

Evaluation of the microbial risk reduction due to selective closure of the raw water intake before drinking water treatment

J. Åström, S. Petterson, O. Bergstedt, T. J. R. Pettersson and T. A. Stenström

ABSTRACT

Short-term peaks in pathogen concentrations may increase the risks for waterborne diseases considerably. In this study the occurrence of indicator organisms and pathogens in the river Göta älv at the raw water intake to Göteborg was evaluated and related to risk for drinking water consumption. About half of the 24 pathogen samples, taken during event and non-event conditions, were positive for at least one of the following: *Cryptosporidium*, *Giardia*, norovirus, enterovirus, *Campylobacter* and *E. coli* O157. Positive pathogen detects were often associated with heavy rainfalls and viruses with a sewage emergency discharge. The annualised probability of infection from this type of event was calculated from pathogen concentrations in a QMRA model. Given that the water intake is not closed, the risk given present water treatment seems to be acceptable for *Giardia*; however, it is at a borderline for *Cryptosporidium* and insufficient for noro- and enteroviruses. Present results emphasise the need for an appropriate intake regulation with respect to high pathogen loads, as the risk increases with time of exposure to pathogen contaminants. Rather than a threshold level on *E. coli*, reports on upstream microbial discharges are valuable for quick pathogen indications.

Key words | indicator organisms, infection risk, microbial barrier, pathogens, raw water, water management

J. Åström (corresponding author)
T. J. R. Pettersson
Department of Civil and Environmental
Engineering,
Water Environment Technology,
Chalmers University of Technology,
SE-412 96 Göteborg,
Sweden
Tel.: +46 31 772 2154
Fax: +46 31 772 5695
E-mail: Johan.I.astrom@chalmers.se

S. Petterson
Centre for Water and Waste Technology,
School of Civil and Environmental Engineering,
University of New South Wales,
UNSW-Sydney, NSW 2052,
Australia

O. Bergstedt
Göteborg Water,
City of Göteborg,
Box 123, SE-424 23 Angered,
Sweden

T. A. Stenström
Department of Parasitology,
Mycology, Water and Environmental Microbiology,
Swedish Institute for Infectious Disease Control,
SE-171 82 Solna,
Sweden

INTRODUCTION

Waterborne diseases caused by pathogens including bacteria, viruses and parasitic protozoa constitute a significant burden on human health in the developing part of the world and have resulted in a number of outbreaks in industrialised countries, where they also influence the endemic background level of disease. The pathogens within a watershed may originate from point sources, such as discharges of treated and untreated wastewater, and diffuse sources, such as manure runoff from agricultural land (Ferguson *et al.* 2003). Pathogen concentration (or density) in surface water varies greatly, and particular events within the catchment can result in significantly higher levels for short periods of time (Kistemann *et al.* 2002; Tyrrel & Quinton 2003).

Short-term peaks in pathogen concentration may increase disease risks considerably, and may result in outbreaks of waterborne diseases when associated with insufficient treatment at the water treatment works (WHO 2004). Furthermore, by the time microbial contamination of distributed drinking water is detected, many people may already have been exposed. Measures to decrease the pathogen in the raw water, in combination with efficient water treatment, is therefore considered as important issues in preventing waterborne diseases.

Treatment failures do occur within the water treatment plants and have resulted in severe outbreaks. In a surface water supply in Milwaukee, USA in 1993, a malfunctioning

filtration system in combination with a microbial peak event in the source resulted in the largest *Cryptosporidium* outbreak in modern times, with more than 400,000 estimated cases of watery diarrhoea (MacKenzie *et al.* 1994) and possibly 50–70 deaths (Hoxie *et al.* 1997). The outbreak of *E. coli* O157:H7 and *Campylobacter* linked to a groundwater supply in Walkerton, Canada due to inadequate chlorination resulted in 2,300 estimated cases and 7 deaths (Hrudey & Hrudey 2004). A large outbreak in Boden, Sweden, in 1988 due to non-functional chlorination resulted in 11,000 cases of illnesses from different pathogens including rotavirus (Andersson 1991). Thus, failures in the drinking water treatment represent a health risk that may result in large outbreaks as catchments may be significantly impacted by numerous sources of pathogens.

These outbreaks have occurred in spite of the general monitoring of faecal indicators. These aim to identify the presence of pathogens in water; however, their validity depended upon the *source(s)* of microorganisms and *distance* from the source(s). Faecal indicators may emanate from different sources including humans, warm-blooded animals (livestock, domestic pets and wild animals) and birds. However, microorganisms that are pathogenic to humans may be expected from only a subset of these. Moreover, the excretion of faecal indicators will exhibit less variability over time in comparison to pathogen concentrations due to prevalence. The adequacy of faecal indicators to identify pathogens is also dependent upon the distance from the source. Once outside their host and exposed to unfavourable environmental conditions, microorganisms are inactivated over time. The inactivation rate varies between organisms; faecal indicator bacteria have been shown to inactivate relatively quickly in comparison to many human pathogens (Medema *et al.* 1997; Allwood *et al.* 2003). In particular, protozoa such as *Cryptosporidium* and *Giardia*, and viruses can persist for relatively long periods of time in environmental waters (Olson *et al.* 1999; Medema & Schijven 2001). Therefore, as travel time from the source increases, the reliability of faecal indicators to identify the presence of pathogens is reduced.

In general, the pathogen concentration in treated drinking water is estimated to be low and these organisms are still complicated and time-consuming to

detect. The pathogen concentration in finished water is expected to be well below limits of detection. Therefore the consumer exposure is estimated based on quantifying source water concentration and treatment removal performance for a given train of water treatment processes (Teunis *et al.* 1998). Microbial indicator organisms in the drinking water indicate a risk; however, the detection of such organisms is not sufficient to estimate the pathogen levels. Bacterial indicator concentrations may be low suggesting safety, while persistent pathogens could still be present in potentially high concentrations (Horan 2003). Samplings for pathogens in the source water and assessing the reduction within the following treatment processes is a way forward to predict the risk from exposure to human pathogens in drinking water, as recently dealt with for a number of European drinking water treatment plants within the MicroRisk project (European Commission).

Quantitative microbial risk assessment (QMRA) is a tool that can be used to predict the risks to public health from waterborne pathogens present in drinking water systems, and models for a range of waterborne pathogens in drinking water have been developed (Gale 2003). A previous microbial risk assessment study for one of the water treatment works in Göteborg indicated that, for a number of selected etiological agents, the main impact on the annual infection risks (*Cryptosporidium parvum*, rotavirus and *Campylobacter jejuni* used as model pathogens) were likely to be due to pathogens passing treatment during normal operation (Westrell *et al.* 2003). In that study, based on literature data for the pathogen occurrence in surface water, the sensitivity analysis indicated that variability in raw water pathogen density was a major contributor to the overall risk and therefore indicated a need for site-specific pathogen sampling in general and to further evaluate the raw water supply system in Göteborg.

In this study the occurrence and exclusion of *E. coli* and pathogens in the river Göta älv at the water intake to Göteborg water treatment plants was investigated. The adequacy of an *E. coli* threshold as a determinant for pathogen-rich water was evaluated in relation to direct pathogen analyses and the implications of failure to exclude indicators/pathogens related to consumer risk of infection within a QMRA framework.

MATERIAL AND METHODS

Study area

In the city of Göteborg on the Swedish west coast about half a million people are served with drinking water produced from the river Göta älv. Water from the river intake basin is transported directly to Alelyckan water treatment plant and to a lake reservoir (Delsjön), thereafter supplying the second main water treatment plant (Lackarebäck) of the city. The microbial point sources in the river Göta älv, upstream of the intake to Göteborg, include eight municipal wastewater treatment plants and urban wastewater (e.g. combined sewer overflows) and stormwater discharges. The diffuse sources include surface runoffs with microbial loads from livestock and wild animals in the catchment area. To limit the impact of contaminated water on the water treatment plant, the intake can be closed during periods of river contamination peaks. This open and closure practice aims to provide a barrier to pathogen passage from catchment to consumer.

Peaks in pathogen concentration at the raw water intake are currently predicted by sampling and on-line monitoring of the water quality along the river, and by information about upstream events. The changes affecting microbial water quality is monitored by faecal indicator bacteria sampling and turbidity measurements at monitoring stations at the intake to Göteborg and at stations located 10, 18 and 35 km upstream. In addition, specific contamination events upstream, such as a wastewater treatment plant overflow, are, to a varying extent, reported to the water treatment plant operator at Alelyckan. At the raw water intake to Göteborg, the faecal indicator levels are compared with the former Swedish national standard for raw water quality (SLVFS 1989:30) that included guidelines for *E. coli* (500 CFU 100 mL⁻¹) and total coliforms (5,000 CFU 100 mL⁻¹). The threshold value for intake closure for *E. coli* is arbitrarily set to 400 MPN 100 mL⁻¹ to be well below the target value. The intake at the river remains closed until a subsequent water sample yields results below this threshold. During the years 2001 to 2004 the threshold of *E. coli* was exceeded in 50 samples and for total coliforms in 15 samples.

