Titre: Title:	Application of Health Canada QMRA Model in 17 Canadian Drinking Water Treatment Plants
Auteur: Author:	Randa Tfaily
Date:	2015
Type:	Mémoire ou thèse / Dissertation or Thesis
Référence: Citation:	Tfaily, R. (2015). Application of Health Canada QMRA Model in 17 Canadian Drinking Water Treatment Plants [Master's thesis, École Polytechnique de Montréal]. PolyPublie. https://publications.polymtl.ca/1746/

URL de PolyPublie: PolyPublie URL:	https://publications.polymtl.ca/1746/
Directeurs de recherche: Advisors:	Benoit Barbeau, & Robert C. Andrews
Programme: Program:	Génie civil

UNIVERSITÉ DE MONTRÉAL

APPLICATION OF HEALTH CANADA QMRA MODEL IN 17 CANADIAN DRINKING WATER TREATMENT PLANTS

RANDA TFAILY

DÉPARTEMENT DES GÉNIES CIVIL, GÉOLOGIQUE ET DES MINES

ÉCOLE POLYTECHNIQUE DE MONTRÉAL

MÉMOIRE PRÉSENTÉ EN VUE DE L'OBTENTION

DU DIPLÔME DE MAÎTRISE ÈS SCIENCES APPLIQUÉES

(GÉNIE CIVIL)

MAI 2015

UNIVERSITÉ DE MONTRÉAL

ÉCOLE POLYTECHNIQUE DE MONTRÉAL

Ce mémoire intitulé :

APPLICATION OF HEALTH CANADA QMRA MODEL IN 17 CANADIAN DRINKING WATER TREATMENT PLANTS

présenté par : TFAILY Randa

en vue de l'obtention du diplôme de : Maîtrise ès sciences appliquées

a été dûment accepté par le jury d'examen constitué de :

Mme PRÉVOST Michèle, Ph. D., présidente

M. BARBEAU Benoit, Ph. D., membre et directeur de recherche

M. ANDREWS Robert C., Ph. D., membre et codirecteur de recherche

Mme McFADYEN Stéphanie, M. Sc., membre

DEDICATION

For my two little kids, my husband, and my parents.

ACKNOWLEDGEMENTS

I thank my director of research, Benoit Barbeau, for his support, advice and listening throughout this project. I thank Isabelle Papineau for her help and support at different stages of the project. I thank my co-director of research, Robert C. Andrews for his efficacious criticism and support.

As part of this project, an important collection of data was carried out with facilities of drinking water treatment Montreal and Toronto regions. I thank all those who provided the various data that have been essential to this project: Jennifer Lee, Josh Elliott and Amélie Lefebvre.

During my studies at Polytechnique, I met some amazing people who have become friends. I want to thank for their support, friendship and all the good times spent together. I know a little less about the students of the Chair, but you have always been very nice and I am delighted to have made your acquaintance.

I thank Julie Philibert and Jacinthe Mailly, which were resource persons important to me.

Finally, I want to thank my family. Thank you my husband for your support, and understanding throughout my studies. Thank you my two little angels Ryan and Mila for your unconditional love. Thank you my mother and my father for your continuous encouragement, my sister, my two brothers, and Ewa for your confidence on me and support during all stages of this project.

RÉSUMÉ

Sécuriser l'approvisionnement en eau potable est l'un des défis les plus importants pour l'industrie de l'eau potable. L'analyse quantitative du risque microbien (AQRM) est un outil qui sert à estimer le risque de la santé publique causé par l'exposition aux microorganismes pathogènes suite à la consommation d'eau potable. Une approche à barrières multiples de traitement d'eau potable est généralement adoptée pour assurer une bonne qualité d'eau potable. L'AQRM peut être considéré comme une étape de l'implantation d'une approche à barrières multiples.

Santé Canada a élaboré un modèle sur Excel d'AQRM qui fournit une évaluation des risques pour la santé humaine suite à l'exposition vi à l'eau potable à cinq agents pathogènes cibles: *E. coli* O157: H7, *Cryptosporidium*, *Giardia*, *Campylobacter* et rotavirus. Ce modèle simplifié est conçu pour un usage par les ingénieurs municipaux, les opérateurs et les décideurs locaux.

L'objectif général de ce projet est d'évaluer le retour d'expérience canadienne en ce qui concerne l'application du modèle d'AQRM de Santé Canada sur 17 installations de traitement. Cet objectif a été réalisé en trois étapes: [1] caractérisation de l'eau de source: Investigation de quatre méthodes disponibles pour représenter la concentration des agents pathogènes dans l'eau brute, [2] Les performances de traitement: évaluation de l'impact de diverses méthodes pour prédire l'inactivation sur les risques, [3] La caractérisation des risques: évaluation des risques microbiens pour la santé dans deux régions du Canada (Ontario et Québec). Cette analyse a permis de proposer une méthodologie d'utilisation du modèle de Santé Canada et mis en évidence les forces et les points de faiblesse du modèle qui devrait être améliorés dans le futur.

Pour mener à bien ce projet, les concentrations de trois organismes pathogènes de référence (*Coliformes fécaux* / *E. Coli, Giardia* et *Cryptosporidium*) ont été recueillies grâce à un suivi historique de la qualité de l'eau brute dans les 17 installations.

Pour la caractérisation de l'eau, une analyse des quatre approches suivantes (moyenne arithmétique avec zéros, moyenne arithmétique avec limites de détection (LD), la régression sur les statistiques d'ordre (ROS) par ProUCL, et la technique de la moyenne de Poisson (point estimate) ont généré des résultats proches pour *E. coli* et *Giardia*. Toutefois, les données LD se sont avérées comme un problème particulièrement important pour *Cryptosporidium*. La concentration moyenne de *Cryptosporidium* de toutes les installations a été augmentée de 8,0 fois lors de l'utilisation de la moyenne arithmétique avec LD au lieu de zéros et 4,5 fois en utilisant la

méthode de régression sur les statistiques d'ordre (ROS) par ProUCL. Enfin, l'utilisation de la moyenne arithmétique avec les zéros a été choisie pour caractériser la source d'eau pour la suite du projet (tel que prescrit actuellement dans le modèle).

La performance des chaînes de traitement a été calculée pour chaque WTP. En ce qui concerne les procédés physiques, les valeurs proposées par défauts par le modèle de Santé Canada ont été utilisées. Pour la désinfection, une comparaison de trois méthodes de calcul de CT (CT_{50}, CT_{10}, et N-CSTR) a été réalisée pour évaluer l'impact de la méthode choisie sur le risque estimé. L'impact de la méthode de calcul du CT était plus important pour *E. coli* que *Giardia*. Les méthodes de CT_{50} et CT_{10} prédisent souvent des valeurs qui atteignent les plafonds de performance arbitrairement imposées dans le modèle de Santé Canada. L'approche N-CSTR prédit des risques plus réalistes car il est moins sensible aux conditions d'inactivations élevées.

Pour estimer le risque des 17 usines, les calculs de CT ont par la suite été effectués avec l'approche CT_{50} (comme proposé dans le modèle Santé Canada).Les résultats de risque annuels pour les 17 usines révèlent que la plupart sont conformes aux niveaux de référence de l'EPA et de l'OMS. Les exceptions ont été trouvées seulement pour deux usines (WTP1 et WTP2) dans lequel les risques calculés pour *Giardia* et *Cryptosporidium* étaient au-dessus des niveaux de référence de l'OMS et de l'EPA.

Le modèle HC AQRM a été relativement simple à mettre en œuvre pour les 17 usines canadiennes. Le modèle s'est avéré utile pour estimer le risque de la santé lié aux agents pathogènes lors de la consommation de l'eau potable. Les différents scénarios évalués dans cette étude illustrent la flexibilité du modèle Santé Canada. Certaines limitations ont été remarqués au sein de cette étude et, en conséquence, des recommandations susceptibles d'améliorer la précision des résultats de risque, ont été proposées et discutées. Enfin, les résultats de risque fournis par le modèle HC d'AQRM sont semi-quantitatifs en raison des nombreuses simplifications et les sources d'incertitude et de la variabilité d'un tel exercice. Néanmoins, le modèle de Santé Canada devrait être vu comme un outil qui peut être intégré dans le contexte plus large d'élaboration d'un plan de sécurisation de l'alimentation en eau.

ABSTRACT

Securing drinking water supply is one of the most significant challenges for the drinking water industry. Quantitative microbial risk assessment (QMRA) is a tool used to estimate the public health risk from exposure to pathogenic microorganisms through drinking water consumption. A multiple barrier approach to drinking water treatment is generally adopted to ensure safe drinking water. QMRA can be used as a part of the multiple barriers approach.

Health Canada developed an Excel based QMRA model providing an assessment for human health risk while exposed to 5 index pathogens: *E. coli* O157:H7, *Cryptosporidium*, *Giardia*, *Campylobacter*, and Rotavirus. This model is designed for the municipal engineers, Water Treatment Plant (WTP) operators and local decision makers.

The general purpose of this project is to evaluate the Canadian experience with regards to the application of Health Canada's QMRA model on 17 WTPs located in Ontario and Quebec. This objective was realized into three steps: [1] The Source water characterization: Investigating four methods available to represent pathogens concentration in raw waters. [2] The treatment performance: Evaluating the impact of various methodologies for predicting inactivation on the overall risk outputs. [3] The risk characterization: Assessing the microbial health risks. These investigations allowed proposing a standardize methodology for using the Health Canada QMRA model, and highlighted the strengths and the weaknesses of the model which should be improved in the future.

For this investigation, the concentrations of three reference pathogens (Fecal coliform/*E. coli*, *Giardia* and *Cryptosporidium*) were collected from historical monitoring of raw water data for the 17 WTPs. For the source water characterization, an analysis of the following four approaches (Arithmetic mean with zeros, Arithmetic mean with DL, regression on order statistics (ROS) by ProUCL, and Point estimate) has been assessed to generate almost similar outputs for *E. coli* and *Giardia*. However, BDL data proved to particularly be an issue for *Cryptosporidium*. The average *Cryptosporidium* concentrations of all WTPs were increased of 8-folds while using the detection limit rather than zeros and 4.5-folds while using the regression on order statistics (ROS) means provided by ProUCL. Following this analysis, the Arithmetic mean was chosen to characterize the source water while proceeding with the rest of the study.

The treatment process performance has been calculated for each WTP. Regarding the physical processes, the HC QMRA model provides default values to represent the physico-chemical. For evaluating disinfection performance, a comparison of three CT calculation methods (CT_{50}, CT_{10}, and N-CSTR) was realized to assess impact of the selected method on the predicted health risk outcomes. The impact of CT calculation methods on risk estimates was more important for *E. coli* than *Giardia*. The CT_{50} and CT_{10} methods are more prone to capping due to their tendency to overestimate inactivation. The N-CSTR approach offered more realistic risk disinfection performances as it proved to be less sensitive to high inactivation conditions.

To evaluate risk estimates in the 17 WTPs, CT calculations were performed with the CT_{50} approach (as proposed in the model). The health risk outcomes predicted for the 17 WTPs revealed that most comply with the DALY and the USEPA reference risk levels. The exceptions were found for two WTPs (WTP 1 and WTP 2) in which *Giardia* and *Cryptosporidium* risk levels were above the WHO and USEPA reference levels.

The HC QMRA model proved to be relatively simple to implement in the 17 Canadian WTPs. The model proved to be useful in estimating pathogen health risk arising from consuming drinking water. The different scenarios assessed within this study illustrate the flexibility of the HC model. Some limitations were noticed within this study and accordingly some recommendations to improve the accuracy of the overall risk outcomes were proposed and discussed.

Finally, the risk outcomes provided by HC QMRA model are semi-quantitative due to the numerous simplifications and sources of uncertainty and variability of such exercise. Nevertheless, the HC model may be used by water treatment utilities as a tool to be integrated within the larger context of developing a water safety plan.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LISTS OF SYMBOLS AND ABREVIATIONS

CSTR	Continuous stirred tank reactor
CT	Disinfection concentration per contact time
DALY	Disability adjusted life year
E. coli	Esherichia coli
EHEC	E. coli enterohemorrhagic
EIEC	E. coli enteroinvasive
ETEC	E. coli enterotoxigenic
FC	Fecal coliform
HC	Health Canada
HRT	Hydraulic retention time
LT2SWTR	Long term 2 surface water treatment rule
LYL	Life years lost
Max	Maximum
Min	Minimum
mL	Milliliters
NPS	Nonpoint source pollution
PDF	Probability distribution function
QMRA	Quantitative microbiological risk assessment
ROS	Regression on order statistics
RTD	Residence time distribution
SWTR	Surface Water Treatment Rule
T_{10}	Baffling factor
T_{10}/T	Hydraulic efficacy

US EPA	United States Environmental Protection Agency
UV	Ultraviolet
WHO	World Health Organization
WSP	Water safety plan
WTP	Water treatment plant
YLD	Years lived with a disability

LIST OF APPENDICES

CHAPTER 1 INTRODUCTION

1.1 Overview of the problem

Water plays an essential role in human life. A poor quality of water can be a real menace to health and well-being. Securing drinking water supply is a significant challenge for the Canadian drinking water industry dealing with aging infrastructures. Risk analysis can be used to assess the impact of pathogens on human health. Ideally, risk assessment would be based on effective on-line pathogens monitoring of treated waters. However, monitoring all pathogens in water is costly and impractical due to the difficulty in their detection and random distribution (Field & Samadpour, 2007). Due to these limitations, good management practices from source to tap as become the recommended approach for the drinking water industry (Rizak & Sinclair, 2001). The multiple barrier approach is a group of procedures, process and tools while implemented; assure the necessary reduction of waterborne pathogens in drinking water prior to reach consumer. It provides a preventive action through the implementation of multiple effective barriers to minimize any possible failures within the system. Quantitative microbial risk assessment (QMRA) can be used as a part of a multiple barriers approach. QMRA is used to estimate the public health risk from exposure to pathogenic microorganisms through drinking water consumption. At this time, QMRA only addresses microbial risks related to treatment processes and as such does not replace a proper multiple barrier approach which also consider the impact of distribution.

Many challenges are encountered during the utilization of QMRA models. Properly characterizing source water contamination can be challenging as pathogen concentrations are often below detection limits. To address the issue of data below the detection limit (BDL) substitution methods are commonly used: where the data BDL are simply replaced by the detection limit, half its value or zero (Travis & Land, 1990). Improper handling of BDL samples can lead to risk calculation outputs that falsely exceed health-based targets (Dechesne & Soyeux, 2007; Jaidi, Brabeau, Carriere, Desjardins, & Prevost, 2009; Parkhurst & Stern, 1998). Secondly, monitoring of pathogens in source waters is not mandatory in all Canadian provinces. For example, Ontario and Quebec do not require *Giardia* and *Cryptosporidium* monitoring (Cook et al., 2013). In the absence of pathogen measurements, bacterial indicators data can be used as

alternative. However, any correlations found between bacterial indicators and protozoan pathogen concentrations have been weak and site specific and generated a lot of uncertainty (Harwood et al., 2005; Howard, Pedley, & Tibatemwa, 2006). Another problem with pathogen concentrations is related to the method efficacy, termed recovery, which is known to vary according to source water characteristics (Schijven, Teunis, Rutjes, Bouwknegt, & Husman, 2011). Proper source water characterization is not the only challenge of using QMRA as of the assessment of treatment process performances can also introduce significant bias. More specifically, as the primary disinfection is key in reducing the burden of waterborne disease, there is a need to use proper models to predict treatment performances. This is of utmost importance as an under- or overestimation of inactivation will either have cost implications due to the increase use of water treatment chemicals and energy or lead to an inadequate conclusion on the safety of a given water system (Jaidi et al., 2009).

Health Canada has developed an *Excel* based QMRA model that allows an assessment of the human health risk following exposure to 5 index pathogens: *E. coli* O157:H7*, Cryptosporidium, Giardia, Campylobacter*, and Rotavirus through drinking water. This model was elaborated in order to provide a user-friendly tool to Canadian water suppliers, which allows them to assess the risk associated to drinking water consumption of their system. The model provides as outputs the risk of infection, the risk of illness as well the health burden assessed according to the disability adjusted life-years (DALY) concept.

The general objective of this project is to evaluate the Canadian experience with regards to the application of Health Canada's QMRA model. This model was used on 17 Canadian water treatment plants (WTPs) located in the regions of Ontario and Quebec to assess the microbial risk associated to drinking water consumption. Considering the numerous challenges of using QMRA described earlier, this project proposes not only an assessment of the model application but also some technical recommendations to improve its performance or facilitate its use by the water industry.

1.2 Research Objectives

As stated above, the general aim of this study is to evaluate the Canadian experience with regards to the application of Health Canada's QMRA model on 17 WTPs. This objective will be investigated in the following manners:

1. Compare four different methods to best represent the microbial concentrations in source waters (arithmetic mean with zeros, arithmetic mean with DL, regression on order statistics (ROS), and point estimate),
2. Assess the impact on the health risk estimates of three alternative methods to evaluate inactivation by chemical disinfection processes, (CT_{50}, CT_{10} and N-CSTR),
3. Calculate and summarize the predicted microbial health risks associated to drinking water in 17 WTPs located in two Canadian provinces (Ontario and Quebec),
4. Recommend improvements to Health Canada QMRA model in order to improve predictions of risk and facilitate the use of the model.

1.3 Research Questions

Based on our research objectives, several questions were raised:

1. How should data below detection be handled during the source water characterization?
2. What is the impact of using CT calculation methods on the risk outcomes and what is the recommended optimal method?
3. Are the 17 WTPs under investigation able to meet the burden disease targeted DALY proposed by Health Canada and the risk of infection objectives proposed by USEPA?
4. What are other improvements that could be made to HC model?

1.4 Research Hypothesis

The Health Canada QMRA model has been developed with the objective of being a user-friendly model. If HC model is distributed within the water suppliers, it will allow them to better understand the microbial risks in their own system. Accordingly two specific hypothesis were suggested below:

1. The treatment of BDL data in source waters may bias concentrations mean by more than one order of magnitude. ***Falsifiability:*** *the assumption is refutable if the concentrations mean are all +/- 1 fold of each other for the four methods of raw water characterization assessed.*
2. The CT calculation methods for representing disinfection performances may bias risk estimates by more than one order of magnitude. ***Falsifiability:*** *the assumption is refutable if the risk estimates are all +/- 1 log of each other for the three CT calculation methods assessed.*
3. All WTPs under investigation meet the targeted risk of infections or DALY in treated waters. ***Falsifiability:*** *the assumption is refutable if one WTP is not meeting the targeted objectives.*

The first chapter of this thesis consists of a literature review on QMRA. Chapter two outlines the methodology used in this research. Chapter three presents the results of this investigation in a research paper submitted for publication to the *Journal of American Water Works Association*. Chapter four consists of a general discussion, which is followed, in chapter 5, by conclusions and recommendations for future research and application of the HC model.

CHAPTER 2 LITERATURE REVIEW

This literature review presents theoretical concepts needed to conduct a microbial risk assessment. First an overview of the framework of quantitative microbial risk assessment (QMRA) is presented. Second, a detailed description of Health Canada's approach to QMRA is described. Finally, the available methodologies to predict chemical disinfection performance are reviewed.

2.1 Risk Assessment

2.1.1 General Concepts and Definitions

a. ***Hazard***

A hazard is a status or agent that threatens the health, life, environment or property and could cause an adverse impact. In other word, the hazard is the potential of some situations to provoke damage. There are three types of hazard: natural hazards (such as epidemics, animal disease outbreaks, earthquakes…), technological hazards caused by accident or systems and structures failures, or human-caused incidents (such as terrorism) ((Homeland Security), 2013).

b. ***Risk***

There is a public dispute between the two terms of hazard and risk. Actually, a hazard combined with risk produces an accident or damage. It is the harmful effect, and the risk is the probability that it will occur. For example, a hurricane is a hazard but may produces very low damage if it does not touch a habited land. Therefore, the risk is a combination of the likelihood of damage or hurt and the degree of probability of such damage (Kaplan & Garrick, 1981).

c. ***Risk assessment***

Risk assessment is the process of qualitative and quantitative identification of the impact related to the exposure of individuals or populations to recognized hazards. The methods for assessing risk may differ from one field to another such as public health, environmental, or ecological risk assessment. Prof. Haas (Drexerl University, USA) was the first to introduce the concept of quantitative microbial risk assessment of waterborne disease through drinking water consumption by using dose-response models (Haas, 1983). Generally, risk assessment is conducted in four

steps: hazard identification, dose-response assessment, exposure assessment and risk characterization. This thesis focuses on microbial risk assessment associated to drinking water.

d. ***Quantitative Microbial Risk Assessment (QMRA)***

The Quantitative Microbial Risk Assessment (QMRA) aims to evaluate the infectious risk related to pathogenic microorganisms. It combines pathogens data (distribution, concentration) with the infectivity of those pathogens on humans, in order to estimate public health risks (WHO & OECD, 2003). Over the years, QMRA has been developed into a workable framework which has helped improve water quality, food safety and public health (Haas, Rose, & Gerba, 1999). Moreover, it helps support risk management decisions on a scientific basis.

In the drinking water industry, QMRA is widely used to estimate the public health risk from exposure to pathogenic microorganisms associated to drinking water consumption (Ashbolt, 2004; Howard et al., 2006; Signor & Ashbolt, 2006). In fact, this approach has been utilized to elaborate drinking water quality regulations worldwide (Bichai & Smeets, 2013; Harwood et al., 2005; Signor & Ashbolt, 2006; WHO, 2004). For example, the United States Environmental Protection Agency (USEPA) has introduced QMRA to set drinking water treatment requirements as early as 1989 for the Safe Drinking Water Treatment Rule. Since then, the World Health Organization (WHO) has promoted the use of water safety plans (WSP) along with health-based target evaluated using QMRA. The Dutch Drinking water Act of 2001 obliges drinking water suppliers to assess the human health risk associated to index pathogens such *Cryptosporidium* and *Giardia* every three years using a QMRA methodology (Schijven et al., 2011). The Australian water authorities are currently evaluating the option of incorporating QMRA process into the national drinking water guidelines (Bichai & Smeets, 2013). Finally, the Guidelines for Canadian Drinking Water Quality (GCDWQ) encourage the implementation of a risk-based, multi-barrier approach that includes QMRA (Health Canada, 2011b, 2012b; Krewski et al., 2004).

QMRA can be considered as additional tool to help improve drinking water quality management. This tool may be used to review treatment strategies to meet regulatory requirements, to evaluate the robustness of a treatment train and to determine set point values or critical situations where the risk of exposure is greater (Bichai & Smeets, 2013; Hartnett, McFadyen, Douglas, Robertson, & Paoli, 2007; Health Canada, 2011b; Howard et al., 2006; McFadyen et al., 2009). QMRA

allows a better identification of the water treatment system and helps in assess the impact of the variations in source water quality and treatment performance on the overall microbial risk. QMRA can also help in elaborating operational guidelines to ensure quality control and to minimize the health risk (Health Canada, 2011b). The operating staff within the water utilities would easily recognize the vulnerabilities within the treatment train and would react accordingly (Health Canada, 2011b).

2.2 QMRA Framework

The QMRA process consists of four steps; (1) hazard identification, (2) exposure assessment, (3) dose response assessment and (4) risk characterization (Haas et al., 1999).

2.2.1 Hazard identification

Hazard identification is defined as the effects of particular hazards on human health. It involves collecting information about pathogenicity and their tendency to cause human disease and illness. The epidemiological data such as endemic and epidemic disease investigations, hospitalization feedback, cases studies are very important to accomplish this step. It allows better understanding of pathogens characteristics, the particularity of host response in regard of immunity and multiple exposures, and highlights the various routes of disease transmission and causes of waterborne diseases (Haas et al., 1999).

While conducting QMRA to assess the human health risk associated to drinking water consumption, the hazard identification consists in the identification of infectious agents responsible of waterborne disease. The majority of hazards in drinking water system are derived from ingested enteric pathogens and their probable gastrointestinal illness (Medema & Ashbolt, 2006).

QMRA is usually performed only for reference pathogens which cover a wide range of health risks (Medema & Ashbolt, 2006) and typically includes bacteria (e.g. pathogenic *E. coli*), viruses (e.g. rotavirus) and protozoan parasites (e.g. *Giardia*). A proper risk assessment conducted on reference pathogens assumes a sufficient protection against other biological agents (Medema & Ashbolt, 2006).

2.2.2 **Exposure assessment**

The exposure assessment consists on defining the individual dose or set of doses to which individual consumers are exposed to (Teunis, P. F. M., Medema, Kruidenier, & Havelaar, 1997).

In this regard, many elements are needed to define proper exposure: the concentration of the pathogens in source water, the impact of the detection methods performance (termed recovery), the fraction of detected microorganisms actually infectious to humans, the performances of water treatment processes, and finally, the daily consumed volume of drinking water (Teunis, P. F. M. & Havelaar, 2002). The dose of ingested organisms (D) is calculated for each pathogen according to Eq. 2-1:

$$D = C_{SW} \times (1/R) \times I_{fraction} \times 10^{-(\text{Log } Removal)} \times V \quad ; \qquad \text{Equation 2- 1}$$

Where C_{SW} the concentration of microorganisms in source water, R the recovery of the analytical method (from 0 to 1), $I_{fraction}$ the infectious fraction of the detected pathogens (from 0 to 1), Log Removal is the overall performance of the water treatment plant (expressed on decimal log reduction and V the daily individual consumption (in Liter) of unboiled drinking water (Teunis, P. F. M. & Havelaar, 2002; Teunis, P. F. M. et al., 1997).

e. ***Concentration microorganisms in source water***

Source water quality is vulnerable to two categories of contamination: point source pollution and nonpoint source pollution (NPS). The point source pollution is defined as a single detectable source of contamination. The sewage treatment plants are considered as the main cause for point source microbial pollution, while for the NPS, the runoff is the major cause of that contamination (Nikolaidis, Heng, Semagin, & Clausen, 1998). The basis of NPS is attributed to wide area instead of particular discharge point, which making its control more difficult (Nikolaidis et al., 1998). These factors need better characterization for improving the risks accuracy (Krewski et al., 2004). The contribution of both contamination sources and the analysis of indicator organisms is recommended to properly assess the microbial quality of a given source water (Health Canada, 2011b).

i Sources of contamination

Understanding the dynamics of contamination sources is very important. It allows proper characterization of raw water quality, which is useful to conduct QMRA. There are two key sources of waterborne pathogens: the fecal and the non-fecal origin. The enteric pathogens with fecal origin are derived fromSaint Laurent, Canada the fecal material of animals (native and domestic) or from the human sewage (Ferguson, Husman, Altavilla, Deere, & Ashbolt, 2003). Both parasites and bacteria can originate from animals (zoonose) or humans while the source of enteric viruses are mainly associated to human sewage (Krewski et al., 2004). Animal slurries and farm waste is considered as an important source of contamination especially for *Cryptosporidium* oocysts, *Giardia* cysts, and Campylobacter (Carey, Lee, & Trevors 2004; Lack, 1999; Monis & Thompson, 2003). Many factors could facilitate the transmission of such contaminants into the environment and consequently lead to source water pollution. These may be related to morphology, hydrology and hydrogeology (water flow, slopes, soils…) or climate impact (rainfall, temperature, snowmelt…) (Dechesne et al., 2006). For example, following a wastewater treatment plant failure, important volumes of untreated sewage can be released and consequently pathogens are dispersed in the environment (Dechesne & Soyeux, 2007). Moreover, combined sewer overflows, storm water discharge and accumulation/release of pathogens from sediment will also lead to water contamination (Dechesne & Soyeux, 2007). Consequently, several challenges render difficult the proper characterization of source water contamination. In fact, the raw water concentrations vary according to many factors such as seasonal variations, sources of contamination, fate and transport of pathogens in the environment. This complexity urges the necessity to obtain site-specific data for pathogen density in source water.

ii Reference pathogens

Microbial indicators are used to estimate the probability of occurrence of other pathogens but are rarely well correlated to pathogens concentrations in source water (Payment & Locas, 2011). Many pathogens of public health importance do not exhibit the same behavior than their reference pathogens, and up to now microbial indicators are utilized mostly to indicate the probability of co-occurrence (Payment & Locas, 2011).

On the other hand, monitoring all pathogens in water is considered costly and impractical due to their rarity, difficulty in the culture, uneven distribution (Field & Samadpour, 2007). In fact,

waterborne microbial pathogens include enteric virus, bacteria and parasitic protozoa (Ferguson et al., 2003). A large fraction of surface water bodies exhibit impaired microbiological qualities (Field & Samadpour, 2007). To date, bacterial indicators (mostly *E. coli* and *enterococci*) are still considered as the most popular tools to assess microbiological quality of water (Field & Samadpour, 2007; Howard et al., 2006; Krewski et al., 2004). It is also a common practice in the field of QMRA (Roser et al., 2007; Soller et al., 2006; Soller et al., 2003) to characterize the risk using a suite of pathogens, which offers the benefit of encompassing a wide range of potentials risks arising from distinct environmental fate and infectivity. Several researchers have proposed lists of waterborne pathogens that should be included in QMRA calculations (Olivieri & Soller, 2002; Rosen, 2000). In general, representative of bacterial, viral and protozoan parasite pathogens are used for this purpose.

f. ***Recovery of detection methods***

The determinations for microbial occurrence, concentration, viability or infectivity have important impact on exposure assessment. Many culture methods have very low recovery rates, which may underestimate the pathogens loads and bias the risk calculation (Dechesne et al., 2006). In general, almost all available methods are at best tentative, given a variable and low recovery, and hard to differentiate the infectious strains to humans from the viable strains (WHO, 2004). For example, culture based methods which are commonly used to assess microorganisms in water often underestimate the overall microbial concentration as these method do not allow a characterization of viable but non-cultivable bacteria (McFeters, Pyle, Lisie, & Broadaway, 1999).

To assess the expected protozoan parasite concentrations, the numbers of observed (oo)cysts are corrected for the recovery of the detection method (Teunis, P. F. M. et al., 1997). The recovery is not constant, it varies from sample to sample, according to the physic-chemical properties of water, temperature, turbidity, the volume analyzed and the methods utilized, the age of (oo)cysts (Teunis, P. F. M. & Havelaar, 2002; WHO, 2004)….

g. ***Infection ability of organisms (Viability/Infectivity)***

Microorganisms viability and infectivity has an important significance while assessing the risk from pathogens in waters (WHO & OECD, 2003). In fact, dead or inactive pathogens will not

threaten public health (USEPA, 2008). Ignorance of the pathogen infectivity will overestimate the exposure assessment. In order to respond properly after pathogen detection, a rapid and accurate differential determination of infectious versus non infectious microbes is necessary (Johnson-White, Lin, & Ligler, 2007). This issue is still a topic of debate. Culture-based methods are classic measurements of viability (USEPA, 2008). However, it is a growth-based and time-consuming method and is not applicable to all target organisms (USEPA, 2008). Hence, molecular methods are a better alternative. Molecular methods allow a more rapid detection, and they are more sensitive and specific than culture-based detection methods (Keer & Birch, 2003). However, the conclusion on the infectivity and or viability may be impacted by the selection of the analytical method (USEPA, 2008).

h. ***Treatment efficiency***

The evaluation of water treatment efficiency is considered an essential part of the exposure assessment, whereas this efficiency is based on the performance of series of barriers. The treatment may be divided into two groups physical-chemical removal and disinfection. Indeed, performance evaluation of each treatment step provides a quantitative understanding of the treatment (LeChevallier & Au, 2004). Water treatment is a dynamic process, which varies according to the treatment train design criteria, operation and source water quality (LeChevallier & Au, 2004). However, assumptions for treatment parameters can cause significant uncertainties (Smeets, P. , Rietveld, Hijnen, Medema, & Stenström, 2006). During QMRA application, the monitoring of full-scale system increase the amount of details necessary while representing the treatment processes (Smeets, P. et al., 2006). Such information, once collected, can assure helpful data base for preventing any possible future risks (Smeets, P. et al., 2006). It was recommended by the Canadian Subcommittee on Drinking Water the use of surrogate or indicator organisms to evaluate water treatment processes performance (Krewski et al., 2004).

i. ***Drinking water consumption***

The number of microorganisms to which an individual is exposed to is defined by the volume of unboiled water consumed (Teunis, P. F. M. et al., 1997) multiplied by the concentration of organisms. The daily water consumption of individuals is variable from one person to another (Krewski et al., 2004; Teunis, P. F. M. & Havelaar, 2002) and from one day to another. The survey of various publications showed different assumptions for the daily consumption of

unboiled water. It is quite linked to the region (climate) and the culture of the population. Some QMRA were previously assessed with an assumed consumption of 2 liters per person per day (Regli, Rose, Haas, & Gerba, 1991; WHO, 2011c). While in the U.S.A., data of a large survey suggest that the average daily consumption per individual is of 0.96 L (Roseberry & Burmaster, 1992). On the other hand, Netherlands considered a smaller water consumption equal to 0.25 L (Teunis, P. F. M. et al., 1997). In Health Canada, the average daily volume of unboiled drinking water is estimated at 1 L per individual (Gale, 1996; Health Canada, 2012b) .

2.2.3 **Dose-response assessment**

The dose-response defines the relation between the dose ingested and the probability of infection or illness within the exposed population (Haas et al., 1999). This relationship are typically derived by applying high dose/risk levels in human studies (Van Ryzin, 1980). An extrapolation using mathematical relationship is necessary to assess the risk at lower exposure (Krewski et al., 2004).

There are two dose-response models utilized in QMRA, initially introduced by Haas (1983), the exponential and the beta-Poisson models. They assume that the risk at low levels is a linear function of the dose and that only one viable microorganism is required to initiate the infection process in vivo (Haas, 2002; Krewski et al., 2004). The exponential model assumes that the probability of a pathogen to cause an infection is independent of dose, whereas the beta-Poisson model follows the same principle as the exponential but it introduces a parameter that models non-constant survival and infectivity. The beta-Poisson model is an approximate of the exact form that uses the confluent hyper geometric function. This can be numerically complex when optimization and uncertainty analysis are needed (Teunis, P. F. & Havelaar, 2000)**.**

The exponential dose–response equation proposed by Haas (1983)

$$P_{inf} = 1 - exp^{-D \times r}; \qquad \text{Equation 2- 2}$$

Where P_{inf} the probability of infection at dose D, D the ingested dose, and *r* a specific parameter for each pathogen (it is considered as the probability that any single pathogen survives all barriers of the host defense systems and succeeds in initiating an infection) (Haas, 1983, 2002).

[2] The beta-Poisson equation according to Haas (1983):

$$P_{inf} = 1 - \left(1 + {}^{D}/_{\beta}\right)^{-\alpha}; \qquad \text{Equation 2- 3}$$

Where: α the model infectivity parameter, β the model shape parameter (Haas, 1983, 2002).

To this day the health impacts from exposure to some pathogens doses are uncertain, researchers need to better understand the dose response relation and its variation within pathogens and humans host conditions (Teunis, P. F. M. & Havelaar, 2002). The selection of the dose-response parameter has a direct impact on the predicted risk estimates. In the case of *Cryptosporidum*, the infectivity parameters (*r*) has been shown to vary as much as two order of magnitudes between three strains (TAMU, IOWA & UCP) (Okhuysen, Chappell, Crabb, Sterling, & DuPont, 1999). There is currently not international consensus on which dose-response parameters should be used but most QMRA have adopted the proposed values of the USEPA or WHO.

2.2.4 **Risk characterization**

In the risk characterization, the information from both exposure and dose-response assessments are integrated to assess the public health outcomes, as example in terms of annual probability of infection or in disability adjusted life years (DALY).

j. ***Annual probability of infection***

The probability of infection is the mostly used to express the risk outcomes for a given consumption of drinking water (Havelaar & Melse, 2003).

The annual probability of one or more infections ($P_{inf/year}$) is given by Eq.2-4:

$$P_{inf/year} = 1-(1-P_{inf})^{365}; \qquad \text{Equation 2- 4}$$

The probability of infection is considered a simple method and concentrates only on health risk, not on the severity of the health outcome. The USEPA use 10^{-4} infection/year as the acceptable annual risk of infection in their analysis (Regli et al., 1991).

k. ***DALYs***

The global burden of disease (GBD) Group of the WHO developed and promoted DALYs as opposed to $P_{inf/year}$ in order to assess the global burden of diseases, to set health policy priorities in different regions in the world (Murray & Lopez, 1997). DALYs is determined by Eq. 2-5:

$$DALY=YLL + YLD; \qquad \text{Equation 2- 5}$$

Where the number of years of life lost due to premature death (YLL) is calculated as the product of the number of deaths with standard life expectancy at the age of death, accumulated over all the health effects an agent is causing or aggravating (Havelaar & Melse, 2003) and the number of years lived with a disability or impairment (YLD), weighted with a factor from 0 to 1 for the severity of disability or disease, and it is calculated as the accumulated product over all diseases related to an agent, of the number of persons affected by a non-lethal disease with the duration of this disease and with a measure for its severity (Havelaar & Melse, 2003).

By considering the outcomes of serious diseases such as mortality and nonfatal health outcomes, DALYs will enable a comprehensive evaluation of health gain and losses of various intervention options. It establishes public health concepts (quality and quantity of life and social magnitude), using time as unit of measurement (Havelaar & Melse, 2003). The utilization of DALYs allows the comparison of various outcomes from different pathogens (Howard et al., 2006). This metric recognizes the difference between the severities of disease for various pathogens. The disease outcomes method required more information about agents and diseases, and implies several normative choices, such which reference life table to use for the lost life expectancy, the severity valuation procedures, etc. These assumed information may lead to important uncertainty (Masago et al., 2006; Xiao et al., 2012). The WHO (2011c) adopted a health based target of 10^{-6} DALY/person/year.

2.3 Health Canada Model

2.3.1 **Overview**

The Health Canada QMRA model was developed as part of the risk assessment process for enteric pathogens in drinking water. The model provides the disease impact for user-defined

scenarios aiming to represent site-specific drinking water systems (Health Canada, 2010, 2011a; McFadyen, Douglas, & J., 2011). The model is realized in a standard *Microsoft Excel* file containing approximately 16 spreadsheets. The first sheet named "Reference" provides technical information about reference values and equations used in the QMRA calculations. The second sheet "UserGuide" contains tips and suggestions for using the QMRA model. The third sheet "input_output" allows the users to input the following data: [1] population, [2] daily consumption, [3] raw water pathogen concentration, [4] and treatment barriers. Subsequently the model calculates in the same sheet the summary log removal, inactivation values and the mean risk estimate along with PDF graphs for each pathogen. The fourth sheet "Treatment" contains details about the treatment parameters used for physical removal values and chemical inactivation. The remaining sheets contain details for risk calculation, individual and overall treatment barriers, PDF for each pathogen and estimates about daily pathogen ingested.

