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Human Health Risks from Tap Water Chlorine Disinfection Byproducts: An Ohio Study

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**Human Health Risks from Tap Water
Chlorine Disinfection Byproducts:
An Ohio Study**

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Honors Research Project Report

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ABSTRACT

The use of chlorine in the disinfection process for drinking water has resulted in the creation of disinfection byproducts in the potable water. These carcinogenic disinfection byproducts (DBP's) pose a risk to consumers. Traveling through the water distribution system, stagnation of water in plumbing pipes, and the heating of the water in a hot water tank are all responsible for increasing the concentration of DBP's. In this report, the existing concentrations of trihalomethanes (THM's) and haloacetic acids (HAA's) were gathered from the consumer confidence reports (CCR's) of eight water treatment plants in five Ohio cities. These values were given assumed values of increased concentration due to the additional time to react after exiting the water treatment plant. These values were then used to calculate the chronic daily intake (CDI) for three paths of exposure; ingestion, inhalation, and dermal contact. The results indicate that Akron is the community with the greatest concentration of DBP's and Columbus: Parson's Ave contains the least. The difference in cancer risk between communities utilizing groundwater sources versus surface water sources is significant. The community utilizing a surface water source with the greatest cancer risk is Akron, Ohio with a value of 0.000569 per million. The community utilizing a groundwater source with the greatest cancer risk of DBP's is Cincinnati: Bolton Water, Ohio with a value of 0.000173 per million. The highest cancer risk within the sample from a surface water source is around 3.3 times the risk from a groundwater source. Cutting shower time from ten minutes to five minutes decreased exposure in both the inhalation and dermal contact pathways. Drinking cold water instead of hot may not be worth the sacrifice.

INTRODUCTION AND PURPOSE

Drinking water sources such as lakes, rivers, reservoirs, and ground water aquifers are often full of dangerous pathogens and viruses. Disinfectants such as chlorine, chloramine, and chlorine dioxide are required to inactivate these pathogens. These disinfectants react chemically with naturally occurring materials in the water to form disinfection byproducts. Trihalomethanes (THM's), haloacetic acids (HAA's), chlorite, and bromate are four of these resulting disinfection byproducts [1]. As the water travels away from the water treatment plant and toward the consumer, more time passes for these chemical reactions with the disinfectants to complete. Heating the tap water for bathing/showering or even making a cup of coffee accelerates these chemical reactions, resulting in even higher concentrations of DBP's in hot water [3].

Information about the concentration of contaminants found in drinking water and the community water systems' compliance with EPA regulations can be found in the

consumer confidence reports (CCR's) [2]. Following the drinking water's exit from the water treatment plants, the travel time through the water distribution system (WDS), stagnation in the plumbing pipes (PP) in homes (i.e. overnight or during work hours), and heating within hot water tanks (HWT) all contribute to the increase of concentrations of DBP's in the potable water. Concern has surfaced over human exposure to these compounds due to their possible association with both cancer and non-cancer risks. Routes of chronic exposure from tap water include ingestion (i.e. drinking water, coffee) as well as inhalation and dermal contact (i.e. bathing and showering) [3].

The purpose of this study and report is to examine the risk associated with using tap water for daily activities in major Ohio cities with different water sources (i.e. surface water vs. groundwater). By calculating and comparing the hazard index (HI) and cancer risk (CR), this report displays the level of risk for consumers based on residency. HI is used to determine the potential of non-cancer related human health risks. HI values range from zero to eight with any value less than one considered to have little potential for adverse health effects [4]. Comparisons between groundwater sources and surface water sources are displayed in order for readers of this report to be able to predict which values are similar to the ones in their community based on information from their local CCR. Lastly, this report also shows the effects of changing certain variables, such as decreasing shower temperature or shower time and drinking cold water instead of hot water, in order to give practical methods for consumers to limit their exposure to harmful DBP's.

METHOD

In the following study, only THM's and HAA's were considered. The THM and HAA levels were extracted from the CCR's of five major Ohio cities. These cities are (with different-multiple water plants):

1. Akron
2. Canton
3. Columbus
 - a. Dublin Road
 - b. Hap Cremean
 - c. Parsons Avenue
4. Cleveland
5. Cincinnati
 - a. Miller Water
 - b. Bolton Water

These values provided in the CCR's were measured in a location furthest from the exit of the water treatment plant in the water distribution system. They were then increased by two assumed values to account for the increase of concentration based stagnation in the plumbing pipes and heating in the hot water tank respectively, shown in Table 2. These values were taken from the *Effects of plumbing systems on human exposure to disinfection byproducts in water: a case study* by Shakhawat Chowdhury done in Saudi Arabia. The actual values of increase will differ greatly based on a home's distance from the water treatment plant, the consumer's schedule (i.e. work hours, time spent asleep), a consumer's shower habits, and a myriad of other factors. Therefore, values were borrowed from the case study in Saudi Arabia for demonstration purposes only.

The CCR's give a range of concentrations for THM's and HAA's depending on both seasonal and daily factors; this study used the maximum values for each community in order to examine the worst cases that residents are exposed to. These values can be scaled based on variable dependent on distance from water treatment plant and other factors. Because the water is likely to have the highest concentration of DBP's in the morning after typical off-peak hours, midnight to morning [3], the values taken from the case study in Saudi Arabia were the samples procured in the morning just before the end of off-peak hours (when consumers commonly take showers before work). These high values will likely coincide with the maximum CCR values taken from the major Ohio cities. Chronic daily intake (CDI) was then calculated for three cases: ingestion, inhalation, and dermal contact. The following calculations for CDI are taken from the *Human Health Risk Assessment Protocol* from USEPA released in 1998.

All of the following variables in every equation for each route of exposure will vary greatly from consumer to consumer [3]. The values used in this study were all taken as middle values given from the *Effects of plumbing systems on human exposure to disinfection byproducts in water: a case study* by Shakhawat Chowdhury. Individual risk can be calculated by using particular values for any of the following variables.

