



## Review article

## Regulation, formation, exposure, and treatment of disinfection by-products (DBPs) in swimming pool waters: A critical review



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## ABSTRACT

The microbial safety of swimming pool waters (SPWs) becomes increasingly important with the popularity of swimming activities. Disinfection aiming at killing microbes in SPWs produces disinfection by-products (DBPs), which has attracted considerable public attentions due to their high frequency of occurrence, considerable concentrations and potent toxicity. We reviewed the latest research progress within the last four decades on the regulation, formation, exposure, and treatment of DBPs in the context of SPWs. This paper specifically discussed DBP regulations in different regions, formation mechanisms related with disinfectants, precursors and other various conditions, human exposure assessment reflected by biomarkers or epidemiological evidence, and the control and treatment of DBPs. Compared to drinking water with natural organic matter as the main organic precursor of DBPs, the additional human inputs (i.e., body fluids and personal care products) to SPWs make the water matrix more complicated and lead to the formation of more types and greater concentrations of DBPs. Dermal absorption and inhalation are two main exposure pathways for trihalomethanes while ingestion for haloacetic acids, reflected by DBP occurrence in human matrices including exhaled air, urine, blood, and plasma. Studies show that membrane filtration, advanced oxidation processes, biodegradation, thermal degradation, chemical reduction, and some hybrid processes are the potential DBP treatment technologies. The removal efficiency, possible mechanisms and future challenges of these DBP treatment methods are summarized in this review, which may facilitate their full-scale applications and provide potential directions for further research extension.

## 1. Introduction

Swimming is a popular activity for exercise and entertainment across the world. During pool activities, human substances (e.g., skin particles, sweat, urine, fecal, and hair) and personal care products (PCPs, e.g., sunscreen, body lotion, shampoos, and other cosmetics) can contaminate the swimming pool waters (SPWs). For outdoor pools, additional pollutants come from leaves and dust in the surrounding environment or from the rain water. These contaminants may carry pathogens, including bacteria, viruses and protozoa, which may result

in some diseases or symptoms such as gastroenteritis and dermatitis (Craun et al., 2005). Centres for Disease Control and Prevention (CDC) and Environmental Protection Agency (EPA) have conducted a 30-year surveillance program on waterborne outbreaks in the United States and the associated causes. During the monitoring period from 1971 to 2000, > 11,000 cases of illness and > 70 waterborne outbreaks were associated with swimming pools (Craun et al., 2005).

Disinfection plays a critical role in killing microbes and preventing the spreading of infectious diseases in SPWs. Nevertheless, disinfectants can react with water constituents of natural and anthropogenic origin to

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yield toxic disinfection by-products (DBPs) (Fischer et al., 2012). Researchers became aware of DBPs since early 1970 and > 600 DBPs have been reported in drinking waters (DWs) in the past 40 years (Richardson et al., 2007). Trihalomethanes (THMs) were the first identified DBP group with a concentration level of low to mid  $\mu\text{g/L}$  in DWs in 1974 (Rook, 1974), which have been linked with diseases such as bladder and colon cancer, asthma, irritation to the eyes and mucous membrane, and reproductive function (Aggazzotti et al., 2004; Erdinger et al., 1998; Goodman and Hays, 2008; Hamidin et al., 2008). Five years later, U.S. EPA constrained THMs with a maximum contaminant level (MCL) of  $100 \mu\text{g/L}$  in DWs (EPA, 1979). In 1998, the MCL of THMs was lowered to  $80 \mu\text{g/L}$  (EPA, 1998). At the same time, haloacetic acids (HAAs), another prevalent class of DBPs, were regulated with a MCL of  $60 \mu\text{g/L}$  for the sum of chloro-, bromo-, dichloro-, dibromo-, and trichloroacetic acids. THMs and HAAs account for nearly 25% of all halogenated DBPs (Krasner et al., 2006), of which chloroform, bromodichloromethane, dichloroacetic acid, and trichloroacetic acid have been classified as contaminants possibly carcinogenic to humans (Group 2B) by International Agency for Research on Cancer (IARC, 1999, 2004, 2014). Some emerging DBPs, though not yet regulated, can be potentially more toxic compared to THMs and HAAs, e.g., halonitromethanes (HNMs), haloamides, and iodo-DBPs (Richardson et al., 2007).

Public attention of DBPs in SPWs is not as much as that in DWs according to the number of publications in the past 20 years (Fig. 1). However, it is becoming an important topic due to the increasing popularity of swimming activities and its associated health side-effects. In addition to natural organic matter (NOM) from the source water, body fluids and PCPs released from the swimmers make the water matrix more complicated and possess a higher DBP formation potential. For example, urea, often found in SPWs at high concentrations, can react with free chlorine to form chloramine (Blatchley III and Cheng, 2010; Schmalz et al., 2011), which may lead to eye and upper respiratory tract irritation, biomarker changes in the lung, and development of asthma (Bernard et al., 2006; Massin et al., 1998).

The DBP concentrations in SPWs are often much or even several orders of magnitude higher than the MCLs in DWs or SPWs (Kanan et al., 2015; Parinet et al., 2012; Simard et al., 2013; Yang et al., 2016). The traditional SPW treatment train based on flocculation, sand filtration, and chlorination is inadequate to meet the current water quality standard. It is worth noting that DBP exposure during swimming can be much greater than that via DWs (Villanueva et al., 2006; Villanueva et al., 2007b), particularly for the more volatile compounds such as THMs. For example, only 1% of THM uptake during pregnancy was from DWs via oral ingestion, and 23% from swimming activities by inhalation and dermal absorption (Villanueva et al., 2007b). In view of

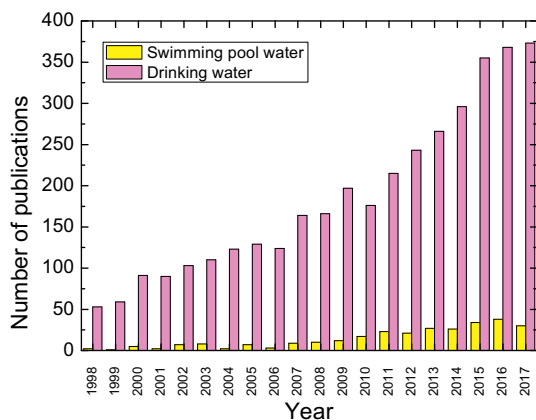


Fig. 1. The number of publications in relation with DBPs in SPWs and DWs in the past 20 years (1998–2017).

Source: Web of Science on Feb. 02, 2018. Keywords for searching: disinfection by-product, swimming pool water/drinking water.

their critical importance, a comprehensive review is warranted to summarize the occurrence, formation and treatment of DBPs in the context of SPWs.

This review overviews the latest research progress of DBPs in SPWs from the following aspects: DBP regulations in different regions and by different organizations, formation mechanisms linked with different disinfectants, precursors and other various conditions, human exposure assessment reflected by biomarkers and epidemiological evidence, and the latest approaches for the control and treatment of DBPs.

## 2. Regulations and guidelines of DBPs

Regulations and guidelines have been established for DBPs in DWs, mainly covering THMs, HAAs, haloacetonitriles (HANs), bromate, chlorate and chlorite, *n*-nitrosodimethylamine (NDMA), cyanogen chloride and 2,4,6-trichlorophenol (Table 1). THMs were first regulated by U.S. EPA in 1979 with a MCL of  $100 \mu\text{g/L}$  in DWs (EPA, 1979). In 1998, the MCL of THMs was lowered to  $80 \mu\text{g/L}$  and meanwhile HAA<sub>5</sub> (the sum of chloro-, bromo-, dichloro-, dibromo-, trichloroacetic acids) were for first time regulated at  $60 \mu\text{g/L}$  (EPA, 1998). Apart from four THM and five HAA compounds regulated by U.S. EPA, the MCLs for bromate and chlorite are 10 and  $1000 \mu\text{g/L}$ , respectively. In contrast, the European Union only regulates THMs ( $< 100 \mu\text{g/L}$ ) and bromate ( $< 10 \mu\text{g/L}$ ) (Directive, 1998). The World Health Organization (WHO, 2008) provides guideline values for a comprehensive list of 15 individual DBPs in DWs. In addition to THMs and HAAs, the WHO guidelines also include NDMA ( $0.1 \mu\text{g/L}$ ), cyanogen chloride ( $70 \mu\text{g/L}$ ), 2,4,6-trichlorophenol ( $200 \mu\text{g/L}$ ), bromate ( $10 \mu\text{g/L}$ ), chlorate ( $700 \mu\text{g/L}$ ), chlorite ( $700 \mu\text{g/L}$ ) and two individual HANs, i.e., dibromoacetonitrile ( $70 \mu\text{g/L}$ ) and dichloroacetonitrile ( $20 \mu\text{g/L}$ ). The DW quality standard prescribed by Singapore is largely based on the WHO guideline (NEA, 2008). DBP guidelines for Australian and Canadian DW quality are shown in Table 1 as well.

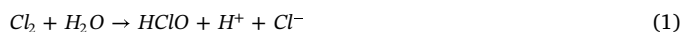
Unlike the systematic regulation of DBPs in DWs, only THMs in SPWs have been regulated in some European countries (Table 2). Based on DIN 19643, the MCL of THMs (calculated as chloroform) in SPWs is  $20 \mu\text{g/L}$  in Germany (DIN, 2012). Switzerland has provided the MCL of THMs of  $30 \mu\text{g/L}$  and Denmark has promulgated the regulation of THMs in SPWs to be under 25 or  $50 \mu\text{g/L}$  depending on the pool types (Simard et al., 2013). The French Agency for Food, Environmental and Occupational Health & Safety (ANSES) has set a mandatory value for THMs of  $100 \mu\text{g/L}$  and a guide value of  $20 \mu\text{g/L}$  (ANSES, 2012). Some other developed countries, for example, Singapore, Canada and the United States, still do not have regulations for DBPs in SPWs in spite of the high popularity of swimming activity in these regions. Based on the existing available regulations and guidelines, the MCLs of DBPs in SPWs are often comparable or even lower than these in DWs. However, the DBP concentrations are generally much higher in SPWs than in DWs (Section 3.3.1), leading to concerns over their potential effect on health.

## 3. DBP formation mechanism

### 3.1. Disinfectants

#### 3.1.1. Disinfectants and their aqueous chemistry

Chlorination is the most commonly used disinfection approach for SPW treatment (Afifi and Blatchley III, 2015; Kogevinas et al., 2010; Weng and Blatchley III, 2011). Chlorine based disinfectants mainly include chlorine gas ( $\text{Cl}_2$ ), sodium hypochlorite ( $\text{NaClO}$ ) and calcium hypochlorite ( $\text{Ca}(\text{ClO})_2$ ) (WHO, 2006) (Table 3). The hydrolysis and/or dissociation of these disinfectants produce hypochlorous acid ( $\text{HClO}$ ) and hypochlorite ion ( $\text{ClO}^-$ ) as the active ingredients for disinfection (Eqs. (1)–(4)). The equilibrium between  $\text{HClO}$  and  $\text{ClO}^-$  is pH and temperature dependent.



**Table 1**  
DBP regulations and guidelines in drinking water.

DBPs	U.S. EPA (EPA, 1998)	WHO (WHO, 2008)	Singapore (NEA, 2008)	European Union (Directive, 1998)	Australia (NHMRC, 2011)	Canada (CDW, 2014)
	MCL <sup>a</sup> (µg/L)					
THMs <sup>b</sup>	80	–	–	100	250	100
Chloroform	70	300	300	–	–	–
Bromodichloromethane	0	60	60	–	–	–
Chlorodibromomethane	60	100	100	–	–	–
Bromoform	0	100	100	–	–	–
HAA <sub>5</sub> <sup>c</sup>	60	–	–	–	–	80
Dichloroacetic acid	0	50	50	–	100	–
Trichloroacetic acid	20	200	200	–	100	–
Monochloroacetic acid	70	20	20	–	150	–
HANs	–	–	–	–	–	–
Dibromoacetonitrile	–	70	70	–	–	–
Dichloroacetonitrile	–	20	20	–	–	–
Others						
Bromate	10	10	10	10	20	10
Chlorate	–	700	700	–	–	1000
Chlorite	1000	700	700	–	800	1000
N-nitrosodimethylamine	–	0.1	–	–	0.1	0.04
Cyanogen chloride	–	70	70	–	80	–
2,4,6-Trichlorophenol <sup>d</sup>	–	200	200	–	20	5

Notes:

<sup>a</sup> MCL means maximum contaminant level.

<sup>b</sup> THMs refer to the summation of chloroform, bromodichloromethane, chlorodibromomethane, and bromoform.

<sup>c</sup> HAA<sub>5</sub> refer to the summation of chloro-, bromo-, dichloro-, dibromo-, and trichloroacetic acids.

<sup>d</sup> 2,4,6-Trichlorophenol has not been detected in swimming pool waters.



Bromine based disinfectants are used in pools under special conditions, e.g., spa. Liquid bromine, following the similar hydrolysis pattern of chlorine gas, is used for the maintenance of SPWs, especially for hot tubs (Eq. (5)). Some European countries have practiced using seawater as the source of SPWs since the early 1900s (Parinet et al., 2012). The bromide ion with a concentration of ~70 mg/L in seawater can be oxidized by HClO to form HBrO which plays a dominant role on disinfection (Eq. (6)) (Borges et al., 2005; Von Gunten and Oliveras, 1998). Parinet et al. (2012) pointed out that the formation of more toxic Br-DBPs (e.g., bromoform, dibromoacetic acid, and tribromoacetic acid) was substantial in seawater pools. Bromine with a lower vapour pressure overcomes the unstable property of chlorine under high temperature, such as spas and hot tubs (WHO, 2018). However, bromine based disinfectants are depleted rapidly under sunlight for outdoor pools and spas leading to the ill-suited applications (WHO, 2006).