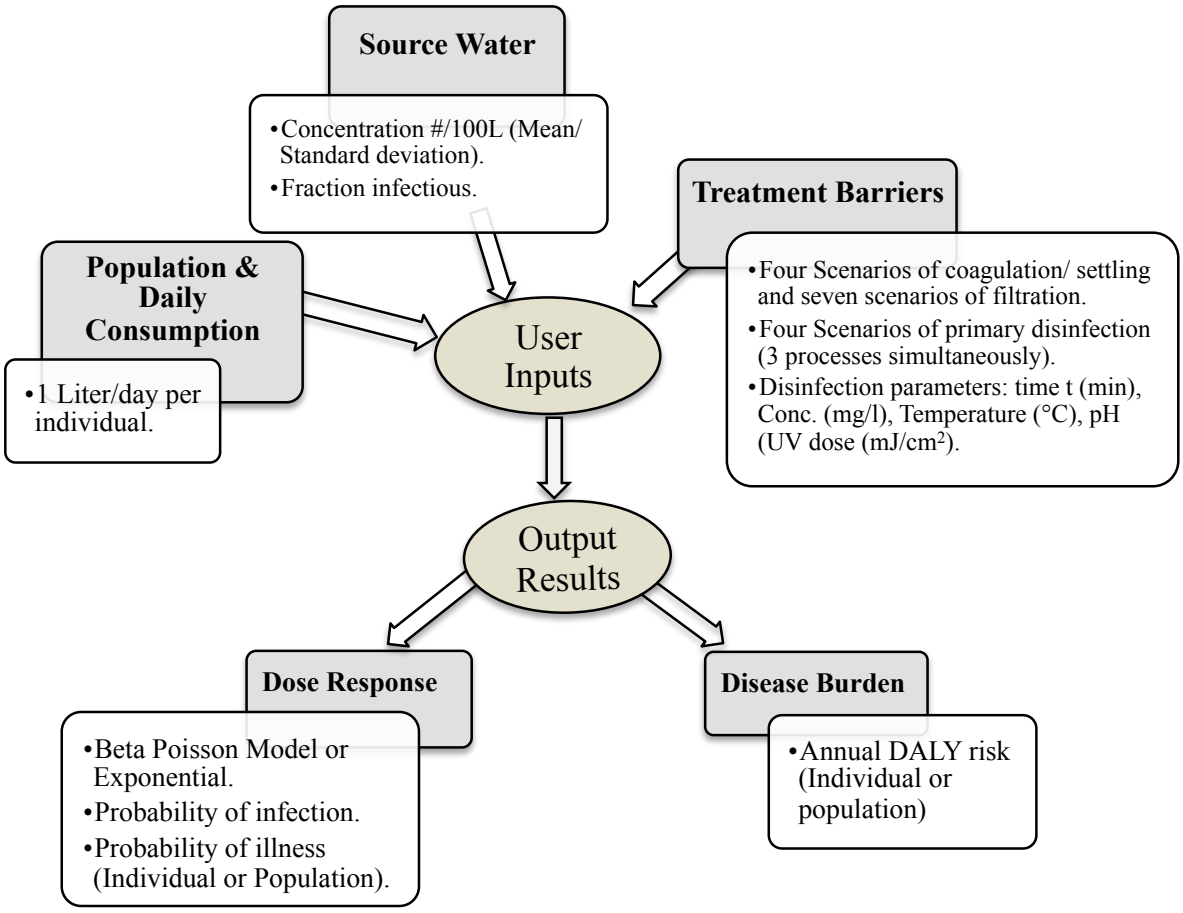

Figure 2-1 Health Canada QMRA model Flow Chart

2.3.2 **Reference pathogens**

Health Canada QMRA model estimates the health risk associated with five reference pathogens: *Cryptosporidium parvum*, *Giardia duodenalis*, Rotavirus, *Campylobacter* and *E. coli* O15:H7. These reference pathogens were selected to represent waterborne pathogenic bacteria, viruses and protozoa. The selection criteria for those pathogens were the occurrence of some organisms such in source water, the resistance to treatment, the high infectivity and virulence and the significant health impact (Hartnett et al., 2007). It is assumed that if health risk from the reference pathogens is acceptable, adequate safety is guaranteed from other waterborne pathogens (Schijven et al., 2011). Health Canada chose *Cryptosporidium parvum* and *Giardia duodenalis* as reference protozoa due to their high infectivity, the well-defined dose-response model for each organism, the resistance of *Cryptoporidium* oocysts to chlorine and the high prevalence of *Giardia* in the

Canadian waters which makes it a useful protozoan reference organism in the Canadian context. Rotaviruses were selected due their high potential to infect children, which could lead to severe outcomes and also to the availability of the dose-response model. Both *E. coli* O157:H7 and *Campylobacter* were considered as of particular significance to water industry. It could be life threatening in the case of *E. coli* O157:H7. The two bacterial organisms also have defined dose-response models (Health Canada, 2012a).

a. ***E. coli***

Escherichia coli (*E. coli*) is a gram negative, non-spore-forming bacteria typically found in humans and warm-blooded animals and does not multiply appreciably in the environment (Edberg, Rice, Karlin, & Allen, 2000). *E. coli* transmission occurs through the fecal-oral route which may be facilitated by water or food consumption (WHO, 2011b). *E. coli* strains are divided into six different major groups (Cabral, 2010), which not all of them is pathogenic. This classification was based on epidemiological evidence, clinical characteristics of disease, phenotypic traits, and specific virulence factors. The more important strains that can be transmitted via drinking water are enterotoxigenic (ETEC, namely O148), enterohemorrhagic (EHEC, namely O157) and enteroinvasive serotypes (EIEC, namely O124) (Scheutz & Strokbine, 2005). The diseases caused by EHEC are such as abdominal cramps; fever, vomiting and diarrhea which could progressed to bloody diarrhea (WHO, 2011b). The infection by EHEC of vulnerable population (such young and elderly) may lead to life-threatening disease (WHO, 2011b). One member of the EHEC group, *E. coli* (O157:H7) was previously involved in many disease outbreaks (Health Canada, 2000; Hunter, 2003). *E. coli* O157:H7 produces toxins known as verotoxins or Shiga-like toxins. The contamination of sources waters is of environmental health significance as *E. coli* O157:H7 can survive up to 4 to 12 weeks in surface waters (Edberg et al., 2000; Wang & Doyle, 1998).

The animals are the important reservoir of *E. coli* O157:H7. In fact cattle are known as the principal reservoir (Haas et al., 1999; WHO, 2004) and other hosts such as sheep, goats, deer, mammals and birds are also known to behave as a reservoir (WHO, 2011b). Finally, human sewage also contributes to *E. coli* O157:H7 contamination of surface waters (Health Canada, 2012a).

b. **Campylobacter**

Campylobacter infections are considered a major cause of bacterial gastroenteritis. *Campylobacter* is a ubiquitous bacteria, it could be shaped in many forms such as spiral, curved, S-shaped or rod-shaped. This bacterial genus is divided into 17 species and 6 subspecies where *C. jejuni* and *C. coli* are the most commonly known to infect humans (Dasti, Tareen, Lugert, Zautner, & Groß, 2010; WHO, 2011a). In fact, more than 90% of *Campylobacter* infections are caused by *C. jejuni* and *C. coli* (Dasti et al., 2010). Campylobacteriosis is characterized by severe diarrhea (sometimes bloody diarrhea), fever, nausea, vomiting and abdominal pain. Infections persist from 5 to 7 days and rarely lead to post-infection problems. Some complications are associated to *C. jejuni* infection, such as Bacteremia (fatal potential for HIV/AIDS), Guillain-Barré syndrome (which is an acute immune mediated disorder of the peripheral nervous system) and reactive arthritis syndrome with severe gastrointestinal associated with joint pain (Altekruse & Tollefson, 2003). Finally some infected individuals remain asymptomatic (Altekruse & Tollefson, 2003). The health significance of *Campylobacter* is not only associated to its clinical features (as described above) but also to the low infectious dose.

Campylobacter infections are transmitted through the fecal-oral route either from direct or indirect contact of infected individuals or from fecal contamination of animal origin. Consequently, human sewage and as well as fecal pollution of warm-blooded animals such wild birds, waterfowl, pigs, cattle, poultry, sheep, goats, dogs and cats have been defined as source of infection (Abulreesh, Paget, & Goulder, 2006; Gölz et al., 2014). After the excretion from animal digestive tract, the bacteria enter a non-cultivable stage. It is hard to multiply outside the host because *Campylobacter* required minimal temperature of growth between 30 to 42˚C and an aquatic environment to survive. *Campylobacter* survives in a non-cultivable form for long duration in water (Abulreesh et al., 2006). Finally, its occurrence in surface water has shown to follow seasonal patterns where higher concentrations are observed in summer and lower concentrations in winter (Jore et al., 2010).

c. ***Rotaviruses***

Rotaviruses are intestinal viruses known as one of the main cause of severe diarrheal disease in kids worldwide. By the age of 5 years old, around 95% of children will have been infected by minimum one rotavirus infection (Matthijnssens et al., 2008). Rotaviruses cause acute, watery,

dehydrating diarrhea in various species like mammals and birds (Rajendran & Kang, 2014). It consists of a double-stranded (ds) RNA genome of 11 segments with six structural viral proteins encodes and six non-structural [NS] (Matthijnssens et al., 2008; Rodger, Bishop, Birch, McLean, & Holmes, 1981).

Rotaviruses infection could lead to HRV disease, which consists in attacking the enterocytes in the small intestine. It lasts from 4 to 7 days with symptoms similar to gastrointestinal diseases. Symptoms of a rotavirus infection are mild fever, vomiting, watery diarrhea and abdominal pain. It appears typically within 2 days after exposure. Rotavirus infection could provoke severe dehydration such as dry, cool skin, dry mouth, and sunken eye in infants and kids (Surendran, 2008). Other diseases are associated to rotaviruses such as upper and lower respiratory infection and intussusception (Public Health Agency of Canada, 2011).

The typical modes of transmission for rotavirus are the fecal-oral, person-to-person or direct contact with contamination (Public Health Agency of Canada, 2011). The typical reservoir of rotaviruses is the humans, although some of the groups of rotaviruses have been found in pigs, foals, cats, dogs, calves and birds (Public Health Agency of Canada, 2011). The rotaviruses can persist and remain infectious in warm temperatures (30-35°C) for many days (Raphael, Sattar, & Springthorpe, 1985).

d. ***Cryptosporidium***

Cryptosporidium is a genus of apicomplexan protozoans (Corso et al., 2003). The taxonomy of *Cryptosporidium* is a serious challenge to molecular epidemiologists and biologist (Tzipori & Ward, 2002). *Cryptosporidium* have been classified in six species; they include *C. parvum* and *C. muris* as mammalian, *C. meleagridis* and *C. baileyi* as avian species *C. serpentis* in the reptiles and *C. nasorum* in fish. The most infectious species for human are *C. parvum* and *C. hominis* (Fayer, Speer, & Dubey, 1997). Its life cycles consists of an asexual reproduction phase, along with sexual reproduction that exhibits an unusual intracellular phase of development within its life cycle (Hunter, 2003).

Cryptosporidium infections cause the gastrointestinal illness cryptosporidiosis (Corso et al., 2003; Hunter, 1997). An infection can be induced with less than 10 oocycts in adult human volunteers (Okhuysen et al., 1999). It is transmitted through the fecal-oral route may it be person-to-person or animal-to-person transmission, water and food consumption (Tzipori & Ward,

2002). Sources of *Cryptosporidium* contamination of surface waters is mainly attributed to cattle and human feces (Health Canada, 2012b). *Cryptosporidium* oocysts can survive from 8 weeks to 24 weeks in surface waters (Carey et al., 2004; King, Keegan, Monis, & Saint, 2005; USEPA, 2010).

e. ***Giardia***

Giardia is a genus of flagellate parasite of the intestinal tract of a wide range of vertebrate hosts (Caccio & Ryan, 2008). It is divided in six species according to the morphology and ultrastructure of their trophozoites; namely *Giardia agilis* in amphibians, *Giardia ardeae* and *psittaci* in birds, *Giardia microti* and *Giardia muris* in rodents and *Giardia duodenalis* in mammals. Symptoms of *Giardia* infections are diarrhea, dehydration, abdominal pain, nausea, vomiting and chronic infections contributing poor growth and many nutritional and health disorders for children and disadvantaged groups of population (Adam, 2000; Thompson, 2004). *Giardia* is a recognized parasite in the humans also in the domestic animals, especially livestock, dogs, cats and numerous species of wild mammals and birds (Thompson, 2004). Due to its simple life cycle, which involves an environmentally resistant cyst, *Giardia* can be transmitted from one infected host to another directly or indirectly. The water is considered an important vehicle of transmission of *Giardia* to humans (Thompson, 2004). The survival of *Giardia* in surface waters varies between weeks to months (deRegnier, Cole, Schupp, & Erlandsen, 1989; Health Canada, 2012b)

2.3.3 **Source water characterization**

Pathogen concentrations in source water are assumed to fit to lognormal distribution. The log-normal PDF parameters are calculated using the arithmetic mean and standard deviation of available data. In absence of site-specific pathogen data the HC QMRA model provides default values for pathogens concentration based on a large literature review. Table 2-1 presents the pathogens concentration proposed by the model classified according to the perceived level of contamination of the source water. However, the use of site-specific data is favored as it provides useful information with regards to the vulnerability of a WTP (Schijven et al., 2011).

Table 2-1 Default values for pathogens concentrations at raw water proposed by Health Canada model.

Source Water	*Crypto.*[1]	*Giardia*	Rotavirus	*Campylobacter*	*E. coli*[2]
	Oocysts/100L	Cysts/100L	Virus/100L	CFU/100L	CFU/100L
Protected	0.1	0.5	0.1	100	100
Slightly impacted	1	5	1	1 000	10 000
Moderately impacted	10	50	10	10 000	100 000
Highly impacted	100	500	100	100 000	1 000 000

[1]*Crypto.* represents *Cryptosporidium*

[2]The values of concentrations presented are for generic *E. coli*; in the model *E. coli* O157:H7 is calculated as being equivalent to 3.49% of the generic *E. coli*.

2.3.4 **Treatment efficiency**

The treatment efficiency is determined by the sum of physical treatment removal and pathogen inactivation by disinfection. The model allows the user to choose from the following water treatment barriers: coagulation (coagulation only, coagulation/ filtration, coagulation/ filtration/ sedimentation), filtration (slow sand, granular high-rate, microfiltration, & ultrafiltration) and disinfection (chlorine, monochloramine, ozone, chlorine dioxide, UV). To assess the overall performance of a given WTP the performance of each individual process within the treatment train must be determined.

a. ***Physical-chemical processes***

Tables 2-2 and 2-3 present the recommended performances according to the type of coagulation and filtration processes. If site-specific data are available (e.g. aerobic spore removals), they can be used to evaluate treatment performance, otherwise the model provides weighted mean values based on an intensive literature review by both Health Canada (2008) and KIWA from Netherlands (2007).

Table 2-2 Recommended performances (as Log removal) used in Health Canada model for various coagulation processes used ahead of filtration.

Processes	*Log removals*				
	Crypto.[1]	*Giardia*	Rotavirus	*Campylobacter*	*E. coli*
Coagulation Only	0.00	0.00	0.00	0.00	0.00
Coagulation, Filtration	0.00	0.00	0.00	0.00	0.00
Coagulation, Filtration, sedimentation	1.86	1.61	1.76	1.55	1.55

[1]*Crypto.* represents *Cryptosporidium*

Table 2-3 Recommended performances (as Log removal) used in Health Canada model for various filtration processes.

Processes	*Log removals*				
	Crypto.[1]	*Giardia*	Rotavirus	*Campylobacter*	*E. coli*
Granular high-rate (no coag.)	1.11	1.23	0.77	0.55	0.55
Granular high-rate (coag.online/ direct.filt)	2.97	2.86	0.59	1.36	1.36
Granular high-rate (coag/direct.filt)	2.41	1.92	1.11	0.87	0.87
Granular rapid (coag/filt/sedim)					
Slow sand	4.66	4.88	2.18	2.69	2.69
Microfiltration	6.13	6.62	1.10	4.60	4.60
Ultrafiltration	6.41	6.18	4.12	10.00	10.00

[1]*Crypto.* represents *Cryptosporidium*

b. ***Inactivation***

For chemical disinfection, the inactivation is calculated using CT_{50} method. The model provides the possibility to calculate the performances of three stages of primary disinfection. Health Canada used the following formulas in the evaluation of disinfection performance for chlorination (Table 2-4) and ozonation (Table 2-5):

Table 2-4 Chlorination inactivation formulas

Organisms	*Equations to predict inactivation*	*Reference*
Giardia	$I = \frac{CT}{0.2828 \times pH^{2,69} \times [Cl_2]^{0.15} \times 0.933^{(Temp-5)}}$	(USEPA, 1999)
Crypto.[1]	$I = \frac{2 \times CT}{7200}$	(Korich et al., 1990
Rotavirus	$I = \frac{CT \times 0{,}3536 \times e^{0{,}0693 \times \mathrm{Temp}}}{-0{,}066658 \times \mathrm{pH}^3 + 1{,}58972 \times \mathrm{pH}^2 - 12{,}4303611 \times \mathrm{pH} + 32{,}336}$	(Sobsey, 1999)
Campylobacter	$I = \left(\frac{3.64}{0.5}\right) CT$	(Blaser et al., 1986)
E. coli	$I = 3.8962 CT^{0.3124}$	(Rice, Clark, & Johnson, 1999)

[1]*Crypto.* represents *Cryptosporidium*

Table 2-5 Ozonation inactivation formulas

Organisms	*Inactivation*	*Reference*
Giardia	$I = CT(0.0087Temp^2 - 0.0334Temp + 1.545)$	(USEPA, 1999)
Crypto.[1]	$I = 0.0397CT(1.09757)^{Temp}$	(USEPA, 2003)
Rotaviruses	$I = CT\mathrm{e}^{0.7423 \times \mathrm{e}^{(0{,}070 \times \mathrm{Temp})}}$	(USEPA, 1999)
Campylobacter	$I = CT60(\frac{S}{\min})(4.828Temp + 31.9)$	(Hunt & Mariñas, 1999)
E. coli	$I = CT60(\frac{S}{\min})(4.828Temp + 31.9)$	(Hunt & Mariñas, 1999)

[1]*Crypto.* represents *Cryptosporidium*

If the calculated inactivation exceeds the maximum values reported in the literature, the QMRA model will replace the extreme values by default capping values. Table 2-6 presents the capping values utilized by the model.

Table 2-6 Capping values used by Health Canada QMRA model

Disinfectants	*Capping Value (Max. Inactivation Log)*				
	Cryptosporidium	*Giardia*	Rotavirus	*Campylobacter*	*E. coli*
Free Chlorine	4	8	8	8	8
Chloramine	5	4	4	8	4
Ozone	6	4	4	8	8
Chlorine Dioxide	6	4	8	4	4
UV	5	4	5	5	5.5

2.3.5 **Water consumption**

In Health Canada, the volume of unboiled drinking water is assumed to be 1 liter daily per individual (Gale, 1996; Health Canada, 2012b) .

2.3.6 **Dose response**

The dose-response models utilized by the HC QMRA model are presented in table 2-7:

Table 2-7 HC model dose-response equations

Pathogens	*Model*	*Dose response parameters*			N_{50}[1]	*Reference*
		r	A	B		
Crypto.[3]	Exponential	0.018	–	–	38.5	(Messner, Chappell, & Okhuysen, 2001)
Giardia	Exponential	0.01982	–	–	35.0	(Rose & Haas, 1991)
Rotavirus	Beta-Poisson	–	0.265	0.4415	5.597	(Haas et al., 1999; Haas, Rose, Gerba, & Regli, 1993)
[2]*Campy.*	Beta-Poisson	–	0.024	0.011		(Teunis, P. F. M. et al., 2005)
E. coli O157:H7	Beta-Poisson	–	0.0571	2.2183	4.15×10^5	(Strachan, Doyle, Kasuga, Rotariu, & Ogden, 2005)

[1] N_{50} the median infectious dose given by:

$N_{50}=\text{Ln}(0.5)/-r$ for exponential dose-response model and $N_{50}= \beta/ (2^{1/\alpha} - 1)$ (Haas et al., 1999).

[2] *Campy.: Campylobacter jejuni*

[3]*Crypto.* represents *Cryptosporidium*

2.3.7 **Risk characterization**

The risk outputs are calculated for each pathogen separately using the appropriate dose-response equation. Instead of calculating point estimates based on the average dose in treated waters (cf. Eq. 1-1), the HC model generates a lognormal distribution from the arithmetic mean and the standard deviation of the pathogens concentrations input by the user as the first step in the model application. The raw water log-normal probability distribution function (PDF) is then divided artificially into 500 segments. The average pathogen dose in treated waters is calculated for each single segment separately by applying the constant log reduction value calculated using the various site-specific process performance inputs. Once this information is available (C_{TWi}), a Poisson distribution is used to predict the probability of finding from 0 to 40 organisms in the treated volume of water given a concentration C_{TW_i} and volume ingested V. The upper number of 40 organisms was selected arbitrarily as the risk of finding more than 40 organisms in 1 L of treated waters is insignificant. The elemental probability of being infected is obtained by multiplying the Poisson probability of having *N* organisms in treated waters by the risk of infection arising from N organisms as predicted using the appropriate dose-response models. This probability is multiplied by the weight-average to account for the relative contribution of each segment (0.002). This process is repeated for the 500-segmented risks. The final risk estimate is obtained by summing up the risk from the 500 segments. Eq. 2-6 summarizes the equation used for the calculation. For this example, the exponential model is presented but may be replaced by the Beta-Poisson model, if pertinent.

$$P_{inf} = \sum_{i=1}^{500}\left[0.002\times\left[\sum_{N=1}^{40}\left(\frac{(V\times C_{TW_i})^N}{N!}\right)\times(1-\exp(-r\times N))\right]\right]; \quad \text{Equation 2- 6}$$

Where C_{TW_i} the concentration of microorganisms in source water, V the volume of unboiled water ingested daily, r is a pathogen specific coefficient used to depict the dose-response curves of each reference pathogen and N doses ranged from 0 to 40 microorganisms per day.

Once the $P_{inf/year}$ is defined the probability of illness per person per year ($P_{ill/year}$) is assessed as follows:

$$P_{ill/year} = P_{inf/year}\times P_{ill.\ given\ infection} \qquad \text{Equation 2- 7}$$

Where the probability of illness given infection ($P_{ill.\ given\ infection}$) determined according to the values published in literature for each reference pathogen (Table 2-8).

Moreover, The HC model expresses the public health risk in terms of DALYs (Eq. 2- 5 & Eq. 2-8) to allow a comparison with the health based target of less than 10^{-6} DALY per person per year ($DALY_{/year}$) (Health Canada, 2011b, 2012b).

$$DALY_{/year} = P_{ill/year} \times DALY_{in\ case\ of\ illness} \qquad \text{Equation 2- 8}$$

Where the DALYs in case of illness per reference pathogen (Table 2-8) determined by Heath Canada based on a literature review and demographic information on the Canadian population.

In summary, the risk calculation outputs and figures provided by the HC model are [1] the probability of infection per person (daily and yearly), [2] the probability of illness yearly (per person and for the given population), [3] the annual DALY risk (per person and for the given population), and [4] graphs showing the probability distribution function (PDF). The acceptable annual DALY is suggested to comply with health-based target of less than 10^{-6} DALY.

Table 2-8 Dose-response models, probability of illness given infection and total DALYs per case of illness according to HC model.

Reference Pathogen	Type of model	Dose-response curves			P_{ill} given infection*		Total DALYs per case of illness†
		R	**β**	**Reference**	**Value**	**Reference**	
Crypto.‡	Exp.§	0.018		Messner et al. (2001)	0.7	Casman, Fischhoff, Palmgren, Small, and Wu (2000); Okhuysen, Chappell, Sterling, Jakubowski, and DuPont (1998)	1.70E -03
Giardia	Exp.	0.01982		Rose, and Gerba (1991)	0.24	Eisenberg et al. (2006); Macler, and Regli (1993)	1.70 E-03
Rotavirus	Beta-Poisson	0.265	0.4415	Haas et al. (1993)	0.88	Havelaar, and Melse (2003)	8.46 E-03
Campylobacter	Beta-Poisson	0.024	0.011	Teunis, P. F. M., Nagelkerke, and Haas (1999)	1**		4.60 E-03
O157	Beta-Poisson	0.0571	2.2183	Strachan et al. (2005)	1**		2.45E-02

*P_{ill}-Probability of illness
†As defined in the HC model
‡ Crypto.- *Cryptosporidium parvum*
§ Exp.- Exponential & ** Illness used in dose response model

2.4 Prediction of chemical disinfection performance

Disinfection has a major role in reducing the exposure to pathogenic microorganisms and the associated human health risk. Consequently, the selected approach used to assess disinfection performance is of importance. An underestimation or overestimation of disinfection performance will either have cost implications, due to unnecessary additional treatment process or create a false sense of safety for a system which is vulnerable to waterborne disease outbreaks. Many variables could influence disinfection performance; such as inactivation kinetics, disinfectant decay in water and reactor hydrodynamics (Pfeiffer & Barbeau, 2014).

There exist several methods to predict the disinfection performance most of which use the CT concept (product of disinfection residual and contact time). The HC QMRA model utilizes the CT_{50} approach. Other approaches such as the CT_{10} or N-CSTR may also be used for that purpose.

Chick-Watson (1906-1908) were the first to introduce the CT concept as an empirical rate equation to describe inactivation (Eq. 2-9) expressed as:

$$K=C^n.t\ ; \qquad \text{Equation 2- 9}$$

Where K is the constant for specific microorganism exposed under specific conditions, C the disinfectant concentration (mg/L); n the so-called coefficient of dilution; t the contact time (min) required for a fixed-percent inactivation (Clark, Read, & Hoff, 1989). Frequently, the value of the constant n is assumed equal to 1.

In 1989, the USEPA introduced the CT concept in the Surface Water Treatment Rule (SWTR) in order to evaluate the inactivation of *Giardia* cysts by disinfection (Clark et al., 1989). Many factors affect CT calculation such water temperature, contact time, pH, degree of mixing, turbidity, and disinfectant concentration (Clark et al., 1989). For chlorination two factors have major impact on the inactivation efficiency: the pH and the water temperature. Meanwhile for ozonation, the water temperature has the greatest influence on inactivation performance. In the SWTR, the EPA recommends a first-order Chick-Watson's law for describing the inactivation kinetics of disinfection processes:

$$ln(N/N_o) = -kCT \qquad \text{Equation 2- 10}$$

Where N and N_0 the concentration of microorganisms at time t and t = 0, C the disinfectant

residual concentrations (mg/L), T the contact time (min) and *k* the inactivation rate constant for a given type of disinfectant (L/mg.min), microorganism, pH, and temperature (Lev O., 1992).

The hydraulic of a system has a significant impact on microbial inactivation. Hence it is of relevance to accurately represent the hydraulic of a contactor in inactivation calculations. While assessing the exposure, several methods for measuring T could be used to predict the reactors performance, and the inactivation kinetics of disinfection. In this project, we compare three different approaches for T (T_{50}, T_{10} and N-CSTR method).

c. ***CT_{50} method***

T_{50} represents the time interval required for assuring the exposition of 50% of treated water to the disinfection. The use of T_{50} compared to the hydraulic retention time gives a very low safety factor (Lev & Regli, 1992). In most cases, it is inadequate measure to predict inactivation levels and does not consider the hydraulic efficacy. Often, the T_{50} is not available in a given water treatment train. The HC model, which uses the T_{50}, recommends using the theoretical average retention time (Volume/Flow) if the T_{50} is not available.

d. ***CT_{10} method***

T_{10} is another method for time characterization (T), recommended by the United States Environmental Protection Agency (USEPA) in 1989 (USEPA, 1991a). This regulatory approach is widely utilized in North America. T_{10} represents the time interval expected for the outlet concentration tracer to achieve 10% of its ultimate response, following inlet step perturbation (Lev & Regli, 1992). The CT_{10} method is generated from the CT by applying a "baffling factor" T_{10}/T which describe the degree of short-circuiting occurred within the basins (Smeets, P. W. M. H. et al., 2006).

$$T_{10}=Baffling\ factor\ x\ HRT; \quad \text{Equation 2- 11}$$

Where HRT is the hydraulic retention time. The baffling factor may vary from a low of 0.1 (perfectly missed reactor) to a high of 1.0 (plug flow contactor). The CT_{10} approach can guarantee a sufficient level of inactivation when low level of inactivation is required and large ratio between T_{10}/T prevails (Lev & Regli, 1992). The drawback of using the CT_{10} is that it

underestimates disinfection performance in conditions where inactivation is low whereas it overestimates disinfection performance in conditions where inactivation is high (Lev & Regli, 1992; Pfeiffer & Barbeau, 2014; Zhang, Huck, Anderson, & Stubley, 2007). In general it's a conservative scale-up design, could lead as much as 9.5 times the design based on HRT (Lev & Regli, 1992). Although this approach is conservative from a regulatory standpoint, it introduces a bias in the evaluation of chemical disinfection performance.

e. ***N-CSTR model***

First proposed by Lawer and Singer (1993), the partially segregated technique (also called N-CSTR method) considers that the contactor is composed of several single completely stirred reactors (CSTRs) in series, with constant disinfection equivalent to the effluent concentration (Pfeiffer & Barbeau, 2014). The N-CSTR model assumes that the liquid is perfectly mixed in each CSTR separately with different reactors. The number of CSTRs utilized does not have to match the actual number of chambers (N) in the contactor and can be fractional value (Pfeiffer & Barbeau, 2014). The number N of CSTRs is calculated based on the fit to a tracer curve of the hydraulics parameters (N and HRT). Based on the baffling factor (T_{10}/T), one can also calculate the theoretical value of N.

This method provides a more accurate description of the hydraulic behavior of a reactor and also considers the disinfectant decay within the reactor. N-CSTR method is less prone to over- or underestimate the inactivation performance in circumstances where low or high disinfectant decay is observed (Pfeiffer & Barbeau, 2014).

The number at the exit of the last contactor (j) is obtained from the following Eq. 2-12:

$$N_j = \frac{N_i}{\left(1+\frac{k_L C_j HRT}{N}\right)^{j-i}}; \qquad \text{Equation 2- 12}$$

Where N_j is the concentration of microorganisms after the j^{th} CSTR in series (mg/L), N_i is the effluent concentration of microorganisms of the previous CSTR (mg/L), k_L is the inactivation rate coefficient (L/mg.min) (Eq. 2-13), HRT is the hydraulic residence time (min), and N is the number of CSTRs in series for the entire contactor (Pfeiffer & Barbeau, 2014).

$$k_L\left(\frac{L}{mg.min}\right) = \frac{ln(10)*Inactivation\ (expressed\ in\ Log)}{CT}\ ; \quad \text{Equation 2- 13}$$

While C_j is the concentration of the disinfectant after the j^{th} CSTR, calculated as follow:

$$C_j = \frac{C_0}{\left(1+\frac{k_D THR}{N}\right)^j}\ ; \quad \text{Equation 2- 14}$$

Where C_0 is the initial disinfectant concentrations (after immediate demand) (mg/L) and k_D is the decay constant of the disinfectant (min^{-1}).

The rate of elimination final (in log) after the j^{th} CSTR in series is determined as:

$$I = -log\left(\frac{Nj}{N_0}\right); \quad \text{Equation 2- 15}$$

Where N_0 is the initial concentration of microorganisms (assumed equal to 1) and N_j is the concentration of microorganisms at time t (mg/L) (Pfeiffer & Barbeau, 2014) calculated with Eq. 2-12.

Although the N-CSTR model may also overestimate or underestimate inactivation due to the implication of the kinetic rate law it uses, it is considered more reliable than the T_{10} method. Smeets, P. W. M. H. et al. (2006) showed that the CSTR method improves prediction of inactivation of microorganisms by full-scale ozonation (Pfeiffer & Barbeau, 2014). Also observed a better prediction of *E. coli* inactivation by free chlorine using this approach.

CHAPTER 3 METHODOLOGY

This section present the methodology used to complete the QMRA of 17 Canadian water treatment plants.

3.1 Source water characterization and target pathogens

First to define the source water quality, the arithmetic mean and standard deviation of the available monitoring data, assumed to fit lognormal distribution by the HC model, were inserted in the model spreadsheet. The concentrations for three reference pathogens (Fecal coliform/*E. coli*, *Giardia* and *Cryptosporidium*) were collected from historical monitoring of raw water data for the 17 WTPs. The assumptions (such as infectivity, recovery etc.) utilized in each WTP to simplify the monitoring of the three reference pathogens were identified separately in order to be identical within WTPs for the project use. The details of the model input assumptions are presented in Table A.1- 1 and Table A.1- 2 at appendix 1.

The arithmetic mean and standard deviation for each pathogen on each WTP were calculated using STATISTICA (Statsoft) (Table A.1-3) and illustrated in box plot for the purpose of assessing four different methods namely, arithmetic mean with data Below Detection Limit (BDL) were substituted with zeros, arithmetic mean with BDL were substituted with Detection Limit (DL), regression on order statistics (ROS) by PROUCL, and point estimate (Poisson mean) to determine the best approach describing the microbial distribution in source waters. The Non-Detects (ND) data in the study were handled in different ways according to the four calculation methods. Finally, the impact of two scenarios of risk estimations was assessed (Refer to appendix 6). In one scenario, the annual risk was predicted according to the annual mean microbial concentrations. In another scenario, an annual risk was calculated based on monthly risk estimates. Each monthly risk calculation used the mean annual concentration of the month.

3.2 Treatment efficiency

The pathogens concentrations in the treated waters is based on the source water concentrations adjusted for the performance of treatments which is the sum of the individual performance of each physic-chemical processes and the disinfection processes within the treatment train. For the physic-chemical treatments, the proposed generic log removals of each physic-chemical process

were used. In circumstances where the calculated inactivation is too optimistic and unrealistic the model capped the performance to the documented values reported in literature. For chemical disinfection, the CT_{50} method was selected for calculating the treatment performance for all WTPs.

However, the impact of the CT calculation method on risk estimate was also conducted on a limited number of WTPs (N=3) by comparing three scenarios: **scenario 1** estimated disinfection efficiency through a CT_{50} approach, **scenario 2** through a CT_{10} approach and **scenario 3** through a N-CSTR approach. These three scenarios were assessed on WTPs which rely both on ozonation and chlorination to comply with primary disinfection requirements and which are fed with variable source water quality; WTP 9 (moderately impacted river), WTP 2 (pristine river) and WTP G (lightly impacted river). T_{10}/T data were collected for both Chlorination (Table A.5-1) and ozonation treatment (Table A.5-2). The source water quality of each WTP was represented by the arithmetic mean and standard deviation of the yearly monitoring data. The BDL data were substituted by the zeros. To assess disinfection performance, average pH temperature and disinfectant concentrations were determined on monthly basis for each water treatment barrier (Refer to appendix 7, Table A.7-1 till A.7-5 for chlorination and Table A.7-6 till A.7-8 for ozonation). Monthly pathogen inactivation by ozonation and chlorination were calculated and transformed into annual risks. Finally, these yearly risk estimates were evaluated for three reference pathogens: *E. coli* O157:H7, *Giardia duodenalis* and *Cryptosporidium parvum*. A nonparametric test named "Wilcoxon test" was used to compare the risk estimates. The Wilcoxon test first computes the difference between the different approaches and then analyzes only the generated list of differences.

3.3 Risk characterization

The HC QMRA model expresses the estimated risk in terms of (i) probability of infection, (ii) probability of illness and (iii) DALYs. To do so, the model used the exponential model for *Cryptosporidium* and *Giardia* whereas the Beta-Poisson model for rotavirus, *Campylobacter* and *E. coli.* The HC model calculated the predicted dose in treated waters for each slices separately after segmenting the raw water PDF into 500 slices. From that predicted dose, the model assessed weighted average risk by assuming the probability of finding from 1 to 40 pathogens in the treated volume of water supposing a Poisson distribution. This approach calculates the risk for

each of these possibilities, which are then summed up. This process is reiterated for the 500 elements and the risk is weight-averaged to account for the relative contribution of each element (0.002).

Finally, the health risks were assessed for the 17 WTPs, and the outcomes were compared to the health-based targets set by USEPA (10E-04 infection/y/person) and Health Canada (10E-06 DALY/year).

CHAPTER 4 ARTICLE 1: QUANTITATIVE MICROBIAL RISK ASSESSMENT AT 17 CANADIAN WATER TREATMENT FACILITIES

Randa TFAILY[1], Isabelle PAPINEAU[2], Robert C. ANDREWS[3], Benoit BARBEAU[4]

[1]Master's Student, NSERC Industrial Chair in Drinking Water, Dept. of Civil, Geological and Mining Engineering, Polytechnique Montreal, P.O. Box 6079, Downtown Station, Montreal, Quebec, Canada, H3C 3A7

[2]Research Associate, NSERC Industrial Chair in Drinking Water, Dept. of Civil, Geological and Mining Engineering, Polytechnique Montreal, P.O. Box 6079, Downtown Station, Montreal, Quebec, Canada, H3C 3A7

[3]Professor, NSERC Industrial Chair on Drinking Water Treatment, Dept. of Civil Engineering, University of Toronto, 35 St. George Street, Toronto, Ontario, Canada, M5S 1A4

[4]Corresponding author: Professor, NSERC Industrial Chair in Drinking Water, Dept. of Civil, Geological and Mining Engineering, Polytechnique Montreal, P.O. Box 6079, Downtown Station, Montreal, Quebec, Canada, H3C 3A7; E-mail: benoit.barbeau@polymtl.ca

Abstract

A QMRA model developed by Health Canada was applied at 17 WTPs located throughout Ontario and Quebec, Canada. Four source water characterization methods were compared that considered *E. coli*, *Giardia* and *Cryptosporidium*. In addition, three strategies to evaluate chemical disinfection performances were compared (CT_{50}, CT_{10}, and N-CSTR). The N-CSTR approach provided more reliable risk estimates as it is less sensitive to high inactivation conditions (when compared to use of CT_{10} or CT_{50}). Predicted risk estimates for the 17 WTPs revealed that only two did not comply with the 10^{-6} DALY (WHO) and 10^{-4} risk of infection (USEPA) reference levels. This publically available QMRA model could help WTP managers to assess overall treatment performance via a systematic evaluation process.

Keywords: QMRA, Source water characterization, Disinfection, N-CSTR, Risk assessment.

4.1 Introduction

The implementation of water safety plans is increasingly being considered as an important approach for the provision of safe drinking water. As such, a need exists to assess the risk of adverse health effects arising from microbial pathogens. The use of quantitative microbial risk assessment (QMRA) is one of the most popular methodologies to achieve this objective. Traditionally QMRA is conducted in four steps: [1] Hazard identification; [2] Exposure assessment; [3] Dose-response; [4] Risk characterization (Coleman & Marks, 1999; Haas et al., 1999).

QMRA has been widely employed within the international drinking water community (Howard et al., 2006; Signor & Ashbolt, 2006), to determine health based targets and help decision makers set regulatory, operational, or research priorities to ensure safe drinking water (WHO, 2004; Signor, R.S. & Ashbolt, N.J., 2006). The United States Environmental Protection Agency (USEPA) first used QMRA to set drinking water treatment requirements (1989 Surface Water Treatment Rule) to ensure $< 10^{-4}$ *Giardia* or virus infection per person per year as a result of drinking water consumption. The World Health Organization (WHO) subsequently promoted the use of water safety plans along with health based targets by applying QMRA (WHO, 2004, 2006). Dutch regulations require drinking water suppliers to assess the human health risk associated with *Cryptosporidium* and *Giardia* using QMRA every three years to compare the risk calculation outputs to health based targets (Schijven et al., 2011). New Zealand has also adopted a QMRA-based regulation for drinking water (New Zealand Ministry of Health, 2008). Finally, the Guidelines for Canadian Drinking Water Quality (GCDWQ) encourage the implementation of a risk-based, multi-barrier approach that includes QMRA (Health Canada, 2012b; Krewski et al., 2004). By offering a quantitative basis for the development of drinking water treatment goals, QMRA allows the identification of operational guidelines to ensure control and minimize public health risk.

QMRA can be applied by end users (e.g. municipal engineers) to evaluate alternative treatment strategies in order to satisfy regulatory requirements, evaluate the robustness of a given treatment train, or determine critical situations where the risk of exposure may be increased (Hartnett et al., 2007; Health Canada, 2011b; McFadyen et al., 2009). Health Canada (HC) has developed a simplified Excel version of QMRA model suitable for use by municipal water utilities (Hartnett et al., 2007; Harwood et al., 2005). This user-friendly model, available as an *Excel* spreadsheet, is

designed to provide municipal engineers, water treatment plant (WTP) operators and local decision makers with a tool to estimate health risk associated with five reference pathogens: *Cryptosporidium parvum, Giardia duodenalis,* Rotavirus*, Campylobacter* and *E. coli* O157:H7. Owing that monitoring all pathogens in water is considered costly and impractical (Schijven et al., 2011), QMRA is generally applied using reference pathogens which cover a broad range of health risks (Medema & Ashbolt, 2006). To assess exposure via drinking water consumption, population size, source water monitoring data and information regarding each process within a given treatment train must be specified. Protozoan parasite or bacterial indicator data are collected to quantify raw water quality. Performance of physical-chemical treatment processes is either described using site-specific data or by adopting default values included in the model, which have been identified following an extensive literature review by Health Canada and KWR Watercycle Research Institute (Netherlands) (Hijnen & Medema, 2010). To allow a site-specific evaluation of chemical disinfection performance, a user must specify the operating conditions (residual concentration x time) of each disinfection process; the spreadsheet then computes inactivation associated with each reference pathogen. Water consumption behaviour as well as the dose-response models for each reference pathogen are incorporated such that calculated risk outcomes may ultimately be expressed in terms of the probability of infection, probability of illness and disability adjusted life years (DALY). An acceptable annual DALY has been established to comply with the health based target of $< 10^{-6}$ DALY (Health Canada, 2011b) while maintaining a tolerable annual probability of infection of 10^{-4}, as suggested by the USEPA (Regli et al., 1991).

