Chapter 20

Disinfection By-Products in Swimming Pool Water: Formation, Modeling, and Control

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Disinfection is practiced to minimize the occurrence and growth of microbial pathogens and thus ensure the hygienic safety of swimming pool water. However, potentially hazardous disinfection by-products (DBPs) are formed from the reaction between disinfectants and DBP precursors. This chapter discusses the formation of these DBPs in swimming pool water and modeling approaches for quantification of their levels given estimates of anthropogenic contaminant input in swimming pools. Promising DBP control technologies are also discussed. Overview of the three key aspects related to swimming pool water help readers to better understand the profile of DBPs and their control in swimming pool water.

Introduction

Swimming, as a physical activity for people of all ages, promotes fitness and has many advantages over land-based activities. It is generally considered as a health-enhancing and relatively injury-free activity; it can be a good aerobic exercise, which contributes to flexibility and muscle strength, and can have positive social aspects. Swimming pools can be found in different kinds and sizes in public areas, hotels and spas, or at private residential homes. In the U.S., more than 368 million pool visits occur each year. In Germany, around 250-300 million pool visits occur each year, averaging 3 visits per capita. In the UK, one-third of the children and around 36% of adults (>15 years of age) visit swimming pools at

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least once a week, and 55% of children (5-9 years of age) use pools at least once a month. In the U.S., there are approximately 250,000 public pools and 10,000,000 residential pools. The highest numbers of existing in-ground and above-ground pools in Europe are found in France (773,000) and Germany (625,000), followed by UK with 155,000, and Italy with 94,000 (*1*).

However, after water leaves a public water system and enters a pool, its quality is no longer regulated as drinking water in the U.S. and many other countries, and in the U.S. the Safe Drinking Water Act makes no stipulation that the tap water meets additional requirements for use in pools or spas. Instead, pools are managed at the local level, usually by state or local public health departments, and sometimes by pool managers or lifeguards. As the pools are filled with swimmers, they contain hundreds to thousands of low levels of chemicals originating from natural and anthropogenic inputs, which complicate the water chemistry. The undesirable substances (e.g., skin, hair, bodily excretions such as sweat, urine, and saliva; pathogens; and personal care products such as lotions and sunscreens) introduced by swimmers also complicate the toxicity and expose swimmers to potential biological and chemical health risks (*2*, *3*).

To conserve the positive aspects of aquatic activities, it is necessary to disinfect swimming pool water to prevent outbreaks of infectious illnesses (*4*, *5*). To date, chlorination is still the most common measure for inactivation of microbial pathogens. Chlorine is added to swimming pools as chlorine gas, calcium/sodium hypochlorite, or through electrolytic generation of sodium hypochlorite. Regardless of the method of application, once the chlorine is in the water it forms hypochlorous acid which dissociates into hydrogen and hypochlorite ions. The sum of the concentrations of hypochlorous acid, hypochlorite ion and aqueous chlorine is referred as free chlorine residual. Chlorine's popularity is not only due to lower cost, but also to its higher oxidizing potential, which provides a minimum level of chlorine residual to inhibit microbial recontamination. Other disinfectants may be used, but less is known about their ability to inactivate pathogens (*6*). For effective disinfection, adequate disinfectant levels are required. In Canada, regulations in Quebec require free chlorine in swimming pools to be between 0.8 and 2.0 mg/L, while the required minimum level for British Columbia varies with pH: 0.5 mg/L for pH 7.4-7.8, and 1.0 mg/L for pH of greater than 7.8 (*7*). In the U.S., a survey of twenty-three indoor pools showed that the median chlorine residual was 3 mg/L (*8*). In practice, chlorine residuals of 2-4 mg/L are generally recommended for chlorinated pools (*9*). However, the reaction of chlorine and chloramines with organic and inorganic precursors, such as natural organic matter (NOM), anthropogenic inputs, bromide, etc, may cause the formation of undesirable disinfection by-products (DBPs). The types and concentrations of DBPs depend on several factors, including the type and amount of disinfectant used, characteristics of the swimming pool and pool water and users' hygiene (*1*). These DBPs can be inhaled or ingested by swimmers during swimming, bathing and showering, or absorbed dermally (*10*), resulting in negative effects on human health (*11*, *12*). DBPs were observed to produce cancer in animal models and to have other toxic consequences, such as reproductive and developmental effects; in particular, users of chlorinated pools may have an elevated risk for bladder

cancer (*13*) and an increased frequency of micronuclei in peripheral blood cells (a marker of DNA damage) (*14*). These published epidemiology studies provide evidences that disinfected pool water might be genotoxic and carcinogenic to humans. Given the popularity of swimming that comes with the increased risk of exposure to pathogenic microorganisms, disinfectants and DBPs, we are only in the early stages of understanding swimming pool chemistry, human exposures, and potential health risks (*6*). Regulations must also ensure that efforts to reduce DBPs do not result in water that is impaired due to microbial contamination: This is the microbe-DBP balancing act of minimizing risks while maximizing beneficial effects.

