Effects of plumbing systems on human exposure to disinfection byproducts in water: a case study

Shakhawat Chowdhury

ABSTRACT

Disinfection byproducts (DBPs) in water distribution systems (WDS) are monitored for regulatory compliance, while populations are exposed to DBPs in tap water that may be different due to stagnation of water in plumbing pipes (PP) and heating in hot water tanks (HWT). This study investigated the effects of water stagnation in PP and HWT on exposure and risk of DBPs to humans. Trihalomethanes (THMs) in PP and HWT were observed to be 1.1–2.4 and 1.6–3.0 times, respectively, to THMs in the WDS, while haloacetic acids (HAAs) were 0.9–1.8 and 1.2–1.9 times, respectively, to HAAs in the WDS. The chronic daily intakes of DBPs from PP and HWT were 0.6–1.8 and 0.5–2.3 times the intakes from WDS. The cancer risks from PP and HWT were 1.46 (0.40–4.3) and 1.68 (0.35–5.1) times the cancer risks from WDS. The findings may assist in regulating DBPs exposure concentrations.

Key words | cancer risk, disinfection byproducts, human exposure, plumbing system

Shakhawat Chowdhury
Department of Civil and Environmental Engineering,
King Fahd University of Petroleum and Minerals,
Dhahran 31261,
Saudi Arabia
E-mail: *schowdhury@kfupm.edu.sa*

INTRODUCTION

Disinfection byproducts (DBPs) in municipal water have been a concern due to their possible association with cancer and non-cancer risks to humans (USEPA 2006, 2014; Richardson *et al.* 2007, 2008; Health Canada 2008). During disinfection, reactions between natural organic matter (NOM) and disinfectant (typically chlorine, chloramines, chlorine dioxide, ozone) form various DBPs, including trihalomethanes (THMs), haloacetic acids (HAAs), haloacetonitriles, haloketones and other known and unknown byproducts (USEPA 2006; Richardson *et al.* 2007, 2008; Health Canada 2008). In addition to the regulated THMs (e.g. chloroform, $CHCl_3$; bromodichloromethane, BDCM; dibromochloromethane, DBCM; and bromoform, $CHBr_3$), emerging THMs including iodo-THMs have been reported in drinking water (Krasner *et al.* 2006). The iodo-THMs are more toxic than the regulated THMs in drinking water (Woo *et al.* 2002) while iodo-THMs are not regulated (USEPA 2006; Health Canada 2008) and not much is known about their occurrences in drinking water. Occurrences of iodo-THMs in drinking water are not regularly monitored in many countries.

Exposure to DBPs can occur through ingestion of drinking water and inhalation and dermal contact during regular indoor activities (e.g. showering, bathing and cooking). Ingestion, inhalation and dermal contact are believed to be the major exposure pathways for DBPs (USEPA 2006; Chowdhury & Champagne 2009; Chowdhury 2013). Early studies have reported that inhalation exposure of volatile organic compounds emitted from supply waters could pose a threat to human health (Andelman 1985; McKone 1987). Past studies have also reported risks to humans from exposure to THMs through inhalation and dermal contact during showering and bathing (Lee *et al.* 2004; Nuckols *et al.* 2005; Xu & Weisel 2005; Savitz *et al.* 2006; Semerjian & Dennis 2007). In most of these studies (Nuckols *et al.* 2005; Xu & Weisel 2005; Uyak 2006; Savitz *et al.* 2006), exposure assessment was performed based on the levels of these compounds measured in water from distribution systems (WDS).

doi: 10.2166/wh.2015.145

Consumers typically use water from taps in the house, while the regulatory/monitoring agencies measure DBPs within the WDS (e.g. Drinking Water Surveillance Program in Ontario, US Environmental Protection Agency). Depending on the size of plumbing system, water may stay in plumbing pipes (PP) for a considerable amount of time before it reaches the tap in the house (Dion-Fortier *et al.* 2009). This stagnation may be even longer during the period of off-peak hours (e.g. midnight to morning). Stagnation of water within PP allows additional reactions between residual NOM and free residual chlorine (FRC), which can increase DBPs. Such increased DBPs may pose an increased risk to human health. In addition, previous studies estimating exposure and risk during showering have assumed that THMs in warm water were equivalent to those of cold water. Warm water (35–45 °C) is typically used for showering, while warming of water during showering can increase THMs formation (Weisel & Chen 1994; Xu *et al.* 2002; Al-Omari *et al.* 2004; Chowdhury & Champagne 2009). Recent research also demonstrated that DBPs could increase between WDS and consumer taps due to stagnation within PP and hot water tanks (HWT) (Wu *et al.* 2001; Weinberg *et al.* 2006; Dion-Fortier *et al.* 2009). Use of water after a long period of stagnation in PP and/or heating in HWT may increase DBPs exposure and risks. A recent study addressed possible increase of THMs and health risks associated with warming of water during showering (Chowdhury 2013). However, changes in DBPs due to stagnation of water in PP and/or heating in HWT were not explained in this study.

Human exposure assessments are associated with uncertainty from different sources, including water ingestion rate, human body weight, exposure frequency, life expectancy, water temperature, shower stall volume, water flow rate, shower duration and air exchange rate (Xu & Weisel 2005; Chowdhury & Champagne 2009). Obtaining precise data for these parameters is difficult. Characterization of uncertainty in exposure assessment may provide flexibility in parameter values and a better understanding of human exposure and risks from DBPs. In this study, occurrences of THMs and HAAs in WDS, PP and HWT were investigated for two houses in Dhahran, Saudi Arabia. Possible effects of water stagnation in PP and HWT were investigated. The seasonal variability of DBPs was assessed. THMs and HAAs in WDS (entry points at the houses), PP and HWT were incorporated for human exposure and risk assessment. For each parameter, statistical distributions were developed to address uncertainty.

METHODOLOGY

Sample collection and analysis

Occurrences of DBPs in municipal water are affected by disinfectant, type and concentration of NOM, pH, bromide ion, water temperature, contact time and seasonal variability (Singer 1994; Chowdhury & Champagne 2008). Concentrations of THMs and HAAs in WDS, PP and HWT were investigated for two houses in Dhahran, Saudi Arabia. Water samples were collected from January 2012 to December 2012 through 12 sampling programs. At each sampling location, seven types of samples were collected at three different times of the day. The samples were collected in the order of: (i) water entry points of the houses (WDS) after the last use of water in late evening (S1); (ii) cold water samples in early morning before the first use, i.e. first flush to represent PP (S2); (iii) HWT samples in the morning (S3); (iv) WDS samples in the morning (S4); (v) cold water samples from PP in afternoon before water use (S5); (vi) HWT samples in afternoon (S6); and (vii) WDS samples in afternoon (S7). For samples of HWT, a temperature of approximately 55 °C was maintained to prevent risk of burn injuries. The sample collections were performed at the same time of the day for each type of sample (e.g. S1, S2) throughout the experimental period.

In this study, THMs, HAAs, FRC, total chlorine (TC), UV absorbance (UV_{254}), total organic carbon (TOC), dissolved organic carbon (DOC), temperature, pH, turbidity and conductivity were measured for each sample. Samples for measuring THMs and HAAs were taken in 100 mL glass vials containing a dechlorinating agent of 100 mg/L (ammonium chloride) and the samples for pH, turbidity, UV_{254}, TOC, DOC and conductivity were collected in 125 mL plastic bottles. The samples were transported to the laboratory in a cooler (4 °C). Temperature and pH were measured *in situ*. THMs were measured by gas chromatography equipped with mass spectroscopy detection (Varian chromatograph, model 3900 equipped with quadrupole mass spectrometer). The analysis was conducted according to the USEPA method 551.1 (USEPA 1995a). The analysis of HAAs was conducted according to USEPA

method 552.2 (USEPA 1995b) using gas chromatography with electron capture detector (Perkin Elmer Chromatograph, model AutoSystem XL). Further details on the extraction of THMs and derivatization of HAAs can be found in USEPA (1995a, 1995b). The FRC and TC were measured by HACH spectrophotometer (HACH DR 3900 model) following HACH methods 8021 and 8167, respectively. Turbidity was measured with a turbidimeter (HACH model 2100N). TOC and DOC were measured with a Shimadzu TOC analyzer (Model: TOC-L-CSN) following standard method 5310B (APHA, AWWA & WEF 1995). UV_{254} was measured using a spectrophotometer (Genesys 10 UV VIS model) at 254 nm with a 10 mm optical path quartz cell. Prior to measuring DOC and UV_{254}, samples were filtered through 0.45 μm membrane filters.

