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Do DBPs swim in salt water pools? Comparison of 60 DBPs formed by electrochemically generated chlorine vs. conventional chlorine ☆

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ABSTRACT

Disinfectants are added to swimming pools to kill harmful pathogens. Although liquid chlorine (sodium hypochlorite) is the most commonly used disinfectant, alternative disinfection techniques like electrochemically generated mixed oxidants or electrochemically generated chlorine, often referred to as salt water pools, are growing in popularity. However, these disinfectants react with natural organic matter and anthropogenic contaminants introduced to the pool water by swimmers to form disinfection byproducts (DBPs). DBPs have been linked to several adverse health effects, such as bladder cancer, adverse birth outcomes, and asthma. In this study, we quantified 60 DBPs using gas chromatography-mass spectrometry and assessed the calculated cytotoxicity and genotoxicity of an indoor community swimming pool before and after switching to a salt water pool with electrochemically generated chlorine. Interestingly, the total DBPs increased by 15% upon implementation of the salt water pool, but the calculated cytotoxicity and genotoxicity decreased by 45% and 15%, respectively. Predominant DBP classes formed were haloacetic acids, with trichloroacetic acid and dichloroacetic acid contributing 57% of the average total DBPs formed. Haloacetonitriles, haloacetic acids, and haloacetaldehydes were the primary drivers of calculated cytotoxicity, and haloacetic acids were the primary driver of calculated genotoxicity. Diiodoacetic acid, a highly toxic iodinated DBP, is reported for the first time in swimming pool water. Bromide impurities in sodium chloride used to electrochemically generate chlorine led to a 73% increase in brominated DBPs, primarily driven by bromochloroacetic acid. This study presents the most extensive DBP study to-date for salt water pools.

Introduction

In the United States, swimming is the fourth most popular recreational activity (U.S. Census Bureau, 2012). To limit swimmers' exposure to harmful viruses, bacteria, fungi, and algae, swimming pools are treated with disinfectants like chlorine, bromine, ultraviolet radiation (UV), or ozone (World Health Organization, 2006). However, these disinfectants react with natural organic matter (NOM) and anthropogenic contami-

☆ This manuscript is dedicated to Prof. Michael Plewa, whose innovative and pioneering research has been a catalyst for improving the safety of drinking water and pools.

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nants introduced to the pool water by swimmers to form disinfection byproducts (DBPs) (Carter and Joll, 2017; Zwiener et al., 2007; Kim et al., 2002; LaKind et al., 2010; Daiber et al., 2016; Allen et al., 2021). Several epidemiologic studies have linked DBPs to bladder cancer, birth defects, miscarriage, and respiratory issues such as asthma (Villanueva et al., 2007a; Cardador and Gallego, 2011; Villanueva and Font-Ribera, 2012; Fornander et al., 2013; Parrat et al., 2012; Wright et al., 2017; Bove et al., 2002; Font-Ribera et al., 2019; Richardson et al., 2007; Bernard et al., 2006). Furthermore, studies have shown that dermal exposure to halogenated DBPs is an important exposure route to consider in swimming pool studies, due to the permeability of some DBPs across the skin (Xu et al, 2002; Xiao et al., 2012).

Studies quantifying DBPs in swimming pools have primarily focused on trihalomethanes (THMs) and haloacetic acids (HAAs). However, with more than 700 DBPs identified to date, many of which are more cytotoxic, genotoxic, or carcinogenic than THMs and HAAs (Richardson and Plewa, 2020), there is a clear need for the expansion of the classes of DBPs quantified in swimming pools. In recent years, brominated and iodinated DBPs have become of particular interest due to their elevated levels of toxicity when compared to chlorinated DBPs (Richardson et al., 2007; Wagner et al., 2017; Yang et al., 2014). Additionally, nitrogenous DBPs (N-DBPs) are generally more toxic than carbonaceous DBPs (C-DBPs) (Plewa et al., 2008). Recent studies completed in the United States and Australia have expanded on the DBPs quantified in pools using a variety of disinfection techniques to include priority, unregulated DBPs such as iodinated trihalomethanes (I-THMs), iodoacetic acids (IAAs), haloacetaldehydes (HALs), haloketones (HKs), haloacetamides (HAMs), haloacetonitriles (HANs), and halonitromethanes (HNMs) (Allen et al., 2021; Carter et al., 2019). Despite chlorine being the most commonly used disinfectant in swimming pools around the world, alternative disinfection techniques are becoming more common. One such alternative disinfection technique is electrochemically generated chlorine (or salt water pools) which works by passing an electric current through a concentrated salt solution (sodium chloride) to produce hypochlorous acid (HOCl) and hypochlorite ions (OCl^-) as the primary oxidants (U.S. Army Center for Health Promotion and Preventive Medicine, 2006). Previous studies have shown that when compared to pools disinfected with chlorine, salt water pools had lower levels of HAAs (dichloroacetic acid and trichloroacetic acid) and trichloroacetaldehyde, but higher levels of Br-DBPs, likely due to bromide impurities in the sodium chloride (Lee et al., 2009, 2010). This increase in bromide is an important factor to monitor because previous studies show that dichloroacetic acid and trichloroacetic acid do not significantly contribute to the cytotoxicity or genotoxicity of pool waters (Yeh et al., 2014). However, there is currently no comprehensive DBP or toxicity data available that provides a direct comparison between conventional chlorine and salt water pools. To address this, a study of 60 DBPs was conducted at an indoor community pool in South Carolina while the pool was disinfecting with conventional liquid chlorine (sodium hypochlorite) and then after the implementation of electrochemically generated chlorine technology (salt water pools). Using DBP data collected over three sampling events, the calculated cytotoxicity and genotoxicity associated with each disinfection type was determined to better understand the impact each disinfection technology has on overall calculated toxicity, as well as the drivers of toxicity for each disinfection technique.

