ENVIRONMENTAL AND HEALTH RISK ASSESSMENT OF TRIHALOMETHANES IN DRINKING WATER A CASE STUDY

SUDIP KAR

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ENVIRONMENTAL AND HEALTH RISK ASSESSMENT OF TRIHALOMETHANES IN DRINKING WATER - A CASE STUDY

By

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Abstract

A comprehensive research related with chlorination by products in drinking water was conducted to assess health-associated risks. Three communities, namely St. John's, Clarenville, and Shoal Harbour were chosen in Newfoundland. Gas Chromatography/ Mass Spectrophotometer (GC/MS) was used for the analysis of the samples at Environmental Quality Laboratory in Newfoundland. Four trihalomethanes (THMs) species, namely chloroform, dichloro-bromomethane, chloro-dibromomethane, and bromoform were analysed. Chloroform was found to be in maximum concentration in comparison to other species. To analyse seasonal variation of the data, both Student's t-test and Mann-Whitney test were conducted. As a result of hypothesis testing, the null hypothesis, which was that the mean chloroform concentrations (for Student's t-test) and median chloroform concentrations (for Mann-Whitney test) respectively for the two seasons were equal, was not rejected for Clarenville and St. John's, whereas rejected for Shoal Harbour. Due to significant presence and known behaviour of chloroform, risk was estimated based on chloroform concentration only. For St. John's the chloroform concentration varied from non detectable level (<1) to 73 μg/L in summer and 3 to 60 μg/L in winter, respectively. For Clarenville the concentration varied from 375 to 512 μg/L in summer and 361 to 557 μg/L in winter. Similarly, for Shoal Harbour, it varied from 203 to 330 μg/L in summer and 155 to 235 μg/L in winter respectively. The lower concentration of chloroform in winter can be attributed to the fact that lesser chlorination practices are performed. The risk associated with chloroform was evaluated through different exposure pathways: ingestion, inhalation and dermal contact through showers. Lifetime risk from water ingestion ranged from 0.08×10^{-4} to 0.82×10^{-4} (summer) and 0.07×10^{-4} to 0.78×10^{-4} (winter). Lifetime risk from normal shower as a result of 10 minutes shower ranged from 0.48×10^{-4} to 6.33×10^{-4} (summer) and 0.40×10^{-4} to 6.07×10^{-4} (winter). To address issues pertaining to limited number of samples, probabilistic risk analysis was also conducted on the original set of data. The software @RISK was used to perform the risk analysis and simulation. Latin Hypercube Simulations was performed to estimate the risk and the results were plotted. The risk values estimated using @RISK were compared with those estimated using deterministic approach.

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List of Symbols

θ : Scale Factor for Maximum Extreme Value Distribution

s : Lognormal Scatter Factor

N : Total Number of Data Points

$F_x(x)$: Cumulative Distribution Function

μ : Mean

σ: Standard Deviation

ϕ^{-1} : Transformation Factor (NORMSINV)

a : Minimum Value

b : Maximum Value

x_0 : Median Value

LC_{50} : Median Lethal Concentration

LD_{50} : Median Lethal Dose

LD_{100} : Dose That Will Kill 100 Percent of Experimental Subjects

LCL_0 : Lowest Published Lethal Concentration

LDL_0 : Lowest Published Lethal Dose

TCL_0 : Lowest Published Toxic Concentration

TDL_0 : Lowest Published Toxic Dose

$\bar{y}_1$: Mean Trihalomethanes Concentrations in Summer

$\bar{y}_2$: Mean Trihalomethanes Concentrations in Winter

S_1^2 : Variance of Trihalomethanes Concentrations in Summer

S_2^2 : Variance of Trihalomethanes Concentrations in Winter

n_1 : Number of Data Points in Summer

n_2 : Number of Data Points in Winter

ν : Degrees of Freedom

H_0 = Null Hypothesis

H_1 = Alternative Hypothesis

μ_1 = Mean Total Trihalomethanes Concentration in Summer Season

μ_2 = Mean Total Trihalomethanes Concentrations in Winter

W = Sum of the Ranks in the Combined Sample Associated With X Observations

η_1 = Median of the Trihalomethanes Concentrations in Summer

η_2 = Median of the Trihalomethanes Concentrations in Winter

List of Acronyms

TOC: Total Organic Carbon

THMs: Trihalomethanes

TTHMs: Total Trihalomethanes

DOC: Dissolved Organic Carbon

POC: Particulate Organic Carbon

NOM: Natural Organic Matter

DBPs: Disinfection By-Products

HAAs: Haloacetic Acids

DCA: Dichloroacetic Acid

TCA: Trichloroacetic Acid

$CHCl_3$: Chloroform

$CHBrCl_2$: Bromodichloromethane

$CHClBr_2$: Chlorodibromomethane

$CHBr_3$: Bromoform

UV: Ultra Violet

TTHMFP: Total Trihalomethanes Formation Potential

GAC: Granular Activated Carbon

AOPs: Advanced Oxidation Processes

PAC: Powdered Activated Carbon

U. S. EPA: United States Environmental Protection Agency

Di: Chloroform Dose from an Inhalation-Only Exposure (μg/Inhalation Exposure- kg)

Er: Chloroform Absorption Efficiency via Respiratory System

Ca: Air Concentration in Shower ($\mu g/m^3$)

R: Breathing Rate (m^3/min)

T: Duration of Shower (min)

Wt: Body Weight of a Reference Person (70-kg)

Dd: Chloroform Dose from a Dermal Exposure (μg/Dermal Exposure-kg)

F: Ratio of the Body Burden from Dermal Exposure to that from Inhalation Exposure

Ei: Absorption Efficiency of Chloroform via the Gastrointestinal Tract

Cw: Chloroform Concentration in the Water (μg/L)

Aw: Quantity of Water Ingested per Day (L/day)

Dig: Dose from Water Ingestion (μg/kg-day)

Pd: Lifetime Risk (Unit Less)

Q: Cancer Risk Potency Slope $(mg/kg\text{-}day)^{-1}$

D: Chloroform Dose (μg/kg-day)

Chapter 1

Introduction

1.1 Drinking Water and Public Health

Drinking water is the source of life. It is the basic substance for sustaining life. Water is considered as the nature's hidden treasure. The drinking water, also known as "potable water", is the water supplied to the consumer that can be safely used for drinking, cooking, and washing. The safe drinking should neither contain the disease causing organisms nor it should contain the minerals and the organic substances at the concentration levels that may cause adverse health effects. It should be aesthetically acceptable and free from the apparent turbidity, odour, colour, and any objectionable taste (AWWA, 1990).

Since the public health aspects of drinking water are very significant and complicated, the concerned health regulatory agencies in the communities undertake reviews, inspection, sample collection, monitoring, and evaluation on a continuous basis of the water supplied to the community with the help of "constantly updated" drinking water standards. Public health regulatory manoeuvrings like these ensure uninterrupted supply of water with the safe limits. In order for the water, delivered to the "ultimate consumer", (at the kitchen faucet) considered safe or potable, it must be scrutinised with a multi-disciplinary approach involving bacteriology, chemistry, physics, engineering and public health, and preventive medicine (Zuane, 1990). Despite advances in the global water supply coverage during and since the 'Water Decade', around one billion persons (20 per cent of the global population) lack access to the safe drinking water (WHO,

1999). In developing countries, 11.000 children die each day of water-related diseases and 2.9 billion people do not have the adequate sanitation facilities (UNICEF, 1999).

Water quality problems can be broadly characterised as microbiological, physical, and chemical. Microbiological problems focus on the waterborne diseases. General or physical parameters include taste and odour, colour, temperature, pH, alkalinity, hardness, solids (total dissolved solids), turbidity, and solubility. Chemical parameters examine the inorganic and organic compounds. The present study concentrates on the water contamination due to the presence of specific chemical compounds i.e. trihalomethanes.

In 1980s, waterborne diseases such as typhoid, cholera, dysentery, amebiasis, salmonellosis, shigellosis, and hepatitis A were estimated to be responsible for the deaths of more than 30, 000 people daily (IRC, 1984). In that context, the United Nations General Assembly declared 1981-1990, as the "International Drinking Water Supply and Sanitation Decade" (WHO, 1984). In the 19th century, major outbreaks of waterborne disease took place in Canada, the United States, and other developed countries. Cholera and dysentery were rampant in the 1800s, and typhoid fever responsible for about 25,000 deaths in the United States as late as 1900 (Akin et al., 1982).

The fundamental objective of the water disinfection is to control the pathogenic bacteria, viruses, helminths, and protozoa that cause the major waterborne diseases. Some outbreaks still occur in the United States owing to continuing problems involving consumption of the untreated water, insufficient or interrupted disinfectants, failure to maintain the adequate levels of residual disinfectants in potable water distribution systems, and /or breaches in the system (Akin et al, 1982).

The etiology of waterborne disease has changed dramatically since the early 1900s. Most outbreaks in the recent years have been caused by the viruses and the protozoan cysts that are generally more resistant to the disinfectant than the pathogenic bacteria (National Academy of Press, 1987).

Chlorine was discovered in 1774 by the Swedish chemist Karl Wilhelm Scheele, while Sir Humphry Davy confirmed it to be an element in 1810 (White, 1992). Semmelweis first introduced the use of chlorine as a disinfectant on the maternity ward of the Vienna General Hospital in 1846 to clean the hands of the medical staff and prevent the puerperal fever (Wigle, 1998). In 1881, Koch was able to demonstrate that the pure cultures of bacteria were destroyed by the hypochlorites (White, 1992). The first continuous usage of the chlorination in the US started in 1908 for the water supply to Jersey City in New Jersey, and at a site that served the Chicago Stockyards to control the sickness in livestock caused by the sewage-contaminated water (White, 1992). In Canada, the earliest use of chlorination found by Wigle was in Peterborough, Ontario, in 1916 (PUC, 1998). In the early years of 20th century, the practice of chlorinating drinking water prompted the elimination of diseases such as the cholera, typhoid fever in addition to other waterborne diseases. This was a phenomenal advancement in the field of public health and safety. Several countries world-wide including Canada, the United States have successfully employed chlorination as a major disinfection process for drinking water for many years. Chlorination has positioned itself as a major offensive against most waterborne pathogens.

Canada has plentiful supplies of good drinking water. In reviewing the human health and water quality issues in Canada, Environment Canada (1999) has stated,

"Water-related illnesses — typhoid fever, cholera, dysentery — are almost unknown in this country today. Waste and wastewater treatment, the development and enforcement of the drinking water guidelines, public health practices and education — all have resulted in a decrease in the water related illnesses in Canada".

Water quality standards and regulations refer to the drinking water in quantitative terms. The term "drinking water standards" typically refers to the numerical limits that define the maximum concentration of contaminants that water may contain to be considered potable (i.e., safe to drink) (Pontius, 1999). In providing an overview on the Safe Drinking Water Act (SDWA) in United States, Pontius in the article "History of the Safe Drinking Water Act (SDWA)", has stated, " The Safe Drinking Water Act (SDWA) is the principal law governing drinking water safety in the United States. Enacted initially in 1974, the SDWA as amended authorises the U.S. Environmental Protection Agency (U. S. EPA) to establish comprehensive national drinking water regulations to ensure drinking water safety. " Similar to U. S. EPA, various other regulatory agencies are constantly involved in ensuring the supply of safe and pure drinking water to the public.

1.2 Waterborne Diseases

Contaminated drinking water always has been an active media in the past for transmitting the infectious diseases. With the technological advancement in the field of water and wastewater treatment, the frequency of infectious diseases has reduced considerably throughout the world. American Water Works Association (AWWA) has classified the water-related diseases into four general groups on the basis of

epidemiological considerations (U. S. EPA, 1993): (1) water -washed diseases, (2) water-based diseases, (3) water-vectored diseases, and (4) waterborne diseases.

Water-washed diseases are associated with the improper hygienic habits and sanitation. These diseases affect the eye and skin. Insufficient water usage for washing and bathing facilitates these categories of diseases.

A significant portion of the pathogen's life is spent in the water. The pathogen is dependent on the aquatic organisms for the completion of its life cycle. The diseases associated with these events are classified as *water-based diseases*. Diseases like schistosomiasis and dracontiasis belong to this group.

Certain group of insects breed in the water or bite in the water neighbourhood. Diseases transmitted by these insects are termed as the *water-vectored diseases*. Yellow fever and malaria are the water-vectored diseases. *Waterborne diseases* are caused by the ingestion of the contaminated water. Cholera and typhoid are well known waterborne diseases. Some diseases are caused by the pathogenic bacteria, viruses, protozoan, helminthes etc. These diseases are mostly caused by the faecal-oral route, from human to human or animal to human. Developing countries are always under the threat of diarrhoea that is a major factor for the infant mortality and morbidity. Examples of some of the waterborne diseases are listed in Table 1.1 (U. S. EPA, 1993).

Waterborne Pathogens Elimination

Microorganisms are present everywhere in our environment. We find them in the soil, air, food, and water. They cannot be seen with the naked eyes. Human beings do not get affected by the microorganisms before their birth but thereafter rapidly get exposed to the microorganisms by virtue of human activities like breathing, eating, and drinking.

Table 1.1 Examples of Some Waterborne Diseases

Name of Organism or Group	Disease	General Symptoms	Primary Sources and Major Reservoirs
Bacteria	Typhoid fever	Fever, nausea, diarrhoea, vomiting, headache, constipation, appetite loss	Human faeces
	Cholera	Vomiting, watery diarrhoea, muscle cramps	Human faeces
	Gastro-enteritis	gastrointestinal disorder	Human faeces, animal faeces
Virus (hepatitis A)	Hepatitis	Fever, jaundice, coloured urine, abdominal discomfort, chills	Human faeces
Virus	Viral Gastro-enteritis	Fever, gastrointestinal disorder, vomiting, headache, diarrhoea	Human faeces
Protozoan	Amebiasis	Fatigue, abdominal discomfort, diarrhoea, flatulence, weight loss	Human faeces
	Cryptosporidiosis	Abdominal discomfort, diarrhoea	Human and animal faeces
	Giardiasis	Abdominal discomfort, diarrhoea	Human and animal faeces

Microorganisms that can cause disease are named as microbial pathogens. They can be harmful to those who become infected. Many diseases fail to have any impact on the healthy individuals but the same diseases may have fatal effects on the individuals not having strong immune systems. There are instances where an infection has led to the creation of "Carrier State" where the body starts to carry the disease- causing agents but does not exhibit any symptoms.

Diseases caused by the consumption of contaminated water are termed as waterborne diseases. EPA has considered the other exposure pathways such as the inhalation of water vapours and dermal contact during bathing in the hospital environment.

Exposure pathways such as the ingestion (drinking water), bathing and ingestion during the water recreational activities (e.g., swimming, and water skiing) are common but the uncontrolled and improper exposure may lead to the widespread outbreaks. Waterborne disease outbreaks are incidents when a) two or more persons report similar illness as a consequence of ingestion or usage of the water intended for drinking and b) epidemiological studies recognise the water as the source of illness. (Levine and Craun, 1990). A single case of chemical poisoning may be considered as an outbreak, if laboratory evidences suggest that the chemicals have contaminated the water. Agencies such as Center for Disease Control, and U. S. EPA study and report the outbreak data and undertake the waterborne disease outbreak investigation and assessment. In addition, the state health departments offer the epidemiological support and service, engineering and environmental consultations in the area of water treatment. The agencies also undertake the water sample collection program to identify the viruses, parasites, and bacterial pathogens. Despite these attempts, the waterborne outbreaks identified, reported and analysed account for only a fraction of the actual occurrences due to the mildness and short duration of the related symptoms. Incidentally, the pathogenic agents are identified only half of the time. Some experts are of the opinion that the contaminated drinking water is the initial source of infection of some foodborne disease outbreaks.

Pathogens associated with the waterborne diseases mostly belong to the group of microbial agents like the bacteria, viruses and protozoa. Theoretically, to remove these pathogens from the drinking water is not an arduous job. We can just add the disinfectants, provide a sufficient contact time to ensure that the disease causing capabilities of the microbes have been completely destroyed and then the disinfected water can be released for the distribution. In practical applications, the process is not so simple because of the fact that many conditions come into the picture.

The physical characteristics of the water like dissolved and suspended solids have the ability to affect the process of disinfection. The chemical parameters like the naturally occurring organic matters and matters produced by human activities can influence the normal chemical reactions expected to take place during treatment and disinfectant process. The pathogens, which are associated with the higher organisms like the algae, rotifers, and worms, may survive the effect of disinfectants. The aforesaid impediments are eliminated in the actual drinking water treatment processes that comprise of screening, coagulation, flocculation, sedimentation, filtration, disinfection, clear water reservoir, and pumping into the main distribution system. After the impurities are removed from the untreated water, sufficient quantity of disinfectants is added to the water. This renders the pathogens harmless. It is imperative to maintain a residual level of disinfectant along the water distribution systems. This is to prevent any recurrence of the microbial growth or invasion of harmful microorganisms into the distribution pipes.

Sometimes untreated or insufficiently treated wastewater is discharged into the fresh water bodies that are used by other communities. This exposes the communities to potential hygienic hazards (U. S. EPA, 1993).

Typhoid fever and amebiasis were the two most deadly waterborne diseases in the United States at the beginning of this century. The continuous decreasing trend in the number of outbreaks and fatalities reinforces the fact that there is a growing progress in the area of waterborne disease prevention. U. S. EPA has attributed this progress to the increased implementation of the important treatment practices such as the filtration and disinfection. The agency has also suggested rigorous monitoring for the indicators of the faecal contamination.

1.3 Disinfection and Disinfection By- Products

Disinfection is a process designed for the reduction of pathogenic microorganisms. Disinfection process is undertaken by a number of physical and chemical agents. Chlorine, chlorine dioxide, ozone are important disinfecting agents or disinfectants. Other methods such as heat, extremes in pH, metals (silver, copper), surfactants, and permanganate can also be used to inactivate the microorganisms.

Disinfection by-products (DBPs) in water are the chemical substances that are formed when the water is subjected to disinfection in the water utilities. Chlorinated disinfection by-products are the by-products found with chlorine. Important classes of compounds (DBPs) are the trihalomethanes (THMs), haloacetic acids (HAAs), haloacetonitriles, halopicrin, chloral hydrate. THMs and HAAs are the major by-products associated with chlorine. The precursor compounds in the water significantly

influence DBPs formation and speciation. In the present scenario, water utilities consider DBPs issues as the most challenging task since there is a potential health effect associated with the exposure to certain DBPs.

Water chlorination causes the formation of several by-products, which can be classified as the halogenated and non-halogenated by-products (Mills et al., 1998; Figure 1.3.1). The halogenated compounds comprise of the trihalomethanes that are the most commonly occurring disinfectant by-products. In addition, the haloacetic acids, which consist of the dichloroacetic acid (DCA) and trichloroacetic acid (TCA) are the member of this group of compounds. The non-halogenated compounds are mostly natural substrates or metabolites. The concentration levels of these by-products are the function of level of the organic material in the source water. As a result, the water supplies that use the surface waters (lakes, rivers, and reservoirs) as their intake source produce the higher levels of by-products than the water supplies that use the ground waters (wells, springs) as their intake source. The type and quantity of the by-products produced depends on the factors such as the amount and character of organic material, ambient pH level and bromide concentration in the water (Mills et al., 1998).

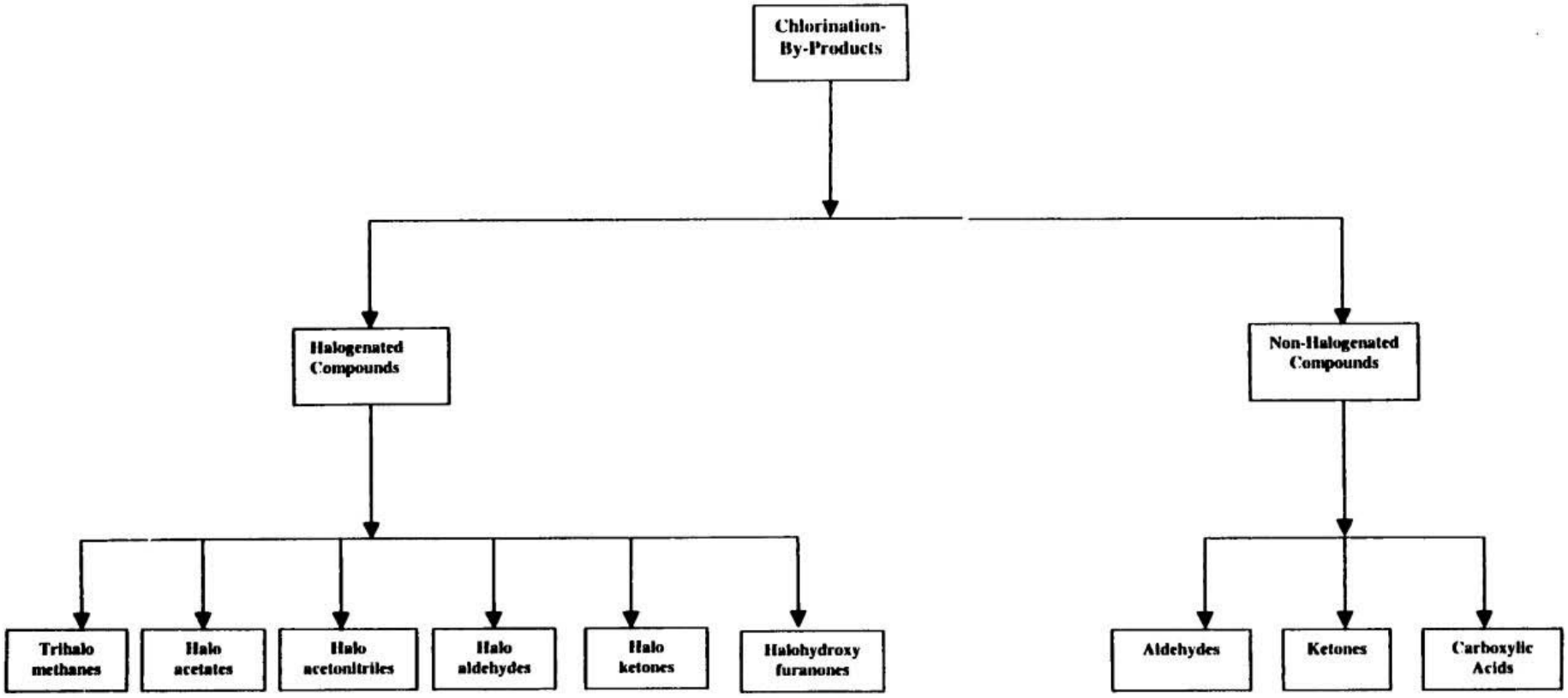

Figure 1.3.1 Classified Chlorinated Disinfection By-Products

1.4 Purpose of Study

The purpose of the present study is to conduct the risk assessment of trihalomethanes (THMs) present in the Canadian drinking water supplies. THMs concentration levels, both at the national and provincial levels in Canada, are considered for the evaluation. Laboratory experiments are performed to analyse and measure all the four chemical compounds of THMs in some selected communities in Newfoundland. The study also aims at undertaking a health impact assessment by estimating the health risk associated with the THMs exposure.

1.5 Significance of Study

Today, most of the Canadian drinking water supplies are free of viruses, bacteria, and protozoa- that cause the fatal diseases like the cholera, and typhoid fever, in many nations (Health Canada, 1999). These advances are mostly attributed to the application of disinfectants such as the chlorine in the water treatment. When the water is subject to chlorination in an attempt to eliminate the disease causing microorganisms, the chlorine comes in contact with the naturally occurring organic matter (e.g., decay products of vegetation). As a result of this reaction, the chlorination by-products are formed in the water.

Considerable research has been conducted to examine the association between the exposure to the trihalomethanes in drinking water and the potential increase in risk of various cancers.

The study is aimed at reviewing the drinking water quality issues due to the formation of disinfection by-products and the related health effects. The fundamental

objective is to estimate the excess cancer risk associated with the use of chlorinated tap water. Attempts are also made to address the emerging questions slated for the environmental engineers and risk managers.

1.6 Outline of Thesis

The review on THMs, their origin, the chemical characteristics, toxicity, and health effects are presented in Chapter 2.

Chapter 3 focuses on the sampling program with details of the sample collection methods and sampling protocols. Results of the laboratory analysis of the drinking water samples are listed in this chapter. Overview of Canadian drinking water is presented in Chapter 4. This Chapter also reviews the National Survey of chlorinated disinfection by-products in Canadian drinking water conducted in 1993 with the risk assessment under different scenarios of its uses.

General overview of the drinking water quality in Newfoundland is presented in Chapter 5. The risk assessment of the water samples under various exposure scenarios is described in this chapter. Chapter 6 presents the probabilistic risk analysis. The procedure proposed for the probabilistic risk analysis includes the normal probability plot and use of the @RISK software. The software @RISK is used to perform the risk analysis and simulation. The concluding remarks and recommendations are given in Chapter 7.

Chapter 2

Literature Review

2.1 THMs and their Origin

2.1.1 Origin

Trihalomethanes are single-carbon compounds having general formula CHX_3, where X may be chlorine, fluorine, bromine or iodine, or combinations. They are halogen substituted. The formation of these compounds takes place in drinking water when the naturally occurring organic matters in raw water are subject to chlorination to kill the microorganisms that cause the various waterborne diseases.

The levels of THMs in drinking water depend on factors like the time and place of water chlorination. THMs levels in drinking water also suggest the seasonal variations. In winter months the concentrations are found to be lower (Otson, 1987; Otson et al., 1981; Otson et al., 1982; Williams et al., 1980). The levels can be lowered, by reducing the concentration of the materials, which enhances the THMs formation. During winter, by reducing the quantity of applied chlorine, the THMs level can be reduced significantly at that time of the year (Kar and Husain, 1999). Since the concentrations of the natural organic matter are lower in the winter, the quantity of chlorine required to disinfect is much less in the winter than in the summer. Hence, the THMs concentration in the drinking water is generally lower in the winter than in the summer. The source of the incoming water is also important. In water bodies like the large lakes and wells, the organic matter content is less, which leads to lower THMs levels in the chlorinated water. Whereas, if the water is taken from the surface water

sources like the river, the level of THMs will be high due to the increased organic matter content.

The THMs mostly found in the drinking water are in the form of chloroform ($CHCl_3$), bromodichloromethane ($CHBrCl_2$), chlorodibromomethane ($CHClBr_2$) and bromoform ($CHBr_3$). All the four compounds are in the liquid state at room temperature and are low soluble in the water with values less than 1 mg/mL at 25^0C. Their volatility also varies between moderate to high range, having vapour pressure values ranging from 0.80 kPa for bromoform to 23.33 kPa for chloroform at 25^0C. The log octanol-water partition coefficients range from 1.97 for chloroform to 2.38 for bromoform. All the four compounds undergo decomposition if exposed to air or light. Chloroform among all the THMs has the most significant presence and highest concentration in drinking water (Health Canada, 1993). The four main constituents of THMs are now discussed.

2.1.2 Different Compounds of THMs

Chloroform. ($CHCl_3$) It is clear, colourless, non-flammable liquid having a characteristic heavy, pleasant and burning sweet taste. It dissolves in acetone and dissolves slightly in water (NAS, 1978).

Bromoform. ($CHBr_3$) Bromoform is a colourless liquid having a strong chloroform-like odour and an acceptable taste. It is less volatile, slightly soluble in water, soluble in the benzene and chloroform. It is also known as tribromomethane or methenyl tribromide (NAS, 1978).

Bromodichloromethane. ($BrCHCl_2$) Bromodichloromethane is a colourless liquid that is insoluble in water and has a high solubility in ethyl alcohol, diethyl ether, acetone, and benzene (NAS, 1978).

Dibromochloromethane. (Br_2CHCl) Dibromochloromethane is a colourless liquid that is insoluble in water but soluble in ethylalcohol, diethyl ether, acetone, and benzene (NAS, 1978).

2.1.3 Formation Mechanism

When chlorine gas is bubbled into pure water, rapid hydrolysis to hydrochloric and hypochlorous (HOCl) acids take place (Larson and Weber, 1994):

Cl_2 (Chlorine) + H_2O (Water) $\longrightarrow$ HCl (Hydrochloric Acid) + HOCl

(Hypochlorous Acid)

The hypochlorous acid undergoes the following reaction, resulting in the formation of THMs and other halogenated DBPs (Singer, 1994).

HOCl (Hypochlorous Acid) + Br^- (Bromide Ion) +

NOM (Natural Organic Matter) $\longrightarrow$ THMs and Other Halogenated DBPs

When the bromide ions (Br^-) are not present, the formation of chlorinated by-products only takes place. When the bromide is present, the free chlorine (hypochlorous acid) oxidises the bromide to hypobromous acid (HOBr). This, together with the residual hypochlorous acid, reacts with the natural organic matter (NOM) resulting in the formation of mixed chloro-bromo substitution products (Singer, 1994).

The rate and degree of THMs formation is directed by the chlorine dose and the humic acid concentration, pH, temperature and bromide ion concentration (Stevens et

al., 1976; Amy et al., 1987). Factors influencing the halogenated DBPs formation include pH, contact time, temperature and season, nature and concentration of NOM, chlorine dose and residual, bromide concentration (Singer, 1994). Presence of bromides facilitates brominated THMs formation and chloroform concentrations decrease proportionally (Aizawa et al., 1989). Trihalomethanes production also depends on the point of chlorination (Health Canada, 1993).

Table 2.1 Four THMs Compounds and Their Characteristics (NAS, 1978)

Parameter	**Chloroform**	**Bromoform**	**Bromo dichloro methane**	**Dibromo chloromethane**
Molecular Weight	119.38	252.7	163.83	208.29
Melting Point	-63.5°C	8.3°C	-57.1°C	-22°C
Boiling Point	61.7°C	149.5°C	90.0°C	119-120°C
Liquid Density	1.483 g/ml (20^0C)		1.980 g/ml (20°C)	(Density) 2.451
Vapour Pressure (kPa at (°C))	21 (20^0C)	0.7 (25^0C)	6.7 (20^0C)	2.0 (10.5^0C)
Vapour Specific Gravity	4.36 g/l (air =1.0) (E)			-
Water Solubility (mg/L at (°C))	8000 (20^0C)	3190 (30^0C)		
Octanol / Water Partition Coefficient (log P_{ow})	1.97	2.30	1.88	2.09

2.1.4 THMs Toxicology Information (NAS, 1978)

A brief description of the toxicological properties of the four compounds of THMs is presented in this section.

Chloroform: When chloroform is inhaled, it is considered many times more potent than carbon tetrachloride as a depressant of the central nervous system, whereas when it is ingested, it is considered less toxic than carbon tetrachloride (NAS, 1978).

Table 2.1.2 Some Toxic Doses of Chloroform in Animals

Rat	Oral	LD_{50}	800 mg/kg
Rat	Inhalation	LCL_0	8,000 ppm/4H
Mouse	Oral	LDL_0	2,400 mg/kg
Mouse	Oral	TDL_0	18 gm/kg/1200
Mouse	Inhalation	LC_{50}	28 ppm
Mouse	Subcutaneous	LD_{50}	704 mg/kg
Dog	Oral	LDL_0	1,000 mg/kg
Dog	Inhalation	LC_{50}	100 ppm
Dog	Intravenous	LDL_0	75 mg/kg
Rabbit	Inhalation	LC_{50}	59 ppm
Rabbit	Subcutaneous	LDL_0	800 mg/kg
Guinea pig	Inhalation	LCL_0	20,000 ppm/2H

Source: NAS, 1978 (The acronyms used in the above table are defined in the List of Acronyms at the beginning)

Chloroform quickly spreads to all the organs of the body after its absorption. When chloroform vapour having concentrations of about 1000 ppm are inhaled for few minutes, it causes moderate toxic effects. However higher concentrations can cause more toxic effects rapidly and exposure to 15,000 ppm for an extended duration poses threat to life. Several cases of acute poisoning have been reported as a result of the

accidental overdose of chloroform during the anaesthesia. Inhalation of chloroform is the major cause of most poisonings. Chloroform poisonings also yield the significant pathological outcomes. The toxic doses of chloroform in animals are listed in Table 2.1.2 (U. S. DHEW, 1975). Chloroform has been classified in Group II-probably carcinogenic to the humans (inadequate evidence in the humans but sufficient data in the animals) (Health Canada, 1993). These health groups are the carcinogenic classification of chemicals. These classifications are developed by the U. S. EPA and Health Canada.

Bromoform: Bromoform is considered to be a highly toxic material. It is more toxic than methylene bromide but it is less toxic than carbon tetrabromide. Iodoform and chloroform seem to be more toxic than bromoform. The LD_{50} in animals due to the exposure to bromoform are listed in Table 2.1.3. Bromoform has been classified in Group IIIB- possibly carcinogenic to the humans on the basis of limited evidence in animals (one species; some evidence in one sex and clear evidence in other sex) and inadequate data in the humans (Health Canada, 1993).

Table 2.1.3 Some Toxic Doses of Bromoform in Animals

Mouse	Subcutaneous	LD_{50}	1,820 mg/kg
Rabbit	Subcutaneous	LDL_0	410 mg/kg

Source: NAS, 1978

Bromodichloromethane: It is the only other THM considered here that has been classified in Group II- probably carcinogenic to the humans (sufficient evidence in the animals; inadequate data in the humans) (Health Canada, 1993).

polar nature. They also have the variable adsorbability, which depends on the source and type of pre-treatment provided. The THMs precursor measurements are expressed in terms of the total organic carbon (TOC). The concentrations of THMs precursors may range from 1 to 10 mg/L approximately. Higher values may be obtained in case of the swampy waters. Humic substances mostly consist of the TOC. Humic substances cannot be measured directly using the analytical techniques due to their heterogeneous and ill-defined character (McPhee, 1992). Humic substances are therefore characterised in terms of non-specific parameters on the basis of their organic carbon content (i.e. DOC), their degree of absorption of ultra violet (UV) light (i.e. UV absorbance at 254 nm [UV-254]), or their potential to form TTHMs (i.e. Total trihalomethanes formation potential {TTHMFP}).

By measuring the total trihalomethanes formation potential (TTHMFP), we can measure the THMs precursors indirectly. But standardisation of the experimental methods to measure TTHMFP is yet to be done. Different pH, temperature, or formation period may be used to measure TTHMFP.

Parameters for measuring the THMs precursors (MacPhee, 1992) are total organic carbon (TOC), UV absorbance, and total trihalomethanes formation potential. These are briefly described as follows:

(a) Total organic carbon is comprised of the dissolved organic carbon (DOC) and particulate organic carbon (POC). In Atlantic Canada, the POC concentration is negligible for many natural waters and the TOC can be considered equal to the DOC. Dissolved organic carbon comprises all of the organically bound carbon present in the

water. Dissolved organic carbon present in most natural waters consists of nearly 50 percent of the aquatic humic substances.

$$TOC = DOC + POC$$

(b) The humic fraction of the NOM is considered as aromatic compounds due to their structural similarity with benzene (C_6H_6). The compounds belonging to this group have the unique property of absorbing light in the ultraviolet (UV) wavelength region. Ultra violet (UV) absorbance method is therefore used to measure the humic substances in raw waters. However, there are some organic compounds in raw waters that may not absorb the UV light. Ultra violet absorbing constituents in a sample absorb the UV light in proportion to their concentration (Macphee, 1992).

(c) Macphee (1992) has defined TTHMFP, as "the concentration of THMs formed in the water buffered at pH 7.0, containing an excess of the free chlorine with a chlorine residual of 1-5 mg/L after being for 168 hrs at 25^0 C."

2.2.2 Disinfection By-Products Control

Studies have reported that the DBPs formation depends on factors such as the precursor concentration, chlorine dose, chlorination pH, temperature, contact time and bromide ion concentration (Health Canada, 1995). The most important chemical variable in chlorination DBPs formation is the pH.