Exposure assessment

Ingestion pathway

Cancer risk through ingestion of DBPs with drinking water is typically predicted following USEPA (1998). The chronic daily intake (*CDI*) of DBPs through the ingestion route is predicted as:

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

where $CDI_{ing} = CDI$ via ingestion (mg/kg-day); Cw = concentration of THMs in drinking water (μg/L); IR = drinking water ingestion rate (L/day); EF = exposure frequency (days/year); ED = exposure duration (year); BW = body weight (kg); AT = averaging time (days); and CF = mass conversion factor from μg to mg (0.001). Details of the parameters for predicting risks are shown in Table 1.

Inhalation pathway

HAAs are non-volatile and thus are unlikely to impose risk through the inhalation pathway. The *CDI* of THMs through the inhalation pathway can be estimated following USEPA (1998) as:

$$CDI_{inh} = \frac{E_r \times C_a \times R \times t \times F \times EF \times ED \times CF}{BW \times AT} \quad (2)$$

where $CDI_{inh} = CDI$ of THMs through inhalation (mg/kg-day); E_r = absorption efficiency through respiratory system; C_a = THMs in shower air (μg/m^3); R = breathing rate (m^3/min); t = shower duration (min/shower); F = shower frequency (shower/day); EF = exposure frequency (days/year); ED = exposure duration (year); BW = body weight (kg); AT = averaging time (days); and CF = mass conversion factor from μg to mg (0.001). C_a depends on several factors, including THMs in shower water, shower stall volume, water flow rate, partition coefficient of THMs from water to air and air exchange rate of shower stall. Chowdhury (2013) predicted C_a using the following approach:

$$\frac{dC_a}{dt} = \frac{1}{V}[Q_w p_v C_w - k_a V C_a] \quad (3)$$

Assuming that the background concentration of THMs is zero prior to the showering event, the boundary condition can be introduced as: $C_a|_{t=0} = 0$. Such that the solution of Equation (3) becomes:

$$C_a(t) = \frac{Q_w p_v C_w}{k_a V}\left(1 - e^{-k_a t}\right) \quad (4)$$

where Q_w = water flow (L/min); V = shower stall volume (m^3); C_w = THMs in cold water (μg/L); k_a = shower air exchange rate (min^{-1}); P_v = transfer efficiency of THMs from water to air. In Equation (4), THMs in shower water are required. In estimating inhalation of THMs during showering, it is essential to predict THMs in shower water. During showering, hot water and cold water are generally mixed to attain a temperature in the range of 35–45 °C. In HWT, THMs increase continuously at a higher rate than that of the cold water, due mainly to a temperature-driven higher reaction rate. Chowdhury *et al.* (2010) reported higher concentrations of THMs in HWT (1.9–2.7 times) than those in the WDS. Other studies have also reported increased formation of THMs in municipal water due to temperature increase (Weisel & Chen 1994; Al-Omari *et al.* 2004). However, prediction of THMs increase in the mixed water during showering requires information on the baseline concentrations of THMs in hot and cold waters coming through the tap, their mixing proportions, FRC, residual organics and temperature of the mixed water. It is to be

Table 1 | Parameters and their values for this study (McKone 1987; USEPA 1998, 2011; Xu *et al.* 2002; Xu & Weisel 2003, 2005; Tan *et al.* 2007; Chowdhury & Champagne 2009; Chowdhury 2012)

Parameter name	Symbol	Value*
DBPs concentrations in water	Cw	Table 3
Water ingestion rate (L/day)	IR	0.74, 1.31, 2.12
Exposure frequency (days/year)	EF	330, 350, 360
Exposure duration (year)	ED	65, 77.1, 82.7
Body weight (kg)	BW	62, 70.4, 81
Averaging time (day)	AT	23,725, 28,142, 30,186
Water flow (L/min)	Q_w	8.7, 10.0, 11.4
Shower stall volume (m^3)	V	1.67, 2, 2.25
Shower time (min/shower event)	T	5, 10, 20
Heated water temperature	T_2	35, 40, 45 °C
Cold water temperature	T_1	15, 20, 25 °C
Air change (ACM)	k_a	0.018, 0.021, 0.023
THMs absorbance through respiratory system	E_r	0.7, 0.77, 0.84
THMs transformation rate from water to air phase (%)	p_v	7.66, 8.76, 9.86
Air intake rate (m^3/min)	R	0.012, 0.014, 0.016
Shower frequency (shower event/day)	F	0.72, 0.74, 0.76
Area of body skin exposed to water (m^2)	S_{skin}	1.69, 1.82, 1.94
Permeability of DBPs through skin (m/min)	P_d	
$CHCl_3$		$(2.54, 2.67, 2.79) \times 10^{-5}$
BDCM		$(2.87, 3.0, 3.13) \times 10^{-5}$
DBCM		$(3.25, 3.33, 3.42) \times 10^{-5}$
$CHBr_3$		$(3.42, 3.50, 3.58) \times 10^{-5}$
MCAA		$(1.5, 1.83, 2.16) \times 10^{-7}$
DCAA		$(2.82, 3.17, 3.52) \times 10^{-7}$
TCAA		$(3.01, 3.17, 3.33) \times 10^{-7}$
MBAA		$(2.01, 2.33, 2.66) \times 10^{-7}$
DBAA		$(3.68, 4.33, 4.98) \times 10^{-7}$
BCAA		$(2.05, 2.67, 3.28) \times 10^{-7}$
CDBAA, BDCAA, TBAA		Not available
Thickness of stratum corneum (cm)	d_{skin}	0.0015, 0.002, 0.003
MW (gm)	MW	$CHCl_3$: 119.4; BDCM: 163.8; DBCM: 208.3; $CHBr_3$: 252.8; MCAA: 94.5; DCAA: 128.9; TCAA: 163.4; MBAA: 138.9; DBAA: 217.8; BCAA: 173.4; BDCAA: 207.8; CDBAA: 252.3; TBAA: 296.7
Octanol-water partition coefficient	k_{ow}	$CHCl_3$: 93; BDCM: 126; DBCM: 127; $CHBr_3$: 128; MCAA: 1.67; DCAA: 8.3; TCAA: 21.4; MBAA: 2.57; DBAA: 16.6; BCAA: 12

*Data represent the minimum, mean and maximum values of parameters.

noted that the increase in THMs during showering is subject to the availability of FRC and organics. As such, concentrations of THMs are generally not constant over the showering period (Chowdhury & Champagne 2009). Chowdhury (2013) used the following equation to predict THMs growth rate and THMs in the shower water:

$$k = 0.0011e^{0.0407T} \tag{5}$$

where T = temperature of water (°C); and k = THMs growth rate at T°C (min^{-1}). Using Equation (5), THMs in the heated water during showering can be estimated as:

$$C_{hw} = C_w e^{(k_1 - k_2)t} \tag{6}$$

where C_{hw} = THMs in heated water (μg/L); C_w = THMs in cold water (μg/L); k_1 = THMs formation rate for heated water (min^{-1}), which can be estimated using Equation (5); k_2 = THMs formation rate for cold water (min^{-1}); and t = shower duration (min). Using C_{hw} instead of C_w in Equation (4), shower air concentration (C_a) is estimated. THMs in the air within the shower stall (C_a) are used in Equation (2) to predict CDI_{inh}. As demonstrated, THMs formation rates (Equations (5) and (6)) are subject to the presence of residual organics and FRC, while both can be available in shower water. As such, an increased temperature during showering may form additional THMs in shower water.

Dermal contact pathway

The molecular weight (MW) and octanol-water partition coefficient (K_{ow}) of THMs are in the ranges of 119.4–252.8 g/mole and 93–128 g/mole, respectively. The MW and K_{ow} of HAAs are 94.5–296.7 g/mole and 1.7–21.4 g/mole, respectively. The technical report on the assessment of non-occupational exposure to nonionizing chemicals noted that chemicals with K_{ow} between 0.1 and 10^5 and MW less than 700 g/mole might be absorbed through human skin (ECETOC 1994). It is plausible that THMs and HAAs may be absorbed through human skin during showering and/or swimming (ECETOC 1994), which may pose a risk to human health.

