Prediction of human exposure and health risk assessment to trihalomethanes in indoor swimming pools and risk reduction strategy

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ABSTRACT
Exposure to disinfection by-products can occur through various pathways such as inhalation, dermal contact, and ingestion for swimmers. In the present study, swimming exposure to disinfection by-products through ingestion and dermal route at the indoor swimming pool was assessed and health risks caused by exposure to these compounds were studied. Water samples were first collected from Eslamshahr (Tehran, Iran) and water quality parameters were analyzed. Then, a multi-pathway model was conducted to assess and estimate the chronic daily intakes (CDI) and lifetime cancer risk (LTCR). The results showed that the mean values of CDIs for chloroform, DCBM, DBCM, and bromoform were $2.12 \times 10^{-6}$, $7.35 \times 10^{-7}$, $8.12 \times 10^{-9}$, and $1.28 \times 10^{-8}$ mg/kg-d, respectively through ingestion pathways, and $3.95 \times 10^{-5}$, $1.56 \times 10^{-6}$, $1.88 \times 10^{-8}$, and $3.08 \times 10^{-8}$ mg/kg-d through the dermal pathway, respectively. Also our findings showed that the mean of total LTCR for swimmers was $4.63 \times 10^{-8}$ and the cancer risks through dermal route were higher than ingestion route. Also, cancer risks of chlorinated compounds were higher to implement a set of strategies in order to improve water treatment of swimming pools.

KEYWORDS disinfection by-products; exposure assessment; multi-pathway risk assessment; indoor swimming pool; trihalomethanes

Introduction
Many studies show that swimming is considered as a widely enjoyed sport for leisure as well as exercise. It is beneficial for the human health of all ages and physical conditions and has some advantages over other physical activities. Various disinfectants including, chlorine, chloramine, chlorine dioxide, and ozone are used in order to inactivate microbial pathogens and prevent waterborne diseases in swimming pools (Chowdhury et al. 2014). Due to its

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ability to meet the demands, such as cost-effectiveness, ease of application, and ability to inactivate a wide variety of pathogenic microorganisms, chlorination is the predominant disinfection method applied in water treatment of swimming pools (Afifi and Blatchley, 2015).