Research has specifically focused on two classes of DBPs from chlorine-based disinfection: trihalomethanes (THMs) and haloacetic acids (HAAs) (*15–19*). THMs, including chloroform, bromodichloromethane, dibromochloromethane, and bromoform, are the best known and most intensively investigated class of DBPs and they are regulated in chlorinated drinking water in the U.S. with a maximum contaminant level at 80 μg/L. Reports on THMs in swimming pools first appeared in 1980s, and they showed a tendency of wide variations in the reported data. Fantuzzi et al. (*20*) found the THM levels ranged from 18 to 71 μg/L in five indoor swimming pools in Italy. Chu and Nieuwenhuijsen (*21*) found the THM levels averaged at 133 μg/L in 44 indoor swimming pools in the UK. HAAs, commonly referred as HAA6 including monochloroacetic acid (MCAA), monobromoacetic acid (MBAA), dichloroacetic acid (DCAA), trichloroacetic acid (TCAA), bromochloroacetic acid (BCAA), and dibromoacetic acid (DBAA), are also regulated for drinking water in the U.S., with a maximum contaminant level at 60 μg/L. There is limited information on HAA levels in swimming pools. Tang (*22*) reported the HAA level stabilized at approximately 1650 μg/L in an indoor swimming pool in the U.S.. Simard et al. (*23*) analyzed 15 indoor and 39 outdoor municipal public pools in Canada and found the HAAs were higher in outdoor pools (808 vs 413 μg/L). Wang et al. (*24*) analyzed 5 outdoor pools in the U.S. and 9 indoor pools in China and found that the HAA levels in the pools in the U.S. averaged at 1440 μg/L while in China the levels averaged at 117 μg/L due to lower chlorine residuals.

Some nitrogen-containing DBPs that were previously reported in drinking water (*25*) are also identified in swimming pool water (*26*), due to the presence of nitrogen sources, such as urine or sweat. Haloacetonitriles (HANs) were the most significant toxic compounds detected by Hansen et al. (*27*), though they were found at much lower concentrations compared to THMs and HAAs. However, it is not known how much the HANs contribute to the overall mixture toxicity. The pH plays an important role on DBP classes. For decreasing pH, THM concentrations are observed to decrease, while HAN concentrations increased. Therefore caution is warranted if the pH level in swimming pool water is lowered for THM control, since the the concentration of more toxic HANs appear to increase. Besides HANs, many other DBPs, including dichloromethylamine (CH_3NCl_2), cyanogen chloride (CNCl), trichloronitromethane (TCNM), chloral hydrate (CH), haloketones (HKs), N-nitrosodimethylamine (NDMA), chloramines and cyanogen bromide (CNBr) have also been reported in swimming pools (*28*, *29*), though at much lower concentrations compared to the predominant

THM and HAA species. To date, their complete chemical and toxicological characterizations are not available.

In this chapter, we examine three key aspects related to swimming pool water.

Formation

DBP precursors lead to the formation of DBPs upon chlorination. Research on DBP precursors in swimming pool water is of keen interest by scientists (*30*, *31*). One option to ensure the safety of public drinking water systems is to improve the quality of source water through pollution prevention and precursor removal. However, a source reduction approach is challenging for swimming pools, because the swimmers themselves are the largest source of organic/inorganic precursors for DBP formation. The incidental anthropogenic contaminant release as a result of human excreta such as urine and sweat, either accidentally or on purpose, is difficult to quantify (*32*, *33*). And its impact on DBP formation in swimming pools is unclear. In this section, we discussed our research on investigating the formation of DBPs and the contributions from various precursors in a sample swimming pool.

Modeling

Models for DBP levels in swimming pool water are needed, aiming at identifying the significance of diverse operational and water quality parameters controlling the formation of DBPs or at investigating the kinetics for their formation. It may consist of empirical and mechanistic relationships of water quality and operational parameters with the prevailing levels of DBPs at various stages of a same single pool. In this section, we discussed our research on developing predictive models for DBPs based on data obtained from field and laboratory-scaled studies.

Control

Swimming pool water treatment in general includes flocculation, sand filtration, and subsequent disinfection with chlorine. The continuous chlorination and input of anthropogenic releases by bathers in combination with recirculation of the pool water may lead to an accumulation of DBPs in the water. Better technologies are needed to solve this issue. In this section, we discussed our research on potential technologies on DBP control from swimming pool water.

By giving an overview of our research efforts on the three topics in this chapter, readers can better understand the profile of DBPs and their precursors in swimming pool water.

Materials and Methods

Reagents

All chemicals and standard solutions, unless noted otherwise, were analytical grade purchased from Sigma-Aldrich.

Analysis of Dissolved Organic Carbon (DOC), Ammonia Nitrogen (NH_3-N), UV Absorbance at 254 nm (UV_{254}), and Chlorine

DOC was measured with a total organic carbon (TOC) analyzer (O.I. Analytical Model 1010, Maryland, USA). NH_3-N was measured using a ammonia-selective electrode method according to Standard Method 4500-NH_3 (*34*). UV_{254} was measured using an Agilent 8453 UV-Visible Spectrophotometer (Agilent technologies, California, USA) with a 10 mm quartz cuvette. Free chlorine and total chlorine were measured immediately using the *N,N*-diethyl-*p*-phenylenediamine (DPD) method with a DR/890 portable colorimeter (Hach Company, Colorado, USA).

Analysis of THMs and HANs

Table 1 shows a list of investigated compounds. Residual chlorine in THM and HAN samples was quenched by adding 5 mg sodium sulfite to 40 mL borosilicate glass vials before they were filled head-space-free with the samples. The samples were stored at 4°C until analysis by a gas chromatograph (GC) (HP 6890 Series GC system, Hewlett Packard) with mass spectrometer (5973 Mass selective detector, Hewlett Packard). A DB-1 capillary column (30 m × 0.32 mm i.d., 1.0 μm film thickness) was used. The temperature ramping program was as follows: Initial at 35°C for 22 minutes, ramp to 145°C at 10°C/min and hold for 2 minutes. This method was also used for the detection of chloral hydrate (CH), trichloronitromethane (TCNM), dichloropropanone (DCP), and trichloropropanone (TCP). For more information refer to EPA 551.1 method.