One limitation of the model is that it uses the CT_{50} method to assess chemical disinfection performance. Such an approach may not be optimal as the T_{50} does not always assure that the minimum inactivation level required by the USEPA Surface Water Treatment Rule (SWTR) regulation (USEPA, 1989) (Lev & Regli, 1992) is achieved. CT_{10} as promulgated by the USEPA (1991a) is widely used throughout North America (Lev & Regli, 1992; Rakness, Najm, Elovitz, Rexing, & Via, 2005). A drawback of using CT_{10} is that it underestimates disinfection performance in conditions where inactivation is low whereas it overestimates disinfection performance in conditions where inactivation is high (Lev & Regli, 1992; Pfeiffer & Barbeau, 2014). An alternative method, using a partially segregated flow technique (also called the N-CSTR method) has been proposed by Lawler, and Singer (1993) to improve accuracy when

predicting disinfection performance. This model was recently shown to improve predictions of *E. coli* inactivation by free chlorine (Pfeiffer, V. & Barbeau, B., 2014). The N-CSTR model considers the reactor to be composed of several single CSTRs in series. In contrast to CT_{10}, it implies a more accurate description of the hydraulic behaviour of a reactor but also assumes that disinfectant decay within the reactor is known. As such, the N-CSTR method offers a more accurate prediction of inactivation and is less prone to over- or underestimation in circumstances where low or high disinfectant decay is observed (Pfeiffer & Barbeau, 2014). As primary disinfection is key in reducing the burden of waterborne disease, there is a need to determine which method should be employed in QMRA modeling to ensure accurate estimation of public health risk while adopting a convenient methodology for end-users. The objectives of this investigation were: [1] to apply the Health Canada QMRA model at seventeen WTPs located in Ontario and Quebec (Canada) having a wide range of treatment technologies, [2] to propose the best method to represent the microbial concentrations in source waters, considering three different calculation techniques (regression on order statistics (ROS), arithmetic mean, and point estimate) in order to improve the accuracy of risk outputs, and [3] to evaluate the impact on risk characterization as a function of the methodology used to predict inactivation by primary disinfection (CT_{50}, CT_{10}, and N-CSTR).

4.2 Methodology

4.2.1 QMRA model

f. *Source water*

Source water quality was characterized using the arithmetic mean and standard deviation of available monitoring data. The model assumes that pathogen concentrations can be fitted to a lognormal distribution. *E. coli* O157:H7 concentrations were estimated using *E. coli* data where the fraction of *E. coli* O157:H7 was assumed to correspond to 3.49% of the total *E. coli* population present in source water (Martins, Rivera, Clark, & Olson, 1992).

g. *Treatment efficiency*

To estimate pathogen concentrations in treated waters the model sums the individual performance of each unit process in the treatment train. The user must select an appropriate type of physical-chemical treatment processes from 4 coagulation/settling scenarios and 7 filtration scenarios. If

site-specific performance data are available, the user may enter it instead. In the absence of such information the model assigns log removal values to each process according to weighted means reported in the literature review (Hijnen & Medema, 2010) and adapted by Health Canada (Table 4.1). The user must enter the theoretical contact time (min), residual disinfectant concentration (mg/L), pH, and temperature (°C) in order to assess the inactivation efficiency of chemical disinfection. In circumstances where the calculated inactivation exceeds values reported in the literature, the model automatically assumes a maximum performance value (Table 4.2).

Table 4-1: Assigned reduction of pathogens by physical-chemical treatment processes.

Reference pathogens	**Reduction per unit process (log values)**									
	Coagulation/ Sedimentation			**Filtration**						**Disinfection**
				Slow	**Rapid granular**			**Membrane**		
	I(c)*	D(c/f)†	C(c/f/s)‡	SS§	NC**	I(d)††	C‡‡	MF§§	UF***	UV†††
Crypto.§§§	0.00	0.00	1.86	4.6(	1.11	2.97	2.41	6.13	6.41	4.43
Giardia	0.00	0.00	1.61	4.88	1.23	2.86	1.92	6.62	6.18	4.00
Rotavirus	0.00	0.00	1.76	2.18	0.77	0.59	1.11	1.10	4.12	4.08
*Campy.*****	0.00	0.00	1.55	2.69	0.55	1.36	0.87	4.60	8.00	5.00
E. coli	0.00	0.00	1.55	2.69	0.55	1.36	0.87	4.60	8.00	5.50

***I(c)-In-line coagulation**
†D(c/f)-Direct (coagulation, flocculation)
‡C(c/f/s)-Conventional (coagulation, flocculation and sedimentation)
§SS-slow sand
****NC-no coagulation**
††I(d)-In-line/direct
‡‡Conventional
§§MF-microfiltration
***UF-ultrafiltration**
†††UV- Ultraviolet disinfection
§§§*Crypto* - *Cryptosporidium*
*********Campy*-Campylobacter**

Table 4-2: Inactivation (I) equations for ozonation and chlorination.

Pathogen	Process	Equation	Reference	Maximum inactivation*
Giardia	O_3	$I = CT(0.0087 \times Temperature^2 - 0.0334 Temperature + 1.545)$	(USEPA, 1991b, 1999)	4 log
	Cl_2	$I = \frac{CT}{0.2828 \times pH^{2.69} \times [Cl_2]^{0.15} \times 0.933^{(Temperature-5)}}$	(USEPA, 1991b, 1999)	8 log
Crypto.	O_3	$I = 0.0397 CT (1.09757)^{Temperature}$	(USEPA, 2006)	6 log
	Cl_2	$I = \frac{2CT}{7200}$	(Korich, Mead, Madore, Sinclair, & Sterling, 1990)	4 log
E.coli	O_3	$I = CT\left(\frac{60s}{min}\right)(4.1828 Temperature + 31.9)$	(Hunt & Mariñas, 1999)	8 log
	Cl_2	$I = 3.8962 CT^{0.3124}$	(Rice et al., 1999)	8 log

*Maximum inactivation values adopted by the model

h. ***Risk characterization***

The model expresses estimated risks in terms of (i) the probability of infection, (ii) the probability of illness and (iii) DALYs, using an exponential dose-response model for *Cryptosporidium* and *Giardia* (Equation 4.1); for rotavirus, *Campylobacter* and *E. coli,* a Beta-Poisson model (Equation 4.2) is applied. For both cases, output is defined as the daily probability of infection (P_{inf}) upon the ingestion of a given pathogens dose.

$$P_{inf} = 1 - exp^{-Dr} \qquad \text{Equation 4.1}$$

$$P_{inf} = 1 - \left(1 + {}^{D}/_{\beta}\right)^{-\alpha} \qquad \text{Equation 4.2}$$

Where r, β, and α are pathogen specific coefficients used to depict the dose-response curves for each reference pathogen (Table 4.3).

The probability of infection per year ($P_{inf/year}$) is calculated as:

$$P_{inf/year} = 1 - (1 - P_{inf})^{365} \qquad \text{Equation 4.3}$$

The dose of ingested organisms (D) is calculated for each pathogen according to Equation 4.4

$$D = C_{SW} \times (1/R) \times I_{fraction} \times 10^{-(\text{Log } Removal)} \times V \qquad \text{Equation 4.4}$$

Where C_{SW} represents the concentration of microorganisms in the source water, R the recovery of the analytical method, $I_{fraction}$ the infectious fraction and V the volume of unboiled water ingested daily.

To conduct risk calculations it is assumed that all pathogens are infectious; recoveries of *Cryptosporidium* and *Giardia* of 40% and 69%, respectively (Jaidi et al., 2009), and that one liter of unboiled water is consumed per person per day (Health Canada, 2012b). Protozoan data were corrected prior to entering (oo)cyst concentrations into the model.

In lieu of calculating point estimates based on the average pathogen dose in treated waters, the model defines the raw water probability distribution function (PDF) as 500 segments, for each of which the predicted dose in treated waters can be calculated using Eq. 4.4 The weighted average risk is subsequently calculated using Eq. 4.5:

$$P_{inf} = \sum_{i=1}^{500}\left[0.002\times\left[\sum_{N=1}^{40}\left(\frac{(V\times C_{TW\_i})^{N}}{N!}\right)\times(1-\exp(-r\times N))\right]\right] \qquad \text{Equation 4.5}$$

This approach calculates the probability of finding 1 to 40 pathogens with concentration (C_{TW_i}) in a volume of treated water (V), assuming a Poisson distribution. For each probability, the risk is calculated and summed. This process is reiterated for the 500 elements and the risk is weight-averaged by multiplying by 0.002 to account for the relative contribution of each. Once the $P_{inf/year}$ is defined, the probability of illness per person per year ($P_{ill/year}$) is assessed as follows:

$$P_{ill/year} = P_{inf/year}\times P_{ill.\ given\ infection} \qquad \text{Equation 4.6}$$

Where the probability of illness given infection ($P_{ill.\ given\ infection}$) is determined according to the values published in literature for each reference pathogen (Table 4.3), and public health risk is expressed in terms of DALYs (Equation 4.7).

$$DALY = LYL + YLD \qquad \text{Equation 4.7}$$

Where LYL is the life years lost due to premature death, YLD is the years lived with disability or impairment.

$$DALY_{/year} = P_{ill/year}\times DALY_{in\ case\ of\ illness} \qquad \text{Equation 4.8}$$

Table 4-3: Dose-response models, probability of illness given infection and total DALYs per case of illness according to the Health Canada model.

Reference Pathogen	Type of model	Dose-response curves				P_{ill} given infection*		Total DALYs per case of illness**
		R	α	β	Reference	Value	Reference	
C. parvum‡	Exp.§	0.018			Messner et al. (2001)	0.7	Casman et al. (2000); Okhuysen et al. (1998)	1.70E -03
Giardia	Exp.	0.01982			Rose, and Gerba (1991)	0.24	Eisenberg et al. (2006); Macler, and Regli (1993)	1.70 E-03
Rotavirus	Beta-Poisson		0.265	0.4415	Haas et al. (1993)	0.88	Havelaar, and Melse (2003)	8.46 E-03
Campy.§§	Beta-Poisson		0.024	0.011	Teunis, P. F. M. et al. (1999)	1†		4.60 E-03
E.coli O157	Beta-Poisson		0.0571	2.2183	Strachan et al. (2005)	1†		2.45E-02

***P_{ill}-Probability of illness**
†Illness used in dose response model
‡ Crypto.- *Cryptosporidium parvum*
§ Exp.- Exponential
§§ Campy- Campylobacter
****As defined in the HC model**

4.2.2 **Water treatment plants**

All 17 Canadian WTPs were located in Ontario (named using alphabetical letter) and in Quebec (numbered). They all received surface waters and varied in terms of treatment schemes (Table 4.4).

Table 4-4: Observed reduction of pathogens at water treatment plants (WTPs)

Province	WTP	Source	Treatment regime	Log removals		
				E. coli	***Giardia***	***Crypto.***
Ontario	A	Lake	Actiflo^{TM} + O_3 + Granular filtration	2.42	3.53	4.27
Ontario	B	Lake	Actiflo^{TM} + O_3 + Granular filtration	2.42	3.53	4.27
Ontario	C	River	Conventional treatment* + Cl_2	2.42	3.53	4.27
Ontario	D	Lake	Microfiltration + GAC contactors + Cl_2	5.15	7.85	7.24
Ontario	E	Lake	Conventional treatment + UV + Cl_2	2.42	3.53	4.27
Ontario	F	Lake	Conventional treatment + Cl_2	2.42	3.53	4.27
Ontario	G	River	Conventional treatment + Cl_2	2.42	3.53	4.27
Ontario	H	Lake	Direct filtration (coag./floc.) + Cl_2	1.36	2.86	2.97
Quebec	1	River	Direct filtration (without coag.) + Cl_2	0.55	1.23	1.11
Quebec	2	River	Direct filtration (without coag.) + O_3 + Cl_2	0.55	1.23	1.11
Quebec	3	River	Conventional treatment + Cl_2	2.42	3.53	4.27
Quebec	4	River	Conventional treatment + Cl_2	2.42	3.53	4.27
Quebec	5	River	O_3 + Conventional treatment + Cl_2	2.42	3.53	4.27
Quebec	6	River	Conventional treatment + O_3 + Cl_2	2.42	3.53	4.27
Quebec	7	River	Conventional treatment + O_3 + Cl_2	2.42	3.53	4.27
Quebec	8	River	Conventional treatment + O_3 + Cl_2	2.42	3.53	4.27
Quebec	9	River	Conventional treatment + O_3 + GAC Filtration + Cl_2	2.97	4.76	5.38

***Conventional treatment: coagulation, flocculation, sedimentation, and granular filtration.**

4.2.3 **Source water data**

Historical *E. coli*/Fecal coliforms, *Giardia* and *Cryptosporidium* raw water data were provided for each WTP (except for parasites which were available in only 12 of 17 plants). The sampling period considered in this study ranged from 2004 to 2010 for WTPs located in Quebec, and 2009 to 2011 for Ontario WTPs. Sampling strategies differed from one WTP to another; *Cryptosporidium/Giardia* and *E.coli*/fecal coliform measurements were conducted on a monthly or biweekly basis, respectively.

4.2.4 **Source water characterization**

Four methods were investigated to determine the most appropriate approach to describe pathogen concentrations in source waters: regression on order statistics (ROS) (ProUCL, USEPA), arithmetic mean (with or without replacement of below detection limit concentrations) and a point estimate (PE). The latter is calculated using Equation 4.9 (Parkhurst & Stern, 1998).

$$PE = {\sum N_x}\Big/{\sum V_x} \qquad \text{Equation 4.9}$$

Where N_x is the number of pathogens detected in the sample *x*, V_x is the sample volume and x the number of collected samples.

Data below detection limit (BDL) were handled according to the method used to describe pathogen concentrations in the source water: [1] Arithmetic mean was calculated by substituting data BDL either by the detection limit or using zeros, [2] ROS extrapolates data BDL according to a log normal distribution performed on data above detection limit, [3] Point estimate represents a Poisson mean which can be calculated including zeros (BDL data).

4.2.5 **Impact of CT calculations on risk assessment**

Three scenarios were compared to determine the most suitable method to assess chemical disinfection performance. Scenarios 1, 2 and 3 estimated disinfection efficiency using CT_{50}, CT_{10}, and N-CSTR approach, respectively. All are derived from the first-order Chick-Watson's law as presented below (Lev & Regli, 1992):

$$ln(N/N_o) = -k \times CT \qquad \text{Equation 4.10}$$

Where N and N_0 are the concentrations of microorganisms at time t and t = 0, C is the disinfectant residual concentration (mg/L), T the theoretical contact time (min) and *k* the inactivation rate which is constant for a given type of disinfectant (L/mg.min), microorganism, pH, and temperature.

A correlation between the T_{10}/T ratio and the corresponding number of CSTR (Carlson et al., 2001; Pfeiffer & Barbeau, 2014) was used to determine the total number of CSTR in series (*n*) for a given contact basin. The concentration of microorganisms following the n^{th} CSTR (N_n), was assessed by predicting the effluent concentration of microorganisms (N_j) of each j^{th} CSTR while considering the effluent concentration of the previous CSTR (N_i) as follows:

$$N_j = \frac{N_i}{\left(1+\frac{kC_jHRT}{n}\right)^{j-i}} \qquad \text{Equation 4.11}$$

Where *k* is the inactivation rate constant and C_j the effluent disinfectant concentration of the j^{th} CSTR. For this study, the disinfectant decay rate was not considered. The residual disinfectant concentrations used to calculate CT_{10} and CT_{50} were also used as C_j in Eq. 4.11. Finally, the overall disinfection process inactivation efficiency (I) was determined as follows:

$$I = -Log\ (N_n/N_0) \quad \text{Equation 4.12}$$

These disinfection calculation scenarios were applied to three WTPs which rely ozonation and/or chlorination to provide primary disinfection and which receive surface water of varying quality; WTP 2 (pristine river), WTP G (lightly impacted river) and WTP 9 (moderately impacted river). The source water quality of each WTP was represented by the arithmetic mean (with BDL data replaced by zeros) and the standard deviation. To assess disinfection performance pH, temperature and disinfectant concentrations were based on monthly averages of ozone and chlorine residuals. Monthly E. coli O157:H7 and Giardia duodenalis inactivation by ozonation and chlorination were calculated using the equations in Table 4.2, and transformed into annual risks. These two organisms were selected to represent a sensitive and resistant pathogen, respectively. Finally, a Wilcoxon matched paired test was performed using Statistica 12 (StatSoft 2012) to compare the risk outcomes of the three scenarios.

4.3 Results

The various approaches examined during the calculation of QMRA risk estimates are summarized in Figure 4.1:

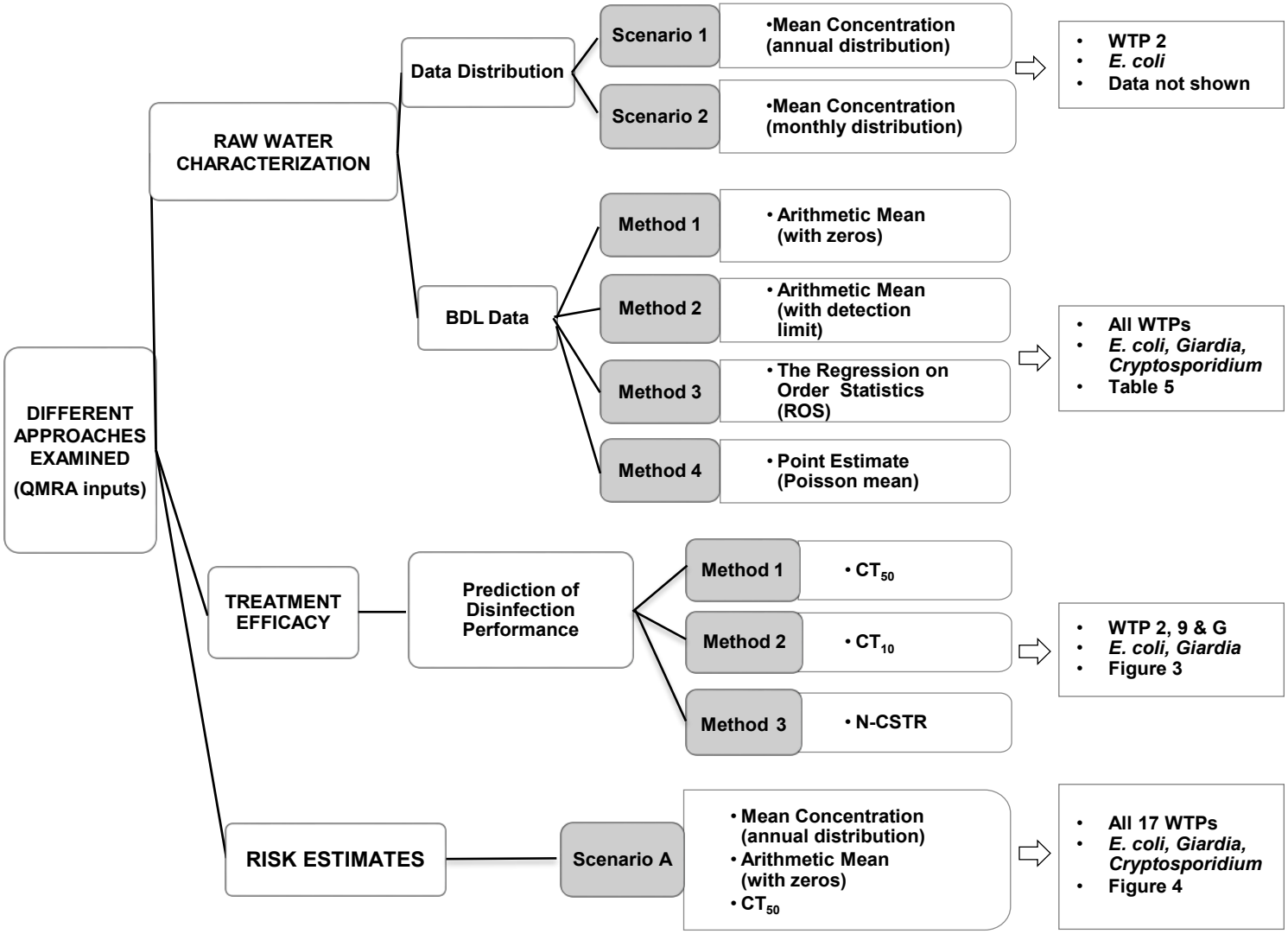

Figure 4-1: Overview of various approaches examined in the framework of this study

4.3.1 **Source water Characterization**

Raw water pathogen concentrations (Figure 4.2) showed significant variability when comparing the 17 WTPs. *E. coli*/fecal coliforms variability was an important factor, as in general fluctuations ranged two orders of magnitude (and as much as 4 log for WTP G). Parasite concentration variability was lower, with most data in the range of 1 to 100 (oo)cysts per 100 L. Point estimate concentrations mainly varied between 1 and 10 (oo)cysts/100 L (*Giardia:* 11 out of 12 WTPs; *Cryptosporidium*: 9 out of 12 WTPs). Finally, *Giardia* proved to be more prevalent than *Cryptosporidium* as observed concentrations of the former were higher than the later in 10 out of the 12 source waters. Such variation within and between the WTPs is consistent with previous studies, which investigated *E.coli*, *Giardia*, and *Cryptosporidium* occurrence in surface waters (Dechesne & Soyeux; Schilling, Zhang, Hill, Jones, & Wolter, 2009; Smith & Grimason, 2003).

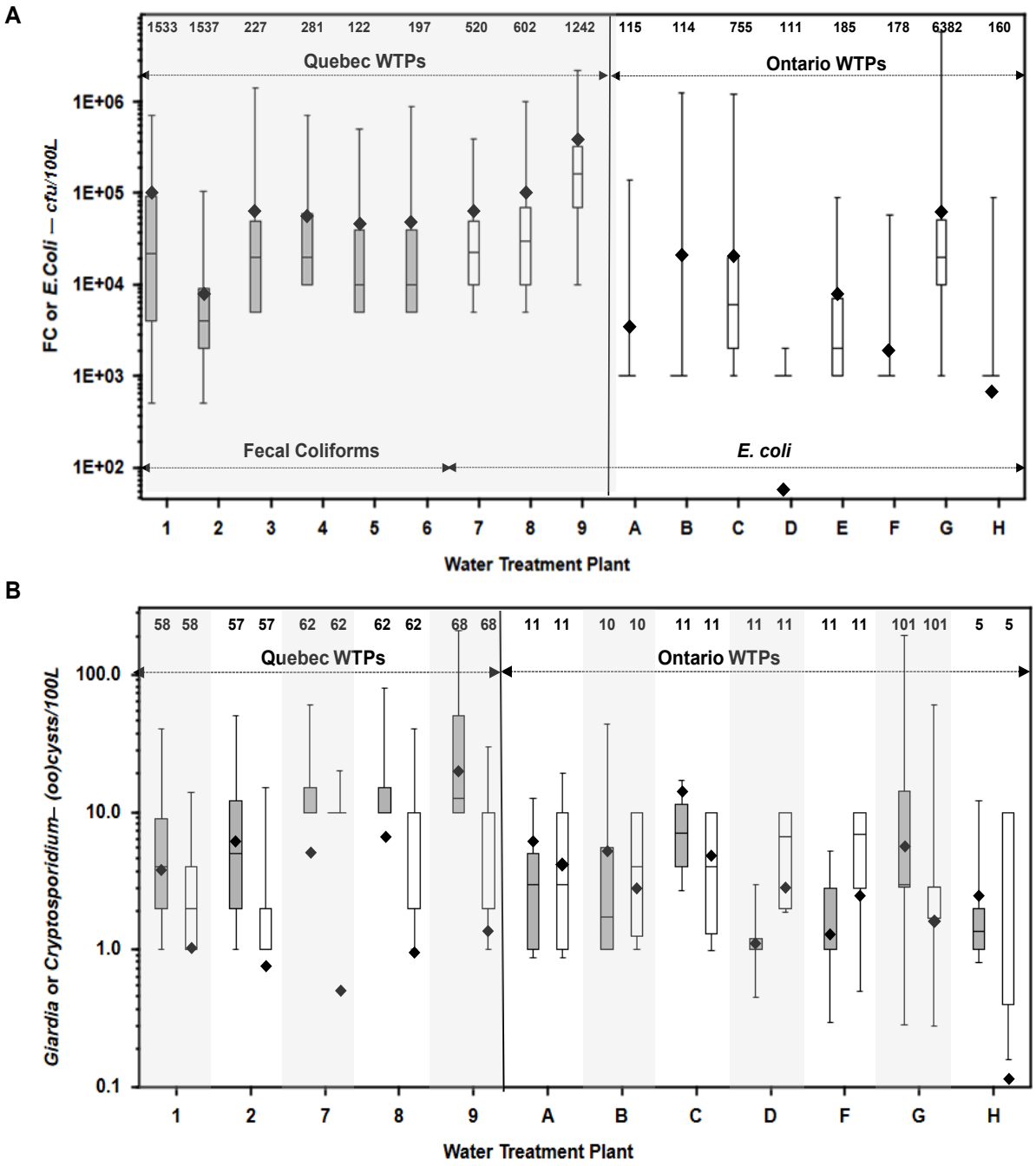

Figure 4-2: Distributions of raw water pathogen concentrations.

(A) fecal coliforms (grey boxes) or *E. coli* (empty boxes), (B) *Giardia* (grey boxes) or *Cryptosporidium* (empty boxes). Boxes represent median, the 25th and 75th percentile while whiskers represent minimum and maximum values. The sample sizes (n) for a given treatment plant are shown above each box. Data, which fall below detection limits, were substituted by the detection limit. Black diamonds represent point estimate means.

E. coli O157:H7 risk estimates were examined by assessing the two following scenarios for WTP 2 (Figure 1): [1] using the mean concentration derived from the annual distribution; [2] using the mean concentration derived from monthly distributions. This site was selected due to the size of its database (1537 samples over five years, 2004-2010) and, its consistent treatment performance. Minimal changes in treatment performance help to better elucidate risk fluctuations attributable to source water variability. Both scenarios 1 and 2 yielded similar risk estimates of 3.49E-16 and 3.92E-16, respectively. The use of an annual probability density function (PDF) was adopted as it simplified data handling.

Four alternative approaches were examined with respect to dealing with data, which fell below the detection limit (BDL) (refer to Figure 4.1 & Table 4.5). The mean concentrations calculated by the different methods were compared to the standard arithmetic mean with zeros (Method 1). [1] The calculated arithmetic means of Method 2, whereby BDL data were substituted using the detection limit, resulted in an overestimation of 8 fold for *Cryptosporidium.* This discrepancy was smaller for *E. coli* and *Giardia*. [2] The regression on order statistics (ROS) (Method 3) provided similar results to the arithmetic mean with zeros (Method 1) for *E. coli and Giardia,* as the average mean concentrations were 53% and 13% smaller respectively than for the ROS technique. However, when considering *Cryptosporidium,* the average arithmetic mean concentrations of all WTPs were 4.5 fold higher than when applying the ROS technique. The observed discrepancy between Methods 1 & 3 increased as the proportion of data BDL also increased. [3] Using Method 4, point estimate (Poisson mean) provided results comparable to those of Method 1. Point estimates were on average higher than the arithmetic means with zeros by 1%, 50% and 14% for *E. coli, Giardia* and *Cryptosporidium,* respectively (Table 4.5). For subsequent calculations, Method 1, consisting of the standard arithmetic mean with zeros was used to calculate the health risk for the 17 WTPs. It corresponds to the proposed approach by Heath Canada and does not considerably differ from other approaches, except with respect to *Cryptosporidium,* for which more refined techniques would be valuable considering the high occurrence of non-detects.

Table 4-5: Comparison of 4 approaches for raw water characterization

Pathogen	Ratio (M2/M1)*	Ratio (M3/M1)†	Ratio (M4/M1)‡
E. coli	2.16	1.53	0.99
Giardia	1.29	1.13	1.5
Cryptosporidium	8.0	4.5	1.14

*M2/M1: Arithmetic mean with DL (Method 2) versus Arithmetic mean with zeros (Method 1)

†M3/M1: ROS (Method 3) versus Arithmetic mean with zeros (Method 1)

‡M4/M1: Point estimate (Method 4) versus Arithmetic mean with zeros (Method 1)

4.3.2 Performance of physical-chemical treatment processes

Predicted removals as a result of physical-chemical treatment processes varied from one WTP to another. Log removals for *E. coli, Giardia* and *Cryptosporidium* ranged from 0.55 to 5.15, 1.23 to 7.85, and 1.11 to 7.24, respectively (Table 4.4). The lowest removals were associated with two WTPs (1 and 2), which use direct filtration without coagulation, whereas the highest performance was attributed to a microfiltration plant with post-GAC contactors (WTP D).

4.3.3 Comparison of alternative methods to predict disinfection performance

Predicted inactivation obtained using CT_{50} often reached the maximum values allocated by the model. When considering *E. coli*, maximum inactivation values were observed for 15 out of 17 WTPs (data not shown). *Giardia* reached maximums for 4 out of 17 WTPs, while *Cryptosporidium* inactivation proved to always be below the maximum values (data not shown). These results highlight the importance of taking into account the hydraulics of a system when calculating disinfection performance, especially for sensitive microorganisms such as viruses or bacteria.

Three strategies were applied (CT_{50}, CT_{10}, and N-CSTR) to assess the impact of hydraulics on inactivation and subsequent yearly probability of infection estimates. WTPs considered included WTP 2 (pristine river), WTP G (lightly impacted river) and WTP 9 (moderately impacted river) (Figure 4.3). As expected, the impact of CT calculation methods on risk estimates was greater for *E. coli* than for *Giardia*. According to a Wilcoxon matched paired test the *E. coli* health risk associated with WTPs 2 and 9 when applying the N-CSTR approach was significantly superior (p

< 0.05) to either CT_{10} and CT_{50}. When applied to WTP G all three methods predicted *E. coli* inactivations which exceeded the maximum values used by the model. As such, the calculated risk outcomes were deemed to be equivalent (Figure 4.3, part A). Similarly, the *Giardia* yearly probability of infection predicted using the CT_{10} and N-CSTR approaches (Figure 4.3, part B) were significantly greater ($p < 0.05$) than predicted using CT_{50}. Observed discrepancies among risk estimates when comparing N-CSTR and CT_{10} are consistent with the bias arising from the CT_{10} concept which is expected to overestimate disinfection performance at higher log inactivation (Lev & Regli, 1992). As such the CT_{50} and CT_{10} methods are more prone to reaching maximum values allocated by the model. Consequently, the N-CSTR approach may provide more reliable risk calculations as it is less sensitive to high inactivation conditions.

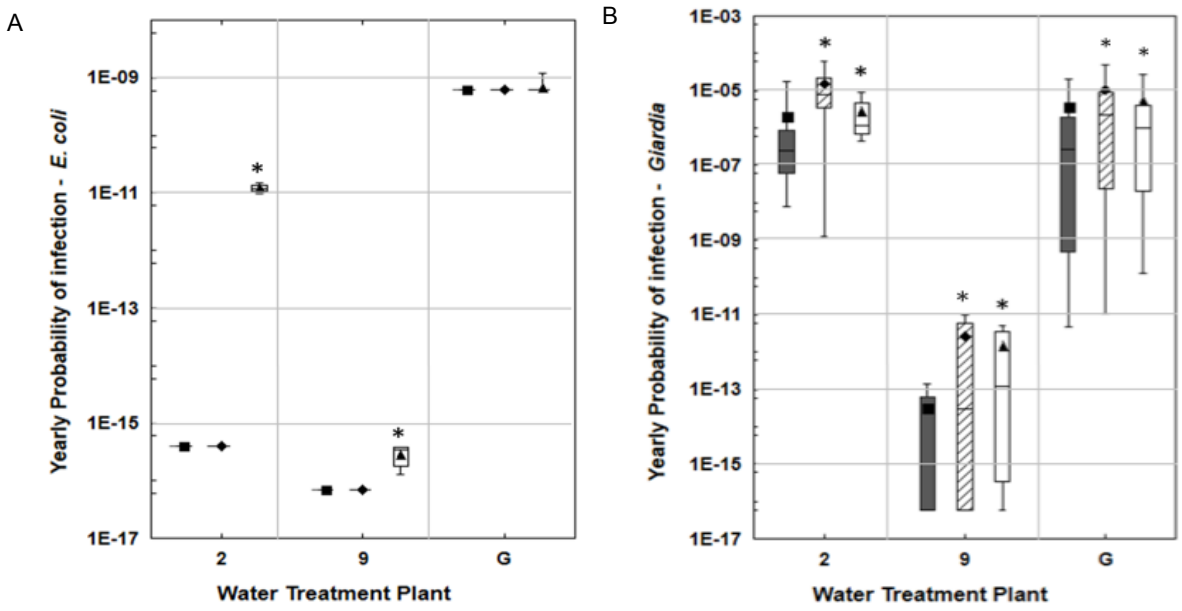

Figure 4-3: Mean yearly probability of infection (A: *E. coli*, B: *Giardia*) for water treatment plants 2, 9 and G using various inactivation models.

CT_{50} (■), CT_{10} (◆) and N-CSTR (▲). The horizontal line within boxes represents the median, boxes the 25th and 75th percentile and whiskers minimum and maximum values of monthly predictions. Significant differences associated with the use CT_{50}, according to a Wilcoxon test are denoted by a star (*) (p_{value}<0.05).

4.3.4 **Overview of risk estimates predicted for the WTPs**

QMRA analyses were performed on the 17 WTPs (Table 4.4) by applying the HC model according to scenario A (Figure 4.1). Due to the use of maximum values allocated by the HC QMRA model, no seasonal variation of *E. coli* inactivation was observed for any WTP. Yearly *E. coli* probability of infection values (left axis) of all 17 WTPs were below 10^{-4} (the basis for the current USEPA regulations) (Regli et al., 1991) (Figure 4.4, part A) and the 10^{-6} DALY value (right axis) (Health Canada, 2011b). In contrast, the annual *Giardia* and *Cryptosporidium* health risk targets were not always achieved for WTP 1 (*Giardia* - Figure 4.4, part B) and WTPs 1 and 2 (*Cryptosporidium* - Figure 4.4, part C), as a result of the poor performance associated with direct filtration without coagulation.

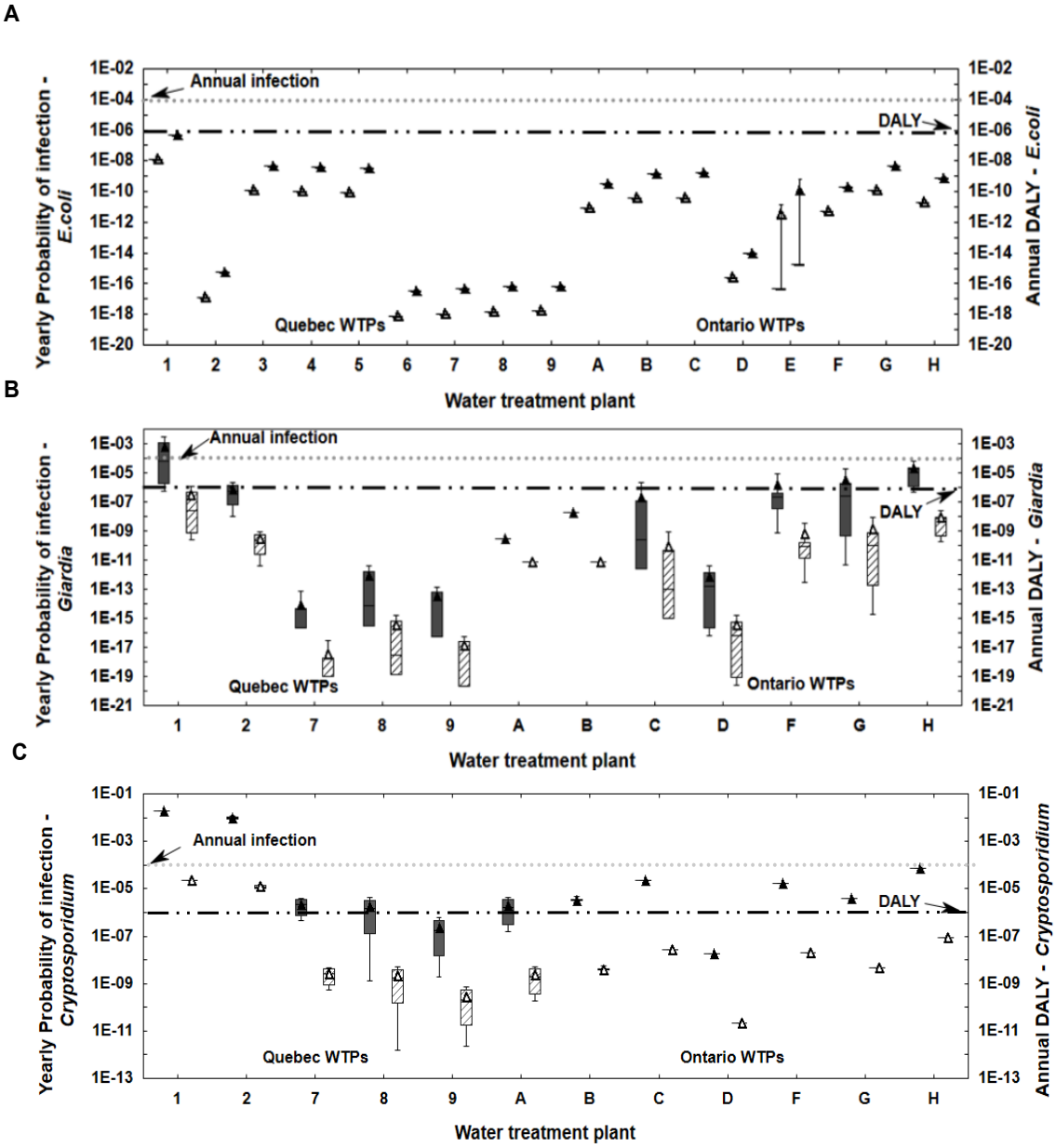

Figure 4-4: Mean yearly probability of infection and mean annual DALY for *E. coli* (A), *Giardia* (B) and *Cryptosporidium* (C) with the CT_{50} inactivation method.

The horizontal line within the boxes represents the median, boxes the 25th and 75th percentile and whiskers the minimum and maximum values of monthly predictions.

▲ Mean yearly probability of infection △ Mean annual DALY

4.4 Discussions

4.4.1 Improving QMRA predictions

As statistical methods to account for data BDL can be complex (Emelko, Schmidt, & Roberson, 2008), it would be useful to provide a tool capable of manipulating source water data for end-users. Recently, the software QMRAspot (Schijven et al., 2011) has been developed in the Netherlands, and includes an automated data fitting procedure. A similar approach should be considered for future versions of HC QMRA model.

A comparison of three CT calculation strategies highlighted the impact of the selected method on predicted health risk outcomes. The use of CT_{50} was shown to overestimate disinfection efficacy. Application of CT_{10} may serve as an attractive approach considering its common adoption in the water industry, however, its use yields overestimation at high inactivation and underestimation at low inactivation, therefore introducing a bias that should be avoided in a QMRA context. Use of the N-CSTR method has been shown to provide improved accuracy when considering predictions of *E. coli* inactivation (Pfeiffer & Barbeau, 2014; Smeets, P. W. M. H. et al., 2006). As such, water utilities are recommended to use this technique when performing a site-specific QMRA on their systems.

QMRA performed on 17 WTPs highlights the importance of chemical disinfection for reducing risk. Use of an N-CSTR model would reduce the need for arbitrary maximum values since disinfection performance is not linear with applied CT. In addition, use of maximum inactivation values introduces a bias in the evaluation of disinfection performance. Other factors such as microorganism clumping or their attachment to particles, may also be an important factor causing deviation from the Chick-Watson kinetics (Barbeau et al., 2005). These effects may be site-specific, vary seasonally, and be important under treatment failure conditions when flocs/aggregates may escape to downstream chemical disinfection. Improving QMRA models should address the issue of refining predictions of chemical disinfection performance at high log inactivation conditions for natural waters.

