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Disinfection byproducts in Canadian provinces: Associated cancer risks and medical expenses

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ABSTRACT

Chlorination for drinking water forms various disinfection byproducts (DBPs). Some DBPs are probably linked to human cancer (e.g., bladder, colorectal cancers) and other chronic and sub-chronic effects. This emphasizes the need to understand and characterize DBPs in drinking water and possible risks to human health. In this study, occurrences of DBPs throughout Canada were investigated. Trihalomethanes (THMs) were observed to be highest in Manitoba followed by Nova Scotia and Saskatchewan, while haloacetic acids were highest in Nova Scotia followed by Newfoundland and Labrador. Based on the characterization of DBPs, risk of cancer from exposure to THMs was predicted using ingestion, inhalation and dermal contact pathways of exposure. In Canada, approximately 700 cancer cases may be caused by exposure to THMs in drinking water. Medical expenses associated with these cancer incidents are estimated at some $140 million/year. Expense may be highest in Ontario (~$47 million/year) followed by Quebec (~$25 million/year) due to a greater population base. This paper suggests improvements in water treatment, source protection and disinfection processes, and caution in the use of alternative disinfectants to reduce DBPs. Finally, elements are provided to mitigate risks and reduce cost estimates in future studies.

1. Introduction

Use of chlorine in drinking water supply systems has been in practice for over a century and has eliminated most waterborne diseases in developed countries. Approximately 90% of water supply systems in Canada use chlorine for disinfection [1,2]. Chlorine is an effective disinfectant against most microorganisms and provides residual protection in water distribution systems (WDS). Chlorine has been reported to be an inexpensive disinfectant to date [3–6]. During disinfection process, reactions between natural organic matter (NOM) and chlorine generate different types of disinfection byproducts (DBPs), including trihalomethanes (THMs), haloacetic acids (HAAs), haloacetonitriles (HANs), haloketones (HKs), N-nitrosodimethylamine (NDMA), iodo THMs and other known and unknown compounds [7,8]. Some DBPs are of concern due to possible cancer risks to human and other sub-chronic/chronic health effects, including cardiac anomalies, stillbirth, miscarriage, low birth weight and pre-term delivery [9–14]. Exposure to DBPs may occur through ingestion of drinking water and inhalation and through dermal contact during regular indoor activities (e.g., showering, bathing, swimming). Recent studies have reported elevated health risks from DBPs exposure through inhalation and dermal contact during bathing and showering [15–19].

In Canada, drinking water in urban areas is typically supplied by municipal water systems, where approximately 75% of populations live [20]. Many of them may be exposed to DBPs throughout their lifetimes. Significant number of the exposed populations may be affected due to chronic exposure to DBPs [2]. To protect human, Health Canada [2] has set guideline concentrations for some groups of DBPs in drinking water: namely, THMs (0.10 mg/L), HAAs (0.08 mg/L), bromate (0.01 mg/L) and chlorite (1 mg/L). Some provinces have established guidelines for THMs based on Canadian guidelines or U.S. EPA regulations.

Alternative disinfection practices may lower chlorinated DBPs; however, these practices can form more toxic byproducts, increase costs and favor incidents of microbiological recontamination in WDS [21]. For example, chloramine, ozone and chlorine dioxide form fewer amounts of chlorinated DBPs. Chloramine is a weaker disinfectant requiring greater contact time for disinfection in water treatment plants (WTP). It may form several regulated and unregulated DBPs including *N*-nitrosodimethylamine (NDMA), which is more toxic than THMs and HAAs. The USEPA sets the maximum allowable concentration of NDMA to 0.7 ng/L [5,7,22]. Ozone can form bromate in the presence of bromide ions (regulatory limit: 0.01 mg/L) and chlorine dioxide may form chlorite (regulatory limit: 1.0 mg/L). Application of chloramine, ozone and chlorine

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dioxide are generally more expensive than chlorine, while ozone and chlorine dioxide may not provide adequate protection in WDS [3,6,23,24]. Inadequate protection of WDS may lead to an increased incidence of waterborne diseases as a result of increased exposure to pathogenic microorganisms, and thus, pose a greater risk to human health [23,25–30]. Some of the recent events of microbiological recontamination include: (i) Walkerton (Ontario, Canada), seven people died and more than 2300 became ill in 2000 after the *E. coli* contamination of the community's municipal water supply system [27]; (ii) the World Health Organization (WHO) reported that approximately 3.4 million people, mostly children, die each year from water-related diseases in developing countries [29,30]; (iii) approximately 366 people died and 9000 were affected by a cholera epidemic in Zimbabwe between August and November 2008; (iv) Most recently, in Haiti, as of November 20, 2010, approximately 1400 deaths and 25,000 hospitalization were reported due to cholera outbreak [31]. The microbiological safety of drinking water is a global issue that warrants proper disinfection for drinking water.

Higher levels of THMs and HAAs in drinking water have been widely reported in Canadian provinces. Variations in their levels can be attributed to source water quality, type of treatment process, type of disinfectant and environmental conditions [1]. To meet Canadian and provincial guidelines, water supply systems require quarterly (4 times/year) reporting of THM concentrations. Therefore, THMs data are generally well maintained by provincial authorities. Some provinces also monitor HAAs and other DBPs, even if monitoring of these compounds is not warranted by any province. For example, in Ontario under the Drinking Water Surveillance Program (DWSP), THMs, HAAs, NDMA, chlorite and bromate are monitored throughout the province. In addition, Health Canada has conducted a survey on the occurrence of THMs and HAAs throughout Canada [1]. In Quebec, THMs monitoring is mandatory at all municipal systems and the Environmental Ministry ensures some measurements of HAAs [32]. In this paper, occurrences of DBPs in Canadian drinking water were investigated. Following a comparative portrait of DBPs occurrence in various provinces, cancer risks from exposure to THMs through ingestion of drinking water and inhalation and dermal contact during showering, were estimated. Cancer risks exceedance probabilities beyond regulatory limits were evaluated. Associated expenses for treatment of a possible increase of cancer cases due to exposure to THMs were also estimated.

2. Experimental

2.1. Canadian portrait of DBPs occurrence

In Canada, formation of DBPs in drinking water is widespread. Concentrations of DBPs from all provinces in Canada [Ontario (ON), Quebec (QC), Manitoba (MB), Saskatchewan (SK), Alberta (AB), British Columbia (BC), Nova Scotia (NS), New Brunswick (NB), Prince Edward Island (PEI) and Newfoundland and Labrador (NL)] were obtained through respective provincial offices and Health Canada reports [1,33–41]. The summary of THMs and HAAs is shown in Tables 1 and 2 and Figs. 1–4, respectively. The data in Table 1 and Figs. 1 and 3 were primarily quarterly THMs data (four times/year) from one or more locations for numerous municipal systems over several years. The period of data collection and number of systems monitored are shown in Table 1. Data from PEI was limited, as they mostly use ground water wells (not always chlorinated) and DBPs occurrences are very low, while data for NB was not available during the study period. THMs data from Health Canada [1] were analyzed for NB. In case of HAAs (Table 2, Figs. 2 and 4), data for AB, SK, MB and NB were from limited

