Faculty of Science and Engineering

Understanding the Chemistry of Disinfection By-Products in Swimming Pools to Minimise Chemical Health Risks

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Declaration

To the best of my knowledge and belief this thesis contains no material previously published by any other person except where due acknowledgement has been made.

This thesis contains no material which has been accepted for the award of any other degree or diploma in any university.

Signature: _____

Rhys Aaron Ainsley Carter

Date: 3rd of July 2019

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Abstract

Disinfection is commonly applied to water in order to protect against microbial disease risk, however, it can lead to the unwanted formation of disinfection by-products (DBPs). Organic DBPs are formed when the added disinfectant reacts with organic matter contained in the water. Organic matter may be introduced into pool water via several sources including filling waters, the water treatment process and swimmers. Filling waters are often distributed drinking water which has been pre-treated (including disinfection) and therefore can introduce natural organic matter, trace amounts of DBPs and residual disinfectants. The swimming pool water treatment process may further introduce organic matter as commercially used disinfectants and other treatment chemicals are often impure. Swimmers are the largest input of organic matter into swimming pools, commonly referred to as 'bather load'. Bather load can be divided into two main categories: human body excretions, and pharmaceuticals and personal care products (PPCPs). Human body excretions introduce organic compounds contained in sweat, urine or saliva, while PPCPs refer to organic compounds such as analgesics, antibiotics, sunscreens, cosmetics, soaps, shampoos or lotions. These organic micropollutants may react with added pool disinfectants. While chlorine is a common disinfectant, other disinfectants include bromine, bromochlorodimethylhydantoin (BCDMH), chloroisocyanurates, chlorine dioxide and electrochemically-generated mixed oxidant (EGMO). With the continual use of pools, hence a continual input of both organic material and disinfectants, combined with recirculation of pool water and minimal freshwater input, DBP formation in pools is a magnified issue compared to drinking waters. Furthermore, while DBP formation occurs in pool waters, volatile DBPs (e.g. trihalomethanes (THMs) or chloramines) may enter the air above pools.

In comparison to DBPs in drinking water, wastewater or recycled water, less is known regarding DBPs in the swimming pool environment. Although many DBPs have been demonstrated to be cytotoxic, neurotoxic and/or genotoxic, with several additionally demonstrating mutagenic, carcinogenic and/or teratogenic nature, these studies are indicative, and they are generally limited to exposure via ingestion (i.e. drinking waters). In addition to ingestion, exposure to DBPs in swimming pools can occur via inhalation and absorption, and, as such, the potential health impact of DBPs in the swimming pool environment is still largely unknown.

This Thesis aims to fill key knowledge gaps on DBPs in the swimming pool environment, with a particular focus on their occurrence and formation, in order to improve the chemical water quality of pools and subsequently minimise potential health risks. **Chapter 2** presents a critical review of DBPs, their occurrence, factors affecting their

formation and potential health impacts, in the unique pool environment. The development of suitable analytical methods for nitrogenous DBPs (N-DBPs) in swimming pool waters and THMs in the air of indoor swimming pool complexes is discussed in **Chapter 3**. **Chapter 4** investigates the occurrence of a range of DBPs and several other general water quality parameters across fifteen pools of different types and/or treatment methods, while a more in-depth study of the chemical water quality of two newly built, filled and opened pools across fifteen months is presented in **Chapter 5**. The impact of the pool building process and/or new pool infrastructure on chemical water quality, particularly the formation of DBPs, is discussed in **Chapter 6**. Finally, conclusions and recommendations of this Thesis are summarised in **Chapter 7**.

The critical review of DBPs in swimming pool waters presented in **Chapter 2** summarises previously reported concentrations of a range of DBPs from various classes in pool and spa waters across the world. The review highlights that while several DBP classes have been well investigated (e.g. THMs, haloacetic acids and halamines), limited information exists for other DBP classes (e.g. haloketones, haloacetaldehydes and N-DBPs: halonitromethanes, haloacetonitriles, haloacetamides and *N*-nitrosamines). The impact of DBP precursor input via filling waters, human body excretions, and PPCPs is summarised, such that it is evident that the transformation of PPCPs and subsequent DBP formation in pool waters is currently an area of limited knowledge. Many additional factors, including type of disinfectant, pH, temperature, secondary treatment methods (e.g. UV (ultraviolet irradiation) and ozone), halide anions (e.g. bromide and chloride), pool location, swimmer activity and pool usage, are discussed as they have been demonstrated to affect the formation of DBPs. For example, high pool usage has been correlated with increasing concentrations of organic carbon, some DBPs (e.g. THMs) and mutagenic potency, with pool usage also reported to increase the volatilisation of volatile DBPs (e.g. THMs and chloramines). Temperature has also been reported to significantly affect the occurrence, formation and volatilisation of DBPs, with increasing water temperatures correlated to an increase in the release of human derived organic compounds (e.g. sweat). Studies focussing on the potential health effects of swimming pools are summarised, and while many have reported a correlation to exist between swimming pool attendance and respiratory issues, conflicting reports exist, suggesting there is currently insufficient information to draw definite conclusions regarding the health impacts of DBPs in the swimming pool environment.

Analytical methods for the analysis of DBPs in swimming pool waters and the ambient air of indoor swimming pool complexes are discussed in **Chapter 3**. A novel analytical method for the simultaneous analysis of 25 N-DBPs (9 haloacetonitriles, 9 halonitromethanes and 7 haloacetamides) in disinfected waters using liquid-liquid extraction followed by gas

chromatography-mass spectrometry (GC-MS) was developed and validated for drinking, pool and spa waters. The use of a programmable temperature vaporiser injector minimises degradation of the thermally labile halonitromethanes, and laboratory and instrumental runtimes were significantly reduced compared to previous methods. Limits of detection of 0.8 to 1.7 μg L^{-1} were achieved and the optimised method was used throughout **Chapters 4** and **5** to investigate these N-DBPs in real pool and spa waters. Furthermore, **Chapter 3.2** discusses the development of a simple method for the collection and analysis of THMs in air samples. The optimised solid-phase microextraction GC-MS method allows for point-in-time quantification of THMs with detection limits between 0.7 to 2.6 μg m^{-3}. The method was used to investigate the occurrence of THMs in the ambient air of several indoor swimming pool complexes, which is the first reported study of its kind in Australia.

The occurrence of sixty-four DBPs across various types of swimming pools with differing treatment methods was investigated and presented in **Chapter 4**. Concentrations of DBPs observed in these pools were similar to, or greater than, concentrations previously reported in other pools. For chloral hydrate, chloroacetic acid, dichloroacetic acid, trichloroacetic acid, dichloroacetamide, dibromoacetamide, dibromochloroacetamide and trichloroacetamide, as well as many of the investigated *N*-nitrosamines, levels of these DBPs were significantly greater than any previously reported concentrations in pools. Where possible, cytotoxicity of the waters was estimated based on DBP concentrations, with chloral hydrate representing over 90% of the total chronic cytotoxicity despite only representing up to 64% of the total molar DBP concentration.

Chapter 5 reports the occurrence of a range of disinfection by-products and several general water quality parameters in two newly built and filled swimming pools over fifteen months, where investigations began prior to opening. DBP concentrations measured in this study were generally similar to (or higher than) those previously reported in chlorinated pools, with concentrations of several DBPs (e.g. chloral hydrate, chloroacetic acid, dichloroacetic acid, trichloroacetic acid; up to 3202, 180, 26 000 and 11 000 μg L^{-1}, respectively) being significantly higher than previously reported maximum values. Additionally, many samples contained DBPs at concentrations greater than their respective Australian Drinking Water Guideline values and/or drinking water guidelines suggested by the World Health Organisation. Pools were found to exhibit significantly higher estimated cytotoxicity than their filling water (between 20 to over 46000 times greater), which reflects the significantly higher concentrations of DBPs measured in the pools in comparison to the filling waters. Chloral hydrate accounted for much of the total estimated cytotoxicity (up to 99%) and was found to be correlated to the number of pool entries, suggesting that swimmers may be a potential source of chloral hydrate precursors in pools. The significant concentrations of non-purgeable

organic carbon (NPOC) and DBPs prior to, and soon after, opening suggest that the pool building process and/or new pool infrastructure appears to have had a major impact on the chemical water quality of the pools.

To assess this possible impact, **Chapter 6** discusses the investigation of several commercial building materials and/or their individual components for their potential to (i) introduce organic compounds into pools via leaching, and (ii) lead to the formation of DBPs under conditions commonly used in Australian swimming pools. The investigated commercial concrete product and latex additive, both used during the construction of the investigated pools (**Chapter 5**) and approved and certified for such application, were demonstrated to leach significant concentrations of NPOC over relatively short periods of time (up to 23 mg L^{-1} over 21 days) under simulated pool conditions. While both the commercial concrete product and latex additive were found to lead to the formation of trichloromethane, dichloroacetic acid and trichloroacetic acid under simulated pool conditions, the latex additive was observed to be a more active precursor of these DBPs. Styrene, a monomer of the styrene-butadiene co-polymer which is a major constituent of the commercial latex additive, led to the formation of trichloromethane, trichloroacetic acid and chloral hydrate upon chlorination. Building materials therefore have the potential to not only act as a source of organic compound input to swimming pools, but also to lead to the formation of DBPs under conditions commonly employed in pools. Additionally, the investigated building materials, while approved for swimming pool applications in Australia, may potentially be responsible for the high concentrations of NPOC and some DBPs measured in the newly built pools investigated in **Chapter 5**. Overall, **Chapter 6** highlights the impact of building materials on the chemical water quality of swimming pools, particularly those newly constructed or having recently undergone maintenance, and the need for further investigations in this area, particularly regarding the potential health impacts.

While many studies have achieved a broadscreen analysis of DBPs across several swimming pools, fewer studies have followed the water quality of pools over time, with information regarding the production and trends of DBPs in pools over extended periods (e.g. >1 year) being very limited. This Thesis reports the first investigation of the occurrence of DBPs in newly built and filled swimming pools, providing information to assess any weekly, monthly or seasonal trends, bridging a significant knowledge gap. This Thesis includes the first investigation of *N*-nitrosamines in a brominated pool, presents the first known quantification of several DBPs (bromochloronitromethane, dichloronitromethane, bromochloroacetaldehyde and bromodichloroacetaldehyde) in chlorinated swimming pools, and provides important new knowledge on the long-term trends of DBPs in pools. Although filling waters, disinfectants and bather load are commonly accepted as sources of organic

material in swimming pools, building materials are yet to be proposed. This Thesis introduces building materials as an additional source of organic compound input to swimming pool waters, and provides information regarding their potential impact on chemical water quality, particularly on DBP formation. While the impact of infrastructure (i.e. building materials) on water quality and DBP formation in drinking waters has been investigated, limited studies of their impact in the swimming pool environment currently exist, further highlighting the novelty of this Thesis.

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List of Abbreviations

1,2-DBP	1,2-Dibromopropane
1,2-DBP-*d6*	1,2-Dibromopropane-*d6*
1HBT	1H-Benzotriazole
2,4,6-TCPh	2,4,6-Trichlorophenol
2,4-DCPh	5-Chloro-(2,4-dichlorophenoxy)phenol
4-DHB	4, 4′-Dihydroxybenzophenone
4-HB	4-Hydroxybenzophenone
4-MBC	4-Methylbenzylidene camphor
5-CBT	5-Cl-1H-Benzotriazole
5-MeBT	5-Methyl-1H-benzotriazole
ABS	Acrylonitrile-Butadiene-Styrene
ADWG	Australian Drinking Water Guidelines
ANSI	American National Standards Institute
AOBr	Adsorbable organic bromine (see also TOBr)
AOCl	Adsorbable organic chlorine (see also TOCl)
AOX	Adsorbable organic halogen (see also TOX)
ATD	Automatic thermal desorption
BCDMH	Bromochlorodimethylhydantoin
BFA	Body fluid analogue
BHT	Butylated hydroxytoluene
BP-1	2,4-Dihydroxybenzophenone
BP-2	2,2′,4,4′-Tetrahydroxybenzophenone
BP-3	Benzophenone-3
BP-8	2,2′-Dihydroxy-4-methoxybenzophenone
Br-DBPs	Brominated disinfection by-products
BuP	Butylparaben
BzP	Benzylparaben
BzS	Benzyl salicylate
CAR	Carboxen
C-DBPs	Carbonaceous disinfection by-products
CDC	Centre for Disease Control
CE	Capillary electrophoresis
CI	Chemical ionisation
CIC	Chloroisocyanurate
CIM	Chloroiodomethane

Cl/P	Chlorine-to-precursor ratio
Cl-DBPs	Chlorinated disinfection by-products
CLSA	Closed loop stripping
CNBr	Cyanogen bromide
CNCl	Cyanogen chloride
DAI	Direct aqueous injection
DBPs	Disinfection by-products
DCICA	Dichloroisocyanuric acid
DI	Direct injection
DMeBT	5,6-Dimethyl-1H-benzotriazole monohydrate
DMH	Dimethylhydantoin
DOC	Dissolved organic carbon
DPD	*N,N*-Diethyl-p-phenylenediamine
DVB	Divinylbenzene
ECD	Electron capture detector
EGMO	Electrochemically-generated mixed oxidant
EI	Electron ionisation
ESI	Electrospray ionisation
EtP	Ethylparaben
Et-PABA	Ethyl-4-aminobenzoate
FID	Flame ionisation detection
FLD	Fluorescence detection
FTIR	Fourier transform infrared spectroscopy
GC	Gas chromatography
HeP	Heptaparaben
HMs	Halomethanes
HNs	Halonitriles
HPLC	High performance liquid chromatography
HS	Headspace
iBuP	Isobutylparaben
IC	Ion chromatography
iPrP	Isopropylparaben
IS	Internal standard
I-THMs	Iodinated trihalomethanes
ITMS	Ion trap mass spectrometry
LD	Liquid desorption

