Risk assessment of haloacetic acids in the water supply of Tehran, Iran

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ABSTRACT

Disinfection by-products are compounds occurring in drinking water as a result of reactions between disinfectants and impurities in raw water, and their occurrence has been a public health concern for the last four decades. Haloacetic acids (HAAs) are one of the major by-products of chlorination. The concentration and variation of HAAs was monitored in 540 samples taken from tap water in six water and wastewater districts of Tehran, Iran. Seasonal variation indicated that natural organic matter and HAA levels were much higher in the spring and fall seasons. The concentrations of HAAs in drinking water samples varied with water sources. They were higher in drinking water obtained from surface water. In this study, the analysis method of human health risk assessment with regard to exposure to HAAs by drinking water in Tehran was based on the United States Environmental Protection Agency (USEPA) guideline. It was found from these studies that the risk to human health appears to be negligible.

Key words | drinking water, haloacetic acids, health risk assessment, seasonal variations, Tehran

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doi: 10.2166/ws.2016.189

INTRODUCTION

Natural organic matters (NOMs) are produced by physical, chemical, and biological activities. They can react with disinfectants and form disinfection by-products (DBPs). The most common method of water disinfection is chlorination. Haloacetic acids (HAAs) are good indicators of overall DBPs in chlorinated waters (Chang *et al.* 2010). HAAs are a combination of nine species: monochloroacetic acid (MCAA), dichloroacetic acid (DCAA), trichloroacetic acid (TCAA), monobromoacetic acid (MBAA), dibromoacetic acid (DBAA), tribromoacetic acid (TBAA), bromochloroacetic acid (BCAA), dichlorobromoacetic acid (DCBAA), and dibromochloroacetic acid (DBCAA). The bromine compound's HAAs are formed in the presence of bromine in water. The USEPA (United States Environmental Protection Agency) has regulated a maximum contaminant level (MCL) of 60 $\mu g\,L^{-1}$ for HAA_5 (MCAA, DCAA, TCAA, MBAA, DBAA). Also, the World Health Organization has published guidelines for MCAA, DCAA, and TCAA as 20, 50 and 200 $\mu g\,L^{-1}$, respectively (WHO 2006). In addition, the USEPA has established an MCL goal (MCLG) of zero for DCAA based on sufficient evidence of carcinogenicity in animals, and an MCLG for TCAA of about 0.3 mgL^{-1} based on developmental toxicity and possible carcinogenicity (EPA 1998). Human consumption of chlorinated drinking water has been epidemiologically linked to bladder, kidney, and rectal cancers (OEHHA 2004). Since HAAs and trihalomethanes (THMs) are the most prevalent and well-documented DBP compounds in drinking water, they are generally considered as indicators of DBP exposure in epidemiological investigations (Kumari *et al.* 2015). Exposure to HAAs has been associated with adverse reproductive outcomes, and cancers of the digestive or genitourinary organs (Llopis-González *et al.* 2011; Pourmoghadas & Kinman 2013; Villanueva *et al.* 2014, 2015). The monitoring of HAAs in water has been carried out in many countries (Rodriguez *et al.* 2007; Lou *et al.* 2010; Zhang *et al.* 2010; Ghoochani *et al.* 2013; Al-shatri *et al.* 2014; Aizawa *et al.* 2015) and a few countries have set regulations to control HAAs in drinking water.

Recent studies have mainly attempted the hazard assessment of DBPs in drinking water to improve exposure assessment, e.g., Lee *et al.* (2004) evaluated the association between THM exposure through three different pathways and lifetime cancer risks in Hong Kong. Results showed that people had a higher risk of cancer through oral ingestion (Lee *et al.* 2004). According to animal studies, DCAA is believed to be a more potent carcinogen than THMs (Bull & Kopfler 1991). Direct human exposure to HAA_5 occurs via ingestion of disinfected tap water, inhalation, and dermal contact. The USEPA has classified TCAA as a possible human carcinogen, Group C, based on a lack of human carcinogenicity data and limited evidence of an increased incidence of liver neoplasms in both sexes of one strain of mice; 7×10^{-2} mg $(kg\text{-}day)^{-1}$ is selected as the oral cancer slope factor for TCAA.