Singer (1994) has suggested the following strategies for controlling the formation of halogenated disinfection by-products (DBPs).

- Source control
- Precursor removal

Enhanced coagulation

Granular activated carbon (GAC) adsorption

Membrane filtration

- Alternative oxidants and disinfectants

Combined chlorine (monochloramine)

Ozone

Chlorine dioxide

Advanced Oxidation Processes (AOPs)

UV light

- Air stripping

Precursor removal is one of the important measures for controlling the DBPs formation (Oxenford, 1996). Natural organic matter, more commonly known as the total organic carbon (TOC) or dissolved organic carbon (DOC), is believed to be the major precursor to the DBPs formation. Precursor removal actions can be classified into three different groups: control at the source, physical/chemical removal, and the oxidation/transformation. Control of the source involves managing the inputs into the watershed. Coagulation, adsorption, and membrane separation are the steps involved in the physical/chemical removal. In oxidation/transformation method, processes that change the form of NOM is employed.

(a) Control at the Source: There are certain parameters that can be used in a water supply management program to reduce the precursors (Cooke and Carlson, 1989).

(b) Physical / Chemical Removal: For removal of the natural organic matter (NOM), three methods have been suggested: membranes, enhanced coagulation, and adsorption.

Using the membranes (Oxenford, 1996), a high percentage removal is possible (up to 95%). Enhanced coagulation is a much-preferred strategy for the water supplies already using conventional coagulation. (AWWA, 1994a). Adsorption of the NOM can be achieved using the granulated activated carbon (GAC), powdered activated carbon (PAC), or other adsorbing materials (Benjamin et al., 1993).

(c) Oxidation / Transformation: Oxidation can remove the NOM by direct oxidation to carbon dioxide, improving coagulation, or by increasing the biodegradability of the NOM. Direct oxidation of the NOM using most oxidants is relatively minor, on the order of 10 to 20% (Oxenford, 1996). Overall NOM removal can be enhanced by the oxidants, by increasing the removals attained by coagulation.

2.2.3 Removal of Disinfection By-Products

Even after their formation, disinfection by-products (DBPs) removal is possible by subsequent treatment processes. United States Environmental Protection Agency (U. S. EPA, 1981) has proposed air stripping and GAC as techniques for THMs removal. With the discovery of haloacetic acids (HAAs), the air stripping technique has become less attractive, and GAC has low capacities for the THMs, especially chloroform (Oxenford, 1996). Study by Hoehn (1994) and Knocke and Iatrou (1993) examined the removal of chlorite, linked with chlorine dioxide. Research has shown that the use of ferrous iron is the most effective technique for chlorite reduction, the majority of ozonation DBPs are biodegradable; however, bromate is not (Oxenford, 1996). Study by Amy and Siddiqui (1994b) has examined the bromate removal. Research by Jacangelo et al., (1995) has reported that the combined ultrafiltration (UF) -powdered

activated carbon (PAC) treatment can be effective for the DBPs precursor removal, depending on the level of removal desired. They also observed, "PAC addition did not impair the permeability of the UF membrane and, in one case, appeared to retard membrane fouling. DBPs precursor removals increased with increasing PAC dosages." Although removal methods after their formation are available, priority should be to minimise the formation of the DBPs in the first place through the precursor removal, manipulation of the water quality parameters, and minimising the use of oxidants while still achieving adequate disinfection (Oxenford, 1996).

2.3 THMs Control

The best available technologies for control of THMs (U. S. EPA, 1981) are:

- Use alternative oxidants and disinfectants
- Remove the precursors by coagulation and settling

Other removal strategies for the THMs precursors include but are not limited to, the granulated activated carbon (GAC) adsorption and membrane technologies such as nanofiltration (NF) (McPhee, 1992).

2.4 Environmental Risk Assessment

With tremendous development in the field of environmental engineering, risk assessment has fast emerged as an integral part of any environmental management planning. Engineering projects, hazardous waste sites and various industrial activities may put the public to considerable risk because of the adverse health and environmental

consequences. Environmental risk assessment has facilitated the scientists and engineers to conceptualise the evaluation of the potential health and environmental hazards.

The various stages of a risk assessment are shown in Figure 2.4.1. The first stage of risk assessment is hazard identification. Hazard identification is defined as a qualitative evaluation of whether the human exposure to an agent has the capability to cause adverse health effects. The second stage of risk assessment process is identifying the actual or potential routes of exposure and type of exposure. The exposure assessment process can be described as an analysis of contaminant release to the environment. The dose-response assessment consists of ascertaining the link between the dose of a chemical and the incidence of the adverse effect caused by the chemical.

The risk characterisation process involves the evaluation of the incidence and the extent of damage to human health and the environment that may be caused by the contaminant exposure. The process systematically characterises the carcinogenic risk, non-carcinogenic risk, environmental risk, and risks to the public welfare. There are various ways to provide the quantitative estimates of carcinogenic risk. First, by estimating the unit cancer risk. Assuming low-dose linearity, this estimate expresses the excess lifetime risk in terms of continuous exposure over an average lifetime corresponding to a particular carcinogen concentration expressed in units of mg/kg/day by ingestion or $\mu g/m^3$ in air.

Second, the estimates can be made of the dose corresponding to a given level of risk. Third, the risk can be stated in terms of excess individual lifetime risk. Fourth, the risk can be correlated to the excess incidence of cancer per annum in the population exposed (Santos, 1987).

Risk characterisation, as described by both the National Academy of Sciences and the U. S. EPA is the estimation of human health risk due to the injurious (i.e., toxic or carcinogenic) compounds or organisms (Naugle and Pierson, 1991). According to this approach, the four components of risk assessment methodology i.e. hazard identification, exposure assessment, dose-response, and risk characterisation are further divided into ten elements: source factors, contaminant concentration, exposure duration/setting, exposure, dosimetry factors, dose, response factor, lifetime individual risk, exposed population, and risk to exposed population. Each element is based on a term in a predictive risk equation. Parameters such as exposure, dose, lifetime individual risk, and risk to exposed populations can be computed independently within the equation itself.

Basic risk may be defined, in the light of the present study as, "the probability that an individual will contract cancer at some time in his/her lifetime based on a daily ingestion of two litres of drinking water or a daily showering of ten minutes, fifteen minutes, or twenty minutes respectively. The risk referred to in this thesis is the risk of getting the disease and not the risk of dying from the disease.

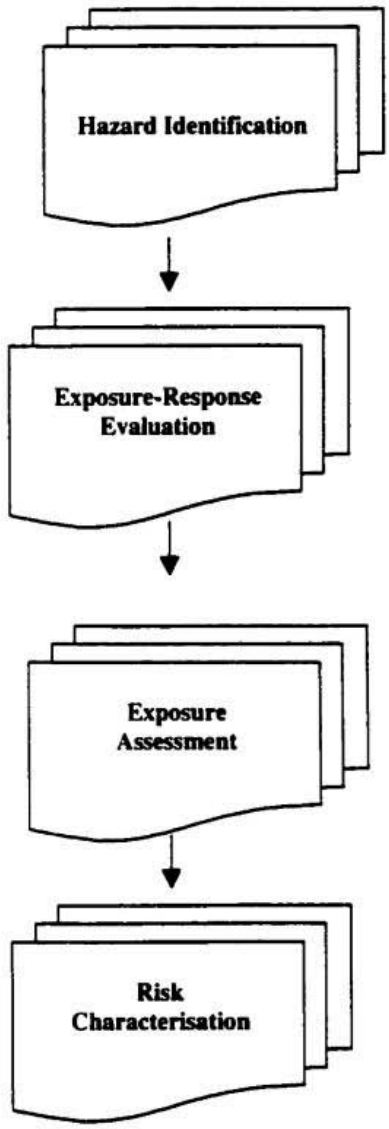

Figure 2.4.1 Fundamental Elements of Risk Assessment: Process Model and Components

Reviewing various studies, Naugle and Pierson (1991) have suggested the formulation of a simple predictive risk equation, which consists of most key elements. The four general equations constituting the predictive risk equation are shown in Figure 2.4.2. Various elements explained in Figure 2.4.2 are as follows:

Column B (Source Factors) is the starting point for the predictive risk equation.

Column C (Contaminant Concentration) accounts for the numerical data of the exposure concentrations for each contaminant under consideration.

Column D (Exposure Duration and Setting) of the framework is the interface between the specification of each environmental setting subject to exposure and the determination of the time spent in that particular environment.

Column E (Exposure) is established as the product of the concentration of the contaminant (an individual is exposed to in a specific setting), and the time spent by that individual in that microenvironment.

Column G (Dose) of the framework is expressed as the product of exposure estimates and the various factors. The dosimetry factors are the contact rate, absorption rate, average body weight, average lifetime etc. Dose is stated as the contaminant mass per kilogram of the body weight per weight day (mg/kg-d).

Column H of the framework (Response Factor) gives the measure of the response of an individual exposed to a certain dose of a substance. The dose-response relationship for carcinogens is described as a potency factor which is the 95 percent upper confidence limit of the human excess lifetime exposure to the carcinogens expressed in $(mg/kg\text{-}day)^{-1}$ (Naugle and Pierson, 1991). Human Health Assessment Group (HHAG) of U. S. EPA has stated the potency factors for many carcinogens. The idea of a

reference dose (RfD) has been preferred to the potency factors by the U. S. EPA in analysing the noncarcinogenic health effects (U. S. EPA, 1988).

The RfD is defined as an estimate of dose, with uncertainty having an order of the magnitude, of a daily exposure to the human population, including the sensitive subgroups, that is likely to be without the appreciable risk of harmful effects during a lifetime (U. S. EPA, 1987). The RfD is expressed in the form of Equation 2.1 (Naugle and Pierson, 1991):

$$RfD = NOAEL / (UF \times MF) \quad \textbf{(2.1)}$$

where

NOAEL is the no observed adverse effect level, using either the human or animal data

UF is the uncertainty factors that reflect the uncertainty in various types of data used to estimate the RfDs

MF is the modifying factors, which reflect the qualitative professional judgements regarding the scientific uncertainties of the entire database of the chemical.

Carcinogenic risks are expressed as the product of a lifetime average daily dose (Column G) which is in units of milligrams per kilogram of the body weight per day, and a potency factor (Column H), which is in units of the lifetime risk per unit of the exposure, $(mg/kg\text{-}day)^{-1}$.

Predictive Risk Equation (A)	Source Factors (B)	Contaminant Concentration (C)	Exposure Duration/ Setting (D)	Exposure (E)	Dosimetry Factors (F)	Dose (G)	Response Factor (H)	Lifetime Individual Risk (I)	Exposed Population (J)	Estimated Cancer Cases in a Community (K)
Qualitative Information	Hazard Identification →									
Quantitative Information	Exposure Assessment →					Dose Response →		Risk Characterisation →		
Qualitative or Quantitative Analysis	Risk Assessment →									

Predictive Risk Equation (A)
Source Factors (B)
Contaminant Concentration (C) × Exposure Duration/Setting (D) = **Exposure** (E) (Total Exposure Over a Lifetime)
Exposure (E) × Dosimetry Factors (F) = **Dose** (G) (Average Dose Per Day Over a Lifetime)
Dose (G) × Response Factor (H) = **Lifetime Individual Risk** (I) (Individual Risk Over a Lifetime or Probability / Lifetime)
Lifetime Individual Risk (I) × Exposed Population (J) = **Estimated Cancer Cases in a Community Over a Lifetime**(K)

Source: Naugle and Pierson (1991).

Figure 2.4.2 Risk Characterisation Framework

Column I (Lifetime Individual Risk) of the framework is defined as the magnitude of the lifetime excess risks of cancer for an individual caused by a given lifetime exposure. It can be expressed as

Lifetime Individual Risk = Dose × Response Factor (Potency or Slope Factor) **(2.2)**

Column J (Exposed Population) in the framework is the affected subpopulations considered in a risk analysis exercise.

Column K (Estimated Cancer Cases in a Community) of the framework is defined as the "expected or observed" number of cases in the population under consideration. It can be expressed as

Estimated Cancer Cases in a Community = Lifetime Individual Risk × Exposed Population **(2.3)**

2.5 Health Risks of Chlorinated Disinfection By-Products in Drinking Water

Many epidemiological studies conducted recently (Health Canada, 1995) have indicated that the people consuming drinking water with high levels of chlorination by-products had an increase in the risk of bladder cancer. Similar studies in chlorination by-products have found a possible increase in the risks of colon and rectal cancers, adverse reproductive and developmental effects like the increased spontaneous abortion rates and foetal anomalies (Mills et al., 1998). Chlorination by-products are formed when water is subjected to chlorination during treatment to check the occurrence of microbial disease. Chlorination is considered to be the most effective disinfectant method as of now. So, while contemplating to adopt the mitigation measures for human health risks from the by-products, it should be kept in mind that those initiatives should

not compromise the microbial disinfection. The acceptable level of the important disinfection by-product, the trihalomethanes (THMs) is 100 µg/L in Canada. Disinfectants like the chloramine and ozone also form the by-products. However, the detailed toxicological information about these compounds is not available at this stage.

Detailed toxicological assessment of chlorination by-products to date has been limited. This is due to the fact that many by-products are involved and different modes of action seemingly result in the carcinogenesis (Mills et al., 1998). Most animal studies undertaken so far have emphasised the by-products having the greatest human exposure or toxic effects. Table 2.5.1 lists the animal studies on exposure to the chlorination by-products. As a matter of fact, the type of tumour mostly observed was liver cancer. This was a result of THMs and haloacetates exposure to mice and rats. Humans never seemed to have been induced with liver cancer as a result of exposure to the chlorinated by-products (Mills et al., 1998). Less significant THMs like the bromodichloro-methane cause colon cancer in mice. These observations are significant in the light of the association of colon cancer with exposure to high levels of THMs in some epidemiological studies. Despite the fact that the data from the animal studies have established that the exposure to by-products at higher concentration levels cause cancer in the laboratory animals, some issues still need to be addressed. There is no indication by any toxicological study that a single chlorinated by-product study seemed to be carcinogenic at the human levels of exposure. Besides, evidence for carcinogenesis for toxicological study is different from that of the epidemiological studies. In animals, the association between by-product exposure and liver cancer could be commonly found, whereas in humans, the association was with bladder cancer.

These variations in information reiterate the need for re-estimating the present cancer risk determined from animal studies. It has been established that summation of

Table 2.5.1 Cancer and Exposure to Chlorination By-Products: Animal Studies[a]

Chlorination By-Product/ Author (year)	Study Animal	Outcome
TRIHALOMETHANES **Chloroform** National Cancer Institute (1976) Jorgenson (1985)	 Mice Rats	 Liver tumours Kidney tumours
Bromodichloromethane National Toxicology Program (1987) National Toxicology Program (1987)	Rats Mice	Colon and kidney tumours Liver and kidney tumours
Chlorodibromomethane National Toxicology Program (1984)	Mice	Liver tumours
Bromoform National Toxicology Program (1989)	Rats	Colon tumours
HALOACETIC ACIDS **Dichloroacetic acid (DCA)** Herren-Freund (1987), Bull (1990), DeAngelo (1991), Daniel (1992), Pereira (1996) DeAngelo (1996)	 Mice Rats	 Liver tumours Liver tumours
Trichloroacetic acid (TCA) Herren-Freund (1987), Bull (1990), Pereira (1996)	Mice	Liver tumours
Dibromoacetic acid So (1995)	Rats	Aberrant crypt foci in colon
HALOACETONITRILES **Brominated haloacetonitriles** Bull (1985) [a] Unpublished studies noted in italics	Mice	Skin tumours

Source: Mills et al., 1998

the toxicological hazard values from individual by-products do not appropriately reflect the risks from chlorinated drinking water. Evidence of adverse health effects could not be strongly established from the initial toxicological studies of mixtures of the by-

products. Extrapolation of this to humans in part is not feasible because the by-product mixtures presently available in treated water exhibit diversity (Mills et al., 1998). A number of epidemiological studies have been conducted to investigate the possible association between cancer and water chlorination by-products. The most common sites of cancer that are found to have association with exposure to chlorinated water are bladder, colon and rectum (Mills et al., 1998).

Table 2.5.2 presents a comprehensive information about nine epidemiological studies ascertaining the risk of colon cancer as a result of chlorinated water by-products exposure. The table reports various relative risks. Relative risks are interpreted as "statistically significant" if their associated 95% confidence intervals (CI) do not include 1.0 and "not statistically significant" if they do so. A result greater than 1.0 is interpreted as a positive risk; less than 1.0, as a negative risk. For simplicity, a single relative risk to summarise a rich and complex body of data is presented (Mills et al., 1998). Two out of seven earlier studies indicated considerably positive result. Two most recent studies (Marrett and King, 1995; and Hildesheim et al., 1998), both of which involved case-control examinations of newly diagnosed disease, showed inconsistent findings. The study conducted by Marrett and King (1995) considered over 5000 people in Ontario. The result found approximately 950 having bladder, colon or rectal cancer. The study had age-and sex-match controls from the general population. A survey of water treatment facility history and estimation of THMs were used to determine THMs levels back to 1950 in regional water supplies. Individuals being exposed to THMs, greater than or equal to 50 μg/L for a period of more than 35 years, had a likelihood of 1.5 times more than of the controls identified from the general

population. to develop colon cancer. A dose-response relationship was established by the data that was valid after taking into consideration potential confounding factors like nutrient, caloric, and fibre intake. The study by Hildesheim and group of researchers (1998) in Iowa identified 685 colon cancer victims. 2400 individuals comprised the control group who were matched for age, sex and had induced "one of five other types

Table 2.5.2 Overview of Epidemiological Studies Related to Colon Cancer and Chlorination By-Products Exposure

Author (year)	Exposure measure	Relative risk (CI)[a]	Association	Dose-response	Duration response	Cancer outcome measure
Hildesheim (1998)	THMs	1.13 (0.7-1.8)	Positive (NS)	No	No	Incidence
Marrett (1995)	THMs	1.5 (1.0-2.2)	Positive (NS)	Yes	N/A	Incidence
Young (1987)	THMs	0.73 (0.44-1.21)	Negative (NS)	No	No	Incidence
Zierler (1986)	Chlorine Vs chloramine[b]	0.89 (0.86-0.93)	Negative *	N/A	N/A	Mortality
Cragle (1985)	Chlorinated water	3.36 (2.41-4.61)	Positive*	N/A	Yes	Incidence
Gottileb (1982)	Surface Vs ground[b]	1.01 (N/A)	Positive (NS)	N/A	N/A	Mortality
Wilkins (1981)	Surface Vs well	0.89 (0.57-1.43)	Negative (NS)	N/A	N/A	Mortality
Brenniman (1980)	Chlorinated groundwater[b]	1.11 (N/A)	Positive (NS)	N/A	N/A	Mortality
Alvanja (1978)	Chlorinated water[b]	1.61 (N/A)	Positive*	N/A	N/A	Mortality

a 95% confidence intervals (CI) in brackets. When only stratified results were reported, the relative risk reported here corresponds to the longest exposure period and greatest exposure.
b Exposure derived from the residence recorded on the death certificate
* Statistically significant, $p < 0.05$
NS = Not statistically significant
N/A = Not applicable/ available

Source: Mills et al., 1998

of cancer." Chlorinated surface water exposure and THMs exposure were evaluated for full lifetime of all the subjects and effects of confounding variables were adjusted for. Escalated risk of colon cancer was not indicated by the Hildesheim et al. study (1998). "While the methods to estimate THMs exposure were somewhat more precise in the Marrett and King study (1995), it is unlikely that this would explain the absence of an association in the Hildesheim study (1998). These contradictory findings are not currently understood. They may be due to chance, to water quality differences between Ontario and Iowa or to other factors. In conclusion, the evidence for an increased risk of colon cancer from exposure to chlorination by-products is inconclusive," Cantor (1998) suggested.

Table 2.5.3 summarises eight studies conducted to examine the possible association between rectal cancer and exposure to chlorinated by-products. Two of the six earliest studies indicated a statistically significant increase in risk of cancer associated with exposure to chlorinated by-products. "Once again, the two most recent studies had inconsistent results: the Marrett and King study showed no association, whereas the Hildesheim study showed a statistically significant positive association and a positive duration-response relationship. In summary, the evidence for an association between rectal cancer and chlorinated by-products is also inconclusive. However, in light of the positive finding in the meta-analysis, the evidence is somewhat stronger for rectal cancer that colon cancer," comments Cantor (1998).

The evidence of an association between exposure to chlorination by-products and bladder cancer is more consistent than it is for rectal and colon cancers (Mills et al., 1998). Table 2.5.4 presents an overview of 11 studies conducted to address the

association between bladder cancer and exposure to THMs. Three of the seven studies published prior to 1990 were statistically significant (Mills et al., 1998). The study by King and Marrett (1996) suggested that exposure to THMs concentration of approximately 50 µg/L or greater for a time period of 35 years or more, yielded a relative risk of 1.61.

Table 2.5.3 Overview of Epidemiological Studies Related to Rectal Cancer and Chlorination By-Products Exposure

Author (year)	Exposure measure	Relative risk (CI)[a]	Association	Dose-response	Duration response	Cancer outcome measure
Hildesheim (1998)	THMs	1.7 (1.1-2.6)	Positive*	Yes	Yes	Incidence
Marrett (1995)	THMs	0.99 (0.5-1.4)	Negative (NS)	No	No	Incidence
Zeiler (1986)	Chlorinated water[b]	0.96 (0.89-1.04)	Negative (NS)	N/A	N/A	Mortality
Gottileb (1982)	Surface Vs ground[b]	1.79 (N/A)	Positive*	N/A	N/A	Mortality
Wilkins (1981)	Surface Vs well	1.42 (0.70-3.16)	Positive (NS)	N/A	N/A	Mortality
Young (1981)	Chlorine dose	1.39 (0.67-2.86)	Positive (NS)	N/A	N/A	Mortality
Brenniman (1980)	Chlorinated groundwater[b]	1.22 (N/A)	Positive (NS)	N/A	N/A	Mortality
Alvanja (1978)	Chlorinated water[b]	1.93 (N/A)	Positive*	N/A	N/A	Mortality

a 95% confidence intervals (CI) in brackets. When only stratified results were reported, the relative risk reported here corresponds to the longest exposure period and greatest exposure.
b Exposure derived from the residence recorded on the death certificate
* Statistically significant, $p < 0.05$
NS = Not statistically significant
N/A = Not applicable/available

Source: Mills et al., 1998

When the exposure period extended to more than 20 years, excess risk was reported. In addition, the researchers observed increases of risk with time.

U. S. EPA has summarised the possible health effects of various contaminants that may be present in drinking water. The document covers an array of organic, inorganic, radionuclides contaminants and contains information on their sources and maximum contaminant levels (MQL). An overview of this document is presented in Appendix 2.

Trihalomethanes is an important chlorinated disinfection by-product found in drinking water. Direct association could not be established between THMs concentrations in drinking water and cancers at various sites. The only exception was a notable increase in pancreatic cancer in white males (Carlo et al., 1980), rectum cancer

Table 2.5.4 Overview of Epidemiological Studies Related to Bladder Cancer and Chlorination By-Products Exposure

Author (year)	Exposure measure	Relative risk (CI)[a]	Association	Dose-response	Duration response	Cancer outcome measure
Cantor (1998)	THMs	1.5 (0.9-2.6)	Positive (NS)	Yes	Yes	Incidence
Freedman (1997)	Municipal water	1.4 (0.7-2.9)	Positive (NS)	N/A	No	Incidence
King (1996)	THMs	1.6 (1.08-2.46)	Positive*	Yes	Yes	Incidence
McGeehin (1993)	THMs	1.8 (1.1-2.9)	Positive*	No	Yes	Incidence
Zierler (1988)	Chlorine Vs chloramine	1.4 (1.20-2.10)	Positive*	N/A	N/A	Mortality
Cantor (1987)	Chlorinated surface water	1.8 (N/A)	Positive*	N/A	Yes	Incidence
Gottileb (1982)	Surface Vs groundwater[b]	1.2 (N/A)	Positive (NS)	N/A	N/A	Mortality
Young (1981)	Chlorine dose[b]	1.04 (0.43-2.50)	Positive (NS)	N/A	N/A	Mortality
Wilkins (1981)	Surface Vs well water	2.2 (0.71-9.39)	Positive (NS)	N/A	N/A	Mortality Incidence
	Males	1.8 (0.84-4.75)	Positive (NS)	N/A	N/A	Incidence
	Females	1.6 (0.54-6.32)	Positive (NS)	N/A	N/A	
Brenniman (1980)	Chlorinated groundwater[b]	0.98 (N/A)	Negative (NS)	N/A	N/A	Mortality
Alvanja (1978)	Chlorinated water[b]	1.69 (N/A)	Positive*	N/A	N/A	Mortality

a 95% confidence intervals (CI) in brackets. When only stratified results were reported, the relative risk reported here corresponds to the longest exposure period and greatest exposure.
b Exposure derived from the residence recorded on the death certificate
* Statistically significant, $p < 0.05$
NS = Not statistically significant
N/A = Not applicable/available

Source: Mills et al., 1998

in males only (Tuthill and Moore, 1980), and stomach cancer in both sexes (Tuthill and Moore, 1980). Tuthill and Moore (1980) were able to identify the association between THMs concentrations and stomach and rectal cancer, although it was not apparent when the population migration patterns were taken into consideration.

To summarise, it may be said, in many epidemiological studies, excesses of cancer at some sites have been correlated with the extent of chlorination of, or levels of THMs in drinking water; however, owing to the difficulty in controlling for the potential confounding factors, such as the population migration, and the lack of consistency of reported results, it is difficult to draw meaningful conclusions about the casualty. Available data are at least consistent with the hypothesis that the ingestion of chlorinated drinking waters, if not THMs specifically, may be causally related to the cancers of the bladder and colon (Health Canada, 1993). The comparison of the actual risk and other risk, people are exposed to, is described in the Conclusion (Chapter 7).

Chapter 3

Research Methodology

3.1 Experimental Methodology

Laboratory experiments were an integral part of this research. The main objective of the laboratory work was to analyse the drinking water samples collected from various communities of this province. The drinking water samples were analysed to determine the concentration levels of trihalomethanes. The concentration levels of all the four components of trihalomethanes namely chloroform, bromoform, chloro-dibromomethane, dichloro-bromomethane were determined.

Three representative sampling sites within the province of Newfoundland, Canada were chosen for the sample collection. These communities are St. John's, Clarenville, and Shoal Harbour (Figure 3.1). Individual communities with the actual sampling locations are shown in Figures 3.2, 3.3, and 3.4 respectively. Clarenville and Shoal Harbour were selected for their known high THMs levels and St. John's was selected for comparison purposes. The water samples were collected from drinking water tap in households, commercial establishments, and university.

The sample collection period was divided into two stages. The first stage of the sample collection took place during the month of July 1998, which is representative of the summer months. The second stage of the sample collection was undertaken during the month of October-November, 1998, which is representative of the winter months. In both the stages, the samples were collected from all the three communities. The sampling program schedule is listed in Table 3.1

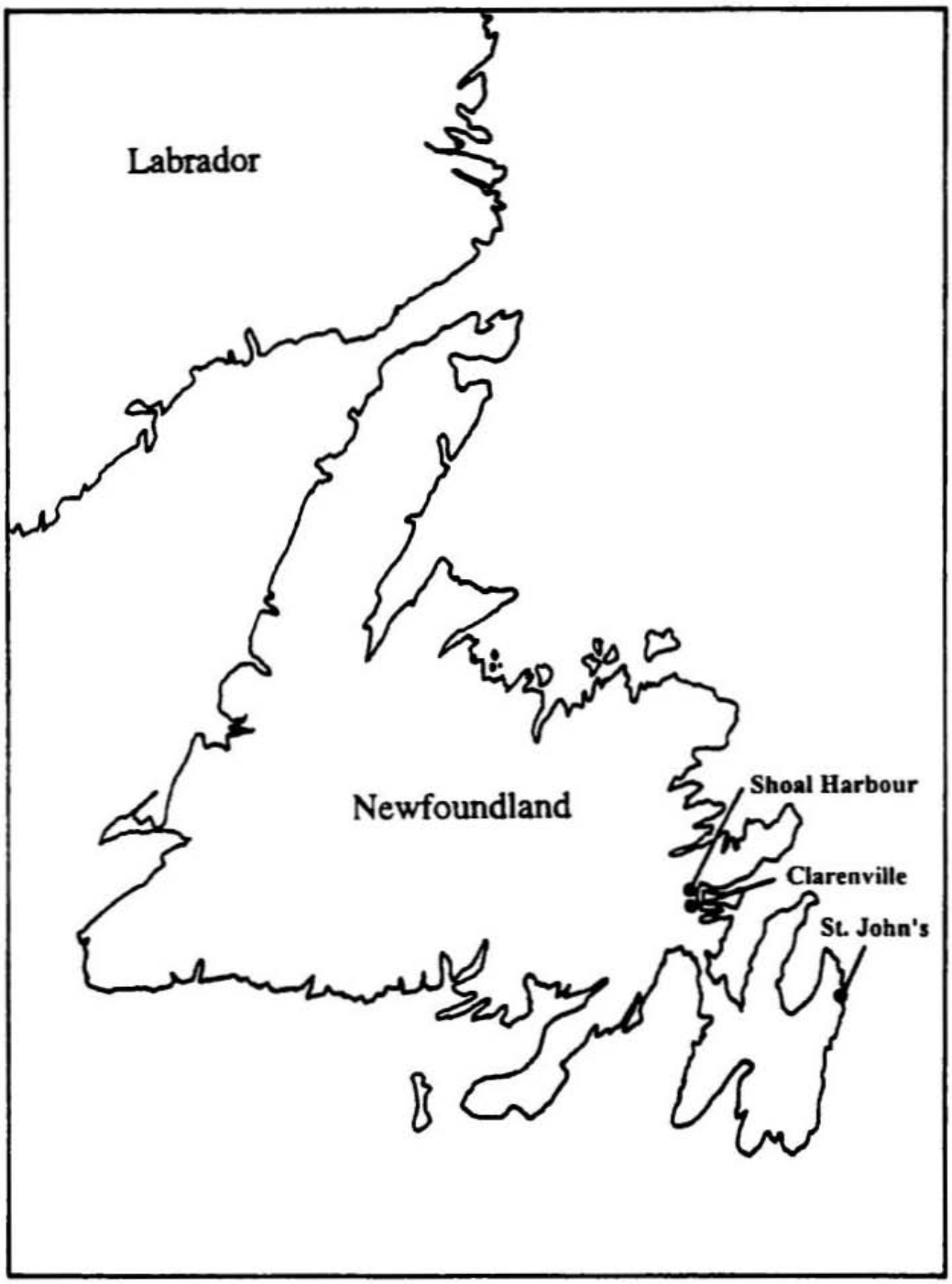

Figure 3.1 Three Sampling Locations, Newfoundland Study, 1998

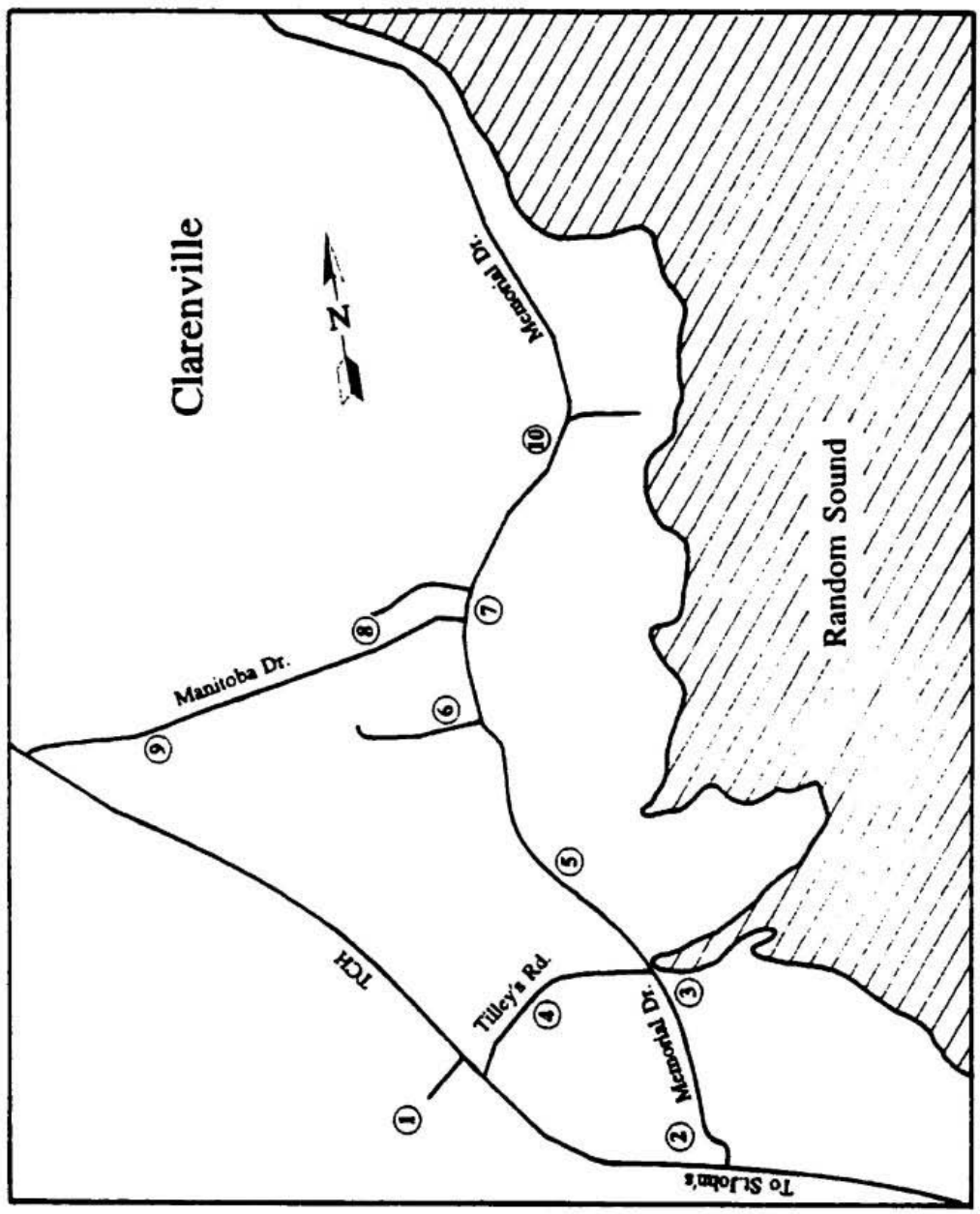

Figure 3.2 Sampling Sites in Clarenville, Newfoundland Study, 1998

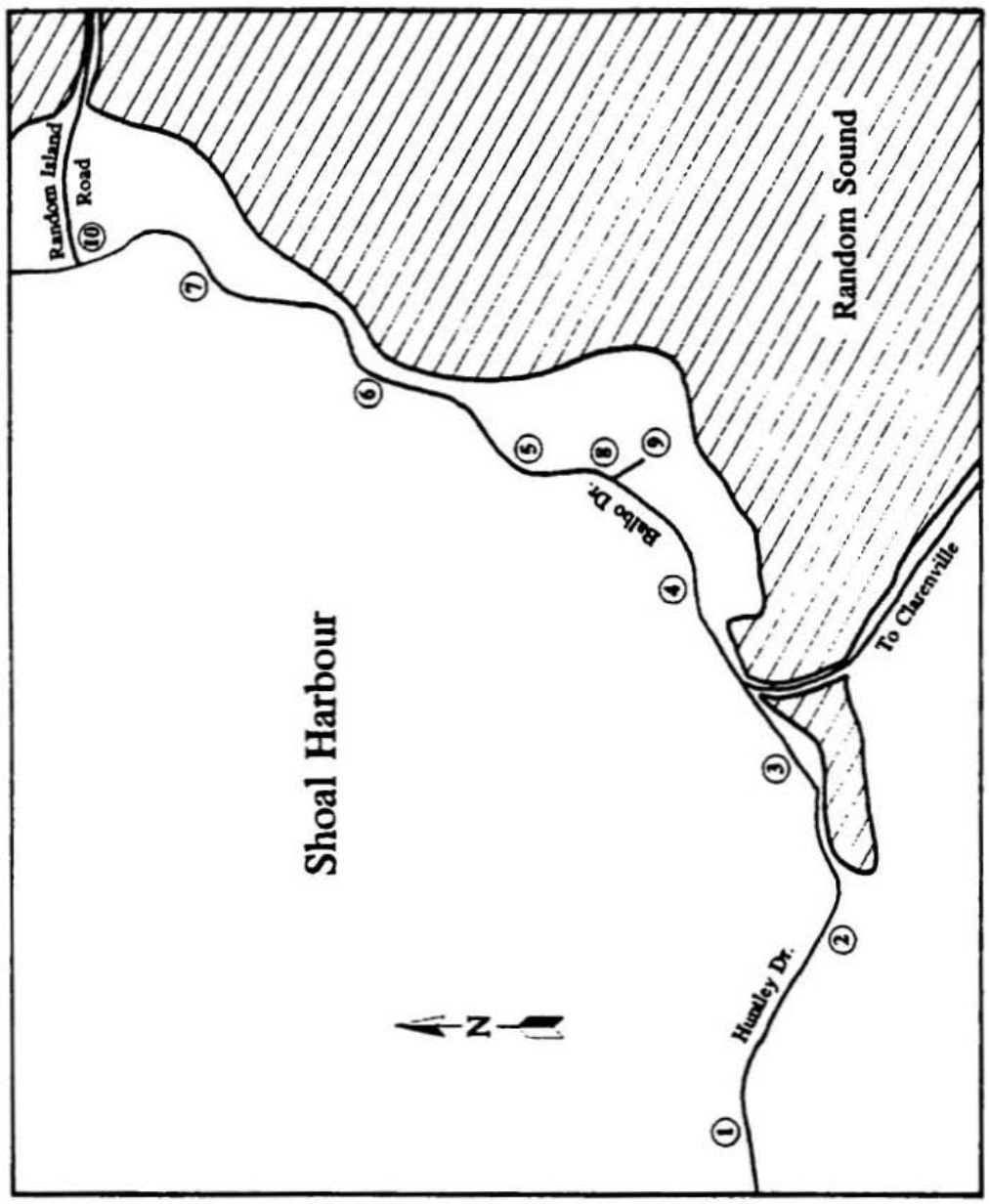

Figure 3.3 Sampling Sites in Shoal Harbour, Newfoundland Study, 1998

Table 3.1 Sampling Program Schedules

Communities	Number of Samples	
	Summer (July 1998)	Winter (Oct-Nov 1998)
St. John's	5	5
Clarenville	10	10
Shoal Harbour	10	10

The plan was to have similar number of samples from all the three sites. However, due to limited resources, a maximum of fifty samples were analysed, and since the THMs concentration in St. John's was much less than in other communities, less number of samples were collected from St. John's.