Ingestion

The CDI of DBP's through means of ingestion is given by:

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

CDI_{ing} = CDI via ingestion (mg/kg-day)

C_w = concentration of THM HAA in drinking water (µg/L)

IR = drinking water ingestion rate (L/day)

EF = exposure frequency (days/year)

ED = exposure duration (year)

CF = mass conversion factor from µg to mg (0.001)

BW = body weight (kg)
 AT = averaging time (days)

Inhalation

Because HAA's are relatively non-volatile, they do not pose significant risk through the inhalation pathway [3]. Therefore, the CDI of THM's through means of inhalation is given by:

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

CDI_{inh} = CDI via inhalation (mg/kg-day) – THM only
 E_r = absorption efficiency through respiratory system (unitless)
 C_a = THM's in shower air ($\mu\text{g}/\text{m}^3$)
 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)
 CF = mass conversion factor from μg to mg (0.001)
 BW = body weight (kg)
 AT = averaging time (days)

The concentration of THM's in the air during the shower (C_a) depends on the concentration of THM's in the in the shower water, shower enclosure volume, water flow rate, etc. [3] Chowdhury predicted the value by the following:

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

Assuming that the concentration of THM's in the air prior to the shower is zero [3], the above equation converts to:

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

Q_w = water flow (L/min)
 p_v = transfer efficiency of THM's from water to air (unitless)
 C_w = concentration of THM in cold water ($\mu\text{g}/\text{L}$)
 k_a = shower air exchange rate (min^{-1})
 V = shower enclosure volume (m^3)

Dermal Contact

The CDI of DBP's through dermal contact must be analyzed through both the unsteady and steady states. Lag times preceding steady state between the water on the skin and the *stratum corneum* of the skin are not the same between compounds, yielding a range of lag times up to 218.5 minutes [3]. This means that for some compounds, the lag time exceeds the shower length and the absorption never reaches steady state [3]. The CDI through means of dermal contact in the unsteady state condition is given by:

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

$CDI_{derm-ust}$ = CDI via dermal contact – unsteady state (mg/kg-day)

J = diffusion through human skin (mg/cm²-min)

S_{skin} = area of skin exposed to water (m²)

t = shower duration (min/shower)

F = shower frequency (shower/day)

EF = exposure frequency (days/year)

ED = exposure duration (year)

CF = mass conversion factor from µg to mg (0.001)

BW = body weight (kg)

AT = averaging time (days)

The diffusion of the DBP's through human skin (J) requires a number of calculations [3].

$$J = \frac{D_{skin} \times \Delta C_{hw}}{d_{skin}} \times \frac{1mg}{1000\mu g} \times \frac{1L}{1000cm^3} \times \frac{1hr}{60min}$$

D_{skin} = molecular diffusion through *stratum corneum* (cm²/hr)

C_{hw} = DBP's in heated water (µg/L)

d_{skin} = thickness of *stratum corneum* (cm)

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

MW = molecular weight of DBP (g/mol)

k_{ow} = octanol-water partition coefficient (unitless)

k_m = partition coefficient between *stratum corneum and chemical in water* (cm)

$$k_m = 0.64 + 0.25k_{ow}^{0.8}$$

To account for the increase of concentration after heating the water, the following equation is used:

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

C_{hw} = DBP's in heated water ($\mu\text{g/L}$)

C_w = DBP's in cold water ($\mu\text{g/L}$)

k_1 = formation rate for heated water (min^{-1})

k_2 = formation rate for cold water (min^{-1})

Finally, in order to obtain a better prediction for J , duration of dermal exposure during unsteady state was discretized into one minute intervals over the period of an hour [3]. Therefore, the equation of J was then altered to:

$$J_i = \frac{D_{skin} \times \Delta C_w e^{(k_1 - k_2)t_i}}{d_{skin}} \times \frac{1\text{mg}}{1000\mu\text{g}} \times \frac{1\text{L}}{1000\text{cm}^3} \times \frac{1\text{hr}}{60\text{min}}$$

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

Where $i = 1, 2, 3 \dots n$; and the time unit is t/n . After J is calculated, $CDI_{derm-ust}$ can be calculated as well [3]. Following the unsteady state calculations, the lag time per compound is calculated by:

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

This equation yields time in hours. Converting these values to minutes and comparing the length of the lag time to the assumed shower length determines if steady state calculations are required [3]. If $L_t < t$, then the following equation is used:

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

$CDI_{derm-ss}$ = CDI via dermal contact – steady state (mg/kg-day)

C_{hw} = DBP's in warm water ($\mu\text{g/L}$)

S_{skin} = area of skin exposed to water (m^2)

P_d = permeability of DBP's through the skin (m/min)

t_{ss} = difference between shower duration and lag time (min/shower)

F = shower frequency (shower/day)

EF = exposure frequency (days/year)

ED = exposure duration (year)

BW = body weight (kg)
 AT = averaging time (days)

After the CDI's during unsteady state and steady state are calculated, the total is a simple sum [3]:

$$CDI_{derm} = CDI_{dem-ust} + CDI_{derm-ss}$$

Cancer Risk

Following the calculations of the CDI's, the cancer risk (CR) can be determined using the following equation [3]:

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

Hazard Index

Also following the calculations of the CDI's, the hazard index (HI) can be calculated using the following equation:

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

Where $i = 1, 2, 3 \dots m$ represents $CHCl_3$, DCAA, and TCAA respectively and $j = 1 \dots n$ represents different exposure pathways (ingestion, inhalation, and dermal contact) [3].

RESULTS

The THM and HAA concentration values extracted from the CCR's were used to calculate the CDI for ingestion, then inhalation, and lastly dermal contact. The CDI's for each exposure pathway were then utilized to calculate the hazard index and the cancer risk for each community. The hazard index and cancer risk values are useful to make comparisons of health risks (non-cancerous and cancerous respectively) between the given sample of communities.