It has been suggested that chlorination lead to the formation of disinfection by-products (DBPs) which occurs mostly via reactions between free chlorine and inputs from bathers through sweat, urine, skin particles, and personal care products as well as natural organic matter in source water (Judd and Black, 2000, Kristensen et al., 2010). Many personal care products have been detected in swimming pools which include parabens, ultraviolet (UV) filters found in personal care products (PCPs) such as lotions and sunscreens and more recently, N, N-diethyl-meta-toluamide (DEET), caffeine and tris(2-carboxyethyl) phosphine (TCEP) (Alcudia-León et al., 2013, Vidal et al., 2010, Weng et al., 2014). Moreover, many studies show the possibility of different reactions between these chemical compounds with the disinfectants, which may transform them into by-products more harmful than the unchanged PCPs (Bottoni et al., 2014). According to WHO guidelines, the tolerable concentration of free chlorine for public/semi-public swimming pools and hot tubes is 3 mg/L and 5 mg/L with maintained 7.2–7.8 pH levels, respectively (WHO, 2006). However, there are other variable practices available for the disinfection of swimming pools. Primary studies reported DBPs in drinking water and subsequently many studies have been carried out to investigate occurrence and toxicity of DBPs in drinking water (Rook, 1974, Richardson et al., 2007). Today, more than 600 DBPs that are mostly mutagenic and carcinogenic have been detected in drinking water (Richardson et al., 2007, Zhai et al., 2014, Zhang et al., 2008). Several aromatic halogenated DBPs also recently have been recognized in drinking water, which have been demonstrated with more toxic properties compared with the previous aliphatic halogenated DBPs (Zhai et al., 2014, Zhang et al., 2008, Yang and Zhang, 2013, Liu and Zhang, 2014). The presence of trihalomethanes (THMs) in swimming pool water has been reported for the first time in 1980 (Weil et al., 1980, Beech et al., 1980). Later in different countries all over the world, various studies have been conducted to determine different categories of DBPs in swimming pool water (Chowdhury et al., 2014). Various factors including higher free residual chlorine (FRC), higher temperature, organic precursors, constant organic loads, exposure routes, contact of water surface with air as well as water recirculation could affect DBPs formation in swimming pool compared with drinking water DBPs (Hang et al., 2016). Many studies show that DBPs have carcinogenic and non-carcinogenic properties (USEPA, 2018). It has been demonstrated that the brominated and nitrogenous DBPs may have considerable cytotoxicity and genotoxicity compared with the chlorinated DBPs (Hang et al., 2016, USEPA, 2018). Also, several studies have reported that low molecular- weight (MW) (<200 g/mole) DBPs are causative for most of the genotoxicity and the pool-water-induced DNA damage in Hep-G2 cells (comet assay) (Glauner et al., 2005). The major categories of the DBPs that were identified in swimming pools include THMs, iodo- THMs, haloacetates, haloacetic acids, haloacids, halodiacids, haloaldehydes, halonitrites, haloketones, halopyrroles, halomethanes, halonitromethanes, bromate, haloamides, haloalcohols, nitrosamines, combined chlorine, halofuranones, halophenols, and halobenzoquinones (HBQs) (Richardson and Postigo, 2011, Xiao et al., 2012, Wang et al., 2013). Air and water qualities are both relevant issues with high importance to human health that must be considered in indoor chlorinated swimming pool facilities. Eleven volatile DBPs have been identified by Li and Blatchley that are formed in chlorinated swimming pools including three inorganic chloramines, four THMs, cyanogen chloride, cyanogen bromide,
dichloroacetonitrile, and dichloromethylamine (Li and Blatchley, 2007, Bernard et al., 2007, Bernard et al., 2003). There have been a few epidemiological studies that were performed specifically to determine exposure and adverse health effects of THMs in indoor swimming pools (Villanueva and Font-Ribera, 2012, Florentin et al., 2011). Different adverse effects associated to THMs ingestion have been determined such as bladder cancer, colon cancer, adverse outcomes on respiratory function, asthma as well as reproductive function (Hamidin et al., 2008, Villanueva et al., 2007, Villanueva et al., 2006, Nickmilder and Bernard, 2007, Aggazzotti et al., 2004, Nieuwenhuijsen, 2005). It has been shown that inhalation, dermal absorption, and ingestion are possible routes of exposure to the volatile DBPs such as THM. However, skin exposure as well as gastrointestinal exposure while swimming is the main intake route of THM that mainly pose cancer risk in comparison to other exposure routes (Panyakapo et al., 2008). The four THMs have been included as human carcinogens by USEPA of which CHCl₃, CHCl₂Br, and CHBr₃ are reported carcinogen type B2 (human carcinogen) and CHClBr₂ with probable human carcinogenicity as carcinogen type C. Various hazards could be associated with these compounds such as abortion or teratogenicity in babies, and children with asthma from inhaling THMs vapor through the respiratory tract (WHO, 2000a) (Environmental Protection Agency, 1999, Amy et al., 2000). Because of this, the indicative concentration of THM in the pool is considered to be the same as THM legal limits in drinking water. 80 µg/L for total THM (sum of the four THM) has been recommended as the guideline value by the US Environmental Protection Agency (USEPA 2003). Also, guideline values for each THM in drinking water have been set by the World Health Organization (WHO, 2008), including 300 µg/L for CF, 60 µg/L for BDCM, and 100 µg/L for DBCM as well as BF. Moreover, it has been indicated by the WHO that the sum of the ratio of each THM concentration with its respective guideline value should not exceed 1. Although, some countries in Europe such as Denmark, Germany, and France have set a maximum value (MV) for total trihalomethanes (TTHMs) in swimming pools water including 50 µg/L, 20 µg/L, and 100 µg/L, respectively (Bisted, 2002, Peng et al., 2016, Wasserweisen, 1997). Many studies were conducted to investigate THMs and Haloacetic acids (HAAs) values in swimming pools or drinking water worldwide (Hang et al., 2016, Dyck et al., 2011, Lee et al., 2009, Lourencetti et al., 2012, Righi et al., 2014, Chowdhury, 2015, Silva et al., 2012, Tardif et al., 2016). However, there are only a few studies conducted in Iran addressed to examine THMs and HAAs in drinking water and swimming pools (Heydari et al., 2013, Fooladvand et al., 2011, Hassani et al., 2010). Because of the health-related issues associated with DBPs, it is of considerable importance to investigate the occurrence of DBPs in swimming pool. DBPs in water and air of swimming pools have been shown to pose adverse effects on human health. The main objective of this study was to investigate the concentrations of THMs as one of the categories of DBPs, in public indoor swimming pool water in Eslamshahr, Tehran, Iran, to evaluate the correlations between this DBP and water quality parameters and to predict the health risks of the swimming pool DBPs to human.

