000)	P2.5	$3.6 \cdot 10^{-7}$	$6.3 \cdot 10^{-6}$	$9.0 \cdot 10^{-4}$	$2.6 \cdot 10^{-7}$
	mean	$1.3 \cdot 10^{-4}$	$3.6 \cdot 10^{-4}$	$1.8 \cdot 10^{-2}$	$9.7 \cdot 10^{-6}$
	P97.5	$1.8 \cdot 10^{-3}$	$2.9 \cdot 10^{-3}$	$9.3 \cdot 10^{-2}$	$9.6 \cdot 10^{-5}$
90-percentile (c. 167,000)	P2.5	$2.0 \cdot 10^{-6}$	$3.4 \cdot 10^{-5}$	$5.7 \cdot 10^{-3}$	$1.5 \cdot 10^{-6}$
	mean	$8.2 \cdot 10^{-4}$	$2.2 \cdot 10^{-3}$	0.12	$6.1 \cdot 10^{-5}$
	P97.5	$1.2 \cdot 10^{-2}$	$1.6 \cdot 10^{-2}$	0.71	$6.7 \cdot 10^{-4}$
Maximum (c. 185,000)	P2.5	$2.6 \cdot 10^{-4}$	$4.5 \cdot 10^{-3}$	0.73	$1.9 \cdot 10^{-4}$
	mean	0.11	0.29	16	$7.8 \cdot 10^{-3}$
	P97.5	1.7	2.2	92	$9.1 \cdot 10^{-2}$

Using the available data, assumptions and calculation models in this paragraph, the exposure risks from Table 5.10 for example would result in the following conclusions:

- The estimated median standard risk of exposure *Campylobacter* due to contamination events in the Netherlands ranges from $9.0 \cdot 10^{-4}$ to $9.3 \cdot 10^{-2}$ (95% confidence interval) with a mean of $1.8 \cdot 10^{-2}$ per person per year depending on the actual *Campylobacter* to *E. coli* ratio, assuming ratios similar to those found in surface water.
- The estimated maximum standard risk of exposure to enterovirus ranges from $4.5 \cdot 10^{-7}$ to $6.0 \cdot 10^{-3}$ (95% confidence interval) with a mean of $9.7 \cdot 10^{-4}$ per person per year.

In Chapter 8, the possible risks of infection to consumers caused by these exposures are calculated and discussed.

6.7 BACKGROUND CONCENTRATIONS

Water companies verify the effectiveness of measures to prevent contamination of drinking-water during distribution by systematically sampling distribution networks periodically and after operations. The statutory monitoring of *E. coli* in distributed drinking-water is conducted in all Member States of the EU. The EU Drinking-water Directive prescribes the sampling frequency for *E. coli* from taps in premises. The sampling frequency depends on the volume of water distributed in the supply zone (for example a supply zone of 1000 m^3/day has to take at least 4 samples per year, a supply zone of 10,000 m^3/day at least 34 samples and a system of 100,000 m^3/day 304 samples). *E. coli* is found very occasionally in tap water in EU Member States (Table 5.11).

The rare detections of *E. coli* in tap water, where a repeat sample is negative, could be the result of (a combination of):

1. Artificial contamination during sampling or in the laboratory. Although this cannot be excluded, QA in the laboratory is rigorous and as long as the QA does not indicate that errors have occurred, the results have to be considered to represent the tap water quality.
2. Low level continuous or semi-continuous contamination of tap water as a result of insufficient treatment or ingress/leaks in the distribution network.
3. Unnoticed contamination events.

If 2 would be the dominant reason, an increase in the sample volume would result in an increase in the frequency of *E. coli* detection. This hypothesis was tested.

Over 300 large volume samples (6 to 200 litre, as compared to the statutory 100 ml) were collected from supply zones in Germany, the Netherlands (Nobel et al., 2005) and the United Kingdom and analysed for the presence of *E. coli* (Table 5.12). Samples were taken in urban and rural supply zones, in supply zones of groundwater and treated surface water, both in chlorinated and unchlorinated supply zones and in sampling sites at different parts of the distribution systems.

No *E. coli* were found in any of the samples collected.

The results show that a background level of *E. coli*, if present at all, is very low in the investigated distribution systems; between $< 1.4 \cdot 10^{-5}$ and $< 7.8 \cdot 10^{-5}$ CFP per 100 ml, depending on the total volume analysed (Table 5.12). This strongly suggests that the occasional presence of *E. coli* in samples collected as a part of periodical monitoring (Table 5.11) or collected after operations is the result of a contamination event.

Table 5.11 Incidence of *E. coli* (or thermotolerant coliforms) in parts of The United Kingdom, France and The Netherlands from 2001 to 2003.

Country		The United Kingdom			La France			Nederland		
Water companies in table		26 in England and Wales			2			9		
Origin of data		www.dwi.gov.uk			Water companies (table 5.15)			Water companies (table 5.15)		
Year		2001	2002	2003	2001	2002	2003	2001	2002	2003
Number of inhabitants (million) supplied by water companies		52.7	52.7	53.3	27.5	27.1	27.8	12.1	12.1	10.4
Volume distributed annually (1.10^6 m^3)		5709	5768	5840	2681	2681	2681	845	846	766
Percentage of all inhabitants per country in table		89%	89%	90%	46%	46%	47%	74%	74%	64%
Mean number of inhabitants per supply zone		22,863	23,023	23,699	387,324	381,690	391,549	98,374	97,581	100,971
Mean annual volume per supply zone (1.10^6 m^3)		2.5	2.5	2.6	37.8	37.8	37.8	6.9	6.8	7.4
Mean annual volume distributed per inhabitant (m^3)		108	109	110	97	99	96	70	70	74
Number of systems investigated	Treatment plants	1386	1344	1303	4328	4256	4248	131	130	110
	Distribution reservoirs	4746	4691	4669	28	28	28	41	41	20
	Supply zones	2305	2289	2249	71	71	71	123	124	103
Number of samples investigated	Treatment plants	164,462	163,779	160,528	14,003	17,153	23,404	11,690	12,065	10,957
	Distribution reservoirs	245,158	243,315	241,105	3401	4016	3835	533	533	260
	Supply zones	149,702	149,393	148,523	37,343	48,163	58,632	34,985	34,518	33,732
Percentage of samples in which thermotolerant coliforms or *E. coli* were found	Treatment plants	0.018%	0.012%	0.017%	1.09%	0.74%	0.38%	0.09%	0.03%	0.01%
	Distribution reservoirs	0.025%	0.023%	0.014%	0.97%	0.67%	0.34%	0.56%	0.19%	0.38%
	Supply zones	0.040%	0.039%	0.031%	1.02%	0.62%	0.38%	0.11%	0.10%	0.09%

Table 5.12 Program for large volume sampling to detect the background level of *E. coli* in supply zones

	Germany[a]	Netherlands	United Kingdom
Sampling period	July 2003 - Jan. 2004	Dec. 2003 – Oct. 2004	July 2003 – July 2004
Sampling intervals	All sites weekly	c. 2 per 2 weeks 5	All sites biweekly
No. supply zones	1	18	2
No. sampling sites	5		7
No. samples	5 * 26 = 130	44	7 * 26 = 182
Sample volumes (l)			
- Minimum	6	30	10
- Median	10	200	10
- Maximum	10	200	10
- Total	1280	7062	1820
Samples with *E. coli*	0	0	0
Detection limit (CFP/l)	$7.8 \cdot 10^{-4}$	$1.4 \cdot 10^{-4}$	$5.5 \cdot 10^{-4}$
Chlorine residual	none	zone 2: ND[b] other: none	Zone 1: 0.05–0.55[c]

[a] All three treatment plants of the supply zone were sampled (10 l) weekly for 3 consecutive weeks in August 2003 and 3 consecutive weeks in January 2004. No *E. coli* or total coliforms were found.

[b] ND = not determined.

[c] 95% of all samples

6.8 PATHOGEN CONCENTRATIONS BASED ON DETECTED *E. COLI* CONCENTRATIONS

Assuming, from paragraph 5.7, the background concentrations (baseline) of *E. coli* in distributed water are undetectable even with large volumes, every *E. coli* found represents a contamination event (also called 'event'). This assumption offers the opportunity to use the frequency of finding *E. coli* as a best estimate of the probability of being exposed to faecal pathogens. The concentrations of *E. coli* can be used as a best estimate of the concentrations of pathogens, resulting in exposure of inhabitants, during these events. As in the evaluation of the outbreak case (paragraph 5.5) and the evaluation of 50 events (paragraph 5.6), pathogen to *E. coli* ratios found in surface water will be used. In the discussion (paragraph 5.9) the uncertainties of the calculation model will be discussed.

5.8.1 *E. coli* in supply zones of 'catchment-to-tap' systems

The frequency and concentrations of thermotolerant coliforms and *E. coli* in samples of finished water and distributed water of the 'catchment-to-tap system' (CTS) treatment plants (described in Chapter 3 and 4) were evaluated to complete the catchment-to-tap approach of estimating pathogen concentrations. As described earlier in this chapter, the approach for contaminations during distribution needs to be different, using other data to estimate pathogen concentrations.

Although from the start it was clear that the frequency of *E. coli* in the distributed water of the CTSs would be too low to conclude anything from differences in CTS characteristics, the characteristics of the distribution systems in the CTS supply zones are presented in Table 5.13.

The highest frequency of samples containing thermotolerant coliforms or *E. coli* was found in tap water in the combined supply zone of CTSs 2 and 9 (table 5.14), treatment plants that do not dose a disinfectant to the finished water, causing an absence of a disinfectant residual in the distributed water (table 5.13). The absence of a disinfectant residual favours the detection of *E. coli*, as these bacteria are relatively sensitive to hypochlorite ions and radicals (see Chapter 4, paragraph 4.2.5). The low leakage rate in the supply zone (3-5%) is an indication of a high maintenance level and there is no cause to assume lower levels of hygiene during operations than in other CTSs. Although other CTSs also have a low or absent disinfectant residual (CTS 7 and 11) and the number of CTSs is too low to conclude anything with a statistical significance, the theory is (weakly) corroborated by the frequency of *E. coli* in finished water. Again, the number of CTSs is too low to be able to conclude more from these results. As the frequency of *E. coli* found is very low in most CTSs, a calculation of pathogen concentrations is not performed.

Table 5.13 Characteristics of the distribution systems in the supply zones of the 'catchment-to-tap' system treatment plants as described in Chapters 3 and 4.

Catchment-to-tap system code	Number of inhabitants in supply zone	Volume distributed		Disinfectant	
		Total	Per person	Type and dose	Residual
		10^6 m^3/yr	l/day	mg/l free Cl_2	mg/l free Cl_2
1	224,168	13.3	163	Cl_2	0.1 - 0.7
2 + 9	883,647	72.8	226	None	0
3	34,000	3.2	259	ClO_2 0.4 – 0.8	0.3
4	18,000	3.8	578	ClO_2 0.35 – 0.55	0.3
7	571,600	49.7	238	ClO_2 0.05	0
8	50,000	6.0	328	Cl_2 1.9 ± 0.4	1.7 ± 0.4
10	47,600	4.1	238	Cl_2 0.5	< 0.5
11	300,000	22.0	201	Cl_2 + ClO_2 0.25	< 0.01 – 0.25
12	24,300	1.9	214	Cl_2 0.7	< 0.7
Total	2,153,315	177	225		

Catchment-to-tap system code	Length and materials of trunk mains and mains[a]									Leakage rate
	Length	CI	CIL	ST	AC	Con	PVC	PE	Cu	
	km	%	%	%	%	%	%	%	%	%
1		75%				5%	20%			16.5%
2 + 9	2612	44%		2%	4%	3%	33%	9%	6%	3 – 5%
3		50%		1%			40%	9%		18.7%
4		6%		80%			4%	5%		17.5%
7	1648	57%	53%							< 5%
8						Trunks			Mains	14%
10		12%		39%			49%			27%
11	1200	---100% ---								12%
12		40%			31%		23%	5%		23%

[a] CI = cast iron; CIL = lined cast iron; ST = steel; AC = asbestos-cement; Con = concrete; PVC = polyvinylchloride; PE = polyethene; Cu = copper

Table 5.14 Frequency of samples containing thermotolerant coliforms or *E. coli* in the finished water and supply zones of the 'catchment-to-tap' system treatment plants as described in Chapters 3 and 4.

Catchment-to-tap system code	Sample Volumes Ml	Analysis method Before 2002	Analysis method Starting 2002	Results from finished water Total no. of samples	Results from finished water No. samples > 0 CFP	Results from finished water Mean CFP/l
1	100	coli44	*E. coli*	1095	0	$< 1.8 \cdot 10^{-3}$
2 + 9	100	coli44	*E. coli*	2190	1	$9.1 \cdot 10^{-4}$
3	100	coli44	*E. coli*	91	0	< 0.11
4	100	coli44	*E. coli*	56	0	< 0.18
7	100	*E. coli*	*E. coli*	3455	0	$< 2.9 \cdot 10^{-3}$
8	100	*E. coli*	*E. coli*	159	0	$< 6.3 \cdot 10^{-2}$
10	100	coli44	*E. coli*	125	0	$< 8.0 \cdot 10^{-2}$
11	100	*E. coli*	*E. coli*	3897	0	$< 2.6 \cdot 10^{-3}$
12	100	coli44	*E. coli*	39	0	< 0.26
Total				11,107	1	$1.8 \cdot 10^{-4}$

Catchment-to-tap System Code	Results from distribution reservoirs Total no. of samples	Results from distribution reservoirs No. samples > 0 CFP	Results from distribution reservoirs Mean CFP/l	Results from tap water Total no. of samples	Results from tap water No. samples > 0 CFP	Results from tap water Mean CFP/l
1	545	1	$1.8 \cdot 10^{-2}$	1283	1	0.13
2 + 9	Included in tap water samples			8282	11	0.58
3				218	0	$< 4.6 \cdot 10^{-2}$
4	271	0	$< 3.7 \cdot 10^{-2}$	33	0	< 0.30
7	1918	0	$< 5.2 \cdot 10^{-3}$	3329	0	$< 3.0 \cdot 10^{-3}$
8	99	0	< 0.10	674	0	$< 1.5 \cdot 10^{-2}$
10	2	0	< 5	373	0	$< 2.7E \cdot 10^{-2}$
11				798	0	$< 1.3E \cdot 10^{-2}$
12	136	3	4.5	79	0	< 0.13
Total	2971	4	0.21	15,069	12	0.33

5.8.2 *E. coli* periodically detected in other supply zones

As the number of CTSs and the frequency of occurrence of *E. coli* in both finished water as well as distributed water are far too low to calculate meaningful pathogen concentrations from the *E. coli* concentrations, *E. coli* occurrence and concentrations were evaluated from other supply zones to estimate exposure to pathogens.

The results of over 400,000 samples collected in 3 years from 211 supply zones with c. 45 million inhabitants were supplied by 22 water companies from 5 countries (Table 5.15). In total, 391 samples (0.34%) of finished water, 80 samples (0.36%) of water leaving distribution reservoirs and 1031 samples (0.38%) of tap water contained *E. coli* or thermotolerant coliforms. These mean percentages might imply that there is

little or no change in *E. coli* occurrence from finished water to tap water, but detailed evaluation would show that in individual supply zones major changes may occur.

As the occurrence of *E. coli* is considered to indicate a contamination event that is an event (event) and not a baseline condition caused by a background concentration, the percentages of samples containing *E. coli* represent the probability of faecal contamination events occurring. In the databases of water companies, *E. coli* concentrations are not always marked as 'first sample' or 'repeat sample', so events that consisted of more than one sample containing *E. coli* were not identified. All *E. coli* concentrations higher than 0 CFP/100 ml were evaluated as individual events or series of separate events per supply zone in this paragraph.

Table 5.15 Evaluated numbers of water companies, supply zones, inhabitants and *E. coli* samples collected during a 3 year period between 2000 and 2003

Country	Water companies	Supply zones	Inhabitants (in million and % per country)	Number of samples collected		
				Finished water after treatment	Distribution reservoirs	Tap water from premises
Australia	1	1	0.05 (0.3%)	159	99	674
France	2	72	27.5 (46%)	54,560	11,252	144,138
Germany	9	13	2.8 (3.4%)	20,737	8820	12,530
Netherlands	9	125	12.1 (74%)	39,454	1599	107,593
Un. Kingdom	1	1	0.22 (0.4%)	1095	545	1283
Total	22	211	42.7[a]	116,005	22,315	266,218

[a] Number of inhabitants in the 209 supply zones for which tap water data were supplied. The number of inhabitants of all 211 supply zones for which finished water data were supplies is c. 45 million

The concentrations of samples of tap water containing *E. coli* were up to 5000 CFP per 100 ml, 96% of these samples were below 100 CFP per 100 ml, 80% were below 10 CFP per 100 ml and 56% were 1 or 2 CFP per 100 ml (Figure 5.8).

The frequency of *E. coli* and thermotolerant detection in samples seems to be highest from July through October (Figure 5.9) and seems to decrease from 2001 (54-65 per month) to 2003 (c. 31-36 per month), possibly as an effect of the EU-wide change of analysis methods from thermotolerant coliforms to *E. coli*, diminishing the frequency of finding false-positives possibly caused by multiplication of thermotolerant coliforms such as *Klebsiella* spp. in drinking-water (Ashbolt *et al.*, 2001). The median concentrations per month stayed between 1 and 3.5 CFP/100 ml throughout the period without noticeable peaks, but monthly 90-percentiles seem to peak in September. These 90-percentiles seem to increase dramatically, although the peaks in June and September 2003 are largely caused by the occurrence of samples containing 5000 CFP of *E. coli* per 100 ml (3 samples in two supply zones in June and 4 samples in one supply zone in September).

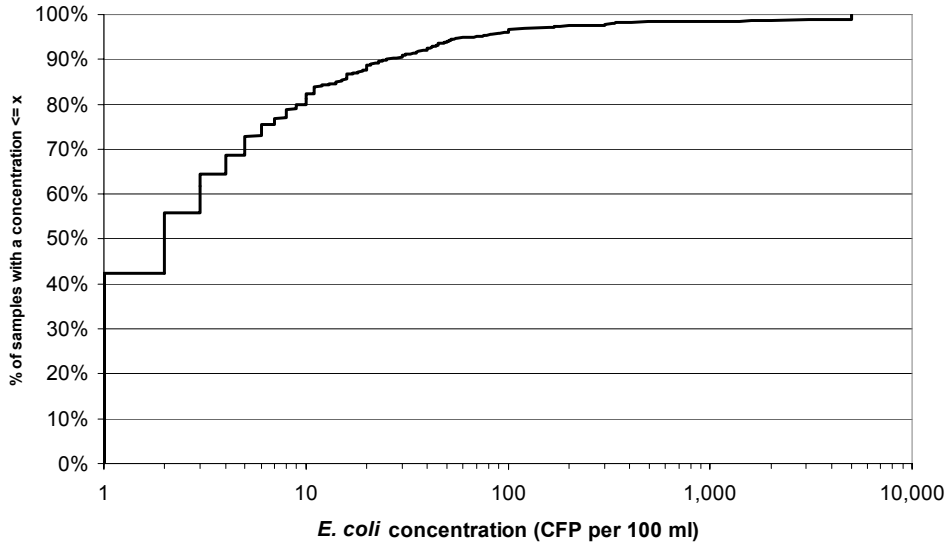


Figure 5.8 CDF of *E. coli* concentrations in all 1031 samples containing > 0 CFP per analysed volume, collected from taps in premises (209 supply zones, 43 million inhabitants, three years from 2000-2003)

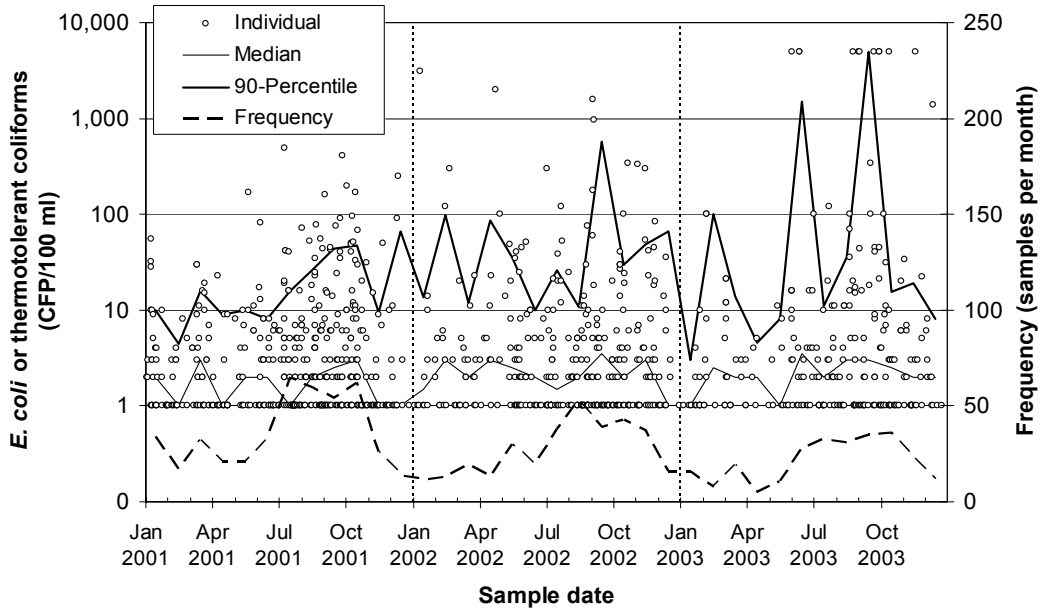


Figure 5.9 *E. coli* concentrations in all 1028 samples containing > 0 CFP per analysed volume, collected from 2001 through 2003 from taps in premises in 209 supply zones supplying 43 million inhabitants. Medians, 90 percentiles and frequencies are calculated per month. Note: from 21 supply zones (1.7 million inhabitants) in the Netherlands data were supplied for the period 2000 through 2002, causing the data from 2003 to represent 188 supply zones and c. 41 million inhabitants (4% less than from 2002).

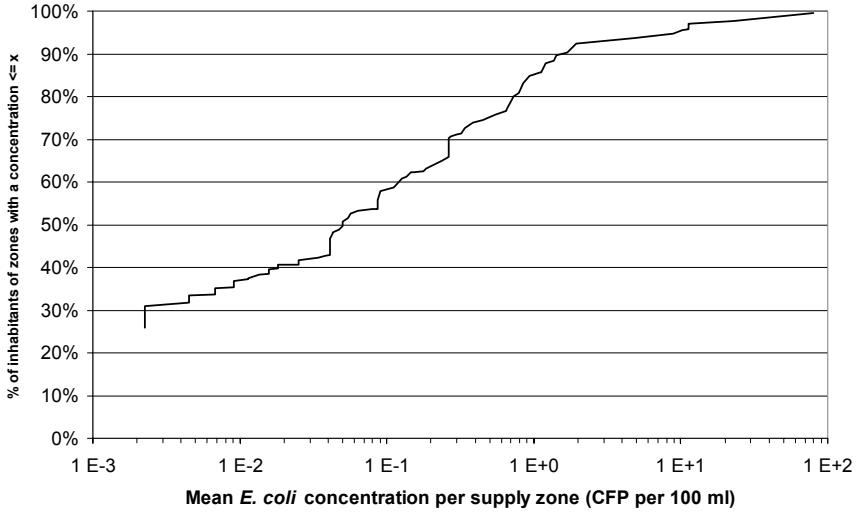


Figure 5.10 CDF of mean *E. coli* concentration of tap water samples per supply zone. CDF-percentiles represent fractions of the total number of inhabitants (209 supply zones, 43 million inhabitants, three years from 2000-2003)

The mean concentration of *E. coli* per supply zone ranged up to 80 CFP per 100 ml (Figure 5.10), the latter concentration being the result of 7 samples collected within 2 months that each contained 5000 CFP of *E. coli* per 100 ml. In the rest of the period (2001-2003), the mean concentration of the 3932 samples in that supply zone was $1.5.10^{-3}$ CFP per 100 ml. The mean concentrations are below 1 CFP per 100 ml for 85% of the population served, below 0.1 CFP per 100 ml for 58% of the population and below 0.01 CFP per 100 ml for 37% of the population. *E. coli* concentrations are below detection limit in all samples for 26% of the population.

5.8.2 *E. coli* detected after invasive operations

Many contamination events are detected after operations that include opening of the mains. The common cause of these events is a combination of a contamination followed by incomplete cleaning.

Common causes of contaminations during operations are:

Ingress into depressurised mains:

- During breaks and leaks, via these openings.
- During operations, via undetected leaks.
- During operations, via opened hydrants (when unaware of the depressurised condition or the negative effects thereof).
- During operations, via the opened main (inadequate hygiene).

Ingress into pressurised mains:

- During operations, via opened hydrants (wrong sequence of opening hydrant and top-piece valves; top-piece valve should always be opened first).

Common causes of incomplete cleaning after operations are:

No cleaning, when falsely assuming the absence of a contamination:

- In mains sections that were depressurised during operations but were not repaired (these may contain undetected leaks).
- After operations were hygiene procedures was considered adequately followed to prevent contamination.

Insufficient flushing velocities:

- When flushing velocities are not calculated and attained by closing appropriate valves.
- When mains are too large to flush.
- When swabbing (pigging) is impossible (presence of valves, absence of entry and exit facilities).
- When contamination material is accumulated in mains couplings.

Insufficient disinfection:

- No disinfection, as refraining from disinfection often does not lead to detecting *E. coli* in 100 ml samples after operations.
- Low disinfectant concentrations.
- Low contact times.

Contaminations that lead to operations or occur during operations are more likely to be detected than other contaminations, as samples are collected 'on the right place at the right time' or at least more near to the site and time of contamination than in other cases of contamination. Notwithstanding this theoretical advantage, the detection may be hampered however:

Samples may be collected at the wrong time:

- Too quick after the operations, causing contaminations not to be spread throughout the mains (especially occurring in large mains).
- Too late after the operations, causing the contaminations to be flushed away.

Samples may be collected at the wrong site:

- At the beginning (upstream) of the mains section that is possibly contaminated, in stead of at the end (downstream).
- At too few locations, therefore missing the contaminated area.

Three water companies in the Netherlands provided results of water quality testing in 47 supply zones (5.8 million inhabitants) after operations that were followed by immediate commencement of supply. Of 16,047 samples collected after operations, 91 contained *E. coli* or thermotolerant coliforms (0,57%). The maximum concentration of *E. coli* found was 900 CFP/100 ml and the mean concentration of samples containing > 0 CFP per sample volume was 22 CFP/100 ml. The mean concentration of all samples was 0.13 CFP/100 ml.

5.8.3 Pathogen exposure during incidental occurrence of *E. coli*

The basics of calculating the infection risk are similar to those in paragraph 5.5.6. The following calculations were performed per supply zone:

1. All *E. coli* (and thermotolerant coliform) concentrations > 0 CFP per sample were selected from the monitoring period (in most cases three years from 2000 through 2003). If none of the samples contained *E. coli*, the concentration of one sample was considered to have been 0.1 CFP per 100 ml[9].
2. The PDFs of the *E. coli* concentrations > 0 CFP per sample and the pathogen to *E. coli* ratios found in surface water (Table 5.7 and 5.8) were bootstrapped 100,000-fold, resulting in a PDF of 100,000 pathogen concentrations.
3. From the PDF of 100,000, N random draws of 10,000 pathogen concentrations were summed, where N = the mean annual number of *E. coli* concentrations > 0 CFP per sample (N is rounded up to the nearest integer).

The result is called the retrospectively expected exposure to pathogens (P_{exp}) per supply zone, i.e. the estimated yearly numbers of pathogens that inhabitants affected by the faecal contamination events were exposed to.

The mean probability (P_I) of being an inhabitant of a faecally contaminated area of the total supply zone is estimated by the weighed[10] fraction of samples containing *E. coli* or thermotolerant coliforms.

The probabilities and exposures are estimated from *E. coli* occurrence and concentrations on different parts and operating conditions in the supply zones:

- Finished water from treatment plants: mean $P_I = 5.7 \cdot 10^{-3}$; P_{exp} in Figure 5.11.
- Drinking-water from distribution reservoirs: mean $P_I = 1.8 \cdot 10^{-2}$; P_{exp} in Figure 5.12.
- Drinking-water from taps in premises: mean $P_I = 4.7 \cdot 10^{-3}$; P_{exp} in Figure 5.13.
- Drinking-water collected from mains after operations directly followed by commencement of supply: mean $P_I = 6.7 \cdot 10^{-3}$; P_{exp} in (Figure 5.14).

[9] The resulting pathogen concentration is an estimate of the maximum pathogen concentration, which is determined by the number of samples collected in a supply zone. Thus, for supply zones in which different numbers of samples were collected, none of which contained *E. coli,* the maximum *E. coli* concentration presented will be fully dependent of the number of samples collected.

[10] Total of affected inhabitants per supply zone (fraction of samples containing *E. coli* * total number of inhabitants) divided by the total number of inhabitants of all supply zones. If *E. coli* is found in none of the samples, the maximum number of affected inhabitants is estimated (total inhabitants divided by the number of samples, i.e. assuming a maximum of 1 samples containing *E. coli*).

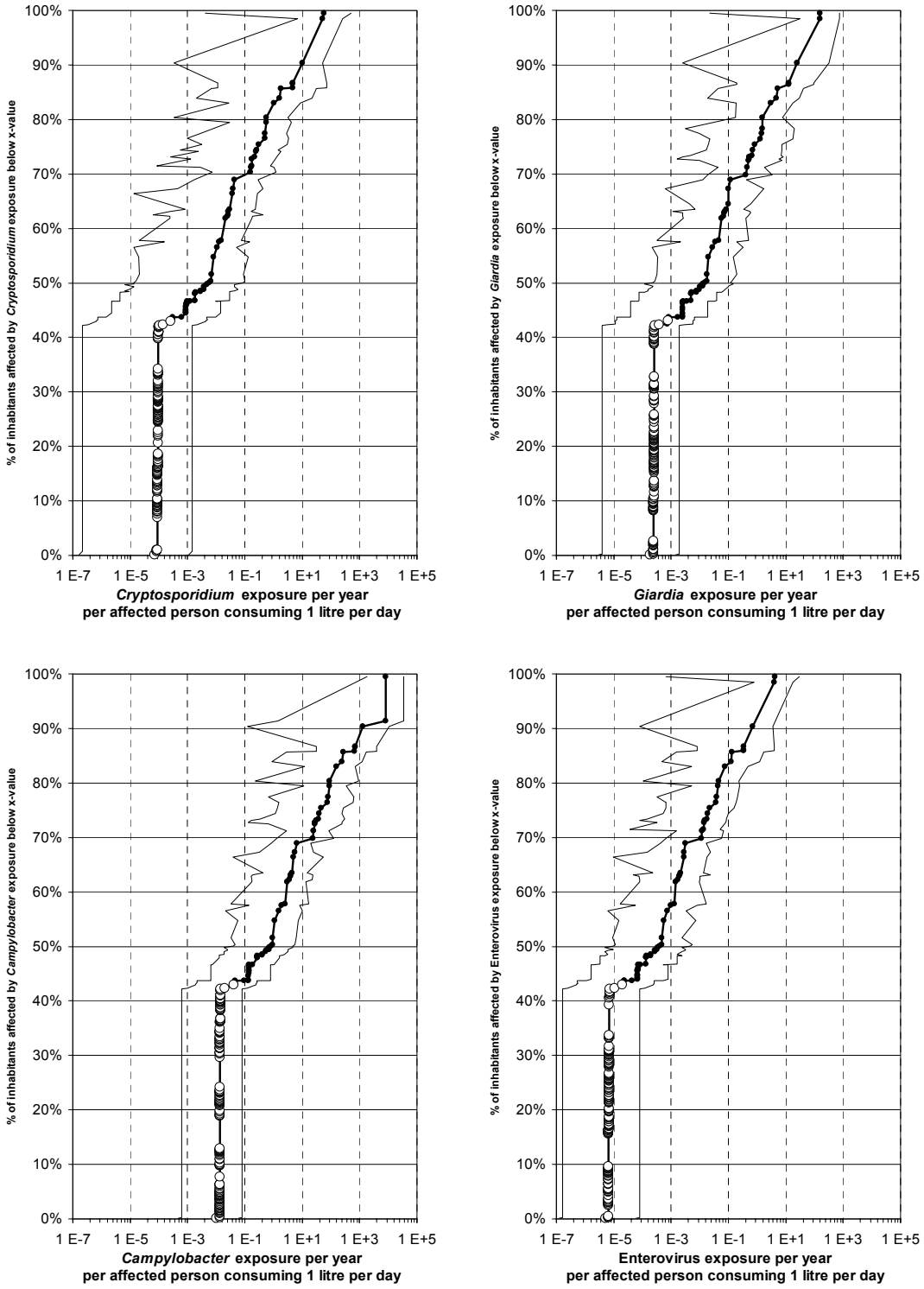


Figure 5.11 Retrospectively expected exposure to pathogens (per person per year) estimated from *E. coli* concentrations > 0 CFP in 391 samples (100-500 ml) of finished water of treatment plants.
(mean, 2.5 and 97.5 percentiles per supply zone, CDF percentiles of c.257,000 inhabitants estimated to be exposed on a population of 45 million people served in 211 supply zones). Open circles marks of mean exposures indicate supply zones were no *E. coli* was detectable in any sample and the maximum exposure to pathogens was estimated by assuming a concentration of 0.1 CFP/100 ml in one sample.

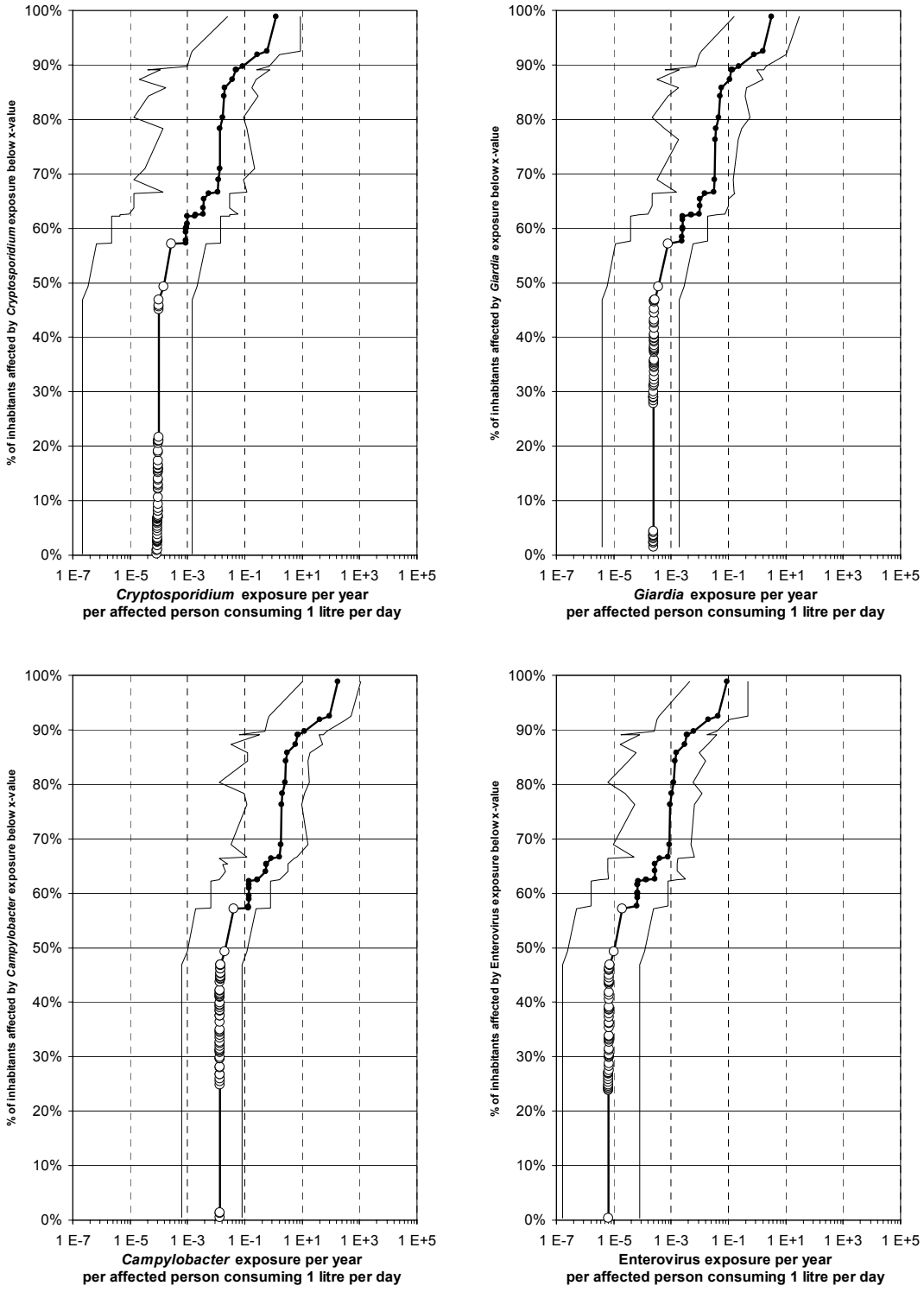


Figure 5.12 Retrospectively expected exposure to pathogens (per person per year) estimated from *E. coli* concentrations > 0 CFP in 80 samples (100 ml) of drinking-water leaving distribution reservoirs (mean, 2.5 and 97.5 percentiles per supply zone, CDF percentiles of c. 385,000 inhabitants estimated to be exposed on a population of 22 million people served in 83 supply zones). Open circles marks of mean exposures indicate supply zones were no *E. coli* was detectable in any sample and the maximum exposure to pathogens was estimated by assuming a concentration of 0.1 CFP/100 ml in one sample.

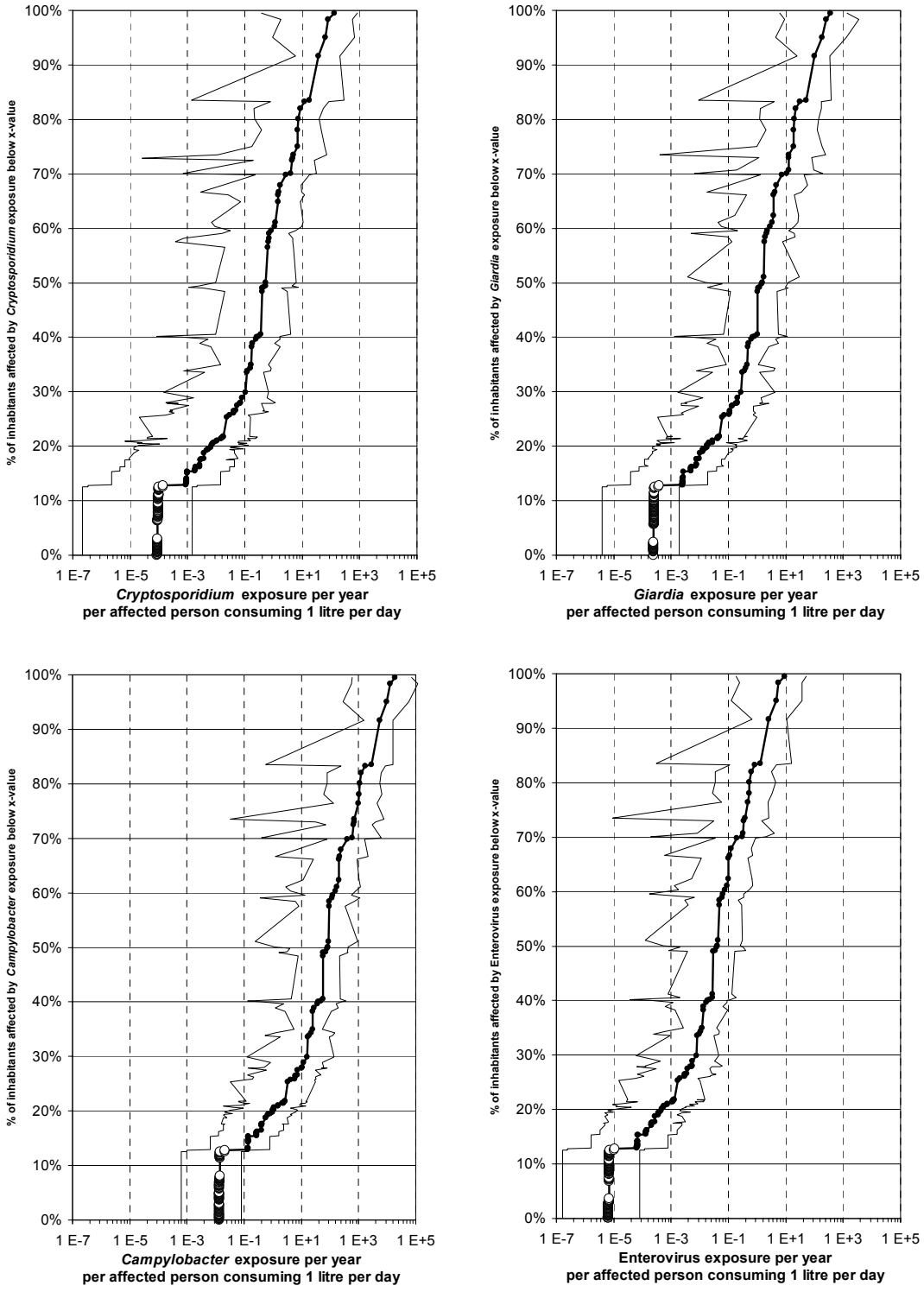


Figure 5.13 Retrospectively expected exposure to pathogens (per person per year) estimated from *E. coli* concentrations > 0 CFP in 1031 samples (100 ml) of drinking-water from taps in premises (mean, 2.5 and 97.5 percentiles per supply zone, CDF percentiles of c. 202,000 inhabitants estimated to be exposed on a population of 43 million people served in 209 supply zones). Open circles marks of mean exposures indicate supply zones were no *E. coli* was detectable in any sample and the maximum exposure to pathogens was estimated by assuming a concentration of 0.1 CFP/100 ml in one sample.

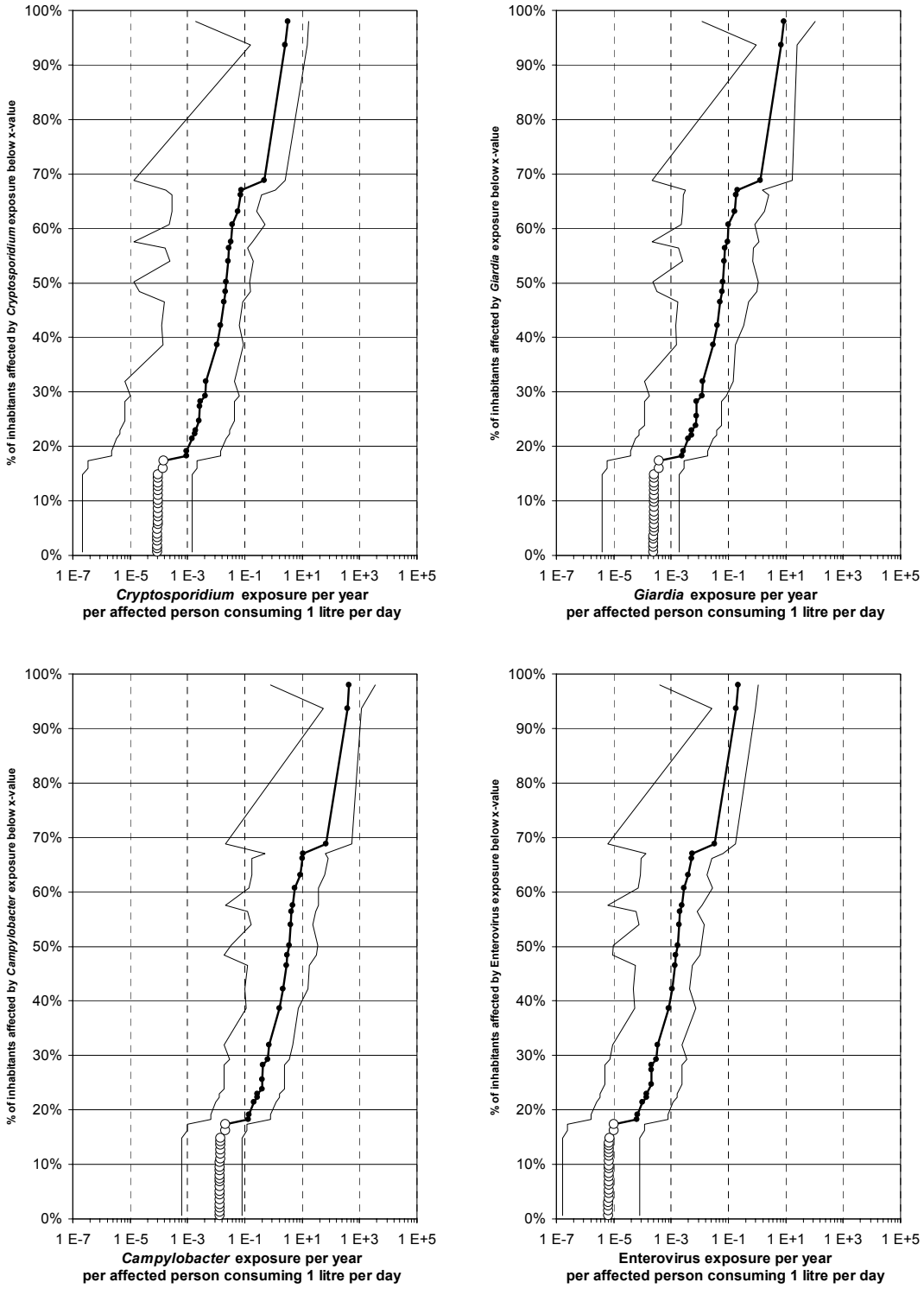


Figure 5.14 Retrospectively expected exposure to pathogens (per person per year) estimated from *E. coli* concentrations > 0 CFP in 91 samples (100 ml) of drinking-water collected after operations (mean, 2.5 and 97.5 percentiles per supply zone, CDF percentiles of c. 39,000 inhabitants estimated to be exposed on a population of 5.8 million people served in 47 supply zones). Open circles marks of mean exposures indicate supply zones were no *E. coli* was detectable in any sample and the maximum exposure to pathogens was estimated by assuming a concentration of 0.1 CFP/100 ml in one sample.

The CDFs in Figure 5.11 through 5.14 are constructed in the same way as in paragraph 5.6.5. On the y-axis, the percentage of inhabitants in affected areas per supply zone is presented. The number of affected inhabitants per supply zone is estimated by multiplying the total number of inhabitants of the supply zones with the fraction of samples containing *E. coli*. On the x-axis, the retrospectively expected standard exposure to pathogens per person per year is presented. The mean exposure is determined by the mean concentration of *E. coli* in the samples containing *E. coli* and the mean pathogen to *E. coli* ratios in the contamination source (assuming surface water, as shown in Tables 5.7 and 5.8). The variation of the exposures is determined by the variation of the *E. coli* concentrations and the pathogen to *E. coli* ratios. Actual exposure in supply zones where *E. coli* was found in none of the samples may be much lower than the estimated maximum (assuming 1 sample to contain 0.1 CFP per 100 ml).

Expected annual exposure from tap water

Taking into account the assumption that the pathogen to *E. coli* ratios in the contamination sources could be any of the ratios found in the limited number of surface water sources, exposure to *Campylobacter* would have been highest during all events, followed by exposure to the other index pathogens *Giardia*, *Cryptosporidium* and enterovirus. Including the probability of c. $4.7 \cdot 10^{-3}$ per person per year to be affected by a contamination event, the ranges of probability of standard exposure to pathogens are presented in table 5.16.

Table 5.16 Maximum of expected standard exposure per person, consuming 1 litre per day, per year to index pathogens due to events (based on detections by periodical monitoring of tap water in premises) including the probability of being an inhabitant of a contaminated area ($4.7 \cdot 10^{-3}$ per person per year). The expected maximum exposures are presented for 12 combinations of 4 percentiles of the events (expressed in numbers of inhabitants affected) and 3 statistical index numbers of standard exposure per event. Risks are based on a standard consumption of 1 litre per person per day. The expected exposures below 1 can be interpreted as probabilities of exposure to 1 index pathogen.

Fraction of supply zones as % of affected inhabitants	Mean and range per supply zone	Maximum risk of exposure			
		Cryptosporidium	*Giardia*	*Campylobacter*	Enterovirus
Minimum	P2.5	$1.0 \cdot 10^{-9}$	$1.8 \cdot 10^{-8}$	$3.0 \cdot 10^{-6}$	$7.7 \cdot 10^{-10}$
(c. 150)	Mean	$4.1 \cdot 10^{-7}$	$1.1 \cdot 10^{-6}$	$6.2 \cdot 10^{-5}$	$3.0 \cdot 10^{-8}$
	P97.5	$6.8 \cdot 10^{-6}$	$9.0 \cdot 10^{-6}$	$3.7 \cdot 10^{-4}$	$3.7 \cdot 10^{-7}$
Median	P2.5	$2.8 \cdot 10^{-5}$	$1.8 \cdot 10^{-4}$	$1.2 \cdot 10^{-2}$	$6.1 \cdot 10^{-6}$
(c. 101,000)	Mean	$2.5 \cdot 10^{-3}$	$7.0 \cdot 10^{-3}$	0.38	$1.8 \cdot 10^{-4}$
	P97.5	$2.4 \cdot 10^{-2}$	$5.5 \cdot 10^{-2}$	2.0	$1.4 \cdot 10^{-3}$
90-percentile	P2.5	$2.8 \cdot 10^{-2}$	0.12	7.4	$3.2 \cdot 10^{-3}$
(c. 182,000)	Mean	0.17	0.47	26	$1.2 \cdot 10^{-2}$
	P97.5	0.57	1.0	46	$3.3 \cdot 10^{-2}$
Maximum	P2.5	$1.8 \cdot 10^{-3}$	$2.9 \cdot 10^{-2}$	2.8	$8.7 \cdot 10^{-4}$
(c. 202,000)	Mean	0.60	1.7	91	$4.4 \cdot 10^{-2}$
	P97.5	4.0	6.3	$3.4 \cdot 10^{2}$	0.32

Using the available data, assumptions and calculation models in this paragraph, the exposure risks from Table 5.16 for example would result in the following conclusions:

- The estimated 90-percentile of the standard risk of exposure to *Giardia* due to incidental contamination of tap water, as indicated by the detection of *E. coli*, in the 209 supply zones ranges from 0.12 to 1.0 (95% confidence interval) with a mean of 0.47 per person per year depending on the actual *Giardia* to *E. coli* ratio, assuming ratios similar to those found in surface water.
- The estimated median standard risk of exposure to *Cryptosporidium* ranges from $2.8 \cdot 10^{-6}$ to $2.4 \cdot 10^{-2}$ (95% confidence interval) with a mean of $2.5 \cdot 10^{-3}$ per person per year.