Samples were collected in decontaminated glass vials. First, the drinking water tap was allowed to run for 5-6 minutes. This was done in order to remove the accumulated metals in the pipe, if any. Then the vial was held right under the flowing tap to collect water. The vial was filled up with water fullest to its brim. No headspace was kept in the vial. After the vial was filled up, it was kept inside a portable ice cooler. The collection exercise was repeated. After the completion of the sampling program, the containers were taken to Environmental Quality Laboratory (EQL) in Newfoundland. Between the period of time of sample collection and transportation of samples to EQL, the sample vials were preserved in refrigerator set at a constant temperature of $4^{\circ}C$.

After the samples were delivered to the EQL, they were again preserved in coolers at a constant temperature of $4^{\circ}C$. All the collected samples were analysed in

the laboratory (EQL) within fourteen days of sample collection as per the usual practice.

3.2 Experimental Procedures

The experimental procedure followed was in accordance with the guidelines prepared by the Environmental Quality Laboratory (EQL) titled "Trihalomethanes in Water"; Method Number M230.01. (EQL, 1998). The systematic procedure compiled in the document is presented in the following section.

The method for trihalomethanes concentration determination in water in the current study employed solid phase micro-extraction / capillary column gas chromatography / mass spectrometer detector (SPME/GC/MS). The method detection limit was 1 µg/L for trihalomethanes. The operating range was 1- 500 µg/L. Some of the samples had contaminant concentrations more than 500 µg/L. These concentrations were measured by using dilution technique.

The water sample was first extracted into the SPME fibre. Subsequently, the desorbed material obtained from the fibre was then analysed with the help of GC/MS. Samples were collected in decontaminated glass vials fitted with teflon lined covers. For the analysis, 2 mL of sample was used. Samples were also analysed within 14 days of collection. Standard laboratory safety measures were practised to minimise exposure to the chemicals, reagents. This was important, as sufficient toxicity information was unavailable. Material safety data sheet (MSDS) of all chemicals necessary for these assignments were readily made available for reference.

Equipment used were Hamilton gas tight syringes, 100 µm polydimethylsiloxane solid phase microextraction fibre, Varian 8200 AutoSampler equipped with SPME accessory, Varian 3400 Gas Chromatography equipped with a Saturn II Mass Spectrometer, Dell 166 Gxi Computer and printer installed with Varian GC/MS software.

The list of reagents used included (a) Methanol- pesticide grade, (b) 19 Mohm deionized water, (c) Supelco EPA 524 Internal Standard Mix. Catalog No.: 4-8948, 2000 µg/mL of flurobenzene and 1,2-dichlorobenzene-d_4 in methanol, (d) Supelco Trihalomethanes Calibration Mix. Catalog No.: 4-8140, 200 µg/mL each of bromodichloromethane, dibromochloromethane, chloroform, bromoform, and (e) Sodium sulphate - TRACEPUR, anhydrous, oven dried at 400°C for 4 hours.

For standards preparation, standard operating procedure (SOP 04.1) was followed. The internal standard was prepared through the following stages. (a) 1.0 mL of Supelco EPA 524 Internal Standard Mix spiking solution was pipetted into a precleaned 50 mL volumetric flask with the help of Hamilton 1001 1000 µL gas tight syringe. It was made to the mark with methanol. (b) This solution was diluted with 1mL in 10mL for working internal standard solution. The solution thus formed was the spiking solution having the following composition, 4.0 µg/mL florobenzene, and 4.0 µg/mL 1,2-dichlorobenzene-d_4. The solution was stored at 4°C. This solution was replaced at the end of each month.

The standard solutions were prepared through the following stages. (a) 1.0 mL of the 200 µg/mL Supelco Trihalomethanes Calibration Mix was pippeted into a precleaned 5 mL volumetric flask with the help of Hamilton 1001 1000 µL gas tight

syringe. It was made to the mark with methanol. (b) The solution thus formed was the standard solution having the following composition. 40.0 µg/mL each of bromodichloromethane, dibromochloromethane, chloroform, and bromoform. (c) 1.0 mL of the 40.0 µg/mL standard solution was pippeted into a precleaned 10mL volumetric flask using the Hamilton 1001 1000 µL gas tight syringe. It was then made up to the mark with methanol. The solution thus formed was the standard solution with the following composition. 4.0 µg/mL each of bromodichloromethane, dibromochloromethane, chloroform, and bromoform. New standards were checked against standard reference materials. The standard solutions were stored at 4°C. The standards were replaced after every six months.

For preparing the sample the following steps were undertaken. (a) Several small samples of sodium sulphate were weighed out. Each of which weighed approximately about 0.28 g of sodium sulphate. Each portion was to be added to each sample vial of water. (b) 1.2 mL of water sample was put into a vial with the help of disposable glass pipette. This volume measurement was done by comparing against other vials that already had exactly 1.20 mL of water. These volume measurements were placed in the vials using Hamilton 1001 1000 µL gas tight syringe. 10 µL of internal standard was pipetted below the water level with the Hamilton 1710 100 µL gas tight syringe. Then the previously weighed portion of 0.28 g (approximately) sodium sulphate was added to this solution. The vial was then tightly capped. (b) In the case of standard solutions, 1.2 mL of water was put in a vial. Then the appropriate volume of 40.0 µg/mL or 4.0 µg/mL standard solution was pipetted below the water level with Hamilton 701N 10µL gas tight syringe. Following that, 10 µL of internal standard was pipetted below the

water level with Hamilton 1710 100 μL gas tight syringe. Thereafter, approximately 0.28g of sodium sulphate was added to the solution. The vial was capped tightly. The method did not require any sample clean up.

The instrument was tuned according to the instrument instruction manual. The system was calibrated before analysis. Then the instrument was set up accordingly. The software Saturn was used for the data acquisition. First the Analysis List was created. A unique data file name was specified and samples information, if any was entered for each entry in the Analysis List. Each entry was highlighted on individual basis in order to edit sample information. The GC and MS Methods appropriate for this study was set at the initial stage. The entries were then checked. After the completion of the Analysis List, the validity of entry list was checked. The next operation was data acquisition. In order to make the instrument automatically ready for data acquisition, the Analysis Program was used. The acquisition of data files of the selected entry in ascending order occurred when the acquisition started. At this point, the Autosampler run was initiated.

The method of data retrieval was carried out by studying the four different chromatograms obtained after the data acquisition process was started. Chromatogram number one gave the peak of chloroform and bromodichloromethane. Chromatogram number two gave the peak of chlorodibromomethane. Chromatogram number three gave the peak of bromoform. Chromatogram number four gave the peak of internal standard (dichlorobenzene). After the four chromatograms appeared, the plots were normalised. The respective peak areas were recorded. In the next step, with the help of spreadsheet, calibration curves were plotted for each trihalomethanes. Consequently, by

using mathematical relationship and the peak area values, the respective concentrations of the four THMs compounds were estimated.

For ensuring quality assurance/quality control, the following steps were implemented. (a) A procedural blank was performed daily by using deionized water. Results were rejected if the blanks were greater than the detection limit. (b) A standard reference material was analysed (in duplicate) with each run. ERA THM Reference 3221 was used. Reading from the calibration curve was used to determine the concentration of reference material. As a quality check, results were not considered if the concentrations did not fall within Performance Acceptance Limits. (c) A duplicate analysis was performed at least once in every ten samples. The analysis was repeated if two numbers varied by greater than twenty percent.

3.3 Experimental Data

In this section the data obtained from laboratory analysis are presented. The sample collection period was divided into two stages.

Sampling in Summer

The concentration levels of THMs compounds in drinking water collected from taps in the three communities of Newfoundland are given below. In the following tabulated data, the second set of values in each row indicates the concentration level of the compounds in the samples analysed after *twenty- four hours* of the first analysis. Moreover, the second set of samples was kept in *uncapped vials* inside the laboratory cooler to determine the effects of volatile nature of THMs on water storage. For the purpose of risk analysis, the first set of data is considered only because studying

variation in THMs concentration levels in various scenarios was not the objective of this research. The *total trihalomethanes (TTHMs)* concentration was determined by summing the concentration level of chloroform, bromoform, chloro-dibromomethane, and dichloro-bromomethane. Concentration levels less than 1 μg/L were *ignored* in the estimation because of the precision error of the instrument and this was a common practice adopted by the laboratory. Such values are shown in the tables as < 1 μg/L. A typical sample of laboratory result data sheet is presented in Appendix 1.

The concentration levels of THMs compounds in drinking water collected from taps in St. John's, Newfoundland, are shown in Table 3.2.

Table 3.2 Concentration Levels of THMs in St. John's, Newfoundland (Summer)

Site#	Bromoform (μg/L)	Chloro-Dibromo Methane (μg/L)	Dichloro-Bromo Methane (μg/L)	Chloroform (μg/L)	Total THMs (μg/L)
St. John's # 1	< 1	< 1	< 1	42	42
St. John's # 2	< 1	< 1	< 1	< 1	0
St. John's # 3	< 1	< 1	< 1	66	66
St. John's # 4	< 1	< 1	< 1	40	40
St. John's # 5	< 1	< 1	8	73	81

The concentration levels of THMs compounds in drinking water collected from taps in Clarenville, Newfoundland are shown in Table 3.3.

Table 3.3 Concentration Levels of THMs in Clarenville, Newfoundland (Summer)

Site#	Bromoform (μg/L)	Chloro Dibromo Methane (μg/L)	Dichloro Bromo Methane (μg/L)	Chloroform (μg/L)	Total THMs (μg/L)
Claren# 1	< 1 , < 1	< 1 , < 1	6 , 5	375 , 279	381
Claren# 2	< 1 , < 1	< 1 , < 1	5 , < 1	473 , 240	478
Claren# 3	< 1 , < 1	< 1 , < 1	6 , 6	480 , 325	486
Claren# 4	< 1 , < 1	< 1 , < 1	6 , 5	508 , 259	514
Claren# 5	< 1 , < 1	< 1 , < 1	6 , 5	445 , 323	451
Claren# 6	< 1	< 1	6	456	462
Claren# 7	< 1 , < 1	< 1 , <1	7 , 5	512 , 290	519
Claren# 8	< 1 , < 1	< 1 , < 1	5 , < 1	476 , 239	481
Claren# 9	< 1 , < 1	< 1 , < 1	5 , 5	459 , 270	464
Claren# 10	< 1 , < 1	< 1 , < 1	5 , < 1	497 , 229	502

The concentration levels of THMs compounds in drinking water collected from taps in Shoal Harbour, Newfoundland are shown in Table 3.4.

This practice of 24 hours uncapped sample was done only for some selected samples to see the effect of volatility although it was not the objective of the study. Therefore this practice was not consistently followed for other samples collected at different times and seasons.

Table 3.4 Concentration Levels of THMs in Shoal Harbour, Newfoundland

(Summer)

Site#	Bromoform (μg/L)	Chloro-Dibromo Methane (μg/L)	Dichloro-Bromo Methane (μg/L)	Chloroform (μg/L)	Total THMs (μg/L)
Shoal # 1	< 1 , < 1	< 1 , < 1	7 , < 1	225 , 112	232
Shoal # 2	< 1 , < 1	< 1 , < 1	9 , 5	330 , 139	339
Shoal # 3	< 1	< 1	6	255	261
Shoal # 4	< 1 , < 1	< 1 , < 1	< 1 , < 1	203 , 78.1	203
Shoal # 5	< 1 , < 1	< 1 , < 1	6 , 5	287 , 111	293
Shoal # 6	< 1 , < 1	< 1 , < 1	6 , < 1	264 , 100	270
Shoal # 7	< 1 , < 1	< 1 , < 1	6 , < 1	267 , 70	273
Shoal # 8	< 1 , < 1	< 1 , < 1	7 , < 1	269 , 90	276
Shoal # 9	< 1 , < 1	< 1 , < 1	6 , 5	234 , 190	240
Shoal # 10	< 1 , < 1	< 1 , < 1	5 , 5	289 , 218	294

Sampling in Winter

The concentration levels of THMs compounds in drinking water collected from taps in St. John's, Newfoundland is shown in Table 3.5.

The concentration levels of THMs compounds in drinking water collected from taps in Clarenville, Newfoundland are shown in Table 3.6.

Table 3.5 Concentration Levels of THMs in St. John's, Newfoundland (Winter)

Site#	Bromoform (μg/L)	Chloro-Dibromo Methane (μg/L)	Dichloro-Bromo Methane (μg/L)	Chloroform (μg/L)	Total THMs (μg/L)
St. John's # 1	<1	<1	<1	3	3
St. John's # 2	<1	2	10	53	65
St. John's # 3	<1	2	8	38	48
St. John's # 4	<1	2	10	39	51
St. John's # 5	<1	2	15	60	77

Table 3.6 Concentration Levels of THMs in Clarenville, Newfoundland (Winter)

Site#	Bromoform (μg/L)	Chloro-Dibromo Methane (μg/L)	Dichloro-Bromo Methane (μg/L)	Chloroform (μg/L)	Total THMs (μg/L)
Claren# 1	<1	<1	4	500	504
Claren# 2	<1	<1	4	382	386
Claren# 3	<1	<1	4	428	432
Claren# 4	<1	<1	4	431	435
Claren# 5	<1	<1	4	455	459
Claren# 6	<1	<1	4	557	561
Claren# 6 "Aerate"	<1	<1	4	173	177
Claren# 6 "Boil"	<1	<1	<1	25	25
Claren# 6 "Filter"	<1	<1	1	174	175
Claren# 6	<1	<1	2	291	293

"Refrigerate"					
Claren# 7	<1	<1	4	472	476
Claren# 8	<1	<1	4	510	514
Claren# 9	<1	<1	3	361	364
Claren# 10	<1	<1	5	400	405

The concentration levels of THMs compounds in drinking water collected from taps in Shoal Harbour, Newfoundland are shown in Table 3.7.

Table 3.7 Concentration Levels of THMs in Shoal Harbour, Newfoundland (Winter)

Site#	Bromoform (μg/L)	Chloro-Dibromo Methane (μg/L)	Dichloro-Bromo Methane (μg/L)	Chloroform (μg/L)	Total THMs (μg/L)
Shoal # 1	<1	<1	7	178	185
Shoal # 2	<1	<1	7	176	183
Shoal # 3	<1	<1	7	173	180
Shoal # 4	<1	<1	7	174	181
Shoal # 5	<1	<1	7	155	162
Shoal # 5 "Boil"	<1	<1	<1	15	15
Shoal # 5 "Filter"	<1	<1	5	62	67
Shoal # 5 "Refrigerate"	<1	<1	7	159	166
Shoal # 5 "Aerate"	<1	<1	7	69	76
Shoal # 6	<1	<1	7	182	189

Shoal # 7	<1	<1	8	175	183
Shoal # 8	<1	<1	8	235	243
Shoal # 9	<1	<1	8	197	205
Shoal # 10	<1	<1	8	192	200

Table 3.8 Summary Statistics of THMs Concentrations (μg/L) by Seasons and Locations

Variable	**Clarenville**		**Shoal Harbour**		**St. John's**	
Seasons	**Summer**	**Winter**	**Summer**	**Winter**	**Summer**	**Winter**
N	10	10	10	10	5	5
Mean	473.8	453.6	268.1	191.10	45.8	48.8
Median	479.5	447.0	271.5	184.00	42.0	51.0
Tr. Mean	479.7	451.4	267.4	188.25	45.8	48.8
Standard Deviation	39.5	61.5	37.7	21.63	30.8	28.1
SE Mean	12.5	19.4	11.9	6.84	13.8	12.6
Minimum	381.0	364.0	203.0	162.00	0.0	3.0
Maximum	519.0	561.0	339.0	243.00	81.0	77.0
Q1	459.2	400.3	238.0	180.75	20.0	25.5
Q2	505.0	506.5	293.2	201.25	73.5	71.0

The summary statistics of THMs concentrations by seasons and locations are shown in Table 3.8.

The boxplots of the concentration levels are shown in Figure 3.5.

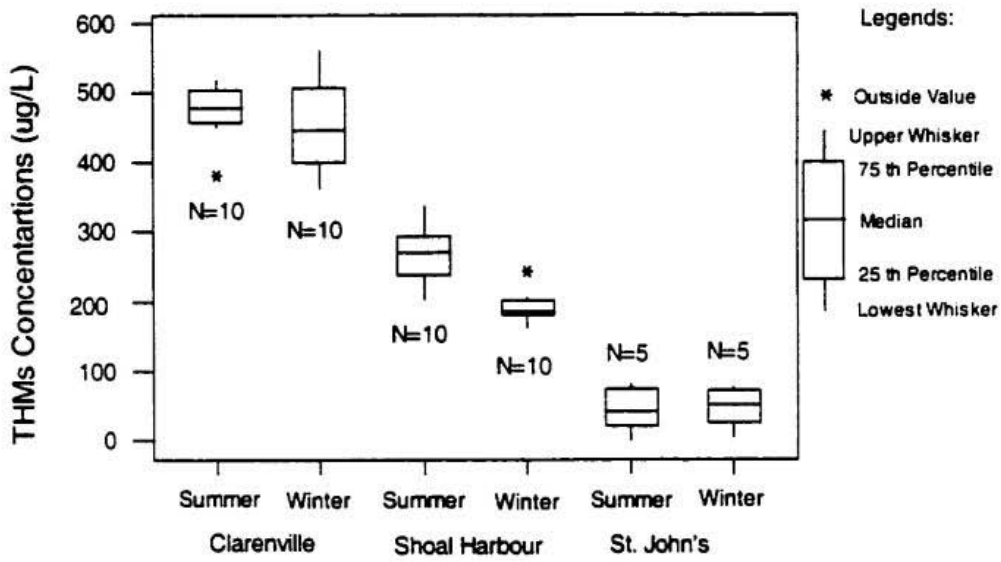

Figure 3.5 Boxplots of THMs Concentrations by Community and Season

From the boxplots, it is seen that the THMs concentration is highest in Clarenville, followed by Shoal Harbour, and St. John's. Concentrations in summer are higher than that in winter months.

Formal statistical tests to check whether there are seasonal differences at each community are given in the next sections.

3.3.1 Student's t-Test

The t-test can be used to determine whether two sample means are equal. If the total trihalomethanes (TTHMs) concentration data for St. John's is considered, we may think that the mean concentrations for the two seasons are equal. This may be stated formally as

$$H_0: \mu_1 = \mu_2 \qquad \textbf{(3.1.1)}$$

Where μ_1 is the mean total trihalomethanes concentration in summer season and μ_2 is the mean total trihalomethanes concentration in winter. The statement $H_0: \mu_1 = \mu_2$ is called the null hypothesis. Suppose that we cannot reasonably assume that the variances of total trihalomethanes concentrations are identical for both the seasons. Then the appropriate test statistic to use for comparing two seasonal mean concentrations in the completely randomised design is

$$t_o = \frac{\bar{y}_1 - \bar{y}_2}{\sqrt{\frac{s_1^2}{n_1} + \frac{s_2^2}{n_2}}} \qquad \textbf{(3.1.2)}$$

where

t_0 is test statistic

$\bar{y}_1$ is mean trihalomethanes concentrations in summer

$\bar{y}_2$ is mean trihalomethanes concentrations in winter

S_1^2 is variance of trihalomethanes concentrations in summer

S_2^2 is variance of trihalomethanes concentrations in winter

n_1 is number of data points in summer

n_2 is number of data points in winter

The degree of freedom is estimated by the following formula. The value is used to obtain a critical value from the t-table.

$$\nu\ (dof) = \frac{\left(\frac{S_1^2}{n_1} + \frac{S_2^2}{n_2}\right)^2}{\frac{\left(S_1^2/n_1\right)^2}{n_1 - 1} + \frac{\left(S_2^2/n_2\right)^2}{n_2 - 1}} \qquad \textbf{(3.1.3)}$$

To determine whether to reject H_0: $\mu_1 = \mu_2$, we would compare t_0 to the t distribution with ν degrees of freedom. If $t_0 > t_{\alpha,\nu}$, where $t_{\alpha,\nu}$ is the upper α percentage point of the t distribution with ν degrees of freedom, we would reject H_0 and conclude that the mean total trihalomethanes concentrations differ.

The summer data is considered as the first range of data. The winter data is considered to be second range of data. The hypothesised mean difference here is zero. A value of 0 (zero) indicates that the sample means are hypothesised to be equal. The alpha level (α) is a significance level .The alpha value (α) is chosen 0.05 here. In the current analysis, two-tailed t-test is performed. The results of the t-test for three communities are shown below.

Table 3.9 Summary of t-Test for Clarenville

Parameter	Summer	Winter
Mean	473.8	453.6
Variance	1559.95	3780.71
Observations	10	10
Hypothesised Mean Difference	0	
ν (degrees of freedom)	15	
t Stat	0.874	
P(T<=t) two-tail	0.395	
t Critical two-tail	2.131	

For Clarenville there are twenty data points. It is assumed that the population variances are unequal. This assumption is made for all the three communities. Therefore, we can use Equation 3.1.2 to test the hypotheses as stated in Equation 3.1.1.

Table 3.10 Summary of t-Test for Shoal Harbour

Parameter	Summer	Winter
Mean	268.1	191.1
Variance	1420.99	467.88
Observations	10	10
Hypothesised Mean Difference	0	
ν (degrees of freedom)	14	
t Stat	5.602	
P(T<=t) two-tail	6.516E-05	
t Critical two-tail	2.144	

As in this case $t_0 = 0.874 < t_{0.025,\ 15} = 2.131$, we would not reject H_0 and conclude that the mean concentrations of total trihalomethanes for the two seasons are not different (Table 3.9).

For Shoal Harbour, $t_0 = 5.602 > t_{0.025,14} = 2.145$, so we would reject H_0 and conclude that the mean concentrations of total trihalomethanes for the two seasons are different (Table 3.10). This may be due to the fact that some of the concentration levels in summer are too high or some of them in winter are too low. Based on available evidence it may be reported that lesser chlorination practice took place in winter than in summer

For St. John's, $t_0 = -0.16089 < t_{0.025,8} = 2.31$, we would not reject H_0 and conclude that the mean concentrations of total trihalomethanes for the two seasons are not different (Table 3.11). The results of the three communities are summarised in Table 3.12.

Table 3.11 Summary of t-Test for St. John's

Parameter	Summer	Winter
Mean	45.8	48.8
Variance	948.2	790.2
Observations	5	5
Hypothesised Mean Difference	0	
ν (degrees of freedom)	8	
t Stat	-0.160	
P(T<=t) two-tail	0.876	
t Critical two-tail	2.306	

Table 3.12 Summary of Hypothesis Testing

Communities	H_0: $\mu_1 = \mu_2$
Clarenville	Not Rejected
Shoal Harbour	Rejected
St. John's	Not Rejected

3.3.2 Mann-Whitney Test

Mann-Whitney gives the results of both a test and confidence interval to compare two independent samples. The results of the Mann-Whitney test for Clarenville is shown in Table 3.13.

Table 3.13 Summary of Mann-Whitney Test for Clarenville

Summer N = 10	Median = 479.50
Winter N = 10	Median = 447.00
Point estimate for $\eta_1 - \eta_2$ is 24.00	
95.5 Percent CI for $\eta_2 - \eta_2$ is (-33.00,77.97)	
W = 118.5	
The test is significant at p = 0.3256 (adjusted for ties)	
Cannot reject at alpha = 0.05	

where

W is the sum of the ranks in the combined sample associated with X observations

η_1 is the median of the trihalomethanes concentrations in summer

η_2 is the median of the trihalomethanes concentrations in winter

The null hypothesis is that the medians of the trihalomethanes concentrations in the two seasons are equal. Non-parametric methods are used to analyse the data.

As the p value 0.3256 >0.05, the null hypothesis is not rejected. That is, the medians of the trihalomethanes concentrations in the two seasons are not significantly different.

The results of the Mann-Whitney test for Shoal Harbour is shown in Table 3.14.

Table 3.14 Summary of Mann-Whitney Test for Shoal Harbour

Summer N = 10	Median = 271.50
Winter N = 10	Median = 184.00
Point estimate for $\eta_1 - \eta_2$ is 81.00	
95.5 Percent CI for $\eta_2 - \eta_2$ is (48.99,108.00)	
W = 151.0	
The test is significant at p = 0.0006 (adjusted for ties)	

As the p value 0.0006 < 0.05, the null hypothesis is rejected. That is, the medians of the trihalomethanes concentrations in the two seasons are different.

The results of the Mann-Whitney test for St. John's is shown in Table 3.15.

As the p value 0.8345>0.05, the null hypothesis is not rejected. That is, the medians of the trihalomethanes concentrations in the two seasons are not significantly different. The summary of the test results is shown in Table 3.16.

Table 3.15 Summary of Mann-Whitney Test for St. John's

Summer N = 5	Median = 42.00
Winter N = 5	Median = 51.00
Point estimate for $\eta_1 - \eta_2$ is -6.00	
96.3 Percent CI for $\eta_2 - \eta_2$ is (-50.97,39.02)	
W = 26.0	
The test is significant at p = 0.8345 (adjusted for ties)	
Cannot reject at alpha = 0.05	

Table 3.16 Summary of Hypothesis Testing

Communities	**H_0: $\eta_1 = \eta_2$**
Clarenville	Not Rejected
Shoal Harbour	Rejected
St. John's	Not Rejected

Water quality data of the municipal water supply in the three communities for other parameters are presented in Appendix 1. The compilation also provides information on relevant drinking water limits. The parameters, which exceed the limits, are assigned star signs. However, it is observed that most of the parameters are within limits. The concentration of dissolved organic carbon (DOC) is particularly significant because it affects the degree of trihalomethanes formation. Dissolved organic carbon concentration is much higher in Clarenville and Shoal Harbour than in St. John's. The higher concentration of trihalomethanes in those areas can be related to the high DOC levels.

3.4 Problem Formulation

The risk assessment study was conducted both at national and provincial level. The main objective was to estimate the health risk associated with multiple use of chlorinated tap water. In the current study, of the four chemical constituents of THMs, chloroform concentration levels are only considered for estimation of health risk due to its significant presence and importance.

Exposure to chloroform resulting from ingestion of chlorinated drinking water poses significant health risk to humans. Inhalation and dermal contact are the two other exposure pathways besides ingestion. Jo et al. (1990) have reported that exposure pathways like inhalation and dermal absorption can cause more exposure to volatile organic compounds (VOC) than pathways like ingestion. Humans are subjected to all these three kind of exposures through activities such as showering, bathing, cooking, toilet use, washing dishes, washing clothes, and drinking.

When an individual is taking a shower, that individual's full body is subjected to dermal exposure as a result of presence of contaminants in the water (in this case, volatile compounds). The entire confined space of the washroom is also filled up with the higher concentration levels of volatile compounds in the air. Thus the same individual is also subjected to inhalation. Many people are habituated to take at least one shower daily for their entire lifetime. In the current research, besides ingestion, the relationship between chloroform concentration in the water from shower and breath is also studied. Chloroform dose and cancer risk due to shower activity and water ingestion were determined by applying the model developed by Jo et al.(1990).

Most drinking water regulations, associated with chemical contaminants, are based on the assumption that daily water consumption is 2 litres by each individual.

Jo et al., (1990) estimated the chloroform exposure and health risk associated with multiple use of chlorinated tap water. The study estimated the increase in risk of cancer from domestic water use for three exposure pathways: ingestion, inhalation, and dermal. As the risks from these routes are similar in nature, all the pathways should be considered if the objective is to estimate the total risk caused by the use of municipal water supply.

Total chloroform dose as a result of taking a shower is estimated from the sum of doses from inhalation and dermal exposure (Jo et al., 1990). The following equation is used to calculate the chloroform dose from inhalation exposure:

$$Di = Er \times Ca \times R \times T/Wt \qquad \textbf{(3.2.1)}$$

where

Di is chloroform dose from an inhalation-only exposure (μg/inhalation exposure- kg)

Er is chloroform absorption efficiency via respiratory system

Ca is air concentration in shower ($\mu g/m^3$)

R is breathing rate (m^3/min)

T is duration of shower (minutes)

Wt is body weight of a reference person (70 kg)

In the above approach, it is assumed that contaminant exposure is occurring uniformly throughout the life of an individual. Therefore, parameters such as exposure frequency (EF), exposure duration (ED), and averaging time (AT) are not considered. The value of Er (chloroform absorption efficiency for the respiratory system) is 0.77

(U. S. EPA, 1980). The value of R (breathing rate) is assumed to be 0.014 m^3/min for a reference 70 kg male adult (Synedar, 1984). To calculate chloroform dose for inhalation pathways during shower, the concentration of chloroform in air during shower (Ca) is required as input in Equation 3.2.1. To estimate Ca, data reported by Jo et al (1990), is used and three different types of regression relationships are developed:

a) Linear Regression

$Y = 10.446X - 99.599$, where R^2 value of the linear regression is 0.87.

b) Exponential Regression

$Y = 1.1X^{1.54}$, where R^2 value of the log-log transformation is 0.82.

c) Non-linear Regression

$Y = 26.46 + 0.2025X^2$, where R^2 value of the non-linear regression is 0.894

These models were applied for lower and higher THM levels prevailing in the three communities, i.e. St John's, Shoal Harbour, and Clarenville respectively. It was found that among all these models, linear regression gave more conservative estimates than the other two models. However, at lower concentration of chloroform in water (<9.5 µg/L), the linear model may not be valid. At such low level, the partitioning of chloroform from water into the air, based on physical properties of chloroform, is considered to be negligible (NAS, 1978). Therefore, for a conservative estimate of chloroform concentration in air during shower, the linear regression equation is used in this study. It is however important to mention that such estimates should be used with caution for decision making purposes due to the following reasons:

(a) Range of data on which regression equation is established does not cover the typical ranges measured in the three communities of Newfoundland.

(b) The set of parameters under which experiment was performed may not be similar to the conditions prevailing in a typical shower in the three communities. The parameters in Jo et al's experiment are water temperature, water flow rate, shower duration, water concentration, showerhead setting, and ventilation. These parameters in shower room may differ from community to community.

For Clarenville, the mean chloroform concentration is 459 μg/L. The corresponding shower air concentration values (Ca) estimated using the above stated non-linear (Ca=42689 $\mu g/m^3$) and exponential (Ca=13822 $\mu g/m^3$) regression relationships are much higher than the shower air concentration value (Ca) estimated using linear regression relationship (Ca=4695 $\mu g/m^3$).

For Shoal Harbour, the mean chloroform concentration is 223 μg/L. The corresponding shower air concentration values (Ca) estimated using the above stated non-linear (Ca=10096 $\mu g/m^3$) and exponential (Ca=4547 $\mu g/m^3$) regression relationships are much higher than the shower air concentration value (Ca) estimated using linear regression relationship (Ca=2230 $\mu g/m^3$).

The values of shower air concentrations (Ca) corresponding to different chloroform concentrations in the National Survey and Newfoundland Study are therefore estimated from the following regression Equation 3.2.2. The regression relationship is as follows:

$$Y = 10.446\ X - 99.599 \qquad \textbf{(3.2.2)}$$

where

X is chloroform concentrations in water (μg/L)

Y is shower air concentrations (Ca) ($\mu g/ m^3$)

This regression equation is developed based on the set of data (Table 3.18) obtained in the study by Jo et al (1990). The values of shower air concentrations (Ca) for National Survey and Newfoundland Study thus determined are put in the Equation 3.2.1 to obtain the values of chloroform dose from an inhalation-only exposure (Di). This regression model is applicable only for $X \geq 9.5$ µg/L. Below this level the concentration of chloroform is considered to be negligible.

From the regression plot (Fig 3.6 and Table 3.17), it is seen that the slope and the intercept are statistically significant at $\alpha = 5\%$, and the $R^2 = 0.863$ is reasonably high.