**Methodology**

**Sampling**

Occurrences of THMs were investigated before swimming and after swimming in public indoor swimming pool water in Eslamshahr, Tehran, Iran. Subjective swimming pools are
fed with groundwater and disinfection has been carried out using chlorine and ozone. Triplicates of water samples were collected in glass bottles of 500 mL twice a day at 8:00 am (at the beginning of swimming) and 11:00 pm (at the end of swimming) for during a week in the winter of 2017. The average value of three samples was considered in this study. Water samples in three different locations around the pool including the shallow end, the middle of the pool, and the deep end were collected in headspace free bottles. Also a distance of 1 m from the side walls and approximately 30 cm in below the water surface has been considered in sample collection. However, only samples of the middle of the pools were collected for physicochemical analysis in which pH, temperature, free residual chlorine, and total chlorine parameters have been included. Also 100 mg of sodium thiosulfate was used to eliminate FRC. The samples were stored in at 2 ± 0.1°C and analyzed within 12 h of collection.

**Analysis**

Temperature and pH were measured in situ; pH (Spectroquant® Picco), temperature (alcohol thermometer), and, according to the 4500-Cl-F method, free residual chlorine (HACH DR 890-MTH 8021) and total chlorine (HACH DR 890-MTH 8167) conductivity measured by Cond 330i and turbidity measured by HACH 2100AN Turbidity meter (Mirzabeygi et al., 2017, Radfard et al., 2018, Soleimani et al., 2018, Yousefi et al., 2018, Yousefi et al., 2018, Asghari et al., 2018, Abbasnia et al., 2018, Takdastan et al., 2018). THMs (chloroform [CHCl₃], bromodichloromethane [BDCM], dibromochloromethane [DBCM], and bromoform [CHBr₃]) in the water were measured by GC-MS by following USEPA method 551.1. THMs were determined through an automatic thermal desorption unit coupled to a GC-FID.

Concentrations of the four forms of THMs were analyzed by VARIAN CP-3800 gas chromatograph and FID detector, Head-space CombiPal Autosampler with VRIAN CP SIL 8CB 30 M × 0.32 mm × 0.25 μm, column, He as carrier gas with a flow rate of 2.9 ml/min, injection temperature at 280°C, GC cycle time 15 min, oven temperature of 35°C, hold for 1 min, then to 100°C with rate of 9°C/min then to 140°C rating 6°C/min, and detector temperature of 250°C. The detection limits of four THMs were 5 ppb.

**Statistical analysis**

To present data, we used mean, standard deviation, median, and range. To assess the normal distribution of data we used Shapiro-Wilk test.

To obtain the correlation of scale variables we used Spearman correlation coefficient. All statistical analyses were performed by SPSS 24. P-values less than 0.05 were considered statistically significant.

**Monte Carlo simulation**

Monte Carlo simulation method was applied for the variation and sensitivity analysis of the forecast of the risk assessment model. In this study, considering the inherited natural variance, equation variables were defined in the terms of a probability density function taken from a little number of measurements.
In order to analyze the data, the distribution of parameters was simulated using software program Crystal Ball® (Version 11.1.2.3, Decisioneering, Inc., Denver, CO, USA). The statistical criterion was the basis for choosing the type of distribution.

The obvious justification for this uncertainty/variability and its impact on the estimation of the risk of cancer and hazard index was derived from a Monte Carlo simulation. Independent runs at 1, 4, 5, and 10 thousand replicates were performed to test convergence and numerical output stability. It should be noted that the sampling of each parameter, independently was performed from the appropriate distribution, at the beginning of each repetition.

According to the result of this research, 10000 repetitions are enough to ensure the consistency of results. The result of Monte Carlo simulation provides a confidence interval of health risk for swimmers exposed to chloroform in indoor swimming pools.