**Table 2**  
DBP regulations and guidelines in SPWs.

Country	MCL <sup>a</sup> (µg/L)	Comments	References
Germany	20	THMs calculated as chloroform	(DIN, 2012)
Switzerland	30	THMs for indoor pools	(Simard et al., 2013)
Denmark	25 or 50	THMs (depending on the type of pools)	(Simard et al., 2013)
Belgium	100	Chloroform	(Simard et al., 2013)
France	100 or 20 <sup>b</sup>	THMs	(ANSES, 2012)
United Kingdom	100	THMs	(Simard et al., 2013)
Finland	100	THMs	(Simard et al., 2013)

Notes:

<sup>a</sup> MCL means maximum contaminant level.

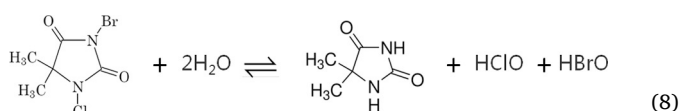
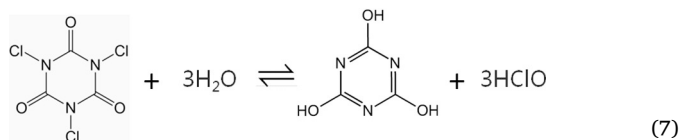
<sup>b</sup> The mandatory value for THMs is 100 µg/L and the guide value for THMs is 20 µg/L.

**Table 3**  
Disinfectant classifications.

Groups	Disinfectants
Chlorine based disinfectants	Chlorine gas Sodium hypochlorite Calcium hypochlorite
Bromine based disinfectants	Bromine liquid Sodium bromide + hypochlorite
Organic based disinfectants	Bromochlorodimethylhydantoin (BCDMH) Trichloroisocyanuric acid (TCCA) Dichloroisocyanuric acid (DCCA)
Others	UV Ozone Chlorine dioxide Hydrogen peroxide/silver/copper Combined disinfectants

Organic based disinfectants mainly include trichloroisocyanuric acid (TCCA), dichloroisocyanuric acid (DCCA), and bromochlorodimethylhydantoin (BCDMH) (Table 3). TCCA and DCCA are widely used in outdoor pools, especially for those exposed under strong sunlight, due to their role of chlorine stabilizer (Yang et al., 2016; Zwiener et al., 2007). The bound forms of chlorine in TCCA, i.e.,

monochloroisocyanurate ion ( $\text{HClCy}^-$ ) and dichloroisocyanurate ion ( $\text{Cl}_2\text{Cy}^-$ ), are more stable than  $\text{HClO}$  and  $\text{ClO}^-$  under the solar exposure, as the formers have much lower maximum absorption wavelengths (Wojtowicz, 1996, 2004). Askins (2013) has suggested that chlorine degradation slowed down with the presence of cyanuric acid under natural sunlight exposure. BCDMH is sometimes used for SPW disinfection (Lourencetti et al., 2012; Richardson et al., 2010). It shows better fecal coliform killing efficiency compared to commonly used  $\text{NaClO}$  (Moffa et al., 2006). However,  $\text{HBrO}$  as the highly reactive disinfection ingredient of BCDMH is unable to sustain the continuous disinfection requirement (Yang et al., 2016). The hydrolysis of TCCA (similar for DCCA) and BCDMH are shown in Eqs. (7) and (8), respectively.



Halogen-free disinfectants, e.g., UV and ozone, offer the advantages of minimizing the formation of halogenated DBPs. However, they are unable to sustain long time disinfection ability due to the lack of residuals. Chlorine dioxide produces chlorite and chlorate (Eq. (9)), which are mandatory regulated species in DWs (Table 1). Combined disinfections (e.g., UV + chlorine) are originally attempted to eliminate DBP formation. However this technology may only eliminate some DBPs (inorganic chloramines for instance) and enhance the formation of other DBPs (e.g., THMs, HANs, etc.) (Cheema et al., 2017; Cimetiere and De Laat, 2014; Weng et al., 2013) (see further discussion in Section 3.3.3).



### 3.1.2. Quantity control of disinfectants

Disinfection prevents some microbial-related infectious diseases spreading among swimmers in pools. The summation of  $\text{HClO}$  and  $\text{ClO}^-$  (active ingredients for disinfection) is defined as free chlorine (White, 1999). The quantity control of free chlorine in SPWs is essential. Its excess may cause discomfort to swimmers such as skin irritation and its insufficiency weakens microbial killing. Many countries have elaborated the free chlorine ranges in SPWs based on water conditions and/or pool types (Table 4). Singapore recommends a range of 1–3 mg/L for free chlorine (NEA, 2005). Based on Germany standard DIN 19643, the free chlorine ranges from 0.3 to 0.6 mg/L for swimming pools and from 0.7 to 1.0 mg/L for hot tubs (DIN, 2012). National Swimming Pool Foundation (NSPF) in the U.S. regulates free chlorine at 1–5 mg/L and recommends an ideal range of 2–4 mg/L (NSPF, 2006). Some states in the U.S. have their own free chlorine guidelines in pools, for example, 0.25–5.0, 1–10, and 1.5–5.0 mg/L in Colorado, Florida, and Georgia, respectively (CDPHE, 1998; DPH, 2013; FAC, 2009).

## 3.2. DBP precursors

DBP precursors in SPWs include NOM from the filling water (surface or ground water from water treatment plants), body fluids from the swimmers (e.g., urine, sweat, hair, saliva, etc.), and PCPs released from human skin or urine (e.g., sunscreen, body lotion, hand soaps, laundry detergents, shampoos, hair gels) (Chowdhury et al., 2014; Kim et al., 2002; Nassan et al., 2017; Zwiener et al., 2007). The additional body fluids and PCPs make SPW matrix more complicated and contribute to the higher potential formation of some conventional or emerging DBPs compared to DWs with NOM as the main organic source.

### 3.2.1. Natural organic matter

NOM in SPWs mainly come from source or make-up water (normally DWs). Humic substances (i.e., humic and fulvic acids) account for the major fraction of NOM (52–70%) (Fan et al., 2001). Other hydrophilic substances (e.g., transphilic acids, proteins, amino acids and carbohydrates) account for 20–40%. NOM has been categorised as humic acids and hydrophobic/hydrophilic acid/base/neutral compounds by fractionation techniques (Matilainen and Sillanpää, 2010). NOM in DWs is in the range of 0.7–6.7 mg TOC/L and 0.1–0.3 mg TN/L in the U.S. (Kanan and Karanfil, 2011), 2.6–4.7 and 1.8–5.4 mg DOC/L in Norway and Australia (Fabris et al., 2008), and 1.1–1.4 mg TOC/L and 2.15 mg TN/L in Singapore (Yang et al., 2016). NOM has been proven to possess a high potential towards the formation of DBPs (e.g., THMs, HAAs, HANs, and HNMs) during chlorination (Kanan and Karanfil, 2011; WHO, 2008; Yang et al., 2016). The reduced TOC in treated SPWs or raw water corresponding to the substantially weakened formation potential of adsorbable organic halogens and THMs (AOX-FP, THM-FP) confirms that NOM acts as effective DBP precursors (Fabris et al., 2008; Glauner et al., 2005a, b). Fabris et al. (2008) reported an empirical relation between NOM and THM-FP as  $\sim 10\text{--}40 \mu\text{g THM-FP/mg DOC}$ . Therefore, it is an effective strategy to remove NOM via SPW treatment to reduce the DBP formation from the source.

### 3.2.2. Body fluids

Human body fluids are released from swimmers during pool activities, where urine and sweat are two main sources of contaminants (Lamont Bradford, 2014). Judd and Black (2000) revealed that the actual pool condition is roughly approximated by 200 ml sweat and 50 ml urine per cubic meter of pool water. The release of urine and sweat into swimming pools has been estimated in a range of 25–77.5 and 200–1000 mL/bather, respectively (De Laat et al., 2011; WHO, 2006). Similar levels of urine and sweat release from swimmers have been reported in other literature studies, i.e., 54.7–117 and 823–1760 mL/bather by Weng and Blatchley III (2011), and 20–80 and 100–1000 mL/bather by Florentin et al. (2011). Sweat release is much higher than urine release especially during intensive exercises in pools operated with high temperatures. The sweat release rate is 0.04–0.91 L/m<sup>2</sup>/h as summarized by Keuten from previous publications and 0.1–0.8 L/m<sup>2</sup>/h as obtained from his own laboratory and on-site experiments (Keuten et al., 2014).

Urea is the main component in urine and sweat with a mean concentration of 10,240 and 680 mg/L, which accounts for 84% and 68% of TN, respectively (WHO, 2006). Urea in SPWs has been reported in many literature studies, e.g., 0.01–0.11 mg/L by Afifi and Blatchley III (2015), 0.50–2.12 mg/L by Schmalz et al. (2011), 0.12–3.6 mg/L by De Laat et al. (2011), 0.01–4.02 mg/L by Blatchley III and Cheng (2010). Our survey in Singapore pools shows a urea concentration of  $0.23 \pm 0.19 \text{ mg/L}$  (Yang et al., 2017b). Urea in an indoor pool increased by 72.4–155  $\mu\text{g/L}$  daily during a national swimming competition, which was equivalent to 0.56–1.20 g urea or 0.26–0.56 g TN released by each bather per day (Weng and Blatchley III, 2011). Similar urea release of 0.8–1.5 g/bather has been reported (WHO, 2006). The average release of pollutants during 30 min exercise is 250, 77.3, 37.1 and 10.1 mg/bather for non-purgeable organic carbon, TN, urea and ammonium, respectively (Keuten et al., 2014). Intensive usage of pools results in the substantial introduction of pollutants (e.g., urea, TOC, and TN), leading to adverse effects on water quality (Afifi and Blatchley III, 2015; De Laat et al., 2011; Weng and Blatchley III, 2011).

Apart from urea, creatinine (670–2150 mg/L), hippuric acid (50–1670 mg/L), citric acid (90–930 mg/L), ammonia (200–730 mg/L), uric acid (40–670 mg/L), glycine (90–450 mg/L), histidine (40–330 mg/L) present at substantial concentrations in urine can act as DBP precursors (Beach, 1971). A large portion of these nitrogenous compounds containing various functional groups ( $-\text{NH}_2$ ,  $-\text{COOH}$ ) and forms of bound nitrogen have been proven as efficient trichloramine precursors, particularly at acidic and neutral pH conditions (Afifi and

**Table 4**  
Regulated conditions for the operation of swimming pools.

Countries	Free chlorine	Combined chlorine (MCL <sup>a</sup> )	pH	References
US	<ul style="list-style-type: none"> <li>• Pools: 1–3 mg/L</li> <li>• Hot tubs, spa: 2–4 mg/L</li> <li>• Health care facilities: 15 mg/L (small hydrotherapy tanks, Hubbard tanks, tubs), 2–5 mg/L (whirlpools and whirlpool spa)</li> <li>• 1–5 mg/L (an ideal range of 2–4 mg/L in real operation)</li> </ul>	<ul style="list-style-type: none"> <li>• Pools: 0.2 mg/L</li> <li>• Spa: 0.5 mg/L</li> </ul>	<ul style="list-style-type: none"> <li>• 7.2–7.8</li> <li>• 7.4–7.6</li> </ul>	(Bottoni et al., 2014)
Canada (Quebec)	<ul style="list-style-type: none"> <li>• Indoor pools: 0.8–2 mg/L</li> <li>• Outdoor pools: 0.8–3 mg/L</li> <li>• Pools (T &gt; 35 °C): 2–3 mg/L</li> </ul>	<ul style="list-style-type: none"> <li>• Indoor pools: 0.5 mg/L</li> <li>• Outdoor pools: 1 mg/L</li> </ul>	<ul style="list-style-type: none"> <li>• 7.2–7.8</li> </ul>	(NSPF, 2006) (EQA, 2016)
Germany	<ul style="list-style-type: none"> <li>• Pools: 0.3–0.6 mg/L</li> <li>• Spa (hot tubs): 0.7–1 mg/L</li> </ul>	<ul style="list-style-type: none"> <li>• 0.2 mg/L</li> </ul>	<ul style="list-style-type: none"> <li>• 6.5–7.6</li> </ul>	(DIN, 2012)
Spain	<ul style="list-style-type: none"> <li>• 0.4–1.5 mg/L</li> </ul>		<ul style="list-style-type: none"> <li>• 6.8–8.0</li> </ul>	(Spain, 2007)
Australia (Queensland)	<ul style="list-style-type: none"> <li>• Indoor pools: &gt; 1.5 mg/L</li> <li>• Heated indoor pools (&gt; 26 °C): &gt; 2 mg/L</li> <li>• Outdoor pools: &gt; 1.5 mg/L</li> <li>• Heated outdoor pools (&gt; 26 °C): &gt; 3 mg/L</li> <li>• Spa (35–40 °C): &gt; 3 mg/L</li> </ul>	<ul style="list-style-type: none"> <li>• 1 mg/L (not exceed half the total chlorine concentration)</li> </ul>	<ul style="list-style-type: none"> <li>• 7.2–7.8</li> </ul>	(Queensland, 2004)
France	<ul style="list-style-type: none"> <li>• Pools (bleach or chlorine gas): 0.4–1.4 or 0.3–0.6 mg/L<sup>b</sup></li> <li>• Pools (isocyanuric acid derivatives): 2–4 mg/L</li> </ul>	<ul style="list-style-type: none"> <li>• 0.6 mg/L</li> </ul>	<ul style="list-style-type: none"> <li>• Pools: 6.9–7.7</li> <li>• Whirlpools: 6.9–8.2</li> </ul>	(ANSES, 2012, 2014)
London	<ul style="list-style-type: none"> <li>• 1–2 mg/L</li> </ul>			(WHO, 2006)
Italy	<ul style="list-style-type: none"> <li>• 0.6–1.2 mg/L</li> </ul>			(WHO, 2006)
Switzerland	<ul style="list-style-type: none"> <li>• Public and semi-public pools: &lt; 3 mg/L</li> <li>• Public and semi-public hot tubs: &lt; 5 mg/L</li> </ul>	<ul style="list-style-type: none"> <li>• 0.2 mg/L</li> </ul>	<ul style="list-style-type: none"> <li>• Chlorine disinfectants: 7.2–7.8</li> <li>• Bromine-based or other non-chlorine disinfectants: 7.2–8.0</li> </ul>	(WHO, 2006)
Singapore	<ul style="list-style-type: none"> <li>• 1–3 mg/L</li> </ul>		<ul style="list-style-type: none"> <li>• 7.2–7.8</li> </ul>	(NEA, 2005)
China	<ul style="list-style-type: none"> <li>• 0.3–0.5 mg/L</li> </ul>		<ul style="list-style-type: none"> <li>• Artificial pools: 6.8–8.5</li> <li>• Natural pools: 6.0–9.0</li> </ul>	(GB, 1996)

## Notes:

<sup>a</sup> MCL means maximum contaminant level.

