An investigation of haloacetic acid occurrence in indoor and outdoor swimming pools in Beijing China

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Abstract. Swimming is one of the most popular amusement activities. In order to keep the swimmers from microbiological, the water need to be constantly disinfected. In this study, several indoor and outdoor swimming pools water and drinking water were investigated, it has found that swimming pool water has much higher HAA concentration and toxicity than the drinking water. DCAA and TCAA are the most abundant species in the indoor and outdoor swimming pool water. In the outdoor swimming pool water, TCAA concentration was higher than DCAA, while [DCAA]/[TCAA] was higher in the indoor swimming pool water.

1. Introduction
Swimming is one of the most popular amusement activities. The swimming pool water needed to be treated by disinfection to keep the water safe from viruses, bacteria, and other microorganisms. During disinfection, the reaction between the disinfectants and organic matters results formation of disinfection by-products. There are several disinfection methods could be chosen in the swimming pool, such as chlorine, ozone, UV, chloramine, etc. Chlorination produces DBPs such as trihalomethane (THMs) and haloacetic acids (HAAs), which have attracted much attention. The other disinfection methods may produce less THMs and HAAs, but other DBPs are produced. On the other hand, these methods could not provide a constant disinfecting effect as the chlorination. Therefore, the chlorination is the most popular in the swimming pool disinfection [1]. Governments of some nations have regulated to adopt chlorination for swimming pool disinfection. The DBPs in drinking water have been studied extensively. More than 600 DBPs have been identified, in which the THMs and HAAs are the two main groups of DBPs in drinking water. The swimming pool water, however, usually contain more DBPs, for mainly two reasons. First, swimmers bring microorganisms and organic substances (saliva, sweat, cosmetics, sunscreen, and urine), which could be precursors of DBPs, and contributes to water contamination [2, 3]. Second, the swimming pool needed more chlorine to disinfection.

The allowable limit of DBPs in drinking water is regulated by guidelines of the WHO and government. China has promulgated new drinking water regulations, in which the highest value of chloroform, dichloroacetic acid, and trichloro acetic acid are 60 µg/L, 50 µg/L and 100µg/L, respectively. WHO regulated HAA5 regulations not exceeding 60 µg/L. In the swimming pool water, however, there are no regulations for THMs and HAAs in recreational water in China. Some Europe countries have regulated total THMs (tTHMs) in swimming pools. France, UK, Finland, and WHO give a maximum tTHMs concentration of 100 µg/L, while the maximum value is 20 µg/L in Germany, Belgium regulates the chloroform level to be less than 100 µg/L. Comparing with the drinking water,
the swimming pool water may cause more severe health problems. One reason is the higher concentrations of DBPs in the swimming pool water. The second reason may be due to primary routes of exposures to DBPs during swimming, i.e. inhalation and dermal absorption [4, 5].

Some researchers have conducted studies on DBPs in swimming pool water in several aspects. Many studies investigated THM and HAA concentration in drinking water and swimming pool water. Some studies have focused on the effects on the health of swimmers and target the main routes of exposure by using plasma, blood, urine or alveolar air as exposure biomarkers [6, 7]. Some others have evaluated parameters that may influence the formation of DBPs, such as pH, temperature, total organic matter, chlorine dose, etc [8-15]. At the same time, the DBPs in the drinking water and swimming pool water behave different. The concentration and occurrence of DBPs in the swimming pool still need further investigation, mechanism, and impacting parameters still and not well understood. Based on which, the regulations need to be set up for more important DBPs. Reports of the occurrence of swimming pool DBPs in China are not abundant. Therefore, the occurrence of swimming pool DBPs in China is still an important topic that calls for investigation.

In this study, one of the most important DBPs group, HAAs, were investigated. We investigated ten indoor swimming pools, and one outdoor swimming pool. In which, the concentration of nine HAA species were tested. At the same time, HAAs in drinking water were also tested to compare with that in the swimming pool water.

2. Methodology

2.1. Study area and sampling strategy
Water samples were collected from indoor and outdoor swimming pools, and also from the distribution system located in Beijing, China. The chosen swimming pools belong to hotels, fitness clubs, schools, and residential communities. All of the water samples were supplied with city tap water and disinfected with NaClO.

The collection method followed the method EPA 551.1 and EPA 552.3. The samples for HAA analysis were collected in 60 mL glass bottles, with teflon lined screw caps. Water below surface 30 mm was collected. The bottles were filled overflowing with pool water and sealed tightly with caps. Prior to sampling, 60 µg of NH4Cl was added to produce a concentration of 100 mg/L for the HAAs test. Samples were analyzed immediately or no later than an overnight. All samples were collected and analyzed in duplicate.

2.2. Analytical method
Chemicals used in the study mainly include methyl tert-butyl ether (MTBE), NH4Cl, and standard samples were bought from the sigma-aldrich company. The water samples undergo treatment process in accordance with EPA552.3, including extraction, methylation with acidic methanol and analysed using an Agilent 7890 gas chromatography with a DB-1701 column for analytes separation and an ECD detector for quantification. In the GC step, the injector temperature was 210°C, a temperature escalating procedure 40°C (10min) → 2.5°C/min → 65°C (0min) → 10°C/min → 85°C (0min) → 20°C/min → 205°C (7min) was used. Concentrations of HAAs were calculated by the comparing the peak areas of each HAA with standards.