The CDI through the dermal route needs to be assessed by incorporating the unsteady and steady states of exposures. For DBPs, lag times to achieve steady state between the water attached to skin and the *stratum corneum* of skin are different, which can be in the range of 7.5–218.3 minutes (Chowdhury 2012). In many cases, showering duration can be less than the lag times. As such, unsteady state analysis is necessary for exposure assessment in these cases (Chowdhury 2012). The unsteady state and steady state estimates of DBPs exposure through the dermal route can be different (Chowdhury 2012). The steady state diffusion of low and high MW compounds through the *stratum corneum* was reported to be in the order of 10^{-13}–10^{-14} m^2/s and 10^{-15}–10^{-17} m^2/s, respectively (Scheuplein & Blank 1971). However, diffusion of chemicals prior to achieving steady state might be different (McKone & Howd 1992). When showering durations are more than the lag times, both unsteady and steady state analyses are necessary (Chowdhury 2012). Chowdhury (2013) applied the unsteady and steady state estimates for DBPs exposure through the dermal route during showering. The equations for predicting dermal exposure are presented following Chowdhury (2013) as:

$$L_t = \frac{d_{skin}^2}{6 \times D_{skin}} \tag{7}$$

where L_t = lag time (h); d_{skin} = thickness of *stratum corneum* (cm); D_{skin} = molecular diffusion of chemical through *stratum corneum* (cm^2/h). The molecular diffusion (D_{skin}) is estimated as:

$$D_{skin} = MW^{-0.6}\left[\frac{(2.4 \times 10^{-6} + 3 \times 10^{-5} K_{ow}^{0.8})}{k_m}\right] \tag{8}$$

where MW = molecular weight (g/mol); K_{ow} = octanol-water partition coefficient; K_m = partition coefficient between *stratum corneum* and chemical in water. This can be estimated as:

$$K_m = 0.64 + 0.25 K_{ow}^{0.8} \tag{9}$$

In predicting influx of DBPs through the *stratum corneum*, Fick's first law of diffusion can be described as:

$$J = \frac{D_{skin} \times \Delta C}{d_{skin}} \tag{10}$$

where d_{skin} = thickness of the *stratum corneum*; D_{skin} = molecular diffusion of chemical through the *stratum corneum*; and ΔC = concentration gradient between the upper and lower layers of the *stratum corneum*. The influx of DBPs is directly related to concentration gradient between the upper and lower layers of the *stratum corneum*. It is anticipated that concentrations of DBPs in shower water change exponentially with shower duration (Equations (5) and (6)). By substituting Equation (6) into Equation (10), we have:

$$J = \frac{D_{skin} \times \Delta C_w e^{(k_1 - k_2)t}}{d_{skin}} \tag{11}$$

It can be reasonably argued that the concentration gradient between DBPs at the upper and lower layers of the *stratum corneum* may not follow a linear pattern. To obtain a better prediction, duration of dermal exposure prior to achieving steady-state was discretized into one-minute intervals. Changes of DBPs were estimated for each interval. Thus, Equation (11) takes the form of:

$$J_i = \frac{D_{skin} \times \Delta C_w e^{(k_1 - k_2)t_i}}{d_{skin}} \tag{12}$$

$$J = \sum_{i=1}^{n} J_i \tag{13}$$

where $i = 1, 2, 3, \ldots\ldots, n$; and the time unit is t/n. The *CDI* of DBPs through dermal contact during showering can be estimated as:

$$CDI_{derm-ust} = \frac{J \times S_{skin} \times t \times F \times EF \times ED \times CF}{BW \times AT} \tag{14}$$

where $CDI_{derm-ust}$ = CDI of DBPs through dermal contact during the unsteady-state condition (mg/kg-day); J = diffusion through human skin (mg/cm^2/min); S_{skin} = area of body skin exposed to water (m^2); t = duration of shower per event (min/event); F = shower frequency (event/day); EF = exposure frequency (day/year); ED = exposure duration (year); BW = human body weight (kg); AT = averaging time (day); and CF = 10,000 (conversion factor for skin area from m^2 to cm^2). If showering duration is more than the lag time, dermal exposure for the steady-state period can be estimated as:

$$CDI_{derm-ss} = \frac{C_{hw} \times S_{skin} \times P_d \times t_{ss} \times F \times EF \times ED}{BW \times AT} \tag{15}$$

where $CDI_{derm-ss}$ = CDI of THMs through dermal contact (mg/kg-day) during steady-state; C_{hw} = THMs in warm water (μg/L); S_{skin} = area of body skin exposed to water (m^2); P_d = permeability of THMs through the skin (m/min); t_{ss} = difference between showering duration and lag time (min/event). The CDI of THMs through the dermal pathway can be obtained as:

$$CDI_{derm} = CDI_{derm-ust} + CDI_{derm-ss} \tag{16}$$

The route specific lifetime cancer risk and hazard index (HI) can be determined as:

$$CR = \sum_{i=1}^{m} \sum_{j=1}^{n} CDI_{ij} \times SF_{ij} \tag{17}$$

$$HI = \sum_{i=1}^{m} \frac{CDI_i}{R_f D_i} \tag{18}$$

where $i = 1, 2, \ldots m$ represents $CHCl_3$, BDCM, DBCM, $CHBr_3$, DCAA (dichloroacetic acid) and TCAA (trichloroacetic acid), respectively; $j = 1, \ldots n$ represents different routes of exposure; CR = cancer risk; SF = slope factor ([mg/kg/day]$^{-1}$) for specific route; and R_fD = reference dose (mg/kg/day). The SF for $CHCl_3$, BDCM, DBCM, $CHBr_3$, DCAA and TCAA are 0.0, 0.062, 0.084, 0.0079, 0.05 and 0.07 (mg/kg/day)$^{-1}$ while the R_fD are 0.01 mg/kg/day, 0.02 mg/kg/day, 0.02 mg/kg/day, 0.02 mg/kg/day, 0.004 mg/kg/day and 0.02 mg/kg/day, respectively (USEPA 2014). The SF represents the 95-percentile upper bound lifetime cancer risk from exposure to a carcinogen; and R_fD represents the safe dose that can be ingested without any adverse effect (USEPA 2014). The CDI were predicted by generating 5,000 random values using the parameter values in Table 1 and the DBPs data in Table 2, which were characterized through statistical distributions in Table 3. For the parameters in Table 1, triangular

Table 2 | THMs and HAAs in plumbing system of WDS

	S1	S2	S3	S4	S5	S6	S7
Turbidity (NTU)	0.47 (0.1)	0.49 (0.15)	0.44 (0.13)	0.45 (0.12)	0.41 (0.16)	0.51 (0.27)	0.44 (0.12)
UV_{254} (/cm)	0.01 (0.003)	0.01 (0.008)	0.01 (0.006)	0.01 (0.007)	0.02 (0.03)	0.01 (0.007)	0.01 (0.005)
pH	7.37 (0.8)	7.96 (0.79)	8.33 (0.74)	6.99 (0.85)	7.26 (0.81)	7.91 (0.64)	6.89 (0.78)
Temp at WDS (°C)	26.2 (2.2)	22.18 (2.02)	53.08 (4.5)	25.86 (2.3)	23.08 (2.25)	50.80 (9.8)	25.57 (2.29)
Conductivity (μS/cm)	1.62 (0.35)	1.63 (0.53)	1.79 (0.75)	1.72 (0.36)	1.89 (0.44)	1.68 (0.57)	1.74 (0.34)
Free chlorine (mg/L)	0.33 (0.14)	0.14 (0.08)	0.11 (0.044)	0.26 (0.16)	0.14 (0.09)	0.12 (0.04)	0.25 (0.11)
TC (mg/L)	0.44 (0.19)	0.23 (0.12)	0.16 (0.046)	0.35 (0.18)	0.19 (0.1)	0.16 (0.04)	0.33 (0.14)
TOC (mg/L)	4.63 (1.45)	3.97 (1.64)	4.10 (1.3)	3.21 (0.59)	2.99 (0.77)	3.83 (1.6)	3.16 (0.54)
DOC (mg/L)	3.46 (1.25)	2.86 (1.28)	3.01 (0.89)	2.23 (0.76)	1.89 (0.71)	2.56 (1.1)	2.12 (0.66)
THMs (μg/L)	6.94 (2.5)	11.1 (3.2)	14.6 (4.0)	6.5 (2.1)	10.4 (2.7)	13.2 (3.4)	6.4 (2.0)
HAAs (μg/L)	6.4 (1.8)	8.6 (2.8)	9.2 (2.2)	6.7 (2.1)	7.9 (2.5)	9.9 (2.7)	6.9 (2.7)

S1: samples from WDS after last use of water in the late evening; S2: cold water samples in the early morning prior to the first water use; S3: hot water samples in the morning; S4: samples from the WDS in the morning; S5: cold water samples in the afternoon, before water use; S6: hot water samples in the afternoon; S7: samples from the WDS in the afternoon; values in brackets represent standard deviations.

distributions were developed with the ranges as the minimum and maximum and the average as the most likely parameter values. The random data have characterized the uncertainties in the estimates.