<sup>b</sup> The mandatory range of chlorine is 0.4–1.4 mg/L. The range can be 0.3–0.6 mg/L when the hygiene conditions of pools are met.

Blatchley III, 2015; Blatchley III and Cheng, 2010; De Laat et al., 2011; Lian et al., 2014; Schmalz et al., 2011). The release of nitrogenous compounds into swimming pools is between 320–840, 30–60, 15–50, 10–25 and 400–1000 mg/bather for urea, ammonia, amino-acids, creatinine and TN, respectively (Florentin et al., 2011).

In some lab-scale experiments, different recipes for body fluid analog (BFA) have been proposed to mimic swimmers' body excretions. A BFA recipe including urea (14,800 mg/L), creatinine (1800 mg/L), hippuric acid (1710 mg/L), histidine (1210 mg/L), citric acid (640 mg/L), uric acid (490 mg/L), ammonium chloride (2000 mg/L), and sodium phosphate (4300 mg/L) has been widely used for DBP formation potential tests in simulated pool conditions (Judd and Bullock, 2003; Kanan and Karanfil, 2011; Yang et al., 2016). This BFA represents a mixture of urine and sweat with a volume ratio of 1:4, in a concentration of ~5700 mg TOC/L and ~8700 mg TN/L. In the early stage, Judd and Black (2000) used a far more complicated BFA recipe with some additional inorganic compounds. Although this recipe is somewhat more representative of the real urine or sweat compositions (Beach, 1971), the additional inorganic constituents do not essentially affect the DBP formation. Kanan and Karanfil (2011) used three BFAs with different organic compositions to study their effect on DBP speciation. DBP formation potential tests of individual component showed that uric acid, citric acid, hippuric acid had significantly higher reactivity towards THM and/or HAA formation (Kanan and Karanfil, 2011; Yang et al., 2016), and that acid amides, particularly urea, ammonium ions and  $\alpha$ -amino acids were efficient trichloramine precursors (Schmalz et al., 2011).

The substantial DBP formation by human fluid raises public awareness of the importance of hygiene behaviour, i.e., have a shower before entering into the pools, do not urinate during swimming. However, a public survey among 1000 adults in the U.S. in 2009 showed that 35% of pool participants skipped the shower before

swimming and 17% had urination experience, and 84% believed their fellow swimmers engaged in unhygienic behaviours at public pools (Wiant, 2011). Keuten et al. (2014) found that 63% of pollutants in pools come from unhygienic behaviour at high exercise levels, of which 31% is caused by none pre-swim shower and 32% results from urination. The elimination of unhygienic behaviour has been shown to reduce the DBP formation by 55% in 35 °C and up to 68% in 25 °C, and these proportions are expected to increase at lower exercise levels (Keuten et al., 2014). A pre-swim shower of as short as 60 s can remove a significant amount of pollutants from human bodies (Keuten et al., 2012). It is essential to encourage or even enforce the swimmers to develop good hygiene habits.

### 3.2.3. Personal care products

Apart from the commonly discussed NOM and BFA, PCPs released by swimmers could be the potential precursors of chlorinated and oxidized or nitrogenous DBPs (Balmer et al., 2005). The more detectable PCPs in SPWs than in DWs are mainly attributed to the lower dilution ratio and high recirculation of SPWs. For example, the concentration of UV filters reached up to  $\mu\text{g/L}$  in SPWs or shower wastewater, which was several orders of magnitude higher than that in surface water (ng/L) (Bottoni et al., 2014; Lambropoulou et al., 2002).

Sunscreen is widely used in summer when pool activities are most popular. It aims at preventing skin damage under solar radiation via some commonly used active ingredients, e.g., 4-methylbenzylidene camphor (Serpone et al., 2002). Ekowati et al. (2016) provided a comprehensive review of 14 UV filters in swimming pools and spas and found that 4-methylbenzylidene camphor and 1H-benzotriazole had the highest concentration of 69.3 ng/L and the highest frequency of occurrence of 59%, respectively. Zwiener et al. (2007) have identified 5 active ingredients of sunscreens, namely 2-hydroxy-4-methoxybenzophenone, ethylhexyl methoxycinnamate,

**Table 5**  
THM concentrations in SPWs.

Country	THMs ( $\mu\text{g/L}$ )					Pool type <sup>a</sup>	References
	$\text{CHCl}_3$	$\text{CHCl}_2\text{Br}$	$\text{CHClBr}_2$	$\text{CHBr}_3$	THMs		
U.S.	71 (25–207)	4 (1–28)	4 (1–10)	< 1	80 (26–213)	23 indoor (Cl)	(Kanan et al., 2015)
	~25	< LOD	~2	< LOD		1 indoor (Cl)	(Weng and Blatchley III, 2011)
	63 (16–138)		6 (1–35)	7 (1–55)	78 (19–146)	11 indoor (Cl)	(Weaver et al., 2009)
Canada	80–130					4 indoor (Cl)	(Li and Blatchley, 2007)
	70–140					2 outdoor (Cl)	(Li and Blatchley, 2007)
	81 (12–282)		2 (N.D.–10)	1 (N.D.–32)	77 (33–282)	1 indoor (Cl)	(Afifi and Blatchley III, 2015)
Germany	18–80				44 (18–114)	15 indoor (Cl)	(Simard et al., 2013)
	29 (13–46)				98 (12–311)	39 outdoor (Cl)	(Simard et al., 2013)
						8 indoor (–)	(Lévesque et al., 2000)
Spain	18 (7–25)				29 (13–46)	2 indoor (Cl)	(Catto et al., 2012)
	23 (21–27)	3 (2–3)	< 0.5	< 0.4	39 (5–125)	2 outdoor (TCCA)	(Zwiener et al., 2007)
	3–28	0.7–6	0.03–7	0.02–0.8	21	1 indoor (Cl)	(Glauner et al., 2005b)
France					35–47	2 outdoor (Cl)	(Glauner et al., 2005b)
	15 (8–21)	14 (9–27)	13 (7–23)	7 (3–17)	50 (35–75)	1 indoor (Cl)	(Richardson et al., 2010)
	< 0.3	< 0.7	2 (2–3)	57 (52–64)	60 (54–67)	1 indoor (BCDMH)	(Richardson et al., 2010)
Australia	13.7 $\pm$ 7.3	1.4 $\pm$ 0.7	0.5 $\pm$ 0.5	0.3 $\pm$ 0.7	15.8 $\pm$ 7.2	20 indoor (–)	(Marina et al., 2009)
					45.4 $\pm$ 7.3	1 indoor (Cl)	(Kogevinas et al., 2010)
	15 (9–20)	14 (9–25)	13 (7–23)	7 (3–16)	50	1 indoor (Cl)	(Lourencetti et al., 2012)
Ireland	< 0.3	< 0.6	2 (2–3)	60 (52–61)	63	1 indoor (BCDMH)	(Lourencetti et al., 2012)
	< 0.3	0.3 (0.1–1.1)	19 (3–64)	300 (29–931)	320 (32–996)	8 indoor (seawater)	(Parinet et al., 2012)
	67 (47–82)	9 (5–12)	3 (1–5)	1 (1–2)	80 (64–98)	3 indoor (Cl)	(Villanueva et al., 2007b)
England	70	8	2	0.6	80	1 outdoor (Cl)	(Manasfi et al., 2016)
	N.D.	N.D.	4 (2–5)	66 (49–87)	70 (50–92)	3 indoor (seawater)	(Manasfi et al., 2016)
	76 (65–84)	2 (2–3)	0.3–0.4	< 0.1	79 (67–87)	1 outdoor (Cl)	(Yeh et al., 2014)
Korea	33.2 $\pm$ 24.6	4.2 $\pm$ 1.3	1.9 $\pm$ 2.0	0.4 $\pm$ 0.5	39.8 $\pm$ 21.7	5 indoor (Cl)	(Fantuzzi et al., 2001)
	88–116	17–18			105–133	3 indoor (Cl)	(Stack et al., 2000)
	121 (45–212)	8 (3–23)	3 (1–7)	1 (1–2)	132 (57–223)	8 indoor (Cl)	(Chu and Nieuwenhuijsen, 2002)
Singapore	21 (N.D.–46)	2 (N.D.–7)	N.D.	N.D.	23	86 indoor (three disinfectants)	(Lee et al., 2010)
	74 (30–167)	4 (1–12)	< 1	< 0.5	90 (32–170)	9 outdoor (–)	(Yang et al., 2016)

Notes: ND: not detectable; LOD: limit of detection.

<sup>a</sup> Chlorinated pools are indicated by Cl in the brackets unless otherwise specified.

2-ethylhexyl-2-cyano-3,3-diphenyl-2-propenoate, 2-phenyl-1H-benzimidazole-5-sulfonic acid, and 4-methylbenzylidene camphor, with their total concentration ranging from single-digit to dozens of  $\mu\text{g/L}$  in outdoor pools. The higher bather load in the baby pool explains its higher sunscreen concentration than that in the adult pool. The 2-hydroxy-4-methoxybenzophenone produced considerably TOX (total organic halides) and THM after chlorination. The solar irradiation of a sunscreen agent, i.e., octyl-dimethyl-*p*-aminobenzoic acid, demonstrated its photolysis mainly via dealkylation and hydroxylation (Sakkas et al., 2003). In addition to the photo-transformation products of octyl-dimethyl-*p*-aminobenzoic acid detected in seawaters, the identification of some chlorinated intermediates in chlorinated SPWs demonstrates its potential role in DBP formation. Vidal et al. (2010) developed a method to identify six sunscreen ingredients (e.g., isoamyl 4-methoxycinnamate, 3-(4'-methylbenzylidene)camphor, 2-hydroxy-4-methoxybenzophenone, 2-ethylhexyl 2-cyano-3,3-diphenylacrylate, 2-ethylhexyl 4-dimethylaminobenzoate, and 2-ethylhexyl 4-methoxycinnamate). Only the former two species were detectable in a public swimming pool with concentrations of  $\sim 0.7 \mu\text{g/L}$  and < LOQ (limit of quantification), respectively. However, they were not detectable in a private pool, possibly due to the low bather load.

There are limited studies reporting the presence of sunscreen constituents in SPWs due to the high detection limit of current used analytical methods and lack of public concerns. Researchers started to conduct laboratory experiments to investigate their roles in DBP formation in the late 2000s. The halogenation of sunscreen reagents relies on many parameters, e.g., chlorine concentration, solution pH, the presence of bromide ions, the compound structure, etc. (Nakajima et al., 2009; Negreira et al., 2008). Wang et al. (2013) conducted the

DBP formation potential tests for 4 body lotions and 4 sunscreens, and found the formation of an emerging DBP group, i.e., halobenzoquinones, such as 2,6-dichloro-1,4-benzoquinone with concentrations up to > 5000 ng/L and 2,6-dichloro-3-methyl-(1,4)benzoquinone with a concentration range of < 0.1–4.0 ng/L. Two orders higher of 2,6-dichloro-1,4-benzoquinone or other more detectable halobenzoquinone (e.g., 2,3,6-trichloro-(1,4)benzoquinone, 2,3-dibromo-5,6-dimethyl-(1,4)benzoquinone, and 2,6-dibromo-(1,4)benzoquinone) in SPWs than in tap water were attributed to the higher presence of these sunscreen precursors in SPWs. Nakajima et al. (2009) tested the chlorination of two sunscreen agents, i.e., octyl dimethyl-*p*-aminobenzoate and octyl-*p*-methoxycinnamate, in model swimming pool conditions and found the intensive appearance of some chlorine-substituted compounds. The mutagenicity of the chlorinated octyl-*p*-methoxycinnamate increased rapidly during the initial reaction stage and reduced to the control level later, indicating the formation of some unstable mutagenic intermediates. Negreira et al. (2008) identified the possible halogenated by-products of 2-ethylhexyl 4-(dimethylamino) benzoate, 2-ethylhexyl salicylate, and 2-hydroxy-4-methoxybenzophenone under chlorine exposure. The DBP formation was observed via aromatic substitution of hydrogen by halogen or cleavage of the carbonyl bond. The occurrence of five commonly used organic UV filters, including dioxybenzone, oxybenzone, avobenzene, 2-ethylhexyl-4-methoxycinnamate, and octocrylene, was reported in chlorinated seawater swimming pools with a total concentration of  $\sim 10 \mu\text{g/L}$  (Manasfi et al., 2015, 2017b). Bromoform and/or bromal hydrate (with higher cytotoxicity and genotoxicity than their chlorinated ones (Manasfi et al., 2017c; Plewa et al., 2008)) were identified as their transformation by-products due to the high presence of bromide in seawater, except