The minimum risks (the risks of exposure in the supply zone with the lowest risk) are calculated from the maximum exposure in a supply zone where *E. coli* was not found in any of the samples and where the highest number of samples was collected. The exposure is calculated based on the assumption that the maximum occurrence of *E. coli* in the supply zone was 0.1 CFP/100 ml in only one of the collected samples.

In Chapter 8, the possible risks of infection to consumers caused by these exposures are calculated and discussed.

Estimated maximum exposure in supply zones where no *E. coli* was detected

Actual exposure in supply zones where *E. coli* was found in none of the samples may be much lower than the estimated maximum (assuming 1 sample contained 0.1 CFP per 100 ml). The percentages of supply zones, based on estimated (maximum) numbers of affected inhabitants, where no *E. coli* were found are:

- c. 43% for finished water of treatment plants, periodical samples (figure 5.11);
- c. 57% for water leaving distribution reservoirs, periodical samples (figure 5.12);
- c. 13% for tap water from premises, periodical samples (figure 5.13);
- c. 17% for water from mains in samples collected after operations (figure 5.13);

The increments in the estimated maximum number of affected inhabitants in some supply zones where no *E. coli* were found are very large, indicating high maximum numbers of affected inhabitants. These estimates can be very high due to anomalies in the data. For instance, in figure 5.12, in one supply zone with c. 275,000 inhabitants, only 1 sample of water from a distribution reservoir was collected yearly, none containing *E. coli.* Assuming 1 sample contained a maximum of 0.1 CFP per 100 ml, the maximum retrospectively expected exposure per person per year of maximally c. 91,500 inhabitants was 0.1 * (pathogen to *E. coli* ratio) / 3 years.

Changes in estimated exposure to pathogens during distribution

In c. 70% of the 209 supply zones (based on affected inhabitants), the estimated mean exposure to *Campylobacter*, based on *E. coli* concentrations, in tap water increased during distribution (Figure 5.15). Especially at high increase ratios (up to c. 5000), no *E. coli* was found in any of the samples collected from finished water, although in the case of the highest ratio, there was. In most of the c. 5% of the supply zones where the exposure notably decreased, exposures from finished water were low (c. 0.05 - 1 per person per year). Exposure from finished water was high from two supply zones with

the highest decrease (c. 700 per person per year). As the ratios are only depending on differences in mean *E. coli* concentrations (*Campylobacter* to *E. coli* ratios are equal for both finished water and tap water), the same ratios apply for estimated exposure to other pathogens as well.

Increased estimated exposure after operations

In c. 70% of the 47 supply zones (based on affected inhabitants) of 3 water companies that supplied data on *E. coli* concentrations after operations, the estimated mean exposure to *Campylobacter* after operations was higher than during normal operations, based on *E. coli* concentrations in periodical samples (Figure 5.15). This is probably due to a multiple factors:

- The increased probability of a contamination occurring.
- The increased probability of detecting a contamination on the right place and time.
- Samples are also collected from special sampling devices and hydrants, increasing the probability of contamination during sampling, although in these cases there is no contamination of samples from contaminated piping in premises.

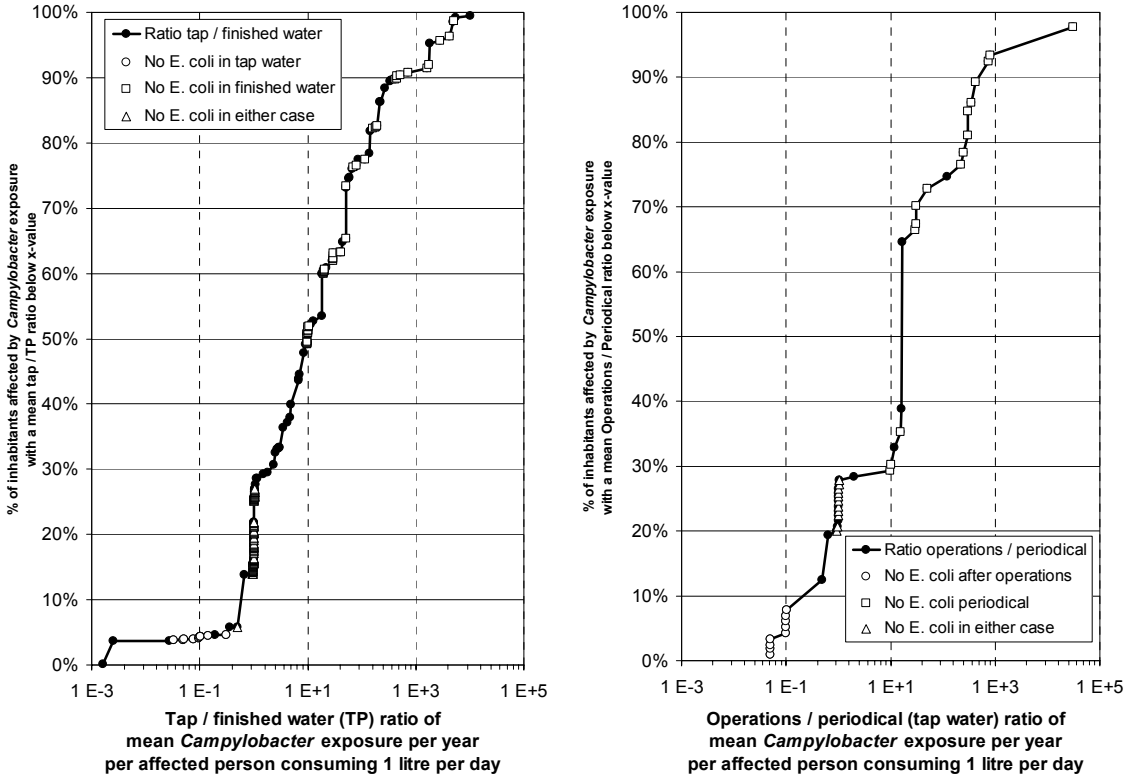


Figure 5.15 Ratios of estimated standard exposure to *Campylobacter* from tap water (Figure 5.13) to finished water from treatment plants (TP) (Figure 5.11) in the same supply zones and ratios of exposures from water after operations (Figure 5.14) and tap water (Figure 5.13) in the same supply zones.

5.9 DISCUSSION

There is a number of uncertainties in the model that hampers its applicability for evaluation of microbial drinking-water safety by water companies and regulators.

5.9.1 Accuracy of estimated *E. coli* concentrations in drinking-water

Underreporting of events

All 50 events evaluated in paragraph 5.6 occurred in the Netherlands. While collecting the event reports from the participating water companies, information on events proved to be difficult to obtain. As water companies in the Netherlands rarely are confronted with an outbreak (after WWII occurring in 1962, 1981 and 2001 only), information on small events usually is not documented and archived. Larger events usually are documented, but these are not occurring on a regular basis (approximately once every two years) and older event reports are not always retrieved or are lacking data on *E. coli* concentrations. In many cases, *E. coli* concentrations from old events could be retrieved from the laboratory database, but data on causes and response measures during the event were lacking.

Probability of detecting contaminations

Although contamination events and outbreaks have been detected as a result of consumer complaints on taste or after detection (and reporting) of technical failures, only larger contamination events usually are confirmed by the presence of indicator bacteria (total coliforms and *E. coli* in 100 ml samples). A recent preliminary simulation study in the Netherlands however revealed that the probability of detecting *E. coli* in 100 ml samples after a contamination of a distribution main in a small town with 16 litre of sewage, ranged from 0 to 15% (n = 9) with a mean of c. 5% (Van Lieverloo et al. in preparation). In this study, no inactivation of *E. coli* during distribution was assumed, resulting in an overestimation of the probability of detection, especially when a disinfectant residual is present.
The study also showed that due to changes of flow direction and velocity during the day, *E. coli* concentrations may vary heavily (over 1000-fold) during the day and that the peaks on most sites occur on regular hours of the day. Therefore, regular sampling hours may influence the probability of detection.

Period between the start and the detection of the contamination

After operations, samples are collected within the first day after the mains (or reservoir) were cleaned. Therefore, a contamination event after operations, if large enough to be detected in a 100 ml sample, is monitored from the first day.
For periodical samples, however, in a best-case scenario, samples are collected daily and on multiple sites from the first day of the contamination. In the worst-case scenario (of a detected event), the first sample contains 1 CFP of *E. coli* per 100 ml and the repeat sample does not contain any indicator bacteria (‘single hit’).

Single hit presence of *E. coli* in periodical samples therefore may represent small contamination events (if it was collected at the start of the event) or large contamination events (if it was collected at the end or the spatial periphery of an event).

The unknown effect of protective measures
When the start of protective measures such as boiling advisories and dosage of disinfectants is documented in the event reports, usually there is little known about the effect of these measures on the consumption of contaminated water. Boiling advisories are not followed by all inhabitants (cf. paragraph 5.6) and disinfectant residuals will inactivate *Cryptosporidium* much less effective than it does *E. coli* (cf. paragraph 4.2.5).

Contamination in piping systems of connected premises
Standard sampling procedures include flushing of the piping systems of connected premises until water temperature is constant. In the Netherlands, one company, until 2002, flushed the tap and piping with only 250 ml. Incidence of *E. coli* was 10-fold higher than found by other water companies, whereas incidence in repeat samples was lower than found by other companies. Repeat samples were always collected after flushing taps and piping according to standard sampling procedures. This indicates a possible contamination of piping systems of connected premises, although no further investigations have yet been conducted (Van Lieverloo et al, 2003).

Contamination during sampling or analysis
For evaluation of 'single hits' (repeat samples not containing *E. coli* or other faecal indicators) especially, contaminations during sampling or analysis are a factor that overestimates infection risks calculated from *E. coli* concentrations in samples collected from water supply. Optimisation of quality assurance during sampling and analysis will diminish this factor. When contamination during sampling or analysis is certain, the results are usually not included in the *E. coli* databases.
Contamination during sampling from hydrants however is a possible cause of contamination that is not excluded from the data. Sampling from hydrants is common after operations.

5.9.3 Pathogen to *E. coli* ratios for unknown sources of contamination

There is a large variation in pathogen to *E. coli* ratios, rendering this variable the most sensitive part of the model. Sources of variation are **conditions in the host**:
- Species of warm-blooded animal or human the faeces originates from.
- Infections and illnesses of the animal or human the faeces originates from.

Conditions in nature influence survival and therefore the ratios:
- Age of the faecal material since defecation;
- Matrix (surface water, soil, groundwater, man-made surfaces) and the resulting environmental conditions (presence of predators, temperature, moisture, exposure to sunlight);
- Conditions in the drinking-water distribution system (temperature, disinfectant residual, flow, biological activity in biofilm and sediments).

Unknown sources of contamination
It is usually hard to identify the source of contaminations leading to faecal contamination events and outbreaks. It is almost impossible to identify the source of 'single hits' (when repeat samples do not contain *E. coli* or other faecal indicators). Therefore, pathogen to *E. coli* ratios are usually unknown and will have to estimated.

Known sources of contamination
Even when the source of a contamination is found, it is usually difficult or impossible to assess the real pathogen to *E. coli* ratio in this source. In many cases in takes at least several days to identify the source, leading at best to unknown changes in the composition of the source and in the worst case to its disappearance. If it is possible to collect samples of the known or most likely source of the contamination, it is important to use this opportunity to minimise the uncertainty and variability of the model.

Sensitivity analysis: impact of pathogen to *E. coli* ratios from different sources
In paragraphs 5.6 (events) and 5.8 (periodical monitoring and operations), pathogen to *E. coli* ratios were used from 26 samples collected in surface water in the Netherlands (table 5.7 and 5.8). To demonstrate the sensitivity of the calculation model for the pathogen to *E. coli* ratio, other ratios were used as well in calculations:

- As found in untreated domestic sewage in the Netherlands and Germany (Table 5.6 in paragraph 5.6).
- As found in soil or shallow groundwater surrounding mains in the United States (Table 5.9 in paragraph 5.6).

The effect of selecting a different type of contamination source is even higher than the variation within a contamination source (Figures 5.16). The highest expected exposures to culturable enteric viruses or PCR-detectable enterovirus are found when assuming soil or shallow groundwater as the contamination source. Enterovirus to *E. coli* ratios (measured as thermotolerant coliforms) in these sources were very much higher than in surface water or untreated sewage. These findings corroborate experiences of higher survival of protozoans and viruses than *E. coli* in natural conditions (paragraph 4.2.5 in chapter 4). It is likely that faecal material in material surrounding mains or surface water is older than faecal material in sewage, causing an increase of the pathogen to *E. coli* ratios, although the absolute levels of pathogens usually are lower in surface water and soil or shallow groundwater.

As most of the recorded events were considered to be caused by operations (paragraph 5.6.1) and small events (paragraph 5.8) are likely to be caused by leaking mains (in combination with loss of pressure) or operations as well, the entry of soil or shallow groundwater is very likely. Although the data on pathogen to *E. coli* ratios in soil or shallow groundwater available is very limited, the available data suggest that every *E. coli* found, when from such a contamination source, would be an indication of very high enterovirus concentrations in drinking-water and resulting high infection risks to consumers.

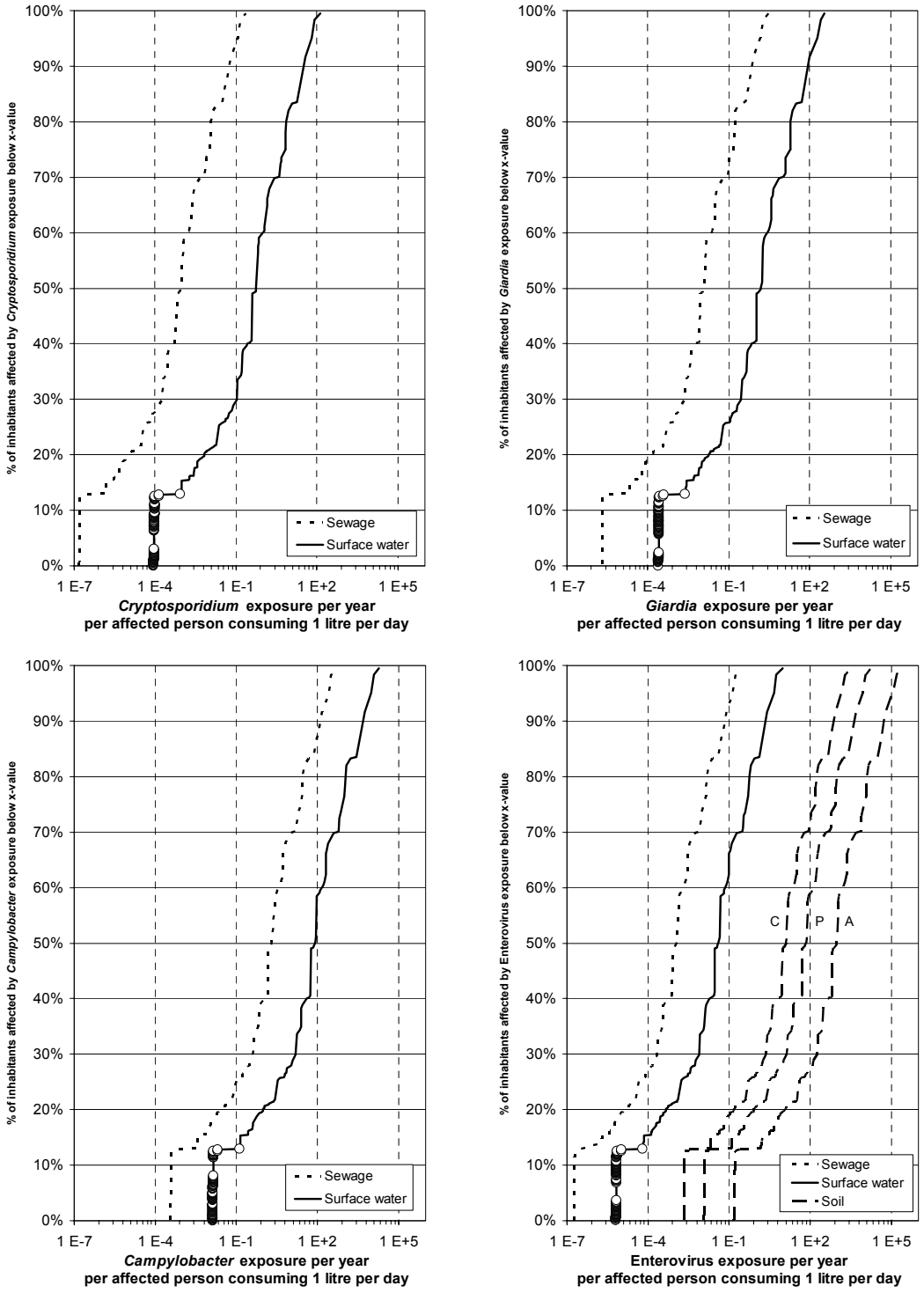


Figure 5.16 Retrospectively expected mean exposures to affected inhabitants (per person per year) in contaminated areas, drinking 1 litre per day, estimated from *E. coli* concentrations in tap water from premises in supply zones, assuming pathogen to *E. coli* ratios from sewage, surface water and soil or shallow groundwater near mains (ratios in Tables 5.6 through 5.9; three enterovirus ratios in soil or shallow groundwater: C = culturable enteric viruses, P = pairs in which both faecal coliforms and viruses (culturable + PCR) are detected, A = all data; CDF percentiles of the mean are based on c. 202,000 affected inhabitants on a total of 43 million people served in 209 supply zones).

5.9.4 Sensitivity to factors affecting variation and uncertainty

The ratio between of the 97.5 percentile and the 2.5 percentile of the mean exposure to pathogens due to differences in mean yearly *E. coli* concentrations determined periodically in tap water between supply zones is c. 915,000, i.e. c. 6.0 on a log-scale or short: 6.0 log. An overview over variation and uncertainty is presented in Table 5.18.

Table 5.18 Indication of variation and uncertainty of estimated exposure to pathogens by factors of the calculation model used in this chapter. Values are presented as ^{10}logarithms of the actual values.

***E. coli* concentration**[a]				
Variation in exposure[b]				
- Periodical (209 supply zones)	6.0			
- Operations (47 supply zones)	4.4			
- Reported events (50)	1.7			
U: Limited detection of events[c]	-1			
U: Limited recording of events[d]	- 0.5			
U: Contamination of samples[e]	+ 0.3			
Pathogen to *E. coli* ratio	*Cryptosporidium*	*Giardia*	*Campylobacter*	Enterovirus
Variation between sources[f]				
- Surface water vs. sewage	2.8	2.1	1.6	1.5
- Soil / groundwater vs. sewage				
- Culturable viruses / det. coli44[g]	NA[g]	NA	NA	4.0
- Viruses / det. coli44	NA	NA	NA	4.8
- Viruses / all coli44	NA	NA	NA	5.9
Variation within sources[k]				
- Sewage	1.0	0.5	4.3	1.5
- Surface water	3.1	3.3	1.9	2.2
- Soil and shallow groundwater				
- Culturable viruses / det. coli44	NA	NA	NA	1.3
- Viruses / det. coli44	NA	NA	NA	2.2
- Viruses / all coli44	NA	NA	NA	4.5

[a] U = uncertainty, leading to underestimation (-) or overestimation (+) of exposure to pathogens

[b] Ratio of 97.5-percentile and 2.5-percentile of supply zones or events (based on affected inhabitants)

[c] Estimate for detection of events longer than one day, this factor probably is higher (Van Lieverloo, in prep.)

[d] Estimate for recording of events longer than one day

[e] Estimate by microbiological laboratory specialist in the Netherlands

[f] Median of the ratios of means per percentile of affected inhabitants (periodical tap water data)

[g] coli44 = thermotolerant coliforms; NA = not available

[k] Ratio of 97.5-percentile and 2.5-percentile of pathogen to *E. coli* ratios in contamination source. When 2.5-percentile = 0, the minimum value > 2.5 is used, thus underestimating the variation within the source

The method for estimating pathogen exposures is very sensitive to the pathogen to *E. coli* ratio: the mean exposure to enterovirus increases almost 6 log (a million) when soil or shallow groundwater is assumed as a contamination source, versus the assumption of untreated sewage as a contamination source. Due to variation within possible contamination sources, these differences may be even higher. The variation within sources (ratio of 97.5 and 2.5 percentile) may be higher than 4 log (Table 5.18). Compared to these sources of variation, the estimated uncertainties due to incomplete recording and detection of events and possible overestimation due to contamination of samples are low, as are the influence of the calculation method.

5.10 CONCLUSIONS

The analysis of large volumes of drinking-water in the United Kingdom, Germany and the Netherlands for the presence of *E. coli* has shown that this indicator of faecal contamination is not present in detectable background concentrations, even when disinfectant residuals are absent that might eliminate the indicator and thus mask faecal contaminations. These results corroborate the hypothesis that faecal contaminations occur as temporary and often local events and that it is not feasible to detect and quantify pathogens as a means of assessing the risks of exposure to and infection by faecal pathogens. The only indicator of faecal contamination that currently may serve as an index of pathogen concentrations is thought to be *E. coli*.

Compared to the method presented in chapters 3 and 4, using pathogen concentrations actually determined in source water and elimination capacities of surrogate variables actually determined in treatment plants, the calculation method based on the occurrence and concentrations of *E. coli* as an index of pathogen concentrations in water samples is very flawed. The variation of the pathogen to *E. coli* ratio between and within possible contamination sources such as untreated sewage, surface water and soil or shallow groundwater is very large. Indeed, this variation is so large, that the uncertainty in the calculation method leaves the method almost, if not entirely, inapplicable for the purpose of estimating pathogen concentrations and resulting infection risks to consumers during short or prolonged faecal contamination events.
As long as quantitative knowledge is limited about the incidence an severity of each type of faecal matter being the source of contamination events, the method can only serve to calculate exposure and infection risks assuming worst conditions. When the type of contamination source is known though (e.g. surface water of sewage), the risk levels can be assumed to be lower. When the pathogen to *E. coli* ratios in the source are characterised, as in the outbreak case presented in paragraph 5.5, the uncertainty and variation is further limited and renders the method more applicable to assess infection risks.

Conclusions regarding the calculation method:

- Uncertainties about actual pathogen to *E. coli* ratios in the contamination source render the calculation method very much flawed and prove the need for assessing pathogen to *E. coli* ratios in sources of each contamination event as well as common contamination sources until a better method is developed.
- Uncertainties about the detectability, start and spatial distribution of events, as well as recording of events by water companies, result in an underestimation of the actual risks of exposure to pathogens. This conclusion founds the need for the quantification of the detectability and for better record keeping of contamination events and possibly the optimisation of monitoring programs as well.

Although the presented method is flawed, the current absence of a better index of pathogen exposure and infection risks during faecal contamination events leaves it the only method available for water companies, inspectorates and regulators to estimate the possible health effects for consumers and the need for reducing risks. The method is applicable to all secondary faecal contaminations, occurring in groundwater wells, (groundwater) treatment plants and distribution systems, detectable by the presence of *E. coli*. Theoretically, the method is equally applicable for other faecal indicators, such as enterococci, providing the quantitative data available is sufficient.

Main conclusions regarding *E. coli* occurrence and inferred exposure to pathogens:

- Background levels of *E. coli* are not detectable in large volumes and may be as low as $< 1.4 \cdot 10^{-4}$ CFP/l.
- The risk of being affected by a faecal contamination indicated by the presence of *E. coli* was c. $5 \cdot 10^{-3}$ in the period 2000-2003 for c. 43 million inhabitants mainly living in France, the Netherlands and Germany.
- There are large differences in mean *E. coli* concentrations between supply zones however (up to a factor of c. a million) and *E. coli* occurrence and concentrations may increase dramatically during distribution (over 1000-fold).
- For the same frequencies of occurrence and concentrations of *E. coli* detected during contamination events, mean exposure to enterovirus is almost a million times higher when assuming enterovirus to *E. coli* ratios encountered in soil or shallow groundwater than when assuming ratios encountered in untreated domestic sewage. Although absolute concentrations of enterovirus in soil and shallow groundwater are much lower than in untreated domestic sewage, exposure to enterovirus has to be assumed to be much higher at the same levels of *E. coli* is found in drinking-water after a contamination with soil or shallow groundwater.
- Due to relatively high ratios of *Campylobacter* to *E. coli* in both untreated sewage as well as surface water, the exposure to this pathogen is relatively high compared to exposure by the other pathogens. Due to higher pathogen to *E. coli* ratios in surface water, the occurrence of the same levels of *E. coli*, a higher exposure to pathogens after a contamination with surface water is assumable.
- Until more is known about the probability of being affected by different contamination sources and the pathogen to *E. coli* ratios therein, high levels of exposure to pathogen have to be assumed when finding levels of > 0 CFP *E. coli* per 100 ml drinking-water.

5.11 RECOMMENDATIONS

Although the purpose of this chapter is limited to calculating pathogen concentrations and exposure, the results are clearly enough cause to recommend limiting the incidence of *E. coli* in drinking-water.
To be able to better assess the risk levels of contamination during distribution, there is a need for research into improving possibilities of estimating frequencies and resulting risks from incidental or prolonged occurrence of faecal matter caused by secondary contaminations in distribution systems or (groundwater) treatment plants. In the mean time, in many supply zones there is enough cause to increase the efforts of preventing these contaminations.

There are three groups of recommendations in this paragraph:
- Limiting the incidence of *E. coli*
- Risk management and reduction options
- Risk assessment during operations
- Risk assessment during research

5.11.1 Limiting the incidence of *E. coli*

The results in this chapter suggest that any consumption of drinking-water with a detectable level of *E. coli* in 100 ml samples, with a high probability leads to high pathogen exposure. Infection risks are high when exposure to pathogens is high (see chapter 7). Therefore, if a water company want to limit infection risks to e.g. $1 \cdot 10^{-4}$ per person per year, the incidence (frequency, probability) of *E. coli* concentrations > 0 CFP/100 ml should at least be lower than this level, i.e. $< 1 \cdot 10^{-4}$.
It is not clear yet whether this level is sufficient for safeguarding drinking-water safety, as the sample volume may need to be larger to do so, maintaining incidence $< 1 \cdot 10^{-4}$.
To assess the sample volume that should not contain *E. coli* (in 99.99% of all samples collected), it is necessary to better assess the distribution of the probability of (common) contamination sources being the cause of *E. coli* occurrence and the pathogen to *E. coli* ratios in these sources. It is likely that the same recommendations are valid for other faecal indicators, but this assumption has not been evaluated.

5.11.1 Risk management and risk reduction options

Recommendations for managing microbial water quality of drinking-water in general and in piped distribution systems specifically have been given in the WHO guidance documents ‘Water Safety Plans’ (Davison *et al.* 2005) and ‘Safe Piped Water’ (Ainsworth, 2004) respectively.

Summarising, there is a need for strengthening the barriers:

- **Primary barrier; limitation of leakage** of mains and distribution reservoirs:
  - Optimisation of mains leak detection and repair.
  - Intensifying programs for refurbishment and/or replacement.
  - Periodic inspection of the constructional integrity of distribution reservoirs.
  - Optimisation of cross-connection and backflow/backpressure prevention.
- **Secondary barrier; limitation of low or no pressure** events:
  - Optimisation of pump design, selection, operation and maintenance;
  - Optimisation of (automated) power failure response measures;
- **Tertiary barrier: limitation of low or no disinfectant residual** events (if residuals are maintained[11]).

In general, risks are managed by a program of systematic quality assessment and quality control, based on quantified deterioration velocities of infrastructural and operational performance:

- **Design of infrastructure and operational procedures**,.
- **Periodic maintenance (condition-independent)**:
  - Maintenance of infrastructure
  - Instruction of company personnel regarding knowledge of procedures and the quality of performance.
- **Condition-dependent maintenance**:
  - Inspection of infrastructure condition and corresponding corrective response measures.
  - Auditing of performance of company personnel and corresponding corrective response measures.
  - Monitoring of water quality variable indicative of infrastructure and operational performance and corresponding corrective response measures.

Depending on local conditions, some of these barriers clearly are performing outside critical limits (e.g. when leakage rates are 30%) and in these cases there is no need for quantification of the effects of low performance on the compliance with microbial safety. However, when microbial safety rarely is outside critical limits, quantification of the effects of failures of parts of risk management structure is necessary to determine quantitative performance criteria for these parts. In these cases, risk assessment is needed to optimise the balance between microbial safety and investments in optimisation and for directing resources to the weakest parts of the fault tree.

[11] In a number of countries in the European Union (Germany, the Netherlands, Denmark), both water companies as well as regulators strive for maintaining microbial safety without the need for the maintenance of a disinfectant residual. One of the reasons is not to mask the presence of a (faecal) contamination by eliminating the indicators (eg. *E. coli*) without eliminating the pathogens.

5.11.2 Risk assessment during operations

- **Events**. Systematic documentation and evaluation of each faecal contamination, including so-called 'single hits'[12] and periodic evaluation of the frequency and severity of these contaminations. It is recommended that each event should be documented and evaluated by performing a fault tree analysis as described in chapter 1.
  - Tracking the source of every faecal contamination, assessing pathogen to *E. coli* ratios in all sources found, in order to assess the possible infection risks to the community served.
  - Describing, as quantitatively as possible, the infrastructural and operational factors leading to the ingress of the contamination source, as well as the amount of contaminative material that has entered the system.

- **Standard operating conditions.** Systematic monitoring (inspection, auditing), documentation and evaluation of infrastructural and operational conditions that are known to be able to lead to faecal contaminations, in combination with conditions occurring beyond the control of the water company (storms, flooding, third-party operations etc.). In their Water Safety Plan approach (Davison *et al.* 2005), the WHO provides guidance for designing these monitoring programs. This includes optimisation of infrastructure maintenance and human resource management in general. Each sub-critical and critical deviation should invoke corrective response measures and protective response measures may be called for as well. Thus, these monitoring programs both act as drivers for risk control in parts of the system (local Deming-circles) as well as input for risk assessment. Periodic evaluation of the performance indicators, preferably in the aforementioned fault tree analysis, will quantify the causal effect of each part of the fault tree and adjustments of critical limits. Furthermore, these evaluations will provide the validation of the effectiveness of the risk management procedures during design, construction and maintenance.

[12] Repeat sample(s) do(es) not contain indicator bacteria. It is important to evaluate whether other indications of persistence of a contamination have occurred or still occur (see paragraph 5.3).

5.11.2 Risk assessment during research

- **Quantitative evaluation of the probability of detecting faecal contamination** in hydraulic model simulations.
- **Quantification of the factors commonly occurring in the fault trees** of contaminations and performing Quantitative Microbial Risk Assessment with these data to assess the risks to the community served:
  - Pathogen to *E. coli* ratios in known sources of contamination (see Risk management during operations) and sources that are most likely to contaminate distribution systems during deviations of operational criteria
    - Soil and shallow groundwater, as these are most likely to enter during:
      - Low or no pressure in combination with leaks in mains;
      - Mains breaks and repairs.
      - Operations (construction, replacement, maintenance).
    - Contents of hydrants, as these are most likely to enter during incorrect application by third parties (illegal or legal), fire-departments, contractors and company personnel
    - Water inside connected premises, especially farms, slaughterhouses, swimming-pools and other premises with potentially high concentrations of pathogens. This water may flow back or be pressed back into the distribution systems when backflow devices are missing or performing outside operational criteria.
  - The probability of performance of infrastructure and operations outside critical limits (available from risk assessment results documented during operation).
  - Amounts of faecally contaminated matter entering the distribution system when infrastructure or operations perform outside critical limits. Experiments simulating conditions that range from common to extreme will provide quantitative information to enter into the fault tree analysis.
  - Survival of pathogens in distribution systems under different circumstances (temperature, disinfect residual concentrations, quantity an composition of biofilm and sediments).

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6. Consumption of tap water

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6.1 INTRODUCTION

When assessing the exposure to pathogens through drinking water, both the concentration of pathogens in drinking water and volume of drinking water consumed are important parameters. In the first Quantitative Microbial Risk assessments (QMRAs) that were conducted on drinking water, a drinking water consumption of 2 litres per person per day has been assumed (Regli *et al.,* 1991). Water intake differs per person however, and Roseberry and Burmaster (1992) used data on water consumption and the variation between persons therein and fitted a statistical distribution to their consumption data. The median value Roseberry and Burmaster reported was 0.96 litres/day (95% confidence interval: 034-2.72 L/day). Several QMRA studies have used this statistical distribution as a description of the water consumption. Statistical distributions are preferable for QMRA, because the variability in the consumption within the consumer population is included in the overall risk assessment. The Roseberry & Burmaster data represent consumption of tap water in total, while for microbial risk assessment only the volume of cold tap water without heat treatment (coffee, tea, cooking) is relevant. Teunis *et al.* (1997) obtained data on cold tap water consumption in the Netherlands for use in QMRA. The median consumption they report is 0.15 litres/day, which is considerably lower than the total tap water consumption reported by Roseberry and Burmaster. Several other authors have assessed the consumption of cold and/or total tap water consumption. This chapter will give an overview of the data that have been reported on consumption of (cold) tap water.

When designing a consumption study or use the data from consumption studies in QMRA it is important to have sufficient insight in the strengths and weaknesses of different study designs. The chapter starts therefore with a brief discussion on study design and recommendations for the design of consumption studies. Subsequently, the results of the consumption data reported in the literature are presented and factors that influence water intake, such as age, social status etc are discussed.

To illustrate the type of data and statistical analysis of consumption data, four studies are presented in more detail. Statistical models were fitted to the data to show how the variability in drinking water consumption can be described. Recommendations are given for consumption estimates within QMRA.

6.2 METHODS FOR COLLECTING CONSUMPTION DATA

6.2.1 Dietary assessment

Drinking water consumption studies have been carried out for several purposes: to determine possible relationships between drinking water quality and human health, to determine the fraction drinking water comprises of the individual's total liquid consumption or just to calculate the amount of water ingested in relation to other uses of drinking water in households, like bathing, dishwashing etc.

Methods to collect consumption data on the individual level can roughly be divided into two categories: short-term and long-term instruments. Short term dietary assessment methods collect dietary information on current intake. They vary from recalling the intake from the previous day (24 h recall) to keeping a record of the intake of food and drinks over one or more days (dietary record). Long-term dietary assessment methods collect information on usual food intake over the previous months or years (dietary history or food frequency questionnaire) (Biró *et al.*, 2002). The drinking water consumption studies reported used similar methods for data collection. Table 5.1 gives an overview of the available literature on drinking water consumption and the study designs applied, with the advantages and disadvantages of the different study designs. Recommendations for the design of future consumption studies are given in paragraph 5.5.

Table 5.1 Consumption study designs; advantages and disadvantages.

Dietary Recall Retrospective research conducted as an interview or questionnaire. Investigator asks respondent to enumerate foods/beverages consumed in the preceding full day, including their quantity.		
Advantages *	**Disadvantages***	**Drinking water studies applying this method**
Applicable for broad populations No literacy requirement Low respondents burden Higher response rate Cost-effective Open-ended** Procedure does not alter intake Characterizes population average intake fairly well Possibility for standardisation	Recall bias Lower accuracy of volume estimation Higher consumption estimates	EPA, 2000

* = Refs: Robertson *et al.* (2000a), Biró *et al.* (2002), Hulshof *et al*. (2002), Dangendorf (2003), Kaur *et al.*, 2004).
** = open-ended (the participants can state themselves what they consumed, instead of choosing from predefined alternatives

Dietary record/Diary Prospective research where respondents record specific data on actual consumption of (tap) water.		
Advantages*	**Disadvantages***	**Drinking water studies applying this method**
Fairly accurate consumption data Reflects current consumption Detailed information No recall problems Completeness of information Open-ended	Time-consuming, possibly resulting in: - lower participation grade - inhibition - fatigue - less accurate reporting Not representative for average consumption throughout a year Less cost-effective	Hopkin and Ellis (1980), EHD (1981), Löwik *et al*., 1994, DWI (1996), Anonymous (1998), Hulshof *et al.* (1999), Petterson and Rasmussen (1999), Gofti-Laroche *et al.* (2001), Sichert-Hellert (2001), Hilbig *et a1*. (2002), and Beaudeau *et al.* (2003).

* = Refs: Callahan *et al*. (1995),DWI (1996), Shimokura *et al*. (1998), Biró *et al.* (2002), (Beaudeau *et al*., 2003), Berg and Viberg (2003)

Food frequency questionnaire Questionnaire regarding usual frequency of consumption during certain periods.		
Advantages*	**Disadvantages***	**Drinking water studies applying this method**
Requires little time Relatively inexpensive Consumption pattern is not influenced Suitable for large population surveys Easy to conduct by telephone	Recall bias Actual consumption may influence estimates from the past Poor estimation of portions and frequencies Not open-ended	Haring *et al.* (1979), Meyer *et al.* (1999), Zender *et al.* (2001), Robertson *et al.* (2002), Dangendorf (2003) and Hunter *et al.* (2004)

* = Refs: Oldendick and Link (1994); cited by Williams *et al.* (2001), Biró *et al.* (2002), Kaur *et al.* (2004)

Combination of questionnaire and diary		
Advantages*	**Disadvantages***	**Drinking water studies applying this method**
See advantages above During recall interview participant can be educated on how to fill in diary	*See disadvantages above* Possible low comparability of results due to different focus questionnaire and diary Elaborate type of study High respondent burden	Ershow *et al*., 1991: Hunt and Waller, 1994; Levallois *et al.*, 1998; Shimokura *et al*., 1998; Robertson *et al*., 2000a; Barbone *et al.*, 2002; Berg and Viberg, 2003; Kaur *et al.*, 2004)

*= Refs: Levallois *et al.* (1998), Robertson *et al.* (2000a)

6.2.2 Assessing the volume of water consumed

To assess the volume of water consumed most studies use the number of cups or glasses as a measure (EHD, 1981, DWI, 1996; Robertson *et al.*, 2000a; Gofti-Laroche *et al.,* 2001; Robertson *et al.*, 2002; Dangendorf, 2003; Hunter pers. comm. 2003; Sinclair pers. comm., 2003; Westrell *et al.*, 2004). This is a very easy way of estimating the water consumption and it is close to the every day habits of the consumer. Disadvantage is that possible bias can be

introduced because glasses and cups of different sizes may be used. In addition it might miss non-glass consumption of drinking water like icecubes, tooth brushing, taking medicines etc.

To enlarge reliability of the volume estimates several studies had the volume of the drinking vessels measured by either the participant or the interviewer (Hopkin and Ellis, 1980; DWI, 1996; EPA, 2000). EHD (1981), Meyer *et al*. (1999) and Beaudeau *et a1*. (2003) used pictures of a cup or glass to make the estimations more accurate (see Figure 6.1 for an example).

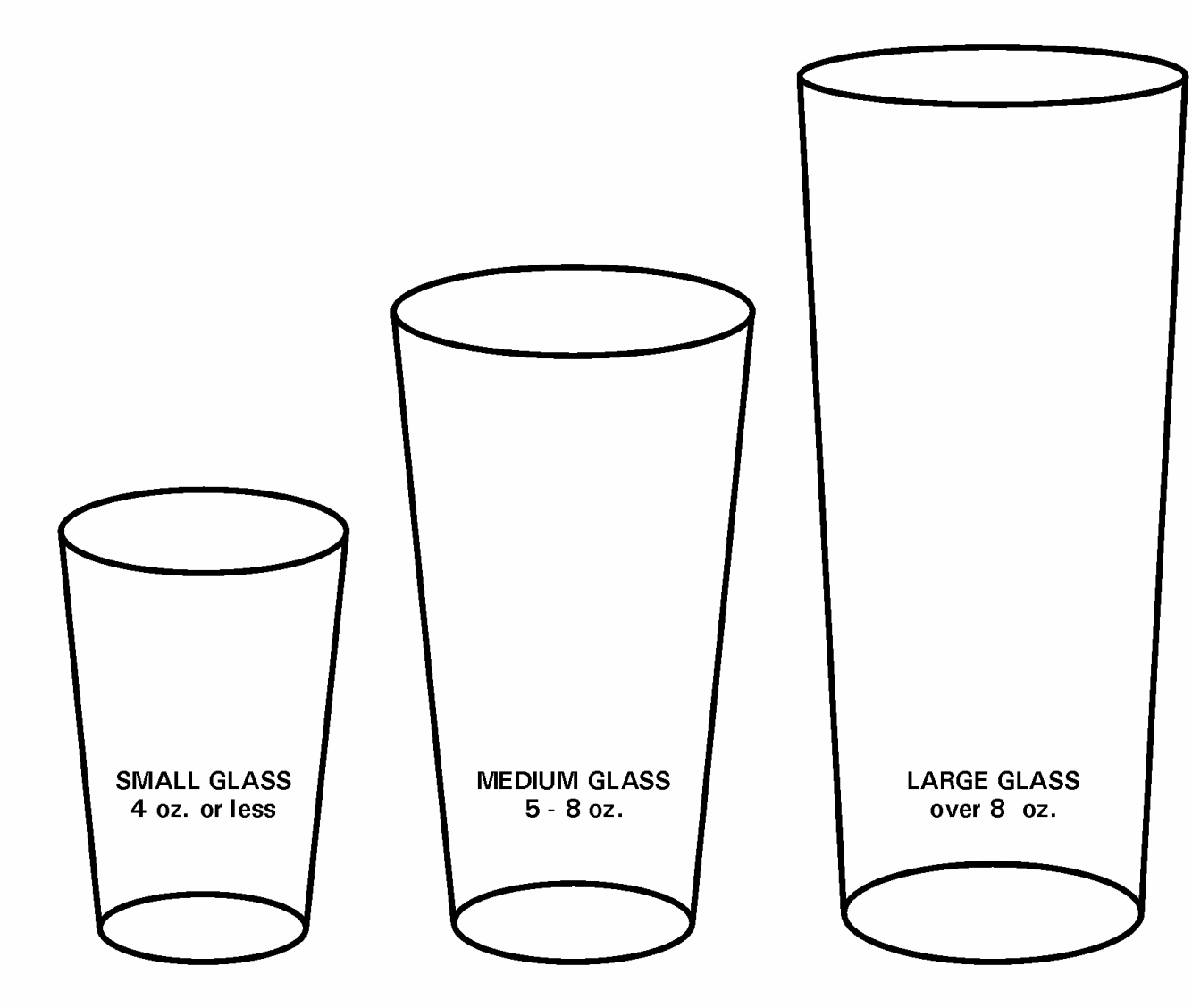

Figure 6.1 Example of pictures used to assess the volume of water consumed in a consumption study (EHD, 1981) (1 oz ≈ 29.6 mL)

The most accurate way of estimating consumption is by determining the amount of water consumed in millilitres, or by weighing, but this is also the most elaborate way. A good alternative in prospective research designs is to give people a standard measuring cup. Alternatives for retrospective research are pictures of drinking vessels, assessment of the volumes of vessels, or the type of cups and glasses used by the consumer.

6.3 CONSUMPTION OF DRINKING WATER

6.3.1 Consumption of cold tap water

Data were collected from 31 studies with consumption data from more than 90,000 people. The mean consumption of tap water is presented in table 6.2. Where the study report allowed, a discrimination was made between total tap water consumption and consumption of cold and heated tap water and bottled water. The mean consumption of the studies on the average consumer was 0.66 L per day and ranged from 0.2 - 1.55 L per day. Total consumed tap water amount for this group was 1.40 L per day and ranged from 0.71 - 2.58 L per day. Figure 6.2 shows the mean consumption and the variation of consumption in several countries. Tap water consumption was relatively high in Sweden and Australia and low in Germany and The Netherlands. Consumption data from the USA, Canada, France, Italy and the UK were quite similar.

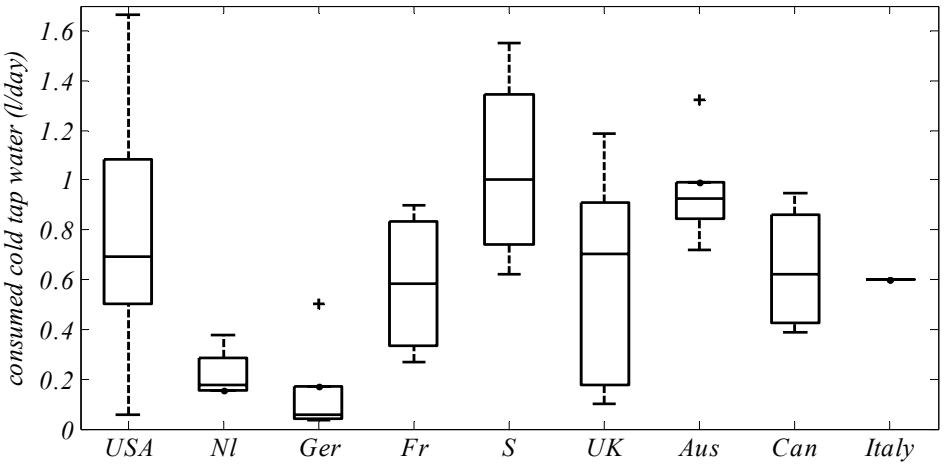

Figure 6.2. Summary results (Box-Whisker plot, showing average (central line) 25-75% (box) and minimum and maximum (error bars) consumption) of cold tap water per country.

Table 6.2. Summary of drinking water consumption data(in liters). If difference was made between consumers and non-consumers, data are presented for the total population incl. non-consumers.