LLE	Liquid-liquid extraction
LOD	Limit of detection
LVI	Large volume injection
m/z	Mass-to-charge ratio
MAHC	Model Aquatic Health Code
MeP	Methylparaben
MIMS	Membrane-inlet mass spectrometry
MLLE	Micro-liquid-liquid extraction
MS	Mass spectrometry
MSD	Mass selective detector
MTBE	Methyl *tert*-butyl ether
N-DBPs	Nitrogenous disinfection by-products
NDIR	Non-dispersive infrared
NHMRC	National Health and Medical Research Council
NOM	Natural organic matter
NPOC	Non-purgeable organic carbon
NSF	National Sanitation Foundation
OC	Organic carbon
OcP	Octylparaben
OCR	Octocrylene
OD-PABA	Octyldimethyl-*p*-aminobenzoic acid
OMC	Octylmethoxycinnamate
OP	Open path
Oz	Ozone
PARAFAC	Parallel factor
PBS	2-Phenyl-3h-benzimidazole-5-sulfonic acid
PDMS	Polydimethylsiloxane
PeP	Pentylparaben
PHBA	*p*-Hydroxybenzoic acid
PPCPs	Pharmaceuticals and personal care products
PrP	Propylparaben
PS	Phenyl salicylate
PT	Purge and trap
PTFE	Polytetrafluoroethylene
PTV	Programmable temperature vaporiser
PVC	Polyvinylchloride

PWAS	Personal whole air sample
PWTAG	Pool Water Treatment Advisory Group
r^2	Correlation coefficient
RSD	Relative standard deviation
SBME	Stir bar microextraction
SDCIC	Sodium dichloroisocyanurate
SDME	Single-drop microextraction
SHS	Static head-space
SIM	Selective ion monitoring
SDCIC	Sodium dichloroisocyanurate
SPE	Solid-phase extraction
SPME	Solid-phase microextraction
SS	Surrogate standard
SBR	Styrene butadiene rubber
$SUVA_{254}$	Specific ultraviolet absorbance at 254 nm
TCC	Triclocarban
TCE-*d2*	1,1,2,2-Tetrachloroethane-*d2*
TCICA	Trichloroisocyanuric acid
THAA/HAA9	Total haloacetic acids
THB	2,3,4-Trihydroxybenzophenone
TL	Thin layer
TN	Total nitrogen
TOBr	Total organic bromide
TOC	Total organic carbon
TOCl	Total organic chloride
TOI	Total organic iodide
TON	Total organic nitrogen
TOX	Total organic halogen
tq	Triple quadrupole
TTHMs	Total trihalomethanes
UPLC	Ultra-performance liquid chromatography
USH	Ultrasound heating
UV	Ultraviolet irradiation
UVD	Ultraviolet detection
WHO	World Health Organisation

*Abbreviations of the investigated disinfection by-products are provided separately in **Table 1-1** (Chapter 1, page 3).

List of Publications and Presentations Arising from this Thesis

Refereed Journal Articles

Carter, R.A.A., Allard. S., Croué, J.-P., and Joll, C.A., *In-Press*. 500 Days of swimmers: The chemical water quality of swimming pool waters from the beginning. Environ. Sci. Pollut. Res.

Carter, R.A.A., West, N., Heitz, A., and Joll, C.A., 2019. An analytical method for the analysis of trihalomethanes in ambient air using solid-phase microextraction gas chromatography-mass spectrometry: An application to indoor swimming pool complexes. Indoor Air 29, 499–509.

Carter, R.A.A., Allard. S., Croué, J.-P., and Joll, C.A., 2019. Occurrence of disinfection by-products in swimming pools and the estimated resulting cytotoxicity. Sci. Total Environ. 664, 851–864.

Carter, R.A.A., Liew, D.S., West, N., Heitz, A., and Joll, C.A., 2019. Simultaneous analysis of haloacetonitriles, haloacetamides and halonitromethanes in chlorinated waters by gas chromatography-mass spectrometry. Chemosphere 220, 314-323.

Carter, R.A.A., and Joll, C.A., 2017. Occurrence and formation of disinfection by-products in the swimming pool environment: A critical review. J. Environ. Sci. 58, 19–50.

Technology Transfer Publications

Carter, R.A.A., 2017. Sharing the Pool with Disinfection By-Products. Aquatic Recreation Australia – Official Journal of the Leisure Institute of Western Australia (LIWA) 1, 26–27.

Submitted Refereed Journal Articles

Carter, R.A.A., and Joll, C.A., Impact of building materials on the chemical water quality of swimming pools. Submitted to Journal of Hazardous Materials.

Oral Presentations

Understanding the Chemistry of Swimming Pool Water to Minimise Chemical Health Risks. Presented at Student Water Prize (Western Australian Branch) presentation evening; 20th September 2018, Perth, Western Australia: Australian Water Association.

500 Days of Swimmers: Variability of the Chemical Water Quality of Swimming Pool Waters from the Beginning. Presented at the 7th International Swimming Pool and Spa Conference; 2nd-5th May 2017, Kos Island, Greece.

Water Chemistry & Associated Health Risks. Presented at the 47th Annual Conference & Trade Display - LIWA Aquatics Conference; 15th-16th August 2016, Perth, Western Australia: Leisure Institute of Western Australia.

Poster Presentation

Simultaneous Analysis of Haloacetonitriles, Haloacetamides and Halonitromethanes by Gas Chromatography-Mass Spectrometry. Presented at the IWA & AWA Young Water Professionals Conference: 18th-19th February 2016, Sydney, New South Wales: Australian Water Association & International Water Association.

CHAPTER 1

INTRODUCTION

1.1. Background and Objectives

The formation of disinfection by-products (DBPs) is an unwanted consequence of the reaction between organic matter contained in water and added disinfectants. While DBPs have been extensively investigated in many water types (e.g. over 700 DBPs have been identified in treated drinking waters, wastewaters and recycled waters (Plewa and Richardson, 2017)), fewer studies have been conducted in swimming pool and/or spa waters (e.g. summary provided by Manasfi et al., 2016). Several studies have reported the occurrence of DBPs in swimming pool or spa waters where these DBPs have not previously been reported in disinfected waters (e.g. Daiber et al., 2016; Manasfi et al., 2016; Richardson et al., 2010; Zwiener et al., 2006). While limited studies have investigated the potential health impact(s) of DBPs specifically in the swimming pool environment (e.g. summary provided by Manasfi et al., 2017), many have demonstrated negative health effects in drinking waters (Richardson et al., 2007).

Four main sources of input of organic compounds to swimming pools are generally accepted. Filling waters (usually distributed drinking water) may introduce natural organic matter (NOM), bromide and DBPs due to prior treatment processes. Added disinfectants and other water treatment chemicals may introduce organic material, where, in addition to the active species, impurities and filler compounds may be introduced. The largest input of organic compounds in swimming pools is via bather load, which is comprised of two categories: human body excretions, as well as pharmaceuticals and personal care products (PPCPs). Human body excretions refer to sweat, urine and saliva, while PPCPs encompass, amongst others, a range of organic sources, including analgesics, antibiotics, sunscreens, cosmetics, soaps, shampoos and lotions. Due to the large variety of organic compounds and usually higher organic content of swimming pool waters, in conjunction with the consistent, and usually higher, employed disinfectant residuals, the formation and occurrence of DBPs in pools is likely greater than other treated waters (e.g. drinking waters). Despite this potential of elevated DBP formation in the swimming pool environment, limited information is currently available, warranting immediate investigation, particularly as the associated public health risk is currently not fully understood.

In this Thesis, an in-depth study of the occurrence, formation and trends of DBPs in the swimming pool environment was conducted, in order to better understand their impact on the chemical water quality of swimming pools. A list of the DBPs investigated throughout this Thesis is presented in **Table 1-1**.

Table 1-1: Disinfection by-products investigated throughout this Thesis. X= Br, Cl, I.

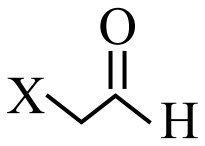
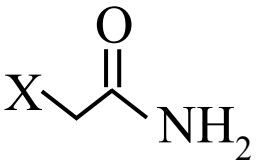
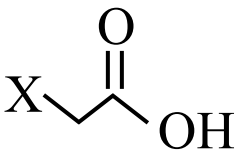
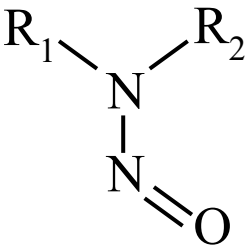
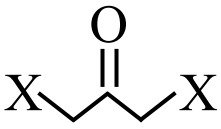
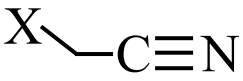
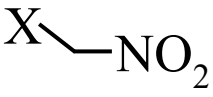
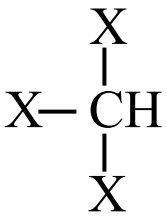
Disinfection By-Product	Abbreviation	General Structure
Haloacetaldehydes (HALs)		
Dichloroacetaldehyde	DCAL	
Dibromoacetaldehyde	DBAL	
Bromochloroacetaldehyde	BCAL	
Dibromochloroacetaldehyde	DBCAL	
Bromodichloroacetaldehyde	BDCAL	
Trichloroacetaldehyde (Chloral hydrate)	CH	
Tribromoacetaldehyde (Bromal)	TBAL	
Haloacetamides (HAAms)		
Dichloroacetamide	DCAAm	
Dibromoacetamide	DBAAm	
Bromochloroacetamide	BCAAm	
Bromodichloroacetamide	BDCAAm	
Dibromochloroacetamide	DBCAAm	
Trichloroacetamide	TCAAm	
Tribromoacetamide	TBAAm	
Haloacetic Acids (HAAs)		
Chloroacetic Acid	CAA	
Bromoacetic Acid	BAA	
Dichloroacetic Acid	DCAA	
Dibromoacetic Acid	DBAA	
Bromochloroacetic Acid	BCAA	
Bromodichloroacetic Acid	BDCAA	
Chlorodibromoacetic Acid	CDBAA	
Trichloroacetic Acid	TCAA	
Tribromoacetic Acid	TBAA	
***N*-Nitrosamines**		
N-Nitrosodiethylamine	NDEA	
N-Nitrosodimethylamine	NDMA	
N-Nitrosodi-*n*-butylamine	NDBA	
N-Nitrosodi-*n*-propylamine	NDPA	
N-Nitrosoethylmethylamine	NEMA	
N-Nitrosomorpholine	NMOR	
N-Nitrosopiperidine	NPIP	
N-Nitrosopyrrolidine	NPYR	

Table 1-1 continued

Disinfection By-Product	Abbreviation	General Structure
Haloketones (HKs)		
Chloropropanone	CP	
1,1-Dichloropropanone	1,1-DCP	
1,2-Dichloropropanone	1,2-DCP	
1,1,1-Trichloropropanone	1,1,1-TCP	
1,1,3,3-Tetrachloropropanone	1,1,3,3,-TeCP	
Haloacetonitriles (HANs)		
Chloroacetonitrile	CAN	
Bromoacetonitrile	BAN	
Bromochloroacetonitrile	BCAN	
Dichloroacetonitrile	DCAN	
Dibromoacetonitrile	DBAN	
Bromodichloroacetonitrile	BDCAN	
Dibromochloroacetonitrile	DBCAN	
Trichloroacetonitrile	TCAN	
Tribromoacetonitrile	TBAN	
Halonitromethanes (HNMs)		
Chloronitromethane	CNM	
Bromonitromethane	BNM	
Dichloronitromethane	DCNM	
Dibromonitromethane	DBNM	
Bromochloronitromethane	BCNM	
Bromodichloronitromethane	BDCNM	
Dibromochloronitromethane	DBCNM	
Trichloronitromethane	TCNM	
Tribromonitromethane	TBNM	
Trihalomethanes (THMs)		
Trichloromethane (Chloroform)	-	
Bromodichloromethane	-	
Dibromochloromethane	-	
Tribromomethane (Bromoform)	-	
Dichloroiodomethane	-	
Chlorodiiodomethane	-	
Dibromoiodomethane	-	
Bromodiiodomethane	-	
Bromochloroiodomethane	-	
Triiodomethane (Iodoform)	-	

The first objective of this Thesis was to carry out a comprehensive and critical review of the existing literature, focusing on the source, occurrence, formation and potential health impacts of DBPs in the swimming pool environment. Additionally, this review aided in identifying several knowledge gaps that existed in the field of DBPs in the swimming pool environment, highlighting key areas and/or topics for further investigation in this Thesis.

The second objective of this Thesis was the development of appropriate analytical methods for the analysis of DBPs in both swimming pool water and the air above pools, since many reported methods (e.g. those validated for drinking water) have previously been demonstrated to be unsuitable for the analysis of these more complex matrices.

A third objective of this Thesis was to apply the developed and validated analytical methods to carry out comprehensive surveys of a suite of DBPs and other general water quality parameters in water and air collected from a range of public swimming pool facilities. These surveys were performed to obtain a better understanding of the occurrence of DBPs in Australian swimming pools, where, in addition to information being currently limited, in comparison to other countries, Australian swimming pools have been demonstrated to be quite unique in terms of their chemical water quality.

A final objective of this study was to investigate, at a laboratory scale, potential precursors to, and formation of, several DBPs that were identified for further investigation due to their unusual concentrations measured during the comprehensive analytical surveys.