**Exposure and risk assessment**

The main exposure pathways of THMs in a swimming pool are dermal contact and ingestion. Time of swimming, body surface area, and ingestion rate of the swimming pool water determine the various exposure of the different age groups. The main important exposure factors and their amounts that are commonly used in the conventional equations are listed in Table 1.

**Ingestion pathway**

There are many data in the literature for accidental ingestion rate of water and duration per event during swimming that are shown in Table 2. In this study, the number of swimmers, duration of swimming, and swimming frequency were monitored. The number of swimmers, duration in the pool, and swimming frequency were found to be in the ranges of 14–62 persons per day, 40–85 min/event, and 26–48 times/year, respectively.

**Table 1.** Parameters and their values for this study.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Symbol</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>DBPs in swimming pool water (μg/L)</td>
<td>( C_w )</td>
<td>This study</td>
</tr>
<tr>
<td>Water ingestion rate (mL/h)</td>
<td>IR</td>
<td>18, 25.6, 34</td>
</tr>
<tr>
<td>Duration of swimming event (h/event)</td>
<td>T</td>
<td>0.67, 0.97, 1.42</td>
</tr>
<tr>
<td>Swimming frequency (events/year)</td>
<td>F</td>
<td>26, 36.3, 48</td>
</tr>
<tr>
<td>Exposure duration (year)</td>
<td>ED</td>
<td>20, 30, 40</td>
</tr>
<tr>
<td>Body weight (kg)</td>
<td>BW</td>
<td>62, 70.4, 81</td>
</tr>
<tr>
<td>Averaging time (day)</td>
<td>AT</td>
<td>23,725; 28,142; 30,186</td>
</tr>
<tr>
<td>Area of body skin exposed to water (m²)</td>
<td>Sskin</td>
<td>1.69, 1.82, 1.94</td>
</tr>
<tr>
<td>Permeability of DBPs (m/min)</td>
<td>( P_D )</td>
<td>(2.54, 2.67, 2.79) × 10⁻⁵</td>
</tr>
<tr>
<td>CHCl₃</td>
<td></td>
<td>(2.87, 3.0, 3.13) × 10⁻⁵</td>
</tr>
<tr>
<td>BDCM</td>
<td></td>
<td>(3.25, 3.33, 3.42) × 10⁻⁵</td>
</tr>
<tr>
<td>CHBr₃</td>
<td></td>
<td>(3.42, 3.53, 3.58) × 10⁻⁵</td>
</tr>
<tr>
<td>Thickness of stratum corneum (cm)</td>
<td>( d_{skin} )</td>
<td>0.0015, 0.002, 0.003</td>
</tr>
<tr>
<td>Molecular weight (g)</td>
<td>MW</td>
<td>CHCl₃ 119.4; BDCM 163.8; DBCM 208.3; CHBr₃ 252.8</td>
</tr>
<tr>
<td>Octanol-water partition coefficient</td>
<td>( K_{ow} )</td>
<td>CHCl₃ 93; BDCM 126; BDCM</td>
</tr>
</tbody>
</table>
In order to assess the risk of THMs through ingestion route, the chronic daily intake (CDI) of DBPs is computed through following equation. (USEPA 1998)

\[
CDI_{\text{ing}} = \frac{C_w \times IR \times T \times F \times ED \times CF}{BW \times AT}
\]

where CDI_{\text{ing}} is chronic daily intake via ingestion (mg/kg-d), \(C_w\) refers to the concentration of DBPs in pool water (µg/L), IR represents the accidental intake of water (mL/h), T is duration of swimming (hour/event), F, ED and BW refer to the swimming frequency (events/year), exposure duration (year) and body weight (kg), respectively. AT and CF are the average time (days) and conversion factor from microgram to milligram; and milliliter to liter (10^{-6}).

**Dermal contact pathway**

The received dose THMs through dermal exposure in the swimming pool water was computed according to the fugacity model. 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, respectively. According to the studies on the assessment of non-occupational exposure to nonionizing chemicals, chemicals with MW less than 700 g/mole and \(K_{ow}\) between 0.1 and 105 may be absorbed significantly through human skin. Thus, it is conceivable that THMs may be significantly absorbed through dermal contact during swimming, which may pose a high risk to human health (Chowdhury et al., 2014).

