



Formation of disinfection by-products in the swimming pool water treated with different disinfection types

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ABSTRACT

Exposure to disinfection by-products (DBPs) could have occurred in swimming pool water disinfected with chlorine. The purpose of this study was to investigate the concentrations of two categories of DBPs including trihalomethanes (THMs) and haloacetic acids (HAAs) in swimming pool water treated with different disinfection types. This study was conducted in ten swimming pools located in Tehran, Iran. Water samples were collected from swimming pools disinfected with various methods: chlorine (Cl_2), ozone/chlorine (O_3/Cl_2), and ultraviolet radiation (UV)/ Cl_2 . Each sample was analyzed to determine the concentrations of subgroups of THMs and HAAs. The concentrations of chloramine, urea, total organic carbon, chloride, and bromide were determined as well. The mean concentration of HAA5 was 945 $\mu\text{g/L}$, which was significantly higher than the overall mean concentration of THMs (548.5 $\mu\text{g/L}$) ($p = 0.04$). The mean concentrations of THMs or HAAs in different pools stratified by disinfectant types were significant ($p < 0.05$). Among subgroup compounds of THMs and HAAs, the highest mean concentrations were related to chloroform (TCM) (247 $\mu\text{g/L}$) and trichloroacetic acid (351 $\mu\text{g/L}$). The concentrations of THMs and HAAs in Pools I–J disinfected by UV/ Cl_2 were lower. Due to the high concentrations of THMs and HAAs, frequent monitoring of chlorine and DBPs, as well as pretreatment of pool water by UV and then O_3 , are needed in swimming pools.

Keywords: Disinfection by-products; Haloacetic acids; Indoor swimming pools; Trihalomethanes

1. Introduction

Swimming pools are applied for recreational activities [1]. The use of disinfectants greatly reduced the incidence of waterborne diseases in swimming pools [2–4]. Chlorine, chlorine dioxide, chloramine, ultraviolet radiation (UV) and ozone (O_3) are the most popular method for disinfecting swimming pool water in many countries [5,6].

Nevertheless, disinfection by-products (DBPs) form in swimming pool water from reactions between disinfectants such as chlorine and organic matter such as urine, skin cells, and sweat [7]. Swimming in indoor pools treated with combined chemical treatments (e.g. UV/ Cl_2 and ozone/

chlorine) may reduce direct exposure to DBPs [5,6]. To date, more than 100 DBPs have been identified in pool water [3]. Trihalomethanes (THMs) and haloacetic acids (HAAs) are two well-known and the most prevalent DBPs [8–10]. Among THM products; chloroform (TCM), bromodichloromethane (BDCM), bromoform (TBM) is listed as probable carcinogenic products (Group B2), while chlorodibromomethane (CDBM) is categorized as a possible carcinogen (Group C) [11]. Two out of nine HAA products, dichloroacetic acid (DCAA) and trichloroacetic acid (TCAA) are the most common products and they are classified as Group C and Group B2 carcinogens by the International Agency for Research on Cancer (IARC), respectively [12].

Several adverse health effects associated with THMs and HAAs have been documented in the literature include respiratory problems and asthma [13], adverse reproductive outcomes, congenital anomalies, destruction of the liver, kidneys and nervous system [14], bladder [15] and colorectal cancers [7,14]. As a result, the maximum contaminant levels (MCLs) have been established by the United States Environmental Protection Agency (USEPA) and World Health Organization (WHO) to reduce the health effects of DBPs [16]. In swimming pool water, the MCLs for THMs and HAAs suggested by USEPA are 80 and 60 µg/L [17,18]. WHO guidelines for the THMs and both CDBM and bromoform are 20 and 10 µg/L, respectively [7,19]. Consequently, measurements of THMs and HAAs concentrations and estimation of their toxicity are necessary for the health studies. There are several studies available measuring the concentrations of THMs and HAAs in swimming pools [20–23]. The mean or median concentrations reported by these studies ranged from 114 to 1,150 µg/L for THMs and 294 to 3,500 µg/L for HAA5 [20–22]. Regarding the concentration of DBPs in Iran, there is only one study available measuring the concentration of HAA5 [23]. The mean concentration of HAA5 in this study was 1,045 µg/L (ranged from 148 to 3,488 µg/L) [23]. Thus, I decided to investigate the concentrations of THMs and HAAs in Iranian swimming pools. This study aimed to investigate the concentrations of THMs and HAAs and estimation of their toxicity in swimming pools in Tehran, Iran.

2. Materials and methods

2.1. Study site and sample collection

This study was performed in 10 crowded swimming pools located in Tehran, Iran. Twelve samples per each pool were collected in summer 2016. Samples were taken in pool water depth about 15–30 cm using 1 L bottle glasses. Prior to sampling, sodium thiosulfate was added to each bottle to quench any chlorine residual. All collected samples were stored at 4°C and tested within 5 d. Five Pools (A–E) disinfected with chlorine (Cl₂), three Pools (F–H) disinfected with ozone/chlorine (O₃/Cl₂), and two Pools (I–J) disinfected with UV radiation followed by chlorine (UV/Cl₂). During sampling simultaneously the temperature, free chlorine, and pH were measured by pH meter HACH HQ40D portable (Loveland, Colorado, USA).

2.2. Analysis of sample

Samples were analyzed to determine four THMs (including TCM, BDCM, Dibromochloromethane (DBCM) and TBM) and five HAAs (including DCAA, TCAA, monochloroacetic acid (MCAA), monobromoacetic acid (MBAA) and dibromoacetic acid (DBAA)) as well as monochloramine.