Analysis of HAAs

For the analysis of HAAs, a modified version of the EPA 552.3 method was used. Sulfuric acid, sodium sulfate, surrogate standard (2-bromobutanoic acid) and methyl-tert-butyl ether (MtBE) was added to the samples which then were hand shaked for two minutes to extract HAAs. The MtBE phase was transferred to a test tube and acidified methanol was added. The samples were placed in a water bath at 50°C for 2 h to methylate the HAAs and then back-extracted with sodium sulfate solution to remove excess methanol. The MtBE phase was transferred to a GC vial and analyzed by a GC (HP 6890 Series GC system, Hewlett Packard) equipped with a DB-1701 capillary column (30 m × 0.25 mm i.d., 0.25 μm film thickness). The temperature ramping program was as follows: Initial at 35°C for 10 minutes, ramp to 75°C at 5°C/min, hold for 16 minutes, ramp to 200°C at 25°C/min and hold for 5 minutes.

DBP Formation Potential (DBPFP) test

A DBPFP test was started by applying an initial chlorine dose to the phosphate buffered samples, which had been pre-diluted to accommodate 3-day free chlorine demands (*35*). The incubation process took 3 days at 25°C in the dark. The DPD method was used to measure the free chlorine and total chlorine. Samples were

then quenched to eliminate chlorine residuals and analyzed for DBPs based on the above protocols. It is noted that five kinds of anthropogenic contaminants, i.e. sweat, saliva, skin wash, hair wash, and urine, were also sampled for the DBPFP tests. The anthropogenic inputs were obtained from a 26-yr-old college male, with sampling protocols approved by the Pennsylvania State University Institutional Review Board and a written informed consent obtained from the participant.

Table 1. List of Investigated Disinfection By-Products

	Compound	*Abbreviation*	*Chemical formula*
THMs	Chloroform	TCM	$CHCl_3$
	Bromodichloromethane	BDCM	$CHBrCl_2$
	Dibromochloromethane	DBCM	$CHBr_2Cl$
	Bromoform	TBM	$CHBr_3$
HAAs	Chloroacetic acid	MCAA	$CH_2ClCOOH$
	Bromoacetic acid	MBAA	$CH_2BrCOOH$
	Dichloroacetic acid	DCAA	$CHCl_2COOH$
	Trichloroacetic acid	TCAA	CCl_3COOH
	Bromochloroacetic acid	BCAA	$CHBrClCOOH$
	Dibromoacetic acid	DBAA	$CHBr_2COOH$
HANs	Dichloroacetonitrile	DCAN	$CHCl_2CN$
	Trichloroacetonitrile	TCAN	CCl_3CN
	Bromochloroacetonitrile	BCAN	$CHBrClCN$
	Dibromoacetonitrile	DBAN	$CHBr_2CN$
Others	Dichloropropanone	DCP	$CHCl_2COCH_3$
	Trichloropropanone	TCP	CCl_3COCH_3
	Trichloronitromethane	TCNM	CCl_3NO_2
	Chloral hydrate	CH	$C_2H_3Cl_3O_2$

Formation

The Swimming Pool under Investigation

The DBP formation in a swimming pool was investigated. The pool, with a volume of 440 m^3, was located in Capitol Union Building at The Pennsylvania State University, Middletown, Pennsylvania, USA. The pool was completely drained, and refilled with fresh water in the second week of June in 2009. Since then, the pool water was periodically sampled and analyzed for DBPs during a

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1.5-yr investigation period. During the operation of the swimming pool, water was filtered and chlorinated through recirculation of the pool water. Some water was lost during back-washing of the filters and by evaporation, therefore fresh water was added to maintain a constant water level at a rate of approximately 1.8 m^3 weekly. Some DBPs might be adsorbed to suspended solids in the pool and removed with the filtration; however, for this study that process and the loss of water through evaporation were neglected, since they account for a negligible fraction of the pool water. The pool was open year-around to students, faculty and staff of the university, members, and guests, i.e. walk-ins of the local community. Table 2 shows the year-around user statistics of the swimming pool. These data show that the pool accepted 1441 users per month or 48 users per day on average. As the typical pool hours were from 9 am to 9 pm in work days except national holidays, the pool accepted an average of 4 users per hour.

Table 2. Year-around User Statistics of the Swimming Pool

	Members	*Students*	*Faculty/Staff*	*Guests**	*Total visits per month*
January	398	255	91	1579	2323
Feburary	353	291	81	1006	1732
March	422	312	70	366	1536
April	346	275	65	665	1351
May	360	140	83	432	1015
June	307	141	78	388	1114
July	315	105	122	892	1434
August	290	164	111	224	789
September	396	231	80	651	1358
October	356	243	133	1148	1879
November	326	169	98	1326	1955
December	325	135	60	896	1416

* Guests include the pool users of swim lessons, swim teams, water fitness classes, and walk-ins of local community.

DBP Levels in the Swimming Pool

Figure 1 shows the profiles of THM and HAA concentrations in the swimming pool following the water change over a 1.5-yr period. After the pool water was completely drained and refilled with fresh water, the THM and HAA concentrations were 81 and 87 μg/L, respectively. These two numbers were similar to those of finished water (80 μg/L for THMs and 60 μg/L for HAAs)

from a typical surface water treatment plant. As the water age increased and more swimmers were admitted, the THM levels were relatively stable, being averaged at 62 µg/L with a standard deviation of 35 µg/L. However, the HAA concentrations substantially increased. Two months following water change, 940 µg/L HAAs were observed in the pool. During the 6th and 8th months following water change, the HAA level appeared to reach a plateau and fluctuated around 1600 µg/L. From the 8th to 12th month, the HAA level consistently declined to 780 µg/L, and after the 12th month, the HAA concentration steadily increased again to the 1600 µg/L level when the 1.5-yr investigation period was close to the end. It is noticeable that the decline in HAA concentrations from January to June could be attributed to the decrease of the number of pool users during the same period, as the monthly number of pool users decreased from 2223 in January to 1114 in June. Figure 2 also shows that a positive correlation between the measured HAA levels in the pool and the numbers of pool users, and the R^2 value was 0.70.