Microbial sampling

Regular monitoring

Regular monitoring during 2004 of indicator bacteria at the intake from the river Göta älv and at Alelyckan raw water included analysis for total coliforms and *E. coli* (three samples per week), intestinal enterococci (monthly) and sulfite-reducing clostridia (three samples per week). Analysis for the pathogens *Giardia* and *Cryptosporidium* were performed six times during the year at the two water treatment plants.

MicroRisk sampling program

A monitoring programme with microbial sampling at the raw water intake in the river Göta älv was undertaken over one year. The samplings and microbial analysis, performed for the period February 2004 to February 2005 are presented in Table 1. Analyses represent regular background measures (nominal) and additional sampling during microbial events. Intensive samplings were made between 21–25 October, following high rainfalls and combined sewer overflow events from the upstream river. During this period a breakage of a high pressure sewage pipe was reported 40 km upstream of the intake on 19 October. This emergency discharge resulted in an input of untreated wastewater to the river from 1,000 persons during a four-day period. Event samples were taken on 21, 22, 24 and 25 October. To ensure safe raw water after this incident, the intake was closed until 29 October.

Microbial analyses

Water samples were transported at +4°C to the laboratory for microbial analyses within 24 h. The indicators analysed included total coliforms, *E. coli*, intestinal enterococci, presumptive clostridia and somatic coliphages. Turbidity within each sample was measured for comparison. The pathogens analysed included *Giardia* and *Cryptosporidium* (oo)cysts, *E. coli* O157, *Campylobacter*, noroviruses, and enteroviruses (Table 1).

The analysis for total coliforms and *E. coli* during the regular monitoring from 2001 to 2003 was made with the membrane filtration method (ISO 2000b). Starting from

Table 1 | Sampling program in Göta älv including the faecal indicators total coliforms (Tc), *E. coli* (Ec), intestinal enterococci (Ie), presumptive clostridia (Pc) and somatic coliphages (Sc) and the pathogens *Giardia* (Gi), *Cryptosporidium* (Cr), *E. coli* O157 (Eo), *Campylobacter* (Ca), noroviruses (Nv) and enteroviruses (Ev)

| Year | Sample date and time | Site/ setting | Sample volume | Indicators analysed | Pathogens analysed ^c |
|--------------|----------------------|---------------|-----------------------|------------------------|---------------------------------|
| 2004 | 24 Feb 16:37 | Intake/open | 25 L | Tc, Ec, Ie, Pc, Sc | Gi, Cr, Eo, Ca, Nv, Ev |
| | 25 Feb 00:37 | Intake/open | 25 L | Tc, Ec, Ie, Pc, Sc | Gi, Cr, Eo, Ca, Nv, Ev |
| | 25 Feb 08:37 | Intake/open | 25 L | Tc, Ec, Ie, Pc, Sc | Gi, Cr, Eo, Ca, Nv, Ev |
| | 29 Feb 16:00 | Intake/open | 25 L | Tc, Ec, Ie, Pc, Sc | |
| | 30 Mar 00:00 | Intake/open | 25 L | Tc, Ec, Ie, Pc, Sc | |
| | 30 Mar 08:00 | Intake/open | 3 × 25 L ^a | Tc, Ec, Ie, Pc, Sc | Gi, Cr, Eo, Ca, Nv, Ev |
| | 27 Apr 08:09 | Intake/open | 25 L ^b | Tc, Ec, Ie, Pc, Sc | Gi, Cr, Eo, Ca, Nv, Ev |
| | 25 May 08:02 | Intake/open | 25 L ^b | Tc, Ec, Ie, Pc, Sc | Gi, Cr, Eo, Ca, Nv, Ev |
| | 07 Jun 20:08 | Intake/open | 25 L | Tc, Ec, Ie, Pc, Sc | |
| | 08 Jun 04:08 | Intake/open | 25 L | Tc, Ec, Ie, Pc, Sc | |
| | 08 Jun 12:08 | Intake/open | 3x25 L ^a | Tc, Ec, Ie, Pc, Sc | Eo, Ca, Nv, Ev |
| | 29 Jun 11:10 | Intake/open | 25 L ^b | Ie, Pc, Sc | Gi, Cr, Eo, Ca, Nv, Ev |
| | 28 Jul 10:20 | Intake/open | 25 L ^b | Tc, Ec, Ie, Pc, Sc | Gi, Cr, Eo, Ca, Nv, Ev |
| | 31 Aug 16:30 | Intake/closed | 25 L | Tc, Ec, Ie, Pc, Sc | Gi, Cr, Eo, Ca, Nv, Ev |
| | 01 Sep 00:30 | Intake/closed | 25 L | Tc, Ec, Ie, Pc, Sc | Gi, Cr, Eo, Ca, Nv, Ev |
| | 01 Sep 08:30 | Intake/closed | 25 L | Tc, Ec, Ie, Pc, Sc | Gi, Cr, Eo, Ca, Nv, Ev |
| | 06 Oct 08:05 | Intake/open | 25 L ^b | Tc, Ec, Ie, Pc, Sc | Gi, Cr, Eo, Ca, Nv, Ev |
| | 21 Oct 10:40 | Intake/closed | 25 L | Tc, Ec, Ie | Gi, Cr, Eo, Ca, Nv, Ev |
| | 21 Oct 15:42 | Intake/closed | 25 L | Tc, Ec, Ie, Pc, Sc | Gi, Cr, Eo, Ca, Nv, Ev |
| | 21 Oct 23:42 | Intake/closed | 25 L | Tc, Ec, Ie, Pc, Sc | Gi, Cr, Eo, Ca, Nv, Ev |
| | 22 Oct 07:42 | Intake/closed | 25 L | Tc, Ec, Ie, Pc, Sc | Gi, Cr, Eo, Ca, Nv, Ev |
| | 24 Oct 15:21 | Intake/closed | 25 L | Tc, Ec, Ie, Pc, Sc | Gi, Cr, Eo, Ca, Nv, Ev |
| | 25 Oct 00:21 | Intake/closed | 25 L | Tc, Ec, Ie, Pc, Sc | Gi, Cr, Eo, Ca, Nv, Ev |
| 25 Oct 08:21 | Intake/closed | 25 L | Tc, Ec, Ie, Pc, Sc | Gi, Cr, Eo, Ca, Nv, Ev | |
| 01 Dec 08:02 | Intake/open | 25 L | Tc, Ec, Ie, Pc, Sc | Gi, Cr, Eo, Ca | |
| 2005 | 20 Feb 19:15 | Intake/closed | 25 L | Tc, Ec, Ie, Pc, Sc | Gi, Cr, Eo, Ca |
| | 21 Feb 03:15 | Intake/closed | 25 L | Tc, Ec, Ie, Pc, Sc | Gi, Cr, Eo, Ca |
| | 21 Feb 11:15 | Intake/closed | 25 L | Tc, Ec, Ie, Pc, Sc | Gi, Cr, Eo, Ca |

^a Samples pooled prior to the analyses.^b Samples concentrated by haemoflow filtration for the pathogen analyses.^c Samples concentrated by membrane filtration for the pathogen analyses. Analyses for pathogenic bacteria were undertaken in parallel directly on the water sample.

2004 and in the MicroRisk sampling programme, the Colilert™ 18 technique was used giving the results as most probable numbers (MPN). The analyses of intestinal enterococci were performed using the membrane filtration method (mEnterococcus agar, Difco, 44 h, 35°C) according to the standard method (ISO 2000a). For the analysis of sulfite-reducing clostridia in regular monitoring, the samples were analysed using the pour plate method (Perfringens agar, Difco, 44 h, 37°C). During the sampling programme in contrast, the samples were heated (70°C, 20 min) prior to the incubation, to give the numbers of presumptive clostridia according to the standard method (ISO 1986). Somatic coliphages were analysed by plaque assay (37°C, 21 h) using *E. coli* 13706 as the host strain (ISO 2000c).

Analyses for pathogens were performed during the sampling programme and for the parasites *Giardia* and *Cryptosporidium* in addition at the water treatment plant during regular monitoring. Analyses for the separate microorganisms were done either directly on the water sample or after concentration using hemoflow or membrane filtration (Table 1). In the analysis of *E. coli* O157, the samples were concentrated and pre-enriched (buffered peptone water, 18 h, 37°C) followed by incubation with Dynabeads coated with antibodies (Dynal O157) and immunomagnetic separation from other particles. Detection by PCR was undertaken (stx1 och stx2 and eae) on the beads and on colonies with morphology as O157 after growth on SMAC and CT-SMAC (18 h, 37°C). *Campylobacter* were analysed on Modified CCDA-Preston agar (Oxoid, 44 h, 42°C microaerophilic). The analyses for noro- and enteroviruses included concentration on positively charged filters (Zetapore; Cuno) and by gradient centrifugation (Centricon) and measurement on PCR (nested PCR for enteroviruses) as described elsewhere (Gilgen *et al.* 1997; Ottoson *et al.* 2006). For the analyses of *Giardia* and *Cryptosporidium* the samples were concentrated by membrane filtration and (oo)cysts separated from other particles by immunomagnetic separation (G/C Combo; Dynal) followed by enumeration with direct microscopy after staining (mAb) with fluorescein isothiocyanate, FITC supplier (USEPA 2001). In the methods used for the pathogen analysis, a 50% recovery was estimated to calculate the final concentrations.