Samples were initially filtered with 0.45 µm pore size. Free chlorine was measured by N,N-diethyl-p-phenylenediamine (DPD) method [24] and monochloramine by the iodophenol method (Hach Co., USA) [25]. The concentration of ammonium was measured by the 4500-NH₃ method, urea and UV₂₅₄ with UV spectrophotometer (Dr5000), chloride and bromide with an ion-chromatograph [24], and total organic carbon (TOC) by TOC analyzer equipped with a chemiluminescence detector (Shimadzu, Japan).

To measure THMs, samples were analyzed using the USEPA method 551.1. In this method, liquid–liquid extraction procedure with methyl tertiary butyl ether (MTBE) coupled with gas chromatography/mass spectrometry (GC-MS) was applied (Agilent 7890A, USA) [26]. The injector and detector temperatures were set at 200°C and 290°C, respectively. For HAAs analysis, USEPA method 552.3 was used. The detail of HAAs analysis was reported by Dehghani et al. [23]. The calibration curve was drawn using five concentrations, ranged from 0.1 to 500 µg/L. The coefficient of variation of an internal standard for both measurements of THMs and HAAs were below 15%. Recovery was achieved between 80%–120%. Limit of detections for THMs and HAAs were 0.1 and 0.5 µg/L, respectively.

2.3. Estimation of toxicity

Based on the measured concentration of the different THMs and HAAs, the cytotoxicity and genotoxicity were estimated as the sum of the concentration of each compound (C_i) divided by its EC₅₀ is defined as given by the following formula:

$$\text{Toxicity} = \sum \frac{C_i}{EC_{50}} \quad (1)$$

All the EC₅₀ values were applied as reported in the literature [27,28] (Table S1).

2.4. Statistical analysis

Statistical analyses were performed with the statistical package R software version 3.3.2 (R Core Team 2018). The Wilcoxon test was employed to compare the concentrations between THMs and HAAs. Differences in concentrations between THMs and HAAs stratified by pools were examined using the Kruskal–Wallis test. Spearman's correlations were applied to evaluate the correlation between parameters.

3. Results

3.1. Physicochemical parameters

The mean number of swimmers per hour was 31.5, ranged from 25 to 45. Temperature and pH of pools ranging from 23°C to 29°C and 6.5 to 8.0, respectively. The concentrations of urea, ammonium, TOC, chloride, bromide, and mono-chloramine stratified by type of pools are given in Figs. 1a–f. The mean concentrations of urea (NH₂CONH₂) and TOC were 5.0 mg/L (ranged from 2.5 to 6.8 and 7.7 mg/L (ranged from 1.7 to 15.5)). The chloride and bromide concentrations ranged from 73 to 92 mg/L and 9.5 to 28.5 mg/L, respectively. Table 1 presents the values of the physicochemical parameters of the collected samples.

3.2. Concentration of THMs and HAA5

The concentrations of different types of THMs and HAAs (µg/L) are presented in Fig. 2. The mean concentrations of THMs and HAA5 were 549 (ranged from 286 to