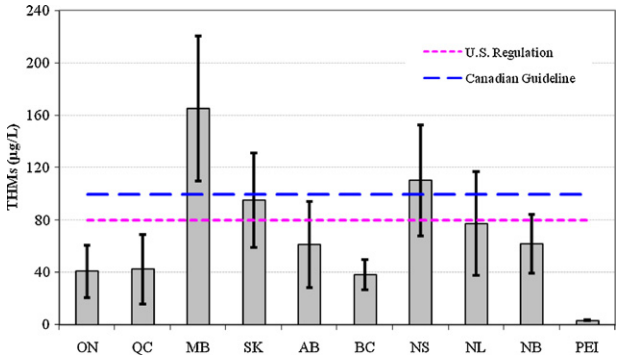

Fig. 1. Average THMs in drinking waters in Canadian provinces (error bars: std dev).

sources [1], while PEI reported no HAAs data. Details on the four THMs [chloroform ($CHCl_3$), bromodichloromethane (BDCM), dibromochloromethane (DBCM) and bromoform ($CHBr_3$)] are shown in Tables 3–6. It should be noted here that THMs in Manitoba were based on unpreserved samples from surface-water-sourced systems only, while preserving samples might have produced different data. Further to this, inclusion of groundwater-sourced systems would reduce average THMs concentrations. As such, THMs from MB are limited in terms of proper representation of all systems. No data for preserved samples were available during the study period [35]. However, THMs in five systems in MB (Winnipeg, Whitemouth, Selkirk, Portage-La-Prairie and Letellier) were fairly consistent with the previously reported data [1].

HAAs has nine species: trichloroacetic acid (TCAA), dichloroacetic acid (DCAA), monochloroacetic acid (MCAA), dibromoacetic acid (DBAA), monobromoacetic acid (MBAA), bromochloroacetic acid (BCAA), bromodichloroacetic acid (BDCAA), chlorodibromoacetic acid (CDBAA) and tribromoacetic acid (TBAA). In drinking water, TCAA, DCAA and MCAA are typically the dominant species, which formed ~95% of HAAs [1]. The USEPA has set maximum allowable concentrations of five HAAs (TCAA, DCAA, MCAA, MBAA and DBAA) to 60 μg/L [5], while the maximum concentration is 80 μg/L in Canada [2]. Table 7 shows the five regulated HAAs as the percentage of total HAAs. In addition, NDMA and bromate were monitored in the municipal systems in ON. NDMA was observed in the range of 1–17 ng/L with an average of 1.6 ng/L [36]. Occurrences of bromate and chlorite were often lower than the detection limits [36]. Provincial averages of THMs were highest in MB followed by NS, SK and NL (Table 1). The exceedance probabilities of THMs (number of occurrences beyond the Canadian guideline of 100 μg/L divided by the total number) are relatively higher in these provinces (Table 1). THMs data were further analyzed for fitting with statistical distributions.

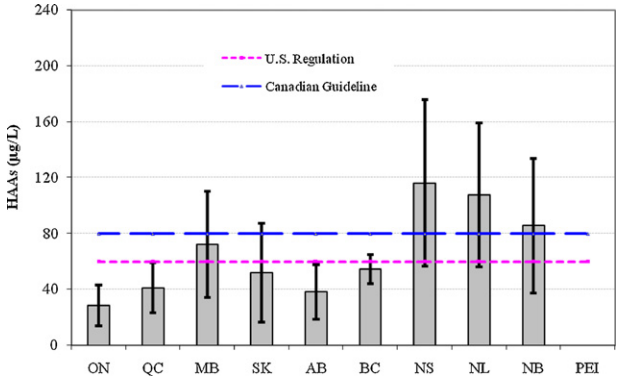

Fig. 2. Average HAAs in drinking waters in Canadian provinces (error bars: std dev).

Table 1
Trihalomethanes (THM) in Canadian drinking waters (μg/L).

Province	Period	WTP	Mean	Range	10%	25%	50%	75%	90%	97.5%	SD	EP	Rem[a]
ON	2000–2004	179	40.9	0.5–343	5.5	13.0	29	58.5	88.1	147.6	39.9	0.066	D
QC	2002–2006	622	42.5	0–565	1.6	7.1	26	56.3	101.1	197.4	53.3	0.104	D
MB[b,c]	2001–2006	74	164.9	0.7–640.3	67.9	100.2	136.5	203	313	518	110.9	0.707	U, D
SK	2002–2006	204	95.3	4.0–445.0	29.5	43	70	129	208	270.8	71.8	0.36	D
AB	2000–2006	449	61.5	0.6–447.3	3.2	12.8	37.6	91.5	147.5	241	66.0	0.21	D
BC	2001–2005	13	38.4	9–116	20	24.5	32	45	66	107.6	22.7	0.051	D
NS	1999–2004	24	110.2	2–640	33.2	52	83.8	145	229.1	328.4	84.9	0.39	D
NL	2001–2007	467	77.3	0–470.2	3.1	3.55	54	106.5	173.1	298.6	79.5	0.29	D
NB[d]	1993	4	62.1	4.1–146.9	7.4	22.3	61.9	74.3	92.4	146.9	45	0.19	D
PEI	2003–2006	–	3.5	1.4–5.9	1.4	2.1	2.7	3.5	3.7	5.9	0.96	0.0	D

[a] U: unpreserved samples; D: distribution system data; SD: standard deviation; EP: exceedance probability beyond regulatory limit of 100 μg/L.
[b] Only the data from surface water sources were available.
[c] Health Canada [1] data was also used for verification.
[d] Health Canada [1] report; WTP: number of water treatment plants.

Table 2
Haloacetic acids (HAA) in Canadian drinking waters (μg/L).

Province	Mean	Range	10%	25%	50%	75%	90%	97.5%	SD	EP
ON	28.6	0.4–244	3.9	8.4	18	40.6	65.5	103.3	28.9	0.054
QC	41.2	3.9–165.8	7.9	14.8	30.8	53.8	88.9	148.2	36.2	0.141
MB	72.4	2.2–249	2.6	10.7	42.8	120.5	207	249	76.1	0.41
SK	51.8	1–238	2.4	7.5	22.5	53	193.9	238	70.8	0.19
AB	38.4	3.2–141.2	3.4	9.6	21.7	52.7	104.5	141.2	39	0.19
BC	54.4	11–117	28.6	39	53	68	81.2	108.2	21	0.109
NS	116.2	8.4–602.6	35.9	51.3	68.8	128.9	227.8	536.1	119	0.42
NL	107.8	0–507.5	8.3	39	83.7	145.4	232.1	394	103	0.52
NB	85.7	10.6–398.5	17	34.8	68.5	131.8	225.9	398.5	96.1	0.43
PEI	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA

SD: standard deviation; EP: exceedance probability.