It is worth noting that the CDI for dermal contact must be evaluated in both the unsteady and steady states of exposures. In addition, the lag times in the steady state between the water contacted to the human skin and the stratum carenum of skin should be considered in order to assess the risk of DBPs that could take into account between 7.5 and 218.3 min (Chowdhury, 2013). Also, the swimming durations is often less than the lag times (Lee et al., 2009). In addition, there is a difference between the unsteady-state and steady-state for estimation of DBP exposure through dermal contact (Chowdhury, 2013). The order of

<table>
<thead>
<tr>
<th>Study</th>
<th>Type</th>
<th>Value (mL/h)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Accidental ingestion rate</td>
<td>Average</td>
<td>25</td>
</tr>
<tr>
<td>American Chemistry Council (ACC)</td>
<td>Men</td>
<td>27–34</td>
</tr>
<tr>
<td>Schets et al. (2011)</td>
<td>Woman</td>
<td>18–23</td>
</tr>
<tr>
<td></td>
<td>Children</td>
<td>31–51</td>
</tr>
<tr>
<td>Evans et al. (2006)</td>
<td>Average</td>
<td>26.5 mL/person/event</td>
</tr>
<tr>
<td></td>
<td>Children</td>
<td>37 mL/person/event</td>
</tr>
<tr>
<td></td>
<td>Adults</td>
<td>16 mL/person/event</td>
</tr>
<tr>
<td>Lee et al. (2009)</td>
<td>Average</td>
<td>25</td>
</tr>
<tr>
<td>This study</td>
<td>Average</td>
<td>18–34</td>
</tr>
<tr>
<td>Duration per event</td>
<td>Schets et al. 2011</td>
<td>41–79 min</td>
</tr>
<tr>
<td></td>
<td>Maximum</td>
<td>67–81 min</td>
</tr>
<tr>
<td>EPA</td>
<td>Average</td>
<td>115 min</td>
</tr>
<tr>
<td>Lee et al. (2009)</td>
<td>Average</td>
<td>78 min</td>
</tr>
</tbody>
</table>
distribution of CDI in the steady-state for low and high-MW chemicals via stratum carennum differs from $10^{-13}$ to $10^{-14}$ and $10^{-15}$ to $10^{-17}$ m$^2$/s, respectively (Chowdhury et al., 2014). However, distribution of compounds before attaining the steady state could be critically varied (Chowdhury, 2013). The unsteady- and steady-state assessments should be carried out when swimming durations are more than the lag times. The dermal contacts with DBPs through swimming were assessed through the following equation (Chowdhury et al., 2014, USEPA, 2018).

$$AD = \frac{C_w \times SA \times PC \times EC \times EF \times ED \times CF}{BW \times AT}$$

where AD is absorbed dose (mg/(kg_d)); CF refers to the volumetric conversion factor for water (1 L/1000 cm$^3$); EF and ET represent the exposure frequency (d/a, events/year) and exposure time (h/d, h/event); and IR, PC, and SA are the intake rate (L/d) chemical-specific dermal permeability constant (cm/h) and skin surface area available for contact (cm$^2$), respectively.

In addition, the route-specific lifetime cancer risk and hazard index can be estimated as follows:

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

$$HI = \sum_{i=1}^{m} \left( \frac{CDI_i}{RfD_i} \right)$$

where i refers to the CHCl$_3$, BDCM, DBCM, CHBr$_3$ which can be varied from 1 to m; j represents the various routes of exposure that could be varied from 1 to n; CR is cancer risk and SF and RfD are the slope factor ([mg/kg/d]$^{-1}$) for specific route and reference dose (mg/kg/d), respectively. SFs for CHCl$_3$, BDCM, DBCM, and CHBr$_3$ are 0, 0.062, 0.084, 0.0079 (mg/kg/d), respectively, while RfDs are 0.01, 0.02, 0.02 and 0.02 mg/kg/d, respectively. The SF factor shows the 95-percentile upper-bound lifetime cancer risk from exposure to the carcinogen compound and RfD is the safe dose that may be swallowed or ingested with no adverse effect (USEPA, 2018).