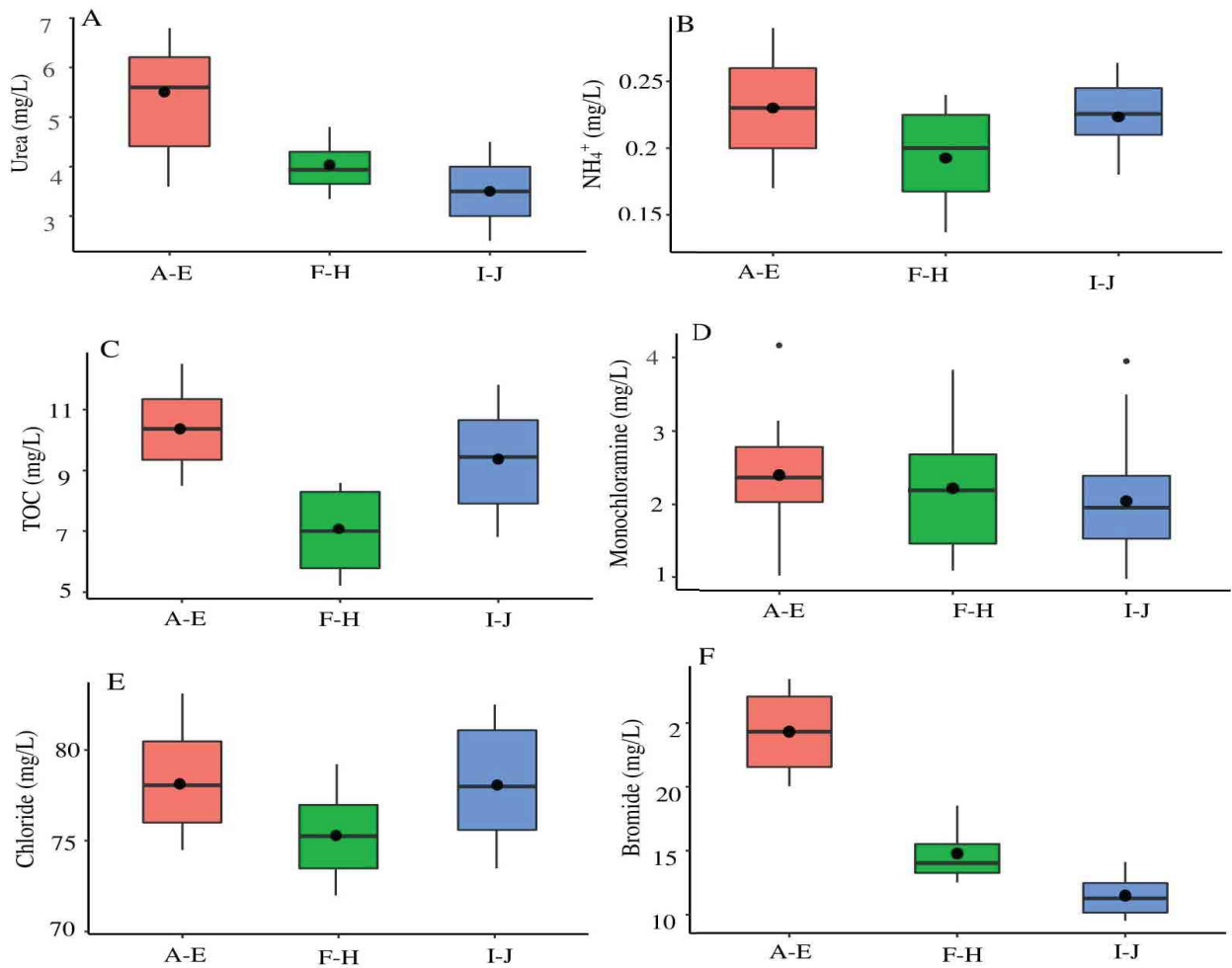


Fig. 1. Concentrations of (a) urea, (b) ammonium, (c) TOC, (d) monochloramine, (e) chloride, and (f) bromide in the swimming water samples collected from Pools A-E, F-H and I-J.

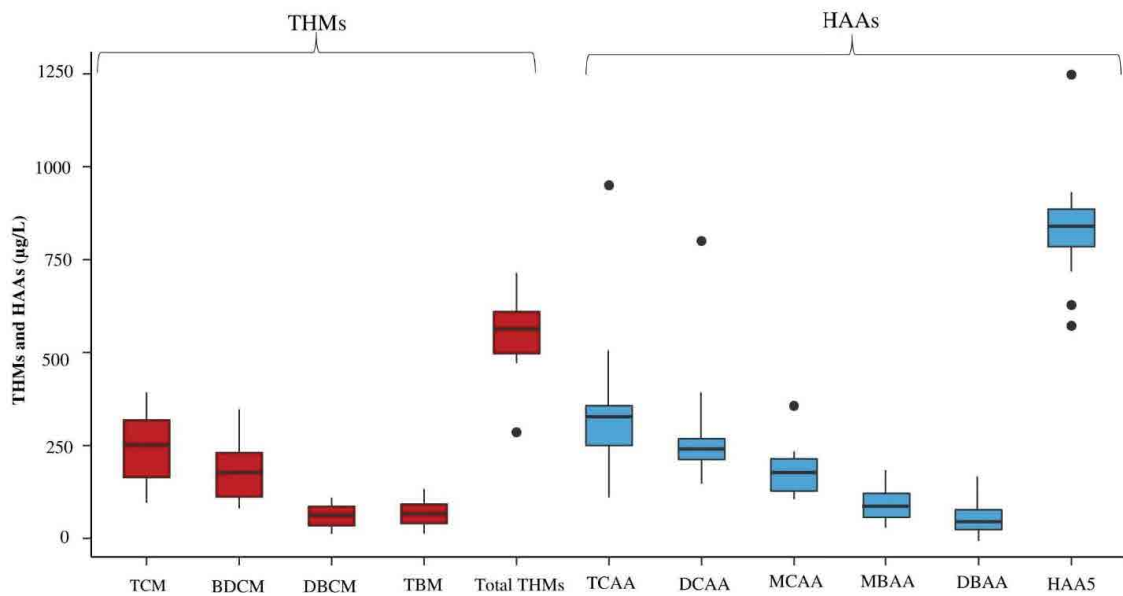


Fig. 2. Concentrations of trihalomethanes (THMs) and haloacetic acids (HAAs) (µg/L) in swimming pools.

Table 1
Physio-chemical characteristics of swimming pool water samples

| Pools | T (°C) | Cl ₂ (mg/L) | Turbidity (mg/L) | pH | NH ₄ ⁺ (mg/L) | Urea (mg/L) | Chloride (mg/L) | Bromide (mg/L) | Monochloramines (mg/L) | Organic chloramines (mg/L) | TOC (mg/L) | UV ₂₅₄ | Number of users per hour |
|-------|--------|------------------------|------------------|------|-------------------------------------|-------------|-----------------|----------------|------------------------|----------------------------|------------|-------------------|--------------------------|
| A | 25.0 | 1.0 | 0.56 | 7.5 | 0.21 | 5.2 | 85.6 | 20.0 | 3.68 | 1.15 | 4.40 | 0.046 | 35.0 |
| B | 28.0 | 1.5 | 0.36 | 7.7 | 0.25 | 6.8 | 78.5 | 28.5 | 2.82 | 0.78 | 15.5 | 0.055 | 45.0 |
| C | 26.5 | 3.5 | 0.35 | 7.3 | 0.29 | 3.2 | 91.1 | 22.0 | 1.86 | 0.78 | 6.30 | 0.042 | 26.0 |
| D | 28.0 | 0.8 | 0.65 | 7.3 | 0.17 | 3.8 | 74.5 | 26.5 | 1.78 | 0.78 | 3.90 | 0.082 | 32.0 |
| E | 26.0 | 4.0 | 0.41 | 8.5 | 0.24 | 3.5 | 88.5 | 18.5 | 1.64 | 0.78 | 15.5 | 0.075 | 28.0 |
| F | 27.5 | 0.8 | 0.37 | 7.2 | 0.38 | 4.8 | 73.0 | 12.5 | 1.58 | 0.97 | 10.5 | 0.094 | 32.0 |
| G | 23.0 | 1.5 | 0.23 | 8.0 | 0.18 | 2.5 | 82.5 | 13.5 | 1.56 | 1.14 | 1.72 | 0.085 | 28.0 |
| H | 24.5 | 3.0 | 0.46 | 7.3 | 0.13 | 4.5 | 86.5 | 14.5 | 2.53 | 0.64 | 5.50 | 0.075 | 30.0 |
| I | 27.0 | 2.5 | 0.54 | 6.7 | 0.38 | 5.5 | 83.2 | 10.8 | 1.78 | 1.50 | 3.50 | 0.068 | 40.0 |
| J | 25.0 | 3.0 | 0.31 | 6.5 | 0.24 | 4.3 | 80.5 | 9.50 | 1.86 | 1.25 | 9.81 | 0.079 | 29.0 |
| Mean | 26.05 | 2.16 | 0.42 | 7.40 | 0.25 | 4.41 | 82.39 | 17.63 | 2.11 | 0.98 | 7.66 | 0.07 | 32.50 |
| SD | 1.57 | 1.12 | 0.12 | 0.48 | 0.08 | 1.18 | 5.57 | 6.23 | 0.66 | 0.26 | 4.68 | 0.02 | 5.66 |