1. Materials and methods

1.1. Swimming pool sampling

Swimming pool samples were collected from an indoor community pool in South Carolina with an estimated total volume of 263,300 liters. This pool was chosen to study due to its consistent bather load (approximately 24 swimmers/day) throughout the year. The first sampling event occurred in May of 2021 when the swimming pool was using sodium hypochlorite (conventional chlorine) to disinfect the swimming pool. Two additional pool samples (November 2021 and January 2022) were collected after the implementation of an electrochemically generated chlorine system (Hayward Saline C 11.0 Commercial Salt Chlorine Generator; Rockville, MD) with a flow rate of 150 gallons per minute and a power supply rated to supply 72 amps. Additional details about the electrochemically generated chlorine system can be found in Appendix A Table S1.

Samples were collected in 1 L amber glass bottles, quenched with ammonium chloride, acidified to pH 3.5-4 with 1 mol/L sulfuric acid (for sample preservation), and filled headspace free. Samples were then shipped overnight on ice to the University of South Carolina and extracted immediately upon arrival. Further sample details can be found in Table 1.

1.2. Chemical and reagents

All solvents (methanol, metyl *tert*-butyl ether, acetonitrile, ethyl acetate) were of the highest purity and were purchased from Sigma-Aldrich (St. Louis, MO) or VWR International (Radnor, PA). General reagents were of ACS reagent grade and were purchased from Sigma-Aldrich and Fisher Scientific (Waltham, MA). DBP standards were purchased from CanSyn Chem. Corp. (Toronto, ON), Sigma-Aldrich, Aldlab Chemicals (Woburn, MA), and TCI America (Waltham, MA) at the highest purity. Specific vendor information can be found in Appendix A Table S2. The internal standard, 1,2-dibromopropane, along with the diazomethane derivatization reagents (Diazald, CARBITOL^{TM}) were purchased from Sigma-Aldrich.

1.3. DBP analysis

Quantification of 60 DBPs was performed in triplicate as described previously (Cuthbertson et al., 2020; Allen et al., 2021, 2022; Aziz et al., 2022; Li et al., 2021). In brief, 100 mL of a sample was placed into a 125 mL amber bottle and acidified to pH < 1 with concentrated sulfuric acid. Then, 5 mL of methyl *tert*-butyl ether was added to each sample along with 30 g of sodium sulfate. Samples were then shaken for 15 min, allowed to settle for 10 min, and the top organic layer was removed and placed into a test tube. This procedure was completed 2 more times for a total extract volume of 15 mL. The organic

Table 1 – Sampling ID (date, time, disinfectant technology used), water quality parameters (pH, residual Cl), estimated bather load, THM levels, HAA levels, and total DBPs.

Sample ID	Sample collection time	Disinfectant	pH	Residual chlorine (mg/L)	Exercise class	THMs (μg/L)	HAAs (μg/L)	Total DBPs (μg/L)
May sample (5/17/2021)	1:00 PM	12% liquid sodium hypochlorite	7.4	6.1	No, typical bather load	777	1066	2541
November sample (11/18/2021)	12:30 PM	Electrochemically generated chlorine*	7.5	3.2	No, typical bather load	322	1798	2613
January sample (1/12/2022)	1:10 PM	Electrochemically generated chlorine*	7.3	2.0	Yes, 8-10 participants	260	2425	3251

* salt water pool.

extract was then dried using sodium sulfate and concentrated to 200 μL using a gentle stream of nitrogen. The concentrated extract was spiked with 4 μL of an internal standard (1,2-dibromopropane) and split into two equal aliquots. The first aliquot was used to analyze for 4 trihalomethanes (THMs), 9 haloketones (HKs), 4 haloacetaldehyde (HALs), 4 halonitromethanes (HNMs), 7 haloacetonitriles (HANs), 13 haloacetamides (HAMs), and 6 iodinated trihalomethanes (I-THMs).

The second aliquot was derivatized using diazomethane for the analysis of 9 haloacetic acids (HAAs) and 4 iodoacetic acids (IAAs). Diazomethane derivatization converts carboxylic acids to methyl esters for analysis by gas chromatography (GC)-mass spectrometry (MS). The diazomethane derivatization was conducted as described by the U.S. Environmental Protection Agency Standard Operating Procedure (Richardson, 2009).In brief, 0.367 g of Diazald and 1.0 mL of CARBITOL™ were placed inside the inner tube of a diazomethane generator. Then, 3.0 mL of methyl *tert*-butyl ether was placed in the outer tube of the generator, and the entire generator was placed in ice. Once on ice, 1.5 mL of 37% potassium hydroxide was added slowly (dropwise) to the inner tube and allowed to react for 1 hr. After 1 hr, 50 μL of diazomethane (dissolved in methyl *tert*-butyl ether) was spiked into a 100 μL organic extract aliquot and allowed to react for 30 min. After 30 min, the reaction was quenched with 10 mg of silica gel and transferred to new vials before analysis.