**Results and discussion**

**Water quality analysis**

The results of the Shapiro-Wilk test indicate that Temp A, TDS, Tub, RH, pH, and Cfm data have a normal distribution (all $P > 0.05$). This test failed to confirm the normal distribution of other variables in this study and indicate deviation from normality (all $P < 0.05$) in Table 3. The correlation analysis showed that there was a statistically significant positive linear relation between temp $W$ ($r = 0.385$, $P = 0.020$). RH ($r = 0.829$, $P = 0.042$) with Cfm. The correlation analysis showed that there was a statistically significant negative linear relation between EC ($r = -0.364$, $P = 0.029$), TDS ($r = -0.414$, $P = 0.012$) with Cfm. Other linear relationships with their correlation coefficients and $P$-value are demonstrated in Table 4.
Exposure assessment for trihalomethanes

In absence of such data, some past studies used the oral slope factor to approximate human health risks from THMs (Lee et al., 2009). In this article, the oral slope factors from USEPA were used to approximate human health risks through all exposure pathways. As CHCl₃ has a threshold, it was not included in cancer risk assessment. The reference dose, which is defined as the maximum level of the safe dose, for CHCl₃, BDCM, DBCM, and CHBr₃ are reported to be 0.01, 0.02, 0.02, and 0.02 mg/kg/d respectively (USEPA, 2018). The assessment of cancer risk of THMs is shown in Figure 1. The probabilistic cancer risk histogram of THMs is shown in Figure 1. The results of cancer risk simulation illustrated that cancer risk related to THMs for swimmers fluctuated from 10⁻⁷ to 4 × 10⁻⁷ (mean of 1.44 × 10⁻⁷). These results plainly presented that exposure to the THMs during swimming demonstrated the high value of cancer risk and risk reduction strategies should be considered for swimmer protection and the overall health risk.

According to the recommendation of health care administrators and health policy, there are many routes of exposure with THMs and then pathway assessment methodology could be employed for prediction of the cancer risk and adverse effects. Ingestion and dermal contact absorption as two main exposure routes were considered in this study.

Table 3. Concentrations of trihalomethanes and other factors for swimming pool water samples.

<table>
<thead>
<tr>
<th></th>
<th>Mean ± SD</th>
<th>Median (range)</th>
<th>P-for normality*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Temp A (°C)</strong></td>
<td>23.98 ± 0.88</td>
<td>24.2 (22.1–25.2)</td>
<td>0.009</td>
</tr>
<tr>
<td><strong>Temp W (°C)</strong></td>
<td>32.42 ± 1.57</td>
<td>33 (29–34)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td><strong>EC (µs/cm)</strong></td>
<td>1396.25 ± 66.07</td>
<td>1373.5 (1335–1527)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td><strong>FRC</strong></td>
<td>2.71 ± 0.96</td>
<td>3 (1.5–4)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td><strong>TDS (mg/L)</strong></td>
<td>754.5 ± 15.59</td>
<td>762 (724–771)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td><strong>Tub (NTU)</strong></td>
<td>0.77 ± 0.33</td>
<td>0.6 (0.37–1.7)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td><strong>RH (%)</strong></td>
<td>81.5 ± 4.85</td>
<td>81.9 (71.3–88.3)</td>
<td>0.002</td>
</tr>
<tr>
<td><strong>pH</strong></td>
<td>7.66 ± 0.2</td>
<td>7.64 (7.28–8)</td>
<td>0.015</td>
</tr>
<tr>
<td><strong>CFm (µg/L)</strong></td>
<td>137.88 ± 114.82</td>
<td>101.21 (5.5–540.08)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td><strong>DCBMm (µg/L)</strong></td>
<td>47.73 ± 48.61</td>
<td>25.12 (0–170.68)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td><strong>DBCMm (µg/L)</strong></td>
<td>0.52 ± 2.26</td>
<td>0 (0–11.68)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td><strong>BFm (µg/L)</strong></td>
<td>0.85 ± 3.49</td>
<td>0 (0–15.45)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Based on Shapiro-Wilk test.

Table 4. Pearson’s correlation coefficients of THMs and other factors for swimming pool water samples.