1.2. Thesis Overview

This Thesis consists of six journal articles, either published, in-press, or under review, which are presented in **Chapters 2** to **6**. Naturally, due to their connected themes, some unavoidable repetition is evident as the papers are presented together in this Thesis, particularly regarding background information and analytical procedures. The supporting information relevant to each journal article, reformatted to match the style of this Thesis, has been provided sequentially as **Appendix 1** to **6**.

An extensive review of the existing literature focusing on the source(s), occurrence, formation and potential health impacts of DBPs in the swimming pool environment was conducted. This work was published as a critical literature review entitled ‘Occurrence and formation of disinfection by-products in the swimming pool environment: A critical review’, in a special edition of the *Journal of Environmental Sciences (58, 19–50)*. Reformatted to the style of this Thesis, the published review is presented in **Chapter 2**, with minor modifications to include more recently available literature.

Chapter 3 exists as two sections and discusses the development of two individual analytical methods for swimming pool water and swimming pool air, respectively. The first section, **Chapter 3.1**, presents the development of a liquid-liquid extraction followed by gas chromatography-mass spectrometry analytical method for the simultaneous analysis of twenty-five nitrogenous DBPs in treated waters, where the developed method was validated for use analysing these DBPs in a range of disinfected waters including swimming pools and spas. A manuscript entitled 'Simultaneous analysis of haloacetonitriles, haloacetamides and halonitromethanes in chlorinated waters by gas chromatography-mass spectrometry' has been published in *Chemosphere (220, 314–323)*; which has been reformatted and presented in this Thesis as **Chapter 3.1**. The second section, **Chapter 3.2**, presents the development of an analytical method for the analysis of trihalomethanes in the ambient air of swimming pool complexes. **Chapter 3.2** is a reformatted version of the manuscript entitled 'An analytical method for the analysis of trihalomethanes in ambient air using solid-phase microextraction gas chromatography-mass spectrometry: An application to indoor swimming pool complexes', which has been accepted for publication in *Indoor Air (29, 499–509).*

A comprehensive survey of a range of public swimming pools where pool type (e.g. lap, leisure, spa and hydrotherapy pools) and treatment practices (e.g. chlorination, bromination and ultraviolet irradiation) varied was undertaken. **Chapter 4** presents the outcomes of this comprehensive survey, particularly the occurrence of a broad suite of DBPs and other general water quality parameters, as well as discussing several observed correlations between, and potential health impacts of, these measured DBPs. A manuscript entitled 'Occurrence of disinfection by-products in swimming pools and the estimated resulting cytotoxicity', which has been published in *Science of the Total Environment (664, 851-864),* has been reformatted as **Chapter 4**.

A key component of this Thesis is the in-depth examination of DBPs in two newly built and opened swimming pools, where investigations began prior to opening. **Chapter 5** presents a study of the occurrence and trends of a range of DBPs in these pools over fifteen months, and the potential health impact they may pose. **Chapter 5** is a reformatted version of the manuscript entitled '500 Days of swimmers: The chemical water quality of swimming pool waters from the beginning' has been accepted for publication in *Environmental Science and Pollution Research (In-Press)*.

Due to some unexpected findings during the in-depth investigation presented in **Chapter 5**, namely the observation of unusually high concentrations of non-purgeable organic carbon and some DBPs prior to, and their subsequent increase after, the opening of the newly built public swimming pool facility, an investigation into building materials as a source of

organic compounds in swimming pools was undertaken. Outcomes from this study are presented in **Chapter 6**. A manuscript entitled 'Impact of Building Materials on the Chemical Water Quality of Swimming Pools' has been submitted to *Journal of Hazardous Materials* where it is currently under review for publication.

Finally, **Chapter 7** provides a summary of the key conclusions arising from the outcomes of the investigations presented in this Thesis. Furthermore, the impact of this research and recommendations for future studies are also presented.

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CHAPTER 2

OCCURRENCE AND FORMATION OF DISINFECTION BY-PRODUCTS IN THE SWIMMING POOL ENVIRONMENT: A CRITICAL REVIEW

Rhys A. A. Carter and Cynthia A. Joll.

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Statement of Contribution to Co-authored Published Paper

This Chapter includes the co-authored paper '*Occurrence and formation of disinfection by-products in the swimming pool environment: A critical review*', published in Journal of Environmental Sciences. The bibliographic details of the co-authored paper, including all authors are:

Carter, R.A.A., Joll, C.A., 2017. Occurrence and formation of disinfection by-products in the swimming pool environment: A critical review. J. Environ. Sci. 58, 19–50. doi:10.1016/j.jes.2017.06.013.

I, Rhys Aaron Ainsley Carter, contributed to the publication by: undertaking an extensive review of the current literature, being the primary writer (including preparing figures and tables), and editing and finalising the manuscript.

I, as a Co-Author, endorse that the level of contribution by the candidate indicated above is appropriate.

Cynthia Joll

2.1. Abstract

Disinfection of water for human use is essential to protect against microbial disease; however, disinfection also leads to formation of disinfection by-products (DBPs), some of which are of health concern. From a chemical perspective, swimming pools are a complex matrix, with continual addition of a wide range of natural and anthropogenic chemicals via filling waters, disinfectant addition, pharmaceuticals and personal care products and human body excretions. Natural organic matter, trace amounts of DBPs and chlorine or chloramines may be introduced by the filling water, which is commonly disinfected distributed drinking water. Chlorine and/or bromine is continually introduced via the addition of chemical disinfectants to the pool. Human body excretions (sweat, urine and saliva) and pharmaceuticals and personal care products (sunscreens, cosmetics, hair products and lotions) are introduced by swimmers. High addition of disinfectant leads to a high formation of DBPs from reaction of some of the chemicals with the disinfectant. Swimming pool air is also of concern as volatile DBPs partition into the air above the pool. The presence of bromine leads to the formation of a wide range of bromo- and bromo/chloro-DBPs, and Br-DBPs are more toxic than their chlorinated analogues. This is particularly important for seawater filled pools or pools using a bromine-based disinfectant. This review summarises chemical contaminants and DBPs in swimming pool waters, as well as in the air above pools. Factors that have been found to affect DBP formation in pools are discussed. The impact of the swimming pool environment on human health is reviewed.

2.2. Introduction

Swimming pool chemical water quality is currently a topic of interest, with many studies occurring in both the United States and Europe. Swimming pool chemical water quality is of possible public health concern due to the formation of disinfection by-products (DBPs), where total DBP concentrations have been shown to progressively increase in pools and spas (up to 610% and 900%, respectively) compared to their respective filling waters (Daiber et al., 2016). Swimming pool DBPs are unwanted consequences from the reactions of components of the swimming pool water and the disinfectant, during the swimming pool disinfection process. There is an increased potential risk to babies and small children where the health effects of DBPs may be more pronounced. Uptake of DBPs are likely increased in children compared to adults due to higher breathing rates of children (up to twice those of adults) and their lesser developed gastrointestinal tracts and blood brain barriers possibly leading to higher absorption of DBPs (Thompson, 2004). Additionally, children's organs are not fully developed, particularly the liver and kidneys which have been shown to be two to nine times slower in the breakdown of chemical compounds compared to adults, and, in

combination with immature metabolite breakdown mechanisms, may not be able to metabolise and remove DBPs sufficiently (Thompson, 2004). DBPs in swimming pools have been potentially linked to several health issues, including asthma, bladder cancer, liver and kidney issues (Villanueva et al., 2007; Villanueva and Font-Ribera, 2012). Swimming pool waters have shown increased genomic DNA damage effects on Chinese hamster ovary cells than the corresponding filling water (Liviac et al., 2010b), which is likely due to more than one mutagen (Honer et al., 1980). Respiratory issues, such as asthma, wheeze, cough and lower respiratory tract infections, have been correlated with swimming pool attendance, which is likely due to chlorinated volatile DBPs, such as chloramines (Bernard et al., 2006; Ferrari et al., 2011; Jacobs et al., 2007; Kaydos-Daniels et al., 2008; Rosenman et al., 2015; Uyan et al., 2009). However, these studies are not conclusive and Goodman and Hays (2008) suggested that "it is premature to draw conclusions about the causal link between swimming and asthma", warranting further investigation of the health effects of the swimming pool environment.

Indoor swimming pools are of particular concern since they may be more regularly used all year round, and volatile DBPs can become trapped within the environmental air of indoor swimming pool complexes. The higher the concentration of these volatile DBPs in the swimming pool water, the higher their concentration in the air above the pool. Volatile compounds of potential health concern in the air pose a risk not only to regular swimmers, but also to regular non-swimmers, such as swimming pool workers and non-swimming visitors.

Disinfection is essential to protect against the microbial disease risk in pools (Montgomery, 1985). Studies of comparison of microbial disease and DBP risks in pools are limited, but, in drinking waters, the risk of death or illness from pathogens is much higher than the risk of cancer from DBPs (Ashbolt, 2004; WHO, 2000). Although chlorine based disinfectants, calcium or sodium hypochlorite and chlorine gas, are more commonly used (Montgomery, 1985), other disinfectants including chlorine dioxide (ClO_2), chloroisocyanurates or their acid counterparts, bromine gas, sodium bromide (in combination with a chlorine oxidiser), bromochlorodimethylhydantoin (BCDMH) or electrochemically-generated mixed oxidant (EGMO) can be employed for swimming pool disinfection.

Chlorine based disinfectants result in the formation of hypochlorous acid (HOCl), whilst bromine based disinfectants predominantly produce hypobromous acid (HOBr). These species react further, producing additional 'active' oxidising species, hypochlorite ($^{-}$OCl) and hypobromite ($^{-}$OBr). All active species (the acids (HOCl/HOBr) and the ions ($^{-}$OCl/$^{-}$OBr)) have the ability to inactivate microorganisms and react with organic matter, leading to the

formation of organic DBPs, chloride (Cl^-) and bromide (Br^-). Hypochlorous acid is approximately 100 times more effective than the hypochlorite ion, whilst HOBr is the stronger oxidising species (Chow et al., 2014). These reactions, and hence disinfectant speciation, are both pH and chloride dependent (E et al., 2016; Hansen et al., 2012b), and care should be taken to maximise the dominance of the more powerful oxidant species to ensure maximum disinfection power, although DBP formation rates and the behaviour of DBP precursors will also be influenced by these more reactive disinfectant species. Unlike other chlorine containing disinfectants, ClO_2 does not produce HOCl: ClO_2 does not hydrolyse in water, rather it remains as a dissolved gas, with oxidation occurring via electron exchange mechanisms (NRC, 1980). The other oxidants, BCDMH or EGMO (the production of oxidants via the electrolysis of waters rich in sodium chloride), are also used as disinfectants in swimming pool waters, however their chemistry is not as straightforward. BCDMH results in both HOCl and HOBr (Elsmore, 1994), whilst EGMO leads to the presence of several oxidising species: HOCl, HOBr, ozone and hydrogen peroxide (Kraft et al., 2008; Patermarakis and Fountoukidis, 1990), with HOBr and HOCl being the dominating species for BCDMH and EGMO, respectively. A detailed discussion of the chemistry of swimming pool disinfectants is provided in **Section 2.3** below.

Table 2-1: Recommended minimum free chlorine equivalent concentrations (mg L^{-1}) and pH values for swimming pools and spas by selected organisations.

Disinfectant	pH Range	Swimming Pools		Spas	Reference
		Unstabilised	Stabilised*		
Chlorine Bromine	7.2-7.8	1 2	2 -	3 6	(NHMRC, 2008)
Chlorine	6.5-7.6	0.3-0.6	0.3-0.6	0.7-1	(German Institute for Standardization, 2012)
Chlorine	7.2-7.4	0.5-1	2.5-5	-	(PWTAG, 2003)
Chlorine Bromine	7.2-7.8	1 3	2 -	3 4	(CDC, 2016)
Chlorine Bromine	7.2-7.8	0.5-1.2 4-6	0.5-1.2 -	2-3 4-6	(WHO, 2006)

*Stabilised refers to the use of cyanuric acid. Stabilisation not possible with bromine based disinfectants

There is currently no international standard for the treatment of swimming pools, with regulations often provided by state or local governing bodies. For example, the USA's Centre for Disease Control (CDC) have released the 'Model Aquatic Health Code' (MAHC) which other governing bodies are encouraged to adopt (CDC, 2016). Similarly, the Australian National Health and Medical Research Centre (NHMRC) encourages Australian pool operators to adopt their 'Guidelines for managing Risks in Recreational Waters' (NHMRC, 2008), whilst the DIN 19643 is regulation in Germany (German Institute for

Standardization, 2012). Other bodies such as the Pool Water Treatment Advisory Group (PWTAG) or the World Health Organisation (WHO) have produced guidelines which have been adopted by various other countries, including the United Kingdom (PWTAG, 2003). The minimum free chlorine equivalent concentrations recommended by the aforementioned organisations are presented as examples in **Table 2-1**. It is evident that recommended guidelines can differ among regulators, suggesting a more complete understanding of treatment methods is required. Development of international swimming pool guideline recommendations, similar to the World Health Organisation's "Guidelines for Drinking Water Quality", would be beneficial in assisting governing bodies worldwide to develop local guidelines based on their local requirements. Since the required free chlorine equivalent residual in swimming pools is higher than that reported in drinking water distribution systems (e.g. minimum 0.2 mg L^{-1}, Chow et al., 2014) and there is a build-up of organic compounds in swimming pool waters such that the total organic carbon content is usually much higher (e.g. <33 mg L^{-1}, Plewa et al., 2011) than that detected in drinking waters (e.g. 1.8 to 3.6 mg L^{-1}, McDonald et al., 2013), DBP formation is a magnified issue in swimming pool waters compared to drinking waters.