Table 3.17 Parametric Values of Equation 3.2.2

Predictor	Coefficient	Standard Deviation	T	P
Constant	-99.60	25.64	-3.88	0.001
X	10.4459	0.9765	10.70	0.000
S = 27.69	R-Sq. = 87.1%	R-Sq.(adj) = 86.3%		

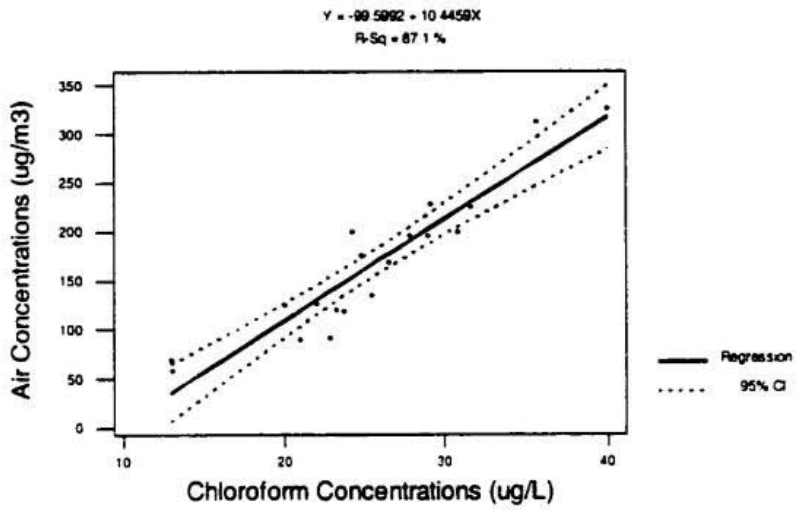

Figure 3.6 Regression Plot for Equation 3.2.2

The goodness of fit is satisfactory and the equation gives reasonable estimates.

Normality of residuals is satisfied as seen in the normal probability plot (Figure 3.7) which shows a straight line. (R = 0.99, P value > 0.1). The figure also shows that the variance is constant and the residuals are randomly scattered. There is no trend along fitted value or order of observation. Figure 3.8 shows the residual model diagnostic of Equation 3.2.2.

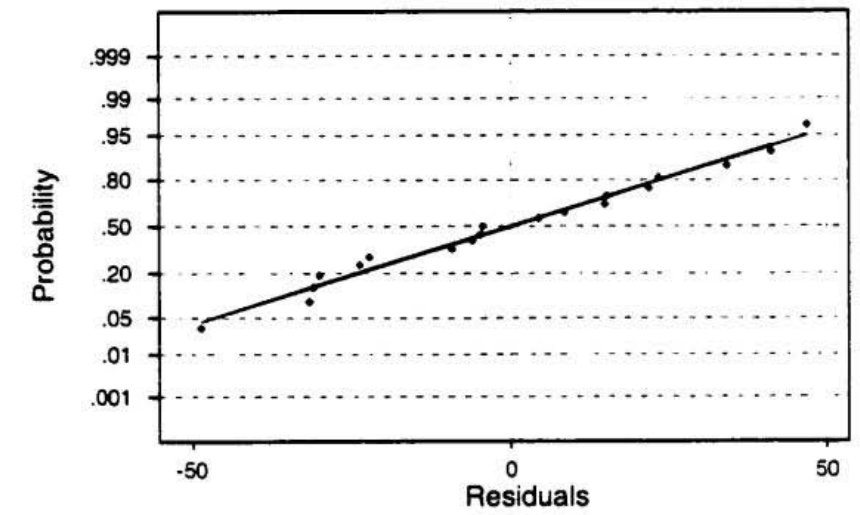

Figure 3.7 Normal Probability Plot for Equation 3.2.2

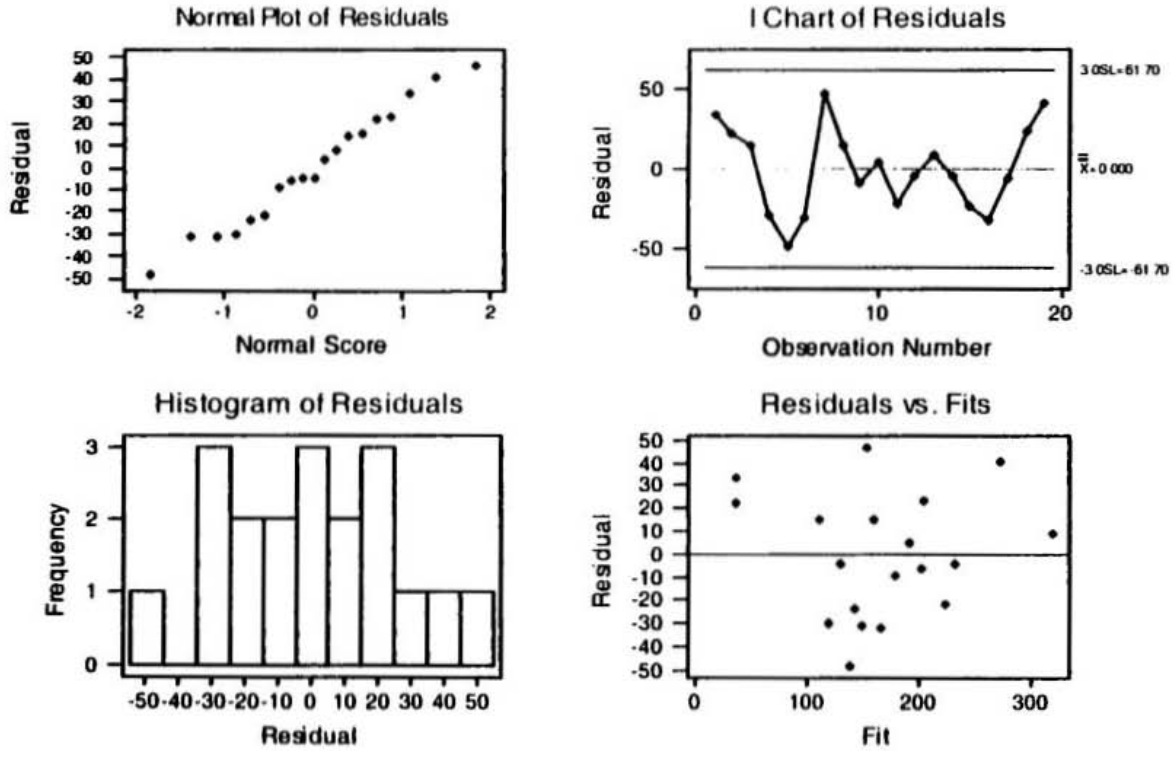

Figure 3.8 Residual Model Diagnostics for Equation 3.2.2

Jo et al., (1990) reported that dermal exposure during showering activity cause chloroform absorption by the body. When the researchers compared chloroform breath concentrations after normal showers with those after inhalation only exposures, they found that "inhalation and dermal exposures result in comparable chloroform doses." It was also reported that the difference was statistically significant at a probability of $p = 0.0001$. Several other studies have reported that VOCs penetrate human and animal skin, which essentially reiterate dermal absorption while showering.

The chloroform dose from dermal exposure is calculated using the following equation:

$$Dd = Di \times F \qquad \textbf{(3.2.3)}$$

where

Dd is chloroform dose from a dermal exposure (μg/dermal exposure-kg)

Di is chloroform dose from an inhalation-only exposure (μg/inhalation exposure-kg)

F is ratio of the body burden from dermal exposure to that from inhalation exposure

Dose from normal showers is the sum of dose from inhalation and dermal exposures.

Step 1: Calculation of Breath Concentrations Obtained after Normal Showers

In order to estimate breath concentrations after normal shower, Jo et al. (1990), collected breath samples from each subject prior to and after each shower. Water samples were collected in the bathroom from the tap after each shower. The shower protocol used was representative of typical shower conditions. The data set obtained in the experiment is given in Table 3.13. A regression relationship (Equation 3.2.4) is developed based on these data. The breath concentrations after normal showers in the

National Survey and Newfoundland Study are estimated using this relationship. The key assumption include the following regression relationship developed in the study (Jo et al., 1990) is valid for the range of chloroform concentrations both in National Survey (1995) and Newfoundland Study (1998).

Table 3.18 Shower Air Concentrations (Ca) vs. Tap Water Concentrations Obtained Without & With a Showering Individual in the Full-Size Shower, Jo et al., Study, 1990

Water Concentrations (μg/L)	Air Concentrations ($\mu g/m^3$)
12.9	69.2
13	58.1
20	124.2
21	89.7
22.8	89.9
23.7	117.2
24.2	200
24.8	174.8
26.5	168.1
27.8	195.2
30.8	200.2
31.8	225.9
40	326.9
22	125.9
23.2	119.2
25.4	134.1
28.9	196.3
29.1	227.8
35.5	313.4

$$Y_1 = 0.4469\ X + 2.907 \qquad \textbf{(3.2.4)}$$

where

X is chloroform concentration in water (μg/L)

Y_1 is breath concentrations obtained after normal showers (μg/ m^3)

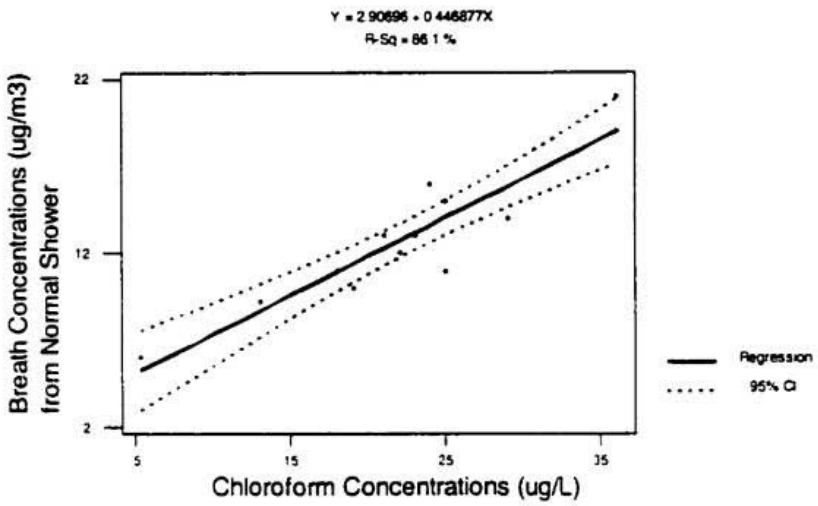

Figure 3.9 Regression Plot for Equation 3.2.4

From the regression plot (Fig 3.10 and Table 3.11), it is seen that the slope and the intercept are statistically significant at $\alpha = 5\%$ with a reasonably high R^2 of 86.1%.

Table 3.19 Parametric Values of Equation 3.2.4

Predictor	Coefficient	Standard Deviation	T	P
Constant	2.907	1.311	2.22	0.049
X	0.44688	0.05424	8.24	0.000
S = 1.574	R-Sq. = 86.1%	R-Sq.(adj) = 84.8%		

Normality of residuals is satisfied as seen from the normal probability plot (Figure 3.12) which shows a straight line. (R = 0.98, P value > 0.1). The figure also shows that the variance is constant and the residuals are randomly scattered. There is no trend along fitted value or order of observation. Figure 3.13 shows the residual model diagnostic of Equation 3.2.4.

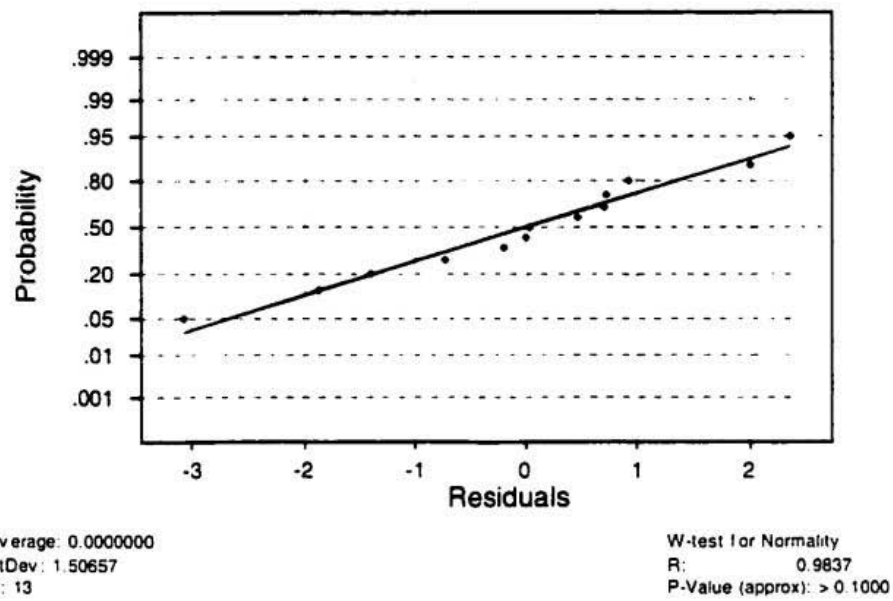

Figure 3.10 Normal Probability Plot for Equation 3.2.4

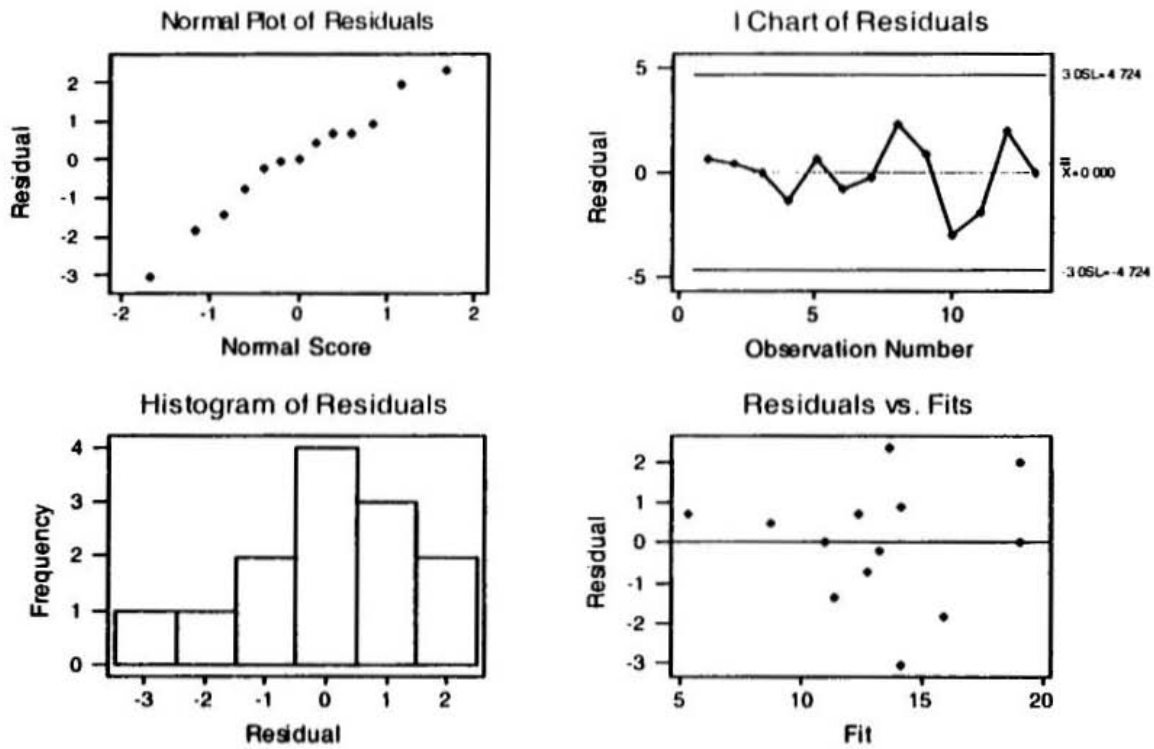

Figure 3.11 Residual Model Diagnostics for Equation 3.2.4

Step 2: Calculation of Breath Concentrations Obtained after Inhalation-Only Exposure

To estimate breath concentrations after inhalation-only exposure, each subject in Jo et al. (1990) study was exposed to chloroform vaporised from shower water while standing within the shower stall. Rubber clothes and boots were worn by the subject during the experiment to avoid dermal contact with the shower water. Prior to each inhalation only exposure, a breath sample was collected from the subject. Chloroform exposures from inhalation only were estimated by measuring chloroform concentration in water samples and breath samples taken from subjects after inhalation-only exposures. The data set obtained in this experiment is given in Table 3.21. A regression relationship (Equation 3.2.5) is developed based on these data. The breath concentrations after inhalation-only exposure in the National Survey and Newfoundland Study are estimated using this relationship.

$$Y_2 = 0.2578\ X + 0.8576 \qquad \textbf{(3.2.5)}$$

where

X is chloroform concentration in water (μg/L)

Y_2 is breath concentrations obtained after inhalation-only exposure (μg/ m^3)

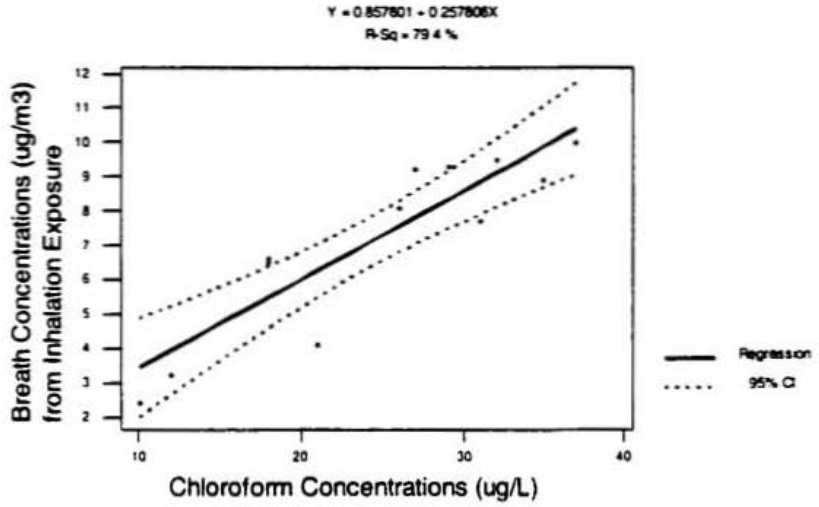

Figure 3.12 Regression Plot for Equation 3.2.5

From the regression plot (Figure 3.12 and Table 3.20), it is seen that the slope is statistically significant at $\alpha = 5\%$. The constant term given by regression equation is not statistically significant at $\alpha = 5\%$. However, this constant is used here because intercept of zero may not be suitable after observing the plot of chloroform concentration versus breath concentration.

The goodness of fit is acceptable with a R^2 of 79.4%.

Table 3.20 Parametric Values of Equation 3.2.5

Predictor	Coefficient	Standard Deviation	T	P
Constant	0.858	1.012	0.85	0.415
X	0.25781	0.03964	6.50	0.000
S = 1.188	R-Sq. = 79.4%	R-Sq.(adj) = 77.5%		

Normality of residuals is satisfied as seen from the normal probability plot (Figure 3.13) (R = 0.96. P value > 0.1). The figure also shows that the variance is constant and the residuals are randomly scattered. There is no trend along fitted value or order of observation. Figure 3.14 shows the residual model diagnostic of Equation 3.2.5.

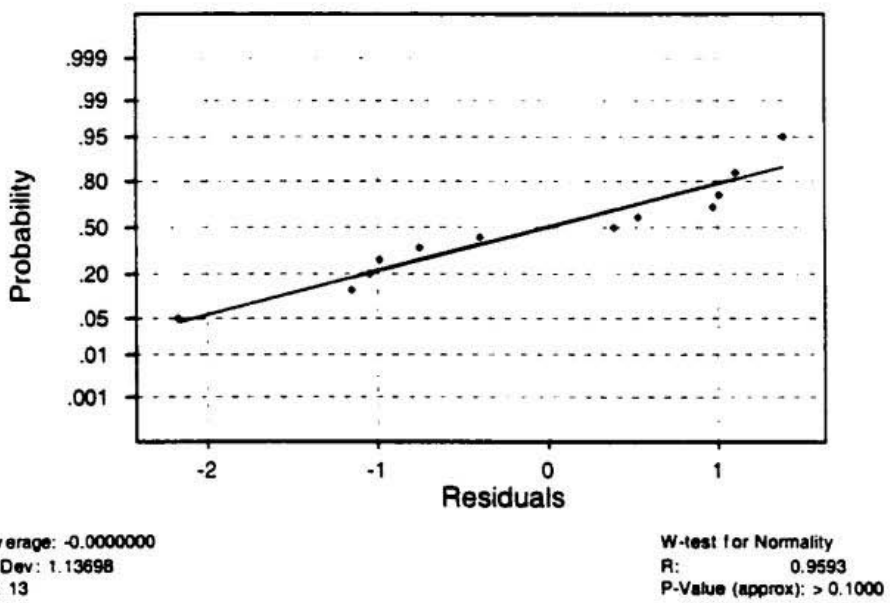

Figure 3.13 Normal Probability Plot for Equation 3.2.5

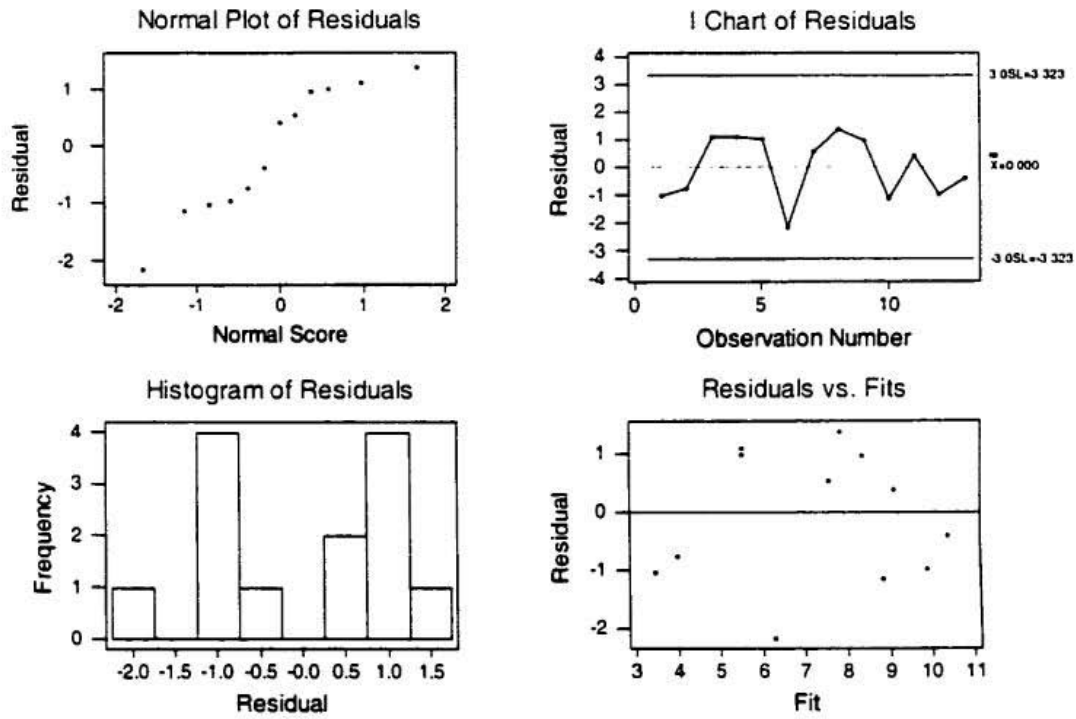

Figure 3.14 Residual Model Diagnostics for Equation 3.2.5

Step 3: Calculations of F

Using the formula for determining F

$$F = \frac{Y_1 - Y_2}{Y_2} \qquad \textbf{(3.2.5a)}$$

where

Y_1 is breath concentrations obtained after normal showers ($\mu g/ m^3$)

Y_2 is breath concentrations obtained after inhalation-only exposure (μg/ m^3)

the value is evaluated. The values of respective Di and F are put in Equation 3.2.3 to estimate the values of chloroform doses from dermal exposure (Dd). Chloroform dose from normal shower is the summation of the dose from inhalation-only exposure and dermal exposure.

D (Normal Showers) = Di (Inhalation-Only Exposure) + Dd (Dermal Exposure)

Hence, the respective doses for inhalation-only and dermal exposures are added to obtain the chloroform doses from normal showers. The chloroform dose from water ingestion is determined from the following relationship:

$$Dig = Ei \times Cw \times Aw/Wt \qquad \textbf{(3.2.6)}$$

where

Ei is absorption efficiency of chloroform via the gastrointestinal tract

Cw is chloroform concentration in the water (μg/L)

Aw is quantity of water ingested per day (L/day)

Wt is body weight of a reference person (70-kg)

Dig is dose from water ingestion (μg/kg-day).

The key assumptions include the gastrointestinal tract had an absorption efficiency of 100% (maximum potential dose to an individual) and daily water ingestion amount of 2L. The chloroform concentration in water is denoted by Cw. The quantity of water ingested per day (Aw) is equal to 2L. Body weight (Wt) of a reference person is equal to 70 kg. The set of values of chloroform doses resulting from water ingestion of 2L per day is then estimated.

The calculated doses Di (inhalation), Dd (dermal exposure) and Dig (water ingestion) are used to estimate the chloroform risk associated with shower and water ingestion for a reference person. Marty (1989) used a linearised model to calculate the cancer potency also known as "slope factor" of the chloroform exposure. The slope factor is a measure of increase in the incidence of cancer resulting from a unit increase in dose. In the model, animal data at high experimental doses were extrapolated to low environmental exposure levels for calculating the cancer risk for humans. The model adopted for estimating the increase in the cancer risk associated with the ingestion exposure was extended to the inhalation and dermal routes of exposure in order to estimate the corresponding cancer risk from shower.

The model is as follows:

$$Pd = q \times D \times 10^{-3} \qquad \textbf{(3.2.7)}$$

where

Pd is lifetime risk (Unit less)

q is cancer risk potency slope $(mg/kg\text{-}day)^{-1}$

D is chloroform dose (μg/kg-day)

Table 3.21 Chloroform Tap Water Concentrations vs. Breath Concentrations Obtained after Normal Showers and Inhalation-Only Exposure Using a Full-Size Shower, Jo et al., Study, 1990

Normal Showers		Inhalation-Only Exposure	
Water Concentrations (μg/L)	Breath Concentrations (μg/ m^3)	Water Concentrations (μg/L)	Breath Concentrations (μg/ m^3)
5.3	6	10	2.4
13	9.2	12	3.2
18	11	18	6.6
19	10	18	6.6
21	13	18	6.5
22	12	21	4.1
23	13	26	8.1
24	16	27	9.2
25	15	29	9.3
25	11	31	7.7
29	14	32	9.5
36	21	35	8.9
36	19	37	10

In the present study, different values of the cancer risk potency slope or the slope factor are used corresponding to the different exposure pathways. The values are taken from the Integrated Risk Information System (IRIS) of United States Environmental Protection Agency (U.S. EPA, 1999). For water ingestion, the cancer risk potency slope value or the slope factor used is 0.0061 $(mg/kg\text{-}day)^{-1}$ (U. S. EPA, 1999). For inhalation exposure, the geometric mean of the cancer risk potency values (U. S. EPA, 1999) for male and female during inhalation exposure is used which is 0.0812 $(mg/kg\text{-}day)^{-1}$. For

dermal exposure, the cancer risk potency slope is assumed to be equal to that of water ingestion (Jo et al., 1990). The slope factor as available in the literature is 0.0061 $(mg/kg\text{-}day)^{-1}$ (U. S. EPA, 1999). The values of chloroform doses (Di, Dd, Dig) from different exposure conditions and shower duration are put in Equation 3.2.7 to obtain the corresponding lifetime cancer risk.

The cancer risk from shower is the summation of cancer risk from inhalation-only exposure and dermal exposure. The cancer risks (Pdi) from inhalation-only exposures are determined by putting the values of chloroform doses from inhalation-only exposures (Di) in Equation 3.2.7. The cancer risks (Pdd) from dermal exposures are determined by putting the values of chloroform doses from dermal exposures (Dd) in Equation 3.2.7. Thus, the cancer risk from showers (Pd) are determined by adding cancer risk values from inhalation-only exposure (Pdi) to cancer risk values from dermal exposure (Pdi). The cancer risk (Pd) from water ingestion is determined by putting the value of chloroform dose from water ingestion (Dig) in Equation 3.2.7.

The estimated number of cancer cases in the communities from chloroform is determined by multiplying the individual lifetime risks calculated by the above procedures with the population of the community exposed. This essentially means the entire population uses the chlorinated tap water.

Hence,

Estimated Cancer Cases in the Community = Lifetime Individual Risk × Exposed Population **(3.2.8)**

where the estimated cancer cases in the community is the number of cancer cases in the exposed population over the lifetime, the lifetime individual risk is the risk to an

individual for getting cancer over the lifetime (dimensionless). and the exposed population is the community size exposed to the contaminant.

Chapter 4

Drinking Water Quality in Canada

4.1 THMs Regulatory Values

Various regulatory agencies have established guidelines for disinfectant by-products like THMs. Among them, prominent ones are listed in Table 4.1.1.

The U. S. EPA maximum contaminant level (MCL) for TTHMs was established at 0.1 mg/L, however, the EPA Federal Register on "Disinfectants and Disinfection By-Products; Proposed Rule" (1994) reports the proposed MCL for TTHMs as 0.08 mg/L and the sum of five haloacetic acids (HAA5) as 0.06 mg/L. Health Canada has set the interim maximum acceptable concentration (IMAC) of trihalomethanes as 0.1 mg/L (running annual average). It is based on the risk associated with chloroform, the trihalomethane most often present in drinking water.

Total trihalomethanes (TTHMs) is the sum of the concentrations of bromodichloromethane, dibromochloromethane, bromoform, and chloroform. Haloacetic acids (five, HAA5) are the sum of the concentrations of mono-, di-, and trichloroacetic acids and mono- and dibromoacetic acids.

U. S. EPA also proposed the following maximum disinfectant residual level goals (MRDLGs) and maximum residual disinfectant levels.

Table 4.1.1 DBPs Guidelines

DBPs	U. S. EPA Proposed MCLG or MCL (mg/L)	WHO (mg/L)	Health Canada IMAC (mg/L)
Total trihalomethanes (TTHMs)	0.080	-	0.1
Haloacetic acids (five) (HAA5)	0.060	-	-
Chloroform	0	0.2	-
Bromodichloro methane	0	0.06	-
Dibromochloro methane	0.06	0.1	-
Bromoform	0	0.1	-
Dichloroacetic acid	0	0.05	-
Trichloroacetic acid	0.3	0.1	-

Table 4.1.2 U. S. EPA Proposed MRDLGs and MRDLs for Disinfectants

Disinfectant Residual	MRDLG (mg/l)	MRDL (mg/l)
(1) Chlorine	4 (as Cl_2)	4.0 (as Cl_2)
(2) Chloramines	4 (as Cl_2)	4.0 (as Cl_2)
(3) Chlorine dioxide	0.3 (as ClO_2)	0.8 (as ClO_2)

Source: U. S. EPA, 1994

Regarding treatment technique for DBP precursors, EPA proposed (1994) that water system that use surface water or ground water under the direct influence of surface water and use conventional filtration treatment be required to remove specified amounts of organic materials (measured as total organic carbon) that may react with disinfectants to form disinfection by-products. Removal would be achieved through a treatment

technique (enhanced coagulation or enhanced softening) unless the system meets certain criteria.

Regarding best available technology (BAT) for disinfectants, EPA proposed (1994) the following options for limiting residual disinfection concentrations in the distribution system.

- To reduce chlorine demand and control of disinfection treatment processes to reduce disinfectant levels
- To reduce chloramine demand and control of disinfection treatment processes to reduce disinfectant levels
- To reduce chlorine dioxide demand and control of disinfection treatment processes to reduce disinfectant levels.

4.2 THMs in Canadian Drinking Water Supply

A nation-wide survey was undertaken by Health Canada in the year 1993 to determine the concentrations of halogenated disinfection by-products in Canadian drinking water. The results of the survey presented significant overview of the prevailing situation in terms of drinking water quality and halogenated disinfection by-products concentration levels as a result of chlorination during water treatment. The prime objective of the study was to analyse the effects of the different disinfectants used (chlorine, chloramine and ozone), seasonal variation (winter and summer) and spatial variation (treatment plant and distribution system).

Disinfection of water supplies at a stage during treatment is very crucial in rendering the human pathogenic microorganisms harmless. Among the microorganisms,

the ones causing typhoid fever and cholera need to be mentioned. However, inadequate disinfection can still result in cholera epidemics (Glass et al., 1992). In Canada, it is reported that disinfection of all surface waters used for human consumption is crucial and that the health risks from pathogenic microorganisms far exceed those potential health risks associated with chemical disinfection by-products (DBPs) formed during drinking water treatment; hence, the trade-off is, to minimise the potential risks from DBPs without compromising disinfection efficiency (Health Canada, 1995). There is no quantitative data on the relative risk comparison of the presence of pathogenic microorganisms and DBPs in the report by Health Canada.

The result of the national survey was published in the form of a comprehensive report titled, "A National Survey of Chlorinated Disinfection By-Products in Canadian Drinking Water," by Environmental Health Directorate, Health Protection Branch, Health Canada (1995). Chlorine has proven highly effective both as a primary and residual disinfection agent and can be easily used. Several studies have also established that chlorine reacts with "biogenic organic matter", as humic and fulvic acids, found in all natural surface waters. The above association results in formation of chlorinated organic by-products that are detected in drinking water supplies. Due to the complexity of the chemistry involved, it is not yet feasible to predict the concentration levels of various DBPs that will be formed in any given water sample (Health Canada, 1995). Although the initial focus was on adverse health effects due to THMs, recent studies have included haloacetic acids (HAAs), haloacetonitriles (HANs), chloropicrin (CPK), chloral hydrate (CH) and other DBPs (Health Canada, 1995).

In the Canadian National Survey (1993), presence of various disinfection by-products was reported in majority of the water treatment facilities throughout the country, of which trihalomethanes and haloacetic acids were the principal ones. Fifty three sites spread over the country were investigated representing the cross-section of the larger population in nine provinces. Prince Edward Island was kept out of the survey due to the limited use of chlorine in the province.

The raw water for the water treatment plants were taken from Canadian lakes, rivers and wells. Water samples were collected from raw, treatment plant and distribution system. The survey included the three major disinfectant methods: chlorine-chlorine, chlorine-chloramine and ozone-chlor (am)ine. Sample collection was undertaken in 1993 during the winter season (February-March) and the summer season (August-September). It was found that on many occasions, the concentration levels of haloacetic acids were equal or greater than that of trihalomethanes. Less significant was the presence of by-products like chloral hydrate, halopropanones, haloacetonitriles and chloropicrin. Three types of treatment processes were considered. chlorine-chlorine, chlorine-chloramine and ozone-chlor (am)ine. It was found that the mean and median trihalomethanes concentration levels in summer exceeded the corresponding values in winter season for all the three types of treatment processes. The values escalated in the case of distribution system with the exception of chlorine-chloramine treatment.

The focus of the survey was on the levels of DBPs in the Canadian drinking water so that the data produced could be used as a reference "in the preparation of future Canadian Drinking Water Guidelines." The outcome of the survey was the determination of 17 different chlorinated DBPs in addition to total bromide ion.

Table 4.1.3 DBPs Analysed in 1993 National Survey (Health Canada, 1995)

Compound	Minimum Quantifiable Limit (MQL)	
Chloroform ($CHCl_3$) [TCM]	0.2	µg/L
Bromodichloromethane ($CHBrCl_2$) [BDCM]	0.1	µg/L
Chlorodibromomethane ($CHBr_2Cl$) [CDBM]	0.1	µg/L
Bromoform ($CHBr_3$) [TBM]	0.1	µg/L
Monochloroacetic acid ($CH_2ClCOOH$) [MCAA]	0.01	µg/L
Dichloroacetic acid ($CHCl_2COOH$) [DCAA]	0.01	µg/L
Trichloroacetic acid (CCl_3COOH) [TCAA]	0.01	µg/L
Monobromoacetic acid ($CH_2BrCOOH$) [MBAA]	0.01	µg/L
Dibromoacetic acid ($CHBr_2COOH$) [DBAA]	0.01	µg/L
Dichloroacetonitrile ($CHCl_2CN$) [DCAN]	0.1	µg/L
Trichloroacetonitrile (CCl_3CN) [TCAN]	0.1	µg/L
Bromochloroacetonitrile (CHBrClCN) [BCAN]	0.1	µg/L
Dibromoacetonitrile ($CHBr_2CN$) [DBAN]	0.1	µg/L
1,1-Dichlro-2-propanone ($CHCl_2COCH_3$) [DCP]	0.1	µg/L
1,1,1-Trichloro-2-propanone (CCl_3COCH_3) [TCP]	0.1	µg/L
Chloral hydrate ($CCl_3CH(OH)_2$) [CH]	0.1	µg/L
Chloropicrin (CCl_3NO_2) [CPK]	0.1	µg/L
Bromide ion (winter)	0.01	mg/L
Bromide ion (summer)	0.002	mg/L
Total organic carbon [TOC]	0.1	mg/L
Total organic halide [TOX]	5.0	µg/L

organic carbon and total organic halides. The report concluded that TTHMs and HAAs were the main DBPs found in all facilities for all treatment processes and HAA levels

often equalled or exceeded TTHMs concentrations, mean and median TTHMs levels were higher in summer than in winter for all three treatment processes, and increased in the distribution system except for chlorine-chloramine treatment (Health Canada, 1995).