<table>
<thead>
<tr>
<th></th>
<th>CFm</th>
<th>DCBMm</th>
<th>DBCMm</th>
<th>BFm</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Location</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>tempA</strong></td>
<td>r</td>
<td>P</td>
<td>r</td>
<td>P</td>
</tr>
<tr>
<td><strong>tempW</strong></td>
<td>0.198</td>
<td>0.247</td>
<td>−0.007</td>
<td>0.966</td>
</tr>
<tr>
<td><strong>cond</strong></td>
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</tr>
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<td><strong>FRC</strong></td>
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<td>0.214</td>
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<td><strong>TDS</strong></td>
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<tr>
<td><strong>Tub</strong></td>
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<td><strong>RH</strong></td>
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<td>0.753</td>
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<td>0.214</td>
</tr>
<tr>
<td><strong>pH</strong></td>
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<td>0.292</td>
<td>−0.126</td>
<td>0.463</td>
</tr>
</tbody>
</table>

r Pearson or Spearman correlation whenever appropriates.
Ingestion pathways

The simulation histogram for CDI imposed by chloroform via ingestion pathways is shown in Figure 2. The results of cancer risk simulation presented that the chloroform carcinogenic risk through ingestion exposure for swimmers varied from $1.05 \times 10^{-6}$ to $9.44 \times 10^{-6}$ mg/kg-d (mean value of $2.12 \times 10^{-6}$ mg/kg-d). According to the simulated results, high exposure to CHCl$_3$ in the swimming pools could increase the carcinogenic risk.

Figure 1. The probabilistic cancer risk histogram of THMs.

Figure 2. Simulation histogram for CDI imposed by chloroform via ingestion pathway.
The simulation histogram for CDI imposed by DCBM via ingestion pathways is shown in Figure 3. The results of CDI simulation for DCBM through ingestion route were less than CHCl₃ and varied from $10^{-6}$ to $3 \times 10^{-6}$ mg/kg-d (mean value of $7.35 \times 10^{-7}$ mg/kg-d).

The simulation histogram for CDI imposed by DBCM via ingestion pathways are shown in Figure 4. The results of CDI simulation for DBCM fluctuated from $1 \times 10^{-9}$ to $1 \times 10^{-8}$ mg/kg-d.
mg/kg-d (mean value of $8.12 \times 10^{-9}$ mg/kg-d), which was less than chloroform and DCBM. Lower CDI of DBCM in comparison with CHCl$_3$ and DCBM was related to low concentration of DBCM in the pool water.

The simulation histogram for CDI imposed by bromoform via ingestion pathways is shown in Figure 5. As seen in Figure 4, the simulated CDI for DBCM changed from $10^{-8}$ to $2 \times 10^{-8}$ mg/kg-d (mean value of $1.28 \times 10^{-8}$ mg/kg-d), which was less than chloroform and DCBM and near to DBCM, which was due to low potential of DCBM and DBCM production in the swimming pool water.

**Life time cancer risk (ingestion pathway)**

The simulation histogram or probabilistic histogram of total carcinogenic risks related to ingestion pathway of DBPs in swimming water is shown in Figure 6. Since swimmers who are frequently in the swimming pool have more exposure with DBPs and must pose higher LTCR in comparison with other swimmers, various swimming frequency was studied. The mean of total LTCR for swimmers was $4.63 \times 10^{-8}$. According to the obtained results, low potential risk of carcinogen may have for swimmers due to low exposure to DBPs. The acceptable risk of lifetime cancer risks for swimmers recommended by USEPA is $10^{-6}$, which was lower than what is considered in this study (USEPA, 2018).

**Dermal pathway**

The CDIs of DBPs through dermal exposures are required to be evaluated to assess the risk of carcinogenicity in swimmers. The simulation histogram for CDI imposed by chloroform via dermal pathway is shown in Figure 7. The average of CDI of CHCl$_3$ was $3.95 \times 10^{-5}$ mg/kg-d which was higher than ingestion route.
The simulation histogram for CDI imposed by DCBM via dermal pathway is presented in Figure 8. According to the results, the higher risks of DCBM through dermal contact compared to ingestion pathway may be elucidated with higher frequency of swimming. The mean of CDI for DCBM through dermal contact obtained is $1.56 \times 10^{-6}$ mg/kg-d.

**Figure 6.** The lifetime cancer risk (LTCR) of THMs through ingestion pathway.

**Figure 7.** Simulation histogram for CDI imposed by chloform via dermal pathway.
The simulation histogram for CDI imposed by DBCM via dermal pathway is illustrated in Figure 9. The average intake of BDCM was predicted to be $1.88 \times 10^{-8}$ mg/kg-d. As seen in Figure 9, cancer risks through dermal contact were lower than chlorinated byproduct due to low concentration of brominated byproduct in this study.