The reference dose (RfD) is used in risk assessments and is an estimate of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. The RfD is useful as a reference point from which to gauge the potential effects of the chemical at other doses. Usually, doses less than the RfD are not likely to be associated with adverse health risks. As the frequency and/or magnitude of exposures exceed the RfD, the probability of adverse effects in a human population increases. It is generally expressed in units of milligrams per kilogram of bodyweight per day. The RfD for oral TCAA is 0.02 mg $(kg\text{-}day)^{-1}$ (USEPA 2011). The EPA has classified DCAA as a probable human carcinogen, Group B2, and derived an oral slope factor of 0.05 mg $(kg\text{-}day)^{-1}$ based on liver adenoma and carcinomas in male B6C3F1 mice (USEPA 2003). The USEPA has derived an RfD of 0.0004 mg $(kg\text{-}day)^{-1}$ for oral DCAA based on lesions observed in the testes, cerebrum, cerebellum, and liver in a subchronic dog oral bioassay (USEPA 2003; WHO 2005).

According to a study on Tehran's outlet water treatment plants in 2013, the presence of HAAs was confirmed (Ghoochani *et al.* 2013). Therefore, the evaluation of exposure concentrations of these chemicals in distribution drinking water and their health risk assessment in the general

population and vulnerable groups was considered as a consequential issue. This is the first time such a study has been carried out in Iran.

METHODS

Water sampling and analysis

Tehran's drinking water is provided from both surface and groundwater resources. Karaj River in the west, Jajrood River in the east, and Lar River in the north and northeast, and more than 400 wells dispersed in various parts of Tehran (mostly in the southern part) supply the required water for the megacity of Tehran. Tehran is divided into six water and wastewater districts. These are managed by the City of Tehran Water and Wastewater Company (CTWWC). The surface water usually supplies about 50% to 65% of the drinking water for the entire city depending on the season and the amount of rainfall (CTWWC 2009). There are five operating water treatment plants in Tehran. Water treatment plants numbers 1 and 2 are fed from Karaj River, water treatment plants numbers 3 and 4 are fed from Jajrood River, and the fifth water treatment plant is fed from Lar River. Well water does not go through any drinking water treatment plants and is only chlorinated before being pumped into the distribution system. Considering the geographical location of these water treatment plants, it seems that processed water is scattered in districts 1, 2, and 3, which are located in the north, northeast, and northwest parts of the city. Well water is mostly distributed in districts 6, 5, and 4, which are located in the south, southwest, and southeast parts of the city, and a small amount of surface water is pumped to these districts. Sometimes, in a surface water shortage situation due to low rainfall, it is possible to pump well water to areas that use surface water.