Population	Country	Study type	N	Mean consumption					Reference
				cold tap water	heated tap water	total tap water	bottled water	total water	
Average consumer	USA	24 h r	15303	0.508		0.927	0.161 0.737*	1.232 1.241*	EPA (2000)
Average consumer	USA	Q/D	26081			1.108		1.785	Roseberry & Burmaster (1992)
Average consumer	USA	Q	1183			1.91			Williams *et al.* (2001)
Average consumer	C	D	970			1.34			EHD (1981)
Average consumer	NL	FFQ	3200			1.5			Foekema& Engelsma (2001)
Average consumer	NL	Q	4620	0.25 0.38*				1.14	Haring *et al.* (1979)
Average consumer	NL	Q	-	0.153					Teunis *et al.* (1997)
Average consumer	**NL**	**D**	**6250**	**0.178**					**Anonymous (1998), Hulshof pers. comm.. (2003)**
Average consumer	**D**	**FFQ**	**195**	**0.5**	**1.08**			**1.58**	**Dangendorf (2003), Dangendorf pers. comm. (2004)**
Average consumer	F	D	373 (w) 427 (s)	0.77** 0.90**	0.54* (w) 0.61* (s)	1.55*(w) 1.78*(s)	0.85*(w) 1.07*(s)	1.83*(w) 2.19*(s)	Gofti-Laroche *et al.* (2001)
Average consumer	S	Q	157	0.86	0.94	1.80	0.06	1.86	Westrell *et al.* (2004)
Average consumer in two cities	S	Q/D	35 40	1.14 1.55	0.81 1.05	1.95 2.58			Berg & Viberg (2003)
Average consumer	UK	D	3564	0.103 0.203*	0.785 1.065*	0.955 0.958*			Hopkin & Ellis (1980)
Average consumer	UK	D	1018			1.138			DWI (1996)
Average consumer	**UK**	**Q**	**416 421**	**0.704 1.187**					**Hunter *et al*., 2004**
Average consumer	**Aus**	**Q D Q**	**253 234 231**	**0.991 0.892 0.964**					**Robertson *et al.* (2000), Sinclair (unpublished data)**
Average consumer	**Aus**	**Q (Melb)**	**950**	**0.842**					**Robertson *et al.* (2002), Sinclair (unpublished data)**
Average consumer	**Aus**	**Q (Adel)**	**644**	**0.718**					**Robertson *et al.* (2000), Sinclair (unpublished data)**
Average adult cons.	Aus	D	10	1.325	0.45			2.7	Froese *et al.* (2002)
Average adult cons. Age 20-64	C	Q/D	125	0.386***		1.617	0.27		Levallois *et al.* (1998)
Average adult cons. Age > 18	C	D	639			1.49			EHD (1981)
Average adult cons. in 2 cities	F	FFQ	100 100			0.783 1.105		1.8 1.8	Meyer *et al.* (1999)

Population	Country	Study type	N	Mean consumption					Reference
				cold tap water	heated tap water	total tap water	bottled water	total water	
Average adult cons. Age 15-65	F	D	1809	0.27 0.4*	0.23 0.21*		0.27 0.19*	1.0 1.0*	Beaudeau *et al.* (2003)
Average male adult consumer	USA	Q/D	33	0.47	0.31	0.78		1.68	Shimokura *et al.* (2002)
Babies < 1 year	USA	24 h r	359	0.058			0.111	0.484 0.563*	EPA (2000)
Breast fed/formula fed babies < 1 yr	D	D	300 BF 758 FF			15 g/kg BF 49 g/kg FF		17 g/kg BF 53 g/kg FF	Hilbig *et al.*(2002)
Mixed fed young children (1-3 yr)	D	D	904			15 g/kg		19 g/kg	Hilbig *et al.*(2002)
Children 9-21 months	S	D	430	0.62					Petterson & Rasmussen (1999)
Children age 1-10	USA	24 h r	3980	0.263			0.071	0.528 0.532*	EPA (2000)
Children age < 3	C	D	34	0.47	0.14	0.57 (s) 0.66 (w)			EHD (1981)
Children age 2-3	D	D	858	0.045	0.077		0.130	1.114	Sichert-Hellert *et al.* (2001)
Children age 3-5	C	D	47	0.77	0.09	0.86 (s) 0.88 (w)			EHD (1981)
Children 4-8	D	D	1795	0.036	0.069		0.179	1.363	Sichert-Hellert *et al.* (2001)
Children age 9-13	D	D	541 (b) 542 (g)	0.062 (b) 0.056 (g)	0.087 (b) 0.087 (g)		0.282(b) 0.242(g)	1.891 (b) 1.676 (g)	Sichert-Hellert *et al.* (2001)
Children age 6-17	C	D	250	0.95	0.19	1.14 (s) 1.13 (w)			EHD (1981)
Children age 11-19	USA	24 h r	1641	0.477			0.118	0.907	EPA (2000)
Pregnant women	USA	24 h r	188	0.695	0.329		0.89	2.076	Ershow *et al.* (1991)
Pregnant women	USA	Q/D	34	0.56	0.23	0.78		1.86	Shimokura *et al.* (1998)
Pregnant women	USA	24 h r	70	0.819 0.872*			0.355	1.318	EPA (2000)
Pregnant women	USA	FFQ	71	3.2	0.2	3.4			Zender *et al.* (2001)
Pregnant women	UK	Q/D	143	0.814	0.57	1.39	0.94	2.33	Kaur *et al.* (2004)
Pregnant women	I	Q/D	210	0.6				2.6	Barbone *et al.* (2002)
Pregnant women	NL	D	52					0.219	Löwik *et al.* (1994)
Pregnant women	NL	D	50	0.153					TNO (1998)
Lactating women	USA	24 h r	77	0.677	0.458		0.178	2.242	Ershow *et al.* (1991)
Lactating women	USA	24 h r	41	1.38 1.665*				1.806	EPA (2000)
Women repr. Age	USA	24 h r	6201	0.583	0.439		0.78	1.940	Ershow *et al.* (1991)
Women repr. Age	USA	24 h r	2332	0.922 0.984*			0.212	1.258	EPA (2000)
Women repr. Age	USA	FFQ	43	2.7	0.3	3.0			Zender *et al.* (2001)

Population	Country	Study type	N	Mean consumption					Reference
				cold tap water	heated tap water	total tap water	bottled water	total water	
Workers in steel industry	Hun	O****	97 67 (w) 30 (z)					2.4 1.8 (w) 3.7 (z)	Toth *et al.* (1977)

data in bold are analysed in this article
* = data for consumers, non-consumers not included
** = cold tap water consumed at home directly from the tap (cold tap water added to e.g. lemonade and cold tap water consumed outside the house are not included in this figure)
*** = cold tap water consumed at home and away (cold tap water added to e.g. lemonade and filtered water not included)
****= drinking water was measured during work by the investigator. Drinking water was ad libitum.
w = winter, s= spring, z= summer
b= boys, g= girls
BF = breast fed, FF = formula fed
Q =Questionnaire, D = Diary, FFQ = food frequency questionnaire 24 h r=24 h recall O = other

6.3.2 Factors influencing water consumption

Several factors might influence the amount of water consumed, like outdoor air temperature (seasonal and/or regional effect), aesthetic quality of drinking water, cultural differences, age, gender, physical activity and diet.

Season/temperature
Considering a possible seasonal effect it could be expected that in summer probably more water is consumed than in winter because of higher ambient temperatures. In the studies evaluated the information regarding possible seasonal effects is limited.
Beaudeau *et al.* (2003) spread their investigations over a year but do not report on the seasonal influences. However, they did not find an increasing tap water consumption in regions with higher temperatures. On the contrary, tap water consumption was higher in the Paris region than in the Mediterranean region but this might also be due to regional problems with aesthetic water quality like high turbidity (Loret, pers. comm., 2004).
Gofti-Laroche *et al*. (2001) conducted one questionnaire in winter and one in spring (May) in France and found a significantly higher consumption of water in spring. Total water consumption amounted to 1.87 L in winter and 2.23 L in spring respectively.
In the study reported by EHD (1981) from Canada consumption of cold tap water-based beverages was higher in summer and consumption of soup and other hot tap water-based beverages was higher in winter. Total tap water intakes (cold and hot tap water) were nearly the same in winter and summer.

In the study by Hopkin and Ellis (1980) only qualitative information was obtained on seasonal effects. Consumers indicated to have higher tap water consumption during summer, but the survey itself was conducted in early spring.
No clear seasonal difference for drinking water ingestion was found in the study by Ershow and Cantor (1989) (as cited by Levallois *et al.,* 1998 and Ershow *et al.,* 1991).

Physical activity
Another factor possibly influencing drinking water consumption is physical activity. No data have been found regarding cold tap water consumption by sportsmen, but consumption can be
expected to be higher than for the general population. Toth *et al.* (1977) investigated water consumption by workers with heavy physical work in the steel industry . The unboiled drinking water consumption was 1.8 L a day in winter and up to 3.7 L in summer. The maximum amount consumed was 8.5 L.
EHD (1981) also investigated (total) daily tap water consumption by adults as function of physical activity at work and in spare time. Non-active people were found to consume about 1.30-1.35 L/day, whereas people that were extremely active during work or in spare time consumed 1.72L and 1.57 L respectively.

Gender

In several studies no significant differences were found, regarding tap water consumption between sexes (Beaudeau *et al.* (2003), Gofti-Laroche *et al.* (2001), EHD (1981) and Hopkin and Ellis (1980)). This is contradicted by others. Haring *et a1*. (1979) found significantly higher water consumption in men, compared to women. This was also the case in the US EPA (2000) study, except for children age <l0 years. Also Levallois *et al*. (1998) reported males to consume more water than females, but the difference was not-significant, while Westrell *et al.* (2004) found higher frequency of women consuming more than 1 litre of cold tap water in a Swedish country based study with more than 10 000 respondents.

Shimokura *et al*. (1998) found only minor differences in water consumption between pregnant women and their male partners. Larger differences were observed by extent of employment (full time vs. part-time).

Age
Varying results were obtained in relation to age and drinking water consumption. Beaudeau *et al.* (2003) and DWI (1996) reported an increase of consumption with age, up to the age of 50 years, where after it gradually decreased.
Levallois *et al*. (1998) found no clear association between the total amounts of water consumed within different age strata, but there was a non-significant tendency for the older people to drink more than the younger ones. Westrell *et al.* (2004) found the highest consumption in people age < 40 and > 60 year.

Other factors
Ershow *et al*. (1991) reported for US that next to pregnancy the most striking effects on total water intake were due to regional variations in residences. Total water intakes were lowest in the northeast, intermediate in the south and Midwest and highest in the west of the USA. In Sweden Westrell *et al.* (2004) found a trend of increasing water consumption the further north the county was located and higher in the countryside than in large cities.
EPA (2000) concluded that pregnant women do not differ significantly in their water intake compared to other women of childbearing age but lactating women consumed significantly more water than pregnant women and women of reproductive age. Zender *et al.* (2001) found no significant differences in tap water consumption at work between pregnant and non-pregnant women.
Also social or cultural differences in drinking water consumption may exist both in relation to the amount and type of water consumed. Differences between countries, as well as between ethnic groups within one country may occur (Williams *et al*., 2001).
Westrell *et al.* (2004) found a significant decrease in cold tap water intake with increasing yearly income. Tenants had a higher water consumption than people who owned their residence.
Another factor influencing drinking water consumption is the medical status of the consumer. Some diseases like diabetes and conditions requiring rapid rehydration needs (gastrointestinal upsets, food poisoning), or disorders of water and sodium metabolism may necessitate high levels of water intake. For other diseases it is sometimes recommended to have low consumption of unboiled drinking water (e.g. HIV infected

persons). Westrell *et al.* (2004) concluded that people who regarded themselves to be of very bad health had high intake of both cold tap water and tap water added to beverages within their home. However, this groupalso included the highest proportion of non-consumers.

6.3.3 Other routes of tap water intake

Other routes of tap water intake should be taken into account in the assessment of the exposure but information is scarce. Routes for intake of cold tap water other than direct consumption include ice cubes, food preparation, intake of medicines with water and tooth brushing. Reported water intake via food was 0.02-0.1 L/day (Levallois *et al.,* 1998; Ershow and Cantor, 1989 cited by Levallois *et al.*, 1998; Gofti-Laroche *et a1*., 2001).

6.4 STATISTICAL ANALYSIS OF WATER CONSUMPTION

Datasets were obtained from Australia, The Netherlands, Great Britain and Germany on consumption of cold unboiled tap water. These datasets and fitted statistical distributions are further described in the following paragraphs. To characterize the gathered data the mean, median and spacing breadth (difference between the 5 and 95% confidence limit) have been calculated for each data-set.

6.4.1 Australia

In a pilot study reported by Robertson *et al.* (2000a) both a questionnaire and a diary study were included. This study was conducted in Melbourne between September and December 1997 with 253 respondents. After the first questionnaire was administered, participants were mailed the diary. Four weeks after the original interview, the same questionnaire was repeated. Water intake was reported in average sized glasses, which were assumed to contain 250 ml. The questionnaire inquired about cold tap water consumption in general (food frequency). Instruction with the diary asked participants to record their intake as soon as possible over a four-day period.

A follow-up investigation with questionnaires was conducted in case-control studies on sporadic cryptosporidiosis (Robertson *et al.,* 2002) . These were conducted in Melbourne from June 1998 to May 2001 and in Adelaide from November 1998 to May 2001. The population did not reflect the general population (median age 11 years).
The questionnaire covered demographic information, clinical details of the case's illness, education level, employment, consumption of tap water on a usual day, consumption of particular food groups and other possible risk factors for cryptosporidiosis.

Results pilot study Melbourne

Table 6.3 presents the statistical consumption data from the questionnaires and diary studies. The empirical data are presented as histograms in figure 6.3-6.5.

Table 6.3. Statistical data characteristics

Parameter	Consumption (glasses/day)		
	Questionnaire 1	Diary	Questionnaire 2
Mean	3.964	3.566	3.856
Median	4.00	3.00	3.00
Spacing breadth	8.00	7.80	9.00
N	253	234	231

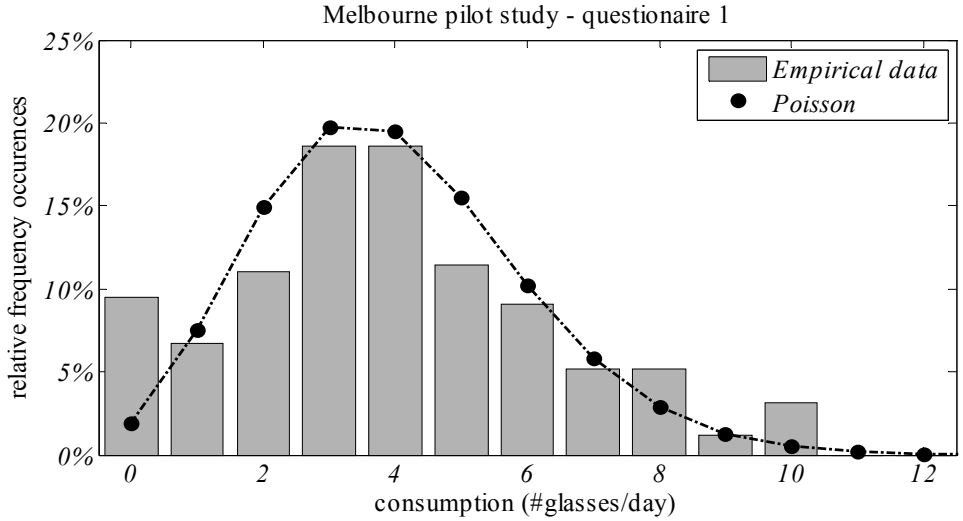

Figure 6.3. Statistical probability distributions for discrete momentous tap water consumption Melbourne pilot study – questionnaire 1

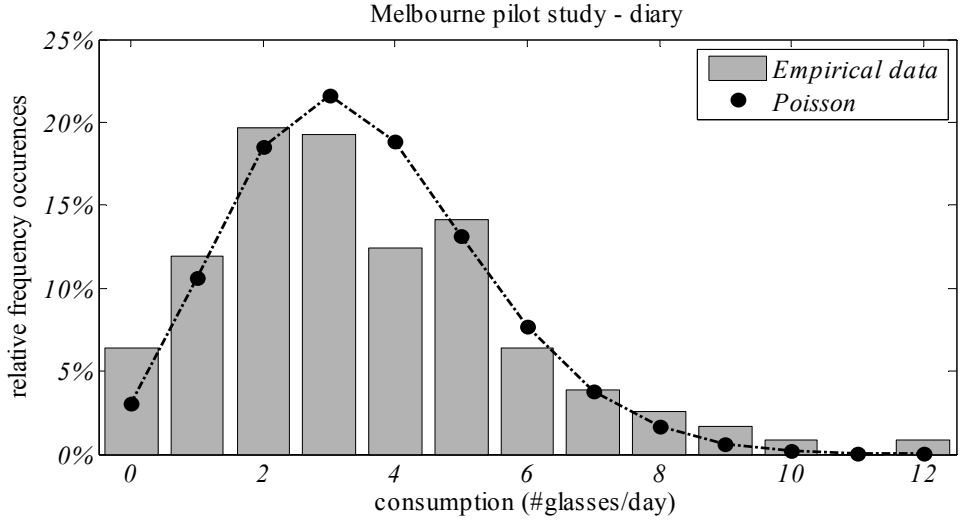

Figure 6.4. Statistical probability distributions for discrete momentous tap water consumption Melbourne pilot study – diary

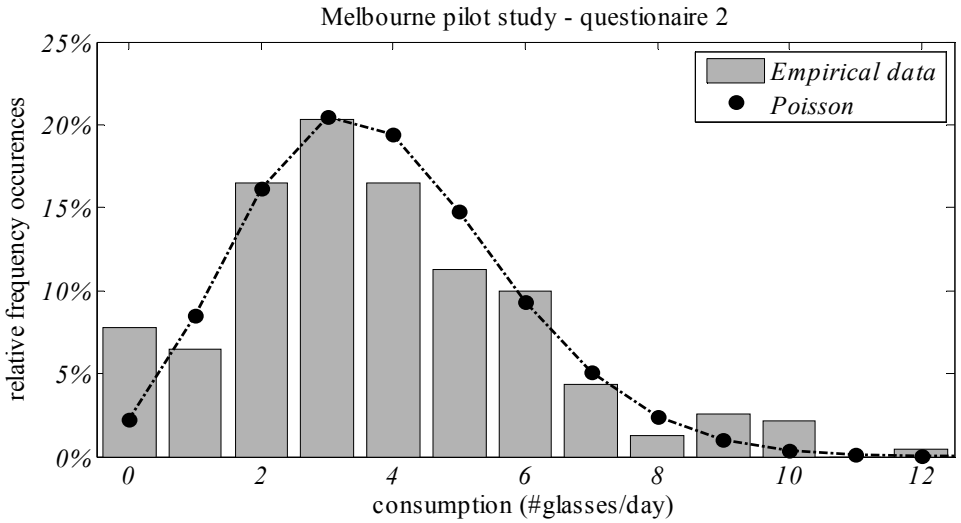

Figure 6.5. Statistical probability distributions for discrete momentous tap water consumption Melbourne pilot study – questionnaire 2

Discussion and conclusions pilot study Melbourne

Robertson *et al.* (2000) concluded that there was only moderate agreement between the telephone questionnaire and diary recordings. This may be true at the individual level, but the differences are much smaller when considering the population as a whole. To analyse possible differences between the questionnaires and the diary in the study population we conducted the non-parametric rank sum test of Wilcoxon (Table 6.4).

Table 6.4. Analysis of differences between questionnaires and diary in pilot study Melbourne

	Questionnaire 2	**Diary**
Questionnaire 1	p=0.4351	p=0.0899
Diary	p=0.2498	

It can be concluded that there are no significant differences between the three parts of the pilot study as $p > 0.05$ for all three comparisons.

Results follow-up study: Melbourne

In the cryptosporidiosis case-control study in Melbourne the same questionnaire was administered as in the pilot study. Table 6.5 presents the obtained statistical data and Figure 6.6 the empirical data. To compare the data and determine the goodness of fit of the different statistical distributions the mean error, Root Mean Square Error (RMSE) and the Fraction Declaring Variance (FDV) were determined for each dataset. For satisfactory fit the mean error should approach zero, the RMSE should be low and the FDV should be close to 100%. These performance indicators are given in Table 6.7 and show that the Poisson distribution fulfils all criteria best.

Table 6.5. Statistical data characteristics

Parameter	**glass/day**

Mean	3.368
Median	3.00
Spacing breadth	8.00
N	950

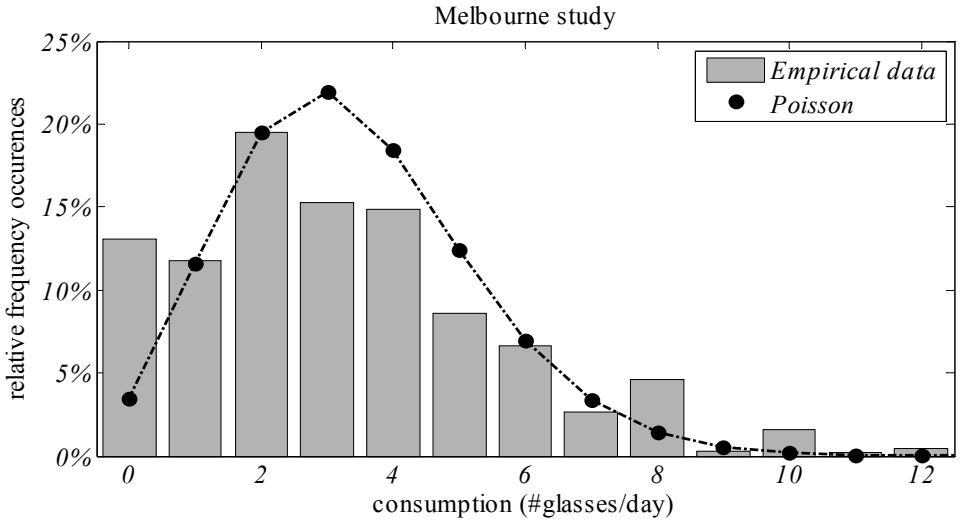

Figure 6.6. Statistical probability distributions for discrete momentous tap water consumption

Table 6.6. Statistical distribution performance indicators discrete momentous tap water consumption

Parameter	**Poisson**	**Exponential**	**Gamma**	**Lognormal**
Mean Error	**0**	**0**	0.0023	**0**
RMSE	**0.0315**	0.1623	0.0382	0.0478
FDV (R^2)	**82.75%**	15.85%	71.52%	60.64%
Distribution parameters	λ= 3.3684	α = 0.4522 β = 0.6394	α = 1.7983 β = 1.8731	m = 1.1467 s = 0.6496

Boldface: best performance

Results follow-up study: Adelaide

In the case-control study in Adelaide the same questionnaire was administered as in the pilot and the follow-up study in Melbourne. Table 6.7 presents the obtained statistical data, Figure 6.7 the empirical data and Table 6.8 the performance indicators.

Table 6.7. Statistical data characteristics

Parameter	**glass/day**
Mean	2.87
Median	2.00
Spacing breadth	8.00
N	644

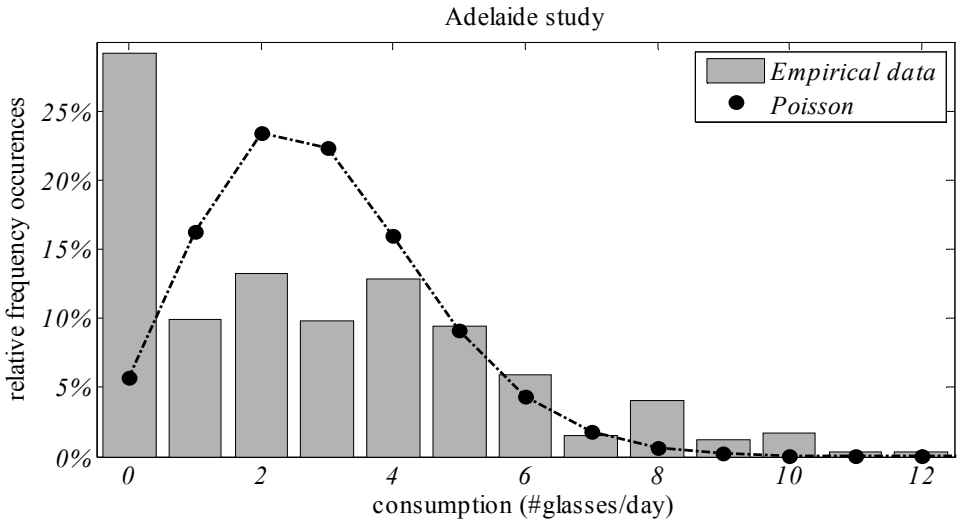

Figure 6.7. Statistical probability distributions for discrete general tap water consumption

Table 6.8. Statistical distribution performance indicators discrete general tap water consumption

Parameter	**Poisson**	**Exponential**	**Gamma**	**Lognormal**
Mean Error	**0**	0	0	0
RMSE	**0.0594**	0.7224	0.0739	0.0663
FDV (R^2)	**41.01%**	15.83%	20.44%	23.26%
Estimated parameters	λ = 2.8676	α = 0.5538 β = 2.4293	α = 0.9542 β = 3.0051	m = 1.1843 s = 0.6854

Discussion and conclusions

From table 6.6 and 6.8 it can be concluded that the Poisson distribution best fits the data both for Melbourne and Adelaide. The Poisson distribution also provided the best fit for the pilot studies (data not shown). The data from Adelaide is however low for R^2 of the FDV compared to the other Australian datasets. This is mainly due to the high percentage of non-consumers in Adelaide, potentially affected by the historically lower aesthetic quality of the Adelaide drinking water. Additional analysis excluding the non-consumers (results not shown) did not increase the performance of the probability distribution functions.

In all Australian recall (questionnaire) studies, the percentage of non-consumers was higher than the percentage of consumers drinking one glass, except for the diary study (pilot Melbourne). This suggests that the recall studies may have overestimated the percentage of non-consumers. We therefore consider the results from the diary study in Melbourne to be the most valuable. This is in line with the conclusions by Robertson *et al.* (2000a) who concluded that the questionnaire was less accurate than the diary.

To analyse for possible difference between the distributions of the final studies in Melbourne and Adelaide and the pilot study the non-parametric rank sum test of Wilcoxon is conducted (Table 6.9).

Table 6.9. Analysis of differences between the results of the final studies in Melbourne and Adelaide and the pilot study in Melbourne

		Adelaide	Melbourne		
		Final study	Final study	Pilot study Quest. 2	Pilot study Diary
Melbourne	Pilot study Quest. 1	0*	0*	0.4351	0.0899
	Pilot study Diary	0*	0.0959	0.2498	-
	Pilot study Quest. 2	0*	0.0027*	-	
	Final Study	0*	-		

* significant difference ($p < 0.05$).

It can be concluded that the data from the follow-up study in Melbourne do not differ significantly from the diary in the pilot study, but they do from the data from the two questionnaires in the pilot study.
The data from the follow-up study in Adelaide differ significantly from the data from both the pilot study and the final study in Melbourne .

6.4.2 The Netherlands

In the Dutch National Food Consumption Survey 1997/1998 data on cold tap water consumption were included (Anonymous, 1998). During this two-day diary survey consumption data on cold tap water were obtained for 6250 respondents. Consumption was registered in grams per person. To obtain a time homogenous dataset each participant wrote down the consumption during two separate random days. Trained dieticians visited the households in advance for instruction and afterwards for collection and control of the diaries and to measure the volume of the used drinking vessels.
Table 6.10 and Figure 6.8 present the characteristics and distribution functions of the tap water consumption data. For discrete analysis of the data, the continuous data in litres per day were translated into discrete values of glasses per day, assuming a glass to be 250 ml. Due to the large number of participants in the survey (6250), the internal variation in glass capacities can be considered irrelevant in comparison with the external variation between the respondents.

Table 6.10. Statistical data characteristics The Netherlands (discrete)

Parameter	Glass/day
Mean	0.706
Median	0.00
Spacing breadth	3.00
N	6250

Discussion and conclusions
The mean consumption of cold tap water (0.177 Liter) is much lower than in the Australian studies, but in line with earlier studies in the Netherlands (see Table 6.2). In The Netherlands a smaller study more specifically on drinking water consumption was analysed by Teunis *et al*.

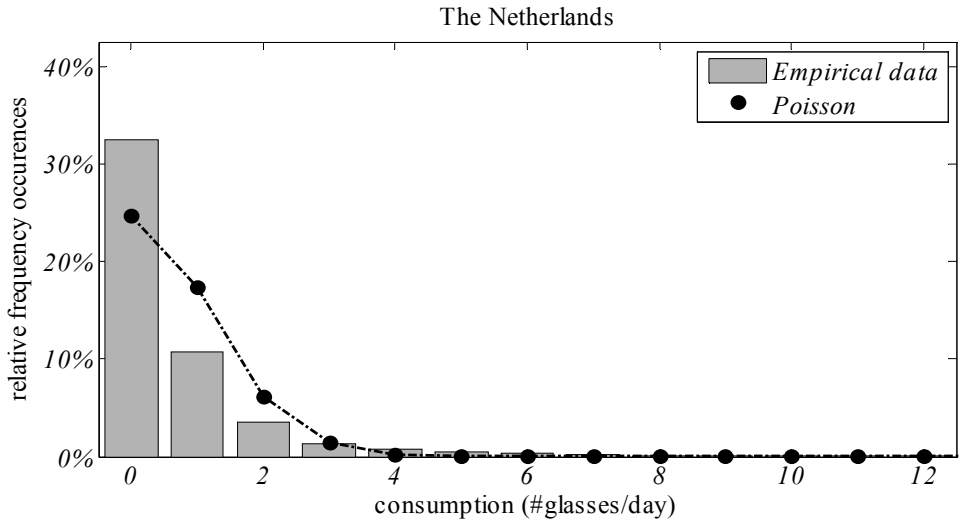

Figure 6.8. Statistical probability distributions for discrete tap water consumption

(1997) who, similar to Roseberry and Burmaster (1992), fitted their data to a Lognormal distribution. The median concentration they reported was 0.153 litres/day and the average 0.222 litres/day. The data in our study yield a median consumption of 0.052 litres/day and an average of 0.177 litres/day.
The distribution of the Dutch consumption data is very skewed towards low consumption volumes (Figure 6.8). It can be seen that the observed number of zeros is higher than expected from the Poisson model. More than half of the respondents (appr. 65%) reported no cold tap water consumption at all. This is considerably higher than earlier Dutch studies and might be the result of the study design. In all the other studies, respondents are specifically asked to answer questions about consumption of tap water. In this food frequency survey, respondents are asked to record all food intake in a diary. A possible explanation for the high percentage of non-consumers is that the respondents have not regarded tap water consumption as *food* intake. Analyses of the data excluding the non-consumers (data not shown) resulted in less performance of the statistical probability distributions than for the overall data including non-consumers. Because of the very skewed distribution it is difficult to derive an average consumption figure from the data. For QMRA, we recommend to use the data themselves to describe the variability of consumption or to use the Poisson distribution since this gives higher probabilities for consumption and is therefore more conservative than the Exponential distribution (see Figure 6.8).

6.4.3 Great-Britain

In Great-Britain a case-control study on sporadic cryptosporidiosis was conducted by Hunter *et al.* (2004) from February 2001 to May 2002. The questionnaire was completed by 427 patients and 427 controls but did not reflect the general population, due to a high percentage of children (50% of the population was of age < 13 years).
Questions were asked on several possible risk factors for cryptosporidiosis. Considering cold tap water consumption the first question was whether the consumer *in general* consumed cold tap water, or drinks containing cold tap water. If the answer was 'yes',

the next question was how many glasses per day, assuming one glass to be 1/3 pint (≈190 mL). These questions were repeated considering cold tap water consumption during the *last two weeks*. The latter was especially important for the water consumption by the cases. However, as it is not clear whether water consumption by the cases was influenced by the fact that they had been ill, these data were left out.

Statistical Analysis

In Table 6.11 the data characteristics are presented for the 2 week based and the general data. Histograms of the consumption data and the Poisson distributions are presented in figure 6.9 and 6.10.

Table 6.11. Statistical data characteristics Great Britain (controls only)

Parameter	**Consumption (glass/day)**	
	2 week recall	**general recall**
Mean	2.815	4.748
Median	2.500	4.00
Spacing breadth	3.00	15
N	416	421

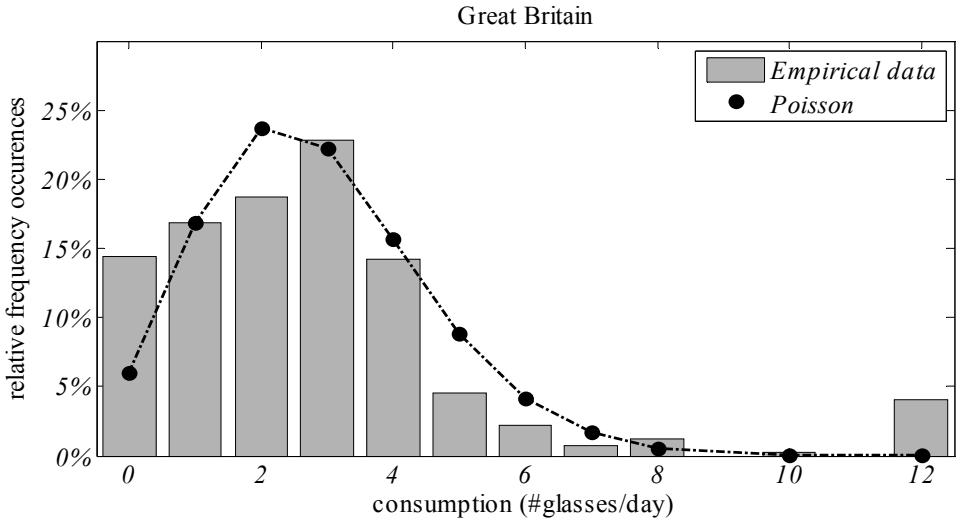

Figure 6.9. Statistical probability distributions for discrete 2 week based tap water consumption (controls only)

Discussion and conclusions

In Figure 6.10 it can be seen that the data regarding general consumption show no smooth distribution, but two separate data blocks. In the first block (0 – 6 glasses per day) all outcomes have more or less similar frequencies. A possible explanation for this result might be that below a certain level (in this case six glasses or less) the respondents' feeling about the general daily consumption is rather indiscriminate. For example the perception that consumption is three glasses per day might be similar to the perception of consuming four or two glasses per day. The empirical distribution of the 2 week based consumption data follow a more smooth line (daily consumption of more than 12 glasses per day were combined into one class).

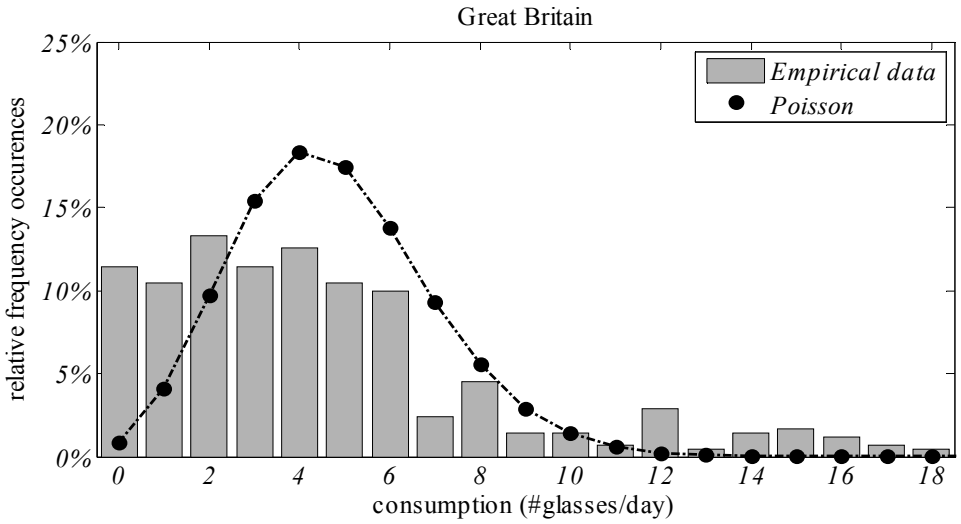

Figure 6.10. Statistical probability distributions for discrete general tap water consumption (controls only)

Visually the distributions of the general consumption and the 2-week consumption (figure 6.9 and 6.10) look quite different from each other. However, despite the apparent differences, statistical analysis does not substantiate this assumption and rejects significant differences. From the data no difference between the medians can be concluded (nonparametric Wilcoxon rank sum test: p=0.1540 and α=5%). Also both empirical distributions do not show significant statistical differences (Pearson Chi-square test: p=1,00 and α=5%). Considering tap water consumption in general none of the distributions performed very good and none surpassed the others. For the 2 week based consumption data the Poisson distribution performed best and the Exponential distribution performed worst.
We consider the 2 week based consumption data to be preferred above the general consumption data because the Poisson distribution on the 2 week based data set obtained the best performance. In addition the empirical distribution of the 2 week based consumption data is smoother. We also believe that the short term data will be more precise because recall bias will be less for recent consumption than for consumption in general.

6.4.4 Germany

Dangendorf (2003) conducted a telephone survey (food frequency questionnaire type) about the distribution of gastrointestinal diseases in a region in Germany (Rheinisch-Bergischer Kreis). In total 195 persons between 14 and 88 years old were interviewed and also asked about the consumption of cold tap water in general. The period of survey covered the summer months of 2000, as well as the winter months January - March of 2001, in order to account for possible seasonal fluctuations of tap water consumption.

Consumption of cold tap water was estimated in cups a day (assuming 150 ml/cup). Consumption of more than 3 cups (ca. 0.5 L) was estimated in multiple units of 0.5 L. (0.5L, 1.0 L, 1.5 L, 2.0 L, 2.5 L).

Statistical Analysis
In table 6.12 and figure 6.11 the data characteristics, a histogram and the modelled Poisson distribution are presented.

Table 6.12. Statistical data characteristics

Parameter	**glass/day**
Mean	2.508
Median	1.00
Spacing breadth	7.00
N	195

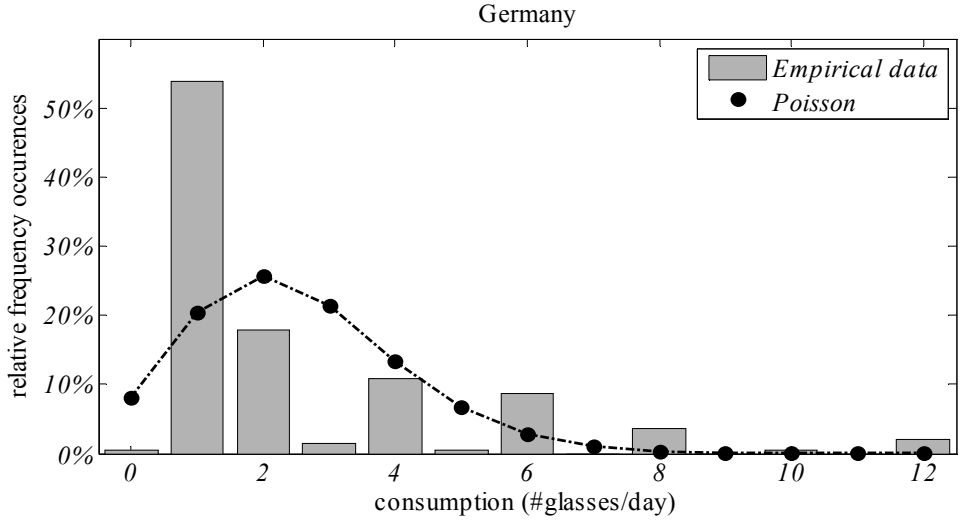

Figure 6.11. Statistical probability distributions for discrete general tap water consumption

Discussion and conclusions
Within the original data set, the results were divided into non-equidistant classes. However, for the fitting of statistical probability distribution functions to discrete consumption in glasses per day, equidistance is recommended. Therefore the original data were transformed into equidistant discrete data (nr of glasses per day, assuming one glass to be 250 mL) before the statistical analysis was conducted. From figure 6 it can be seen that the obtained empirical distribution does not follow a smooth line. One of the causes is the fact that the number of non-consumers is remarkably low compared with the number of people drinking one glass per day. This disjunction is also expressed by the fitted statistical probability distribution functions. None of the proposed functions is able to fit both the low value for non consumption as well as the high frequency for one glass per day.
This is possibly caused by the design of the interview and the way of questioning. In this study questions on consumption were asked like: "How much plain tap water do you consume?" (And then suggesting:) "2 or 3 cups, less or more?" This way of questioning can suggest the consumer that the answer of consumption of 0 cups is less

likely, or maybe even less preferred. In the German data set only one respondent indicated to drink no tap water (0.5%) whereas more than half of the respondents (54%) indicated to drink one glass.

Because of the lack of harmony between the zero and one value of the empirical distribution as well as the non-equidistance of the original gathered data there is not a satisfactory way for statistical analysis. Therefore, it is not possible to draw conclusions about the underlying statistical probability distribution function and about the consumption behaviour of the respondents.

6.5 DISCUSSION & RECOMMENDATIONS

6.5.1 Design of the study

The evaluation of the literature leads to several recommendations about study design:

1. Use a diary study.
In the analysis of the Australian data it was demonstrated that estimations of drinking water consumption were higher in the questionnaires than in the diaries. Similar findings were reported by Kaur *et al.* (2002), and Levallois *et al.*(1998). Also the number of non-consumers was higher in the questionnaire studies compared to the diary study. Therefore we believe the diary is to be preferred for collecting water consumption data.

2. Record consumption for 3-4 days.
The longer the period for data collection, the more representative data can be obtained. On the other hand, if the duration of the study is too long this might result in less accurate reporting. We believe that probably 3-4 days would be most feasible.

3. 24-h recall can be an alternative.
If a diary study is not possible because of limitations in time or money, a 24-h recall is an appropriate alternative. In order to get more information of the within-person variation, it is advisable to repeat the 24 h recall at least once on a non-consecutive day (Brussaard *et al.*, 2002).

4. Use more than 2000 persons.
To include variation between respondents a large number of respondents should be questioned However, the number of repeated measurements and participants needed in dietary surveys are often a compromise between theoretical considerations (e.g. reliability of the index number calculated) and practical constraints (costs, respondent burden etc). Taking such considerations into account Brussaard *et al.(* 2002) concluded that a minimum sample size of 2000 adults in each country will be needed in order to identify trends in the mean intakes of foods and nutrients in Europe. To increase the

participation rate, measures such as sending a letter in advance explaining the study, special training of interviewers and money incentives should be considered.

5. Distribute moments of data collection over a year.
To obtain a generalisation in time, the moments of data collection should be homogenously distributed over one or more years.

6. Ask for number of glasses/cups and use pictures.
The water consumption data can be collected as continuous data (e.g. grams or litres per day) or as discrete data (e.g. glasses per day). From a statistical point of view, continuous data are preferable above discrete data because of the lack of classes. However, it can be questioned whether in theory tap water consumption is distributed continuous or discrete. During continuous measurements the consumer is often asked the number of glasses or cups consumed and afterwards this is recalculated to millilitres or litres. The actual result of this way of gathering data is false continuous data. Discrete data also have the advantage that they are easier to collect than continuous data. When collecting data in discrete measures the volume consumed will be best estimated by measuring the volume of the used drinking vessels by the interviewer or with the use of pictures of cups and glasses.

Example questions/questionnaire:

- *Do you drink plain unboiled water? (Yes/No)*
  (if yes:)
- *How many average sized glasses over 24 hours on a usual weekday including water you put into cordials and juices?(NB use 250 mL glass as average)*

During the study attention should be paid to the way of questioning to avoid wrong representation of non-consumers. E.g. avoid suggestive questions like: How much water do you drink, 1 cup, 2 cups, or more?
In addition avoid discrete classes of unequal size (e.g. 1 cup, 2 cups, 3 cups, 1 L, 1,5L, 2L)

7. Consider differences in study design when comparing different studies.
When comparing studies on tap water consumption conclusions regarding differences in consumption between countries, sexes etc. should be drawn very carefully taking into account the many differences in study design. Attention should be paid to the study population (specific group or whole population), the moment/season of data collection within a year, the methods of data collection (e.g. diary record or recall), the method to assess the volume tap water consumed and the types of water included in the surveys (food, medicines, lemonade, ice cubes etc.). The experiences of this study illustrate that these factors can have large impact on the (distribution of the) consumption data.

6.5.2 Statistical distribution

1. Describe the data with the Poisson distribution.

To account for the variability in water consumption over the population, a statistical distribution can be fitted to the consumption data. The Lognormal distribution, as suggested by Roseberry and Burmaster (1992), did not provide the best fit to the consumption datasets we examined. In the Lognormal distribution the number of non-consumers is per definition 0, while the UK, Australian and Dutch dataset contained 7-65% non-consumers. Tap water consumption (or at least the way information on consumption is collected) is more a discrete than a continuous parameter Therefore, the Poisson distribution is more appropriate and proved to have a good fit to the datasets. The Poisson distribution also has the advantage that parameter estimation is easy.

2. Give attention to non-consumers.
In the Dutch data, the fraction of non-consumers was approximately 65%, which is very high compared to the data from Melbourne and Great Britain. The fraction of non-consumers is an aspect that needs attention in the statistics of water consumption studies. In most data-sets that were tested, the number of observed non-consumers differed considerably from the number of non-consumers estimated by the Poisson distribution (and the other statistical distibutions). Because the fraction of non-consumers did not fit the statistical distributions well, a second method of statistical data analysis was applied to the studies. The idea was that by eliminating the derogatory value of non-consumption a more smooth empirical distribution could be obtained. However, the fitted statistical distributions performed less. Therefore, it was better to fit the statistical probability distribution functions on the total dataset, including the non-consumers.

6.5.3 Recommendations for the estimation of water consumption in QMRA

1. Use country specific data.
For QMRA, it would be best to use country specific consumption data and statistical distributions, if available. For the average consumer, the reported mean consumption of cold tap water varies between 0.10 – 1.55 litres. Differences occur between countries, but also within countries (see Table 6.2).

2. Use data from the best study design.
If more datasets are available for a country, we recommend to select the data that have been collected with the best study design. If the selection cannot be based on study design, the study that yields the highest consumption data should be used, as conservative estimate of the consumption of cold tap water.

3. Describe the variation with a Poisson distribution.
To be able to include the variation in tap water consumption in the QMRA, the data can be fitted to a Poisson distribution. If the observed distribution of the water consumption (the histogram of the data) shows large discrepancies with the Poisson distribution that is fitted to the data (for instance in the number of non-consumers), it is possible to use the data themselves, rather than the Poisson distribution.

As the only parameter for the Poisson distribution is the mean, the distribution can be used to estimate the variation of consumption if only the mean consumption is known.

4. Consumption data for Great Britain, Australia, The Netherlands and Germany.
For Great Britain, a Poisson distribution with a mean of 2.81 glasses/day (2 week-recall) can be used in QMRA and for Australia a Poisson distribution with a mean of 3.49 glasses/day (diary study, Melbourne) can be used. For The Netherlands, the Poisson distribution with a mean of 0.71 glasses/day can be used. For Germany, the Poisson distribution did not match the observed data and it is suggested to draw from the observed dataset themselves and not from a statistical distribution.

5. And if no country specific data are available?
If no country specific data are available we recommend to use the Australian distribution data from the Melbourne diary study (Poisson, λ=3.49 glasses/day) as conservative estimate, because the water consumption in these data is relatively high and the data are collected in a well-designed study.

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7. QMRA methodology

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Quantitative microbial risk assessment (QMRA) of drinking water systems requires the quantification of pathogen occurrence in source water and their removal through various treatment barriers to the consumer's tap. When pathogen occurrence at the tap is combined with consumption patterns and pathogen dose-response relationships, the risk of infection (or other end-points) can be estimated.

In this chapter, a framework for calculating and characterising the microbial risk from drinking water is presented (Figure 7.1). The process of quantifying model inputs and choosing numerical values for each variable is critical to the QMRA process, and yet potentially daunting for the risk analyst. Pathogen sources, transport and removal are complex processes dependent on many factors including hydrology, climate, land use, hydrodynamics, disease incidence, process design and performance, and unpredictable human behaviour.

Experimental data is key, however datasets that relate directly to the variables of interest are limited. Analyses are costly, and pathogens generally occur at low densities in environmental waters making detection difficult. Datasets are characteristically small in size and often contain low numbers and many non-detects. Statistically, analysis of microbial datasets for characterising inputs to QMRA is therefore a less usual case. Whereas most traditional statistical methods are designed to analyse large datasets of relatively low variance, microbial datasets are generally small datasets of high variance with large uncertainties.

The role of the risk analyst is to determine what the limited monitoring or experimental results reveal regarding the magnitude and variability of each quantitative input to the risk model. Statistical approaches appropriate for describing model inputs for QMRA from microbial data are therefore presented. Examples are given that require a specific and sometimes rigorous consideration of the relevant experimental data collected during the MicroRisk project. The authors argue that the greatest value will be obtained from the experimental data when the statistical analysis approach is tailored to the individual dataset. The aim of these detailed analyses is to learn as much as possible from the available information regarding appropriate quantitative estimates of model inputs, and the uncertainties associated with these estimates.

Uncertainties[1] in microbial risk modelling are important to untangle from variability[2]. The risk management implications of an isolated probability of infection estimate can be difficult to interpret without further understanding of how and why model inputs may vary and where the most important sources of uncertainty lie. The aim of this chapter is not only to demonstrate how risk calculations were undertaken, but also to

[1] Uncertainties arise due to lack of precise knowledge of the input values or to lack of knowledge of the system being modelled, more data typically reduces these uncertainties.

[2] Variability refers to observed differences attributable to true heterogeneity or natural diversity in a parameter that cannot be reduced by additional data collection (but can be better characterised).

give some guidance regarding the interpretation of uncertainty associated with modelling results.

In the MicroRisk project, a pragmatic approach to characterising uncertainty was applied by combining traditional quantitative methods and a more semi-quantitative approach drawing on expert opinion. An expert may have prior knowledge that the estimated value for a model variable (quantified based on a small or surrogate dataset) is unlikely to be representative. In fact, discussion of this prior knowledge, aimed at ensuring the representativeness of data and assumptions to the real systems and processes being studied, was identified as a critical component of the quantitative risk assessment process. The importance of these kinds of uncertainties (or scepticisms) on risk calculations was explored using sensitivity analysis.

A general framework for estimating pathogen risks from drinking water is illustrated in Figure 7.1.

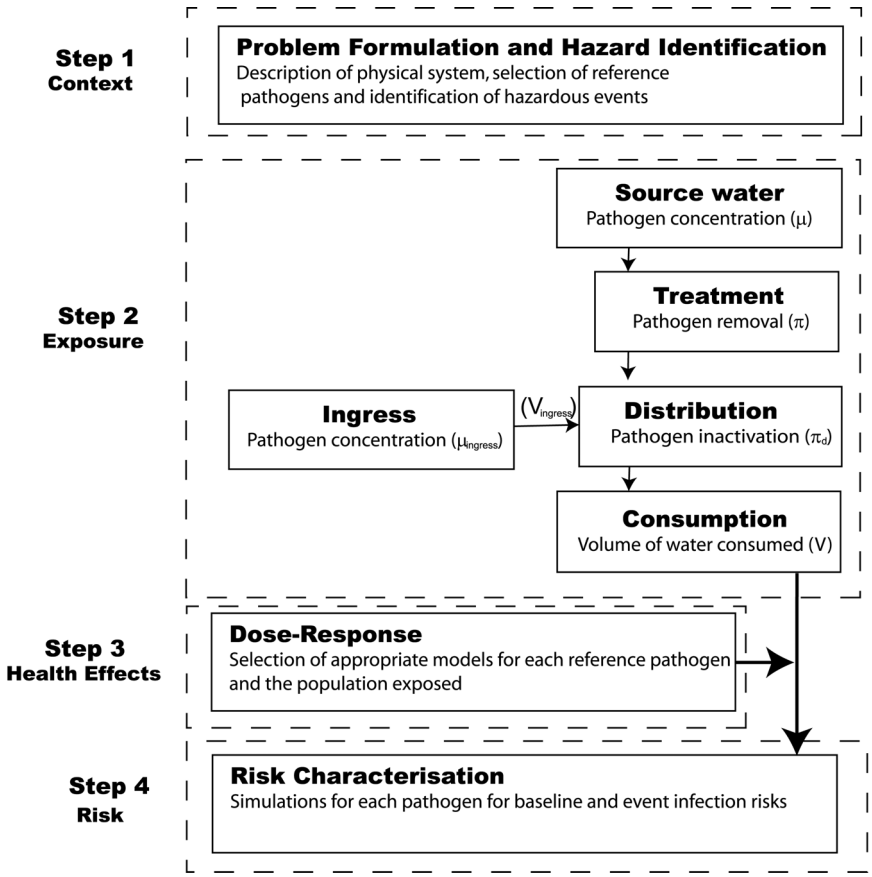


Figure 7.1 General framework for calculating microbial risk from drinking water

7.1.1. Step 1 – Context

The first step in the QMRA process is to define which pathogens will be modelled and what conditions will be investigated. These choices should be made so that the range of pathogen types are modelled under both baseline and hazardous event conditions, providing the context for the QMRA.