Table 1: CCR information [1]

PWSID	PLANT	WATER SOURCE TYPE	ORGANIC					
			THM's (ppb)			HAA's (ppb)		
			MIN	MAX	YEAR	MIN	MAX	YEAR
OH7700011	AKRON	SURFACE	17.4	171.0	2014	15.5	84.2	2014
OH7608112	CANTON	GROUND	10.9	42.2	2014	1.1	16.1	2014
OH2504412	COLUMBUS: DUBLIN ROAD	SURFACE	17.3	83.1	2014	23.3	56.5	2014
OH2504412	COLUMBUS: HAP CREMEAN	SURFACE	24.2	89.3	2014	12.6	61.9	2014
OH2504412	COLUMBUS: PARSONS AVENUE	GROUND	12.0	27.1	2014	3.3	5.6	2014
OH1801212	CLEVELAND	SURFACE	8.4	48.3	2014	10.6	43.7	2014
OH3102612	CINCINNATI: MILLER WATER	SURFACE	12.6	53.8	2014	4.8	14.0	2014
OH3102612	CINCINNATI: BOLTON WATER	GROUND	12.6	53.8	2014	4.8	14.0	2014

Table 2: Demonstration values from case study [3]

MORNING STAGNATION EFFECTS					
THM's (ppb)			HAA's (ppb)		
PP	HWT	WDS	PP	HWT	WDS
11.1	14.6	--	8.6	9.2	--

Table 3: Maximum values from Table 1 + values from Table 2

MAXIMUM MORNING CONCENTRATIONS						
THM's (ppb)			HAA's (ppb)			
PP	HWT	WDS	PP	HWT	WDS	
182.1	185.6	171.0	92.8	93.4	84.2	AKRON
53.3	56.8	42.2	24.7	25.3	16.1	CANTON
94.2	97.7	83.1	65.1	65.7	56.5	COLUMBUS: DUBLIN ROAD
100.4	103.9	89.3	70.5	71.1	61.9	COLUMBUS: HAP CREMEAN
38.2	41.7	27.1	14.2	14.8	5.6	COLUMBUS: PARSONS AVENUE
59.4	62.9	48.3	52.3	52.9	43.7	CLEVELAND
64.9	68.4	53.8	22.6	23.2	14.0	CINCINNATI: MILLER WATER
64.9	68.4	53.8	22.6	23.2	14.0	CINCINNATI: BOLTON WATER

Table 4: Equation values from case study [3]

IR =	1.31			MW =	119.4	THM's	Chloroform
EF =	350				128.9	HAA's	DCAA
ED =	77.1				163.4		TCAA
BW =	70.4			k_{ow} =	93	THM's	Chloroform
AT =	28142				8.3	HAA's	DCAA
Q_w =	10				21.4		TCAA
V =	2			k_m =	10.031	THM's	Chloroform
T =	10				1.999	HAA's	DCAA
T_2 =	40				3.539		TCAA
T_1 =	20			D_{skin} =	6.387E-06	THM's	Chloroform
k_a =	0.021				4.485E-06	HAA's	DCAA
E_r =	0.77				4.651E-06		TCAA
p_v =	8.76			L_t =	6.263	THM's	Chloroform
R =	0.014				8.918	HAA's	DCAA
F =	0.74				8.599		TCAA
S_{skin} =	1.82			SF =	0.0061	THM's	Chloroform
P_d =	0.0000267	THM's	Chloroform		0.05	HAA's	DCAA
	0.000000317	HAA's	DCAA/TCAA		0.07		TCAA
d_{skin} =	0.002			$R_f D$ =	0.01	THM's	Chloroform
k_2 =	0.005603				0.004	HAA's	DCAA
k_1 =	0.002483				0.02		TCAA

Table 5: Calculated CDI values for ingestion pathway

CHRONIC DAILY INTAKE BY INGESTION						
THM's (mg/kg-day)			HAA's (mg/kg-day)			
PP	HWT	WDS	PP	HWT	WDS	
0.00325	0.00331	0.00305	0.00166	0.00167	0.00150	AKRON
0.000951	0.00101	0.000753	0.000441	0.000451	0.000287	CANTON
0.00168	0.00174	0.00148	0.00116	0.00117	0.00101	COLUMBUS: DUBLIN ROAD
0.00179	0.00185	0.00159	0.00126	0.00127	0.00110	COLUMBUS: HAP CREMEAN
0.000682	0.000744	0.000484	0.000253	0.000264	9.99E-05	COLUMBUS: PARSONS AVENUE
0.00106	0.001122	0.000862	0.000933	0.000944	0.000780	CLEVELAND
0.00116	0.00122	0.000960	0.000403	0.000414	0.000250	CINCINNATI: MILLER WATER
0.00116	0.00122	0.000960	0.000403	0.000414	0.000250	CINCINNATI: BOLTON WATER

Table 6: Calculated CDI values for inhalation pathway

CDI BY INHALATION			
THM's (mg/kg-day)			
PP	HWT	WDS	
0.0782	0.0797	0.0734	AKRON
0.0229	0.0244	0.0181	CANTON
0.0404	0.0419	0.0357	COLUMBUS: DUBLIN ROAD
0.0431	0.0446	0.0383	COLUMBUS: HAP CREMEAN
0.0164	0.0179	0.0116	COLUMBUS: PARSONS AVENUE
0.0255	0.0270	0.0207	CLEVELAND
0.0279	0.0294	0.0231	CINCINNATI: MILLER WATER
0.0279	0.0294	0.0231	CINCINNATI: BOLTON WATER

Table 7: Calculated values for J

J (mg/cm ² /min)			
CHCl ₃	DCAA	TCAA	
WDS	WDS	WDS	
2.79E-11	4.82E-12	5.00E-12	AKRON
6.88E-12	9.22E-13	9.56E-13	CANTON
1.35E-11	3.23E-12	3.35E-12	COLUMBUS: DUBLIN ROAD
1.46E-11	3.54E-12	3.68E-12	COLUMBUS: HAP CREMEAN
4.42E-12	3.21E-13	3.32E-13	COLUMBUS: PARSONS AVENUE
7.88E-12	2.50E-12	2.59E-12	CLEVELAND
8.77E-12	8.02E-13	8.31E-13	CINCINNATI: MILLER WATER
8.77E-12	8.02E-13	8.31E-13	CINCINNATI: BOLTON WATER

Table 8: Calculated CDI values for dermal contact (all units are mg/kg-day)

CDI _{derm-ust}			CDI _{derm-ss}			CDI _{derm} = CDI _{derm-ust} + CDI _{derm-ss}		
CHCl ₃	DCAA	TCAA	CHCl ₃	DCAA	TCAA	CHCl ₃	DCAA	TCAA
WDS	WDS	WDS	HWT	HWT	HWT	TOTAL	TOTAL	TOTAL
5.11E-08	8.84E-09	9.17E-09	0.000340	2.94E-07	3.80E-07	0.000340	3.03E-07	3.90E-07
1.26E-08	1.69E-09	1.75E-09	0.000104	7.96E-08	1.03E-07	0.000104	8.13E-08	1.05E-07
2.49E-08	5.93E-09	6.15E-09	0.000179	2.07E-07	2.68E-07	0.000179	2.13E-07	2.74E-07
2.67E-08	6.50E-09	6.74E-09	0.000190	2.24E-07	2.90E-07	0.000190	2.30E-07	2.96E-07
8.11E-09	5.88E-10	6.10E-10	7.63E-05	4.65E-08	6.03E-08	7.63E-05	4.71E-08	6.09E-08
1.44E-08	4.59E-09	4.76E-09	0.000115	1.66E-07	2.15E-07	0.000115	1.71E-07	2.20E-07
1.61E-08	1.47E-09	1.52E-09	0.000125	7.30E-08	9.45E-08	0.000125	7.44E-08	9.60E-08
1.61E-08	1.47E-09	1.52E-09	0.000125	7.30E-08	9.45E-08	0.000125	7.44E-08	9.60E-08