THMs in PEI and NB were uniformly distributed; however, the data were limited (Fig. 3). THMs in MB were normally distributed (approx.), while THMs in NL, NS, QC, ON, SK, AB and BC have skewed distributions. A fraction of THMs data were beyond the limits of Box-plots in NS, QC, ON, MB, SK and AB, which can be statistically considered as possible outliers (Fig. 3). However, these data might be important for risk assessment [9]. HAAs were highest in NS followed by NL, NB and MB, respectively (Table 2; Fig. 2). Fig. 4 shows that a fraction of HAAs can be statistically considered as outliers in NL, NS and ON. HAAs in NB, MB, SK and AB can be approximated by the uniform distributions; however, this judgment is based on limited data (Fig. 4). HAAs data in NL, NS, QC, and ON were positively skewed (Fig. 4).

Concentrations of $CHCl_3$, BDCM, DBCM and $CHBr_3$ for different provinces are shown in Tables 3–6, respectively. Averages of $CHCl_3$ were lowest in PEI (2.8 μg/L) and highest in MB (139.9 μg/L). Averages of BDCM were 0.5–22.3 μg/L in various provinces (Table 4). DBCM (0.2–6.4 μg/L) and $CHBr_3$ (0.1–2.2 μg/L) were insignificant in most cases (Tables 5 and 6). From Tables 3–6, $CHCl_3$ comprised 74–97%, while BDCM comprised 3–14% of THMs. The larger pro-

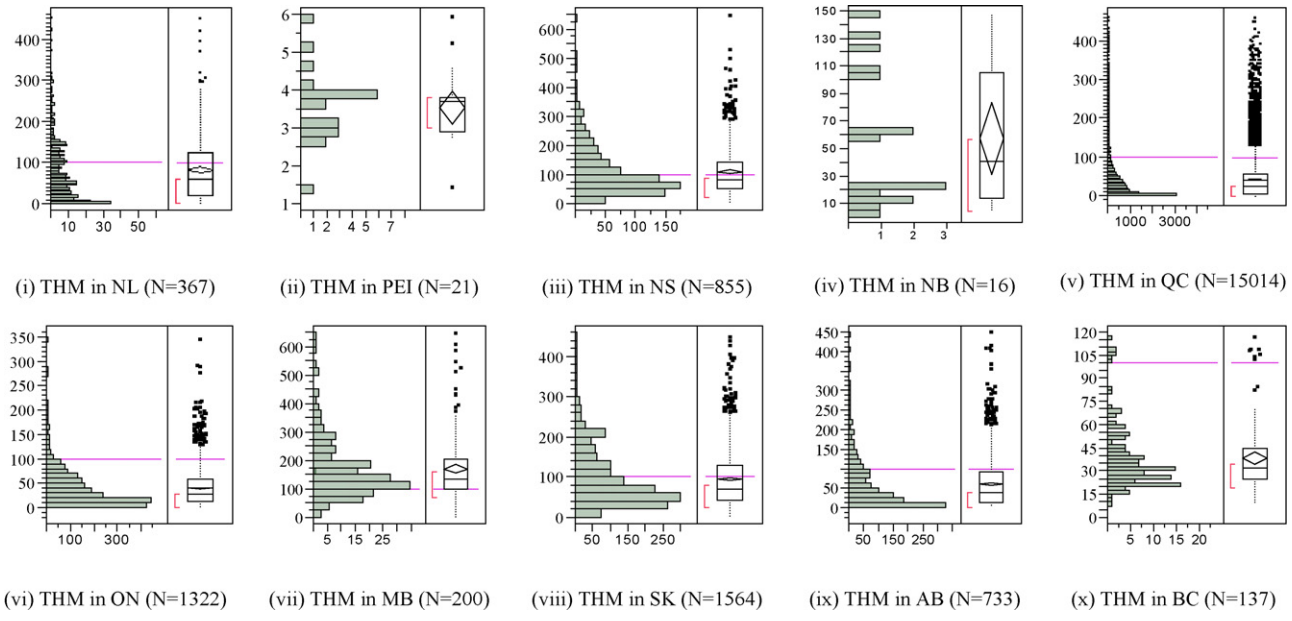

Fig. 3. Distributions plots for THMs in Canadian provinces (*N*: nos. of data; Purple line: Canadian regulatory limit of 100 μg/L).

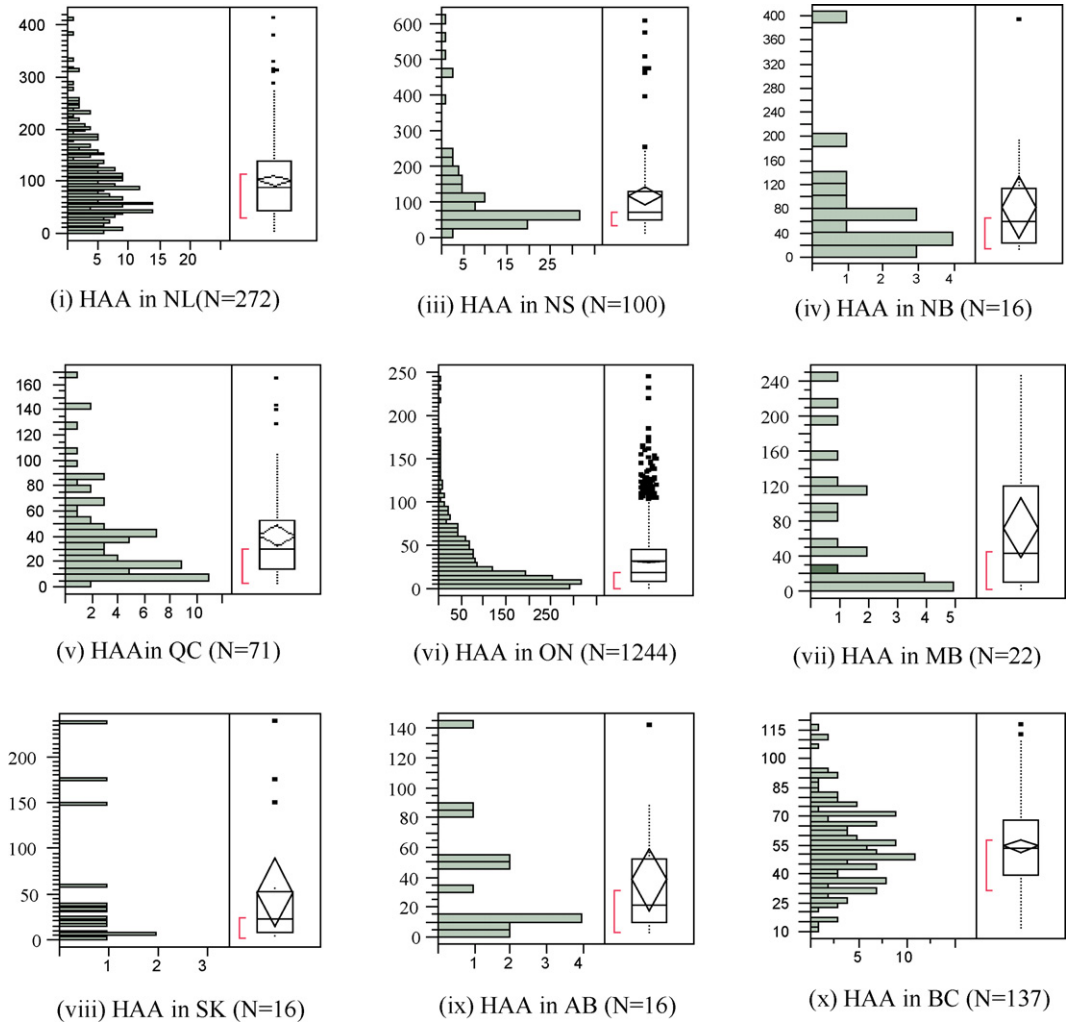

Fig. 4. Distributions plots for HAAs in Canadian provinces.

