

Including operational data in QMRA model: development and impact of model inputs

Kenza Jaidi, Benoit Barbeau, Annie Carrière, Raymond Desjardins and Michèle Prévost

ABSTRACT

A Monte Carlo model, based on the Quantitative Microbial Risk Analysis approach (QMRA), has been developed to assess the relative risks of infection associated with the presence of *Cryptosporidium* and *Giardia* in drinking water. The impact of various approaches for modelling the initial parameters of the model on the final risk assessments is evaluated. The Monte Carlo simulations that we performed showed that the occurrence of parasites in raw water was best described by a mixed distribution: log-Normal for concentrations > detection limit (DL), and a uniform distribution for concentrations < DL. The selection of process performance distributions for modelling the performance of treatment (filtration and ozonation) influences the estimated risks significantly. The mean annual risks for conventional treatment are: $1.97E - 03$ (removal credit adjusted by log parasite = log spores), $1.58E - 05$ (log parasite = $1.7 \times$ log spores) or $9.33E - 03$ (regulatory credits based on the turbidity measurement in filtered water). Using full scale validated SCADA data, the simplified calculation of CT performed at the plant was shown to largely underestimate the risk relative to a more detailed CT calculation, which takes into consideration the downtime and system failure events identified at the plant ($1.46E - 03$ vs. $3.93E - 02$ for the mean risk).

Key words | filtration, Monte Carlo, ozonation, QMRA, risk assessment, SCADA

Kenza Jaidi
Benoit Barbeau
Annie Carrière
Raymond Desjardins
Michèle Prévost (corresponding author)
Department of Civil, Geological and Mining
Engineering,
Ecole Polytechnique de Montréal,
Industrial-NSERC Chair in Drinking Water,
CP 6079, Succ. Centre-ville, Montréal,
Québec H3C 3A7,
Canada
Tel.: +1514 340 4711 x5924
E-mail: michele.prevost@polymtl.ca

NOMENCLATURE

D	expected number of parasites ingested daily	θ and η	parameters of distribution f
r	organism-specific infectivity parameter	$C_{\text{res}(i)}$	ozone residual in cell i (mg/L)
$C_{p,RW}$	concentration of parasitic organisms (<i>Giardia</i> or <i>Cryptosporidium</i>) in raw water entering the treatment plant ((oo)cysts/100 l)	T	hydraulic retention time (min)
R	recovery of the detection method	T_{10}/T	hydraulic efficacy
I	fraction of the organisms capable of infection	$V_{\text{cell},i}$	volume of cell i (m^3)
DR	decimal reduction of the treatment process	$Q_{d,\text{max},LP(i)}$	maximum daily flow with low pressure in cell i (m^3/s)
R_p	removal achieved from a physical process	$I(p,i)$	parasite inactivation in cell i
I_{O_3}	inactivation resulting from ozonation	X_h	temperature data from $h = 1$ to n ($^{\circ}\text{C}$)
V	individual's daily consumption of drinking water (L/d)	n	number of temperature data
μ	mean	\bar{X}	mean temperature
σ	standard deviation	$S_{\text{prediction}}^2$	unbiased predicted (or estimated) standard deviation
ε and τ	parameters of log-Normal distribution	MSE	mean square error or mean square residual
		SSE	sum of squares error or sum of squares residual

doi: 10.2166/wh.2009.133

INTRODUCTION

The use of indicator bacteria, whether total/fecal coliform or *E. coli*, as general indicators of microbiological water quality has been overall successful in reducing the risk of waterborne illnesses. However, it is no longer considered sufficient for assessing and managing the risk associated with some pathogenic micro-organisms, especially chlorine resistant pathogenic micro-organisms such as *Cryptosporidium* and *Giardia* (Ashbolt *et al.* 2001; Hunter *et al.* 2003). The latest international guidelines on drinking water quality promote a preventive, and integrated (source to tap) framework for assessing and managing the safety of drinking water. For drinking water, risk reduction involves identifying and managing the contaminants present in source water, installing treatment barriers appropriate for handling the level of contamination, monitoring treatment performance and ensuring the maintenance of water quality throughout the distribution network. Quantitative microbial risk assessment (QMRA) is a method providing a scientific basis for risk management decisions. Its use by water suppliers is highly beneficial for supporting risk management, as it helps define when safe is 'safe enough' (Medema & Ashbolt 2006).

There are several QMRA frameworks, and they differ significantly in statistical approach and complexity (Haas *et al.* 1999). Simple models, which produce point risk estimates using average contaminant values, and may include treatment removal performance, can provide a useful estimate of microbial risk (Aboytes *et al.* 2004). More complex models produce risk distributions integrating various factors, such as detailed process performance and actual estimates of disability adjusted life years (DALYs), and allow for the identification and subsequent management of high-risk situations (Haas *et al.* 1996a; Teunis *et al.* 1997; Gale 1998; Barbeau *et al.* 2000; Havelaar *et al.* 2000; Messner *et al.* 2001, 2006; Medema *et al.* 2003; Masago *et al.* 2004; Signor & Ashbolt 2006; Smeets *et al.* 2006a; Petterson 2006). Few authors have investigated the impact of the choice of model inputs on risk estimates (Barbeau *et al.* 2000) and analysed the benefits of using the information contained in the Supervisory Control and Data Acquisition (SCADA) data (Nilsson 2006). Data from SCADA systems allow for the detailed investigation of events through the analysis of the distributed monitoring data and reporting of control actions.

Current knowledge on pathogen removal by physico-chemical treatment has recently been reviewed, providing mean, minimum and maximum elimination capacity figures (LeChevallier & Au 2004; Emelko *et al.* 2005; Smeets *et al.* 2006a). However, recent data from a large number of full-scale plants suggest that high-performance chemically-assisted filtration does not constitute a robust enough barrier to reduce the relative infection risk below the reference $10E - 4$ infection/yr annual goal used by the USEPA (Aboytes *et al.* 2004). By contrast, the results of the physical removal of parasites and the spores of aerobic spore-forming bacteria (ASF) observed in full-scale and pilot filters are often significantly higher than the corresponding *Cryptosporidium* removal regulatory credits (Mazounie *et al.* 2000; Cornwell *et al.* 2003; Emelko *et al.* 2003). Existing models of pathogen removal or inactivation based on CT calculations during drinking water treatment processes have been shown to over or underestimate the inactivation of micro organisms, and with large margins of uncertainty. The inactivation of pathogens can be predicted using CT models of varying complexity (CT_{10} , CT_{IDDF} , $CT_{average}$, CT_{CFD}) (Teefy & Singer 1990; Haas *et al.* 1996b; Bellamy *et al.* 1998; Zhang *et al.* 2005). The traditional CT_{10} concept overestimates inactivations due to the use of the linear Chick Watson inactivation model as shown by the unrealistic high log values calculated for chlorine inactivation in warm water. Recent comparisons of inactivation levels predicted by various models have shown large (2 to 4 fold) discrepancies between advanced inactivation models (Zhang *et al.* 2006; Broséus *et al.* 2006). Major issues with treatment models which remain unresolved include: (1) the lack of established validation with inactivation/treatment data from full-scale plants which include actual operational data (SCADA); (2) the inability of these models to fully take into account the impact of water quality (Mysore *et al.* 2003; Hijnen *et al.* 2004; Barbeau *et al.* 2004, 2005) and the initial pathogen concentration (Haas & Kaymak 2002, 2003). Recent work has shown the importance of the actual measurement of pathogen survival during disinfection (Smeets *et al.* 2006a,b). Although disinfection and filtration models are already useful for operational and design purposes, a true evaluation of health risk reduction by treatment must be founded on plant-validated pathogen removal figures. Actual measurements of indicators and

pathogens can resolve model discrepancies and decrease the level of uncertainty in the predictions (Smeets *et al.* 2006b; Smeets & Medema 2006).

Our study focuses on two treatment processes: filtration and disinfection (ozonation). The objectives of this work are the following: (1) Develop a Monte Carlo probabilistic QMRA model to estimate the relative risk of infection associated with the ingestion of *Cryptosporidium* and *Giardia* in drinking water. The distinctive feature of this part of the research is the inclusion of SCADA data to integrate the reality of full-scale operations; (2) Evaluate the impact of input models on risk estimates (such as those that describe parasite distribution and the correlation between pathogen concentration and recovery); (3) Evaluate the impact of the detailed features of disinfection and filtration models, such as CT calculation methods and the application of filter performance credits, on relative risk estimates.

METHODOLOGY: DESCRIPTION OF THE MODEL DEVELOPED

The concentration of parasites in treated water is quantified by the measurement of the occurrence of the pathogens in raw water and its subsequent reduction by treatment barriers. Interpretation of the measurement data is only possible within the framework of a mathematical model. The choice of the model's parameters must be rigorously justified, as a single series of data may lead to different interpretations, depending on the model used. The application of the exponential model of infection for *Cryptosporidium* and *Giardia* has been extensively used (Haas 1983; Regli *et al.* 1991; Rose *et al.* 1991; Haas *et al.* 1996a; Eisenberg *et al.* 1998; Teunis *et al.* 1997, 1999; Barbeau *et al.* 2000) and is based on the work carried out by Rendtorff (1954), DuPont *et al.* (1995) and Okhuysen *et al.* (1999). The model developed in this project to express the risk of infection, and described by Equation (1), is built on this exponential model.

$$P_{\text{infection}} = 1 - \exp(-r \times D) \quad (1)$$

The dose D can be expressed as follows (Teunis *et al.* 1997; Barbeau *et al.* 2000; Havelaar *et al.* 2000; Teunis &

Havelaar 2002; Hunter *et al.* 2003):

$$D = C_{p,RW} \times (1/R) \times I \times 10^{-(DR)} \times V \quad (2)$$

Considering consecutive exposures as the independent variable, the annual probability of 1 or more infections may be calculated under the assumptions of binomial process (infection: $P_{\text{infection}}$ or not infection: $1 - P_{\text{infection}}$) (Haas *et al.* 1999; Petterson *et al.* 2006). However, estimates of the probability of infection after a single exposure (e.g. daily exposure, $P_{\text{infection},d}$) can be converted into the probability of infection after n exposures ($P_{\text{infection},n}$) (Haas *et al.* 1999; Hunter *et al.* 2003; Petterson *et al.* 2006):

$$P_{\text{infection},n} = 1 - (1 - P_{\text{infection},d})^n \quad (3)$$

When $P_{\text{infection},d} \ll 1$, $P_{\text{infection},n}$ may be approximated as $P_{\text{infection},n} = n \times P_{\text{infection},d}$ (Haas *et al.* 1999).