The HC QMRA model provides default values to represent performance of the physical-chemical treatment processes, based on extensive data presented in the literature. However, consideration of site-specific parameters (e.g. filtered water turbidity) or site-specific performance data would

reduce uncertainty in calculated risk assessment outcomes (Smeets, P., Medema, Kruidenier, & Havelaar, 2007; Teunis, P. F. M. et al., 1997). Site-specific performance evaluations using microbial indicator organisms would represent a good opportunity for drinking water operations personnel aiming to reduce uncertainty in QMRA risk estimates. In Canada, aerobic spore-formers have been examined as a practical alternative for the purpose of evaluating treatment performance (Barbeau et al., 2005), whereas in Netherlands, *Clostridia* spores have been commonly used for this purpose. Further studies should aim to provide advice to QMRA users willing to integrate site-specific performance indicators in their evaluations.

4.4.2 **QMRA risk estimates**

Health risk outcomes predicted using the Health Canada approach in this study revealed that the majority comply with the DALY and the USEPA reference levels. Exceptions were observed for two WTPs with respect to *Giardia* and *Cryptosporidium* associated risk. Both WTPs which used direct filtration without coagulation at the time of the data collection, have recently implemented coagulation, ozonation and UV disinfection.

4.5 Conclusion

The HC QMRA model proved to be useful to assess overall treatment performance and compare a wide range of treatment scenarios. The greatest value of using this tool may reside in the systematic evaluation process that WTP managers must follow to implement it. It should however be stated that the risk outcomes are semi-quantitative due to numerous simplifications and sources of uncertainty. Standardization of input source water data handling would improve the accuracy of risk estimates. In addition, the use of an N-CSTR method is suggested as an alternative to the CT_{10} or CT_{50} approach to calculate chemical disinfection performance. Future work should also evaluate techniques to improve the prediction of chemical disinfection in natural waters considering that these processes largely contribute to the reduction of risk. Finally, existing QMRA models do not address microbial risks to consumers during distribution. Nevertheless, the HC model may be used by water treatment utilities as a tool to be integrated within the larger context of developing a water safety plan.

Acknowledgements

The authors acknowledge the support of the NSERC Industrial Chair on Drinking Water (Polytechnique Montreal) and the NSERC Industrial Chair in Drinking Water Research (University of Toronto) and their industrial partners. They also thank participating water utilities which provided all the necessary data to conduct this research. Finally, they acknowledge Joshua Elliott and Jennifer Lee for their support and technical assistance in this work.

Reference

Barbeau, B., Huffman, D., Mysore, C., Desjardins, R., Clement, B., & Prevost, M. (2005). Examination of discrete and counfounding effects of water quality parameters during the inactivation of MS2 phages and Bacillus subtilis spores with chlorine dioxide. *Journal of Environmental Engineering and Science, 4* 139-151.

Carlson, K., Pier, D., Bellamy, W., Carlson, M., Ducoste, J., Amy, G., . . . Via, S. (2001). *Implementation of the integrated disinfection design framework*. Denver, Colorado, USA: American Water Works Association Research Foundation.

Casman, E. A., Fischhoff, B., Palmgren, C., Small, M. J., & Wu, F. (2000). An Integrated Risk Model of a Drinking-Water – Borne Cryptosporidiosis Outbreak. *Risk Analysis, 20*(4), 495-511.

Coleman, M. E., & Marks, H. M. (1999). Qualitative and quantitative risk assessment. *Food Control, 10* 289-297.

Dechesne, M., & Soyeux, E.

Eisenberg, J. N., Cevallos, W., Ponce, K., Levy, K., Bates, S. J., Scott, J. C., . . . Trostle, J. (2006). Environmental change and infectious disease: How new roads affect the transmission of diarrheal pathogens in rural Ecuador. *Proceedings of the National Academy of Sciences of the United States of America, 103*(51), 19460–19465.

Emelko, M. B., Schmidt, P. J., & Roberson, J. A. (2008). Quantitification of uncertainty in microbial data - reporting and regulatory implications. *Journal American Water Works Association, 100*(3), 94-104.

Haas, C. N., Rose, J. B., & Gerba, C. P. (1999). *Quantitative microbial risk assessment* (Vol. 04). New York: John Wiley.

Haas, C. N., Rose, J. B., Gerba, C. P., & Regli, S. (1993). Risk assessment of virus in drinking water. *Risk Analysis, 13*(5), 545-552.

Hartnett, E., McFadyen, S., Douglas, I., Robertson, W., & Paoli, G. (2007). *Quantitative microbiological risk assessment; New tools to assess and manage risks from pathogens in drinking water*. Paper presented at American Water Works Association: Water Quality Technology Conference and Exposition 2007: Fast tracks to Water Quality, Ottawa.

Harwood, V. J., Levine, A. D., Scott, T. M., Chivukula, V., Lukasik, J., Farrah, S. R., & Rose, J. B. (2005). Validity of the Indicator Organism Paradigm for Pathogen Reduction in Reclaimed Water and Public Health Protection. *Applied and Environmental Microbiology, 71*(6), 3163-3170.

Havelaar, A. H., & Melse, J. M. (2003). *Quantifying public health risk in the WHO Guidelines for Drinking-Water quality: a burden of disease approach* (Report No. RIVM report 734301022). Bilthoven, Netherlands: World Health Organization

Health Canada. (2011). *Guidelines for Canadian Drinking Water Quality: guidline technical document- Enteric Viruses*.

Health Canada. (2012). *Guidelines for Canadian drinking water quality: guideline technical document- Enteric Protozoa: Giardia and Cryptosporidium*.

Hijnen, W. A. M., & Medema, G. J. (2010). *Elimination of micro-organisms by drinking water treatment processes* (3rd edition ed.). Nieuwegein: IWA publishing.

Howard, G., Pedley, S., & Tibatemwa, S. (2006). Quantitative microbial risk assessment to estimate health risks attributable to water supply: can the technique be applied in developing countries with limited data? *Journal of Water and Health, 4*(1), 49-65.

Hunt, N. K., & Mariñas, B. J. (1999). Inactivation of Escherichia coli with ozone: chemical and inactivation kinetics. *Water Research, 33*(11), 2633-2641.

Jaidi, K., Brabeau, B., Carriere, A., Desjardins, R., & Prevost, M. (2009). Including operational data in QMRA model: development and impact of model inputs. *Journal of Water and Health, 7*(1), 77-95.

Korich, D. G., Mead, J. R., Madore, M. S., Sinclair, N. A., & Sterling, C. R. (1990). Effects of ozone, chlorine dioxide, chlorine and monochloramine on Cryptosporidium parvum oocyst viability. *Applied and Environmental Microbiology, 56*(5), 1423-1428.

Krewski, D., J., B., Butler-Jones, D., Haas, C. N., Isaac-Renton, J., Roberts, K. J., & Sinclair, M. (2004). Managing the microbiological risks of drinking water. *Journal of Toxicology and Environmental Health. Part A, 67*(20-22), 1591-1617.

Lawler, D. F., & Singer, P. C. (1993). Analyzing disinfection kinetics and reactor design: a conceptual approach versus the SWTR. *Journal of the American Water Works Association, 85*(11), 67-76.

Lev, O., & Regli, S. (1992). Evaluation of ozone disinfection systems: characteristic time T. *Environmental Engineering, 118*(2), 268-285.

Macler, B. A., & Regli, S. (1993). Use of microbial risk assessment in setting US drinking water standards. *International Journal of Food Microbiology, 18*(4), 245-256.

Martins, M. T., Rivera, I. G., Clark, D. L., & Olson, B. H. (1992). Detection of virulence factors in culturable Escherichia coli isolates from water samples by DNA probes and recovery of toxin-bearing strains in minimal o-nitrophenol-beta-D-galactopyranoside-4-methylumbelliferyl-beta-D-g luc uronide media. *Applied and Environmental Microbiology, 58*(9), 3095-3100.

McFadyen, S., Brooks, T., Andrews, R., Baxter, C., Hartnett, E., & Paoli, G. (2009). *QMRA experiences in the development of Canadian Drinking Water Guidelines and in water treatment plants.* Paper presented at AWWA WQCT Conference Proceedings (pp. 1-2).

Medema, G., & Ashbolt, N. (2006). *MicroRisk (Microbiological risk assessment: A scientific basis for managing drinking water safety from source to tap). QMRA: its value for risk management*.

Messner, M. J., Chappell, C. L., & Okhuysen, P. C. (2001). Risk assessment for cryptosporidium: A hierarchical Bayesian analysis of human dose response data. *Water Research, 35*(16), 3934-3940.

New Zealand Ministry of Health. (2008). *Drinking-water standards for New Zealand 2005 (revised 2008)*. Wellington: New Zealand Ministry of Health

Okhuysen, P. C., Chappell, C. L., Sterling, C. R., Jakubowski, W., & DuPont, H. L. (1998). Susceptibility and serologic response of healthy adults to reinfection with Cryptosporidium parvum. *Infection and Immunity, 66*(2), 441-443.

Parkhurst, D. F., & Stern, D. A. (1998). Determining Average Concentrations of Cryptosporidium and Other Pathogens in Water. *Enviromental Science and Technology, 32*(21), 3424-3429.

Pfeiffer, V., & Barbeau, B. (2014). Validation of a Simple Method for Predicting the Disinfection Performance in a Flow-through Contactor. *Water Research, 49*(1), 144-156.

Rakness, K. L., Najm, I., Elovitz, M., Rexing, D., & Via, S. (2005). Cryptosporidium log-inactivation with ozone using effluent CT10, geometric mean CT10, extended integrated CT10 and extended CSTR calculations. *Ozone Science & Engineering Journal, 27*(5), 335-350.

Regli, S., Rose, J. B., Haas, C. N., & Gerba, C. P. (1991). Modeling the risk from Giardia and viruses in drinking water. *Journal of the American Water Works Association 83*(11), 76-84.

Rice, E. W., Clark, R. M., & Johnson, C. H. (1999). Chlorine inactivation of Escherichia coli O157:H7. *Emerging Infectious Disease, 5*(3), 461-463.

Rose, J. B., & Gerba, C. P. (1991). Use of risk assessment for development of microbial standards. *Water Science and Technology, 24*(2), 29-34.

Schijven, J. F., Teunis, P. F. M., Rutjes, S. A., Bouwknegt, M., & Husman, A. M. d. R. (2011). QMRAspot: a tool for quantitative microbial risk assessment from surface water to potable water. *Water Research, 45*(17), 5564-5576.

Schilling, K. E., Zhang, Y., Hill, D. R., Jones, C. S., & Wolter, C. F. (2009). Temporal variations of Escherichia coli concentrations in a large Midwestern river. *Journal of Hydrology, 365* 79-85.

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 Science and Technology, 54*(3), 261-268.

Smeets, P., Medema, G., Kruidenier, L., & Havelaar, A. H. (2007). How can the UK statutory Cryptosporidium monitoring be used for Quantitative Risk Assessment of Cryptosporidium in drinking water. *Journal of Water and Health 05.S1* 107-118. doi: 10.2166/wh.2007.140.

Smeets, P. W. M. H., Van der Helm, A. W. C., Dullemont, Y. J., L.C., R., Van Dijik, J. C., & Medema, G. J. (2006). Inactivation of Escherichia coli by ozone under bench-scale plug flow and full-scale hydraulic conditions. *Water Research, 40*(17), 3239-3248.

Smith, H., & Grimason, A. (2003). Giardia and Cryptosporidium in Water and Wastewater. In D. Mara & N. J. Horan (Eds.), *The Handbook of Water and Wastewater Microbiology* (pp. 696-755). Academic, London.

Strachan, N. J., Doyle, M. P., Kasuga, F., Rotariu, O., & Ogden, I. D. (2005). Dose response modelling of Escherichia coli O157 incorporating data from foodborne and environmental outbreaks. *International Journal of Food Microbiology, 103*(1), 35-47.

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 Research, 31*(6), 1333-1346.

Teunis, P. F. M., Nagelkerke, N. J. D., & Haas, C. N. (1999). Dose response models for infectious gastroenteritis. *Risk Analysis, 19*(6), 1251-1260.

USEPA. (1989). *National primary drinking water regulations: Filtration, disinfection, turbidity, Giardia*
lamblia, viruses, Legionella, and heterotrophic bacteria. Washington, D.C.: U.S. Environmental Protection Agency,.
USEPA. (1991a). *Guidance manual for compliance with the filtration and disinfection requirements for public water systems using surface water sources*. Washington, D.C.: U.S. Environmental Protection Agency,.
USEPA. (1991b). *Guidance manual for compliance with the surface water treatment requirements for public water systems*. Washington, D.C.: U.S. Environmental Protection agency,.
USEPA. (1999). *Disinfection profiling and benchmarking manual*.
USEPA. (2006). *Federal Register. Part II. National primary drinking water regulations: Long Term 2 Enhanced Surface Water Treatment Rule; final rule*. Washington, DC, USA: Office of Science and Technology, Office of Water.
WHO. (2004). *Guidelines for drinking-water quality*. Geneva: World Health Organization,.
WHO. (2006). *Guidelines for drinking-water quality: incorporating first addendum.* . Geneva: World Health Organization.

CHAPTER 5 GENERAL DISCUSSIONS

In this chapter, we will synthesize and review the results presented, either in the article or in the appendix, in order to answer the main research questions: of our research objective: How should data below detection be handled during the source water characterization? What is the impact of using CT calculation methods on the risk outcomes and what is the recommended optimal method? Are the 17 WTPs under investigation able to meet the targeted DALY and risk of infection objectives proposed by Health Canada and USEPA? What are other improvements that could be made to HC model? Our discussion will be divided into three parts: source water characterization, treatment performance, and risk characterization.

5.1 Source water characterization

An accurate source water characterization is a big challenge in the area of QMRA. Knowing that pathogens concentration distributions are usually undefined and vary on a log-scale: few samples could have very high concentrations of infectious organisms while others have concentrations below detection levels. Therefore, there is a necessity for caution when selecting the best-fitted distribution in the presence of the non-detectable data (Taylor, Kupper, Rappaport, & Lyles, 2001). Many authors worked on describing the best-fitted statistical distributions for pathogens using Lognormal, Binomial, Negative Binomial, Poisson, and Gamma distributions, etc.… As previously mentioned, Health Canada model assumes that the 5 pathogens studied obey to a lognormal distribution. The lognormal distribution is a commonly used distribution for describing microbial populations (Williams & Ebel, 2014). One advantage of the log normal distribution is that while combining the oocysts/cycts in source water with treatment efficacy, the overall variance can be easily defined from the variance of each individual elements (Medema et al., 2009). When the proportion of data below detection limit become important, Taylor et al. (2001) suggested the use of alternative models (other than the lognormal distribution) to account for these data in order avoid biased parameter estimates and potentially misleading inferences. Furthermore, the results illustrated in appendix 3 proved that the assumption of the HC model is not always valid since pathogens concentrations do not always follow a log normal distribution (Refer to appendix 3). This is one of the limitations within HC QMRA model. Moreover, BDL

data proved to be an issue mostly for *Cryptosporidium* data (Refer to Table A.2-1). Subsequently, and since statistical techniques can be complexes, it would be beneficial if source water data fitting could be performed automatically by a macro and transformed to usable PDFs within the HC model. Such improvements would facilitate the use of the software by the end-users and minimize the bias of the parameters estimates. The Dutch experience is a good example of this approach. The software QMRAspot (Schijven et al., 2011) collect uniformly through automated reading of the unprocessed raw water data, entailing index pathogens data from source water and indicator organism data for treatment efficiency. The software analyses the collected raw microbial data automatically, and assumes that concentrations can be fitted with negative binomial distribution and takes into consideration the non-detects data. Moreover, we used the CCFD method (Smeets et al. 2010) to define if extremes events are dominating the mean pathogen concentrations in raw water. The outcomes presented in appendix 4 is coherent with the vision of Smeets, P., Rietveld, Van Dijik, and Medema (2010) that we should not always rely on the statistical methods and sometimes the visual analysis of a PDF may replace the use of the P-value to assess if the mean concentration is representative of the PDF.

Furthermore, we assessed several correlations (presented in appendix 2) in order to better understand the raw water data. First, *Giardia* and *Cryptosporidium* concentrations were found to be uncorrelated (p-value equal to 0.15) (figure A.2-1) as opposed to many previous studies in the literature (Crabtree, Ruskin, Shaw, & Rose, 1996; LeChevallier, Norton, & Lee, 1991; Rose, Darbin, & Gerba, 1988; Rose & Gerba, 1991).This discrepancy can most likely be traced back to the different sources of contamination (river or lake) and the various detection methods utilized in the various studies.

In figure A.2- 2 to A.2- 4, we studied the correlation between the number of samples and the variance, which is the ratio between standard deviation and mean (SD/Mean). Our first expectation was that while the sample size increases, the variance (SD/Mean) would shrink, and consequently reduce our uncertainty about the estimated concentration mean. While, the results did not show any clear correlation between those elements and somehow for some WTPs the variance actually went up. Further research on how the number of samples could impact the HC QMRA outcomes and the identification to which extent we need to proceed with our sampling data would be valuable information to provide to the user of HC QMRA model.

5.2 Treatment Performance

As discussed in the article, the physical-chemical treatment process performances are assessed independently from the raw water data within the HC model, and none of the site-specific parameters such as (pH, Temperature, Turbidity…) are considered. Indeed the HC model provides default values to represent the physico-chemical process performance. Whereas the potential of microbial indicator organisms and particle concentrations to be used in assessing pathogen risk was previously investigated by Brookes et al. (2005), further studies should focus on the use of microbial indicator organisms (such aerobic spore-formers) that could be integrated within the HC QMRA model to improve the accuracy of risk calculations while bearing in mind that this model was created in a perspective to provide a user-friendly tool to end-users.

Our study showed clearly the significant impact of the treatment prediction methods on the health risk estimates, and proved our second hypothesis about the bias generated from the selected CT method. This phenomenon has been described earlier by Lev, and Regli (1992). The use of the CT_{50} will overestimate disinfection efficacy (the hydraulic efficacy not considered) (Figure A.8-1) while CT_{10} tends to overestimate at high inactivation and underestimate at low inactivation the disinfection. The regulatory method CT_{10} is not recommended in the QMRA context as it could produce bias risk estimates (Pfeiffer & Barbeau, 2014). The N-CSTR method is the best alternative simple method as it showed to be less impacted by the inactivation conditions (Pfeiffer & Barbeau, 2014). Consequently, more research is required for finding an optimal approach representing the treatment performance of chemical disinfection processes. In the meantime, water utilities are recommended to use the N-CSTR method while performing a QMRA on their system.

5.3 Risk characterization

By using HC QMRA on 17 WTPs, a global view was provided about the risk estimates predicted in these systems. We conclude that 15 WTPs out of 17 are meeting the WHO and USEPA reference levels under normal operating conditions. The exceptions were found for two WTPs (WTP 1 and WTP 2) in which *Giardia* and *Cryptosporidium* control was above the WHO and USEPA reference risk levels due to the use of direct filtration without coagulation. This allows

the identification of the strength and weaknesses within a treatment train, which could form a semi-quantitative basis for a higher scale risk communication to the higher-level water managers and politicians.

HC QMRA model ignores all exposure to pathogenic microorganisms that could occur through drinking water distribution (Payment, 1989). Therefore, the microbial risk provided by the HC model is considered as a partial view. A provision to account the microbial risk of the distribution systems within the overall microbial risk management strategy is recommended. Consequently, more research is required for incorporating the microbial risk from drinking water distribution within the HC QMRA model.

One limitation within this study is that the sensibility of the HC QMRA model over different parameters such as recovery, infectious rate, ingested volume, physical-chemical treatment or disinfection, etc. was not evaluated…. Further assessment would allow defining the critical parameters, that is the ones, which have the highest impact on the risk estimates.

CONCLUSION AND RECOMMENDATIONS

The HC QMRA model proved to be useful to estimate pathogen health risk from drinking water consumption. The USEPA and WHO reference risk levels were met in all 17 Canadian WTPs plants, except for two WTPs where protozoan removals were too low. The different scenarios assessed within this study are relatively simple to implement by water utilities and risk managers and illustrate the flexibility of the HC model. However, we propose some recommendations, which should improve the accuracy of the overall risk outcomes. First the source water characterization showed to be complex; a standardization of source water data handling using macros within the HC model would facilitate its application and improve the accuracy of the risk estimates. Second, the health risk provided by the model represents the risk that pathogens be present in the source water and inactivated or removed by the treatment process and therefore ignores the vulnerability of the distribution system. A provision to consider this issue in the model would increase the usefulness of the overall risk outcomes. Second, for the physical-chemical treatment, integrating performance indicators or additional parameters such turbidity, would improve the accuracy of risk calculations. Fourth, N-CSTR method proved to be the most optimum method in contrast with the CT_{50} and CT_{10}. Integrating such treatment prediction method in HC QMRA model will ameliorate the accuracy of the health risk outcomes.

Finally, knowing that each water utility is unique, the benefice from utilizing HC QMRA would be related to the users learning more about their own system. HC model may be used by water treatment utilities as a tool to be integrated within the larger context of developing a water safety plan. In that way, the Health Canada QMRA model provides at the same time an opportunity and challenges for water utilities users. However, this experience gained from implementing a site-specific QMRA should proved to be useful in the management of their water facility

REFERENCES

(Homeland Security). (2013). *Threat and hazard identification and risk assessment guide - Comprehensive Preparedness Guide (CPG) 201*.

Abulreesh, H. H., Paget, T. A., & Goulder, R. (2006). Campylobacter in Waterfowl and Aquatic Environments: Incidence and Methods of Detection. *Enviromental Science and Technology, 40*(23), 7122-7131.

Adam, R. D. (2000). The Gardia lamblia genome. *International Journal for Parasitology, 30* 475-484.

Altekruse, S. F., & Tollefson, L. K. (2003). Human campylobacteriosis: a challenge for the veterinary profession. *Journal of the American Veterinary Medical Association, 223*(4), 445-452.

Ashbolt, N. J. (2004). Risk analysis of drinking microbial contamination versus disinfection by-products (DBPs). *Toxicology, 198* 255-262.

Barbeau, B., Huffman, D., Mysore, C., Desjardins, R., Clement, B., & Prevost, M. (2005). Examination of discrete and counfounding effects of water quality parameters during the inactivation of MS2 phages and Bacillus subtilis spores with chlorine dioxide. *Journal of Environmental Engineering and Science, 4* 139-151.

Bichai, F., & Smeets, P. W. M. H. (2013). Using QMRA-based regulation as a water quality management tool in the water security challenge: Experience from the Netherlands and Australia. *Water Research, 47*(20), 7315-7326.

Blaser, M. J., Perez, G. P., Smith, P. F., Patton, C., Tenover, F. C., Lastovica, A. J., & Wang, W. I. (1986). Extraintestinal Campylobacter jejuni and Campylobacter coli infections: host factors and strain characteristics. *Journal of Infectious Diseases, 153*(3), 552-559.

Brookes, J. D., Hipsey, M. R., Burch, M. D., Regel, R. H., Linden, L. G., Ferguson, C. M., & Antenucci, J. P. (2005). Relative value of surrogate indicators for detecting pathogens in lakes and reservoirs. *Enviromental Science and Technology, 39* 8614-8621.

Cabral, J. P. S. (2010). Water microbiology. bacterial pathogens and water. *Enviromental Research and Public Health, 7*(10), 3657-3703.

Caccio, S. M., & Ryan, U. (2008). Molecular epidemiology of Giardiasis. *Molecular & Biochemical Parasitology, 160*(2), 75-80.

Carey, C. M., Lee, H., & Trevors , J. T. (2004). Biology, persistence and detection of Cryptosporidium parvum and Cryptosporidium hominis oocyst. *Water Research, 38*(4), 818-862.

Carlson, K., Pier, D., Bellamy, W., Carlson, M., Ducoste, J., Amy, G., . . . Via, S. (2001). *Implementation of the integrated disinfection design framework*. Denver, Colorado, USA: American Water Works Association Research Foundation.

Casman, E. A., Fischhoff, B., Palmgren, C., Small, M. J., & Wu, F. (2000). An Integrated Risk Model of a Drinking-Water – Borne Cryptosporidiosis Outbreak. *Risk Analysis, 20*(4), 495-511.

Clark, R. M., Read, E. J., & Hoff, J. C. (1989). Analysis of inactivation of Giardia Lamblia by chlorine. *Journal of Environmental Engineering, 115*(1), 80-90.

Coleman, M. E., & Marks, H. M. (1999). Qualitative and quantitative risk assessment. *Food Control, 10* 289-297.

Cook, C., Prystajecky, N., Feze, I. N., Joly, Y., Dunn, G., Kirby, E., . . . Isaac-Renton, J. (2013). A comparison of the regulatory frameworks governing microbial testing of drinking water in three Canadian provinces. *Canadian Water Resources Journal, 38*(3), 185-195.

Corso, P. S., Kramer, M. H., Blair, K. A., Addiss, D. G., Davis, J. P., & Haddix, A. C. (2003). Cost of illness in the 1993 waterborne Cryptosporidium outbreak, Milwaukee, Wisconsin. *Emerging Infectious Diseases, 9*(4), 426-431. doi: 10.3201/eid0904.020417

Crabtree, K. D., Ruskin, R. H., Shaw, S. B., & Rose, J. B. (1996). The detection of Cryptosporidium oocysts and Giardia cysts in cistern water in the U.S. Virgin Islands. *Water Research, 30*(1), 208-216.

Dasti, J. I., Tareen, A. M., Lugert, R., Zautner, A. E., & Groß, U. (2010). Campylobacter jejuni: A brief overview on pathogenicity-associated factors and disease-mediating mechanisms. *International journal of Medical Microbiology, 300*(4), 205-211.

Dechesne, M., & Soyeux, E.

Dechesne, M., Soyeux, E., Loret, J. F., Westrell, T., Stenstrom, T. A., Gornik, V., . . . Medema, G. J. (2006). *MicroRisk (Microbiological risk assessment: A scientific basis for managing drinking water safety from source to tap). Pathogens in source water*.

Dechesne, M., & Soyeux, M. (2007). Assessment of source water pathogen contamination. *Journal of Water and Health, 5*(1), 39-50.

deRegnier, D. P., Cole, L., Schupp, D. G., & Erlandsen, S. L. (1989). Viability of Giardia Cysts Suspended in Lake, River, and Tap Water. *Applied and Environmental Microbiology, 55*(5), 1223-1229.

Edberg, S. C., Rice, E. W., Karlin, R. J., & Allen, M. J. (2000). Escherichia coli: The best biological drinking water indicator for public health protection. *Journal of Applied Microbiology, 88* 106S-116S.

Eisenberg, J. N., Cevallos, W., Ponce, K., Levy, K., Bates, S. J., Scott, J. C., . . . Trostle, J. (2006). Environmental change and infectious disease: How new roads affect the transmission of diarrheal pathogens in rural Ecuador. *Proceedings of the National Academy of Sciences of the United States of America, 103*(51), 19460–19465.

Emelko, M. B., Schmidt, P. J., & Roberson, J. A. (2008). Quantitification of uncertainty in microbial data - reporting and regulatory implications. *Journal American Water Works Association, 100*(3), 94-104.

Fayer, R., Speer, C. A., & Dubey, J. P. (1997). *Cryptosporidium and Cryptosporidiosis*. Boka Raton: CRC Press.

Ferguson, C., Husman, A. M. d. R., Altavilla, N., Deere, D., & Ashbolt, N. (2003). Fate and transport of surface water pathogens in watersheds. *Enviromental Science and Technology, 33*(3), 299-361.

Field, K. G., & Samadpour, M. (2007). Fecal source tracking, the indicator paradigm, and managing water quality. *Water Research, 41* 3517-3538.

Gale, P. (1996). Developments in microbiological risk assessment models for drinking water-a short review. *Journal of Applied Bacteriology, 81*(4), 403-410.

Gölz, G., Rosner, B., Hofreuter, D., Josenhans, C., Kreienbrock, L., Löwenstein, A., . . . Alter, T. (2014). Relevance of Campylobacter to public health—The need for a One Health approach. *International Journal of Medical Microbiology, 304*(7), 817-823.

Haas, C. N. (1983). Estimation of risk due to low doses of microorganisms: a comparison of alternative methodologies. *American Journal of Epidemiology, 118*(4), 573-582.

Haas, C. N. (2002). Conditional Dose-Response Relationships for Microorganisms: Development and Application. *Risk Analysis, 22*(3), 455-463.

Haas, C. N., Rose, J. B., & Gerba, C. P. (1999). *Quantitative microbial risk assessment* (Vol. 04). New York: John Wiley.

Haas, C. N., Rose, J. B., Gerba, C. P., & Regli, S. (1993). Risk assessment of virus in drinking water. *Risk Analysis, 13*(5), 545-552.

Hartnett, E., McFadyen, S., Douglas, I., Robertson, W., & Paoli, G. (2007). *Quantitative microbiological risk assessment; New tools to assess and manage risks from pathogens in drinking water*. Paper presented at American Water Works Association: Water Quality Technology Conference and Exposition 2007: Fast tracks to Water Quality, Ottawa.

Harwood, V. J., Levine, A. D., Scott, T. M., Chivukula, V., Lukasik, J., Farrah, S. R., & Rose, J. B. (2005). Validity of the Indicator Organism Paradigm for Pathogen Reduction in Reclaimed Water and Public Health Protection. *Applied and Environmental Microbiology, 71*(6), 3163-3170.

Havelaar, A. H., & Melse, J. M. (2003). *Quantifying public health risk in the WHO Guidelines for Drinking-Water quality: a burden of disease approach* (Report No. RIVM report 734301022). Bilthoven, Netherlands: World Health Organization

Health Canada. (2000). *waterborne outbreak of gastroenteritis associated with contaminated municipal water supply*. Walkerton, Ontario:

Health Canada. (2010). *Enteric viruses in drinking water*. Federal-Provincial-Territorial Committee on Drinking Water. Retrieved from http://www.hc-sc.gc.ca/ewh-semt/alt_formats/hecs-sesc/pdf/consult/_2010/enteric-enteriques/enteric-enteriques-eng.pdf

Health Canada. (2011a). *Enteric protozoa: Giardia and Cryptosporidium*. Ottawa, ON, Canada: Federal-Provincial-Territorial Committee on Drinking Water.

Health Canada. (2011b). *Guidelines for Canadian Drinking Water Quality: guidline technical document- Enteric Viruses*.

Health Canada. (2012a). *Guidance on Waterborne Bacterial Pathogens*.

Health Canada. (2012b). *Guidelines for Canadian drinking water quality: guideline technical document- Enteric Protozoa: Giardia and Cryptosporidium*.

Hijnen, W. A. M., & Medema, G. J. (2010). *Elimination of micro-organisms by drinking water treatment processes* (3rd edition ed.). Nieuwegein: IWA publishing.

Howard, G., Pedley, S., & Tibatemwa, S. (2006). Quantitative microbial risk assessment to estimate health risks attributable to water supply: can the technique be applied in developing countries with limited data? *Journal of Water and Health, 4*(1), 49-65.

Hunt, N. K., & Mariñas, B. J. (1999). Inactivation of Escherichia coli with ozone: chemical and inactivation kinetics. *Water Research, 33*(11), 2633-2641.

Hunter, P. B. (1997). *Waterborne Disease: Epidemiology and ecology*. Chichester: Wiley.

Hunter, P. B. (2003). Drinking water and diarrheal disease due to Escherichia coli. *Journal of Water and Health, 1*(2), 65-72.

Jaidi, K., Brabeau, B., Carriere, A., Desjardins, R., & Prevost, M. (2009). Including operational data in QMRA model: development and impact of model inputs. *Journal of Water and Health, 7*(1), 77-95.

Johnson-White, B., Lin, B., & Ligler, F. S. (2007). Combination of immunosensor detection with viability testing and confirmation using the polymerase chain reaction and culture. *Analytical Chemistry, 79*(1), 140-146.

Jore, S., Viljugrein, H., Brun, E., Heier, B. T., Borck, B., Ethelberg, S., . . . Hofshagen, M. (2010). Trends in Campylobacter incidence in broilers and humans in six European countries, 1997–2007. *Preventive Veterinary Medicine, 93*(1), 33-41.

Kaplan, S., & Garrick, B. J. (1981). On The Quantitative Definition of Risk. *Risk Analysis, 1*(1), 11-27.

Keer, J. T., & Birch, L. (2003). Molecular methods for the assessment of bacterial viability. *Journal of Microbiological Methods, 53*(2), 175-183.

King, J. B., Keegan, A. R., Monis, P. T., & Saint, C. P. (2005). Environmental temperature controls Cryptosporidium oocsyt metabolic rate and associated retention of infectivity. *Applied and Environmental Microbiology, 71*(7), 3848-3857.

Korich, D. G., Mead, J. R., Madore, M. S., Sinclair, N. A., & Sterling, C. R. (1990). Effects of ozone, chlorine dioxide, chlorine and monochloramine on Cryptosporidium parvum oocyst viability. *Applied and Environmental Microbiology, 56*(5), 1423-1428.

Krewski, D., J., B., Butler-Jones, D., Haas, C. N., Isaac-Renton, J., Roberts, K. J., & Sinclair, M. (2004). Managing the microbiological risks of drinking water. *Journal of Toxicology and Environmental Health. Part A, 67*(20-22), 1591-1617.

Lack, T. (1999). Water and health in Europe: an overview. *BMJ, 318*(7199), 1678-1682.

Lawler, D. F., & Singer, P. C. (1993). Analyzing disinfection kinetics and reactor design: a conceptual approach versus the SWTR. *Journal of the American Water Works Association, 85*(11), 67-76.

LeChevallier, M. W., & Au, K.-K. (2004). *Water Treatment and Pathogen Control - Process Efficiency in Achieving Safe Drinking Water*. World Health Organization (WHO)

LeChevallier, M. W., Norton, W. D., & Lee, R. G. (1991). Occurrence of Giardia and Cryptosporidium spp. in surface water supplies. *Applied and Environmental Microbiology, 57*(9), 2610-2616.

Lev, O., & Regli, S. (1992). Evaluation of ozone disinfection systems: characteristic time T. *Environmental Engineering, 118*(2), 268-285.

Macler, B. A., & Regli, S. (1993). Use of microbial risk assessment in setting US drinking water standards. *International Journal of Food Microbiology, 18*(4), 245-256.

Martins, M. T., Rivera, I. G., Clark, D. L., & Olson, B. H. (1992). Detection of virulence factors in culturable Escherichia coli isolates from water samples by DNA probes and recovery of toxin-bearing strains in minimal o-nitrophenol-beta-D-galactopyranoside-4-methylumbelliferyl-beta-D-g luc uronide media. *Applied and Environmental Microbiology, 58*(9), 3095-3100.

Masago, Y., Katayama, H., Watanabe, T., Haramoto, E., Hashimoto, A., Omura, T., . . . Ohgaki, S. (2006). Quantitative Risk Assessment of Noroviruses in Drinking Water Based on Qualitative Data in Japan. *Environmental Science and Technology, 40*(23), 7428-7433.

Matthijnssens, J., Ciarlet, M., Rahman, M., Attoui, H., Ba´nyai, K., Estes , M. K., . . . Van Ranst, M. (2008). Recommendations for the classification of group A rotaviruses using all 11 genomic RNA segments. *Archives of Virology, 153*(8), 1621-1629.

McFadyen, S., Brooks, T., Andrews, R., Baxter, C., Hartnett, E., & Paoli, G. (2009). *QMRA experiences in the development of Canadian Drinking Water Guidelines and in water treatment plants.* Paper presented at AWWA WQCT Conference Proceedings (pp. 1-2).

McFadyen, S., Douglas, I., & J., E. (2011). *QMRA- Probabilistic model to estimate annual risk of illness & DALYs based on source water pathogens and treatment barriers. Version 11-07-Rev.*

McFeters, G. A., Pyle, B. H., Lisie, J. T., & Broadaway, S. C. (1999). Rapid direct methods for enumeration of specific, active bacteria in water and biofilms. *Symposium series (Society for applied microbiology), 85*(28), 193S-200S.

Medema, G., & Ashbolt, N. (2006). *MicroRisk (Microbiological risk assessment: A scientific basis for managing drinking water safety from source to tap). QMRA: its value for risk management*.

Medema, G., Teunis , P. F. M., Blokker, M., Deere, D., Davison, A., Charles, P., & Loret, J. F. (2009). *Risk assessment for Cryptosporidium in drinking-water*. Geneva: World Health Organization,.

Messner, M. J., Chappell, C. L., & Okhuysen, P. C. (2001). Risk assessment for cryptosporidium: A hierarchical Bayesian analysis of human dose response data. *Water Research, 35*(16), 3934-3940.

Monis, P. T., & Thompson, R. C. A. (2003). Cryptosporidium and Giardia-zoonoses: fact or fiction? . *Infection, Genetic and Evolution, 3*(4), 233-244.

Murray, C. J., & Lopez, A. D. (1997). Alternative projections of mortality and disability by cause 1990–2020: Global Burden of Disease Study. *The Lancet, 349*(9064), 1498-1504.

New Zealand Ministry of Health. (2008). *Drinking-water standards for New Zealand 2005 (revised 2008)*. Wellington: New Zealand Ministry of Health

Nikolaidis, N. P., Heng, H., Semagin, R., & Clausen, J. C. (1998). Non-linear response of a mixed land use watershed to nitrogen loading. *Agriculture, Ecosystems and Environment, 67*(2-3), 251-265.

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. *Journal of Infectious Diseases, 180*(4), 1275-1281.

Okhuysen, P. C., Chappell, C. L., Sterling, C. R., Jakubowski, W., & DuPont, H. L. (1998). Susceptibility and serologic response of healthy adults to reinfection with Cryptosporidium parvum. *Infection and Immunity, 66*(2), 441-443.

Olivieri, A., & Soller, j. (2002). *Evaluation of the public health risks concerning infectious disease agents associated with exposure to treated wastewater disharged by the city of Vacaville, Easterly wastewater treatment plant*. Vacaville:

Parkhurst, D. F., & Stern, D. A. (1998). Determining Average Concentrations of Cryptosporidium and Other Pathogens in Water. *Enviromental Science and Technology, 32*(21), 3424-3429.

Payment, P. (1989). Bacterial colonization of domestic reverse-osmosis water filtration units. *Canadian Journal of Microbiology, 35*(11), 1065-1067.

Payment, P., & Locas, A. (2011). Pathogens in water: value and limits of correlation with microbial indicators. *Ground Water, 49*(1), 4-11.

Pfeiffer, V., & Barbeau, B. (2014). Validation of a Simple Method for Predicting the Disinfection Performance in a Flow-through Contactor. *Water Research, 49*(1), 144-156.

Public Health Agency of Canada. (2011). *Human rotavirus-pathogen safety data sheet- infectious substances*.

Rajendran, P., & Kang, G. (2014). Molecular epidemiology of rotavirus in children and animals and characterization of an unusual G10P[15] strain associated with bovine diarrhea in south India. *Vaccine, 32S* A89-A94.

Rakness, K. L., Najm, I., Elovitz, M., Rexing, D., & Via, S. (2005). Cryptosporidium log-inactivation with ozone using effluent CT10, geometric mean CT10, extended integrated CT10 and extended CSTR calculations. *Ozone Science & Engineering Journal, 27*(5), 335-350.

Raphael, R. A., Sattar, S. A., & Springthorpe, V. S. (1985). Long-term survival of human rotavirus in raw and treated river water. *Canadian Journal of Microbiology, 31*(2), 124-128.

Regli, S., Rose, J. B., Haas, C. N., & Gerba, C. P. (1991). Modeling the risk from Giardia and viruses in drinking water. *Journal of the American Water Works Association 83*(11), 76-84.

Rice, E. W., Clark, R. M., & Johnson, C. H. (1999). Chlorine inactivation of Escherichia coli O157:H7. *Emerging Infectious Disease, 5*(3), 461-463.

Rizak, S., & Sinclair, M. (2001, June 2001). Drinking water quality management: Australia's new approach. *Water21*(3.3)*,* 18-23.

Rodger, S. M., Bishop, R. F., Birch, C., McLean, B., & Holmes, I. H. (1981). Molecular epidemiology of human rotaviruses in Melbourne, Australia, from 1973 to 1979, as determined by electrophoresis of genome ribonucleic acid. *Journal of Clinical Microbiology, 13*(2), 272-278.

Rose, J. B., Darbin, H., & Gerba, C. P. (1988). Correlations of the Protozoa, Cryptosporidium and Giardia, with Water Quality Variables in a Watershed. *Water Science and Technology, 20*(11-12), 271-276.

Rose, J. B., & Gerba, C. P. (1991). Use of risk assessment for development of microbial standards. *Water Science and Technology, 24*(2), 29-34.