Table 3
Chloroform ($CHCl_3$) in Canadian drinking waters (μg/L).

Province	Mean	Range	10%	25%	50%	75%	90%	97.5%	SD
ON	33.1	0.1–285	1.9	5.4	20.3	50.5	81.1	132	36.9
QC	35.4	0.1–540.3	1.3	6.0	21.6	47.2	84.4	167.3	41.2
MB	139.9	0.3–620	42	69.3	100	180	290	509.3	112.5
SK	70.0	0.1–380	19	30	51	94	166	200	15.4
AB	52.1	0.02–422	3.2	7.7	37.6	80.5	132.8	210.4	60.6
BC	37.6	4–87	18.3	22.7	35.5	42.8	68.7	84.3	14.9
NS	101.6	0.2–630	28	44	74	140	220	320	83.6
NL	64.6	0–391	2.6	3.11	45.3	88.2	144	242	69.2
NB	49	3.1–126	5.9	18.2	47.4	86.7	93	114.1	37.3
PEI	2.8	1.1–4.8	1.3	1.7	2.3	3.0	3.2	4.5	2.1

SD: standard deviation.

Table 4
Bromodichloromethane (BDCM) in Canadian drinking waters (μg/L).

Province	Mean	Range	10%	25%	50%	75%	90%	97.5%	SD
ON	5.2	0–117	1.0	2.0	3.8	6.6	9.8	16.7	6.4
QC	3.4	0.01–220	0.4	0.94	2	4.2	7.5	14	5.7
MB	22.3	0.01–110	2.5	5.2	15	34	54.7	73.8	21.4
SK	13.0	0.5–138	3.0	5	7.0	14	33.0	64.8	56.7
AB	5.9	0.1–106.8	0.51	1.1	3.5	7.2	13.4	30.3	8.7
BC	1.1	0.5–5.2	0.5	0.6	0.75	1.1	2.5	5.2	0.96
NS	7.8	1–53	2.7	4	6.6	10	13.4	22.7	5.4
NL	7.2	0–64.4	1.2	3.1	5.5	9.3	15.3	26.5	7.2
NB	2.9	0.7–5.8	0.8	1.8	2.4	4.4	5.2	5.8	1.6
PEI	0.5	0.1–1.0	0.2	0.3	0.5	0.8	1.0	1.0	0.28

SD: standard deviation.

Table 5
Dibromochloromethane (DBCM) in Canadian drinking waters (μg/L).

Province	Mean	Range	10%	25%	50%	75%	90%	97.5%	SD
ON	2.0	0–22.6	0.4	0.8	2.0	3.2	4.2	7.5	4.1
QC	1.7	0.01–19	0.3	0.64	1.9	2.9	4.5	12.6	3.7
MB	6.4	0.1–66	0.1	0.1	1.1	6.2	22.1	44.9	11.4
SK	4.4	0.01–86	0.1	1	1	1	3.0	37	9.5
AB	2.0	0.01–108	0.1	0.1	0.2	0.8	4.2	17.3	7.4
BC	0.2	0–0.8	0.1	0.1	0.1	0.2	0.5	0.9	0.4
NS	1.5	0.4–25	1	1	1	1	2	4.9	2.2
NL	0.5	0–41.4	0	0.2	0.3	0.3	0.8	4.0	2.3
NB	0.5	0–1.7	0.0	0.1	0.4	0.5	1.1	1.6	1.4
PEI	0.2	0–0.8	0.0	0.0	0.0	0.3	0.6	0.7	0.2

SD: standard deviation.

Table 6
Bromoform ($CHBr_3$) in Canadian drinking waters (μg/L).

Province	Mean	Range	10%	25%	50%	75%	90%	97.5%	SD
ON	0.8	0.1–23	0.5	0.5	0.5	0.5	0.5	2.2	2.9
QC	0.7	0.1–14	0.5	0.5	0.5	0.5	0.5	1.8	2.6
MB	1.2	0.1–22	0.2	0.2	0.2	0.2	2.3	5.0	2.2
SK	2.2	0.01–78	1	1	1	1	2.0	13.0	6.9
AB	1.5	0.1–8.7	0.5	0.5	0.5	0.5	0.5	7.3	7.9
BC	0.1	0–0.9	0.1	0.1	0.1	0.2	0.4	0.7	0.4
NS	1.2	0–20	1	1	1	1	1	1	1.5
NL	0.1	0–51.6	0	0	0.2	0.3	0.3	0.8	1.5
NB	0.4	0–1.6	0.0	0.1	0.3	0.3	0.8	1.4	1.2
PEI	0.1	0–0.5	0.0	0.0	0.0	0.2	0.3	0.5	0.2

SD: standard deviation.

portion of $CHCl_3$ may be explained by the relatively low bromide in source water [1]. In HAAs, average TCAA were 18.7–65.8 μg/L, while DCAA and MCAA were 7.6–44.5 μg/L and 1.2–4.2 μg/L, respectively. These three compounds (TCAA, DCAA and MCAA) formed 86–98% (~94.6%) of HAAs (Table 7). The other two regulated HAAs (MBAA and DBAA) were 0.2–3.4% only (Table 7). The remaining four HAAs (BCAA, BDCAA, CDBAA and TBAA) contributed negligible percentages.

Concentrations of DBPs in different provinces were significantly different. Formation of THMs can be much higher during the summer months than those in the winter months [61], while formation of HAAs can be variable. Higher THMs in summer can be explained by: (i) higher reaction rates between chlorine and DBP precursors during the warm months than those of the cold months; (ii) higher chlorine doses in the warm months to deal with the higher chlorine demand and biological activity; (iii) higher amounts of NOM in summer compared to winter. HAAs can be affected by these factors and the presence of microorganisms in WDS. Biological activity of microorganisms may be increased significantly at the extremities of the large WDS during summer, where residual chlorine levels are generally low and the action of biofilms may degrade HAAs [68]. Differences in water sources, treatment approaches, disinfectants, distribution and amounts of NOM, bromide ions, seasonal variability and reaction periods can have significant role in such variability of DBPs formation. Another reason of DBPs variability among the provinces might be due to the differences in provincial regulations. Different provinces have different allowable limits of DBPs in drinking water (for example, in Ontario, THMs = 100 μg/L, while in Quebec it is 80 μg/L), which might have effects in the levels of efforts of reducing DBPs precursors prior to disinfection. Understanding of differences in these factors and DBPs variability can provide better idea on the selection of control measures.