Swimming pools have a wide variety of uses and therefore the swimming pool water matrix is quite unique. Not only does the type of swimming pool affect the water matrix, but other factors, including temperature, climate, location and swimming habits, particularly swimmer hygiene, all have an impact. Both organic and inorganic compounds may enter a swimming pool in a variety of ways, as illustrated in **Figure 2-1**. The filling water, or water used to fill the swimming pool, is commonly disinfected distributed drinking water (freshwater swimming pools), although seawater is sometimes used, and the filling water can introduce species such as natural organic matter (NOM), trace amounts of DBPs and chlorine or chloramines. Compounds introduced in the filling water are highly dependent on the disinfection method used for the distributed water. Due to the constant addition of a disinfectant to the pool, chlorine and/or bromine are introduced. Personal care products, such as sunscreens, hair products, lotions/soaps and cosmetics, as well as human body excretions (sweat, urine, saliva and body cells), are also introduced into the swimming pool water, with urea being detected up to 17 mg L^{-1} (Yang et al., 2018). These two categories together have been termed bather load as they are introduced by swimmers or 'bathers'. The continual use of the swimming pool, input from bather load, continual addition of a disinfectant, combined with minimal freshwater input and continual recirculation of the same water, can cause these contaminants to become highly concentrated within swimming pool waters. Swimming pool waters are commonly subject to filtration by either sand, diatomaceous earth or membrane filters, however this predominantly removes the physical contaminants, such as hair and lint,

rather than the chemical contaminants, although some dissolved compounds (e.g. DBPs and their precursors) may be adsorbed. Filter media have also shown potential to form some DBPs (Hansen et al., 2012b) and proper operation of filters should seek to minimise them as a source and proper operation of filters should seek to minimise them as a source of DBPs in swimming pool waters. Some swimming pools employ additional treatment to improve microbiological inactivation, such as ozone or ultraviolet (UV) irradiation, however, while these methods commonly decrease some chemical contaminants, they can increase the formation of others. For example, UV followed by post-chlorination of swimming pool water has been shown to decrease chloramine concentrations, however the formation of trihalomethanes increased (Cimetiere and De Laat, 2014). Other studies have reported contradictory findings (discussed in more detail in **Section 2.6.1**), highlighting the complex nature of the chemistry involved in secondary treatment of swimming pools.

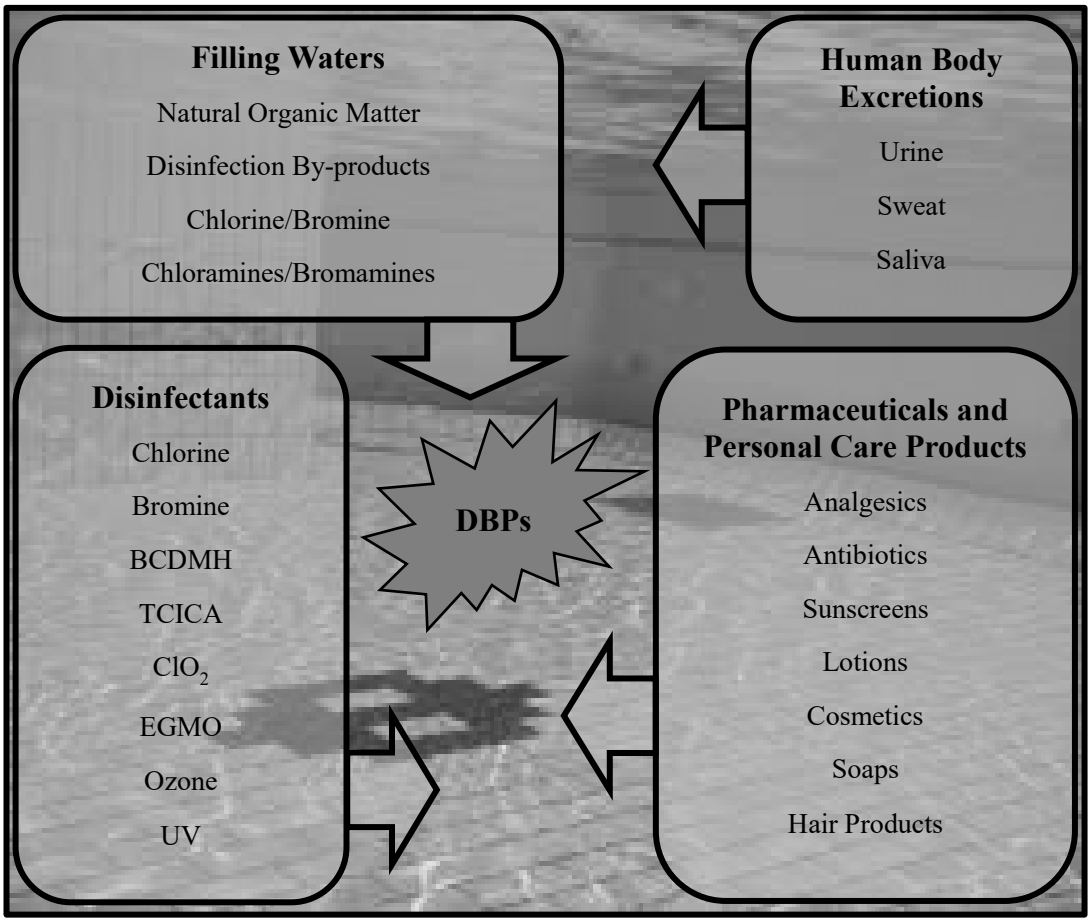


Figure 2-1: Disinfection by-product precursors and disinfectants in swimming pool and spa waters. Adapted from Carter et al. (2015). **BCDMH**: Bromochlorodimethylhydantoin. **DBPs**: Disinfection by-products. **EGMO**: Electrochemically-generated mixed oxidant. **TCICA**: Trichloroisocyanuric acid. **UV**: Ultraviolet irradiation.

There are three DBP uptake mechanisms applicable to the swimming pool environment: ingestion, absorption and inhalation. Ingestion and absorption both occur during swimming activities as some water is often accidentally swallowed and DBPs may be absorbed through the skin. For example, Dufour et al. (2006) found the average volume of water ingested by adults during a 45 minute swim was 16 mL (21 mL hr^{-1}), with non-adults (<18 years) swallowing twice as much as adults. A recent study, however, reported that previous investigations of swimming pool water ingestion may be underestimated by up to 15% (Sinclair et al., 2016). The skin permeability of some DBPs has been studied. Xu et al. (2002) investigated trihalomethanes (THMs), haloketones (HKs) and haloacetic acids (HAAs), reporting that THMs had the highest skin permeability, with brominated THMs being more permeable than chlorinated THMs. HKs were reported to be less permeable to human skin than THMs, but more permeable than HAAs, which showed almost no permeability (Xu et al., 2002). Haloacetonitriles (HANs) were investigated by Trabaris et al. (2012), with dibromoacetonitrile being found to have the highest permeability to human skin, whilst chloroacetonitrile had the least. HANs were shown to be less permeable to human skin than chloral hydrate (Trabaris et al., 2012). Both studies correlated an increase in temperature to increased human skin permeability of selected DBPs (Trabaris et al., 2012; Xu et al., 2002). Inhalation is particularly important for volatile DBPs and has been reported to be the major route of human exposure of DBPs in the swimming pool environment (Aggazzotti et al., 1998; Aprea et al., 2010; Chen et al., 2011; Erdinger et al., 2004). In the swimming pool environment, THM uptake via inhalation has been estimated to have a higher associated cancer risk than uptake via ingestion or dermal routes (Lee et al. 2009), where estimations were calculated using the US EPA guidelines for carcinogen risk assessment and the Swimmer Exposure Assessment Model using standard values from the US EPA Exposure Factors Handbook. Similar results were reported by Chen et al. (2011), who found that 99% of the risk arising from THM exposure was due to inhalation of chloroform. An increase in water temperature, swimming activity and blowers/jets causes an increased volatilization rate of volatile DBPs and hence this inhalation uptake mechanism can become of high importance, particularly for indoor heated swimming pools (Aggazzotti et al., 1998; Kristensen et al., 2010; Marco et al., 2015).

Currently, few guidelines appear to exist worldwide for the concentrations of DBPs specifically in the swimming pool environment. DBPs are regulated in drinking waters; however, due to the uptake mechanism ratio shift from ingestion (drinking waters) to inhalation (swimming pool waters), the drinking water DBP guidelines may not be directly applicable to assess the health risk associated with DBPs in swimming pool waters. Drinking water guidelines may, however, act as an indicative health guideline value where no

swimming pool specific guideline value exists. Current swimming pool specific regulations mainly provide health guidelines for chloramines (measured as combined chlorine), which are encouraged to be no greater than half that of the free chlorine equivalent concentrations in pool water, although lower ideal concentrations (less than 0.2 to 0.4 mg L^{-1}) have been suggested (CDC, 2016; WHO, 2006). Trichloramine in the swimming pool air has also been regulated, with WHO (2006) recommending maximum concentrations of 0.5 mg m^{-3}, although some European countries propose a lower guideline for trichloramine in the air of indoor swimming pool complexes, 0.2 to 0.3 mg m^{-3} (Cassan et al., 2011; Umweltbundesamtes, 2011). THMs are the only organic DBP class known to be regulated in swimming pool waters. For the total THM concentrations (sum of trichloromethane, bromodichloromethane, dibromochloromethane and tribromomethane), the German standard DIN 19643 suggests a guideline value of 20 µg L^{-1} in swimming pool waters (German Institute for Standardization, 2012), whilst Denmark's Statutory Order no 623 recommends total THM concentrations do not exceed 25 µg L^{-1} in pool waters (Lovtidende, 2012).

This critical review summarises chemical contaminants and DBPs reported in swimming pool waters, as well as in air above swimming pools. Factors that have been found to affect DBP formation in pools are also discussed. The impact of the swimming pool environment on human health is reviewed.

2.3. Disinfectants and their Associated Chemistry in Swimming Pools and Spas

Although many oxidants can form by the addition of one disinfectant to water, the type of the disinfectant (chlorine or bromine based) refers to the most dominant species. Although specific disinfectants will be provided in some examples, for the purpose of this review, chlorination (treated by chlorine) refers to the use of disinfectants where HOCl is the primary oxidant (sodium hypochlorite (NaOCl), calcium hypochlorite ($Ca(OCl)_2$), chlorine gas (Cl_2), sodium dichloroisocyanurate (SDCIC), chloroisocyanurate (CIC), dichloroisocyanuric acid (DCICA) or trichloroisocyanuric acid (TCICA)), whilst bromination (treated by bromine) refers to the use of disinfectants where HOBr is the primary oxidant, the main examples being bromochlorodimethylhydantoin (BCDMH) and sodium bromide (NaBr) in the presence of an oxidant. EGMO and ClO_2 will be discussed separately to other disinfectants, where possible. Waters which are treated in combination with a secondary treatment (e.g. UV or ozone) will be distinguished from those treated solely by disinfectants.

2.3.1. Chlorine Based Disinfectants

The addition of the major chlorine based disinfectants (chlorine gas, sodium and calcium hypochlorite) to water results in the formation of hypochlorous acid (HOCl) as per **Eq. (2-i)**, **(2-ii)** and **(2-iii)**, respectively.

$$Cl_2 + H_2O \leftrightharpoons HOCl + H^+ + Cl^- \quad \textbf{Eq. (2-i)}$$

$$NaOCl + H_2O \rightarrow HOCl + Na^+ + OH^- \quad \textbf{Eq. (2-ii)}$$

$$Ca(OCl)_2 + H_2O \rightarrow 2HOCl + Ca^{2+} + 2OH^- \quad \textbf{Eq. (2-iii)}$$

HOCl further dissociates in water producing hypochlorite as per **Eq. (2-iv)**.

$$HOCl \leftrightharpoons OCl^- + H^+ \quad \textbf{Eq. (2-iv)}$$

Both HOCl and OCl^- have the ability to inactivate microorganisms (disinfect), as well as react with organic matter leading to the formation of Cl-DBPs and oxidised organic matter/ chloride. Although the predominant chlorine species is HOCl, the highly reactive, but less abundant, electrophiles, Cl_2O and Cl_2, may also be present in swimming pool waters and, due to their higher reactivity compared to HOCl (De La Mare et al., 1975), may be responsible for the formation of some Cl-DBPs. In a study of aromatic ethers, although unexplored specifically in swimming pools, Cl_2O and Cl_2 were shown to be important species in generation of DBPs from aromatic ether DBP precursors of moderate reactivity (Sivey and Roberts, 2012).

Cyanurates and their acids all result in the formation of HOCl (and hence OCl^-), via twelve simultaneous chemical equilibrium reactions (dissociation or chlorination) of the cyanurates, their acids, and their chlorinated counterparts. In the case of chlorinated cyanuric acids, the most commonly used in pools are trichloroisocyanuric acid (TCICA) and dichloroisocyanuric acid (DCICA), which are often added as their salt form; in these cases, HOCl will be formed directly via dissociation in water, as shown in **Figure 2-2 (a)** and **(b)** where DCICA and TCICA are used as examples, respectively.