715 µg/L and 945 µg/L (ranged from 571.8 and 2247.5 µg/L), respectively (Table 2). The Wilcoxon test results showed a significant difference between the mean concentrations of THMs and HAAs ($p < 0.05$). The concentrations of THMs and HAAs subgroup, stratified by disinfectant methods are presented in Table 3. The estimated cytotoxicity and genotoxicity of THMs and HAAs subgroup, stratified by disinfectant type are presented in Fig. 3.

3.3. Correlation analysis

Spearman’s correlation analysis between measured variables is presented in Fig. 4. A positive significant correlation was found between concentrations of THMs and HAAs with temperature, chlorine, urea, ammonium, TOC, chloride, bromide and the number of swimmers.

4. Discussion

Several factors such as the number of swimmers, temperatures, pH, the chlorine dose, the bromide, and chloride content, the extent of TOC, urea and ammonium ions have been suggested to influence on the THMs and HAAs variations in swimming pools water [29]. The mean concentration of free chlorine was 2.2 mg/L (ranged from 0.8 to 4 mg/L). About 65% of our samples had chlorine residual concentration higher than the value suggested by WHO (1.4 mg/L) [19]. Consequently, the formation potentials of THMs and HAAs were increased in a high level of chlorine. The concentration of urea (NH₂CONH₂), as a precursor of DBPs formation, ranged from 2.5 to 6.8 mg/L (mean 5.0 mg/L), and it was in agreement with concentrations reported by Abidi et al. [30] and De Laat et al. [31] in other swimming pools. The most common sources of urea in pool water are swimmer body fluid discharges [8]. The mean concentration of TOC as a second precursor of DBPs formation ranged from 9.15 to 15.5 mg/L. Previous studies similarly showed that the mean concentrations of TOC changing between 1.5 to 39.3 mg/L [20,31–33]. The high variabilities of TOC concentrations in swimming pools were associated with three different sources: organic loads introduced by swimmer’s bodies (e.g. urine, sweat, hair, skin particles, mucus, lotion, sunscreen, and cosmetics), characteristics of the filling water, and disinfection methods (e.g. Cl₂, UV or ozone) [34]. Other important factors in THMs and HAAs formation are related to chloride and bromide concentrations. The lower concentration of chloride and bromide in Pools F–G was related to the ozone/chlorine (O₃/Cl₂) method [33]. Chloride and bromide were contributed to the formation of several chlorinated and brominated DBPs [33]. Consequently, lower concentrations of chloride and bromide associated with the minor form of these DBPs [33]. Furthermore, the increase in UV₂₅₄ values was associated with greater concentrations of TOC and indicate that the swimming pool was contaminated by organic compounds.

The measured concentrations of THMs were exceeded from the concentration of 80 µg/l suggested by the EPA [18,35]. The mean concentration of THMs in Pools I–J was borderline different than the concentrations in Pools A–E and F–G ($p = 0.05$) (Table 3). In parallel to our result, the highest mean concentration of THMs in pool water reported by

Table 2
Mean concentrations of subgroup compounds of THMs and HAAs in each pool

| Pools | THMs ($\mu\text{g/L}$) | | | | | HAAs ($\mu\text{g/L}$) | | | | | |
|-------|--------------------------|-------|------|-------|------------|--------------------------|--------|-------|------|------|--------|
| | TCM | BDCM | DBCM | TBM | Total THMs | TCAA | DCAA | MCAA | MBAA | DBAA | HAA5 |
| A | 225.5 | 234 | 15.5 | 12.6 | 487.6 | 110 | 175.5 | 220.5 | 95.5 | 26.5 | 628 |
| B | 343.5 | 189 | 12 | 68 | 612.5 | 339 | 263 | 128.5 | 65.5 | 60.9 | 856.9 |
| C | 320.5 | 220.5 | 18.5 | 45 | 604.5 | 950 | 800 | 356.5 | 95.5 | 45.5 | 2247.5 |
| D | 140.7 | 348 | 15.5 | 38.5 | 542.7 | 379.5 | 242.5 | 105.5 | 66.5 | 28.5 | 822.5 |
| E | 202.5 | 80 | 110 | 133.5 | 526.0 | 352.5 | 265 | 106 | 58.5 | 35.5 | 817.5 |
| F | 292 | 268 | 90 | 45 | 695.0 | 358.5 | 202 | 127 | 87.5 | 30.5 | 805.5 |
| G | 393.5 | 80 | 33 | 38.5 | 545.0 | 316 | 270 | 235 | 55.6 | 55.5 | 932.1 |
| H | 143 | 189 | 45 | 95 | 472.0 | 180 | 147.36 | 187.5 | 28.5 | 28.5 | 571.86 |
| I | 95.5 | 85.5 | 15 | 89.5 | 285.5 | 285 | 255 | 186.5 | 95 | 66.5 | 888 |
| J | 312 | 214.5 | 95 | 93 | 714.5 | 238.5 | 300 | 195 | 75.5 | 70.5 | 879.5 |
| Mean | 246.9 | 190.9 | 45.0 | 65.9 | 548.5 | 350.9 | 292.0 | 184.8 | 72.4 | 44.8 | 944.9 |
| SD | 94.8 | 83.4 | 36.5 | 34.5 | 116.4 | 215.7 | 175.1 | 72.4 | 20.7 | 16.3 | 447.5 |