2.2. Assessment of human health cancer risk

In risk assessment, understanding slope factor and chronic daily intakes (CDI) of contaminants is crucial. Slope factor is estimated as the upper 95% confidence value of cancer potency from animal bioassay data using various models [42,43]. Literally, it can be defined as the 95 percentile upper bound life-time probability of an individual's developing cancer as a result of exposure to a potential carcinogen. It has been reported recently that $CHCl_3$, which was known to be a probable human carcinogen, may not be a carcinogen [43–45]. The Integrated Risk Information System (IRIS) has characterized $CHCl_3$ as either human carcinogen or non-carcinogen [9]. To be protective against risk, use of a safe dose

Table 7
HAA_5 as percentage of total HAAs (mean HAAs values in μg/L).

Province	HAA	TCAA (%)	DCAA (%)	MCAA (%)	MBAA (%)	DBAA (%)
ON	28.60	65.38	26.57	5.24	1.0	1.4
QC	41.20	62.86	27.91	5.83	1.9	1.0
MB	72.40	67.27	23.76	3.31	1.4	0.7
SK	51.80	75.68	20.46	2.32	0.2	0.2
AB	38.40	61.46	26.82	4.17	0.3	0.3
BC	54.40	63.79	25.18	3.68	0.2	0.2
NS	116.20	54.73	38.30	3.61	1.7	1.6
NL	107.80	61.04	22.63	2.41	1.1	2.0
NB	85.72	60.78	32.90	3.50	0.1	0.1
PEI	–	–	–	–	–	–

SD: standard deviation.

Table 8
Human health toxicological data for THMs components.

Compounds	USEPA Group	Oral (SF) [mg/kg/day]$^{-1}$	Inhalation SF [mg/kg/day]$^{-1}$ [a]
Chloroform	B-2	0.0061	0.081
Bromodichloromethane	B-2	0.062	
Bromoform	B-2	0.0079	0.0039
Dibromochloromethane	C	0.0084	

B-2: probable human carcinogen; C: possible human carcinogen.
[a] For inhalation and dermal risk, where slope factors are not available, oral slope factors have been used in calculation.

of 0.01 mg/kg/day has been recommended [9]. The results of a reassessment study on $CHCl_3$ may be available in 2011 [9], which can provide better evidence in future. To be protective against cancer risks, this study used previously reported slope factor for $CHCl_3$. For the other THMs (e.g., BDCM, DBCM and $CHBr_3$), slope factors from IRIS [9] were used (Table 8). Upon availability of new information, risk assessment can be updated in future. In addition to THMs, many other possible/probable carcinogenic DBPs can be present in drinking water (e.g., DCAA, TCAA, NDMA and bromate). For example, DCAA is likely to be a human carcinogen (oral slope factor = 0.05 risk per mg/kg/day), TCAA is a possible human carcinogen, NDMA is a probable human carcinogen (oral slope factor = 51 risk per mg/kg/day) and bromate is a probable human carcinogen (oral slope factor = 0.7 risk per mg/kg/day). DCAA, NDMA and bromate have higher slope factors (DCAA/THMs = 0.8–8.2; bromate/THMs = 11.2–114.5; NDMA/THMs = 822.6–8360.7) than THMs (Table 8), indicating higher cancer potency. Occurrences of DCAA, TCAA, NDMA, bromate, chlorite and other common/emerging DBPs (e.g., haloacetonitriles, haloketones) are not regularly monitored in all provinces in Canada. Further to this, toxicities of many DBPs are not well understood [9]. Upon availability of required information, these DBPs can be included for human risk estimation. This study considers human health risk assessments from THMs in drinking water through ingestion, inhalation and dermal contact pathways. The details of the methodologies for human health cancer risk assessments through ingestion, inhalation and dermal contact pathways are available in literature [15–18,46,47,61]. In this paper, the approaches are summarized below.

2.2.1. Ingestion pathway

Cancer risks through ingestion of THMs with drinking waters are typically predicted [46] as:

$$CDI_{ing} = \frac{C_w \times IR \times EF \times ED \times CF}{BW \times AT} \quad (1)$$

where CDI_{ing} is the chronic daily intake via ingestion (mg/kg/day); C_w is the concentration of THMs in drinking water (μg/L); IR is the drinking water ingestion rate (L/day); EF is the exposure frequency (days/year); ED is the exposure duration (year); BW is the body weight (kg) and AT is the averaging time (days); CF is the mass conversion factor from μg to mg (0.001).

$$CR = CDI_{CHCl_3} \times SF_{CHCl_3} + CDI_{BDCM} \times SF_{BDCM} + CDI_{DBCM} \times SF_{DBCM} + CDI_{CHBr_3} \times SF_{CHBr_3} \quad (2)$$

where CR is the cancer risk; SF is the slope factor ([mg/kg/day]$^{-1}$).

The values C_w, IR, EF, ED, BW and AT are shown in Table 9. These parameters are random variables and prone to uncertainty [60]. In risk assessment, the 10th and 90th percentile values are often considered as the reasonable range for parameter values [46]. The 10th and 90th percentiles range minimize the biases associated with possible outliers. In this study, the 10th, 50th and 90th percentiles for the parameters were used to generate random numbers following triangular distributions. A total of 20,000 iterations were performed with MINITAB™ to determine the chronic daily intake of THMs.

2.2.2. Inhalation pathway

Showering can be an important indoor activity posing inhalation risk from THMs [15,48]. During showering, water temperature ranges 35–45 °C, increasing mass transfer of THMs. In a closed shower stall, THMs concentrations in shower air typically increase with the increase of water temperature and shower duration. Inhalation of THMs can occur from elevated concentrations of THMs in shower air and the impact of water on skin while showering. Jo et al. [17,18] predicted chloroform dose through inhalation as equivalent to 35% of the ingestion dose (0.7 L water ingestion) from a 10-min shower. However, this study did not consider variability in parameters, including effects of water flow rates, air exchange in the shower stall, implications of shower stall volume and variable human exposure scenarios. The chronic daily intakes of THMs through inhalation pathway can be estimated as:

$$CDI_{inh} = \frac{E_r \times C_a \times R \times t \times F \times EF \times ED \times CF}{BW \times AT} \quad (3)$$

where CDI_{inh} is the chronic daily intake of THMs through inhalation pathway (mg/kg/day); Er is the absorption efficiency of THMs through respiratory system; C_a is the THMs in shower air (μg/m^3); R is the breathing rate (m^3/min); t is the shower duration (min/shower); F is the shower frequency (shower/day); EF is the exposure frequency (days/year); ED is the exposure duration (year); BW is the body weight (kg), and AT is the averaging time (days); CF is the mass conversion factor from μg to mg (0.001). C_a can be modeled [49] as:

$$\frac{dC_a}{dt} = \frac{1}{V}[Q_w p_v C_w - k_a V C_a] \quad (4)$$

By introducing the boundary conditions $C_a|_{t=0} = 0$, Eq. (4) can be solved to obtain $C_a(t)$ as:

$$C_a(t) = \frac{Q_w p_v C_w}{k_a V}(1 - e^{-k_a t}) \quad (5)$$

where Q_w is the water flow (L/min); V is the shower stall volume (m^3); C_w is the THMs in cold water (μg/L); k_a is the shower air exchange rate (min^{-1}); P_v is the transfer efficiency of THMs from water to air.