3. Results and discussion

3.1. Occurrence of DBPs in pools
Nine HAA were tested, and the peaks in the GC-ECD were monitored. In the indoor swimming pool water, only five of the HAA9 could be identified. In the drinking water, less HAAs were observed. In the outdoor swimming pool water, however, eight of the HAA9 could be identified. The concentrations of HAAs in the drinking water and swimming pool water are shown in the Table 1. DCAA, TCAA, BCAA, BDCAA, DBAA, and DBAA in the table stand for Dichloroacetic acid,
Trichloroacetic acid, Bromo chloroacetic acid, Bromo dichloroacetic acid, Dibromo acetic acid, respectively.

Table 1. HAA concentrations in the drinking water and swimming pool water (µg/L).

|       | DCAA | TCAA | BCAA | BDCAA | DBAA | HAA9 |
|-------|------|------|------|-------|------|------|
| DW1   | 2.40 | 1.00 | 0.00 | 0.00  | 0.00 | 3.40 |
| DW2   | 4.93 | 2.29 | 1.48 | 0.00  | 0.00 | 8.70 |
| DW3   | 2.37 | 1.19 | 1.29 | 0.00  | 0.06 | 4.91 |
| SW1   | 261.27 | 153.15 | 11.55 | 4.88  | 0.71 | 431.56 |
| SW2   | 31.11 | 39.52 | 4.98 | 2.05  | 1.36 | 79.02 |
| SW3   | 229.43 | 171.28 | 4.75 | 0.94  | 0.00 | 406.40 |
| SW4   | 24.94 | 20.06 | 3.70 | 0.89  | 0.66 | 50.25 |
| SW5   | 116.38 | 167.85 | 3.09 | 0.87  | 0.31 | 288.50 |
| SW6   | 176.92 | 91.23 | 17.77 | 1.29  | 0.20 | 287.41 |
| SW7   | 203.49 | 128.45 | 6.08 | 1.37  | 0.30 | 339.69 |
| SW8   | 22.72 | 28.24 | 1.23 | 0.00  | 0.00 | 52.19 |
| SW9   | 99.97 | 101.92 | 5.12 | 1.80  | 0.30 | 209.11 |
| SW10  | 23.67 | 14.47 | 2.26 | 0.77  | 0.00 | 41.17 |
| Outdoor | 125.70 | 168.23 | 11.10 | 4.31  | 1.57 | 335.98 |

In the indoor swimming pool water, five HAAs could be identified. The chlorinated HAAs were higher than the brominated HAAs. The order of concentrations of the brominated HAAs were BCAA, > BDCAA, > and DBAA. Concentrations of DBPs in the indoor swimming pools water are shown in boxplot in the Figure 1. Mean value of DCAA and TCAA were 118.99 µg/L and 91.62 µg/L, with standard derivations of 93.23 µg/L and 62.55 µg/L, respectively. Mean value of BCAA, BDCAA, and DBAA, were 6.05 µg/L, 1.49 µg/L, 0.38 µg/L, with standard derivation of 4.98 µg/L, 1.32 µg/L, and 0.38 µg/L, respectively. The sample labeled SW10 was collected from a diving pool, which has much less swimming activities, and therefore the concentrations of HAAs were lower than that in most swimming pool water. It is clearly that DCAA and TCAA were the dominant HAAs formed, which is comparable with other studies [2, 8, 16].

Compared to swimming pools water, drinking water contained much lower HAA concentrations. The concentrations of HAAs were within national regulation. In the swimming pool water, higher content precursors are usually identified. As well known, precursors of drinking water are natural organic matter, and the precursors of swimming pool water are both natural organic matter and body fluid analog. According to Kanan and Karenfil (2011) [2], body fluid analogs introduced by the swimmer were more reactive than the organic matter from the filling water. This is might be the reason for the higher HAA value in the swimming pool water. In addition, the precursors in the swimming pool accumulated along with the operation of the swimming pool. Operation time, number of swimmers also influences the amount of the precursors in the water [8]. Therefore, the standard deviation of every HAA species was high.

The HAA concentrations in the outdoor swimming pool are comparable with that in the indoor swimming pool water. Simard (2013) have found two times concentrations of HAAs in the outdoor swimming pool water than the indoor swimming pool water [8]. It was explained by more swimmers and more type of precursor, and higher TOC. Exposure to UV (medium-pressure lamp) was also found to increasing their HAA content [17]. Therefore, the higher UV exposure by sunlight produced more DBPs in the outdoor swimming pool water.

The different ratios of [DCAA]/[TCAA] in the indoor and outdoor swimming pool water were also observed. For outdoor pools, it has found that the average concentrations of DCAA and TCAA were practically equivalent. In the indoor swimming pools, the average concentration of TCAA was slightly lower than DCAA [8]. In our study, the results were similar. TCAA concentration in the outdoor
swimming poor water was higher than DCAA. In the indoor swimming pool water, the ratio [DCAA]/[TCAA] were higher in the water of most indoor swimming pools.