7.1.2. Step 2 - Exposure

Source Water

Pathogens may be present in the water column at the treatment plant off-take due to human and/or animal inputs (waterborne enteric viruses being assumed to only come from human excreta) within the catchment. The density of pathogens at the treatment plant off-take is dependent upon the magnitude of pathogen inputs and the environmental processes affecting their transport and inactivation; and is expected to vary both over time and between pathogen groups as described in Chapters 3-4.

To calculate microbial risk the density of pathogens (number of micro-organisms per litre) in the source water must be quantified and entered into the risk model. The main sources of information for quantifying pathogen density in source waters are:

- Water samples collected from the site and analysed directly for the presence of pathogens;
- Water samples collected from the site and analysed for (pathogen) index organisms combined with some assumptions regarding the ratio of index organisms to pathogens (direct analysis of pathogens is often not undertaken as analytical methods for detection are costly, and pathogens are often present in low densities requiring the collection of large volume samples); and
- Literature data from a catchment of similar pathogen sources and physical characteristics.

For some systems, significant changes in pathogen density can be linked to specific events affecting the mobilisation of micro-organisms from their source to surface waters such as rainfall induced runoff, and discharge of sewage overflows. Identifying the occurrence and impact of such hazardous events can facilitate understanding and management of the microbial risks for a given system. Describing the source water concentration for such a system by a single estimate may underestimate the peak risks, as high pathogen densities can be dampened by nominal low densities. A simple approach to address this is to describe separately pathogen densities under "event" and "nominal" conditions, leading to a bimodal description of pathogen density.

Treatment Efficacy

A wide range of treatment processes exist for the physical, chemical and microbiological purification of drinking water as indicated in Chapter 4. Each of these processes contributes to the removal or inactivation of pathogens from the water column. The effectiveness of each process in removing pathogens is variable: between different types of processes; between the same processes operated at different treatment facilities; and even variable over time for an individual process at a specified treatment plant.

Quantifying treatment removal performance for a drinking water CTS, accounting for the individual characteristics of the system being studied, and the expected temporal variability in performance for each process unit is a great challenge. Careful consideration of the available data is essential. In the QMRA framework, removal performance is represented by π (Figure 7.1) which is the fraction of organisms passing any treatment barrier (or barriers). When multiplied by the source water concentration ($\mu\pi$), the pathogen density in finished (treated) water may be estimated.

The primary sources of data for quantifying treatment performance (π) include:

- Pathogen densities at the inlet and outlet for a process or treatment plant;

- Surrogate densities at the inlet and outlet for a process or treatment plant; and
- Online performance data including turbidity, and chlorine residual.

Treatment efficacy is often reported as decimal elimination or Log_{10} reduction in micro-organism density. Log_{10} reduction is simply the Log_{10} of π, and therefore can be directly transformed to an estimate of π for input to the risk model ($\pi = 10^{Log10 reduction}$).

Distribution

An ideal distribution system protects water quality as it transports treated water from the plant to the consumer's tap. The only effect on pathogen density should be a reduction due to inactivation with travel time (increased in the presence of a disinfectant residual), and incorporation into pipe biofilms.

In reality however two types of events in the distribution system may lead to an increase in the pathogen concentration between the treatment plant and the consumer:

1. **Deficiencies in the distribution system** may lead to the ingress of pathogen contaminated material including cross-connections, contamination while in storage, contamination during construction or repair, and broken or leaking mains. In addition, common hydraulic transients may lead to contamination through negative pressure and subsequent intrusion of soil water.
2. **Biofilm sloughing events** (caused by shear force from changes in water flow or change in disinfectant concentration) may lead to incorporation of pathogen rich material from the internal pipe surface into the water column [Storey and Ashbolt, 2003].

Calculation of the impact of events within the distribution system on the microbial risk to the consumer requires the quantification of the frequency and duration of each type of event, along with the numbers of pathogens incorporated into the drinking water. Techniques for identifying the occurrence and impact of these events are still in their infancy and there is a great need for research in this area, as described in Chapter 5.

The current risk model oversimplifies the problem by looking at the impact of an ingress event on the pathogen concentration at the consumer's tap by considering the volume of contaminated material entering the water ($V_{ingress}$) and the pathogen concentration in the contaminated material ($\mu_{ingress}$). Within this framework, the relative importance of ingress events on consumer risk can be explored. The concentration of pathogens at the tap may be calculated using Equation 1.

$$\mu_{tap} = X\mu_{ingress} + (1 - X)(\mu.\pi)$$

Equation 1

Where: $X = \dfrac{V_{ingress}}{Q \times t}$ if no ingress event occurred, then $X = 0$

(X) represents the proportion of external (ingressed) material present in the water column at the tap; $V_{ingress}$ is the volume of contaminated material entering the water column over time t for an ingress event; and Q is the flow rate in the pipe at the time of the ingress event.

No attempt has been made to quantify the impact of pathogen incorporation into and subsequent sloughing of biofilms. Given the oversimplified framework, however, the sloughing of biofilm could be tested as a special case of an ingress event where the

estimated pathogen density in the biofilm is combined with the volume of material sloughed into the water with the subsequent concentration at the tap calculated using Equation 1. The likelihood and overall impact of these ingress events on the probability of infection to the consumer may then also be characterised (see section 7.1.4).

Consumption

The volume of water consumed must be quantified in order to estimate the dose of pathogens. Results from analysis of unboiled tap water consumption patterns (Chapter 6) indicate that the amount of water consumed is influenced by many factors including age, culture (or nationality) and level of physical activity. The volume of water consumed (litres per day) is multiplied by the pathogen concentration at the tap to calculate the total exposure or dose ($Dose = \mu_{tap} \times V_{consumed}$) per day.

7.1.3. Step 3 – Dose-Response

Dose-response modelling is the key to microbial risk assessment as it provides a link between exposure dose and the probability of infection. Prior to dose-response relationships, human feeding experiments were only used to estimate infectious doses such as ID_{50} or minimum infective dose (MID). However, in more recent years it has become clear that infection is theoretically possible from exposure to a single organism, and the use of models based on the 'single-hit' theory of dose-response have increased [Regli *et al.*, 1991; Haas *et al.*, 1993; Gerba *et al.*, 1996b].

Dose –response models

Quantitative dose-response models have been developed to estimate the probability of infection based on the average pathogen dose [Haas *et al.*, 1983]. While the average dose of pathogens is continuous and can potentially take any value, the actual number of organism that an individual may consume is a discrete quantity (i.e. it is not possible to consume 2.67 *Cryptosporidium* oocysts, but rather given an average dose of 2.67 most individuals would consume 2 or 3 oocysts with a fewer number consuming lower [0, 1] or higher numbers [6, 7]). Beginning with the average dose, the calculation of probability of infection is a two step process, being the combined probability of exposure and infection shown in Equation 2.

$$P(\text{inf} \mid \mu) = \sum_{n=0}^{\infty} P(n \mid \mu) \times P(\text{inf} \mid n)$$

Equation 2

Where: $P(\text{inf} \mid \mu)$ is the probability of infection given the mean pathogen density.

$P(n \mid \mu)$ is the probability of exposure to *n* organisms given the mean pathogen density μ.

$P(\text{inf} \mid n)$ is the probability of infection given exposure to *n* organisms

The distribution of pathogens in the exposure media is assumed to be random, and therefore the probability of exposure to *n* organisms when the mean concentration is equal to μ (P(n| μ)) is given by the Poisson distribution.

When an individual organism is ingested, the probability of that organism successfully overcoming host barriers and reaching a site for infection may be

represented by *r*. If every organism is assumed to behave independently from other organisms within the host, then the overall probability of infection may be described as a binomial process. That is, each ingested organism may result in one of two outcomes; infection or not infection. If the probability that an individual organism may cause infection is denoted by *r*, then the probability of not being infection is equal to (1-*r*). Over a series of *n* independent trials (in this case, number of organisms consumed), the probability of not being infection is equal to $(1\text{-}r)^n$, and hence the probability of at least one organism being successful in causing infection is the complement:

$$P(\inf \mid n) = 1-(1-r)^n$$

Equation 3

The implementation of the Poisson pathogen distribution and binomial probability of infection (Equation 3) leads to a family of models referred to as single-hit models, where the name relates to the concept that only a single organism is necessary to cause infection. The simplest form of the single-hit model assumes that for a given pathogen, every pathogenic particle within every host has the same constant probability of survival, given by *r*. When combined with the P(n| μ), the dose-response relationship is the exponential model.

Exponential model: When organisms are distributed randomly (Poisson) and the probability of infection for any organism equals r then:

$$P_{\inf} = 1-e^{-r\mu}$$

Equation 4

While the exponential model is simple, the practical implications are unsatisfying since the between pathogen variation in infectivity, and between host variation in susceptibility is ignored. This limitation is partially overcome by Beta Poisson model.

Beta Poisson model: When *r* is assumed vary according to a beta distribution, a complicated dose-response relationship emerges containing a confluent hypergeometric function [Haas *et al.*, 1999]. Furumoto and Mickey [1967] made some simplifying assumptions to this relationship, and derived a simple dose-response relationship referred to as the Beta Poisson:

$$P_{\inf} \approx 1-\left(1+\frac{\mu}{\beta}\right)^{-\alpha} \quad \text{which holds when } \beta \geq 1 \text{ and } \alpha \leq \beta$$

Equation 5

The Beta Poisson approximation has been widely applied for describing dose-response relationships for QMRA. In some studies, the Beta-Poisson approximation has been applied even when the criteria for the parameter values (Equation 5) are not satisfied. A notable example is the dose-response relationship for Rotavirus infection fitted to data from Ward *et al.* [1986] with maximum likelihood parameters of ($\hat{\alpha}$ = 0.253, $\hat{\beta}$ = 0.422). The implications of this inappropriate application, particularly as it relates to the maximum risk curve are discussed below.

Maximum Risk Model: An important property of the single-hit relationship is that a maximum risk curve exists. The maximum risk curve is calculated when the probability that an ingested organism will pass the host's defense mechanisms and find a site suitable for colonisation is maximised and assumed equal to 1. The resulting equation is therefore the exponential dose-response function with $r = 1$.
This property is not retained by the Beta-Poisson approximation. In a study aimed at investigating the Rotavirus Beta-Poisson model fitted to data from Ward *et al.* [1986] with maximum likelihood parameters of ($\hat{\alpha}$ = 0.253, $\hat{\beta}$ = 0.422), the upper confidence level of the dose-response relation was shown to exceed the maximum risk curve [Teunis and Havelaar, 2000].
In addition for some models used in the MicroRisk calculations, the Beta-Poisson approximation was shown to exceed the maximum risk curve at low doses. This exceedance is illustrated for the *Campylobacter* model (again $\hat{\alpha}$ and $\hat{\beta}$ do not satisfy the criteria of approximation), in Figure 7.2[3]. The implication was that at low doses, the dose-response model was predicting theoretically impossible probability of infection estimates. As an alternative, for low doses (< 0.1 org.L^{-1}) the exact Beta Poisson model can be approximated by setting r (Equation 4) equal to the expected value of the Beta distribution ($\alpha/\alpha+\beta$)), thus avoiding this complication.

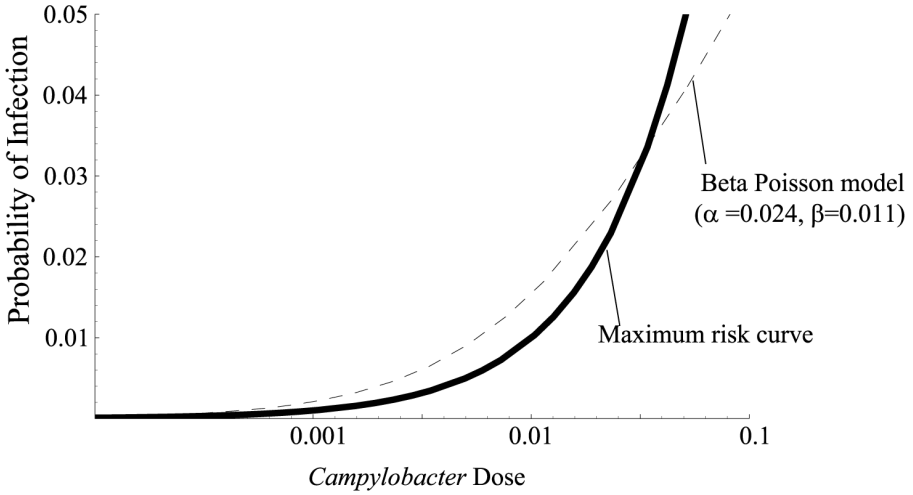


Figure 7.2. *Campylobacter* and maximum risk dose response curves at low doses

The maximum risk curve is also an important tool for uncertainty analysis, providing the upper bound of possible infection response. The importance of uncertainties in the calculation of the dose-response relationship can be screened using the maximum risk curve as a worst case sensitivity input.
The maximum risk curve could also be applied for risk assessment of pathogens with unknown properties. While for highly infectious pathogens the maximum risk curve appears to be a reasonable conservative assumption, it is however important to be aware that for less infectious pathogens, the maximum risk curve may significantly overestimate infection risk.

[3] Teunis *et al.* (2005) fitted the exact Beta Poisson model to the dose-response data to find estimates of the parameter values α and β. Here, the parameter values have been used in the Beta Poisson approximation (Equation 5).

Experimental Data

In order to estimate the parameters of a dose-response model it is necessary to fit the dose-response relationship to some data. There are two primary sources of relevant data; they are from human feeding trials and unsolicited outbreaks.

Human Feeding Trials: Human feeding trials are controlled experiments where "volunteers" are administered doses of different pathogen concentrations. The number of volunteers who then exhibit an infective response are recorded. Important uncertainties associated with these studies include:

- The number of *viable* particles in the dose is unknown. Depending on the source of the inoculum and the individual pathogen, there is uncertainty as to how many of the administered particles were actually infectious at the time of consumption.
- *Strain* of the micro-organisms contained in the inoculum. Practicalities drive the sourcing of pathogens for the feeding trials. In some circumstances the strain of the administered organisms varied from the strain most likely to cause infection in humans, for example, most *Cryptosporidium* feeding trials have been undertaken using *Cryptosporidium parvum,* whereas most human infections are thought to be caused by various strains of *Cryptosporidium hominis*.
- Representativeness of volunteers. For ethical reasons human feeding trials are conducted on healthy adults who's immune response may not be representative of the entire population.

Outbreak data: In more recent years, information from outbreaks of enteric illness has been used to estimate dose-response parameters [Teunis *et al.*, 2005; Teunis *et al.*, 2004]. The great advantage of data from a real outbreak is that it demonstrates an actual response to exposure to human pathogens, without the constraints and simplifications necessary for a controlled study; pathogens are native to the system, and those exposed are a true sample from the susceptible population. Conversely however, additional uncertainties are introduced including:

- Estimating the dose. There is an incubation period between the time a pathogen is ingested and when a response (illness) is identified. Due to this incubation period, by the time an outbreak is identified, the source material is unlikely to be available for direct analysis. If it is available, the pathogen density may no longer be representative of the density at the time of exposure (due to inactivation or growth).
- Illness rather than infection is the endpoint. In a controlled feeding trial, blood serum can be analysed on a daily interval following exposure to identify whether or not an individual has been infected. For a real outbreak, identification of a response is limited to those who report *symptoms* of infection (illness) which is only a portion of the total infected population.

7.1.4. Step 4 – Risk Characterisation

The aim of risk characterisation is to integrate information from exposure and dose-response assessment to express public health outcomes. Dose-response models are concerned with estimating probability of infection. Infection has been defined as a situation in which the pathogen, after ingestion and surviving all host barriers,

actively grows at its target site [Last, 1995]. Infection may or may not result in illness, as asymptomatic infection can be common for some pathogens.

Incorporating events into overall probability of infection

Probability of infection estimates are based on the calculated exposure to pathogens. While the baseline (nominal) exposure can be calculated based on the expected variability in model inputs, it is often desirable to incorporate the likelihood and magnitude of certain events into the overall probability of infection estimate. One approach for undertaking this analysis is to calculate the probability of infection for each event condition that is to be investigated, and then to combine all events and nominal conditions based on their probability of occurrence (Equation 6).

$$P\inf = \sum_{i=1}^{n} Pevent_i \times P\inf i + (1 - \sum_{i=1}^{n} Pevent_i) \times P\inf_{no\min al}$$

Equation 6

Where: Pinf is the overall probability of infection
n is the total number of event conditions to be included
$Pevent_i$ is the probability of event *i* occurring
$Pinf_i$ is the probability of infection given that event *i* has occurred
$Pinf_{nominal}$ is the probability of infection under baseline or nominal conditions.

Predicting the number of infections from multiple exposures

When multiple exposures (either due to many individuals being exposed at the one time, one individual being exposed on multiple occasions, or a combination) are assumed to be independent events, then the number of infections (successes) may be described as a binomial random variable (X). The probability that the number of infections will equal a given number (k) is:

$$P(X = k) = \binom{n}{k} p^k (1-p)^{n-k}$$

Equation 7

Where: *k* is the number of infections
n is the number of trials (i.e. for the number of infections per year for an individual, n = 365; for the number of infections per year for a population of 10 000, n = 3 650 000)
p is the probability of infection

This distribution can be maximized to find the most likely number of infections based on the calculated P_{inf}.

Annual probability of one or more infections

If consecutive exposures are assumed to be independent, the annual probability of 1 or more infections may be calculated under the assumptions of a binomial process (a series of trials with one of two possible outcomes – infection or not infection). If the probability of infection for an individual exposure is given by P_{inf}, then the probability of not being infected is $(1-P_{inf})$. For *n* exposures, the probability of not being infected is given by $(1-P_{inf})^n$. The annual probability of one or more infections is the corollary of this for *n* = 365, and is given by Equation 8 :

$$P_{ann} = 1 - (1 - P_{\inf})^{365}$$

Equation 8

When $P_{inf} << 1$, this may be approximated as $P_{ann} = 365 \times P_{inf}$

Incorporating the impact of events into the annual probability of one or more infections

Equation 6 calculated the probability of infection given the likelihood of a range of possible event scenarios. It is also possible to consider the impact of events on the yearly probability of one or more infections when it is assumed that one (or more) events occurred during the year for a known duration (days). In this situation, the binomial assumption can be expanded:

$$P_{ann} = 1 - (1 - P_{\inf(no\,\min al)})^{t(no\,\min al)} \prod_{n=1}^{i} (1 - P_{\inf(n)})^{t(n)}$$

Equation 9

For example, consider a scenario when an event was known to occur in a given treatment plant for 2 days during the year. The probability of infection during that event was calculated to be 0.01. For the remainder of the year (363 days) the probability of infection was calculated to be 0.00001 (1×10^{-5}). The overall probability of one or more infections during that year was = $1- (1-0.00001)^{363} \times (1-0.01)^2 = 0.023$, if the event had not occurred the probability of one or more infections would have been = $1 - (1-0.00001)^{365} = 0.0036$.

Disease Outcomes

Infection is necessary to cause disease, however not all infections will result in symptoms of illness. While asymptomatic infections may be important for disease transmission, they do not in themselves contribute to the disease burden on a community. Evaluating the disease burden requires consideration of illness outcomes including the likelihood, severity and duration.

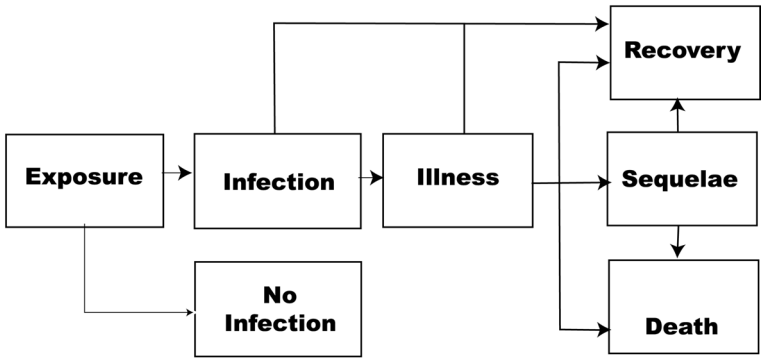


Figure 7.3 Outcomes of exposure to pathogens

Disability Adjusted Life Years (DALYs) is as a metric for translating the risk of disease burden a general health burden per case of illness, as discussed in Chapter 2. The DALY accounts for the years lived with a disability (YLD) plus the years of life lost (YLL) due to the hazard (compared to the average expected age of death in a community). One DALY per million people a year roughly equates to one cancer death per 100 000 in a 70 year lifetime (a benchmark often used in chemical risk assessments) [WHO, 2004]. The DALY is calculated as the product of the probability of each illness outcome with a severity factor and the duration (years). Calculation of the DALY contribution per infection is undertaken using Equation 10.

$$DALY = \sum_{i=1}^{n} P(ill \mid \inf) \times P(outcome_i \mid ill) \times Duration_i \times Severity_i$$

Equation 10

Where n is the total number of outcomes considered

$P\ (ill|inf)$ is the probability of illness given infection
$P\ (outcome|ill)$ is the probability of outcome i given illness
$Duration_i$ is the duration (years) of outcome i
$Severity_i$ is the severity weighting for outcome i

The advantage of using DALYs over an infection risk end point is that it not only reflects the effects of acute end-points (e.g. diarrhoeal illness) but also the likelihood and severity of more serious disease outcomes (e.g. Guillain-Barré syndrome associated with *Campylobacter*). Disease burden per case varies widely, but can be focused on a locality. 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. Disease burden estimates for different drinking water contaminants is summarised in Table 7.1.

Table 7.1 Summary of disease burden estimates for different drinking-water contaminants*

	Disease burden per 1000 cases		
	YLD	**YLL**	**DALY**
Cryptosporidium parvum	1.34	0.13	1.47
Campylobacter spp	3.2	1.4	4.6
STEC O157	13.8	40.9	54.7
Rotavirus			
High income countries	2.0	12	14
Low income countries	2.2	480	482
Hepatitis-A virus			
High income countries, 15-49yr	5	250	255
Low income countries	3	74	77

* Reproduced from Havelaar and Melse [2003]

While the use of DALYs has many conceptual advantages, research is necessary to facilitate its implementation. Estimates of incidence, severity and duration of disease outcomes based on epidemiologic data have only been presented in the literature for Rotavirus [Havelaar and Melse, 2003], *Campylobacter* [Havelaar *et al.*, 2000b], *E. coli* O157 [Havelaar *et al.*, 2003] and *Cryptosporidium* [Havelaar *et al*., 2000]. These inputs for DALY calculations are extremely uncertain and the variability in severity and duration between cases is still poorly understood. Havelaar *et al.* [2000b] however, argue that for *Campylobacter* spp that the uncertainty is relatively small and

that the DALY remains a robust measure even when input parameters are varied. Nevertheless, risk calculations undertaken as part of the MicroRisk project maintained probability of infection as the endpoint. The implementation of the DALY metric is demonstrated in an illustrative example only.

7.1.5 Example: Implementation of DALY metric for interpreting probability of infection estimates for *Cryptosporidium*.

Estimates of severity and duration of health outcomes following infection with *Cryptosporidium*, based primarily on the Global Burden of Disease (GBD) project, have been presented by Havelaar *et al.* [2000a] and reviewed by Havelaar and Melse [2003]. In developed countries 71% of immunocompetent individuals infected with *Cryptosporidium* develop gastroenteritis. The mean duration and severity weightings are summarised in Table 7.2.

Table 7.2 Summary of input assumptions for Cryptosporidium DALY calculations

Outcome	Probability of outcome given illness	Duration	Severity
Diarrhoea (mild)	1	7.2 (days)	0.067
Death	0.00001	13.2 (years)	1

Implementing
Equation 10 with data for *Cryptosporidium* from Table 7.2:

$$DALY = \underbrace{0.71 \times 1 \times 7.2/365 \times 0.067}_{Diarrhoea} + \underbrace{0.71 \times 10^{-5} \times 13.2 \times 1}_{Death} = 0.00103$$

Note: the probability of diarrhoea given illness is 1 since in the case of *Cryptosporidium* infection, all ill individuals are assumed to have diarrhoea.

The disease burden based on DALYs would therefore be calculated using the expected number of infections per year (maximising Equation 7) for the population multiplied by the DALY contribution per infection (0.00103).

7.2. TIERED APPROACH TO QMRA

QMRA can be undertaken at various levels of detail, from a deterministic analysis aiming to characterise, say, worst or best case risk scenarios, to a full scale stochastic analysis. More detail is not always advantageous, but rather the QMRA scope and the perceived risk level of the system should govern what an assessor considers an appropriate level of detail. Figure 4 illustrates an iterative approach for conducting QMRA that aims to:

i) Assess the health risks associated with a water supply system;
ii) compare the estimated risks to the health targets; and
iii) if necessary, identify points in the system whereby either more data is required to better characterise the risks, or, where management strategies could best be deployed to improve the overall system performance.

The level of detail required for each iteration cannot be prescriptive; instead it will depend on the exercise scope and the available data and resources. While any level of detail or method sophistication can be employed for the first or subsequent iterations, it is advisable to begin with a simple approach, *i.e.* to conduct a screening-level assessment, the simplest approach for QMRA is to describe each model input as a point estimate. As the aim of each iteration is to identify if further consideration of the microbial risk from the system of interest is necessary, any deterministic parameter estimates should be based on the best information available at the time, and be conservative. Should the infection risk estimate be well below some health target level, the management outcome may be interpreted as that the system is performing adequately, and that current practices are adequately safe. Alternatively, it may be that even following simple analyses while the infection risk is not below the target, that management options can be identified to reduce the risk to an acceptable level. In this circumstance, further data collection or analysis may also be considered unnecessary.

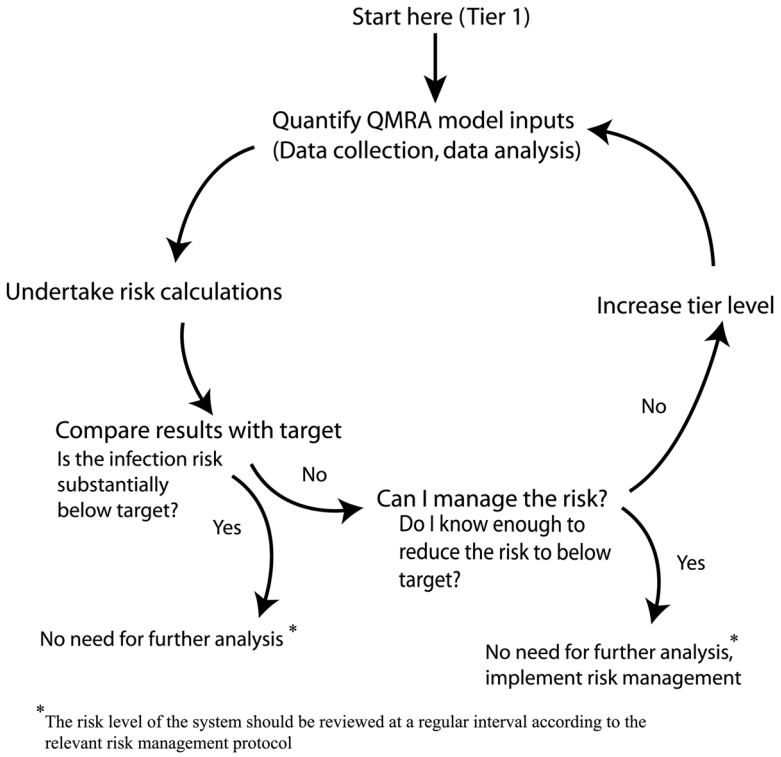


Figure 7.4 Iterative tiered approach for undertaking QMRA

In many situations however, an effective risk management approach is not clear unless further analysis is undertaken to characterise the variability in risk and the important determinants that drive such variability for the specific system. In this case, higher level analyses, whereby parameters are described not as point estimates, but as variable quantities may be useful.

7.2.1. (Higher level) probabilistic analyses

All inputs in a QMRA model are likely to vary. Source water quality varies with time dependent on catchment activity, seasonal climate changes, or specific point-source contamination such as a sewer overflow entering the waterway. Treatment efficacy varies depending on a host of factors (Chapter 4) such as plant design, treatment methods, and plant breakdown. Water consumption and susceptibility to pathogens varies between consumers. Understanding the impact of this variability on consumer risk is important, especially in management terms, as such understanding will aid answering why and how higher risk periods may occur, and provide insight into controlling those effects.

The central tool for describing variability is the Probability Density Function (PDF). When a model input is considered to be a variable rather than a constant, the input may be quantified using a PDF. When described by a PDF, the variable may take one of a range of values, each with a known probability of occurrence. The variable risk estimates may then be made using Monte Carlo simulation.

It is necessary to distinguish between the true PDF and an estimated PDF of an input variable. Each variable could be considered to have a true PDF; that is the actual frequency/duration of the range of values that the variable may take. This true PDF however, is unknown. For example, consider the *Campylobacter* density in a particular source water, which is known to be constantly changing. We may also know that it is higher at some times than others, perhaps due to season or hydrology. What we do not know is how high it can become, or for exactly how long it may be elevated.

At best, we can use available data along with some understanding of the system and formulate a PDF that is an *estimate* of the true PDF. Hopefully the estimate will encapsulate the key features of the true distribution and provide a realistic representation of the variable of interest. In order to construct an estimate of the true PDF we rely on experimental data and process or statistical models.

The manner in which a parameter is estimated and described depends on various factors, not least of all an understanding of the processes and mechanisms that may dictate the 'true' value of a parameter or the nature of its variability. The following sections (1.3 & 1.4) detail manners in which variable parameters (and the related concept of uncertainty) may be estimated using different types of relevant datasets.

7.3. QUANTIFYING VARIABILITY FROM MICROBIOLOGICAL DATASETS

Experimental data provides the most important insight into the quantitative value of each model variable. Numbers can be comforting, and tend to create an aura of certainty and accuracy; however numbers can be easily misinterpreted and inappropriately applied. Some elementary considerations when approaching a dataset for analysis include:

1. Is the dataset a random representative sample? It is important to consider if the available dataset is representative of the variable being quantified. For example: The aim may be to estimate the PDF for source water density of a particular pathogen. Were the samples taken randomly? If the dataset was collected as part of a short term study and all results were collected say during winter, or if the data was collected to investigate the impact of rainfall events and every sample

was collected following rain, then the results would not represent a random sample of the source water microbial density. Conversely, if samples were collected according to an arbitrary (or randomised) time factor, unrelated to water quality processes (e.g. the first Tuesday of every month) then the sample would be assumed to be random.

2. Is the dataset representative of my system? There are countless factors that vary between water supply systems that affect microbial risk, including: catchment land use, climate, hydrology, water chemistry and treatment process performance. Direct application of published literature data, or data provided by a colleague from an apparently similar system is appealing, however processes that affect pathogen risk are complex and one system cannot necessarily be directly applied elsewhere.

3. What was the method of detection and how accurate may I expect it to be? Microbiological detection methods are constantly improving, but may differ due to changing water (matrix) effects and technician performance.

4. What is the source of the numbers being used? Reported laboratory results may have already undergone transformation from their raw state due to averaging of replicate samples or translation into concentration estimates. Translation of raw laboratory results into reported densities can ignore underlying uncertainties arising from the detection and quantification process. It is extremely relevant to understand exactly what reported numbers represent.

7.3.1. Characteristics of microbial data

Microbiological datasets have many unique characteristics and represent a less usual case for statistical analysis. Understanding the source of microbial data, and where uncertainties may lie in their generation and analysis is important for QMRA.

Detection and Quantification

Microbiological species are small, and present in highly variable densities in environmental samples (ranging from <1 micro-organism per L to potentially $>10^8$ micro-organism per L (e.g. for sewage)). Technical assay procedures rely on a range of approaches for developing a quantitative estimate of the micro-organism density in a water sample. Understanding the basis of quantitative density estimates is important for interpreting the inherent uncertainties associated with reported results. Hence, it is important to provide a brief description of microbiological methods employed so as to give the necessary background to understand the statistical difference between different types of microbiological data.

There are three approaches for identifying the presence of an organism in the analysed sample including:

- **Visual identification:** The presence of the organism is identified using a microscope. For example, analysis of *Cryptosporidium* and *Giardia* typically involves filtering a water sample concentrate through a membrane, staining oo/cysts then counting them with the aid of a microscope. The particular strain and infectivity status are not resolved.

- **Culture:** The most common methods of pathogen detection and enumeration rely on culturing organisms (allowing organism to multiply under favourable conditions) in the laboratory. Viable organisms are quantified by the growth of colonies or diagnostic changes in liquid media (bacteria), or by measuring their effect on established host cell lines (viruses).
- **Molecular methods:** Molecular methods are designed to detect and analyse specific genetic material unique to the group being enumerated. The genetic material is present in the sample whether or not the pathogen is infectious, and therefore routine molecular methods cannot distinguish between infectious and non-infectious organisms. Polymerase Chain Reaction (PCR) involves the specific amplification of DNA from the genome of the organism with the aid of primers. PCR can be undertaken as a non-quantitative presence/absence test, or as a semi-quantitative analysis (real-time PCR).

A combination of methods may be implemented such as the culture/ enrichment of organisms prior to PCR identification. In this case, the organisms are cultured to increase their concentration prior to PCR, therefore improving the sensitivity of the PCR identification and largely detecting viable micro-organisms.

The results of these analytical processes are translated into a quantitative estimate of micro-organism densities using:

- **Count**: A directly quantitative approach where the number of micro-organisms (*Giardia* (cysts), *Cryptosporidium* (oocysts)), plaques (viruses), or colonies are counted (bacteria). The concentration is then estimated based on the volume of original sample analysed.
- **Most Probable Number**: Results from a series of presence/absence analysis are used to predict the most probable number of organisms in the original sample based on the assumption of a Poisson distribution.

Table 7.3. Summary of analytical approaches and datatypes obtained from MicroRisk CTSs

	Detection method:	**Quantitative: P/A or Count**	**Reported value:**
Campylobacter spp.	Culture or enrichment + PCR	P/A	$MPN.vol^{-1}$
E. coli O157	Culture or enrichment + PCR	P/A	Estimated concentration
Norovirus	PCR	P/A	Estimated concentration
Enteroviruses	Cell Culture	P/A or count	PFU or $TCID_{50}$. L^{-1}
Cryptosporidium spp.	Visual identification	count	$Oocysts.10L^{-1}$
Giardia spp.	Visual identification	count	$Cysts.10L^{-1}$
Indicators and Surrogate Organisms			
E. coli	Plate Culture	count	$CFU.100mL^{-1}$
	Culture	P/A	$MPN.100mL^{-1}$
Clostridium perfringens	Plate Culture	count	$CFU.100mL^{-1}$
	Culture	P/A	$MPN.100mL^{-1}$

Key sources of uncertainty associated with these methods include:

- **One micro-organism or a cluster?** In many of these methods, one organism or a cluster can initiate a positive reaction. For example, when counting colonies (bacteria) or plaques (viruses) either one or a cluster of cells/virions may have contributed to each colony or plaque. When a result is reported as PFU (plaque forming units) or CFU (colony forming units) in a sample volume, this is interpreted directly as the concentration estimate. In reality, the

PFU or CFU may in fact underestimate the number of cells originally present in the sample. Similarly, for a Presence/Absence MPN a positive result may have been caused by either one or a cluster of organism. The estimated MPN is based on the assumption of a Poisson distribution which only accounts for random distribution of cells in the sample rather than clustering (see Section 7.3.2). If micro-organisms are clustered, the MPN may also underestimate the original concentration.

- **Non-culturable but still infectious?** Under environmental conditions, micro-organisms may become stressed and as a result may be non-culturable in the laboratory. There is evidence however to suggest that such non-culturable organisms may still be infectious [McFeters, 1990; Barer and Harwood, 1999]. Though still controversial, the concept of cells being infectious but not culturable has been raised for a number of the bacterial pathogens [Federighi *et al.*, 1998].
- Lastly, visual identification of pathogens, such as oo/cysts of *Cryptosporidium* or *Giardia* is fraught with additional problems to those outline above. Primarily, standard methods estimate total oo/cysts, or at best the presence of potentially infectious structures within these oo/cysts [US-EPA, 1999; Smith *et al.*, 2004). Nonetheless, differences between strains, and indeed species can be missed, if not totally miss-identified by confounding microorganisms, such as algae [Rodgers *et al.*, 1995].

Recovery and Imperfect Detection

Analytical methods for identifying microbial species in water are imperfect. Imperfect methods are evidenced by the presence of a detection limit; a value below which organisms cannot be detected. For example, consider an assay known to have a detection limit of 5 pathogens.L^{-1}. If a sample containing 3 pathogens.L^{-1} is analysed, the result will be zero as the sample density is below the limit of detection. This detection limit may be caused by:

- **Method sensitivity -** The detection limit may exist because a critical mass is necessary to perform a successful analysis. Once the detection limit is exceeded the analytical result is a direct reflection of the original organism density. Consider a sample containing 6 pathogens.L^{-1} to be analysed by a method with a detection limit of 5 pathogens.L^{-1}. Under this explanation, the expected analytical result would be 6 pathogens.L^{-1}, as the density is greater than the detection limit. This explanation may be true of many chemical analysis methods, and may also be true of microbiological analysis that aims to identify the presence or absence of the target organism in a sample volume.
- **Inactivation or loss-** A portion of original microorganisms may be inactivated or lost during the assay process. Consider again the previous example of a sample containing 6 pathogens.L^{-1}, and a detection limit of 5 pathogens.L^{-1}. The result would be expected to be 2 pathogens.L^{-1} where four pathogens may be "lost" and two detected. The inactivation or loss of organisms throughout the analytical process potentially affects all methods.

These underlying mechanisms[4] are relevant for predicting original source water density from analytical results. Many results may be interpreted as though the cause

[4] A third notable interpretation of the detection limit relates to the sample volume. The lower limit of detectable concentration is also limited by the size of sample, for example, if the sample

of the detection limit was method sensitivity, i.e. any value above the detection limit is assumed representative. It may be more realistic to assume, particularly with microbiological species, that the detection limit exists due to inactivation or loss, and that those "lost" organisms should be accounted for over all reported values. This loss is described by the method recovery.

Techniques for assaying microbial constituents in water samples can involve many processes and steps each of which may lead to loss or inactivation of some micro-organisms. Recovery is the portion of micro-organisms "recovered" by a particular method. If the recovery was 100%, then there would be no loss, and the analytical result would be a direct refection of the original micro-organism density. Alternatively, if the recovery was say 40%, then the original density would be estimated at 60% higher than the analytical result. For example, if the analytical result predicted a *Cryptosporidium* density of 10 oocysts.L^{-1}, and the recovery of the method was 40%, then the original sample density would be estimated to be $10 \times 1/0.4 = 25$ oocysts.L^{-1}

Little has been reported regarding the recovery of *Campylobacter* and *E. coli* 0157, however recoveries of *Cryptosporidium* and *Giardia* may vary from <10% to >80% (US-EPA Method 1623) and viruses enumerated by plaque assays from ~10% to 90%. *Cryptosporidium* and *Giardia* have received the most attention in the literature regarding recovery experimentation. US-EPA methods 1622 and 1623 for enumerating oocysts and oo/cysts respectively from environmental samples have been shown to yield highly variable recoveries [Kuhn and Oshima, 2002]. Many studies have sought to quantify the dependency between sample characteristics and recovery, however the results have been inconclusive. While some studies have identified a drop in recovery at high turbidities (e.g. 159 NTU) [Kuhn and Oshima, 2002; Digiorgio *et al*., 2002] a continuous relationship is not easily defined and may not exist. Digiorgio *et al.* [2002] noted that the nature of the turbidity and the background water matrix is likely to be just as important as the absolute NTU. Consequently, there is currently no easily measurable native surrogate for estimating recovery of *Cryptosporidium* and *Giardia* in water samples; hence the recommendation of an internal control with each sample assayed.

Sampling Effects

Microbiological species consist of discrete entities or particles that cannot be assumed to be uniformly distributed throughout the water body. Rather, due to the random variation in the location of microbial particles, microbiological counts enumerated from a single well-mixed sample will rarely yield a series of identical numbers [Tillet and Lightfoot, 1995]. At low densities, the impact of sampling variability may be large.

For example, consider a volume of water containing an unknown density of *Cryptosporidium*. Suppose that the density of organisms in the volume of water is to be estimated by taking several 1L samples at random. The first sample contains 5, second 3, third 2 and finally 1. Each of these counts is an estimate of the actual mean pathogen density, illustrated in Figure 7.5.

volume was 200mL, and one organism was found the estimated concentration would be reported as 5 org.L^{-1}. Consequently, if no organisms were found, the concentration would be reported as < 5 org.L^{-1}. This interpretation does not directly relate to the recovery, but represents negative results.

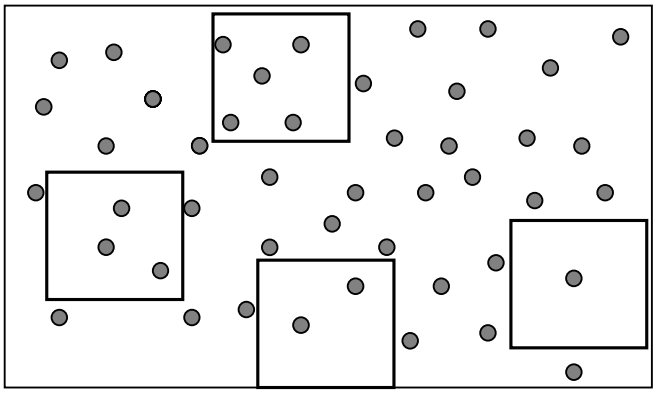


Figure 7.5. Random sampling of oocysts in a fixed sample volume

If the volume of water is considered well-mixed, the counts may be expected to follow a Poisson distribution [Haas *et al*., 1999]. An illustration of the Poisson count distribution with a mean (μ) of three is shown in Figure 7.6.

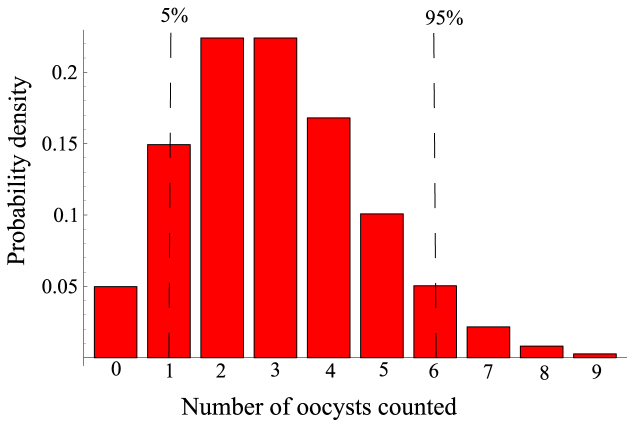


Figure 7.6 Poisson distribution (μ=3) highlighting 5th and 95th percentiles

In a well-mixed water body, with a mean *Cryptosporidium* density of three oocysts per litre, replicate counts would be expected to vary from 1 to 6, 90% of the time, with 10% of samples outside these limits. It is therefore possible that while the mean density is three, samples may be collected from which eight oocysts are enumerated.
This expected variability has implications for interpreting a pathogen's density in source water from analytical results. Suppose the number of organisms enumerated from a 1 L sample was three, what was the actual organism density in the water body at that time? A common assumption would be to consider the result a direct measure of the mean organism density, at 3.oocysts L^{-1}. However it is evident that due to sampling variability this count could have been enumerated from waters with a much higher or lower mean organism density.
Similarly, for analysis techniques that rely on identifying the presence or absence of a target organism in the sample volume, sampling variability leads to uncertainty in interpreting analytical results. While the target organism may not have been identified in a particular sample volume, it is possible due to sampling variability that the mean density in the original sample was greater than zero.

7.3.2. Model Fitting and Parameter Uncertainty

Statistical models can be used to enable variability and uncertainty associated with model inputs to be quantified from microbiological data. Models are idealisations of reality that facilitate a description of the true situation. No model presents reality, however certain models are more useful descriptors than others. The aim is to choose a model that facilitates the description of the target variable for the purposes of the QMRA.

The type of statistical model selected and implemented to describe a model variable will depend on the experimental data including the type of data available (e.g. continuous or discrete; raw data or reported densities); the size of the dataset (number of data points); and perhaps the appearance (the data may appear to have come from a particular type of underlying distribution).

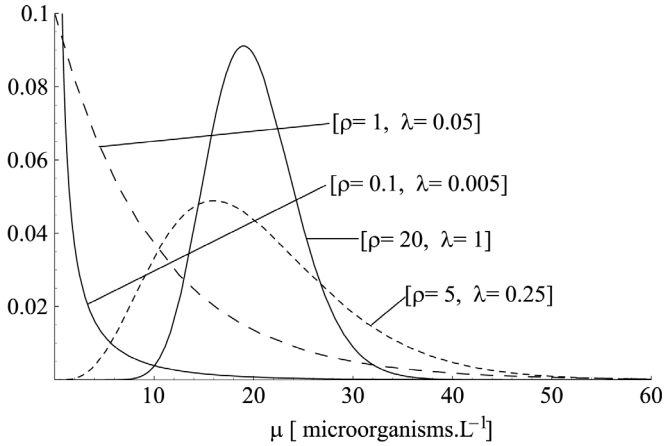
Model choice will also depend on how much is known about the process or system being studied. If the process is poorly understood, a simple empirical model that simply describes the dataset may be selected. Alternatively, if the underlying processes are well known, a model may account for the environmental, mechanistic or social processes that drive the value of the variable. Finally, the choice of model will depend on the aims of the individual risk investigation. A screening-level (tier 1) risk assessment, may intentionally select an overly simple approach.

Parametric distributions

Parametric distributions are important modelling tools for describing variability. A great number of distributions are available; however this section is limited to a description of the distributions applied as part of the MicroRisk project. In this context, the choice of distribution depended on the type of data (continuous or discrete), and the constraints (or domain) of the target variable.

Continuous data

A continuous variable can take on any value within a specified range and is not limited to discrete integer values. For continuous variables limited to positive values (such as pathogen density in source water, which cannot be negative), the Gamma distribution was applied for describing the probability density. The gamma distribution is a family of curves described by two parameters, shape (ρ) and scale (λ), of which the exponential and Chi-square distributions are special cases. The gamma distribution is particularly flexible for describing PDFs of different shapes (Figure 7.7). When ρ is large, the gamma distribution closely approximates the normal distribution however gamma only has density for positive numbers.



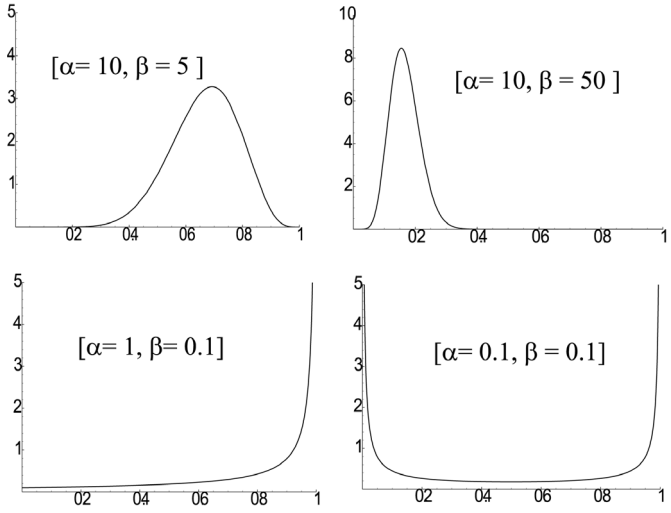
Gamma distribution:

$$g(\mu \mid \rho, \lambda) = \frac{\lambda^{\rho}}{\Gamma(\rho)} \mu^{\rho-1} e^{-\lambda\mu}$$

Where Γ represents the Euler gamma function

Figure 7.7. Shape of the gamma distribution for different combinations of shape and scale parameter values

For continuous variables limited to values between 0 and 1, the Beta distribution was applied for describing the probability density. The beta distribution is described by two parameters α and β and is extremely flexible for describing PDFs for binomial probabilities which will always lie between 0 and 1 (such as method recovery and probability of passage through a treatment barrier), some parameter combinations are illustrated in Figure 7.8.



Beta distribution:

$$g(\pi \mid \alpha, \beta) = \frac{(1-\pi)^{\beta-1} \pi^{\alpha-1}}{Beta(\alpha, \beta)}$$

Where $Beta(\alpha, \beta)$ represents the Euler beta function

Figure 7.8. Shape of the Beta distribution for combinations of parameter values

Discrete data

Microbial datasets frequently consist of discrete counts of micro-organisms, colonies or plaques in a certain sample volume. At low microbial densities, sampling variability should be incorporated into the statistical model structure using discrete distributions. Apart from satisfying statistical correctness, there are two practical advantages associated with accounting for sampling variability:

1. Provides greater flexibility in describing the target variable. If a particular analytical result could have eventuated from a range of source water densities, then to only consider the most likely density limits the flexibility of

the statistical model to predict the most likely parameter values for the PDF; and

2. Handling zero counts. Under the assumption of a discrete counting distribution, zeros are a result with a known probability of occurrence and can therefore be directly included within the model. There is no need to substitute zero values with a less than, or a detection limit, this approach describes what actually happened.

Two types of discrete distributions are presented here for describing microbial counts: the Poisson and the negative binomial distributions.

When particles are assumed to be randomly distributed in the water body, then a series of counts enumerated from water samples may be described by a Poisson distribution. The Poisson distribution assumes that the mean density of particles is a constant value. In reality, the mean density of micro-organisms in a water body may be expected to vary both spatially and temporally. This variability in mean density implies that micro-organisms are overdispersed, rather than randomly dispersed, in the waterbody (Figure 7.9). When that variability is described by a gamma distribution, the result is a Poisson-gamma mixture model, which is a form of the negative binomial distribution (BOX 7.1). The negative binomial distribution has been widely used to describe microbial count data [Haas *et al.*, 1999; Teunis *et al.*, 1999a; Teunis *et al.*, 1999b; DeVires and Hamilton, 1999; Pipes *et al.*, 1977].