In Eq. (5), THM concentrations in warm water are required. Chowdhury and Champagne [15] developed THMs growth rate model to predict THMs in warm water. The properties of the parameters considered in this model were found to be variable (temperature: 15–30 °C, pH: 7.6–8.4, TOC: 0.7–3.1 mg/L, bromide ions: 0.92–2.14 mg/L, chlorine dose: 1–5 mg/L, and contact time: 0.5–24 h) [2,15,36]. Understanding relative distributions of hydrophobic/hydrophilic fractions of NOM, variability in treatment processes, free residual chlorine and implications of geographical variability could further improve this model [49].

Table 9
Parameters and their values for THMs risk estimations (modified after Chowdhury and Hall [61]).

Parameter name	Symbol	Value	Reference
THM concentrations in water (μg/L); [$CHCl_3$, BDCM, DBCM, $CHBr_3$]	C_w	10, 50 and 90 percentile values in Tables 3–6	This paper
Water ingestion rate (L/day)	*IR*	0.74, 1.31, and 2.12	[46,58]
Exposure frequency (days/year)	*EF*	330, 350, and 360	[46,61]
Exposure duration (year)	*ED*	65, 77.1, and 82.7	[46,61]
Body weight (kg)	*BW*	62, 70.4, and 81	[46,61]
Averaging time (day)	*AT*	23725, 28142, and 30186	[46,61]
Water flow (L/min)	Q_w	8.7, 10.0, and 11.4	[17,18,48]
Shower stall volume (m^3)	*V*	1.67, 2, and 2.25	[17,18,48]
Shower time (min/shower)	*T*	7, 10, and 13	[17,18,48]
Air change (ACM)	k_a	0.021	[17,18,48]
THM absorbance through respiratory system	E_r	0.7, 0.77, and 0.84	[17,18,60]
THM transformation rate from water to air phase (%)	p_v	7.66, 8.76, and 9.86	[17,18,60]
Air intake rate (m^3/min)	*R*	0.014	[46,61]
Shower frequency (shower/day)	*F*	0.74	[46,61,48]
Area of body skin exposed to water (m^2)	A_s	1.69, 1.82, 1.94	[46,61]
Permeability of THM through skin (m/min)	P_d	(2.67, 3.0, and 3.5) × 10^{-5}	[50]

The THMs growth rate model for warm water can be presented as:

$$k = 0.0011\, e^{0.0407T} \tag{6}$$

where T is the temperature of water (°C), and k is the THMs growth rate at T°C (min^{-1}). Using Eq. (6), THMs in the warm water during shower can be estimated as:

$$C_{hw} = C_w e^{(k_{hw}-k_w)t} \tag{7}$$

where C_{hw} is the THM concentrations in warm water (μg/L); C_w is the THMs in cold water (μg/L); k_{hw} is the THMs formation rate for warm water (min^{-1}), which can be estimated using Eq. (6); k_w is the THMs formation rates for cold water (min^{-1}), and t is the shower duration (min). Using C_{hw} instead of C_w in Eq. (5), the shower air concentrations (C_a) can be estimated. THM concentrations in the air of shower stall (C_a) were used in Eq. (3) to predict CDI_{inh}.

WDS requires adequate protection against microbial recontamination [2,5], which warrants sufficient free residual chlorine in the WDS. Past studies have shown that complete removal of organics from source water is extremely costly [49]. As a result, it is likely that water will have a fraction of NOM throughout WDS, which can also be justified by the formation of additional THMs in the WDS [36]. As such, increasing temperature during showering and bathing may increase THMs in warm water.

2.2.3. Dermal contact pathway

During indoor activities, such as cooking, bathing, swimming in a pool and showering, health risks from exposure to THMs can occur through dermal contact. THMs are permeable through human skin at the rate of 0.16–0.21 cm/h [16,48,50,59]. Health risks from exposure to DBPs through dermal contact have been predicted in some past studies [15,17–19,61]. Jo et al. [17,18] reported that the dermal doses were equivalent to 32.5% of the ingestion dose (0.65 L water ingestion). Cleek and Bunge [51] demonstrated that the daily dermal doses from THMs were 40–70% of daily ingestion doses. However, variability in exposure, THMs permeability through skin, shower frequency, exposure frequency, exposure duration, human body weight and averaging time were not considered in some of these studies. It may be beneficial to improve risk assessment through incorporating variability in these parameters. Human cancer risk from THMs exposure through dermal contact can be estimated following:

$$CDI_{derm} = \frac{C_{hw} \times A_s \times P_d \times t \times F \times EF \times ED}{BW \times AT} \tag{8}$$

where CDI_{derm} is the chronic daily intake of THMs through dermal contact (mg/kg/day); C_{hw} is the THM concentrations in warm water (μg/L); A_s is the area of body skin exposed to water (m^2); P_d is the permeability of THMs through the skin (m/min). The *CDI* through ingestion, inhalation and dermal contact were used in Eq. (2) to obtain cancer risk through each exposure route. In Eq. (2), route specific slope factors were used (Table 8). While route specific slope factors were not available, oral slope factors were used [9].

In predicting *CDI* of THMs, it can be noted that the populations and the occurrence of THMs were not evenly distributed among the cities and rural areas within the provinces. The bigger cities and metropolitan areas generally have higher population bases than the sub-urban and rural areas. For example, populations in Ontario was approximately 12.6 million, while the populations in Toronto was nearly 5.3 million (42% of the province) in 2008. However, THMs in Toronto drinking water were 4–27.2 μg/L, with a mean of 12.4 μg/L, while the provincial THMs were 0.5–343 μg/L with a mean of 40.9 μg/L [36]. Approximately 42% of the populations of Quebec (3.1 million) live in Montreal, 54% of the population of British Columbia live in Vancouver (2.3 million) and 67% of the population of Alberta live in Calgary and Edmonton (2.2 million) [20]. Concentrations of THMs in Montreal were 0.1–132 μg/L with an average of 32.2 μg/L [39], while the drinking water in Vancouver reported THMs of 9–102.1 μg/L with an average of 37.3 μg/L [34]. THMs in Calgary and Edmonton were 0.6–187.3 μg/L with an average of 53.2 μg/L. To incorporate this variability, *CDI* for the populations in bigger cities (populations >1 millions, e.g., ON, QC, AB and BC) were predicted independently by using THM concentrations from the respective cities. The *CDI* for the remaining populations were predicted using THMs in their drinking water and, finally, *CDI* were normalized to the total populations of the respective provinces.