4.3 Risk Assessment of THMs in Canadian Drinking Water

In this section the health risk assessment of trihalomethanes in Canadian drinking water is discussed. The concentration levels of chloroform in the water distribution system for winter and summer months are given in the Appendix 1. The mean chloroform concentrations are shown in Table 4.3.1.

Table 4.3.1 Mean Chloroform Concentrations (Cw) at National Level, 1993

Provinces	Mean Concentration Values (μg/L)	
	Winter	Summer
	Cw	
Alberta	7.43	18.93
British Columbia	18.52	19.13
Manitoba	59.06	115.54
New Brunswick	24.75	65.70
Newfoundland	4.75	8.50
Nova Scotia	33.17	85.14
Ontario	12.67	29.81
Quebec	14.82	50.89
Saskatchewan	42.20	60.35

4.3.1 Risk Estimation for Normal Shower

As discussed in Chapter 3, the model used by Jo et al (1990) is used in the current study. The chloroform dose from inhalation exposure is estimated using the Equation 3.2.1. The values of shower air concentrations (Ca) corresponding to different

chloroform concentrations in the National Survey (Table 4.3.2) are estimated from Equation 3.2.2.

Table 4.3.2 Shower Air Concentrations (Ca) for National Survey, 1993

Provinces	Air Concentration ($\mu g/ m^3$) Values	
	Ca	
	Winter	Summer
Alberta	< 1	98.09
British Columbia	93.83	100.27
Manitoba	517.34	1107.33
New Brunswick	158.94	586.70
Newfoundland	< 1	< 1
Nova Scotia	246.91	789.80
Ontario	32.75	211.80
Quebec	55.16	432.02
Saskatchewan	341.22	530.82

The values of Ca from Table 4.3.2 are then put in the Equation 3.2.1 to obtain the values of chloroform dose from an inhalation-only exposure (Di). The breath concentrations after normal shower are calculated using Equation 3.2.4. The breath concentrations estimated for National Survey are given in Table 4.3.3. The breath concentrations obtained after inhalation-only exposure are determined using Equation 3.2.5. The breath concentrations obtained after inhalation-only exposure estimated for National Survey are given in Table 4.3.3.

Table 4.3.3 Breath Concentrations ($\mu g/ m^3$) Obtained after Normal Shower and Inhalation-Only Exposure, National Survey, 1993

Provinces	Normal Shower		Inhalation-Only Exposure	
	Winter	Summer	Winter	Summer
Alberta	6.23	11.36	2.77	5.74
British Columbia	11.18	11.46	5.63	5.79
Manitoba	29.30	54.54	16.08	30.64
New Brunswick	13.97	32.27	7.24	17.80
Newfoundland	5.03	6.71	2.08	3.05
Nova Scotia	17.73	40.96	9.41	22.81
Ontario	8.57	16.23	4.12	8.54
Quebec	9.53	25.65	4.68	13.98
Saskatchewan	21.77	29.88	11.74	16.42

Using Equation 3.2.5a, the value of F is evaluated. The values of Di and F are then put in Equation 3.2.3 to estimate the values of chloroform doses from dermal exposure (Dd). The respective doses (Di and Dd) are added to obtain the chloroform doses from normal shower.

The cancer risks (Pdi) from inhalation-only exposure are determined by putting the values of chloroform doses from inhalation-only exposure (Di) in Equation 3.2.7. The calculated values are shown in Table 4.3.4 (10 minutes shower) and Figure 4.3.1. The cancer risks (Pdd) from dermal exposures are determined by putting the values of chloroform doses from dermal exposures (Dd) in Equation 3.2.7. The calculated values are shown in Table 4.3.4 (10 minutes shower) and Figure 4.3.2. Similarly, the cancer risks from normal shower (Pd) are determined by adding cancer risk values from inhalation-only exposure (Pdi) to cancer risk values from dermal exposure (Pdd). The final values, lifetime risk from normal shower (Pd) are shown in the subsequent columns

of Table 4.3.4 (10 minutes shower). All the lifetime risks estimated above are the probabilities of excess cancer over an individual's lifetime due to exposures to THMs.

Table 4.3.4 Lifetime Risk from Different Exposure Pathways

Provinces	Inhalation-Only Exposure (Pdi)		Dermal Exposure (Pdd)		Normal Shower (Pd)		Water Ingestion Based on 2L/day	
	$Pdi = q \times Di \times 10^{-3}$		$Pdd = q \times Dd \times 10^{-3}$		$Pd = Pdi + Pdd$		$Pd = q \times Dig \times 10^{-3}$	
	Winter	Summer	Winter	Summer	Winter	Summer	Winter	Summer
Alberta	$< 0.01 \times 10^{-4}$	0.12×10^{-4}	$< 0.01 \times 10^{-4}$	0.01×10^{-4}	$< 0.01 \times 10^{-4}$	0.13×10^{-4}	0.01×10^{-4}	0.03×10^{-4}
British Columbia	0.12×10^{-4}	0.13×10^{-4}	0.01×10^{-4}	0.01×10^{-4}	0.13×10^{-4}	0.13×10^{-4}	0.03×10^{-4}	0.03×10^{-4}
Manitoba	0.65×10^{-4}	1.38×10^{-4}	0.04×10^{-4}	0.08×10^{-4}	0.69×10^{-4}	1.47×10^{-4}	0.10×10^{-4}	0.20×10^{-4}
New Brunswick	0.20×10^{-4}	0.73×10^{-4}	0.01×10^{-4}	0.04×10^{-4}	0.21×10^{-4}	0.78×10^{-4}	0.04×10^{-4}	0.11×10^{-4}
Newfoundland	$< 0.01 \times 10^{-4}$	$< 0.01 \times 10^{-4}$	$< 0.01 \times 10^{-4}$	$< 0.01 \times 10^{-4}$	$< 0.01 \times 10^{-4}$	$< 0.01 \times 10^{-4}$	0.01×10^{-4}	0.01×10^{-4}
Nova Scotia	0.31×10^{-4}	0.99×10^{-4}	0.02×10^{-4}	0.06×10^{-4}	0.33×10^{-4}	1.05×10^{-4}	0.06×10^{-4}	0.15×10^{-4}
Ontario	0.04×10^{-4}	0.26×10^{-4}	0.00×10^{-4}	0.02×10^{-4}	0.04×10^{-4}	0.28×10^{-4}	0.02×10^{-4}	0.05×10^{-4}
Quebec	0.07×10^{-4}	0.54×10^{-4}	0.01×10^{-4}	0.03×10^{-4}	0.07×10^{-4}	0.57×10^{-4}	0.03×10^{-4}	0.09×10^{-4}
Saskatchewan	0.43×10^{-4}	0.66×10^{-4}	0.03×10^{-4}	0.04×10^{-4}	0.45×10^{-4}	0.70×10^{-4}	0.07×10^{-4}	0.11×10^{-4}

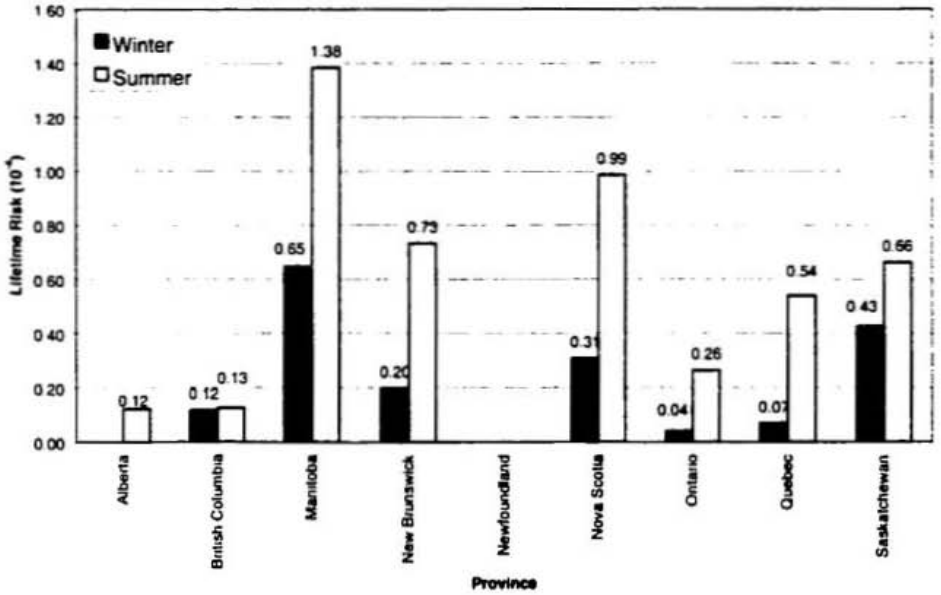

Figure 4.3.1 Lifetime Risk from Inhalation-Only Exposure (10 minutes)

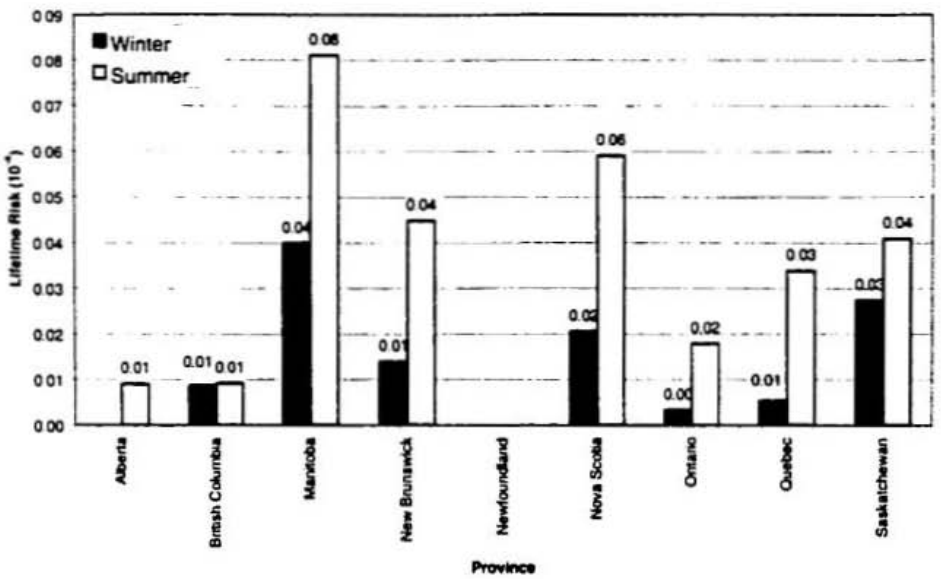

Figure 4.3.2 Lifetime Risk from Dermal Exposure (10 minutes)

4.3.2 Risk Estimation for Water Ingestion

The chloroform dose from water ingestion (Dig) is determined from Equation 3.2.6. The cancer risk (Pd) from water ingestion is determined, by putting the value of chloroform dose from water ingestion (Dig) estimated above, in Equation 3.2.7. The calculated values are shown in Table 4.3.4 and Figure 4.3.3. All the lifetime risks estimated are risks of getting cancer over an individual's lifetime due to various exposures.

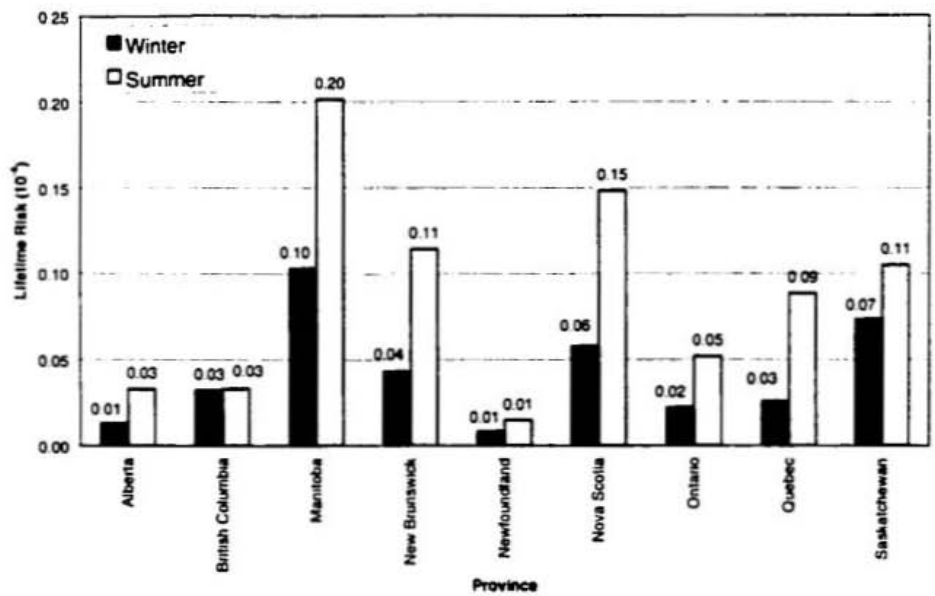

Figure 4.3.3 Lifetime Risk from Water Ingestion Based on 2L/day

4.3.3 Estimation of Cancer Cases in the Exposed Population

For determining the cancer cases in various provinces of Canada, the 1996 census for Canadian population is used. The population is shown in Table 4.3.5 and Figure 4.3.4.

Thus, the number of cancer cases, under various exposure conditions, in various provinces of Canada are estimated with the help of Equation 3.2.8 and the results are listed in Table 4.3.6 and shown in Figures 4.3.5 and 4.3.6 respectively.

Table 4.3.5 Canadian Population (1996 Census, Source: Statistics Canada)

Provinces	Population Exposed
Alberta	2,696,826
British Columbia	3,724,500
Manitoba	1,113,898
New Brunswick	738,133
Newfoundland	551,792
Nova Scotia	909,282
Ontario	10,753,573
Quebec	7,138,795
Saskatchewan	990,237

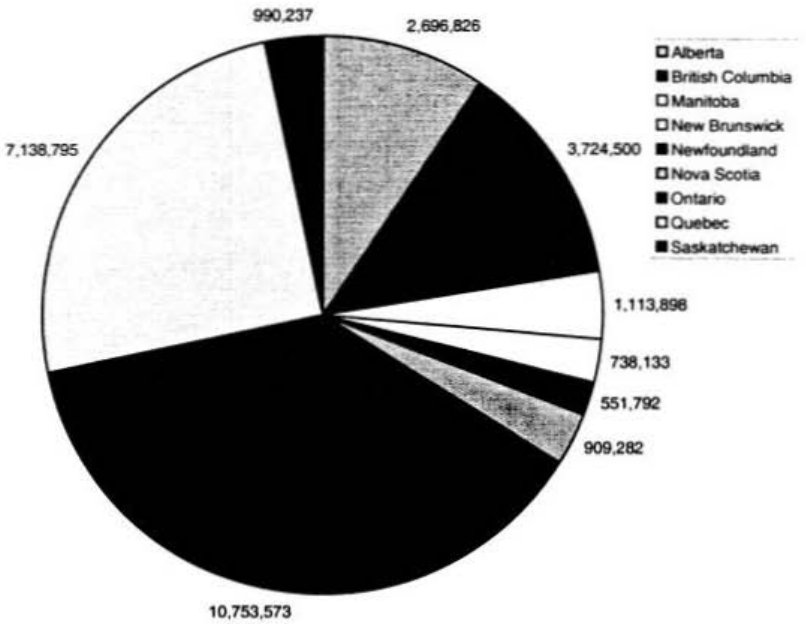

Figure 4.3.4 Distribution of Canadian Population (1996 Census)

Table 4.3.6 Estimated Cancer Cases in the Communities from Different Exposure Pathways

Provinces	Inhalation-Only Exposure		Dermal Exposure		Normal Shower		Water Ingestion	
	Winter	Summer	Winter	Summer	Winter	Summer	Winter	Summer
Alberta	< 1	33.08	< 1	2.44	< 1	35.52	3.49	8.90
British Columbia	43.70	46.70	3.24	3.43	46.93	50.13	12.02	12.42
Manitoba	72.06	154.24	4.45	9.04	76.51	163.28	11.47	22.43
New Brunswick	14.67	54.15	1.02	3.31	15.70	57.46	3.18	8.45
Newfoundland	< 1	< 1	< 1	< 1	< 1	< 1	0.46	0.82
Nova Scotia	28.07	89.80	1.87	5.37	29.94	95.17	5.26	13.49
Ontario	44.04	284.81	3.57	19.25	47.61	304.06	23.75	55.87
Quebec	49.24	385.66	3.84	24.20	53.08	409.86	18.43	63.32
Saskatchewan	42.25	65.73	2.71	4.05	44.96	69.78	7.28	10.42

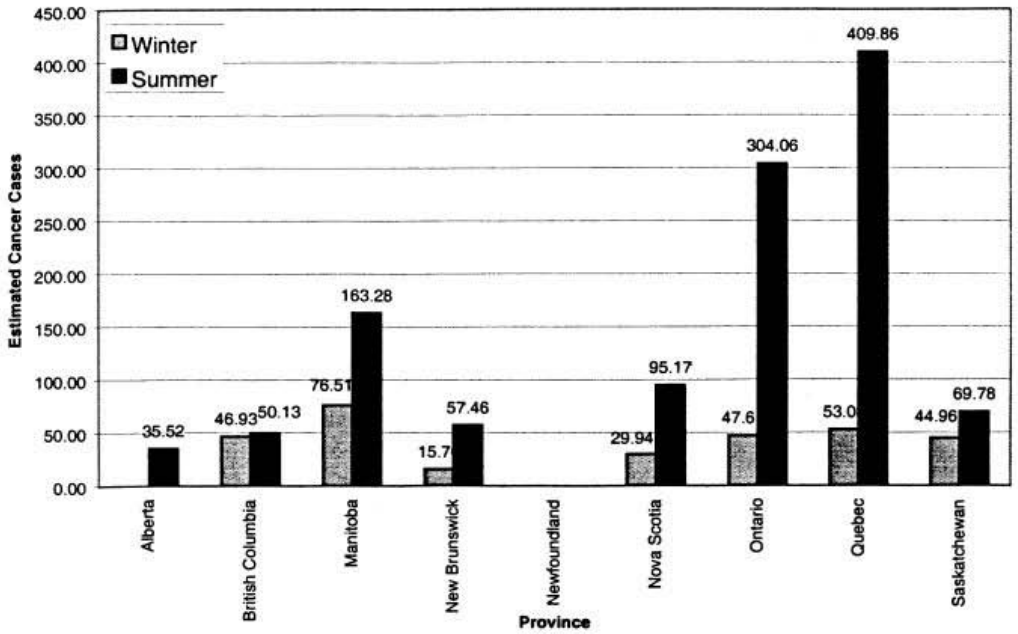

Figure 4.3.5 Estimated Cancer Cases in the Communities from Normal Shower (10 minutes)

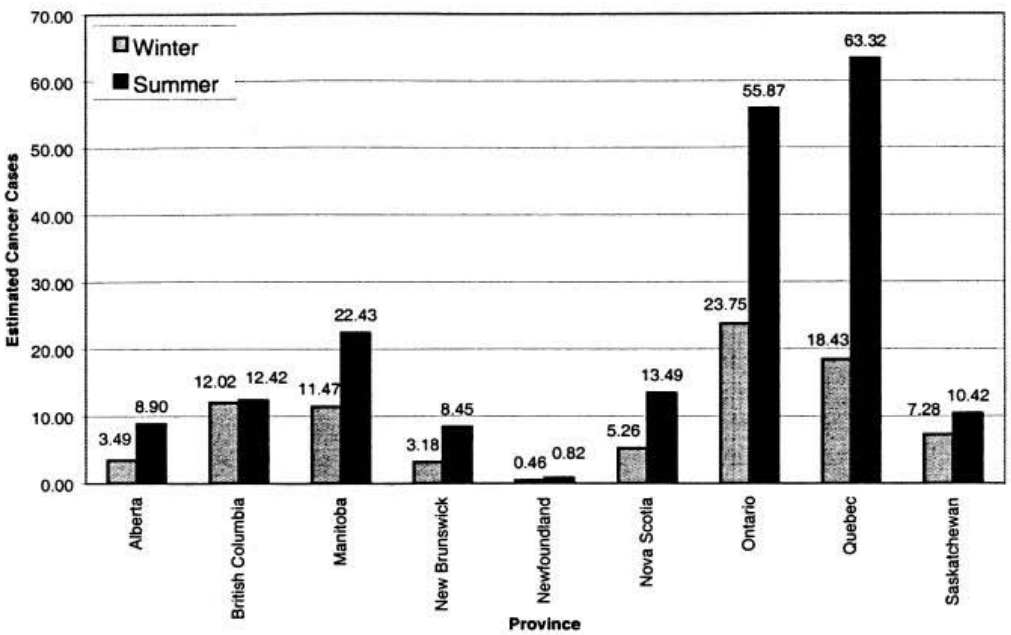

Figure 4.3.6 Estimated Cancer Cases in the Communities from Water Ingestion of 2L/day

A deterministic approach is adopted in the risk analysis. From the data, it is observed that chloroform concentrations in summer are higher than in winter. The

province of Manitoba has the highest chloroform level in water in both the seasons. The level in summer exceeded the interim maximum acceptable concentration (IMAC) established by Health Canada. Nova Scotia has the second highest concentration followed by New Brunswick in summer. In winter, Saskatchewan has the second highest chloroform concentration followed by Nova Scotia. The concentration levels in Newfoundland are found to be very low whereas in the following chapter it is observed that the levels are significantly high. This apparent contradiction can be explained by the fact that in the National Survey, the water samples were collected from St. John's only, where the concentration is generally low. St. John's does not appropriately reflect the true scenario of the province. In the Newfoundland Study (1998), water samples were collected from three different communities including St. John's. Except for St. John's, the other two communities have high chloroform concentration in their drinking water.

Standard shower duration of 10 minutes was considered while estimating risk from normal shower. Lifetime risk from normal shower was found to be highest in Manitoba in both the seasons. The risk reduced by 47% in winter. This province also had the highest risk from water ingestion. The risk reduction in this case was 50% in winter. In summer, the risk from normal shower was almost 14 times than the risk from ingestion in Manitoba. In winter, the corresponding risk was 7 times. Risk from inhalation-only exposure was more than that from dermal exposure for all the provinces. Risk from ingestion in summer was 20 times in Manitoba than that in Newfoundland. In winter it was 10 times. Risk from ingestion was almost same in Alberta and Newfoundland. Risk from ingestion in Newfoundland was almost equal in both the seasons.

From the results it can be concluded that number of cancer cases is highest in Quebec (summer) and Ontario (winter) for ingestion. For normal shower, cancer cases in the exposed population are highest in Quebec (summer) and Manitoba (winter).

Chapter 5

Risk Assessment of THMs in Newfoundland Drinking Water

In this section the health risk associated with multiple use of chlorinated tap water in Newfoundland, Canada is estimated. The raw water is subject to chlorination during water treatment. To examine the effect of seasonal variation, water samples were collected in two stages. A deterministic approach is adopted to estimate the various health-related risks in the current section. A probabilistic risk analysis was also conducted and is presented in Chapter 6.

For conducting risk assessment in the study, chloroform concentrations are considered only due to their significant presence and importance. The chloroform concentrations measured in two stages of the study are shown in Table 5.1 and Figure 5.1. Figures 5.2 and 5.3. show levels of total THMs and chloroform in the three communities in both summer and winter respectively. Figure 5.4. shows the total THMs concentrations in both the seasons.

Table 5.1 Mean Chloroform Concentrations for Two Seasons, Newfoundland Study

Communities	Mean Chloroform Concentration (μg/L)		Average Values for Two Seasons (μg/L)
	Summer	Winter	
Clarenville	468	450	459
Shoal Harbour	262	184	223
St. John's	44	39	41

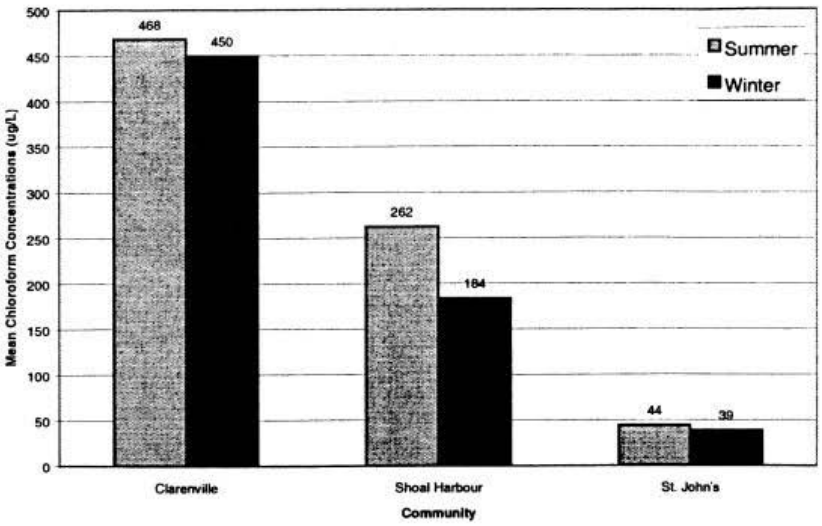

Figure 5.1 Mean Chloroform Concentrations (μg/L) for Two Seasons, Newfoundland Study

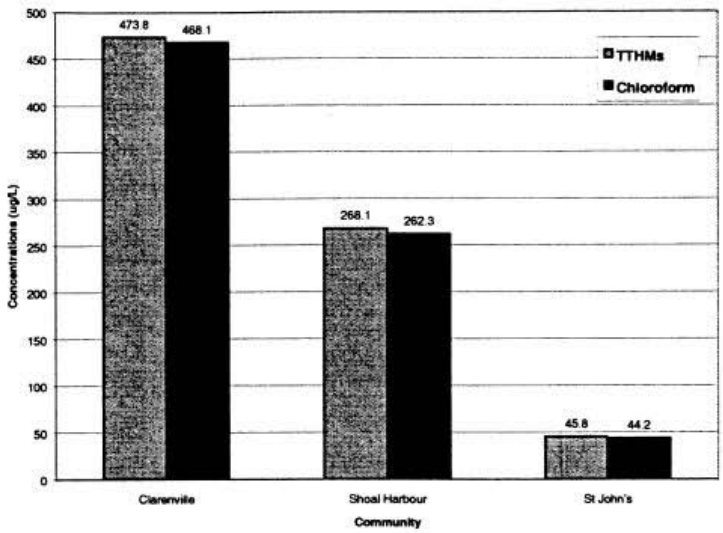

Figure 5.2 Total THMs and Chloroform Levels for Summer (Mean Concentrations), Newfoundland Study

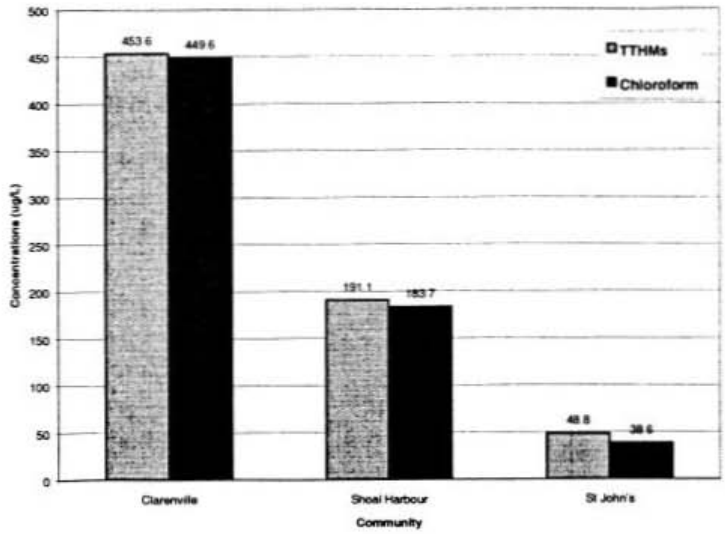

Figure 5.3 Total THMs and Chloroform Levels for Winter (Mean Concentrations), Newfoundland Study

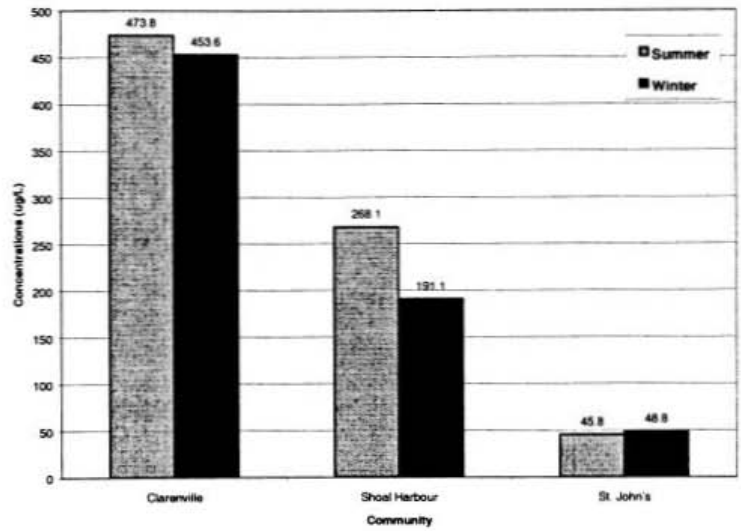

Figure 5.4 Total THMs Concentrations (Mean) for Two Seasons, Newfoundland Study

5.1 Risk Estimation for Normal Shower

As mentioned earlier, the model used by Jo et al. (1990) is used in the current study. The chloroform dose from inhalation exposure is estimated using Equation 3.2.1.

Three different shower duration of 10 minutes, 15 minutes, and 20 minutes are considered. The values of shower air concentrations (Ca) corresponding to different chloroform concentrations in the Newfoundland Study (Table 5.2) are estimated from Equation 3.2.2. All the lifetime risks estimated are risks of getting cancer over an individual's lifetime due to various exposures.

Table 5.2 Shower Air Concentrations (Ca) for Newfoundland Study, 1998

Communities	Air ($\mu g/m^3$) Concentration Values	
	Summer	Winter
Clarenville	4790.17	4596.92
Shoal Harbour	2640.39	1819.33
St. John's	362.11	303.62

The values of Ca from Table 5.2 are put in the Equation 3.2.1 to obtain the values of chloroform dose from an inhalation-only exposure (Di). The study by Jo et al (1990) considered shower duration of 10 minutes. It has been assumed that the model developed in that study can be extended to consider shower duration of any time periods like 15 and 20 minutes. The chloroform dose from dermal exposure is calculated using Equation 3.2.3.

The breath concentrations after normal shower are calculated using Equation 3.2.4. The breath concentrations after normal shower estimated for Newfoundland Study is given in Table 5.3. The breath concentrations obtained after inhalation-only exposures are determined using Equation 3.2.5. The breath concentrations obtained after inhalation-only exposures estimated for Newfoundland Study are given in Table 5.3.

Table 5.3 Breath Concentrations ($\mu g/m^3$) Obtained after Normal Shower and Inhalation-Only Exposure, Newfoundland Study, 1998

Communities	Normal Shower		Inhalation-Only Exposure	
	Summer	Winter	Summer	Winter
Clarenville	212.10	203.83	121.53	116.76
Shoal Harbour	120.13	85.00	68.48	48.22
St. John's	22.66	20.16	12.25	10.81

Using Equation 3.2.5a, the value of F is evaluated. The values of Di and F are put in Equation 3.2.3 to estimate the values of chloroform doses from dermal exposure (Dd). The respective doses from inhalation-only and dermal exposures are then added to obtain the chloroform doses from normal shower.

The cancer risks (Pdi) from inhalation-only exposures are determined by putting the values of chloroform doses from inhalation-only exposures (Di) in Equation 3.2.7. The calculated values are shown in Table 5.4 (10 minutes, 15 minutes, and 20 minutes shower) and Figure 5.5 respectively.

Table 5.4 Lifetime Risk (Pdi) from Inhalation-Only Exposure

Communities	Lifetime Risk					
	Summer			Winter		
	10 minutes	15 minutes	20 minutes	10 minutes	15 minutes	20 minutes
Clarenville	5.99×10^{-4}	8.99×10^{-4}	11.98×10^{-4}	5.75×10^{-4}	8.62×10^{-4}	11.50×10^{-4}
Shoal Harbour	3.30×10^{-4}	4.95×10^{-4}	6.60×10^{-4}	2.28×10^{-4}	3.41×10^{-4}	4.55×10^{-4}
St. John's	0.45×10^{-4}	0.68×10^{-4}	0.91×10^{-4}	0.38×10^{-4}	0.57×10^{-4}	0.76×10^{-4}

The cancer risks (Pdd) from dermal exposures are determined by putting the values of chloroform doses from dermal exposures (Dd) in Equation 3.2.7. The calculated values are shown in Table 5.5 (10 minutes, 15 minutes, 20 minutes) and Figure 5.6 respectively.

Similarly, the cancer risks from normal shower (Pd) are determined by adding cancer risk values from inhalation-only exposure (Pdi) shown in Table 5.4, to cancer risk values from dermal exposure (Pdi) shown in Table 5.5. The final values, lifetime risk from shower (Pd) are shown in Table 5.6 (10 minutes, 15 minutes, 20 minutes) and Figure 5.7 respectively.