Table 9: Calculated individual Hazard Index values for ingestion

HAZARD INDEX FOR INGESTION								
CHCl ₃			DCAA			TCAA		
PP	HWT	WDS	PP	HWT	WDS	PP	HWT	WDS
0.325	0.3312	0.3051	0.207	0.208	0.188	0.0414	0.0417	0.0376
0.095	0.1013	0.0753	0.055	0.056	0.036	0.0110	0.0113	0.0072
0.168	0.1743	0.1483	0.145	0.147	0.126	0.0290	0.0293	0.0252
0.179	0.1854	0.1593	0.157	0.159	0.138	0.0314	0.0317	0.0276
0.068	0.0744	0.0484	0.0317	0.0330	0.0125	0.00633	0.00660	0.00250
0.106	0.1122	0.0862	0.117	0.118	0.0975	0.0233	0.0236	0.0195
0.116	0.1220	0.0960	0.0504	0.0517	0.0312	0.0101	0.0103	0.00625
0.116	0.1220	0.0960	0.0504	0.0517	0.0312	0.0101	0.0103	0.00625

Table 10: Calculated total Hazard Index values for ingestion

INGESTION			
TOTAL HAZARD INDEX (HI)			
PP	HWT	WDS	
0.573	0.581	0.530	AKRON
0.161	0.169	0.118	CANTON
0.342	0.350	0.299	COLUMBUS: DUBLIN ROAD
0.368	0.376	0.325	COLUMBUS: HAP CREMEAN
0.106	0.114	0.0633	COLUMBUS: PARSONS AVENUE
0.246	0.254	0.203	CLEVELAND
0.176	0.184	0.133	CINCINNATI: MILLER WATER
0.176	0.184	0.133	CINCINNATI: BOLTON WATER

Table 11: Calculated Hazard Index values for inhalation

HAZARD INDEX INHALATION			
THM's			
PP	HWT	WDS	
7.817	7.967	7.340	AKRON
2.288	2.438	1.811	CANTON
4.044	4.194	3.567	COLUMBUS: DUBLIN ROAD
4.310	4.460	3.833	COLUMBUS: HAP CREMEAN
1.640	1.790	1.163	COLUMBUS: PARSONS AVENUE
2.550	2.700	2.073	CLEVELAND
2.786	2.936	2.309	CINCINNATI: MILLER WATER
2.786	2.936	2.309	CINCINNATI: BOLTON WATER

Table 12: Calculated Hazard Index values for dermal contact

DERMAL CONTACT				
HI BY THM vs. HAA			TOTAL	
CHCl ₃	DCAA	TCAA		
0.0340	7.56E-05	1.95E-05	0.0341	AKRON
0.0104	2.03E-05	5.24E-06	0.0104	CANTON
0.0179	5.31E-05	1.37E-05	0.0180	COLUMBUS: DUBLIN ROAD
0.0190	5.75E-05	1.48E-05	0.0191	COLUMBUS: HAP CREMEAN
0.00763	1.18E-05	3.04E-06	0.0076	COLUMBUS: PARSONS AVENUE
0.0115	4.27E-05	1.1E-05	0.0116	CLEVELAND
0.0125	1.86E-05	4.8E-06	0.0125	CINCINNATI: MILLER WATER
0.0125	1.86E-05	4.8E-06	0.0125	CINCINNATI: BOLTON WATER

Table 13: Calculated individual Cancer Risk values per million

CANCER RISK									
INGESTION			INHALATION			DERMAL CONTACT			
CHCl ₃	DCAA	TCAA	CHCl ₃	DCAA	TCAA	CHCl ₃	DCAA	TCAA	
1.98E-05	4.14E-05	5.80E-05	0.000448	0	0	2.07E-06	1.51E-08	2.73E-08	AKRON
5.8E-06	1.10E-05	1.54E-05	0.000110	0	0	6.34E-07	4.06E-09	7.33E-09	CANTON
1.03E-05	2.90E-05	4.07E-05	0.000218	0	0	1.09E-06	1.06E-08	1.92E-08	COLUMBUS: DR
1.09E-05	3.14E-05	4.40E-05	0.000234	0	0	1.16E-06	1.15E-08	2.07E-08	COLUMBUS: HC
4.16E-06	6.33E-06	8.87E-06	7.10E-05	0	0	4.66E-07	2.36E-09	4.26E-09	COLUMBUS: PA
6.47E-06	2.33E-05	3.27E-05	0.000126	0	0	7.02E-07	8.55E-09	1.54E-08	CLEVELAND
7.06E-06	1.01E-05	1.41E-05	0.000141	0	0	7.64E-07	3.72E-09	6.72E-09	CINCINNATI: MW
7.06E-06	1.01E-05	1.41E-05	0.000141	0	0	7.64E-07	3.72E-09	6.72E-09	CINCINNATI: BW

Table 14: Calculated total Cancer Risk values per million

CANCER RISK				
ING.	INH.	D.C.	TOTAL	
1.19E-04	0.000448	2.11E-06	5.69E-04	AKRON
3.22E-05	0.00011	6.46E-07	1.43E-04	CANTON
7.99E-05	0.000218	1.12E-06	2.99E-04	COLUMBUS: DUBLIN ROAD
8.64E-05	0.000234	1.19E-06	3.21E-04	COLUMBUS: HAP CREMEAN
1.94E-05	7.1E-05	4.72E-07	9.08E-05	COLUMBUS: PARSONS AVENUE
6.25E-05	0.000126	7.26E-07	1.90E-04	CLEVELAND
3.13E-05	0.000141	7.74E-07	1.73E-04	CINCINNATI: MILLER WATER
3.13E-05	0.000141	7.74E-07	1.73E-04	CINCINNATI: BOLTON WATER