In pools, the majority of chlorine (97%) is bound to the cyanurates which are poor oxidants compared to HOCl, but despite this and due to the equilibrium, as HOCl is depleted, chlorinated cyanurates 'release' chlorine to reform HOCl, and hence maintain the desired free chlorine residual. These types of chemicals are referred to as chlorine stabilisers, as cyanurates are less susceptible to solar degradation than HOCl itself. This lower chlorine decay observed is seen as stabilisation of the chlorine, hence the name chlorine stabiliser. Stabilised chlorine may also be formed by the addition of cyanurate to swimming pools that use chlorine based disinfectants, as in the reverse reactions of **Figure 2-2**.

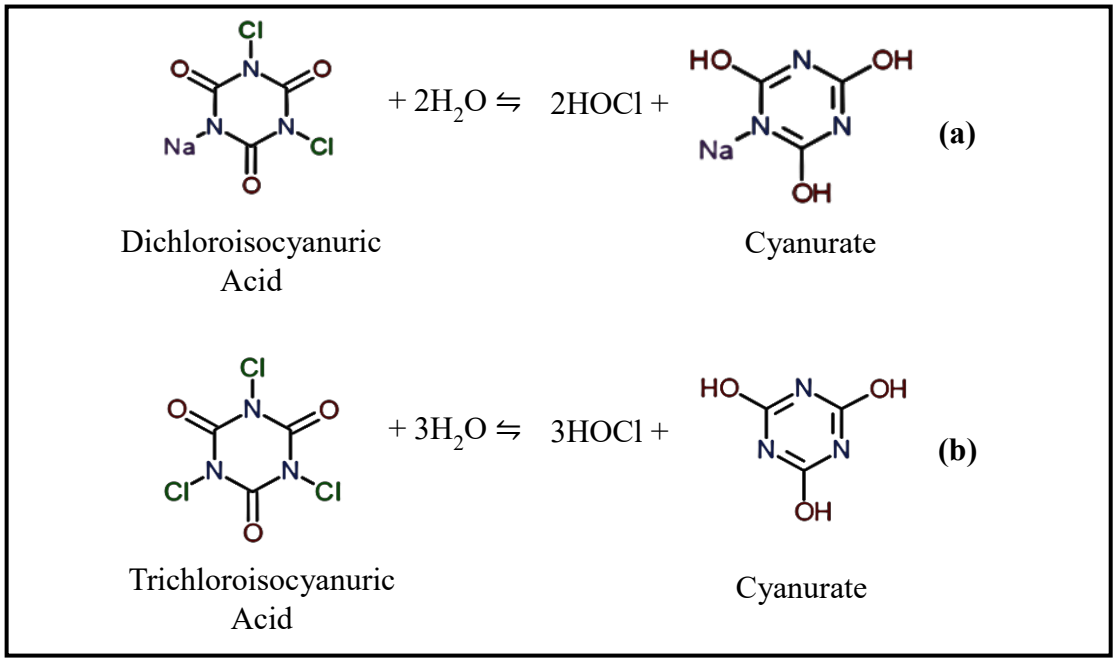


Figure 2-2: Formation of HOCl by the use of **(a)** dichloroisocyanuric acid (DCICA, as its sodium salt) and **(b)** trichloroisocyanuric acid (TCICA).

2.3.2. Bromine Based Disinfectants

Similar to the formation of HOCl and OCl^- via chlorine based disinfectants, bromine based disinfectants result in the formation of HOBr, which further dissociates to OBr^- as per **Eq. (2-v)** and **(2-vi)**.

$$Br_2 + H_2O \leftrightharpoons HOBr + H^+ + Br^- \quad \textbf{Eq. (2-v)}$$

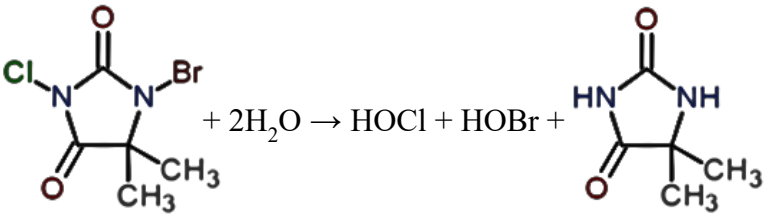
$$HOBr \leftrightharpoons OBr^- + H^+ \quad \textbf{Eq. (2-vi)}$$

Unlike Cl_2, bromine gas is rarely used as a disinfectant, instead HOBr may be formed by combining sodium bromide (NaBr) with an oxidant (usually chlorine based, although ozone is often used), as per **Eq. (2-vii).**

$$NaBr + HOCl \rightarrow HOBr + Na^+ + Cl^- \quad \textbf{Eq. (2-vii)}$$

Analogous to HOCl, both HOBr and OBr^- have the ability to inactivate microorganisms (disinfect), as well as react with organic matter leading to the formation of Br-DBPs and oxidised organic matter/bromide (Br^-).

The most common bromine based disinfectant is BCDMH, which forms both HOBr and HOCl in the presence of water, as shown in **Figure 2-3**.



Bromochlorohydantoin (BCDMH) | Dimethyhydantoin (DMH)

Figure 2-3: Formation of HOBr and HOCl via the hydrolysis of bromochlorodimethylhydantoin (BCDMH).

HOCl and HOBr further dissociate as per **Eq. (2-iv)** and **(2-vi)**, respectively. Despite the formation of HOCl, BCDMH is considered a bromine based disinfectant as (i) HOBr is a stronger oxidising agent than HOCl and (ii) the HOCl is mainly associated with the bromide recycling reaction, that is, the regeneration of HOBr via oxidation of bromide as demonstrated in **Eq. (2-viii)**; HOCl still has the ability to inactivate microorganisms and form Cl-DBPs, although these are minor reactions in comparison to those involving HOBr (Hunter and Jiang, 2010).

$$Br^- + HOCl \rightarrow HOBr + Cl^- \qquad \textbf{Eq. (2-viii)}$$

2.3.3. Chlorine Dioxide

Although not as commonly used as other chlorine based disinfectants, chlorine dioxide may be used in swimming pools or spas, however, unlike other chlorine containing disinfectants, ClO_2 does not produce HOCl as it does not hydrolyse in water. ClO_2 remains as a stable free radical ($ClO_2^{\bullet}$), where it reacts by radical mechanisms with electron rich moieties following **Eq. (2-ix)**, where the source of the e^- is electron rich moieties (e.g. lone electron pairs on amines or phenolates) present in organic matter, and leads to the formation of chlorite (ClO_2^-).

$$ClO_2^{\bullet} + e^- \rightarrow ClO_2^- \qquad \textbf{Eq. (2-ix)}$$

This reaction leads to the formation of highly reactive organic radical moieties which may be further oxidised by ClO_2 or react with other organic moieties. Although chlorite is relatively stable in conditions commonly used in swimming pools, it may lead to the formation of chloride and chlorate as per **Eq. (2-x)** and **(2-xi)**.

$$2ClO_2^- + HOCl + H^+ \rightarrow 2ClO_2 + Cl^- + H_2O \qquad \textbf{Eq. (2-x)}$$

$$ClO_2^- + Cl_2 + H_2O \rightarrow ClO_3^- + 2Cl^- + 2H^+ \qquad \textbf{Eq. (2-xi)}$$

2.3.4. Electrochemically-Generated Mixed Oxidant

Electrochemically-generated mixed oxidant (EGMO) disinfection is achieved by applying an electric current to water with a high chloride content and, despite the production of several oxidising species, the predominant species formed is Cl_2. Electrolysis of water involves an anodic reaction (**Eq. (2-xii)**) and a cathodic reaction (**Eq. (2-xiii)**), where the overall process is shown in **Eq. (2-xiv)**.

$$2H_2O \rightarrow O_2 + 4H^+ + 4e^- \quad \textbf{Eq. (2-xii)}$$

$$2H_2O + 2e^- \rightarrow H_2 + 2OH^- \quad \textbf{Eq. (2-xiii)}$$

$$2H_2O \rightarrow O_2 + 2H_2 \quad \textbf{Eq. (2-xiv)}$$

Disinfection is achieved by the production of Cl_2, which is formed as a secondary reaction at the anode (**Eq. (2-xv)**), where **Eq. (2-xvi)** represents the overall equation.

$$2Cl^- \rightarrow Cl_2 + 2e^- \quad \textbf{Eq. (2-xv)}$$

$$2NaCl + 2H_2O \rightarrow Cl_2 + 2NaOH + H_2 \quad \textbf{Eq. (2-xvi)}$$

In conjunction with **Eq. (2-i),** HOCl may be formed as per **Eq. (2-xvii)**. The reformation of chloride allows the EGMO process to repeat as long as an electric current is applied.

$$Cl_2 + NaOH \rightarrow OCl^- + NaCl + H^+ \quad \textbf{Eq. (2-xvii)}$$

Ozone and hydrogen peroxide are also formed during EGMO disinfection, although to a much smaller extent than the formation of Cl_2, as per **Eq. (2-xviii)** and (**2-xix)**, respectively. Furthermore, if bromide is present, HOBr may be produced as per **Eq. 2-viii**.

$$3H_2O \rightarrow O_3 + 6e^- + 6H^+ \quad \textbf{Eq. (2-xviii)}$$

$$O_2 + 2H_2O + 2e^- \rightarrow H_2O_2 + 2OH^- \quad \textbf{Eq. (2-xix)}$$

2.4. Disinfection By-Products: Occurrence in Swimming Pool and Spa Waters and the Ambient Air Above Pools

Many studies have investigated the occurrence of DBPs and other chemical contaminants in both drinking waters and wastewaters, with fewer studies of swimming pool waters being available. Arguably the most comprehensive study of DBPs in swimming pools was that of Richardson et al. (2010), who identified over 100 DBPs (including 8 haloalkanes, 9 haloacetic acids, 22 other haloacids, 9 halodiacids, 8 haloaldehydes, 24 halonitriles, 6 haloamides, 18 haloalcohols and 7 non-halogenated DBPs) in their investigation of five chlorinated and two brominated public swimming pools in Spain. Similarly, Daiber et al

(2016) identified over 100 DBPs (bromoimidazoles, bromoanilines, haloacids, halonitriles, haloamides, halonitromethanes, haloketones, haloaldehydes, halophenols, halobenzenes, halobenzenediols, bromomethanesulfenic acid esters, aldehydes, ketones, an iodo-THM and an organic chloramine), including a range of newly reported DBPs (bromoimidazoles, bromoanilines, bromomethanesulfenic acid esters), in a range of swimming pools and spas treated by either chlorine or bromine based disinfectants. More recently, Joseph (2017) identified a range of DBPs, including several halomethanes, haloacids, haloacetonitriles, haloaldehydes, haloketones, halonitromethanes, haloamides, haloalcohols and halophenols, as well as benzaldehyde, in her comprehensive investigation of DBPs in several chlorinated swimming pools across Spain. Although many different types of swimming pools exist, the majority of studies have primarily focused on chlorinated swimming pools, particularly those located indoors. Similarly, the ambient air of indoor swimming pool complexes has received some attention. Due to the vast numbers of reports of some DBPs (particularly trihalomethanes, haloacetic acids, chloramines and haloacetonitriles), this review will discuss overall trends for the occurrence of DBPs in the swimming pool environment: both swimming pool water and the ambient air of indoor swimming pool complexes. **Table 2-2** summarises the range of average concentrations for selected DBPs in swimming pool waters, with **Tables A1-1** to **A1-11** providing a complete summary. Similarly, **Tables A1-12** and **A1-13** provide a summary of THM and trichloramine concentrations reported in the ambient air of indoor swimming pool complexes.

2.4.1. Occurrence in Swimming Pool and Spa Waters

2.4.1.1. Trihalomethanes

Rook (1974) was the first to investigate trihalomethanes (THMs) in drinking water, with attention turning to swimming pool waters less than 6 years later (Beech et al., 1980; Norin and Renberg, 1980). To date, THMs, along with HAAs (discussed in **Section 2.4.1.2**), are the most commonly reported class of DBPs in the swimming pool environment. **Table 2-2** summarises the average occurrence of brominated and chlorinated THMs in a variety of swimming pool waters, with **Table A1-1** providing a more complete summary.

As opposed to reporting individual THM species, a value known as TTHM is often presented and refers to the sum of trichloromethane, bromodichloromethane, dibromochloromethane and tribromomethane. TTHM concentrations were generally lower in chlorinated pools that employ ozone compared to those reported in pools treated solely by chlorination (Kelsall and Sim, 2001; Zhang et al., 2015), whilst chlorinated pools filled with salt water contained on average the highest TTHMs concentrations (Beech et al., 1980; Boudenne et al., 2017; Chowdhury, 2016; Chowdhury et al., 2016; Manasfi et al., 2016;

Manasfi et al., 2017b; Parinet et al., 2012). Lower TTHM concentrations (12 to 311 μg L^{-1}; Glauner et al., 2005; Manasfi et al., 2016; Panyakapo et al., 2008; Simard et al., 2013; Tang and Xie, 2016; Yang et al., 2018, 2016; Zhang et al., 2015) were reported for outdoor pools treated by chlorination compared to chlorinated pools located indoors, which is likely due to the increased volatilisation and UV degradation in outdoor pools, which is discussed further in **Sections 2.6.1** and **2.6.3**.