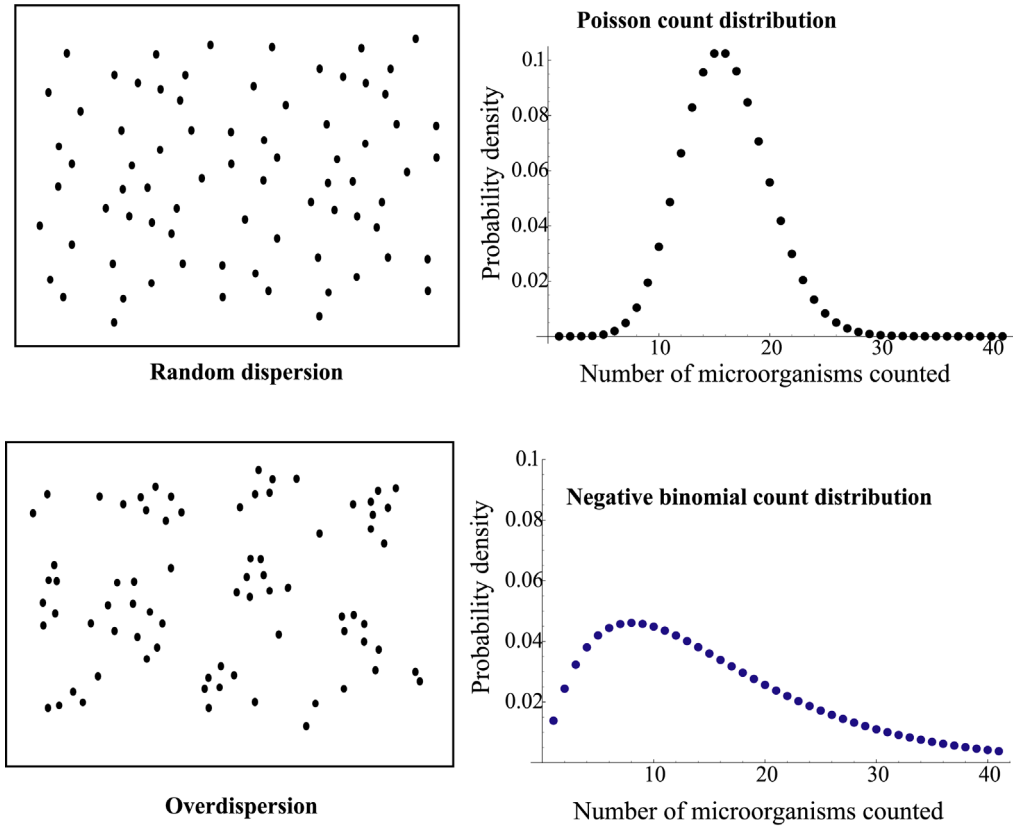


Figure 7.9. Illustration of the expected distribution of counts resulting from random and over-dispersion of micro-organisms.

BOX 7.1 - Parameterisation of the Poisson-gamma mixture model

While a range of equivalent parameterisations are available, the following description is reproduced from the work of Teunis *et al.* [1999a, b].

When counts are assumed to be generated from a Poisson (random) process, then the probability of counting *n* organisms given a mean concentration (μ) and sample volume (V) is given by:

$$P(n|V) = \frac{(\mu.V)^n e^{-(\mu.V)}}{n!}$$

Equation 11

If that mean concentration (μ) is assumed to follow a gamma distribution, then the distribution of counts (n) is given by:

$$g(n|\lambda,\rho,V) = \int \frac{(\mu.V)^n e^{-(\mu.V)}}{n!} \frac{\lambda^\rho}{\Gamma(\rho)} \mu^{\rho-1} e^{-\lambda\mu} d\mu$$

Equation 12

The solution to the integral can be rearranged into the form of the negative binomial count distribution described by gamma parameters ρ and λ:

$$g(n|\lambda,\rho,V) = \frac{\Gamma(\rho+n)}{n!\Gamma(\rho)} \frac{\lambda^\rho V^n}{(\lambda+V)^{\rho+n}}$$

Equation 13

This function can be used to construct a likelihood function based on measured counts. The maximum likelihood estimators for the gamma distribution parameters describe the variability in mean concentration μ.

Parameter estimation and uncertainty

Models used to predict and describe process variables are defined by *parameters*. Once a model has been selected, and it is hypothesised to be a useful representation of the underlying variable or system, appropriate values for the model parameters need to be estimated.

Given the experimental data (observations), the aim is to infer the parameter values of the selected distribution describing them. Several combinations of parameter values may be possible, and could have led to the observations, however the objective is to find the *most likely* parameter values, along with the probable region within which the parameter values may be expected to lie. The size of this region is reduced as the number of observations is increased. This uncertainty is referred to as *parameter uncertainty* and can be significant for small datasets.

Method of Maximum Likelihood

The concept of likelihood has been widely applied in the development of statistical models, and refers to the probability that the experimental data was generated from the assumed model [Edwards, 1992]. Construction of the likelihood function

facilitates the inference of parameters values and evaluation of their uncertainty. Values of the model parameters that maximise the value of the likelihood function are termed the Maximum Likelihood Estimators (MLE), and are deemed the parameter values that are most consistent with the observations (data). It is also possible to construct a confidence region for a parameter vector based on the likelihood function[5]. For a full explanation of constructing likelihood functions and the method of maximum likelihood see a standard text, such as Montgomery and Runger [1999].

Bayesian Inference and MCMC

For complex models containing large numbers of parameters, numerical optimization of the likelihood function can be laborious. Simulation techniques using Markov Chain Monte Carlo (MCMC) analysis, are available that allow the characteristics of the likelihood function to be explored within a Bayesian framework[6]

MCMC methods are well established and have been used for parameter estimation and uncertainty analysis in a range of modelling applications [Gilks *et al.*, 1996; Gelman *et al.*, 2004], particularly hydrology [Campbell *et al.*, 1999; Bates and Campbell, 2001]. The approach is well suited to risk assessment for evaluating uncertainty associated with models fitted to small datasets [Teunis *et al*., 1997; Teunis *et al*., 1999]. In the examples presented in this chapter, MCMC has been applied to quantify the uncertainty associated with parameter estimates. For a detailed explanation of the MCMC techniques and applications see Gilks *et al.* [1996] and Gelman *et al.* [2004].

For MicroRisk, models were constructed in Mathematica® software package (Wolfram Research, Inc.) and a Markov Chain Monte Carlo approach using the Metropolis-Hastings algorithm was used to obtain a sample of the posterior distribution for model parameters. These samples were used to construct credible intervals for the PDF (see Figure 7.10). The posterior sample of parameter values was used to construct a sample of PDFs, one PDF representing each sample of the parameter vector (gray lines, Figure 7.10). For each value of the given variable (x-axis), the lower 2.5% and upper 97.5% quantiles of the sample of PDFs were selected. These quantiles were joined, resulting in a 95% credible region for the PDF

[5] Confidence region for parameter vector $\theta = (\theta_1, \theta_2, ...\theta_k)$ consists of all parameter vector values that do not lead to rejection of the hypothesis $H_0 : \theta = \theta_0$. Leading to a 100(1-α)% confidence region: $\left\{ \theta, 2Ln\left(\frac{L(\hat{\theta})}{L(\theta)} \right) < \chi^2_{[v;1-\alpha]} \right\}$

[6] Statistical methods have great difficulty in determining uncertainty distributions for two or more parameters from the same data set when these distributions are correlated. Classical statistical methods either assume that the uncertainty distributions are Normally distributed, and then use a covariance matrix to create the correlation, or use resampling methods (bootstrapping). MCMC is a technique to obtain a required Bayesian posterior distribution and is particularly useful for multi-parameter models where it is difficult to algebraically define, normalise and draw from a posterior distribution. The method is based on Markov chain simulation: a technique that creates a Markov process (a type of random walk) whose stationary distribution (the distribution of the values it will take after a very large number of steps) is the required posterior distribution. The technique requires that one runs the Markov chain a sufficiently large number of steps to be close to the stationary distribution, and then record the generated values [Vose, 2004].

of interest. The credible region is a representation of the parameter uncertainty, and represents the region within which the PDF is expected to lie – with 95% confidence.

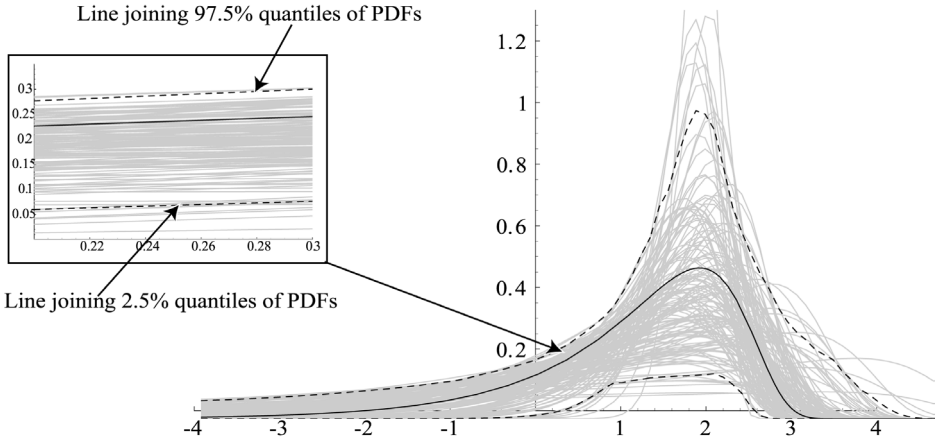


Figure 7.10 Illustration of method for constructing 95% credible interval (dashed line) from posterior sample of parameter pairs (PDFs constructed from posterior sample shown in grey)

7.3.3. Triangular Distributions

For some of the MicroRisk systems examined representative pathogen data was completely lacking and attempting to predict the shape of the PDF for certain variables was considered to be inappropriate. Other forms of information were however often accessible including related literature data, MicroRisk data from other similar systems and expert opinion. It was desirable to be able to quantitatively describe this prior knowledge or expert opinion in a simple way for the purposes of a low tier assessment. While many formal approaches for incorporating prior knowledge into risk calculations are available in the statistical literature – the complexity of implementation did not fit well with the objective of undertaking a simple analysis. In these situations the triangular distribution was considered to be a useful representation of the region within which the variable may be expected to lie. The triangular distribution is defined by a minimum, most likely and maximum value, these limits of the distribution could be estimated based on general information. While the true distribution of environmental variables may never be expected to be triangularly distributed, it was considered to be useful representation of existing knowledge surrounding the value of variables at a low tier level.

7.4. QUANTIFYING UNCERTAINTY

Once a PDF has been constructed to estimate the target variable – it is relevant to ask, how good is the estimate? How confident may I be that the estimate is a realistic representation of reality? There are many sources of uncertainty associated with predicting PDFs from experimental data. The key to accounting for uncertainty is to be precise about the uncertainty: that is, to be precise about the source of uncertainty and quantify it accordingly.

Two approaches are presented here for quantifying uncertainty. The first is to use statistical methods to account for and quantify uncertainty based on experimental data and model selection. These methods answer the question of: given my selected model, and experimental data, how much could the predictions deviate from the best estimate. These methods are presented in Section 7.3.2.
But what if the selected model is not only wrong (as all models are) but is a major misrepresentation of the system? or, What if the dataset is not-representative (perhaps all source water samples were collected under low flow conditions missing potential event driven spikes)? The second approach to quantifying uncertainty relies on expert opinion to explore the impact of these possible underlying errors or inadequacies on the overall risk estimates. Sensitivity analysis is used to investigate the sensitivity of the risk model to such underlying assumptions.

7.4.1. Expert opinion and worst case sensitivity

Limited datasets available for QMRA rarely tell the whole story. Expert opinion has an important role to play in interpreting environmental and risk implications of the data available. In particular, incorporating known sources of uncertainty, even when they cannot be easily quantified is desirable. Two such sources include:

Uncertainty regarding the representativeness of experimental data: The risk analyst may consider a small dataset of pathogen counts from their water supply system. Due to some prior experience or knowledge (for example the range of pathogen densities expected given catchment sources), the analyst may question whether the dataset is in fact representative of the system. Incorporation of this uncertainty or scepticism is relevant for understanding the risk. Ignoring all prior knowledge from literature studies, other datasets or epidemiologic experience in favour of local data alone is irrational, particularly if the local dataset was extremely small, or from an unknown source. The data itself is subject to many uncertainties including influences from random sampling, and method recovery. It is therefore desirable to be able to use small local datasets for estimating the PDF, but then also to test the importance of any perceived inadequacies.

Uncertainty regarding model selection: The basis of some models may be relatively poorly understood, containing necessary but questionable simplifications. While the risk analyst may believe that the selected model is the most appropriate choice given the available data and understanding of the system, they may also be interested to test the importance of this model choice on the calculated probability of infection. The selection of a second possible model may lead to much higher probability of infection, highlighting the need to consider carefully which model is chosen, and perhaps the need for further data collection to understand which model is likely to be more representative.
In order to provide a quantitative framework for the consideration of these uncertainties, a pragmatic approach, using a sensitivity analysis calculation was proposed.
In a model that contains a series of steps, sensitivity analysis may be used to identify which components or variables within the model are most important to the outcome. Sensitivity analysis allows for the effect of changing assumptions to be assessed and is a valuable tool for determining the critical drivers of microbial risk within the system. Using sensitivity analysis tools, uncertainties can be evaluated for the

purpose of prioritising data collection and research. Methods for undertaking sensitivity analysis have been reviewed by Frey and Patil [2002]. In that article, sensitivity analysis methods were categorized into three groups: mathematical, statistical or graphical. The method adopted to evaluate the sensitivity of the model to uncertainty in variable estimation was the worst case sensitivity. This method was presented by Zwietering and van Gerwen [2000] in the context of food safety and risk assessment.

Worst case sensitivity: The importance of uncertainty in each model component may be evaluated by calculating the factor sensitivity at each step. The factor sensitivity compares the impact of worst case, or extreme assumptions relative to the average.

$$FS_K = Log\left(\frac{N_k(extreme)}{N_k(average)}\right)$$

Equation 14

The risk model was initially constructed and simulated using best-estimates of all model variables. The results from this analysis were used to find the dose under "average" conditions (N_k (*average*)). A worst-case value was then selected for each of the model variables. Keeping all other variables at their average or "best" estimates, the model was simulated to find the dose under "extreme" conditions (N_k (*extreme*)), with reference to each individual variable. The resulting factor sensitivity for each step indicates the relative importance of uncertainties associated with each model variable. Given that the dose under average and extreme conditions is described by a PDF, the FS is also represented by a PDF, and was calculated for both the average and the 95th percentile of the PDF.

Selection of worst case value: For each model variable, a "worst case" value was selected, the basis of which depended on the particular variable in question and the perceived uncertainty associated with the estimation of that variable. Sources included:

1. **Parameter uncertainty:** There is uncertainty associated with the parameter values fitted to local datasets. For small datasets, this uncertainty can be significant. The worst case value was selected as the conservative (i.e. for source water concentration the upper, for treatment performance the lower) 95% credible limit of the estimated PDF.
2. **Data from another system or literature** – Perhaps the *Cryptosporidium* density in source waters was estimated from a small experimental dataset to be 0.001 oocysts. L^{-1}. However results from another similar catchment indicate densities closer to 2 oocysts.L^{-1}. This higher density of 2 could be adopted as a worst case value, to test the sensitivity of the risk model to the uncertainty in source water *Cryptosporidium* density. If found to be important, further investigation of the source waters may then be justified.
3. **Event impact** – Particularly for treatment performance, the impact of loss of a treatment barrier in the process was estimated by assuming that removal performance was zero as an extreme value.

The aim of the framework is to allow the sensitivity of any assumed variable value to be tested. Whether selected arbitrarily or as a set percentile from parameter

uncertainty, the influence of any hypothesis or assumption can be tested in a structure way allowing for uncertainties that are important to the risk outcomes to be identified and prioritised.

7.4.2. Auditing Score

Each model input, for each system studied was quantified with a varying degree of precision and complexity. This variation depended on the quantity and quality of data available, the importance of the model input to the overall quantitative risk outcomes and the tier level of the investigation (Figure 7.4). The level of knowledge and uncertainty associated with quantifying each model input needs to be weighed in the light of the QMRA outcomes. For example, consider the case where a system's source water pathogen content was estimated from measured pathogens in the specific source water, while removal by sedimentation was based on observed removals in particle count data at the treatment plant, and removal by filtration was estimated based on removals of an indicator organism at a similar plant elsewhere. A sensitivity analysis result might imply that the source water content of pathogens most heavily dictates the risks, and so should be the first point for a water manager to begin implementing management resources. However, there is greater unquantified uncertainty regarding how representative the estimates of removal by sedimentation, and removal by filtration were compared to the true values for the system of interest. A framework for testing the importance of these uncertainties on the risk outcomes has been presented (see section 7.4.1), however there is also a need to *evaluate* and *document* the level of detail and confidence associated with each model input, alongside the QMRA calculations. The need to consider and communicate such information suitability uncertainties for QMRAs has been raised before (*e.g.* Fewtrell, *et al*. [2000]).
To facilitate this auditing process in the MicroRisk project, each variable in the QMRA model was given an audit score as described in (Appendix 2, Chapter 8). When determining data needs for future iterations of the QMRA models, consideration needs to be given both to the quantified assessment results as well as the data quality audit scores.

7.5. IMPLEMENTATION FOR CTS'S

Ideally, each variable should be described by a PDF, with the resulting variability in exposure characterized by Monte Carlo simulation. Variability and uncertainty are inevitably woven together, however at least conceptually, they were separated for this risk analysis. PDFs were limited to the description of variability. Second order analysis (where PDFs are also used to describe uncertainty) was not undertaken. This was a deliberate choice, given the magnitude of variability, and the limited datasets available (leading to high uncertainty). An exposure characterisation that incorporates all variability and uncertainty was considered to be so broad as to limit its practical application.
As an alternative, PDFs for model variables were estimated based on relevant data and parameter uncertainty associated with those PDFs was predicted. The importance of uncertainty due to model assumptions and adequacy of experimental

data was evaluated within a more pragmatic framework. The sensitivity of the risk model to uncertainty associated with each variable was estimated by choosing a worst case value (i.e. how high/low could an expert realistically expect this variable to be) and calculating the Factor Sensitivity.
The resulting risk characterisation, therefore reflects variability in model inputs, described using the best available data. Factor sensitivity results identify the most important sources of uncertainty in terms of risk outcomes, therefore identifying where additional information is required to improve risk predictions.
The following sections outline specific techniques applied for estimating PDFs from microbiological data for source water concentration, recovery and treatment performance; distribution and consumption are not included in this section since results from Chapters 5 and 6 were directly applied for the risk calculations in Chapter 8. Published dose-response parameter values are included along with some discussion of the influence of model choice on the estimated probability of infection estimates.

7.5.1. Source water pathogen densities

Pathogens may be present in the source water due to human or animal inputs (waterborne enteric viruses being assumed to only come from human excreta). The density of pathogens at the treatment plant off-take is dependent upon the magnitude of pathogen inputs and the environmental processes affecting the transport and inactivation; and is expected to vary both over time and between pathogen groups.
Literature and Chapter 3 data was used to describe pathogen densities at the off-take for each studied system. With few exceptions, the Gamma distribution was selected for describing variability in source water pathogen density due to its flexibility. The modelling approach adopted for fitting a PDF for source water density depended upon the type of experimental data provided.

Microbial Counts

Consider *Giardia* counts from the raw water source for CTS 7 (shown in Table 7.4). Direct conversion of these counts to concentrations (i.e. number cysts counted/Volume = cysts.L^{-1}) leads to a mean cyst concentration of 0.117 cysts. L^{-1} with a maximum concentration of 0.97 cysts L^{-1}. Describing these counts directly as densities ignores the influence of sampling variability (the mean bulk water density at the time the sample was taken is assumed to be exactly equal to No. cysts/Volume), and necessitates the substitution of zero counts with some positive value. To obtain a more realistic picture of the source water concentration, these data should be analysed as a discrete dataset using counting statistics (relying on each raw count in the measured sample volume) rather than a continuous distribution.
Assuming that these discrete counts reflect random samples (Poisson process) from the source water with mean *Giardia* concentration (μ), and that the mean concentration varies according to a gamma distribution leads to a negative binomial count distribution (Equation 13). When the negative binomial distribution was fitted to the counts and volumes (Equation 3), the maximum likelihood gamma distribution describing *Giardia* cyst concentration in source water for CTS 7 was found and is illustrated in Figure 7.11.

Table 7.4. *Giardia* cysts counts from CTS7 source water

Count	Volume (L)
8	16.25
9	9.25
8	65
7	67.5
9	92.5
1	110
3	130.75
4	134
5	105
2	76.25
0	137.5
3	125
2	125
2	125
0	125
0	125
1	112.25

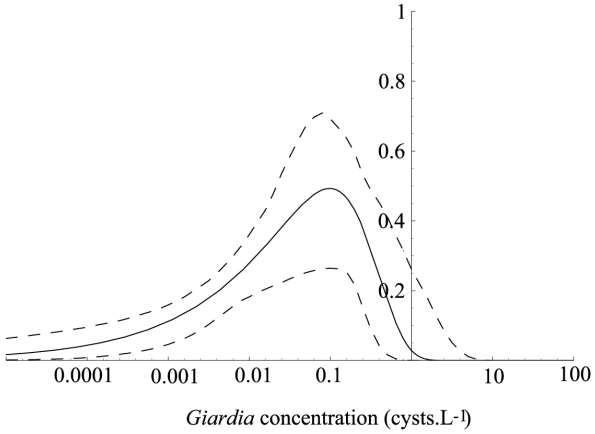


Figure 7.11. PDF for the *Giardia* cyst density in the source water for CTS 7 – Maximum likelihood Gamma distribution $\rho = 0.41$ and $\lambda = 0.24$ (solid line) and 95% Bayesian credible intervals (dashed lines) constructed from posterior MCMC samples.

Parameter uncertainty was explored by constructing a sample of the Bayesian posterior distribution of ρ and λ using MCMC simulation with uninformative priors. The variable *Giardia* cyst concentration in source water for CTS7 was included in the risk model as a gamma distribution defined by maximum likelihood values of $\rho = 0.41$ and $\lambda = 0.24$. While the upper 95% of the best fit PDF was 1.12 cysts.L^{-1}, the uncertainty analysis indicates that given the data, the mean *Giardia* cyst concentration could reach concentrations as high as 10 cysts.L^{-1} (upper credible interval).

Incorporation of Events

For some source waters, elevated concentrations of pathogens may be directly linked to events that mobilise pathogens in the catchment such as rainfall induced runoff, or sewage discharges. In these situations, rather than fitting one distribution to all data points, it may be more representative to describe the source water pathogen concentration separately for event and nominal (baseline) conditions.

For the *Giardia* dataset from CTS 7, elevated pathogen concentrations were hypothesised to be associated with periods following a rapid rise in water level in the source river. To investigate this hypothesis, when the samples were collected, the operator identified whether the conditions were classified as “event” or “nominal”. The same dataset from Table 7.4 is categorised as event and nominal in Table 7.5.

Table 7.5. *Giardia* cyst counts from CTS7 source water. Samples classified as "event affected" and "nominal"

Event affected	
Count	Analysed Volume (L)
8	16.25
9	9.25
8	65
7	67.5
9	92.5
1	110
3	130.75
4	134
5	105
2	76.25
Nominal	
Count	Analysed Volume (L)
0	137.5
3	125
2	125
2	125
0	125
0	125
1	122.25

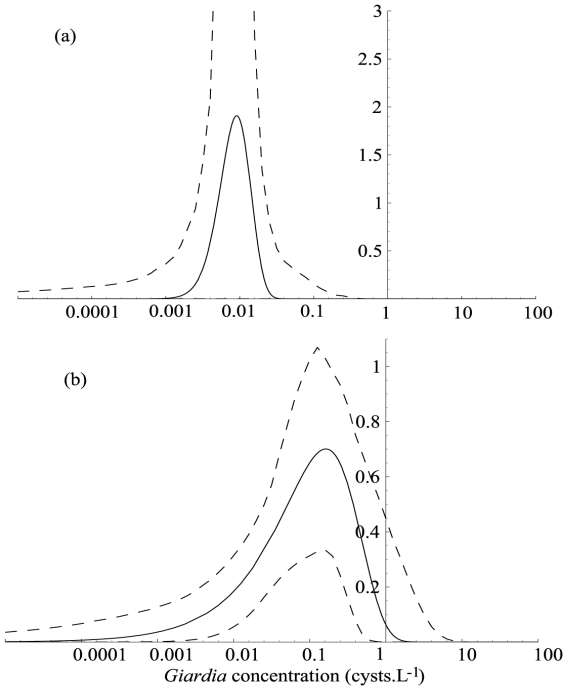


Figure 7.12 PDF for variability in the mean *Giardia* density (μ) under nominal (a) [$\hat{\lambda}$ =0.002 , $\hat{\rho}$ =4.48] and event (b) [$\hat{\lambda}$ = 0.22, $\hat{\rho}$ = 0.72] conditions. Maximum likelihood gamma distribution (solid line), with 95% credible intervals from MCMC analysis (dashed lines).

The aim of the analysis was firstly to determine if there was a significant difference between the *Giardia* density under event and nominal conditions; and secondly if appropriate, to estimate the PDF for each condition. The maximum likelihood estimates of the parameter values and deviance were calculated for each separate condition and the pooled (combined) dataset. A comparison of the deviance indicated that there was a significant improvement in fit achieved by separating the datasets (Combined (101.23) - (Nominal (20.33) + Event (68.34)) = 12.56 > 5.991, Chi squared distribution at 95% level with 2 degrees of freedom), and describing the PDF for nominal and event conditions separately. The PDFs for nominal and event conditions are illustrated in Figure 7.12 with their credible intervals.

The expected value of the *Giardia* density under event conditions was 0.16 cysts.L^{-1} in comparison to 0.009 cysts.L^{-1} under nominal conditions. *Giardia* density was clearly higher during events.

The separation of data for describing event and nominal conditions affected the spread of the distribution, and hence the assumed variability associated with the mean density. Firstly, when counts measured under event conditions were removed, the

baseline or nominal dataset showed very little variability and was equally well fit by the Poisson distribution (Deviance = 0.11 < Chi-Square at 95% level with 1 degree of freedom = 3.841), indicating no evidence in the nominal data for variability in the mean density (μ). Under nominal conditions, the source water could be assumed constant at 0.009 cysts.L^{-1} (The upper 95% quantile of the MCMC posterior sample of the Poisson parameter μ was 0.016 cyst.L^{-1}).

Secondly, predictions of upper concentration values were reduced by considering event conditions separately. Comparison of the upper 95 quantile of the distribution for event conditions (0.69 cysts.L^{-1}) with the upper 95 quantile of the pooled dataset (Table 7.4 and Figure 7.11) (1.12 cysts.L^{-1}) demonstrates that the assumed peaks in concentration are in this case reduced by considering events separately. Similarly, the upper credible interval of parameter uncertainty was lower for the event only dataset (1.57 cysts.L^{-1}) in comparison to the pooled dataset, where the upper credible interval for the upper 95 quantile approached 10 cysts.L^{-1}.

Exposure to pathogens could be calculated in the risk model for event and nominal conditions separately, in which case the relative importance of each condition could be examined during risk characterisation. Alternatively, the two distributions could be combined to form one single PDF for source water *Giardia* concentration.

Combining the two PDFs for a single model input requires a representation of the proportion of time that the water quality is represented by each condition. If the dataset itself was representative, the river would be under event conditions 10/17= 58.8% of the time. More accurate data relating to the cause of events would be expected to exist, that would provide a better estimate of this parameter [Signor *et al.*, 2005 application to CTS 8]. The overall PDF for source water density would be given by Equation 15:

$$\mu_{Sourcewater} = A \times \mu_{event} + (1 - A) \times \mu_{no\,\min al}$$

Equation 15

Where:

A is the proportion of time that source water is under event conditions

μ_{event} is the *Giardia* concentration under event conditions

$\mu_{nominal}$ is the *Giardia* concentration under nominal conditions

Given the analysis of the *Giardia* count data, the best estimate for implementing Equation 15 and describing source water density of *Giardia* at CTS 7 is: $\mu_{Sourcewater} = 0.588 \times GammaDistribution[\rho = 0.72, \lambda = 0.22] + 0.422 \times 0.009$.

Presence/Absence results

For many pathogens, the number of pathogens present cannot be directly identified and analytical methods are limited to identifying the presence or absence of the target organism in a sample volume. A presence/absence approach can however be used quantitatively when several replicate samples at different dilutions are analysed in parallel.

One such organism that is analysed for presence or absence is *E. coli* O157. Sampling and analytical procedure for quantifying *E. coli* O157 concentration in source water for CTS 10 included 15 samples, each of which was sub-sampled at three ten-fold dilutions. The presence or absence of *E. coli* O157 was identified in each sub-sample. The results, along with reported density estimates are included in Table 7.6.

Table 7.6. Presence/Absence results for *E. coli* O157 from CTS 10 source water samples

Volume (L)			*Estimate org.L^{-1}
0.01	0.1	1	
0	0	0	<1
0	0	0	<1
0	0	1	1-10
0	0	0	<1
0	0	0	<1
0	0	0	<1
0	0	0	<1
0	0	0	<1
0	0	1	1-10
1	1	1	>100
0	1	1	10-100
1	1	1	>100
1	1	1	>100
0	0	1	1-10
0	0	0	<1

*Without taking into account recovery of the method

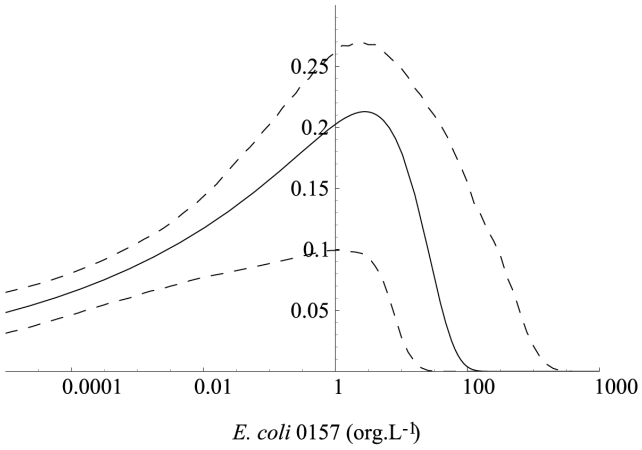


Figure 7.13 PDF for *E. coli* O157 density based on presence/absence data from CTS10. Maximum posterior gamma distribution (solid line) with 95% credible intervals (dashed line) from MCMC analysis.

The estimated concentrations give some idea of the expected range of how many *E. coli* O157 may have been present in the source water; however quantifying the PDF of *E. coli* O157 concentration for input into the risk model is more complicated. A statistical approach is required that allows the shape of the PDF to be estimated (including a realistic representation of the parameter uncertainty) based on the presence/absence results.

In order to undertake this analysis, some relatively simple assumptions regarding the underlying processes influencing the pathogen density were made:

1. The three sub-samples for each sampling day were assumed to be random samples (Poisson process,) from the source water with mean *E. coli* O157 concentration μ.
2. The mean density (μ) was assumed to vary between sampling occasions according to a gamma distribution.

The model is no-longer a straight forward Poisson-gamma mixed model but rather a special case where gamma dispersion is only assumed between sampling days; on any individual sampling day, the dispersion between sub-samples is assumed random (Poisson). Implementing these assumptions by constructing a likelihood function is mathematically complex, however when the model is constructed within a Bayesian hierarchical framework, the calculations are simplified.

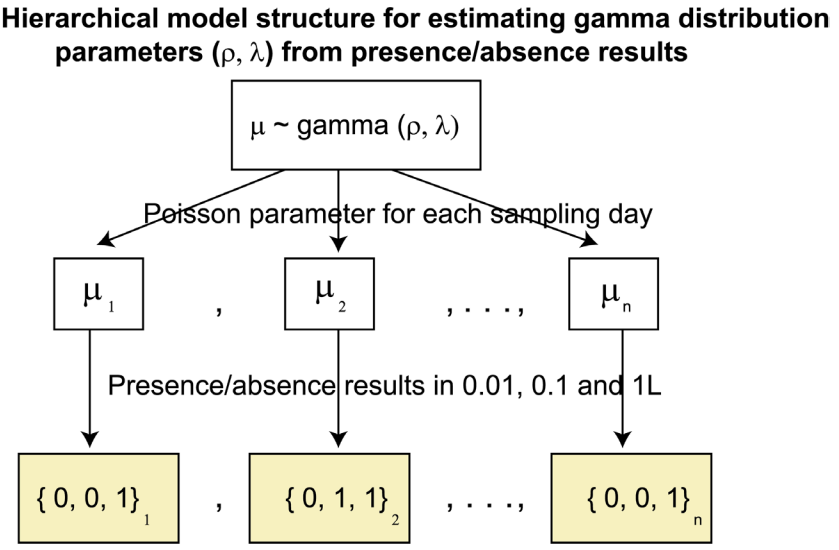


Figure 7.14 Structure of the hierarchical model for estimating gamma distribution parameters from Presence/Absence results

The posterior distribution of ρ and λ can be investigated by simulation using MCMC. The maximum posterior gamma distribution with 95% credible intervals for the PDF of *E. coli* O157 density illustrated in Figure 7.13. Sampling days that consisted of all negative results led to a lack of convergence in the posterior samples of ρ and λ. To avoid this problem, sampling days where all results were negative, were modelled as half the detection limit, rather than zero. This was achieved by substituting a positive result for the 1L sample volume, and estimating μ as half the predicted value for that day. The expected value of the *E. coli* O157 density was 2.78 org.L^{-1} with an upper 95% quantile of variability of 15.73 org.L^{-1}. The upper 95% quantile of parameter uncertainty was 134.8 org.L^{-1}. The clear benefit of this approach is that the shape of the PDF, along with associated uncertainty can be estimated directly from the presence/absence results.

Index Organisms

Index organisms are microbial species that are present in water samples at a known ratio to one or more human pathogens [Ashbolt *et al.*, 2001]. Not only do index organisms indicate the presence of human pathogens, but they can be used to quantitatively estimate the concentration of a particular pathogen using the ratio between their densities. In order to be useful as an index organism, a microbial species should be from the same source as the human pathogen, and respond similarly to environmental conditions.

Given the complexity and hence expense associated with analysing directly for pathogens in source water samples, the use of index organisms for quantifying source water pathogen concentration is desirable.

Within the MicroRisk project, *E. coli* and thermotolerant coliforms (Coli 44C) have been used as an index for quantifying pathogen densities in surface waters and sewage from reported concentrations (Chapter 5). These estimated pathogen to *E. coli* or thermotolerant coliform ratios were used to predict possible pathogen densities in distribution systems based on *E. coli* measurements. The data used to estimate these ratios are summarised in Figures 7.15 & 7.16 for surface waters and sewage respectively. The illustrated PDFs were constructed by fitting a gamma distribution

separately to the sample of pathogen and index densities, and then calculating the PDF for the ratio between the two gamma distributions. The estimated ratios based on paired index and pathogen reported densities are also shown for each figure.

The illustrations demonstrate the variability in the estimated ratios based on reported concentrations, spanning several orders of magnitude for all organisms. For some organisms such as *Campylobacter* in sewage, the ratio with *E. coli* varied by nearly 6 orders of magnitude. The uncertainty associated with the estimated ratios was not possible to capture since the underlying (raw) data was not available for analysis.

While the theory behind the use of index organisms is attractive, the practical application is subject to both the existence and quantitative description of the ratio between the particular index organism and pathogen under consideration. It may be that to assume that a ratio exists at all between *E. coli* or thermotolerant coliforms and pathogens is erroneous. Nonetheless, if these assumptions are applied for the purposes quantifying pathogen densities, then the variability and uncertainty associated with the estimated ratio need to be incorporated into the calculations. In particular, the implications of significantly underestimating pathogen densitie should be thoroughly explored.

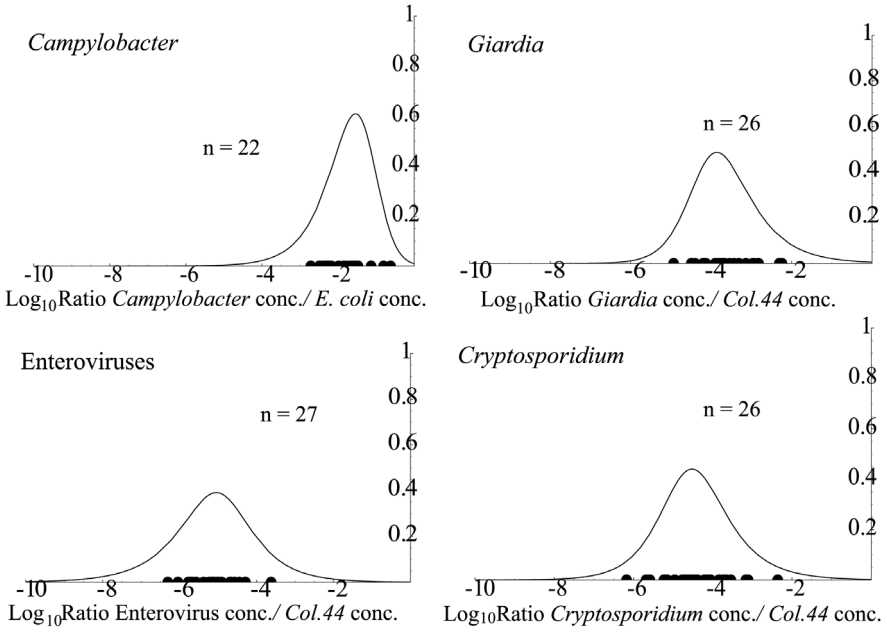


Figure 7.15. PDFs for the ratio between maximum likelihood gamma distributions for index organisms (*E. coli* and Coliforms at 44°C) and pathogen concentration, based on reported concentrations in surface waters (*n* is the number of paired concentrations). Data source: Medema *et al.* [2000]

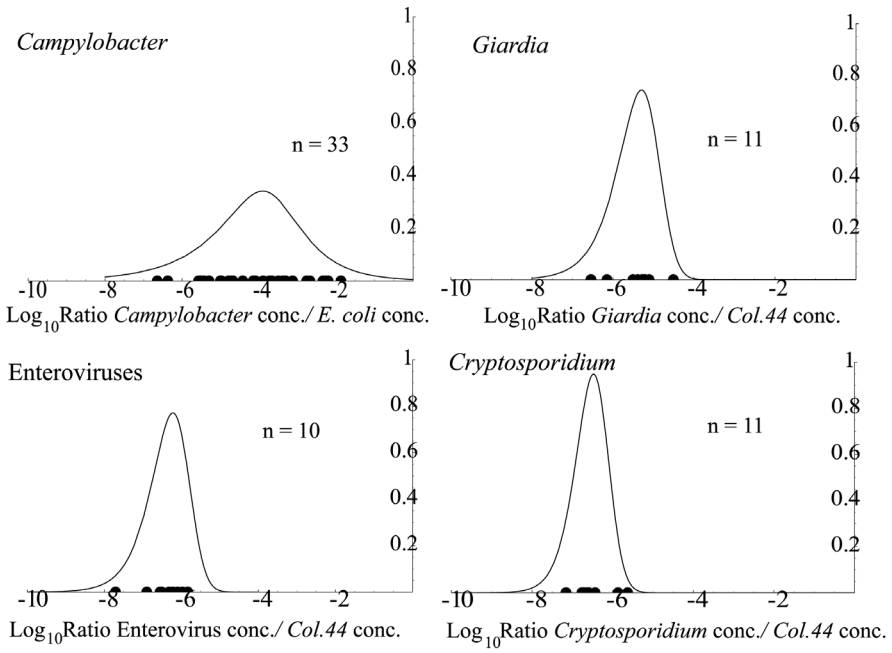


Figure 7.16. PDFs for the ratio between maximum likelihood gamma distributions for index organisms (*E. coli* and Coliforms at 44°C) and pathogen concentration, based on reported concentrations in sewage (*n* is the number of paired concentrations) Data source: *Campylobacter* [Hőller,1988], *Giardia, Cryptosporidium* and Enteroviruses [Medema *et al.*, 2000]

7.5.2. Quantifying Method Recovery

Analytical methods are imperfect, not all organisms present in the original sample may be recovered and enumerated in the laboratory. The result of a microbiological analysis is therefore a reflection of the number of identifiable micro-organisms present at the *conclusion* of the assay method. Interpretation of the original sample density from the assay results requires a quantitative understanding of the method recovery. Assuming that analytical results are precise representations of the original organism numbers may significantly underestimate the density. In addition, unaccounted for variability in method recovery may lead to apparent high variability in micro-organism density that is actually a product of the analytical process rather than of the original water quality.

Quantifying the magnitude and variability of recovery is important for interpreting analytical results. Experiments specifically designed to estimate recovery involve spiking a known number of micro-organisms into a sample volume that is subsequently analysed using the relevant protocol. Results of such experiments indicate that recovery varies between micro-organisms, between analytical methods and between laboratories. Recovery may also be expected to vary between subsequent samples even when analysis is undertaken using the same method at a single laboratory. There is also uncertainty that the spiked microbial preparation behaves the same as 'native' microorganisms or how difference in the water matrix between laboratory and natural samples influence the method recovery.

Microbial counts

In conventional seeding procedures for *Cryptosporidium* and *Giardia*, one sample is typically split into two for analysis. One sample is spiked with a known number of oo/cysts, while the other is unseeded. Statistical models for analysing this type of recovery data have been published [Teunis *et al.*, 1999a]. A modification to this approach has been developed [Francey *et al.*, 2004] involving the use of labelled oo/cysts known as ColorSeed™ (BTF Pty. Ltd., Sydney) that are spiked into a single sample, enabling the analyst to estimate the number of recovered seeded organisms (due to a unique colour), along with the native organisms from a single sample. Every sample analysed for *Cryptosporidium* and *Giardia* from the CTS 8 source water reservoir received a ColorSeed™ internal spike. The recovered ColorSeed™ counts (100 ±1 oocysts seeded from 110 samples analysed for *Cryptosporidium* are illustrated in Figure 7.17.

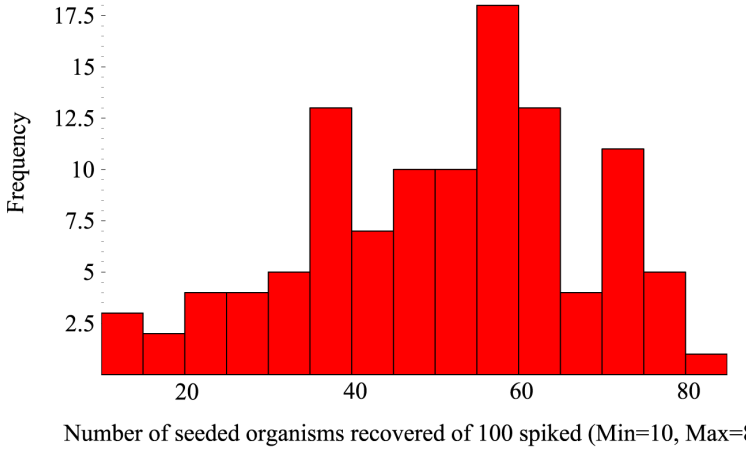


Figure 7.17 Histogram of the number of seeded oocysts recovered (from 100 spiked) for *Cryptosporidium* samples from raw river water at CTC 1

The variability in recovery between samples is clearly evident. These results are from the same source water, analysed at the same laboratory, using the same experimental protocol, and yet the variability is still high. The great advantage of the ColorSeed™ internal spike, is that a sample specific estimate of the recovery is obtained for each native count. Factors that drive the variability in recovery are still largely unknown, and therefore the internal spike reduces the uncertainty associated with the unknown influence of sample characteristics (including turbidity, temperature and pH) on the estimated recovery.

When an internal spike result is available for each native count, the recovery can be accounted for directly when estimating the PDF for source water concentration. If the recovery is assumed to be a binomial process (each organism may have one of two outcomes – it will be recovered or not recovered), where every organism has a certain probability (p) of being recovered. The number of spiked organisms recovered is an estimator of the probability of recovery (p). In this example, the probability of recovery on each sampling day was assumed to be independent of other sampling occasions.

Native counts were assumed to follow a negative binomial distribution and a likelihood function was constructed to account for the binomial probability of recovery (BOX 7.2).

BOX 7.2- Incorporating recovery into the negative binomial count distribution

If the counts are assumed to be generated from a Poisson process, with a probability of detection (*p)*, then the probability of counting *n* organisms given a sample concentration (μ) is:

$$P(n|\mu, p) = \frac{(\mu.p)^n e^{-(\mu.p)}}{n!}$$

Equation 16

When the mean source water concentration (μ) is assumed to follow a gamma distribution. The solution can be rearranged into the form of the negative binomial count distribution:

$$g(n|\lambda, \rho, p) = \frac{\Gamma(\rho + n)}{n!\Gamma(\rho)} \frac{\lambda^{\rho} p.^n}{(\lambda + p)^{\rho+n}}$$

Equation 17

If the number of recovered organisms k_i is assumed to be a precise estimate of the probability of recovery, then the likelihood function may be constructed as:

$$L(\lambda, \rho \mid n_{1-m}, k_{1-m}) = \prod_{i=1}^{n} g(n_i \mid \lambda, \rho, \frac{k_i}{100})$$

Equation 18

Allowing for uncertainty in estimation of binomial parameter (*p*)

The uncertainty associated with estimating *p* from the number of recovered organisms may be incorporated into the model using a hierarchical structure. Within a Bayesian framework, the posterior distribution of λ and ρ is proportional to the likelihood multiplied by the prior:

$$P(\lambda, \rho \mid n_{1-m}, k_{1-m}) \propto \underbrace{P(\lambda, \rho, p_{1-m})}_{\text{Prior}} . \underbrace{\prod_{i=1}^{m} P(\lambda, \rho \mid n_i, p_i)}_{Negativebinomal} . \underbrace{\prod_{i=1}^{m} P(p_i \mid k_i)}_{Binomial}$$

Equation 19

Where the binomial likelihood is described by: $l(p;k) = \binom{100}{k} p^k (1-p)^{100-k}$

An MCMC procedure then allows the stationary posterior distribution of λ and ρ to be characterised.

The maximum likelihood gamma distribution (expected value = 2.02 oocysts.L^{-1} and upper 95 percentile (variability) = 8.59 oocysts.L^{-1}) and credible intervals from MCMC (upper 95 percentile = 11.57 oocysts.L^{-1}) for the source water *Cryptosporidium* concentration CTS 8 is illustrated in Figure 7.18. The size of the dataset and the incorporation of recovery estimates for each day leads to a small credible interval surrounding the maximum likelihood gamma distribution.

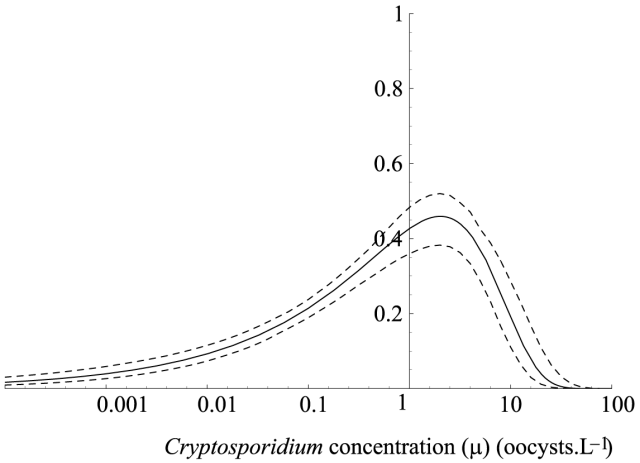


Figure 7.18. PDF for *Cryptosporidium* oocyst density in the raw river water for CTC 1: accounting for method recovery. Maximum likelihood gamma distribution (solid line), and 95% credible intervals (dashed lines) from MCMC modelling.

Internal spike material appears to be the most appropriate approach for estimating sample specific recovery for *Cryptosporidium* and *Giardia* oo/cysts. Unfortunately, this analysis was not provided with the dataset from any of the other CTSs studied. At best, laboratories provided a sample of recovery results believed to be representative for the entire dataset. In this case, recovery may be included as a variable in the risk model, described by a PDF [Teunis *et al.*, 1996]. In comparison to the 110 recovery results provided for CTS 8, three recovery experiments were undertaken for CTS 11. These experiments were also undertaken using the ColorSeed™ internal spike, however as they were not undertaken for every sample, the direct sample specific recoveries cannot be applied to the native results in the same manner as the previous example for CTS 8. Rather, by fitting a distribution to the recovery results, recovery may be included in the risk model as an independent variable. The results of these experiments are included in Table 7.7. Based on these results, the average recovery for *Cryptosporidium* oocysts may be expected to be 12%. This would be an appropriate point estimate assumption based on this data, however a point estimate does not allow for variability in the recovery between samples.