Water samples were taken randomly from six water and wastewater districts (20 locations) from April to December 2013. Three replicate analyses were done for each location. The samples were taken directly from the taps of consumers after letting the water run for several minutes. The samples were analyzed for pH, free residual chlorine, and HAAs. The pH and free residual chlorine (mg L^{-1}) were measured onsite, and for HAA analysis samples were stored in 40 mL amber glass containers with screw caps and PTFE-faced septa. A dechlorinating agent (ammonium chloride) to convert free chlorine to monochloramine was added to the sample bottles onsite (Chang *et al.* 2010). Method No. 6251 Standard Water and Wastewater analysis is no longer commonly used in new research as it utilizes diazomethane. Recent researchers use EPA Method No. 552.2, 552.3; however, we used a different method via direct derivatization of HAAs by dimethyl sulfate to reduce the analysis time and in consideration of the need for more green chemistry in research. Using a new static headspace gas chromatography–electron capture detector (GC-ECD) method and without a manual pre-concentration, HAAs were analyzed. The calibration curve was designed by injection of 10, 20, 40, 60, 100, and 200 μg L^{-1} concentrations of HAA_5 and 2-3 dibromopropionic acid as internal standard. A 5 mL water sample was taken into a 10 mL headspace vial without pre-filtration. The headspace vial was closed with a gas-tight cap after adding 5 g of pure Na_2SO_4 (anhydrous), 800 μg tetrabutyl ammonium hydrogen sulfate (TBA-HSO_4) as an aqueous solution and 100 μL dimethyl sulfate. The headspace vial was moved to a 60 °C water bath, where the solution was mixed for 30 min with a glassy magnetic stirring bar, allowing *in situ* derivatization. After these procedures, the sample was ready for analysis in only a few minutes. A Varian cp-3800 gas chromatograph with electron capture detection was used for HAA analysis. Separations were done on a fused-silica capillary column (50 m, 0.32 mm, 0.25 μm), with helium as a carrier gas, at a linear velocity of 35 mL/min and pressure 5 psi. Nitrogen was used as makeup gas. The instrumental temperatures were as follows: injector temperature 275 °C; initial oven temperature 40 °C (held for 3 min), increased to 50 °C at a rate of 5 °C min^{-1} (held for 2 min), increased to 110 °C at a rate of 5 °C min^{-1} (held for 2 min), then increased to the final temperature of 250 °C at a rate of 30 °C min^{-1}, where it was held for 1 min. The inlet was operated in splitless mode and the detector temperature was set at 300 °C. The detection limits for all of the compounds (HAA_5) at a signal-to-noise (S/N) ratio of 3 was 0.5 μg L^{-1}. The limits of detection for MCAA, DCAA, TCAA, and MBAA were recognized as 1.7, 1, 1.4, and 1 μg L^{-1} . This was not detected for DCAA.

Daily intake and health risk assessment

For assessing human exposure to chemical contaminants, risk assessment through exposure assessment of specific environmental media can be used. In this study, the carcinogenic and non-carcinogenic risk assessment for exposure through oral ingestion of TCAA and DCAA was considered. Potential risks to human health can be evaluated quantitatively by combining potential exposure and toxicity data. A distinction is also made between non-carcinogenic and carcinogen end points. For the non-carcinogenic risk assessment, the hazard quotient (HQ) of DCAA and TCAA was calculated. Carcinogenic risk assessment for DCAA and TCAA was determined by excess lifetime cancer risk (ELCR).

Estimated daily intake (EDI) of HAAs is based on the daily average consumption of drinking water, concentration of HAAs in drinking water, and bodyweight. EDI is expressed in units of micrograms per kilogram of bodyweight per day. Therefore, it is necessary to calculate the EDI from two types information: (1) the concentration of DCAA and TCAA in water and (2) the recommended water intake per day based on bodyweight:

$$\text{EDI} = \text{MC} \times \text{waterconsumption}$$

where MC is the concentration of DCAA and TCAA.

The water consumption data in target groups (infants, children, teenagers, and adults) were estimated according to a study by Karyab *et al.* (in press) in Iran. The water consumption rates in infants (0–2 years old), children (2–6 years old), teenagers (6–16 years old), and adults (≥16 years old) were 0.095, 0.05, 0.04 and 0.03, L kg bodyweight^{-1} day^{-1}, respectively.

The HQ is the ratio between the RfD and the EDI; an HQ value less than 1 indicates that it is unlikely even for sensitive populations to experience adverse health effects. If the HQ is greater than 1 (i.e., if exposure/RfD exceeds unity), then adverse health effects are possible:

$$\text{HQ} = \frac{\text{EDI}}{\text{RfD}}$$

The health risk assessment for carcinogenic effect was estimated by the ELCR. ELCR is an estimate of the potential increased risk of cancer resulting from lifetime exposure to constituents detected in media at the facility. ELCR is calculated based on the following formula:

$$\text{ELCR} = \text{drinking water unit risk} \times \text{MC}$$

and 1.4×10^{-6} and 2×10^{-6} per μg L^{-1} are selected for drinking water unit risk by the EPA (IRIS).