Considering individual THMs, chloroform and bromoform were the most abundant THMs in pools treated with chlorine and bromine, respectively, with concentrations of up to 551 and 400 μg L^{-1} reported in these pools for chloroform and bromoform, respectively (Klosok-Bazan et al., 2018; Norin and Renberg, 1980). On average, chlorinated pools had higher concentrations of bromodichloromethane and dibromochloromethane than pools treated with bromine. Pools where ozone in addition to chlorine was employed generally reported lower average concentrations of THMs compared to pools where only chlorination was employed. Whilst the average concentrations of THMs were lower compared to chlorinated pools, pools where EGMO was employed reported a higher ratio of bromo- and mixed bromochloro-THMs, which is likely due to the higher bromide content, which, as discussed in **Section 2.5.2**, is likely added as an impurity in the added salt. A similar trend was observed for seawater filled pools treated by chlorination, which is likely due to the higher bromide content of the filling water, as discussed in **Section 2.5.1**. Spas treated with chlorine generally had lower concentrations of THMs than chlorinated pools, whilst bromine treated spas contained higher levels of THMs than those reported in brominated pools. These observations are likely due to (i) the increased formation of THMs at the higher temperatures of spas compared to pools, and (ii) the increased partitioning of THMs into the air above the pool as a result of the higher temperature and water agitation in spas, which would have an increased effect on chlorinated THMs due to their higher volatility.

While Daiber et al. (2016) identified the presence of bromochloroiodomethane in one brominated public spa, only two published studies have reported iodinated THMs (I-THMs), where individual concentrations up to 17 and 8.5 μg L^{-1} were reported for chlorinated pools and a spa, respectively (Carter et al., 2015; Yeh et al., 2014).

2.4.1.2. Haloacetic Acids

Haloacetic acids (HAAs) have been extensively studied in drinking water, however studies investigating their occurrence in swimming pool waters only began to be published in 1999 (Martínez et al., 1999). Early studies investigated only chlorinated HAAs, but attention quickly expanded to the brominated and mixed chlorinated/brominated analogues. As presented in **Table 2-2**, the average concentrations of HAAs will be the focus of discussion

in this section, whilst **Table A1-2** provides a more complete summary of the occurrence of HAAs in a variety of swimming pool and spa waters.

Excluding bromine treated or seawater filled pools and spas, the most abundant of the HAAs are dichloroacetic acid (DCAA) and trichloroacetic acid (TCAA), where concentrations of up to 2400 and 2600 μg L^{-1} have been reported, respectively (Yeh et al., 2014). As observed with THMs and presented in **Table 2-2**, for chlorinated pools, HAA concentrations generally decrease as bromine substitution increases, which is likely a result of the low bromide concentrations in chlorinated pools. Likewise, for bromine treated pools, brominated HAAs generally increase in concentration as bromine substitution increase, with dibromoacetic acid and tribromoacetic acid the dominant species. Chlorinated pools where ozone is employed contained lower HAAs on average than those treated solely by chlorine, whilst EGMO treated pools generally contained the lowest concentrations of HAAs.

Chlorinated spas generally contained similar or lower concentrations of HAAs compared to chlorinated pools, excluding TCAA where concentrations were approximately double, whilst brominated spas were reported to generally contain higher concentrations of HAAs than brominated pools. As discussed in **Sections 2.6.4** and **2.6.5**, factors including water agitation and temperature are likely to affect the formation of HAAs.

2.4.1.3. Inorganic Halamines

Chloramines have been investigated in swimming pools and spas, with their average occurrence summarised in **Table 2-2** and a more complete summary provided in **Table A1-3**. Regardless of pool type or treatment method, trichloramine was generally the dominant chloramine, followed by di- and mono-chloramine, although, as discussed in **Section 2.6.5**, pH plays an important role in DBP occurrence, particularly for chloramines. On average, chloramines were detected at higher concentrations in chlorinated pools filled with fresh water compared to those filled with seawater, where maximum concentrations of between 11 to 3412 μg L^{-1} and between 110 to 490 μg L^{-1} have been reported, respectively (Chowdhury et al., 2016; Wang et al., 2014; Weaver et al., 2009). Generally (**Table 2-2**), brominated pools had lower concentrations of chloramines compared to chlorinated pools, where maximum concentrations between 18 and 300 μg L^{-1} have been reported (Daiber et al., 2016; Richardson et al., 2010). Brominated spas contained slightly higher concentrations of chloramines than brominated pools, up to 363 μg L^{-1} (Daiber et al., 2016), which is likely due to the higher release of human derived chloramine precursors and an increased formation due to the higher operating temperature used in spas, as discussed in **Section 2.6.5**.

Bromamines are known to form in the presence of ammonia in chlorinated waters (NRC, 1980). Despite the possibility that other halamines (e.g. bromamines, mixed bromochloramines) may be present in the swimming pool environment, their occurrence has yet to be investigated due to the lack of suitable analytical methods. Further work is required to develop such analytical methods in order to fully understand halamines in the swimming pool environment.

2.4.1.4. Haloacetonitriles

Few studies investigating haloacetonitriles (HANs) in swimming pool waters and spas have been reported to date and they have mainly focused on bromochloroacetonitrile, dibromoacetonitrile, dichloroacetonitrile and trichloroacetonitrile (BCAN, DBAN, DCAN and TCAN, respectively), as summarised in **Table 2-2** and **Table A1-4**. Chlorinated and brominated species dominated pools treated with chlorine and bromine, respectively, with BCAN concentrations reported higher in chlorinated pools compared to those treated with bromine. Of the HANs investigated, DCAN and DBAN were generally the most abundant in pools treated with chlorine and bromine, respectively, with maximum concentrations of 89 and 39 μg L^{-1} being reported (Daiber et al., 2016; Hang et al., 2016). Where disinfection was achieved by bromine, Daiber et al. (2016) reported a higher concentration of DBAN in spas compared to pools, which is likely due to the higher release of nitrogen containing anthropogenic chemicals due to the elevated temperatures, as discussed in **Section 2.6.5**. As shown in **Table A1-4**, limited reports of other HANs have been published, although concentrations are much lower, up to 3.0 μg L^{-1} (Carter et al., 2015; Kanan, 2010), compared to those discussed above.

2.4.1.5. *N*-Nitrosamines

Few studies have investigated the occurrence of *N*-nitrosamines in swimming pool waters, as summarised in **Table 2-2**, with a more complete summary of the studies provided in **Table A1-5**. Walse and Mitch (2008) were the first researchers to investigate *N*-nitrosamines in chlorinated swimming pool waters, reporting maximum concentrations of *N*-nitrosodimethylamine (NDMA) of 44, 6.9 and 429 ng L^{-1} in indoor pools, outdoor pools and a heated spa, respectively. Considering average concentrations, NDMA is generally higher in chlorinated spas than chlorinated pools, which is likely due to the increased release of anthropogenic NDMA precursors from swimmers due to the higher water temperature (discussed further in **Section 2.6.5**). Although similar average concentrations have been reported for NDMA, maximum concentrations have been reported between 6.9 and 208 ng L^{-1} for pools treated by chlorination (Kim and Han, 2011; Wang et al., 2011). Other nitrosamines, *N*-nitrosodiethylamine, *N*-nitrosomorpholine and *N*-nitrosoethylmethylamine

have been detected in chlorinated pools at concentrations up to 72, 34 and 26 ng L^{-1}, respectively (Kim and Han, 2011; Wang et al., 2011), with *N*-nitrosopyrrolidine reported at concentrations up to 127 ng L^{-1} (Pozzi et al., 2011), although information regarding pool type or treatment method was not presented.

2.4.1.6. Haloacetaldehydes

Very limited information exists on haloacetaldehydes (HALs) in swimming pool waters, as summarised in **Table 2-2** and **Table A1-6**. Chloral hydrate (CH), the monohydrate of trichloroacetaldehyde, has been the most commonly investigated, with concentrations of up to 400, 190, 10 and 23 µg L^{-1} in chlorinated indoor, chlorinated outdoor, chlorinated with ozone and EGMO treated swimming pools, respectively (Carter et al., 2015; Lee et al., 2010; Manasfi et al., 2016). CH was not detected in seawater filled or brominated pools and was detected at lower concentrations in brominated spas compared to those that were chlorinated, up to 2.9 and 405 µg L^{-1}, respectively (Carter et al., 2015; Daiber et al., 2016; Manasfi et al., 2016; Manasfi et al., 2017a), which is likely due to the higher availability and reactivity of bromine based disinfectants present in these waters. Dichloroacetaldehyde (up to 23 µg L^{-1}) has been reported in one study, although pool type and treatment method were not presented (Serrano et al., 2011). Although at much lower concentrations (0.3 to 2.4 µg L^{-1}), a few studies have reported the occurrence of other HALs (dibromoacetaldehyde, dibromochloroacetaldehyde and tribromoacetaldehyde) in swimming pool waters (Carter et al., 2015; Manasfi et al., 2016). Concentrations of up to 12.4 µg L^{-1} of tribromoacetaldehyde have been reported in some seawater filled, chlorinated indoor pools (Manasfi et al., 2017b).

2.4.1.7. Haloketones

Little is known about haloketones (HKs) in swimming pool or spa waters, with their known occurrence summarised in **Table 2-2** and **Table A1-7**. The majority of researchers have investigated chlorinated pools, reporting 1,1-dichloropropanone (1,1-DCP) and 1,1,1-trichloropropanone (1,1,1-TCP) at concentrations up to 7.7 and 15 µg L^{-1}, respectively (Hang et al., 2016), although 1,1,1-TCP was reported at significantly higher concentrations (up to 180 µg L^{-1}; Hang et al. (2016)) in some pools. Carter et al. (2015) was the only known study to investigate other HKs, 1,2-DCP and chloropropanone, where concentrations up to 1.8 µg L^{-1} were reported. Only one study has investigated HKs in brominated (treated with BCDMH or sodium bromide in combination with TCICA) pools or spas, although both 1,1,1-TCP and 1,2-DCP were below detection limits in all cases (Daiber et al., 2016). No studies have reported the occurrence of brominated ketones, despite their likely occurrence in pools treated with bromine.

Table 2-2: Summary of the occurrence of disinfection by-products in swimming pool and spa waters. Unless otherwise stated, concentrations are presented in μg L^{-1} and represent a range of the average concentrations reported. Where only one value is present, either only one known report exists or, of the existing reports, only one presented data regarding average concentrations. Where no data is included, either the concentration(s) were below the limit of detection or there is no known report of the given disinfection by-product(s). More complete summaries can be found in **Tables A1-1** to **A1-12**.

Disinfection By-Product	Swimming Pools					Spas		Reference(s)
	Chlorine Based	Bromine Based	EGMO	Chlorine & Ozone	Seawater Filled	Chlorine Based	Bromine Based	
Trihalomethanes (THMs)								
Bromodichloromethane	0.13-167	0.5-0.7	9.8-10	1.1-106	0.29-5	0.1	2.9	(Abbasnia et al., 2018; Aprea et al., 2010; Beech et al., 1980; Benoit and Jackson, 1987; Carter et al., 2015; Chowdhury et al., 2016; Daiber et al., 2016; Font-Ribera et al., 2010a; Glauner et al., 2005; Golfinopoulos, 2000; Hang et al., 2016; Kelsall and Sim, 2001; Klosok-Bazan et al., 2018; Lee et al., 2010, 2009; Lourencetti et al., 2012; Manasfi et al., 2016; Parinet et al., 2012; Richardson et al., 2010; Weaver et al., 2009; Zhang et al., 2015)
Dibromochloromethane	0.49-120	2.4-2.5	8.9-9.1	0.2-2	3.57-27	0.14	4.67	
Tribromomethane	0.04-64	57-152	4.1-19	0.9-47	50-651	0.11	182-1253	
Trichloromethane	8.65-243	0.2-0.21	14-27	7.4-141	0.1-6	19-264[b]	1.6	
Haloacetonitriles (HANs)								
Bromoacetonitrile	1							(Carter et al., 2015; Daiber et al., 2016; Hang et al., 2016; Kanan, 2010; Lee et al., 2010; Li and Blatchley, 2007; Manasfi et al., 2016; Manasfi et al., 2017b; Tardif et al., 2016a; Yang et al., 2018; Yeh et al., 2014; Zhang et al., 2015)
Bromochloroacetonitrile	0.63-9.2	1.8	3.5	0.4	0.93		1.8-5.6	
Chloroacetonitrile	1.14-2.4							
Dibromoacetonitrile	0.1-5.8	37	2.6	0.4	19-27		80-219	
Dichloroacetonitrile	0.1-75		3.8	1.3-5.3	8.99	14[b]		
Trichloroacetonitrile	0.03-1.2							

Table 2-2 continued

Disinfection By-Product	Swimming Pools					Spas		Reference(s)
	Chlorine Based	Bromine Based	EGMO	Chlorine & Ozone	Seawater Filled	Chlorine Based	Bromine Based	
Haloacetic Acids (HAAs)								
Bromoacetic Acid	2-37	4.7		16	3.75-55		46-62	(Catto et al., 2012b; Chowdhury et al., 2016; Daiber et al., 2016; Dehghani et al., 2018; Font-Ribera et al., 2016; Hang et al., 2016; Kanan, 2010; Lee et al., 2010; Manasfi et al., 2016; Manasfi et al., 2017b; Parinet et al., 2012; Sarrión et al., 2000; Tang and Xie, 2016; Wang, 2011; Yeh et al., 2014; Zhang et al., 2015)
Bromochloroacetic Acid	1.8-510	2.2		425	4.27-65	2.6[b]	13-294	
Bromodichloroacetic Acid	2.71-61	8.9			2.00-12	12[b]	10-117	
Chloroacetic Acid	4.22-113			41	1.25-96	31[b]	-3.9	
Dibromoacetic Acid	1-28	123			16-307		337-1795	
Dibromochloroacetic Acid	2.7-367	4.05		1.2	3.1-103		4.4-14	
Dichloroacetic Acid	23-982	2.2	34	12-200	1.67-4.79	343[b]	27-89	
Tribromoacetic Acid	5.6-19	72		8.1	43-186		73-97	
Trichloroacetic Acid	19-978	64	97	17-20	2.56-27	1865[b]	13-37	
Halamines and Cyanogen Halides								
Dichloramine	11-430	51			220		40-142	(Catto et al., 2012b; Chowdhury et al., 2016; Daiber et al., 2016; Font-Ribera et al., 2010b, 2016; Lian et al., 2014; Richardson et al., 2010; Simard et al., 2013; Weaver et al., 2009; Zare Afifi and Blatchley, 2016)
Monochloramine	10-323	67-270			220		48-205	
Trichloramine	7-1500	12			70		91-183	
Cyanogen Chloride	4.4-24	3.7					3.2	
Cyanogen Bromide	3.3-25	19-52					4.9-125	
Haloacetaldehydes (HALs)								
Dibromoacetaldehyde	2.4							(Carter et al., 2015; Daiber et al., 2016; Lee et al., 2010; Manasfi et al., 2016; Manasfi et al., 2017b; Serrano et al., 2011)
Dibromochloroacetaldehyde	0.3							
Dichloroacetaldehyde	1.8-23[c]							
Tribromoactetaldehyde					8.4			
Trichloroacetaldehyde	17-301		10	3.6	190	405	2.9	