**Table 6**  
HAA concentrations in SPWs.

Country	HAAs (µg/L)										Pool type <sup>a</sup>	References	
	MCAA	MBAA	DCAA	TCAA	BCAA	DBAA	BDCAA	DBCAA	TBAA	HAAs			
U.S.			700 (310–1330)	680 (370–1140)							1442 (800–2430)	6 outdoor (CI)	(Wang et al., 2014)
			727 (50–2040)	890 (20–2970)							1613 (70–3980)	6 indoor (CI)	(Wang et al., 2014)
			450 (50–750)	330 (40–530)							1067 (690–1360)	3 spa (CI)	(Wang et al., 2014)
Canada		2 (MRL-5)	777 (52–6787)	437 (76–1925)	21 (1–176)	8 (1–25)	34 (8–110)	6 (1–32)			1541 (172–9005)	23 indoor (CI)	(Kanan et al., 2015)
			93 (48–192)	10 (54–191)	2 (1–3)		15 (< LOD-24)				364 (104–1195)	15 indoor (CI)	(Simard et al., 2013)
				206 (175–245)			1 (1–2)	< 0.5			808 (155–2224)	39 outdoor (CI)	(Simard et al., 2013)
Germany											218 (111–390)	2 outdoor (CI)	(Catto et al., 2012)
												3 indoor (–)	(Cammann and Hübner, 1993)
	26 (3–81)	0.3 (< 0.5–3)	23 (2–192)	42 (4–199)		0.6 (0.2–8)						Indoor (–)	(Stottmeister and Naglitsch, 1996)
	32 (3–174)	0.2 (< 0.5–2)	9 (2–27)	15 (1–45)		0.6 (0.2–5)						Hydrotherapy (–)	(Stottmeister and Naglitsch, 1996)
	26 (3–112)	0.1 (< 0.5–2)	132 (6–562)	249 (8–887)		0.1 (0.2–1)						Outdoor (–)	(Stottmeister and Naglitsch, 1996)
Spain												Indoor (–)	(Schmalz, 2012)
	2 (1–5)		19 (4–59)	10 (4–21)	2 (< 0.6–4)	< 1	5 (2–10)				39 (11–99)	Indoor (–)	(Cardador and Gallego, 2011)
	20 (9–36)		71 (60–91)	105 (85–125)							207 (154–251)	1 indoor (CI)	(Cardador and Gallego, 2011)
	26 (20–34)		154 (135–170)	122 (99–146)							302 (254–350)	1 outdoor (CI)	(Cardador and Gallego, 2011)
	15–1000			1000–1700			208–912	< LOD-62	< LOD-15		1300–3200	3 (–)	(Loos and Barceló, 2001)
	4	N.D.	45	155	11	2	61	33	19		330	1 (–)	(Sarrion et al., 2000)
France	15 (1–96)	42 (4–155)	4 (1–9)	22 (3–87)	50 (5–216)	234 (11–1089)	10 (1–20)	92 (36–243)	124 (4–428)		594 (84–2233)	8 indoor (seawater)	(Parinet et al., 2012)
	N.D.	N.D.	23	461	2	2	7	3	N.D.		498	1 outdoor (CI)	(Manasfi et al., 2016)
	N.D.	N.D.	N.D.	N.D.	4 (4–5)	66 (63–72)	N.D.	3 (3–4)	43 (36–53)		116 (109–132)	3 indoor (seawater)	(Manasfi et al., 2016)
Australia	59 (< 0.5–120)	< 0.5	1001 (230–2400)	1151 (110–2600)	< 0.5	< 0.5	12 (< 0.5–22)	< 0.5	–		2204 (366–5126)	7 outdoor + 5 indoor (CI)	(Yeh et al., 2014)
Korea			68 (14–246)	156 (20–636)								86 indoor (three disinfectants)	(Lee et al., 2010)
Singapore	22 (14–45)	< 2	370 (45–828)	437 (114–1020)	5 (1–9)	1 (0–1)	11 (5–19)	< 2	< 5		798 (191–1906)	9 outdoor (–)	(Yang et al., 2016)
China			81 (44–195)	52 (33–98)							156 (89–332)	5 outdoor (CI)	(Wang et al., 2014)
			34 (5–60)	53 (6–90)							95 (13–133)	9 indoor (CI)	(Wang et al., 2014)

Notes: ND: not detectable; LOD: limit of detection; MRL: minimum reporting levels. MCAA: chloroacetic acid; MBAA: bromoacetic acid; DCAA: dichloroacetic acid; BCAA: bromochloroacetic acid; TBAA: tribromoacetic acid; TCAA: trichloroacetic acid; BDCAA: bromodichloroacetic acid; DBCAA: dibromochloroacetic acid; DBCAA: dibromochloroacetic acid; TBAA: tribromoacetic acid.

<sup>a</sup> Chlorinated pools are indicated by CI in the brackets unless otherwise specified.

octocrylene as a chlorine resistant species.

Weng et al. (2014) found that only *N,N*-diethyl-*m*-toluamide, caffeine, and tri(2-chloroethyl)-phosphate were detectable in SPWs although totally 32 pharmaceuticals and personal care products (PPCPs) were tested. Teo et al. (2016) found the high occurrence of caffeine (up to 1540 ng/L) and ibuprofen (up to 83 ng/L) in chlorinated SPWs after a screen of 30 PPCPs. The absence of other PPCPs may be attributed to their chlorine-susceptible properties (transfer to DBPs by chlorination). Conversely, the accumulation of some PPCPs in SPWs may be caused by the slow reaction rate with chlorine, slow transfer from liquid to gas phases, or inefficient metabolism in humans. The insect repellent *N,N*-diethyltoluamide (DEET), commonly detected in swimming pool water samples, has been linked to the formation of nitrosamines during chloramination and chlorination (Shen and Andrews, 2011; Suppes et al., 2017). Parabens (known as hydroxybenzoate esters) are also widely used as preservatives in PCPs due to their broad antimicrobial spectrum and solubility in water (Alcudia-León et al., 2013; Terasaki and Makino, 2008). Terasaki and Makino (2008) for first time showed the appearance of parabens and its corresponding Cl-DBPs in SPWs. They found that benzylparaben as one of the parabens presents in SPWs at a concentration up to 28 ng/L (Terasaki and Makino, 2008). These authors also reported that a dichlorinated isopropylparaben was present in SPWs with a maximum concentration of 25 ng/L, and a dichlorinated methylparaben and a monochlorinated benzylparaben were detectable but lower than LOQs. Triclosan as an antibacterial agent, widely used in hand soaps, toothpastes, deodorants, mouthwash, etc., is a chlorine-sensitive compound. Its formation of chloroform and other Cl-DBPs under chlorination or chloramination has been reported (Greyschock and Vikesland, 2006; Rule et al., 2005).

### 3.3. Factors affecting DBP formation

#### 3.3.1. DBPs in SPWs and DWs

Richardson et al. (2007) have summarized that a list of > 600 DBPs have been previously detected in DWs. However, a so-far comprehensive field survey conducted also by Richardson in Spain in 2010 showed > 100 DBPs in two large public swimming pools disinfected with NaClO or BCDMH. These DBPs included THMs, HAAs, HNMs, haloacids, halodiacids, haloaldehydes, halonitriles, halo ketones, haloamides, haloalcohols, and other halogenated and non-halogenated DBPs, many of which have yet been reported in SPWs and even do not exist in the current mass spectral library database (Richardson et al., 2010). Zwiener et al. (2007) has used GCMS to identify ~20 DBPs, including HAAs, other haloacids (e.g., 2,2-dichloropropanoic acid, 2-chloro-2-methylpropanoic acid, etc.), halo ketones (e.g., 1,3-dichloropropanone, etc.), and dichloromethyl methyl sulfone in two private outdoor swimming pools treated with stabilized chlorine in the U.S. In swimming pool treatment system, chlorination is the most widely applied or only designated disinfection approach. For example, France only permits the use of chlorine-based disinfectants (e.g., sodium hypochlorite, calcium hypochlorite or stabilized chlorine) in public pools since 2011 (Parinet et al., 2012). In Singapore, NEA authorizes chlorination as the sole disinfection method in SPW treatment. Other potential alternatives, e.g., ozone, chloramines, chlorine dioxide, commonly applied in DWs originally aiming at reducing Cl-DBP formation, may produce far more kinds of emerging non-chlorinated DBPs (Richardson et al., 2007; Sadiq and Rodriguez, 2004).

A large population-based survey (n = 500) conducted in the U.S. reported the average THM and HAA concentrations of 38 and 23 µg/L respectively in DWs (Richardson et al., 2007). However, DBPs (HAAs in particular) in U.S. pools are far more than these values (see Tables 5 and 6). The higher DBP concentrations in SPWs than in DWs could be attributed to the following reasons: 1) the concentration of organic matter in SPWs (high-ppm levels, single- or even two-digit) is higher than that in DWs (low-ppm levels, single-digit or even less) (Kanan and Karanfil, 2011; Kanan et al., 2015; Wang et al., 2014); 2) there are more

organic species in SPWs (body fluids and PCPs in addition to NOM, see Section 3.2); 3) the recirculation of SPWs results in the accumulation of some stable and non-volatile DBPs, HAAs for instance (Kanan and Karanfil, 2011; Yang et al., 2016) (refer to Sections 5.2–5.4 for the DBP reactivity). A study shows that only about 1% of fresh water is added to the pools to offset the daily water consumption via evaporation and other losses, and therefore the retention time of SPWs reaches as high as 100 days (Arnaud, 2016).

#### 3.3.2. DBPs in different countries

DBPs in pools in different countries have been reviewed. A recent research article has summarized the occurrence of DBPs such as THMs, HAAs, HANs, haloacetaldehydes, haloamines, nitrosamines, and halo-benzoquinones in SPWs (Manasfi et al., 2017a). In the current paper, we aim to provide further insights towards the environmental occurrence of DBPs based on a more comprehensive review of THMs and HAAs (as a screen of other potential DBPs due to their abundant data availability) in SPWs. The THM concentrations in different countries listed in Table 5 are mostly in two-digit µg/L levels or occasionally slightly higher than 100 µg/L with one exception, i.e., 320 µg/L in a French study. The high THM concentration (CHBr<sub>3</sub> accounts for ~94%) in this study is mainly attributed to the use of bromide-rich seawater (68.1–106.7 mg/L as Br<sup>-</sup>) as the pool filling water (Parinet et al., 2012) (see more discussions in Section 3.3.4). In addition, CHBr<sub>3</sub> has higher molar mass and is less volatile than CHCl<sub>3</sub> (vapour pressure: 5.4 vs. 196 mm Hg), contributing to more severe contaminant accumulation.

The HAA concentrations in SPWs varied significantly in different countries (Table 6). For example, HAAs were in average only 39 µg/L in a German pool (Schmalz, 2012) and 95 or 156 µg/L in China pools (Wang et al., 2014). Nevertheless, several studies have reported the average HAA concentrations with > 1000 µg/L in the U.S. pools (Kanan et al., 2015; Wang et al., 2014) and 798 µg/L in Singapore pools (Yang et al., 2016). Moderate HAA concentrations were reported for most of Spain and France pools (~100–600 µg/L), with one exception that a Spain pool contains HAAs as high as 3200 µg/L (Loos and Barceló, 2001). The significant gaps between these values attribute in a large extent to the different ranges of disinfectant concentrations in pools regulated by different countries, for example, only 0.3–0.6 mg/L for German pools and 0.3–0.5 mg/L for China pools, but 1–5 mg/L for U.S. pools by NSPF and 1–3 mg/L for Singapore pools (Table 4). Fig. 2 illustrates that HAA concentrations increase significantly with the increasing chlorine in some countries where swimming activities are popular. In addition to chlorine regulation, other SPW characteristics (e.g., the loading and characteristics of organic inputs, the frequency of pool usage, and the hygiene habits of the bathers) and treatment conditions (e.g., filtration rate, water renewal rate, filtration media, etc.),

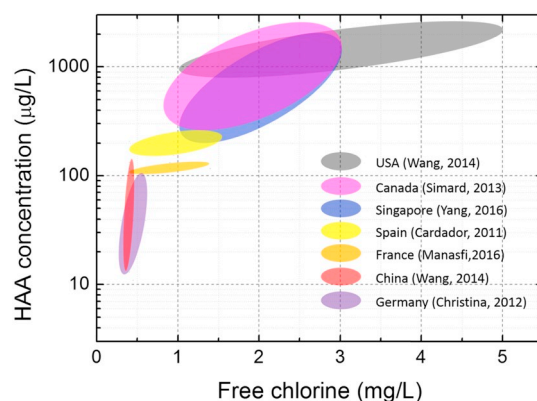


Fig. 2. The relationship between regulated ranges of free chlorine and HAA concentrations in SPWs in different countries. The coordinates of the ellipse vertexes represent the regulated chlorine ranges and reported HAA concentrations.



may contribute to the discrepancies of DBP concentrations as well.

### 3.3.3. DBPs formed by different disinfectants

Chlorination is the most commonly used disinfection technique for SPW treatment. Chloroform, di- and trichloroacetic acids are the main THM and HAA species for chlorinated pools, and bromoform, di- and tribromoacetic acids for brominated pools or chlorinated pools with bromide content (Tables 5 and 6). Bromoform accounted for only 14% for chlorinated pools with minor  $\text{Br}^-$  in source water and it increased up to 95% during bromination by BCDMH (Lourencetti et al., 2012; Richardson et al., 2010). Chlorinated isocyanurates (e.g., TCCA and DCCA) as stabilized forms of chlorine are easy for handling (manual input) and resistant to UV irradiation, therefore commonly used in private or outdoor pools exposed under sunlight (Bernard et al., 2008; Yang et al., 2016; Zwiener et al., 2007). The stabilized chlorine reduced chlorine consumption and DBP formation, mainly attributed to the more stable bound-chlorine species, i.e., monochloroisocyanurate ion ( $\text{HClCy}^-$ ) and dichloroisocyanurate ion ( $\text{Cl}_2\text{Cy}^-$ ) instead of  $\text{HClO}$  and  $\text{ClO}^-$  (Wojtowicz, 2004; Yang et al., 2016). A field study showed that DCCA reduced DBPs up to one order of magnitude compared to the common bleach (e.g., sodium hypochlorite, calcium hypochlorite) (Parinet et al., 2012).