Rose, J. B., & Haas, C. N. (1991). Risk assessment and control of waterborne Giardiasis. *American Journal of Epidemiology, 81*(6), 709-713.

Roseberry, A. M., & Burmaster, D. E. (1992). Log-normal distributions for water intake by children and adults. *Risk Analysis, 12*(1), 99-104.

Rosen, B. H. (2000). *Waterborne pathogens in agricultural watersheds*. NY: Watershed science institute,.

Roser, D. J., Ashbolt, N. J., Davies, C. M., Glamore, W. C., Hawker, K. M., & Miller, B. M. (2007). *Assessment principles for pathogen management at an urban recreation lake.* Paper presented, Texas (pp. 420-426).

Scheutz, F., & Strokbine, N. (2005). Genus Escherichia. In *Bergey's manual of Systematic Bacteriology* (2nd edition ed., Vol. 2, pp. 607-623). USA.

Schijven, J. F., Teunis, P. F. M., Rutjes, S. A., Bouwknegt, M., & Husman, A. M. d. R. (2011). QMRAspot: a tool for quantitative microbial risk assessment from surface water to potable water. *Water Research, 45*(17), 5564-5576.

Schilling, K. E., Zhang, Y., Hill, D. R., Jones, C. S., & Wolter, C. F. (2009). Temporal variations of Escherichia coli concentrations in a large Midwestern river. *Journal of Hydrology, 365* 79-85.

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 Science and Technology, 54*(3), 261-268.

Smeets, P., Medema, G., Kruidenier, L., & Havelaar, A. H. (2007). How can the UK statutory Cryptosporidium monitoring be used for Quantitative Risk Assessment of Cryptosporidium in drinking water. *Journal of Water and Health 05.S1* 107-118. doi: 10.2166/wh.2007.140.

Smeets, P., Rietveld, L., Hijnen, W., Medema, G., & Stenström, T. (2006). *MicroRisk (Microbiological Risk Assessment: A Scientific Basis for Managing Drinking Water Safety from Source to Tap). Efficacy of water treatment processes*.

Smeets, P., Rietveld, L., Van Dijik, J. C., & Medema, G. (2010). Practical applications of quantitative microbial risk assessment (QMRA) for water safety plans. *Water Science and Technology, 61*(6), 1561-1568.

Smeets, P. W. M. H., Van der Helm, A. W. C., Dullemont, Y. J., L.C., R., Van Dijik, J. C., & Medema, G. J. (2006). Inactivation of Escherichia coli by ozone under bench-scale plug flow and full-scale hydraulic conditions. *Water Research, 40*(17), 3239-3248.

Smith, H., & Grimason, A. (2003). Giardia and Cryptosporidium in Water and Wastewater. In D. Mara & N. J. Horan (Eds.), *The Handbook of Water and Wastewater Microbiology* (pp. 696-755). Academic, London.

Sobsey, M. D. (1999). Methods to identify and detect microbial contaminants in drinking water. In *Identifying future drinking water contaminants* (pp. 173-205). Ottawa.

Soller, J. A., Eisenberg, J. N. S., DeGearge, J. F., Cooper, R. C., Tchobanoglous, G., & Olivieri, A. W. (2006). A public health evaluation of recreational water impairment. *Journal of Water and Health* 1-19.

Soller, J. A., Olivieri, A. W., Crook, J., Cooper, R. C., Tchobanoglous, G., Parkin, R. T., & Eisenberg, J. N. (2003). Risk-based approach to evaluate the public health benefit of additional wastewater treatment *Environmental Science and Technology, 37*(9), 1882-1891.

Strachan, N. J., Doyle, M. P., Kasuga, F., Rotariu, O., & Ogden, I. D. (2005). Dose response modelling of Escherichia coli O157 incorporating data from foodborne and environmental outbreaks. *International Journal of Food Microbiology, 103*(1), 35-47.

Surendran, S. (2008). Rotavirus infection: molecular changes and pathophysiology *EXCLI Journal 7*154-162.

Taylor, D. J., Kupper, L. L., Rappaport, S. M., & Lyles, R. H. (2001). A mixture model for occupational exposure mean testing with a limit of detection. *Biometrics, 57* 681-688.

Teunis, P. F., & Havelaar, A. H. (2000). The beta poisson dose-response model is not a single-hit model. *Risk analysis, 20*(4), 513-520. Retrieved from http://onlinelibrary.wiley.com/doi/10.1111/0272-4332.204048/pdf

Teunis, P. F. M., & Havelaar, A. H. (2002). Risk assessment for protozoan parasites. *International Biodeterioration and Biodegradation, 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 Research, 31*(6), 1333-1346.

Teunis, P. F. M., Nagelkerke, N. J. D., & Haas, C. N. (1999). Dose response models for infectious gastroenteritis. *Risk Analysis, 19*(6), 1251-1260.

Teunis, P. F. M., Van Den Brandhof, W., Nauta, M., Wagenaar, J., Van Den Kerkhof, H., & Van Pelt, W. (2005). A reconsideration of the Campylobacter dose-response relation. *Epidemiology and Infection, 133*(04), 583-592.

Thompson, R. C. A. (2004). The zoonotic significance and molecular epidemiology of Giardia and giardiasis. *Veterinary Parasitology, 126*(1-2), 15-35. Retrieved from http://www.sciencedirect.com/science/article/B6TD7-4DN1HXV-5/2/96ef1972b8a8927803a0513e9b83ee20

Travis, C. C., & Land, M. L. (1990). Estimating the mean of data sets with nondetectable values. *Enviromental Science and Technology, 24*(7), 961-962.

Tzipori, S., & Ward, H. (2002). sporidiosis: biology, pathogenesis and disease. *Microbes and Infection, 4*(10), 1047-1058. doi: 10.1016/S1286-4579(02)01629-5

USEPA. (1989). *National primary drinking water regulations: Filtration, disinfection, turbidity, Giardia*

lamblia, viruses, Legionella, and heterotrophic bacteria. Washington, D.C.: U.S. Environmental Protection Agency,.

USEPA. (1991a). *Guidance manual for compliance with the filtration and disinfection requirements for public water systems using surface water sources*. Washington, D.C.: U.S. Environmental Protection Agency,.

USEPA. (1991b). *Guidance manual for compliance with the surface water treatment requirements for public water systems*. Washington, D.C.: U.S. Environmental Protection agency,.

USEPA. (1999). *Disinfection profiling and benchmarking manual*.

USEPA. (2003). *LT2ESWTR Long Term Second Enhanced Surface Water Treatment Rule and Draft*

Toolbox Guidance Manual USEPA. Washington DC, USA: U.S. Environmental Protection agency,.

USEPA. (2006). *Federal Register. Part II. National primary drinking water regulations: Long Term 2 Enhanced Surface Water Treatment Rule; final rule*. Washington, DC, USA: Office of Science and Technology, Office of Water.

USEPA. (2008). *Literature review of molecular methods for simultaneous detection of pathogens in water*. Cincinnati, OH: U.S. Environmental Protection Agency,.

USEPA. (2010). *Quantitative microbial risk assessment to estimate illness in freshwater impacted by agricultural animal sources of fecal contamination*.

Van Ryzin, J. (1980). Quantitative risk assessment. *Occupational and Environmental Medicine, 22*(5), 321-326.

Wang, G., & Doyle, M. P. (1998). Survival of Enterohemorrhagic Escherichia coli O157:H7 in Water. *Journal of Food Protection, 61*(6), 662-667.

WHO. (2004). *Guidelines for drinking-water quality*. Geneva: World Health Organization,.

WHO. (2006). *Guidelines for drinking-water quality: incorporating first addendum.* . Geneva: World Health Organization,.

WHO. (2011a, October 2011). Campylobacter. Retrieved from http://www.who.int/mediacentre/factsheets/fs255/en/

WHO. (2011b, December 2011). Enterohaemorrhagic Esherchia coli (EHEC). Retrieved from http://www.who.int/mediacentre/factsheets/fs125/en/

WHO. (2011c). *Guidelines for Drinking-Water Quality*. Geneve, Zwitserland: World Health Organization,.

WHO, & OECD. (2003). *Assessing Microbial Safety of Drinking Water -Improving approaches and methods* IWA Published on behalf of the World Health Organization and the Organisation for Economic Co-operation and Development by.

Williams, M. S., & Ebel, E. D. (2014). Estimating the correlation between concentrations of two species of bacteria with censored microbial testing data. *International Journal of Food Microbiology, 175* 1-5.

Xiao, S., An, W., Chen, Z., Zhang, D., Yu, J., & Yang, M. (2012). The burden of drinking water-associated cryptosporidiosis in China: The large contribution of the immunodeficient population identified by quantitative microbial risk assessment. *Water Research, 46*(13), 4272–4280.

Zhang, J., Huck, P. M., Anderson, W. B., & Stubley, G. D. (2007). A Computational Fluid Dynamics Based Integrated Disinfection Design Approach for Improvement of Full-scale Ozone Contactor Performance. *Ozone: Science & Engineering: The Journal of the International Ozone Association, 29*(6), 451-460.

APPENDICES

APPENDIX 1– Model Input Assumptions & Source Water Data

Table A.1- 1 Model Input Assumptions- (Part 1)

Parameter	Microorganism	Standardized assumption
Source Water Characterization		
Standard deviation	All	▪ It has little impact on output.
Infectivity	All	▪ Assumed equal to 100%.
Recovery	*Cryptosporidium* *Giardia*	▪ 40% (Jaidi et al., 2009). ▪ 69% (Jaidi et al., 2009).
Source water characterization	All	▪ Yearly mean and standard deviation are used in the calculation.
Log normality distribution	All	▪ Using CCDF method to evaluate the quality of the means.
Data Below detection limit	All	▪ Using Arithmetic mean calculated in Statistica while zero value substituted by detection limit.
In absence of data	Rotavirus & Campylobacter *E. coli*	▪ Excluded from the calculation and no investigation was done. ▪ 0.75*fecal coliforms.
Physic-chemical Treatment		
Conventional treatment Direct filtration (with coagulation) Direct filtration (no coagulation) Microfiltration	All	▪ Log reduction as defined by Health Canada QMRA Model.
GAC contactor	All	▪ Considered as rapid granular (no coagulation).

Table A.1- 2 Model Input Assumptions- (Part 2)

Parameter	Microorganism	Standardized assumption
Disinfection		
Chlorination	All	▪ Daily CT calculations paired with microbial measurements according to (pH at reservoir effluent- highest pH) to adopt a conservative approach. ▪ CT calculation is based on maximum inlet flow rate. No info on T_{50}. The T_{50} value was back calculated with CT and residual. ▪ If more than one reservoir is used for post-chlorination, to adopt a conservative approach the inactivation was determined according to the smallest CT value (shortest residence time).
	Cryptosporidium	▪ No impact of chlorination on *Cryptosporidium*, it was removed from the calculation.
Ozonation	All	▪ While having many ozone reservoirs, CT calculation was done in segments, and then the total inactivation was calculated.
Dose Response		
Pathogens	All	▪ As defined by the Heath Canada Model.
Risk Calculation		
Pathogens	All	▪ Risk calculation based on yearly basis for source water and monthly basis for the treatment performance.

Table A.1- 3 Source Water Data

Plant	*Giardia*			*Cryptosporidium*			*E. coli* or Fecal Coliform		
	Mean	STD	Ratio	Mean	STD	Ratio	Mean	STD	Ratio
1	5.4	7.5	0.72	1.466	2.1	0.70	64251	87000	0.74
2	8.2	10	0.82	1.12	2.3	0.49	6879	9200	0.75
3							45303	110000	0.41
4							41099	59000	0.70
5							32377	63000	0.51
6							38548	80000	0.48
7	7.8	13	0.60	0.645	3.5	0.18	43803	53000	0.83
8	10.1	18.55	0.54	1.2	5.6	0.21	61257	81000	0.76
9	31.1	42.5	0.73	1.83	5.3	0.35	238417	250000	0.95
A	4.1	3.6	1.14	2.73	5.5	0.50	3254	13820	0.24
B	6.1	13	0.47	1.9	2	0.95	15956	120750	0.13
C	7.9	5.6	1.41	2.51	2.9	0.87	15750	49300	0.32
D	0.6	1	0.60	1.96	2.4	0.82	54	265	0.20
E							6000	12500	0.48
F	1.2	1.8	0.67	2	2.5	0.8	2050	6570	0.31
G	15.3	31.15	0.49	2.97	7.99	0.37	45433	128000	0.35
H	3.2	4.9	0.65	0.12	0.17	0.71	662	7120	0.09

APPENDIX 2– Correlations ***Correlation Cryptosporidium versus Giardia***

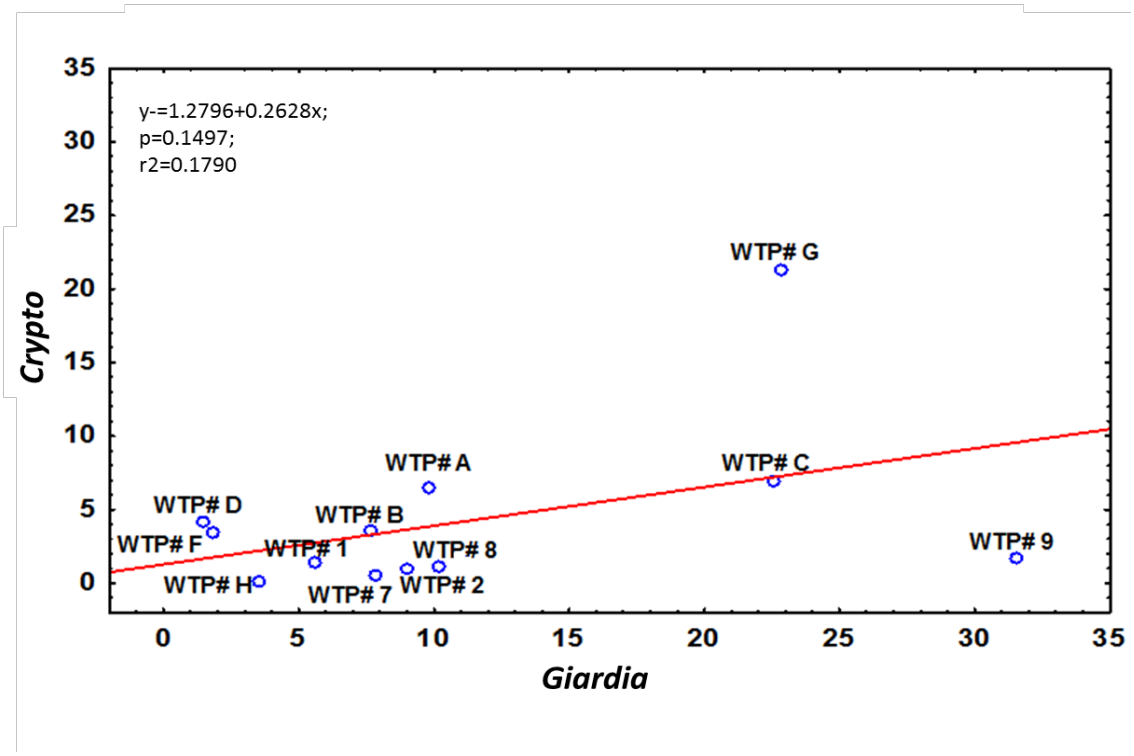

Figure A.2- 1 Correlation *Cryptosporidium/Giardia* (Point estimate)

Correlations between SD/Mean and number of samples for Arithmetic mean

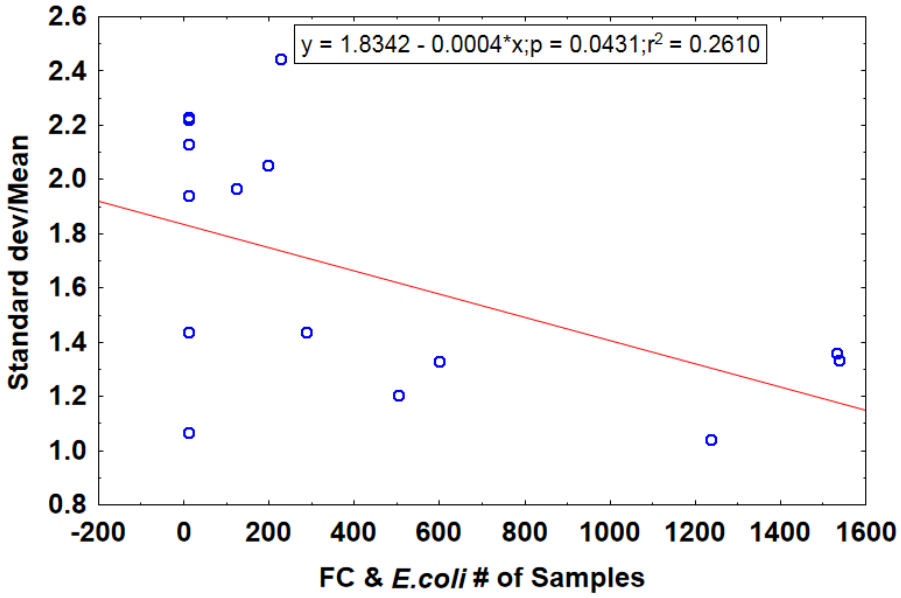

Figure A.2- 2 Correlation SD/Mean versus Number of Samples for FC& *E. coli*- Arithmetic Mean

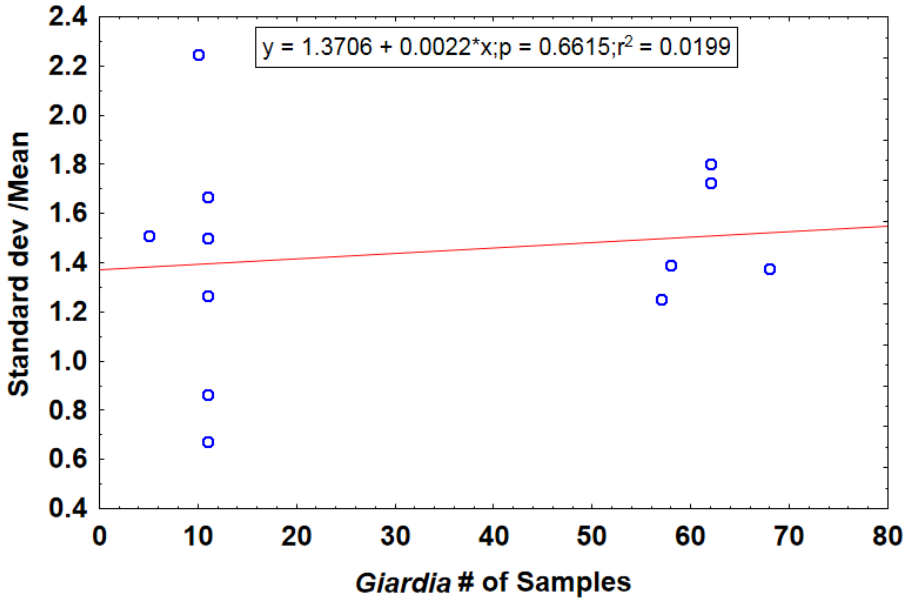

Figure A.2- 3 Correlation SD/Mean versus Number of Samples for *Giardia*-Arithmetic Mean

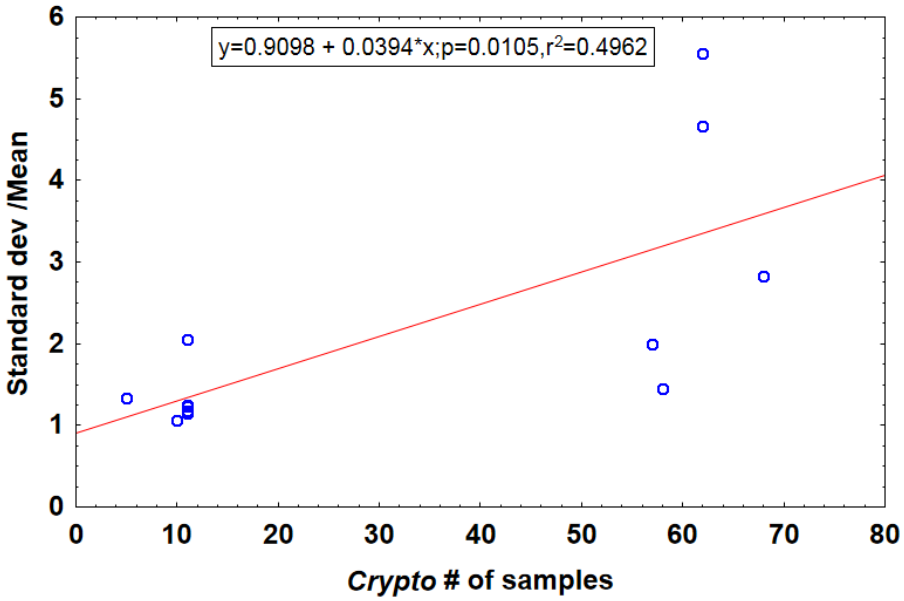

Figure A.2- 4 Correlation SD/Mean versus Number of Samples for *Crypto*-Arithmetic Mean

Correlations between the four approaches of raw water characterization

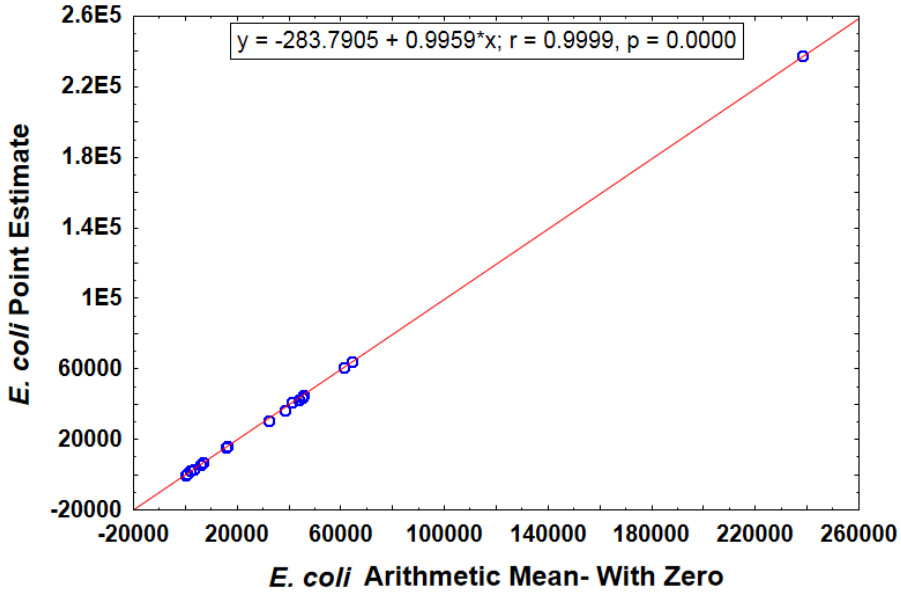

Figure A.2- 5 Correlation *E. coli* Point Estimate versus Arithmetic mean with zero

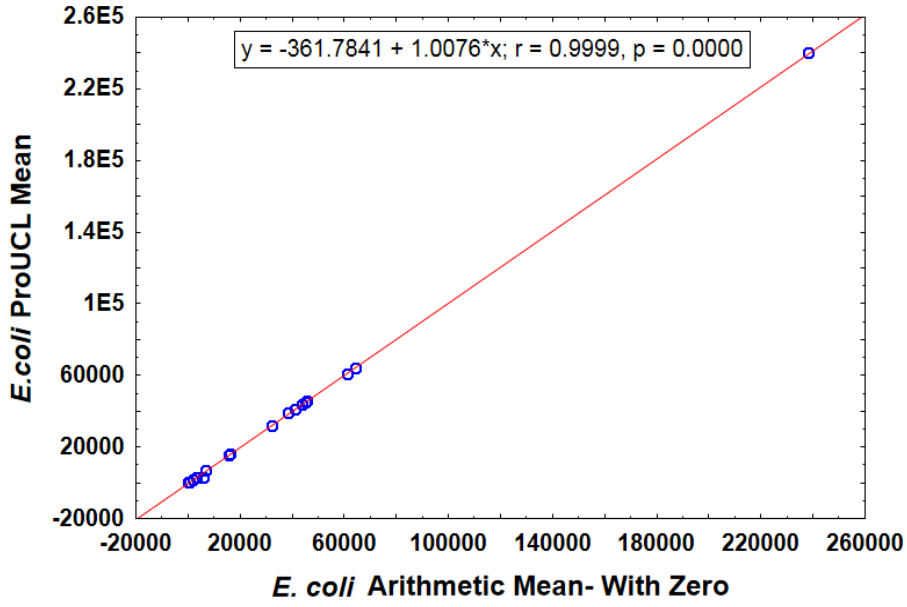

Figure A.2- 6 Correlation *E. coli* ProUCL versus Arithmetic mean with zero

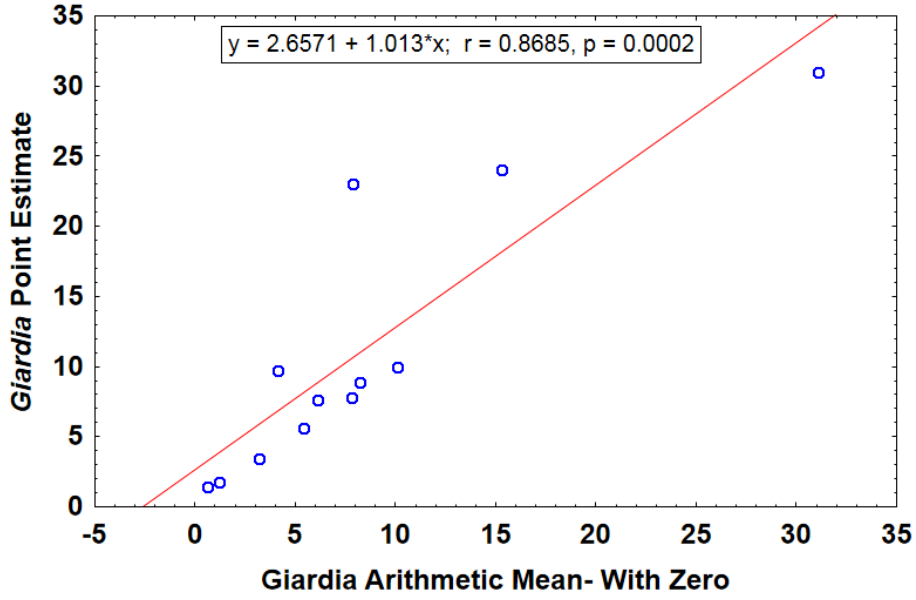

Figure A.2- 7 Correlation *Giardia* Point Estimate versus Arithmetic mean with zero

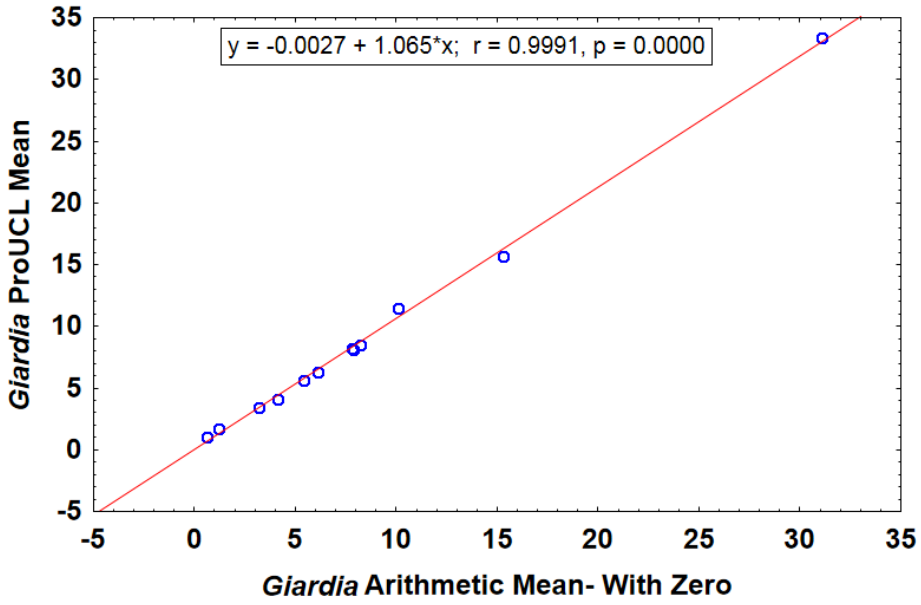

Figure A.2- 8 Correlation *Giardia* ProUCL versus Arithmetic mean with zero

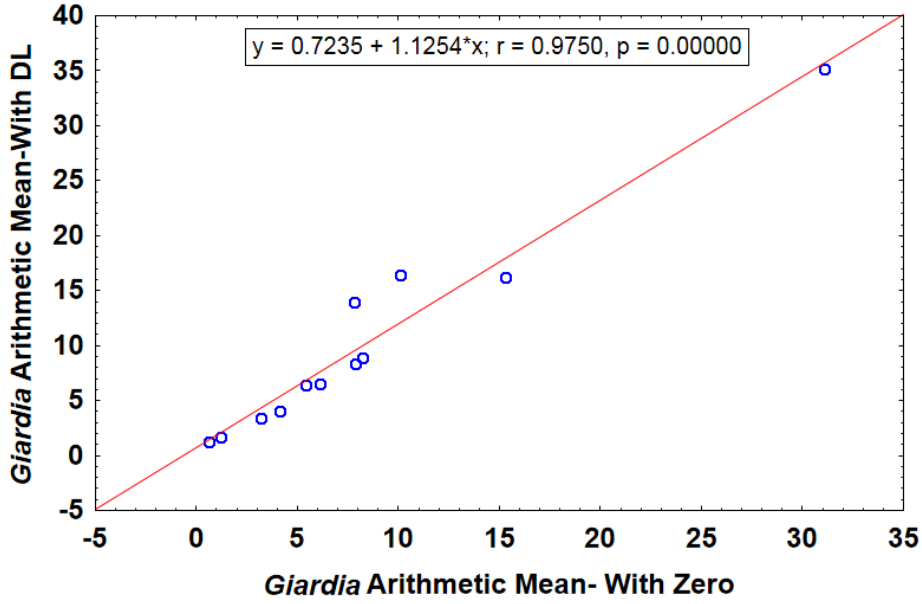

Figure A.2- 9 Correlation *Giardia* Arithmetic mean with DL versus Arithmetic mean with zero

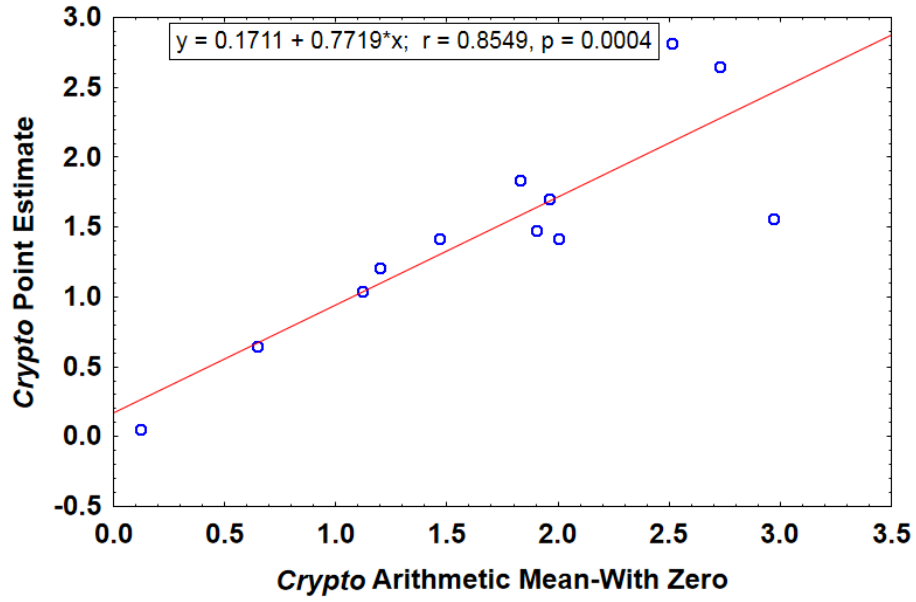

Figure A.2- 10 Correlation *Cryptosporidium*Point Estimate versus Arithmetic mean with zero

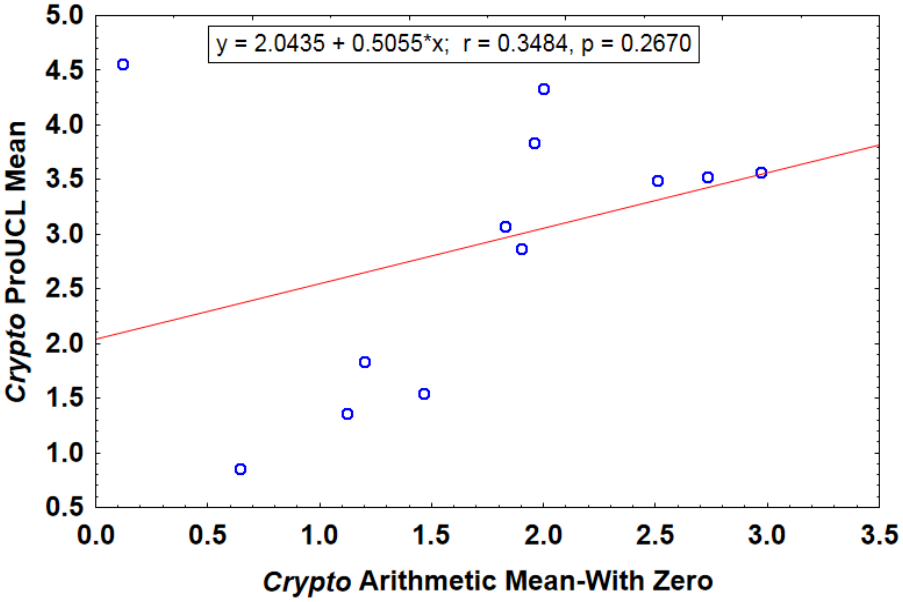

Figure A.2- 11 Correlation *Cryptosporidium*ProUCL versus Arithmetic mean with zero

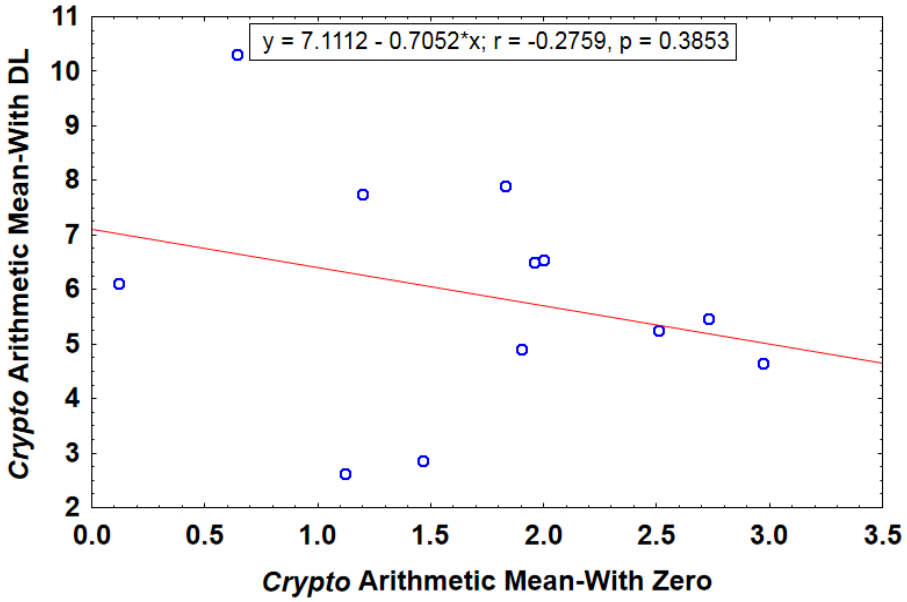

Figure A.2- 12 Correlation *Cryptosporidium* Arithmetic mean with DL versus Arithmetic mean with zero

Table A.2- 1 Correlation summary between the four approaches

	Point Estimate		**ProUCL**		**Arithmetic with DL**	
	Ratio	**R(p-value)**	**Ratio**	**R(p-value)**	**Ratio**	**R(p-value)**
***E.coli*- Arithmetic with zero**	0.99	0.99 (0.00)	1.53	0.99 (0.00)	2.16	--(--)
***Giardia*- Arithmetic with zero**	1.51	0.86 (0.00)	1.13	0.99 (0.00)	1.29	0.97 (0.00)
***Crypto.*- Arithmetic with zero**	1.14	0.85 (0.00)	4.53	0.34 (0.26)	8.08	-0.27 (0.38)

APPENDIX 3–Log normality Graphs

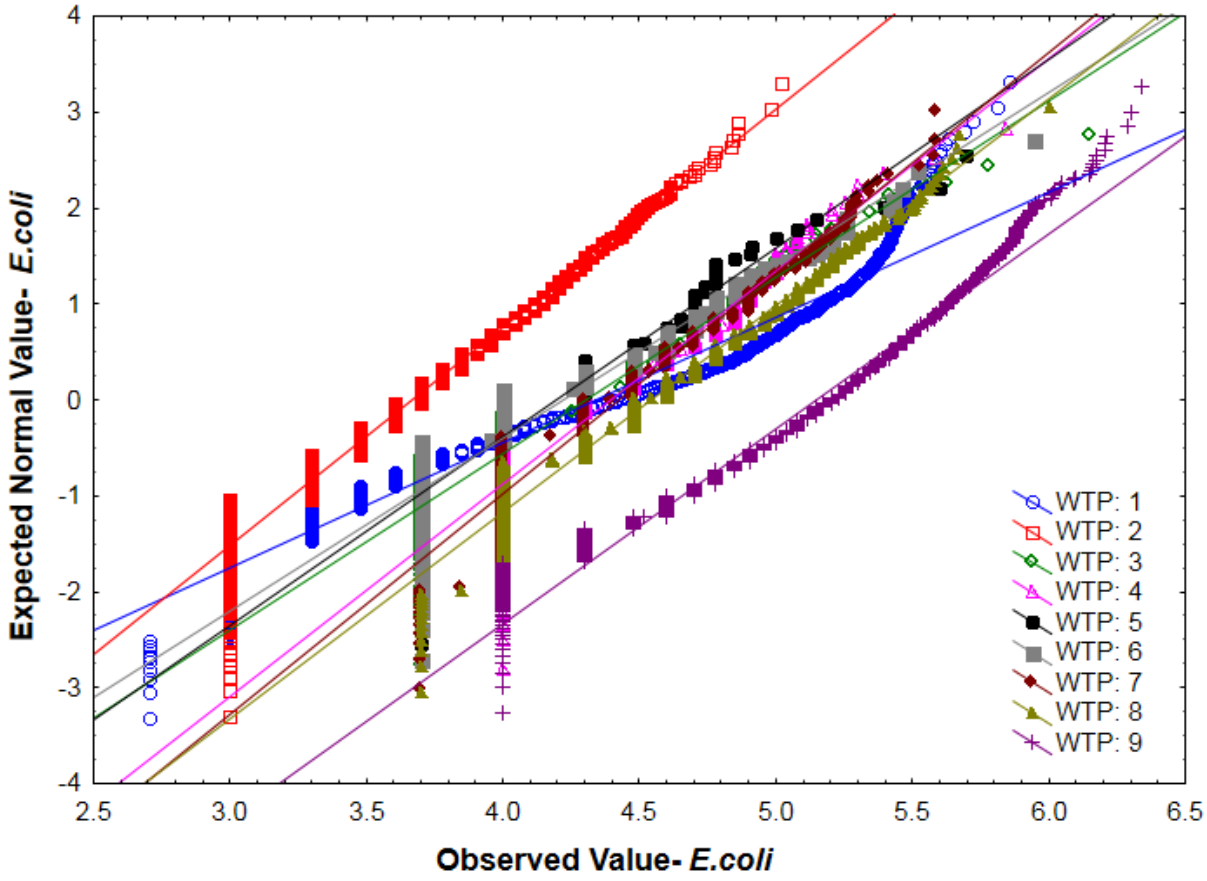

Figure A.3- 1 Log normality graphs for *E. coli* Quebec

Table A.3- 1 Statistics Summary for *E. coli* Quebec

WTP	SW-W	P-VALUE
1	0.9562	0.0000
2	0.9653	0.0000
3	0.9106	0.0000
4	0.844	0.0000
5	0.8832	0.0000
6	0.877	0.0000
7	0.9256	0.0000
8	0.9561	0.0000
9	0.9747	0.0000