In the case of the exponential dose-response model, Equation (3) is equal to Equation (4) (Haas *et al.* 1999):

$$P_{\text{infection},n} = 1 - \exp\left(-r \times \sum_{i=1}^n \text{Dose}_i\right) \quad (4)$$

The annual risk of infection can then be calculated for $n = 365$. The general equation for risk of infection is thus written as follows:

$$P_{\text{infection}} = 1 - \exp(-r \times C_{p,RW} \times (1/R) \times I \times 10^{-(DR)} \times V \times 3.65) \quad (5)$$

Because of the uncertainty on the various parameters involved in the exposure process or their variability, or a combination of these two factors, these parameters will be characterized by probability distribution functions (PDF). With the probabilistic approach, it is possible to quantify the weight of the various parameters in the final assessment of the risk and of its uncertainty. The method most widely used in this quantitative risk analysis is Monte Carlo (MC) simulation (Haas *et al.* 1999), which was adopted for this study. The application of the MC method is based on statistical sampling techniques which make it possible to produce a stochastic approximation of the result (solution of a mathematical equation or model), instead of calculating a single, or mean value of the risk.

The Monte Carlo simulations were performed using the software Crystal Ball[®] 7.2 (Decisioneering, USA). The PDFs were defined for each variable in Equation (5). The calculation was made iteratively, and the parameters were randomly sampled. The sum of the iterations forms the probability distribution of the assessed risk. The most appropriate distribution for each variable is identified by two tests: the Kolmogorov–Smirnov (K–S) test, which is sensitive to mean values, and the Anderson–Darling (A–D) test, which is sensitive to extreme values (Barbeau *et al.* 2000). The two tests consist of measuring the largest vertical distance between the theoretical distribution and the experimental distribution.

Uncertainty and variability

While performing a QMRA, it is useful to distinguish between variability and uncertainty. Variability corresponds to the difference observed in a given population due to heterogeneity or to natural diversity, and reflects fluctuations linked to a statistical phenomenon, while uncertainty is caused by methodological limitations or by a lack of knowledge of the initial parameters of the model (Firestone *et al.* 1997; Haas *et al.* 1999). Consequently, variability and uncertainty are represented by probability distributions on the understanding that they describe two different phenomena. At the same time, certain parameters describing the variability of the exposed population can constitute sources of uncertainty (the parameters are not measured with certainty). They are referred to as uncertain variables which can be represented by second-order random variables (Haas *et al.* 1999). With the Monte Carlo approach, which is a two-dimensional simulation (2-D MCA), it is possible to integrate second-order variables by combining the distributions describing variability and uncertainty (USEPA 2001a, b). This analysis also makes it possible to evaluate the uncertainty surrounding estimated values, represented by credibility intervals.

Occurrence of parasites in raw water, $C_{p,RW}$

Two water treatment plants were considered for this study. Both draw their source water from the St. Lawrence River water. Plant A is currently equipped with direct filtration without coagulation, followed by chlorination. At Plant B,

the treatment includes direct filtration without chemical assistance, followed by ozone disinfection and chlorination.

Parasites data from the two plants were collected. The period considered covers January 2000 to June 2006, with one microbiological sample analyzed every two months. The analytical methods used to measure the concentrations of *Cryptosporidium* and *Giardia* in the raw water were the ICR method (USEPA 2003b), applied between January 2000 and October 2003, and method 1623 (USEPA 2001c), applied from February 2004 to June 2006.

The occurrence of *Cryptosporidium* and *Giardia* is relatively low in the raw water from both sources. The proportion of positive samples is relatively high in the case of plant A (43% *Cryptosporidium* and 69% *Giardia* positive) and more modest for plant B (17% *Cryptosporidium* and 48% *Giardia*). Mean concentrations measured for Plant A ($n = 35$) were 3.16 ± 8.04 oocysts/100 L and 6.08 ± 7.43 cysts/100 L and were 3.49 ± 16.96 oocysts/100 L and 2.62 ± 4.97 cysts/100 L for plant B ($n = 42$). The concentrations of the oo(cysts) were highly variable (from non-detected to 28 cysts/100 L for *Giardia*, and from non-detected to 108 oocysts/100 L for *Cryptosporidium*), and the data is characterized by the predominance of non-detected oocysts. These limitations constitute a significant modelling challenge, and the following mathematical approaches have been proposed:

- (1) The use of mixed distribution to describe parasites density (Haas *et al.* 1999; Barbeau *et al.* 2000; Medema *et al.* 2003; Masago *et al.* 2004);
- (2) The use of discrete distributions to describe the counts, instead of considering continuous distributions of parasite density. A Poisson distribution can be defined for the combinations of count/volume pairs (USEPA 2003b; Petterson *et al.* 2006). This approach takes into account the influence of the volume analyzed on the probability of detecting an event. By contrast, at low densities, the variability of the volume analyzed has a major impact, which increases the uncertainty of the concentration estimate (low counts, low volumes).

In fact, no model describes the true behaviour of the micro-organisms in the environment. Since our objective was to choose the model providing an adequate and simplified description of the parasite occurrence, we opted for mixed distributions to describe the concentrations of

Cryptosporidium and *Giardia*. Distributions were modeled using the raw counts and volumes analyzed. Three forms of distribution were tested (Barbeau *et al.* 2000):

$$C_{p,EB} = \begin{cases} f_1(\varepsilon, \tau) & \text{for } C_{p,RW} \geq 1/100 \text{ L} \\ f_2(\theta, \eta) & \text{for } C_{p,RW} < 1/100 \text{ L} \\ f_3(\theta, \eta) & \text{for } 0 \leq C_{p,RW} \leq n/100 \text{ L} \end{cases} \quad (6)$$

Concentrations of *Cryptosporidium* and *Giardia* which are above the detection limit (DL = 1 oocyst/100 L) follow a log-Normal distribution (f_1) in a satisfactory manner (Table 1). To model concentrations under the detection limit, two distributions were tested: a constant distribution and a uniform distribution. The first distribution (f_2 -a) is the most conservative approach, and presupposes that the concentration under the detection limit is constant and equal to 1 oocyst/100 L. The second distribution is a uniform distribution (f_2 -b) defined by an interval varying from 0 to 1 oocyst or cyst/100 L. This approach is neutral, and desirable in the absence of information on the underlying data distribution. The third distribution is a log-Normal (f_3) distribution on the population of measured data, defined by a combination of (i) the 50th percentile or a percentile > the 50th adjusted to the percentage of non-detected concentrations, and (ii) the 95th percentile. In summary, the three mixed distributions are: log-Normal (> LD) + Constant (< LD): $f_1 + f_2 - a$, log-Normal (> LD) + Uniform (< LD): $f_1 + f_2 - b$ and log-Normal (x th, y th): f_3 .

Recovery, R

There was insufficient historical data on the recovery measured on the samples from plants A and B to generate probability distributions. As a result, the recoveries evaluated for the LT2ESWTR (USEPA 2003b; Messner & Wolpert 2002) were applied. Recoveries data for *Giardia* for the 1623 method were supplied by Dr. Michael Messner of the USEPA (personal communication). The PDF parameters are presented in Table 1. For plants A and B, the distributions of recoveries for the ICR and 1623 methods were combined, given that the parasite concentrations in raw water were measured by these two methods. The percentage of data measured by the ICR method is 63% for plant A and 52% for plant B, while the percentage for the 1623 method is

37% for plant A and 48% for plant B. The use of recoveries data other than those obtained for the measured concentrations creates a source of uncertainty for the model, since recovery is not always constant. It varies from sample to sample, depending on the composition of the water, turbidity, temperature, the number of organisms present and the volume analyzed (Teunis & Havelaar 2002).

Infectivity, I

The analytical methods currently used to detect parasites in source water do not allow a distinction to be made between infectious and non-infectious oo(cysts). Failure to take this distinction into account in assessing risk leads to an overestimation of the risk of infection, since only a fraction of the oocysts and cysts detected in the environment is capable of causing an infection. A recent study has shown that the immunofluorescence cell culture techniques are not sensitive enough to detect infectious oocysts present in low concentrations (Schets *et al.* 2005). Pending the development of more precise techniques, the fraction of infectious *Cryptosporidium* (37%) determined by Di Giovanni *et al.* (1999) and LeChevallier *et al.* (2003) and that of infectious *Giardia* (80%) reported by Zmirou-Navier *et al.* (2006) were considered in this study. These high values may lead to an overestimation of risk. In fact, the reported fractions of fresh oocysts capable of causing *Cryptosporidium* infection in experiments using cell cultures and in mice are lower and vary from 10.7–13.9% (Rochelle *et al.* 2002).

Dose–response parameter, r

The USEPA has conducted a meta analysis on data from clinical studies carried out on volunteers for three strains of *Cryptosporidium*: IOWA, (DuPont *et al.* 1995), TAMU and UCP (Okhuysen *et al.* 1999). The results obtained show that the mean probability of infection following the ingestion of a single oocyst varies from 7% to 9% (USEPA 2003a). By comparison, estimation of the dose–response parameter of *C. parvum* based solely on the IOWA strain is considerably lower, because of the low infectivity of that strain. The mean calculated was 0.4% (Haas *et al.* 1996a; Teunis *et al.* 1997; Barbeau *et al.* 2000; Havelaar *et al.* 2000) which was the value considered for the IESWTR (USEPA 1998).

Table 1 | Summary of input variables and probability distributions of model parameters