Table 2-2 continued

Disinfection By-Product	Swimming Pools					Spas		Reference(s)
	Chlorine Based	Bromine Based	EGMO	Chlorine & Ozone	Seawater Filled	Chlorine Based	Bromine Based	
Haloketones (HKs)								
Chloropropanone	1.9							(Carter et al., 2015; Hang et al., 2016; Manasfi et al., 2016; Spiliotopoulou et al., 2015; Yeh et al., 2014)
1,2-Dichloropropanone	0.8							
1,1-Dichloropropanone	0.4-21							
1,1,1-Trichloropropanone	1.3-46			11				
***N*-Nitrosamines**								
N-Nitrosodiethylamine	1.2-35[d]							(Carter et al., 2015; Jurado-Sánchez et al., 2010; Kim and Han, 2011; Walse and Mitch, 2008; Wang, 2011)
N-Nitrosodimethylamine	5.3-52					5.5-313		
N-Nitrosodi-*n*-butylamine	15-141							
N-Nitrosoethylmethylamine	7.1-16							
N-Nitrosomorpholine	3.1-26							
N-Nitrosopiperidine	4.4							
N-Nitrosopyrrolidine	4.5-77[d]							
Halonitromethane (HNMs)								
Bromochloronitromethane	4							(Kanan, 2010; Montesinos and Gallego, 2012; Yang et al., 2018; Yeh et al., 2014; Zhang et al., 2015)
Bromonitromethane	1.5							
Tribromonitromethane	1.2							
Trichloronitromethane	0.1-2.7			0.4				
Inorganic Anions								
Bromate	3	10-900[b,c]						(Chowdhury et al., 2016; E et al., 2016; Lee et al., 2010; Manasfi et al., 2016; Parinet et al., 2012; Righi et al., 2014; Spiliotopoulou et al., 2015)
Bromide	0.2-79[a]				0.6-86[a]			
Chlorate	0.04-37[a]							
Chlorite	20-22[c]							
Nitrate	0.004-63[a]		23[a]	13[a]				

Table 2-2 continued

Disinfection By-Product	Swimming Pools					Spas		Reference(s)
	Chlorine Based	Bromine Based	EGMO	Chlorine & Ozone	Seawater Filled	Chlorine Based	Bromine Based	
Haloacetamides (HAAms)								
Dibromoacetamide	0.6-1.9							(Carter et al., 2015; Yeh et al., 2014)
Dichloroacetamide	1.5							
Trichloroacetamide	2-2.7							
Total Organic Halogen (TOX)								
Total Organic Halogen (TOX)	140-480		47-1215	880-1080[c]				(Daiber et al., 2016; Font-Ribera et al., 2016; Kelsall and Sim, 2001; Manasfi et al., 2017b; Yeh et al., 2014)
Total Organic…. Bromine (TOBr)	0.75-200	4897	0.3-16	53-84[c]			4197-18239	
Chlorine (TOCl)	139-3682	1337	47-1198	1081-9512[c]	707		1213-13860	
Iodine (TOI)	0.63		0.04-1.9					

(a) Reported in mg L^{-1}. **(b)** Ozone also employed. **(c)** Range presented. **(d)** Treatment method not provided. **EGMO**: Electrochemically-generated mixed-oxidant.

2.4.1.8. Halonitromethanes

As summarised in **Table 2-2** and **Table A1-8**, few studies have investigated halonitromethanes (HNMs) in pool or spa waters. Trichloronitromethane (TCNM) is the most investigated HNM, where concentrations up to 5 μg L^{-1} have been reported for chlorinated pools (Tardif et al., 2015). Daiber et al. (2016) is the only known study of HNMs in spas or brominated pools, although TCNM was not detected in any waters investigated. Several other HNMs (tribromonitromethane, bromochloronitromethane and bromonitromethane) have been investigated, where maximum concentrations between 1.2 and 11 μg L^{-1} were reported (Kanan, 2010; Yeh et al., 2014).

2.4.1.9. Haloacetamides

Haloacetamides (HAAms) are an almost unexplored DBP class in swimming pool waters, with only two publications to date. Not all investigated HAAms were detected, and as summarised in **Table 2-2** and **Table A1-9**, on average dibromoacetamide, dichloroacetamide and trichloroacetamide were reported at similar concentrations in the investigated chlorinated pools, where maximum concentrations between 2 and 3.1 μg L^{-1} were reported (Carter et al., 2015; Yeh et al., 2014).

2.4.1.10. Inorganic Anions

A few studies have investigated the occurrence of inorganic anions which are DBPs in swimming pool waters. The relevant results from these studies are summarised in **Table A1-10**, with a simplified summary provided in **Table 2-2**. The inorganic anions bromide and chloride play multiple roles in disinfected water systems and these roles will be discussed in **Sections 2.6.1** and **2.6.2**. Additionally, although measured in swimming pool waters, fluoride, sulfate and phosphate have been excluded from this review as they are unlikely to directly take part in DBP formation under the conditions commonly found in swimming pool waters.

The occurrence of bromate has been limited to pools where ozone was employed as a secondary treatment, with concentrations below detection in all other investigated pools (Kelsall and Sim, 2001; Michalski and Mathews, 2007). As bromide was not detected in the pools treated with ozone, but was present in the chlorinated swimming pools in the study by Michalski and Mathews (2007), bromide was likely oxidised to bromate in the presence of ozone, as observed in bromide containing waters (von Gunten and Hoigne, 1994). Where bromide is limited, the bromide to bromate oxidation likely goes to completion. However, where there is a continual input of bromide (e.g. via bromine based disinfectant), the oxidation process may not go to completion and both bromide and bromate may exist. It is

evident that both the bromide availability and treatment process have an effect on the chemical water quality of pools, particularly the occurrence of bromide and bromate, with further investigations required to fully understand their implications (discussed further in **Sections 2.5.1, 2.5.2, 2.6.1** and **2.6.2**). Although reported at higher concentrations in pools treated with with ozone, chlorate has also been detected in pools where ozone was not employed as a secondary treatment. For example, Michalski and Mathews (2007) reported the occurrence of chlorate (ClO_3^-) in two pools treated with ozone (2.1 to 3.2 mg L^{-1}), three pools treated with chlorine dioxide (22 to 23 mg L^{-1}) and at even higher concentrations in two pools treated with sodium hypochlorite (29 to 32 mg L^{-1}), where all pools were located indoors. The occurrence of chlorate in pools can be explained by (i) the direct addition as a DBP in pre-treated filling water, (ii) the direct addition as a degradation product of hypochlorite stock solutions (Garcia-Villanova et al., 2010), (iii) formation in pools due to the degradation of hypochlorite which has been shown to increase in the presence of metal oxides (Liu et al., 2012; Sharma et al., 2017), (iv) or in cases where ozone is employed as secondary treatment, formation due to oxidation of hypochlorite by ozone and hydroxyl radicals via a several step mechanism (Von Gunten, 2003). Similarly, chlorite has been detected in pools treated with chlorine and chlorine dioxide, where concentrations up to 2.5 mg L^{-1} have been reported (Michalski and Mathews, 2007).

2.4.1.11. Total Organic Halogen

The structures of many DBPs in swimming pool waters remain unknown and, therefore, not all DBPs can be individually identified. However, the bulk parameter, total organic halogen (TOX), sometimes referred to as adsorbable organic halogen (AOX), can be used as a measure of all halogenated organic compounds in a water sample. As a bulk measurement of halogen, TOX is reported as a chloride equivalent concentration (Kristiana et al., 2015). Furthermore, individual measurement of chlorine, bromine and iodine incorporated into NOM can be carried out, known as total organic chlorine (TOCl), total organic bromine (TOBr) and total organic iodine (TOI), respectively. TOCl, TOBr and TOI are reported as chloride, bromide and iodide concentrations, respectively (Kristiana et al., 2015). **Table A1-11** summarises the total TOX and individual TOCl, TOBr, and TOI concentrations previously reported in swimming pools and spas, with **Table 2-2** presenting a more simplified version.

Considering average concentrations (**Table 2-2**), TOCl dominated chlorinated pools, whilst TOBr was highest in pools treated with bromine, which is likely due to the higher availability of chlorine and bromine in these waters, respectively. Brominated spas had significantly higher concentrations of TOBr than those reported for brominated pools,

indicating that larger concentrations of brominated organic compounds exist in these spa waters, which is likely due to the higher release of anthropogenic precursors and faster reaction rates as a result of the elevated temperatures, as discussed in **Section 2.6.5**. Manasfi et al. (2017b) is the only known report of TOCl (measured as extractable organic chlorine (EOX)) in chlorinated pools filled with seawater, where concentrations up to 920 μg L^{-1} were measured. Furthermore, only one study has reported the occurrence of TOI, which was present in significantly lower concentrations compared to TOCl or TOBr, attributable to the minimal availability of iodine in pools or spas (Yeh et al., 2014).

2.4.1.12. Cyanogen Halides

A limited number of studies have investigated cyanogen halides in swimming pools or spas. All known studies have focused on the two cyanogen halide species, cyanogen chloride (CNCl) and cyanogen bromide (CNBr), as summarised in **Table 2-2**. As observed with other DBP classes, CNCl concentrations were reported to be higher than CNBr in pools treated with chlorine, although Weaver et al. (2009) observed cyanogen bromide up to 325 μg L^{-1} in a study of eleven indoor pools treated by chlorination. CNBr concentrations were higher in brominated spas compared to pools treated by bromine, with maximum concentrations reported to be 125 and 52 μg L^{-1}, respectively (Daiber et al., 2016), and were comparable to CNCl concentrations in chlorinated pools (Daiber et al., 2016; Lian et al., 2014; Weaver et al., 2009; Weng and Blatchley, 2011; Zare Afifi and Blatchley, 2015).

2.4.2. Occurrence in the Ambient Air Above Swimming Pool and Spa Waters

Although the focus was not specifically DBPs, a comprehensive analysis of the quality of air contained in an indoor swimming pool facility in Greece was undertaken (Tolis et al., 2018). The authors monitored the levels of a range of compounds, including NO_2, O_3, a range of anions (Cl^-, $NO_3^-$and SO_4^{2-}) and cations (Na^+, NH_4^+, K^+, Mg^{2+} and Ca^{2+}), several volatile organic compounds (benzene, tetrachloroethylene, octane, ethylbenzene, *p*-xylene, *m*-xylene, *o*-xylene, 1,2,4-trimethylbenzene, toluene, styrene, α-pinene, D-limonene, naphthalene, 1,2,3-trimethylbenzene, trichloromethane) and the more general parameter PM 2.5 (particulate matter larger than 2.5 microns), where many of the measured concentrations were significantly higher than those measured directly in the ambient air outside the swimming pool facility. Operational conditions (e.g. chlorination practices and relatively high humidity) were suggested to be the major factors affecting the air quality (Tolis et al., 2018), which aligns with operational conditions and other external factors being highlighted as key factors to affect DBP formation and occurrence in the swimming pool environment, as discussed in **Sections 2.5** and **2.6**.

2.4.2.1. Trihalomethanes

Several studies have investigated the occurrence of THMs in the ambient air of indoor swimming pool facilities, with a summary provided in **Table A1-12**. Unless otherwise stated, all reports of THMs in the ambient air were for swimming pools located indoors and treated by chlorination.