Retrospective *E. coli* dataset evaluation

The efficiency of the open/close strategy in eliminating *E. coli* concentrations in excess of the design threshold of 400 MPN 100 mL⁻¹ was assessed through a compiled dataset of reported *E. coli* concentrations in samples from the raw water intake and the inlet to the water treatment plant. The overall concentrations, before and after the barrier, were compared and the frequency of *E. coli* samples exceeding the threshold at the river intake summarised. The retrospective dataset comprised *E. coli* results from 829 samples collected at the intake from the river and 751 samples collected at the inlet to Alelyckan water treatment plant, between January 2001 and March 2005.

In order to illustrate the overall change in the distribution of *E. coli* concentration as a result of the intake closure, the probability density function of *E. coli* concentration in the river and at the inlet to the water treatment plant was estimated by fitting a Gamma distribution to the reported *E. coli* concentrations using the method of maximum likelihood (Montgomery & Runger 2003). The number of samples in the river and at the water treatment plant in exceedence of the threshold 400 MPN 100 mL⁻¹ was identified. The data with membrane filtration (ISO 2000b) and results from the MPN method (Colilert™) was combined, as previous site-specific data from these two methods have provided comparable results (Braathén *et al.* 2005).

Evaluation of MicroRisk dataset

The adequacy of *E. coli* for indicating the presence of human pathogens in the Göta älv was investigated by undertaking two data comparisons. Firstly, during the known four-day human sewage contamination event (sampled 21–25 October 2004) the concentration of microbial indicators at the raw water intake were compared with the presence or absence of human pathogens in the same sample. Secondly, for the entire sampling programme, the concentration of microbial indicators was tabulated for any sample that was positive for at least one human pathogen.

QMRA

The implications of closure efficiency on consumer infection risk were investigated within a QMRA model. This

model involves estimating the exposure of the consumer to human pathogens (i.e. number of organisms consumed), and then the likely consequence (probability of infection) of that exposure.

Source water concentration

For noroviruses, enteroviruses, *Cryptosporidium* and *Giardia*, the analytical results from the sewage discharge event in October 2004 were used to estimate an event mean pathogen concentration. Since no *Campylobacter* were detected during this event, an estimate of peak *Campylobacter* concentration in the Göta älv was based on the one positive result (1 December 2004).

Data analysis

All pathogens were assumed to be Poisson distributed (well-mixed) within the river over the course of the event. While this is a simplification, it is not unrealistic given that the contamination sources gave a relatively constant input (broken sewer pipe), and the flow in the river was high (during 21–25 October in the range of 180–650 m³ s⁻¹), implying rapid mixing. *Cryptosporidium* and *Giardia* counts¹ and presence/absence results² for noroviruses, enteroviruses, and *Campylobacter* were used to estimate the Poisson parameter (μ) for each pathogen by the method of maximum likelihood. The upper 95 credible limit for the value of μ , given the data, was estimated using Markov Chain Monte Carlo simulation. These 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). The approach is well suited for assessing uncertainty associated with model parameters fitted to small microbial datasets (Teunis et al. 1998). These event pathogen concentrations would, according to the system set-up, enter the Alelyckan water treatment plant if the intake from the Göta älv was not closed.

1 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 $P(n|V) = \frac{(\mu V)^n e^{-\mu V}}{n!}$.

2 Assuming the above Poisson distribution of organisms, the probability of obtaining a negative result is given by: $P(0|V) = e^{-\mu V}$. Hence the probability of a positive result is given by: $P(n \geq 1|V) = 1 - e^{-\mu V}$.

Water treatment plant removal

The treatment train at Alelyckan water treatment plant, in consecutive order, consists of conventional chemical treatment (flocculation, sedimentation), disinfection with chlorine, filtration by Granular Activated Carbon (GAC), followed by disinfection with chlorine and chlorine dioxide in combination. The influence of variation in process design and site specific water quality on estimated pathogen removal rates is unknown. Therefore, the risk to the consumer was estimated for the range of treatment removal performances from 1 to 8 total log-reduction. The results of these analyses can therefore provide a tool for identifying what level of treatment would be necessary to protect consumer health, given the inlet raw water pathogen concentrations. A treatment removal performance was estimated from literature data and local pilot plant data on pathogen surrogate organisms.

Consumption

Data on drinking water consumption in Sweden was taken from Westrell et al. (2006). Daily consumption (in mL) was best fit by a lognormal distribution ($\mu = 6.61$, $\sigma = 0.57$).

Dose-response functions

Published dose-response functions from the literature were applied in the risk model to estimate the probability of infection as listed in Table 2.

For all beta-Poisson models, low doses were approximated using the exponential model with

$$r = \frac{\alpha}{\alpha + \beta}$$

This is the expected value of the beta distribution. The beta-Poisson approximation is given by the equation

$$P_{\text{inf}} \approx 1 - \left(1 + \frac{\mu}{\beta}\right)^{-\alpha}$$

which holds when $\beta \geq 1$ and $\alpha \leq \beta$ (Furumoto & Mickey 1967).

Table 2 | Dose-response functions applied in the risk model to estimate probability of infection from drinking water consumption

| Reference pathogen | Study organism | Model | Parameters | Original data source | Analysis source |
|------------------------|--|----------------------------|---------------------------|-----------------------------|-----------------------------|
| <i>Cryptosporidium</i> | Combined dataset of three isolates collected from neonatal calves. | Beta-Poisson | $\alpha = 0.115$ | Teunis <i>et al.</i> (2002) | Teunis <i>et al.</i> (2002) |
| <i>Giardia</i> | <i>Giardia lamblia</i> Human feeding trial | Exponential | $r = 1.99 \times 10^{-2}$ | Rendtorff (1954) | Teunis <i>et al.</i> (1996) |
| Norovirus | Rotavirus strain C/JN (clinical isolate, not passed prior to administration) Human feeding trial | Beta-Poisson | $\alpha = 0.167$ | Ward <i>et al.</i> (1986) | Teunis & Havelaar (2000) |
| Enterovirus | Echovirus 12 clinical isolate Human feeding trial | Beta-Poisson approximation | $\alpha = 0.401$ | Schiff <i>et al.</i> (1984) | Teunis <i>et al.</i> (1996) |

Risk characterisation

The risk model was simulated to estimate the probability of infection for the consumer when peak pathogen concentrations are allowed to intrude to the treatment plant intakes. The treatment performance required in order to achieve the level of 1 infection per 10,000 persons (10^{-4}), a benchmark accepted by the USEPA, was assessed in the evaluations. The annualised probability of one or more infections (P_{ann}) was calculated using the following equation:

$$P_{ann} = 1 - ((1 - P_{inf(nominal)})^{t(nominal)}(1 - P_{inf(event)})^{t(event)})$$

where the $P_{inf(nominal)}$ is the daily probability of infection under nominal conditions that is assumed to dictate $t(nominal)$ days during the year and $P_{inf(event)}$ is the daily probability of infection under event conditions assumed to dictate the remaining $t(event)$ days during the year. The $P_{inf(nominal)}$ was calculated using a nominal pathogen input concentration, while all other model assumptions remained unchanged. The nominal pathogen concentration was calculated for *Cryptosporidium* and *Giardia* based on regular samplings in raw water entering Alelyckan during 2003 and 2004 ($n = 12$) but corresponding samplings were not performed for pathogenic viruses or bacteria. Relying on the assumption that the intake regulations efficiently reduce pathogen peaks, sampling results for these parasites taken at Alelyckan were used as representative for non-event periods. These results were used to estimate the parameters of a negative binomial distribution for describing nominal (background) concentration of parasites (Teunis *et al.* 1998).

RESULTS

The overall reduction in *E. coli* concentration as a result of the operation of the intake closure barrier is illustrated in Figure 1. The difference between the PDFs demonstrates the reduction in frequency of high *E. coli* concentrations following the intake barrier. 65 of the 829 river samples (7.8%) exceeded the threshold, in comparison to 8 of the 729 samples (1.1%) from the treatment plant intake. These exceedences demonstrate events when the operation procedure failed to eliminate water deemed to be of

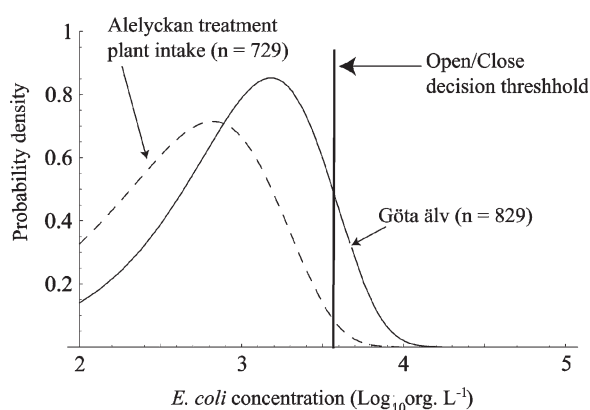


Figure 1 | Gamma distributed PDFs for *E. coli* concentration fitted to data from the river source (Göta älv) and the treatment plant intake. The decision threshold represents the level 400 MPN 100 mL⁻¹.

unacceptable quality according to the protocol, or there was a raw water shortage.