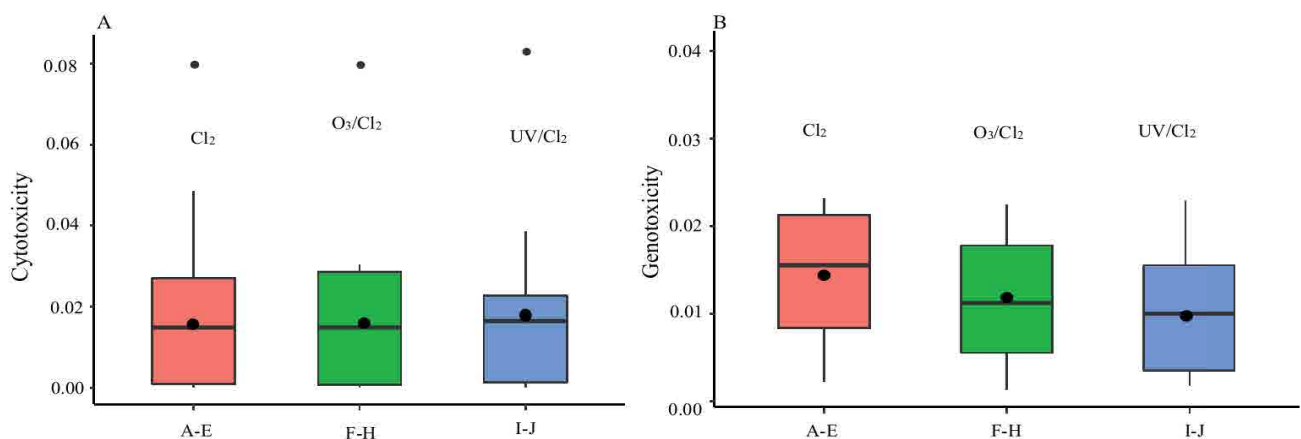


Fig. 3. Estimated cytotoxicity and genotoxicity in the swimming water samples collected from Pools A–E, F–H and I–J.

other studies ranged from 49.6 to 1,150 $\mu\text{g/l}$ [4,20,34,36–38]. Using Cl_2 as a disinfectant in pool water leads to the formation of two compounds, including chloride and oxychlorines ions, which related to the high concentration of THMs [39]. Among subgroup compounds of THMs, TCM (chloroform) was the most abundant compound with a mean concentration of 252 $\mu\text{g/L}$ (Table 2 and Fig. 2), which was two-fold higher than the concentrations reported by Chu et al. [20] (113 $\mu\text{g/L}$) and Chen [40] (71.3 $\mu\text{g/L}$) studies. Moreover, our concentration exceeded the value (200 $\mu\text{g/L}$) suggested by WHO guidelines [19]. DBCM as a THMs compound was detectable with a mean concentration of 45 $\mu\text{g/L}$. Finally, the concentration of BDCM in Pools A–E was significantly higher than the other pools, ($p = 0.04$) (Table 3).

The mean concentration of HAA5 was 945 $\mu\text{g/L}$, ranged from 571.8 to 2,247.5 $\mu\text{g/L}$. This concentration was similar to the concentration (2,232.9 $\mu\text{g/L}$) reported by Parinet et al. [41] in seawater pools, but lower the concentrations (413 $\mu\text{g/L}$) reported by Simard et al. [34] (413 $\mu\text{g/L}$) and Tardif et al. [21] (294.8 $\mu\text{g/L}$) in indoor tap water pools. Among subgroup compounds of HAAs, the highest concentration was related

to the TCAA compound with a mean of 365 $\mu\text{g/L}$ (Table 2). Similar findings were found by other studies [10,42]. The higher mean concentration of HAAs compared with THMs is probably associated with the higher molecular stability of HAAs rather than THMs [10,43]. The mean concentrations of THMs and HAAs in Pools F–G disinfected with UV/Cl_2 and Pools I–J disinfected with ozone/chlorination were lower than the concentrations in Pools A–E.

The absolute value of THMs toxicity associated with the increasing concentration of THMs in pools disinfected with chlorine followed by O_3/Cl_2 and UV/Cl_2 . Based on the above, HAAs are the most potent DBP group investigated in this study and are the main contributor of the investigated compounds to the cytotoxicity and genotoxicity of the chlorinated swimming pools. Similar results were reported by Hansen et al. [27].

5. Conclusions

The concentrations of THMs and HAAs were found to be distinctly high (HAA5 was significantly higher than THMs).