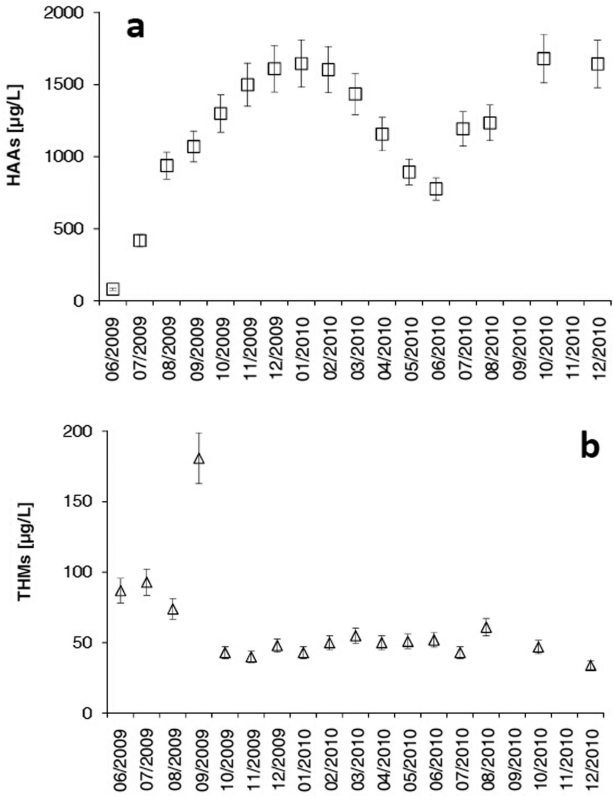

Figure 1. THM and HAA levels in an indoor swimming pool after water change.

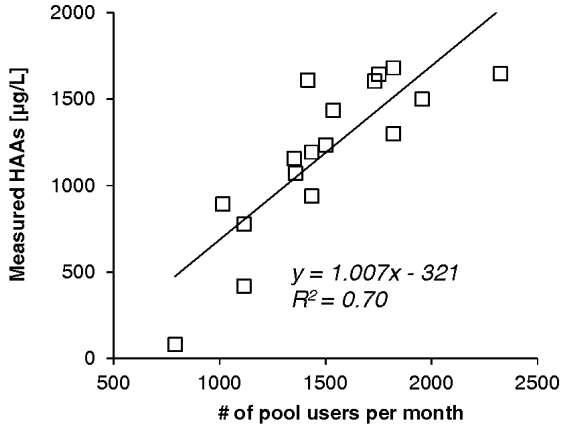

Figure 2. Correlation between the measured HAAs and the numbers of pool users.

The predominant species of HAAs, THMs, HANs, and HKs in the swimming pool water were DCAA and TCAA, TCM, DCAN, and TCP, respectively. Table 3 shows their concentrations in the pool water during the 1.5-yr investigation period since water change. DCAA was found to be in a higher concentration than TCAA. Because DCAA and TCAA are believed to be produced as a result of different mechanistic pathways (*36*), the ratio of DCAA/TCAA implies that the formation of DCAA is favoured in the swimming pool water, possibly due to the presence of more precursors for DCAA in pool water. HANs and HKs in swimming pools were investigated by fewer studies than those of THMs and HAAs. Lee et al. (*37*) reported DCAN in the chlorinated pools as 3.9 ± 3.3 µg/L, while TCAN was not reported. In this research, the DCAN in the swimming pool water was 1.3 ± 0.7 µg/L. The HAN concentrations were much lower than THM and HAA concentrations, possibly due to their decomposition to form HAAs at pH conditions higher than 7.0 (*27*). As the DBP formation is also attributable to precursors for DBPs, the profiles of individual species need to be reviewed to explore the contribution from their precursors.

DBP Precursors in the Swimming Pool

The widely accepted precursors for DBP formation in drinking water is the natural organic matter (NOM). However, this phenomenon is not completely applicable to swimming pools. Researchers have associated anthropogenic inputs, i.e. sweat and urine, by swimmers with main sources of DBP precursors (*38–41*). It was estimated that a swimmer introduces 25-80 mL of urine and 200-1000 mL of sweat into swimming pool water each swim session (*42*, *43*). As anthropogenic contaminants are continuously brought in by swimmers and pools are continuously exposed to disinfectant, the disinfection process resembles

a DBPFP test, which maintains a sufficient free chlorine residual and a long reaction time to convert precursors to DBPs. To further explore their impact on DBP formation in swimming pools, five kinds of anthropogenic contaminants, i.e. sweat, saliva, skin wash, hair wash, and urine, were analyzed for DBPFPs

Table 3. Concentrations of Individual Species and the Ratio of DCAA/TCAA

Month No.	*DCAA*	*TCAA*	*TCM*	*DCAN*	*TCP*	*DCAA/ TCAA**
1	51	18	87	0.0	0.0	2.8
2	276	65	93	0.8	2.3	4.2
3	636	186	74	1.6	2.1	3.4
4	750	201	181	0.9	1.5	3.7
5	975	290	43	2.3	2.0	3.4
6	1100	326	40	2.7	2.5	3.4
7	1095	366	48	1.6	2.5	3.0
8	1119	381	43	1.8	1.8	2.9
9	1074	489	50	1.5	1.5	2.2
10	942	444	55	1.0	1.3	2.1
11	715	400	50	2.2	1.1	1.8
12	493	362	51	1.4	1.9	1.4
13	504	249	52	0.8	1.6	2.0
14	849	321	43	0.6	1.2	2.6
15	861	345	61	0.4	1.6	2.5
17	1167	480	47	1.6	2.1	2.4
19	1116	504	34	1.3	2.9	2.2

* Data in this ratio column are unitless. Data in the DCAA, TCAA, TCM, DCAN, and TCP columns are concentrations represented. in μg/L.