1.4. GC-MS analysis

Both extracts were analyzed using a gas chromatograph-mass spectrometer (Agilent 7890 GC, 5977A mass spectrometer, Agilent Technologies, Santa Clara, CA) with electron ionization (EI) at 70 eV in selection ion monitoring (SIM) mode. Sample extracts (1.0 μL) were injected into a multimode inlet (MMI) in pulsed splitless mode. Analytes were chromatographically separated using a Restek Rtx-200 column (30 m × 0.25 mm × 0.25 μm film thickness; Restek Corporation, Bellefonte, PA). This column provides improved separation and detection limits for iodo-THMs and haloacetamides, which tend to tail and give lower responses using a DB-5 column (Cuthbertson et al., 2020). The GC temperature program for the analysis of the 4 THMs, 9 HKs, 4 HALs, 4 HNMs, 7 HANs, 13 HAMs, 6 I-THMs was as follows: initial temperature of 35 °C for 5 min, increased to 220°C at 9 °C/min, ramped at 20 °C/min to 280 °C, and held for 15 min. The GC temperature program for the analysis of the 9 HAAs and 4 IAAs was as follows: initial temperature held at 35 °C for 5 min, increased to 280 °C at 9 °C/min, and held for 15 min. Both methods held the transfer line at 280 °C and source temperature at 200 °C. Quantifier and qualifier ions along with limits of quantification (LOQ) for each DBP are listed in Appendix A Table S2.

1.5. Bromide and iodide measurements

To quantify the amount of bromide and iodide present in the sodium chloride used for salt water pools, a solid sodium chloride sample used at this community pool was collected and dissolved in ultrapure water for analysis. Bromide and iodide were quantified via a Dionex Integrion high performance ion chromatography (HPIC) system (Sunnyvale, CA) with an IonPac AS20 guard column and an IonPac AS20 analytical column. The system included a 500 μL sample loop and 50 mmol/L NaOH as the eluent. An external calibration curve was prepared in ultrapure water (1, 5, 10, 20, and 30 μg/L) using sodium bromide and sodium iodide. The limits of quantification (LOQs) for both bromide and iodide are 1.0 μg/L.

1.6. "TIC-Tox": calculated cytotoxicity and genotoxicity

"TIC-Tox" is a metric previously used in several studies to calculate cytotoxicity and genotoxicity of water samples and predict the drivers of toxicity Smith et al., 2010; Allard et al., 2015; Plewa et al., 2017; Cuthbertson et al., 2019; Carter et al., 2019; Allen et al., 2021; Aziz et al., 2022). In brief, "TIC-Tox" calculates cytotoxicity and genotoxicity by multiplying the molar concentration of each individual DBP by their corresponding cytotoxicity or genotoxicity index values for Chinese hamster ovary (CHO) cells reported in literature (Wagner and Plewa, 2017; Plewa et al., 2017). Each product is then multiplied by a normalization factor (10^6) and summed together (Eqs. (1) and 2).

$$\text{Total calculated cytotoxicity} = \sum \left([\text{DBP}] \times \text{LC}_{50}{}^{-1} \times 10^6 \right) \quad (1)$$

$$\text{Total calculated genotoxicity} = \sum \left([\text{DBP}] \times 50\%\text{TDNA}^{-1} \times 10^6 \right) \quad (2)$$

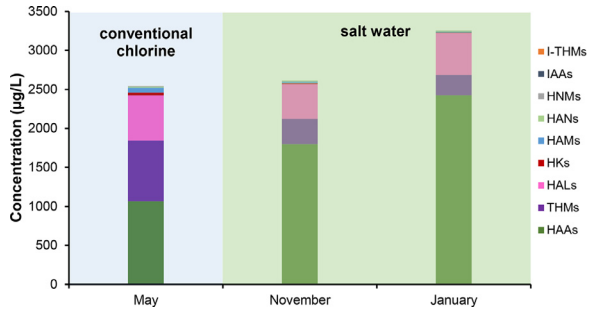

Fig. 1 – Total concentration of DBPs by class in conventional chlorine and salt water pool samples.

where LC_{50}^{-1} is the inverse of the lethal concentration at 50% in molarity (mol/L) and $TDNA^{-1}$ is the inverse of the 50% tail DNA measurement in molarity (mol/L). "TIC-Tox" assumes that the toxicity of individual DBPs is additive, an assumption shown to be accurate in a recently published study (Lau et al., 2020). Note that haloketones (HKs) are not included in "TIC-Tox" calculations because there are no cytotoxicity or genotoxicity index values currently available in the literature. Additional details about 'TIC-Tox" and the determination of cytotoxicity and genotoxicity index values can be found in previous studies (Wagner and Plewa, 2017; Plewa et al., 2017; Tice et al., 2000; Rundell et al., 2003).