Other disinfectants are also used in SPWs in some regions. In Korea, chlorine alone, the combined use of ozone and chlorine (ozone/chlorine), and a technique that uses electrochemically generated mixed oxidants (EGMOs), are three most commonly used disinfection methods. Ozone/chlorine seems to be the most chemical-safe method in terms of the formation of THMs, HAAs, HANs and chloral hydrate (Lee et al., 2009; Lee et al., 2010). The geometric mean concentrations of THMs in indoor SPWs were  $32.9 \pm 2.4$ ,  $23.3 \pm 2.2$ , and  $58.2 \pm 1.7 \mu\text{g/L}$  for chlorine ( $n = 72$ ), ozone/chlorine ( $n = 86$ ) and EGMO ( $n = 25$ ), respectively (Lee et al., 2009). Ozone as a partial substitute of chlorine reduced the total chlorine dosage therefore reduced the formation of Cl-DBPs. Nevertheless, ozone promoted the formation of some extra DBPs, e.g., bromate and nitrate, which are undesirable contaminants as well (Lee et al., 2010; Michalski and Mathews, 2007). A study in Poland showed that SPWs treated with chlorine dioxide generated more chlorite and chlorate compared to these with ozone (Michalski and Mathews, 2007). These DBPs have also been found in DWs treated with ozone or chlorine dioxide (Sadiq and Rodriguez, 2004). The summation of DBPs (including THMs, HAAs, HANs, and chloral hydrate) in chlorinated SPWs was significantly higher than that by combined chlorine and ozone based on an indoor pool survey (183 vs. 33  $\mu\text{g/L}$ ,  $n = 86$ ) (Lee et al., 2010). On the other hand, Richardson et al. (1999) reported that some emerging DBPs, e.g., 1,1-dichloropropanone and dichloroacetaldehyde, appeared at higher concentrations during the combined disinfection (chlorine + ozone) compared to chlorination only. The combined use of chlorine and UV had advantages of reducing genotoxicity compared to chlorination alone, which might be due to the less formation of Cl-DBPs (chloramine and HAAs for instance) (Cheema et al., 2017; Cimetiere and De Laat, 2014; Liviatic et al., 2010). However, UV irradiation promotes the formation of THMs and some nitrogenous DBPs (e.g.,  $\text{CH}_3\text{NCl}_2$ ,  $\text{CNCHCl}_2$ , and  $\text{CNCl}$ ) which might be more toxic than the carbon-based DBPs (Muellner et al., 2007; Weng et al., 2012; Weng et al., 2013). UV irradiation contributes to the simultaneous photolysis and UV-induced formation of NDMA, which is the most abundant toxic species of nitrosamines (i.e., as biomarkers of bladder cancer in humans (Walse and Mitch, 2008)), in the presence of chlorinated dimethylamine and monochloramine (Soltermann et al., 2013). Cassan et al. (2006) found the medium-pressure UV irradiation increased chloroform and bromodichloromethane via increased active chlorine or a progressive transformation of  $\text{CHBr}_3$ . Chloramination reduced the overall levels of some DBPs (e.g., THMs and HAAs) compared to chlorination but produced other DBPs, e.g., cyanogen chloride (CNCl) and NDMA (Richardson et al., 2007; WHO, 2008).

### 3.3.4. Impact of filling water characteristics

In addition to the types and nature of disinfectants or organics (see Sections 3.1 and 3.2) in SPWs, the filling water characteristics including pH, temperature and the presence of bromide play significant roles in DBP formation as well. Proper pH adjustment in SPWs is necessary to ensure disinfection efficiency, secure user comfort and avoid pipe corrosion. The pH values have been regulated in different countries based on pool types or disinfectants (see Table 4). The pH range is 7.2–7.8 for pools in the U.S., Canada, Australia and Singapore, and wider ranges in Germany (6.5–7.6), Spain (6.8–8.0), and China (6.8–8.5 or 6.0–9.0). In general, lower pH is favoured as  $\text{HClO}$  is a more effective disinfection species than  $\text{ClO}^-$  (Sadiq and Rodriguez, 2004). Hansen and co-workers performed systematic investigations towards the pH effect on DBP formation (Hansen et al., 2012a, b, 2013). In general, with the decreasing pH within 6.0–8.0, THM formation decreased gradually, HAN and  $\text{NCl}_3$  formation increased, and HAA formation remained constant or decreased. Kanan (2010) similarly found that the formation of THMs and HAAs decreased by 40–60% with decreasing pHs from 8.0 to 6.0. The formation of THMs contains the alternate steps of hydrolysis and halogenation, which are favoured under high pH conditions (Bougeard et al., 2008). HANs are expected to be increased due to their reduced hydrolysis under lower pH (Teo et al., 2015). The conflicting results for HAA formation may be attributed to the difference of organic sources in these experiments. The HAA formation was reduced by decreasing pH during the chlorination of filter particles from swimming pools (Hansen et al., 2012b). However, HAAs remained constant with the decreasing pH for BFA chlorination experiments (Hansen et al., 2012a). Acidic and neutral pHs are the most favourable conditions for  $\text{NCl}_3$  formation. Urea as one of the most effective  $\text{NCl}_3$  precursors has a relative  $\text{NCl}_3$  formation of 96% at pH 2.5 however only 24% at pH 7.7 (Schmalz et al., 2011). Low pH promotes  $\text{HClO}$  formation and therefore results in the protonation of bound nitrogen. In addition, low pH slows down  $\text{NCl}_3$  decay (Schmalz et al., 2011). Hansen et al. (2013) recommended a pH range of 6.8–7.5 for SPWs based on the calculated genotoxicity of DBPs (including THMs, HANs and  $\text{NCl}_3$ ). Overall, a comprehensive assessment based on disinfection efficiency, swimmer comfort, system stability and DBP formation is essential to establish the optimal pH range.

The temperature of SPWs influences the DBP formation as well. Simard et al. (2013) found higher concentrations of THMs, HAAs and inorganic chloramine in heated than non-heated outdoor pools (26 vs. 23 °C). An indoor swimming pool survey ( $n = 10$ ) in Canada illustrated higher halobenzoquinone formation at higher temperatures (Wang et al., 2013). Yang et al. (2016) also showed that THM concentrations almost or more than doubled when the temperature was increased from 25 to 40 °C. However, the increase of HAAs was not as severe as that of THMs, which is possibly due to thermal decarboxylation of HAAs to the corresponding THMs (Lifongo et al., 2010). Kanan and Karanfil (2011) similarly found higher temperature contributed to higher THM than HAA yields. The DBP formation affected by temperature could be a combined result of several contributing factors, e.g.,  $\text{HClO}$  ionization, the kinetics of reactions between chlorine and organic matter, heat-promoted hydrolysis of bound chlorine/bromine if stabilized disinfectants are used, thermal stability of DBPs, etc. In addition, the sweat release rate increased from 0.1–0.2 to 0.8  $\text{L/m}^2/\text{h}$  with the temperature increase from 29 to 35 °C, which indicates that low temperature could restrain the continual anthropogenic pollutants released from the swimmers (Keuten et al., 2014).

The bromide present in the source water or as impurities in chemicals for SPW treatment plays a noticeable role in the formation of Br-DBPs. The SPWs are mainly supplied by DWs with a  $\text{Br}^-$  range of 36–2230  $\mu\text{g/L}$  based on a survey during 1997–1998 by U.S. EPA or with a small portion supplied by seawater with  $\sim 70$ –90  $\text{mg/L}$   $\text{Br}^-$  (Kanan, 2010; Manasfi et al., 2016; Parinet et al., 2012). Bromoform, dibromoacetic acid, tribromoacetic acid, dibromoacetonitrile, tribromonitromethane and bromonitromethane are the predominant

species of THMs, HAAs, HANs and HNMs in pools with the presence of bromide (Kanan, 2010; Manasfi et al., 2016; Parinet et al., 2012). The THM and HAA concentrations reached as high as 996 and 2233  $\mu\text{g/L}$  respectively (bromoform accounted for 93% of THMs, and dibromoacetic acid and tribromoacetic acid accounted for 70% of HAAs) in one of the eight chlorinated seawater pools (Parinet et al., 2012), which are far more than the average in chlorinated freshwater pools as shown in Tables 5 and 6. However, except THMs with comparable values, the concentrations of HANs, halo ketones, HAAs, trihaloacetaldehydes and HNMs in chlorinated freshwater pools were much higher than those in chlorinated seawater pools (Manasfi et al., 2016). This discrepancy may be related to other factors, e.g., the number of swimmers, solution pH and temperature, location of pools, physicochemical properties of DBPs, etc. Nevertheless, the cytotoxicity and genotoxicity study based on 18 DBPs (including 6 HAAs, 6 haloacetamides, 3 HANs and 3 THMs) showed that Br-DBPs were substantially more toxic than their chlorinated analogs (Plewa et al., 2008). Hence, the use of seawater or freshwater with high bromide as pool filling water is undesirable with respect to Br-DBP elimination.

### 3.3.5. Impact of indoor and outdoor pools

The coverage of the pools, e.g., indoor or outdoor, has an impact on DBP formation as well. THM and HAA concentrations in outdoor pools were higher than those in indoor pools based on two studies (Cardador and Gallego, 2011; Simard et al., 2013). However, Li and Blatchley (2007) and Wang et al. (2014) found both indoor and outdoor pools in the U.S. had similar THM and HAA concentrations. The entries of additional precursors from the external environment (e.g., rain, dust, leaves) and the higher attendance rate in summer in outdoor pools may contribute to the higher DBP formation. Prolonged exposure of outdoor pools under solar radiation could also contribute to higher THM and HAA yields (Liu et al., 2006). However, the competing effects, such as solar photolysis of THM and HAA in outdoor pools (Chen et al., 2010), have a tendency to moderate their concentrations. The lower concentration of inorganic chloramines ( $\text{NCl}_3$  for instance) in outdoor pools was mainly caused by the solar UV photolysis and higher volatility of these compounds (Simard et al., 2013). Indoor environment may accumulate these volatile DBPs in the gaseous phases above the pool water levels if the ventilation are insufficient (Catto et al., 2012). Zwiener et al. (2007) pointed out that swimmers and even non-swimmers in indoor pools are exposed to a larger extent than in outdoor pools via inhalation of volatile DBPs (e.g., THMs and  $\text{NCl}_3$ ). Similarly, higher DCAA and TCAA were detected in workers' urine in indoor pools than outdoor pools although outdoor pools generally had higher HAAs (Cardador and Gallego, 2011). Therefore, the final DBP concentration in pools relies on the combined effects of formation, transformation and degradation.

## 4. Human exposure and risk assessment

### 4.1. Human exposure

Human bodies are exposed to DBPs mainly via three pathways: 1) inhalation of volatile compounds or aerosolized solutes; 2) dermal absorption by skin; 3) ingestion of DBP-contaminated water. In this section, we mainly reviewed the DBP exposure by assessing the corresponding DBP concentrations in bathers' or workers' body matrices including exhaled air, urine, blood, and plasma.

#### 4.1.1. THMs

Dermal absorption and inhalation are two main exposure routes of THMs (due to their percutaneous absorbable and volatile properties) during activities such as bathing, showering and swimming in indoor pools (Villanueva et al., 2007a, b; Xu et al., 2002). A level III fugacity model demonstrated the similar exposure level for dermal contact and inhalation for chloroform but higher for dermal contact for Br-THMs

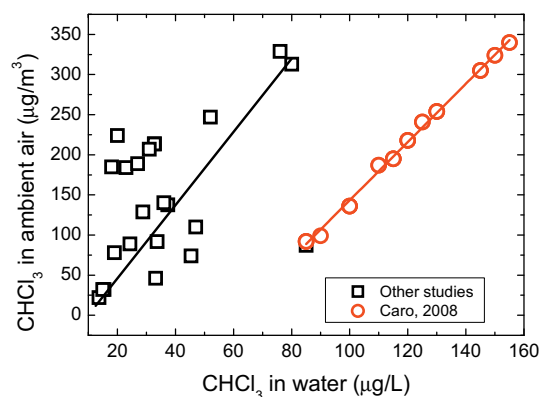
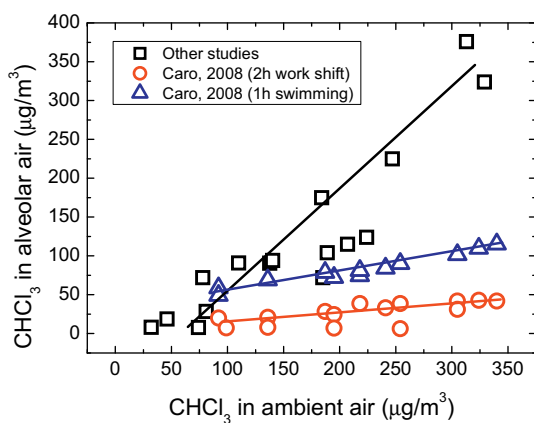


Fig. 3. The relationship between  $\text{CHCl}_3$  concentrations in swimming pool water and in ambient air. Other studies include (Catto et al., 2012; Fantuzzi et al., 2001, 2010; Kogevinas et al., 2010; Lévesque et al., 2000; Lourencetti et al., 2012; Marina et al., 2009; Richardson et al., 2010).