The WHO (1996) in the drinking-water quality guidelines considers, in relation to genotoxic carcinogens, that a lifetime cancer risk of less than 10^{-5} for consumers is a tolerable risk. Guideline values associated with the ELCRs of 10^{-4} and 10^{-6} are also presented for genotoxic carcinogens to emphasize the fact that each country should select its own appropriate risk level. The calculation of cancer slope factors and unit risk estimates were performed to evaluate the carcinogenic effects.

RESULTS AND DISCUSSION

The concentration and variation of HAAs was monitored in 540 water samples taken from tap water from 20 sites selected in six water and wastewater districts in Tehran during spring, summer, and fall seasons. In addition, human health risk assessments (carcinogenic and non-carcinogenic) were estimated based on the USEPA guidelines for exposure to HAAs from drinking water.

The water quality parameters that can also influence DBP formation (temperature, pH, free residual chlorine) were also measured in three seasons (spring, summer, and fall) and are shown in Table 1. The average concentration of free residual chlorine in the six districts was found to be in the range of 0.5–0.8 mg Cl_2 L^{-1}. As shown in Table 1, pH was in the range of 7 to 8. The average concentrations of HAAs in water samples are shown in Table 2. Average total HAAs varied between 8.67 μg L^{-1} and 58.11 μg L^{-1}. The data presented in Table 2 showed that DCAA and TCAA had the highest values of HAA compounds in all samples. MCAA and MBAA were not detected among the five HAAs in the measured samples. MCAA, MBAA, and DBAA measured lower than the detection limit, which can be explained primarily by the trace amounts of bromide ions in the water sources leading to very low concentrations of brominated DBPs. The results of various studies have shown that DCAA and TCAA

Table 1 | The mean measured tap water quality parameters in different districts

District	Season	Sample size[a]	pH	Temperature[b]	Free residual chlorine[c]
1	Spring	27	7.7	18.83	0.53
	Summer	27	7.7	24.22	0.89
	Fall	27	7.2	13.11	0.87
2	Spring	27	7.8	18.99	0.59
	Summer	27	7.9	24.11	0.71
	Fall	27	7.2	11.50	0.77
3	Spring	18	7.9	19.03	0.63
	Summer	18	8.0	23.92	0.82
	Fall	18	7.5	12.50	0.72
4	Spring	27	8.1	19.18	0.48
	Summer	27	8.1	24.17	0.7
	Fall	27	8.0	13.11	0.78
5	Spring	45	8.0	17.8	0.46
	Summer	45	7.9	23.33	0.75
	Fall	45	7.9	12.63	0.77
6	Spring	36	8.0	18.88	0.54
	Summer	36	7.8	24.25	0.73
	Fall	36	7.9	12.83	0.65

[a]Sample size (*n*).
[b]Temperature (°C).
[c]Free residual chlorine (mg Cl_2 L^{-1}).

are the major HAA species even if there are brominated HAAs in the water (Golfinopoulos *et al.* 2003; Rodriguez *et al.* 2004; Uyak *et al.* 2008; Chang *et al.* 2010; Lou *et al.* 2010; Zhang *et al.* 2010; Ghoochani *et al.* 2013). In all the six districts in spring and fall, the concentration of HAA_5 was higher than in summer. This high concentration is probably due to the greater presence of precursors favoring the formation of HAAs (Ghoochani *et al.* 2013).

Ghoochani *et al.* (2013) reported NOM concentration in raw waters (Karaj, Jajrood, and Lar rivers). NOM content was characterized using two indicators, total organic carbon (TOC) and UV-254. The results showed that their concentrations during spring and fall were significantly higher than during summer. NOM concentrations in different seasons were consistent with concentrations of HAA_5 in different districts.