Concentrations of monochloro acetic acid, monobromo acetic acid, chloro-dibromo acetic acid in the outdoor swimming pool water was 22.7 µg/L, 1.77 µg/L, and 0.62 µg/L, which was slightly higher than that observed for the indoor swimming pool water, however, not statistically t significant. The formation of brominated DBPs takes place upon chlorination of bromide-containing waters. In such environments, bromine is the predominant disinfectant since bromide ions react with chlorine and undergo rapid oxidation to form hypobromous acid [18-20]. The brominated HAAs concentrations are much lower than the chlorinated HAAs. Low concentrations of bromide and quicker degradation than chlorinated DBPs might have led to this result. However, brominated DBPs usually have higher toxicity, which needs further evaluation.

In the previous studies, DCAA and TCAA in swimming pool are mostly studied, some of the results are listed in the Table 2.

| DCAA (µg/L) | TCAA (µg/L) | Origin             |
|------------|------------|--------------------|
| 68.3       | 180.9      | [21] Lee et al. (2010) |
| 76         | 44.9       | [22] Berg et al. (2000) |
| 419        | 420        | [23] Kim and Weisel (1998) |
| 419(HAA9)  |            | [24] Parinet et al. (2012) |
| 23         | 466.1      | [25] Manasfi et al. (2016) |
| 474(HAA9)  |            | [8] Simard et al. (2013) |
| 295.9      | 21.3       | [14] Hang et al. (2016) |
| 27.4–500   | 24.1–249.6 | [26] Tardif et al. (2016) |
| 15.4–51.7  | 39.2–83.4  | [27] Font-Ribera et al. (2016) |
| 118.99     | 91.62      | In this study       |

3.2. Cytotoxicity and genotoxicity in the swimming pool water

The cytotoxicity and genotoxicity of the swimming pool water and drinking water were calculated using the formula 1.

\[
Toxicty = \sum_i^i \frac{C_i}{EC_{50,i}} \tag{1}
\]

Where \( C_i \) stands for concentration of the \( i \)th species, and \( EC_{50,i} \) stands for the concentration which cause 50 % positive effects.

The \( EC_{50,i} \) values for all the HAA species were taken from previous article [28], which are listed in the table 3. As shown in Table 3, all the HAAs detected have cytotoxicity, lower \( EC_{50,i} \) means higher cytotoxicity. Some of the HAAs have genotoxicity, and others not. The water sample from outdoor swimming pool contain chlorodibromoacetic acid (CDBAA), therefore the calculation contained genotoxicity caused by the CDBAA. Calculation result is shown in the Figure 2.

As shown in the figure, the swimming pool water were about two orders of magnitudes higher than the drinking water in the cytotoxicity, and one to two orders of magnitudes higher in the genotoxicity. In the swimming pool water, cytotoxicity could be divided into two groups, a high toxicity group, and a lower toxicity group, with difference of about one order of magnitude. Considering the difference between \( EC_{50,i} \) values of chlorinated and brominated HAAs, the contribution of chlorinated HAA to cytotoxicity were calculated, which reveals that about half of cytotoxicity were contributed from the chlorinated HAAs in the drinking water samples, and 70 % -90 % in the swimming pool water samples. Higher contribution in the swimming pool water was caused by the high content of chlorinated HAAs in water samples. All the genotoxicity was caused by brominated HAAs. Another study revealed that swimming pool water resulted in higher mutagenicity than drinking water. It has also been reported that recreational pool waters showed higher toxicity than their tap water sources [29].
Figure 1. Concentrations of five kinds of HAAs in indoor swimming pool water.

Figure 2. Cytotoxicity and genotoxicity of the water samples.
Table 3. EC_{50,i} values of cytotoxicity and genotoxicity for every species.

|        | cytotoxicity | genotoxicity |
|--------|--------------|--------------|
| DCAA   | 7.1e-3       | /            |
| TCAA   | 2.3e-3       | /            |
| BCAA   | 7.9e-4       | 3.7e-3       |
| BDCAA  | 6.9e-4       | /            |
| DBAA   | 5.2e-4       | 1.8e-3       |
| CDBAA  | 2e-4         | 1.2e-2       |

4. Conclusions
Concentrations of HAAs in swimming pool water were investigated. The findings are:

In the same locality, HAA concentrations were significantly higher in the swimming pool water than that in the drinking water;

HAA concentrations in the swimming pool can be classified into two groups, one was much higher than another. The most abundant HAAs in the swimming pool water were DCAA and TCAA, others were much lower.

Brominated HAAs, including BCAA, BDCAA, and DBAA were observed, in descending order.

In the outdoor swimming pool water, TCAA was higher than DCAA. In the indoor swimming pool water, the DCAA concentrations were slightly higher than TCAA.

The swimming pool water had much higher cytotoxicity and genotoxicity than drinking water. The cytotoxicity of indoor swimming pool water can be classified into two groups: contribution of one group to the toxicity was much higher than another.

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