(Dyck et al., 2011). Erdinger et al. (2004) found that 2/3 of chloroform in the blood was taken up via inhalation and 1/3 via dermal absorption, assuming that accidental swallowing of water by divers is negligible. Similarly, Lourencetti et al. (2012) assessed the THM exposure by two predetermined actions, i.e., bathing in the water without exercise, and standing beside the pools. It showed dermal absorption (by subtracting the exposures in these two cases) accounted for ~40% of inhalation. The significant role of inhalation is mainly attributed to the high volatility of these compounds. For example, the vapour pressure of chloroform (196 mm Hg) is two orders of magnitude higher than that of trichloroacetic acid (1 mm Hg).

The crucial exposure of THM by inhalation attracts the public awareness of THM concentration in the ambient air especially in indoor environment with a relatively enclosed space. It is not surprising to observe a positive correlation between chloroform concentrations in SPWs and its corresponding ambient air as shown in Fig. 3 (Catto et al., 2012; Fantuzzi et al., 2001, 2010; Kogevinas et al., 2010; Lévesque et al., 2000; Lourencetti et al., 2012; Marina et al., 2009; Richardson et al., 2010). The chloroform concentrations obtained from an indoor public pool in Spain demonstrated an excellent linear correlation (Caro and Gallego, 2008). Bromoform as an abundant THM species in brominated and chlorinated bromide-rich SPWs, demonstrated its positive correlation between water and air phases as well (Bouduenne et al., 2017; Manasfi et al., 2017a). The prediction of air concentration based on aqueous samples may be possible to avoid the complicated air sampling procedures. It should be noted that air concentrations are location-dependent. Higher THM concentrations were observed at lower levels above the water surface (Nitter et al., 2018). The average THM concentration in air in poolside ( $58.0 \mu\text{g}/\text{m}^3$ ) was more than double of that in the reception area and engine room (26.1 and  $25.6 \mu\text{g}/\text{m}^3$ ) (Fantuzzi et al., 2001). Aprea et al. (2010) similarly found that the THM concentration in air near the pool was higher than those in places far away (e.g., change rooms, passageways and offices).

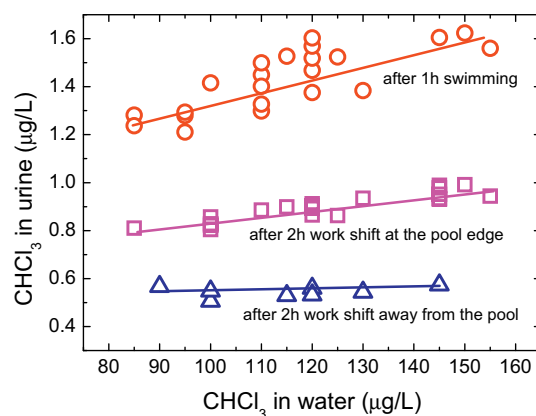
The THM concentrations in alveolar air of swimmers or workers could be a good indicator to evaluate human exposure. The  $\text{CHCl}_3$  in swimmers' alveolar air had a quite wide concentration range of ~10–350  $\mu\text{g}/\text{m}^3$  (shown as black squares in Fig. 4) (Catto et al., 2012; Fantuzzi et al., 2001, 2010; Kogevinas et al., 2010; Lévesque et al., 2000; Lourencetti et al., 2012; Marina et al., 2009; Richardson et al., 2010). Caro and Gallego (2008) have systematically investigated the THM exposure by human groups with different activities. The alveolar  $\text{CHCl}_3$  concentration increased 20 times for swimmers after 1 h swimming and only 8 times for workers after 2 h work shift. Similarly, Fantuzzi et al. (2001) found that the alveolar  $\text{CHCl}_3$  concentrations of pool trainers were almost doubled compared to those worked as reception participants and engine room technicians (25.1 vs.  $14.8 \mu\text{g}/\text{m}^3$ ).



**Fig. 4.** The relationship between  $\text{CHCl}_3$  concentrations in ambient air and alveolar air of swimmers. Other studies include (Catto et al., 2012; Fantuzzi et al., 2001, 2010; Kogevinas et al., 2010; Lévesque et al., 2000; Lourencetti et al., 2012; Marina et al., 2009; Richardson et al., 2010).

The THM concentrations in alveolar air were highest for swimmers followed by those bathing in the pools without exercise and were lowest for those standing beside the pools (Lourencetti et al., 2012). It is worth noting that  $\text{CHCl}_3$  concentration in breathing zones of swimmers (i.e., boundary layer) was around 40 times higher than that in the ambient indoor air, which indicates that inhalation of  $\text{CHCl}_3$  by swimmers is more substantial than other non-swimmers (Chen et al., 2011). A good linear correlation between the  $\text{CHCl}_3$  concentrations in alveolar and ambient air further demonstrates the significance of inhalation for human exposure (Fig. 4). A strong correlation between the total THM levels in ambient air and exhaled breath of pool attendants have been reported as well (Fantuzzi et al., 2001). The  $\text{CHCl}_3$  concentration in the alveolar air reached up to  $124 \mu\text{g}/\text{m}^3$  after 1 h leisure activity, however  $376 \mu\text{g}/\text{m}^3$  for competition swimmers (Lévesque et al., 2000). Lourencetti et al. (2012) found that physical activity enhanced THM exposure by comparing swimmers with swimming activities and volunteers with bathing only. Intensive exercises speed up swimmers' breathing and THM mass transfer, and therefore enhance THM exposure via inhalation. In brominated pools,  $\text{CHBr}_3$  in swimmers' alveolar air reached up to  $\sim 10 \mu\text{g}/\text{m}^3$  and showed a correlation with its corresponding ambient air concentrations as well (Lourencetti et al., 2012). Overall, human exposure reflected by alveolar air mainly attributes to the THM concentration in ambient air and water, exposure time and human activities.

Urine is another matrix for human exposure evaluation. Aprea et al. (2010) found the  $\text{CHCl}_3$  concentration in urine increased from  $0.123$  to  $0.404 \mu\text{g}/\text{L}$  after some water activities. Caro and Gallego (2007) found the  $\text{CHCl}_3$  and  $\text{CHBrCl}_2$  concentrations in human urine were around  $0.5$  and  $0.02 \mu\text{g}/\text{L}$  respectively before any exposure in swimming pool environment. The concentrations increased 3 times after 1 h swimming and only 2 times after 2 h work shift. Even after 4 h work shift, the concentration cannot reach as high as that after swimming (Caro and Gallego, 2007, 2008; Caro et al., 2007). Both swimmers and workers suffer from THM exposure via inhalation, however, swimmers with more strenuous exercises than workers inhale more air with higher THM concentration (just above the water level). In addition, swimmers exposed directly in SPWs experienced two additional exposure routes, i.e., dermal absorption and ingestion. Fig. 5 shows that the staffs working near the pool (e.g., monitors and pool guardians) experienced much higher  $\text{CHCl}_3$  exposure than those working far away (e.g., receptionists and maintenance technician), which indicates the importance of the relative position to THM exposure. Fig. 5 also shows a good linear relationship between  $\text{CHCl}_3$  concentrations in SPWs and urine.



**Fig. 5.** The relationship between  $\text{CHCl}_3$  concentrations in swimming pool water and in the urine of swimmers after 1 h swimming and workers after 2 h work shift.

Data are from (Caro and Gallego, 2007, 2008).

#### 4.1.2. HAAs

The more polar HAAs are less volatile and permeable to skin, and therefore have a higher potential of exposure by ingestion (Xu et al., 2002). HAA concentrations in SPWs (Table 6) are generally much higher than those reported in DWs (Richardson et al., 2007), thus even a small amount of SPW ingestion may result in a substantial intake. Cardador and Gallego (2010) developed a sensitive and straightforward method (headspace gas chromatography - mass spectrometry, HS-GCMS) to detect HAAs in human urine with detection limits as low as  $\text{ng}/\text{L}$ , which established a technique foundation for the further investigation of HAA exposure among humans. Later these authors tested the urinary HAA concentrations of three groups of subjects to distinguish the respective role of three exposure pathways (Cardador and Gallego, 2011). They found that ingestion ( $\sim 94\%$ ) outcompeted inhalation ( $\sim 5\%$ ) and dermal absorption ( $\sim 1\%$ ) and became a major exposure route. The finding that the urinary HAA concentration of the testers after 1 h swimming was more than an order of magnitude higher than that after 2 h work shift further validates ingestion as a more decisive exposure route than inhalation (Cardador and Gallego, 2011). More specifically, TCAA, DCAA and MCAA were present in swimmers' urine with concentrations of  $\sim 4.4$ ,  $\sim 2.3$ , and  $\sim 0.56 \mu\text{g}/\text{L}$  respectively after 1 h swimming in indoor pools. Font-Ribera et al. (2016) similarly reported the urinary TCAA increased from  $3.9$  to  $12.3 \mu\text{g}/\text{L}$  after 40 min swimming in a chlorinated indoor pool. Urinary TCAA seems a better biomarker for ingestion exposure evaluation due to its longer biological half-life than other HAAs (Bader et al., 2004; Kim et al., 1999).

HAAs regardless of the low volatility were still detectable in air phase with a concentration range of  $5\text{--}64 \mu\text{g}/\text{m}^3$ , owing to a great amount of aerosols (reflected by the humidity) in some indoor swimming pools (Pereira et al., 2012). Workers around the pools without touching SPWs still can be exposed via inhalation of HAA-contaminated aerosols. Cardador and Gallego (2011) reported that DCAA and TCAA reached  $\sim 300$  and  $\sim 120 \text{ng}/\text{L}$  in the urine of workers after 2 h work shift in indoor swimming pools. These values were several times higher than those reported in outdoor pools and the exposure increased with time (2 h vs. 4 h as shown in Fig. 6A, B). It indicates that sufficient ventilation in indoor pools is of high importance to ensure workers' safety. The linear relationship between HAA concentrations in human urine and SPWs as shown in Fig. 6 offers the possibility to estimate the human HAA exposure by known HAA concentration in SPWs (Cardador and Gallego, 2011; Cardador and Gallego, 2010). Hence, HAA exposure by pool participants varied with a number of factors, e.g., HAA concentrations in SPWs, pool types (indoor or outdoor), exposure time, human activities, etc.

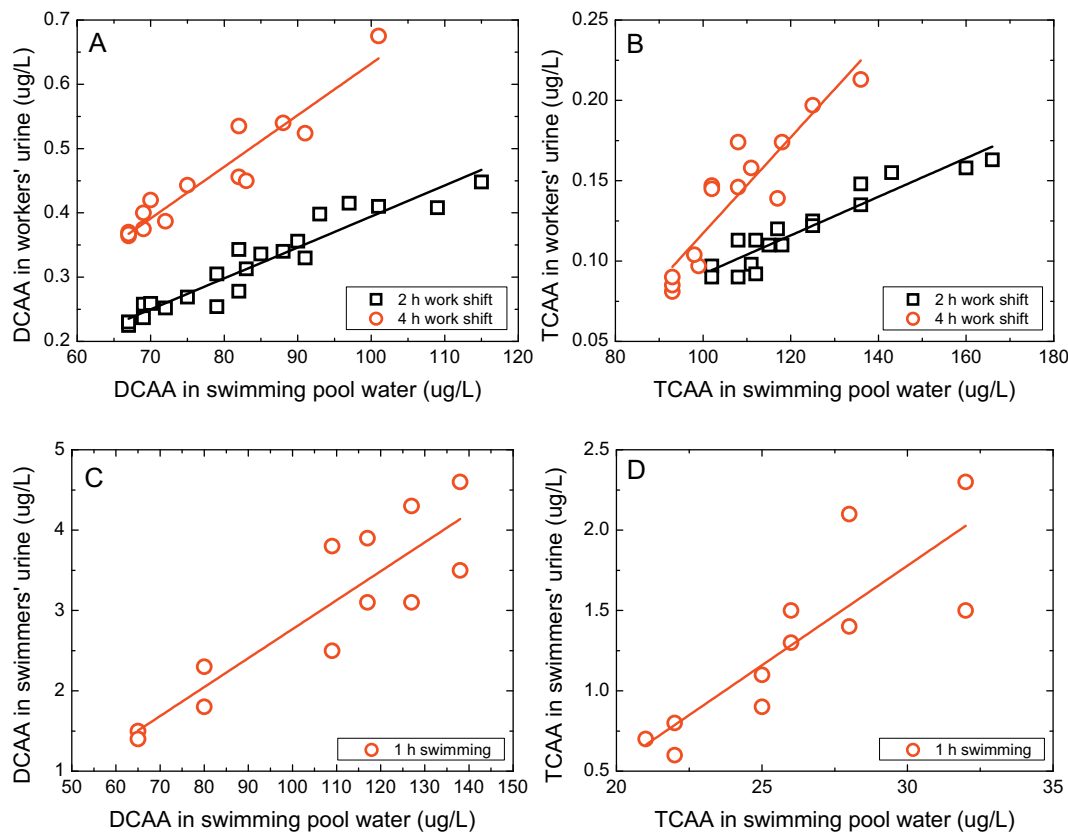


Fig. 6. The relationship between HAA concentrations in SPWs and human urine. Data are from (Cardador and Gallego, 2011; Cardador and Gallego, 2010).

#### 4.1.3. Other DBPs

Apart from these commonly mentioned regulated DBPs (i.e., THMs and HAAs), other DBPs in SPWs may also have the potential to be absorbed via human skin during swimming. For example, Xiao et al. (2012) newly identified some halo(nitro)phenols in SPWs, among which 2,4-dibromophenol, 2,4-dichlorophenol, and 2-bromophenol have the skin permeability of 0.031, 0.021, and 0.023 cm/h, respectively. Haloketones have a skin permeation range of 0.024–0.043 cm/h which is more than one order of magnitude higher than HAAs (0.001–0.003 cm/h) (Xu et al., 2002). The permeability of haloketones triples with the temperature increased from 20 to 40 °C. These permeation studies may further promote researchers' interests of quantifying these unregulated DBPs in human matrix for exposure estimation. A comprehensive identification and characterization of DBP mixture in SPWs is essential for human exposure assessment.