Input parameters	Symbol	Distributions and parameters	5th and 95th centiles	Goodness-of-fit
Occurrence of <i>Cryptosporidium</i> (oocysts/100 L)	$C_{C,RW}$ in plants B	$f_2: C_{p,RW} < LD$: constant ($f_{2-a} = 1$) or uniform (f_{2-b} : [0–1])		
		$f_1: C_{C,RW} \geq LD$: log-Normal ($\epsilon = 1.91$; $\tau = 1.46$) with $\mu = 21.49$; $\sigma = 57.39$	1.39–78.70	N.A.
		1) $f_1 + f_{2-a}: 0 \leq C_{C,RW} \leq n$: log Normal + constant ($\mu = 4.47$; $\sigma = 23.33$)	1.0–16.83	N.A.
		2) $f_1 + f_{2-b}: 0 \leq C_{C,RW} \leq n$: log-Normal + uniform ($\mu = 4.06$; $\sigma = 22.40$)	0.06–16.83	N.A.
		3) $f_3: 0 \leq C_{C,RW} \leq n$: log-Normal (C83rd = 0.4, C95th = 8.0) ($\mu = 44.55$; $\sigma = 2,979$)	4.94E-6-146.43	N.A.
	$C_{C,RW}$ in plant A	$f_1: C_{C,RW} \geq LD$: log-Normal ($\epsilon = 1.45$; $\tau = 1.01$) with $\mu = 7.58$; $\sigma = 9.51$	1.31–23.24	K-S = 0.16; A-D = 0.37
		1) $f_1 + f_{2-a}: 0 \leq C_{C,RW} \leq n$: log-Normal + constant ($\mu = 3.82$; $\sigma = 7.09$)	1.0–14.77	N.A.
		2) $f_1 + f_{2-b}: 0 \leq C_{C,RW} \leq n$: log-Normal + uniform ($\mu = 3.53$; $\sigma = 7.21$)	0.09–14.77	N.A.
		3) $f_3: 0 \leq C_{C,RW} \leq n$: log-Normal (C60th = 1.16; C95th = 10,3) $\mu = 2.64$; $\sigma = 7.97$	0.06–10.35	N.A.
		Occurrence of <i>Giardia</i> (cysts/100 L)	$C_{G,RW}$ in plant B	$f_1: C_{G,RW} \geq LD$: log-Normal ($\epsilon = 1.22$; $\tau = 1.00$) with $\mu = 6.21$; $\sigma = 7.55$
1) $f_1 + f_{2-a}: 0 \leq C_{G,RW} \leq n$: log-Normal + constant ($\mu = 3,50$; $\sigma = 5.88$)	1.0–12.83			N.A.
2) $f_1 + f_{2-b}: 0 \leq C_{G,RW} \leq n$: log-Normal + uniform ($\mu = 3.24$; $\sigma = 6.00$)	0,1–12.83			N.A.
$C_{G,RW}$ in plant A	3) $f_3: 0 \leq C_{G,RW} \leq n$: log-Normal (C60th = 1.0; C95th = 12.38) $\mu = 3.21$; $\sigma = 13.26$		0.03–12.6	N.A.
	$f_1: C_{G,RW} \geq LD$: log-Normal ($\epsilon = 1.81$; $\tau = 0.94$) with $\mu = 9.67$; $\sigma = 11.31$		1.59–28.80	K-S = 0.12; A-D = 0.37
	1) $f_1 + f_{2-a}: 0 \leq C_{G,RW} \leq n$: log-Normal + constant ($\mu = 6.97$; $\sigma = 10.33$)		1.0–24.29	N.A.
Dose–response parameters	$r_{Giardia}$	log-Normal ($\epsilon = -4.12$; $\tau = 0.37$) with $\mu = 0.017$; $\sigma = 6.43E - 3$	8.7E – 3–2.9E – 2	K-S = 0.07; A-D = 6.2
	$r_{Cryptosporidium}$	log-Normal ($\epsilon = -2.99$; $\tau = 1.14$) with $\mu = 0.096$; $\sigma = 1.57E - 1$	7.31E – 3–3.02E – 1	K-S = 0.03; A-D = 54.8
Recovery (%)	$R_{Giardia-M1623}$	Beta ($\alpha = 4.5$; $\beta = 2$; min-max = 1%–100%) ($N = 270$)	37.8%–92.9%	K-S: 0.04; A-D: 0.53
	$R_{Giardia-ICR}$	Beta ($\alpha = 3.28$; $\beta = 8.91$; min-max = 1%–100%)	9.07%–49%	N.A.
	$R_{Cryptos-M1623}$	Beta ($\alpha = 2$; $\beta = 3$; min-max = 1%–100%)	9.56%–74.9%	N.A.
	$R_{Cryptos-ICR}$	Beta ($\alpha = 1.44$; $\beta = 11.2$; min-max = 1%–100%)	1.35%–28.31%	N.A.
Infectivity, I	$I_{Giardia}$	80%	N.A.	N.A.

Table 1 | (continued)

Input parameters	Symbol	Distributions and parameters	5th and 95th centiles	Goodness-of-fit
Physical removal, Rp (log)	$I_{Cryptos}$	37%	N.A.	N.A.
	DFNC	Triangular (likeliest = 0.5; min-max = 0.0-1.0)	0.17-0.84	N.A.
	DFSOC	Triangular (likeliest = 0.98; min-max = 0.36-1.66)	0.56-1.44	N.A.
	DFOC	Triangular (likeliest = 1.7; min-max = 0.36-2.8)	1.03-2.45	N.A.
	CONV*	Normal ($\mu = 3.38$; $\sigma = 0.38$)*	3.31-3.38*	K-S:S; A-D:S*
Inactivation with ozone: detailed calculation (log)	I_{O_3} : Plant B	Temperature-dependent with 10.4% downtime. ($N = 17,032$)		
	<i>Cryptosporidium</i>	$\mu = 0.12$; $\sigma = 0.08$; min-max = 0.0-1.02	0.0-0.28	N.A.
	<i>Giardia</i>	$\mu = 1.35$; $\sigma = 0.90$; min-max = 0.0-8.26	0.0-2.99	N.A.
Inactivation with ozone: Plant calculation (mg.min/L)	$CT_{10\text{mean}}$: Plant B	Log-Normal ($\varepsilon = 0.54$; $\tau = 0.55$) with $\mu = 2.00$; $\sigma = 1.20$ ($N = 16,997$)	0.69-4.27	K-S = 0.08; A-D = 134
	Temperature (°C)	T: Plant B Custom ($\mu = 10.59$; $\sigma = 8.68$) ($N = 17,032$)	0.25-23.72	N.A.
Water consumption (L/d)	V	Max extreme (likeliest = 1.25; scale = 0.61) ($N = 1,139$)	0.56-3.03	K-S = 0.04; A-D = 1.12

*Barbeau et al. (2000); K-S: Kolmogorov-Smirnov; A-D: Anderson-Darling; N.A.: Not Available.

For this study, the distribution of the dose–response parameter r of *Cryptosporidium* retained for the LT2ESWTR was obtained from Dr. Michael Messner of the USEPA (personal communication). For *Giardia*, the dose–response parameter r data were taken from a Bootstrap study of the initial Rendtorff data (Rendtorff 1954; Barbeau et al. 2000) yielding results similar to those reported by Rose et al. (1991).

Physical abatement, R_p

Evaluating the performance of physical treatment by directly measuring pathogens such as *Cryptosporidium* and *Giardia* is restrictive. The low concentration of these pathogens prevents any reliable measurement of abatement as a result of the treatment stages. Turbidity removal is used as an indicator of parasite removal; however the validity of this indicator has been seriously questioned (LeChevallier et al. 1991; Scott et al. 1997; Mazoua & Chauveheid 2005). The spores of aerobic spore-forming (ASFB) bacteria can serve as an indicator of the physical removal and chemical inactivation of these oocysts by physical means (Barbeau 1996; Rice et al. 1996; Scott et al. 1997; Yates et al. 1997; Nieminski et al. 2000; Mazoua & Chauveheid 2005; Brown & Cornwell 2005). The ASFB removal capacity is to some extent site specific and site demonstration of actual log removal is now considered as the acceptable approach to demonstrate a system's performance.

Experimental data from pilot studies were available for the source water studied and provide actual measured removals of ASFB by direct filtration without coagulation (DFNC), direct filtration with sub-optimal coagulation (DFSOC), direct filtration with optimal coagulation (DFOC) (Barbeau et al. 1999, 2006) and conventional treatment (CONV) (Barbeau et al. 2000). This data was used to generate probability distributions for these types of filtration. Table 1 summarizes the parameters for the spore removal according to the physical treatment considered.

Once ASFB removal by the various physical treatments has been evaluated, the estimation of parasite removal (*Cryptosporidium* and *Giardia*) can be integrated into the model developed in accordance with three modalities (Figure 1):

1. The first modality constitutes a conservative approach, in which the removal of spores is considered to be the

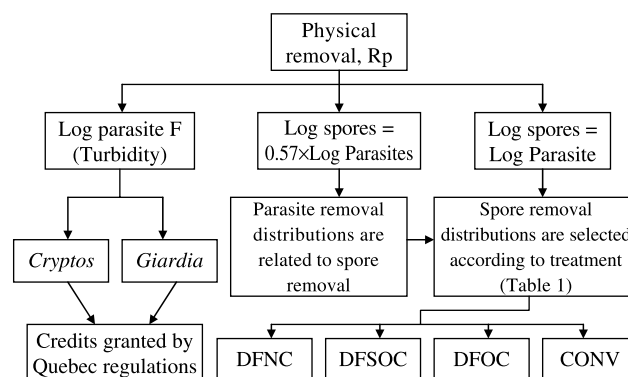


Figure 1 | Modelling approaches to *Cryptosporidium* and *Giardia* removal by a physical process. DFNC: direct filtration without coagulation; DFSOC: direct filtration with sub-optimal coagulation; DFOC: direct filtration with optimal coagulation; CONV: conventional treatment.

equivalent to the removal of oo(cysts) (Teunis et al. 1997; Barbeau et al. 2000; Havelaar et al. 2000).

2. The second approach is the result of a synthesis of a number of studies conducted on the removal of spores as an indicator of the abatement of *Cryptosporidium* (Scott et al. 1997; Yates et al. 1997; Swertfeger et al. 1999; Coffey et al. 1999; Mazounie et al. 2000; Huck et al. 2001; Emelko 2001; Dugan et al. 2001; Clark et al. 2001). From this synthesis, the following correlation can be applied: $\log \text{ spores} = 0.57 \times \log \text{ Cryptosporidium}$ with $R^2 = 0.74$ (Figure 2).
3. When ASFB removal data are not available at a given study site, removals could be set as a function of the prescribed regulatory credits. In this case, the (oo)cyst removal credits are granted in accordance with the Government of Québec regulation, which adjusts the credits as a function of the 95th percentile turbidity value measured in the filter effluent (MDDEP 2002). This approach is essentially identical to the LT2SWTR toolbox (USEPA 2003a).

Ozone inactivation, I_{O_3}

Data source

To estimate inactivation by ozonation, the following data were collected at plant B from 2002 to 2005 recorded bi-hourly: pH, temperature, residual ozone, flow rate at the ozonation tank inflow point (maximum daily flow at low pressure, $Q_{d,max(LP)}$). The operational data were taken from the plant's SCADA system. Some of the values in the SCADA database can be interpreted in different ways. For example,

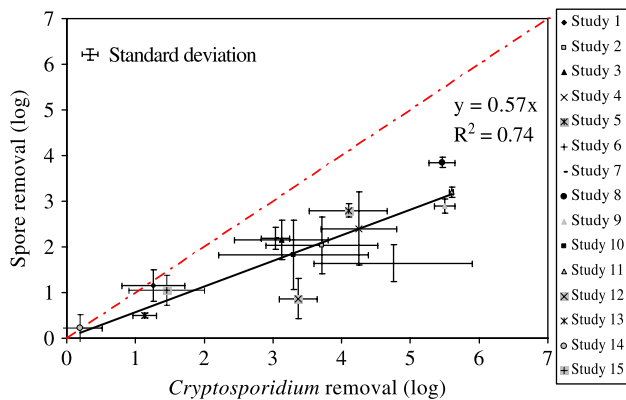


Figure 2 | Correlation between aerobic spore and *Cryptosporidium* removals. Study 1 Dugan et al. (2001): Clarification + optimal coagulation; Study 2 Dugan et al. (2001): Filtration + optimal coagulation; Study 3 Swertfeger et al. (1999); Study 4 Coffey et al. (1999); Study 5 Yates et al. (1997); Study 6 Clark et al. (2001); Study 7 Huck et al. (2001); Study 8 Emelko (2001); Study 9 Mazounie et al. (2000); Study 10 Scott et al. (1997); Study 11 Huck et al. (2001): Stable operation without coagulation; Study 12 Huck et al. (2001): Sub-optimal coagulation, Ottawa; Study 13 Huck et al. (2001): Sub-optimal coagulation, MWD; Study 14 Dugan et al. (2001): Clarification + sub-optimal coagulation; Study 15 Dugan et al. (2001): Filtration with sub-optimal coagulation.

zero residual ozone values may correspond to downtime of ozone injection, a contactor offline, a defective analyzer, etc. Therefore, the log books and the summary event reports were consulted to sort out the events corresponding to the null values, as well as to determine what caused these events to occur (tank maintenance, ozone generator maintenance or downtime, analyzer malfunction, insufficient ozonation, etc.). This information provided a more accurate evaluation of the actual operation of the ozonation contactors at plant B.