Table 7.7. *Cryptosporidium* oocyst recovery results CTS 11

***Cryptosporidium* oocysts**	
Number Spiked	Number Recovered
100	12
100	10
100	14

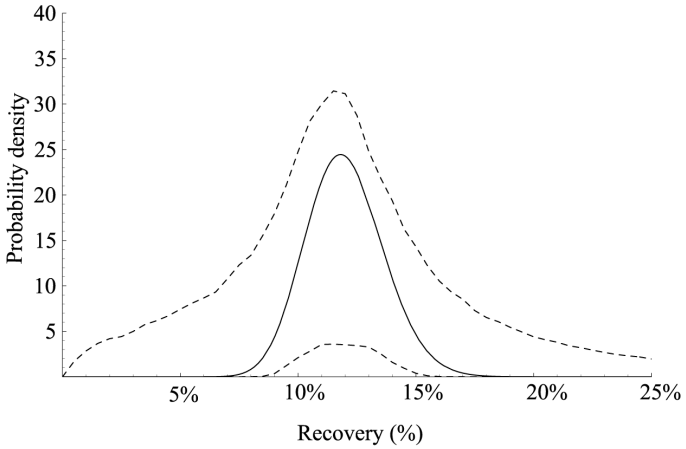


Figure 7.19. PDF for *Cryptosporidium* oocyst recovery with Maximum likelihood Beta Distribution(α= 47.1, β=345.43) (solid line), and 95% credible intervals from MCMC analysis.

To incorporate variability of recovery in the QMRA model, the probability distribution for recovery needs to be estimated. The variability in recovery has previously been described using a Beta distribution [Teunis *et al.*, 1999a, Teunis *et al.*, 1996] which is considered suitable as it is flexible and bound by 0 and 1. The Beta distribution was fitted to the data points (Table 7.7) using the method of maximum likelihood to obtain a best estimate of the PDF for recovery. Figure 7.19 show the shape of the beta distribution with the maximum likelihood estimates for α and β, and the 95% credible region constructed using an MCMC approach. Additional uncertainty is introduced when the recovery is incorporated into the model as an independent variable. Firstly, when the range of between sample variability is applied to every result, the variability and uncertainty associated with the source density may be expected to be increased. Secondly, there is danger that a small sample from a highly variable recovery may lead to an unrepresentative PDF. Comparison of the results in Table 7.7 with the variability in results illustrated in Figure 7.17, along with consideration of recoveries reported in the literature [Kuhn and Oshima, 2002], suggest that the three datapoints from CTS 11 may not be representative of the entire distribution of recovery. The reported recoveries are however low, leading to conservative estimates of source water density when the distribution in Figure 7.19 is applied to the native counts.

An important consideration of this approach for describing variability is the impact of fitting a continuous distribution to the experimental datapoints. The beta distribution projects to very low values close to 0. The true recovery would not however be expected to approach zero, but rather there would be minimum result below which, samples would be disregarded (based on laboratory QA protocols). When running the Monte Carlo simulation for QMRA, these unrealistic, very low values lead to occasional excessively high pathogen densities. These high pathogen densities are not considered to be representative of the system, but rather a consequence of the model assumptions. The parametric distributions are a tool to enable the estimation of the true PDF, and when they project beyond the realistic range of values, it is pragmatic to truncate them. It is therefore advisable to select a lower value at which to truncate the PDF for recovery, such as 1%, to avoid the generation of unrealistic values.

Presence/Absence results

Quantifying method recovery is also important for analytical methods that rely on identifying the presence/absence of the target organism in a sample volume. The assumptions of the previous examples involving microbial counts where recovery was assumed to be a binomial process may be extended to the presence/absence scenario. A specific experiment was used to investigate the recovery of *E. coli* O157 at one CTS [Suez Environnement, 2005]. This investigation consisted of three separate spiked solutions of known density (BioBall^{TM}). Each of these three solutions was sub-sampled (1L) and analysed 10 times for the presence or absence of the *E. coli* O157:H7. The results are included in Table 7.8.

Assuming that the spiked solution has a known concentration (μ), then the probability of the analysis yielding a positive ($n \geq 1$) or negative ($n = 0$) will follow a Poisson distribution with mean ($p\mu$, Equation 16) - where p is the binomial probability of recovery. A likelihood function was constructed based on the analytical outcomes (Table 7.8) to estimate the constant probability of recovery (p). The maximum likelihood estimator for the probability of recovery was 0.4, and the posterior sample for p based on MCMC sampling is illustrated in Figure 7.20.

Table 7.8. Results from *E. coli* 0157 detection limit experiment [Suez Environment, 2005]

Spike (org.L^{-1})	No. Pos (+)	No. Neg (-)
1	2	8
5	9	1
10	10	0

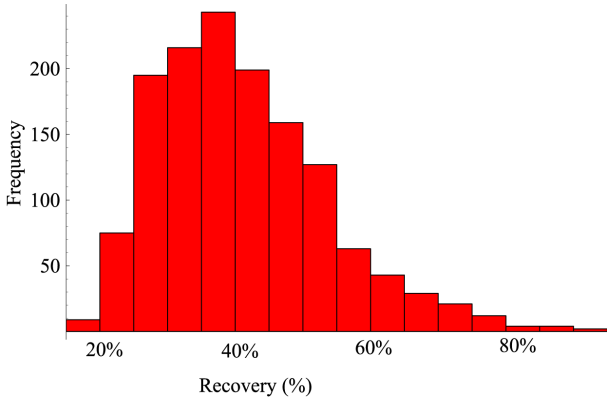


Figure 7.20. Posterior sample of probability of *E coli* O157 recovery parameter (p) from MCMC analysis

Figure 7.20 illustrates the uncertainty associated with the estimation of the probability of recovery. When these results are used in the QMRA the estimated recovery must be assumed to be representative of all *E. coli* 0157 analyses to which it is applied; therefore assuming that native organisms behave in the same manner as the spiked organisms regardless of water quality. The current model was chosen for simplicity and considered appropriate to the available data, however the recovery is only described as a point estimate and variation is not accounted for.

7.5.3. Quantifying Treatment Performance

A wide range of treatment processes exist for the physical, chemical and microbiological purification of drinking water. Each of these processes contributes to the removal or inactivation of pathogens from the water column. The effectiveness of each process in removing pathogens is variable: variable between different types of processes; between the same processes operated at different treatment facilities; and even variable over time for an individual process at a specified treatment plant.
The same treatment process may perform differently with respect to pathogen removal at different plants due to a number of factors including:

- process design – processes are optimized for the treatment of specific source waters, within the physical constraints of each specific sight;
- Source water- Different physical and chemical characteristics of source waters may be expected to affect the treatment performance; and
- Management – management protocol can vary between different managing agencies.

The performance of any given process may be expected to vary over time depending upon:

- Inlet water quality (including chemical, e.g pH, physical, e.g turbidity, and microbiological, e.g. algae count);
- Process conditions (e.g. chemical dosing, flow rate); and
- Maintenance (e.g. age of filter media).

Quantifying treatment removal performance for a drinking water CTS, accounting for the individual characteristics of the system being studied, and the expected temporal variability in performance for each process unit is a great challenge. Careful

consideration of the available data is essential. Incorporation of different types of data (including literature data, online data and surrogates) for estimating removal performance has been discussed in Chapter 4. In this section, modelling techniques for quantifying variability (and the associated uncertainty) in treatment performance based on microbiological data are discussed.

Two general approaches to modelling treatment performance can be applied:

- **Mechanistic process model:** Treatment performance is predicted based on models describing the mechanisms of pathogen removal/inactivation. For example, disinfection is modelled based on hydraulic flow characteristics, disinfection dosage and individual pathogen inactivation kinetics (Chapter 4).
- **Empirical transformation model:** Without specific consideration of the individual process, the outflow pathogen concentration is compared with the inflow pathogen concentration. This approach has great value for many processes, where quantitative information regarding the mechanisms of removal is very limited. For example, when modelling filtration performance, a mechanistic process model would give specific consideration to adsorption/desorption of microorganisms to the filter media, straining efficiency and inactivation rates within the filter. In contrast, a transformation model simply estimates total removal across the filter (or series of filters) based on outflow concentration as a fraction of the inflow.

The obvious limitation of simplifying a treatment process to a simple transformation is that any estimated removal is specific to the individual process and system studied, for the time frame represented by the available dataset. Since the underlying mechanisms and process characteristics are not accounted for (e.g. flow rate, source water quality, hydraulic design etc.) the impact of modifications to the system on pathogen removal cannot be quantitatively projected. Similarly, direct translation of results from one system to another is difficult, since the impact of even apparently minor differences in design or source water quality is unknown. In this section, approaches for modelling treatment processes by simple transformation are presented. The emphasis is placed on describing the variability and uncertainty associated with the transformation from the data available.

Transformation model assumptions

Pairing data points

Samples collected at the inlet and outlet may or may not be *paired*. Given a set of data, where samples have been collected and analysed from the inlet and the outlet of a water treatment process, should samples collected on the same day be assumed to be paired? For example, consider results from the enumeration of *Giardia* from water samples collected from the inlet and outlet of the water treatment process at CTS 10 (Table 7.9). For these results, is the reduction in *Giardia* cysts calculated on a daily basis actually representative of the variability in removal performance of the process? Or is it better to look at the overall removal as a point estimate of the process performance?

When two samples are assumed to be paired, they are assumed to represent the "same" water as it enters and leaves the treatment process. To obtain a truly paired sample, the outlet sample must be delayed from the inlet sample by the hydraulic retention time of the process. In reality this is rarely the case. Results from CTS 10, while not technically "paired", are clearly correlated (Spearman rank correlation

coefficient = 0.37)[7], with high inflow samples coinciding with high outflow samples (e.g. day 11) and vice versa (Day 5 and 6). It is often reasonable, for rapid processes, to assume that samples collected at approximately the same time, on a given sampling occasion are paired. Analysis can be undertaken with or without the assumption of pairing, however assuming that samples are paired allows for between sampling day variability to be characterized.

Table 7.9. *Giardia* counts enumerated from inflow and outflow samples collected from CTS10, with estimated concentrations and Log_{10} removal rates[8]

Sampling Day	Inflow			Outflow			REDUCTION
	Count	Volume	Estimated Conc. (cysts.L^{-1})	Count	Volume	Estimated Conc. (cysts.L^{-1})	Estimated Removal (Log_{10} reduction)
1	3	10	0.3	0	100	0	> 0.52
2	6	10	0.6	14	100	0.14	0.63
3	2	10	0.2	7	100	0.07	0.46
4	2	10	0.2	3	100	0.03	0.82
5	0	10	0	0	100	0	-
6	0	10	0	0	100	0	-
7	1	10	0.1	6	100	0.06	0.22
8	3	10	0.3	0	100	0	> 0.52
9	6	10	0.6	0	50	0	> 0.22
10	2	10	0.2	0	50	0	> 0.70
11	30	10	3	18	100	0.18	1.22
12	0	5	0	2	100	0.02	-
Average of all days			0.46			0.042	1.04

Variability in source water concentration

Constantly varying source water concentrations and random sampling effects can influence the representativeness of direct concentration comparisons, particularly for small microbial datasets. When outflow density estimates are "paired" with inflow samples collected on the same day, perplexing results can emerge including the apparent increase in microbial densities on some days (e.g. Table 7.9, Day 12). It is conceivable that for some processes, during a particular event, the microbiological concentration in the water column may be increased as a result of passage (for example regrowth of bacterial pathogens in filter media, or sloughing of a filter), however a far more likely and common explanation leans on the knowledge that source water concentration is constantly varying. By implication, if the number of

[7]Spearman rank correlation coefficient: $\tau = 1 - \frac{6\sum_{i=1}^{k}(r_{i,1} - r_{i,2})^2}{k(k^2-1)}$, Where k is the number of paired sample points and r_1 and r_2 are the within sample ranks for each inflow and outflow sample [Haas *et al.*, 1999, p343]

[8] Raw results are the counted number of organism ("Count") in sample volume ("Volume"). These results were used directly to find an estimate of the cyst density for that sample (Count/Volume), and then the estimated Log reduction was calculated from those densities (Log_{10}Reduction = $Log_{10}(Conc_{out}/Conc_{in})$). When there were no cysts found in the outflow concentration, the estimated Log_{10}Reduction was reported as $>Log_{10}Conc_{in}$, when there were no organisms reported in the inflow and outflow, then no estimate of reduction could be made. Overall removal was calculated based on the average $Conc_{in}$ and average $Conc_{out}$ over all samples (including zeros).

organisms in the sample collected at the inlet was an instantaneous low density, while the outflow sample contained an instantaneous high density, the apparent change in density would be an increase, even though the underlying mean concentration may have decreased. Similarly, as a result of random sampling variability (see Section 7.3.1 p15) if the inflow sample contained a small number or organisms given the mean density, and the outflow sample contained a large number of organisms, an apparent increase in concentration may also result.

Accounting for random sampling variability and a varying source water concentration in the transformation model allows the underlying treatment removal performance to be characterised. In the same way as for source water characterisation, for the analysis of MicroRisk datasets, sampling variability was accounted for using the Poisson distribution, and mean sample concentration was assumed to vary according to a Gamma distribution (see Section 7.3.2).

Ratio of outflow concentration to inflow concentration

The probability that any individual organism will pass the treatment barrier (π, see Figure 7.1) can be estimated by the ratio of the outflow concentration to the inflow concentration. The Log_{10} of π is the Log_{10} removal of the process(es). For example, in Table 7.9, the Log10 removal is estimated for each day by calculated the ratio of the outflow concentration to the inflow concentration for each sampling day. This concept may be extended to account for variability in the inflow and outflow concentrations by finding the ratio of the outflow concentration PDF to the inflow concentration PDF. The resulting PDF for the ratio is therefore the PDF for π. An example of this approach is illustrated using Giardia results from CTS 10 (Table 7.9). This example is illustrative of using pathogen data to characterise treatment performance. In this situation, if the only aim was to calculate microbial risk, the outflow *Giardia* density PDF would be a suitable input to the QMRA model. By characterising the process performance, however, opportunities for management are possible (i.e. is the process working according as expected) and source water pathogen densities (where monitoring is more feasible) can be translated to expected outflow density.

A gamma distribution was fitted to the *Giardia* counts using the Poisson-gamma (negative binomial) mixture model (Equation 13), the maximum likelihood gamma distributions for the inflow and outflow are illustrated in Figure 7.21.

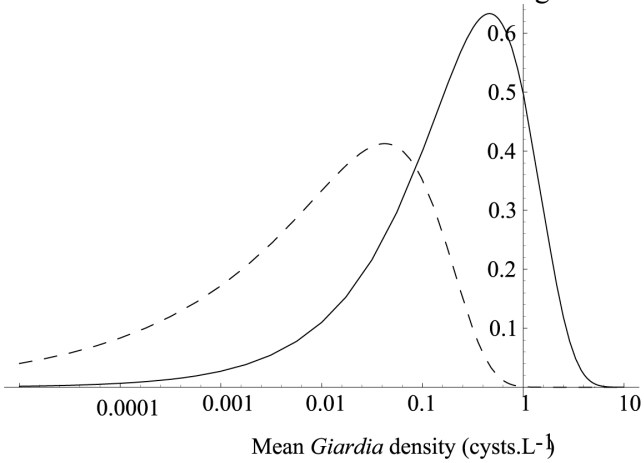


Figure 7.21. Maximum Likelihood Gamma distributions for mean *Giardia* density in the inflow (solid line) and outflow (dashed line) at CTS 10. Inflow: Gamma [$\hat{\rho}$ =0.61, $\hat{\lambda}$ =0.75], Outflow: Gamma [$\hat{\rho}$ =0.31, $\hat{\lambda}$ =0.13].

The distribution for the ratio of the outflow gamma distribution to the inflow gamma distribution was calculated using a random sampling procedure (Monte Carlo analysis) were random sample were drawn from the inflow and outflow distribution, and the ratio calculated for each random sample. A histogram of 10 000 random samples is illustrated in Figure 7.22a).
The great advantage of using the ratio to estimate treatment performance is simplicity; the estimate is a direct comparison between outflow and inflow. In addition the assumptions associated with the removal performance are limited, relying only on the assumed pathogen density distribution. The ratio is not constrained to be less than one, and therefore the removal performance can take any value, and may even be positive (increase in pathogen density). Application of this ratio, to any *Giardia* inflow density sample, should provide an indicative estimate of the outflow density at CTS 10^9.

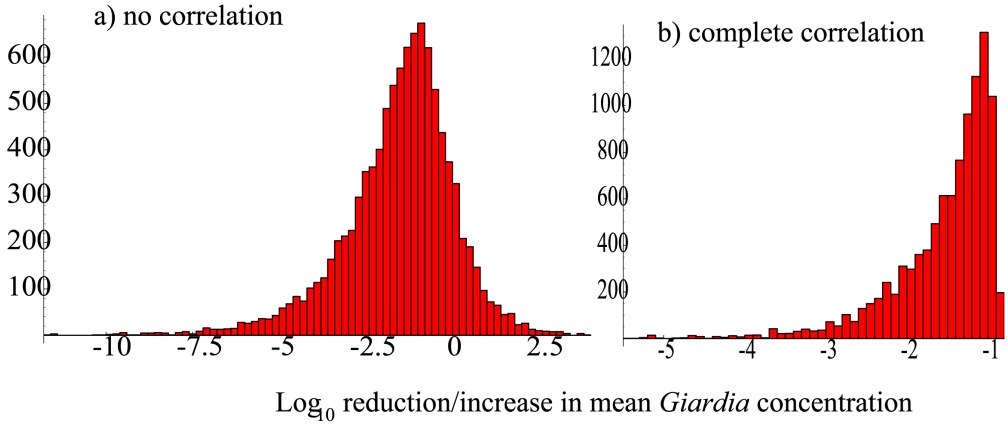


Figure 7.22. Histogram of Monte Carlo sample for the ratio between inflow and outflow Gamma distributions for *Giardia* density. a) no correlation - Lower 5%, 50% and Upper 95% quantiles of the sample were [- 4.51, -1.33, 0.70] b) complete correlation – random samples sorted before ratios calculated Lower 5%, 50% and Upper 95% quantiles of the sample were[-2.76, -1.34, -0.92]

The ratio distribution illustrated in Figure 7.22a) was constructed assuming that the inflow and outflow concentrations were independent. This is however counter intuitive. Since the mean concentration would be expected to be reduced as a result of treatment, the random sample from the outflow distribution should always be less than the random sample from the inflow distribution. A simple modification to the Monte Carlo sampling procedure was undertaken assuming 100% correlation between the variables: random samples of inflow and outflow distributions were sorted (forcing complete rank correlation). The ratio was then calculated on these rank paired random samples, the resulting histogram of the Log_{10} reduction is illustrated in Figure 7.22b. The expected values of both analysis are similar (-1.33 versus -1.34), however the variability (and uncertainty, since they are not separated in this model) in the distribution for *Giardia* removal is greatly reduced in the correlated model in comparison to the independent model. The true result would be expected to lie somewhere between these two unrealistic extremes.

[9] Parameter uncertainty associated with the fit of the Gamma distributions was not accounted for in this analysis.

Binomial models

Models have been presented in the literature that describes organism passage through treatment as a binomial process [Teunis *et al.*, 1999a], where each microorganism faces one of two possible outcomes, passage or removal. Mean microorganism concentration in the inflow (μ) is assumed to follow a gamma distribution. Microorganism concentration in the finished water is then assumed to equal $\pi.\mu$, where π is the binomial probability of passage. A joint likelihood function can then be constructed that describes the inflow and outflow counts by a single gamma distribution, scaled by the binomial probability of passage (π). Models for estimating the beta distributed probability of passage, under the assumptions of paired and unpaired data, as presented by [Teunis *et al.*, 1999a] were applied to the *Giardia* data from CTS 10 (Table 7.9). The results from likelihood analysis, including maximum likelihood parameter values are given in Table 7.10.

Table 7.10 Results from likelihood analysis of paired and unpaired binomial models fitted to *Giardia* counts from CTS 10 (Table 7.9)

	-2Loglik	$\hat{\rho}$	$\hat{\lambda}$	$\hat{\alpha}$	$\hat{\beta}$	$Log_{10}(\alpha/\alpha+\beta)$*
Unpaired	116.3	0.62	0.73	0.63	5.23	- 0.97
Paired	112.9	0.67	0.67	0.51	3.72	- 0.92

*Log_{10} of the expected value of the beta distribution

Results from the likelihood analysis indicate that estimated reduction in *Giardia* was similar under either the paired or unpaired data assumptions. The paired model achieved a slightly better fit than the unpaired model (compare -2Loglik, deviance = 3.7) for the given dataset. The maximum likelihood beta distribution is illustrated with credible intervals in Figure 7.23. The expected removal of *Giardia* was 0.92 Log_{10} units with a 95% interval of variability ranging from 3.3 to 0.37 Log_{10} units.

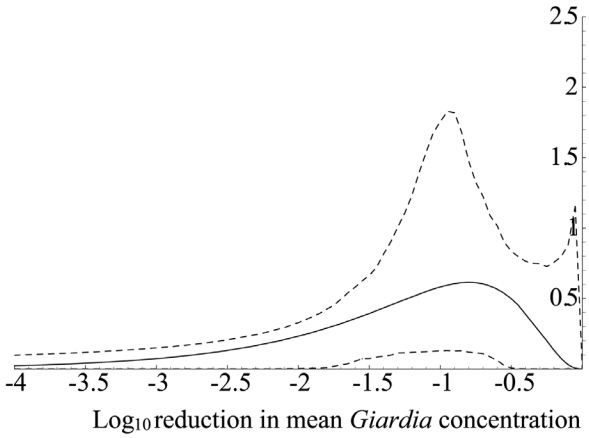


Figure 7.23 Maximum likelihood Beta distribution (solid line) and 95% credible intervals (dashed lines) for *Giardia* removal at CTS 10

The binomial modelling approach goes beyond the ratio calculations presented previously by making additional assumptions, in particular, that the passage of organisms through a treatment barrier is a binomial process. If each organism is assumed to have a certain probability π of passage, then the estimation of π is restricted to values between 0 and 1, eliminating the issue of negative removal. Within this approach, the uncertainty associated with the shape of the beta distribution can

also be explored. The calculations involved in undertaking this analysis are relatively straight forward to implement in a mathematical software package (equations available in Teunis *et al.* [1999]), however the likelihood functions can become complex requiring not insignificant computational time. The implementation of a hierarchical modelling approach can simplify these computational issues (see Gelman *et al.* [2004] for full explanation of methodology) while achieving equivalent results.

Surrogates

In order to apply a transformation model, and estimate the removal of organisms, it is necessary to have a sample of data points from the inlet and the outlet of the individual process or treatment chain to be studied. While some negative results can be easily managed through the application of a discrete distribution, the outlet sample must contain some positive results. Pathogens are usually present in finished waters at densities well below the limits of detection, and therefore this data requirement can be difficult to meet. The analysis and interpretation of surrogates (native or spiked) can then be necessary. While the same models can be used to evaluate treatment removal of microbiological surrogates, it is important to not ignore the uncertainty associated with assuming a given surrogate is representative of the pathogen of interest. In order to account for this quantitatively, data must be available to quantify the ratio or relationship between the surrogate and the pathogen for the given treatment process. When this ratio (including uncertainty) is applied to the surrogate removal PDF, a more representative estimate of the uncertainty associated with the pathogen removal PDF may be obtained. This type of data is rarely available, however the implications of uncertainty associated with the application of treatment performance surrogates must be accounted for. One proposal would be to use evaluate the assumption using sensitivity analysis (see Section 7.4.1).

7.5.4. Dose-Response

Many studies have been published that estimate dose-response relationships for human pathogens. Those studies relevant to the MicroRisk project are summarised in Table 7.11 and in the following sections. There are numerous sources of uncertainty regarding how adequately the cited dose-response models reflect the true impact of pathogen consumption on the population. Frequently, more than one model is relevant for consideration. Rather than proposing one single model as correct for each reference pathogen, some notes providing guidance on the different models considered and the data on which they are based are included in the following sections.

Campylobacter

Two studies have been conducted for estimating parameter values for the dose-response relationship of *Campylobacter jejuni.* The first fitted the Beta-Poisson model to data from a single human feeding trial, where administered doses were generally high [Black *et al.* 1988; Medema *et al.* 1996]. More recently, a second study has been presented that fits the dose-response relationship to both the first human feeding study and also two small outbreaks related to the consumption of raw milk [Teunis *et al.* 2005]. This second study gives consideration to low dose behaviour and indicates that health risks may be higher at lower doses than previously assumed from the first published parameter estimates. This second model is therefore more conservative and

may be more representative of the entire population (including children) rather than simply healthy adults.

Table 7.11. Summary of key dose-response studies and associated results to be used for risk analysis

Reference Pathogen	DR Study Organism	Model	Parameters		Original Data Source	DR Analysis Source
Campylobacter	*Campylobacter jejuni*- Human feeding study	Beta-Poisson	α=0.145	β=7.59	Black *et al.* [1988]	Medema *et al.* [1996]
	Campylobacter jejuni –Outbreak data*	Beta-Poisson**	α=0.024	β=0.011	Van den Brandhof *et al.* [2003] Evans *et al.* [1996]	Teunis *et al.* [2005]
E. coli 0157	Enteropathogenic *E. coli* (EPEC) *Shigella disenteriae* (combined data)	Beta-Poisson	α=0.22	β=8.7 $\times 10^3$	Levine *et al.* [1973]	Powel *et al.* [2000]
	E. coli 0157 – Outbreak data Adults: Children:	Beta-Poisson**	α=0.084 α=0.050	β=1.44 β=1.001	Shinagawa *et al.* [1997]	Teunis *et al.* [2004]
	Shigella disenteriae	Beta-Poisson	α=0.157	β=9.16	Levine *et al.* [1973]	Teunis *et al.* [1996]
Norovirus	Rotavirus strain CJN(clinical isolate, not passed prior to administration) Human feeding trial	Beta - Poisson	α = 0.253	β = 0.422	Ward *et al.* [1986]	Teunis *et al.* [1996]
Enterovirus	Echovirus 12 clinical isolate Human feeding trial	Beta – Poisson	α = 0.401	β = 227.2	Schiff *et al*. [1984]	Teunis *et al.* [1996]
	Coxsackie A	Exponential	r = 0.014493			
Cryptosporidium	*Cryptosporidium parvum* (isolate from a calf) Human feeding trial	Exponential	r =4.005 $\times 10^{-3}$		DuPont *et al.* [1995]	Teunis *et al.* [1996]
	Combined dataset of three isolates collected from neonatal calves.	Beta-Poisson	α = *0.115*	β = *0.176*	Teunis *et al*., 2002a	Teunis *et al*., 2002a
Giardia	*Giardia lamblia* Human feeding trial	Exponential	r = 1.99$\times 10^{-2}$		Rendtorff (1954)	Teunis *et al.* 1996

*In this study, outbreak data was combined with the previous human feeding study to find overall dose-response parameter estimates.

**This study used the exact Beta-Poisson relationship rather than the approximation used in the other studies cited in the table.

Figure 7.24 illustrates the difference between the two published *Campylobacter* models. Teunis *et al.* [2005] assumes higher infectivity at low doses. If this model were used instead of the previous model Medema *et al.* [1996], predicted infection

estimates would be more than an order of magnitude higher at low doses. Conversely, at high doses, the model from Teunis *et al.* [2005] would predict lower infection rates.

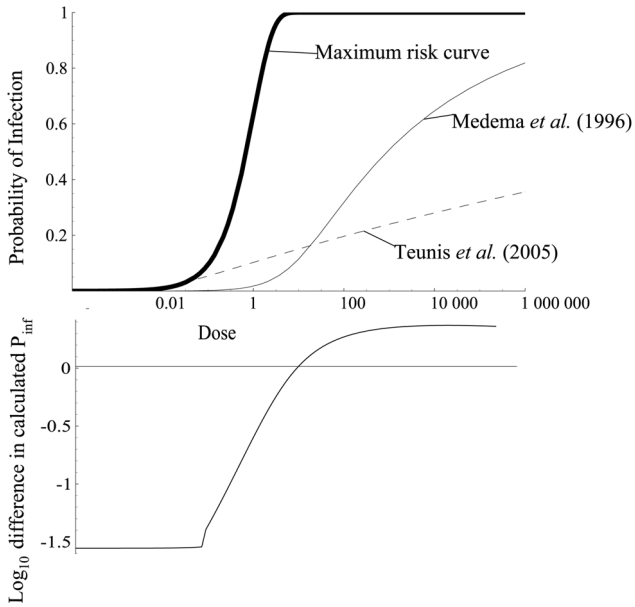


Figure 7.24. Dose-response relationships for *Campylobacter* and maximum risk curve, including the Log_{10} difference in calculated probability of infection with dose

E. coli 0157

Powell *et al.* [2000] combined human feeding study results from two surrogate organisms Enteropathogenic *E. coli* (EPEC) and *Shigella disenteriae* to provide a relationship for *E. coli* 0157:H7. Teunis *et al.* [2004] analysed actual outbreak data from school children and teachers who ate contaminated lunch [Shinagawa *et al.* 1997]. This later study compared their results with the former and identified the results from *Shigella disenteriae* (analysed by Teunis *et al.* [1996]) appeared to have the greatest agreement with the actual outbreak data [Teunis *et al.* 2004].

Viruses

While under development, the dose-response model for *Norovirus* is yet to be published. The dose-response relationships for Rotavirus, Echovirus and Coxsackie A are illustrated in Figure 7.25 with the maximum risk curve.

Figure 7.25 illustrates the relative infectivity assumed for each virus dose-response model. Echovirus 12 is the least infectious, followed by Coxsackie A and Rotavirus. The comparison of each individual model prediction with the maximum risk demonstrates that model choice has a large impact on estimated probability of infection at low doses. For viruses of relatively low infectivity, such as Echovirus 12, the choice of a conservative model such as Rotavirus or the maximum risk curve, may overestimate infection risk by more than 2 orders of magnitude. Conversely for pathogens that are known to be highly infectious, the assumption of the maximum risk curve may be considered, particularly in the absence of pathogen specific information.

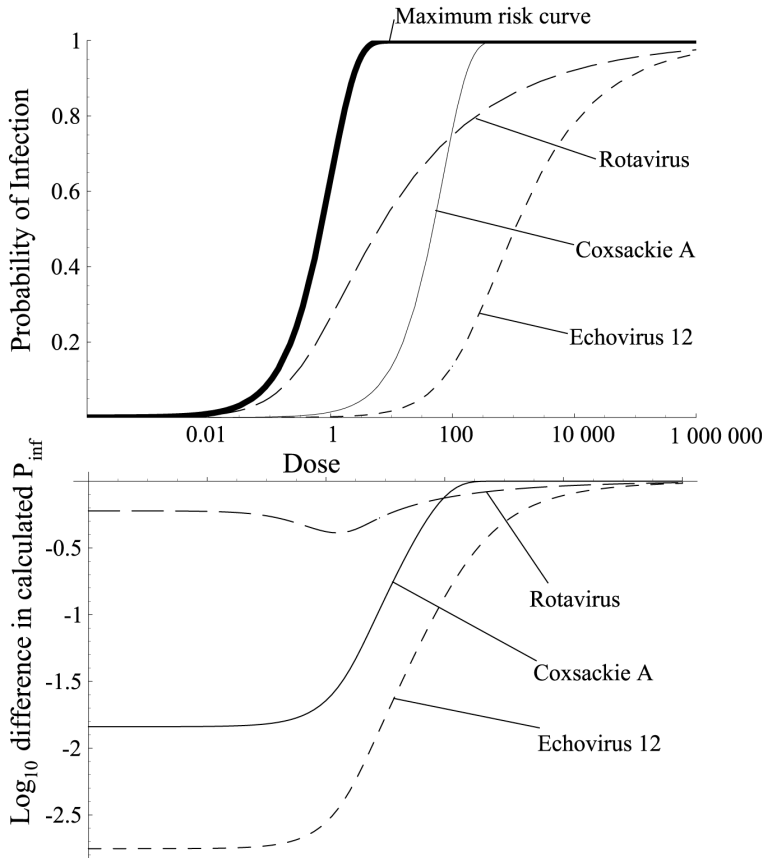


Figure 7.25 Dose-response relationships for Rotavirus, Coxsackie A, Echovirus 12 and the maximum risk curve, including the Log10 difference in calculated probability of infection between each individual model and the maximum risk.

Giardia

Results from human feeding trial with *Giardia lamblia* in were reported more than 50 years ago [Rendtorff, 1954]. The data was adequately fitted by an exponential distribution.

Cryptosporidium

Results from a human feeding trial with *Cryptosporidium parvum* were published by DuPont *et al.* [1995]. The data was adequately fit by the exponential model. More recently, work has been undertaken to investigate the variability in infectivity for *Cryptosporidium* between isolates [Teunis *et al.* 2002a] and between hosts [Teunis *et al.* 2002b]. The maximum likelihood estimates for parameter values when the Beta-Poisson model was fitted to the combined isolates dataset is included in Figure 7.26 The dose-response relationships for *Cryptosporidium* and *Giardia* are illustrated in Figure 7.26.

Figure 7.26 illustrates the comparison between the two published *Cryptosporidium* models. Selection of the more recent *Cryptosporidium* dose-response model instead of the previous model [Teunis *et al.*, 1996] may increase probability of infection estimates at low doses by more than 2 orders of magnitude.

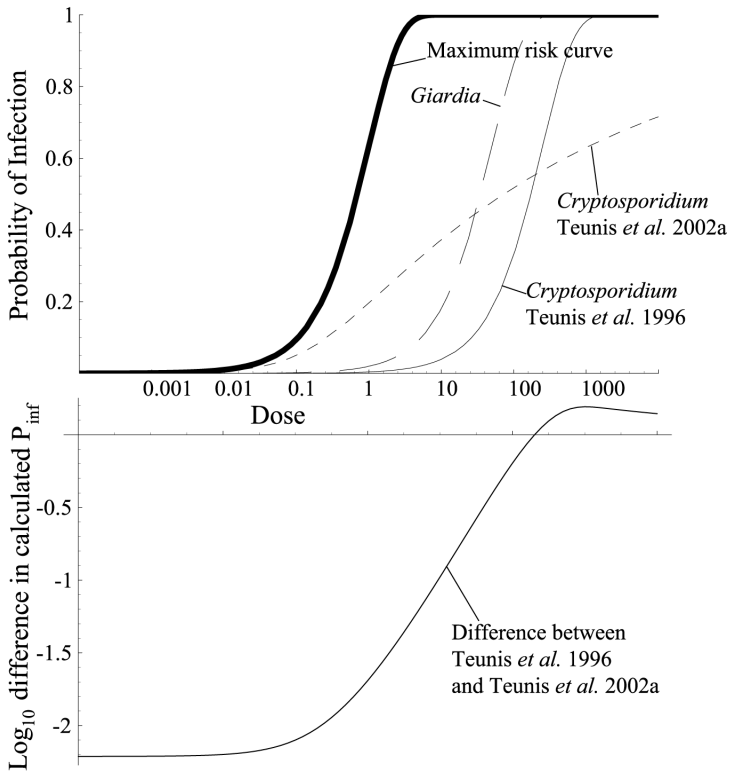


Figure 7.26 Two dose-response relationships for *Cryptosporidium, Giardia* and the maximum risk curve, including the Log_{10} difference in calculated probability of infection between the two *Cryptosporidium* curves

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8 How to Implement QMRA to Estimate Baseline and Hazardous Event Risks with Management End Uses In Mind

David Roser, Susan Petterson, Ryan Signor, Nicholas Ashbolt, Per Nilsson, Rebecka Thorwaldsdotter

8.1 INTRODUCTION

The goal of this chapter is to illustrate how Quantitative Microbial Risk Assessment (QMRA) can be undertaken in practice utilising case studies from the MicroRisk project. It outlines the data analysis strategies and describes the simulation methods using illustrative input and output data collected on selected Catchment-to-Tap Systems (CTSs). It is designed to show:

- What system-level QMRA analyses require;
- How sub-model component (e.g. models describing each barrier) vary depending on such factors as data quality, data selection, source water quality and treatment system design;
- How widely the risk magnitudes estimated vary depending on the issue being addressed, the level of analysis sophistication (Tier), the pathogens modelled, and CTS structure;
- How the data output varies with aim of a simulation (e.g. estimation of 'Baseline' or 'Hazardous Event' effects); and
- How risk estimates might be used to support water management activities.

It is proposed that for each CTS/pathogen combination a full risk analysis involve 4 stages.

1. Baseline QMRA
Firstly risks to consumers from their water supply would be estimated for 'nominal' or Baseline conditions; that is the predominant operating conditions under which a CTS is understood to supply water to consumers. By virtue of their predominance, Baseline conditions should have the most comprehensive associated sets of system performance and operations information. Risks associated with Baseline conditions would by definition be the minimum achievable and hence the first to be assessed for acceptability.

2. Sensitivity Analysis
The second step is to undertake 'Sensitivity Analysis' on Baseline risk probability. Each barrier or stage is set in turn to its worst reasonable value and the simulation model is rerun. The process forces the water manager to critically consider vulnerable points (Control Points) within the supply train and estimate a worst case situation. The Factor Sensitivity (*FS*) values calculated by dividing each extreme (perturbed) Baseline simulate risk value by the normal Baseline risk, which serves to identify where Hazardous Events might lead to elevated risks.

3. Hazardous Events QMRA
Next the risks arising from specific 'Hazardous Events' [Nadebaum *et al.* 2004] are modelled. Hazardous Event conditions are assumed to apply when there is a perturbation from Baseline conditions which increases the infection risk to consumers. The increase in risk

is estimated by simulating Baseline and Hazardous Event conditions and combining the risks in proportion to the time the CTS is in one or other state to estimate a Baseline+Hazardous Event risk. Sensitivity Analysis issues and outputs act as guides to scenario construction. Though the risk increase from several concurrent Hazardous Events can be simulated, a meaningful total Baseline+Hazardous Events risk cannot be calculated in practice because of the conceptual uncertainties. Currently there is little data suited to QMRA on the magnitude, duration and diverse attributes of many common Hazardous Events. And by definition there is almost no useful data on rare, high impact Hazardous Events.

4. Use of QMRA results in risk management
Finally the Baseline and Hazardous Event risk estimates and models are used to inform water supply management. The Risk probability estimates (probability infection.person^{-1}.y^{-1}) are used in two ways. Baseline and Hazardous Event risk estimates provide relative measures of risk which can be used to judge whether an alternative scenario (new plant, altered source water etc.) leads to a marked increase in risk compared to that already existing. Additionally where data is considered reliable enough, risk estimates may be treated as providing absolute measures of risk which can be compared to predetermined Tolerable Risk limits to determine whether a operating conditions under a given water supply scenario provides 'safe' or whether specific processes are within acceptable 'Critical Limits'.

This chapter concludes with a discussion outlining the strengths and limitations of the actual QMRA input and output data and suggests how the QMRA approach presented here can support and be integrated in practice with Water Safety Planning.

This chapter is structured as follows:
- An outline of how microbial data has been used for QMRA analysis and considerations informing these analyses;
- An illustrative worked example of one CTS (CTS 8) detailing the calculation of Baseline risks posed by one index pathogen, *Campylobacter*;
- Extension of the basic risk estimation approach to Sensitivity Analysis, the estimation of the risk arising from Hazardous Events, management including the estimation of Critical Limits and supplementation of Baseline risks with those resulting from Hazardous Events in the Distribution system;
- Illustration of the diversity of risks estimates encountered when a range of CTSs and pathogens are analysed concurrently; and
- A discussion of the uses and limitations of QMRA, its benefits and strengths.

8.2 METHODS AND ISSUES FOR A WATER MANAGER'S CONSIDERATION

8.2.1 Simulation of Risk within the Context of Water Safety Planning

QMRA application is a moderately complex procedure. Consequently the Water Manager should be clear about why they are doing QMRA, what information they intend to obtain from the process by way of better understanding and managing risks in their CTS and how far they intend to take the process. They should also be prepared to stop the process early as a contingency. This could occur because a simple analysis is all that is required as when absolute risks are either trivial or very severe and the answer to the issue being considered is

evident. At the other extreme a critical data gap may exist such as a lack of credible source water data on the pathogen of interest. In this case new field measurements of water quality or treatment processes may be necessary before a useful outcome can be provided..

8.2.2 Decision Making and QMRA in Practice

Science and mathematics underpin pathogen behaviour concepts, analytical methods, and the probability theory underlying QMRA and modelling technology. This association tacitly suggests that water treatment Decision-Making (e.g. process management, disaster response) based on QMRA is also 'scientific' and 'objective'. This is however a misapprehension which water managers must avoid if they are to use QMRA appropriately.

The application of science to environmental policy and decisions has numerous limitations and complications which have become evident with increased understanding of how science works in practice [e.g. see Giampetro *et al.* 2006]. This does not imply science has no place in management decisions but rather its roles and relationship need clear definition.

Dowie [2005] outlines the problem as it applies to 'Evidence Based Medicine' of how to translate probabilistic scientific knowledge into risk decisions which minimise human health risks. He proposes a range of solutions based "Decision Making" concepts arising consideration of how Bayesian Statistics work (term proposed by the author in Bayesian Decision Analysis). This same general objective, of wishing to apply sound statistics (on water treatment process effectiveness) to decision making (e.g. need to upgrade treatment) is faced by water supply managers.

From consideration of Dowie's [2005] arguments the following is proposed, by analogy, regarding pathogen risk assessment and management and where QMRA might fit in:

- Management decisions based on traditional water supply concepts (e.g. no coliforms = safe water) and the newer qualitative risk assessment approach are in actuality based on risk probability estimation. But the probability estimation process is tacit and is based on intuition informed by expert knowledge of waterborne pathogens etc. rather than strict statistical approaches.
- Experts can consider a range of diverse information of waterborne pathogen risks and through experience integrate and reduce risks. But they are not good at combining multiple numerical probabilities and estimating aggregate risks intuitively.
- To improve 'Decision Making' aggregate probabilities can be better estimated by substituting intuited probabilities where possible with ones calculated systematically using probability theory.

In summary QMRA appears to have great potential as an adjunct to water management decision making and qualitative risk assessment not only intuitively but from theory being currently developed in allied disciplines.

8.2.3 Risk Assessment Tiers

A concept often encountered in different risk assessment fields in that of Tiers [e.g. KarDouzas and Capri 2004; CMPHU; 2005; Hendley *et al.* 1998; Hart *et al.* 2005].

Practically Tiers have two common roles. Firstly they identify to a reviewer the general extent to which a risk analysis has been undertaken and hence its general strengths and limitations in support of decisions and whether it is sufficient for the desired decision support role. Secondly as outlined in Chapter 7, they provide a logical sequence whereby the complexity of a risk assessment exercise may be varied according to what information is needed. This sequence is summarised in Figure 7.4.

With this in mind Dowie's [2005] recommendations were reconsidered with a view to identifying a possible basis for a QMRA Tier classification. Four ways were identified in which intuition dominated CTS risk assessments are replaced by systematic and statistical data treatment as proposed within QMRA:

1. Replacement of the zero risk concept (no indicators present) with numerical probability estimates;
2. Replacement of end of system risk assessment (e.g. coliform testing on finished water) with assessment of the contribution of all water supply stages (i.e. analysis of barrier by barrier removal);
3. Recognition that water quality, barrier process effectiveness and risk probability data are better described by probability density functions (PDF) than point values; and
4. Recognition that the future behaviour of a specific CTS is being predicted from historical data sets whose applicability to the system of interest varies.

As each of these enhancements represents a different way in which BDA principles can be applied to CTS risk assessment it is proposed that the extent to which these enhancements are implemented be the basis for a series of QMRA Tiers, specifically that:

- Tier 1 be equivalent to enhancements 1 and 2
- Tier 2 be equivalent to enhancements 1, 2 and 3
- Tier 3 be equivalent to enhancements 1, 2, 3 and 4.

In this scheme the Tiers roughly equate to:

- Modelling of pathogen reduction by barriers using point values also known as screening level risk assessment (Tier 1);
- Monte Carlo style modelling of source water concentration and barrier effects as simple distributions based on minimal assumptions of PDF attributes largely using data derived from general literature (Tier 2); and
- Monte Carlo style barrier modelling using CTS specific data to estimate PDFs, variability and uncertainty (Tier 3).

The practicality and use of this Tier assignment classification model is illustrated in Section 8.3. The primary aim is not to establish a specific Tier classification scheme but to demonstrate how the Tier concept could work, trial it on a real set of data and illustrate the different possible QMRA outputs and model assumptions which may be developed for even a single CTS/pathogen risk assessment.

8.2.4 An Illustrative CTS and Index Pathogen

The system selected for illustrative purposes was CTS 8. This system was selected as:

- This catchment-to-consumer system is essentially a simple linear one with a single source dominant water and homogenous water treatment plant wherein all water receives exactly the same type and degree of treatment;

- A large body of historical pathogen and indicator information was available for this CTS and additional data was collected on most barriers during the course of the MicroRisk project; and
- Good ancillary data was available including catchment hydrology information, and treatment plant SCADA data.

The basic structure of CTS 8 is shown in Figure 8-1. The four principle barriers modelled (a reservoir, a flocculation/coagulation step, a particulate filter and disinfection system) were commonly encountered among the other MicroRisk CTSs. Also the conceptual barrier configuration of CTS 6 was very similar (River source > reservoir > flocculation > particle filter > disinfection > storage > distribution). The modelling process necessarily involved some simplifications of the system i.e. omission of pre-chlorination and sludge feedback, but this was seen as reasonable as the former process was not used during the study period and the quantity of water recycled was small compared to the total volume.

Campylobacter was selected as the illustrative index pathogen as available data was extensive and of a high quality. Measurements of full-scale removal of indicators and surrogates of this pathogen by several system barriers were also available.

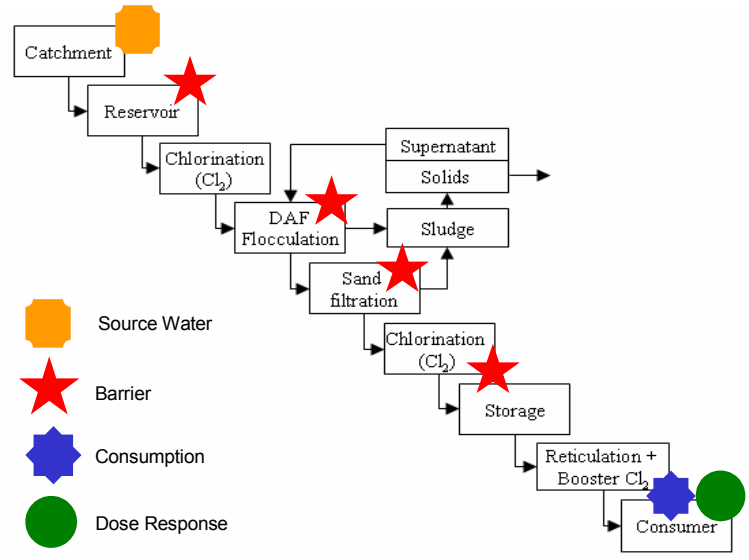

Figure 8-1. Process diagram of CTS 8

8.2.5 The Logistics of Barrier Modelling

The challenge posed initially to the MicroRisk team of modeling 12 CTSs, each with several barriers, six index pathogens and a range of Hazardous Events and other factors influencing water quality, brought home the need for strategies for implementing QMRA efficiently and addressing logistics issues such as:

1. The range of pathogens to be assessed;
2. Data management (i.e. review, collation, archiving selection, analysis, model modification and quality assurance);
3. Water managers having responsibility for tens or many more CTSs and the need for a prioritization system;

4. Baseline water quality variability;
5. Hazardous Events of variable type, duration, impact and magnitude;
6. Hazardous Events of extreme impact potential for which there is little or no data;
7. The task of comparing different risk reduction options;
8. Data gap filling and model revision in light of initial model development; and
9. The need for systematic data management to account for the above issues.

Three strategies were identified to help address these issues and the need for 'living' risk assessment systems. Firstly application of the Tier concept (see above and Chapter 7) provided a scheme for only undertaking as much risk assessment as decision making warranted. It also allowed the quality of barrier models to be assessed and data gaps and limitations to be identified.

Secondly we adapted the concept of Hazardous Events [Nadebaum *et al.* 2004] to define their converse – Baseline or 'nominal' water supply conditions when there are no notable perturbations in pathogen concentrations or their removal. This concept outlined by Teunis *et al*. [in press] provides a natural starting point for risk modelling and was incorporated into the Metamodel Design (Section 8.2.7). Data sampling strategies developed with Hazardous Events in mind could be adapted to allow modelling of other aspects of water quality and treatment variability such as that occurring between different seasons. Simulation of Hazardous Events also provided a means for exploring potential high risk situations identified via Sensitivity Analysis and the conceptual basis for quantitative setting of Critical Limits. The relationship between barriers simulation and Baselines, Sensitivity Analysis, Hazardous Events and Critical Limits are summarized in Figure 8-2. Figure 8-2 expands on key parts of the general assessment framework (Figure 7-1) to show how the latter has been applied in practice.

The third strategy pertained to programming philosophy. Creating simple barrier simulation models with or without Monte Carlo models is straightforward. It can be undertaken with a range of software and even with Microsoft Excel spreadsheets enhanced with add-ins such as Crystal-Ball [Decisioneering Inc.] or @Risk [Palisade] [Haas *et al.* 1999]. Such programs, however, become difficult to manage as the number of models, and sub-models describing each barrier step increases. This is unfortunate for two reasons. Firstly Excel has the great advantage that most engineers and scientists are sufficiently familiar with its workings to also understand the operation of the probabilistic add-in functions. Secondly because of their transparency, communicating the structure of spreadsheet models is relatively straightforward. The solution found to this was to adapt the concept of the 'Metamodel' [e.g. Schimoeller 2004] to an @Risk version 4.5 enhanced workbook program (Section 8.2.7).

The idea of a Metamodel [Harvey, 2005] is that when developing a simulation system, rather than constructing a purpose designed 'monolithic' model which runs in isolation, construct first a software framework or domain within which different modules can be inserted or replaced according to needs. The Metamodel based system provided a way of efficiently compiling large numbers of alternate source water concentration, barrier consumption and dose-response sub-models. These were then assembled into the different simulation models for each CTS, pathogen and scenario. The main benefit was that the large number of moderately complex simulation models could be easily managed within an Excel spreadsheet environment. Other benefits included ease of checking for model errors, revision and storage for future use or modification, and recording of reservations relating to data quality for future attention.