Table 5.5 Lifetime Risk (Pdd) from Dermal Exposure

Communities	Lifetime Risk					
	Summer			Winter		
	10 minutes	15 minutes	20 minutes	10 minutes	15 minutes	20 minutes
Clarenville	0.34×10^{-4}	0.50×10^{-4}	0.67×10^{-4}	0.32×10^{-4}	0.48×10^{-4}	0.64×10^{-4}
Shoal Harbour	0.19×10^{-4}	0.28×10^{-4}	0.37×10^{-4}	0.13×10^{-4}	0.20×10^{-4}	0.26×10^{-4}
St. John's	0.03×10^{-4}	0.04×10^{-4}	0.06×10^{-4}	0.02×10^{-4}	0.04×10^{-4}	0.05×10^{-4}

Table 5.6 Lifetime Risk (Pd) from Normal Shower

Communities	Lifetime Risk					
	Summer			Winter		
	10 minutes	15 minutes	20 minutes	10 minutes	15 minutes	20 minutes
Clarenville	6.33×10^{-4}	9.49×10^{-4}	12.65×10^{-4}	6.07×10^{-4}	9.11×10^{-4}	12.14×10^{-4}
Shoal Harbour	3.49×10^{-4}	5.23×10^{-4}	6.98×10^{-4}	2.41×10^{-4}	3.61×10^{-4}	4.81×10^{-4}
St. John's	0.48×10^{-4}	0.72×10^{-4}	0.96×10^{-4}	0.40×10^{-4}	0.61×10^{-4}	0.81×10^{-4}

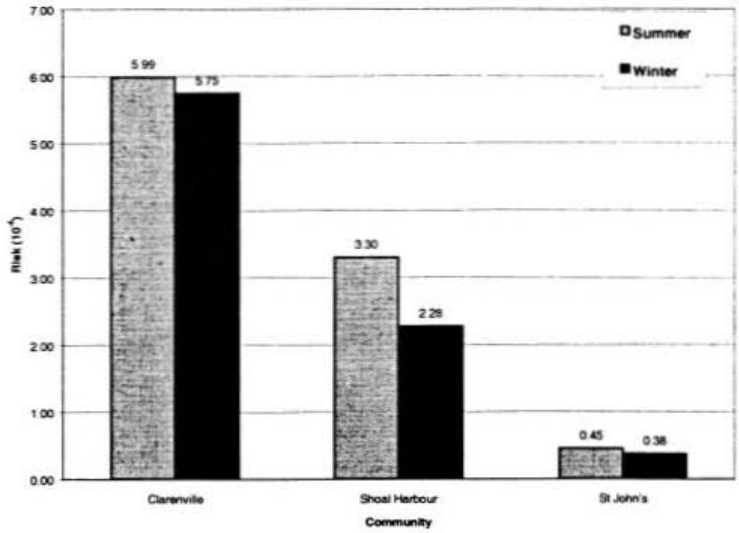

Figure 5.5 Lifetime Risk from Inhalation-Only Exposure (10 minutes)

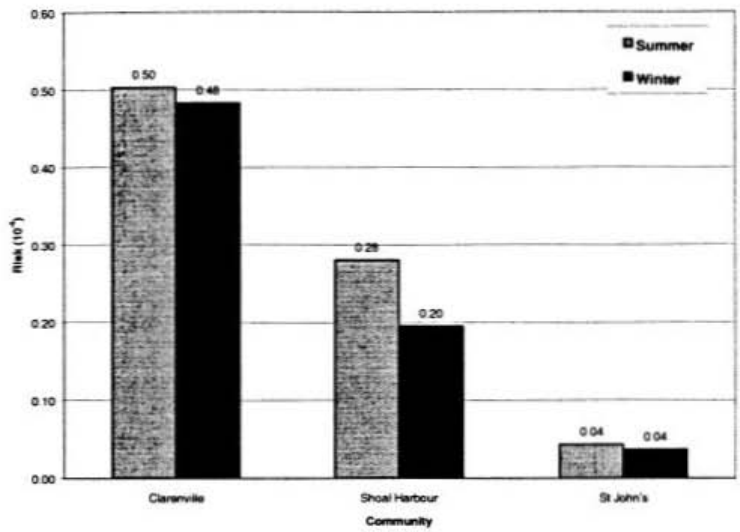

Figure 5.6 Lifetime Risk from Dermal Exposure (15 minutes)

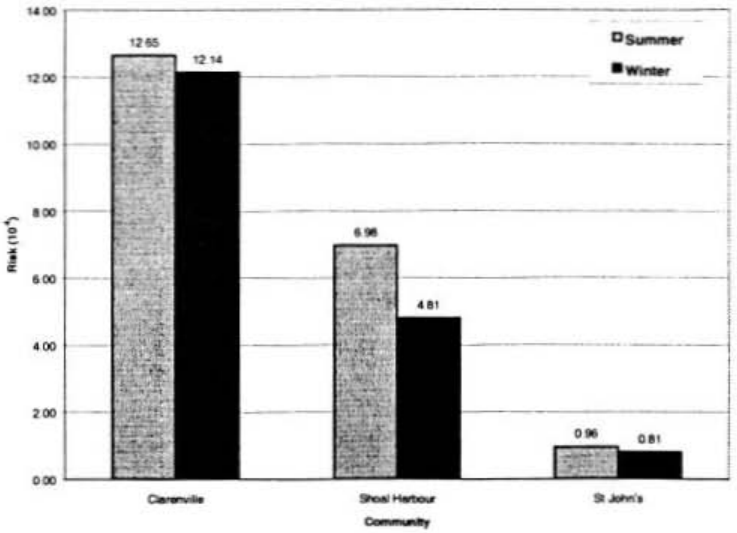

Figure 5.7 Lifetime Risk from Normal Shower (20 minutes)

5.2 Risk Estimation for Water Ingestion

The chloroform dose (Dig) from water ingestion is determined from Equation 3.2.6. The cancer risk (Pd) from water ingestion is determined by putting the value of chloroform dose from water ingestion (Dig) in Equation 3.2.7. The calculated values are shown in Table 5.7 and Figure 5.8 respectively. All the lifetime risks estimated are risks of getting cancer over an individual's lifetime due to various exposures.

Table 5.7 Lifetime Risk (Pd) from Water Ingestion of 2L Per Day

Communities	Lifetime Risk		
	Summer	Winter	Average Values
Clarenville	0.82×10^{-4}	0.78×10^{-4}	0.80×10^{-4}
Shoal Harbour	0.46×10^{-4}	0.32×10^{-4}	0.39×10^{-4}
St. John's	0.08×10^{-4}	0.07×10^{-4}	0.07×10^{-4}

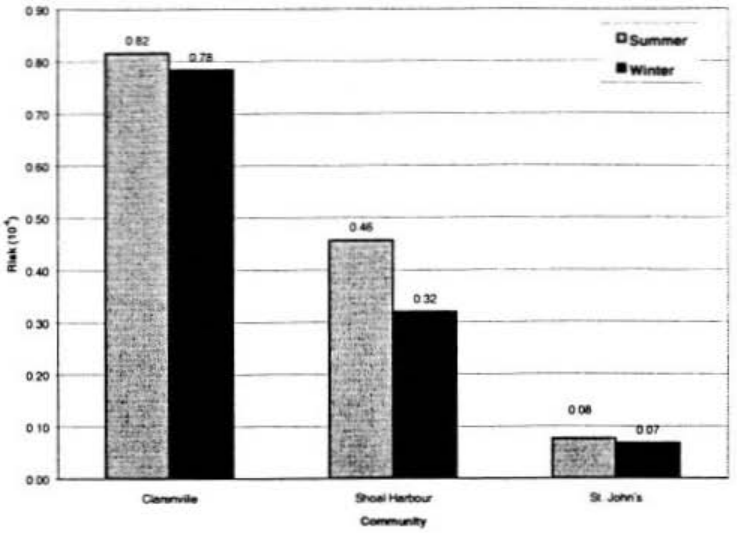

Figure 5.8 Lifetime Risk from Water Ingestion Based on 2L/day

5.3 Estimation of Cancer Cases in the Exposed Population

For determining the cancer cases in various communities of Newfoundland, the most recent available population figures are considered. The population is shown in Table 5.8 and Figure 5.9 respectively. The population of central St. John's is considered because all the samples were collected from this area. Moreover, for risk estimation purpose, population of central St. John's is assumed to represent entire St. John's city.

Table 5.8 Population of Three Communities (1996 Census)

Communities	Population Exposed
Clarenville	5335
Shoal Harbour	1500
St. John's (Central)	101936

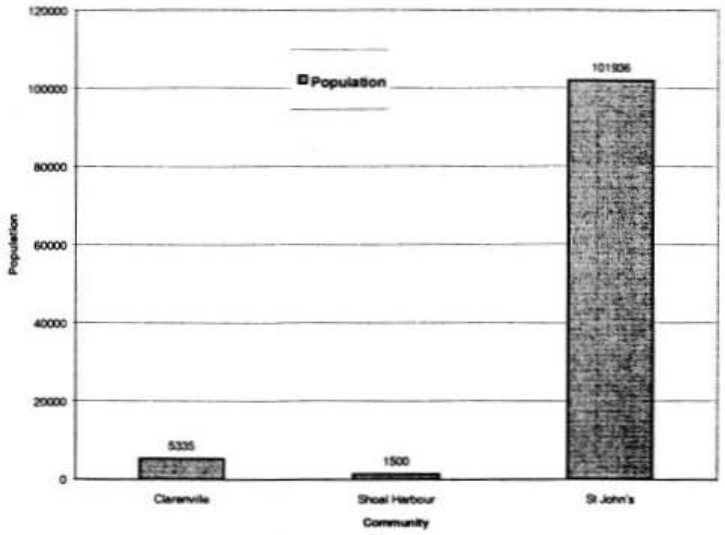

Figure 5.9 Population of the Three Communities (1996 Census)

The cancer cases, under various exposure conditions, in the three communities are estimated using Equation 3.2.8. The values are listed in Tables 5.9, 5.10, 5.11, and 5.12 respectively. The results are represented in Figures 5.10, 5.11, 5.12, and 5.13 respectively.

Table 5.9 Estimated Cancer Cases in the Communities from Inhalation-Only Exposure

Communities	Estimated Cancer Cases								
	Summer			Winter			Average Values		
	10 minutes	15 minutes	20 minutes	10 minutes	15 minutes	20 minutes	10 minutes	15 minutes	20 minutes
Clarenville	3.20	4.79	6.39	3.07	4.60	6.13	3.13	4.70	6.26
Shoal Harbour	0.50	0.74	0.99	0.34	0.51	0.68	0.42	0.63	0.84
St. John's	4.62	6.92	9.23	3.87	5.81	7.74	4.24	6.36	8.49

Table 5.10 Estimated Cancer Cases in the Communities from Dermal Exposure

Communities	Estimated Cancer Cases								
	Summer			Winter			Average Values		
	10 minutes	15 minutes	20 minutes	10 minutes	15 minutes	20 minutes	10 minutes	15 minutes	20 minutes
Clarenville	0.18	0.27	0.36	0.17	0.26	0.34	0.18	0.26	0.35
Shoal Harbour	0.03	0.04	0.06	0.02	0.03	0.04	0.02	0.04	0.05
St. John's	0.29	0.44	0.59	0.25	0.38	0.50	0.27	0.41	0.55

Table 5.11 Estimated Cancer Cases in the Communities from Normal Shower

Communities	Estimated Cancer Cases								
	Summer			Winter			Average Values		
	10 minutes	15 minutes	20 minutes	10 minutes	15 minutes	20 minutes	10 minutes	15 minutes	20 minutes
Clarenville	3.37	5.06	6.75	3.24	4.86	6.48	3.31	4.96	6.61
Shoal Harbour	0.52	0.78	1.05	0.36	0.54	0.72	0.44	0.66	0.88
St. John's	4.91	7.37	9.82	4.12	6.18	8.24	4.52	6.77	9.03

Table 5.12 Estimated Cancer Cases in the Communities from Water Ingestion of 2L per day

Communities	Estimated Cancer Cases		Average Values
	Summer	Winter	
Clarenville	0.44	0.42	0.43
Shoal Harbour	0.07	0.05	0.06
St. John's	0.79	0.69	0.74

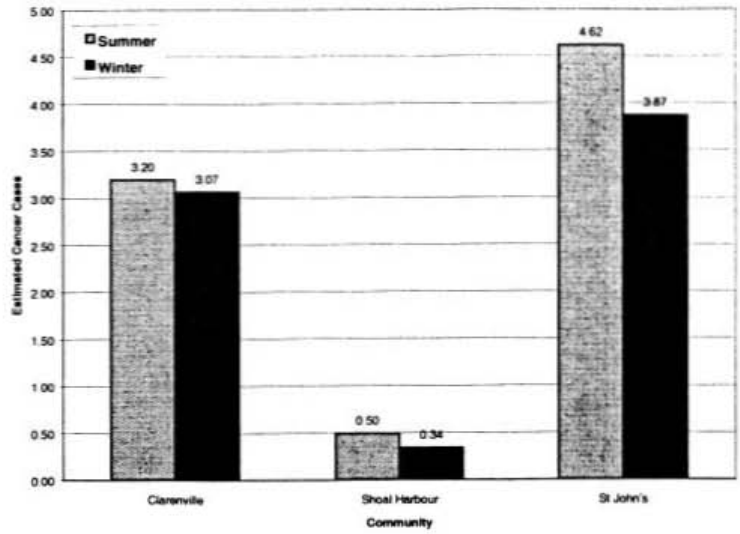

Figure 5.10 Estimated Cancer Cases in the Communities from Inhalation-Only Exposure (10 minutes)

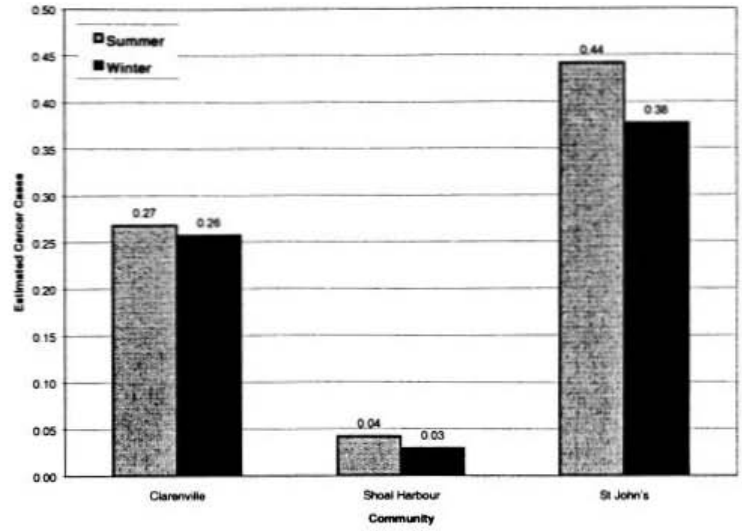

Figure 5.11 Estimated Cancer Cases in the Communities from Dermal Exposure (15 minutes)

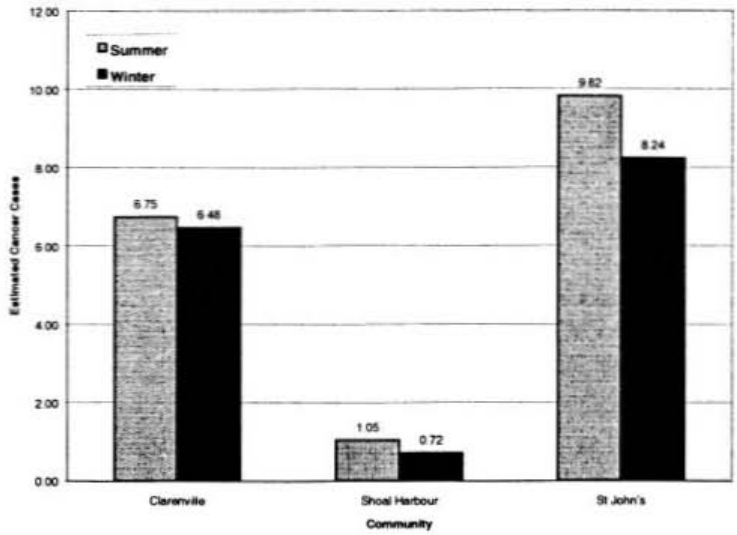

Figure 5.12 Estimated Cancer Cases in the Communities from Normal Shower (20 minutes)

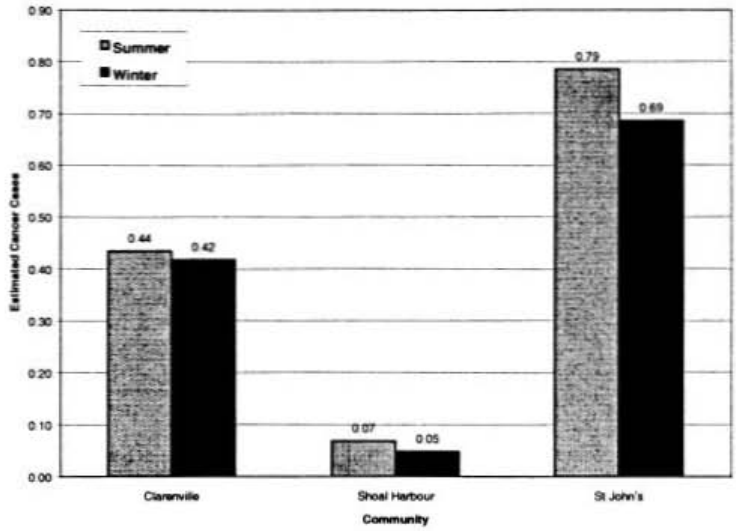

Figure 5.13 Estimated Cancer Cases in the Communities from Water Ingestion Based on 2L/day

The risk assessment presented in this chapter is based on a deterministic approach. From the results of the laboratory analysis, it can be said that trihalomethanes contain mostly chloroform. Figures 5.2 and 5.3 show levels of total THMs and

chloroform in the three communities in both summer and winter respectively. From these figures it is obvious that the chloroform constitutes more than 90% of the total THMs and less than 10% are other by-products.

The next important member in this group of compounds in terms of presence is dichloro-bromomethane. The key factor for the entire health risk estimation is the concentration of chloroform in the drinking water. From Table 5.1, it is observed that chloroform concentration in the winter season is lower than that in the summer. This can be attributed to many compounding factors. However, it is mostly due to lesser chlorination practice in winter. The other factors influencing formation of trihalomethanes / chloroform in treated water in the light of seasonal variation is discussed in section 2.1. Lower trihalomethanes / chloroform level in winter has been reported in several studies. In St. John's, this seasonal variation is not sharply observed. This is because, although the sample collection periods in the study were referred as summer and winter seasons, the months during which samples were collected do not truly represent those seasons.

The concentration levels of trihalomethanes / chloroform in drinking water of Clarenville and Shoal Harbour exceed the permissible limit (100 μg/L) set by Health Canada. It is satisfactory to note that in St. John's, the levels are well within the permissible limit. Although thorough investigation of the factors responsible for high occurrence of trihalomethanes / chloroform in the two above-mentioned communities was beyond the scope of this research, some general observations can be reported. High natural organic matter (NOM) content and improper chlorination / water treatment practices in the two communities appeared to be the major causes. The province of

Newfoundland has a scattered population. Several small water treatment facilities were therefore developed in the closed vicinities of the local population. These plants were not of high efficient standards. This paved the way for many drinking water related problems in the past.

Samples were collected from many locations in each community and each sample was analysed for the concentration levels. However, for the purpose of deterministic risk analysis, average values of the concentrations were used for simplicity. So for three communities three concentration levels were obtained. The mean concentration values may not have reflected the true nature of the set of data. Considering all the three communities, in summer, the chloroform concentration ranged from 0 μg/L to 512 μg/L, and in winter from 3 μg/L to 557 μg/L.

Lifetime risk from inhalation-only exposure during showering activity increased with longer shower duration. For both the seasons, lifetime risk from inhalation increased by about 50% for a shower of 15 minutes when compared to a 10 minutes shower. The risk increase was approximately 33% for a 20 minutes shower when compared to a 15 minutes shower. In summer, the risk varied from 0.45×10^{-4} to 5.99×10^{-4} (10 minutes), 0.68×10^{-4} to 8.99×10^{-4} (15 minutes), and 0.91×10^{-4} to 11.98×10^{-4} (20 minutes). In winter, the risk ranged from 0.38×10^{-4} to 5.75×10^{-4} (10 minutes), 0.57×10^{-4} to 8.62×10^{-4} (15 minutes), and 0.76×10^{-4} to 11.50×10^{-4} (20 minutes). In winter the risk reduced approximately by 4% for Clarenville, 31% for Shoal Harbour, and 16% for St. John's when compared to that in summer season. The figures were consistent for shower durations of 10 minutes, 15 minutes, and 20 minutes respectively.

Lifetime risk from dermal exposure during showering activity increased with longer shower duration. In summer, risk increase from inhalation ranged from 33% to 47% for a shower of 15 minutes over a 10 minutes shower. The risk increase ranged from 32% to 50% for a 20 minutes shower over a 15 minutes shower. During winter, risk increase from inhalation ranged from 50% to 100% for a shower of 15 minutes over a 10 minutes shower. The risk increase ranged from 25% to 33% for a 20 minutes shower over a 15 minutes shower. In summer, the risk varied from 0.03×10^{-4} to 0.34×10^{-4} (10 minutes), 0.04×10^{-4} to 0.50×10^{-4} (15 minutes), and 0.06×10^{-4} to 0.67×10^{-4} (20 minutes). In winter, the risk varied from 0.02×10^{-4} to 0.32×10^{-4} (10 minutes), 0.04×10^{-4} to 0.48×10^{-4} (15 minutes), and 0.05×10^{-4} to 0.64×10^{-4} (20 minutes). In winter, the reduction in risk ranged from approximately 5.8% to 33.3% (10 minutes), 0% to 28.5% (15 minutes) and 4.4% to 29.7% (20 minutes) respectively.

Lifetime risk from water ingestion based on intake of 2 litres per day, ranged from 0.08×10^{-4} to 0.82×10^{-4} (summer), and 0.07×10^{-4} to 0.78×10^{-4} (winter). In winter, the risk reduced by 4.8% for Clarenville, 30.4% for Shoal Harbour, and 12.5% for St. John's respectively.

Risk from normal shower activity is the summation of risk from inhalation and dermal exposures. Inhalation and dermal exposures are the two possible sources of exposure during a normal shower activity. In risk assessment cancer risk from normal shower is often compared with risk from water ingestion. In the study, it is observed that the risk from normal shower is significantly more than that from water ingestion. For instance, risk from a 10-minute shower is 5.7 to 7.8 times more than risk from a daily intake of two litres chlorinated tap water. Estimated cancer cases are found to be more in

St. John's than Clarenville and Shoal Harbour. This is because St. John's has a higher population than the other two communities.

Chapter 6

Probabilistic Risk Analysis

6.1 Introduction

Probability and statistics have played a significant role in engineering applications. The rationality and utility of probabilistic models is of phenomenal interest to all engineers. There has been a widespread multipurpose adoption of such models in engineering disciplines. A unified probabilistic approach to water-resources planning, design, construction planning, environmental engineering, and many other subjects has led to the development of various tools for more sophisticated analysis.

In the current study, limited number of drinking water samples has been collected due to time and resource constraints and deterministic risk analysis was described in Chapter 5 based on the limited sets of data without considering variability and uncertainty in the analysis. To include uncertainty effects in the health risk analysis, quantification of uncertainty measures is important. To address these issues, probabilistic analysis is incorporated in the risk assessment. When adopting a probabilistic approach, one cannot fully set aside deterministic models. Although probabilistic methods usher a scientific, workable alternative tool to resolve engineering problems, they are, in essence complementary to physically based deterministic models.

In this chapter, probabilistic risk analysis of trihalomethanes is described. Various concentration levels in drinking water obtained in the Newfoundland Study (1998) are considered. As chloroform constitutes a significant portion of total trihalomethanes (TTHMs), for the purpose of analysis, chloroform concentration is considered to be equal to the total trihalomethanes concentration. The procedure proposed for probabilistic risk

analysis, which includes use of normal probability plot and software @RISK is discussed in the following sections.

For the purpose of analysis, the concentration levels for the two seasons are combined since limited number of data points is available. Hence, risk using probabilistic model is calculated on yearly basis rather than on seasonal basis.

6.2 Normal Probability Plot

For each of the three communities, the data points are combined to produce the normal probability plots and the box plots. They are shown in Figures 6.1, 6.2, 6.3, 6.4, 6.5, and 6.6 respectively. For all the three communities, the p values are greater than 0.1. Hence, it is concluded that the data set for each community follows normal distribution. Table 6.1 gives the summary of distribution parameters.

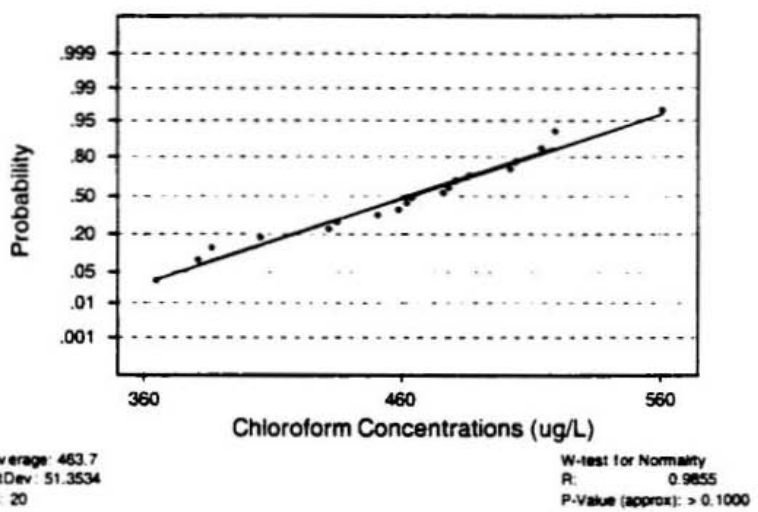

Figure 6.1 Normal Probability Plot of Chloroform Concentrations for Clarenville

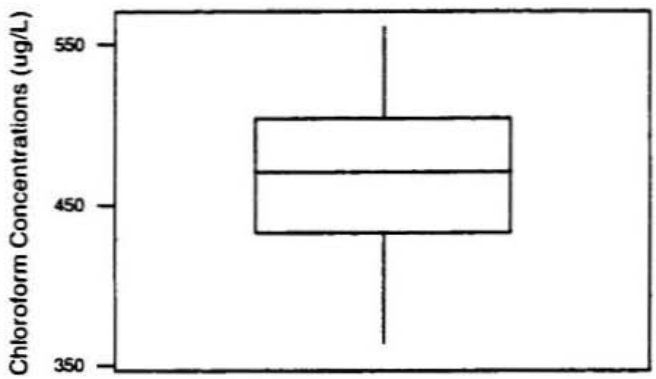

Figure 6.2 Box Plot of Chloroform Concentrations for Clarenville

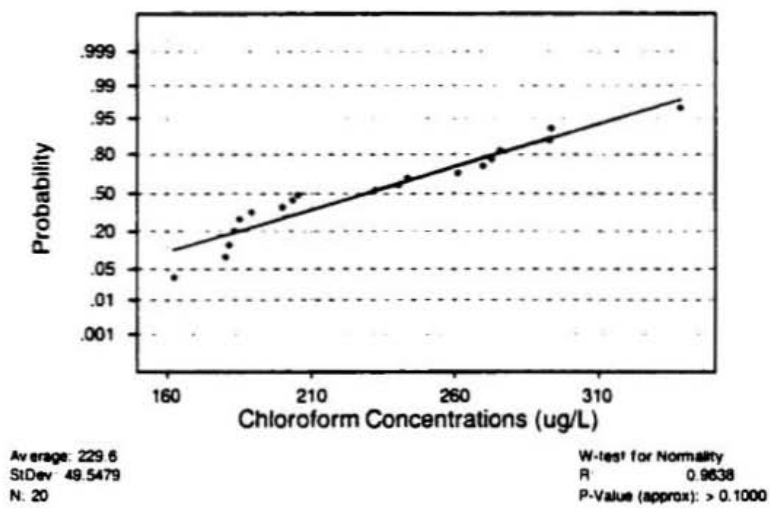

Figure 6.3 Normal Probability Plot of Chloroform Concentrations for Shoal Harbour

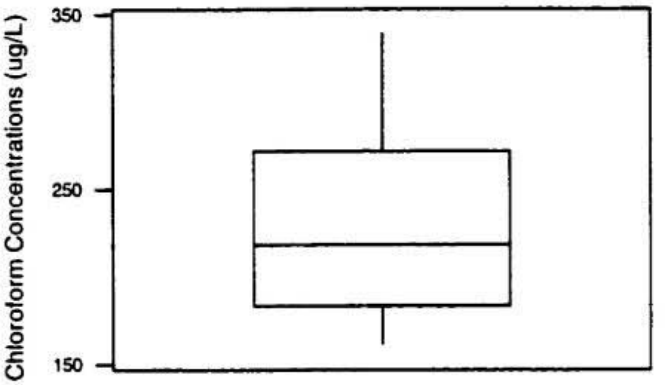

Figure 6.4 Box Plot of Chloroform Concentrations for Shoal Harbour

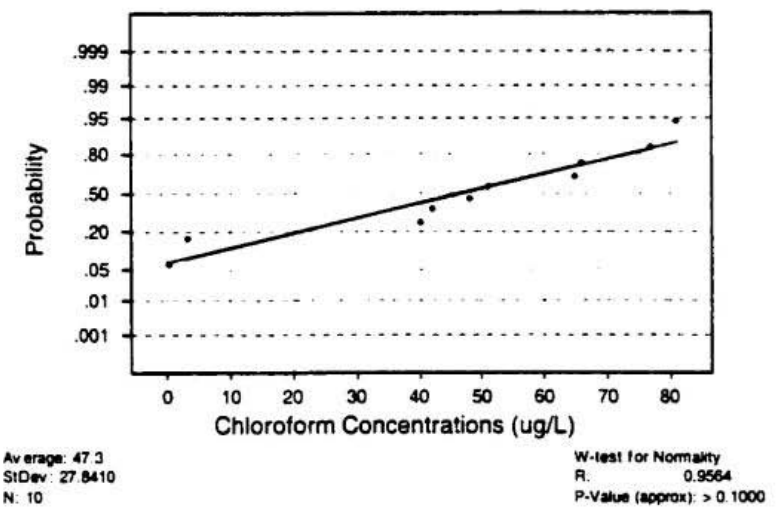

Figure 6.5 Normal Probability Plot of Chloroform Concentrations for St. John's

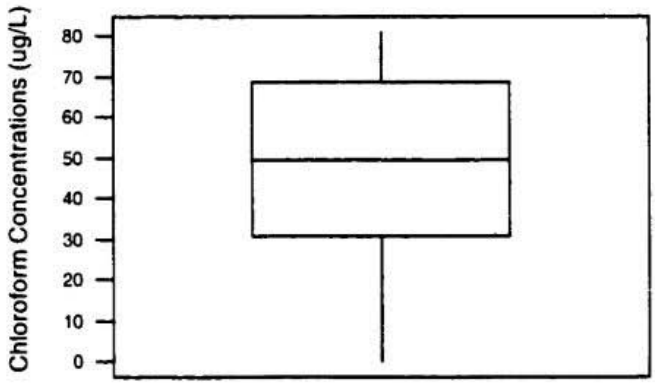

Figure 6.6 Box Plot of Chloroform Concentrations for St. John's

Table 6.1 Summary of Distribution Parameters

Communities	Normal Distributions (μg/L) (μ, σ)
St. John's	(47.3, 26.4)
Clarenville	(463.7, 50)
Shoal Harbour	(229.6, 48.3)

6.3 @RISK Analysis

In order to perform risk analysis and simulation, Window® version of the software @RISK was used. @RISK, developed by Palisade Corporation, is an add-in for Microsoft® Excel or Lotus® 1-2-3. With @RISK, risk analysis model can be designed and any uncertainty present in the estimates can be explicitly included to generate results

that present all possible outcomes. This software uses a technique called "simulation" to combine all the uncertainties identified in the modelling situation. The @RISK has all the tools for setting up, executing and viewing the results of Risk Analyses. The uncertain cell values in Excel are defined as probability distributions using functions. The various functions enable us to specify a different distribution type for cell values. @RISK is able to specify and execute simulations of Excel or 1-2-3 models. It can perform both Monte Carlo and Latin Hypercube sampling techniques. The output distributions from @RISK simulations can be presented using high quality graphs. These graphs may be further transported to Excel or 1-2-3 for enhancement. (Guide to @RISK, 1997). The generation of stochastic output is possible following any of the three combinations shown in Table 6.2. In the present analysis, the inputs are stochastic, the system is deterministic, and the outputs are stochastic. It is assumed that the regression models are deterministic.

Table 6.2 Generation of Stochastic Output

Input	System	Output
Stochastic	Deterministic	Stochastic
Deterministic	Stochastic	Stochastic
Stochastic	Stochastic	Stochastic

6.3.1 Risk Estimation for Normal Shower

The model by Jo et al (1990) is used in the analysis. In order to estimate the chloroform dose from an inhalation-only exposure, Equation 3.2.1 is used. The cancer risks (Pdi) from inhalation-only exposures are determined by substituting the values of chloroform doses from inhalation-only exposures (Di) in Equation 3.2.7. Each input component of Equation 3.2.1 and 3.2.7 is ascertained. The various input variables of the

model used by Jo et al are assumed to follow specific distributions. They are listed in Table 6.3. In ascertaining the nature of these distributions, generally, the statistical data is collected before such an analysis. However, that was not the objective of the study. Therefore assumptions were made on the choice of distributions for demonstration purpose only and due to lack of availability of the data and time. Dose and Risk are selected as the output variables. Using the Latin Hypercube sampling technique, 1000 iterations were performed which provide both the dose and risk values.

Similarly, the dose and risk from dermal exposure is estimated using Equations 3.2.4, 3.2.5, 3.2.5a, 3.2.3, and 3.2.7 respectively.

6.3.2 Risk Estimation for Water Ingestion

The chloroform dose (Dig) and risk (Pd) from water ingestion are determined from Equation 3.2.6 and 3.2.7. All the risk values are plotted in Figures 6.7, 6.8, and 6.9 respectively.

The Figures 6.7, 6.8, and 6.9 show the uncertainty associated with the increase in risk level due to inhalation, dermal and ingestion respectively. For example, from Figure 6.9, it is seen that the uncertainty in the increase of risk value by 2.25×10^{-4} due to inhalation of chloroform for St. John's is 18%.

In this analysis, the concentration levels for the two seasons are not considered separately. So the seasonal variation is not reflected in the analysis. The mean and the various percentile risk values are listed in Table 6.4. The risk values obtained from the deterministic analysis are also listed in the same table. These values are the seasonal

averages. The deterministic risk values are compared with the values obtained from @RISK analysis.

For Clarenville, the deterministic risk estimate due to inhalation exposure corresponds to 20% (approximately) percentile value of the @RISK analysis. Similarly, the deterministic risk estimate due to dermal exposure corresponds to 22% (approximately) percentile value of the @RISK analysis. The deterministic risk estimate due to ingestion corresponds to 22% (approximately) percentile value of the @RISK analysis.

For Shoal Harbour, the deterministic risk estimate due to inhalation exposure corresponds to 20% (approximately) percentile value of the @RISK analysis. Similarly, the deterministic risk estimate due to dermal exposure corresponds to 30% (approximately) percentile value of the @RISK analysis. The deterministic risk estimate due to ingestion corresponds to 21% (approximately) percentile value of the @RISK analysis.

For St. John's, the deterministic risk estimate due to inhalation exposure corresponds to 28 % (approximately) percentile value of the @RISK analysis. Similarly, the deterministic risk estimate due to dermal exposure corresponds to 40% (approximately) percentile value of the @RISK analysis. The deterministic risk estimate due to ingestion corresponds to 26% (approximately) percentile value of the @RISK analysis.