Table 3
THMs and HAAs concentrations and their cytotoxicity stratified by type of disinfectants used

| DBPs | Pools A–E (Chlorine) | | | Pools F–G (Ozone/chlorination) | | | Pools I–J (UV/Chlorine) | | | p ^a |
|-------------|----------------------|-------------|--------------|--------------------------------|-------------|--------------|-------------------------|-------------|--------------|----------------|
| | Mean ± SD | Range | Cytotoxicity | Mean ± SD | Range | Cytotoxicity | Mean ± SD | Range | Cytotoxicity | |
| TCM (µg/L) | 246.5 ± 84.3 | 140.7–343.5 | 0.03 | 276.2 ± 126.0 | 143–393.5 | 0.03 | 203.7 ± 153.1 | 95.5–312 | 0.02 | 0.08 |
| BDCM (µg/L) | 214.3 ± 96.1 | 80–348 | 0.02 | 179.0 ± 94.4 | 80–268 | 0.02 | 150 ± 91.2 | 85.5–214.5 | 0.02 | 0.04 |
| DBCM (µg/L) | 34.3 ± 42.4 | 12–110 | 0.01 | 56.0 ± 30.0 | 33–90 | 0.01 | 55 ± 56.6 | 15–95 | 0.01 | 0.22 |
| TBM (µg/L) | 59.52 ± 45.8 | 12.6–133.5 | 0.01 | 59.5 ± 30.9 | 38.5–95 | 0.01 | 91.25 ± 2.5 | 93–89.5 | 0.02 | 0.18 |
| TTHM (µg/L) | 554.7 ± 53.1 | 487.6–612.5 | – | 570.7 ± 113.7 | 472–695 | – | 500.0 ± 303.3 | 285.5–714.5 | – | 0.51 |
| TCAA (µg/L) | 426.2 ± 312.1 | 110–950 | 0.00 | 284.8 ± 93.2 | 180–358.5 | 0.00 | 261.75 ± 32.9 | 238.5–285 | 0.00 | 0.06 |
| DCAA (µg/L) | 349.2 ± 254.6 | 175.5–800 | 0.05 | 206.5 ± 61.4 | 147.4–270 | 0.03 | 277.5 ± 31.8 | 255–300 | 0.04 | 0.08 |
| MCAA (µg/L) | 183.4 ± 107.7 | 105.5–356.5 | 0.08 | 183.2 ± 54.1 | 127–235 | 0.08 | 190.75 ± 6.0 | 186.5–195 | 0.08 | 0.55 |
| MBAA (µg/L) | 76.3 ± 17.8 | 58.5–95.5 | 0.00 | 57.2 ± 29.5 | 28.5–87.5 | 0.00 | 85.25 ± 13.8 | 75.5–95 | 0.00 | 0.54 |
| DBAA (µg/L) | 39.38 ± 14.1 | 26.5–60.9 | 0.00 | 38.2 ± 15.0 | 28.5–55.5 | 0.00 | 68.5 ± 2.8 | 66.5–70.5 | 0.00 | 0.08 |
| HAA5 (µg/L) | 1074.5 ± 661.9 | 628–2,247.5 | – | 769.8 ± 182.8 | 571.9–932.1 | – | 883.8 ± 6.0 | 879.5–888 | – | 0.65 |

Note: Significant P-values are shown in bold.
^ap-values from Kruskal–Wallis test between three different pools.

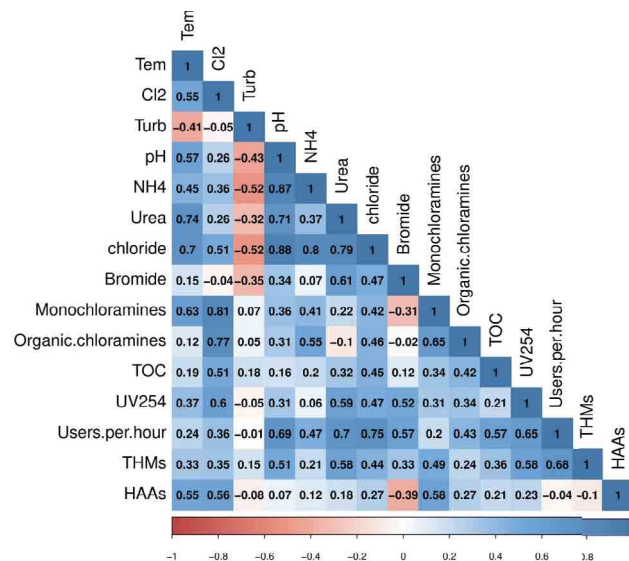


Fig. 4. Spearman's correlation test between variables.

The concentrations of THMs or HAAs in diverse swimming pool water were dependent on the type of disinfectants and precursor concentrations, including ammonium, TOC, urea chloride and bromide ions. TCM and TCAA were the most dominant compounds among THMs and HAAs, respectively. The concentrations of all categories of THMs and HAAs in the pool disinfected with UV/Cl₂ and ozone/chlorination was lower than those in the pool disinfected with chlorination. Therefore, genotoxicity and cytotoxicity are reduced in pools disinfected with UV/Cl₂. The controls of the organic precursor entrance into swimming pools and disinfection with UV/chlorine and then ozone/chlorine were the most effective ways to improve the chemical quality of the swimming pool water.

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References

- [1] D. Hendrickx, A. Stephen, D. Lehmann, D. Silva, M. Boelaert, J. Carapetis, R. Walker, A systematic review of the evidence that swimming pools improve health and wellbeing in remote Aboriginal communities in Australia, *Aust. N.Z. J. Public Health*, 40 (2016) 30–36.
- [2] S.J. Judd, G. Bullock, The fate of chlorine and organic materials in swimming pools, *Chemosphere*, 51 (2003) 869–879.
- [3] W.A. Cheema, K.M.S. Kaarsholm, H.R. Andersen, Combined UV treatment and ozonation for the removal of by-product precursors in swimming pool water, *Water Res.*, 110 (2017) 141–149.
- [4] J. Lee, K.-T. Ha, K.-D. Zoh, Characteristics of trihalomethane (THM) production and associated health risk assessment in swimming pool waters treated with different disinfection methods, *Sci. Total Environ.*, 407 (2009) 1990–1997.
- [5] W.A. Cheema, K.M.S. Kaarsholm, H.R. Andersen, Treatment of Swimming Pool Water with UV Followed by Ozone,