Modelling

Two approaches were retained to calculate CT: (1) the CT_{10} calculation performed in the plant, an approach applied by the USEPA and adopted by the Government of Québec (MDDEP 2002); and (2) the detailed CT_{10} calculation with corrections based on the detailed operations data at the plants, i.e. taking into account conditions in each contactor including the periods of underperformance, malfunction and downtime.

Plant calculation

The CT calculation at plant B is based on a mean CT_{10} averaged over all six parallel ozone contactors. This figure is calculated and readjusted once a day to obtain a

3-log inactivation of *Giardia*, taking into account water temperature. It is described by the following equation: $\overline{CT}_{10} = \overline{C}_{res} \times \overline{T} \times (T_{10}/T)$.

The mean CT_{10} data collected ($N = 16997$) covers the period 2002 to 2005 (the same period covered for the detailed calculation). The probability distribution of this figure can be described by a log-Normal distribution ($\mu = 2.0$; $\sigma = 1.2$). The mean parasite inactivation is obtained by the equations adopted by the USEPA and by the Guidance Manual for the Design of Drinking Water Installations in Québec (MDDEP 2002).

Detailed calculation

The detailed calculation for the inactivation of *Cryptosporidium* and *Giardia* follows these steps: (1) Calculation of contact time in each contactor: $T_i = V_{cell,i}/Q_{d,max LP(i)}$; (2) Calculation of CT in each contactor i : $CT_i = C_{res(i)} \times T_i \times (T/T_{10})$; (3) Calculation of parasite inactivation in each contactor; (4) Calculation of total parasite inactivation by the weighted mean over the six contactors (Equation (7)).

$$\text{Log parasite} = -\log \left[\left(\frac{1}{6} \right) \times 10^{-I(p,i=1)} + \dots + \left(\frac{1}{6} \right) \times 10^{-I(p,i=6)} \right] \quad (7)$$

Three situations of ozone operation were distinguished during the analysis of the plant data:

- (1) *Normal operation*: the ozone residual in the tanks was greater than 0;
- (2) *Partial operation*: ozonation in one or more tanks is stopped, i.e. the tank was empty (tank = E) or the water was circulating in the tank without ozone injection (tank = NO). According to the SCADA data, the following four conditions occurred: a) 1 contactor = NO; b) 2 contactors = NO c) 1 contactor = E and 1 contactor = NO; d) 2 contactors = E and 1 contactor = NO;
- (3) *Downtime operation*: ozonation in all six contactors is stopped. Either the tanks were empty, or the water was circulating through the tanks without ozone injection.

In the case of a “partial operation” of the ozonation process, *Giardia* inactivation was found to be constant

for each condition previously identified (Figure 3). This situation is explained by the use of weighted mean, which accurately reflects the importance of short-circuiting on the overall inactivation (Equation (7)).

In the case of “normal operation” of the ozonation process, parasite inactivation varied considerably as a function of temperature. In order to take this effect into account, regressions were established between the log of parasite inactivation and water temperature. Figures 4 (a) and (b) illustrate the removal of parasites by ozonation as a function of temperature.

The variability of the log of inactivation relative to the established regressions was taken into account by calculating the error on the regressions, according to the following equation (Neter et al. 1985):

$$S_{\text{prediction}} = \sqrt{\text{MSE} \cdot \left[1 + \frac{1}{n} + \frac{(X_h - \bar{X})^2}{\text{SSX}} \right]} \quad (8)$$

$$\text{With } \text{SSX} = \sum (X_h - \bar{X})^2 \quad (9)$$

In our model, we calculated parasite inactivation as a function of temperature, in the “normal operation” case, according to the following reasoning:

Posing $y = \text{Log } \textit{Giardia}$ or $\text{Log } \textit{Cryptosporidium}$ and $Y = \text{Ln } y$:

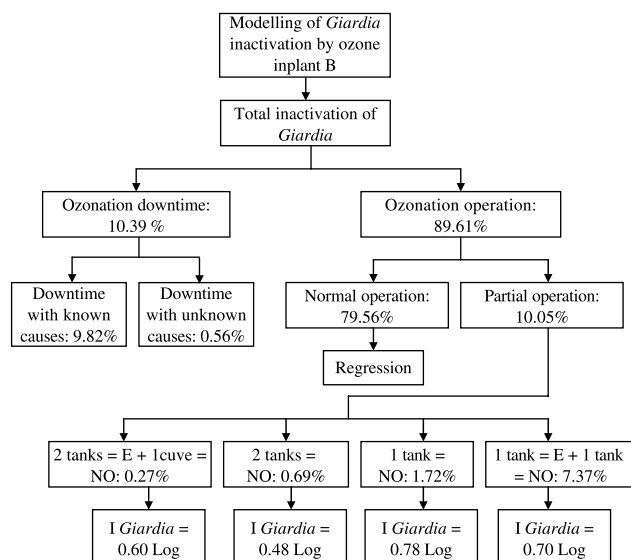


Figure 3 | Modelling of *Giardia* inactivation by ozone in plant B.

Find a random value in the temperature distribution: (1) Calculate: $Y = b + aT$ (regression equation); (2) Calculate: $S_{\text{prediction}}$ with $T = X_h$, knowing n , SSX and MSE ; (3) Find a random value from a Normal law centered at 0 with $\sigma = 1$; (4) Calculate: $Y_{\text{adjusted}} = Y + S_{\text{prediction}} * \text{Normal law}$; (5) Calculate: $y = \exp(Y)$. The temperature of the raw water at plant B is defined by a custom distribution (Table 1).

Daily drinking water consumption, V

The data on the daily consumption of drinking water were taken from a study conducted by Payment et al. (1997) in Quebec, Canada. The data fits in a satisfactory manner the extreme value PDF with a mean of 1.6 L/d (Table 1).

Stability of the model

The stability of the output of the model is influenced by the specifics of the model developed and the intrinsic heterogeneity of the results (Haas et al. 1999). This stability was evaluated using the mean, the standard deviation and coefficient of variation of the model outputs of occurrence of *Cryptosporidium* and risk of infection obtained as a function of the number of MC simulations. This analysis indicated that the mean concentration of *Cryptosporidium* in source water is stable for the various numbers of simulations tested, while the mean risk of infection only stabilizes after 50,000 runs. The analysis also shows a systematic reduction in the standard deviation with an increase in the number of simulations. Moreover, if a coefficient of variation of 5% is deemed acceptable, then 100,000 would be the most appropriate number of simulations. In this study, the Monte Carlo simulations were carried out with 100,000 trials.

RESULTS AND DISCUSSION

Impact of input parameters to the model on risk assessment

The impacts of several approaches to model the input parameters on the final assessment of the risk of infection via drinking water were assessed.

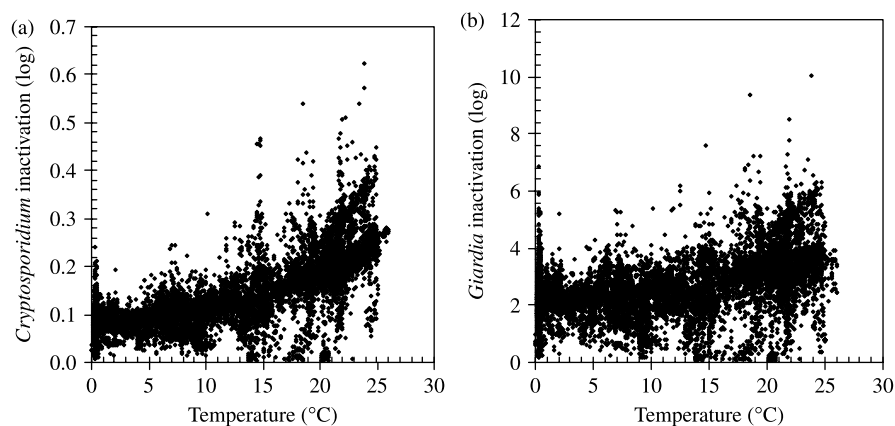


Figure 4 | Parasite inactivation by ozone according to temperature, and without downtime in plant B: a) *Giardia* inactivation; b) *Cryptosporidium* inactivation.

Selection of occurrence distribution

Figure 5 illustrates the distributions tested in the form of cumulative percentiles. A comparison of the three distributions shows that the constant distribution (f_{2-a}) is the most conservative approach, as it most likely overestimates the occurrence of parasites for concentrations under the detection limit. Furthermore, the log-normal distribution defined on the data set (including zero counts) shows a great sensitivity to the number of data available and the number of zero counts. In fact, the more zero counts there are, the lower the predicted concentrations under the detection limit will be. Compared with the measured concentrations ($> DL$), the predicted concentrations are lower, with the exception of the values at the higher end of the distribution (95th, 99th and 100th percentiles). These high concentrations in the extremities of the distribution exceed 1,000 oocysts/100 L, and sometimes even 10,000 oocysts/100 L, when the number of data available is limited and the percentage of zero counts is high (as is the case with *Cryptosporidium* occurrence at plant B). These extreme values are more characteristic of undiluted waste waters, and in no way reflect the concentrations realistically present in most surface waters, particularly in good quality surface waters. By contrast, the mixed distribution (log-Normal $> DL$ and uniform $< DL$) retained for this study, closely describes the observed parasite occurrence in raw water for values over the DL. The log-Normal distribution adequately describes the measured concentrations, while

the uniform distribution provides a probability equivalent to the non-detected data of having concentrations between 0 and the detection limit (1 oocyst/100 L). These results show the importance of the selection of the distribution on the production of unrealistically high estimates in the tailing of the distribution.