In the analysis for pathogens during the sampling programme, half of the samples (12 out of 24 samples) were negative for all pathogens. The indicator concentrations for every sample positive for at least one human pathogen are summarised in Table 3. The first three samples, positive for *Giardia* or *Cryptosporidium*, were taken during nominal conditions when the intake was open. In these samples the microbial indicator concentrations were relatively low and *E. coli* was well below the threshold. The next two samples were positive for *Cryptosporidium* and *Giardia* (samples 4 and 5). In connection to these samples, elevated levels of *E. coli* and total coliforms (8200 and 100,000 MPN L⁻¹) were observed in samples at the station 35 km upstream the intake to Göteborg.

Only one of the samples was positive for *Campylobacter* (from 1 December) but other tested pathogens were absent in this sample. None of the samples were positive for *E. coli* O157. Pathogen detections were in several instances associated with high rainfall, either on the same day or accumulated during four days prior to the sampling (Table 3).

In Figure 2 the reported concentration of microbial indicators sampled after the sewer breakage in a municipality 40 km upstream is illustrated. As seen in the figure, human pathogens are present in the river throughout the event. Notably, persistent protozoa and viruses were present; however, the less persistent bacterial pathogens were not identified. At the start of the event, the *E. coli* concentration was observed below the threshold and did

not exceed the threshold until 24 October. Without the aid of direct reporting, this incident would not have been identified by the routine monitoring at the intake until 24 October when the *E. coli* concentration exceeded 400 MPN 100 mL⁻¹ (5700 MPN L⁻¹). By this time, the duration of event conditions reaching the treatment plant intake would have been at least 3 d (positive pathogen samples on 21, 22 and 23 October) after the discharges on 19 October.

Pathogen event concentrations

The estimated pathogen concentrations in river water sampled at the intake during the event from 21–25 October are summarised in Table 4. The estimated concentrations for entero- and noroviruses were several orders of magnitude higher than for the parasites and the uncertainty in the predicted concentrations was indicated by the high credible limits. Only one sample was positive for *Campylobacter* and the concentration estimate given in the table is based on only that one positive result.

QMRA results

The probability of infections calculated using the QMRA model with the MLE values of the event pathogen concentration (samples 6–12, Table 4) are shown in Table 5. These results describe a situation where the intake is open and the pathogens, originating from a sewer emergency discharge upstream, are allowed to penetrate the drinking water treatment. Such a situation may occur either as a result of an unregistered high pathogen level or in the case of raw water shortage, e.g. the reservoir (the lake Delsjön) is not able to provide with raw water. According to Table 5, the highest risks may result from the noroviruses followed by enteroviruses. Viruses were present in high concentrations during the event studied and are modelled as highly infectious agents through the assumed dose–response models. Event concentrations of *Cryptosporidium* and *Giardia* were lower, leading to lower calculated daily probability of infection.

The annualised probabilities of infection results from simulation of the QMRA model are illustrated in Figures 3 and 4. The figures demonstrate the annual probability of infection (y axis) with the duration of event conditions

Table 3 | Microbial samples positive for pathogens at the river intake during 2004. None of the three samples from 2005 were positive for the analysed pathogens. ND = not determined

| Sample and date | Inlet Status (O = open; C = Closed) | Reason for closure ^b | <i>Cryptosporidium</i> | <i>Giardia</i> | Noroviruses | Enteroviruses | <i>Campylobacter</i> | <i>E. coli</i> O157:H7 | <i>E. coli</i> (MPN L ⁻¹) ^a | Intestinal enterococci (CFU L ⁻¹) | Coliphage (PFU L ⁻¹) | Presumptive clostridia (CFU L ⁻¹) | Rain falls (mm) ^c | Accum. rainfalls (mm) ^d | Turbidity (FNU) |
|------------------|---|---------------------------------------|------------------------|----------------|-------------|---------------|----------------------|---------------------------|---|---|-------------------------------------|---|------------------------------------|--|--------------------|
| 1. 25 Feb 0:37 | O | | √ | □ | □ | □ | □ | □ | 1100 | 380 | 59 | 80 | 0 | 23.0 | 5.2 |
| 2. 30 Mar 8:00 | O | | √ | □ | □ | □ | □ | □ | 410 | 160 | 62 | 60 | 0 | 0 | 4.5 |
| 3. 28 Jul 10:20 | O | | | √ | □ | □ | □ | □ | 410 | 80 | 50 | 100 | 0 | 3.4 | 5.7 |
| 4. 31 Aug 16:30 | C | A | √ | □ | □ | □ | □ | □ | 1400 | 1200 | 270 | 210 | 2.4 | 35 | 6.9 |
| 5. 1 Sep 0:30 | C | A | □ | √ | □ | □ | □ | □ | 2200 | 1800 | 730 | 330 | 0.4 | 31 | 7.4 |
| 6. 21 Oct 10:40 | C | B | □ | √ | □ | □ | □ | □ | 2300 | 1100 | ND | ND | 14 | 20 | 13 |
| 7. 21 Oct 15:42 | C | B | √ | √ | √ | □ | □ | □ | 2500 | 1100 | 620 | 230 | 14 | 20 | 12 |
| 8. 21 Oct 23:42 | C | B | □ | √ | □ | □ | □ | □ | 1900 | 500 | 340 | 260 | 14 | 20 | 13 |
| 9. 22 Oct 7:42 | C | B | √ | □ | □ | √ | □ | □ | 1600 | 600 | 500 | 210 | 33 | 19 | 12 |
| 10. 24 Oct 16:21 | C | B | √ | √ | √ | √ | □ | □ | 5700 | 3800 | 1700 | 500 | 0 | 50 | 28 |
| 11. 25 Oct 0:21 | C | B | √ | □ | √ | √ | □ | □ | 7500 | 3700 | 2000 | 500 | 15 | 50 | 28 |
| 12. 25 Oct 8:21 | C | B | √ | □ | □ | □ | □ | □ | 8300 | 4300 | 2000 | 500 | 15 | 50 | 26 |
| 13. 1 Dec 8:02 | C | C | □ | □ | □ | □ | √ | □ | 2000 | 1000 | ND | 150 | 0 | 1.9 | 6.4 |

^a Samples with *E. coli* concentrations above the 400 MPN mL⁻¹ threshold are marked in bold.

^b Events included bacteria registrations 35 km upstream (A), report on sewer emergency discharge upstream (B) and unspecified contamination (C).

^c Precipitation data was collected 35 km upstream of the intake. ^d Accumulated precipitation at 4 to 1 days prior to the sample day.

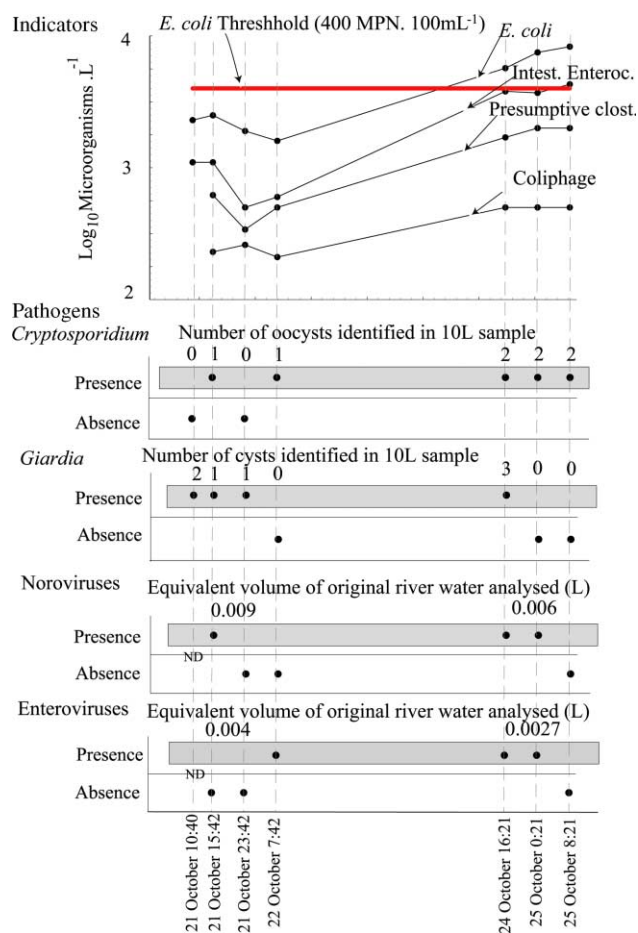


Figure 2 | Comparison of indicator and pathogen results over the course of an event in the Göta älv, compared with the presence of human pathogens (21–25 October 2004). ND = analysis not done (represents sample on 10:40 21 October).

(x axis). The duration of the event refers to the total number of days in the year where peak pathogen concentrations (as defined by the statistically estimated concentrations based on analytical results from samples collected during

one such event) were assumed to penetrate the barrier and reach the treatment plant intake. Each line on the graph then represents the relationship between annualised probability of infection and time under event conditions, for a hypothetical treatment plant performance at Alelyckan water treatment plant (ranging from 1 to 8 log reduction).