- International Ozone Association Pan American Group, Las Vegas, United States, 2016.
- [6] M.H. Dehghani, G.-R. Jahed, A. Zarei, Investigation of low-pressure ultraviolet radiation on inactivation of rhabditidae nematode from water, Iran. *J. Public Health*, 42 (2013) 314–319.
 - [7] R.A.A. Carter, C.A. Joll, Occurrence and formation of disinfection by-products in the swimming pool environment: a critical review, *J. Environ. Sci.*, 58 (2017) 19–50.
 - [8] S. Chowdhury, K. Alhooshani, T. Karanfil, Disinfection by products in swimming pool: occurrences, implications and future needs, *Water Res.*, 53 (2014) 68–109.
 - [9] M.J. Plewa, E.D. Wagner, W.A. Mitch, Comparative mammalian cell cytotoxicity of water concentrates from disinfected recreational pools, *Environ. Sci. Technol.*, 45 (2011) 4159–4165.
 - [10] T. Manasfi, B. Coulomb, J.-L. Boudenne, Occurrence, origin, and toxicity of disinfection byproducts in chlorinated swimming pools: an overview, *Int. J. Hyg. Environ. Health*, 220 (2017) 591–603.
 - [11] US. EPA, Integrated Risk Information System, 2008. Available at: <http://www.epa.gov/iris/backgrd.html>.
 - [12] WHO, IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Some Drinking-Water Disinfectants and Contaminants, Including Arsenic, IARC Press, Lyon, France, 2004. Available at: <https://monographs.iarc.fr/iarc-monographs-on-the-evaluation-of-carcinogenic-risks-to-humans-37/>.
 - [13] A. Bernard, C. Voisin, A. Sardella, Con: respiratory risks associated with chlorinated swimming pools: a complex pattern of exposure and effects, *Am. J. Respir. Critical Care Med.*, 183 (2011) 570–572.
 - [14] A. Florentin, A. Hautemanière, P. Hartemann, Health effects of disinfection by-products in chlorinated swimming pools, *Int. J. Hyg. Environ. Health*, 214 (2011) 461–469.
 - [15] C.M. Villanueva, K.P. Cantor, J.O. Grimalt, N. Malats, D. Silverman, A. Tardon, R. Garcia-Closas, C. Serra, A. Carrato, G. Castaño-Vinyals, R. Marcos, N. Rothman, F.X. Real, M. Dosemeci, M. Kogevinas, Bladder cancer and exposure to water disinfection by-products through ingestion, bathing, showering, and swimming in pools, *Am. J. Epidemiol.*, 165 (2006) 148–156.
 - [16] C.M. Villanueva, S. Cordier, L. Font-Ribera, L.A. Salas, P. Levallois, Overview of disinfection by-products and associated health effects, *Curr. Environ. Health Rep.*, 2 (2015) 107–115.
 - [17] USEPA, National Primary Drinking Water Regulations: Disinfectants and Disinfection By-Products, Notice of Data Availability, Final Rule, Federal Register, 63 (1998) 69390–69476. Available at: <https://www.federalregister.gov/documents/2006/01/04/06-3/national-primary-drinking-water-regulations-stage-2-disinfectants-and-disinfection-byproducts-rule>.
 - [18] USEPA, National Primary Drinking Water Regulations: Stage 2 Disinfectants and Disinfection By-products Rule: Final Rule, Federal Register, 71 (2006) 388. Available at: <https://www.federalregister.gov/documents/2006/01/04/06-3/national-primary-drinking-water-regulations-stage-2-disinfectants-and-disinfection-byproducts-rule>.
 - [19] WHO, Guidelines for Safe Recreational Water Environments: Swimming Pools and Similar Environments, Guidelines for Safe Recreational Water Environments: Swimming Pools and Similar Environments, 2006. Available at: https://www.who.int/water_sanitation_health/publications/safe-recreational-water-guidelines-2/en/.
 - [20] H. Chu, M.J. Nieuwenhuijsen, Distribution and determinants of trihalomethane concentrations in indoor swimming pools, *Occup. Environ. Med.*, 59 (2002) 243–247.
 - [21] R. Tardif, C. Catto, S. Haddad, S. Simard, M. Rodriguez, Assessment of air and water contamination by disinfection by-products at 41 indoor swimming pools, *Environ. Res.*, 148 (2016) 411–420.
 - [22] X.M. Wang, G.L. MI, X.L. Zhang, H.W. Yang, Y.F. Xie, Haloacetic acids in swimming pool and spa water in the United States and China, *Front. Environ. Sci. Eng.*, 8 (2014) 820–824.
 - [23] M.H. Dehghani, M. Farhang, A. Zarei, Data on the level of haloacetic acids in indoor swimming pools of Iran: a case study of Tehran, *Data Brief*, 19 (2018) 326–330.
 - [24] WEF, APHA, Standard Methods for the Examination of Water and Wastewater, American Public Health Association (APHA), Washington, D.C., USA, 2005.
 - [25] W. Lee, P. Westerhoff, Formation of organic chloramines during water disinfection: chlorination versus chloramination, *Water Res.*, 43 (2009) 2233–2239.
 - [26] Appropriate Preparation Technique, Method 8260B Volatile Organic Compounds by Gas Chromatography/Mass Spectrometry (GC/MS), US Environmental Protection Agency, Cincinnati, Ohio, 1996.
 - [27] K.M. Hansen, S. Willach, M.G. Antoniou, H. Mosbæk, H.-J. Albrechtsen, H.R. Andersen, Effect of pH on the formation of disinfection byproducts in swimming pool water—is less THM better?, *Water Res.*, 46 (2012) 6399–6409.
 - [28] M.J. Plewa, E.D. Wagner, M.G. Muellner, K.-M. Hsu, S.D. Richardson, Occurrence, Formation, Health Effects and Control of Disinfection By-Products in Drinking Water, Chapter 3, T. Karanfil, S.W. Krasner, P. Westerhoff, Y. Xie, Eds., Comparative Mammalian Cell Toxicity of N-DBPs and C-DBPs, American Chemical Society, Washington, D.C., 2008, pp. 36–50.
 - [29] H. Kim, J. Shim, S. Lee, Formation of disinfection by-products in chlorinated swimming pool water, *Chemosphere*, 46 (2002) 123–130.
 - [30] H. Abidi, J.-L. Gass, M.-F. Grenier-Loustalot, Analyse quantitative de l'urée dans l'eau par HPLC-APCI-MS-MS et HPLC-ES-MS-MS, *Actual. Chim.*, (2001) 33–36.
 - [31] J. De Laet, W. Feng, D.A. Freyfer, F. Dossier-Berne, Concentration levels of urea in swimming pool water and reactivity of chlorine with urea, *Water Res.*, 45 (2011) 1139–1146.
 - [32] N.P. Thacker, V. Nitnaware, Factors influencing formation of trihalomethanes in swimming pool water, *Bull. Environ. Contam. Toxicol.*, 71 (2003) 633–640.
 - [33] C. Hang, B. Zhang, T. Gong, Q. Xian, Occurrence and health risk assessment of halogenated disinfection byproducts in indoor swimming pool water, *Sci. Total Environ.*, 543 (2016) 425–431.
 - [34] S. Simard, R. Tardif, M.J. Rodriguez, Variability of chlorination by-product occurrence in water of indoor and outdoor swimming pools, *Water Res.*, 47 (2013) 1763–1772.
 - [35] B. Ljubic, L. Sundac, [Council] Directive 98/83/EC [of 3 November 1998] on the Quality of Water Intended for Human Consumption: Review and Integral Translation [From English into Serbian], *Voda i sanitarna tehnika (Serbia and Montenegro)*, 1998.
 - [36] W.A. Weaver, J. Li, Y. Wen, J. Johnston, M.R. Blatchley, E.R. Blatchley III, Volatile disinfection by-product analysis from chlorinated indoor swimming pools, *Water Res.*, 43 (2009) 3308–3318.
 - [37] C. Lourencetti, J.O. Grimalt, E. Marco, P. Fernandez, L. Font-Ribera, C.M. Villanueva, M. Kogevinas, Trihalomethanes in chlorine and bromine disinfected swimming pools: air-water distributions and human exposure, *Environ. Int.*, 45 (2012) 59–67.
 - [38] E. Righi, G. Fantuzzi, G. Predieri, G. Aggazzotti, Bromate, chlorite, chlorate, haloacetic acids, and trihalomethanes occurrence in indoor swimming pool waters in Italy, *Microchem. J.*, 113 (2014) 23–29.
 - [39] J. Jin, M.G. El-Din, J.R. Bolton, Assessment of the UV/chlorine process as an advanced oxidation process, *Water Res.*, 45 (2011) 1890–1896.
 - [40] M.J. Chen, J.M. Duh, R.H. Shie, J.H. Weng, H.T. Hsu, Dynamic real-time monitoring of chloroform in an indoor swimming pool air using open-path Fourier transform infrared spectroscopy, *Indoor Air*, 26 (2016) 457–467.
 - [41] J. Parinet, S. Tabaries, B. Coulomb, L. Vassalo, J.-L. Boudenne, Exposure levels to brominated compounds in seawater swimming pools treated with chlorine, *Water Res.*, 46 (2012) 828–836.
 - [42] R.Y.L. Yeh, M.J. Farre, D. Stalter, J.Y.M. Tang, J. Molendijk, B.I. Escher, Bioanalytical and chemical evaluation of disinfection by-products in swimming pool water, *Water Res.*, 59 (2014) 172–184.
 - [43] A. Kanan, T. Karanfil, Formation of disinfection by-products in indoor swimming pool water: the contribution from filling water natural organic matter and swimmer body fluids, *Water Res.*, 45 (2011) 926–932.