Table 4 shows the DBPFPs of the anthropogenic input samples. All the anthropogenic contaminants were found to contribute to the DBP formation, which was consistent with the results reported by Kim et al. (*39*). The HAA yields of chlorinated sweat, saliva, skin wash, hair wash, and urine samples were 300, 390, 380, 160, and 160 μg/mg C, respectively, while the THM yields of the corresponding samples were 67, 130, 96, 50, and 74 μg/mg C, respectively. The relative abundance of HAAs and THMs was presented by the ratio of HAA/THM. These anthropogenic contaminants contributed to more HAAs than THMs, with the HAA/THM ratio in the range of 2.1 and 4.5. It is noted that urine had the

lowest HAA/THM ratio of the 5 contaminants. For the 3-day Cl_2 demand per DOC, Kanan and Karanfil (*8*) reported 2-8 mg/L Cl_2 demand for 1 mg DOC of filling water NOM and 17-25 mg/L Cl_2 demand for 1 mg DOC of body fluid analogue. A slight higher number, 21-40 mg/L Cl_2 demand for 1 mg DOC of real anthropogenic contaminants, was found in this research. Table 4 also presents the yields of HAAs and THMs by normalizing HAAFP and THMFP based on DOC, to compare different anthropogenic contaminants regardless of their dilution ratios. Sweat was observed to have an HAA yield of 300 μg/mg C. Urine and hair were observed to have relatively low HAA yields (160 μg/mg C) compared to three other anthropogenic contaminants. The THM yields of sweat and urine were 67 and 74 μg/mg C, respectively. These DBP yield data will be used in the later modeling section.

Table 4. DBPFPs and DBP Yields of Anthropogenic Input Samples

	Sweat[1]	*Saliva*[1]	*Skin wash*[1]	*Hair wash*[1]	*Urine*[2]
3-day Cl_2 demand [mg Cl_2/L]	170	170	210	230	410
THMFP [μg/L]	430	1010	870	560	740
HAAFP [μg/L]	1930	3050	3420	1750	1560
3-day Cl_2 demand per DOC [mg Cl_2/mg C]	27	22	23	21	41
THM yield [μg/mg C]	67	130	96	50	74
HAA yield [μg/mg C]	300	390	380	160	160
HAA/THM	4.5	3.0	3.9	3.1	2.1

[1] A dilution ratio of 1:25 was applied to accommodate free Cl_2 demand in DBPFP test. [2] A dilution ratio of 1:33 was applied to accommodate free Cl_2 demand in DBPFP test. Data presented are average values of duplicate sets.

Interpretation of DBP Profile in Swimming Pools

Swimming pool water is found to have high DBP levels, especially in forms of HAAs. THMs, however, did not show an increase trend with water age but were relatively constant in the pool water. We have summarized four factors that lead to this specific DBP profile in swimming pools:

Firstly, the long water retention time in swimming pools is special. As the pool water is continuously exposed to disinfectants, all DBP precursors have a potential to be driven to DBPs. This tends to result in high DBP levels.

Secondly, continuous introduction of anthropogenic contaminants leads to high DBP levels. With an R^2 of 0.70, a positive correlation between the HAA levels and the numbers of pool users exists (Figure 2).

Thirdly, discrepancies in the chemical loss rates could lead to the high HAA but low THM profile. Benoit and Jackson (*44*) observed stable THM levels in 25 whirlpool spas affected by heat, aeration, and agitation. Although these factors, i.e. the degree of water agitation by pool users, were difficult to measure, the THM loss rate appeared to be faster than the HAA loss rate because of much higher Henry's law constants for THMs. The loss rate includes the effects of evaporation, adsorption, degradation, and other incurred processes.

Finally, the anthropogenic inputs of swimming pools consist of more HAA precursors than THM precursors, as data of this research presents (Table 4). It is obvious that reduction of anthropogenic contaminants will lead to reduction of DBPs. This research demonstrated the actual effect of anthropogenic contaminants on the level of DBPs.

Modeling

Model Development

In recent years, a particular interest has been grown on the development of models to estimate the formation and fate of DBPs. Several predictive models have been reported (*45*). These models used different types of explanatory variables for variety of applications in drinking water industry. The DBP profile in a swimming pool system can be conceptualized based on mass balance and decay kinetics (*46*, *47*). For chlorination of pool water, a comprehensive model for DBPs should consider variation of DBP precursors, i.e. sweat and urine from anthropogenic inputs. Efforts should be focused on the gaps between specific models and their actual applications.

The concentration data used to model DBP formation in swimming pool water were obtained from the study by Tang (*22*) where samples were collected from an indoor pool once a month over a 1.5-yr period. The DBP model was developed based on four assumptions: (1) All DBP precursors from anthropogenic inputs have been converted to DBPs under the chlorination scenarios of swimming pools; (2) There is no water change during the entire modeling period and the amount of make-up water is negligible, because a 1.8 m^3 weekly make-up water versus the pool volume of 440 m^3 makes the influence of dilution within the range of variability for DBP analysis. (3) The DBP loss due to evaporation, degradation, dilution, and adsorption follows first-order kinetics (*48*); and (4) The DBP loss rates maintain constant since water temperature of the pool is stable. Equation 1 shows the mass balance of DBPs in swimming pools. It can be reformed to represent the computational algorithm on the basis of the contribution from daily anthropogenic inputs to DBPs (Equation 2 and 3).