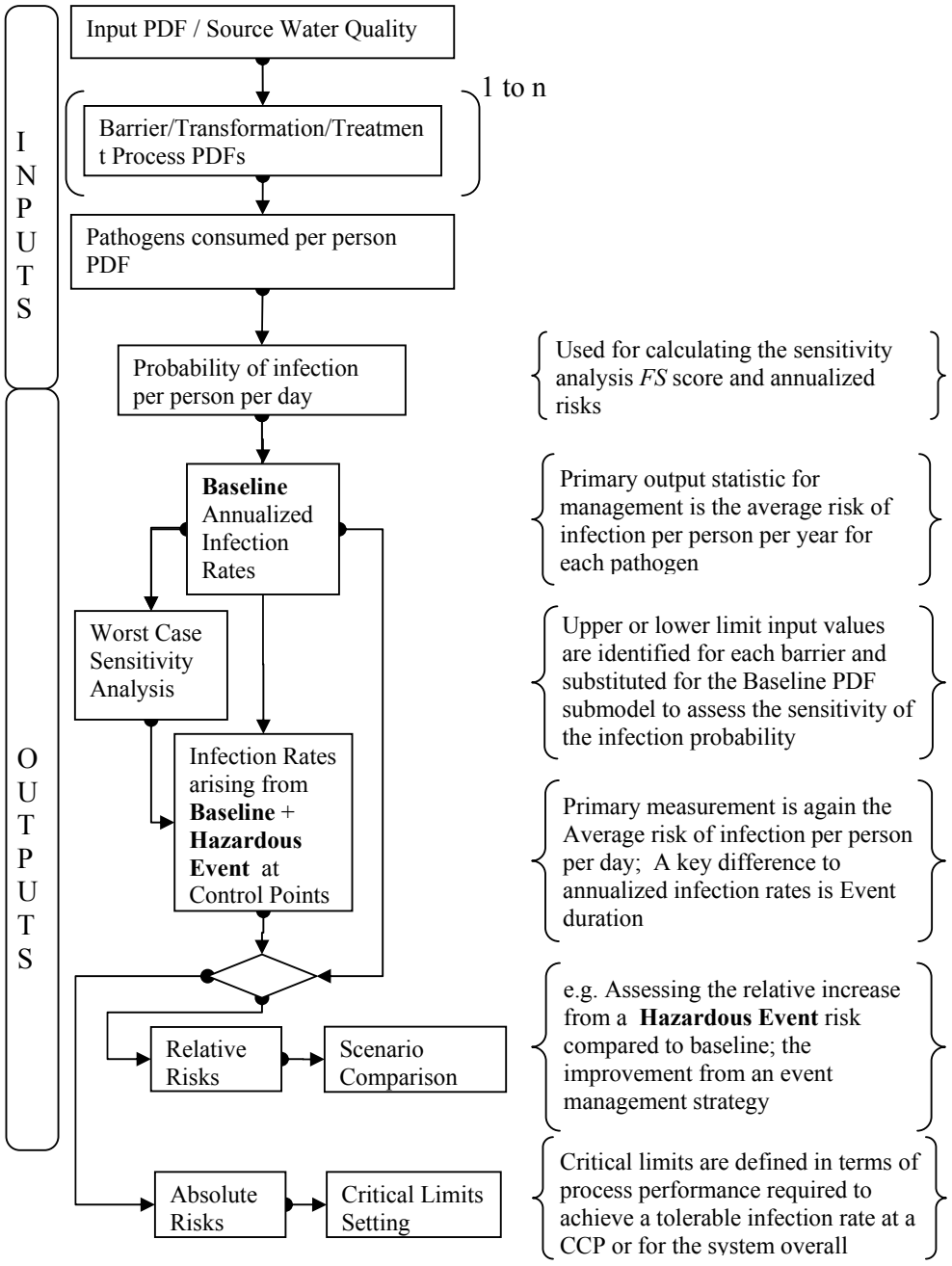

Figure 8-2. Relationship between CTS modelling and the risk estimation process

8.2.6 Simulation Model Outputs

Excel add-ins such as @Risk generate a wide range of output statistics with potential to inform a water manager/decision maker. This section describes further those statistics which were found most useful for estimating risk. Most were obtained using standard Excel routines and simple @Risk statistics functions.

Secondary features of value were @Risk *Goal Seek* Wizard which was useful for Critical Limit estimation, and the *Distribution Fitting* wizard which was useful for preliminary estimation of coefficients for PDF functions from sets of pathogen or surrogate concentration data or barrier removal data. This fitting wizard is probably most useful for Tier 1 or perhaps Tier 2 assessments. If possible, and for higher Tiers data should be fitted by the more sophisticated techniques presented in Chapter 7.

The detailed *@Risk Output* window provided access to the actual output of each model iteration. The @Risk Wizards for *Advanced Sensitivity Testing* and *Stress Analysis* were not used for work described here. This is not to suggest that water managers will not find them useful but that they were not essential to the risk assessment tasks here and in Chapter 7.

8.2.6.1 Baseline Risk Estimates

Baseline risks are exposure and risk probability estimates calculated assuming nominal operating conditions i.e. where source waters are not exposed to unusual contamination inputs and treatment processes are operating according to specifications. Simulation risks are expressed in two principle formats:

- Daily probability of infection by pathogen (prob. of infection.person^{-1}.d^{-1}); and
- Annualized infection rates (prob. of infection.person^{-1}.y^{-1}).

As the pathogen risk estimates are by their nature PDFs with variable associated distribution, the following statistics were routinely calculated for each pathogen:

- The arithmetic average of all risk simulation iterations (= Average Risk) which accounts for distribution skew;
- 95th and 99th percentiles which indicated the robustness of the Average Risk estimate; and
- The Median risk that indicated mid-range risk.

8.2.6.2 Factor Sensitivity Values

A full discussion of Factor Sensitivity and its uses is presented in Chapter 7. In this Chapter Sensitivity analyses were undertaken mainly to help indicate:

- Which stages are most critical to maintaining acceptable water quality and hence which should be most closely managed;
- How different stages and barriers compare to one another in importance and variance;
- At which stage(s) might Hazardous Events have a major impact; and
- If it was likely that there may be rare periods of much higher and/or low risk (extremes of PDFs).

Factor Sensitivity is calculated by dividing the daily risk estimate obtained when an input value is set to a credible but extreme value, by the Baseline risk and then $\log_{10}$ transforming the ratio to generate 'Factor Sensitivity' or *FS* values [Zwietering and van Gerwen, 2000] i.e. *FS* = $\log_{10}$ (average annualized infection risk arising when the baseline model (e.g. PDF for sand filtration) has been substituted with a credible extreme value, divided by the average annualized Baseline infection risk).

An indication of Factor Sensitivity can be gleaned by comparing the changes in concentrations of pathogens across different barriers. Further, the Factor Sensitivity calculation standardises the comparison parameter and thereby aids comparison between all barriers.

8.2.6.3 Additional Risk Arising from a Hazardous Event

Hazard analysis critical control point (HACCP) analysis is being promoted increasingly in the water industry (Chapter 2). Integrating QMRA into this framework is a simple way by which QMRA can be made and accessible to commercial water managers. To this end we have developed an approach for modelling Hazardous Events occurring at Control Points and proposed how Baseline and Hazardous Event modelling can be used to estimate Critical Limits needed at these control points in order not to exceed Tolerable Risk levels.

The daily risk from a Hazardous Event is calculated in the same manner as the Baseline risk estimates. Then because the durations of Hazardous Events vary greatly, a total Baseline+Hazardous Event risk is estimated over an extended period (e.g. 1 year). This is done by simulating in parallel Baseline and Event conditions and then choosing the output of one or other iteration in proportion to the proportion of time spent in each state. The impact of the Hazardous Event is then assessed by comparison of the total risk estimate with the Baseline risk estimate.

8.2.6.4 Critical Limits

Baseline and Baseline+Hazardous Event simulations allow the impact of normal operation and system failures at each Control Point to be quantified and compared to target values. This provides the basis for quantitatively defining and setting Critical Limits or 'Action Levels' a central concept in the HACCP process which addresses the need of managers for clear guidance of when a CTS is operating satisfactorily and when it needs attention. Setting of Critical Limits and associated operational (target) limits has various potential uses including:

1. Estimation of tolerable maintenance and failure periods for water treatment barriers;
2. Standard reference points against which safety factors may be develop;
3. Definition of minimum treatment efficiency under Baseline and Hazardous Event conditions which can be used to assess treatment plant function and propose methods to avoid or reduce adverse impacts;
4. Setting of regulatory legislation and guidelines;
5. Measurement of system performance (e.g. in audits); and
6. Simulation of what monitoring parameters will provide information suited to management and whether existing monitoring needs revision.

Two methods for Critical Limit setting were identified, graphing of similar scenarios with variable event duration and the use of @Risk *Goal Seek* to estimate the performance required of a barrier to achieve a particular target risk value.

8.2.6.5 Monte Carlo Probabilities as Relative and Absolute Estimates of Risk

Risk estimates derived from Monte Carlo style simulation modelling are typically of the form - number of infections.person^{-1}.time period^{-1}. In this form they appear superficially absolute. However, as discussed in Section 8.2.2, they are in fact a best estimate of likely health risks for a population based on a combination of statistical analysis of historical data and intuition rather than a perfect prediction of illness rates in the CTS of interest (Chapter 1). Accordingly the decision maker must recognize that risk estimates can be used either as relative or 'absolute' measures and considered this in their use.

Water managers are commonly faced with three basic questions where QMRA risk estimation can be useful:

- Where action has been determined necessary, what is the preferred option out of a number of choices?

- Where monitoring (new research or ongoing audit) data has been gathered, do the results indicate a need for action based on an explicit existing triggers or general principles?
- Where there are social concerns and calls to initiate water management, what advice should be given to senior management and communications departments as to whether action is warranted?

In the first instance QMRA is well suited to providing the relative assessments required as it implicitly weights the benefits and limitations of different technical options. For example it allows the effectiveness of two treatment processes to be compared or two sources of risk. It also allows the severity of different Events to be compared to one another and to the general Baseline Risk, and hence whether a given risk is likely to be of concern.

In the second and third instances, prediction of 'absolute' risks is not possible for reasons described by Dowie [2005]. What can be provided is still arguably the best estimate possible of the absolute risk probability estimate based on scientific data.

8.2.7 Model Coding

QMRA models for simulating pathogen reduction, consumption and infection were constructed using MS Excel program v. 8 [2002] enhanced with @Risk v. 4.5 [Palisade, Ithaca, NY]. As discussed earlier the program was designed using the Metamodel concept whose relevance for environmental and risk management has been discussed by Harvey [2005] in respect to hydrological modelling.

The procedure for constructing a Baseline risk simulation model was as follows:
Excel was enhanced with the @Risk 4.5 Professional Add-in;
A quantitative probabilistic model describing each simulation stage/barrier was developed and entered as a single Excel table record (i.e. one line in a worksheet table);
Specific QMRA stage/barrier models were then assembled in a 'Scenario' worksheet. This was achieved using Excel "Lookup and Reference" functions such as *Indirect()* and *Offset()* to extract each stage/barrier model in a sequence reflecting CTS structure;
The final model was run using the @Risk *Start Simulation* tool; and
Infection rate, Factor Sensitivity, and Critical Limit output data were generated using the RiskOutput() and @Risk statistics functions.

Typically each barrier/stage model comprised a) descriptive information such as algorithm source and pathogen and b) algorithms comprised of @Risk *Distribution* functions and lookup tables which together generated a probability density function which defined stage/barrier process. Different primary tables were constructed to store source water concentration, barrier performance, consumption and dose-response models. Using this system it was possible to rapidly assemble QMRA models and explore model variations e.g. by constructing and selecting alternate barrier functions, dose-response curves etc.

Calculation of the impact of Hazardous Events required an additional 'Event Scenario' module. This module was run in parallel with the Baseline Scenario module. The outputs of the two modules were then sampled in proportion to the fraction of time the CTS was in its Baseline or Hazardous Event state. Risks were then combined using the procedure summarised in Figure 8-3. The effect was to simulate infection risk PDF function distribution

similar to the Baseline but with a few high risk outlier values with potentially large influence over the aggregate average infection rates.

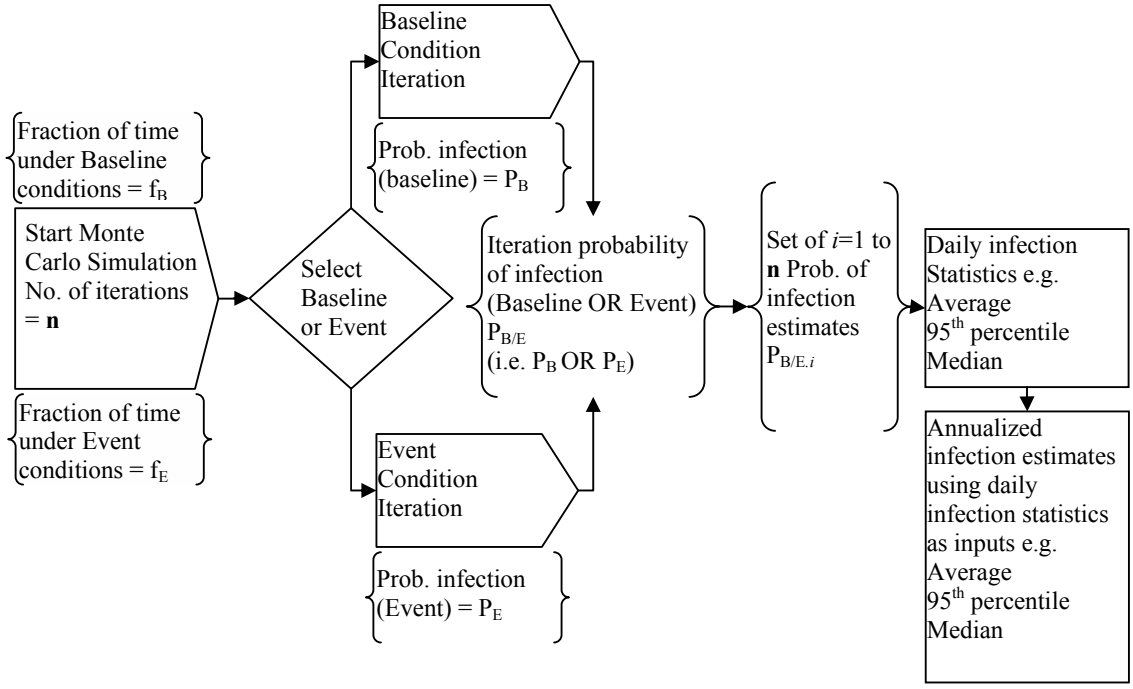

Figure 8-3. Procedure for Simulating the Risk Arising from Baseline + Hazardous Event Conditions

This sub-model library and selective extraction design features had a range of benefits compared to a series of specific task models. Reuse of a common framework allowed extensive familiarisation, design refinement and checking for errors. It was possible to rapidly recreate older models for review and updating as well as to rapidly build new barrier models by copying an existing structure and altering the model descriptors and function coefficients. When modifications were introduced, older modules could be maintained as a contingency. As a result only one moderate sized workbook was required to accommodate the full range of MicroRisk models. Also, recording of risk scenario settings for documentation and further use was straightforward.

8.3 CTS 8: AN ILLUSTRATIVE EXAMPLE

This section presents the inputs and outputs for CTS 8 to illustrate the modelling process and outcomes associated with Baseline risk estimation. It identifies and discusses key features of each barrier and shows how the data can be interpreted.

8.3.1 System Conceptualisation

Initial steps in developing a general modelling scheme for CTS 8 are covered in detail in Chapter 7. Using the information collected the critical stages and barriers in the water supply

system were defined (Table 8-1, Figure 8-1). In developing the Baseline models the following principles were employed:

1. Initially undertake the preliminary Water Safety Plan [WHO, 2004] steps;
2. Use the best available local data when defining each barrier/stage;
3. Where there are a choice of stage/barrier PDFs more conservative ones based on local data should be preferred;
4. Remove Hazardous Events data from Baseline datasets except where the data is insufficient and/or Events data too ill-defined to include in Baseline scenario;
5. Assume the CTS operates in a linear fashion, with each barrier functioning independently to treat the product of the previous barrier; and
6. Where 'Absolute' Risks are to be estimated, threshold targets values should be identified first to minimise interpretation biases.

The first practical step was to fix the starting point of the simulations, i.e. the Source Water location and its attributes. Two options were considered. Pathogen and indicator data were available for the CTS 8 reservoir (see Chapter 3) and the CTS 8 river immediately upstream of the reservoir. The river water dataset was chosen because it provided a very well defined starting point, downstream of the most impacted land, *Campylobacter* were present in significant concentrations allowing estimation of PDF coefficients under dry and wet conditions, and 10 minute hydrologic data was available allowing differentiation of high run-off event periods.

The next step was to define each barrier conceptually and quantitatively. For each barrier a probability density function (PDF) was constructed, which generated a spread of $\log_{10}$ decimal elimination capacity (DEC) factors [Hijnen *et al.* 2005] when sampled in a Monte Carlo fashion. All four barriers (the CTS 8 reservoir, the system flocculation-coagulation-dissolved air flotation (F/C/DAF) unit, the rapid sand filter (RSF) and the chlorination) were similarly definable. There was no large contact chlorination tank, however, water was stored on site for several hours providing an opportunity for chlorine mediated inactivation to taken place. Nonetheless, data on storage tank operating temperature, flow and filling cycles was available.

Consideration of the source water and barrier data showed that in general a Tier 3 level risk assessment was possible. As this would not always be the case we also undertook Tier 1 and Tier 2 simulations and these are presented for illustrative & comparison purposes. The considerations, analysis principles, programming and data management methods applied equally to Tiers 1, 2 and 3 style simulations. The main difference in the simulations was the origin and form of the input data.

Once assembled the Tier 1, 2 and 3 data were entered into the Excel sub-model library tables. Simulations were undertaken using the default @Risk settings (Latin hypercube sampling, randomly generated seed) to generate final infection risk rates. Run times ranged from *ca* 30 seconds for 1000 simulation iterations to 2 hours for 100 000 on a later model PC (1024 Gigabytes RAM, 1400 gigahertz Centrino Processor, Windows XP). Repeated simulations showed that there was little difference in the Baseline risk estimates generated by 1000 and 10 000 iterations.

8.3.1.1 Tier 1 and Tier 2 Input Data

Tier 1 and Tier 2 data were obtained from the same sources but were used in slightly different ways to compare and contrast the outputs. With the exception of the source water

concentration data all were generic and readily available. Source water quality data was taken as the *Campylobacter* concentration data collected during dry weather conditions. From this dataset the 5th percentile, mode and 95th percentile were estimated. Barrier removal for bacteria were obtained from three literature sources [LeChevallier and Au, 2004; Hijnen *et al.*, 2005; Westrell *et al.,* 2003]. Again the 5th percentiles, modes and 95th percentiles were estimated. Consumption used the default Melbourne Australia PDF recommended by Mons *et al.* [2005]. The dose-response relationship used the *Campylobacter* beta Poisson curve in Haas and Eisenberg's [2001] summary table.

8.3.1.2 Tier 3 Input Data

No measurements were made of *Campylobacter* in the reservoir because it was judged from protozoa inactivation rates that their concentration would be too low to usefully estimate with the available assay technology (sensitivity *ca* 1 bacteria .L^{-1}). Instead the long timeseries data set of *E. coli* concentrations at the water treatment plant off-take was combined with *E. coli* data for the river to estimate a reduction factor distribution function for vegetative coliform-like bacterial cells. F/C/DAF and RSF removal were estimated by measuring Total Coliforms concentrations before and after each of these processes.

Independent analytical support for the DEC probability density function soundness was obtained in the case of the reservoir, F/C/DAF and RSF barriers. In the reservoir, reduction in the much more durable *Cryptosporidium* was observed to be *ca* 1.4 orders of magnitude. The removal estimates for bacteria by the physical water treatment plant processes were consistent with the experimentally observed removal of inoculated *Saccharomyces cerevisiae* and particles in size band ranges between 1 and 20 μm. It was not possible to confirm disinfection inactivation rates, but sufficient information on flow, water temperature, chlorine concentration and storage tank operation were available to use the conservative Complete Stirred Tank Reactor (CSTR) equation proposed in Chapter 4 from estimating chemical disinfection barrier effectiveness.

Reduction in concentration within the distribution system was included in the Baseline simulations as conceptually water should have been fit to drink at the exit from the treatment plant and no further disinfection should ideally have been required. Distribution ingress was seen as better modelled as a class of Hazardous Event. Other reasons were that distribution system was not conceptually an inactivation barrier, no inactivation model was available and data on pathogen presence was very limited (see Chapter 5).

Good estimates of local consumption rates were available from Mons *et al.* [2005] who included, among their reported PDFs, functions for South Australia where CTS 8 was located. Several dose-response relationships were reviewed. That selected [Van den Brandhof *et al.* 2003] was conservative (i.e. *Campylobacter* assumed to be highly infectious) and most recent in derivation.

Table 8-1. CTS 8 Stages and Barriers Described Quantitatively

Stage/Barrier	Stage/Barrier Description	Input Data Origin (Tier 3)	How Stage Modeled in Maximum Detail (Tier 3)
Input Source Water	River water running off an intensively developed agricultural catchment. Catchment water quality and quantity modelled based on microbial quality and flow measurements taken over a total period of *ca* 3 years from the key stream monitoring station located immediately above its entry point to the CTS 8 reservoir.	Primary input information was concentrations of *Campylobacter* spp. measured during dry and wet weather during different seasons and years.	*Campylobacter* probability density functions were estimated for dry and wet weather. The proportion of time that the river was under dry or wet weather flow was calculated as a proportion of each season. The source PDF inputted into the model was designed to select values from one to the eight possible states (4 seasons X dry and wet weather flow conditions).
Barrier A	Large reservoir designed primarily to store winter rains and minimize the impact of drought.	Reduction in *Campylobacter* spp. concentration by natural processes (e.g. predation, thermal inactivation) based on the observed reduction in *E. coli* concentration between source water station located at the head of the Reservoir and the Water Treatment Plant intake.	The PDFs for of *E. coli* concentrations in the river under dry and wet weather conditions are combined with flow data to estimate the total loads entering the reservoir during each season and average initial concentration in the reservoir headwaters. This is then compared with the seasonal *E. coli* concentrations at the extraction point to estimate a seasonal reduction (DEC) factor.
Barrier B	Dissolved Air Flotation treatment stage designed to remove the majority of flocculated and coagulated particles (Alum, polymer coagulant)	Reduction in *Campylobacter* based on reductions in total coliform concentrations measured between the beginning of the F/C/DAF process mixing zone and underside of the DAF sludge layer.	Three treatment modules were monitored over the course of a full water production cycle (production is effective in batches due to Rapid Sand Filtration cycles). Reductions in total coliform concentrations were measured used to estimate the DEC function.
Barrier C	Rapid Sand Filter designed to remove particles remaining after the DAF process on a batch cycle of 10-20 hours.	Reduction in *Campylobacter* based on reductions measured in total coliform concentration above and below the sand filter	Three treatment modules were monitored over the course of a full batch treatment run. Reductions in total coliform concentrations were measured used to estimate the DEC.
Barrier D	Chlorination + on plant storage	Reduction in bacterial concentration is estimated using a combination of local and literature data and other information. Chlorine inactivation kinetics use published inactivation coefficients. Local measurements of water temperature, total production flow rate, tank volume and filling/emptying statistics. Disinfection was modelled assuming continuously stirred tank reactor conditions apply.	Key equations were: Decimal ($\log_{10}$) elimination capacity = $\log_{10}$ (1/(1+(Concentration Cl_2 * Contact time)*(1^{st} order reaction constant k')) (Cl_2 concentration * Contact time) = $[Cl_2]$*[Storage Volume]*[Fraction of storage tank full]/[Flow rate] $k' = A.e^{(-E/(R.T))}$

Stage/Barrier	Stage/Barrier Description	Input Data Origin (Tier 3)	How Stage Modeled in Maximum Detail (Tier 3)
Consumption	Daily consumption of cold tap water person	Australian state specific drinking water consumption pattern.	Consumption rate modeled as a Poisson distribution
Dose Response	Dose-response curve for *Campylobacter* which estimates infection rate.	Dose-response curve calculated from multiple sources including outbreak data.	Beta Poisson dose-response curve + Maximum risk curve (where the infection probability calculated from beta Poisson is greater than that for the Maximum Risk Curve, see Chapter 7)

8.3.2 CTS 8 Baseline Risk Estimation Based on Stage / Barrier Simulations

8.3.2.1 Tier 1

The Tier 1 simulation first used mid-range point values (Table 8-2) to estimate the risks of infection arising from consumption of drinking water. The Average Risk probability estimates were $1.0x10^{-8}$. $person^{-1}.d^{-1}$ and $3.7x10^{-6}$. $person^{-1}.y^{-1}$ corresponding to an overall reduction in *Campylobacter* concentration by a factor of $6x10^{7}$. This high level of removal and the fact that the annualised risk of infection was much less than the mooted Benchmark probability of $1.0x10^{-4}$ $.person^{-1}.y^{-1}$ [Hunter and Fewtrell 2001; Chapter 2] suggested that the water quality achieved was satisfactory.

However when more conservative 'worst case' inputs were used as is done in the case of chemical exposures (e.g. CMPHU 2005) the equivalent risk probability estimates were $6x10^{-2}$.$person^{-1}.d^{-1}$ and 1.0 $.person^{-1}.y^{-1}$. This was despite the fact that worst case values were not used for dose-response and water consumption. The difference between the mid-range and worst case scenarios highlights the uncertainty associated with point value simulations and their sensitivity to input values chosen.

Nonetheless, Tier 1 style screening still had value. If the mid-range risk estimates had been judged to be high then more detailed conservative simulations would most likely have yielded the same result and need for some action would require little further confirmation. Similarly if the worst case simulation showed risk to be low then the real risk would be likely to be very low and remedial action would not be required and further simulations would be unnecessary.

In the present instance though the risk estimates provided very different conclusions regarding risk. In this case the appropriate response to enhance the quality of the statistical assessment was to move to a Tier 2 assessment.

The annualised Average Risk values equate approximately to the commonly seen "n_1 infections per n_2 population per year". In the current instance (Average Risk probability $3.7x10^{-6}$. $person^{-1}.y^{-1}$) the equivalent mid-range value would be "0.037 infections per 10 000 persons per year". The individual infection risk probability measures (10^{power}) were used as the primary simulation output format because the meaning of the "infections per 10 000" style formats are ambiguous where the probability of multiple infections per year is significant.

Table 8-2. CTS_8 Tier 1 Mid-range and 'Worst Case' Modelling Inputs

Stage/Barrier	Simple and/or Literature Functions Describing Stage/Barrier	Mid Range Input Values	Worst Case Input Values	Rationale for Choices	Information Source
Input Source Water	Dry weather probability density function for *Campylobacter*.L^{-1} is described by the following statistics:	Median = 43	95^{th} percentile = 903	Measurements of pathogen concentration will tend to be undertaken under dry weather conditions. Ten to 20 measurements would be considered a good	Dry weather data from CTS 8

Stage/Barrier	Simple and/or Literature Functions Describing Stage/Barrier	Mid Range Input Values	Worst Case Input Values	Rationale for Choices	Information Source
				sized small sample set.	
Barrier A (Reservoir)	Decimal elimination capacity (DEC) with the following coefficients:	Mode = 1.45	Minimum = 0.70	The minimum and maximum are reductions in bacteria reported to occur in reservoirs	LeChevallier and Au [2004] Bacterial reduction in reservoirs (Chapter 3)
Barrier B (Coagulation + Flocculation + DAF)	Triangular DEC with the following coefficients:	Mode = 1.55	Minimum = 0.55	Conservative removal based on literature data on flocculation removal of bacteria.	Hijnen *et al.* [2005] collation
Barrier C (Rapid Sand Filter)	DEC with the following coefficients:	Mode = 1.29	Minimum = 0.1	Conservative removal based on literature data on rapid sand filtration removal of bacteria.	Hijnen *et al.* [2005] collation
Barrier D (Chlorination + Short term storage)	DEC with the following coefficients:	Mode = 3.5	Minimum = 2.5	Removal based on literature data on chlorination reduction of bacteria	Westrell *et al.* [2003]
Consumption[1]	PDF of litres consumed per day per person (Consumption PDF) with the following statistics: 5^{th} percentile = 0 L; Mode = 0.75 L; 95^{th} percentile = 1.5 L			Recommended as a default value in the absence of local consumption data	Mons *et al.* [2005] Poisson based on conservative data for Melbourne Australia
Dose-response[1]	$P=1-(1+(dose/896)*(2^{(1/0.145-1)}))^{-0.145}$			Widely available reference making *de facto* choice	Haas and Eisenberg [2001] Table 8.1

Note:
No special point estimates were used for consumption and dose-response as this would be unnecessary in practice because of the extent of data now available (Chapter 7).

8.3.2.2 Tier 2

Tier 2 employed simple PDFs (Table 8-3) to estimate the order of magnitude of risk. The Average Risk probability estimates $2.0x10^{-7}$.person^{-1}.d^{-1} and $7.4x10^{-5}$.person^{-1}.y^{-1}. The annualized risk of infection was considerably higher than the point value supporting the (simulated) decision to undertake a higher Tier assessment.

The Average annualised risk probability also approached the mooted Benchmark of $1.0x10^{-4}$ .person^{-1}.y^{-1} (Chapter 2) suggesting that the water quality achieved was satisfactory. Yet bearing in mind that the Tier 2 estimate is still not an optimal simulation, a conservative decision maker using the $1.0x10^{-4}$ value as a Benchmark might desire further evidence before finalising a decision on whether further management was needed. One possible response in this case would be to undertake a Tier 3 assessment based on more appropriate inputs to determine whether a similar conclusion on risk would be reached.

Table 8-3. CTS_8 Tier 2 System Modelling Inputs

Stage/Barrier	Simple and/or Literature Functions Describing Stage/Barrier	Rationale for Choice	Information Source
Input Source Water	Dry weather probability density function for *Campylobacter*.L^{-1} is described by the following statistics: 5^{th} percentile = 2 ; Median = 43; 95^{th} percentile = 903 These values were assumed to be the minimum, mode and maximum of a triangular distribution.	Measurements of pathogen concentration will tend to be undertaken under dry weather conditions. Ten to 20 measurements would be considered a good sized small sample set.	Dry weather data from CTS 8
Barrier A (Reservoir)	Uniform decimal elimination capacity (DEC) with the following coefficients: Minimum = 0.70; Maximum = 2.2; (Mode) = 1.45	The minimum and maximum are reductions in bacteria reported to occur in reservoirs	LeChevallier and Au [2004] Bacterial reduction in reservoirs (Chapter 3)
Barrier B (Coagulation + Flocculation + DAF)	Triangular DEC with the following coefficients: Minimum = 0.55; Maximum = 3.7; Mode = 1.55	Conservative removal based on literature data on flocculation removal of bacteria.	Hijnen *et al.* [2005] collation
Barrier C (Rapid Sand Filter)	Triangular DEC with the following coefficients: Minimum = 0.1; Maximum = 3.4; Mode = 1.29	Conservative removal based on literature data on rapid sand filtration removal of bacteria.	Hijnen *et al.* [2005] collation
Barrier D (Chlorination + Short term storage)	Triangular DEC with the following coefficients: Minimum = 2.5; Maximum = 5.0; Mode = 3.5	Removal based on literature data on chlorination reduction of bacteria	Westrell *et al.* [2003]
Consumption	PDF of litres consumed per day per person described by Poisson distribution with the following coefficient: Gamma = 3.37 Consumption PDF has the following statistics: 5^{th} percentile = 0 L; Mode = 0.75 L; 95^{th} percentile = 1.5 L	Recommended as a default value in the absence of local consumption data	Mons *et al.* [2005] Poisson based on conservative data for Melbourne Australia
Dose-response	$P=1-(1+(dose/896)*(2^{(1/0.145-1)}))^{-0.145}$	Widely available reference making *de facto* choice	Haas and Eisenberg [2001] Table 8.1

8.3.2.3 Tier 3

Using the complete set of available data a range of functions and coefficient values were developed to quantify the effectiveness of each barrier (Table 8-4) and produce Tier 3 risk estimate statistics (Table 8-5). Though the analysis was superficially similar to that undertaken for Tier 2, the data inputs and barrier models can be seen from Table 8-4 to be much more complex reflecting the local origin of the data and mechanistic barrier behaviour functions. The final calculation products were the daily and annualised Average Risk estimates (risk probability *Campylobacter* infection $4.8x10^{-8}$.$person^{-1}$.d^{-1}/$1.7x10^{-5}$.$person^{-1}$.y^{-1}).

A range of features can be seen in the Tier 1, 2, and 3 input and output data (Tables 8-2 to 8-4) which will be commonly encountered:

- Source water pathogen concentrations are highly variable;
- Pathogen measurement is most useful for characterising source water concentrations;

- Useful barriers need to reduce microbial concentrations by at least one or two factors of 10;
- The output data from each barrier simulation is itself a probability density function whose statistics can be extracted to provide insight into relative barrier effectiveness;
- Describing the behaviour of a barrier may require a complex algorithm or model as in the case of disinfection. The complexity required for the illustrated level of QMRA simulation is within the capacity of water managers moderately skilled in Excel;
- Dose-response curves currently available still have limitations (Chapter 7); and
- Simulations require collection and interpretation of substantial quantities of information. Accordingly good data management and documentation are essential.

8.3.3 Comparison of Simulation Tier Outputs

Side by side comparison of the outputs of the three simulation Tiers is shown in
Table 8-6 and Table 8-7. Overall the lower Tier 1 (mid-range) and two subsequent simulations yielded similar risk estimates the other Tiers, though the estimated influence of the same barrier or stage varied considerably between each simulation (compare concentrations inputted into Dose-response stage). The fact that Tier 2 and Tier 3 yielded similar risk estimate values suggests intuitively that the estimations are robust and could be used as reference points for assessing the impact of Hazardous Events or for comparison with other treatment systems assessed in a similar manner.

The issue of whether risk was very much less or greater than the Average Annualised 10^{-4} value was largely resolved. As far as it was possible to determine the average annualised risk was below this mooted Benchmark value though only by an order of magnitude. As to whether it necessitated management intervention would depend on how risk averse the exposed community and supply manager were and how much of a safety margin they desired. The latter might be based on the 95^{th} percentile.

The sequential application of Tier 1, 2 and 3 appeared to work well. The iterative decision sequence simulated (summarised in Chapter 7) addresses in principle a key question for water managers intending to do risk assessment – "How far should QMRA be undertaken on any given CTS?". The generic answer is, as far as is necessary to provide the manager with sufficient information to make an informed recommendation or decision on the need for water management consistent with their policy on what levels of risk are tolerable, the remediation options available and other decision affecting factors such as resources.

One minor difficulty encountered with the Tier classification scheme was that the best data available for any given CTS could fit into more than one Tier. In the illustrative CTS 8 Baseline simulation the river water concentration inputs were of Tier 3 quality, whereas the dose-response curve reduced to a single threshold infection fraction and was at best Tier 2. As a result the case for assigning the equivalent of Tiers on a barrier by barrier basis was investigated using the CTS 8 simulations. A composite 'Data Audit' [Hunter and Fewtrell, 2001] score was assessed for each barrier using criteria shown Appendix 1. The latter were developed with each ideal Tier assessment in mind. The resulting Data Audit scores are shown in Table 8-7. The scores were as expected marginally lower than the nominal Tier in the case of the Tier 2 and Tier 3 assessments.

Table 8-4. Stage and Barrier Input and Output Values for a Tier 3

Stage/Barrier	Functions and Coefficients Describing Stages and Barriers	Calculated Pathogen Quantity at Stage Endpoint	Data Source
Input Source Water	Dry weather concentration *Campylobacter*.L^{-1} described by a lognormal PDF with coefficients: μ (log_{10}) = 1.46; sigma (log_{10})= 1.85 Wet weather *Campylobacter*.L^{-1} concentration described by a Gamma PDF distribution with coefficients: alpha = 1.98; beta = 24.7 Proportion of time in dry weather based on flow analysis: Spring = 0.16; Summer = 0.017; Autumn =0.065; Winter = 0.329	*Campylobacter*.L^{-1} intermediate output PDF statistics describing river water were: 5^{th} percentile = 30; Average = 270; 95^{th} percentile = 855 (note these represent a combination of the 4 seasons and the dry and wet data)	Signor *et al.* [2005]
Barrier A (Reservoir)	Point estimates for seasonal decimal (log_{10}) elimination capacity (DEC's) were: Spring = 2.69; Summer = 2.46; Autumn =2.16; Winter = 2.37 These reductions were averaged to generate the reduction figure used.	*Campylobacter*.L^{-1} intermediate output PDF statistics describing water harvested at the treatment plant inlet were: Median= 0.44; Average = 1.03; 95^{th} percentile = 3.2	(South Australia Water + Signor Ph.D. data)
Barrier B (Coagulation + Flocculation + DAF)	DEC described by a normal PDF: μ = 2.38; sigma = 0.38 Decimal (i.e. log_{10}) reduction statistics are: 5^{th} percentile = 1.75; Mode = 2.38; 95^{th} percentile = 3.00	*Campylobacter*.L^{-1} intermediate output PDF statistics describing water after DAF treatment were: Median= 0.0019; Average = 0.0061; 95^{th} percentile = 0.021	(United Water + Signor Ph.D. data)
Barrier C (Rapid Sand Filter)	DEC described by a normal PDF: μ = 1.12; sigma = 0.40 Decimal (i.e. log_{10}) reduction statistics are: 5^{th} percentile = 0.46; Mode = 1.12; 95^{th} percentile = 1.78	*Campylobacter* .L^{-1} intermediate output PDF statistics describing water after Rapid Sand Filtration were: Median= $7.0x10^{-4}$; Average = $1.4x10^{-4}$; 95^{th} percentile = $2.5x10^{-3}$	(United Water + Signor Ph.D. data)
Barrier D (Chlorination + Short term storage)	Complex DEC with the following statistics: 5^{th} percentile = 3.49; Mode = 3.88; 95^{th} percentile = 6.53 Coefficients and inputs used in reduction calculation were: A = 6.31E9; E = 48699; R = 8.314; T = 273 + Temperature in ^{o}C where ^{o}C was obtained by re-sampling of a table of percentiles of water temperature entering the water treatment plant ; [Cl_2] $mg.L^{-1}$ = lognormal PDF with (μ = 3.86, sigma = 0.44, correction factor = -2.13); Fraction of storage volume = 0.1*beta function; Flow ($ML.d^{-1}$) = resample of percentile lookup table of flows into the plant.	*Campylobacter*.L^{-1} intermediate output PDF statistics describing water after chlorination were: Median= $1.3x10^{-8}$; Average = $9.9x10^{-8}$; 95^{th} percentile = $3.7x10^{-7}$ Selected secondary statistics are: ^{o}C minimum = 10.25, average = 16.5, maximum = 22.75; average [Cl_2] = 1.73 $mg.L^{-1}$; average storage volume = 7.59 ML (% fill = 76%); average flow = 25.9 $ML.d^{-1}$	(Kiwa CSTR disinfection model, and local flow, temperature chlorine and reservoir %full data)
Consumption	PDF of litres consumed per day per person described by Poisson distribution with the following coefficient: Gamma = 2.86 Consumption PDF has the following statistics:	PDF statistics describing *Campylobacter* no.s consumed. d^{-1} after full treatment were: Median= $7.1x10^{-9}$; Average = $7.2x10^{-8}$; 95^{th} percentile = $2.7x10^{-7}$	[Mons *et al.* 2005]

Stage/Barrier	Functions and Coefficients Describing Stages and Barriers	Calculated Pathogen Quantity at Stage Endpoint	Data Source
Dose-response	5^{th} percentile = 0 L; Mode = 0.75 L; 95^{th} percentile = 1.5 L Variation on beta Poisson where dose is always 0 or 1: Prob. of infection (P) = $e^{(-(alpha/(alpha + beta))*Dose)}$ Where beta = 0.011 and alpha =0.024 The Maximum likelihood curve is: P=1-$e^{(-Dose)}$	PDF statistics describing probability of *Campylobacter* infection.person^{-1}. d^{-1} after full treatment are: Median= 5.410^{-9}; Average = $4.1x10^{-8}$; 95^{th} percentile = $1.8x10^{-7}$	[Van den Brandhof *et al.*, 2003; Evans *et al.* 1996; Teunis pers. Com]

Table 8-5. Final Tier 3 CTS 8 Baseline Risk Estimate Statistics for *Campylobacter*

Statistic	**Average**	**Median**	**95th percentile**	**99th percentile**
Probability of infection (person.d^{-1})	4.8x10^{-8}	4.9x10^{-9}	2.6x10^{-7}	1.1x10^{-6}
Annualised probability of infection (person^{-1}.y^{-1})	1.7x10^{-5}	1.8x10^{-6}	6.5x10^{-5}	4.0x10^{-4}

Notes:
Statistics calculated from Monte Carlo simulation outputs.
Annual estimates calculated using equivalent daily estimate for that statistic using the equation $P_{ann} = 1-(1-P_{daily})^{365}$.

Table 8-6. Average Output of Each CTS 8 Stage/Barrier Simulated with Different Tier Data

Stage/Barrier	**Units**	**Average Output of Each Barrier Algorithm**			
		Tier 1 Mid range Case	Tier 1 Worst Case	Tier 2	Tier 3
Input Source Water	Campylobacter.L^{-1}	43	903	316	275
Barrier A (Reservoir)	Campylobacter.L^{-1}	1.5	180	17.8	1.03
Barrier B (Coagulation + Flocculation + DAF)	Campylobacter.L^{-1}	4.3x10^{2}	51	5.8x10^{-1}	6.1x10^{-3}
Barrier C (Rapid Sand Filter)	Campylobacter.L^{-1}	2.2x10^{-3}	40	4.0x10^{-2}	1.4x10^{-4}
Barrier D (Chlorination + Short term storage)	Campylobacter.L^{-1}	7.0x10^{-7}	0.13	1.5x10^{-5}	9.9x10^{-8}
Consumption	Campylobacter.d^{-1}	5.9x10^{-7}	0.096	1.1x10^{-5}	7.2x10^{-8}
Dose-response	prob. *Campylobacter* infection. person^{-1}.d^{-1}	1.0x10^{-8}	6.0x10^{-2}	2.0x10^{-7}	4.1x10^{-8}
Annualized Risk	prob. *Campylobacter* infection. person^{-1}.y^{-1}	3.7x10^{-6}	1.0	7.4x10^{-5}	1.7x10^{-5}

Table 8-7. Comparison of final Risk Statistics Estimated for CTS_8 Using the Four Alternative **Tier** Value Input Date Sets

General Tier Assessment Level	**Probability of infection.person^{-1}.d^{-1}**			**Data Audit Scores**
	Average	Median	95th percentile	
Tier 1 – Point Values (mid range)[1]	1.0x10^{-8}	9.3x10^{-9}	2.2x10^{-8}	mean=1.14, sd=0.37, minimum = 1 (n=7)
Tier 1 – Point Values (worst case)[1]	6.0x10^{-2}	6.4x10^{-2}	1.2x10^{-1}	mean=1.14, sd=0.37, minimum = 1 (n=7)
Tier 2 – Uniform or Triangular Distributions	2.0x10^{-7}	6.5x10^{-9}	7.7x10^{-7}	mean=1.85, sd=0.24, minimum = 1.5 (n=7)
Tier 3 – Best available location specific modelling Inputs	4.1x10^{-8}	4.9x10^{-9}	2.6x10^{-7}	mean=2.78, sd=0.39, minimum = 2 (n=7)

Note.
1. The slight variability in the 'point estimate' risks arises from the use of the default consumption distribution function.

8.4 EXTENSION OF THE RISK ESTIMATION PROCESS

Once a Baseline CTS model and Scenario have been developed it could be systematically expanded and altered to explore the sources and importance of the different factors contributing to pathogen risks. Three stages in this process are considered in this section using CTS 8 again as the principle example:

- Factor Sensitivity Analysis;
- Simulation of Hazardous Events; and
- Estimation of Critical Limits.

8.4.1 Sensitivity Analysis

Sensitivity Analysis was undertaken by replacing the Tier 3 PDFs with point values based on our knowledge of CTS 8. Bearing in mind the need to move away from intuited probabilities, selection or 'extreme values' posed a challenge. In the end the criteria adopted were:
95^{th} or 99^{th} percentiles of PDFs or equivalent.
Extreme values observed in the literature for similar CTS barriers or stages.
Worst long term barrier failure based on the team's expert opinion.
Default maximum values (= total barrier failure, maximum conceivable water consumption (taken as 6 Litres per day), maximum infectivity).

Comparison of the *FS* values (Table 8-8) indicated that for *Campylobacter* the most critical were the disinfection barrier followed by the reservoir. Four stages showed *FS* values > 1 $\log_{10}$ units and hence the potential for markedly increasing long term risk if they were not functioning nominally. This analysis also indicates that management of all stages upstream of consumption was important in protecting consumers, as under poor conditions all could lead to degradation of treatment by an order of magnitude or more.

These *FS* scores provided a rational order for prioritising investigations and water treatment plant upgrades. In the case of CTS 8 understanding and improving chlorination appears to be the best way to improve protection against *Campylobacter* infection in the consumer population, followed by reservoir management.

A related use of this priority list is to identify where Hazardous Events might have the most severe effects and where impact modelling and Critical Limit development would be most useful.

Table 8-8. Average Factor Sensitivity Values – Based on Sensitivity of Daily Infection Rate (persons^{-1}.d^{-1})

Stage/Barrier	**Sensitivity Value Tested**	**Average Tier 3 Baseline**	**Sensitivity Value Inputted**	**Average *FS* Value**
Input Source Water	95^{th} percentile Tier 3 Baseline *Campylobacter*.L^{-1}	270	2500	1.01
Barrier A (Reservoir)	Decimal Reduction due only to dilution arising when input flows short circuit [Hipsey *et al.* 2005]	2.42	1.0	1.48
Barrier B (Coagulation + Flocculation + DAF)	Worst Case Decimal Reduction – "expert opinion"	2.38	1.0	1.32
Barrier C (Rapid Sand Filter)	Worst Case Decimal Reduction – "expert opinion"	1.12	0.2	0.82
Barrier D (Chlorination + Short term storage)	Worst Case Decimal Reduction – "expert opinion"	3.88	1.0	2.92
Consumption	Extreme High Consumption Netherlands (litres)	0.75	6	0.90
Dose-Response	Maximum infectivity	0.6 infections per organism	$P=1-e^{(-dose)}$ (=1 infection per organism)	0.44

8.4.2 Hazardous Event Characterization

Simulation of a Hazardous Event involved the following steps:

- Quantitative definition of event characteristics;
- Compilation of appropriate algorithms in the program library;
- Creation of an Hazardous Event simulation model to run in parallel with the Baseline simulation; and
- Running sufficient iterations to capture the influence of the Hazardous Events.

From the Sensitivity Analysis of CTS 8 chlorination was identified as the most important process where the impact of a Hazardous Event should be considered. To explore the impact of a Hazardous Event the following conditions were modelled:
Chlorination failure for 0.1, 0.5, 2, 5, 20 and 365 days.
Outside of these times Baseline conditions for CTS_8 prevailed on 365 days minus each of the failure periods.

These Baseline+Hazardous Event scenarios were simulated in parallel for sufficient iterations (50 000) to ensure that a substantial number of event iterations reflecting the shorter failure periods (0.1 or 0.5 days) were included in the total risk estimation process. The assumption of failure for 365 days was also undertaken so as to be able to place the shorter term events

The statistics of the combined PDFs are shown in Table 8-9 and demonstrate the potential impact of chlorination failure and the information gained by simulating a step series of such failure modes. Effective chlorination is clearly essential to infection minimisation despite the protection afforded by the reservoir, F/C/DAF and RSF treatment stages. Some notable features were:

- A noticeable increase in Average Risk occurred for failure duration periods as short as 0.1 days. However the risks arising from such a short event were still dominated by those associated with Baseline conditions;
- This increase was mostly noticeable in the Average Risk estimate. The median risk estimates did not change appreciably. Impact was only noticeable with the 95th percentile for failure periods > 1 day; and
- For events of greater duration than 0.5 days the Average Risk exceeded the threshold value of an annualised risk probability of 10^{-4}. person.$^{-1}$.y^{-1}.

This data was compared to hourly SCADA free chlorine data collected for CTS 8 immediately after chlorination and at the exit to the treatment plant after storage but before distribution. At the point of chlorination only one measurement < 0.5 mg.L^{-1} was recorded from a total of 17 000, equivalent to a total failure period of 0.04 days. This indicated that actual failure occurred less frequently than the worst simulated aggregate Hazardous Event period and the resultant increase in risk was small enough to be tolerable.

However at the storage off-take Cl_2 < 0.5 mg.L^{-1} occurred at a rate of 13.6 days per year. This was a concern as the average annualised risk probability arising would have been of the order of 1x10^{-3} .person^{-1}. y^{-1} compared to the Baseline of 1.7x10^{-5} person^{-1}. y^{-1}. How well the loss of chlorine at the plant exit reflected reduced chlorination effect in the storage tank is unclear. Yet the Hazardous Event simulation highlights the need to investigate the efficiency of disinfection in the storage tank.