Figure 8. Simulation histogram for CDI imposed by DCBM via dermal pathway.

Figure 9. Simulation histogram for CDI imposed by DBCM via dermal pathway.
The simulation histogram for CDI imposed by bromoform via dermal pathway is shown in Figure 10. The results of bromoform were predicted like DBCM, which were lower than CHCl₃ and DCBM. The mean of CDIs for bromoform was $3.08 \times 10^{-8}$ mg/kg-d.

The CDIs of DBPs were high that may be contributed to the low $K_{ow}$ of DBPs, causing higher permeability through dermal contact (Chowdhury et al., 2014). In addition, according to the results, the cancer risks through dermal route was higher than ingestion route. Also, cancer risks of chlorinated compounds were higher than brominated compounds. The higher amounts of CDIs could display much higher risks of cancer for human especially for swimmers. According to the previous researches, the mean CDIs in the city of Toronto for chloroform, BDCM, DBCM, and bromoform were predicted as $1.52 \times 10^{-4}$, $1.31 \times 10^{-4}$, $8.15 \times 10^{-5}$ and $1.60 \times 10^{-5}$ mg/kg-d respectively, which was higher than this study. In addition, cancer risk of brominated compound was higher than chlorinated compounds that was not confirmed in this study. The mean intakes of DBPs in Dhahran city were much higher than CDIs obtained in the present research (Fooladvand et al., 2011). Similar study has been conducted by Lee et al. (2009) to estimate the concentrations of TTHMs in indoor swimming pools, and the result showed that while risks from oral ingestion and dermal exposure to THMs are mostly less than $10^{-6}$, which is the negligible risk level defined by the USEPA, swimmers can also be at the greater risk from inhalation exposure ($7.77 \times 10^{-4}$–$1.36 \times 10^{-3}$) (Lee et al., 2009).

In another study by Siddique et al. (2015), it is reported the human health cancer risk for TTHMs through ingestion and dermal routes were estimated in “acceptable-low risk” ($\geq 1.0 \times 10^{-6}$; $\leq 5.10 \times 10^{-5}$), whereas through inhalation route it was estimated under “acceptable-high risk” ($\geq 5.10 \times 10^{-5}$; $\leq 1.0 \times 10^{-4}$) category (Siddique et al., 2015).

The results predicted in the present study displayed high potential risk of cancer for swimmers. Therefore, reduction of DBPs precursors such as urine content, personnel care, and pharmaceutical products could play a significant role for decreasing the DBPs 

![Figure 10. Simulation histogram for CDI imposed by bromoform via dermal pathway.](image)
formation. Several strategies can be followed to reduce the risks, such as creating gap between showers, taking shower prior to entering in swimming pool, lowering the retention time of water, adequate airflow in pool area, using appropriate disinfectant according to the water quality such as combined treatments, no use of water sources with precursors DBPs that can illustrate higher risk should be created.

**Conclusion**

The results of this study indicate that exposure to DBPs in indoor swimming pools could cause high potential risk of cancer for swimmers. The results showed that the mean values of CDIs for chloroform, DCBM, DBCM, and bromoform were $2.12 \times 10^{-6}$, $7.35 \times 10^{-7}$, $8.12 \times 10^{-9}$, and $1.28 \times 10^{-8}$ mg/kg-d through ingestion pathways and $3.95 \times 10^{-5}$, $1.56 \times 10^{-6}$, $1.88 \times 10^{-8}$, and $3.08 \times 10^{-8}$ mg/kg-d through dermal pathway, respectively. In addition, according to the results, the mean of total LTCR for swimmers was $4.63 \times 10^{-8}$. In addition, the cancer risks through dermal route were higher than ingestion route. Also, cancer risks of chlorinated compounds were higher than brominated compounds. According to the results, exposure frequencies had a significant effect on the carcinogenic risk of DBPs. Finally, the results of this study suggested that some strategies such as establishing some rules and guidelines for water treatment, water resources used, and swimmers can be useful for decreasing the production of DBPs and risk of human exposure.

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**Conflict of interest**

The authors declare that they have no conflict of interests.

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