Equation 1.

$$DBP\ Accumulation = DBP\ In + DBP\ Production - DBP\ Out$$

Equation 2.

$$DBP_i = DBP_{new,i} + DBP_{i-1} \times e^{-k}$$

Where
DBP_i = Accumulated DBP at *Day i*, *μg DBP/L*;
$DBP_{new,i}$ = New *DBP In* due to the anthropogenic input at *Day i*, *μg DBP/L*;
DBP_{i-1} = Accumulated DBP at the day before *Day i*, *μg DBP/L*;
$DBP_{i-1} \times e^{-k}$ = Remaining DBP after 1-day first-order decrease, *μg DBP/L*;
k = DBP loss coefficient, *day*$^{-1}$.

Equation 3.

$$DBP_{new,i} = [N_i(Y_S C_S + Y_U C_U)]/V$$

Where
N_i = Number of pool users at *Day i*, *persons*;
Y_S = DBP yield for sweat, *μg DBP/mg C*;
C_S = Sweat carbon released per person, *mg C/person*;
Y_U = DBP yield for urine, *μg DBP/mg C*;
C_U = Urine carbon released per person, *mg C/person*;
V = Volume of the swimming pool, *Liter*.

It is noted that C_S, C_U, and k were difficult to measure and show variances from one swimming pool to another. They need to be calibrated by computer-based regression programs. Shuffled complex evolution (SCE), a global optimization method developed by Duan et al. (*49*), was used to calibrate these difficult-to-measure parameters of the swimming pool. The calibration process used 48 (approximately 70%) of data sets (12 months × 4 classes of DBPs) based on statistical requirements. The C_S, C_U, and k were assigned an initial value, respectively, to start the calibration. If the values were perfect, there would be an approximate prediction but not necessarily an exact match. To take care of small discrepancies, small adjustments were made to these parameters until the predicted data matches the measured ones. The SCE algorithm ensured that the root mean squared value between the predicted data and the measured ones was the lowest. The remaining 30% of data sets were used for validation. The coefficient of determination (R^2) was used to verify the regressions between the predicted DBP data the measured ones in the pool water.

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Model Performance

The obtained parameters of the DBP model for the swimming pool after calibration were as follows:

C_S , Sweat carbon released per person = 1000 *mg C/person*;
C_U , Urine carbon released per person = 480 *mg C/person*;
k_{HAA} , HAA loss coefficient = 0.035 day^{-1};
k_{THM} , THM loss coefficient = 0.23 day^{-1};

Figure 3 presents the calibration and validation results of comparing the predicted and measured data without distinguishing different DBP classes. The 45° line the figure depicted the predicted DBP data that were precisely equal to the measured ones. As shown in Figure 3a, with an R^2 of 0.97, the predicted data showed a good fit to the measured ones. The validation process used the remaining 30% of data sets, and was still able to show a good fit to the measured data, with an R^2 of 0.96 (Figure 3b). Therefore, the model calibration led to sound results for the coefficients of the swimming pool system.

Figure 4 presents the predicted profiles of HAAs and THMs in comparison to the actual ones. The patterns were developed as a result of a combined effect of DBP formation and loss rates as described in the previous section. The predicted HAA curve captured the rising trends and descending trends very well. The predicted THM pattern also gave a quantitatively correct range. Because of the high loss rates, THM variations with water age were not as significant as those of HAAs. An extraordinary THM observation of 181 µg/L at the 104th day could be attributed to a peak hourly loading of swimmers during the sampling.

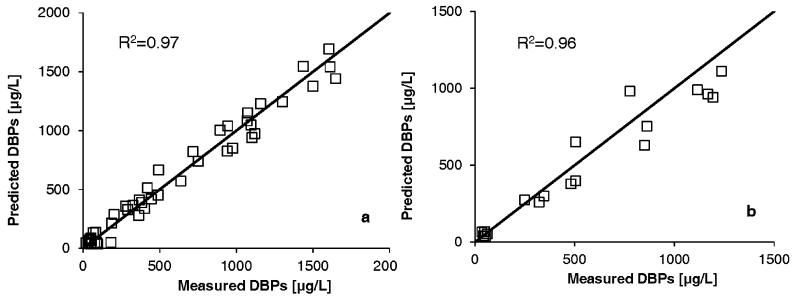

Figure 3. Calibration and validation results. (a) calibration based on 70% data; (b) validation based on 30% data.

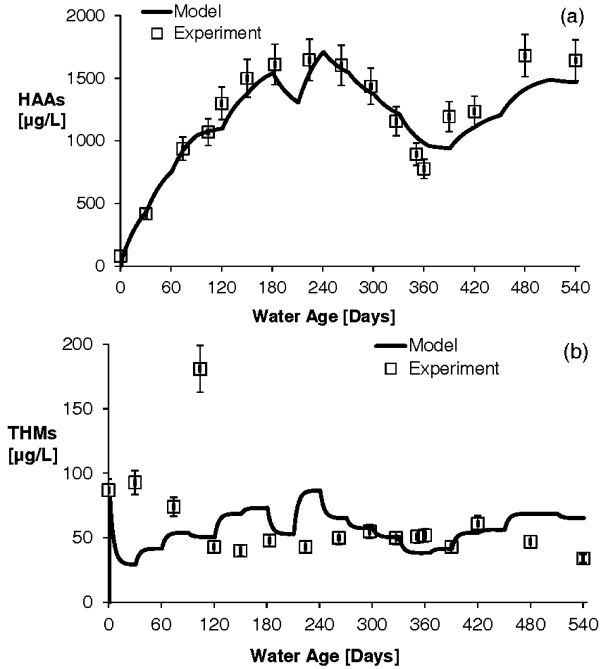

Figure 4. Predicted DBP profiles as a function of water age. (a) HAAs; (b) THMs.