2. Results and discussion

2.1. *Overall findings*

Upon implementation of an electrochemically generated chlorine system, there was a 15% increase in average total DBPs compared to the conventionally chlorinated pool sample. Of the 60 DBPs measured in this study, 68% were detected at least once. Table 2 shows the 60 DBPs quantified during each sampling event. The dominant DBP classes quantified were haloacetic acids (HAAs), followed by haloacetaldehydes (HALs), and trihalomethanes (THMs) (Fig. 1). HAAs, which accounted for 63% of the average total DBPs present, are known to accumulate in pools due to their lack of volatility (Carter and Joll, 2017; Simard et al., 2013; Daiber et al., 2016; Allen et al., 2021). Total HAA concentrations ranged from 1066 to 2425 µg/L and were dominated by Cl-HAAs (sum of chloroacetic acid, dichloroaceitc acid, and trichloroacetic acid). Dichloroacetic acid (DCAA) and trichloroacetic acid (TCAA) were the dominant HAAs detected with an average concentration of 1332 and 277 µg/L, respectively. This finding matches well with previously published data from our lab in which another indoor pool had DCAA and TCAA at levels as high as 1230 and 275 µg/L, respectively (Allen et al., 2021). Of the haloacetaldehydes quantified in this study, trichloroacetaldehyde (TCAL) was the most commonly detected, and accounted for 19% of the average total DBPs formed. Trichloromethane (TCM) was the third most abundant DBP quantified with an average concentration of 446 µg/L. Interestingly, the January salt water pool sample contained the highest level of total DBPs (3251 µg/L) despite having the lowest residual chlorine measured (Table 1). Prior to the January sampling, an exercise class was offered, thus the bather load was higher compared to the other two sampling events (Table 1). An increase in turbulence in the pool prior to sampling resulted in lower THMs levels (260 µg/L) but higher levels of non-volatile DBPs, particularly HAAs (2425 µg/L). This finding matches well with previous studies that noted as the turbulence in swimming pools increases, so does the THM concentration in air samples collected at indoor pools (Aggazzotti et al., 1998; Catto et al., 2012), thus, decreasing THM levels in the water.

Iodinated trihalomethanes (I-THMs), iodoacetic acids (IAAs), and halonitromethanes (HNMs) were present at the lowest levels. On average, these classes represented <1% of the average DBPs present in all pool samples. Trichloronitromethane (TCNM) was the most frequently detected HNM in pool samples, with average levels ranging from 2.1 µg/L in the salt water pool samples to 4.6 µg/L in the conventional chlorinated pool sample. The only I-THM detected in this study was bromodiiodomethane (BDIM), which was found in the conventional chlorinated pool sample at 0.5 µg/L. Iodoacetic acid (IAA) was also detected in the conventional chlorinated pool at 0.2 µg/L and diiodoacetic acid (DIAA) and chloroiodoacetic acid (CIAA) were detected in the January salt water pool sample, both at 0.3 µg/L. This is the first report of DIAA in swimming pool water and is a significant finding due to its elevated level of toxicity. For example, DIAA is 1.8 × more cytotoxic than DBAA. Ion chromatography analysis of the salt used in the salt water pool revealed that iodide was not present as an impurity, suggesting that the presence of the I-DBPs was the result of iodide in disinfected source water used to fill the pool.

2.2. *Conventional chlorine vs. salt water: C-DBPs*

2.2.1. *Haloacetic acids (HAAs)*

The 15% increase in the average total DBPs in the salt water pool samples was driven by HAAs. The January salt water pool sample had the lowest residual chlorine but the highest bather load prior to sampling. This increase in DBP formation was driven by dichloroacetic acid and trichloroacetic acid, which saw a 124% and 25% increase, respectively. Dichloroacetic acid accounted for 69% (730 µg/L) of the HAAs formed in the conventional chlorine pool and 77% (1633 µg/L) of the HAAs present in the salt water pool, a 124% increase. Trichloroacetic acid accounted for 22% (238 µg/L) of HAAs present in the conventional chlorine pool sample and 14% (297 µg/L) in salt water pool samples, a 25% increase. A previous study noted a similar increasing trend when comparing levels of dichloroacetic acid (196% increase) and trichloroacetic (229% increase) in conventional chlorine and salt water pools (Yeh et al., 2014).

2.2.2. *Haloacetaldehydes (HALs)*

The concentration of trichloroacetaldehyde (TCAL) was 580 µg/L in the conventional chlorine pool and 490 µg/L on average in the salt water pool, a 16% decrease. Lee et al. (2010) also noted a decrease (40%) in the formation of trichloroacetaldehyde between conventional chlorine and salt water pools. This decrease in trichloroacetaldehyde is an important finding when considering previous studies

Table 2 – DBPs quantified in conventional chlorine or salt water pools (μg/L).[a]