3. Results and discussion

3.1. Cancer risks

Cancer risks from THMs in different provinces through ingestion, inhalation and dermal contact pathways are shown in Fig. 5. Cancer risks through ingestion of THMs were highest, followed by inhalation and dermal contact pathways. Both inhalation and dermal contacts contribute to approximately 40% of total cancer risks. Overall cancer risks are shown in Table 10. Total cancer risks were predicted to be highest in MB (6.98×10^{-5}) followed by NS (4.46×10^{-5}) and SK (4.26×10^{-5}). Cancer risk exceedance probabilities beyond the regulatory limits [1.0×10^{-6} to 1.0×10^{-4}] are shown in Fig. 6. Cancer risk exceedance probabilities for MB, NS, SK and NL were higher at different risk levels (Fig. 6), indicating that these provinces might have greater chances of cancer from THMs. At risk level 1.0×10^{-6}, cancer risks exceedance probability is unity

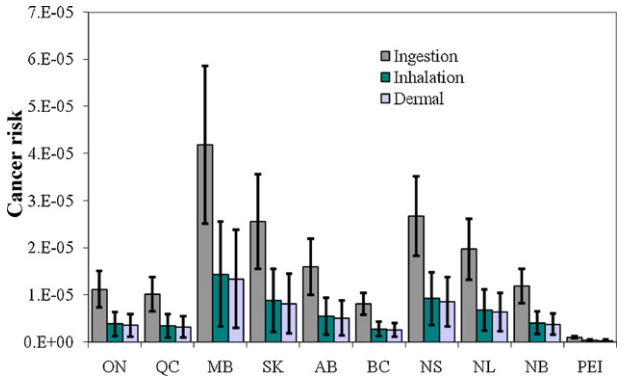

Fig. 5. Risk comparison (THMs) for ingestion, inhalation and dermal contact pathway.

Table 10
Human health cancer risks of THM in Canadian provinces.

Province	Cancer risk		
	10th	Mean	90th
ON	3.41E-06	1.87E – 05	5.47E – 05
QC	1.41E – 06	1.69E – 05	5.32E – 05
MB	1.47E – 05	6.98E – 05	2.81E – 04
SK	5.42E – 06	4.26E – 05	1.41E – 04
AB	3.17E – 06	2.67E – 05	9.34E – 05
BC	3.69E – 06	1.36E – 05	4.07E – 05
NS	1.09E – 05	4.46E – 05	1.39E – 04
NL	3.96E – 06	3.29E – 05	9.77E – 05
NB	3.14E – 06	1.99E – 05	5.48E – 05
PEI	5.82E – 07	1.80E – 06	4.30E – 06

for all provinces, meaning that there is perfect chance of at least one cancer incident per million of population in the provinces (Fig. 6). At risk level 1.0×10^{-5}, cancer risk exceedance probabilities for ON, QC, MB, SK, AB, BC, NS, NL, NB and PEI were 0.93, 0.84, 1.0, 1.0, 0.98, 0.84, 1.0, 0.99, 0.94 and 0.0, respectively, indicating corresponding probabilities of having 10 cancer incidents per million populations (Fig. 6). There is 34 percent chance of one cancer incident per 10,000 populations in MB. The higher cancer risks for MB may be better described by the fact that occurrences of THMs in MB are relatively high and the data used in this study were from the analysis of unpreserved samples and from the surface water-sourced systems only [35]. Preservation of samples prior to analysis and inclusion of groundwater sourced-systems could produce different results.

Possible cancer incidents from THMs are shown in Table 11. Total cancer incidents from THMs have been predicted to be 703 per year in Canada (Table 11). The highest cancer incidents were predicted for ON followed by QC, due to the fact that more than 60% of

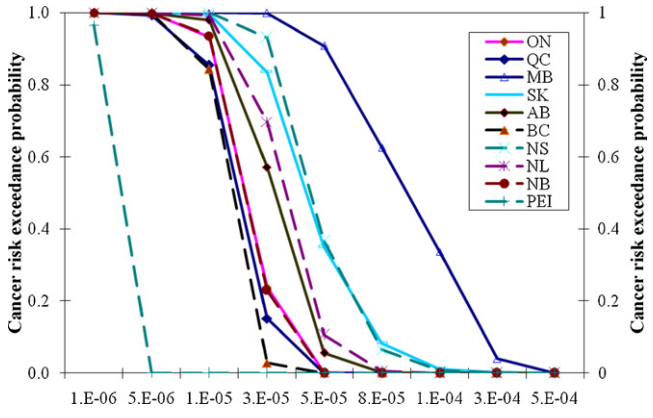

Fig. 6. Exceedance probability of cancer risk limits in the Canadian provinces.

the Canadian populations live in ON and QC. Previous studies have reported that DBPs may be associated with human bladder and colorectal cancer incidents [11,52–55]. The Canadian Cancer Society reported that occurrences of total bladder cancer incidents in ON varied in the range of 1570–1910 with an average of 1660 per year [56]. Based on the epidemiological evidence, King and Marrett [57] reported that approximately 14–16% bladder cancers in Ontario, which is equivalent to 232–265 bladder cancer incidents per year, might be attributed to the drinking water containing higher concentrations of DBPs. The current study predicted possible cancer incidents in Ontario from THMs exposure to be 235 per year, which is fairly consistent with the King and Marrett [57] study.

3.2. Cost associated with cancer risks

As THMs in drinking water may be a possible source of human cancer, regulatory agencies and authorities might be interested in gaining an idea of medical expenses associated with THMs. THMs is considered to have possible association with bladder and colorectal cancer incidents [11–13,52–55]. Chowdhury and Hall [61] estimated approximate costs of bladder and colorectal cancers associated with THMs exposure for some cities in Canada. This study used the values reported in that study, where average costs for typical bladder and colorectal cancers were estimated to be $208,000 and $187,000, respectively [58,61]. Using an average value of $200,000 for each cancer incident, medical expenses were estimated and shown in Table 11. Total medical costs of treating 703 cancer patients in all provinces were estimated to be $140.7 million. The highest additional costs of treatment were predicted for ON ($47.1 million/year) followed by QC ($25.3 million/year). In addition to cancer risks, chronic and sub-chronic effects such as cardiac anomalies, stillbirth, miscarriage, low birth weight and pre-term delivery could also be attributed to chlorinated drinking water. The cost of dealing with these effects may be high. For example, a low birth weight baby may need a total of $600,000 for special care, education, grade repetition and medical treatment for a full lifetime of 75 years and a cardiac anomaly patient may need $300,000 for his/her lifetime treatment [58].

3.3. Other DBPs and sources of risk

The estimated cancer risks in this study are from exposure to THMs only. In addition to THMs, many other DBPs have been reported in drinking water. To date, the reported DBPs include THMs, HAAs, haloacetonitriles, haloketones, nitrosamines, MX and MX Analogues, haloaldehydes, halomethanes, haloamides, tribromopyrrole, halonitromethanes, iodo-THMs, iodo-acids, haloacids, haloacetates, non-halogenated aldehydes and ketones, volatile organic compounds, oxyhalides, carboxylic acids and miscellaneous DBPs [7,9,10]. Many of these DBPs are more genotoxic than the regulated DBPs. For example, iodo-acetic acid, one of the five iodo-acids, is 2 times more genotoxic than bromoacetic acids, which is the most genotoxic of regulated HAAs [7]. Iodo-acids have been reported in chloraminated drinking water [7]. Dibromonitromethane, a compound of halonitromethanes, was found to be at least an order of magnitude more genotoxic to mammalian cells than MX, and is more genotoxic than all of the regulated DBPs, except for MBAA. Halonitromethanes were reported to be at least 10 times more cytotoxic than the regulated THMs [62]. Iodo-THMs might be more toxic than brominated and chlorinated compounds [7,10]. MX can account for as much as 20–50% of the total mutagenicity in chlorinated drinking water [63]. The mammalian cell toxicity testing showed that tribromopyrrole could be 8 times more cytotoxic than DBAA and to have about the same genotoxic potency as MX [7]. Nitrosamines can be another potent human carcinogen in drinking water (e.g., NDMA), while chloraminated drinking water

Table 11
Average number of cancer incidents from THMs exposure and cost.