Adjustment factor

The USEPA demonstrates that early-life exposure has a greater contribution to cancer appearing later in life. The USEPA suggested the following age-dependent adjustment factors (ADAF) to represent such effects (USEPA 2005):

1. For exposure before 2 years of age (i.e. spanning a 2-year time interval from the first day of birth up until a child's second birthday), a 10-fold adjustment.
2. For exposures between 2 and <16 years of age (i.e. spanning a 14-year time interval from a child's second birthday up until their sixteenth birthday), a three-fold adjustment.
3. For exposures after turning 16 years of age, no adjustment.

It is important to emphasize that these adjustments are combined with corresponding age-specific estimates of exposure to assess cancer risk. This is a departure from the way cancer risks have historically been based upon the premise that risk is proportional to the daily average of lifetime dose. The USEPA (2005) showed an example of lifetime exposure for a carcinogenic chemical with slope factor of 2.0 per mg/kg-day and lifetime average dose of 0.0001 mg/kg-day. Without considering the ADAF, risk was estimated to be 0.0002. With ADAF, the risk was estimated to be 0.00033, which is 1.63 times the risk without the ADAF. In this study, the estimated cancer risks were multiplied by 1.63 to better protect human health. However, upon availability of precise exposure data for these age groups (e.g. 0–2, 2–16 and 16 + years), cancer risks can be predicted for each age group and then normalized over the lifetime.

RESULTS

Occurrences of DBPs in WDS, PP and HWT

The averages and standard deviations of THMs and HAAs are presented in Figure 1. Concentrations of THMs were higher than HAAs in most of the sampling scenarios. For example, THMs and HAAs in S3 (morning hot water) were 14.6 μg/L and 9.2 μg/L, respectively, while for S2 (cold water in early morning prior to first use), these were 11.1 μg/L and 8.6 μg/L, respectively (Figure 1). The water quality parameters and averages of THMs and HAAs are shown in Table 2. Averages of THMs in WDS (S1, S4 and

Table 3 | Statistical distributions of DBPs in WDS, PP and HWT (μg/L)

Variable	Mean	St. Dev	Minimum	Maximum	Distribution
$CHCl_3$-WDS	5.511	2.177	1.264	9.3	T(1.26, 5.5, 9.3)
$CHCl_3$-PP	9.371	3.113	3.46	16.1	T(3.46, 9.37, 16.1)
$CHCl_3$-HWT	12.422	3.894	2.66	18.93	W(3.80, 13.76))
BDCM-WDS	0.8003	0.1808	0.53	1.3087	Ln(−0.2467, 0.218)
BDCM-PP	0.9272	0.2164	0.594	1.388	Ln(−01021, 0.233)
BDCM-HWT	1.1124	0.2209	0.744	1.6422	Ln(0.0888, 0.192)
DBCM-WDS	0.20465	0.06634	0.118	0.376	Ln(−1.632, 0.298)
DBCM-PP	0.2586	0.1722	0.126	1.08	Ln(−1.469, 0.429)
DBCM-HWT	0.2384	0.082	0.144	0.448	Ln(−1.48, 0.31)
$CHBr_3$-WDS	0.11532	0.03009	0.1	0.24	T(0, 0.115, 0.2)
$CHBr_3$-PP	0.1075	0.0112	0.1	0.13	T(0, 0.126, 0.25)
$CHBr_3$-HWT	0.1075	0.0112	0.1	0.13	T(0, 0.126, 0.22)
MCAA-WDS	0.548	0.3621	0	2.338	T(0, 0.548, 2.33)
MCAA-PP	0.5543	0.3035	0	1.2075	T(0, 0.5543, 1.20)
MCAA-HWT	0.7947	0.3147	0.216	1.3752	T(0.22, 0.79, 1.38)
DCAA-WDS	2.304	1.008	0.16	4.79	Ln(0.718, 0.54)
DCAA-PP	3.544	1.641	0.204	6.696	Gamma(2.77, 1.279)
DCAA-HWT	5.61	1.785	1.73	8.432	Gamma(7.787, 0.72)
TCAA-WDS	1.905	1.127	0.326	4.901	Ln(0.44, 0.679)
TCAA-PP	2.193	1.029	0.46	4.35	Ln(0.65, 0.565)
TCAA-HWT	1.586	0.726	0.286	3.328	Ln(0.328, 0.574)
MBAA-WDS	0.01014	0.03139	0	0.128	T(0, 0.01, 0.13)
MBAA-PP	0.00621	0.01947	0	0.088	T(0,0.006, 0.09)
MBAA-HWT	0.06354	0.05326	0	0.1748	T(0,0.063, 0.17)
DBAA-WDS	0.1508	0.0859	0	0.2938	T(0, 0.15, 0.29)
DBAA-PP	0.1688	0.0782	0	0.3042	T(0, 0.17, 0.31)
DBAA-HWT	0.1381	0.0905	0	0.2964	T(0, 0.14, 0.30)
BCAA-WDS	0.1263	0.0965	0	0.351	T(0, 0.126, 0.35)
BCAA-PP	0.1575	0.1218	0	0.3822	T(0, 0.16, 0.38)
BCAA-HWT	0.2082	0.128	0	0.4316	T(0, 0.21, 0.43)
CDBAA-WDS	0.5208	0.3681	0	1.114	T(0, 0.52, 1.11)
CDBAA-PP	0.5557	0.3559	0	1.22	T(0, 0.56, 1.22)
CDBAA-HWT	0.3705	0.3895	0	1.246	T(0, 0.37, 1.25)
BDCAA-WDS	0.5325	0.1259	0.29	0.774	T(0.29, 0.53, 0.77)
BDCAA-PP	0.4287	0.1629	0	0.752	T(0, 0.42, 0.75)
BDCAA-HWT	0.2249	0.1933	0	0.496	T(0, 0.22, 0.5)
TBAA-WDS	0.5543	0.2205	0	1.118	T(0, 0.55, 1.11)
TBAA-PP	0.6635	0.2583	0	1.474	T(0, 0.66, 1.47)
TBAA-HWT	0.5156	0.3116	0	1.568	T(0, 0.52, 1.56)

WDS: water distribution system; PP: plumbing pipes; HWT: hot water tanks; T: triangular distribution; Ln: lognormal distribution; Gamma: gamma distribution; W: Weibull distribution.

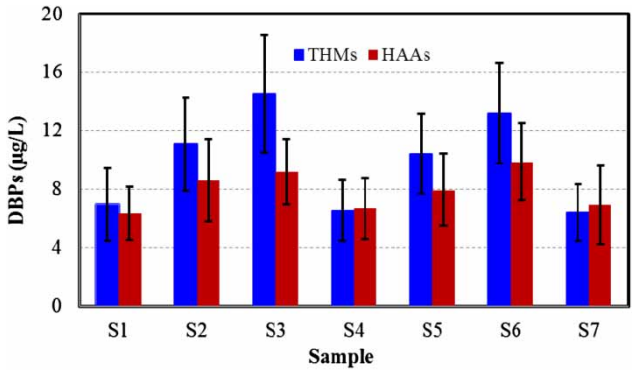

Figure 1 | THMs and HAAs concentrations in the different samples considered in the study (S1: samples from WDS after last use of water in the late evening; S2: cold water samples in the early morning prior to the first water use (first flush); S3: hot water samples in the morning; S4: samples from the WDS in the morning; S5: cold water samples in the afternoon before water use; S6: hot water samples in the afternoon; S7: samples from the WDS in the afternoon; error bars represent standard deviations).

S7), PP (S2, S5) and HWT (S3, S6) were 6.4–6.94 μg/L, 10.4–11.1 μg/L and 13.2–14.6 μg/L, respectively, while averages of HAAs in WDS, PP and HWT were 6.4–6.9 μg/L, 7.9–8.6 μg/L and 9.2–9.9 μg/L, respectively (Table 2). THMs in PP were 20–120% higher than the THMs in WDS. THMs in HWT were 80–260% higher than the THMs in WDS and 20–60% higher than the THMs in PP. A possible explanation for the increase of THMs may include extended and accelerated reactions between FRC and residual NOM in PP and HWT as well as decomposition of trihaloacetic acids (Zhang & Minear 2002). In almost all cases, THMs and HAAs in PP and HWT were higher than the THMs and HAAs in the WDS. Depending on sampling months, THMs in PP and HWT were up to 2.2 and 3.6 times the THMs in the WDS, respectively, while HAAs in PP and HWT were up to 2.1 and 2.2 times the HAAs in the WDS, respectively. The variability of THMs and HAAs due to sampling time (e.g. morning versus afternoon) was insignificant. THMs and HAAs in S1, S4 and S7 were statistically comparable (THMs: $P > 0.65$; HAAs: $P > 0.46$). For example, THMs in WDS were 6.9 μg/L, 6.5 μg/L and 6.4 μg/L in S1, S4 and S7, respectively. In these samples, HAAs were 6.4 μg/L, 6.7 μg/L and 6.9 μg/L, respectively. THMs and HAAs in PP were statistically comparable (THMs: $P = 0.45$; HAAs: $P > 0.57$). THMs in the PP were 11.1 μg/L and 10.4 μg/L in S2 and S5, respectively, while HAAs were 8.6 μg/L and 7.9 μg/L, respectively. No significant change in THMs and HAAs from HWT samples were observed (THMs: $P = 0.78$; HAAs: $P > 0.59$). THMs in S3 were 14.6 μg/L, while they were 13.2 μg/L in S6.