Uncertainty on parasite occurrence in raw water

The uncertainty on *Cryptosporidium* occurrence data (log-Normal distribution for concentrations $> DL$) from plant A is described by normal distributions of the mean ($\mu = 7.58$; $\sigma_{moy} = 2.9$) and standard deviation ($\mu_{ET} = 9.51$; $\sigma_{ET} = 2$). The results obtained by 2-D MCA can be represented by cumulative distributions to describe the variability, and credibility intervals to describe the uncertainty. The 2-D MCA simulations executed were based on 10,000 simulations (internal loop), each simulation using 250 iterations (external loop). Figure 6 presents the cumulative distributions of the median of the 5th percentile (limit under 95%) and the 95th percentile of the plant A *Cryptosporidium* occurrence data. The credibility interval at 90% of the median (4.80 oocysts/100 L) is [2.52 – 9.30]. Differentiating uncertainty from variability may be important in a risk assessment. However, experience shows that, for QMRA, it is possible that variability and uncertainty can be confounded, to the extent that it becomes difficult to consider them separately (Soller 2006). Indeed, in this study, the information about uncertainty is not available for all inputs parameters. Also, the use of

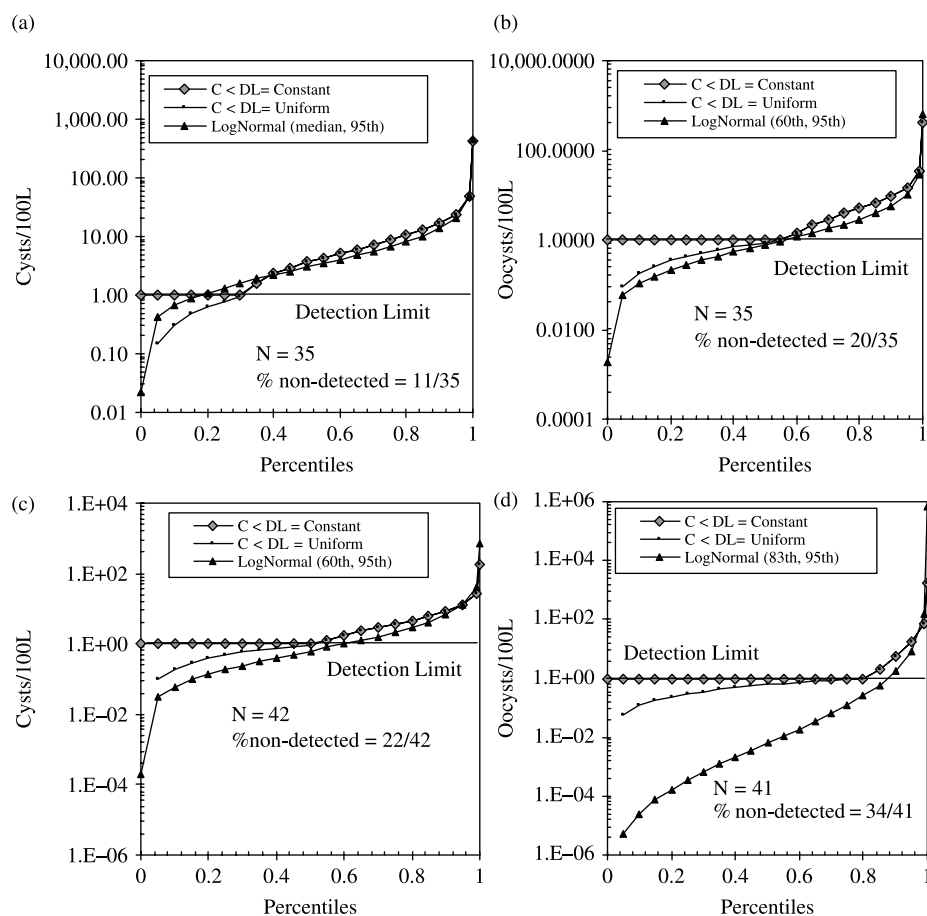


Figure 5 | Three distributions tested for parasite occurrence modelling: (a) *Giardia* in plant A; (b) *Cryptosporidium* in plant A; (c) *Giardia* in plant B; (d) *Cryptosporidium* in plant B. C: concentration, DL: detection limit.

2-DMCA (with Crystal-Ball) is long and limited for complex model. However, for purposes of simplification, the distinction between uncertainty and variability was not made in this study as our efforts were directed to quantify the influence of the treatment PDFs.

Adjustment of recovery by concentration

The concentrations must be adjusted to account for the partial recovery of parasites while performing ICR or 1623 methods. A MC simulation normally considers input variables as independent. However, it is quite common to encounter correlated variables. Taking into account the correlation between two independent variables can significantly influence the output and increase the precision of the model (Firestone *et al.* 1997). Using USEPA data on

recovery for method 1623 (Dr M. Messner, personal communication), correlation coefficients (R) of 0.8 and 0.7 were calculated between the measured concentrations and the recoveries of *Cryptosporidium* and *Giardia*, respectively. Consequently, we integrated this correlation into our model, which diminished the probability of the combination of a high concentration of parasites with a low recovery. For simplification purposes, a combined coefficient of correlation of 0.75 was considered for the entire data set.

Figure 7 illustrates the influence of integrating the correlation on (i) the evaluation of adjusted concentrations and (ii) the risk of infection. The results in Figure 7 (a) show a 52% reduction in mean concentration due to the autocorrelation effect (from 37.58 to 17.88 oocysts/100 L). Similarly, Figure 7 (b) shows a 28% reduction in the mean risk of infection (from 11.4 to 8.2 infections per 1,000).

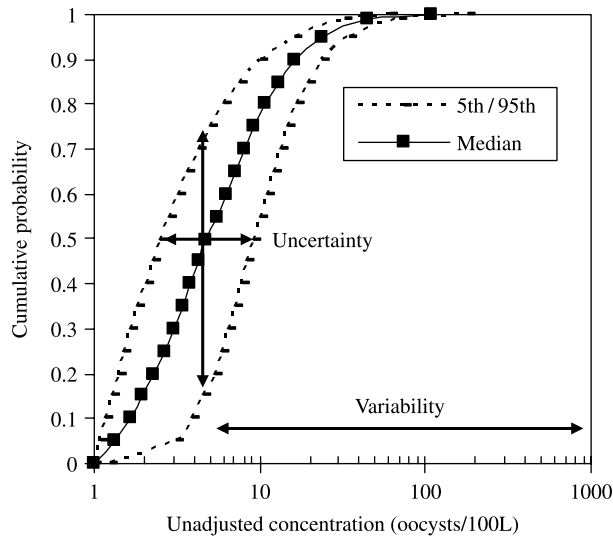


Figure 6 | *Cryptosporidium* concentration (above detection limit) in plant A with 90% credible interval. Concentrations are not adjusted for recovery.

More importantly, the reduction in the estimated values is greater at the mean than at the 90th and 95th percentile levels, particularly for the occurrence data. The results at the 99th percentile level may be less reliable, because they are subject to the accumulation of the extremities of the various parameter distributions of the model, which are most sensitive to the sampling process. Based on these results, we selected to integrate the correlation between parasite concentration and the rate of recuperation into our model.

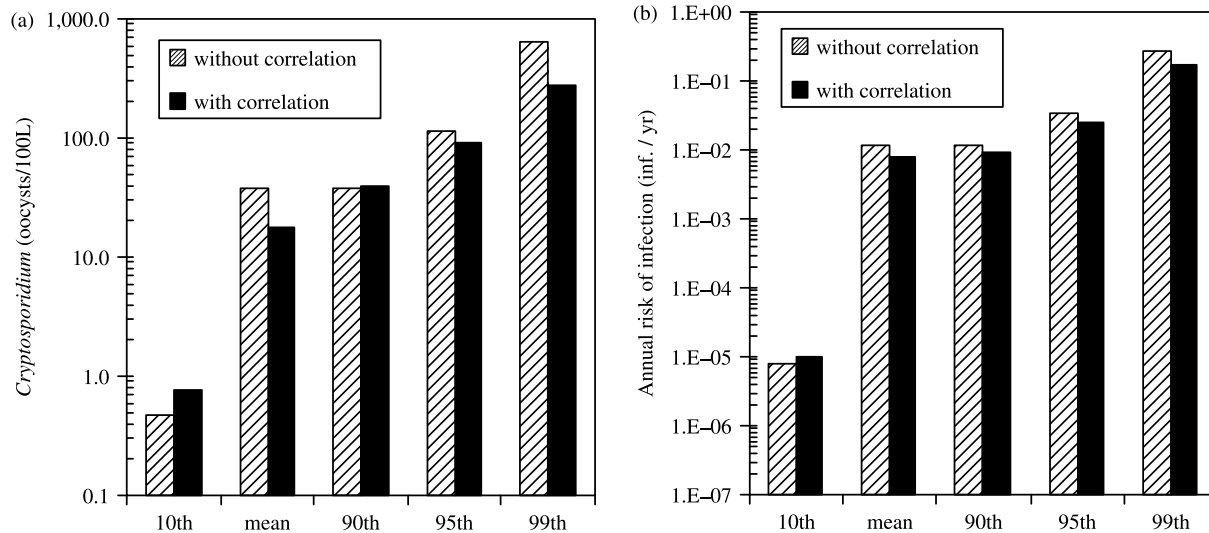


Figure 7 | Influence of correlating parasite concentrations in source water and analytical parasite recovery on the risk output (*Cryptosporidium* data in plant B): (a) unadjusted concentration; (b) annual risk of infection.

Selection of filtration models

Figure 8 presents the impact that the various approaches of modelling parasite removal by means of physical treatment by filtration on the estimated risk of infection. The three filtration approaches evaluated are summarized in Figure 1. The results of the simulations performed reveal fairly major differences in the risk outputs from the various approaches. For example, approach 2, which considers that $\log_{\text{parasites}} = \log_{\text{spores}}/0.57$, produces a lower risk than the risk computed using approach 1 ($\log_{\text{parasites}} = \log_{\text{spores}}$) and approach 3 ($\log_{\text{parasites}}$ fixed).