Supplementary information*S1. EC₅₀ values for toxicity estimation*

Table S1

EC50 values for cytotoxicity and genotoxicity

| | | Cytotoxicity EC ₅₀ (mol/L) | Genotoxicity EC ₅₀ (mol/L) |
|-----|---------------------------------|---------------------------------------|---------------------------------------|
| THM | Trichlormethan (TCM) | 9.1×10^{-3} | * |
| | Bromodichloromethane (BDCM) | 9.1×10^{-3} | * |
| | Dibromochloromethane (DBCM) | 5.2×10^{-3} | * |
| | Tribromomethane (TBM) | 4.0×10^{-3} | * |
| | Chloroacetic acid (CAA) | 9.0×10^{-4} | 4.1×10^{-4} |
| HAA | Bromoacetic acid (BAA) | 9.8×10^{-6} | 1.6×10^{-5} |
| | Dichloroacetic acid (DCAA) | 7.2×10^{-3} | * |
| | Trichloroacetic acid (TCAA) | 2.3×10^{-3} | * |
| | Bromochloroacetic acid (BCAA) | 8.4×10^{-4} | 3.7×10^{-3} |
| | Dibromoacetic acid (DBAA) | 5.2×10^{-4} | 1.7×10^{-3} |
| | Trichloronitromethane (TCnitro) | 5.2×10^{-4} | 9.1×10^{-5} |

*Compounds were not found genotoxic in the assay used.