DISCUSSION

The horizontal axis values by community were assigned as follows:

1. Akron
2. Canton
3. Columbus: Dublin Road
4. Columbus: Hap Cremean
5. Columbus: Parsons Avenue
6. Cleveland
7. Cincinnati: Miller Water
8. Cincinnati: Bolton Water

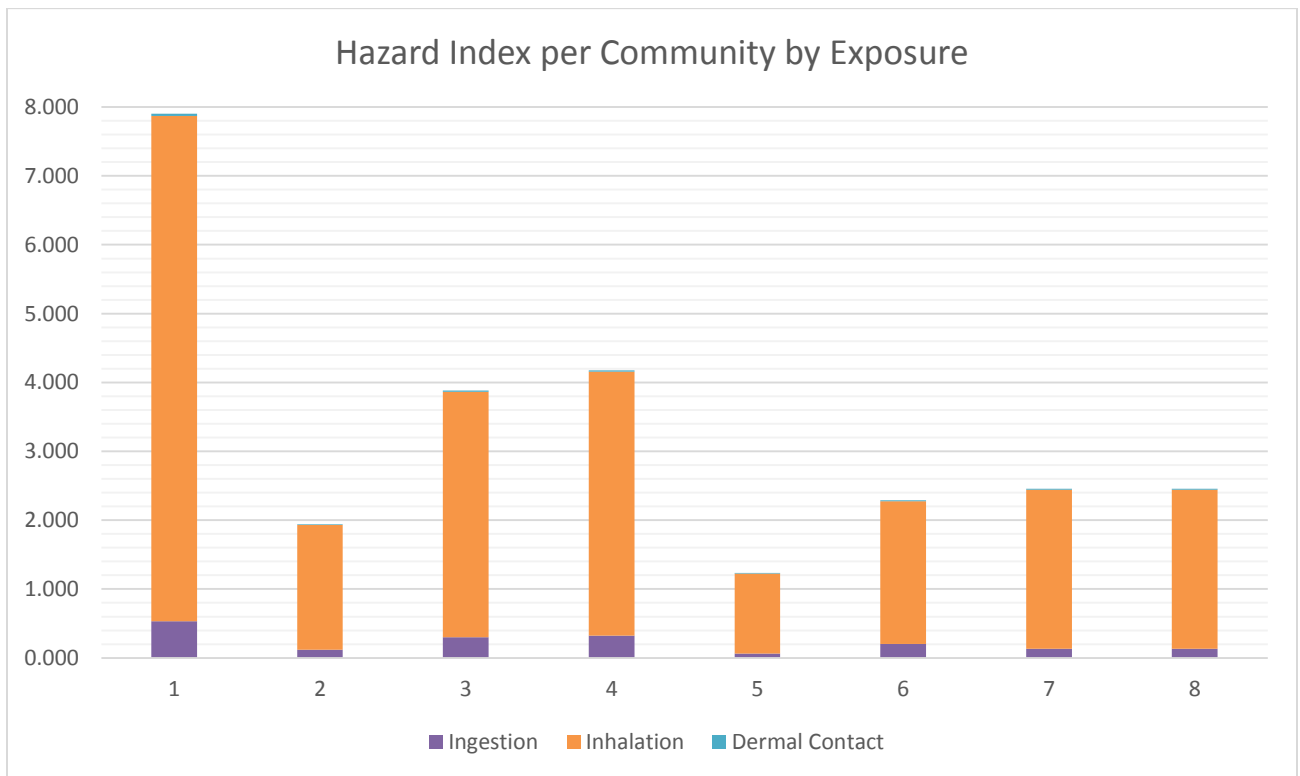


Figure 1: Hazard index per community by exposure

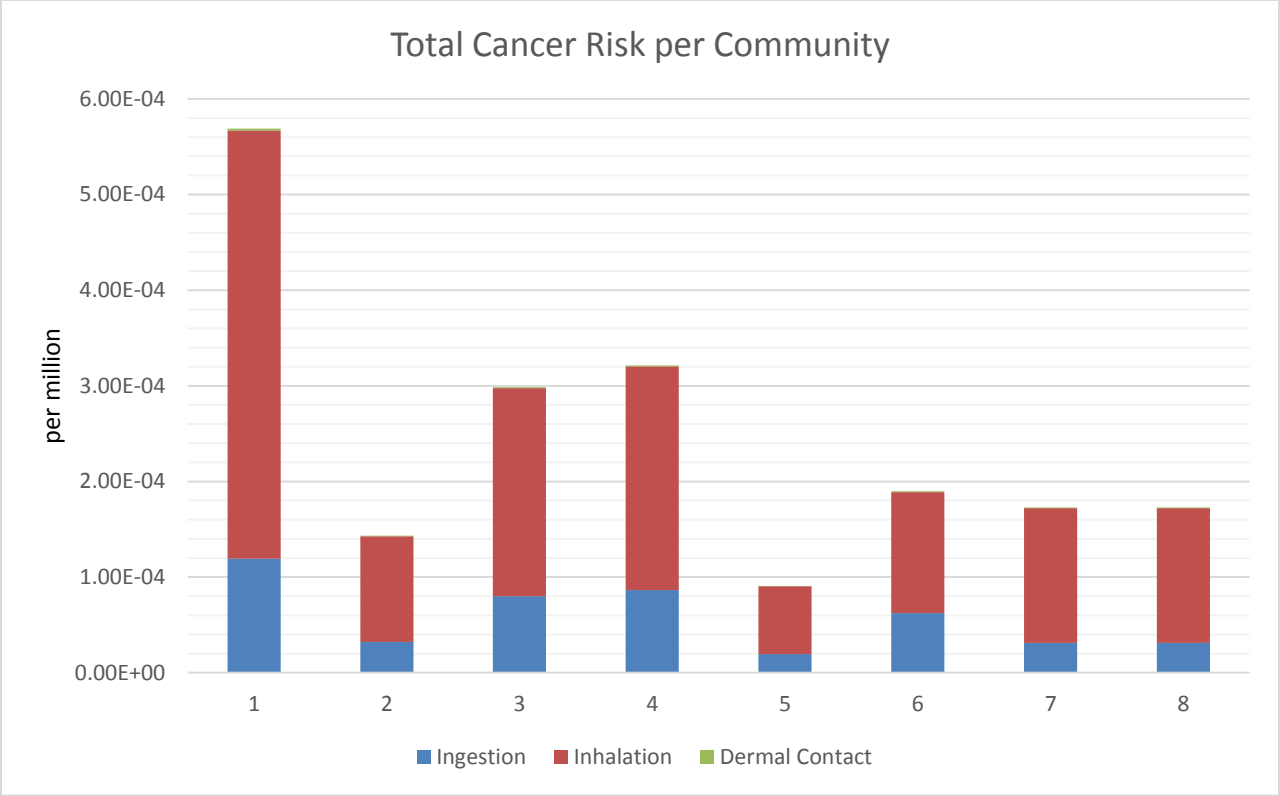


Figure 2: Total Cancer Risk per million by community

Because the concentrations of THM’s and HAA’s are the greatest in the community of Akron, Ohio, residents there are at the greatest risk within this set of community water systems (CWS’s). On the other side of the spectrum, the community members in the Parsons Avenue community in Columbus, Ohio are under the least risk. The major difference between these two extremes stems from their respective drinking water sources; Akron has a surface water source while Columbus: Parsons Avenue has a ground water source. Surface water is exposed to the elements and wildlife use and is therefore likely to contain more contaminants and pathogens requiring treatment. Because groundwater is naturally filtered through the ground water table and is also protected from the elements and wildlife, it typically requires less treatment. This results in drinking water containing fewer disinfection byproducts.

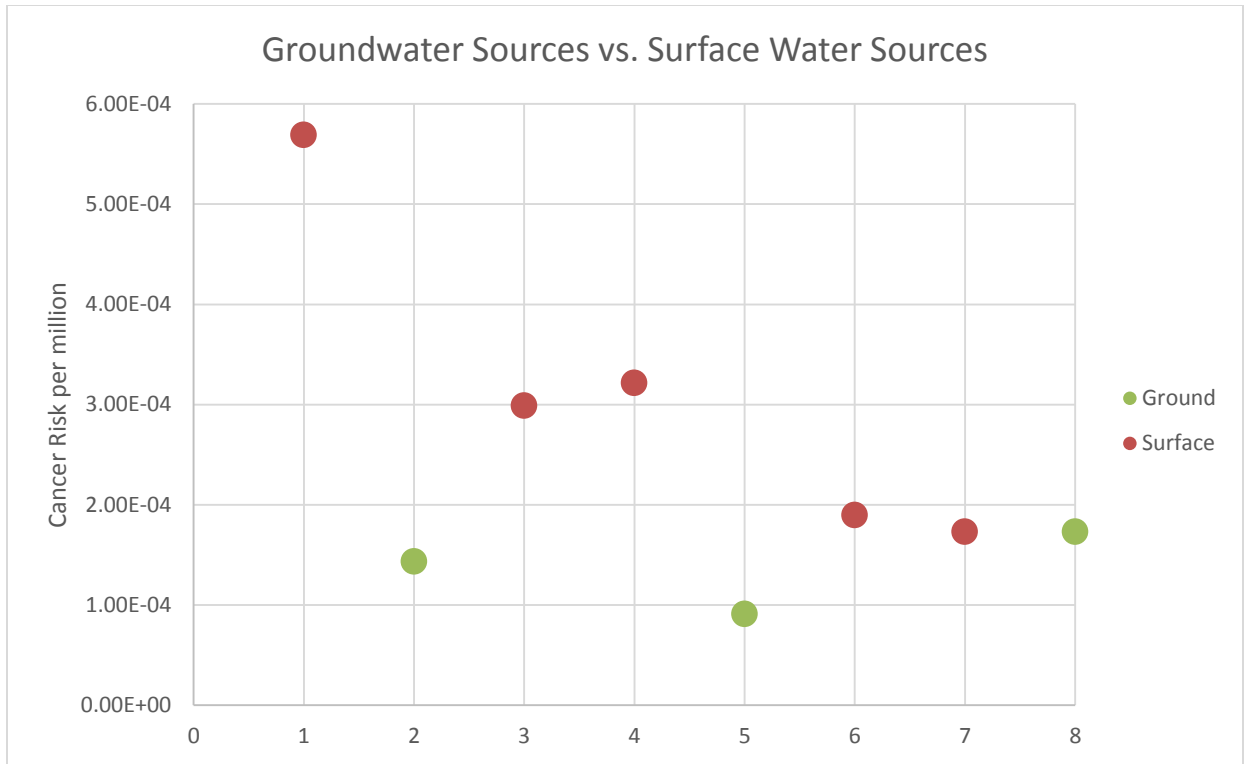