DBP class	Name	Abbreviation	Conventional chlorine	Salt water	
			May	November	January
HALs	Trichloroacetaldehyde	TCAL	580 ± 15.0	448 ± 10.7	532 ± 58.3
	Bromodichloroacetaldehyde	BDCAL	1.0 ± 0.0	ND	4.8 ± 0.2
	Dibromochloroacetaldehyde	DBCAL	0.2 ± 0.0	ND	0.1 ± 0.0
	Tribromoacetaldehyde	TBAL	ND	ND	0.1 ± 0.0
HANs	Trichloroacetonitrile	TCAN	0.6 ± 0.0	0.3 ± 0.0	0.5 ± 0.0
	Dichloroacetonitrile	DCAN	5.2 ± 0.3	5.4 ± 0.2	4.2 ± 0.4
	Chloroacetonitrile	CAN	6.3 ± 0.1	2.6 ± 0.2	1.4 ± 0.0
	Bromochloroacetonitrile	BCAN	0.2 ± 0.0	0.3 ± 0.0	0.5 ± 0.0
	Bromoacetonitrile	BAN	3.1 ± 0.0	ND	ND
	Dibromoacetonitrile	DBAN	0.2 ± 0.0	0.1 ± 0.0	0.4 ± 0.0
	Iodoacetonitrile	IAN	ND	ND	ND
	1,1-Dichloropropanone	11DCP	2.8 ± 0.1	ND	ND
HKs	Chloropropanone	CP	18.0 ± 1.0	4.6 ± 0.2	3.5 ± 0.4
	1,1,1-Trichloropropanone	111TCP	8.2 ± 0.3	1.0 ± 0.0	0.9 ± 0.0
	1,1-Dibromopropanone	11DBP	ND	ND	ND
	1-Bromo-1,1-dichloropropanone	1B11DCP	0.3 ± 0.0	0.2 ± 0.0	0.3 ± 0.0
	1,3-Dichloropropanone	13DCP	2.8 ± 0.0	0.7 ± 0.1	0.7 ± 0.1
	1,1,3-Trichloropropanone	113TCP	1.6 ± 0.1	0.2 ± 0.0	ND
	1,1,3,3-Tetrachloropropanone	1133TeCP	0.8 ± 0.0	ND	ND
	1,1,3,3-Tetrabromopropanone	1133TeBP	ND	3.1 ± 0.3	1.7 ± 0.4
HNMs	Trichloronitromethane	TCNM	4.6 ± 0.1	2.4 ± 0.1	1.7 ± 0.1
	Dichloronitromethane	DCNM	0.4 ± 0.0	ND	ND
	Bromochloronitromethane	BCNM	0.2 ± 0.0	ND	ND
	Dibromonitromethane	DBNM	ND	ND	ND
THMs	Trichloromethane	TCM	764 ± 14.8	318 ± 29.0	257 ± 38.1
	Tribromomethane	TBM	0.3 ± 0.0	ND	ND
	Dibromochloromethane	DBCM	0.9 ± 0.1	0.2 ± 0.0	0.3 ± 0.0
	Bromodichloromethane	BDCM	11.8 ± 0.1	3.5 ± 0.1	3.0 ± 0.1
	Dichloroiodomethane	DCIM	ND	ND	ND
I-THMs	Bromochloroiodomethane	BCIM	ND	ND	ND
	Dibromoiodomethane	DBIM	ND	ND	ND
	Chlorodiiodomethane	CDIM	ND	ND	ND
	Bromodiiodomethane	BDIM	0.5 ± 0.0	ND	ND
	Iodoform	TIM	ND	ND	ND
HAMs	Chloroacetamide	CAM	ND	ND	ND
	Bromoacetamide	BAM	ND	ND	ND
	Dichloroacetamide	DCAM	21.5 ± 0.9	ND	3.4 ± 0.2
	Bromochloroacetamide	BCAM	ND	ND	ND
	Iodoacetamide	IAM	ND	ND	ND
	Trichloroacetamide	TCAM	37.0 ± 1.8	23.1 ± 0.8	9.0 ± 0.7
	Dibromoacetamide	DBAM	ND	ND	ND
	Bromodichloroacetamide	BDCAM	1.5 ± 0.1	1.9 ± 0.0	0.6 ± 0.0
	Chloroiodoacetamide	CIAM	ND	ND	ND
	Bromoiodoacetamide	BIAM	ND	ND	ND
	Dibromochloroacetamide	DBCAM	ND	ND	ND
	Tribromoacetamide	TBAM	ND	ND	ND
	Diiodoacetamide	DIAM	ND	ND	ND
	Chloroacetic acid	CAA	67.5 ± 6.8	120 ± 5.6	90.9 ± 5.5
HAAs	Bromoacetic acid	BAA	1.2 ± 0.0	ND	ND
	Dichloroacetic acid	DCAA	730 ± 113	1298 ± 150	1969 ± 32.5
	Trichloroacetic acid	TCAA	238 ± 23.4	317 ± 17.6	277 ± 24.6
	Bromochloroacetic acid	BCAA	14.9 ± 0.7	40.7 ± 3.6	69.1 ± 2.7
	Bromodichloroacetic acid	BDCAA	7.0 ± 0.2	9.0 ± 0.3	6.5 ± 0.4[b]
	Dibromoacetic acid	DBAA	2.8 ± 0.1	7.5 ± 0.5	12.0 ± 0.7[b]
	Dibromochloroacetic acid	DBCAA	3.9 ± 0.1	4.2 ± 0.0	0.5 ± 0.0[b]
	Tribromoacetic acid	TBAA	ND	1.6 ± 0.0	0.2 ± 0.0[b]
IAAs	Iodoacetic acid	IAA	0.2 ± 0.0	ND	ND
	Chloroiodoacetic acid	CIAA	ND	ND	0.3 ± 0.1[b]
	Bromoiodoacetic acid	BIAA	ND	ND	ND
	Diiodoacetic acid	DIAA	ND	ND	0.3 ± 0.0[b]

[a] Values reported as average ± standard deviation of triplicate measurements.
[b] Values reported as average ± standard error of duplicate measurements; ND = not detected.

have cited trichloroacetaldehyde as the primary driver of calculated chronic cytotoxicity in pools (Carter et al., 2019).