For the purpose of comparison, two configurations of filtration were considered: direct filtration with optimal coagulation (DFOC) and conventional treatment (CONV). They were then applied to the parasite occurrence distribution. The difference between approach 1 and approach 2 is greater with conventional chemically assisted filtration–CONV (Figure 8(b)) than with optimized direct filtration–DFOC (Figure 8(a)). For DFOC, the mean risk of infection estimated with approach 2 is 5.8-fold times (0.8 log) lower than the one estimated with approach 1 ($4.72E - 2$ as opposed to $8.15E - 3$). This reduction in risk reaches 125-fold (2 log) with CONV ($1.97E - 03$ as opposed to $1.58E - 05$). The same results are observed for the risk at the 90th and 95th percentile levels. Moreover, the risks estimated by approach 3 are generally comparable to

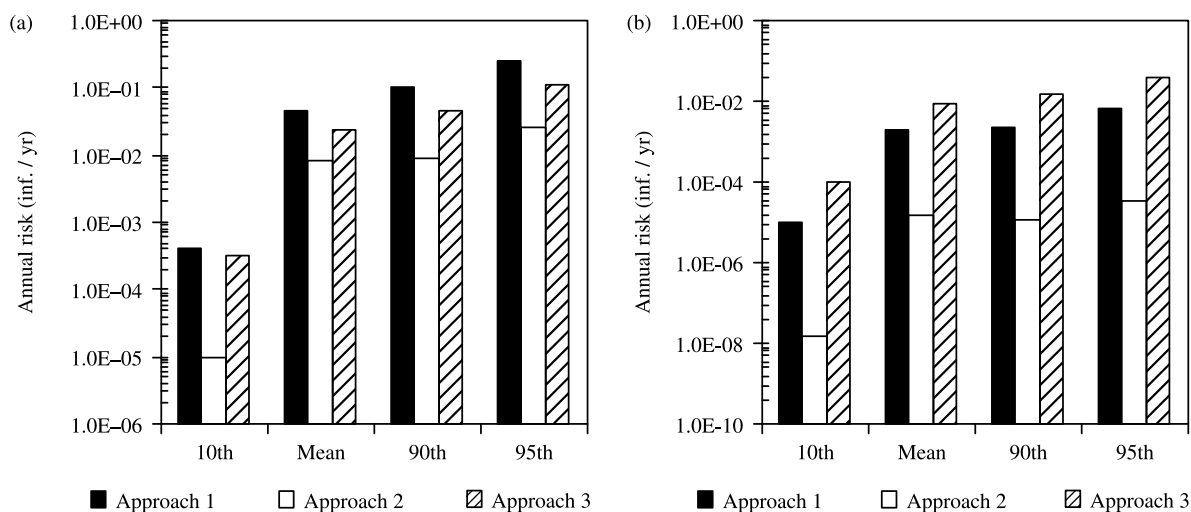


Figure 8 | Annual risk of *Cryptosporidium* infection in plant B according to three physical removal modelling approaches: (a) direct filtration with optimal coagulation (DFOC); (b) conventional treatment (CONV). Approach 1: $\log(\text{parasites}) = \log(\text{spores})$; Approach 2: $\log(\text{parasites}) = 0.57/\log(\text{spores})$; Approach 3: 2 log for DFOC and 2.5 log for CONV, if individual filtered turbidity are under 0.3 NTU (MDDEP 2002).

those estimated by approach 1 ($4.72\text{E} - 02$ as opposed to $2.46\text{E} - 02$ and $1.97\text{E} - 03$ as opposed to $9.33\text{E} - 03$ for DFOC and CONV respectively).

The first approach granting as *Cryptosporidium* log credits the observed ASF log removal appears most conservative. The second approach based on equivalent *Cryptosporidium*/spore log credits must be used with caution, because the correlation between spore removal and *Cryptosporidium* removal (Figure 2) is based entirely on pilot tests using spiked seeded parasites and relatively low duration of spiking. This correlation is appealing, but its application to full scale removal of pathogen may be premature. Hijnen *et al.* (2006) consider the data on prediction of oocyst removal on the basis of spores and sulphite reducing Clostridia inconclusive and lacking in the ability to account for the accumulation and delayed passage of parasites. Therefore, such correlation should be validated by actual plant pathogen removal measurements and longer term spiking (Hijnen *et al.* 2006). Finally, the third approach, based on a simplified regulatory assessment, can be used as an alternative to approach 1. It is evidently simple but possibly underestimates the true abatement of parasites in a given system. It is in line with the established relationship between turbidity removal and oocyst removal. However, this relationship has been questioned by the documented passage of total and viable infectious oocysts in filter effluents with very low (<0.1 NTU) turbidity (Aboytes *et al.* 2004).

CT calculation method for ozone

Modelling parasite inactivation by ozonation was integrated into the model developed in accordance with the two approaches described previously: (1) a detailed calculation in each tank corrected with SCADA and log book entries and (2) a plant simplified calculation based on the average residence time and the average residual which is commonly used in plants.

For the plant calculation, the variation in mean CT as a function of temperature is taken into consideration in the evaluation of parasite inactivation. The analysis of the mean CT_{10} data at plant B indicates a negative coefficient of correlation of 0.79 (obtained by the Crystal Ball software), since the efficacy of ozone in inactivating *Cryptosporidium* oocysts decreases considerably at low temperatures (Finch & Li 1999; Rennecker *et al.* 1999).

Figure 9 illustrates the impact of the two modelling approaches to calculate CT on the estimated reduction risk of infection by *Giardia* by ozonation alone. The comparison shows an underestimation of the risk in the plant calculation relative to the detailed calculation, which was fully expected. The extent of this underestimation shows the importance of using adequate CT calculation procedures rather than relying on a simplified approach sometimes favoured by utilities. Estimates were produced for the detailed CT calculation with (Figure 9(b)) and without (Figure 9(a)) actual ozonation

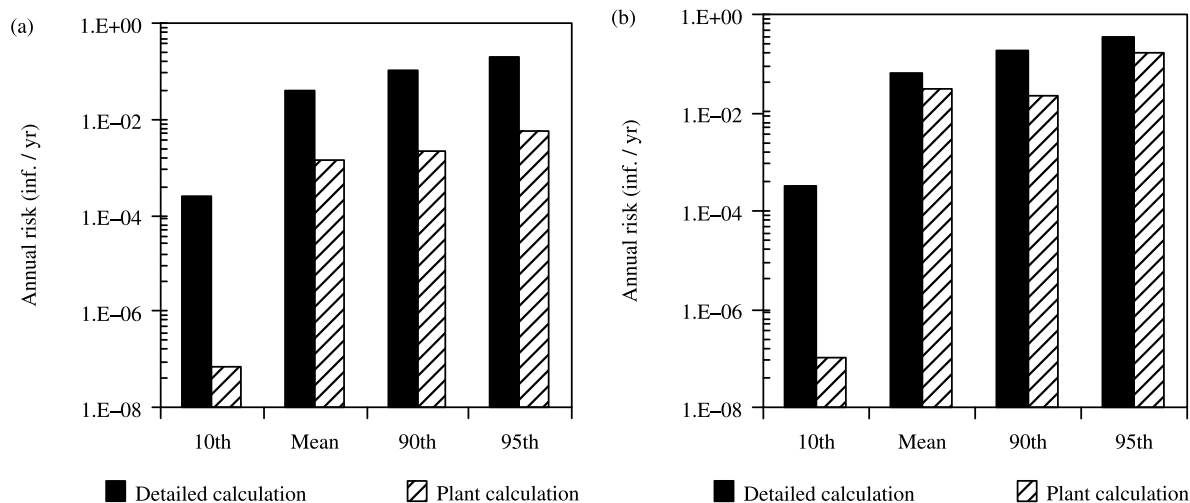


Figure 9 | Annual risk of infection with *Giardia* at plant B according to two CT calculation methods (Plant and Detailed) for ozone: (a) without downtime; (b) with downtime. Approach 1: Detailed Calculation; Approach 2: Plant Calculation.

downtime. With respect to the ozonation process without downtime (Figure 9(a)), we observe that the mean annual risk of infection by *Giardia* based on the detailed calculation is reduced by 27-fold (1.4 log) when the mean CT₁₀ approach (plant calculation) is considered (from $3.93E-02$ to $1.46E-03$). Incorporating the ozonation downtime (10.4% measured at plant B) in the simulation (Figure 9(b)) increases the mean annual risk of *Giardia* infection by about 1.5-fold (0.2 log) for the plant calculation. However, the mean risk is increased roughly 20-fold (1.3 log). The risks are $6.07E-02$ for detailed calculation to $3.02E-02$ for plant calculation. This situation reflects the fact that the higher the disinfection performance of a process is, the more the mean risk of infection will be impacted by short term treatment deficiencies (such as downtime). These results show the importance of adequately calculating CT and incorporating the downtime of the ozonation process in the risk calculation.

CONCLUSION

The QMRA model presented in this study was developed with the objective of assessing the risk of infection due to the presence of *Cryptosporidium* and *Giardia* in raw and treated waters at two filtration plants. The application of the model aimed to show the relative impact of the selection of the model's input parameters, particularly the distributions

of parasite occurrence in source water, of granular filtration performance and of ozone inactivation.

The Monte Carlo simulations performed demonstrate the importance of the selection of PDF for modelling the physical treatment (DFCO and CONV) and the method of calculating inactivation by ozonation on the measurement of the risk of infection by *Cryptosporidium* and *Giardia*. For the physical treatment, there are fairly major differences in the risk of infection by *Cryptosporidium*, depending on the modelling approach considered. The three approaches evaluated describe the removal of oo(cysts) by various configuration of filtration: (1) $\log_{\text{parasites}} = \log_{\text{spores}}$; (2) $\log_{\text{parasites}} = 1.7 \times \log_{\text{spores}}$; and (3) $\log_{\text{parasites}} = f(\text{Turbidity-UTN})$. The results indicate a significantly lower relative risk while using the second approach. The residual mean annual risks of infection by *Cryptosporidium* evaluated after conventional treatment are $1.97E-03$, $1.58E-05$ and $9.33E-03$ for the first, second and third filtration approach respectively. Granting a minimum value of equivalent log removal credits based on the spore removal performance could be conservative, if data from seeded *Cryptosporidium* pilot filters is indicative of actual oocyst removal in full-scale filters. By contrast, granting larger log *Cryptosporidium* removal credits than those observed for spores should be used with care, because the correlation between spore removal and *Cryptosporidium* removal (Figure 2) is most likely site specific, reflecting local process operation and

water quality fluctuations. A higher credit should be validated by parasite abatement measurements at full-scale, which remains a challenging task. The third approach, based on a simplified regulatory estimation, can be used as an alternative in the case where spore removal data are not available.

It is well established that the predicted inactivation by ozonation is directly influenced by the method used for CT calculation. Therefore, the risk of infection by *Giardia* assessed for the ozonation process will also depend on the method of calculation. Two methods were evaluated: a simplified calculation performed in the plant, which corresponds to a mean CT_{10} on all the ozone contactors; and a detailed calculation, which takes into consideration the dominant effect on total parasite inactivation of the tank which performs less well. The Monte Carlo simulations reveal the importance of the underestimation of the risk by the inexact simplified calculation relative to the detailed calculation ($1.46E - 03$ as opposed to $3.93E - 02$ for the mean risk of infection by *Giardia*). The practice of averaging residual concentrations is incorrect as it does not account for the severe impact on inactivation of blending waters with large differences in ozone exposure. Unfortunately, it is used by several utilities. This situation reflects the fact that inactivation is an exponential phenomenon: the lowest inactivation on parallel ozone contactors will dominate the overall performance of the process.

Risk estimates derived from this work are significantly higher than the proposed USEPA target of $1E - 04$ annual infection risk for *Giardia*. Including the validated SCADA data to evaluate risk estimate has an important impact on the outcome of the risk analysis. However, in the absence of significant epidemiological evidence of disease in the area served by the plants (less than 7 cases per 100,000), it is believed that the model used overestimates the actual risk of infection. The risk estimated by the model is a relative risk of infection, which does not take into account the variability of the immune response in the exposed population. The values of risk of infection do not translate directly to the number of illness cases reported. Therefore, this model under its current form should be used to compare the relative improvement of risk associated with various treatment options rather than to predict actual health outcomes.

Future efforts to reduce uncertainty should be targeted towards a better estimation of (i) parasite occurrence in raw

water, (ii) the dose-response relation and (iii) the infectious oo(cyst) fraction. Similarly, the spore removal data could play the role of a parasite removal indicator, but only with adequate plant validation. Finally, the correlation between parasite inactivation and parasite concentration in raw water should be evaluated.