Province	Population (million)	Cancer risk per million	Possible cancer incidents/year	Average cost/year (million US$)
ON	12.587	19	235	47.08
QC	7.482	17	126	25.29
MB	1.163	70	81	16.24
SK	0.982	43	42	8.37
AB	3.268	27	87	17.45
BC	4.185	14	57	11.38
NS	0.942	45	42	8.40
NL	0.515	33	17	3.39
NB	0.760	20	15	3.02
PEI	0.141	2	0	0.05
Total	32.025		703	140.7

might have higher concentrations of NDMA [9,10,22,36]. Bromate as well as brominated DBPs has been reported to be more toxic to human, while ozonation of bromide rich water can form bromate in drinking water [9,10]. Moreover, approximately 50% of the DBPs in drinking water are yet to be identified. To date, limited number of the known DBPs has been characterized in context to human and/or animal toxicity. Inclusion of these DBPs in risk assessment is likely to increase cancer risk. Understanding the occurrences of different DBPs and their effects to human is important to evaluate the overall risk from DBPs in drinking water.

Among the possible sources of cancer risks from DBPs in municipal water, chlorinated swimming pools have been given much attention in the recent years. Richardson et al. [64] reported more than 100 different types of DBPs in swimming pools in which many were nitrogen containing DBPs. Human inputs (e.g., urine, sweat, skin cells) might be the precursors for these nitrogen-containing DBPs [64]. Their study demonstrated that swimming pool waters might be as mutagenic as of drinking waters [64]. Mallika et al. [65] predicted cancer risk from exposure to THMs in swimming pool in Nakhon Pathom, Thailand. This study reported that approximately 94% of total cancer risk from THMs was due to the THMs in swimming pools [65]. The non-swimmers and swimmers had average cancer risks of 2.19×10^{-5} and 7.99×10^{-4}, respectively in their study [65]. The World Health Organization [66] reported $CHCl_3$ in the ranges of 4–420 μg/L and 0.69–112 μg/L in outdoor swimming pools in the US and Germany, respectively, whereas indoor swimming pools had higher concentrations. Nieuwenhuijsen [67] observed higher THMs in swimming pool water than tap water. If swimmers were in a swimming pool for 1 h, they would be exposed to THMs at a level of 141 times higher than showering in tap water for 10 min [65,67]. Indoor swimming pools in Canada can pose significant risks from exposure to DBPs. No systematic monitoring of DBPs in swimming pools in Canada has been administered to date. Only recent information generated in Quebec City exists [69]. This suggests understanding of DBPs occurrences and exposure to human while in swimming pools can provide better estimation of human cancer risk [70]. A new study to monitor DBPs in swimming pools in Quebec (Canada) is underway. Upon completion of this study, better idea on exposure can be achieved.

3.4. Reduction of risk of DBPs

Water treatment systems must provide microbiologically safe drinking water to the consumers [1,2,5,26–31]. Microbiological risks from improperly disinfected drinking water are more evident than those from DBPs in drinking water [27–31], and compromising of proper disinfection has never been suggested [26]. A number of municipal water systems have implemented alternative disinfectants to reduce DBPs without compromising proper disinfection [1,5,33–41]. Introducing alternative disinfectants might reduce regulated THMs and HAAs. However, unregulated and emerging DBPs with higher toxicities can be formed in drinking water in such alterations, which needs to be better understood in the event of introducing alternative disinfectants. Further to this, cost can be an important issue in using alternative disinfectants. Post reduction of DBPs might not be feasible in some classes of DBPs [22]. Reduction of precursors for DBPs can be achieved through enhanced treatments (e.g., membrane filtration, granular activate carbon, reverse osmosis) and/or source protection, which can form lower DBPs. However, enhanced treatments are typically more costly than the currently available treatment approaches. Further to this, removal of bromide ion and hydrophilic NOM is complex, while these can form significant fractions of brominated DBPs. These brominated DBPs can pose higher risks to human. Risk–risk or risk–cost tradeoffs can be introduced to better explain the complexity of controlling risk for drinking water.

This study also has limitations from population distributions and THMs occurrences within the major provinces. To obtain a clearer picture of risk from THMs, it may be helpful to study individual provinces through subdivisions based on populations and occurrences of THMs in respective areas. Risk assessment can be affected by individual habits associated with the use of chlorinated water, supplementary use of bottled water, swimming in chlorinated indoor/outdoor pools, etc. Use of THMs data from WDS (and not from the residential point of use such as: filtered tap water, stored water, etc.), inter and intra species variation of toxicity data and uncertainty associated with toxicity characterization also increase the variability in these estimates. Further, the estimated cancer risks are bound to slope factors, while the slope factors are the 95 percentile upper values. As such, the estimated cancer risks are likely to be the 95 percentile upper values, which can be further improved through better understanding of the slope factors. THMs in tap water (exposure points) in the house may be different from the THMs in the WDS, possibly due to stagnation of water in the plumbing pipes, heating in hot water tanks and other indoor handling of water prior to use (e.g., filtration, storing in a refrigerator). In dealing with uncertainty, there are several methods available to incorporate uncertainty associated with inadequate and/or imprecise data (e.g., probabilistic approaches, fuzzy rule based modeling, possibility theory, interval analysis). However, none of these approaches can be considered to be universal for all types of uncertainty encountered in environmental risk assessment. Based on the availability of data and the nature of problems, the appropriate approach can be selected to perform uncertainty analysis for environmental risk. Finally, the costs of cancer were estimated based on U.S. data, due to the fact that the estimates on the costs of cancer are rarely available in the Canadian context. As a result, deviations may be expected in the Canadian context.

Future studies are required to investigate the effects of plumbing pipes, hot water tanks, indoor use of water, and short-term and seasonal variability of THM occurrences on human health cancer risk assessment.

4. Conclusions

In this study, characterization of DBPs in Canadian drinking water and risk assessment from THMs exposure were performed. Approximate treatment costs for the increased numbers of cancer patients resulting from exposure to THMs were calculated. In summary:

- THMs and HAAs varied widely throughout the Canadian provinces.
- $CHCl_3$ and BDCM formed most of the THMs, while TCAA, DCAA and MCAA formed most of the HAAs.
- Cancer risk exceedance probabilities were highest in MB and NL for THMs and HAAs, respectively.
- Cancer risks from THMs might be associated with significant medical expenses.
- Better understanding of various DBPs in drinking water and their toxicities can provide better assessment for risk in future.
- Swimming pools can be a significant source of human cancer risks, which needs to be better explained in future.
- Introduction of alternative disinfectants needs to be carefully evaluated in context to variety of DBPs and their toxicities.

This study is associated with several limitations including those mentioned in the previous section. Despite these limitations, this study provides a basis for the estimation of THMs-associated risk in Canadian provinces. It also sets the direction for possible future research in comprehensive human health risk assessment involving exposure to DBPs through drinking water and a more detailed investigation of associated medical expenses in Canada.

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