THMs and HAAs in WDS, PP and HWT showed seasonal variability (Figure 2). In January, THMs in S1 were in the range of 2.3–2.8 μg/L, while in August the range was 7.1–7.6 μg/L (Figure 2). In January and August, THMs in PP were in the ranges of 5.4–5.9 μg/L and 12.7–13.2 μg/L, respectively. In HWT, ranges of THMs were 5.9–6.5 μg/L and 15.3–16.1 μg/L in January and August, respectively. HAAs showed a variable pattern. Higher concentrations of HAAs were observed during September–November. In contrast to THMs, a decreasing trend of HAAs was noted in summer (June–August), which might be explained by the degradation of HAAs due to higher levels of microbial activity in summer. Overall, THMs and HAAs in PP and HWT were higher than THMs and HAAs in WDS. Increase of THMs and HAAs in PP and HWT may have implications for human exposure and risks.

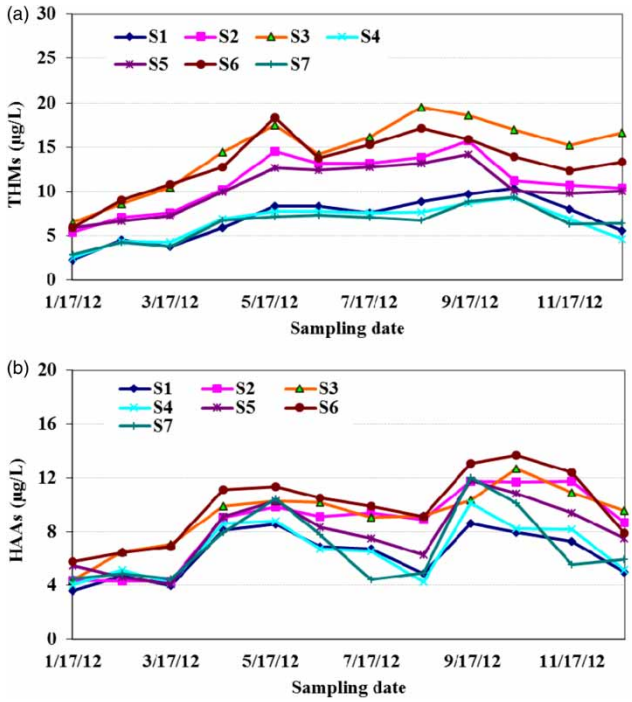

Figure 2 | Seasonal variability of THMs and HAAs in the WDS, PP and HWT (S1: samples from WDS after last use of water in the late evening; S2: cold water samples in the early morning prior to the first water use; S3: hot water samples in the morning; S4: samples from the WDS in the morning; S5: cold water samples in the afternoon, before water use; S6: hot water samples in the afternoon; S7: samples from the WDS in the afternoon).

Human exposure to THMs and HAAs

Time of sampling did not affect the concentrations of THMs and HAAs. As such, THMs and HAAs in WDS (S1, S4 and S7) were combined to obtain a single exposure scenario. The inclusion of data from different times represents overall diurnal variability. Similarly, THMs and HAAs in PP (S2 and S5) and HWT (S3 and S6) were combined to obtain independent exposure scenarios for PP and HWT. These data were characterized through statistical distributions (Table 3). Using these distributions, 5,000 random values were generated through statistical software (Minitab). The statistical distributions for the other input parameters were obtained from Table 1. The CDI for 13 DBPs (four THMs and nine HAAs) are shown in Table 4. The highest CDI was noted for $CHCl_3$ followed by DCAA, TCAA and BDCM. In most cases, CDI from PP and HWT were higher than from WDS (Table 4). It is to be noted that the CDI represents ingestion, inhalation and dermal routes of exposure for THMs, and ingestion and dermal routes for HAAs, because HAAs are not partitioned significantly from water to air during showering.

On average, CDI of $CHCl_3$ for PP and HWT were 1.8 and 2.3 times, respectively, higher than the CDI for WDS. The CDI of DCAA and TCAA for PP and HWT were also higher than the CDI for WDS. For DCAA, ratios of CDI for PP to WDS, HWT to WDS, and HWT to PP were 1.5, 2.4 and 1.6, respectively (Table 4). For TCAA, ratios of CDI for PP to WDS, HWT to WDS and HWT to PP were 1.2, 0.8 and 0.7, respectively, indicating that the concentrations of TCAA might have been decreased in the HWT. Past studies have reported that the increase in water temperature could transform TCAA into $CHCl_3$ and CO_2 (Wu *et al.* 2001). This transformation phenomenon was observed when water temperature exceeded 40 °C (Dion-Fortier *et al.* 2009). Overall, for the 13 DBPs, CDI for PP and HWT were 0.6–1.8 and 0.5–2.3 times, respectively, higher than the CDI for WDS. The CDI for DBCM, $CHBr_3$, monobromoacetic acid (MBAA), DBAA and BCAA were much lower in comparison to the other DBPs (Table 4). It is to be noted that CDI of HAAs were through the ingestion and dermal routes and the intake through inhalation was assumed negligible due mainly to their non-volatile nature (Chowdhury *et al.* 2014). Further, coefficients for dermal permeation

Table 4 | Summary of CDIs of THMs and HAAs (mg/kg-day)

	WDS				PP				HWT			
	Average	Min	Max	Std. dev.	Average	Min	Max	Std. dev	Average	Min	Max	Std. dev
$CHCl_3$	1.7×10^{-4}	3.6×10^{-5}	4.5×10^{-4}	6.5×10^{-5}	3.0×10^{-4}	6.5×10^{-5}	7.6×10^{-4}	1.1×10^{-4}	3.9×10^{-4}	3.8×10^{-5}	1.1×10^{-3}	1.5×10^{-4}
BDCM	2.5×10^{-5}	7.7×10^{-6}	7.5×10^{-5}	8.1×10^{-6}	3.2×10^{-5}	9.2×10^{-6}	9.2×10^{-5}	1.0×10^{-5}	3.5×10^{-5}	1.2×10^{-5}	9.0×10^{-5}	1.0×10^{-5}
DBCM	6.4×10^{-6}	1.4×10^{-6}	2.4×10^{-5}	2.5×10^{-6}	7.9×10^{-6}	1.1×10^{-6}	4.8×10^{-5}	4.1×10^{-6}	7.5×10^{-6}	1.7×10^{-6}	2.4×10^{-5}	3.0×10^{-6}
$CHBr_3$	3.2×10^{-6}	7.1×10^{-8}	9.5×10^{-6}	1.5×10^{-6}	4.0×10^{-6}	6.7×10^{-8}	1.2×10^{-5}	1.8×10^{-6}	3.6×10^{-6}	5.2×10^{-8}	1.0×10^{-5}	1.6×10^{-6}
MCAA	2.4×10^{-5}	3.4×10^{-7}	8.9×10^{-5}	1.4×10^{-5}	1.5×10^{-5}	4.5×10^{-7}	4.4×10^{-5}	6.9×10^{-6}	2.0×10^{-5}	4.4×10^{-6}	4.9×10^{-5}	7.3×10^{-6}
DCAA	6.0×10^{-5}	5.4×10^{-6}	4.3×10^{-4}	3.7×10^{-5}	9.0×10^{-5}	2.8×10^{-6}	5.0×10^{-4}	5.9×10^{-5}	1.4×10^{-4}	1.7×10^{-5}	5.2×10^{-4}	5.8×10^{-5}
TCAA	4.9×10^{-5}	2.7×10^{-6}	5.5×10^{-4}	3.9×10^{-5}	5.6×10^{-5}	4.8×10^{-6}	4.2×10^{-4}	3.7×10^{-5}	4.1×10^{-5}	3.4×10^{-6}	4.1×10^{-4}	2.7×10^{-5}
MBAA	1.2×10^{-6}	2.2×10^{-8}	4.7×10^{-6}	7.9×10^{-7}	8.0×10^{-7}	7.0×10^{-9}	3.0×10^{-6}	5.5×10^{-7}	2.0×10^{-6}	3.3×10^{-8}	6.0×10^{-6}	9.7×10^{-7}
DBAA	3.7×10^{-6}	7.0×10^{-8}	1.0×10^{-5}	1.7×10^{-6}	3.7×10^{-6}	4.1×10^{-8}	1.1×10^{-5}	1.7×10^{-6}	4.0×10^{-6}	2.4×10^{-8}	1.1×10^{-5}	1.8×10^{-6}
BCAA	4.0×10^{-6}	6.8×10^{-8}	1.2×10^{-5}	2.0×10^{-6}	4.5×10^{-6}	3.1×10^{-8}	1.4×10^{-5}	2.2×10^{-6}	5.4×10^{-6}	8.4×10^{-8}	1.5×10^{-5}	2.5×10^{-6}
CDBAA	1.4×10^{-5}	1.5×10^{-7}	4.1×10^{-5}	6.4×10^{-6}	1.5×10^{-5}	2.5×10^{-7}	4.3×10^{-5}	7.1×10^{-6}	1.3×10^{-5}	2.4×10^{-7}	4.4×10^{-5}	7.2×10^{-6}
BDCAA	1.3×10^{-5}	4.4×10^{-6}	3.1×10^{-5}	3.6×10^{-6}	9.9×10^{-6}	9.6×10^{-8}	2.8×10^{-5}	4.4×10^{-6}	6.1×10^{-5}	1.2×10^{-7}	1.9×10^{-5}	2.9×10^{-6}
TBAA	1.4×10^{-5}	1.7×10^{-7}	4.0×10^{-5}	6.4×10^{-6}	1.8×10^{-5}	5.1×10^{-7}	5.1×10^{-5}	8.5×10^{-6}	1.7×10^{-5}	1.1×10^{-7}	5.2×10^{-5}	8.9×10^{-6}