The trend for seasonal HAA variability was similar to that found in other studies in the literature (Nikolaou *et al.* 2004; Uyak *et al.* 2008; Zhang *et al.* 2010). The levels of HAAs in drinking water varied with different water sources and followed the order of: surface water > groundwater reflecting lower NOM concentrations (Wei *et al.* 2010). The results showed that the HAA concentrations in water samples were much higher in districts 1, 2, and 3, which likely received the highest ratio of surface water with regard to well water, compared with districts 4, 5, and 6, which likely received mostly well water sources.

Table 2 | The average concentration (SD) of HAA compounds ($\mu g L^{-1}$) in water samples

District	Season	Sample size[a]	DCAA	TCAA	Total HAAs
1	Spring	27	33.72 (4.06)	14.38 (0.27)	48.11 (4.10)
	Summer	27	19.82 (2.15)	15.19 (0.42)	35.07 (2.19)
	Fall	27	34.61 (2.01)	17.15 (0.36)	51.80 (2.09)
2	Spring	27	37.56 (3.59)	15.36 (0.31)	52.93 (3.71)
	Summer	27	30.27 (1.46)	15.4 (1.14)	45.67 (2.16)
	Fall	27	39.10 (1.09)	18.98 (1.08)	58.11 (0.67)
3	Spring	18	35.66 (2.59)	15.26 (0.49)	50.96 (2.68)
	Summer	18	22.75 (3.12)	15.08 (0.92)	37.84 (3.51)
	Fall	18	39.12 (1.71)	17.65 (1.10)	56.67 (1.40)
4	Spring	27	10.12 (1.22)	7.01 (1.08)	17.14 (1.97)
	Summer	27	6.20 (1.82)	5.31 (1.15)	11.51 (2.89)
	Fall	27	10.47 (1.11)	8.18 (0.56)	18.74 (0.71)
5	Spring	45	9.30 (0.80)	6.46 (1.17)	15.78 (1.68)
	Summer	45	5.93 (1.41)	4.99 (0.87)	10.92 (2.27)
	Fall	45	10.52 (0.98)	8.47 (0.80)	19.00 (1.09)
6	Spring	36	5.72 (0.64)	3.40 (0.62)	9.13 (0.7)
	Summer	36	4.76 (0.87)	3.90 (0.81)	8.67 (1.14)
	Fall	36	8.08 (1.07)	7.55 (1.48)	15.64 (2.08)

[a]Sample size (*n*).

The one-way analysis of variance was used to determine whether there are any significant differences between the means of HAA concentration in the six water districts. The significance level was given as 0.001.

The correlations between different districts were as follows: sig = 0.14 between districts 1 and 2, sig = 0.89 between 1 and 3, sig = 0.001 between 1 and 4, 5, and 6, confirming that surface water organic matter is the primary driver of HAA concentrations. The water treatment plants are located in districts 1, 2, and 3. In these districts, most of the required water is supplied from surface water with storage reservoirs used when necessary. The estimation of the carcinogenic and non-carcinogenic health risk assessment of HAAs via consumption of tap water is shown in Table 3. The mean concentrations of DCAA in six districts and in spring, summer and fall seasons were measured as 22.01, 14.95, and 23.65 μg L^{-1}, respectively. Also, the mean concentrations of TCAA in spring, summer, and fall seasons were determined as 10.31, 9.98, and 12.99 μg L^{-1}, respectively. Based on the mean concentrations of DCAA and TCAA, the EDI, RfD, HQ, and ELCR were estimated (the calculation has been explained in the section 'Daily intake and health risk assessment'). For the non-carcinogenic risk assessment, the HQ of DCAA and TCAA in the target groups was calculated to be less than 1, which indicates that it is unlikely even for sensitive populations to experience adverse health effects. The HQ values for DCAA in infants, children, teenagers, and adults in spring were 0.523, 0.275, 0.220, and 0.165, respectively. The HQ values for TCAA in infants, children, teenagers, and adults in spring were 0.049, 0.026, 0.021, and 0.015, respectively. The HQ values for DCAA were found to be more than TCAA in all of the target groups and also it was found that the HQ in fall seasons was higher than in other seasons.