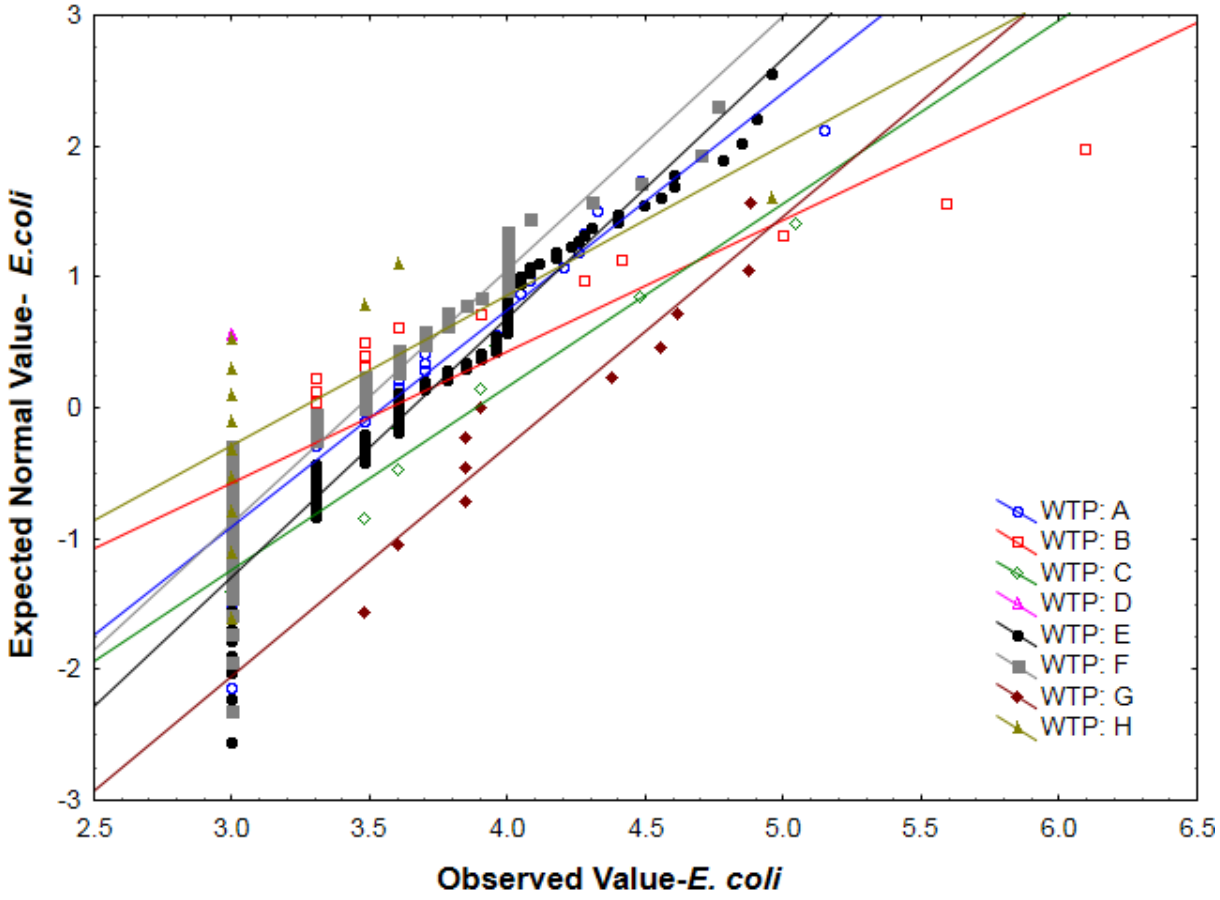

Figure A.3- 2 Log normality graphs for *E. coli* Ontario

Table A.3- 2 Statistics Summary for *E. coli* Ontario

WTP	SW-W	P-VALUE
A	0.8721	0.0004
B	0.7284	0.00002
C	0.9479	0.6897
D	Bad numerical conditions for statistics	
E	0.9404	0.00004
F	0.8608	0.00001
G	0.8957	0.164
H	0.5243	0.00004

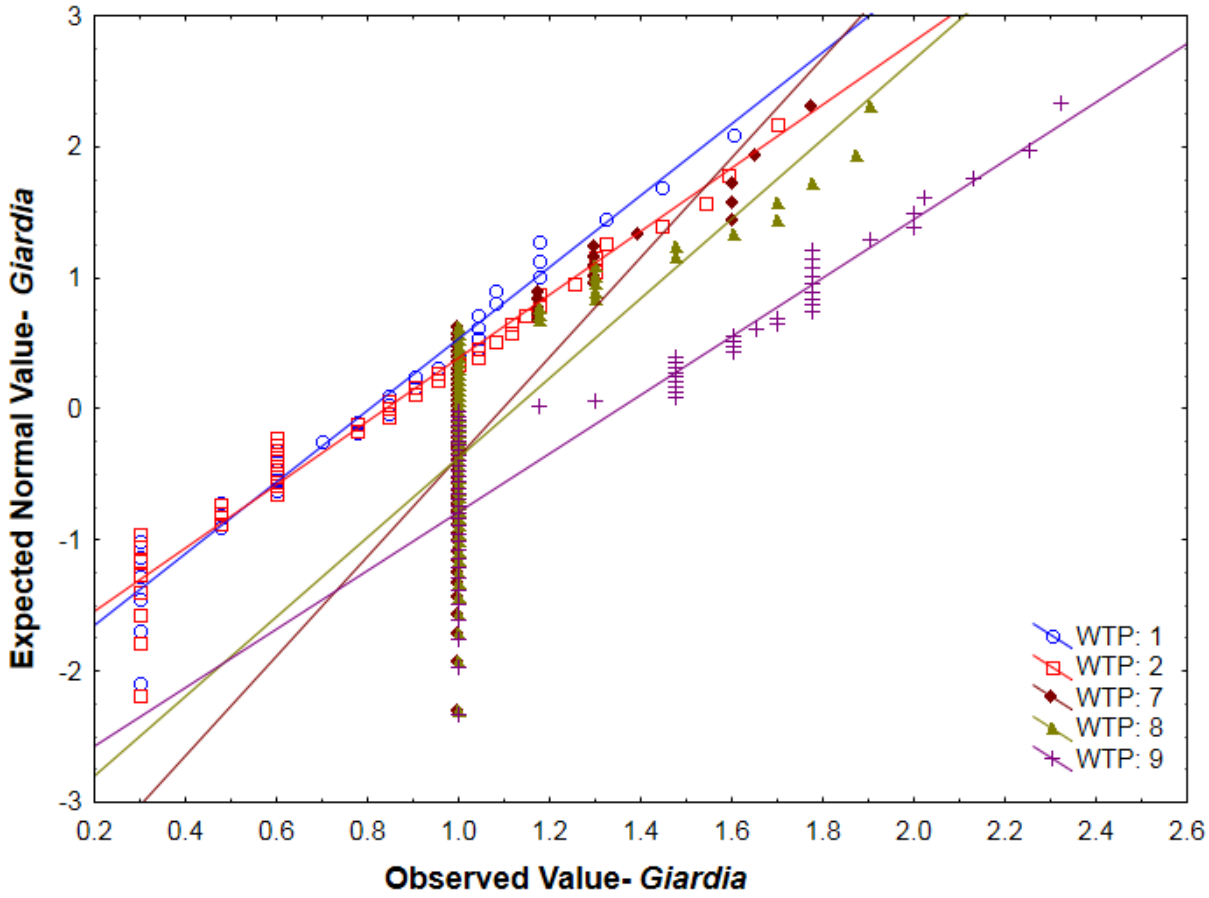

Figure A.3- 3 Log normality graphs for *Giardia* Quebec

Table A.3- 3 Statistics Summary for *Giardia* Quebec

WTP	SW-W	P-VALUE
1	0.9545	0.1447
2	0.9474	0.0406
7	0.5705	0.0001
8	0.5701	0.0001
9	0.8073	0.00001

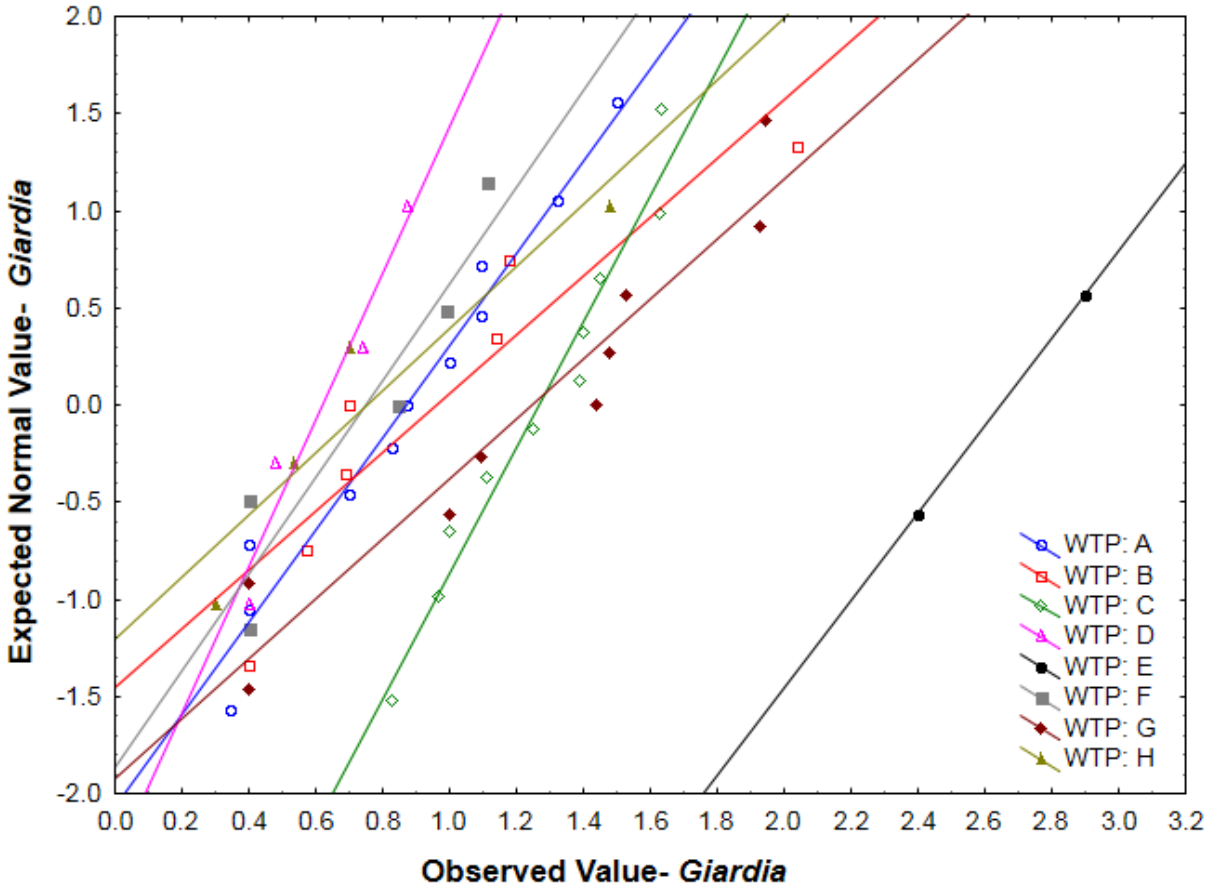

Figure A.3- 4 Log normality graphs for *Giardia* Ontario

Table A.3- 4 Statistics Summary for *Giardia* Ontario

WTP	SW-W	P-VALUE
A	0.9456	0.5882
B	0.8697	0.1845
C	0.9416	0.5709
D	0.9257	0.5697
E	Bad numerical conditions for statistics	
F	0.8569	0.2173
G	0.9048	0.2811
H	0.8959	0.4112

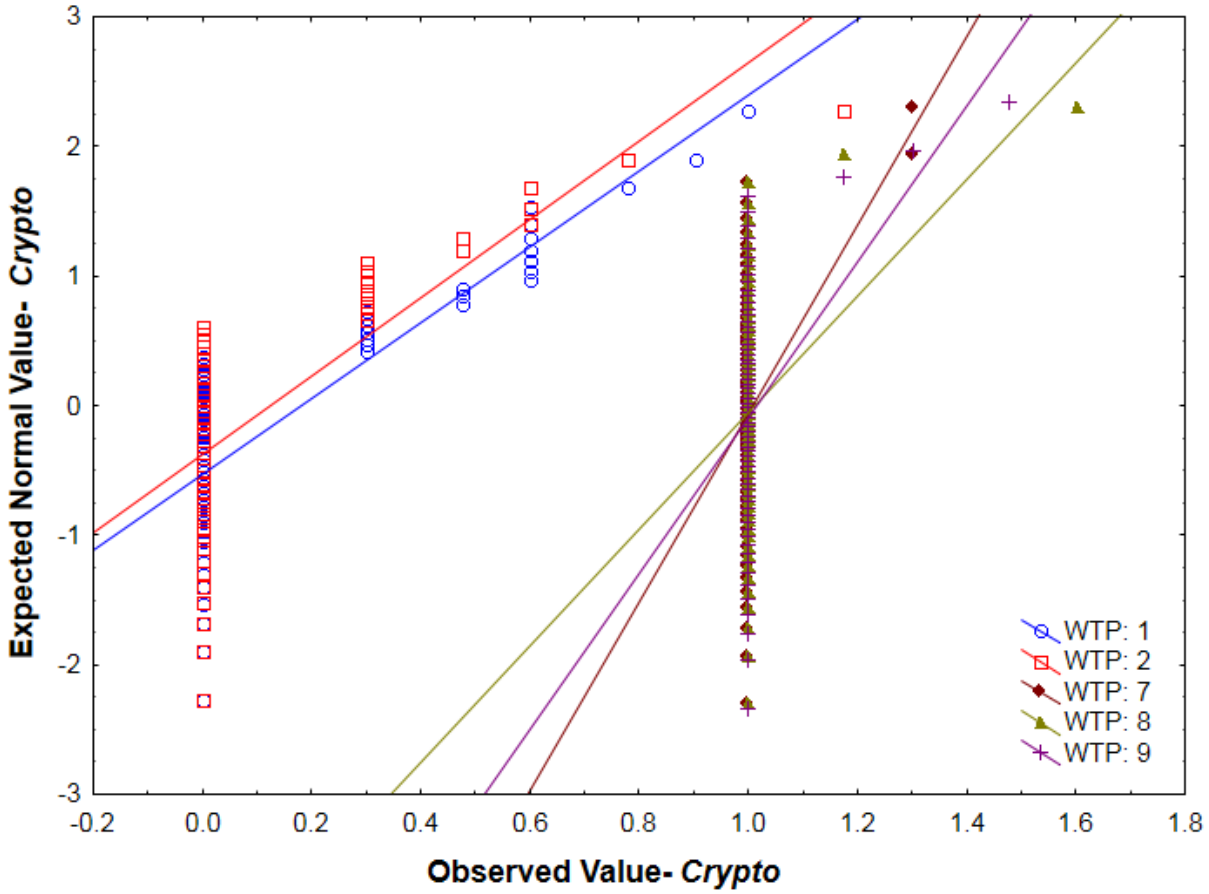

Figure A.3- 5 Log normality graphs for *Cryptosporidium* Quebec

Table A.3- 5 Statistics Summary for *Cryptosporidium* Quebec

WTP	SW-W	P-VALUE
1	0.6865	0.0000
2	0.5827	0.0000
7	0.1714	0.0000
8	0.1499	0.0000
9	0.2036	0.0000

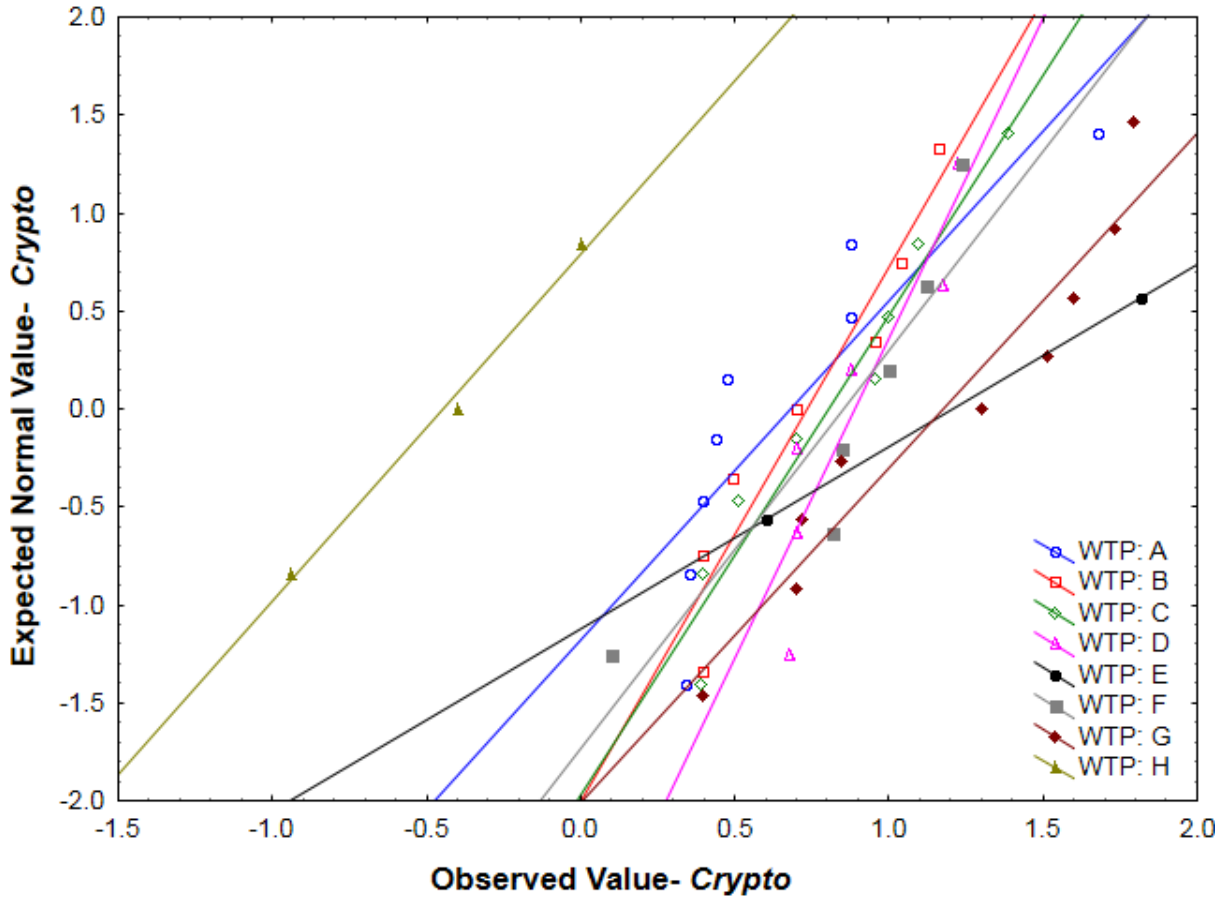

Figure A.3- 6 Log normality graphs for *Cryptosporidium* Ontario

Table A.3- 6 Statistics Summary for *Cryptosporidium* Ontario

WTP	SW-W	P-VALUE
A	0.7623	0.0112
B	0.88	0.2592
C	0.9298	0.5145
D	0.8083	0.0697
E	Bad numerical conditions for statistics	
F	0.8525	0.1649
G	0.905	0.2827
H	0.9919	0.8278

APPENDIX 4–CCDF Graphs

Table A.4- 1 CCDF Results Summary

WTP	1	2	3	4	5	6	7	8	9	A	B	C	D	E	F	G	H
F.C.	96	90	72	100	67	66	N.A.										
E. coli	N.A.						100	100	100	35	**25**	**1**	2	67	35	**0.2**	**6**
Giardia	66	83	N.A.				36	44	78	100	**70**	91	36	100	36	**9**	80
Crypto	52	42	N.A.				3	6	**13**	**73**	70	73	**55**	100	55	8	0

Not enough data available (number of measurement above DL <= 4)

The mean is driven by extreme events (low occurrence of high microbial concentrations)

Data is well distributed

The numbers inside the boxes represent the % of available data above detection limit