were not available for three HAAs (chlorodibromoacetic acid, CDBAA; bromodichloroacetic acid, BDCAA; and tribromoacetic acid, TBAA). As such, CDI through dermal contact was predicted for the remaining six HAAs. The coefficients of variation (CV), defined as the ratio of standard deviation to mean, were calculated for CDI. For CDI of THMs, CV varied in the ranges of 0.32–0.47, 0.33–0.52 and 0.30–0.45 for the WDS, PP and HWT, respectively. For HAAs, CV was in the ranges of 0.27–0.8, 0.44–0.68 and 0.36–0.52 for the WDS, PP and HWT, respectively.

Risk of THMs and HAAs

Three regulated THMs (BDCM, DBCM and $CHBr_3$) and two HAAs (DCAA and TCAA) were used to predict human health risks. In predicting cancer risks, it is to be noted that the route-specific CDI need to be multiplied by the route-specific SF. However, route-specific SF were not available for all routes. For comparative purposes, the SF through ingestion route from USEPA were used. Upon availability of route-specific SF, these values can be updated in future.

The cancer risks and hazard indices for WDS, PP and HWT sourced water are shown in Table 5. The cancer risks from PP and HWT were 1.46 (0.40–4.3) and 1.68 (0.35–5.1) times the cancer risks from WDS, while the cancer risks from the HWT water were 1.27 (0.46–3.97) times the cancer risks from the PP water. The HI for PP and HWT were 1.73 (0.63–4.37) and 2.37 (0.53–5.12) times the HI for the WDS water. Cancer risks from HAAs were 3.0–3.5 times the cancer risks of THMs. Average cancer risk from THMs and HAAs in WDS were 2.12×10^{-06} and 6.38×10^{-06}, respectively. HAAs in PP and HWT also had higher cancer risks than the corresponding THMs (Table 5). The higher cancer risks from HAAs may be partially explained by the higher concentrations of DCAA and TCAA than the three THMs (BDCM, DBCM and $CHBr_3$) considered for cancer risk assessment. Although $CHCl_3$ concentrations (Table 3) were much higher than DCAA and TCAA, $CHCl_3$ was not included in cancer risk assessment. However, in case of non-cancer risks, all four THMs were included, and the HI of THMs were higher than the HI from HAAs (e.g. DCAA and TCAA). The ratios of HI from HAAs to THMs were in the range of 0.78–0.93.

Average cancer risks from THMs and HAAs are shown in Figure 3. Ingestion route was the highest contributor of risks from THMs and HAAs. The risks of HAAs were higher than the risks of THMs through ingestion and dermal routes. For THMs, the ingestion route had the risks

Table 5 | Cancer risks and hazard indices for different sources of exposure

			Average	Min	Max	Std. dev.
Cancer risk	WDS	THMs	2.12×10^{-6}	7.11×10^{-7}	5.73×10^{-6}	6.18×10^{-7}
		HAAs	6.38×10^{-6}	8.64×10^{-7}	3.99×10^{-5}	3.37×10^{-6}
	WDS – Total		8.51×10^{-6}	2.28×10^{-6}	4.17×10^{-5}	3.56×10^{-6}
	PP	THMs	2.68×10^{-6}	7.68×10^{-7}	7.44×10^{-6}	8.23×10^{-7}
		HAAs	8.46×10^{-6}	8.11×10^{-7}	3.60×10^{-5}	4.10×10^{-6}
	PP – Total		1.11×10^{-5}	2.16×10^{-6}	4.02×10^{-5}	4.37×10^{-6}
	HWT	THMs	2.82×10^{-6}	1.06×10^{-6}	6.90×10^{-6}	7.81×10^{-7}
		HAAs	9.95×10^{-6}	1.73×10^{-6}	3.55×10^{-5}	3.67×10^{-6}
	HWT – Total		1.28×10^{-5}	3.34×10^{-6}	3.88×10^{-5}	4.03×10^{-6}
HI	WDS	THMs	1.86×10^{-2}	4.64×10^{-3}	4.81×10^{-2}	6.69×10^{-3}
		HAAs	1.73×10^{-2}	2.07×10^{-3}	1.10×10^{-1}	9.53×10^{-3}
	WDS – Total		3.59×10^{-2}	8.02×10^{-3}	1.36×10^{-1}	1.26×10^{-2}
	PP	THMs	3.24×10^{-2}	7.16×10^{-3}	7.97×10^{-2}	1.09×10^{-2}
		HAAs	2.53×10^{-2}	2.23×10^{-3}	1.33×10^{-1}	1.50×10^{-2}
	PP – Total		5.77×10^{-2}	1.58×10^{-2}	1.85×10^{-1}	2.01×10^{-2}
	HWT	THMs	4.13×10^{-2}	6.00×10^{-3}	1.10×10^{-1}	1.49×10^{-2}
		HAAs	3.74×10^{-2}	4.97×10^{-3}	1.33×10^{-1}	1.48×10^{-2}
	HWT – Total		7.88×10^{-2}	2.12×10^{-2}	2.03×10^{-1}	2.38×10^{-2}

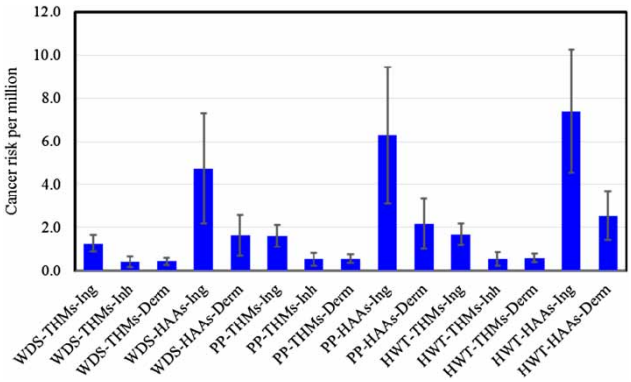

Figure 3 | Cancer risk of THMs and HAAs through different sources of exposure (WDS: water distribution system; PP: plumbing pipes; HWT: hot water tanks; Ing: ingestion route; Inh: inhalation route; Derm: dermal pathway).