The health risk assessment for carcinogenic effect was estimated by the ELCR. The ELCR values were estimated in the range 10^{-5} for DCAA and TCAA, and human health risk assessment for exposure to these was shown to be negligible. The median risk of TCAA was highest among the DBPs considered in research done by Pan *et al.* (2014).

Table 3 | Estimation of carcinogenic and non-carcinogenic health risk assessment of HAAs in the water supply of Tehran

	HAAs					
	DCAA			**TCAA**		
	Spring	**Summer**	**Fall**	**Spring**	**Summer**	**Fall**
Detected mean concentration [μg/L]	**22.01**	**14.95**	**23.65**	**10.31**	**9.98**	**12.99**
EDI[a]						
Infant (<2 yr)	2.091	1.420	2.247	0.979	0.948	1.234
Children (2–6 yr)	1.101	0.748	1.183	0.516	0.499	0.650
Teenagers (6–12 yr)	0.880	0.598	0.946	0.412	0.399	0.520
Adults (≥16 yr)	0.660	0.449	0.710	0.309	0.299	0.390
RfD[b]	**4**	**4**	**4**	**20**	**20**	**20**
HQ[c]						
Infant (<2 yr)	0.523	0.355	0.562	0.049	0.047	0.062
Children (2–6 yr)	0.275	0.187	0.296	0.026	0.025	0.032
Teenagers (6–12 yr)	0.220	0.150	0.237	0.021	0.020	0.026
Adults (≥16 yr)	0.165	0.112	0.177	0.015	0.015	0.019
Drinking water unit risk[d]	$\mathbf{1.4\times10^{-6}}$	$\mathbf{1.4\times10^{-6}}$	$\mathbf{1.4\times10^{-6}}$	$\mathbf{2\times10^{-6}}$	$\mathbf{2\times10^{-6}}$	$\mathbf{2\times10^{-6}}$
ELCR[e]	$\mathbf{3.08\times10^{-5}}$	$\mathbf{2.09\times10^{-5}}$	$\mathbf{3.31\times10^{-5}}$	$\mathbf{2\times10^{-5}}$	$\mathbf{2\times10^{-5}}$	$\mathbf{2\times10^{-5}}$

[a]Estimated daily intake via drinking water (EDI: μg/kg bodyweight/day).
[b]IRIS RfD chronic non-carcinogenic effect risk (RfD, μg/kg bw/day). The TCAA oral RfD is 0.02 mg (kg-day)$^{-1}$ and oral RfD of 0.0004 mg (kg-day)$^{-1}$ for DCAA.
[c]Hazard quotient (HQ): ratio of EDI to RfD.
[d]Drinking water unit risk (μg L^{-1}) (EPA, IRIS).
[e]Excess lifetime cancer risk (ELCR).

CONCLUSION

The HAA concentration in Tehran's drinking water was observed to be less than the MCL established by the EPA (60 μg L^{-1}) and the WHO guideline values for DCAA and TCAA. The concentrations were close to standard values in spring and fall.

HAAs can form especially in districts being supplied by surface water sources. From this approach to human health risk assessment for exposure to HAAs from drinking water in Tehran, the risk appeared to be negligible.

ACKNOWLEDGEMENTS

This research was funded by the Institute for Environmental, Tehran University of Medical Sciences. The authors would like to express their thanks to the Department of Environmental Health Engineering, School of Public Health, Tehran University of Medical Sciences for their collaboration.

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First received 21 March 2016; accepted in revised form 8 November 2016. Available online 8 December 2016