Figure 3: Groundwater vs. surface water cancer risk comparison

According to Figure 1, inhalation poses the greatest risk between the three pathways. Therefore, an effective way to reduce risk of exposure to carcinogens is to reduce shower time. The shower time for the previous calculations was assumed to be ten minutes. If time were to be reduced by five minutes (by showering more quickly, turning the water off when washing hair, etc.), the shower time would be less than the lag time for all three compounds investigated in this report. This means absorption of the DBP's through the skin would never reach steady state. This therefore not only reduces exposure by inhalation, but also by dermal contact.

Table 15: Hazard Index for five-minute showers

HAZARD INDEX INHALATION			DERMAL CONTACT				
THM's			HI BY THM vs. HAA			TOTAL	
PP	HWT	WDS	CHCl ₃	DCAA	TCAA		
2.057	2.096	1.931	2.56E-06	1.11E-06	2.29E-07	3.89E-06	AKRON
0.602	0.642	0.477	6.31E-07	2.11E-07	4.38E-08	8.86E-07	CANTON
1.064	1.103	0.939	1.24E-06	7.42E-07	1.54E-07	2.14E-06	COLUMBUS: DR
1.134	1.173	1.009	1.34E-06	8.13E-07	1.69E-07	2.32E-06	COLUMBUS: HC
0.431	0.471	0.306	4.05E-07	7.35E-08	1.52E-08	4.94E-07	COLUMBUS: PA
0.671	0.710	0.546	7.22E-07	5.74E-07	1.19E-07	1.42E-06	CLEVELAND
0.733	0.773	0.608	8.05E-07	1.84E-07	3.81E-08	1.03E-06	CINCINNATI: MW
0.733	0.773	0.608	8.05E-07	1.84E-07	3.81E-08	1.03E-06	CINCINNATI: BW