CCDF Graphs

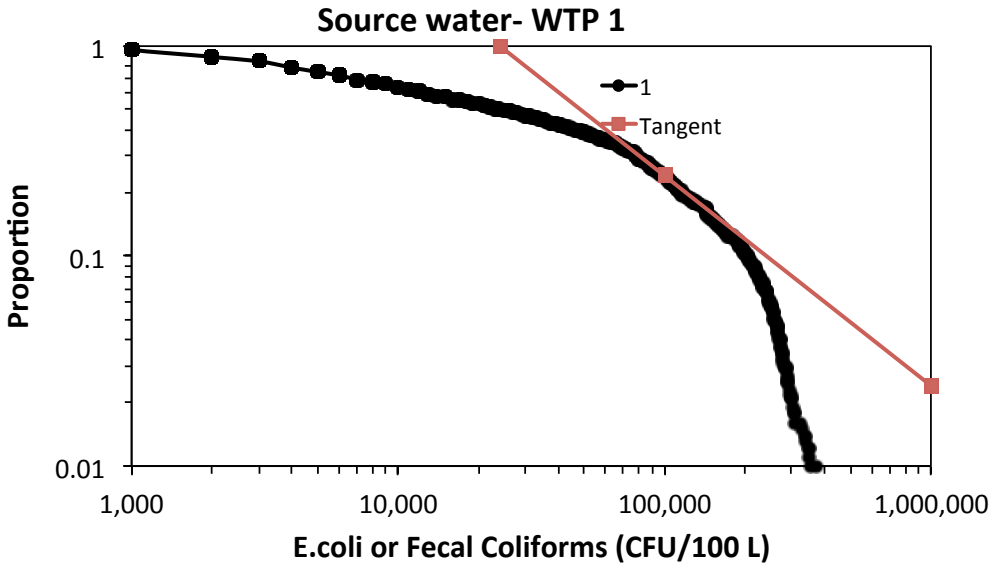

Figure A.4- 1 *E. coli* CCDF Graph for WTP1

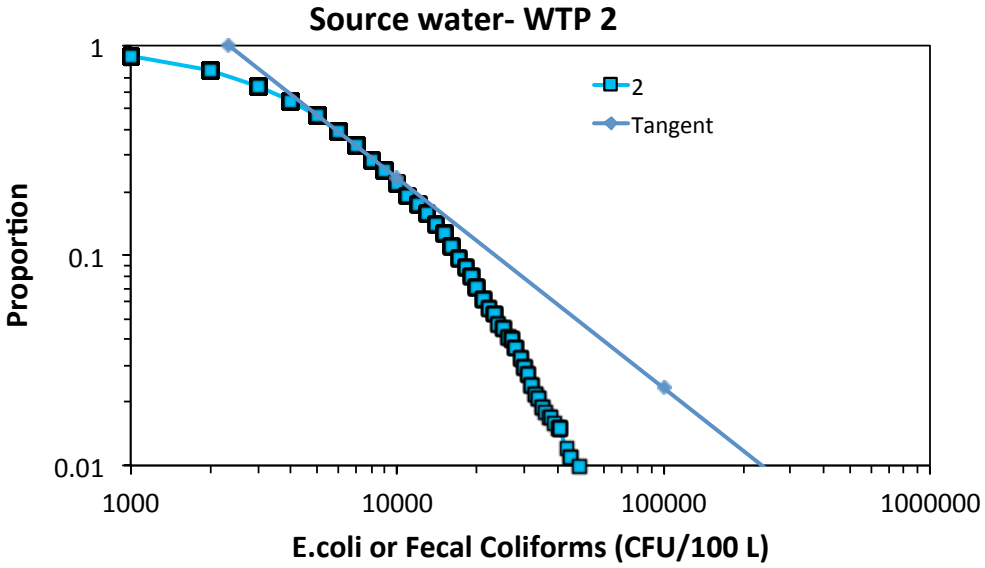

Figure A.4- 2 *E. coli* CCDF Graph for WTP 2

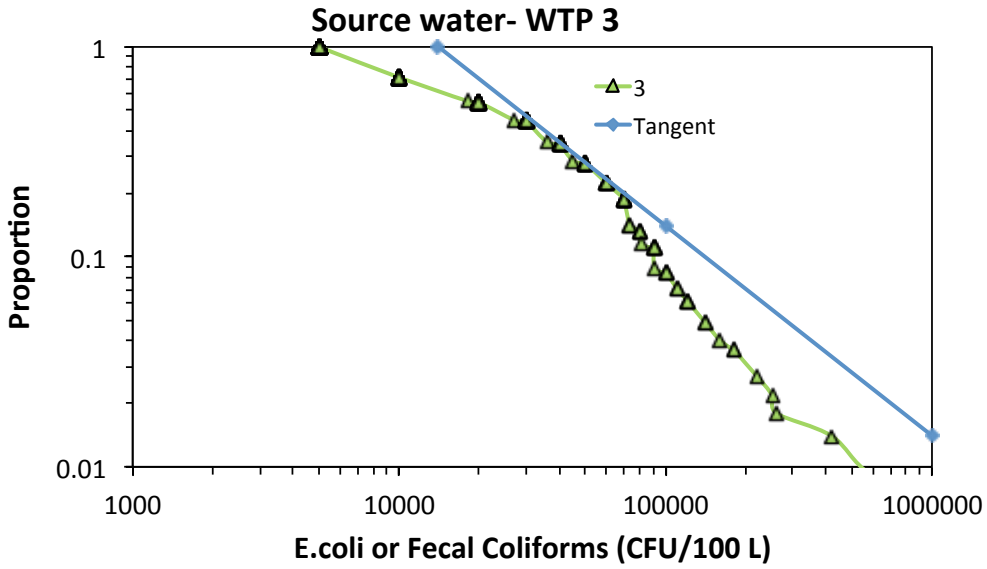

Figure A.4- 3 *E. coli* CCDF Graph for WTP 3

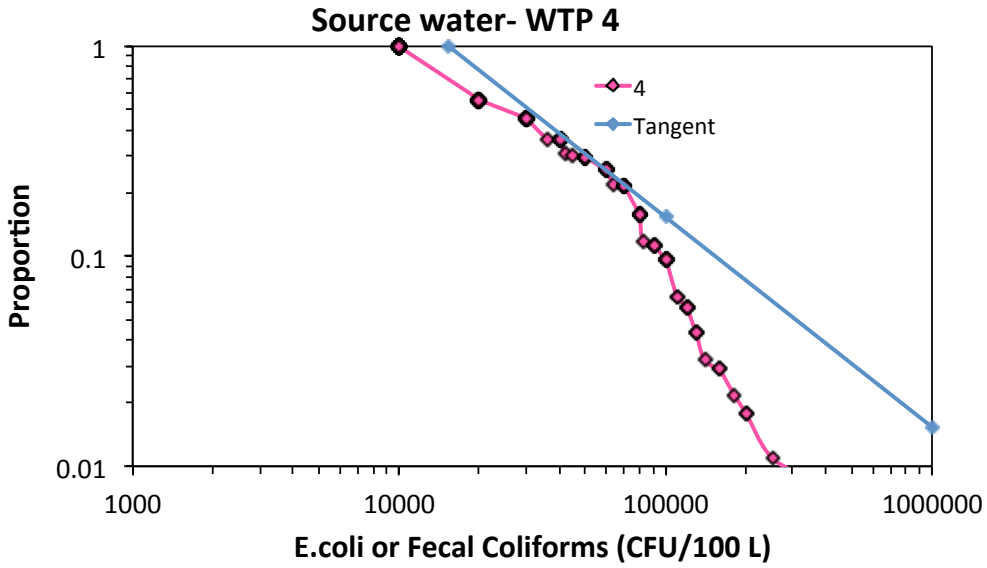

Figure A.4- 4 *E. coli* CCDF Graph for WTP 4

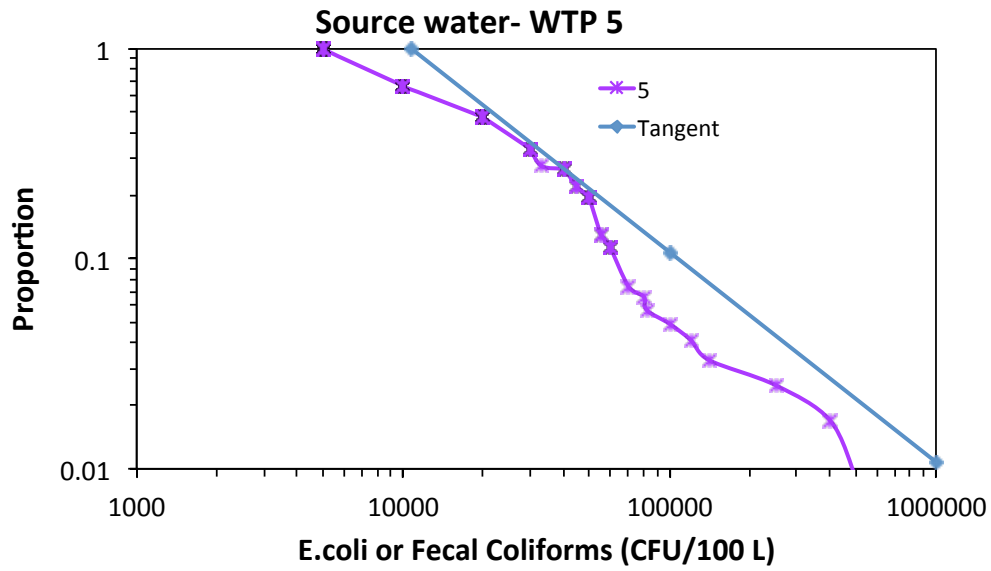

Figure A.4- 5 *E. coli* CCDF Graph for WTP 5

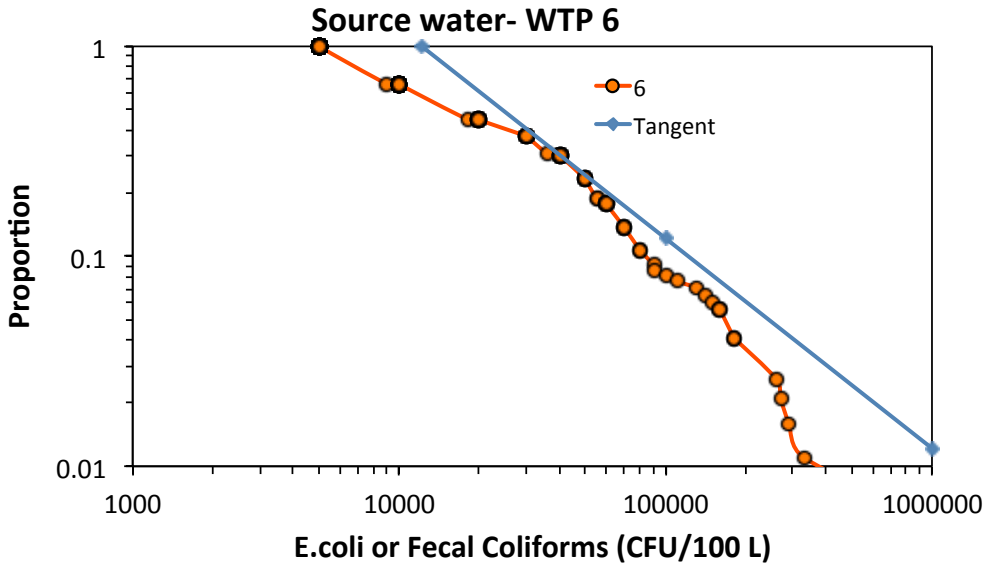

Figure A.4- 6 *E. coli* CCDF Graph for WTP 6

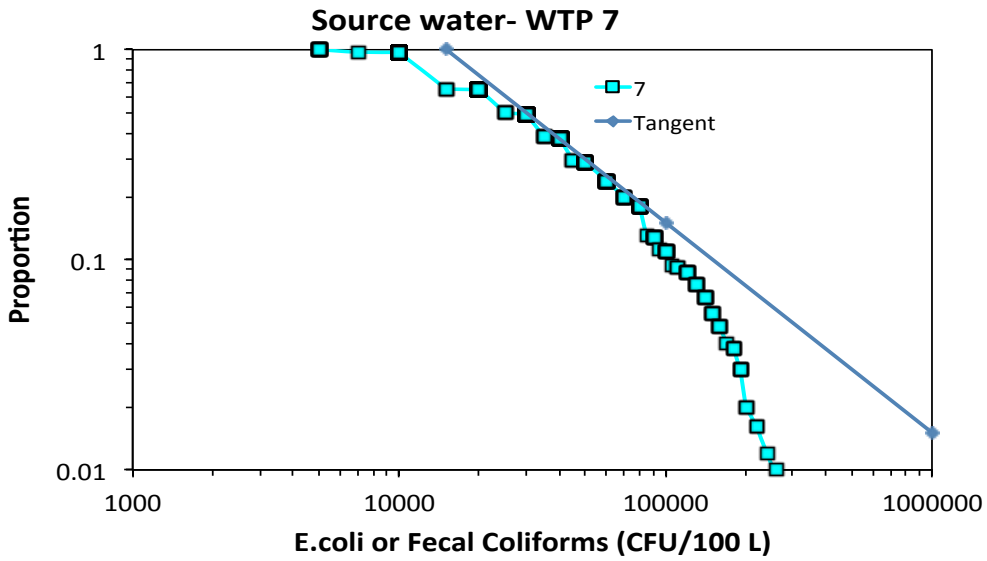

Figure A.4- 7 *E. coli* CCDF Graph for WTP 7

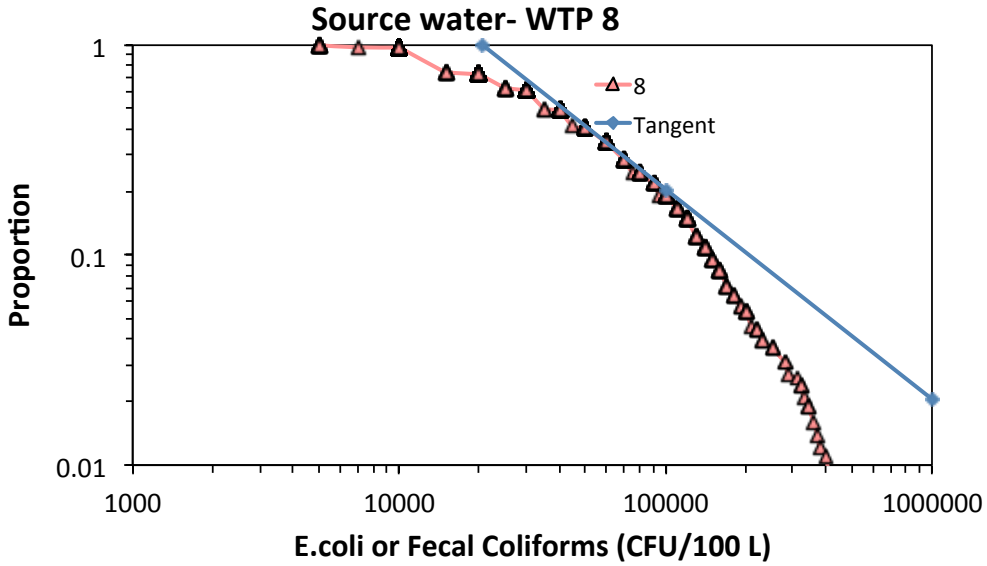

Figure A.4- 8 *E. coli* CCDF Graph for WTP 8

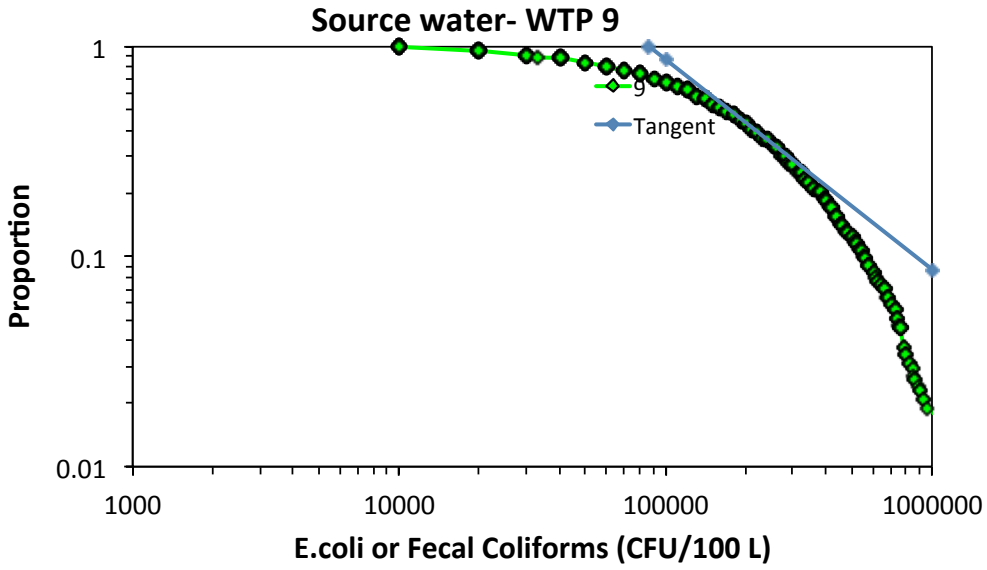

Figure A.4- 9 *E. coli* CCDF Graph for WTP 9

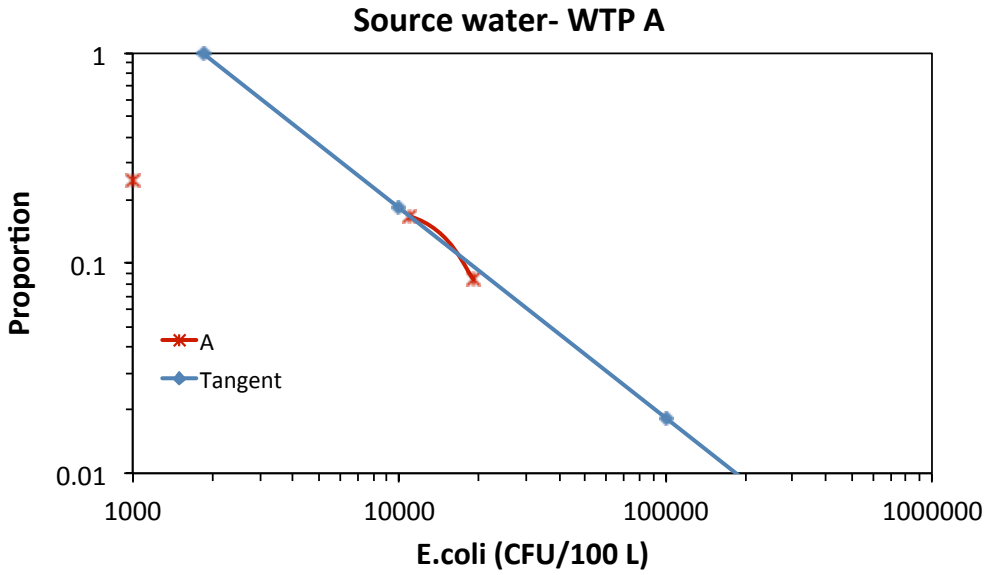

Figure A.4- 10 *E. coli* CCDF Graph for WTP A

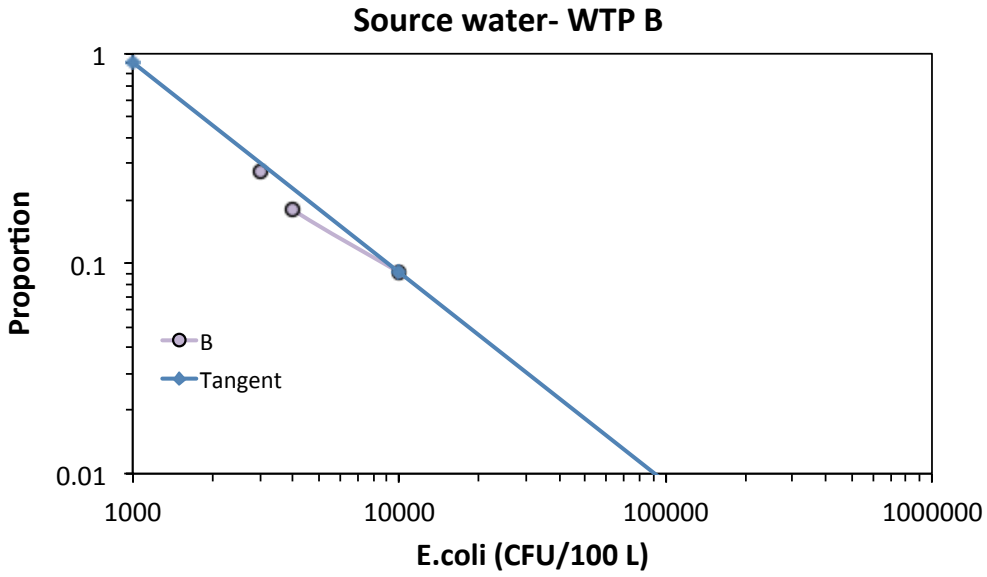

Figure A.4- 11 *E. coli* CCDF Graph for WTP B

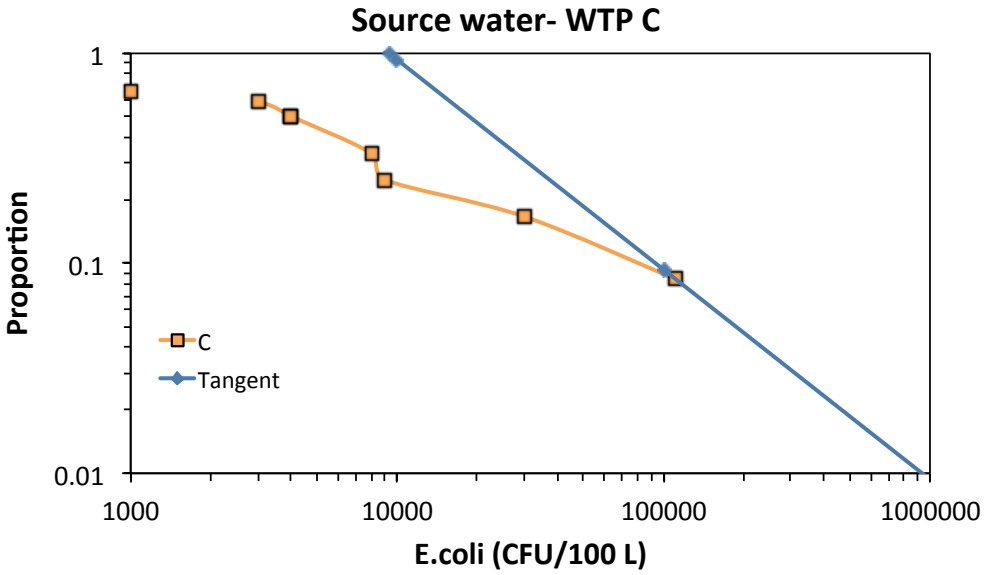

Figure A.4- 12 *E. coli* CCDF Graph for WTP C

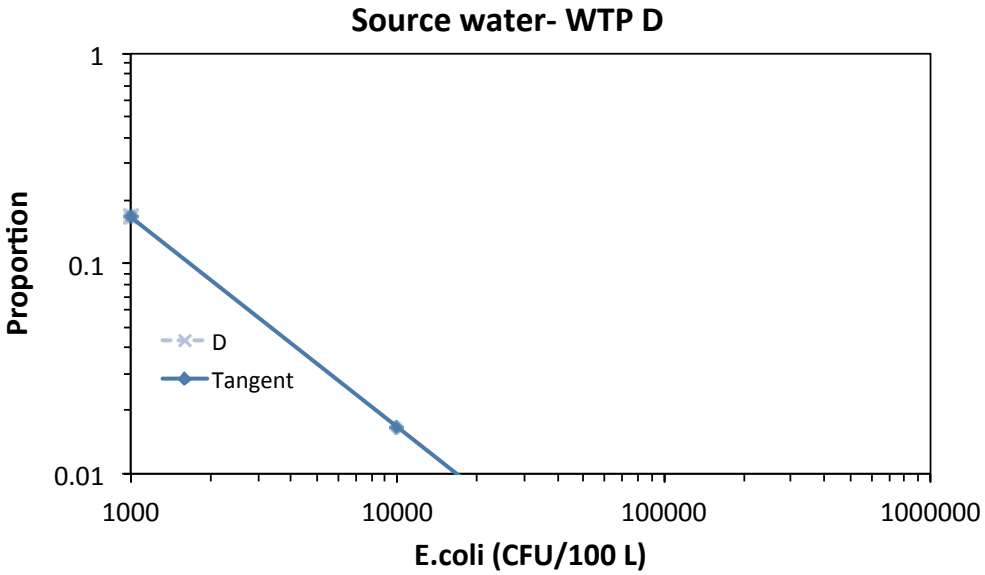

Figure A.4- 13 *E. coli* CCDF Graph for WTP D

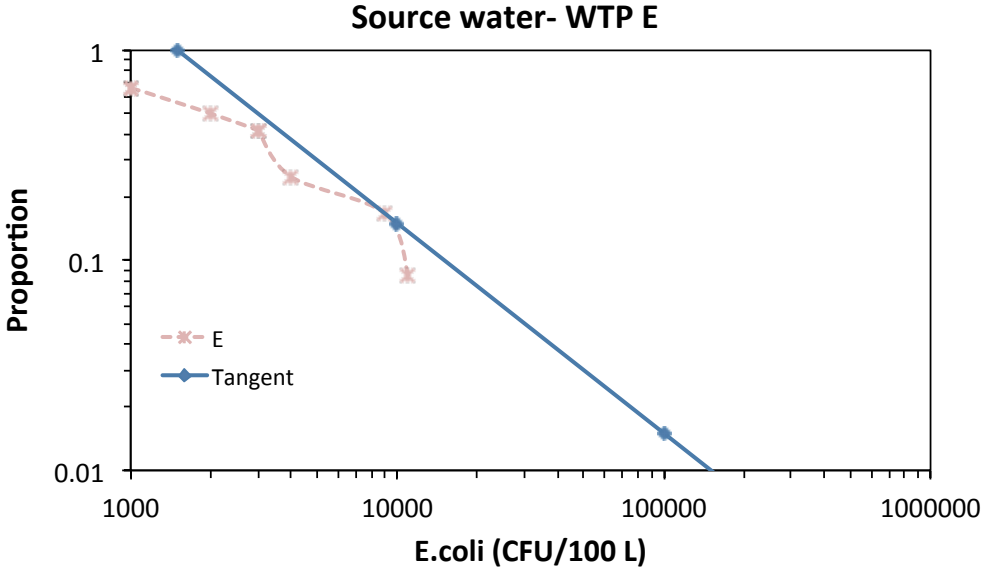

Figure A.4- 14 *E. coli* CCDF Graph for WTP E

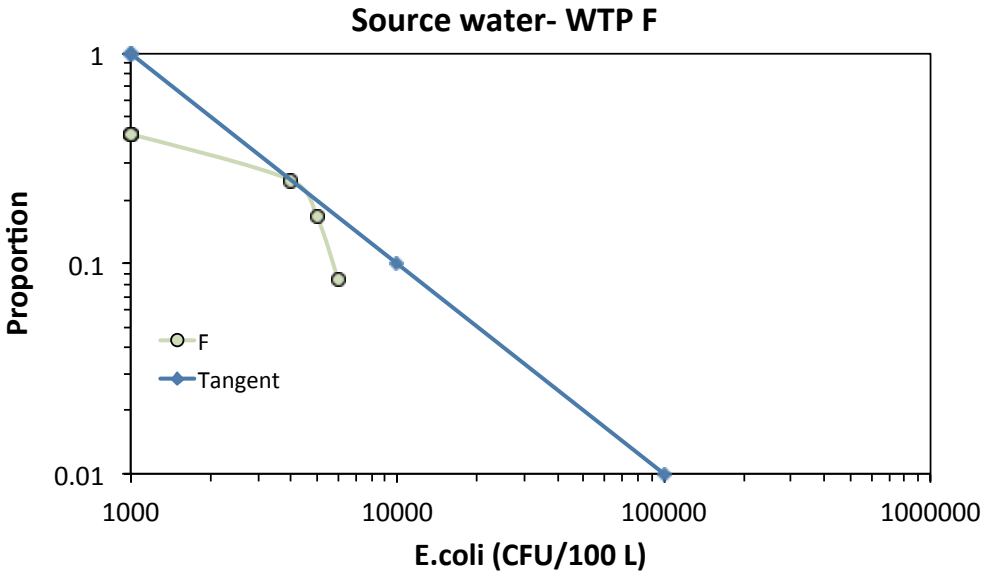

Figure A.4- 15 *E. coli* CCDF Graph for WTP F

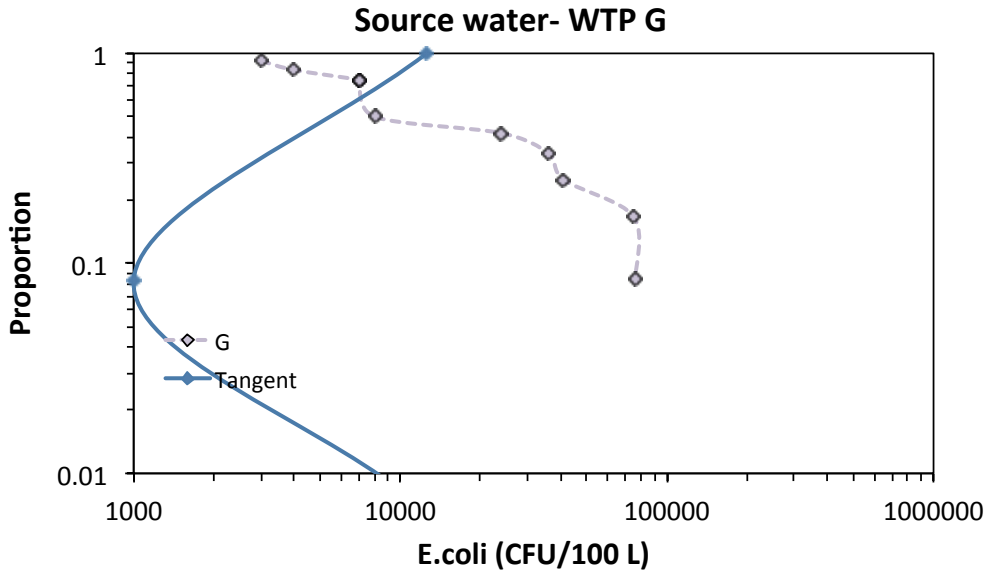

Figure A.4- 16 *E. coli* CCDF Graph for WTP G

Giardia CCDF Graphs

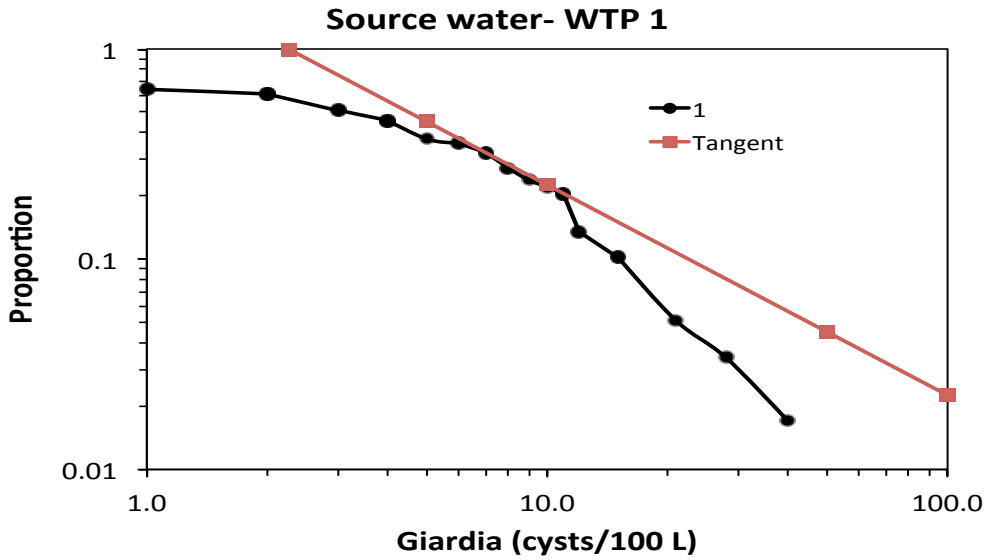

Figure A.4- 17 *Giardia* CCDF Graph for WTP 1

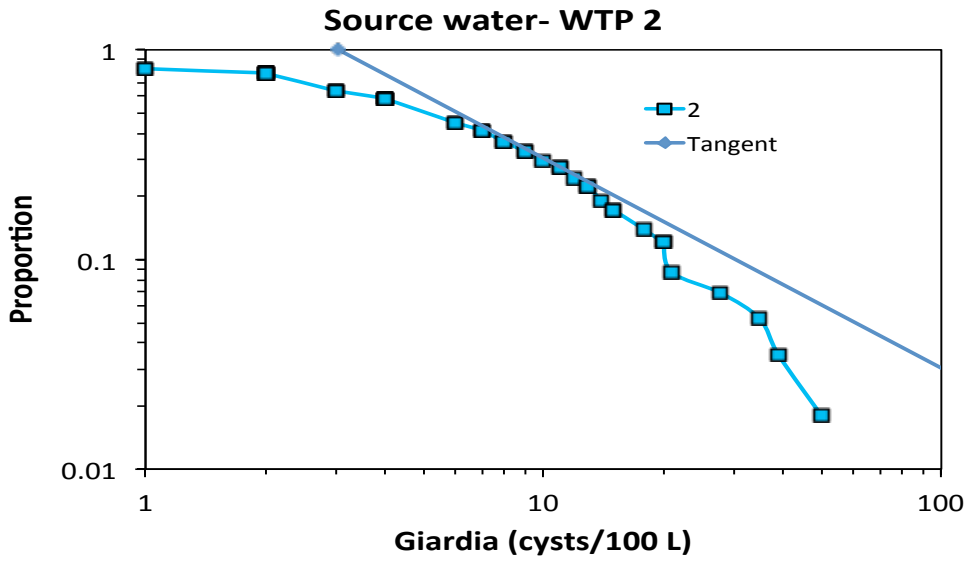

Figure A.4- 18 *Giardia* CCDF Graph for WTP 2

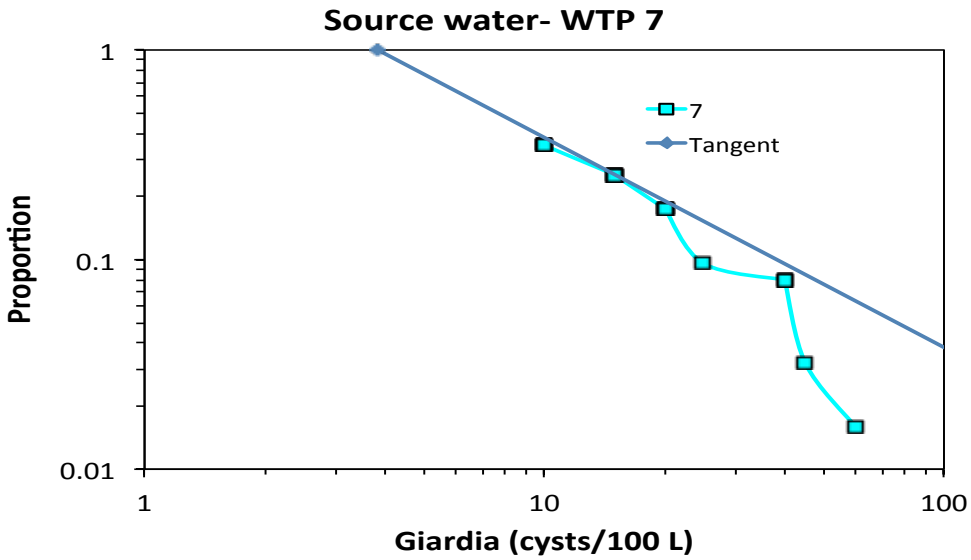

Figure A.4- 19 *Giardia* CCDF Graph for WTP 7

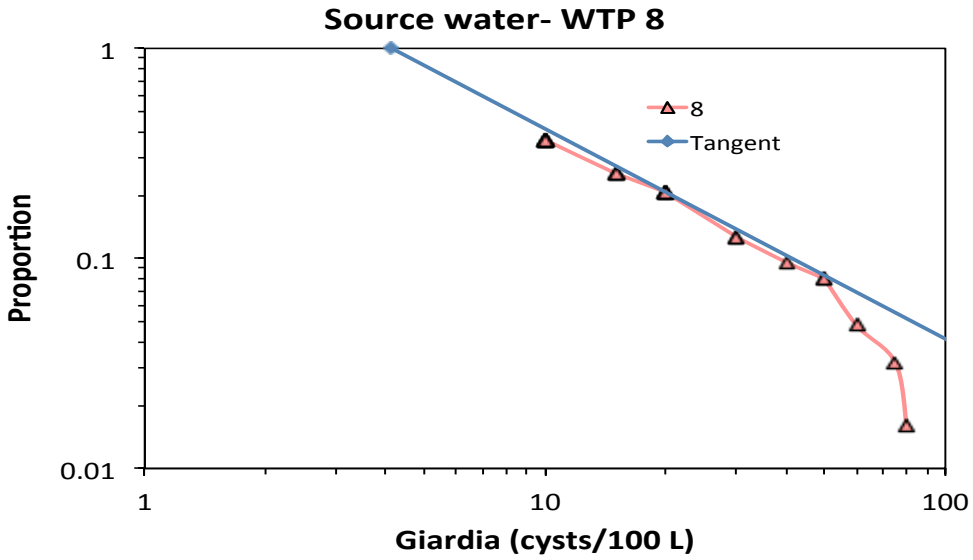

Figure A.4- 20 *Giardia* CCDF Graph for WTP 8

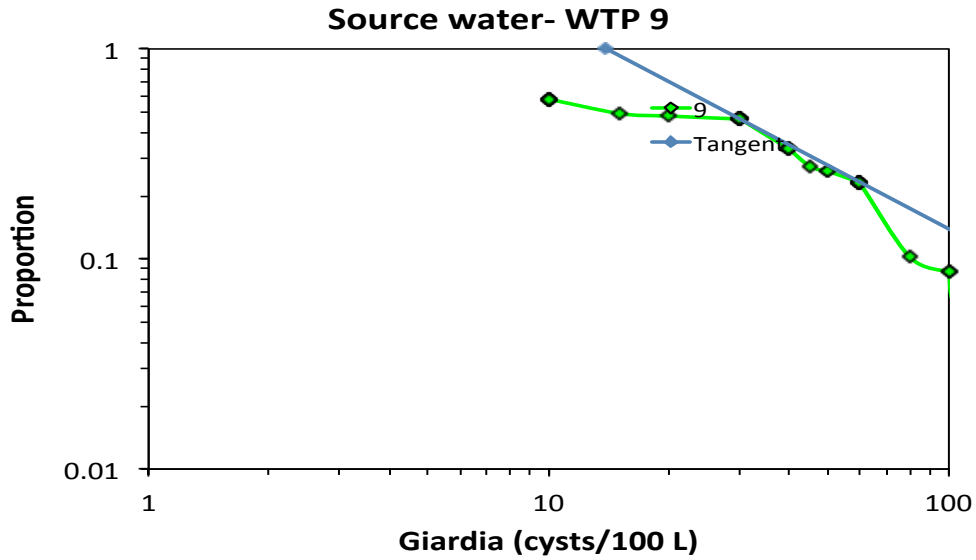

Figure A.4- 21 *Giardia* CCDF Graph for WTP 9

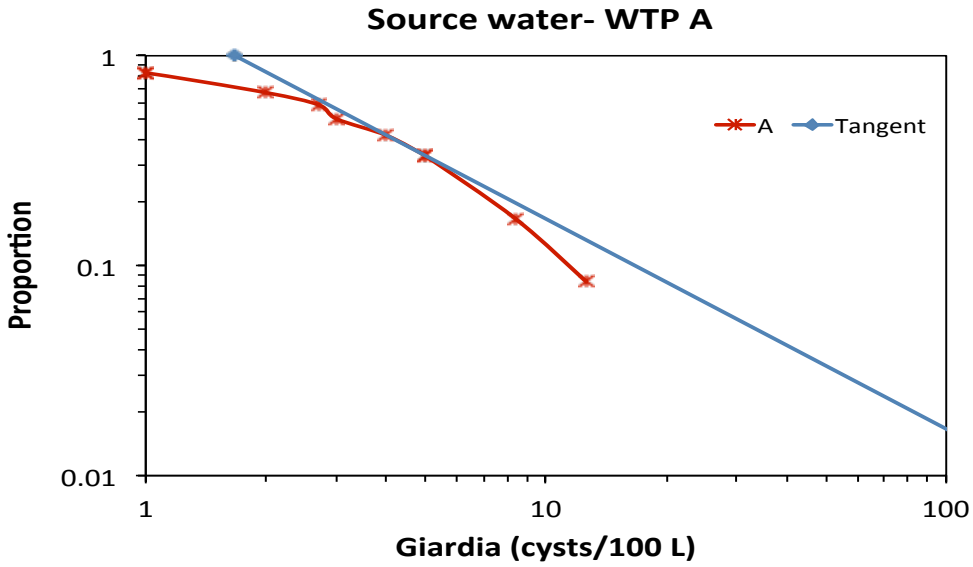

Figure A.4- 22 *Giardia* CCDF Graph for WTP A

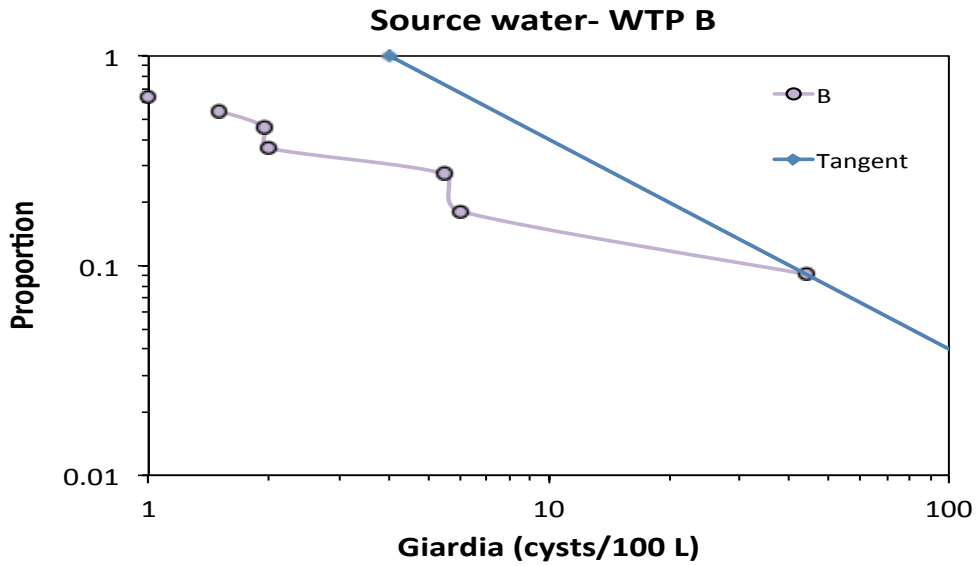

Figure A.4- 23 *Giardia* CCDF Graph for WTP B

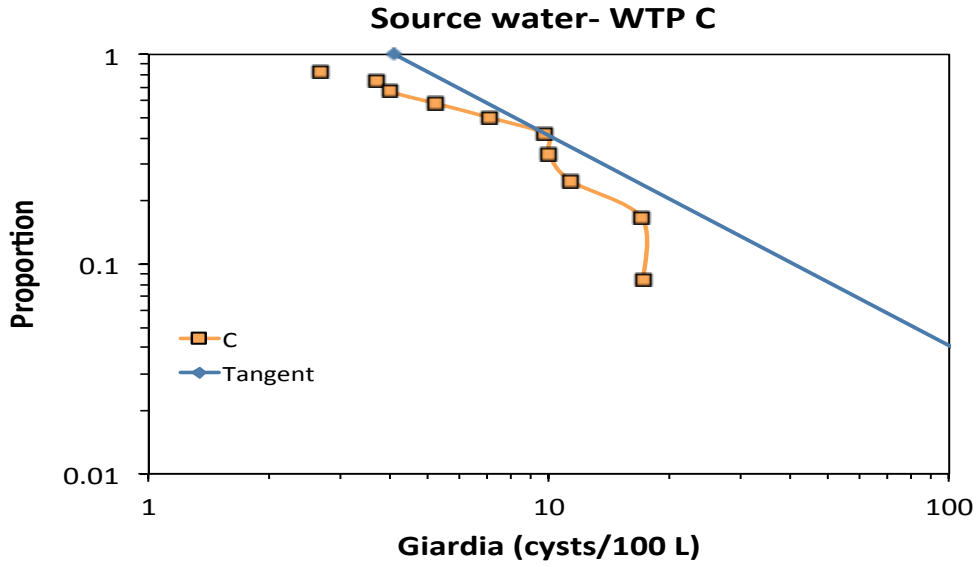

Figure A.4- 24 *Giardia* CCDF Graph for WTP C

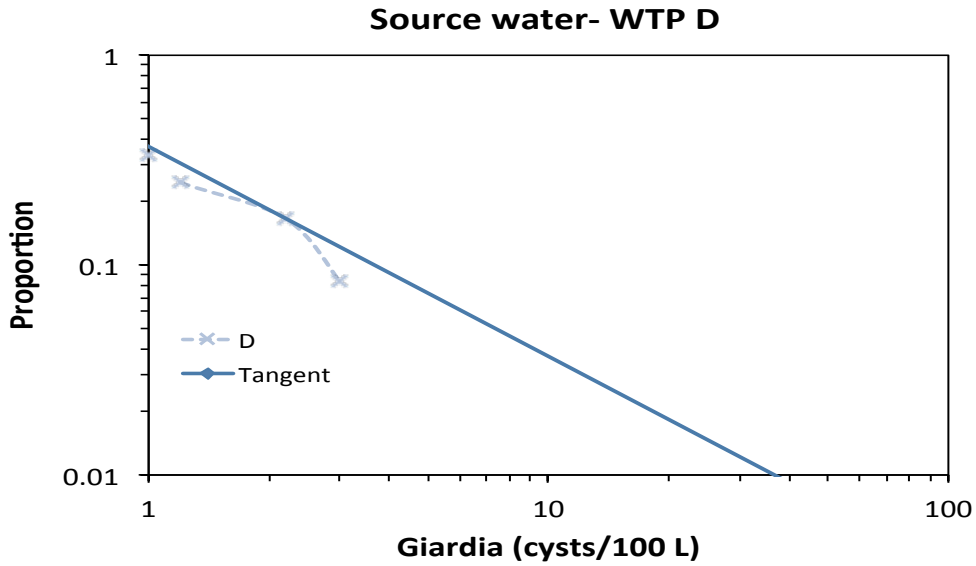

Figure A.4- 25 *Giardia* CCDF Graph for WTP D

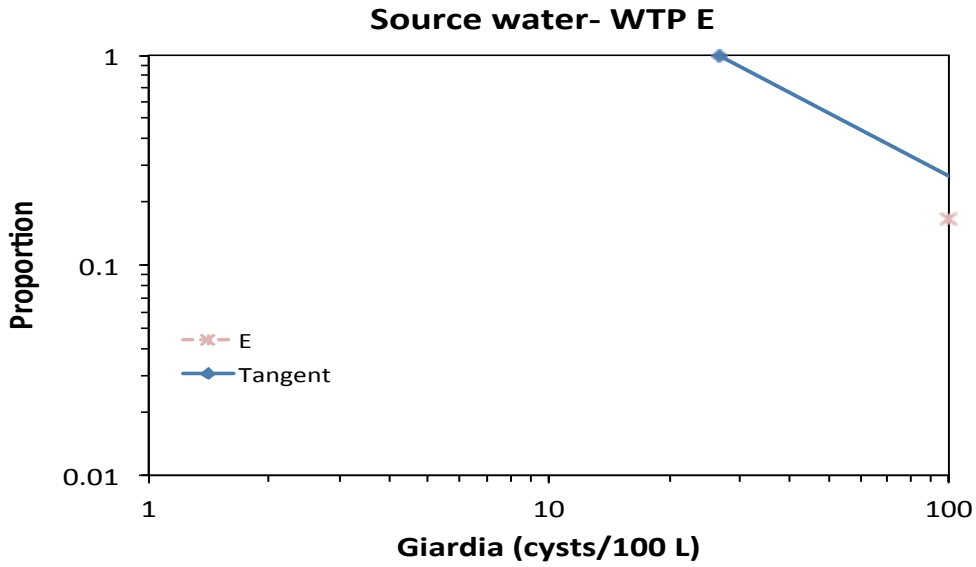

Figure A.4- 26 *Giardia* CCDF Graph for WTP E

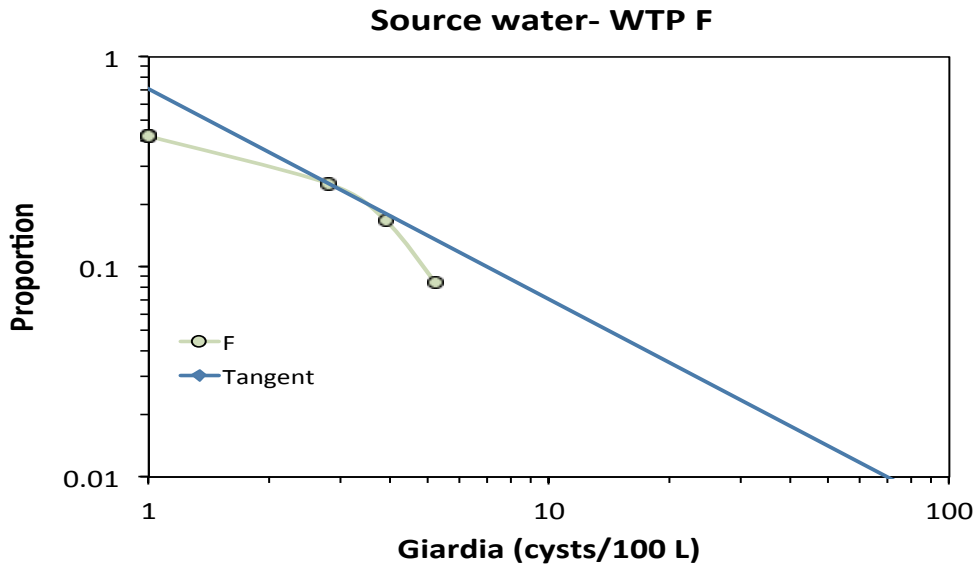

Figure A.4- 27 G*iardia* CCDF Graph for WTP F

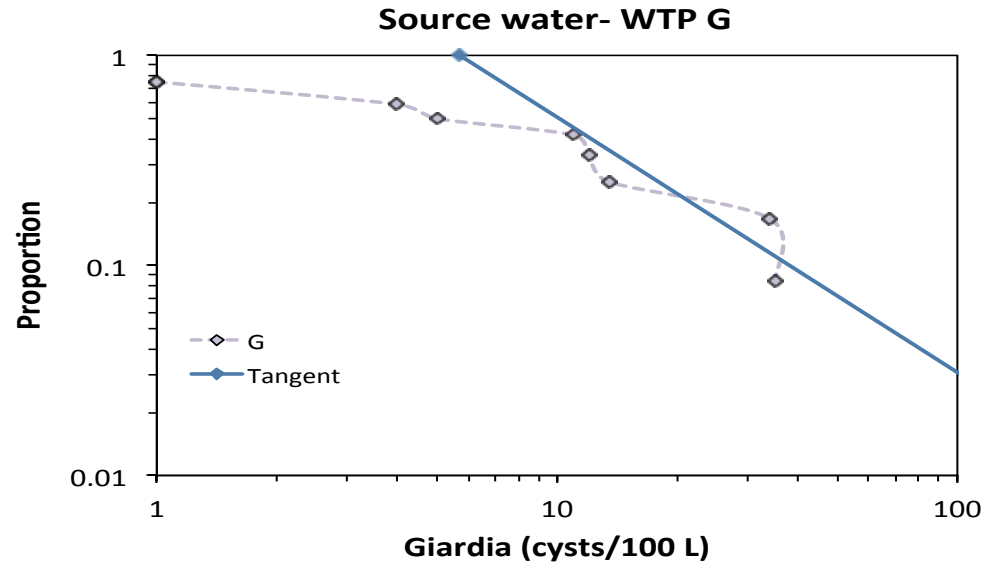

Figure A.4- 28 *Giardia* CCDF Graph for WTP G

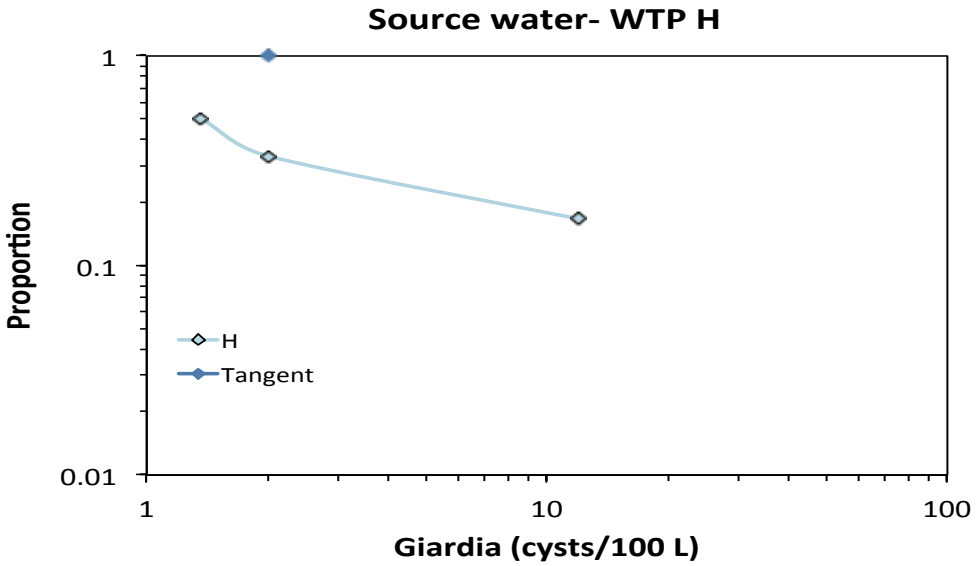

Figure A.4- 29 *Giardia* CCDF Graph for WTP H

***Cryptosporidium* CCDF Graphs**

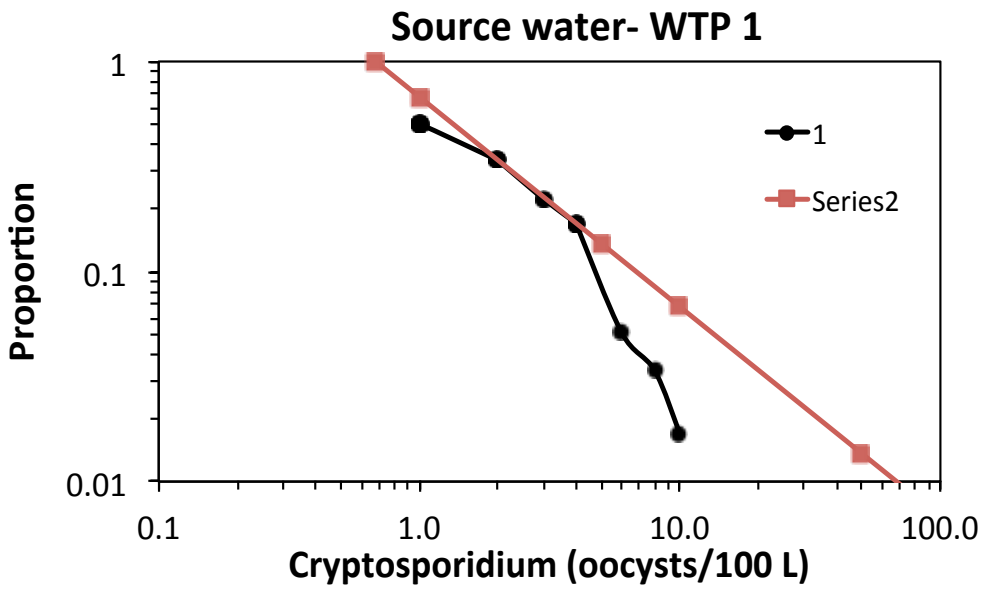

Figure A.4- 30 *Cryptosporidium* CCDF Graph for WTP1

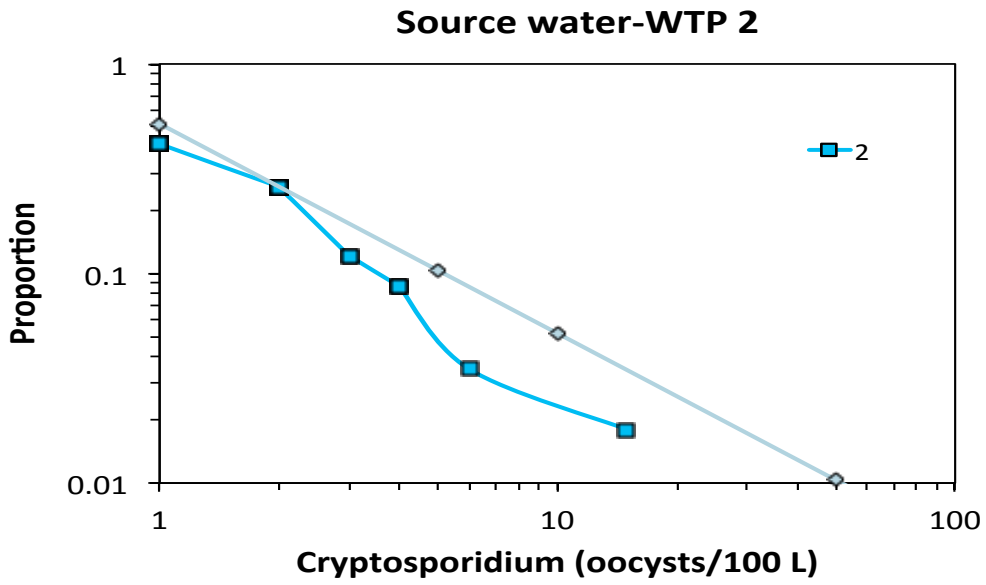

Figure A.4- 31 *Cryptosporidium* CCDF Graph for WTP 2

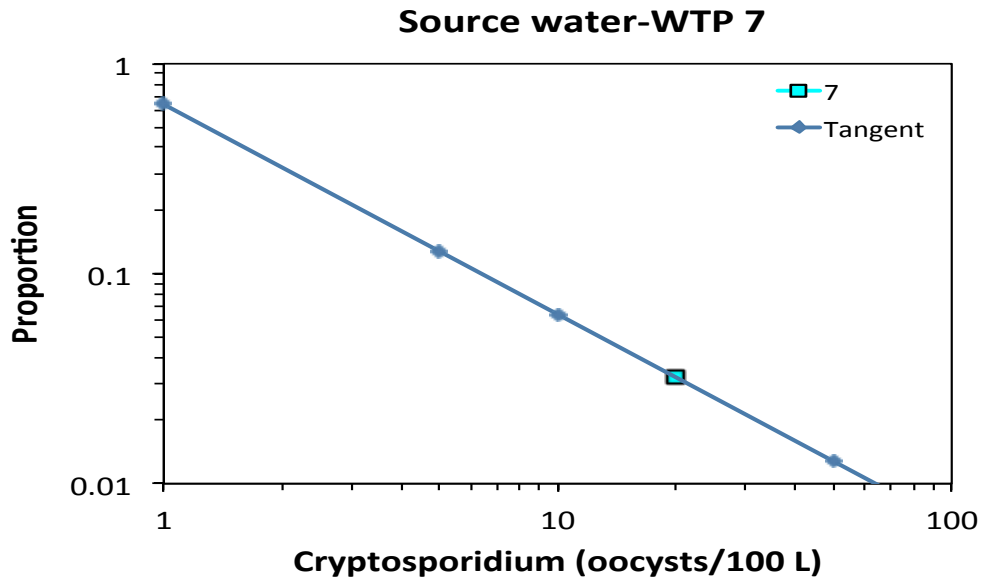

Figure A.4- 32 *Cryptosporidium* CCDF Graph for WTP 7

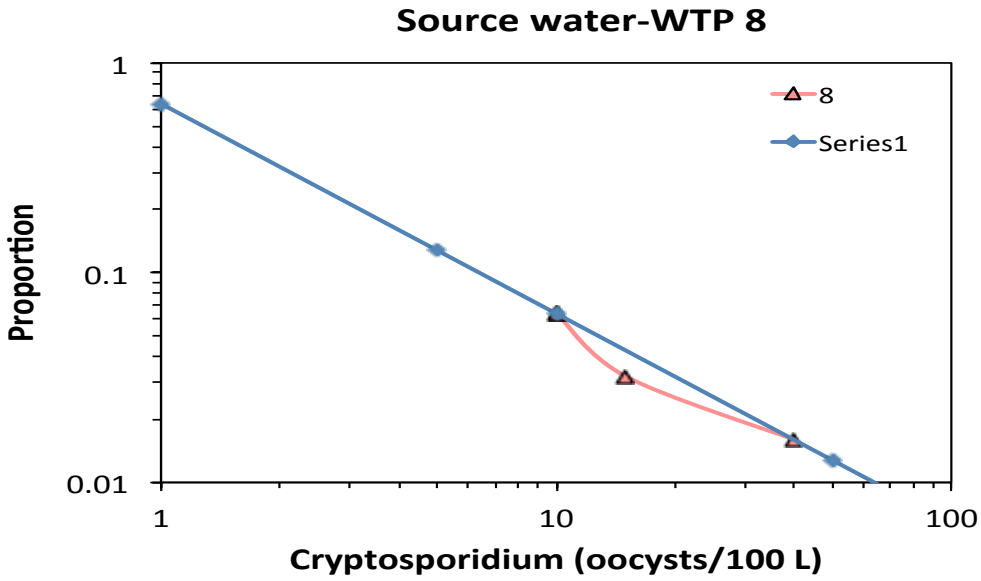

Figure A.4- 33 *Cryptosporidium* CCDF Graph for WTP 8

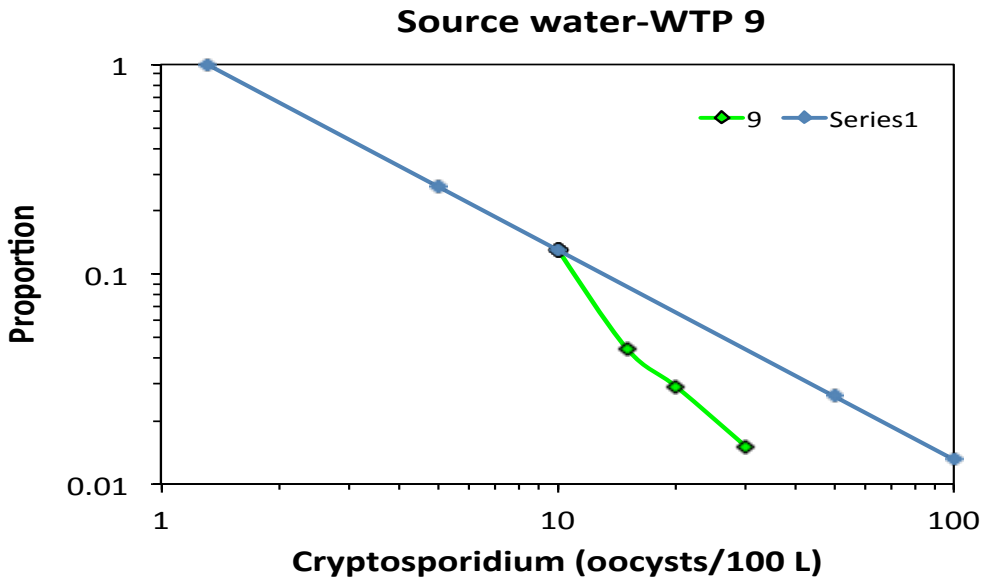

Figure A.4- 34 *Cryptosporidium* CCDF Graph for WTP 9

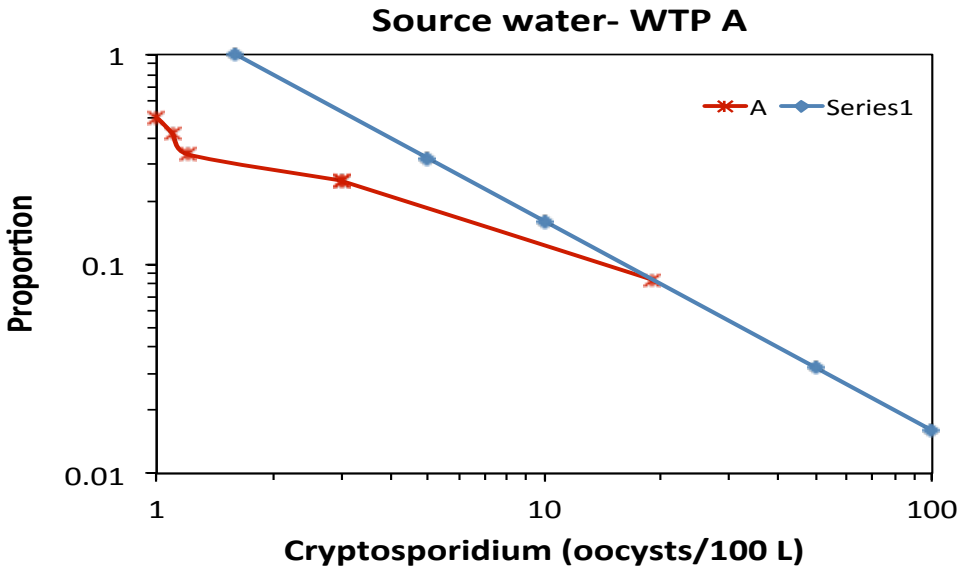

Figure A.4- 35 *Cryptosporidium* CCDF Graph for WTP A

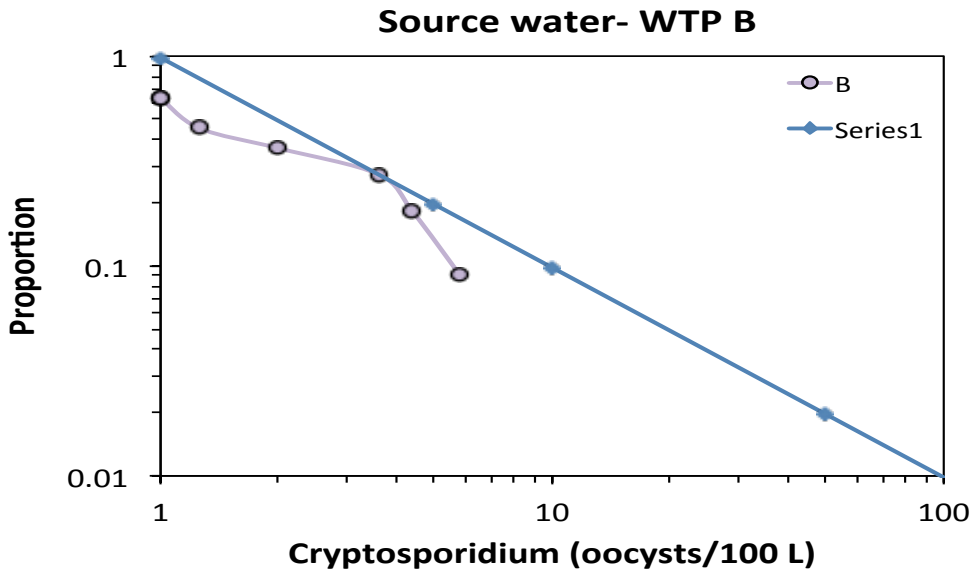

Figure A.4- 36 *Cryptosporidium* CCDF Graph for WTP B

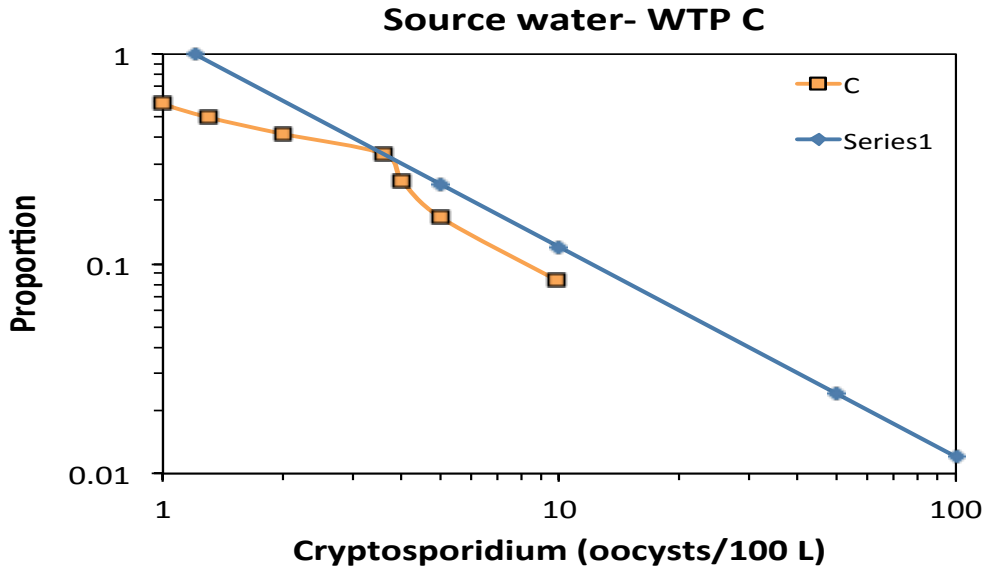

Figure A.4- 37 *Cryptosporidium* CCDF Graph for WTP C

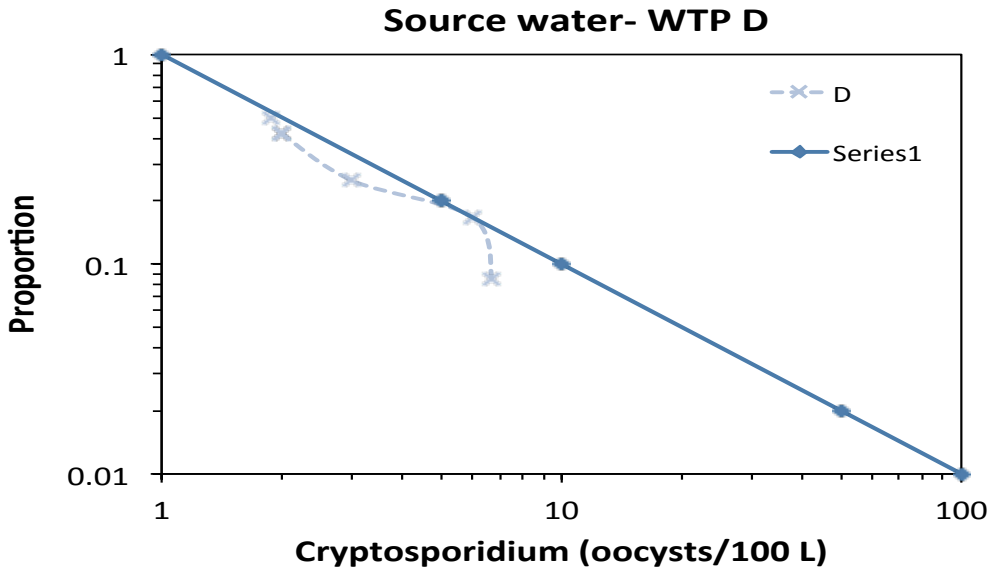

Figure A.4- 38 *Cryptosporidium* CCDF Graph for WTP D

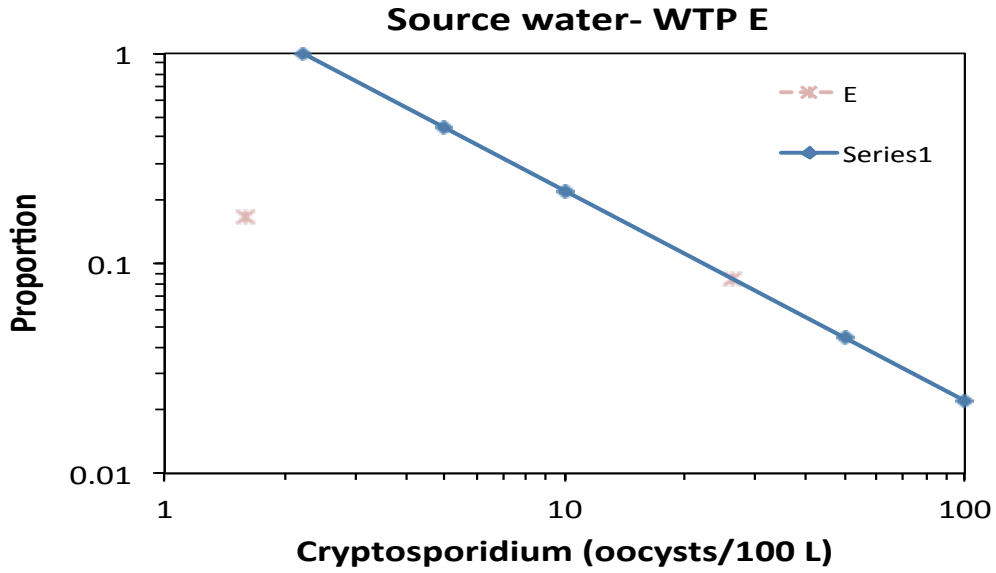

Figure A.4- 39 *Cryptosporidium* CCDF Graph for WTP E

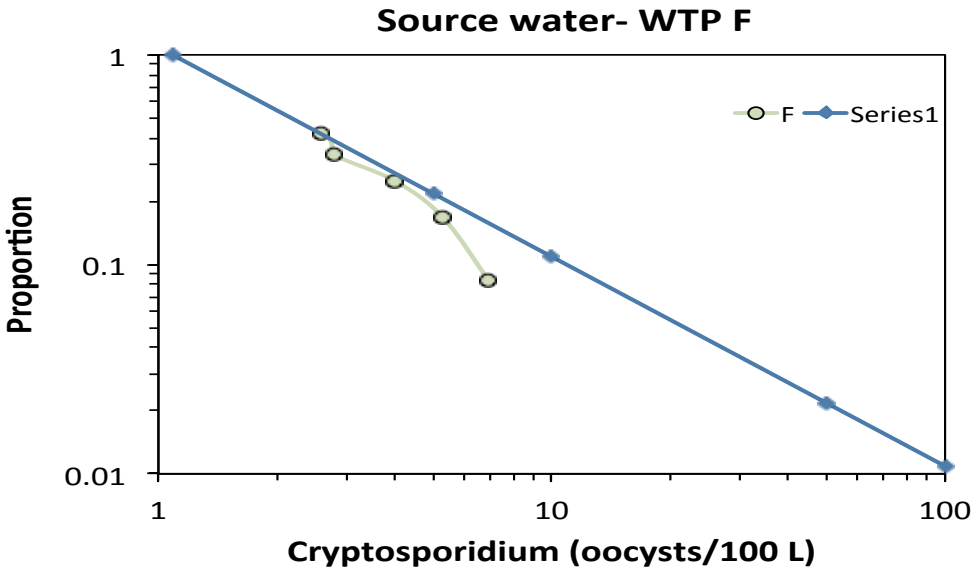

Figure A.4- 40 *Cryptosporidium* CCDF Graph for WTP F

APPENDIX 5- T_{10}/T Source of Data

Table A.5- 1 T_{10}/T Source of Data for Chlorination

WTP	**T_{10}/T**	**Source/Comment**
1	0.2	Assumption taken by WTP in 2000 (Mr. Robert Millette)
2	0.4	Assumption taken by WTP
3	0.3	Theoretical value taken by the WTP
4	0.6	Tracer study
5	0.35	Tracer study
6	0.55	Tracer study
7	0.6	Value taken from literature
8	0.6	Value taken from literature
9	0.6	Value taken from literature
A to D & G	N/A	No data available
E	0.6	As per EPA & Procedure of Disinfection of DW in Ontario
F	1 to 0.4	To be conservative, 0.4 is used in calculations
H	0.4 & 0.3	0.3 is used in calculations.

Table A.5- 2 T_{10}/T Source of data for Ozonation

APPENDIX 6-

Risk comparison of 2 scenarios:

A nnual versus Yearly source water distribution –WTP 2

WTP 2 risk estimates for *E. coli* assessed through two different scenarios.

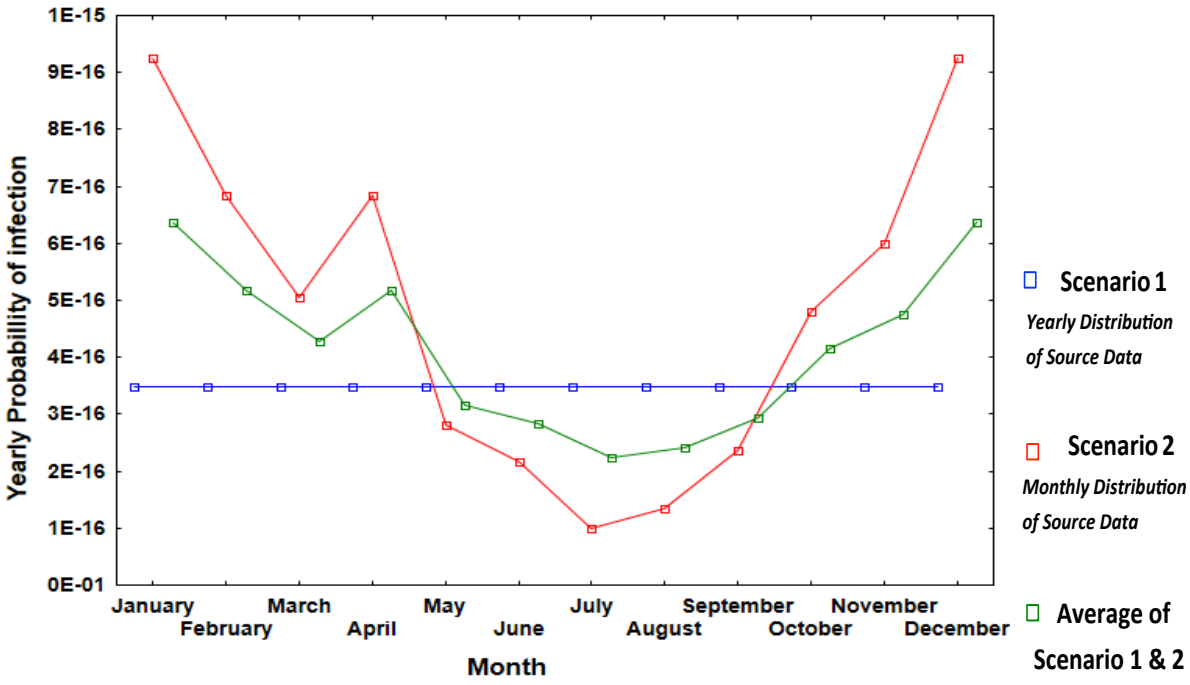

Figure A.6- 1 Risk estimate of two scenarios – WTP 2

APPENDIX 7- Disinfection Data

Table A.7- 1 Chlorination data for WTP 1 and 2

Month	WTP 1			WTP 2		
	Chlorination (Low pH prior to softening)			Chlorination (Low pH prior to softening)		
	Residual (mg/L)	pH	Temp. (^{0}C)	Residual (mg/L)	pH	Temp. (^{0}C)
January	1.1	7.84	0	0.98	7.69	0.04
February	1.01	7.84	0	0.99	7.75	0.04
March	1.08	7.79	4	0.99	7.73	0.61
April	1.19	7.84	9	1	7.64	4.92
May	1.3	7.74	14	0.99	7.56	11.1
June	1.32	7.77	20	0.99	7.56	15.89
July	1.25	7.9	22	0.97	7.54	19.98
August	1.25	7.91	24	1.03	7.56	22.96
September	1.24	7.95	23	1.04	7.71	19.82
October	1.18	7.95	15	1.04	7.72	12.6
November	1.09	7.88	10	1.01	7.72	8.81
December	1.05	7.88	4	1.03	7.76	3.02

Table A.7- 2 Chlorination data for WTP 3 and 6

Month	WTP 3			WTP 6		
	Chlorination (Low pH prior to softening)			Chlorination (Low pH prior to softening)		
	Residual (mg/L)	pH	Temp. (^{0}C)	Residual (mg/L)	pH	Temp. (^{0}C)
January	0.9	7.81	0.82	1.1	7.15	0.64
February	0.91	7.82	0.87	1.39	7.19	0.65
March	0.91	7.91	1.24	1.38	7.16	0.76
April	0.89	7.87	6.23	1.4	6.96	8.83
May	0.9	7.63	13.43	1.39	6.98	12.75
June	0.94	7.38	17.89	1.42	7.11	18.23
July	0.92	7.37	21.58	1.51	7.16	21.84
August	1	7.42	23.54	1.49	7.17	23.18
September	1	7.37	19.38	1.56	7.15	19.33
October	0.96	7.43	11.39	1.4	7.2	11.33
November	0.95	7.58	7.21	1.41	7.13	6.89
December	1	7.66	2.46	1.35	7.09	1.82

Table A.7- 3 Chlorination data for WTP 8

Month	WTP 8		
	Chlorination (Low pH prior to softening)		
	Residual (mg/L)	pH	Temp. (^{0}C)
January	0.88	7.41	0.62
February	0.88	7.4	0.4
March	0.97	7.46	0.52
April	1.02	7.48	5.53
May	1.13	7.5	12.95
June	1.31	7.48	18.72
July	1.43	7.52	21.99
August	1.46	7.67	23.54
September	1.36	7.67	19.13
October	1.06	7.68	10.85
November	0.95	7.72	6.77
December	0.86	7.77	1.75

Table A.7- 4 Chlorination data for WTP C and D

Date	WTP C			WTP D		
	Chlorination (Low pH prior to softening)			Chlorination (Low pH prior to softening)		
	Residual (mg/L)	pH	Temp. (^{0}C)	Residual (mg/L)	pH	Temp. (^{0}C)
09-Aug	1.28	6.52	25	1.03	7.7	16.78
09-Oct	1.32	6.36	8.1	0.95	8.22	13.22
09-Dec	1.65	6.48	16	1.05	8.03	6.56
10-Feb	1.38	6.53	0.1	1.21	7.49	14.05
10-Apr	1.24	6.72	12.3	1.12	7.88	9.01
10-Jul	1.57	6.5	24.5	1.39	7.51	16.1
10-Oct	1.51	7.46	14.3	1.2	7.79	18.2
10-Dec	1.28	7.6	0.8	1.1	7.92	7.7
11-Feb	1.21	6.59	0	1.7	8.05	6.43
11-May	1.21	6.78	13.9	1.3	8.1	7.3
11-Jun	1.36	6.76	20	1.35	7.41	13.8

Table A.7- 5 Chlorination and UV data for WTP E and H

WTP E					WTP H			
Date	Chlorination (Low pH prior to softening)			UV	Date	Chlorination (Low pH prior to softening)		
	Residual (mg/L)	pH	Temp. (^{0}C)	Dose (mJ/cm2)		Residual (mg/L)	pH	Temp. (^{0}C)
09-Oct	1.2	8.19	14.08	0	10-Dec	2.62	8.07	4.6
09-Nov	1.25	8.17	9.54	0	11-Jan	2.05	7.77	2.7
10-Apr	1.36	8.76	7.58	158.98	11-Mar	2.33	8.23	3.2
10-Jun	1.37	7.94	14.02	138.4	11-May	2.2	8.22	5.3
10-Oct	1.31	8.36	13.65	139.08	11-Jul	2.25	8.37	8.9
10-Nov	1.3	8.28	10.65	140.16				
11-Jan	1.28	7.92	0	155.87				
11-Mar	1.31	8.13	1.09	143.09				
11-Apr	1.22	8.2	4.3	138.4				
11-Aug	1.31	8.59	23.33	138.4				

Table A.7- 6 Chlorination data for WTP F and G

WTP F				WTP G			
Date	Chlorination (Low pH prior to softening)			Date	Chlorination (Low pH prior to softening)		