ACKNOWLEDGEMENTS

We wish to acknowledge the support of the Industrial-NSERC Chair in Drinking Water at École Polytechnique de Montréal. The industrial partners of the Chair include the City of Montreal, the City of Laval and John-Meunier Inc/Veolia. We also wish to acknowledge the support of Dr. Michael Messner (USEPA) for some helpful discussions and for providing some data set needed to complete this work.

REFERENCES

- Aboytes, R., Di Giovanni, G. D., Abrams, F. A., Rheinecker, C., Mcelroy, W., Shaw, N. & LeChevallier, M. W. 2004 Detection of infectious *Cryptosporidium* in filtered drinking water. *J. AWWA* **96**(9), 88–98.
- Ashbolt, N. J., Grabow, W. O. K. & Snozzi, M. 2001 Indicators of microbial water quality. In *Water Quality: Guidelines, Standards and Health Risk Assessment and Management for Water-related Infectious Disease* (ed. L. Fewtrell & J. Barttram). IWA Publishing and World Health Organization (WHO), London, United Kingdom, chapter 13.
- Barbeau, B. 1996 *Évaluation des Bactéries Sporulantes Aérobies comme Indicateur de l'Efficacité du Traitement d'une Filière d'Eau Potable*. École Polytechnique de Montréal-Génies Civil, Géologique et des Mines.
- Barbeau, B., Morissette, C. & Prévost, M. 1999 *Évaluation de la filtration directe à l'usine Atwater de la Ville de Montréal (rapport final)*. Chaire Industrielle CRSNG en Eau Potable, Montréal, Québec, Canada.
- Barbeau, B., Payment, P., Coallier, J., Clément, B. & Prévost, M. 2000 Evaluating the risk of infection from the presence of *Giardia* and *Cryptosporidium* in drinking water. *Quant. Microbiol.* **2**(1), 37–54.
- Barbeau, B., Huffman, D., Mysore, C., Desjardins, R. & Prévost, M. 2004 Examination of discrete and confounding effects of water quality parameters during the inactivation of MS2 phages and *Bacillus subtilis* spores with free chlorine. *J. Environ. Eng. Sci.* **3**(4), 255–268.
- Barbeau, B., Huffman, D., Mysore, C., Desjardins, R., Clément, B. & Prévost, M. 2005 Examination of discrete and confounding

- effects of water quality parameters during the inactivation of MS2 phages and *Bacillus subtilis* spores with chlorine dioxide. *J. Environ. Eng. Sci.* **4**(2), 139–151.
- Barbeau, B., Carrière, A. & Prévost, M. 2006 *2006 Rapport Synthèse des Essais Pilotes de Filtration Granulaire à l'Installation de Traitement Charles-J. Des Bailleurs de la Ville de Montréal*. École Polytechnique de Montréal, Chaire Industrielle CRSNG en traitement et distributions des eaux potables, Montréal, Québec, Canada.
- Bellamy, W. D., Finch, G. R. & Haas, C. N. 1998 *Integrated Disinfection Design Framework*. American Water Works Association Research Foundation and American Water Works Association, Denver, Colorado, USA.
- Broséus, R., Barbeau, B. & Bouchard, C. 2006 *Validation of Full-scale Ozone Contactor Performance using Biodosimetry*. American Water Works Association-Water Quality Technology Conference, Denver, Colorado, USA.
- Brown, R. A. & Cornwell, D. A. 2005 *Utilizing Spore Removal to Monitor Plant Performance for Cryptosporidium Removal*. American Water Works Association-Annual Conference, San Francisco, California, USA.
- Clark, S. C., McGuire, P. E., Morabbi, M., Hargy, T., Chandler, J. & Wiginton, J. 2001 *Softening: the Ultimate Microbial Tool?* American Water Works Association-Water Quality Technology Conference, Nashville, Tennessee, USA.
- Coffey, B. M., Huck, P. M., Maurizio, D. D., Emelko, M. B., Douglas, I. P. & Van Den Oever, J. 1999 *The Effect of Optimizing Coagulation on the Removal of Cryptosporidium parvum and Bacillus subtilis*. American Water Works Association-Water Quality Technology Conference, Tampa, Florida, USA.
- Cornwell, D. A., MacPhee, M. J., Brown, R. A. & Via, S. H. 2003 Demonstrating *Cryptosporidium* removal using spore monitoring at lime-softening plants. *J. AWWA* **95**(5), 124–133.
- Di Giovanni, G. D., Hashemi, F. H., Shaw, N. J., Abrams, F. A., LeChevallier, M. W. & Abbaszadegan, M. 1999 Detection of infectious *Cryptosporidium parvum* oocysts in surface and filter backwash water samples by immunomagnetic separation and integrated cell culture-PCR. *Appl. Environ. Microbiol.* **65**(8), 3427–3432.
- Dugan, N. R., Fox, K. R., Owens, J. H. & Miltner, R. J. 2001 Controlling *Cryptosporidium* oocysts using conventional treatment. *J. AWWA* **93**(12), 64–76.
- DuPont, H. L., Chappell, C. L., Sterling, C. R., Okhuysen, P. C., Rose, J. B. & Jakubowski, W. 1995 *The infectivity of Cryptosporidium parvum in healthy volunteers*. *N. Engl. J. Med.* **332**(13), 855–859.
- Eisenberg, J. N., Seto, E. Y., Colford, J. M. Jr., Olivieri, A. & Spear, R. C. 1998 An analysis of the Milwaukee *Cryptosporidiosis* outbreak based on a dynamic model of the infection process. *Epidemiology* **9**(3), 255–263.
- Emelko, M. B. 2001 *Removal of Cryptosporidium parvum by Granular Media Filtration (Thesis)*. University of Waterloo, Department of Civil Engineering, Ontario, Canada.
- Emelko, M. B., Huck, P. M. & Douglas, I. P. 2003 *Cryptosporidium* and microsphere removal during late in-cycle filtration. *J. AWWA* **95**(5), 173–182.
- Emelko, M. B., Huck, P. M. & Coffey, B. M. 2005 A review of *Cryptosporidium* removal by granular media filtration. *J. AWWA* **97**(12), 101–115.
- Finch, G. R. & Li, H. 1999 Inactivation of *Cryptosporidium* at 1°C using ozone or chlorine dioxide. *Ozone Sci. Eng.* **21**(5), 477–486.
- Firestone, M., Fenner-Crisp, P., Barry, T., Bennett, D., Chang, S., Callahan, M., Burke, A. M., Michaud, J., Olsen, M., Cirone, P., Barnes, D., Wood, W. P. & Knott, S. M. 1997 *Guiding Principles for Monte Carlo Analysis*. Risk Assessment Forum, United States Environmental Protection Agency (USEPA), Washington, DC, USA.
- Gale, P. 1998 *Simulating Cryptosporidium exposures in drinking water during an outbreak*. *Water Sci. Technol.* **38**(12), 7–13.
- Haas, C. N. 1983 Estimation of risk due to low doses of microorganisms: a comparison of alternative methodologies. *Am. J. Epidemiol.* **118**(4), 573–582.
- Haas, C. N. & Kaymak, B. 2002 *Effect of Initial Microbial Concentration on Disinfectant Efficiency*. American Water Works Association Research Foundation, Denver, Colorado, USA.
- Haas, C. N. & Kaymak, B. 2003 *Effect of initial microbial density on inactivation of Giardia muris by ozone*. *Water Res.* **37**(12), 2980–2988.
- Haas, C. N., Crockett, C. S., Rose, J. B., Gerba, C. P. & Fazil, A. M. 1996a *Assessing the risk posed by oocysts in drinking water*. *J. AWWA* **88**(9), 131–136.
- Haas, C. N., Joffe, J., Anmangandla, U., Jacangelo, J. G. & Heath, M. 1996b *Water quality and disinfection kinetics*. *JAWWA* **88**(3), 95–103.
- Haas, C. N., Rose, J. B. & Gerba, C. P. 1999 *Quantitative Microbial Risk Assessment*. John Wiley & Sons, New York, USA.
- Havelaar, A. H., De Hollander, A. E. M., Teunis, P. F. M., Evers, E. G., Van Kranen, H. J., Versteegh, J. F. M., Van Koten, J. E. M. & Slob, W. 2000 *Balancing the risks and benefits of drinking water disinfection: disability adjusted life-years on the scale*. *Environ. Health Persp.* **108**(4), 315–321.
- Hijnen, W. A. M., Medema, G. J. & van der Kooij, D. 2004 Quantitative assessment of the removal of indicator bacteria in full-scale treatment plants. *Wat. Sci. Technol. Wat. Supply.* **4**(2), 47–54.
- Hijnen, W. A. M., Dullefont, Y. J., Bosklopper, K., Th, G. J., Schijven, J. F. & Medema, G. 2006 *Assessment of the Capacity of Slow sand Filtration to Eliminate Cryptosporidium oocysts*. American Water Works Association-Water Quality Technology Conference, Denver, Colorado, USA.
- Huck, P. M., Emelko, M. B., Coffey, B. M., Maurizio, D. D. & O'Melia, C. R. 2001 *Filter Operation Effects on Pathogen Passage*. American Water Works Association Research Foundation and United States Environmental Protection Agency, Denver, Colorado, USA.
- Hunter, P. R., Payment, P., Ashbolt, N. & Bartram, J. 2003 *Assessing Microbial Safety of Drinking Water: Improving*