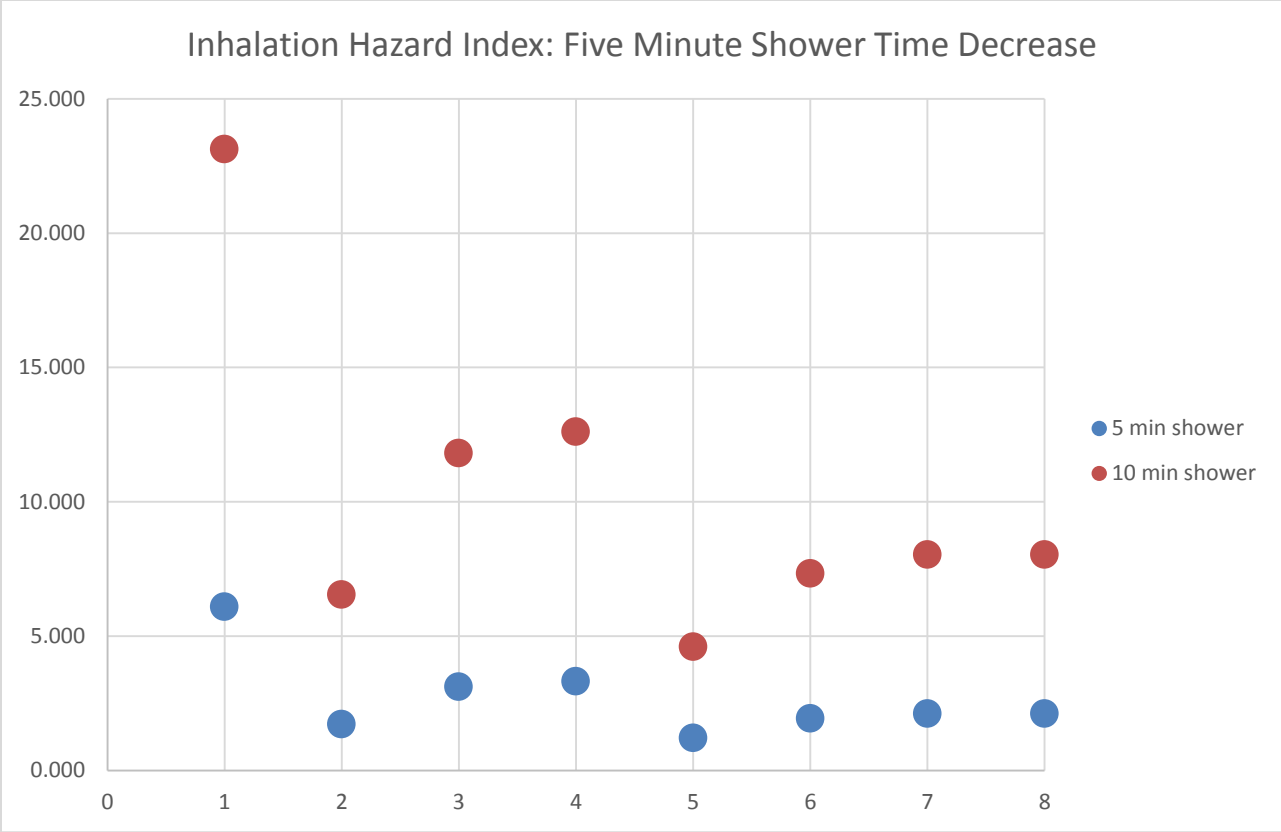


Figure 4: Inhalation Hazard Index decrease due to shower time decrease

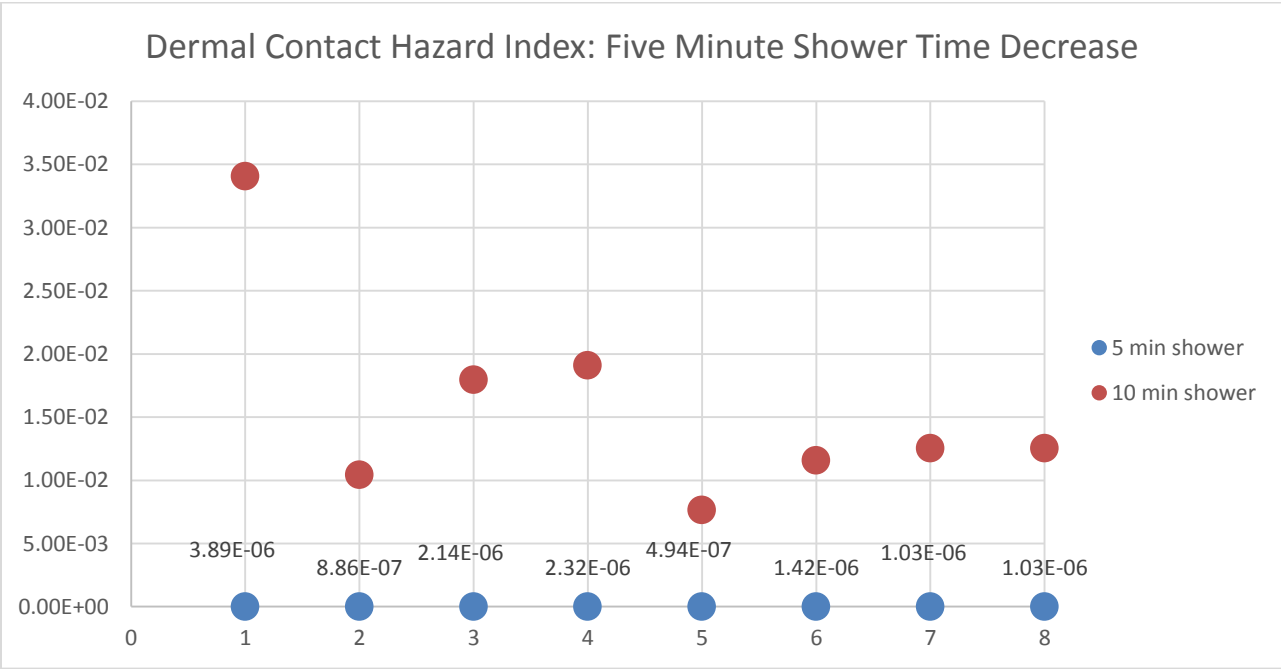


Figure 5: Dermal Contact Hazard Index decrease due to shower time decrease

It is important to note that these dramatic decreases in dermal contact risk are due to the shower time falling below lag time of skin absorption for these compounds. The lag time is 6.3 minutes for chloroform, 8.9 minutes for DCAA, and 8.6 minutes for TCAA (Table 4). These values all fall below ten minutes and above five minutes, causing a drastic decrease of dermal contact risk when decreasing the shower length by this time. These results are not always the case when decreasing the shower time by five minutes, such as from fifteen minutes to ten minutes. However, the inhalation risk still decreases significantly with any cut in shower length because there is no lag time associated with inhalation exposure.