Table 8-9. Effect of Different Durations of Chlorination Failure

Risk Measurement	Statistic	Scenario						
		Baseline Conditions (100%)	Baseline (99.97%)+ vent (0.027%) (0.1 days)[1]	Baseline (99.86%)+ vent (0.13%) (0.5 days)[1]	Baseline (99.5%)+ vent (0.55%) (2 days)[3]	Baseline (98.6%)+ vent (1.4%) (5 days)[3]	Baseline (94.5%)+ vent (5.5%) (20 days)[3]	Event Conditions (100%) (365 days)
Annualized Probability (person^{-1}. y^{-1})	Average	$1.7x10^{-5(2)}$	$2.4x10^{-5}$	$1.7x10^{-4}$	$5.1x10^{-4}$	$8.8x10^{-4}$	$5.8x10^{-3}$	$1.6x10^{-1}$
	95th percentile	$6.5x10^{-5}$	$6.2x10^{-5}$	$6.6x10^{-5}$	$7.3x10^{-5}$	$1.0x10^{-4}$	$3.1x10^{-3}$	$4.8x10^{-1}$
	Median	$1.8x10^{-6}$	$1.8x10^{-6}$	$1.8x10^{-6}$	$1.8x10^{-6}$	$1.7x10^{-6}$	$2.2x10^{-6}$	$3.1x10^{-2}$
	99th percentile	$4.0x10^{-4}$	$4.3x10^{-4}$	$4.9x10^{-4}$	$2.6x10^{-3}$	$5.1x10^{-2}$	$2.0x10^{-1}$	$9.2x10^{-1}$

Notes:
Based on 50 000 iterations.
Bold entries are within one order of magnitude of the target risk, bold and underlined are more than 1 order of magnitude greater than the target risk.

8.4.3 Critical Limit Setting

QMRA provides a means for generating scientifically-based Critical Limits to manage control points and evaluating the appropriateness of existing ones against Benchmark risk levels. In this section we have considered how QMRA could address the setting of tolerable failure periods for chlorination in the CTS 8 treatment works.

The first manner discussed already in Section 8.3.2 is to treat infection risk probabilities as representing acceptable estimates of 'absolute' risk to consumers and comparing them to agreed risk 'Benchmarks' e.g. infection probability of 10^{-4} .person^{-1}.y^{-1} Benchmark [Hunter and Fewtrell 2001; Macler and Regli 1993]. In this instance comparison of the CTS 8 risk estimates (Table 8-5) indicated that the Baseline Risk was acceptable when compared to this Benchmark and this conclusion was robust as indicated by the 95th percentile (prob. infection = $6.5x10^{-5}$.person^{-1}. y^{-1}) being less than 10^{-4} .person^{-1}.y^{-1} and the Tier 2 assessment yielding a similar risk estimate.

Another way is to use relative risks to identify the better indicators of 'absolute' risk e.g. high risk index pathogens. For example the annualised risk probability estimated for *Giardia* in CTS 8 was $8.8x10^{-10}$.person^{-1}.year^{-1}. This showed that *Giardia* was of much less concern than *Campylobacter* at the treatment plant exit. Hence public health protection is better served by focusing on *Campylobacter* control at CTS 8 in limit setting, monitoring and management strategies.

Another approach is indicated from consideration of the data in Table 8-9. It can be seen that it is possible to estimate the risks arising from varying periods of chlorination loss Hazardous Events which would cause increased risks or exceed a predetermined tolerance threshold. The actual Critical Limit could be expressed in generic terms of a maximum acceptable process downtime and expressed in the following manner:

- "The chlorination failure duration period should not lead to an annualised risk > than 10^{-4} person^{-1}. y^{-1}"; or
- "The chlorination failure duration period should not lead to an annualised risk increase double the existing annualized risk".

In the case of CTS 8 the above 'Critical Limits' would correspond to process failure periods of 0.5 and 0.25 days respectively (compare Table 8-9 and Figure 8-4).

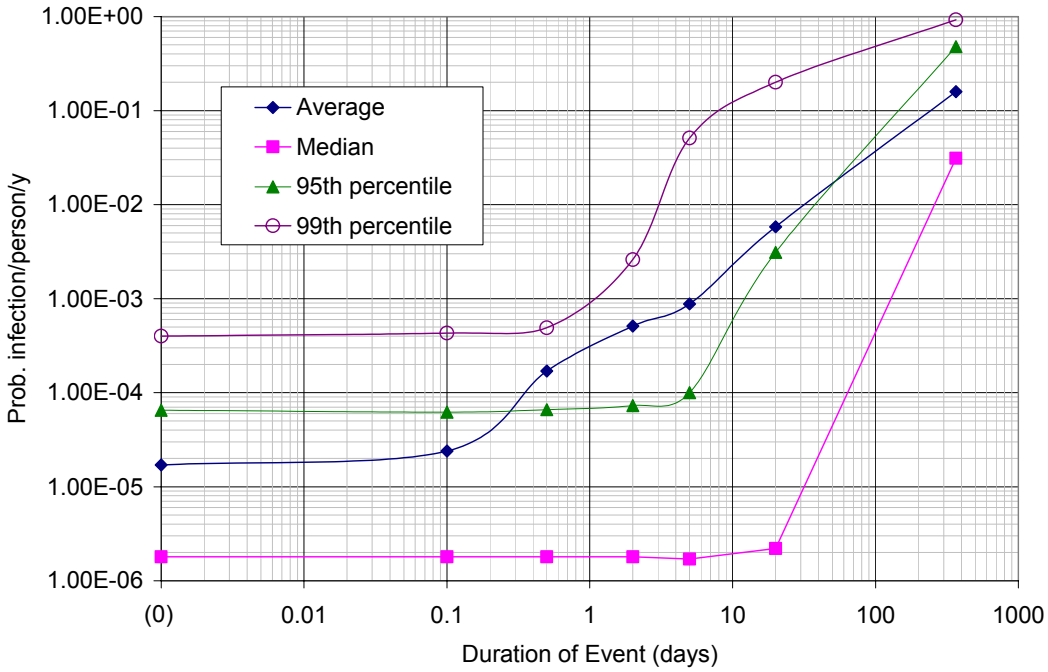

Figure 8-4. Changes in annualised infection risk with increasing simulated duration of chlorination failure

A further application of the Critical Limit concept, which may be explored through modelling, is support for treatment plant upgrade planning. To illustrate using the CTS 8 example, the current modelling inputs yielded an average annualized risk probability of $1.7x10^{-5}$ .person^{-1}.y^{-1} and the 95th percentile of $6.0x10^{-5}$ .person^{-1}.y^{-1}. An improvement target might be set such that the Baseline risk was to be decreased so as to achieve a 95th percentile risk probability for *Campylobacter* that was $1.0x10^{-5}$ .person^{-1}.y^{-1} leading to a greater margin of treatment safety for consumers than with the current arrangements.

From consideration of the current treatment plant scheme and the CSTR relationship used to estimate disinfection, one approach might be to increase chlorination effectiveness. The first step in the planning process would be to determine how much protection would be required from this barrier i.e. the minimum (Critical Limit) barrier performance. Using @Risk *Goal Seek* different possible chlorination efficiency values were trialed. It was determined that the DEC value would need to be on average 4.75 compared to the current value of 3.88. Different design configurations could then be compared with this target in mind.

8.4.4 Bacterial Indicators and the Detection of Elevated Risk

The best known Critical Limit trigger currently used at water treatment plants is the detection of *E. coli* in finished water. The question is in light of the low concentrations of pathogens required to generate significant risk, how well does bacterial indicator measurement detect significant risk of pathogen presence. It is possible to investigate this question using QMRA?

Using *Goal Seek* it was estimated that given the Baseline operating conditions at CTS 8 *Campylobacter* would pose an Average Annualised Risk probability of 10^{-4} .person^{-1}.y^{-1} if the source water contained 1050 *Campylobacter*.L^{-1}. Based on 12 dry and wet weather measurements of CTS 8 river, a median *Campylobacter* : *E. coli* ratio of 0.021 it was in turn estimated that this level of pathogen contamination would correspond to *ca* 5 000 *E. coli* 100mL^{-1}. The question arises as to whether a risk probability of 10^{-4}.person^{-1}.y^{-1} would be revealed by *E. coli* monitoring?

Assuming *E. coli* concentrations are reduced in the same manner as *Campylobacter* (i.e. Tier 3 removal assumptions) it is possible to simulate the concentrations that would be seen at the treatment plant exit. Given a starting value of 5000 cfu. 100mL^{-1} the expected median concentration would be expected to be $8x10^{-6}$ *E. coli*.L^{-1}. Even the 99.9th percentile concentration would only have been $6x10^{-4}$ *E. coli*.L^{-1}.

Further if a Hazardous Event occurred sufficient to generate an infection risk probability of $\underline{10^{-4}\text{ .person}^{-1}\text{.day}^{-1}}$ the median *E. coli* concentration encountered in the finished water would still have be *ca* $4.0x10^{-3}$ *E. coli*.L^{-1} given this median ratio applied.

These simulations show clearly that in the case of *Campylobacter* and CTS 8 not only was end point monitoring not timely, but the ability of *E. coli* monitoring to detect elevated risk from even this very similar pathogen could be very low. The more general question arising is of what use is random end point in general. These simulation suggest that if it is to continue is should be better linked to risk assessment e.g. through challenges of treatment units.

8.4.5 Distribution System Hazardous Events

As discussed in Section 8.3.1 it is seen as more appropriate to view distribution system contamination as arising from Hazardous Events. This section illustrates two model approaches to distribution system risk analysis based on the Hazardous Event concept.

From Chapter 5 it can be seen that microbial contamination of the distribution system is commonly encountered in two different forms. Firstly it is associated with clear ingress incidents which are conceptually similar to other Hazardous Events like disinfection breakdown.

Secondly there occur sporadic positive indicator samples whose cause is never determined. As observed frequencies indicator detection are less than 1 in 1000 samples on average some could conceivably be analysis ‘false positives’. It is also well recognised however, that distribution systems have significant leakage (Australian urban figure often used is 14% of total flow) there is clearly interconnectivity between distribution systems and their surroundings which could lead to ingress during transitory periods of negative pressures in pipes. Thus these sporadic positive samples must be assumed to reflect ingress even it there is some testing signal ‘noise’ from false positives unless it can be demonstrated otherwise.

Calculating the impacts of these two forms of distribution system Hazardous Event required somewhat different data analysis approaches. So we have assigned different names to the two types, ‘Acute’ meaning incident associated, and ‘Cryptic’ meaning ‘hidden’, ‘unseen’ or ‘mysteriously obscure’ for those transitory detections (single or multiple) of microbial indicators where no cause is ever identified.

8.4.5.1 Acute Distribution System Events (i.e. Incident Associated)

Based on data collected in Chapter 5 the measured probability of an incident affecting a consumer in a well maintained distribution system was 0.00154 .person^{-1}.y^{-1} or 4.2x10^{-6}. person^{-1}.day^{-1}. The latter event probability value is, however, too small to simulate using @Risk and the Monte Carlo fault tree modelling (Figure 8-3).

This is because to sample the Hazardous Event portion of Baseline+Hazardous Event PDF as few as 10 times it would still be necessary to undertake *ca* 2.3 million Monte Carlo sampling iterations in total, a task not practical with current PCs running @Risk. A 100 000 iteration sampling with @Risk requires *ca* 2 hours and calculation speed decreases markedly as memory is exhausted. As a result full simulation of distribution system events occurring at such low frequencies needed to be undertaken using more efficient software and/or faster computer types than that selected to develop the model platform.

This limitation required us to modify our modelling strategy. Instead of considering the whole supply system we instead modelled only the worst case of those supplies impacted using the following statistics:
The likelihood of an individual within one of the impacted zones being exposed to an incident was 0.00031 d^{-1} (4.2x10^{-6} .d^{-1} / 1.5% of the total population affected);
Each incident was associated with *E. coli* concentrations of *ca* 10, 3 and 0.5 organisms.L^{-1} on 3 consecutive days; and
Assuming sewage was the source of the contamination: the average ratio of *E. coli* : *Campylobacter* in sewage was *ca* 1100.

This data was used to construct a 'worst case' Baseline+Hazardous Event model where the water quality achieved by CTS 8 was simulated to be periodically (0.031% of iterations) subject to an increase in *Campylobacter* concentration of 0.01, 0.003 or 0.0005.L^{-1}. The total number of iterations used was 100 000 simulating *ca* 30 intrusion events. *Campylobacter* was seen as the most useful model as it is biologically similar to *E. coli* and has a high dose-response relationship.

The risk estimate statistics are shown as Event 1 in Table 8-10. Within the worst case zones actually affected by an acute ingress event the simulation outputs suggest that their annualized Average Risk increased by a factor of 10 and exceeded the 10^{-4} .person^{-1}.y^{-1} Benchmark probability. If these areas were in fact especially prone to ingress this would be a concern. If, however, they were merely subject to ingress events by chance, and overall had no more chance of being affected by an ingress event than the overall population surveyed, then the increase in risk would need to take into account i.e. that only 1.5% of the total population supplied was affected by ingress events. In this case the overall risk probability would be lower than this figure by a factor of about 60.

A quandary for water managers considering such risk estimates is whether to view the ingress affected sub-populations to be special high risk populations or whether they were simply unlucky. This question cannot be answered from the data presented here alone. The next step needed is to test the hypothesis that any high risk zones existed whose risk of ingress was significantly elevated. If such zones exist then it would be appropriate to rerun the simulation model with any revised statistics on the incidents per number of people to see if a risk requiring management existed.

Table 8-10. Risk estimates for simulated distribution system events

Simulation	Probability statistic person^{-1}.y^{-1}			
	Average	95th perc-entile	Median	99th perc-entile
Baseline	1.7x10^{-5}	6.5x10^{-5}	1.8x10^{-6}	4.8x10^{-4}
Baseline+Hazardous Event 1(Acute) Infiltration of contaminated water into distribution system is shown by *E. coli*. *E. coli*.L^{-1} concentration during Event described by Triangular distribution with coefficients (0.5,1,10); Ratio of *E. coli: Campylobacter* = 1000; Probability of being affected = 0.00031 person^{-1}.d^{-1}. Duration of event = 3 days.	1.7x10^{-4}	6.6x10^{-5}	1.8x10^{-6}	4.8x10^{-4}
Baseline+Hazardous Event 2(Cryptic): Infiltration of contaminated water into distribution system is shown by sporadic *E. coli* detection. *E. coli*.L^{-1} concentration at such times is 0.2 L^{-1}. Cryptic event described by point value; Ratio of *E. coli: Campylobacter* = 1000; Probability of being affected = 0.0003 person^{-1}.y^{-1}.	2.7x10^{-5}	6.5x10^{-5}	1.8x10^{-6}	4.3x10^{-4}

8.4.5.2 Cryptic Distribution System Events

Modelling of Cryptic events was more straightforward. Data from Chapter 5 indicated that the rate of detection was 1 positive sample per 3 000, and the concentration of *E. coli* in positive samples was *ca* 0.2 .L^{-1} and hence the *Campylobacter* concentrations at such times were 0.0002 L^{-1}. These indicated that a consumer would encounter water impacted by a Cryptic event with a probability of *ca* 0.00033.

This data was used to construct a **Hazardous Event** scenario whereby the treated water quality produced by CTS 8 was subject to an increase in *Campylobacter* concentration of 0.0002.L^{-1} during 0.033% of simulations. The total number of iterations modeled was 100 000 simulating *ca* 30 iterations that included intrusion. The simulation outputs showed (Event 2. Table 8-10) that there was an increase in annualized risk by 70% which could be ascribed to 'Cryptic' intrusion. As the increase did not lead to a combined **Average Risk** probability exceeding the 10^{-4} .person.y^{-1} **Benchmark**, this may be seen as tolerable. Interestingly, though the concentration of indicators was lower in the Cryptic event simulation than in the Acute events, comparison of the risk estimates suggested that Cryptic contamination is more important from a total supply system perspective.

8.5 RISKS ESTIMATES FOR OTHER CTSs AND PATHOGENS

8.5.1 Baseline Risk

Average Baseline risk estimates for CTS 1, CTS 5, CTS 6 and CTS 8 for all pathogens assessed are shown in Table 8-11. It can be seen clearly that the estimated risks can vary between both pathogens and CTSs by several orders of magnitude. The reasons for the large differences in the estimated risks, particularly between CTS 8 and CTS 1 on one hand, and CTS 5 and CTS 6 on the other, were apparent when the detailed inputs, system assumptions and barrier performance equivalent to those shown in Table 8-4 were compared.

Cryptosporidium posed the highest risk in all four systems. This result was consistent with widespread concerns regarding this pathogen and supports the belief that QMRA generates risk estimates consistent with general experience. This high risk occurred despite CTS 5 and CTS 6 having 3 and 4 barriers to pathogens respectively. The reason identified for their poor simulated performance was that the physical treatment plant removal processes (Flocculation + Sedimentation and Activated Carbon filtration) only reduced protozoa by *ca* 2 $\log_{10}$ units. The lower risks arising from water at CTS 6 were due to the additional protective effect (Median DEC = 0.88) provided by a storage reservoir located between the treatment plant intake and the river.

The differences in estimated barrier performance between CTS 6 and CTS 8 were noteworthy for related reasons. Both had river water as their primary source water. Both had a reservoir and two physical processes as their main barriers to *Cryptosporidium.* Further, barrier performances in both instances were estimated using local data. However the protective effect at each of these three barriers was *ca* 1 $\log_{10}$ unit greater in the case of the CTS 8 units than with CTS 6.

The generally low risk for CTS 1 was surprising in light of the concentrations of indicators in the source water (>10^4 *E. coli*.L^{-1}) and the nature of the source, a major river draining urban and intensive agricultural areas. The low estimated risk was largely due to there being five treatment barriers within the treatment plant, none of which was predominant and four of which were expected to reduce *Cryptosporidium.* Bacteria and viruses appeared to pose little problem for CTS 1, CTS 5 and CTS 6 because of the effectiveness of the disinfection process under nominal conditions.

The poor simulated ability of CTS 5 and CTS 6 to reduce protozoan numbers generated some discussion and disagreement within the MicroRisk team. On the one hand particle size data from the actual treatment systems was used to estimate the 2 $\log_{10}$ reduction credit. However the survey of Hijnen *et al.* [2005] suggested that 3 to 4 $\log_{10}$ reduction might have been expected.

This discussion again highlighted how risk estimates can vary significantly according to the choice of input assumptions, the need to develop agreement among CTS stakeholders on the assumptions to be used in any given risk estimation exercise and the uncertainties which may be disguised numerical process data. In the case of CTS 5 and CTS 6 this was a particularly

Table 8-11. Comparison of Baseline Risk Estimates Calculated for 4 CTSs

Measure	**CTS**	**Pathogen**					
		Crypto-sporidium	**Giardia**	**Campylo-bacter**	**E. coli O157**	**Norovirus**	**Enterovirus**
Annualised Probability of infection. person^{-1}.y^{-1}	CTS 8	$1.4x10^{-5}$	$1.8x10^{-10}$	$1.7x10^{-5}$	-[2]	-	-
	CTS 1	$7.7x10^{-6}$	$4.7x10^{-11}$	$3.7\ x10^{-11}$	$6.5x10^{-13}$	-	$5.3x10^{-11}$
	CTS 5	$9.0x10^{-2}$	$2.9x10^{-5}$	$1.7x10^{-4}$	-	$(5.8x10^{-4})^1$	$(7.8x10^{-5})^1$
	CTS 6	$1.3x10^{-3}$	$2.8x10^{-5}$	$2.5x10^{-6}$	-	$(1.7x10^{-5})^1$	$(2.2x10^{-6})^1$

Notes:
The risk estimates in brackets are based on upper 95th percentile uncertainty and are derived from upper limit inputs rather than typical source water concentrations.
E. coli O157 and O111 biotypes were both tested for but not detected in 20 composited cow faeces samples from within the catchment. Other Shiga toxin producing *E. coli* were detected but their significance is uncertain.

difficult one to resolve. The choice was between literature data based on microbial removal and high quality local particle sizing data. Because the particle size data were conservative and local (and therefore nominally of a higher Tier based on the classification applied here) they were used to generate the data in Table 8-11. One safe conclusion is that there is an urgent need for more data on factors controlling the removal of microorganisms at the physical barriers at CTS 5 and CTS 6. The best way to gain such removal data is probably to experimentally using microbial tracers to isolated treatment subunits isolated.

8.5.2 Sensitivity Analysis

In Section 8.2.6.2 a full system Sensitivity Analysis is illustrated. In the latter instance the main aim of the analysis was to identify the most important barriers to pathogens in CTS 8. There are other potential uses of Sensitivity Analysis, three of which are illustrated in Table 8-12.

Table 8-12. Further examples of the value **Sensitivity Analysis**

CTS	**Stage / Barrier ; Pathogen Considered**	**Management Issue**	**Baseline Assumption**	**Sensitivity Test Value**	**Average Baseline Annual-ised Risk .person^{-1} .y^{-1}**	**Annual-ised Risk with Test Value(s) .person^{-1} .y^{-1}**	FS
CTS_1	River Source Water; *E. coli* O157	Concentrations measured are much less than normally expected for river water. How much of a problem would poor analysis recovery pose for risk estimates.	Average is 0.081 organisms .L^{-1}. Fifth and 95th percentiles are 0.06 and 0.1 respectively	4000 organisms .L^{-1} reported in Chapter 5.	$5.0x10^{-12}$	$2.2x10^{-7}$	4.7
CTS_1	Ozonation Treatments; *Crypto-sporidium*	Ozonation, often seen as a major barrier for protozoa may need to be stopped when Bromide concentrations lead to unacceptable bromate formation	Pre-ozonation and Ozonation reduces *Crypto-sporidium* by on average 0.33 and 0.4 $\log_{10}$ units	No ozonation	$1.7x10^{-5}$	$7.2x10^{-5}$	0.62
CTS_5	Extraction of River Water; Norovirus	CTS extracts river water when there is low pollution i.e. closed intake during Hazardous Events. How sensitive is viral risk to this policy.	No viruses detected. Upper 95th percentile point value used = 0.23 pfu.L^{-1}	River water during Hazardous Event periods has mean concentration of 36 pfu.L^{-1}	$1.4x10^{-3}$ (worst case)	$1.9x10^{-1}$	>2.2

In Chapter 3 (source water chapter) one striking difference was between the concentration of *E. coli* O157 reported and that considered to occur typically in river sources. The reason for this discrepancy was not clear but it could have been due to analytical problems or real differences in the quality of the river water. One question arising was how urgently this issue needed resolving. Sensitivity Analysis using the potential concentration value of 4 000 *E. coli* O157 (Chapter 3) showed that while the potential discrepancy was very high (*FS*=4.7), the maximum annualized risk to consumers under Baseline simulations conditions was still well below a Benchmark probability value of 10^{-4}.person^{-1}.y^{-1}.

A second issue at CTS 1 was the potential for increased risk arising if ozonation was shut down. Ozonation at the treatment plant was used routinely to oxidise organic matter with disinfection of protozoa was seen as a opportune secondary benefit. However there was potential for the two ozonation barriers to be disabled in the event of excessive Bromate formation leading to reduced removal of *Cryptosporidium.* Sensitivity Analysis showed that because of the effect of other barriers, the impact on risk even over 1 year would be marginal.

The third case considered was a policy of selective extraction employed at CTS 8. Sensitivity Analysis indicated the policy had a major protective effect.

8.5.3 Hazardous Events & Critical Limits

The range of Hazardous Events which may impact on any given CTS is very large. So it was not practical to do an exhaustive set of simulations. Nonetheless a number of additional events were identified in discussions with local CTS stakeholders, from SCADA data and from Chapters 3-6. Of these five were selected for simulation to assess the diversity of information that could be gleaned by Hazardous Event analysis (Table 8-13).

In the case of the CTS 1 the local managers were concerned about the prospect of a motorway fuel spill and its potential impact on the treatment plant. It was speculated that even small quantities could foul major filters (Rapid Sand Filter and GAC) and reactors (Ozone contact tanks) and necessitate cleaning. This led us to simulate a clean-up period of 7 days during which protection was provided by chlorination and hence the system was vulnerable to *Cryptosporidium* contamination because of its resistance to chlorine. It can be seen that the annualised risk rises by a factor of 1 000 and the estimated probability of illness is much higher than 10^{-4} .person^{-1}.y^{-1}. Further, even if the repair period could be reduced to 1-2 days the additional risk would still be great and hence other action such as a boiled water alert on top of chlorination would need to be considered.

Sensitivity Analysis of CTS 5 performance highlighted water quality sensitivity to intake gate operation. Hazardous Event scenario analysis provided further information. Were no gate management in place average annualized risk would have been at least 19 times higher. The impact of a delay in closing the intake was also substantial. This highlighted the need for timely warning of event onset where source extraction is being managed.

CTS 6 included extensive diary and SCADA data detailing barrier performance (next section). This information allowed among other assessments determination of whether chlorination failure was occurring at tolerable rates. Analysis of the in line chlorine monitoring data indicated that at worst chlorine dosing failed for a total time of 1.5 hours

over a 12 month period. The impact of simulated worst case failure on *Campylobacter* showed a detectable but only small increase in the Annualized Risk probability compared to the 10^{-4}.person^{-1}.y^{-1} threshold.

The final Event scenario considered was that of multiple concurrent Events. A concern for CTS 8 and CTS 6 type systems which draw their supply from a reservoir is that during high run-off events there can be concurrent polluted input and short-circuiting [Hipsey *et al.* 2005]. Further, storms frequently cause power failures which could affect treatment plant equipment such as dosing pumps. Two scenarios were considered with these three Events in mind. Concurrent contamination of run-off and short circuiting were estimated to double the Annualized Risk probability for *Campylobacter* to 3.4x10^{-5}.person^{-1}.y^{-1}. When combined with a short duration power failure leading to chlorination loss during a storm event they could increase annualized risk 11 fold in a short time, confirming the need for avoiding or actively managing periods of concurrent Hazardous Events.

Table 8-13. Illustrative Hazardous Event Impacts on Risk

CTS	Pathogen: Stages/Barriers Altered	Simulated Event (= Variations from Baseline	Total Duration of Event Conditions	Average Baseline Risk (Annualized)	Baseline + Hazardous Event Risk (Annualized)
				.person^{-1}.y^{-1}	
CTS_1	*Cryptosporidium* / Loss of major barriers	Loss of physical barriers due petroleum spill necessitating clean-up. Only remaining treatment is chlorination.	7 days	1.4x10^{-5}	1.7x10^{-2}
CTS_5	*Norovirus*/ Evaluation of intake operation	No gate operation leading to exposure to periodic Hazardous Events	57 days	<5.8x10^{-4}	2.7x10^{-2}
		Delay in intake gate closure of 4 h for each of 29 Events per year due to time needed for rapid assay incubation	4.75 days		3.4x10^{-3}
CTS_6	*Campylobacter*/ Loss of disinfection capacity	Total suboptimal chlorination periods based on analysis of SCADA data – worst case of total loss of disinfection assumed	1.5 hours	2.5x10^{-6}	3.2x10^{-6}
CTS_8	*Campylobacter*/ Impact of concurrent catchment and treatment plant events	Short circuiting leads to reservoir creating protection factor of 10 for 24 hours through dilution (i.e. DEC = 1). Nine short circuiting events occur per year.	9 days	1.7x10^{-5}	3.4x10^{-5}
		Short circuiting leads to reservoir creating protection factor of 10 for 24 hours through dilution (i.e. DEC = 1). Nine short circuiting events occur per year. During this period chlorination loss occurs due to power failure for 2.4 hours (0.1 days).	0.1 days		1.8x10^{-4}

The risk estimates in brackets are based on upper 95th percentile uncertainty and are derived from upper limit inputs rather than typical source water concentrations.

The value of the Event analyses illustrated lies not only in the actual estimates presented. They also demonstrate how QMRA can simulate Events and other hazardous scenarios to produce risk estimates useful for management and development of Critical Limits. In the case of CTS 1 it was clear that plant shut down even for a short periods posed high risks because

of the contamination levels in the source water. Selective water intake at CTS 5 is a beneficial management activity. However risk was not reduced below the 10^{-4} probability threshold so additional management should be investigated e.g. to reduce the response period before intake closure. At CTS 6 chlorine dosing was shown to be maintained at a level sufficient to reduce risks arising from plant failure. The CTS 8 analysis showed that Baseline operating conditions provide sufficient barrier protection to mitigate two concurrent environmental risks. But three concurrent events pose a significant threat.

8.5.4 SCADA Data Analysis

To ensure that water treatment processes work properly many Water Treatment Plants are monitored in real time by online control and monitoring systems, that is Supervisory Control and Data Acquisition (SCADA) systems. On a regular basis these systems collect parameters such as flow, turbidity, pH, disinfectant residuals and temperature. Although these measures of process performance cannot be directly translated into pathogen removal, they still provide a valuable source of (event frequency/duration) information for undertaking assessments of risks. Concurrent with the analysis in this Chapter analysis of one such system was undertaken [Nilsson 2006]. This section presents the key findings of this work.

The overall objective of Nilsson's [2006] MSc project was to identify, compile, and critically evaluate the use of SCADA data sets in QMRA and the implications of its use for risk management. By analysing diary records and deviation reports in parallel with SCADA data sets, advantages and limitations to SCADA in its ability to identify frequencies, durations and magnitude of events were assessed. Ten-minute mean values for the time period 01/Oct/2004 to 19/Sep/2005 were collated for CST 6 for the following analytes judged relevant to pathogen risk assessment:

1. Turbidity in raw water, filtrate water and drinking water.
2. Chlorine residual in raw water weir and in drinking water.
3. pH in flocculation chamber one.

Interpretation of this data raised three critical challenges:

1. The task of managing and analysing in a PC environment data sets amounting to gigabytes;
2. How to recognise Events in a timeseries record – known as the 'Change Point' problem. (There is no single statistical technique available for addressing this and simple visual inspection is arguably as effective as many approaches as a first step).
3. Linking timeseries data to actual plant operation.

All three issues were sufficiently overcome to generate useful system performance statistics. Using a combination of visual assessment of the SCADA record and CUSUM analysis [Taylor, 2000] a total 119 candidate 'events' were provisionally identified. Seventy one percent were assessed as being non-hazardous whereas the other 29 % were considered being possibly hazardous based on their general characteristics and examination of concurrent treatment plant diary records. Of those considered non-hazardous, 85 % were the result of maintenance and 15 % the result of incidents. Of those considered possibly hazardous, 76 % were of unknown cause and 24 % were caused by maintenance or incidents.

The most immediate use of the timeseries data was to estimate the frequency and duration of treatment failure. Estimation of impact magnitude was more problematic but for modelling purposes total process failure could be used to assess the worst case and hence the need for further work.

The duration of most identified events ranged between 0.5 and 2.3 hours. The CUSUM was most useful for the detection of longer term trends in timeseries which were thought likely to result from early summer algae blooms (Figure 8-5) or adjustment of dosing levels (pH, Cl_2). SCADA analysis was used as the basis for estimating Hazardous Event duration in regard to disinfection (e.g. CTS 6 and CTS 8, Table 8-13).

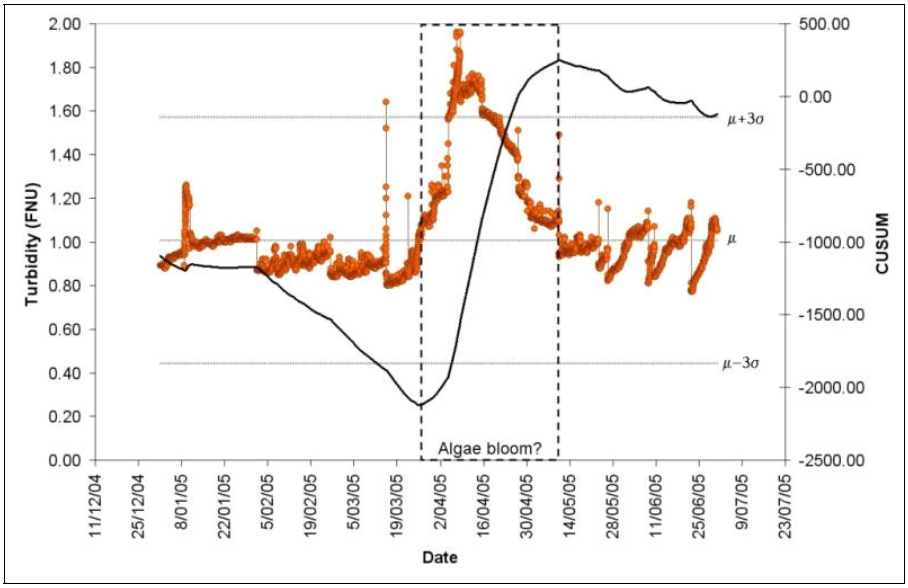

Figure 8-5. Raw Water Turbidity series showing CUSUM plot based identification of the Event period of a possible algal bloom

8.6 DISCUSSION

8.6.1 Uses and Limitations of QMRA Risk Estimates

From the CTS simulation outputs it appears that QMRA has reached the point where it can be operationally used by water utilities to produce a range of risk estimate based products which can be used to inform pathogen management. In this success and the ease with which it is possible to generate risk estimates though also lies the temptation to misuse. This section outlines the strengths and limitations of full CTS QMRA undertaken here to alert users of the need to balance its strengths and limitations.

8.6.1.1 QMRA Limitations

QMRA models and simulations are not reality but idealisations. Thus the input PDFs (source water and barriers) and output infection rates should never be seen as final fixed representations of water quality but rather best approximations which need ongoing revision and care in use and which will always have a level of associated uncertainty and variability. Accordingly newcomers to QMRA should view output risk estimates not as absolute guides to water management to be used in isolation but rather as information to be interpreted in

light of other best practice management principles. Consideration of data variability and uncertainty should be routine. Tier assessment is one possible aid for identifying the best possible inputs and highlighting uncertainties. But there will often be multiple choices available which can yield markedly different risk estimates as was encountered with CTS 5 and CTS 6.

A related need is to recognise the principle "the whole is not necessarily the sum of the parts" which in a sophisticated way QMRA simulations tacitly imply. At present QMRA is well suited to linear risk calculations and as good data is available for most barriers credible modelling is possible. However the PDFs do not as yet account all sources of variance and simple proportional decimal reductions are an empirical approximation. Current algorithms do not recognise that pathogens may exist as a number of subpopulations with differing values, such as resistance to disinfection. Simple DECs do not account for contaminant antagonisms which can affect removal. It is unclear how colloids in different waters might block pathogen binding sites in flocs and on filter media [Song *et al*. 2005]. The issue of hydraulic flow complicates the estimation of disinfection and probably the effectiveness of other processes effectiveness. Finally uncertainty arises from translating laboratory data or surrogate data to the behaviour of pathogens in full-scale systems.

8.6.1.2 QMRA Benefits and Strengths

While recognizing these limitations the strengths and potential of QMRA simulations undertaken here are also clear. For all its limitations QMRA still appears to provide the most credible quantitative synthesis of currently available data and knowledge on water treatment and risks. So its introduction into widespread use seems reasonable provided the opportunities for misuse are avoided and revision of assessments is routine.

The recognition that for any given barrier there are alternative credible filtration, coagulation and disinfection removal models highlights implicitly the uncertainties in current knowledge exists and posits hypothetical pathogen removal barrier effects which may be tested on a CTS. Water treatment is generally analysed on a barrier by barrier basis. QMRA makes possible a start on quantitative analysis of complete systems.

Easily conceived endpoint risk measurements (annualised infection rates, DALYs, Chapter 2) which address the primary concern of minimising risks to human health provide clear targets for setting Critical Limits for upstream barriers or a CTS as a whole and management action triggers. This contrasts with older coliform-based targets which did not have as clear a quantitative relationship to risk levels.

Preliminary desktop simulations form are an aid conceiving understanding water treatment systems as they forces the water manager to define the system and the way it is believed to function, its effectiveness in light of available knowledge and their current assumptions about it. The same process exposes knowledge gaps. By simple modification of models it is possible to explore the impacts of Events and prioritise them. Further it is possible to assess the impact of past, potential single and multiple Hazardous Events. It allows Hazardous Events to be differentiated from non-Hazardous Events based on their impact on infection rates.

8.6.1.3 Technology Use Principles

A feature of the above benefits of QMRA is that they arise from QMRA functioning as tool for better understanding the structure and function of CTSs. Like all tools, QMRA has its

limits and needs a guide to appropriate use. Based on experience gained during the MicroRisk project the following application principles and approaches are proposed to promote balanced use:

1. QMRA is well adapted for use as an hypothesis testing and generating tool and should be used in this fashion;
2. The existence often of competing alternate input data makes possible a broader sensitivity testing. This should be undertaken where ever warranted;
3. WHO [2003] recreational water guidelines propose a hybrid matrix for assessing microbiological risks which combines both qualitative and quantitative criteria. This model could be adapted for QMRA use where pathogen management decisions are based on other criteria as well as QMRA assessments;
4. Lack of transparency can be a frustrating feature of computer model outputs. QMRA input and output data should be well documented and transparent to aid auditing and revision;
5. Reports on water quality and barrier effectiveness should routinely include estimates of variability and uncertainty;
6. Primary data should be managed so as to promote sharing and reanalysis;
7. Protocols should be developed for periodically reviewing CTS QMRAs in light of new knowledge; and
8. Reports should be divided into two distinct sections with:
   a. The first part providing a basic interpretation of risk estimates in terms of Benchmarks, guidelines, Critical Limits, Tolerable Risks and action levels;
   b. The second part identifying caveats to the basic interpretation.

8.6.2 Water Safety Plans, Hypothesis Falsification and QMRA

Water Safety Plan [WHO, 2004] implementation as currently promoted is focused on Qualitative Risk Assessment or a screening level risk assessment. At the heart of this process is the risk assessment matrix where qualitative risk scores are assigned to hazards and Hazardous Events based on expert perception of risk severity and likelihood. This process of risk estimation and assignment is in effect an application of the first stage of scientific methodology i.e. generating hypotheses about risks based on current water science paradigms and expert opinion. That hypotheses generated in the process of developing WSPs should then be tested is also clearly supported in WSP guidelines. In addition to the emphasis on the use of statistics, there is in the HACCP process summary a clear proposal to undertake robust hypothesis testing as well i.e. “validate and verify management”, “produce and verify flow charts”, “test management actions”.

The difficulty with Qualitative Risk Assessment scores is that they are not well suited to robust statistical testing. Some non-parametric testing of the scores of each risk may in theory be undertaken but Qualitative Risk Assessment scores are necessarily very value judgment based because the scores are really numerical equivalents of “good, fair, etc. A second limitation is that while Qualitative Risk Assessment assessments can easily evaluate single hazards, how to assess the impact of concurrent multiple risks is less clear. The problem of how to consistently amalgamate qualitative assessment scores is a common problem in State of the Environment reporting. One approach is to introduce weightings. But these again are prone to value judgment bias.

Alternatively QMRA can be used to provide a measure of risk frequency and severity which is conceptually the same as that generated by the qualitative matrix system (Figure 8-6). Quantitative Microbial Risk Assessment appears to support the need for risk hypothesis falsification in a range of ways:

1. The process of infection risk calculation yields product numbers which directly incorporate scientific quantitative data on a CTS;
2. The infection risk rate calculation yields in effect a Hazardous Event likelihood X severity measurement (Figure 8-6);
3. There is no need to introduce ad-hoc weightings to account for the relative importance of different barriers, as infection rate calculation takes the magnitude and effect of different barriers into account automatically;
4. QMRA could in theory be used to test/verify Qualitative Risk Assessment derived hypotheses;
5. The use of QMRA necessitates the framing of risks identified by Qualitative Risk Assessment in as precise a mathematical format as possible with available data;
6. The selection of input data on source water pathogen levels and barrier effectiveness for new systems in effect posits hypotheses about source water pathogen concentrations and the effectiveness of barriers which can be tested experimentally;
7. Qualitative Risk Assessment has difficulty generating whole of system risk assessments which balance the significance of the different steps. QMRA fills this gap;
8. Events of the same class (e.g. high run-off) will inevitably vary in magnitude. QMRA provides a means of quantifying the impact of magnitude differences;
9. Using the data and system definition developed through QMRA can be used to explore the impact of possible rare and multiple barrier failure scenarios to see if they deserve further qualitative risk assessment or other study; and
10. The concept of Tolerable Risk defined in terms of risk of infection or equivalent appears to address the need for a consistent approach to defining Critical Limits.

The MicroRisk data and project itself also appears to provide significant assistance to WSP method development. This is because the project has generated a large range of data on CTSs which can be used for those which have not been previously the subject of risk assessment in part or whole for first cut simulations.

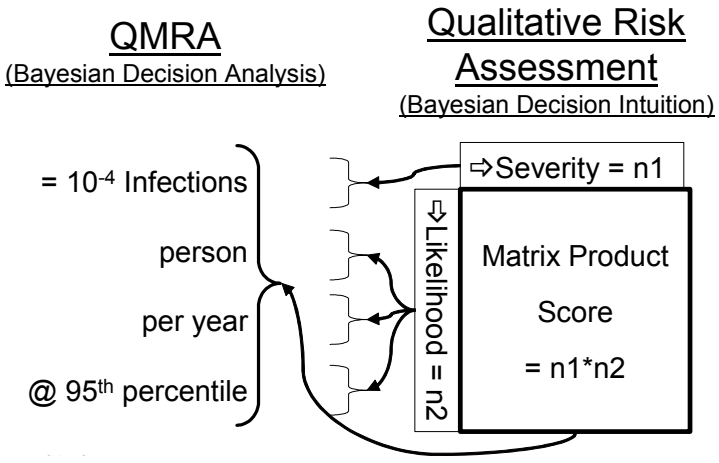

Figure 8-6. The equivalence of QMRA and Qualitative Risk Assessment Processes

8.7 CONCLUSIONS - HOW TO USE QMRA

QMRA does not replace Qualitative Risk Assessment and Water Safety planning but appears to greatly support these developments. In particular:

1. Risk estimation appears capable of replacing the less reliable numerical scoring used in qualitative risk assessment matrix completion;
2. QMRA appears to provide a means of assessing whether the treatment system as a whole is vulnerable to malfunction or failure and which individual components are most vulnerable/critical/suboptimal; and
3. QMRA can provide a rational basis for setting numerical Critical Limit targets.

Despite the inherent variability and uncertainty of input PDF and output statistics from risk simulations, QMRA methodology appears capable of providing a range of objective numerical assessments of system performance as well as uncertainty in such performance measures which can be used to improve existing water treatment management and evaluate the need for upgraded or new source water management or water treatment. Uses for such information includes: the setting of management targets, triggering of actions such as boiled water directives, identifying research priorities and estimating the effect of rare, high impact events. The following is a check list advice notes which tries to capture the messages above in simplified form:

1. Use QMRA as a tool to ask Questions and Test Hypotheses in the context of a larger Water Safety Plan.
2. Define clearly in mathematical form the barriers and inputs including surface waters and reservoirs.
3. Define first a Baseline, nominal or reference conditions noting any Hazardous Events which may be included inadvertently (e.g. periodic high run-off; seasonal variation).
4. For conceptual and actual Hazardous Events quantify not only their size but also their frequency and duration.
5. When interpreting outputs give equal weight to uncertainty and variability as is given to modal data.
6. When generating PDFs recognize the existence of a range of sophisticated mathematical tools and programs which will yield the best functions possible. Don't hesitate to work with a biometrician.
7. Water Safety Plans are likely to be developed by committees. Given the possible permutations and combinations in simulation inputs, (healthy) disagreement is likely and a practical way forward is needed. The following scheme is proposed for use when and after a set of primary simulation models are developed which involve periodic revision and gap identification:
    a. initially select a set of provisional input data as the starting Baseline;
    b. calculate provisional Baseline (or parts thereof) risks and circulate them for review and feedback(the inputs and outputs in this document) is subject to review;
    c. refined the provisional assessment to generate the first consensus Baseline CTS risk model for a CTS/pathogen and risk estimates;
    d. subject the management framework to periodic review; and alter/refine as necessary (post MicroRisk use of simulation products);
    e. generate risk estimates associated with derivative simulation products such as priority Events, and barriers. Critical Limit setting should be similarly subject to provisional development, review and modification as necessary; and

f. repeat the review process as needed e.g. in response to changes in general scientific knowledge, a local CTS review or experimental work which leads to Baseline modification.

8. In line with recreation risk guidelines it is proposed that the concept of provisional assessments be used where appropriate e.g. where a consultant develops a first cut plan for review and refinement prior to being adopted as v. 1.0 consensus model. Such a scheme would be aided by having a simulation process which is flexible, straightforward to do and interpret and hopefully simple enough to interactively explore the numerous scenario in local manager workshops. Transparency in input assumptions is likely to be essential.
9. Recognize that Hazardous Events include not only short duration shocks to CTS functioning but may be chronic such as poorly defined long term infiltration of water or cyclic such as seasonal contamination of source water from snow melt.
10. Establish a generally acceptable quantitative measure of risk (e.g. 1 infection per 10 000 population per pathogen per year at a 95% confidence level).
11. Use QMRA to answer four basic questions about a water supply system in respect to each pathogen with a view to risk minimization.
    a. Is the nominal/Baseline risk tolerable? (Baseline Scenario simulation). If yes how much safety margin is there? If no how much additional?
    b. Which barriers and sources of variability in the process appear to be most critical to maintain (Sensitivity simulation)?
    c. What is the potential impact of a hazard (at a control point)? What is the potential impact of concurrent Hazardous Events?
    d. What (Critical) Limits need to aimed for / maintained overall and individually?
12. In respect to Critical Limits some critical questions are:
    a. What performance has to be maintained v. How much short of my desired target am I?
    b. What safety factors should be applied to Hazardous Events compared to the tolerable Baseline risk?
    c. How close to the 95^{th} percentile limit is acceptable given uncertainty and Hazardous Events?

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APPENDIX 1. DATA AUDIT SCORING KEY

Simulation Stage Class	Audit Score = 1	Audit Score = 2	Audit Score = 3	Comments on each Stage Class
1. Source water concentration	Point estimate of pathogen input/source water concentration based on: local indicator data & generic ratios of indicators: pathogens for land use or PDF of pathogens from similar source water type	Source water pathogen PDF estimate based on local indicator data and local pathogen:indicator ratio data or Basic pathogen PDF (e.g. triangular or uniform) based on local summary statistics data	System specific pathogen PDF estimate with uncertainty and/or recovery estimates.	For maximum precision this should be the concentration at the entrance to the water treatment plant.
2. Reduction by pre WTP processes and WTP Physical/ chemical processes not designed explicitly for disinfection (e.g. reservoir sedimentation, riverbank filtration)	Generic literature reduction for treatment process with credible effectiveness range such that a simple PDF (e.g. uniform or triangular) can be constructed or Reduction variability based on removal algorithm function which can be used with good specific point removal estimates for the treatment system	High quality pathogen removal estimation PDF function based on: Local, credible, relevant surrogate data (particle size removal for the appropriate size band) or PDF based on compilation/collation of data for closely comparable systems [e.g. relevant subset of Hijnen *et al.* 2005]	Very high quality reduction PDF function based on: Local measurements of microbial removal using indicator microorganisms in actual system or pilot plant results or Surrogate PDFs calibrated with microbial removal such that the relationship between particle and pathogen removal is credible.	Includes organic oxidation processes which might have some impact on microbial concentrations but are not optimised for this purpose. Can include reservoir and off bank filtration.
3. Disinfection	Disinfection reduction estimate in the form of a simple PDF based on credible relevant data such as disinfection PDF for a similar water treatment plant disinfection: or Disinfection based on USEPA CT methodology	Reduction PDF that accounts for local disinfection system design and incorporates the following: Hydraulics based empirical CSTR assumption Temperature variation Disinfectant concentration as measured/expressed as a simple PDF or point value based on dosing rates. Pathogen group response (k value).	Optimal PDF that accurately accounts for all local variables including: Hydraulics (detention time and extent of mixing) Temperature variation Disinfectant concentration, availability and decomposition rate Specific pathogen response. Reliability of disinfection measurements. Variability in the coefficients. Correlation between these variables	Disinfection separated from other process. Criteria need to be expanded to include ultraviolet radiation systems
4. Distribution	See below	See below	See below	Not included currently in

Simulation Stage Class	Audit Score = 1	Audit Score = 2	Audit Score = 3	Comments on each Stage Class
				Baseline model
5. Consumption	General water consumption PDF – proposed one is conservative Melbourne Australia data	Country specific water consumption PDF	Subpopulation specific water consumption (age-group, city, region, sensitive subpopulation)	Consumption PDFs appear to have the least variation of any stage.
6. Infectivity	Dose response curve for generic bacteria, virus or protozoa similar to the organism of interest	Dose response curve for same bacteria, virus or protozoa as the organism of interest.	Dose response curve for the same bacteria, virus or protozoa as the organism of interest and: The same exposed population or Uncertainty/variability estimates for the curve's coefficients	Care needs to be taken in using some distributions to calculate infection rates where the consumption is notionally < 1 organism per person
7. Total Disease Burden	DALY for generic bacteria, virus or protozoa similar to the organism of interest	DALY for same bacteria, virus or protozoa as the organism of interest.	DALY for same bacteria, virus or protozoa as the organism of interest and population of concern.	Not included currently in model. Included for consideration

Notes:
This Audit Score assignment table is applicable only for a Baseline assessment. A system also needs to be developed also for Hazardous Events which balance the extent of the data available with the need to model extreme events for which only limited data if any is available.
Audit Score assignment for distribution systems needs to be added. It has been omitted for the moment as the assessment is for Baseline conditions where quality at the point of exit from the treatment works should be drinkable.
In assigning a rating, a score intermediate between the different optimal classes may be assigned – e.g. 2.5 for disinfection where the local temperature and disinfectant decomposition are known but where hydraulics is not well characterised. Because of the range of numerical inputs into disinfection effectiveness estimation and the varying degrees with which the mid point variability and uncertainty of this data can because of known would particularly need as expert assessment of where a system lay between the Audit Score 2 and 3 criteria.
Overall score is estimated as arithmetic mean +- standard deviation for the combined scores for all significant transformation stages.
Weighting might be considered for some stages e.g. to provide greater emphasis on the quality of the disinfection analysis because of its importance or less on consumption because the variability in consumption is much less than the other stages.