- Approaches and Methods*. IWA Publishing, London, World Health Organization - Drinking Water Quality Series.
- LeChevallier, M. W. & Au, K.-K. 2004 *Water Treatment and Pathogen Control: Process Efficiency in Achieving Safe Drinking Water*. World Health Organization and International Water Association Publishing, London.
- LeChevallier, M. W., Norton, W. D. & Lee, R. G. 1991 Occurrence of *Giardia* and *Cryptosporidium* spp. in surface water supplies. *Appl. Environ. Microbiol.* **57**(9), 2610–2616.
- LeChevallier, M. W., Di Giovanni, G. D., Clancy, J. L., Bukhari, Z., Bukhari, S., Rosen, J. S., Sobrinho, J. & Frey, M. M. 2003 [Comparison of method 1623 and cell culture-PCR for detection of *Cryptosporidium* spp. in source waters](#). *Appl. Environ. Microbiol.* **69**(2), 971–979.
- Masago, Y., Oguma, K., Katayama, H., Hirata, T. & Ohgaki, S. 2004 *Cryptosporidium* monitoring system at a water treatment plant, based on waterborne risk assessment. *Water Sci. Technol.* **50**(1), 293–299.
- Mazoua, S. & Chauveheid, E. 2005 [Aerobic spore-forming bacteria for assessing quality of drinking water produced from surface water](#). *Water Res.* **39**(20), 5186–5198.
- Mazounie, P., Bernazeau, F. & Alla, P. 2000 Removal of *Cryptosporidium* by high rate contact filtration: the performance of the prospect water filtration plant during the Sydney water crisis. *Water Sci. Technol.* **41**(7), 93–101.
- Medema, G. & Ashbolt, N. 2006 *Microbiological Risk Assessment: a Scientific Basis for Managing Drinking Water Safety from Source to Tap. QMRA: its Value for Risk Management (preliminary web version)*. Microrisk. Available online: http://www.microrisk.com/uploads/microrisk_value_of_qmra_for_risk_management.pdf
- Medema, G. J., Hoogenboezem, W., van der Veer, A. J., Ketelaars, H. A. M., Hijnen, W. A. M. & Nobel, P. J. 2003 Quantitative risk assessment of *Cryptosporidium* in surface water treatment. *Water Sci. Technol.* **47**(3), 241–247.
- Messner, M. J. & Wolpert, R. L. 2002 *Information Collection Rule Data Analysis*, McGuire, M.J. *Cryptosporidium and Giardia Occurrence in ICR Drinking Sources - Statistical Analyses of ICR Data (Chapter 19)*. American Water Works Association and United States Environmental Protection Agency, Washington, DC, USA.
- Messner, M. J., Chappell, C. T. & Okhuysen, P. C. 2001 [Risk assessment for *Cryptosporidium*: a hierarchical Bayesian analysis of human dose response data](#). *Water Res.* **35**(16), 3934–3940.
- Messner, M., Shaw, S., Regli, S., Rotert, K., Blank, V. & Soller, J. 2006 [An approach for developing a national estimate of waterborne disease due to drinking water and a national estimate model application](#). *J. Water Health* **4**(Suppl. 2), 201–240.
- Ministère du développement durable, de l'environnement et des parcs (MDDEP) 2002 *Guide de Conception des Installations de Protection d'Eau Potable (Volume 1)*. Gouvernement du Québec, Québec, Canada.
- Mysore, C., Leparac, J., Barbeau, B., Amy, G., Hernandez, M., Dow, S., Huffman, D. & Marinas, B. 2003 *Impact of Water Quality on the Inactivation of Bacterial and Viral Pathogens*. American Water Works Association Research Foundation and United States Environmental Protection Agency, Denver, Colorado, USA.
- Neter, J., Wasserman, W. & Kutner, M. H. 1985 *Applied Linear Statistical Models*, 2nd edition. Richard D. Irwin, Inc., Homewood, Illinois, USA.
- Nieminski, E. C., Bellamy, W. D. & Moss, L. R. 2000 Using surrogates to improve plant performance. *J. AWWA* **92**(3), 67–78.
- Nilsson, P. 2006 *The Use of Water Treatment SCADA Data to Quantify Hazardous Microbiological Events and Risks Arising. A Case Study from Sweden*. Lund University, Department of Design Sciences, Division of Ergonomics and Aerosol Technology. Lund, Sweden.
- Okhuysen, P. C., Chappell, C. L., Crabb, J. H., Sterling, C. R. & DuPont, H. L. 1999 [Virulence of three distinct *Cryptosporidium parvum* isolates for healthy adults](#). *J. Infect. Dis.* **180**(4), 1275–1281.
- Payment, P., Berte, A., Ménard, B., Prévost, M. & Barbeau, B. 1997 *Évaluation des Risques à la Santé Humaine pour les Usines de Traitement de l'Eau Potable Tirant leur Source du Fleuve Saint-Laurent et de ses Tributaires*. Réseau Environnement–9^e Atelier sur l'Eau Potable, Montréal, Québec.
- Petterson, S., Signor, R., Ashbolt, N. & Roser, D. 2006 *Microbiological Risk Assessment: a Scientific Basis for Managing Drinking Water Safety from Source to Tap*. QMRA methodology (preliminary web version). Microrisk. Available online: http://www.microrisk.com/uploads/microrisk_qmra_methodology.pdf
- Regli, S., Rose, J. B., Haas, C. N. & Gerba, C. P. 1991 Modeling the risk from *Giardia* and viruses in drinking water. *J. AWWA* **83**(11), 76–84.
- Rendtorff, R. C. 1954 The experimental transmission of human intestinal protozoan parasites. II. *Giardia lamblia* cysts given in capsules. *Am. J. Epidemiol.* **59**(2), 209–220.
- Rennecker, J. L., Marinas, B. J., Owens, J. H. & Rice, E. W. 1999 [Inactivation of *Cryptosporidium parvum* oocysts with Ozone](#). *Water Res.* **33**(11), 2481–2488.
- Rice, E. W., Fox, K. R., Miltner, R. J., Lytle, D. A. & Johnson, C. H. 1996 Evaluating plant performance with endospores. *J. AWWA* **88**(9), 122–130.
- Rochelle, P. A., Marshall, M. M., Mead, J. R., Johnson, A. M., Korich, D. G., Rosen, J. S. & De Leon, R. 2002 [Comparison of in vitro cell culture and a mouse assay for measuring infectivity of *Cryptosporidium parvum*](#). *Appl. Environ. Microbiol.* **68**(8), 3809–3817.
- Rose, J. B., Haas, C. N. & Regli, S. 1991 Risk assessment and control of waterborne Giardiasis. *Am. J. Public Health.* **81**, 709–713.
- Schets, F. M., Engels, G. B., During, M. & de Roda Husman, A. M. 2005 [Detection of infectious *Cryptosporidium* oocysts by cell culture immunofluorescence assay: applicability to environmental samples](#). *Appl. Environ. Microbiol.* **71**(11), 6793–6798.
- Scott, K. N., Palencia, L. S., Merlo, R. P., Yates, R. S., Liang, S., DeLeon, R. 1997 Evaluation of *Cryptosporidium* and Surrogate Removal through a Full-scale Treatment Plant. *International*

- Symposium on Cryptosporidium*. Newport Beach, California, USA. Poster presentation.
- Signor, R. S. & Ashbolt, N. J. 2006 Pathogen monitoring offers questionable protection against drinking-water risks: a QMRA (quantitative microbial risk analysis) approach to assess management strategies. *Water Sci. Technol.* **54**(3), 261–268.
- Smeets, W. M. & Medema, G. J. 2006 Combined use of microbiological and non-microbiological data to assess treatment efficacy. *Water Sci. Technol.* **54**(3), 35–40.
- Smeets, P., Rietveld, L., Hijnen, W., Medema, G. & Stenström, T. A. 2006a *Microbiological Risk Assessment: a Scientific Basis for Managing Drinking Water Safety from Source to Tap. Efficacy of Water Treatment Processes* (preliminary web version). Microrisk. Available online: http://www.microrisk.com/uploads/microrisk_efficiency_of_water_treatment_processes.pdf
- Smeets, P. W. M. H., van Dijk, J. C. & Medema, G. J. 2006b *Pathogen Elimination by Drinking Water Treatment for Quantitative Risk Assessment*. American Water Works Association-Water Quality Technology Conference, Denver, Colorado, USA.
- Soller, J. A. 2006 Use of microbial risk assessment to inform the national estimate of acute gastrointestinal illness attributable to microbes in drinking water. *J. Water Health* **4**(Suppl 2), 165–186.
- Swertfeger, J., Metz, D. H., DeMarco, J., Braghetta, A. & Jacangelo, J. G. 1999 Effect of filter media on cyst and oocyst removal. *J. AWWA* **91**(9), 90–100.
- Teefy, S. M. & Singer, P. C. 1990 Performance and analysis of tracer tests to determine compliance of a disinfection scheme with the SWTR. *J. AWWA* **82**(12), 88–98.
- Teunis, P. F. M. & Havelaar, A. H. 2002 Risk assessment for protozoan parasites. *Int. Biodeter. Biodegr.* **50**(3–4), 185–193.
- Teunis, P. F. M., Medema, G. J., Kruidenier, L. & Havelaar, A. H. 1997 Assessment of the risk of infection by *Cryptosporidium* or *Giardia* in drinking water from a surface water source. *Water Res.* **31**(6), 1333–1346.
- Teunis, P. F. M., Nagelkerke, N. J. D. & Haas, C. N. 1999 Dose response models for infectious gastroenteritis. *Risk Anal.* **19**(6), 1251–1260.
- United States Environmental Protection Agency (USEPA) 1998 *Federal Register. Part V. National Primary Drinking Water Regulations: Interim Enhanced Surface Water Treatment (Final Rule)*. Office of Science and Technology-Office of Water, Washington, DC, USA.
- United States Environmental Protection Agency (USEPA) 2001a *Risk Assessment Guidance for Superfund. Volume III–Part A: Process for Conducting Probabilistic Risk Assessment. Advanced Modeling Approaches for Characterizing Variability and Uncertainty (Appendix D)*. Office of Emergency and Remedial Response, Washington, DC, USA.
- United States Environmental Protection Agency (USEPA) 2001b *Risk Assessment Guidance for Superfund. Volume III–Part A: Process for Conducting Probabilistic Risk Assessment. Communicating Risk and Uncertainties in Probabilistic Risk Assessments (Chapter 6)*. Office of Emergency and Remedial Response, Washington, DC, USA.
- United States Environmental Protection Agency (USEPA) 2001c *Method 1623: Cryptosporidium and Giardia in Water by Filtration/IMS/FA*. Office of Water (4603), Washington, DC, USA.
- United States Environmental Protection Agency (USEPA) 2003a *Federal Register. Part II. National Primary Drinking Water Regulations: Long Term 2 Enhanced Surface Water Treatment Rule; Proposed Rule*. Environmental Protection Agency-Office of Science and Technology-Office of Water, Washington, DC, USA.
- United States Environmental Protection Agency (USEPA) 2003b *Occurrence and Exposure Assessment for the LT2ESWTR Proposal*. The Cadmus Group, Watertown, Massachusetts.
- Yates, R. S., Green, J. F., Liang, S., Merlo, R. P. & De Leon, R. 1997 *Optimizing Direct Filtration Processes for Cryptosporidium Removal*. American Water Works Association-Water Quality Technology Conference, Denver, Colorado, USA.
- Zhang, J., Anderson, W. B., Smith, E. F., Barbeau, B., Desjardins, R. & Huck, P. M. 2005 *Development, Validation and Implementation of a Multiphase CFD Model for Optimization of Full-scale Ozone Disinfection Processes*. American Water Works Association-Water Quality Technology Conference, Québec, City Québec, Canada.
- Zhang, J., Huck, P. M., Stubble, G. C., Anderson, W. B. & Barbeau, B. 2006 A Comparison between a CFD Approach and CT₁₀ Method for Evaluation of Drinking Water Ozone Disinfection performance. *12th National Conference and 3rd Policy Forum on Drinking Water. Saint-John, New Brunswick, Canada*.
- Zmirou-Navier, D., Gofti-Laroche, L. & Hartemann, P. 2006 Waterborne microbial risk assessment: a population-based dose-response function for *Giardia* spp (E.MI.R.A study). *BMC Public Health* **6**, 122.

First received 1 November 2007; accepted in revised form 1 April 2008. Available online October 2008.