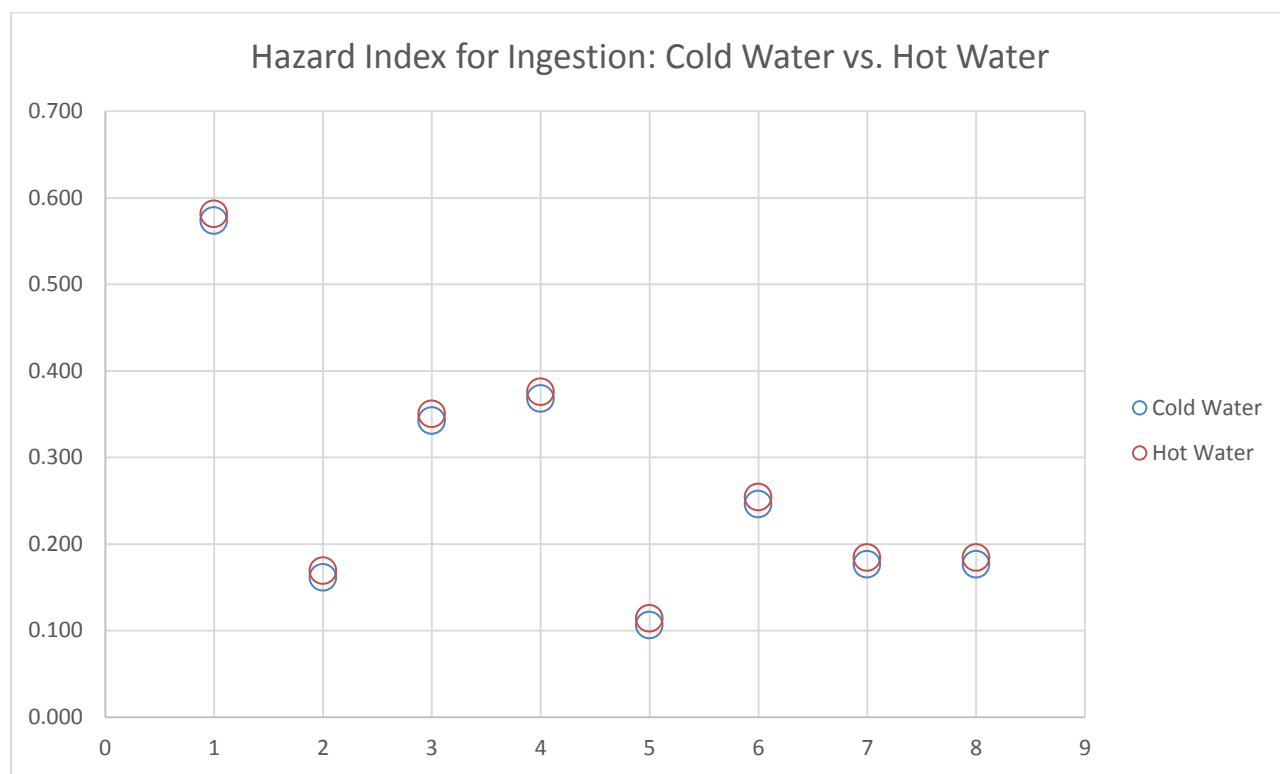


Figure 6: Hazard Index for Ingestion: cold water vs. hot water

The last comparison made is between the consumption of cold water versus hot water. In the morning, the time of day that these concentrations pertain to, many Americans drink coffee or tea in order to help wake themselves up. There are cold drinks that also contain caffeine that could replace these hot drinks. Figure 6 examines whether it is worth replacing these hot caffeinated drinks with cold ones.

CONCLUSIONS

According to Figure 1, inhalation is the biggest concern for non-cancerous human health effects. The HI's for all communities exceeded the benchmark value of 1. According to

Figure 2, inhalation poses the greatest cancer risk. Although all three exposure pathways are displayed on both figures, dermal contact does not contribute to the visible portion.

The first way for residents to reduce exposure to DBP's is achievable when an individual is shopping for a new home. People can research their prospective community water systems in order to find out the levels of disinfection byproducts. Home shoppers can keep in mind that groundwater sources typically require less treatment and therefore have lower levels of disinfection byproducts in the drinking water. According to Figure 3, the difference of cancer risk between communities utilizing groundwater sources versus surface water sources is significant.

According to Figure 4, cutting a ten-minute shower in half will decrease the hazard index for inhalation by significantly more than half. This means that decreasing shower time does not linearly decrease risk; cutting shower time by half can disproportionately benefit a consumer. A ten-minute shower exceeds the lag time for Chloroform, DCAA, and TCAA. This length of time allows the absorption of these compounds through the skin to reach steady state. Reducing shower time to five minutes prevents absorption rates from reaching steady state. Although the overall risk from dermal contact is not very high even in a ten-minute shower, decreasing the shower time to five minutes causes the dermal contact pathway hazard index to be approximately zero (Figure 5).

Lastly, according to Figure 6, drinking cold water beverages do not cause a significant decrease in the Hazard Index. Because many Americans enjoy their hot beverages, giving them up would likely require a large amount of self-control and personal sacrifice. Therefore, it may not be conducive for consumers give up their pleasurable cup of hot coffee or tea in the morning in order to lower risk from DBP's because the benefits may not outweigh the sacrifice.

SUMMARY OF CONCLUSIONS

- HI exceeds the benchmark value of 1 for all sampled communities
- Inhalation is responsible for the majority of both hazard index and cancer risk values
- In the sample provided, surface water sources made up the top contributors to human health risks over groundwater sources
- Taking a five-minute shower rather than a ten-minute shower decreases the hazard index by more than half
- Replacing hot water drinks with cold water drinks causes an insignificant decrease in risk

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