2.2.3. *Trihalomethanes (THMs)*

Trichloromethane concentrations in the conventional chlorine and salt water pool samples were 764 µg/L and 288 µg/L, respectively. Notably, 764 µg/L of trichloromethane in the conventional chlorinated indoor pool is the second highest reported level of trichloromethane in the literature, second to only 980 µg/L reported in a study conducted by Lahl et al. (1981). Elevated levels of trichloromethane in indoor pools underlines the importance of maintaining a low residual chlorine and having adequate ventilation in indoor pools to decrease swimmers' exposure to volatile DBPs via inhalation (Villanueva et al., 2007b), especially when there is an increase in bather load like during the January sampling.

2.2.4. *Haloketones (HKs)*

The average concentration of haloketones (HKs) decreased by 76% in salt water pool samples (8.4 µg/L) compared to the conventional chlorine pool sample (34.5 µg/L). Of the 8 HKs detected in one or more pool samples, 7 of them decreased in concentration, ranging from a 10% decrease to a 100% decrease, with the exception of 1,1,3,3-tetrabromopropanone (1133TeBP), which was not detected in conventional chlorinated waters, but had an average concentration of 2.4 µg/L in salt water pool samples. The formation of 1133TeBP indicates the presence of a bromide impurity in the salt used in the salt water pool, which would lead to the formation of Br-DBPs. Further discussion of bromide levels and resulting Br-DBPs can be found in Section 2.4 (Br-DBPs in pool samples).

2.3. *Conventional chlorine vs. salt water: N-DBPs*

2.3.1. *Haloacetamides (HAMs)*

This study presents the first report of the quantification of two haloacetamides (dichloroacetamide and bromodichloroacetamide) in a salt water pool. Of the 13 HAMs quantified in this study, only 3 were detected above the limit of quantification. Of those, trichloroacetamide (TCAM) was quantified at the highest level, with an average concentration of 23.0 µg/L, followed by dichloroacetamide (DCAM) at 8.3 µg/L and bromodichloroacetamide (BDCAM) at 1.3 µg/L. For trichloroacetamide and dichloroacetamide, maximum concentrations occurred in the conventional chlorinated pool water at 37.0 µg/L and 21.5 µg/L, respectively. The maximum concentration of bromodichloroacetamide occurred in the November salt water pool sampling event at 1.9 µg/L. On average, salt water pool samples showed a decrease in trichloroacetamide (57%), dichloroacetamide (92%), and bromodichloroacetamide (17%) when compared to the conventional chlorinated pool sample.

2.3.2. *Haloacetonitriles (HANs)*

Dichloroacetonitirle (DCAN) and chloroacetonitrile (CAN) were present at the highest level of all HANs quantified in this study, with an average concentration of 4.9 µg/L and 3.4 µg/L, respectively. HANs in salt water pool samples consistently decreased when compared to the conventional chlorine pool sample, with the exception of bromochloroacetonitrile (BCAN) and dibromoacetonitrile (DBAN), likely due to the presence of bromide in the salt used in the salt water pool.

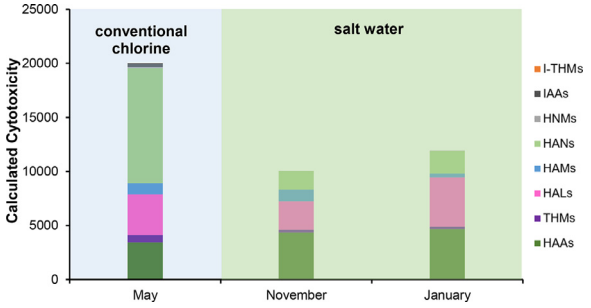

Fig. 2 – Calculated cytotoxicity of DBPs by class in conventional chlorine and salt water pool samples. Note that cytotoxicity data for haloketones (HKs) are currently not available in literature.

2.4. *Br-DBPs in pool samples*

Brominated DBPs (Br-DBPs) are of interest due to their elevated levels of toxicity compared to their chlorinated analogues (Richardson et al., 2007; Wagner and Plewa, 2017). Previous salt water pool studies, which measured a smaller number of DBPs, have attributed the formation of Br-THMs, Br-HAAs, and Br-HANs to bromide present in sodium chloride and emphasized the importance of using high purity sodium chloride (Beech et al., 1980; Whitaker et al., 2003; Lee et al., 2010, 2009). Ion chromatography analysis of the sodium chloride used in the salt water pool in this study revealed that the salt contained approximately 0.05% bromide. Assuming the salinity of salt water pools are typically maintained around 3,000 to 5,000 mg/L, at 4,000 mg/L salinity, a 0.05% bromide impurity will contribute approximately 118 µg/L of bromide to the pool. This impurity is a significant contribution to the bromide levels in this pool, considering that the tap water in the city where the community pool is located only contained 22 µg/L of bromide. Overall, the switch to a salt water pool led to a 73% increase in Br-DBPs, primarily driven by bromochloroacetic acid (BCAA), which saw a 268% increase to an average of 54.9 µg/L.