Model Implications

Computer-based modeling is a useful tool for a number of tasks performed by water and wastewater professionals. Because of the high R^2 values of both calibration and validation, the DBP model developed based on mass balance and first-order kinetics is competent to predict DBPs in swimming pools. The model basically attributes the formation of DBPs to the continuous introduction of anthropogenic contaminants or the number of pool users.

Anthropogenic inputs represent the major cause of high DBP levels in swimming pool water. The model suggests 1000 mg carbon from sweat per person and 480 mg carbon from urine per person were released to the pool every day. According to Judd and Black (*46*), body fluids had a carbon content of approximately 6000 mg/L. The model implies that each pool user released approximately 170 mL sweat and 80 mL urine to the pool. These values were in the range reported by Gunkel and Jessen (*42*) and Erdinger et al. (*43*), who estimated that a swimmer introduces in water 25-80 mL/h of urine and 200-1000 mL/h of sweat. The model also presents an approach to estimate the DBP loss coefficients. It is believed that water agitation affects the DBP levels in swimming pool water. However, the degree of water agitation is difficult to measure. According to the model, the HAA loss coefficient was 0.035 per day while the THM loss coefficient was 0.23 per day in the investigated pool. Lower coefficient implies longer half life and thus higher concentration.

Control

Control Factors

DBPs in drinking water are typically controlled by limiting both the amount of disinfectants and the amount of NOM. In swimming pool water, the DBP formation may be limited by three factors: disinfectant residual, the amount of organic material in the water, and the recirculation of the pool water (*50*).

The amount of chlorine entering the pool water can be easily controlled, as the chlorine, in forms of granular powder or tablets, can be added by hand or by a mechanical dosing system. Proper dosing ensures chlorine residual kept at a minimum and still effectively eradicate pathogenic microorganisms, in the meanwhile keeps DBP concentrations at low levels. This is the microbe-DBP balancing act of minimizing risks while maximizing beneficial effects.

The amount of organic material can be controlled by reducing the input of DBP precursors (NOM and anthropogenic input). The anthropogenic input can be reduced considerably by the behavior of swimmers before and during swimming. For example, showering and using toilet facilities, washing off sunscreen lotions, and applying water-tight diapers can reduce the anthropogenic load and help to reduce DBP formation. Keuten et al. (*51*) found that the duration of shower has the largest influence on the removal of contaminants and most of contaminants can be removed within a 60-second shower. Although a showering protocol is an important first step to inform pool users about their personal influence on the pool water quality, showering protocols alone might not be enough. The aquatic staff also needs to be aware of the necessity of pre-swim showering. And finally, supportive policies (e.g., maintaining clean, well-stock bathroom facilities that not only encourage showering but also toileting) should be established, implemented, and enforced.

The recirculation of the pool water leads to an accumulation of DBPs in the water. From the technological point of view, the treatment efficiency to reduce DBP precursors and already-formed DBPs can be improved by more efficient filtration and oxidation. This includes tight membrane (e.g. nano or reverse osmosis membrane) filtration, carbon filtration, biological filtration, and advanced oxidation (*52*). Removal of the low-molecular-weight fraction would be advised because it contains a large part of the toxicity according to Zwiener et al. (*1*). Their toxicity data also suggest keeping bromide and iodide concentrations in pool water low and not using bromine because brominated and iodinated DBPs have been found to be even more toxic than chlorinated DBPs. It is also beneficial to increase air circulation in indoor pool settings to reduce the levels of volatile DBPs.

Filtration

Typical swimming pool water treatment includes sand filtration and disinfection with chlorine. Hansen et al. (*27*) reported a few swimming pools in Denmark use drum filters based on a weaved microsieve (cutoff 10 or 20 μm) which removes the particles from the pool water fast by washing the filter. Although most swimming pools remove particles through a filtration unit, the

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ACS Symposium Series; American Chemical Society: Washington, DC, 2015.

filtration units are not designed to remove organic matter. Control of DBPs and their organic precursors using adsorbent, i.e. GAC, could be an effective approach.

Two types of GAC were investigated in bench-scale filters: virgin GAC supplied by Waterlink (USA) and biologically activated carbon (BAC) collected at a local drinking water treatment facility. The schematic setup of the GAC filters is depicted in Figure 5. Swimming pool water was directly added to the filter columns loaded with 40 mL of GAC and the flow rates were adjusted to 2 mL/min to have an empty bed contact time (EBCT) of 20 minutes.

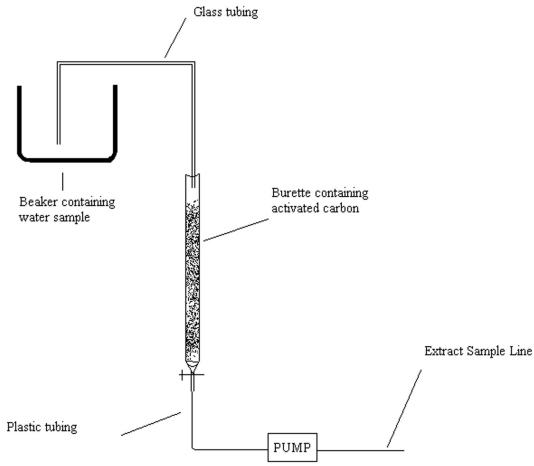

Figure 5. Schematic setup of bench-scale GAC filters.