#### 4.2. The relevance between pool activities and health risks

Health risks of pool activities are commonly assessed by the association with THMs due to their sufficient data availability. The prevalence of health symptoms including red eyes, itchy eyes, dyspnea/asthma, and blocked nose was positively linked to THM concentration in alveolar air ( $> 21 \mu\text{g}/\text{m}^3$ ) based on a survey in twenty indoor pools (Fantuzzi et al., 2010). Kogevinas et al. (2010) mentioned that urine mutagenicity correlated proportionally with exhaled  $\text{CHBr}_3$  ( $p = 0.004$ ) and the frequency change of micronucleated lymphocytes correlated with Br-THMs ( $p \leq 0.05$ ). Rahman et al. (2010) found that the increase of THM concentration and exposure duration enhanced the risk of colorectal cancer. Panyakapo et al. (2008) assessed the cancer risk of swimmers exposed to THM-contaminated SPWs as  $7.99 \times 10^{-4}$  in average which exceeds the acceptable level based on U.S. EPA standard, while only  $2.19 \times 10^{-5}$  for non-swimmers. Lee et al. (2009) assessed

the lifetime cancer risk via THM inhalation during swimming in a range of  $7.77 \times 10^{-4}$  to  $1.36 \times 10^{-3}$  based on a study of 183 indoor swimming pools in Korea. The cancer risk via THM exposure was attributed mainly by swimming activities (~94.2%), compared to a relatively low percentage of 4.1% for tap water intake (Panyakapo et al., 2008). Similarly, Chowdhury (2015) found that the cancer risk of THM exposure in SPWs was more than four times higher than that in DWs. Villanueva et al. (2007b) reported 23% of the total THM uptake from swimming and only 1% from DWs for pregnant women.

Pool attendants are eventually exposed to a mixture of varieties of DBPs in aqueous and gaseous phases during pool activities. Many researchers directly assessed the relevance of health symptoms with pool activities. Suffering of dermatitis by swimmers in brominated swimming pools has been reported (Pardo et al., 2007). Fantuzzi et al. (2010) conducted a survey to record the declared health symptoms (e.g., ocular, cutaneous, asthma-related and other respiratory symptoms) among pool workers. It showed that workers in swimming pools suffered frequently from cold (65.4%), sneezing (52.6%), red eyes (48.9%) and itchy eyes (44.4%). Workers with the frequent touch of SPWs, e.g., lifeguards and trainers, were much easier to catch symptoms like red eyes, runny nose, voice loss and cold, than other pool employees (e.g., office and cafe). Villanueva and Font Ribera (2012) pointed out that pool participants had higher risks of suffering bladder cancer and asthma. Villanueva et al. (2007a) found that the risk of suffering bladder cancer increased with swimming activities and the duration of exposure to THM-contaminated water. Bernard et al. (2008) similarly found that the increase of exposure time in pools increased the prevalence of asthma, respiratory allergies or airways inflammation for the adolescents. However, Font-Ribera et al. (2009) pointed out that swimming pool attendance in childhood was associated with slightly lower prevalence of current asthma. The controversial association between swimming activities and health risks needs further investigation.

A comprehensive consideration of all existed DBPs in SPWs and their associated health effects will be of great importance for the overall risk assessment.

## 5. DBP treatment

The high frequency of occurrence and high concentration of DBPs in SPWs demonstrate that the conventional pool water treatment system (flocculation-sand filtration-chlorination) cannot effectively remove DBPs or DBP precursors to achieve good water quality. Some novel DBP control processes, such as membrane filtration, AOPs, and biodegradation, are beneficial to improve the chemical safety of pool participates. In this section, we review several advanced DBP treatment approaches reported in the past two decades.

### 5.1. Membrane filtration

Membrane has been widely used in the water and wastewater treatment and seawater desalination with its maturely developed technology and operation. The enhanced safety awareness towards DBPs in recent years promotes membrane filtration as a potential technology for DBP treatment. Laboratory experiments reveal that DBP treatment by forward osmosis (FO), nanofiltration (NF), and reverse osmosis (RO) has achieved good results. Kong et al. (2014) for the first time used FO to treat 9 HAAs and found that the rejection of each HAA reached > 94.6% for the orientation of active layer facing the feed water using 1 mol/L NaCl as the draw solution. The rejection of five regulated HAAs reached 90–100% by a dense negatively charged NF membrane (Chalati et al., 2009). Kimura et al. (2003) found 91–96% of DCAA and TCAA were removed by NF/RO membranes. Yang et al. (2017a) also showed that the rejection of 9 HAAs reached equal or higher than 90% by commercial NF/RO membranes (XLE, NF90 and SB50). At usual solution pHs of SPWs (7.2–7.8), RO/NF membranes, typically of polyamide or cellulose acetate chemistry, are generally negatively charged. HAAs with pKa values between 0.05 and 2.73 are present mainly in the form of HAA anions. The charge repulsion between membranes and HAAs, in addition to size exclusion, contributes significantly to the consistently high HAA rejection in the above studies. Uyak et al. (2008) found that the rejection of total THMs reached as high as 95% by NF membranes. The rejection of individual THM followed the order of  $\text{CHClBr}_2 > \text{CHCl}_2\text{Br} > \text{CHCl}_3$ , attributed to the role of the molecular size. Xu et al. (2005) found that high initial rejections of  $\text{CHCl}_3$  (> 80%) and  $\text{CHBr}_3$  (> 90%) by 3 NF/RO membranes was attributed to the effect of adsorption (hydrophobic-hydrophobic solute-membrane interactions) in addition to size exclusion. The rejections were reduced to 20–35% and 35–45%, respectively, at steady state conditions. Doederer et al. (2014) reported a wide range of rejections (i.e., 5–100%) for 29 DBPs (e.g., THMs, I-THMs, HANs, HNMs, chloral hydrate, halo ketones, and haloacetamides) by NF/RO membranes. These rejection data correlated well with the intrinsic DBP molecular properties (e.g., polar surface area, molecular volume, molecular weight, dipole moment, etc.) and operational parameters (e.g., temperature and flux) by a multiple linear regression method. Molecular properties were found to have greater impacts on DBP rejection than operational parameters (polar surface area > molecular volume > dipole moment > temperature > flux).

In addition to bench-scale experiments, Klüpfel et al. (2011) applied NF with the membrane installed at the outlet of a traditional sand filter in a by-pass to investigate its effect on a large pool. The rejections of DBPs and DBP precursors reached as high as 80% and 70%, respectively. High rejections (86–94%) for charged HAAs and low rejections (as low as 55%) for neutral and low-molecular-weight DBPs (e.g.,  $\text{CHCl}_3$  and  $\text{CHBrClCN}$ ) were shown in a pilot-scale RO plant in California (Agus and Sedlak, 2010). Fujioka et al. (2012) reviewed NDMA removal based on the findings in some laboratory-, pilot- and full-scale RO plants and found significantly different rejections (from negligible up to

86%) caused by the varied operating parameters, e.g., feed solution temperature and pH, ionic strength, and membrane permeate flux.

Removal of DBP precursors by membrane filtration could be an effective method to minimize DBP formation at source. Sentana et al. (2010) found that NF membranes reduced the formation potential of HAAs and THMs (deemed as HAA-FP and THM-FP) by > 82%. The rejection of THM and HAA precursors reached 77–96% and 75–92%, respectively, by NF membranes under optimized conditions (Chellam, 2000). However, the high TOC rejection (71–94%) coupled with the extremely low bromide rejection (< 10%) shifted Cl-DBPs to Br-DBPs upon chlorination to permeate water (Chellam, 2000).

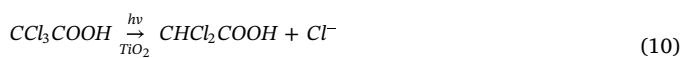
Existing studies demonstrate the potential of using membrane filtration for DBP treatment in SPWs. However, there are still several critical aspects to be further addressed: 1) the commonly used polyamide-based membranes show high DBP rejections but are sensitive to chlorine which is ubiquitously present in SPWs (Do et al., 2012a; Do et al., 2012b, c; Klüpfel et al., 2011). 2) The DBP concentrations used in laboratory experiments varied significantly which may produce completely different rejections. 3) The SPW matrix with the co-presence of dissolved organic matter and divalent cations (e.g., calcium) may lead to severe organic fouling and finally affect DBP rejection. 4) Special attention should be paid to the further treatment of DBP-accumulated retentate generated from the filtration process. Addressing these concerns will assist the full application of membrane technology for DBP treatment in SPWs.

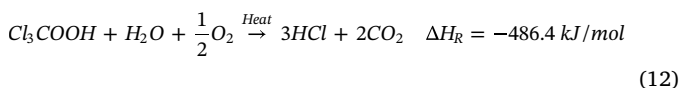
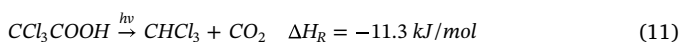
### 5.2. Advanced oxidation processes

The application of AOPs in the SPW treatment, often involving the use of UV,  $\text{H}_2\text{O}_2$  or  $\text{O}_3$  to generate hydroxyl radicals ( $\cdot\text{OH}$ ) for the degradation of organic matter, is promising to eliminate DBPs and DBP precursors.

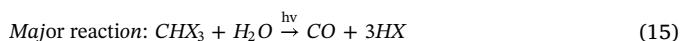
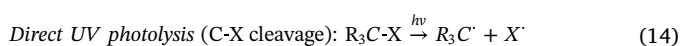
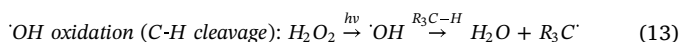
Direct sunlight is an economic natural source for DBP degradation in outdoor pools. Chen et al. (2010) investigated the photolysis of a variety of DBPs under natural solar irradiation and found that the stability of DBPs generally followed the order of N-DBPs < C-DBPs, Br-, I-DBPs < Cl-DBPs, T-DBPs < D-DBPs < M-DBPs (T-, D-, M-DBPs refer to tri-halogenated, di-halogenated, and mono-halogenated DBPs respectively). Some THMs and HAAs showed high persistence to solar irradiation. For example, 5–10% of THMs was removed after 6 h solar exposure (1150–1300  $\text{W}/\text{m}^2$  irradiation intensities), revealing that solar photolysis is insufficient for the degradation of these DBPs. It partly explains the high concentrations of DBPs especially non-volatile HAAs detected in outdoor pools (see Tables 5 and 6). The fate of  $\text{CH}_2\text{I}_2$ ,  $\text{CH}_2\text{Cl}$ , and  $\text{CH}_2\text{Br}$  under natural solar photolysis demonstrated that one iodide was substituted by one chlorine (Jones and Carpenter, 2005).

DBPs exposed under UV or visible light in the presence of catalyst undergo good photocatalysis degradation. Lifongo et al. (2004) found that HAAs had better photocatalysis degradation under visible light ( $\lambda > 400$ ) in the presence of  $\text{TiO}_2$ , compared to photolysis only (without  $\text{TiO}_2$ ). HAAs with a higher number of halogens had higher degradation rate constants and no obvious photolysis was observed for mono-halogen HAAs. Dehalogenation of HAAs was the main mechanism since the halide ions were produced during the reactions (for example, TCAA may be degraded to form DCAA and chloride ions (Eq. (10))). HAAs under high temperatures underwent thermal degradation as well (Lifongo et al., 2004). Spangenberg et al. (1996) have proposed two possible degradation mechanisms for TCAA: 1) photolysis by radical decarboxylation to form  $\text{CHCl}_3$  and  $\text{CO}_2$  (Eq. (11)); 2) pyrolysis by dehalogenation to form HCl and  $\text{CO}_2$  (Eq. (12)). The commonly used metal catalysts (e.g. Cu, Fe, Mn, Ti, V, Mo and Zn) showed no direct effect for HAA degradation (Lifongo et al., 2010).





UV/H<sub>2</sub>O<sub>2</sub> based AOPs are widely used in the treatment of DBPs. Rudra et al. (2005) found that 70 min UV irradiation completely degraded THMs except for CHCl<sub>3</sub> (46% removal) using an initial concentration of 50 µg/L for each THM. The addition of 0.1% H<sub>2</sub>O<sub>2</sub> significantly enhanced CHCl<sub>3</sub> degradation to 92%. However, only 10–12% THMs were removed by 0.1% H<sub>2</sub>O<sub>2</sub> solution without UV exposure, indicating sole H<sub>2</sub>O<sub>2</sub> to be a mild oxidizer. The UV/H<sub>2</sub>O<sub>2</sub> provides oxidation via ·OH generation by H<sub>2</sub>O<sub>2</sub> photolysis, which is consistent with ·OH dependent degradation of Cl-DBPs (Eq. (13)) (Jo et al., 2011; Rudra et al., 2005). Jo et al. (2011) found that the removal of Br-THMs and Br-HAAs reached > 80% at a UV dose of 1200 mJ/cm<sup>2</sup> in the presence of 6 mg/L H<sub>2</sub>O<sub>2</sub> and the removal efficiency increased with the increased bromine number in the molecules, e.g., CHBr<sub>3</sub> > CHBr<sub>2</sub>Cl and TBAA > DBAA. The degradation of Br-DBPs mainly rely on UV hydrolysis (see Eq. (14), where X refers to a halogen atom) in the UV/H<sub>2</sub>O<sub>2</sub> process. This UV hydrolysis mechanism is attributed to high UV absorption by Br-DBPs and enough energy from UV photon (113 kcal/mol at 253.7 nm) for the cleavage of C–Br bond (70.4 kcal/mol for CHBr<sub>3</sub>) (Jo et al., 2011). Xiao et al. (2014) performed degradation experiments of I-THMs by UV 254 irradiation. The I-THMs underwent a rapid first-order photodegradation process via the cleavage of carbon-halogen bond, with reaction rate constants (in the range of 0.1–0.6 min<sup>−1</sup>) proportional to their corresponding molar extinction coefficients (an indicator of UV absorption). End-product identification revealed that THMs were mainly converted to hydrogen halide (92–109% recovery) and also produced minor HCOOH (5% carbon conversion) as shown in Eqs. (15) and (16). Similar observation was reported by Kwok et al. (2004). The degradation rate constants of I-THMs in UV/H<sub>2</sub>O<sub>2</sub> system increased to > 2 min<sup>−1</sup>, indicating the ·OH promoted photolysis in addition to direct UV photolysis (Xiao et al., 2014). The combined UV radiation and ultrasound sonolysis (UV-US) showed a greater degradation rate for TCAA than UV or US alone, attributed to dual effects of free radical excitation by UV and thermal degradation by US (Wu et al., 2001). NDMA was degradable under UV with a reaction rate constant up to 0.36 min<sup>−1</sup> (Sharpless and Linden, 2003). While in the presence of chlorinated dimethylamine and monochloramine, UV irradiation contributes to simultaneous NDMA photolysis and formation (Soltermann et al., 2013). Although the addition of H<sub>2</sub>O<sub>2</sub> to UV treatment has usually been applied to improve NDMA removal, Sharpless and Linden (2003) reported that the addition of H<sub>2</sub>O<sub>2</sub> slightly assisted or seriously hindered NDMA degradation due to the competing effects of light screening by H<sub>2</sub>O<sub>2</sub> and ·OH promoted NDMA degradation.