2.5. *Calculated cytotoxicity*

Calculated cytotoxicity decreased by 45% after implementation of the salt water pool system. Overall, the calculated cytotoxicity in pool samples was driven by HANs, HAAs, and HALs, which accounted for 34%, 30%, and 26%, respectively, of the average calculated cytotoxicity in conventional chlorinated and salt water pool samples combined (Fig. 2). In pool waters disinfected with conventional chlorine, the calculated cytotoxicity was driven by HANs (53%), followed by HALs (19%) and HAAs (17%). The 45% decrease in calculated cytotoxicity of salt water pools was primarily driven by HANs. Although the concentration of BCAN and DBAN increased in salt water pools, the concentration of BAN increased in conventional chlorine pool samples compared to salt water pool samples (3.1 µg/L and ND, respectively). Therefore, despite the overall increase in Br-DBPs upon implementing the electrochemically

generated chlorine system, an increase in the formation of BAN (contributing 0.1% of the total DBPs formed) in the conventional chlorine pool sample resulted in a substantial increase in calculated cytotoxicity and accounted for 40% of the calculated cytotoxicity in the conventional chlorine sample.

Trichloroacetaldehyde, despite being the least cytotoxic haloacetaldehyde quantified, contributed 22% of the average calculated cytotoxicity of all pool samples. In the conventional chlorinated pool sample, trichloroacetaldehyde accounted for 17% of the total calculated cytotoxicity and 26% in the salt water pool samples. Although to a lesser degree, this finding is consistent with a previous study in which trichloroacetaldehyde was cited as a significant driver of calculated cytotoxicity in swimming pools (Carter et al., 2019). Unlike HANs and HALs, HAAs did not have a clear driver of cytotoxicity in conventional chlorinated pool samples, but was driven by several HAAs like chloroacetic acid (4%), bromoacetic acid (5%), dichloroacetic acid (4%), and trichloroacetic acid (3%).

In salt water pools, the calculated cytotoxicity was driven by HAAs (41%), HALs (33%), and HANs (17%). On average, dichloroacetic acid contributed 39% to the calculated cytotoxicity for HAAs despite contributing 77% of the HAAs detected. Chloroacetic acid, which contributed 31% of the calculated cytotoxicity of the HAAs, only accounted for 5% of the average HAAs formed in salt water pool samples. Trichloroacetaldehyde contributed to 26% of the total calculated cytotoxicity but only 17% of the average total DBPs in salt water pool samples. Dichloroacetonitrile (7%) and dibromoacetonitrile (4%) were the primary HANs contributing to calculated cytotoxicity in salt water pool samples. The cases described above further showcase the importance of utilizing "TIC-Tox" to determine drivers of calculated cytotoxicity rather than inferring toxicity based on total DBP concentrations.

All classes of DBPs saw a decrease in calculated cytotoxicity when comparing the conventional chlorine pool to the salt water pool, with the exception of HAAs that saw an increase of 31%. When compared to conventional chlorinated pool samples, Br-HAAs and Br/Cl-HAAs were major contributors to the increase in calculated cytotoxicity. The calculated cytotoxicity contributed by bromochloroacetic acid and dibromoacetic acid saw a 268% and 250% increase, respectively, in the salt water pool. Interestingly, bromoacetic acid was not detected in salt water pool samples but was present at low levels (1.2 μg/L) in the conventional chlorinated pool sample and contributed 5% of the total calculated cytotoxicity of that sample. C hloroacetic acid, dichloroacetic acid, and trichloroacetic acid also saw an increase in calculated cytotoxicity (56%, 124%, and 25%, respectively) when compared to the conventional chlorine pool sample, likely due to the increase in bather load prior to the January sampling event (Table 1).

2.6. *Calculated genotoxicity*

Calculated genotoxicity decreased by 15% upon implementation of a salt water system. Primary drivers of calculated genotoxicity were HAAs, which accounted for 80% of the average calculated genotoxicity in all pool samples. The calculated genotoxicity of the conventional chlorine pool samples was driven by a combination of HAAs (62%) and HANs (23%). Chloroacetic acid (47%) and bromoacetic acid (14%) were the main contributors to calculated genotoxicity (Fig. 3), despite only contributing 3% and <1% of the total DBPs in the conventional chlorine pool sample, respectively. Like with calculated cytotoxicity, bromoacetonitrile (18%) was also the primary driver of calculated genotoxicity in the conventional chlorine pool sample, despite contributing <1% of the total DBPs formed. Chloroacetic acid (86%) was the calculated genotoxicity driver in salt water pools samples, despite only contributing 4% of the total DBPs formed.

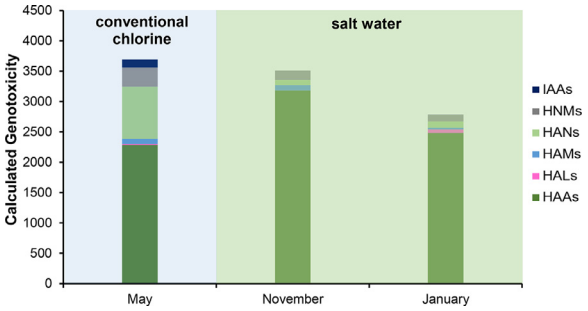

Fig. 3 – Calculated genotoxicity of DBPs by class in conventional chlorine and salt water pool samples. Note that genotoxicity data for HKs are currently not available in literature. I-THMs and THMs are not shown in this figure due to their presence at low or non-detect levels and/or their low genotoxicity values reported in literature.