Table 6.3 Summary of Input Parameters for Risk Analysis

Parameters	**Chloroform Concentrations (μg/L)**	**Absorption Efficiency via Respiratory System (Er) (Unitless)**	**Breathing Rate (R) (m^3/min)**	**Shower Duration (T) (min)**	**Body Weight (Wt) (kg)**	**Absorption Efficiency via Gastro-Intestinal Tract (Ei) (Unitless)**	**Ingestion Rate (Aw) (L/day)**
Nature of Distribution	Normal	N/A	Triangular	Normal	Uniform	N/A	Triangular
Values	Table 6.1	0.77	Minimum (a) = 0.005 Most Likely (b) = 0.014 Maximum (c) = 0.03	$\mu = 10$ $\sigma = 3$	a = 5 b = 80	1	Minimum (a) = 0.5 Most Likely (b) = 2 Maximum (c) = 3.5

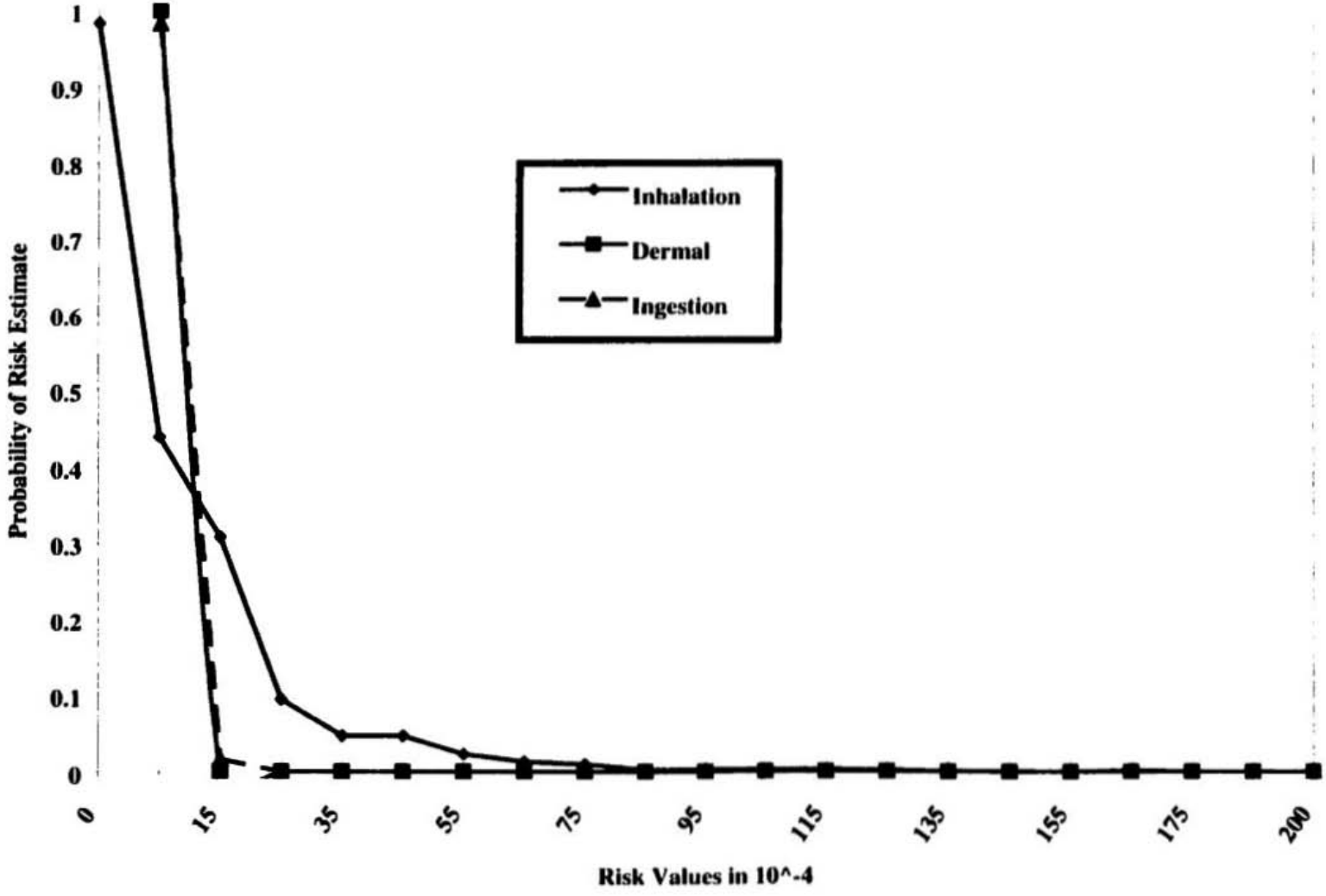

Figure 6.7 Risk Distribution for Clarenville

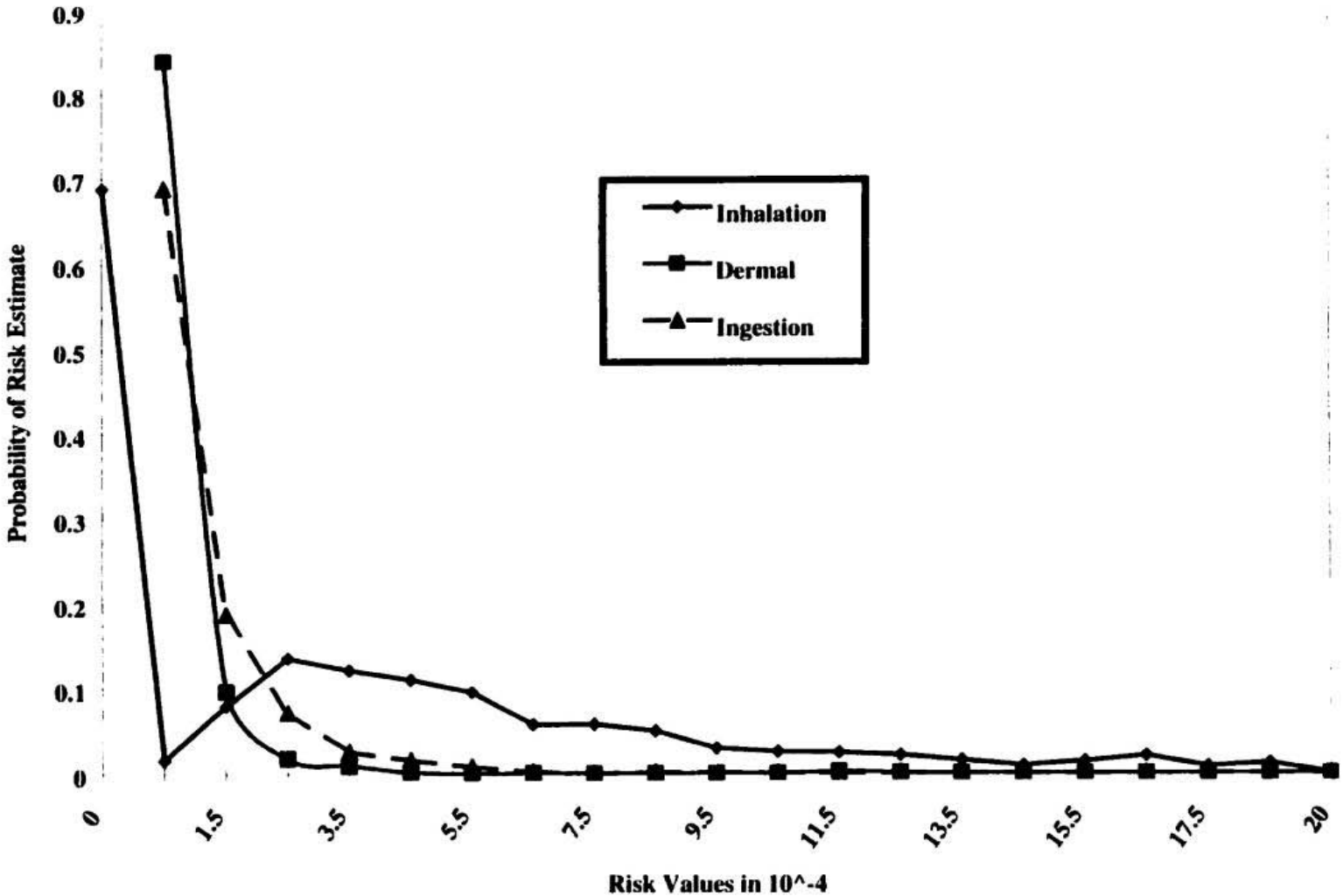

Figure 6.8 Risk Distribution for Shoal Harbour

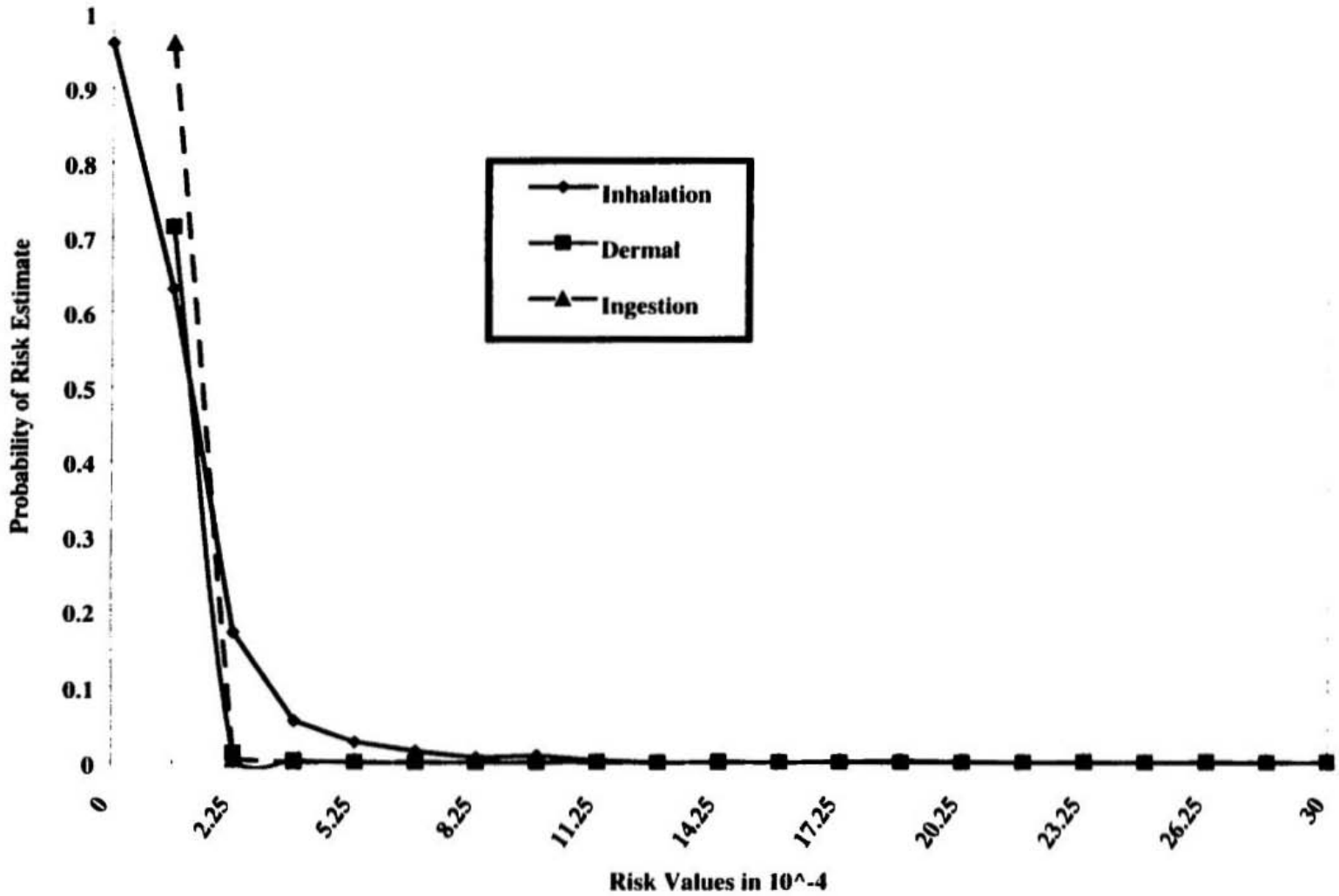

Figure 6.9 Risk Distribution for St. John's

Table 6.4 Summary of Risk Values

Exposure		Inhalation			Dermal			Ingestion		
Communities		Claren-ville	Shoal Harbour	St. John's	Claren-ville	Shoal Harbour	St. John's	Claren-ville	Shoal Harbour	St. John's
Risk Values										
Probabilistic Values from @RISK	Mean	1.81E-03	8.77E-04	1.45E-04	1.01E-04	5.43E-05	1.39E-05	2.13E-04	1.03E-04	2.10E-05
	5%	3.54E-04	1.52E-04	<1.00E-06	1.53E-05	2.83E-06	<1.00E-06	4.86E-05	2.45E-05	9.18E-07
	10%	4.40E-04	2.03E-04	6.09E-06	2.21E-05	5.69E-06	<1.00E-06	6.27E-05	2.93E-05	3.19E-06
	25%	*6.65E-04*	*3.16E-04*	*3.83E-05*	*3.62E-05*	*1.35E-05*	<1.00E-06	*8.60E-05*	*4.25E-05*	*6.70E-06*
	50%	**1.15E-03**	**5.35E-04**	**8.44E-05**	**6.22E-05**	**2.74E-05**	**3.38E-06**	**1.33E-04**	**6.45E-05**	**1.31E-05**
	75%	2.02E-03	1.00E-03	1.68E-04	1.13E-04	6.20E-05	1.42E-05	2.44E-04	1.19E-04	2.47E-05
	90%	4.30E-03	1.94E-03	3.47E-04	2.23E-04	1.21E-04	3.50E-05	4.72E-04	2.28E-04	5.15E-05
	95%	5.51E-03	2.58E-03	5.14E-04	3.34E-04	1.74E-04	6.95E-05	6.38E-04	3.33E-04	7.16E-05
(10 minutes)										
Deterministic Values (Seasonal Average)		**5.87E-04**	**2.79E-04**	**0.41E-04**	**0.33E-04**	**0.16E-04**	**0.02E-04**	**0.80E-04**	**0.39E-04**	**0.07E-04**

**Italic number shows the closest percentile values in comparison to deterministic values.*

Chapter 7

Concluding Remarks and Recommendations

7.1 Concluding Remarks

1. Among the three communities sampled, tap water of Clarenville has the highest mean trihalomethanes concentrations in both summer and winter. Total trihalomethanes concentrations in Clarenville and Shoal Harbour exceeded the interim maximum acceptable concentration (IMAC) established by Health Canada.

2. Chloroform among all the THMs has the most significant presence and highest concentration in drinking water. Chloroform constitutes more than 90% of the total THMs and less than 10% are other by-products.

3. In the present study, there is significant reduction in total trihalomethanes concentration levels in drinking water samples when the samples are boiled, aerated, filtered with activated carbon, or refrigerated for twenty-four hours. For Clarenville (winter), reduction rate is 95% for boiling, 68% both for filtration and aeration, and 48% for refrigeration.

4. Lifetime risk to an individual taking a normal shower with chlorinated water is significantly more than the risk from drinking the same. For example, in summer, the risk from a ten minute shower was found to be approximately 6-7 times more than that from drinking chlorinated water at a rate of two litres a day. However care should be taken in using these numbers since these are based on extrapolation of regression equation which was developed using data published by Joe et al. (1990). At the national level, Manitoba drinking water had the highest chloroform content in both the seasons.

5. Lifetime risk from inhalation-only exposure during showering activity increased with longer shower duration. For both the seasons, lifetime risk from inhalation increased by about 50%

for a shower of 15 minutes when compared to a 10 minutes shower. The risk increase was approximately 33% for a 20 minutes shower when compared to a 15 minutes shower. In summer, the risk varied from 0.45 $\times 10^{-4}$ to 5.99 $\times 10^{-4}$ (10 minutes shower), 0.68 $\times 10^{-4}$ to 8.99 $\times 10^{-4}$ (15 minutes shower), and 0.91 $\times 10^{-4}$ to 11.98 $\times 10^{-4}$ (20 minutes shower). In winter, the risk ranged from 0.38 $\times 10^{-4}$ to 5.75 $\times 10^{-4}$ (10 minutes shower), 0.57 $\times 10^{-4}$ to 8.62 $\times 10^{-4}$ (15 minutes shower), and 0.76 $\times 10^{-4}$ to 11.50 $\times 10^{-4}$ (20 minutes shower). In winter the risk reduced approximately by 4% for Clarenville, 31% for Shoal Harbour, and 16% for St. John's when compared to that in summer season. The figures were consistent for shower durations of 10 minutes, 15 minutes, and 20 minutes respectively.

6. Lifetime risk from dermal exposure during showering activity increased with longer shower duration. In summer, risk increase from inhalation ranged from 33% to 47% for a shower of 15 minutes over a 10 minutes shower. The risk increase ranged from 32% to 50% for a 20 minutes shower over a 15 minutes shower. During winter, risk increase from inhalation ranged from 50% to 100% for a shower of 15 minutes over a 10 minutes shower. The risk increase ranged from 25% to 33% for a 20 minutes shower over a 15 minutes shower. In summer, the risk varied from 0.03×10^{-4} to 0.34×10^{-4} (10 minutes), 0.04×10^{-4} to 0.50×10^{-4} (15 minutes), and 0.06×10^{-4} to 0.67×10^{-4} (20 minutes). In winter, the risk varied from 0.02×10^{-4} to 0.32×10^{-4} (10 minutes), 0.04×10^{-4} to 0.48×10^{-4} (15 minutes), and 0.05×10^{-4} to 0.64×10^{-4} (20 minutes). In winter, the reduction in risk ranged from approximately 5.8% to 33.3% (10 minutes), 0 to 28.5% (15 minutes) and 4.4% to 29.7% (20 minutes) respectively.
7. Lifetime risk from water ingestion based on intake of 2 litres per day, ranged from 0.08×10^{-4} to 0.82 $\times 10^{-4}$ (summer), and 0.07 $\times 10^{-4}$ to 0.78 $\times 10^{-4}$ (winter). In winter, the risk reduced by 4.8% for Clarenville, 30.4% for Shoal Harbour, and 12.5% for St. John's respectively.

8. The mean chloroform concentration values for Clarenville, Shoal Harbour, and St. John's are 463.7, 229.6, and 47.3 μg/L respectively. The standard deviation for Clarenville, Shoal Harbour, and St. John's are 50, 48.3 and 26.4 μg/L respectively.
9. Risk analysis was performed using @RISK software. Latin Hypercube sampling technique was used for simulation. One thousand iterations were performed. The simulation outputs (risks) from three exposure pathways were overlaid for obtaining the output graphs. Selected simulation statistics results are reported and compared with results from deterministic analysis.
10. The seasonal mean and median values are not significantly different for Clarenville and St. John's but significantly different for Shoal Harbour.
11. Domestic water uses such as, bathing, washing clothes, washing dishes, and cooking cause additional risk to an individual from chloroform. Besides, many people shower longer that 10 minutes and /or more than once per day. This practice is prevalent more in developing countries and countries having hot climate. Therefore, the risk associated with chloroform exposure from total household water use may be higher than that estimated by the showering rate and duration, and daily water ingestion rate (Jo et. al, 1990).
12. While doing risk estimates, one considers both voluntary and involuntary risks. Table 7.1 provides examples of some voluntary risks with the related risks involved. For example, death per billion persons with one hour of swimming is 3650. Assuming, an individual swims for an average of one hour in a week, the death risk due to year long swimming is $3650 \times 10^{-9} \times 52 = 1.9 \times 10^{-4}$. On the other hand, for a community like Clarenville, individual risk over a lifetime from a daily normal shower of ten minutes and water intake of 2 liters based on THMs concentrations in summer season is 7.15×10^{-4}. This risk value is not

alarming if compared to the death risk from swimming over a lifetime. It might be noted in this regard that the acceptable risk is one in million. For people in occupational safety and health, the baseline risk is one in ten thousand to one in one hundred thousand.

Table 7.1 Comparative Probabilities of Death for Different Activities (Wilson, 1984; and Wilson and Crouch, 1987)

	Deaths Per Billion Persons With One Hour Risk Exposure
Being vaccinated or inoculated	1.3
Exposure to radiation in a two hour altitude flight during solar flare	2.5
Living in area where snakes are present	3.8
Radiation exposure of world population in majority nuclear war (areas away from conflict)	5.0
Railroad or bus travel (USA) (Britain)	10.0 50.0
Child asleep in crib	140.0
Being struck by lightning	200.0
Coal mining (Br.)	400.0
Amateur boxing (Br.)	450.0
Climbing stairs	550.0
Coal mining (USA)	910.0
Hunting	950.0
Automobile travel	1200.0
Air travel	1450.0
Cigarette smoking	2600.0
Mountain climbing (USA)	2700.0
Boating (small boats)	3000.0
Motor scooter riding (Br.)	3000.0
Swimming	3650.0
Motor cycle rising (Canada) (USA) (Br.)	4420.0 6280.0 6600.0
Armed forces in Viet Nam	7935.0
Canoeing	10000.0
Motor cycle racing (Br.)	35000.0
Mountain climbing (Alpine)	40000.0
Professional boxing	70000.0
Being born	80000.0

One in a million risk of death from the following:
1 ½ cigarettes
50 miles by car
250 miles by air
1 ½ minutes rock climbing
6 minutes canoeing
20 minutes being a man aged 60
1 or 2 weeks' typical factory work

7.2 Recommendations

The following recommendations are made:

1. Drinking water from smaller communities in Newfoundland should be further analysed for determination of trihalomethanes concentrations. The potential of health related risk associated with multiple use of chlorinated water seems to be much more in smaller municipalities. Similar risk assessment studies are also recommended. Additional samples should be collected from each study location in order to obtain more conclusive results.
2. As health risks from inhalation and dermal exposures while taking normal showers are found to be significantly more than that from ingestion, further research is suggested in this direction.
3. Drinking water that is already in compliance with the guidelines for *Canadian Drinking Water Quality* does not require additional treatment for health-related reasons. However, there is no one easy way that consumer can remove all of the disinfectant by-products. The volatile or easily evaporating by-products like THMs can be partially removed if the consumer boils the water or lets it sit in the refrigerator overnight or simply aerates the water in a blender. Commercially available water treatment devices containing activated carbon filters are also capable of adsorbing chlorine and chlorinated disinfection by-products (CDBPs). In the process, the by-products are removed from the tap water. It is further recommended that to reduce the possible chemical and microbiological risks, the activated carbon filters are to be

replaced at the frequency recommended by the manufacturer and that the filters be flushed before every use. Given the current unregulated water treatment device industry in Canada, the consumers should be careful in choosing the appropriate products for themselves. The devices complying health- based standard certification are recommended for use (Health Canada, 1999).

4. In the present study, it is noted that the chloroform dose and the cancer risk from a single, 10-minute shower is greater than that from the daily water ingestion. The chloroform dose received from the showers and other uses of chlorinated tap water should be considered when regulatory and health agencies conduct the water quality evaluation of a chlorinated water supply.

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APPENDIX 1
WATER QUALITY DATA

Table 1 Chloroform Concentration Levels, National Survey, 1993

Province	Municipality	Chloroform Concentration (μg/L)	
		Winter	Summer
Alberta	Calgary	9.10	41.90
	Edmonton	1.00	2.70
	Lethbridge	3.00	4.10
	Red Deer	16.60	27.00
British Columbia	Chilliwack	14.70	17.80
	Kamloops	37.80	27.80
	Nanaimo	19.10	28.10
	Penticon	21.10	12.80
	Vancouver	15.40	24.70
	Victoria	3.00	3.60
Manitoba	Letellier	12.90	44.50
	Portage-La-Prairie	4.30	53.50
	Selkirk	0.50	0.40
	Whitemouth	216.20	335.90
	Winnipeg	61.40	143.40
New Brunswick	Fredericton	17.40	57.60
	Moncton	21.40	59.10
	Oromocto	44.60	126.00
	Saint John	15.60	20.10
Newfoundland	St. John's Source#1	7.50	13.30
	St. John's Source#2	2.00	3.70

Nova Scotia	Dartmouth	85.30	130.60
	Halifax	20.70	71.30
	New Glasgow	67.80	210.70
	Truro	24.30	112.20
	Barrie	2.20	2.30
	Brantford	31.30	67.70
	Guelph	0.60	1.20
Ontario	Kingston	14.40	11.50
	Grand Bend	6.60	7.60
	Mississauga	4.70	5.50
	North Bay	7.20	14.20
	Ottawa Source#1	10.30	60.10
	Ottawa Source#2	9.80	67.50
	Peterborough	50.20	99.60
	St. Catharines	4.10	4.70
	Sudbury	16.30	22.80
	Toronto	3.10	4.60
Quebec	Drummondville	33.10	91.10
	Gatineau	17.20	91.40
	Granby	26.00	54.10
	Laval	13.50	100.80
	Levis	16.60	40.30
	Pierrefonds	13.00	90.20
	Quebec	5.20	87.20
	Repentigny	4.40	23.70
	St-Jean-sur-Richelieu	4.00	21.00
	Trois-Rivieres	19.90	38.70
	Montreal	6.00	9.20
	Moose Jaw	22.90	13.60
	Prince Albert	10.80	0.30

Saskatchewan	Saskatoon	13.10	25.30
	Swift Current	71.30	95.40

Source: Health Canada, 1995

Table 2 Typical Laboratory Data

Chapter 2 * * * ENVIRONMENTAL QUALITY LABORATORY / NF * * *

REPORT DATE : July 25. 1998

LABORATORY # : N375
DATE SUBMITTED : 20/07/98
DATE SAMPLED : 17/07/98
FILE NUMBER :

SAMPLE DESCRIPTION : SAMPLE # 3
JULY 17. 1998

TEST #	DESCRIPTION	RESULT	UNITS	DATE ANALYSED	FLAGS (S)
753 W	BROMOFORM	< 1	µg / L	28/07/98	
752 W	CHLORO – DIBROMO METHANE	< 1	µg / L	28/07/98	
751 W	DICHLORO-BROMO METHANE	< 1	µg / L	28/07/98	
750 W	CHLOROFORM	66	µg / L	28/07/98	
	TOTAL THM	66	µg / L		

Approved : _____

Table 3 Water Quality Data of Clarenville Municipal Water Supply, Newfoundland

Parameters	Units	Drinking Water Limits	Sampling Data		
			YY/MM/DD	YY/MM/DD	YY/MM/DD
			87/ 09 / 28	90 / 06 / 04	90 /11/ 20
Analysing Laboratory			MUN	WAL	WAL
Alkalinity	mg/L		4.70	6.00	3.10
Aluminium	mg/L		1.2600	0.2200	0.0250
Arsenic	mg/L	0.025	0.0025	0.0025	0.0025
Cadmium	mg/L	0.005	0.00020	0.00020	0.00020
Calcium	mg/L		0.97	1.42	1.96
Chloride	mg/L	250	3.90	1.80	3.80
Chromium	mg/L	0.05	0.00250	0.00250	0.00250
Copper	mg/L	1.0	0.00500	0.00200	0.00200
DOC	mg/L		N/A	N/A	N/A
Fluoride	mg/L	1.5	0.020	0.020	0.190
Iron	mg/L	0.3	0.005	0.240	0.450*
Potassium	mg/L		0.22	0.33	0.39
Kejlhal Nit.	mg/L		0.19	0.19	0.25
Lead	mg/L	0.01	0.0005	0.0005	0.0020
Magnesium	mg/L		0.60	0.34	0.60
Manganese	mg/L	0.05	0.010	0.002	0.050
Mercury	mg/L	1.0	N/A	N/A	N/A
Sodium	mg/L	200	3.18	1.95	2.69
Nickel	mg/L		0.0050	0.0025	0.0025
Nitrate (ite)	mg/L	10	N/A	N/A	N/A
pH	(pH units)	6.5-8.5	6.05*	6.08*	5.38*
Tot. Phosphorus	mg/L		0.0440	0.0400	0.0500
Sulphate	mg/L	500	4.30	3.50	4.80

T D S	mg/L	500	23	35	49
Zinc	mg/L	5.0	0.013	0.010	0.030
Colour	(TCU)	15	70.0*	94.0*	152.0*
Spec. Cond.	(uS/cm)		22.0	19.1	34.5
Turbidity	(NTU)	1 & 5	2.20*	0.26	0.25
Temperature	(C)	15	N/A	N/A	N/A
T S S	mg/L		4	2	2
Total Col.	(/100ml)	0.0	N/A	N/A	N/A
Faecal Col..	(/100mL)	0.0	N/A	N/A	N/A

Table 4 Water Quality Data of Clarenville Municipal Water Supply, Newfoundland

Parameters	**Units**	**Drinking Water Limits**	**Sampling Data**		
			YY/MM/DD	**YY/MM/DD**	**YY/MM/DD**
			94 / 05 / 25	**94 / 11 / 09**	**98 /05/ 19**
Analysing Laboratory			**WAL**	**WAL**	**WAL**
Alkalinity	mg/L		3.40	2.60	1.90
Aluminium	mg/L		0.1330	0.3110	0.1900
Arsenic	mg/L	0.025	N/A	N/A	N/A
Cadmium	mg/L	0.005	0.00030	0.00030	N/A
Calcium	mg/L		1.12	1.68	0.87
Chloride	mg/L	250	2.60	4.00	1.40
Chromium	mg/L	0.05	0.00250	0.00050	N/A
Copper	mg/L	1.0	0.00100	0.00500	0.00500
DOC	mg/L		4.50	8.40	7.40
Fluoride	mg/L	1.5	0.030	0.110	N/A
Iron	mg/L	0.3	0.124	0.379*	0.280
Potassium	mg/L		0.30	0.20	0.01

Kejlhal Nit.	mg/L		0.16	0.38	0.13
Lead	mg/L	0.01	0.0005	0.0005	0.0005
Magnesium	mg/L		0.35	0.56	N/A
Manganese	mg/L	0.05	0.005	0.021	0.005
Mercury	mg/L	1.0	N/A	N/A	N/A
Sodium	mg/L	200	1.75	2.19	1.14
Nickel	mg/L		0.0010	0.0020	N/A
Nitrate (ite)	mg/L	10	0.009	0.002	0.003
pH	(pH units)	6.5-8.5	6.19*	5.59*	6.22*
Tot. Phosphorus	mg/L		0.0100	0.0070	0.0100
Sulphate	mg/L	500	1.50	1.70	0.90
T D S	mg/L	500	14	20	14
Zinc	mg/L	5.0	0.001	0.003	0.005
Colour	(TCU)	15	65.0*	70.0*	92.0*
Spec. Cond.	(uS/cm)		10.8	20.5	14.8
Turbidity	(NTU)	1 & 5	0.40	0.60	0.47
Temperature	(C)	15	11.9	4.1	99.9
T S S	mg/L		2	2	2
Total Col.	(/100ml)	0.0	N/A	34*	N/A
Faecal Col..	(/100mL)	0.0	N/A	38*	N/A

Table 5 Water Quality Data of Clarenville Municipal Water Supply, Newfoundland

Parameters	Units	Drinking Water Limits	Sampling Date
			YY /MM / DD
			98 / 11 / 03
Analysing Laboratory			WAL
Alkalinity	mg/L		0.70
Aluminium	mg/L		0.4600
Arsenic	mg/L	0.025	N/A
Cadmium	mg/L	0.005	N/A
Calcium	mg/L		1.48
Chloride	mg/L	250	3.90
Chromium	mg/L	0.05	N/A
Copper	mg/L	1.0	0.00500
DOC	mg/L		10.00
Fluoride	mg/L	1.5	N/A
Iron	mg/L	0.3	0.390*
Potassium	mg/L		0.17
Kejlhal Nit.	mg/L		0.32
Lead	mg/L	0.01	0.0005
Magnesium	mg/L		N/A
Manganese	mg/L	0.05	0.030
Mercury	mg/L	1.0	N/A
Sodium	mg/L	200	1.71
Nickel	mg/L		N/A
Nitrate (ite)	mg/L	10	0.003
pH	(pH units)	6.5-8.5	5.40*
Tot. Phosphorus	mg/L		0.0050
Sulphate	mg/L	500	1.60

T D S	mg/L	500	20
Zinc	mg/L	5.0	0.005
Colour	(TCU)	15	142.0*
Spec. Cond.	(uS/cm)		23.5
Turbidity	(NTU)	1 & 5	1.30
Temperature	(C)	15	N/A
T S S	mg/L		1
Total Col.	(/100ml)	0.0	80*
Faecal Col..	(/100mL)	0.0	60*

Note: 'N/A' indicates this parameter was not tested.