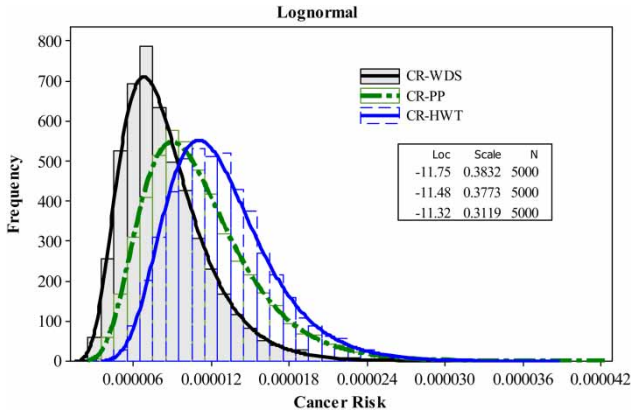

Figure 4 | Frequency distributions of cancer risks from WDS, PP and HWT sourced water (CR-WDS: cancer risk from water distribution system; CR-PP: cancer risk from plumbing pipes; CR-HWT: cancer risk from hot water tanks).

of 1.27×10^{-6}, 1.6×10^{-6} and 1.7×10^{-6} for WDS, PP and HWT, respectively. The risks through the inhalation route were 0.42×10^{-6}, 0.5×10^{-6} and 0.6×10^{-6}, respectively, while the risks through the dermal route were comparable to the inhalation route. Overall, ingestion, inhalation and dermal routes contributed approximately 59.7%, 19.7% and 20.6%, respectively, of the risks of THMs (Figure 3). While assessing exposure and risks from HAAs, ingestion and dermal routes were considered. Average risks of HAAs through ingestion for WDS, PP and HWT sourced water were 4.7×10^{-6}, 6.3×10^{-6} and 7.4×10^{-6}, respectively. The dermal route had risks of about 34.6% of the risks of ingestion route. Overall, ingestion and dermal routes contributed 74.3 and 25.7%, respectively, of HAAs risks.

The frequency distributions of cancer risks for WDS, PP and HWT sourced water are shown in Figure 4. Among 5,000 simulated scenarios, none had cancer risk of 1.0×10^{-6} or less. The cancer risks for WDS, PP and HWT showed lognormal distributions as: Ln(−11.75, 0.3832), Ln (−11.48, 0.3773) and Ln(−11.32, 0.3119), respectively. The 1st and 2nd parameters represent the location and scale, respectively, which are the median of the natural logarithm transformed data and the standard deviation, respectively. The skewness of risk distributions was also supported by the CV and skewness coefficients. The CV for WDS, PP and HWT were 0.42, 0.39 and 0.32, respectively, while the skewness coefficients were 1.74, 1.22 and 0.91, respectively. The right-tailed distributions of cancer risks from WDS, PP and HWT indicate the possibility of some events with higher risks. To explain the high-risk events, the cumulative distribution function (CDF) of total cancer risks for WDS, PP and HWT are plotted in Figure 5. In all cases, cancer risks were higher than 1.0×10^{-6} (Figures 4 and 5). At risk level 1.0×10^{-5}, the exceedance probabilities of cancer risks from WDS, PP and HWT are 26.8%, 53.8% and 73.5%, respectively (Figure 5). The PP and HWT had 27.0 and 46.7% more chance of exceeding a cancer risk of 1.0×10^{-5} than the WDS. There is a 4.7%, 16.4% and 25.1% chance that cancer risks exceed the risk level of 1.5×10^{-5} in WDS, PP and HWT, respectively. The PP and HWT had 11.7% and 20.4% more chance of exceeding a cancer risk of 1.5×10^{-5} than the WDS. At a risk level of 2.0×10^{-5}, the exceedance probabilities are 0.8%, 4.1% and

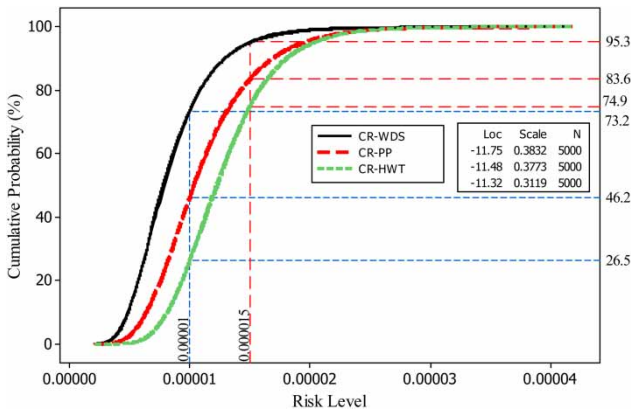

Figure 5 | Cumulative probability plots for the cancer risks from WDS, PP and HWT sourced water (CR-WDS: cancer risk from water distribution system; CR-PP: cancer risk from plumbing pipes; CR-HWT: cancer risk from hot water tanks).

5.6% in these sources, respectively. At a risk level of 5.0×10^{-5}, the exceedance probability is zero in all scenarios (Figure 5).

DISCUSSION

Various forms of chlorine or chloramines are used to protect drinking water throughout the WDS. Despite the efforts to remove NOM from drinking water, it is not entirely free from NOM. During the stagnation of water in PP and HWT, reactions between FRC and NOM are likely to form additional DBPs. In this study, water stagnation in PP and HWT has shown to increase the concentrations of DBPs in tap water. Such an increase may be attributed to extended reaction periods, temperature-driven higher reaction rates and/or decomposition of some DBPs. Past studies have reported decomposition of TCAA into $CHCl_3$ and CO_2 when temperature exceeded 40 °C (Wu *et al.* 2001; Dion-Fortier *et al.* 2009). This study observed seasonal variability of THMs and HAAs in WDS, PP and HWT. The higher temperature in summer increased THMs significantly while HAAs were variable. Past studies reported that some HAAs could serve as a source of nutrient for microbiological regrowth in WDS (McRae *et al.* 2004; Tung & Xie 2009). McRae *et al.* (2004) reported that monochloroacetic acid (MCAA) culture degraded MCAA and MBAA, while TCAA culture degraded TCAA and MCAA. Past studies have documented lower concentrations of HAAs at the extremities of a large WDS where bacterial activities were much higher, which might have degraded HAAs (Baribeau *et al.* 2004).

In multi-storey residential or office buildings, this can be an issue as water can be stagnant for hours to several days. Such stagnation can form additional DBPs and the FRC can be depleted, which can compromise microbiological water quality. There is a need to assess water quality from the taps in high-rise buildings. Stagnation of water is possible at the dead zone of a large WDS, which can form additional DBPs in this zone. Due to extended stay, FRC can be exhausted and microbiological quality of water can be compromised in the dead zones. Availability of a chlorine-boosting station may resolve this problem. Further, few regulatory agencies (e.g. USEPA, Health Canada) require DBPs concentrations on quarterly samples. The sampling program should be designed to represent the dead zone of a large WDS. In this study, DBPs were below the regulatory limits of the World Health Organization (WHO 2011) while local regulations are not available. The TOC and DOC were relatively higher while FRC were lower, which can be an issue with respect to water quality at the consumption points. Additional treatment may reduce TOC and DOC. Future study should perform comprehensive investigation through incorporating more sampling points from the dead zones of a WDS and the taps of high-rise buildings to protect water quality. A further challenge related to sampling points is to decide whether samples should be collected from the WDS or from the tap in the house. The populations are generally exposed to tap water. Assessment of human exposure based on tap water concentration may better protect human health.

CONCLUSIONS

This study analyzed THMs and HAAs in the WDS, PP and HWT, and predicted cancer risks to humans. Cancer risks from DBPs in the PP and HWT can be higher than the cancer risks from WDS. The study thus demonstrates that the use of DBPs data from the WDS is not adequate to represent the real exposure and risks of DBPs. The study has some limitations. The toxicological information for THMs and HAAs provided by the USEPA was developed using a set of transformations and extrapolations from animal bioassay data. Determination of slope factors was subjected to further assumptions, including a linear relationship between dose and response at low doses and 95-percentile upper value, while the true relationship may be different. Hence, the predicted cancer risks represent 95 percentile upper values. In addition to PP and HWT, indoor handling of drinking water such as aeration, freezing and filtration may also affect risks of DBPs. Further, DBPs are the mixture of many known and unknown compounds. Populations are generally exposed to the DBPs mixture, while many DBPs are yet to be identified. However, approaches to characterize cancer risks from DBPs mixture are not fully established yet. Despite several limitations, this study sheds light on the implications of plumbing systems for

human exposure and risks from DBPs in drinking water, which may better protect human health in future.

ACKNOWLEDGEMENTS

The author would like to acknowledge the support provided by King Abdulaziz City for Science and Technology (KACST) through the Science & Technology Unit at King Fahd University of Petroleum & Minerals (KFUPM) for funding this work through project No. 12-WAT2402-04 as part of the National Science, Technology and Innovation Plan.

REFERENCES

Al-Omari, A., Fayyad, M. & Qader, A. A. 2004 Modeling trihalomethanes formation for Jabal Amman water supply system in Jordan. *Environmental Modeling and Assessment* **9**, 245–252.

Andelman, J. B. 1985 Inhalation exposure in the home to volatile organic contaminants of drinking water. *Science of the Total Environment* **47**, 443–460.

APHA (American Public Health Association), AWWA (American Water Works Association) & WEF (Water Environment Federation) 1995 *Standard Methods for the Examination of Water and Wastewater*, 19th edn, Washington, DC.