The bench-scale GAC filter was operated for 400 hours. Results showed that TCAA was completely removed during the beginning of the experiment and the effluent concentration of DCAA steadily increased. Before the GAC column reached saturation, biological degradation of DCAA was observed after 128 hours of operation. This is shown in Figure 6 at the highest peak of the curve. At this point, the total HAA was 566 μg/L, and there was 48% reduction in total HAAs, a 9% reduction in DCAA and 99% reduction in TCAA. The results imply that GAC was effective in removing TCAA while the adsorption capacity for DCAA was relatively low.

The bench-scale BAC filter was initially operated for 2 hours filtering deionized water. Then it started filtering swimming pool water for 480 hours at the same settings with the GAC filter. Since BAC has almost no adsorption capacity, it became saturated almost immediately for TCAA (Figure 7). There was approximately 90% removal of DCAA throughout the entire experiment. Opposite to what was observed in the bench-scale GAC filter, this occurred because the bacteria capable of degrading DCAA were already established on the BAC. The TCAA degrading bacteria, however, took approximately 30 hours to

be established, and then the TCAA concentration in the effluent rapidly decreased throughout the experiment. In the end of the experiment, approximately 70% of TCAA was removed.

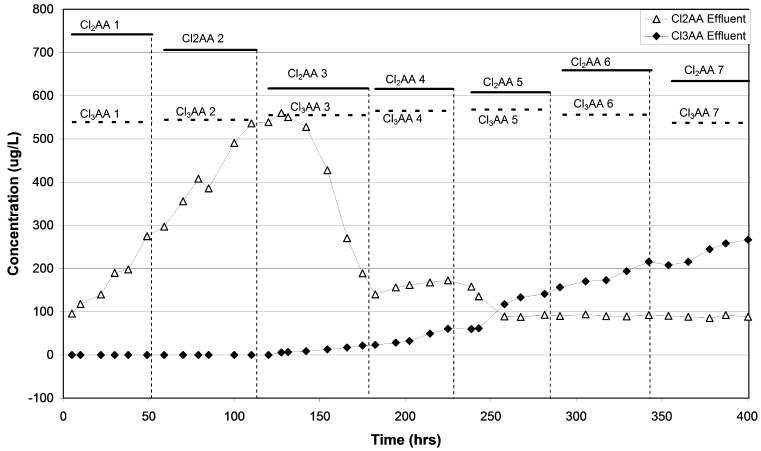

Figure 6. HAA removal from swimming pool water by a bench-scale GAC filter.

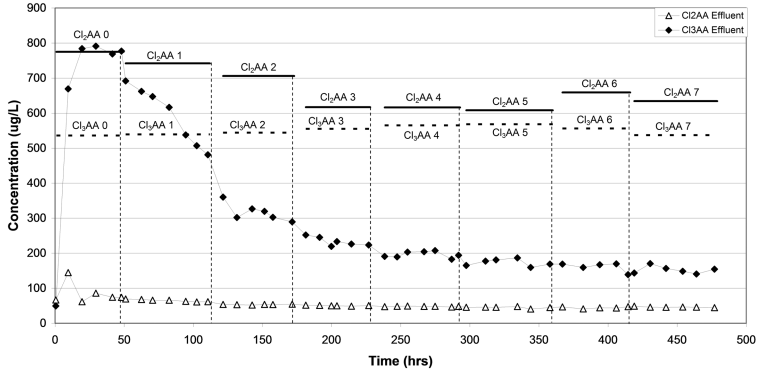

Figure 7. HAA removal from swimming pool water by a bench-scale BAC filter.

These experiments show that GAC is able to adsorb TCAA and BAC can remove DCAA efficiently at the beginning. Once TCAA degrading bacteria become established, BAC can efficiently remove HAAs from swimming pool water and outperforms GAC. The study also implies that, given enough time, bacteria capable of degrading HAAs will develop on the surface of GAC, converting it to BAC and reducing the need to replace the GAC. A disadvantage associated with both studies is that the filter effluent had zero chlorine residual. This may lead to problems when used on an actual swimming pool, because the

pool water needs to be continuously chlorinated to keep a residual typically in a range of 2-4 mg/L. Elevated costs for the use of additional chlorine could be expected.

Besides adsorption and biodegradation during the filtration process for DBP control in swimming pool water, other filtration technologies, i.e. membrane, zero-valent iron, etc, are also being investigated by scientists. Cost-effective HAA removal technologies for swimming pool water represent a promising direction of future research.

Conclusions

In this chapter, three key aspects related to swimming pool water were investigated:

Formation

We measured DBP formation in a swimming pool over a 1.5-yr period since water change. We found the HAA levels could go very high in the swimming pool, while THM levels remained relatively constant. By exploring the DBPFPs from anthropogenic contaminants, we found the anthropogenic inputs to swimming pools contributed to high HAAs and significantly impacted certain DBP species in the water.

Modeling

The DBP profile in swimming pools can be modeled and predicted. The DBP model was developed using the DBP data of the investigated swimming pool and a computer-based derivatization method based on mass balance and first-order kinetics. The predictive DBP model was competent to provide quantitatively acceptable DBP data. The model attributes the formation of DBPs to the continuous introduction of anthropogenic contaminants and the number of pool users, which helps the interpretation of the special DBP profile in swimming pool water.

Control

Of the three limiting factors to DBP formation in swimming pools, reducing the input of anthropogenic contaminants is critical. With the purpose of reducing DBP precursors and already-formed DBPs, filtration technologies using GAC and BAC were investigated by bench-scale experiments. GAC adsorption and BAC biodegradation were both found to be effective options on certain HAA species removal.

Future research efforts should be focused on three aspects: (1) monitoring and assessment of other emerging/remerging DBPs in swimming pools; (2) the potential health impact of swimming pool exposure to high level HAAs; and (3) cost-effective HAA removal technologies for swimming pools.

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