Two plots are presented in order to illustrate the results for *Cryptosporidium* (Figure 3(A)). Firstly, assuming that the concentration of oocysts under nominal conditions is zero, and that the treatment plant provides a 4 log reduction of *Cryptosporidium*. The results indicate that the treatment plant, given an open intake from the river, could tolerate event conditions for 10–15 d before the 10^{-4} annual threshold (marked by a horizontal line) is exceeded. Taking into consideration a background concentration of *Cryptosporidium* (0.016 oocysts L⁻¹) however substantially increases the predicted risk and a greater than 4 log reduction would then be necessary, regardless of the event duration.

The corresponding plots for *Giardia* are illustrated in Figure 3(B). The estimated concentration for *Giardia* during the event was relatively low, and the results show that, given an event duration of 3–5 d and a zero nominal concentration of *Giardia*, a 2 log reduction at the treatment plant would provide adequate protection. When the background concentration is assumed to be 0.0049 cysts L⁻¹, for the same event duration, a 3–4 log removal would be necessary to keep the annual probability of infection below the 10^{-4} benchmark.

The QMRA results for noro- and enteroviruses are illustrated in Figure 4. Unfortunately no relevant data was available for estimating the likely nominal concentration of viruses in the river. During the event in October 2004, the

Table 4 | Maximum likelihood estimate (MLE) and upper 95 credible limit for the Poisson parameter (μ) describing pathogen concentration from counts (*Cryptosporidium* and *Giardia*), and presence/absence results (enteroviruses, noroviruses and *Campylobacter*) in Göta älv at the river intake to Göteborg during the sewer breakage event in October, 2004. vu = Virus Units

| | <i>Crypto-sporidium</i> (oocysts L ⁻¹) | <i>Giardia</i> (cysts L ⁻¹) | Noroviruses (vu L ⁻¹) | Enteroviruses (vu L ⁻¹) | <i>Campylobacter</i> (no. L ⁻¹) |
|---------|--|---|-----------------------------------|-------------------------------------|---|
| MLE | 0.13 | 0.083 | 148 | 201 | 10 ^b |
| μ^a | 0.22 | 0.16 | 289 | 439 | |

^a The upper 95% credible limit is an indication of the upper bound of uncertainty. The actual pathogen concentration is unlikely (2.5%) to have exceeded this limit given the analytical result and the assumed model.

^b The result was positive in one sample in 100 mL, and the concentration estimate is therefore conservative.

Table 5 | Daily probability of infection during the sewer breakage event in October 2004 due to *Giardia*, *Cryptosporidium*, enterovirus and norovirus from QMRA modelling, given for the range of potential log reductions in a drinking water treatment assuming open intake

| Log ₁₀ reduction by treatment | Daily probability of infection | | | |
|--|--------------------------------|-----------------------|----------------------|----------------------|
| | <i>Cryptosporidium</i> | <i>Giardia</i> | Norovirus | Enterovirus |
| 1 | 0.005 | 1.4×10^{-4} | 1 | 0.22 |
| 2 | 4.5×10^{-4} | 1.4×10^{-5} | 0.45 | 0.025 |
| 3 | 4.5×10^{-5} | 1.4×10^{-6} | 0.058 | 0.003 |
| 4 | 4.5×10^{-6} | 1.4×10^{-7} | 6.0×10^{-3} | 2.6×10^{-4} |
| 5 | 4.5×10^{-7} | 1.4×10^{-8} | 6.0×10^{-4} | 2.6×10^{-5} |
| 6 | 4.5×10^{-8} | 1.4×10^{-9} | 6.0×10^{-5} | 2.6×10^{-6} |
| 7 | 4.5×10^{-9} | 1.4×10^{-10} | 6.0×10^{-6} | 2.6×10^{-7} |
| 8 | 4.5×10^{-10} | 1.4×10^{-11} | 6.0×10^{-7} | 2.6×10^{-8} |

virus concentrations estimated from analytical results were high, and therefore the QMRA results indicate that, if such an event persisted for 3–5 d, a 6–7 log removal for noroviruses and 5–6 log removal for enteroviruses would be necessary to keep the annual risk below the 10^{-4} benchmark. If the background virus concentration was accounted for in the model, it may be expected that even greater removal capacity would be required to keep the probability of infection below the benchmark.

DISCUSSION

The risk reduction by an active exclusion of microbial contamination peaks in raw water before drinking water treatment has been evaluated from retrospective data records of indicator bacteria concentrations and pathogen sampling data. The retrospective data at the raw water intake from Göta älv indicated an efficient, though not perfect, reduction of *E. coli* levels in relation to the management threshold level at 400 MPN 100 mL⁻¹. The performance of the intake closure as a barrier in eliminating *E. coli* depends on the appropriateness of the monitoring, reporting and decision-making systems. Even when relying on an online (Colilert[™]) assay, a 100% exclusion will not be achieved due to two reasons. Firstly, all microbial monitoring are based on time-interval based samples of

waters entering the intake and the variability in concentration will lead to portions of water with elevated microbial loads being undetected. Secondly, there is a logistical delay between sample collection and the decision to close the intake. At the investigated site, this latter issue has been addressed during the recent year by installing field equipment for online *E. coli* assays within the upstream area using the automated Colifast At-Line Monitor technique[™] (Braathen *et al.* 2005).

How well does the observed elimination of *E. coli* achieved by the water intake regulation reflect the elimination of human pathogens? The weakness of using a threshold for *E. coli* as an indication for pathogen occurrence was illustrated for the sewage emergency discharge in the municipality 40 km upstream of the intake. During this event, positive pathogen data coincided with *E. coli* quantities below the arbitrarily set threshold level. Without the aid of direct reporting, this incident would not have been identified until 3 d after the first positive pathogen samples at the intake. In this example, the action taken based on the reporting of upstream incidents was the key to the efficiency of the intake closure as a barrier.

The drawbacks in relying on different faecal indicators to assess the pathogen load in environmental samples has been summarised by Ashbolt *et al.* (2001). In Finnish lakes and rivers, Hörman *et al.* (2004) concluded that, while the

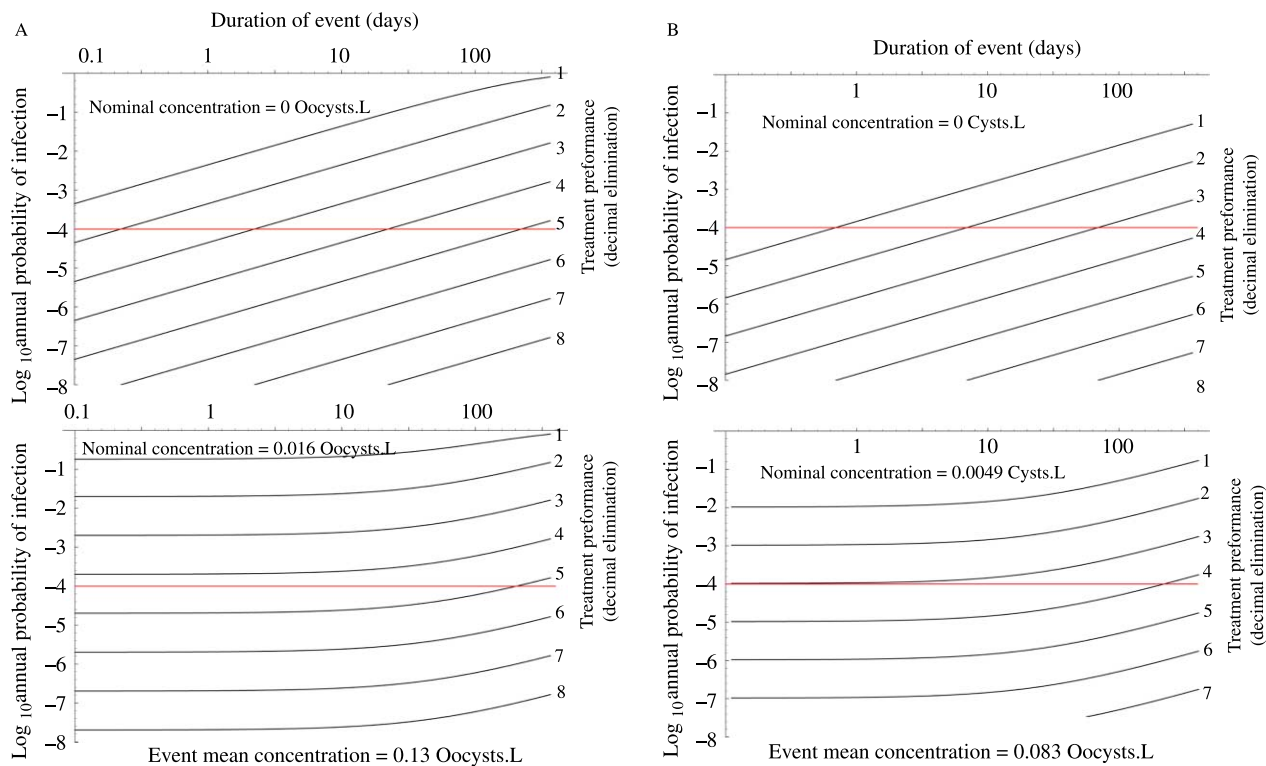


Figure 3 | Annual probability of infection for different water treatment performance, as governed by the duration of sewage discharge events, related to (A) *Cryptosporidium* and (B) *Giardia*. The upper figures illustrate a situation with a nominal concentration equal to zero and the lower a nominal concentration calculated from regular samplings at the Alelyckan water treatment plant.

presence of *E. coli* is correlated to the presence of pathogens, there was no evidence for a quantitative relationship. In that study, pathogens were found as frequently in waters with low *E. coli* concentrations as when the concentrations were high. Mussels grown at the west coast of Sweden did not show significant correlation between occurrence of viruses, noro- and enteroviruses among others and quantities of *E. coli*, somatic coliphages, F-specific phages and phages infecting *B. fragilis* (Hernroth et al. 2002). A similarly low predictive value was observed within the present results, where the current threshold for *E. coli* was shown not to be valid a guide for safe raw water. As shown in Figure 2, the levels of *E. coli* increased during the duration of the event and a similar trend was observed for the other indicators.