WAL - Water Analysis Laboratory, Mt. Pearl
WQL - Water Quality Laboratory, Env. Canada, Moncton
VGH - Victoria General Hospital, Halifax
MUN - Memorial University of Newfoundland, St. John's
NBE - N.B. Environmental Services Lab, Fredericton

Source: Water Resources Management Division, Department of Environment and Labour, Government of Newfoundland and Labrador, 1999

Table 6 Water Quality Data of Shoal Harbour Municipal Water Supply, Newfoundland

Parameters	**Units**	**Drinking Water Limits**	**Sampling Data**		
			YY/MM/DD	**YY/MM/DD**	**YY/MM/DD**
			87 / 09 / 28	**89 / 06 / 15**	**89 / 10 / 23**
Analysing Laboratory			**MUN**	**WQL**	**WQL**
Alkalinity	mg/L		6.90	4.80	4.90
Aluminium	mg/L		0.1000	0.0770	0.0870
Arsenic	mg/L	0.025	0.0025	0.0003	0.0003
Cadmium	mg/L	0.005	0.00020	0.00050	0.00050
Calcium	mg/L		1.75	2.50	2.35

Chloride	mg/L	250	5.90	6.70	5.80
Chromium	mg/L	0.05	0.00250	N/A	0.00020
Copper	mg/L	1.0	0.00500	0.00100	0.00100
DOC	mg/L		N/A	3.10	4.85
Fluoride	mg/L	1.5	0.020	0.030	0.030
Iron	mg/L	0.3	0.010	0.180	0.185
Potassium	mg/L		0.18	0.16	0.24
Kejlhal Nit.	mg/L		0.14	0.02	N/A
Lead	mg/L	0.01	0.0005	0.0010	0.0010
Magnesium	mg/L		0.50	0.53	0.50
Manganese	mg/L	0.05	0.002	0.040	0.020
Mercury	mg/L	1.0	N/A	0.0100	0.0100
Sodium	mg/L	200	4.8	4.40	3.90
Nickel	mg/L		0.0050	0.0010	0.0010
Nitrate (ite)	mg/L	10	N/A	0.020	0.005
pH	(pH units)	6.5-8.5	6.12*	6.60	6.65
Tot. Phosphorus	mg/L		0.0050	N/A	N/A
Sulphate	mg/L	500	3.10	2.20	1.70
T D S	mg/L	500	28	N/A	N/A
Zinc	mg/L	5.0	0.009	0.005	0.005
Colour	(TCU)	15	35.0*	30.0*	30.0*
Spec. Cond.	(uS/cm)		28.0	40.0	39.0
Turbidity	(NTU)	1 & 5	0.50	0.30	0.55
Temperature	(C)	15	N/A	N/A	N/A
T S S	mg/L		1	N/A	N/A
Total Col.	(/100ml)	0.0	N/A	N/A	N/A
Faecal Col..	(/100mL)	0.0	N/A	N/A	N/A

Table 7 Water Quality Data of Shoal Harbour Municipal Water Supply, Newfoundland

Parameters	Units	Drinking Water Limits	Sampling Data		
			YY/MM/DD	YY/MM/DD	YY/MM/DD
			92 / 06 /09	92 / 10 / 29	95 / 06 / 07
Analysing Laboratory			WQL	WQL	NBE
Alkalinity	mg/L		4.30	2.50	3.57
Aluminium	mg/L		0.1200	0.1700	0.1110
Arsenic	mg/L	0.025	0.0003	0.0003	N/A
Cadmium	mg/L	0.005	0.00050	0.00050	0.00005
Calcium	mg/L		2.10	1.60	2.00
Chloride	mg/L	250	5.70	4.40	5.08
Chromium	mg/L	0.05	0.00010	N/A	0.00025
Copper	mg/L	1.0	0.00100	0.00100	0.00025
DOC	mg/L		5.70	7.00	6.00
Fluoride	mg/L	1.5	0.030	0.030	0.070
Iron	mg/L	0.3	0.210	0.300	0.140
Potassium	mg/L		0.29	0.20	0.17
Kejlhal Nit.	mg/L		N/A	N/A	0.12
Lead	mg/L	0.01	0.0010	0.0010	0.0005
Magnesium	mg/L		0.45	0.46	0.40
Manganese	mg/L	0.05	0.015	0.025	0.012
Mercury	mg/L	1.0	N/A	0.0100	N/A
Sodium	mg/L	200	4.00	3.00	3.60
Nickel	mg/L		0.0010	0.0010	N/A
Nitrate (ite)	mg/L	10	N/A	N/A	0.025
pH	(pH units)	6.5-8.5	6.50	6.15*	6.71
Tot. Phosphorus	mg/L		N/A	N/A	0.0025
Sulphate	mg/L	500	1.70	1.60	1.40

T D S	mg/L	500	N/A	N/A	30
Zinc	mg/L	5.0	0.005	0.005	0.005
Colour	(TCU)	15	55.0*	65.0*	25.0*
Spec. Cond.	(uS/cm)		34.0	26.0	30.2
Turbidity	(NTU)	1 & 5	0.30	N/A	0.35
Temperature	(C)	15	N/A	N/A	9.8
T S S	mg/L		N/A	N/A	N/A
Total Col.	(/100ml)	0.0	N/A	N/A	15*
Faecal Col..	(/100mL)	0.0	N/A	N/A	8*

Table 8 Water Quality Data of Shoal Harbour Municipal Water Supply, Newfoundland

Parameters	**Units**	**Drinking Water Limits**	**Sampling Data**		
			YY/MM/DD	**YY/MM/DD**	**YY/MM/DD**
			95 / 10 / 10	**96 / 06 / 18**	**96 / 10 / 01**
Analysing Laboratory			**NBE**	**NBE**	**WAL**
Alkalinity	mg/L		6.53	3.53	4.80
Aluminium	mg/L		0.1600	0.1300	0.0800
Arsenic	mg/L	0.025	N/A	N/A	N/A
Cadmium	mg/L	0.005	0.00005	N/A	N/A
Calcium	mg/L		2.88	1.86	1.58
Chloride	mg/L	250	4.36	2.63	4.20
Chromium	mg/L	0.05	0.00025	N/A	N/A
Copper	mg/L	1.0	0.00500	N/A	N/A
DOC	mg/L		9.20	8.20	4.70
Fluoride	mg/L	1.5	0.050	N/A	N/A
Iron	mg/L	0.3	0.133	0.149	0.170
Potassium	mg/L		0.22	N/A	N/A

Kejlhal Nit.	mg/L		0.30	N/A	N/A
Lead	mg/L	0.01	0.0005	0.0005	0.0005
Magnesium	mg/L		0.67	0.47	0.42
Manganese	mg/L	0.05	0.037	0.013	0.003
Mercury	mg/L	1.0	N/A	N/A	N/A
Sodium	mg/L	200	3.59	3.55	2.73
Nickel	mg/L		N/A	N/A	N/A
Nitrate (ite)	mg/L	10	0.025	0.025	0.002
pH	(pH units)	6.5-8.5	6.84	6.46*	6.35*
Tot. Phosphorus	mg/L		0.0080	N/A	N/A
Sulphate	mg/L	500	1.83	3.30	2.60
T D S	mg/L	500	30	30	22
Zinc	mg/L	5.0	0.005	N/A	N/A
Colour	(TCU)	15	100.0*	50.0*	42.0*
Spec. Cond.	(uS/cm)		18.6	29.6	27.0
Turbidity	(NTU)	1 & 5	0.80	0.30	0.62
Temperature	(C)	15	8.9	12.9	11.6
T S S	mg/L		N/A	N/A	N/A
Total Col.	(/100ml)	0.0	57*	16*	33*
Faecal Col..	(/100mL)	0.0	55*	12*	14*

Table 9 Water Quality Data of Shoal Harbour Municipal Water Supply, Newfoundland

Parameters	Units	Drinking Water Limits	Sampling Data	
			YY/MM/DD	YY/MM/DD
			98 / 05 / 19	98 / 10 / 15
Analysing Laboratory			WAL	WAL
Alkalinity	mg/L		2.90	3.80
Aluminium	mg/L		0.1600	0.0250
Arsenic	mg/L	0.025	N/A	N/A
Cadmium	mg/L	0.005	N/A	N/A
Calcium	mg/L		1.47	2.10
Chloride	mg/L	250	4.60	5.30
Chromium	mg/L	0.05	N/A	N/A
Copper	mg/L	1.0	0.00500	0.00500
DOC	mg/L		7.40	7.00
Fluoride	mg/L	1.5	N/A	N/A
Iron	mg/L	0.3	0.170	0.210
Potassium	mg/L		0.15	0.18
Kejlhal Nit.	mg/L		0.11	0.23
Lead	mg/L	0.01	0.0005	0.0005
Magnesium	mg/L		N/A	N/A
Manganese	mg/L	0.05	0.010	0.020
Mercury	mg/L	1.0	N/A	N/A
Sodium	mg/L	200	3.35	3.04
Nickel	mg/L		N/A	N/A
Nitrate (ite)	mg/L	10	0.018	0.015
pH	(pH units)	6.5-8.5	6.21*	6.60
Tot. Phosphorus	mg/L		0.0050	0.0050
Sulphate	mg/L	500	1.10	1.00

T D S	mg/L	500	24	21
Zinc	mg/L	5.0	0.005	0.005
Colour	(TCU)	15	72.0*	62.0*
Spec. Cond.	(uS/cm)		30.7	30.6
Turbidity	(NTU)	1 & 5	0.23	1.86*
Temperature	(C)	15	N/A	N/A
T S S	mg/L		1	1
Total Col.	(/100ml)	0.0	N/A	0
Faecal Col..	(/100mL)	0.0	N/A	0

Table 10 Water Quality Data of St. John's (Windsor Lake) Municipal Water Supply, Newfoundland

Parameters	**Units**	**Drinking Water Limits**	**Sampling Data**	
			YY/MM/DD	**YY/MM/DD**
			95 / 10 / 03	**97 / 10 / 07**
Analysing Laboratory			**WAL**	**WAL**
Alkalinity	mg/L		3.00	3.10
Aluminium	mg/L		0.0250	0.0250
Arsenic	mg/L	0.025	N/A	N/A
Cadmium	mg/L	0.005	0.00025	0.00050
Calcium	mg/L		0.87	1.26
Chloride	mg/L	250	9.30	12.80
Chromium	mg/L	0.05	N/A	N/A
Copper	mg/L	1.0	0.00250	0.00500
DOC	mg/L		2.30	1.90
Fluoride	mg/L	1.5	0.025	0.025
Iron	mg/L	0.3	0.010	0.440*

Potassium	mg/L		0.49	0.43
Kejlhal Nit.	mg/L		0.11	0.03
Lead	mg/L	0.01	0.0005	0.0005
Magnesium	mg/L		0.82	0.66
Manganese	mg/L	0.05	0.020	0.110*
Mercury	mg/L	1.0	N/A	N/A
Sodium	mg/L	200	5.20	7.06
Nickel	mg/L		N/A	N/A
Nitrate (ite)	mg/L	10	0.009	0.005
pH	(pH units)	6.5-8.5	6.24*	6.38*
Tot. Phosphorus	mg/L		0.0100	0.0050
Sulphate	mg/L	500	4.70	4.90
T D S	mg/L	500	34	42
Zinc	mg/L	5.0	0.003	0.005
Colour	(TCU)	15	10.0	12.0
Spec. Cond.	(uS/cm)		46.3	61.0
Turbidity	(NTU)	1 & 5	0.30	1.17
Temperature	(C)	15	N/A	N/A
T S S	mg/L		2	2
Total Col.	(/100ml)	0.0	N/A	N/A
Faecal Col..	(/100mL)	0.0	N/A	N/A

Note: ' N/A ' indicates this parameter was not tested.

WAL - Water Analysis Laboratory, Mt. Pearl
WQL - Water Quality Laboratory, Env. Canada, Moncton
VGH - Victoria General Hospital, Halifax
MUN - Memorial University of Newfoundland, St. John's
NBE - N.B. Environmental Services Lab, Fredericton

Source: Water Resources Management Division, Department of Environment and Labour, Government of Newfoundland and Labrador, 1999

Table 11 Water Quality Data of St. John's (Windsor Lake) Municipal Water Supply, Newfoundland

Sample Identification: WINDSOR LAKE RAW WATER 99 \ 06 \ 13.
Date Submitted: 05 / 13 / 1999.

Parameters	Values	Units
Alkalinity	1.2	mg/L $CaCO_3$
pH	5.98	Units
True Colour	5	TCU
Specific Conductance	58.5	µS/cm
Turbidity	0.60	NTU
Hardness	4.5	mg/L $CaCO_3$
Calcium	0.96	mg/L Ca
Magnesium	0.52	mg/L Mg
Manganese	0.01	mg/L Mn
Iron	0.01	mg/L Fe
Copper	< 0.01	mg/L Cu
Zinc	< 0.01	mg/L Zn
Potassium	0.32	mg/L K
Sodium	7.44	mg/L Na
Chloride	11.1	mg/L Cl
Fluoride	<0.05	mg/L F
Sulphate	2.7	mg/L SO_4
Dissolved Organic Carbon	1.4	mg/L C
Total Solids	37	mg/L
Total Suspended Solids	< 2	mg/L
Total Dissolved Solids	37	mg/L
Nitrate	< 0.005	mg/L N
Ammonia	< 0.01	mg/L N
Kjeldahl Nitrogen	0.17	mg/L N
Total Phosphorus	< 0.01	mg/L P
Cadmium	< 0.001	mg/L Cd

Lead	0.023	mg/L Pb
Aluminium	< 0.05	mg/L Al
Chromium	< 0.005	mg/L Cr
Nickel	< 0.005	mg/L NI
Silicate (reactive)	0.29	mg/L SI
Nitrite	< 0.001	mg/L N
Orthophosphate	< 0.01	mg/L P
Bromide	< 0.05	mg/L Br
Cobalt	< 0.005	mg/L Co
Vanadium	<0.05	mg/L V
Arsenic	< 0.01	mg/L As

Table 12 Water Quality Data of St. John's (Windsor Lake) Municipal Water Supply, Newfoundland

Sample Identification: MEMORIAL STADIUM (Windsor Lake Treated Water)
Date Submitted: 05 / 13 / 1999.

Parameters	Values	Units
Alkalinity	5.0	mg/L $CaCO_3$
pH	7.06	Units
True Colour	14	TCU
Specific Conductance	54.1	µS/cm
Turbidity	0.37	NTU
Hardness	5.9	mg/L $CaCO_3$
Calcium	2.71	mg/L Ca
Magnesium	0.54	mg/L Mg
Manganese	< 0.01	mg/L Mn
Iron	0.36	mg/L Fe
Copper	< 0.01	mg/L Cu
Zinc	< 0.01	mg/L Zn

Potassium	0.27	mg/L K
Sodium	7.06	mg/L Na
Chloride	9.1	mg/L Cl
Fluoride	< 0.05	mg/L F
Sulphate	2.2	mg/L SO_4
Dissolved Organic Carbon	1.1	mg/L C
Total Solids	43	mg/L
Total Suspended Solids	< 2	mg/L
Total Dissolved Solids	43	mg/L
Nitrate	< 0.005	mg/L N
Ammonia	< 0.01	mg/L N
Kjeldahl Nitrogen	0.16	mg/L N
Total Phosphorus	< 0.01	mg/L P
Cadmium	< 0.001	mg/L Cd
Lead	< 0.001	mg/L Pb
Aluminium	< 0.05	mg/L Al
Chromium	< 0.005	mg/L Cr
Nickel	< 0.005	mg/L NI
Silicate (reactive)	0.35	mg/L SI
Nitrite	< 0.001	mg/L N
Orthophosphate	< 0.01	mg/L P
Bromide	< 0.05	mg/L Br
Cobalt	< 0.005	mg/L Co
Vanadium	< 0.05	mg/L V
Arsenic	< 0.01	mg/L As

Table 13 Water Quality Data of St. John's (Windsor Lake) Municipal Water Supply, Newfoundland

Sample Identification: WINDSOR LAKE TREATED WATER 98 \ 10 \ 15
Date Submitted: 10 / 15 / 1998.

Parameters	Values	Units
Alkalinity	3.1	mg/L $CaCO_3$
pH	6.79	Units
True Colour	4	TCU
Specific Conductance	58.9	µS/cm
Turbidity	0.40	NTU
Hardness	8.0	mg/L $CaCO_3$
Calcium	2.36	mg/L Ca
Magnesium	0.51	mg/L Mg
Manganese	< 0.01	mg/L Mn
Iron	< 0.01	mg/L Fe
Copper	< 0.01	mg/L Cu
Zinc	< 0.01	mg/L Zn
Potassium	0.30	mg/L K
Sodium	7.50	mg/L Na
Chloride	13.8	mg/L Cl
Fluoride	< 0.05	mg/L F
Sulphate	2.6	mg/L SO_4
Dissolved Organic Carbon	1.2	mg/L C
Total Solids	34	mg/L
Total Suspended Solids	< 2	mg/L
Total Dissolved Solids	34	mg/L
Nitrate + Nitrite	< 0.005	mg/L N
Ammonia	< 0.01	mg/L N
Kjeldahl Nitrogen	0.13	mg/L N
Total Phosphorus	< 0.01	mg/L P
Cadmium	< 0.001	mg/L Cd

Lead	< 0.001	mg/L Pb
Aluminium	< 0.05	mg/L Al
Chromium	< 0.005	mg/L Cr
Nickel	< 0.005	mg/L NI
Silicate (reactive)	0.14	mg/L SI
Nitrite	< 0.001	mg/L N
Orthophosphate	< 0.01	mg/L P
Bromide	< 0.05	mg/L Br

Table 14 Water Quality Data of St. John's (Windsor Lake) Municipal Water Supply, Newfoundland

Sample Identification: WINDSOR LAKE RAW WATER 98 \ 10 \ 15
Date Submitted: 10 / 15 / 1998.

Parameters	Values	Units
Alkalinity	2.3	mg/L $CaCO_3$
pH	6.38	Units
True Colour	5	TCU
Specific Conductance	55.8	µS/cm
Turbidity	0.47	NTU
Hardness	4.9	mg/L $CaCO_3$
Calcium	0.95	mg/L Ca
Magnesium	0.62	mg/L Mg
Manganese	< 0.01	mg/L Mn
Iron	< 0.01	mg/L Fe
Copper	< 0.01	mg/L Cu
Zinc	< 0.01	mg/L Zn
Potassium	< 0.01	mg/L K
Sodium	0.31	mg/L Na
Chloride	8.03	mg/L Cl

Fluoride	13.8	mg/L F
Sulphate	< 0.05	mg/L SO_4
Dissolved Organic Carbon	2.7	mg/L C
Total Solids	1.2	mg/L
Total Suspended Solids	34	mg/L
Total Dissolved Solids	< 2	mg/L
Nitrate + Nitrite	34	mg/L N
Ammonia	< 0.01	mg/L N
Kjeldahl Nitrogen	0.15	mg/L N
Total Phosphorus	< 0.01	mg/L P
Cadmium	< 0.01	mg/L Cd
Lead	< 0.001	mg/L Pb
Aluminium	< 0.05	mg/L Al
Chromium	< 0.005	mg/L Cr
Nickel	< 0.005	mg/L NI
Silicate (reactive)	0.20	mg/L SI
Nitrite	< 0.001	mg/L N
Orthophosphate	< 0.01	mg/L P
Bromide	< 0.05	mg/L Br

Table 15 Water Quality Data of St. John's (Windsor Lake) Municipal Water Supply, Newfoundland

Sample Identification: WINDSOR LAKE TREATED WATER. STADIUM 98 \ 05 \ 14
Date Submitted: 05 / 14 / 1998.

Parameters	Values	Units
Alkalinity	4.4	mg/L $CaCO_3$
pH	6.56	Units
True Colour	17	TCU
Specific Conductance	64.8	µS/cm
Turbidity	0.40	NTU

Hardness	6.24	mg/L $CaCO_3$
Calcium	2.07	mg/L Ca
Magnesium	0.26	mg/L Mg
Manganese	< 0.01	mg/L Mn
Iron	0.54	mg/L Fe
Copper	0.06	mg/L Cu
Zinc	< 0.01	mg/L Zn
Potassium	0.30	mg/L K
Sodium	8.27	mg/L Na
Chloride	14.6	mg/L Cl
Fluoride	< 0.05	mg/L F
Sulphate	2.4	mg/L SO_4
Dissolved Organic Carbon	1.6	mg/L C
Total Solids	47	mg/L
Total Suspended Solids	< 2	mg/L
Total Dissolved Solids	47	mg/L
Nitrate + Nitrite	< 0.005	mg/L N
Ammonia	< 0.01	mg/L N
Kjeldahl Nitrogen	0.14	mg/L N
Total Phosphorus	< 0.01	mg/L P
Cadmium	< 0.001	mg/L Cd
Lead	< 0.001	mg/L Pb
Aluminium	0.06	mg/L Al
Chromium	< 0.005	mg/L Cr
Nickel	< 0.005	mg/L NI
Silicate (reactive)	0.18	mg/L SI
Nitrite	< 0.001	mg/L N
Orthophosphate	< 0.01	mg/L P
Bromide	< 0.05	mg/L Br

Table 16 Water Quality Data of St. John's (Windsor Lake) Municipal Water Supply, Newfoundland

Sample Identification: WINDSOR LAKE RAW WATER 98 \ 05 \ 14
Date Submitted: 05 / 14 / 1998.

Parameters	Values	Units
Alkalinity	2.1	mg/L $CaCO_3$
pH	6.38	Units
True Colour	5	TCU
Specific Conductance	63.5	µS/cm
Turbidity	0.18	NTU
Hardness	5.11	mg/L $CaCO_3$
Calcium	1.24	mg/L Ca
Magnesium	0.51	mg/L Mg
Manganese	0.01	mg/L Mn
Iron	0.02	mg/L Fe
Copper	0.06	mg/L Cu
Zinc	< 0.01	mg/L Zn
Potassium	0.42	mg/L K
Sodium	8.27	mg/L Na
Chloride	14.1	mg/L Cl
Fluoride	< 0.05	mg/L F
Sulphate	3.6	mg/L SO_4
Dissolved Organic Carbon	1.5	mg/L C
Total Solids	46	mg/L
Total Suspended Solids	< 2	mg/L
Total Dissolved Solids	4.6	mg/L
Nitrate + Nitrite	< 0.005	mg/L N
Ammonia	< 0.01	mg/L N
Kjeldahl Nitrogen	0.15	mg/L N
Total Phosphorus	< 0.01	mg/L P
Cadmium	< 0.001	mg/L Cd

Lead	< 0.001	mg/L Pb
Aluminium	0.06	mg/L Al
Chromium	< 0.005	mg/L Cr
Nickel	. 0.005	mg/L NI
Silicate (reactive)	0.18	mg/L SI
Nitrite	< 0.001	mg/L N
Orthophosphate	< 0.01	mg/L P
Bromide	< 0.05	mg/L Br

Venue of Water Analysis: Water Analysis Laboratories, Mount Pearl, Newfoundland
Source: Paul Kieley, St John's City Council, Newfoundland, 1999

APPENDIX 2
CONTAMINANTS THAT MAY CAUSE HEALTH EFFECTS, THEIR POTENTIAL SOURCES, AND THEIR POSSIBLE CHRONIC HEALTH EFFECTS

Office of Water, United States Environmental Protection Agency has established current drinking water standards. The regulations are categorised into two groups, National Primary and Secondary Drinking Water Regulations. National Primary Drinking Water Regulations (NPDWRs or primary standards) legally enforceable standards applicable to public water systems. The objective of the primary standards is to ensure protection of drinking water quality by limiting the levels of specific contaminants that has potential adverse public health effects. They are known or anticipated to occur in public water systems.

National Primary Drinking Water Regulations

Table 1 U. S. EPA Primary Drinking Water Contaminants (Inorganic Chemicals), Their Potential Sources, Possible Chronic Health Effects, And Maximum Contaminant Levels (MCLs) as of July, 1999.

INORGANIC CONTAMINATS	USES AND/OR SOURCES	POTENTIAL HEALTH EFFECTS FROM INGESTION OF WATER	MCL[2] or TT[3] (mg/L)[4]	MCLG[1] (mg/L)[4]
Antimony	Discharge from petroleum refineries; fire retardants; ceramics; electronics; solder	Increase in blood cholesterol; decrease in blood glucose	0.006	0.006
Arsenic	Corrosion of asbestos cement pipe in water distribution systems; manufacture of: cement products, paper, floor	Skin damage; circulatory system problems; increased risk of cancer	0.05	None[5]

	tiles, paint, caulking, textiles, and plastics; natural deposits; discharge from semiconductor manufacturing; petroleum refining			
Asbestos (fibre > 10 micrometers)	Decay of asbestos cement in water mains; erosion of natural deposits; manufacture of: cement products, paper, floor tiles, paint, caulking, textiles, and plastics	Increased risk of developing benign intestinal polyps, cancer	7 million fibres per Litre	7 MFL
Barium	Discharge of drilling wastes; discharge from metal refineries; erosion of natural deposits	Increase in blood pressure	2	2
Beryllium	Discharge from metal refineries and coal-burning factories; discharge from electrical, aerospace, and defence industries	Intestinal lesions	0.004	0.004

Cadmium	Corrosion of galvanised pipes; erosion of natural deposits; discharge from metal refineries; runoff from waste batteries and paints	Kidney damage	0.005	0.005
Chromium (total)	Discharge from steel and pulp mills; erosion of natural deposits	Some people who use water containing chromium well in excess of the MCL over many years could experience allergic dermatitis	0.1	0.1
Copper	Corrosion of household plumbing systems; erosion of natural deposits; leaching from wood preservatives	Short term exposure: Gastrointestinal distress. Long term exposure: Liver or kidney damage. Those with Wilson's Disease should consult their personal doctor if their water systems exceed the copper action level.	Action Level=1.3; TT[6]	1.3
Cyanide (as free cyanide)	Discharge from steel/metal factories; discharge from plastic and fertiliser factories	Nerve damage or thyroid problems	0.2	0.2
Fluoride	Water additive which promotes strong teeth; erosion of	Bone disease (pain and tenderness of the bones); Children may get mottled teeth.	4.0	4.0

	natural deposits; discharge from fertiliser and aluminium factories			
Lead	Corrosion of household plumbing systems; erosion of natural deposits	Infants and children: Delays in physical or mental development. Adults: Kidney problems; high blood pressure	Action Level=0.015; TT[6]	zero
Inorganic Mercury	Erosion of natural deposits; discharge from refineries and factories; runoff from landfills and cropland	Kidney damage	0.002	0.002
Nitrate (measured as Nitrogen)	Runoff from fertiliser use; leaching from septic tanks, sewage; erosion of natural deposits	"Blue baby syndrome" in infants under six months - life threatening without immediate medical attention. Symptoms: Infant looks blue and has shortness of breath.	[10]	10
Nitrite (measured as Nitrogen)	Runoff from fertiliser use; leaching from septic tanks, sewage; erosion of natural deposits	"Blue baby syndrome" in infants under six months - life threatening without immediate medical attention. Symptoms: Infant looks blue and has shortness of breath.	1	1
Selenium	Discharge from petroleum refineries;	Hair or fingernail loss; numbness in fingers or toes;	0.05	0.05

	erosion of natural deposits; discharge from mines	circulatory problems		
Thallium	Leaching from ore-processing sites; discharge from electronics, glass, and pharmaceutical companies	Hair loss; changes in blood; kidney, intestine, or liver problems	0.002	0.0005

Table 2 U. S. EPA Primary Drinking Water Contaminants (Organic Chemicals), Their Potential Sources, Possible Chronic Health Effects, And Maximum Contaminant Levels (MCLs) as of July, 1999.

ORGANIC CONTAMINATS	USES AND/OR SOURCES	POSSIBLE HEALTH EFFECTS	MCL[2] or TT[3] (mg/L)[4]	MCLG[1] (mg/L)[4]
Acrylamide	Added to water during sewage/wastewater treatment	Nervous system or blood problems; increased risk of cancer	TT[7]	zero
Alachlor	Runoff from herbicide used on row crops	Eye, liver, kidney or spleen problems; anaemia; increased risk of cancer	0.002	zero
Atrazine	Runoff from herbicide used on row crops	Cardiovascular system problems; reproductive difficulties	0.003	0.003
Benzene	Discharge from factories; leaching from gas storage tanks and landfills	Anaemia; decrease in blood platelets; increased risk of cancer	0.005	zero

Benzo(a)pyrene	Leaching from linings of water storage tanks and distribution lines	Reproductive difficulties; increased risk of cancer	0.0002	zero
Carbofuran	Leaching of soil fumigant used on rice and alfalfa	Problems with blood or nervous system; reproductive difficulties.	0.04	0.04
Carbon tetrachloride	Discharge from chemical plants and other industrial activities	Liver problems; increased risk of cancer	.005	zero
Chlordane	Residue of banned termiticide	Liver or nervous system problems; increased risk of cancer	0.002	zero
Chlorobenzene	Discharge from chemical and agricultural chemical factories	Liver or kidney problems	0.1	0.1
2,4-D	Runoff from herbicide used on row crops	Kidney, liver, or adrenal gland problems	0.07	0.07
Dalapon	Runoff from herbicide used on rights of way	Minor kidney changes	0.2	0.2
1,2-Dibromo-3-chloropropane (DBCP)	Runoff/leaching from soil fumigant used on soybeans, cotton, pineapples, and orchards	Reproductive difficulties; increased risk of cancer	0.0002	zero
o-Dichlorobenzene	Discharge from industrial chemical factories	Liver, kidney, or circulatory system problems	0.6	0.6
p-Dichlorobenzene	Discharge from industrial chemical factories	Anaemia; liver, kidney or spleen damage; changes in blood	0.075	0.075
1,2-Dichloroethane	Discharge from industrial chemical factories	Increased risk of cancer	0.005	zero
1-1-Dichloroethylene	Discharge from industrial chemical factories	Liver problems	0.007	0.007

cis-1, 2-Dichloroethylene	Discharge from industrial chemical factories	Liver problems	0.07	0.07
trans-1,2-Dichloroethylene	Discharge from industrial chemical factories	Liver problems	0.1	0.1
Dichloromethane	Discharge from pharmaceutical and chemical factories	Liver problems; increased risk of cancer	0.005	zero
1-2-Dichloropropane	Discharge from industrial chemical factories	Increased risk of cancer	0.005	zero
Di(2-ethylhexyl)adipate	Leaching from PVC plumbing systems; discharge from chemical factories	General toxic effects or reproductive difficulties	0.4	0.4
Di(2-ethylhexyl)phthalate	Discharge from rubber and chemical factories	Reproductive difficulties; liver problems; increased risk of cancer	0.006	zero
Dinoseb	Runoff from herbicide used on soybeans and vegetables	Reproductive difficulties	0.007	0.007
Dioxin (2,3,7,8-TCDD)	Emissions from waste incineration and other combustion; discharge from chemical factories	Reproductive difficulties; increased risk of cancer	0.00000003	zero
Diquat	Runoff from herbicide use	Cataracts	0.02	0.02
Endothall	Runoff from herbicide use	Stomach and intestinal problems	0.1	0.1
Endrin	Residue of banned insecticide	Nervous system effects	0.002	0.002
Epichlorohydrin	Discharge from industrial chemical factories; added to water during treatment process	Stomach problems; reproductive difficulties; increased risk of cancer	TT[7]	zero
Ethylbenzene	Discharge from petroleum refineries	Liver or kidney problems	0.7	0.7

Ethylene dibromide	Discharge from petroleum refineries	Stomach problems; reproductive difficulties; increased risk of cancer	0.00005	zero
Glyphosate	Runoff from herbicide use	Kidney problems; reproductive difficulties	0.7	0.7
Heptachlor	Residue of banned termiticide	Liver damage; increased risk of cancer	0.0004	zero
Heptachlor epoxide	Breakdown of heptachlor	Liver damage; increased risk of cancer	0.0002	zero
Hexachlorobenzene	Discharge from metal refineries and agricultural chemical factories	Liver or kidney problems; reproductive difficulties; increased risk of cancer	0.001	zero
Hexachloro cyclopentadiene	Discharge from chemical factories	Kidney or stomach problems	0.05	0.05
Lindane	Runoff/leaching from insecticide used on cattle, lumber, gardens	Liver or kidney problems	0.0002	0.0002
Methoxychlor	Runoff/leaching from insecticide used on fruits, vegetables, alfalfa	Reproductive difficulties	0.04	0.04
Oxamyl (Vydate)	Runoff/leaching from insecticide used on apples, potatoes, and tomatoes	Slight nervous system effects	0.2	0.2
Polychlorinated biphenyls (PCBs)	Runoff from landfills; discharge of waste chemicals	Skin changes; thymus gland problems; immune deficiencies; reproductive or nervous system difficulties; increased risk of cancer	0.0005	zero
Pentachlorophenol	Discharge from wood preserving factories	Liver or kidney problems; increased risk of cancer	0.001	zero
Picloram	Herbicide runoff	Liver problems	0.5	0.5

Simazine	Herbicide runoff	Problems with blood	0.004	0.004
Styrene	Discharge from rubber and plastic factories; leaching from landfills	Liver, kidney, and circulatory problems	0.1	0.1
Tetrachloroethylene	Leaching from PVC pipes; discharge from factories and dry cleaners	Liver problems; increased risk of cancer	0.005	zero
Toluene	Discharge from petroleum factories	Nervous system, kidney, or liver problems	1	1
Total Trihalomethanes (TTHMs)	By-product of drinking water disinfection	Liver, kidney or central nervous system problems; increased risk of cancer	0.10	none[5]
Toxaphene	Runoff/leaching from insecticide used on cotton and cattle	Kidney, liver, or thyroid problems; increased risk of cancer	0.003	zero
2,4,5-TP (Silvex)	Residue of banned herbicide	Liver problems	0.05	0.05
1,2,4-Trichlorobenzene	Discharge from textile finishing factories	Changes in adrenal glands	0.07	0.07
1,1,1-Trichloroethane	Discharge from metal degreasing sites and other factories	Liver, nervous system, or circulatory problems	0.2	0.20
1,1,2-Trichloroethane	Discharge from industrial chemical factories	Liver, kidney, or immune system problems	0.005	0.003
Trichloroethylene	Discharge from petroleum refineries	Liver problems; increased risk of cancer	0.005	zero
Vinyl chloride	Leaching from PVC pipes; discharge from plastic factories	Increased risk of cancer	0.002	zero
Xylenes (total)	Discharge from petroleum factories; discharge from chemical factories	Nervous system damage	10	10

Table 3 U. S. EPA Primary Drinking Water Contaminants (Radionuclides), Their Potential Sources, Possible Chronic Health Effects, And Maximum Contaminant Levels (MCLs) as of July, 1999.

RADIONUCLIDES	USES AND/OR SOURCES	POSSIBLE HEALTH EFFECTS	MCL[2] or TT[3] (mg/L)[4]	MCLG[1] (mg/L)[4]
Beta particles and photon emitters	Decay of natural and man-made deposits	Increased risk of cancer	4 millirems per year	none[5]
Gross alpha particle activity	Erosion of natural deposits	Increased risk of cancer	15 picocuries per Litre (pCi/L)	none[5]
Radium 226 and Radium 228 (combined)	Erosion of natural deposits	Increased risk of cancer	5 pCi/L	none[5]

Table 4 U. S. EPA Primary Drinking Water Contaminants (Microorganisms), Their Potential Sources, Possible Chronic Health Effects, and Maximum Contaminant Levels (MCLs) as of July, 1999.

MICROORGANISMS	USES AND/OR SOURCES	POSSIBLE HEALTH EFFECTS	MCL[2] or TT[3] (mg/L)[4]	MCLG[1] (mg/L)[4]
Giardia lamblia	Human and animal faecal waste	Giardiasis, a gastroenteric disease	TT[8]	zero
Heterotrophic plate count	n/a	HPC has no health effects, but can indicate how effective treatment is at controlling microorganisms.	TT[8]	N/A

Legionella	Found naturally in	Legionnaire's Disease.	TT[8]	zero
Total Coliforms (including faecal coliform and E. Coli)	Human and animal faecal waste	Used as an indicator that other potentially harmful bacteria may be present[10]	5.0%[10]	zero
Turbidity	Soil runoff	Turbidity has no health effects but can interfere with disinfection and provide a medium for microbial growth. It may indicate the presence of microbes.	TT[8]	N/A
Viruses (enteric)	Human and animal faecal waste	Gastroenteric disease	TT[8]	zero

National Secondary Drinking Water Regulations

National Secondary Drinking Water Regulations (NSDWRs or secondary standards) are non-enforceable guidelines that regulate contaminants causing cosmetic effects (such as skin or tooth discoloration) or aesthetic effects (such as taste, odour, or colour) in drinking water. EPA recommends the secondary standards to water systems. However, the systems are not required to comply. It may so happen that the states choose to adopt them as enforceable standards.

Table 5 Contaminant and Secondary Standards

Contaminant	Secondary Standard
Aluminium	0.05 to 0.2 mg/L
Chloride	250 mg/L
Colour	15 (colour units)
Copper	1.0 mg/L
Corrosivity	noncorrosive
Fluoride	2.0 mg/L
Foaming Agents	0.5 mg/L

Iron	0.3 mg/L
Manganese	0.05 mg/L
Odour	3 threshold odour number
pH	6.5-8.5
Silver	0.10 mg/L
Sulphate	250 mg/L
Total Dissolved Solids	500 mg/L
Zinc	5 mg/L

Notes

[1] Maximum Contaminant Level Goal (MCLG) - The maximum level of a contaminant in drinking water at which no known or anticipated adverse effect on the health effect of persons would occur, and which allows for an adequate margin of safety. MCLGs are non-enforceable public health goals.

[2] Maximum Contaminant Level (MCL) - The maximum permissible level of a contaminant in water, which is delivered to any user of a public water system. MCLs are enforceable standards. The margins of safety in MCLGs ensure that exceeding the MCL slightly does not pose significant risk to public health.

[3] Treatment Technique - An enforceable procedure or level of technical performance which public water systems must follow to ensure control of a contaminant.

[4] Units are in milligrams per Litre (mg/L) unless otherwise noted.

[5] MCLGs were not established before the 1986 Amendments to the Safe Drinking Water Act. Therefore, there is no MCLG for this contaminant.

[6] Lead and copper are regulated in a Treatment Technique which requires systems to take tap water samples at sites with lead pipes or copper pipes that have lead solder and/or are served by lead service lines. The action level, which triggers water systems into taking treatment steps, if exceeded in more than 10% of tap water samples, for copper is 1.3 mg/L, and for lead is 0.015mg/L.

[7] Each water system must certify, in writing, to the state (using third-party or manufacturer's certification) that when acrylamide and epichlorohydrin are used in drinking water systems, the combination (or product) of dose and monomer level does not exceed the levels specified, as follows:

•Acrylamide = 0.05% dosed at 1 mg/L (or equivalent)

•Epichlorohydrin = 0.01% dosed at 20 mg/L (or equivalent)

[8] The Surface Water Treatment Rule requires systems using surface water or ground water under the direct influence of surface water to (1) disinfect their water, and (2) filter their water to meet criteria for avoiding filtration so that the following contaminants are controlled at the following levels:

•Giardia lamblia: 99.9% killed/inactivated

Viruses: 99.99% killed/inactivated

•Legionella: No limit, but EPA believes that if Giardia and viruses are inactivated, Legionella will also be controlled.

•Turbidity: At no time can turbidity (cloudiness of water) go above 5 nephelometric turbidity units (NTU); systems that filter must ensure that the turbidity go no higher than 1 NTU (0.5 NTU for conventional or direct filtration) in at least 95% of the daily samples in any month.

•HPC: NO more than 500 bacterial colonies per millilitre.

[9] No more than 5.0% samples total coliform-positive in a month. (For water systems that collect fewer than 40 routine samples per month, no more than one sample can be total coliform-positive). Every sample that has total coliforms must be analysed for faecal coliforms. There cannot be any faecal coliforms.

[10] Faecal coliform and E. coli are bacteria whose presence indicates that the water may be contaminated with human animal wastes. Microbes in these wastes can cause diarrhoea, cramps, nausea, headaches, or other symptoms.