All classes of DBPs saw a decrease in calculated genotoxicity in salt water pool samples when compared to the conventional chlorinated pool, with the exception of HALs and HAAs, which saw an increase of 89% and 24%, respectively. IAAs (99%), HANs (89%), and HNMs (59%) saw the largest percent reduction in calculated genotoxicity, but were only responsible for 7% of the total calculated genotoxicity in the salt water pool. Bromochloroacetic acid and dibromoacetic acid saw the largest increase in calculated genotoxicity, with a 268% and 250% increase, respectively. However, this increase in Br-HAAs only contributed 4% of the total genotoxicity of the pool samples. Furthermore, bromoacetic acid was not detected in salt water pool samples, but was present at low levels (1.2 μg/L) in the conventional chlorine pool sample, contributing 14% of the total calculated genotoxicity of that sample.

3. Conclusions

This study provides an extensive analysis of 60 DBPs in the same indoor community pool treated with either conventional chlorine or electrochemically generated chlorine (salt water). Of the 60 DBPs measured, 68% were detected at least once, with dominant DBP classes including HAAs, HALs, and THMs, with average concentrations of 1763 μg/L, 522 μg/L, and 453 μg/L, respectively. DBP levels were consistent with previous studies that reported these 3 classes, with the exception of trichloromethane, which was present at 764 μg/L in the conventional chlorine pool sample, likely due to a high residual chlorine (6.1 mg/L). This finding highlights the importance of maintaining a lower residual chlorine (1.0 to 2.0 mg/L) and ensuring adequate ventilation in indoor pools to decrease

swimmers' exposure to volatile DBPs. The switch from conventional chlorine to a salt water system saw a 15% increase in the average total DBPs present, driven by trichloroacetic acid and dichloroacetic acid. The overall increase in total DBPs in the salt water pool samples was driven by the January sample which was collected after an exercise class and contained 28% and 24% more total DBPs compared to the conventional chlorine sample and the November salt water pool sample, respectively.

However, the implementation of a salt water system led to a 45% and 15% decrease in calculated cytotoxicity and genotoxicity, respectively. Calculated cytotoxicity values for both conventional chlorine and salt water pool samples were driven by HALs, HANs, and HAAs. This decrease in calculated cytotoxicity and genotoxicity further indicates that maintaining a low residual chlorine is also just as important as limiting the bather load. Further, our calculated cytotoxicity findings match well with a previous drinking water study that demonstrated a statistically significant correlation between the concentration of N-DBPs and cytotoxicity (Allen et al., 2021). Therefore, limiting the formation of N-DBPs by reducing the amount of nitrogen sources like sweat and urine (Li and Blatchley, 2007; Yeh et al., 2014; Shah and Mitch, 2012) in swimming pools will be crucial in reducing the overall toxicity of swimming pools.

I-THMs, HNMs, HAMs, and THMs contributed only 9% on average to the total calculated cytotoxicity of all three pool samples. IAAs, despite their elevated levels of toxicity, only contributed 1% of the calculated cytotoxicity, due to their presence at low or non-detect levels. Trichloroacetaldehyde was the primary driver of calculated cytotoxicity, contributing 22% of the calculated cytotoxicity on average.

Calculated genotoxicity values for both conventional chlorine and salt water pool samples were driven by HNMs, HANs, and HAAs, with chloroacetic acid contributing 72% on average, despite only accounting for 3% of the average total DBPs. HAMs, HALs, and I-THMs were not significant contributors to calculated genotoxicity due to their presence at low or non-detect levels. Further, despite their high levels, THMs are not genotoxic in CHO cells (Wagner and Plewa, 2017), so they did not contribute to the calculated genotoxicity for these pool samples.

Ion chromatography analysis of the sodium chloride used in the salt water pool system revealed a 0.05% bromide impurity. Based on the average salinity required for salt water pools, this 0.05% impurity results in an increase of bromide levels by more than 100 μg/L. As a result, the concentration of Br-DBPs and Br/Cl-DBPs increased from 49.9 μg/L to 86.1 μg/L, a 73% increase. This increase in Br-DBPs was primarily driven by bromochloroacetic acid, which increased by 268% to 54.9 μg/L, but did not substantially contribute to the calculated genotoxicity.

This study provides important insights for pools utilizing conventional chlorine vs electrochemically generated chlorine (salt water). Overall, the change from a conventional chlorinated pool to a salt water pool system reduced the calculated cytotoxicity and genotoxicity despite the presence of a bromide impurity and the increase in bather load prior to the second (January) salt water sample. Due to the increasing popularity of salt water pools, future work focusing on controlled lab reactions and measurements of whole water toxicity of salt water pools using a variety of sodium chloride salts would be beneficial to better understand the impact bromide impurities may have on the toxicity of the pool water. Additionally, future research studying a larger number of both indoor and outdoor pools (utilizing both salt water and conventional chlorine) will aid in a more robust understanding of the factors that drive toxicity in each treatment technique.

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Appendix A Supplementary data

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.jes.2022.04.044.

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