A dose-response relationship with both gastro-intestinal and acute febrile respiratory illnesses in marine bathing waters has been shown for intestinal enterococci (Prüss 1998; WHO 2003). In the present results, high concen-

trations of intestinal enterococci ($>1,000$ CFU L⁻¹) coincided with positive detections of either *Giardia* or *Cryptosporidium*, which suggests that enterococci complement the *E. coli* to indicate the presence of parasitic protozoa. For the presumptive clostridia in the present results, no clear trend was observed in relation to the pathogen samples. Results for other studies have shown significant correlations between clostridia and pathogens in rivers (Payment & Franco 1993; Ferguson et al. 1996) and retrospective data analysis on sulfite-reducing clostridia in Göta älv gave a positive correlation to the turbidity and the precipitation upstream, probably due to surface runoffs (Åström et al. 2007). The somatic coliphages, on the other hand, were observed at levels above 620 PFU L⁻¹ in samples positive for noro- or enteroviruses. Bacteriophages are suggested by the WHO as models to assess the behaviour of enteric viruses in water environments as representing similarities in composition and morphology (WHO 2004).

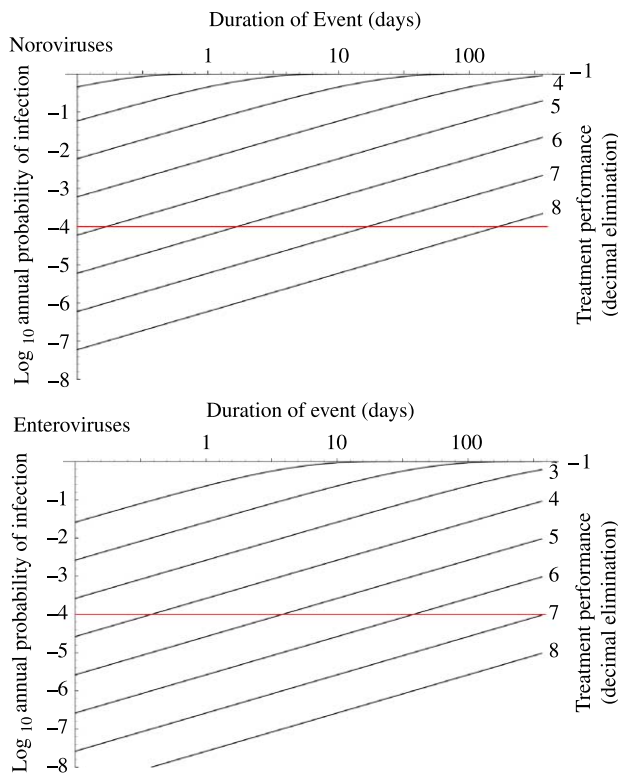


Figure 4 | Annual probability of infection for different water treatment performance, as governed by the duration of sewage discharge events for noro- and enteroviruses. The figures illustrate a situation with a nominal concentration assumed to be equal to zero.

If the present action threshold level of *E. coli* at the intake is lowered the intake will be closed more frequently, but would not guarantee a lower annual probability of infections; rather it would lead to raw water shortage. Additional safety may instead be gained by reducing the impact from pathogen sources and frequency of risk events upstream as verified by monitoring. As seen in the present study positive pathogen detections were, in general, associated with previous rainfalls, as has also been well reported in the literature (Atherholt *et al.* 1998; Kistemann *et al.* 2002; Signor *et al.* 2005). In Göta älv, significant correlation exists between accumulated precipitation in the upstream area and the indicator concentrations downstream (Åström *et al.* 2007), as well as for the pathogenic viruses and parasites occasionally observed during the rain intensive period in the end of October (Table 3). The turbidity in the pathogen samples was highly varying and the peaks at 28 FNU coincided with positive virus

detections. During the sampling programme, positive correlations were observed between turbidity and the indicators, and highest with *E. coli* ($\rho = 0.79$) and sulfite-reducing clostridia ($\rho = 0.88$).

Noroviruses and enteroviruses were detected at the end of October after the sewer breakage upstream. During the same day as this emergency discharge, heavy rainfalls were reported that probably further enhanced the load of human viruses to the river. Due to the small analysed water volumes in the virus assays, absence of viruses during other periods of the year does not mean that virus concentrations are very low (absence in 9 mL simply indicates a concentration less than 110 virus units L^{-1}). It may therefore be that the significance of event periods has been over-stated and the nominal concentrations may be higher than expected (Figure 4). In Sweden, outbreaks of noroviruses are shown to be a significant cause of community-associated outbreaks of acute gastroenteritis (Hedlund *et al.* 2000) and sewer discharges have previously been shown to be the origin for norovirus outbreaks in drinking water (Carrique-Mas *et al.* 2003; Nygård *et al.* 2003). The positive virus detections obtained in the river Göta älv in the present study, at such low sample volumes, clearly indicate significant virus loads in the river water. Additional investigations on viruses, including higher sample volumes, should be prioritized in the future to assess the variability of concentrations during nominal and event conditions.

The parasites *Giardia* and *Cryptosporidium*, known to be very resistant towards chlorination, were observed in about half of the samples during the sampling programme. Concentrations during periods considered as events were similar to the background level measured by regular monitoring at Alelyckan. Given that parasite peaks in the river actually were sampled, this either means that human sewage discharge events do not have a significant impact on *Cryptosporidium* and *Giardia* concentrations at the intake, or that event concentrations regularly penetrate the barrier (intake) undetected. A high survival is reported for *Giardia* and *Cryptosporidium* compared to other microorganisms (deRegnier *et al.* 1989; Medema *et al.* 1997) and may therefore survive transport from far upstream in the river. The higher detection frequency for these protozoa compared to the other pathogens probably also resulted

from the higher volumes analysed, about 10 L compared to a few millilitres for the noro- and enteroviruses given from the virus detection assay.

In contrast to the parasites, pathogenic bacteria were detected in only one of the samples including *Campylobacter*. The presence of gulls near to the intake, reported to occur in the order of several hundreds, represent a potential risk for transmission of *Campylobacter* to the water treatment plants. The prevalence of this organism in black-headed gulls has been found to vary with the season, with highest rates found in late autumn (Broman et al. 2002) which is in line with the positive detection of *Campylocater* as observed in December. In contrast, none of the samples were positive for *E. coli* O157 despite a high prevalence of EHEC reported in this region of Sweden (SMI 2006). Infected cattle within the catchment represent a potential source for zoonotic spread to the river water including, among others, the pathogens *E. coli* O157, *Giardia* and *Cryptosporidium*. The absence of *E. coli* O157 in the present results may reflect a combined low prevalence and a low impact from animals on the microbial contaminations in the river.

The regulation of the raw water intake at the river Göta älv exemplifies a risk management strategy that is not commonly practiced in other parts of Sweden. In general, the objective with risk management is to reduce the risk to an acceptable level and to minimise risk by optimising the reduction throughout the system including all available barriers (Deere et al. 2001). Expected ranges of pathogen reduction given for the major water treatment steps in Alelyckan water treatment plant are reported in Table 6

based on studies published in the literature. Chlorination used intermediately and, in combination with chlorine dioxide in the last step, increase the reduction of bacterial and virus pathogens one order of magnitude.

Local pilot data for surrogate removal within the Alelyckan water treatment plant in Göteborg including fluorescent micro algae (1–15 µm), used as surrogates for protozoan oo(cysts), showed a 2 log removal in the chemical flocculation and sedimentation (Bergstedt & Rydberg 2002). This is within the lower range of values presented in the literature, verifying the infection risks from the present QMRA calculations. The removal of the bacteriophages φx174 and MS2 by chemical flocculation and sedimentation from local pilot studies are reported in the range of 3.8 and 6.2 log (phages added prior to the flocculants; Heinicke 2005). Thus, this treatment step may perform better than indicated from literature data. The reduction given by the intermediate chlorination remains unclear. In addition, treatment failures with sub-optimal processes may result in no pathogen removal within separate treatment steps (Westrell et al. 2003)

The decimal reductions needed in relation to the tolerable risk level, defined as 10^{-4} annual probability of infections, within the connected population, was indicated from the QMRA results. Characterised for a sewage emergency discharge upstream in the river the risk (for varying durations and treatment removal) was presented in relation to background levels, as illustrated for the parasites (Figure 3) and viruses (Figure 4). From the literature data in Table 6, a treatment removal can be expected for *Cryptosporidium* and *Giardia* in the range of 1.6 to 5.7 log units.