Baribeau, H., Boulos, L., Haileselassie, H., Singer, P., Nichols, C., Schlesinger, S., Williams, S. & Gullick, R. 2004 Formation and decay of THMs and HAAs in five full-scale distribution systems. In *Proceedings of the Water Quality Technology Conference*. American Water Works Association, San Antonio, TX.

Chowdhury, S. 2012 Implications of using steady-state conditions in estimating dermal uptakes for volatile compounds in municipal drinking water: an example of THMs. *Human and Ecological Risk Assessment* **18** (5), 1051–1068.

Chowdhury, S. 2013 Exposure assessment for trihalomethanes in municipal drinking water and risk reduction strategy. *Science of the Total Environment* **463–464**, 922–930.

Chowdhury, S. & Champagne, P. 2008 An investigation on parameters for modeling THMs formation. *Global Nest Journal* **10** (1), 80–91.

Chowdhury, S. & Champagne, P. 2009 Risk from exposure to trihalomethanes during shower: probabilistic assessment and control. *Science of the Total Environment* **407** (5), 1570–1578.

Chowdhury, S., Rodriguez, M. J., Sadiq, R. & Serodes, J. 2010 Modeling DBPs formation in drinking water in residential plumbing pipes and hot water tanks. *Water Research* **45** (1), 337–347.

Chowdhury, S., Alhooshani, K. & Karanfil, T. 2014 Disinfection byproducts in swimming pool: occurrences, implications and future needs. *Water Research* **53**, 68–109.

Dion-Fortier, A. F., Rodriguez, M. J., Serodes, J. & Proulx, F. 2009 Impact of water stagnation in residential cold and hot water plumbing on concentrations of trihalomethanes and haloacetic acids. *Water Research* **43**, 3057–3066.

ECETOC (European Chemical Industry Ecology, Toxicology Centre) 1994 *Assessment of Non-occupational Exposure to Chemicals*. European Centre for Ecotoxicology and Toxicology of Chemicals. Technical Report 58, Brussels, Belgium.

Health Canada 2008 *Guidelines for Canadian Drinking Water Quality*. Prepared by the Federal-Provincial-Territorial Committee on Health and the Environment. Health Canada, Ottawa, Ontario.

Krasner, S. W., Weinberg, H. S., Richardson, S. D., Pastor, S. J., Chinn, R., Sclimenti, M. J., Onstad, G. D. & Thruston Jr, A. D. 2006 Occurrences of a new generation of disinfection byproducts. *Environmental Science and Technology* **40** (23), 7175–7185.

Lee, S. C., Guo, H., Lam, S. M. & Lau, S. L. 2004 Multipathway risk assessment on disinfection by-products of drinking water in Hong Kong. *Environmental Research* **94** (1), 47–56.

McKone, T. E. 1987 Human exposure to volatile organic compounds in household tap water: the indoor inhalation pathway. *Environmental Science and Technology* **21** (2), 1194–1201.

McKone, T. E. & Howd, R. A. 1992 Estimating dermal uptake of nonionic organic chemicals from water and soil: I. unified fugacity-based models for risk assessments. *Risk Analysis* **12** (4), 543–556.

McRae, B. M., LaPara, T. M. & Hozalski, R. M. 2004 Biodegradation of haloacetic acids by bacterial enrichment cultures. *Chemosphere* **55**, 915–925.

Nuckols, J. R., Ashley, D. L., Lyu, C., Gordon, S. M., Hinckley, A. F. & Singer, P. 2005 Influence of tap water quality and household water use activities on indoor air and internal dose levels of trihalomethanes. *Environmental Health Perspectives* **113** (7), 863–870.

Richardson, S. D., Plewa, M. J., Wagner, E. D., Schoeny, R. & DeMarini, D. M. 2007 Occurrence, genotoxicity, and carcinogenicity of regulated and emerging disinfection by-products in drinking water: a review and roadmap for research. *Mutation Research* **636**, 178–242.

Richardson, S. D., Fasano, F., Ellington, J. J., Crumley, F. G., Buettner, K. M., Evans, J. J., Blount, B. C., Silva, L. K., Waite, T. J., Luther, G. W., Mckague, A. B., Miltner, R. J., Wagner, E. D. & Plewa, P. J. 2008 Occurrences and mammalian cell toxicity of iodinated disinfection byproducts in drinking water. *Environmental Science and Technology* **42** (22), 8330–8338.

Savitz, D. A., Singer, P. C. & Herring, A. H. 2006 Exposure to drinking water disinfection by-products and pregnancy loss. *Am J. Epidemiology* **164** (11), 1043–1051.

Scheuplein, R. J. & Blank, I. H. 1971 Permeability of skin. *Physiological Reviews* **51**, 702–747.

Semerjian, L. & Dennis, J. 2007 Multipathway risk assessment of trihalomethanes exposure in drinking water of Lebanon. *Journal of Water and Health* **5** (4), 511–522.

Singer, P. C. 1994 Control of disinfection by-products in drinking water. *Journal of Environmental Engineering* **120** (4), 727–744.

Tan, Y. M., Lao, K. H. & Clewell, H. J. 2007 Reverse dosimetry: interpreting trihalomethanes bio-monitoring data using physiologically based pharmacokinetic modeling. *Journal of Exposure Science and Environmental Epidemiology* **17**, 591–603.

Tung, H. H. & Xie, Y. F. 2009 Association between haloacetic acid degradation and heterotrophic bacteria in water distribution systems. *Water Research* **43**, 971–978.

USEPA (US Environmental Protection Agency) 1995a *USEPA Method 551.1: Determination of Chlorination Disinfection Byproducts, Chlorinated Solvents and Halogenated Pesticides/Herbicides in Drinking Water by Liquid-Liquid Extraction and Gas Chromatography with Electron Capture Detection*. Office of Research and Development, USEPA, Ohio, USA.

USEPA 1995b *USEPA Method 552.2. Determination of Haloacetic Acids and Dalapon in Drinking Water by Liquid-Liquid Extraction, Derivatization and Gas Chromatography with Electron Capture Detection*. Revision 1.0, Office of Research and Development, USEPA, Ohio, USA.

USEPA 1998 *Human Health Risk Assessment Protocol*. EPA-530-D-98–001A; Office of Solid Waste, USA.

USEPA 2005 *Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens*. EPA/630/R-03/003F, Washington, DC.

USEPA 2006 National primary drinking water regulations: stage 2 disinfectants and disinfection byproducts rule: final rule. *Federal Register* **71** (2), January 4, 388–493.

USEPA 2011 *Exposure Factors Handbook*. Office of Research and Development, Washington, DC.

USEPA 2014 USEPA Integrated Risk Information System (IRIS) online database, Washington, DC. http://www.epa.gov/iris/subst/index.html (accessed December 2014).

Uyak, V. 2006 Multi-pathway risk assessment of trihalomethanes exposure in Istanbul drinking water supplies. *Environment International* **32** (1), 12–21.

Weinberg, H. S., Pereira, V. R. P. J., Singer, P. C. & Savitz, D. A. 2006 Considerations for improving the accuracy of exposure to disinfection by-products by ingestion in epidemiologic studies. *Science of the Total Environment* **354** (1), 35–42.

Weisel, C. P. & Chen, W. J. 1994 Exposure to chlorination byproducts from hot water use. *Risk Analysis* **14** (1), 101–106.

WHO (World Health Organization) 2011 *Guidelines for Drinking-Water Quality*. 4th edn. World Health Organization, Geneva.

Woo, Y. T., Lai, D., McLain, J. L., Manibusan, M. K. & Dellarco, V. 2002 Use of mechanism-based structure-activity relationships analysis in carcinogenic potential ranking for drinking water disinfection by-products. *Environmental Health Perspectives* **110** (Suppl. 1), 75–87.

Wu, W. W., Benjamin, M. M. & Korshin, G. V. 2001 Effects of thermal treatment on halogenated disinfection by-products in drinking water. *Water Research* **35** (15), 3545–3550.

Xu, X. & Weisel, C. P. 2003 Inhalation exposure to haloacetic acids and haloketones during showering. *Environmental Science and Technology* **37**, 569–576.

Xu, X. & Weisel, C. P. 2005 Human respiratory uptake of chloroform and haloketones during showering. *Journal of Exposure Analysis and Environmental Epidemiology* **15**, 6–16.

Xu, X., Mariano, T. M., Laskin, J. D. & Weisel, C. P. 2002 Percutaneous absorption of trihalomethanes, haloacetic acids and haloketones. *Toxicology and Applied Pharmacology* **184**, 19–26.

Zhang, X. & Minear, R. A. 2002 Decomposition of trihaloacetic acids and formation of the corresponding trihalomethanes in drinking water. *Water Research* **36** (14), 3665–3673.

First received 28 June 2015; accepted in revised form 1 November 2015. Available online 4 December 2015