	Residual (mg/L)	pH	Temp. (^{0}C)		Residual (mg/L)	pH	Temp. (^{0}C)
09-Aug	1.17	7.1	21.6	09-Sep	1.05	5.92	20.8
09-Oct	1.21	7.1	12	09-Nov	1.03	6	7.8
09-Nov	1.22	7.1	9.7	10-Jan	0.93	5.97	0.2
10-Apr	1.16	7.2	9.5	10-Mar	1.48	6.02	3.2
10-Jun	1.3	7.3	15.51	10-May	1.02	5.99	10.6
10-Oct	1.35	7.15	13.2	10-Aug	1.05	5.92	21.6
10-Nov	1.21	7.2	8.8	10-Oct	1.01	6.08	10
10-Nov	1.16	7.14	8.33	11-Jan	0.94	6.08	0.4
11-Mar	0.46	7.32	1.68	11-Mar	1.07	5.97	0.8
11-Apr	0.51	7.24	3.52	11-May	0.79	6.03	9.1
11-Aug	0.35	7.14	23.62	11-Jul	1.11	5.86	23.5

Table A.7- 7 Chlorination data for WTP 4 and 5

WTP	Month	Chlorination (Low pH prior to softening)			Chlorination (High pH prior to softening)		
		Residual (mg/L)	pH	Temp. (^{0}C)	Residual (mg/L)	pH	Temp. (^{0}C)
4	January	0.99	7.33	2.45	1.02	7.35	2.45
	February	0.98	7.22	2.54	0.95	7.27	2.54
	March	1.01	7.39	2.89	0.98	7.39	2.89
	April	1	7.22	6.33	0.95	7.04	6.33
	May	0.98	7.15	13.09	0.96	7.07	13.09
	June	0.99	7.19	17.78	0.96	7.33	17.78
	July	0.97	7.24	21.52	0.95	7.25	21.52
	August	1.01	7.18	23.8	0.99	7.15	23.8
	September	0.98	7.26	20.04	0.99	7.27	20.04
	October	0.97	7.35	12.25	1	7.4	12.25
	November	0.99	7.11	7.84	0.95	7.1	7.84
	December	1	7.36	2.57	0.98	7.23	2.57
5	January	1.03	6.49	1.43	0.87	7.14	1.43
	February	1.07	6.51	1.35	0.9	7.33	1.35
	March	1.04	6.45	1.66	0.91	7.26	1.66
	April	1.04	6.56	6.83	0.96	7.23	6.83
	May	1.06	6.48	13.9	0.8	7.29	13.9
	June	1.03	6.62	18.27	0.84	7.17	18.27
	July	1.12	6.73	21.55	0.94	7.39	21.55
	August	1.33	6.61	23.9	1.07	7.27	23.9
	September	1.2	6.59	20.31	1.01	7.32	20.31
	October	1.12	6.46	13.94	0.88	7.25	13.94
	November	1.1	6.46	7.51	1.05	7.58	7.51
	December	1.11	6.56	2.7	0.92	7.13	2.7

Table A.7- 8 Chlorination data for WTP 7 and 9

WTP	Month	Chlorination (Low pH prior to softening)			Chlorination (High pH prior to softening)		
		Residual (mg/L)	pH	Temp. (^{0}C)	Residual (mg/L)	pH	Temp. (^{0}C)
7	January	1.03	6.27	0.89	1.03	7.11	0.89
	February	1.2	6.25	0.9	1.2	6.98	0.9
	March	1.15	6.37	0.95	1.15	7	0.95
	April	0.97	6.18	5.57	0.97	7.49	5.57
	May	1.09	5.91	12.68	1.09	7.58	12.68
	June	1.3	5.68	17.94	1.3	7.43	17.94
	July	1.43	5.74	21.58	1.43	7.36	21.58
	August	1.41	5.9	23.07	1.41	7.58	23.07
	September	1.36	6.21	19.21	1.36	7.73	19.21
	October	1.06	6.22	11.69	1.06	7.67	11.69
	November	0.87	6.39	7.37	0.87	7.64	7.37
	December	0.79	6.4	2.44	0.79	7.59	2.44
9	January	0.79	6.02	0.4	0.79	7.33	0.4
	February	0.81	6.06	0.4	0.81	7.33	0.4
	March	0.82	6.1	0.52	0.82	7.35	0.52
	April	0.89	5.98	5.53	0.89	7.49	5.53
	May	1.12	5.65	12.95	1.12	7.51	12.95
	June	1.3	5.76	18.72	1.3	7.54	18.72
	July	1.41	6.21	21.99	1.41	7.46	21.99
	August	1.42	6.62	23.55	1.42	7.55	23.55
	September	1.31	6.33	19.13	1.31	7.58	19.13
	October	1.06	5.73	10.85	0.8	7.48	10.85
	November	0.9	5.81	6.77	0.79	7.34	6.77
	December	0.81	6.07	1.65	0.79	7.16	1.65

Table A.7- 9 Ozonation data for WTP 2

Month	Temp. (^{0}C)	WTP 2											
		Tank 1		Tank 2		Tank 3		Tank 4		Tank 5		Tank 6	
		Residual (mg/L)	T_{50} (min.)	Residual (mg/L)	T_{50} (min.)	Residual (mg/L)	T_{50} (min.)	Residual (mg/L)	T_{50} (min.)	Residual (mg/L)	T_{50} (min.)	Residual (mg/L)	T_{50} (min.)
January	0.04	0.59	4.2	0.6	4.2	0.59	4.2	0.6	4.2	0.61	4.2	0.6	4.2
February	0.04	0.64	4.28	0.66	4.28	0.66	4.28	0.66	4.28	0.67	4.28	0.66	4.28
March	0.61	0.72	4.31	0.73	4.31	0.73	4.31	0.73	4.31	0.75	4.31	0.73	4.31
April	4.92	0.47	4.27	0.51	4.27	0.51	4.27	0.5	4.27	0.54	4.27	0.53	4.27
May	11.1	0.26	4.41	0.31	4.41	0.29	4.41	0.31	4.41	0.3	4.41	0.38	4.41
June	15.89	0.27	4.16	0.29	4.16	0.32	4.16	0.33	4.16	0.3	4.16	0.41	4.16
July	19.98	0.22	4.22	0.23	4.22	0.23	4.22	0.24	4.22	0.21	4.22	0.23	4.22
August	22.96	0.17	4.22	0.18	4.22	0.17	4.22	0.19	4.22	0.17	4.22	0.17	4.22
September	19.82	0.22	4.27	0.23	4.27	0.22	4.27	0.24	4.27	0.22	4.27	0.2	4.27
October	12.6	0.36	4.41	0.38	4.41	0.39	4.41	0.41	4.41	0.38	4.41	0.35	4.41
November	8.81	0.46	4.42	0.48	4.42	0.48	4.42	0.5	4.42	0.48	4.42	0.46	4.42
December	3.02	0.73	4.49	0.73	4.49	0.72	4.49	0.73	4.49	0.72	4.49	0.71	4.49

Table A.7- 10 Ozonation data for WTP 6

Month	WTP 6										
	Temp. (^{0}C)	Tank 1		Tank 2		Tank 3		Tank 4		Tank 5	
		Residual (mg/L)	T_{50} (min.)	Residual (mg/L)	T_{50} (min.)	Residual (mg/L)	T_{50} (min.)	Residual (mg/L)	T_{50} (min.)	Residual (mg/L)	T_{50} (min.)
January	0.64	0.08	1.59	0.08	0.53	0.18	2.30	0.18	0.53	0.18	2.83
February	0.65	0.12	1.65	0.12	0.55	0.31	2.39	0.31	0.55	0.31	2.94
March	0.76	0.21	1.70	0.21	0.57	0.37	2.45	0.37	0.57	0.37	3.02
April	8.83	0.13	1.70	0.04	0.57	0.03	2.45	0.03	0.57	0.03	3.02
May	12.75	0.13	1.54	0.00	0.51	0.00	2.23	0.00	0.51	0.00	2.74
June	18.23	0.13	1.48	0.01	0.49	0.08	2.13	0.08	0.49	0.08	2.62
July	21.84	0.09	1.60	0.09	0.53	0.76	2.31	0.76	0.53	0.76	2.84
August	23.18	0.13	1.45	0.04	0.48	0.86	2.09	0.86	0.48	0.86	2.57
September	19.33	0.16	1.44	0.16	0.48	1.40	2.08	1.40	0.48	1.40	2.56
October	11.33	0.31	1.83	0.31	0.61	1.20	2.64	1.20	0.61	1.20	3.25
November	6.89	0.30	1.86	0.30	0.62	0.84	2.69	0.84	0.62	0.84	3.31
December	1.82	0.18	1.75	0.18	0.58	0.70	2.53	0.70	0.58	0.70	3.12

Table A.7- 11 Ozonation data for WTP 7, 8 and 9

Month	WTP 7			WTP 8			WTP 9				
	Temp. (^{0}C)	Tank 1		Temp. (^{0}C)	Tank 1		Temp. (^{0}C)	Tank 1		Tank 2	
		Residual (mg/L)	T_{50} (min.)		Residual (mg/L)	T_{50} (min.)		Residual (mg/L)	T_{50} (min.)	Residual (mg/L)	T_{50} (min.)
January	0.89	0.27	10.41	0.62	0.29	15.68	0.40	0.35	25.53	0.37	25.53
February	0.9	0.27	10.26	0.4	0.29	14.89	0.40	0.28	25.40	0.29	25.40
March	0.95	0.27	10.3	0.52	0.29	15.25	0.52	0.38	24.61	0.38	24.61
April	5.57	0.31	10.34	5.53	0.26	15.02	5.53	0.38	22.69	0.38	22.69
May	12.68	0.44	10.11	12.95	0.32	13.42	12.95	0.34	22.19	0.38	22.19
June	17.94	0.38	10.14	18.72	0.29	12.6	18.72	0.37	21.66	0.38	21.66
July	21.58	0.32	11.12	21.99	0.42	13.8	21.99	0.37	22.56	0.40	22.56
August	23.07	0.29	10.54	23.54	0.48	12.71	23.55	0.38	20.36	0.39	20.36
September	19.21	0.38	10.29	19.13	0.54	13.46	19.13	0.41	21.51	0.40	21.51
October	11.69	0.36	11.64	10.85	0.5	0	10.85	0.40	25.38	0.39	25.38
November	7.37	0.35	11.51	6.77	0.31	0	6.77	0.37	26.69	0.36	26.69

Table A.7- 1 Ozonation data for WTP A and B

WTP A				WTP B			
Date	Ozonation- Total Log Removal			Date	Ozonation- Total Log Removal		
	E. coli	*Giardia*	*Crypto.*		*E. coli*	*Giardia*	*Crypto.*
09-Jul	8	9.98	1.69	09-Jun	8	8.39	0.65
09-Sep	8	8.66	2.02	09-Nov	8	7.53	0.65
10-Jan	8	7.83	0.81	10-Jan	8	7.76	0.56
10-Apr	8	7.27	0.75	10-Apr	8	7.3	0.72
10-Jun	8	10.87	2.19	10-Nov	8	8.3	0.72
10-Sep	8	10.84	1.69	11-Jan	8	7	0.68
10-Nov	8	9.12	1.17	11-Mar	8	7.01	0.7
11-Jan	8	7.77	0.78	11-Apr	8	7.86	0.9
11-Mar	8	8.02	0.83	11-Jul	8	6.58	0.81
11-Apr	8	8.32	0.93				
11-Jul	8	10.98	1.87				

APPENDIX 8- Predicted inactivation using CT_{50} calculations

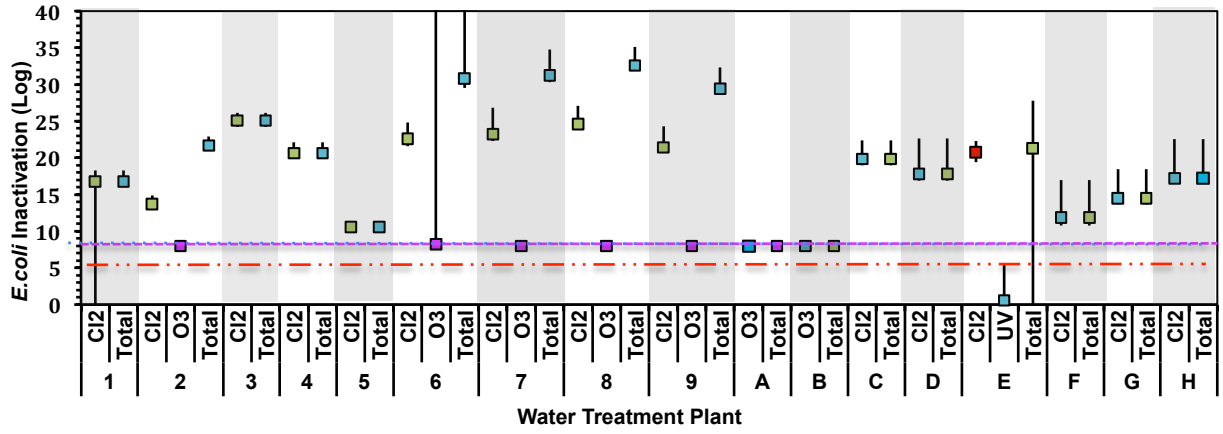

Figure A.8- 41. Mean annual inactivation *E.coli* predicted using CT_{50} calculations.

* Capping values used for chlorination (Cl_2) (dotted line) ozonation (O_3) (dashed line) and UV irradiation (intermittent dashed and dotted line) in the HC QMRA model. Error bars represent minimum and maximum monthly predictions.

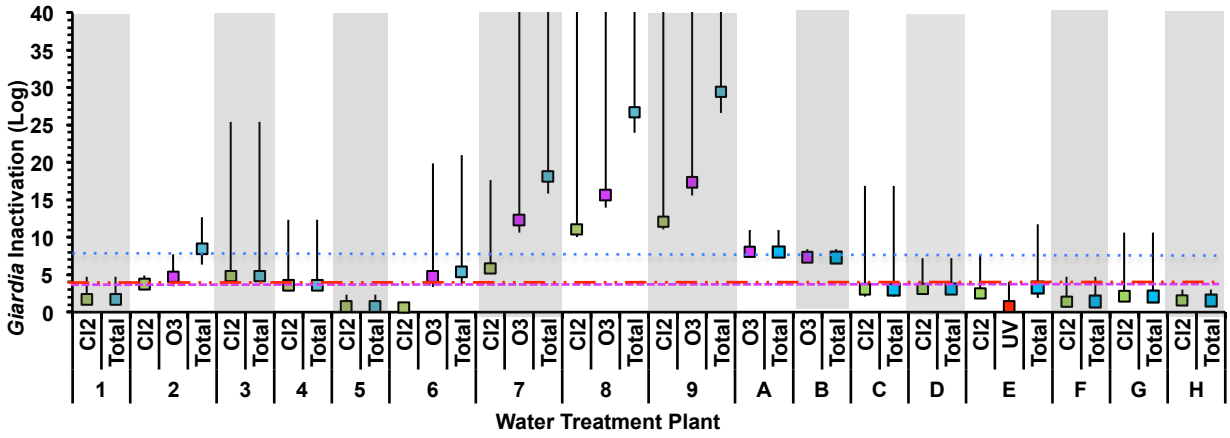

Figure A.8- 42. Mean annual inactivation *Giardia* predicted using CT_{50} calculations.

* Capping values used for chlorination (Cl_2) (dotted line) ozonation (O_3) (dashed line) and UV irradiation (intermittent dashed and dotted line) in the HC QMRA model. Error bars represent minimum and maximum monthly predictions.

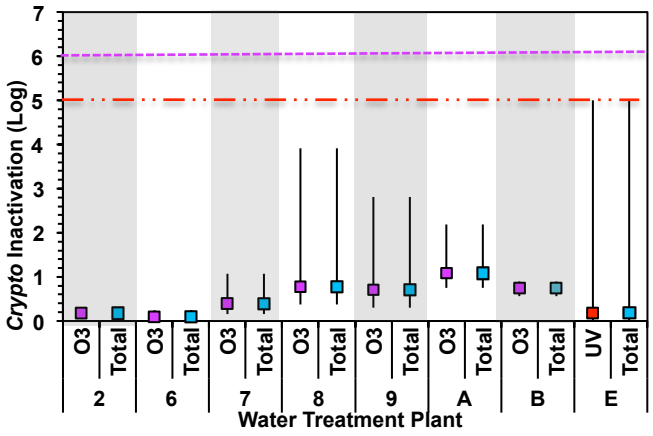

Figure A.8- 43. Mean annual inactivation *Cryptosporidium* predicted using CT_{50} calculations.

* Capping values used for chlorination (Cl_2) (dotted line) ozonation (O_3) (dashed line) and UV irradiation (intermittent dashed and dotted line) in the HC QMRA model. Error bars represent minimum and maximum monthly predictions. Chlorine inactivation of *Cryptosporidium* are not considered presented due to the very low predicted.