Table 6 | Expected range of reduction performance (\log_{10}) for selected pathogens in drinking water treatment based on published literature. Modified from Hijnen et al. (2005) and Thorwaldsdotter (2006)

| | <i>Cryptosporidium</i> and <i>Giardia</i> | | | Noroviruses and enteroviruses | | | <i>Campylobacter</i> | | |
|-------------------------------|---|-------------|------------------|-------------------------------|-------------|------------------|----------------------|-------------|------------------|
| | Min ^a | Most likely | Max ^a | Min ^a | Most likely | Max ^a | Min ^a | Most likely | Max ^a |
| Flocculation/Sedimentation | 0.5 | 1.54 | 2.7 | 0.49 | 1.42 | 2.7 | 0.4 | 1.6 | 2.53 |
| GAC filtration | 0.7 | 1.24 | 2.1 | 0.3 | 1.0 | 1.2 | 0.5 | 1.07 | 2.9 |
| Chlorine dioxide disinfection | 0.41 | 0.57 | 0.9 | 2.21 | 2.43 | 2.77 | 3.77 | 3.89 | 4.08 |

^aMin and Max represent the lower and upper limits of expected removal performance for Flocc/Sed and GAC. However, for chlorine dioxide disinfection these lower and upper values are the lower 5% and upper 95% quantiles of the expected variability in disinfection performance.

Given a mean value of the reduction at 3.4 log, and a nominal concentration at zero oocysts per litre, this means that the tolerable annual risk level is exceeded for *Cryptosporidium* already within one week of these parasite levels in the raw water, given a penetration of the intake microbial barrier. Assuming a nominal concentration at 0.016 oocysts, the corresponding probability for infection increased to levels constantly above the threshold, indicating an inadequate treatment for this organism at the water treatment plant. A lower risk was calculated for the *Giardia* where literature data for the treatment (Table 6) indicate that the infection risk is kept below the tolerable risk level for longer event periods. The results for *Giardia* indicated that the current level of treatment would effectively cope with events lasting for over 100 d given nominal concentrations at zero, and up to 50 d given nominal concentrations at 0.0079 cysts per litre.

During the evaluated sewage emergency discharge event, the viruses were estimated at higher concentrations compared to the parasites. Given the assumptions of the QMRA model, and that the expected treatment performance is between 3–6.7 log (Table 6), the annual infection risk exceeded the threshold for both noroviruses and enteroviruses within a few days of event conditions. The intermediate chlorination (Cl_2), however, is expected to enhance the treatment removal of viruses, lowering the risk for virus passage through the treatment. In the QMRA model, the nominal concentrations for viruses were assumed to be zero. Since there are known to be constant human faecal sources in the catchment (such as treated sewage), this is not a realistic assumption. Instead, it is likely that human enteric viruses would be present in significant levels in the river water also during non-event periods. The results demonstrated in this paper are therefore likely to be an underestimate of the actual annual infection risk with respect to viruses.

A limitation of focusing on an annualised probability of infection is that moderate pathogen loading events of moderate duration may be potentially equated with high pathogen loading events of short duration. These two scenarios are not equivalent from a public health point of view, particularly with regard to the outbreak potential of short-term peaks in the probability of infection. The daily event risks (Table 5) show the magnitude of these potential

risk peaks. A scenario where a pathogen loading event coincides with some form of treatment failure may further increase the peak daily infection risk.

CONCLUSIONS

In this study, the microbial risk due to drinking water consumption was assessed from the occurrence of faecal indicators and pathogens in a river source water. Closing the raw water intake in the river protects the water treatment against pathogen penetrations and therefore serve as a microbial barrier. The efficiency in this barrier was shown to depend upon the closure precision with respect to high pathogen loads in the river. With the present pathogen removals at the water treatment plant the infection risk was calculated in a QMRA model. Given that the intake is not closed in time, the results show that the annual risk level associated with a sewage emergency discharge may be acceptable with respect to *Giardia*, at a borderline for *Cryptosporidium* and not sufficient for noro- and enteroviruses. This emphasises the need for the additional microbial barrier created by an effective raw water intake regulation. Rather than using a threshold level on *E. coli* as a guide for this regulation, information sent about microbial discharges upstream was shown to be helpful within this regulation practice.

ACKNOWLEDGEMENTS

This study has been performed partly within the MicroRisk project, funded by the European Commission (contract EVK1-CT-2002-00123), and by grants from the Swedish Water and Wastewater Association (VA-Forsk). We would like to thank Göteborg Water for their positive collaborations and are grateful for skilful laboratory analysis, performed by the water laboratory at the Swedish Institute for Infectious Disease Control (SMI) and, in relation to some of the indicators, by the laboratory at Alelyckan.

REFERENCES

- Allwood, P. B., Malik, Y. S., Hedberg, C. W. & Goyal, S. M. 2003
Survival of F-specific RNA coliphage, feline calicivirus, and

- Escherichia coli in water: a comparative study. *Appl. Environ. Microbiol.* **69**(9), 5707–5710.
- Andersson, Y. 1991 A waterborne disease outbreak. *Wat. Sci. Technol.* **24**(2), 13–15.
- Ashbolt, N., Grabow, W. & Snoazzi, M. 2001 Indicators of microbial water quality. In *Water Quality: Guidelines, Standards and Health* (ed. L. Fewtrell & J. Bartram), IWA Publishing, London. pp. 289–316.
- Åström, J., Pettersson, T. J. R. & Stenström, T. A. 2007 Identification and management of microbial contaminations in a surface drinking water source. *J. Wat. Health* **5**(Suppl), 67–79.
- Atherholt, T. B., LeChevallier, M. W., Norton, W. D. & Rosen, J. S. 1998 Effect of rainfall on Giardia and Crypto. *J. AWWA* **90**(9), 66–80.
- Bergstedt, O. & Rydberg, H. 2002 Naturally occurring autofluorescent particles as surrogate indicators of sub-optimal pathogen removal in drinking water. In *Chemical Water and Waste Water Treatment VII* (ed. H. Hahn, H. Hoffmann & H. Ødegaard), IWA Publishing, London. pp. 191–200.
- Braathén, H., Ranneklev, S., Sagstad, H. & Rydberg, H. 2005 Fully automated monitor reduces intake of E. coli- contaminated raw water. *Wat. Wastewater Int.* **20**(2), 35.
- Broman, T., Palmgren, H., Bergström, S., Sellin, M., Waldenström, J., Danielsson-Tham, M. L. & Olsen, B. 2002 Campylobacter jejuni in black-headed gulls (*Larus ridibundus*): Prevalence, genotypes, and influence on C. jejuni epidemiology. *J. Clin. Microbiol.* **40**(12), 4594–4602.
- Carrique-Mas, J., Andersson, Y., Petersen, B., Hedlund, K. O., Sjögren, N. & Giesecke, J. 2003 A norwalk-like virus waterborne community outbreak in a Swedish village during peak holiday season. *Epidemiol. Infect.* **131**(1), 737–744.
- Deere, D., Stevens, M., Davison, A., Helm, G. & Dufour, A. 2001 Management strategies. In *Water Quality: Guidelines, Standards and Health* (ed. L. Fewtrell & J. Bartram), World Health Organization Water Series, IWA Publishing, London. pp. 257–288.
- deRegnier, D. P., Cole, L., Schupp, D. G. & Erlandsen, S. L. 1989 Viability of Giardia cysts suspended in lake, river, and tap water. *Appl. Environ. Microbiol.* **55**(5), 1223–1229.
- Ferguson, C., Coote, B. G., Ashbolt, N. J. & Stevenson, I. M. 1996 Relationships between indicators, pathogens and water quality in an estuarine system. *Wat. Res.* **30**(9), 2045–2054.
- Ferguson, C., Husman, A. M. D., Altavilla, N., Deere, D. & Ashbolt, N. 2003 Fate and transport of surface water pathogens in watersheds. *Crit. Rev. Environ. Sci. Technol.* **33**(3), 299–361.
- Furumoto, W. A. & Mickey, R. 1967 A Mathematical model for infectivity-dilution curve of tobacco mosaic virus - experimental tests. *Virology* **32**(2), 224–233.
- Gale, P. 2003 Developing risk assessments of waterborne microbial contaminations. In *The Handbook of Water and Wastewater Microbiology* (ed. D. Mara & N. J. Horan), Academic, London. pp. 263–280.
- Gelman, A., Carlin, J. B., Stern, H. A. & Rubin, D. B. 2004 *Bayesian Data Analysis*. Chapman & Hall, London.
- Gilgen, M., Germann, D., Luthy, J. & Hubner, P. 1997 Three-step isolation method for sensitive detection of enterovirus, rotavirus, hepatitis A virus, and small round structured viruses in water samples. *Int. J. Food Microbiol.* **37**(2–3), 189–199.
- Gilks, W. R., Richardson, S. & Spiegelhalter, D. J. 1996 *Markov Chain Monte Carlo in Practice*. Chapman & Hall, London.
- Hedlund, K. O., Rubilar-Abreu, E. & Svensson, L. 2000 *Epidemiology of calicivirus infections in Sweden 1994–1998*. *J. Infect. Dis.* **181**(Suppl 2), S275–S280.
- Heinicke, G. 2005 *Biological Pre-filtration and Surface Water Treatment. Microbial Barrier Function and Removal of Natural Inorganic and Organic Compounds*. PhD thesis, Chalmers University of Technology, Göteborg.
- Henroth, B. E., Candén-Hansson, A. C., Rehnstam-Holm, A. S., Girones, R. & Allard, A. K. 2002 Environmental factors influencing human viral pathogens and their potential indicator organisms in the blue mussel, *Mytilus edulis*: the first scandinavian report. *Appl. Environ. Microbiol.* **68**(9), 4523–4533.
- Hijnen, W. A., Beerendonk, E. & Medema, G. 2005 *Elimination of Microorganisms by Drinking Water Treatment Processes*. Kiwa NV, Nieuwegein, The Netherlands.
- Horan, N. J. 2003 Fecal indicator organisms. In *The Handbook of Water and Wastewater Microbiology* (ed. D. Mara & N. J. Horan), Academic, London. pp. 105–112.
- Hörman, A., Rimhanen-Finne, R., Maunula, L., von Bonsdorff, C. H., Torvela, N., Heikinheimo, A. & Hanninen, M. L. 2004 *Campylobacter spp., Giardia spp., Cryptosporidium spp., noroviruses, and indicator organisms in surface water in southwestern Finland, 2000–2001*. *Appl. Environ. Microbiol.* **70**(1), 87–95.
- Hoxie, N. J., Davis, J. P., Vergeront, J. M., Nashold, R. D. & Blair, K. A. 1997 Cryptosporidiosis-associated mortality following a massive waterborne outbreak in Milwaukee Wisconsin. *Am. J. Public Health* **87**(12), 2032–2035.
- Hrudey, S. E. & Hrudey, E. J. 2004 *Safe Drinking Water. Lessons from Recent Outbreaks in Affluent Nations*. IWA Publishing, London.
- ISO 1986 *ISO 6461-2, Water Quality-Detection and Enumeration of the Spores of Sulfite-Reducing Anaerobes (Clostridia)-Part 2: Membrane Filtration Method*. International Standardisation Organisation, Geneva.
- ISO 2000a *ISO 7899-2, Water Quality-Detection and Enumeration of Intestinal Enterococci-Part 2: Membrane Filtration Method*. International Standardisation Organisation, Geneva.
- ISO 2000b *ISO 9308-1: Water Quality-Detection and Enumeration of Escherichia coli and Coliform Bacteria-Part 1: Membrane Filtration Method*. International Standardisation Organisation, Geneva.
- ISO 2000c *ISO 10705-2 Water Quality-Detection and Enumeration of Bacteriophages - Part 2: Enumeration of Somatic Coliphages*. International Standardisation Organisation, Geneva.
- Kistemann, T., Classen, T., Koch, C., Dangendorf, F., Fischer, R., Gebel, J., Vacata, V. & Exner, M. 2002 Microbial load of drinking water reservoir tributaries during extreme