Some unregulated but potentially more toxic nitrogenous DBPs are degradable by AOPs as well. HNMs underwent homolysis or heterolysis to produce halides, nitrite, nitrate, and/or carbon dioxide under UV254 radiation (Fang et al., 2013). Neutral to alkaline pHs which are typical for SPWs, contribute to HNM degradation due to their high UV molar absorptivity at these pHs. Two nitrogenous DBPs, *N*-nitrosopyrrolidine and *N*-nitrosopiperidine, were almost completely photodegraded (> 99%) after only 5 min UV 254 exposure (Xu et al., 2009). The N–N bond fission of these two compounds was proposed as the first

photodegradation step followed by the further degradation to form aliphatic amines.

AOPs are also used to reduce the amount of DBP precursors and thus control the DBPs in the treated water. Combined AOPs, i.e., O<sub>3</sub>/UV and O<sub>3</sub>/H<sub>2</sub>O<sub>2</sub>, showed better elimination of DBP precursors (using TOC, AOX-FP, THM-FP, and/or HAA-FP as indicators) than O<sub>3</sub> and UV alone for both SPW and surface water treatment (Chin and Bérubé, 2005; Glauner et al., 2005a). The removal efficiency of TOC, THM-FP and HAA-FP after 1 h O<sub>3</sub>/UV treatment reached 50%, 80% and 70%, respectively, and reaction rate constants were 0.04, 0.12 and 0.15 min<sup>−1</sup>, respectively (Chin and Bérubé, 2005). Wang et al. (2000) found UV/H<sub>2</sub>O<sub>2</sub> as an effective method for the remediation of a group of important DBP precursors, i.e., humic acids (~90% removal within 1 h with a reaction rate constant of 0.037 min<sup>−1</sup>).

In conclusion, considerable laboratory studies reviewed above have demonstrated the effectiveness of AOPs used for DBP degradation via means of dehalogenation, decarboxylation, and others, depending on the specific AOPs and DBP species. Nevertheless, there are still some important concerns or limitations we need to pay attention to. It is essential to optimize H<sub>2</sub>O<sub>2</sub> dosage to the UV/H<sub>2</sub>O<sub>2</sub> system as an excess addition may result in a scavenging effect to the hydroxyl radical or light screening, eventually decreases the removal efficiency of DBPs or DBP precursors (Jo et al., 2011; Rudra et al., 2005; Sharpless and Linden, 2003; Wang et al., 2000). Humic acids, nitrate, chloride, and sulphate commonly detected in SPWs may affect the DBP photodegradation efficiency via shielding or activating ·OH (Chen et al., 2010; Xiao et al., 2014). In addition, the application of medium-pressure UV lamps in an indoor chlorinated public swimming pool could even result in a significant increase of THMs, particularly CHCl<sub>3</sub> and CHBrCl<sub>2</sub>, due to the increase in active chlorine and radical initiation by UV radiation (Cassan et al., 2006). Therefore, the systematic understanding of real SPW characteristics and the fundamental reaction mechanisms are critical to ensure the effective AOP applications in real SPWs. Furthermore, the comprehensive identification of end-products after AOPs is essential as DBPs are commonly degraded to a variety of small weight compounds with unknown or even higher toxicity (Glauner et al., 2005b; Xu et al., 2009). Hence, it is far from enough to obtain a high degradation rate. An overall risk assessment for the degradation process is essential.

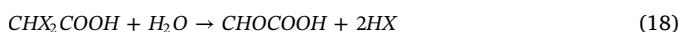
### 5.3. Biodegradation

Biodegradation is another potential technique for the removal of DBPs in water systems. HAAs are shown to be biodegradable under conditions of interest (Chuang et al., 2011; Pluchon et al., 2013; Tung and Xie, 2009). Chlorine < 0.3 mg/L and heterotrophic bacteria > 10,000 HPC/mL were necessary conditions for effective HAA removal (Chuang et al., 2011; Tung and Xie, 2009). Chuang et al. (2011) observed the removal of MCAA and DCAA reached up to 80% by sand filtration. The bioactivity on the sand surface made a significant contribution to the HAA removal due to the biofilm development under perennial high temperature. Nevertheless, Yang et al. (2016) still observed an average HAA concentration of ~1000 µg/L in a public campus pool treated with sand filtration. These contradictory findings might be explained by the difference of chlorine residuals in these two systems. The average chlorine concentration of 0.57 mg/L in the campus pool in Yang's study (higher than the threshold 0.5 mg/L) may deactivate the corresponding bio-degraders and thus prevent effective HAA biodegradation (Chuang et al., 2011). The inverse relationship between heterotrophic bacteria and HAA concentrations under low chlorine concentration further emphasized the important role of bacteria for HAA degradation (Tung and Xie, 2009). Bacteria, e.g., *S. natatoria* and *P. donghaensis*, have been isolated and proven to be responsible for HAA degradation (Chuang et al., 2011). Other parameters including water temperature, retention time, the composition of organic matter, etc., also affect HAA degradation (Pluchon et al., 2013).

On the contrary, biodegradation may be far less effective for THMs. A recent study by [Tung and Xie \(2009\)](#) even reported a proportional relationship between bacteria and THM concentration in some water distribution systems. The application of DBP biodegradation in SPWs still has a long way to go. The specific SPW matrix in particular constant high chlorine (e.g., 1–3 mg/L) may inhibit the growth of some contributing bacteria. It is worthwhile to find robust bacteria which are DBP biodegradable and stable under high chlorine conditions.

#### 5.4. Thermal degradation and chemical reduction

[Lifongo et al. \(2010\)](#) explored the thermal degradation of HAAs and found trihalogenated haloacetic acids (T-HAAs) were degraded by decarboxylation (as shown in Eq. (11) for TCAA), via C–C bond crack. The higher activation energy of decarboxylation reaction (C–C break) for TCAA compared to TBAA (151 vs. 139 kJ/mol/k) was consistent to its lower reaction rate constant ( $4.80 \times 10^{-10}$  vs.  $7.77 \times 10^{-8} \text{ s}^{-1}$ , at 15 °C). Monohalogenated and dihalogenated haloacetic acids (M-HAAs, D-HAAs) were hydrolysed via oxygen attack to the  $\alpha$ -carbon of the molecules, producing glycolic and glyoxalic acids, and its corresponding hydrogen halide (as shown in Eqs. (17) and (18), X represents halogens). The reaction rate for decarboxylation followed an order of T-HAAs > D-HAAs > M-HAAs, as halogen attachment to the  $\alpha$ -carbon weakens the C–C bond. [Zhang and Minear \(2002\)](#) found the temperature-dependent decomposition of T-HAAs produce the corresponding THMs via decarboxylation.



Zero-valent iron ( $\text{Fe}^0$ ) in the iron pipe system is an effective substance to degrade HAAs by dehalogenation ([Hozalski et al., 2001](#); [Tang et al., 2013](#)). Debromination happened prior to dechlorination for the mixed chloro-bromo species, e.g., BDCAA, and therefore Br-HAAs were much easier to be removed than chlorinated ones (reaction rate constants followed  $\text{BDCAA} > \text{CDBAA} \approx \text{TBAA} \gg \text{TCAA}$  as shown in [Table 7](#)) ([Hozalski et al., 2001](#)). [Tang et al. \(2013\)](#) found that DCAA and MCAA as the subsequent dehalogenation products of TCAA by  $\text{Fe}^0$  were completely biodegraded via a subsequent active carbon treatment process with a retention time of 10 min. [Korshin and Jensen \(2001\)](#) observed complete dehalogenation of Br-HAAs and partial dehalogenation of Cl-HAAs (produced notable MCAA whose direct reduction was difficult) by an electrochemical reduction method using copper or gold as the electrode. Other DBPs, e.g., chloropicrin, trichloroacetonitrile, and dibromoacetonitrile, also underwent dehalogenation in the presence of sulphite as a reductant ([Croue and Reckhow, 1989](#)).

**Table 7**  
HAA degradation rate constant.

	HAA degradation rate constant ( $\text{min}^{-1}$ )									References
	MCAA	MBAA	DCAA	TCAA	BCAA	DBAA	BDCAA	DBCAA	TBAA	
Solar photolysis	3.48E-05	1.87E-04	1.18E-05	4.00E-06	6.34E-05	3.48E-04	2.20E-05	1.18E-04	6.34E-04	<a href="#">(Chen et al., 2010)</a> <a href="#">(Lifongo et al., 2004)</a>
Photocatalysis by solar light + $\text{TiO}_2$	1.15E-05	7.68E-06	4.61E-05	7.68E-05	2.69E-05	3.46E-05			5.76E-05	
AOP by $\text{H}_2\text{O}_2$ + UV	3.48E-02	6.60E-02	3.00E-02	1.74E-02		7.80E-01			3.84E+00	<a href="#">(Jo et al., 2011)</a> <a href="#">(Jo et al., 2011)</a>
AOP by UV		1.26E-01				9.00E-01			4.44E+00	
Biodegradation by rapid sand filtration	1.70E-01		1.50E-01							<a href="#">(Chuang et al., 2011)</a>
Reduction and biodegradation by $\text{Fe}^0$ + BAC	4.00E-01		4.00E-01	5.00E-01						<a href="#">(Tang et al., 2013)</a>
Reduction by $\text{Fe}^0$				1.33E-03			1.77E-01	2.38E-02	2.35E-02	
Thermal degradation at 82–83 °C	8.94E-05	1.39E-03	2.69E-05		2.36E-04	1.78E-04				<a href="#">(Lifongo et al., 2010)</a>

#### 5.5. Hybrid process

The investigation of multi-stage DBP purification in SPWs is of great interests. For a municipal swimming pool in France, a hybrid process (hollow fibre ultrafiltration (UF) + granular activated carbon adsorption) was included after a conventional SPW treatment system (coagulation-sand filtration-disinfection) ([Barbot and Moulin, 2008](#)). This 18-month study illustrated that the hybrid process limited the combined chlorine (as an indicator of DBPs) to be lower than 0.35 mg/L. [Glauner et al. \(2005b\)](#) used a two-step membrane filtration (UF followed by NF) to separate the compounds into three categories, i.e., < 200, 200–1000, > 1000 g/mol. DBPs with molecular weight < 1000 g/mol accounted for > 80% in both indoor and outdoor SPWs. The extract with lower molecular weight had higher genotoxicity. It is essential to use membranes with molecular weight cut-offs (MWCOs) < 200 g/mol to minimize its potential toxicity. Meanwhile, [Glauner et al. \(2005a\)](#) also attempted to apply UF or NF prior to AOPs for the treatment of SPWs and found that the overall eliminations of DBPs and DBP precursors reached up to 80%. The  $\text{Fe}^0$ -active carbon treatment process contributed to TCAA dehalogenation by  $\text{Fe}^0$  and the subsequent complete biodegradation of DCAA and MCAA (as the dehalogenated products of TCAA) by active carbon within 10 min ([Tang et al., 2013](#)). The hybrid processes can potentially substitute or supplement the conventional SPW treatment process to achieve better water quality. However, a full-scale application needs further validation, e.g., the stability of long-term operation, membrane fouling-induced side-effects.

## 6. Conclusions

Previous research has mainly focused on the DBPs in the context of DWs subject to regulations by some global organizations or local government (e.g., EPA, WHO). Unlike DWs, SPWs are more complicated and may present higher DBP-related health risks due to the multi-various organic inputs from the bathers (body fluids and PCPs) in addition to NOM from source water. This paper critically reviewed the latest research progress on the regulation, formation, exposure, and treatment of DBPs in the context of SPWs, with a total of ~200 references. DBPs in SPWs, with the main focus on HAAs and THMs, are often much or several orders of magnitude higher than the MCLs in DWs or SPWs. A more comprehensive DBP identification targeting at SPW context is necessary. Swimmers and workers are exposed to THMs mainly via dermal absorption and inhalation and HAAs via ingestion, reflecting by DBPs detected in human matrix including exhaled air, urine, blood, and plasma. Membrane filtration, AOPs, biodegradation, thermal degradation, chemical reduction, and some hybrid processes are the latest approaches for the control and treatment of DBPs. The

removal efficiency, possible mechanisms and future challenges of these DBP treatment methods have been discussed to facilitate their full-scale applications and to provide potential directions for further research roadmap.

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