- rainfall and runoff. *Appl. Environ. Microbiol.* **68**(5), 2188–2197.
- MacKenzie, W. R., Hoxie, N. J., Proctor, M. E., Gradus, M. S., Blair, K. A., Peterson, D. E., Kazmierczak, J. J., Addiss, D. G., Fox, K. R., Rose, J. B. & Davis, J. P. 1994 A massive outbreak in Milwaukee of cryptosporidium infection transmitted through the public water supply. *N. Engl. J. Med.* **331**(3), 161–167.
- Medema, G. J., Bahar, M. & Schets, F. M. 1997 Survival of *Cryptosporidium parvum*, *Escherichia coli*, faecal enterococci and *Clostridium perfringens* in river water: Influence of temperature and autochthonous microorganisms. *Wat. Sci. Technol.* **35**(11–12), 249–252.
- Medema, G. J. & Schijven, J. F. 2001 Modelling the sewage discharge and dispersion of *Cryptosporidium* and *Giardia* in surface water. *Wat. Res.* **35**(18), 4307–4316.
- Montgomery, D. C. & Runger, G. C. 2003 *Applied Statistics and Probability for Engineers*. Wiley, New York.
- Nygård, K., Torven, M., Ancker, C., Knauth, S. B., Hedlund, K. O., Giesecke, J., Andersson, Y. & Svensson, L. 2003 Emerging genotype (GGIIB) of norovirus in drinking water. *Sweden. Emerg. Infect. Dis.* **9**(12), 1548–1552.
- Olson, M. E., Goh, J., Phillips, M., Guselle, N. & McAllister, T. A. 1999 *Giardia* cyst and *Cryptosporidium* oocyst survival in water, soil, and cattle feces. *J. Environ. Qual.* **28**(6), 1991–1996.
- Ottoson, J., Hansen, A., Björleinius, B., Norder, H. & Stenström, T. A. 2006 Removal of viruses, parasitic protozoa and microbial indicators in conventional and membrane processes in a wastewater pilot plant. *Wat. Res.* **40**(7), 1449–1457.
- Payment, P. & Franco, E. 1993 *Clostridium perfringens* and somatic coliphages as indicators of the efficiency of drinking water treatment for viruses and protozoan cysts. *Appl. Environ. Microbiol.* **59**(8), 2418–2424.
- Prüss, A. 1998 Review of epidemiological studies on health effects from exposure to recreational water. *Int. J. Epidemiol.* **27**(1), 1–9.
- Rendtorff, R. C. 1954 The experimental transmission of human intestinal protozoan parasites. II. *Giardia lamblia* cysts given in capsules. *Am. J. Hygiene.* **59**(2), 209–220.
- Schiff, G. M., Stefanovic, G. M., Young, E. C., Sander, D. S., Pennekamp, J. K. & Ward, R. L. 1984 Studies of echovirus-12 in volunteers: determination of minimal infectious dose and the effect of previous infection on infectious dose. *J. Infect. Dis.* **150**(6), 858–866.
- Signor, R. S., Roser, D. J., Ashbolt, N. J. & Ball, J. E. 2005 Quantifying the impact of runoff events on microbiological contaminant concentrations entering surface drinking source waters. *J. Wat. Health* **3**(4), 453–468.
- SMI 2006 *Statistics from the Swedish Institute for Infectious Disease Control*. Accessed on 12 October 2006. Available at: <http://www.smittskyddsinstitutet.se>
- Teunis, P., Medema, G., Kruidenier, L. & Havelaar, A. 1998 Assessment of the risk of infection by *Cryptosporidium* or *Giardia* in drinking water from a surface water source. *Wat. Res.* **31**(6), 1333–1346.
- Teunis, P. F. & Havelaar, A. H. 2000 The Beta Poisson dose-response model is not a single-hit model. *Risk Anal.* **20**(4), 513–520.
- Teunis, P. F. M., Chappell, C. L. & Okhuysen, P. C. 2002 *Cryptosporidium* dose response studies: variation between isolates. *Risk Anal.* **22**(1), 175–183.
- Teunis, P. F. M., van der Heijden, O. G., van der Giessen, J. W. B. & Havelaar, A. H. 1996 *The Dose-response Relation in Human Volunteers for Gastro-intestinal Pathogens*. Rijksinstituut voor Volksgezondheid en Milieu RIVM.
- Thorwaldsdotter, R. 2006 *Use of Quantitative Microbial Risk Assessment (QMRA) as a Tool in the Hazard Analysis and Critical Control Point (HACCP) Management System for Water Treatment Plants - Especially for Development of Critical Limits*. Lund University, Lund.
- Tyrrel, S. F. & Quinton, J. N. 2003 Overland flow transport of pathogens from agricultural land receiving faecal wastes. *J. Appl. Microbiol.* **94** (Suppl), 87–93.
- USEPA 2001 *Method 1623. Cryptosporidium and Giardia in Water by Filtration/IMS/IFA*. US Environmental Protection Agency, Washington, DC.
- Ward, R. L., Bernstein, D. I., Young, E. C., Sherwood, J. R., Knowlton, D. R. & Schiff, G. M. 1986 Human rotavirus studies in volunteers: determination of infectious dose and serological response to infection. *J. Infect. Dis.* **154**(5), 871–880.
- Westrell, T., Andersson, Y. & Stenström, T. A. 2006 Drinking water consumption patterns in Sweden. *J. Wat. Health* **4**(4), 511–522.
- Westrell, T., Bergstedt, O., Stenström, T. A. & Ashbolt, N. J. 2003 A theoretical approach to assess microbial risks due to failures in drinking water systems. *Int. J. Environ. Health Res.* **13**(2), 181–197.
- WHO 2003 *Guidelines for Safe Recreational-Water Environments*, vol. 1. *Coastal and Fresh-Waters*. World Health Organization, Geneva.
- WHO 2004 *Guidelines for Drinking Water Quality*, 3rd edn. World Health Organization, Geneva.