Trichloromethane (chloroform), the most investigated THM in the ambient air of swimming pools, is generally reported to be between 12 and 320 µg m^{-3} (Aggazzotti et al., 1998; Aprea et al., 2010; Cammann and Hübner, 1995; Caro and Gallego, 2008; Catto et al., 2012b; Lévesque et al., 2000; Silva et al., 2012; Tardif et al., 2015, 2016), although other studies have reported significantly lower concentrations (12 to 81 µg m^{-3}) (Font-Ribera et al., 2010b; Lourencetti et al., 2012; Richardson et al., 2010; Thiriat et al., 2009; Westerlund et al., 2018). Compared to other reported studies, Aggazzotti et al. (1990) reported much higher chloroform concentrations (66 to 650 µg m^{-3}), although these are consistent with concentrations reported in their later investigations (16 to 853 µg m^{-3}) (Aggazzotti et al., 1995), as well as those reported in a study of sixteen chlorinated whirlpool spas (4 to 750 µg m^{-3}) (Benoit and Jackson, 1987) and several chlorinated pools in Norway (89 to 477 µg m^{-3}) (Nitter et al., 2017). Other THMs, bromodichloromethane, dibromochloromethane and tribromomethane, have been reported in the ambient air above indoor chlorinated swimming pools at concentrations of below detection to 24, below detection to 26 and 0.2 to 23 µg m^{-3}, respectively (Aggazzotti et al., 1998; Cammann and Hübner, 1995; Caro and Gallego, 2008; Catto et al., 2012b; Font-Ribera et al., 2010b; Lourencetti et al., 2012; Richardson et al., 2010), with a few studies reporting higher concentrations, up to 155, 205 and 103 µg m^{-3} for bromodichloromethane, dibromochloromethane and tribromomethane, respectively (Nitter et al., 2017; Tardif et al., 2016a, 2016b, 2015). Lahl et al. (1981) measured trichloromethane and bromodichloromethane in the ambient air above eight chlorinated pools in concentrations between 10 to 384 and 0.1 to 39 µg m^{-3}, respectively, although whether the pools were located indoors or outdoors was not specified. Similarly, trichloromethane, bromodichloromethane, dibromochloromethane and tribromomethane have been found in the ambient air of other indoor swimming pools in concentrations of 11 to 13000, 8.7, 3.1 and 0.8 µg m^{-3}, respectively, although the disinfection methods of these pools were not provided (Chen et al., 2011; Erdinger et al., 2004; Fantuzzi et al., 2001; Hsu et al., 2009; Lévesque et al., 1994). Although not detected in all samples, THMs have also been reported in the ambient air above chlorinated seawater filled swimming pools, up to 29, 19, 150 and 1600 µg m^{-3} for trichloromethane, bromodichloromethane, dibromochloromethane and tribromomethane, respectively (Boudenne et al., 2017; Chowdhury, 2016; Chowdhury et al., 2016; Manasfi et al., 2017b). THMs (up to 477, 94, 38

and 319 μg m^{-3} for trichloromethane, bromodichloromethane, dibromochloromethane and tribromomethane, respectively) have also been reported in the ambient air above chlorinated pools filled with a mix of freshwater and seawater (Nitter et al., 2017).

To the best of our knowledge, only two studies have investigated THMs in the ambient air of swimming pools treated with bromine based disinfectants. Lourencetti et al. (2012) reported concentrations of 1.8 to 6.9, 1.9 to 4.2, 6.4 to 8.7 and 55 to 928 μg m^{-3} for trichloromethane, bromodichloromethane, dibromochloromethane and tribromomethane, in the air above an indoor swimming pool treated by bromination. Similarly, Richardson et al. (2010) found concentrations of 1.7 to 9.4, 1.7 to 4.8, 6.1 to 9.7 and 53 to 101 μg m^{-3} for trichloromethane, bromodichloromethane, dibromochloromethane and tribromomethane, respectively, in their investigation of an indoor swimming pool treated with BCDMH. As observed in swimming pool water and discussed further in **Sections 2.5** and **2.6**, the speciation of THMs in the ambient air is influenced by the water quality of the corresponding swimming pool, particularly by filling water composition and disinfection practices.

2.4.2.2. Inorganic Halamines

A few studies have investigated halamines in the air above swimming pools, although, to date, studies have focused on trichloramine in the air of indoor swimming pool complexes (summarised in **Table A1-13**). Although not detected in all samples, trichloramine has been reported in the ambient air of indoor swimming pool complexes that employ chlorination, at concentrations between 1 and 1340 μg m^{-3}, although, on average, concentrations are generally between 23 and 637 μg m^{-3} (Andersson et al., 2018; Bernard et al., 2011; Bessonneau et al., 2011; Catto et al., 2012b; Chu et al., 2013; Font-Ribera et al., 2016; Fornander et al., 2013; Gomà et al., 2017; Jacobs et al., 2007; Lévesque et al., 2015; Parrat et al., 2012; Predieri and Giacobazzi, 2012; Richardson et al., 2010; Schmalz et al., 2011a; Seys et al., 2015; Tardif et al., 2016b; Westerlund et al., 2018; Zare Afifi and Blatchley, 2015; Zwiener and Schmalz, 2015). Richardson et al. (2010) reported an average trichloramine concentration of 80 μg m^{-3} in the air of an indoor swimming pool treated with BCDMH, with concentrations varying (70 to 100 μg m^{-3}) over the twelve samples taken. Monochloramine was investigated in the ambient air of forty-one indoor and chlorinated swimming pool complexes, with concentrations of 70, 320 and 150 μg m^{-3} reported for the minimum, maximum and average, respectively (Tardif et al., 2016a).

2.5. Disinfection By-Products in Swimming Pools and Spas: Precursor Input and Implications

Compared to drinking waters, DBP formation in pools and spas is increased due to the higher input of organic matter and constant addition of disinfectants. Whilst DBP formation has been extensively studied in many different waters (e.g: Richardson et al., 2007, 2010; Richardson, 2009; Richardson and Kimura, 2016; Richardson and Ternes, 2014), and despite the efforts of the many pool and spa studies covered in this review, much is still unknown about DBP formation in the swimming pool environment. Daiber et al. (2016) were the first to follow the water quality from source to pool, by investigating the DBP occurrence and mutagenicity of the waters at each stage: in source, finished, tap, pool and spa waters. Considering average total molar concentrations of DBPs, an increase of 610% was observed between filling and pool waters, whilst a 900% increase was observed between filling and spa waters. Where pools and spas were at the same location, spas contained approximately 140% more DBPs than pools. Their results provide evidence that DBP formation is prominent in swimming pool and spa waters, proposed to be mainly attributable to human input (human body excretions, pharmaceuticals and personal care products) (Daiber et al., 2016). In addition to input via humans, two additional sources of contaminants (and hence possible DBP precursors) have been identified: the filling water and the chemicals (particularly the disinfectants) used during treatment. Bradford (2014) summarises the input, and subsequent reaction(s) of, organic nitrogen compounds likely to be present in pools, paying particular attention to those contained in body fluids and the formation of organic chloramines. Although providing detailed information, the review is more focused on the fundamental chemistry behind the origin and fate of human derived nitrogen containing compounds. With limited information provided regarding DBPs in a broader sense, the review by Bradford (2014) is outside the scope of the current review, and as such will not be further discussed. Furthermore, while their input may be a result of one or more of the categories discussed here, the impact of a range of substances, including nanoparticles, metal oxides and metal ions, has been reviewed elsewhere (Sharma et al., 2017). Although these compounds are potentially relevant to the swimming pool environment, Sharam et al. (2017) discuss their impact on DBPs in a broader sense (i.e. in chlorinated waters) and, as such, their review will be only briefly discussed in the current review.

As discussed in **Section 2.4**, due to the wide variety of DBPs and lack of suitable analytical methods, not all individual DBPs and their precursors can be investigated in the swimming pool environment. However, bulk parameters, like total organic carbon (TOC) and dissolved organic carbon (DOC), are easily measured and are often used to assess the quality of the water, in terms of DBPs and their precursors. TOC refers to the dissolved,

particulate and colloidal organic matter contained within water, whilst DOC refers to the soluble organic matter that cannot be removed by a 0.45 μm filter (Potter and Wimsatt, 2009). **Table A1-12** summarises the occurrence of these bulk parameters in swimming pool and spa waters to date.

For chlorinated indoor swimming pools, TOC concentrations are generally reported to be between 0.02 and 7.3 mg L^{-1} (Bessonneau et al., 2011; Carter et al., 2015; De Laat et al., 2011; Glauner et al., 2005; Lee et al., 2010; Sa et al., 2011; Wang, 2011; Xiao et al., 2012), although some studies have reported maximum concentrations of 16 to 71 mg L^{-1} (Dehghani et al., 2018; Kanan, 2010; Lee et al., 2009; Plewa et al., 2011; Wang et al., 2013). Similar TOC concentrations have been reported for chlorinated pools located outdoors (0.02 to 39 mg L^{-1}) (Glauner et al., 2005; Klosok-Bazan et al., 2018; Manasfi et al., 2016; Plewa et al., 2011; Tang and Xie, 2016; Wang, 2011; Xiao et al., 2012; Yang et al., 2018, 2016; Yeh et al., 2014; Zhang et al., 2015). Where all pools were located indoors, TOC concentrations have been measured for chlorinated pools additionally treated with UV or ozone (5.2 to 18 and 0.7 to 27 mg L^{-1}, respectively), although a higher maximum concentration was reported in some ozonated pools (82 mg L^{-1}) (Lee et al., 2009, 2010; Plewa et al., 2011; Wang et al., 2013; Zhang et al., 2015). TOC concentration has been measured in two studies of twenty-five and twenty-six indoor pools treated with EGMO, where concentrations of 0.4 to 12 and 1.9 to 5.8 mg L^{-1} were reported (Lee et al., 2009, 2010). DOC concentration has been studied in several indoor swimming pools (1.3 to 39, 4.9 to 9.5 and 8.0 to 25 mg L^{-1}), where pools were treated by chlorination, chlorination with UV and chlorination with ozone, respectively (Hang et al., 2016; Schmalz et al., 2011b; Tardif et al., 2016a; Wang et al., 2013). TOC and DOC concentrations have been presented in several other studies (below detection to 28 and 0.6 to 14 mg L^{-1}, respectively), although not all pool details were provided (Chu and Nieuwenhuijsen, 2002; Font-Ribera et al., 2016; Jmaiff Blackstock et al., 2017; Lempart et al., 2018b; Maia et al., 2014; Panyakapo et al., 2008; Prieto-Blanco et al., 2012; Spiliotopoulou et al., 2015; Wang et al., 2014). Generally, chlorinated spas reported higher TOC/DOC concentrations (up to 155 mg L^{-1}) than those found in chlorinated swimming pools (Benoit and Jackson, 1987; Carter et al., 2015; Jmaiff Blackstock et al., 2017; Plewa et al., 2011; Wang et al., 2014), which may be due to the higher operation temperature of spas, promoting anthropogenic release of TOC from bathers, discussed further in **Section 2.6.5**. One study presented even higher concentrations for whirlpool spas treated with bromine, up to 345 mg L^{-1} (Benoit and Jackson, 1987). Only one known study has provided TOC concentrations for indoor pools treated with bromine, where concentrations were generally higher than indoor pools treated with chlorine, being up to 125 mg L^{-1} (Plewa et al., 2011). Concentrations of DOC and TOC of 1.0 to 3.6 and 0.5 to 8.6 mg L^{-1}, respectively, were

found in seawater filled, chlorinated swimming pools (Boudenne et al., 2017; Chowdhury, 2016, 2015; Parinet et al., 2012; Manasfi et al., 2017b), with another study finding more elevated TOC concentrations (up to 12 mg L^{-1}) (Manasfi et al., 2016). One study investigated the organic loading of swimming pools using fluorescence excitation-emission matrix spectroscopy with parallel factor analysis (PARAFAC), reporting that organic matter fluorescence was characterised by five different components, one of which was identified to be unique to swimming pool waters (Seredyńska-Sobecka et al., 2011). The authors suggested the use of monitoring fluorescence at 420 nm as a specific indicator of organic loading in swimming pools, reporting its increase during swimming pool opening hours and seeing a gradual accumulation of organic matter over this period (Seredyńska-Sobecka et al., 2011).

The following subsections present a review on current knowledge of the input of organic and inorganic matter in the swimming pool environment, with a particular focus on the impact on DBP formation.

2.5.1. Filling Waters

One major factor that influences the occurrence of DBPs in swimming pool waters is the filling water, which can introduce a range of species, e.g. natural organic matter (NOM), trace amounts of DBPs chlorine and bromine, species that are dependent on both the quality and prior treatment of the filling water. The majority of swimming pools are filled with disinfected distributed drinking water (freshwater), however, the use of other natural waters, e.g. seawater, may become the norm in the future for some countries, where there is an increasing scarcity of freshwater.

Whilst TOC concentrations are often low in filling waters and not the major input of TOC for pools and spas (Daiber et al., 2016), filling waters may introduce bromide/bromine species, with bromide reported at 65 to 89 and up to 0.5 mg L^{-1}, for sea and fresh water, respectively (WHO, 2009), which is consistent with bromide levels reported in seawater filled swimming pools of 49 to 107 mg L^{-1} (Boudenne et al., 2017; Manasfi et al., 2016; Manasfi et al., 2017b; Parinet et al., 2012). Bromide can also be detected at higher concentrations in freshwaters, with Heeb et al. (2014) detailing a concentration range of ~10 to >1000 µg L^{-1} in their critical review of aqueous reactions of bromine and concentrations up to 8.5 mg L^{-1} being measured in Western Australian groundwaters (Gruchlik et al., 2014).

Although only studied in chlorinated pools, bromide (after quenching the oxidant residual) was reported at significantly lower concentrations than chloride, being below the detection limit in some studies (Cardador and Gallego, 2011; E et al., 2016) and ranging from 0.002 to 1.8 mg L^{-1} in other pools located indoors (Michalski and Mathews, 2007; Xiao

et al., 2012). Similar results were reported for chlorinated swimming pools located outdoors, where bromide was below detection limits in some studies (Cardador and Gallego, 2011; Yeh et al., 2014) and 0.002 to 0.2 mg L^{-1} in others (Manasfi et al., 2016; Xiao et al., 2012).

As discussed in **Section 2.3.2**, the presence of bromide in swimming pools disinfected with chlorine can lead to the formation of HOBr, as previously shown in **Eq (2-viii)**, which is known to occur inpools treated by BCDMH, sodium bromide in combination with a chlorine based oxidant or EGMO. As the reaction of HOBr with organic compounds in pools results in the formation of Br-DBPs which are generally more toxic than their chlorinated counterparts (Plewa et al., 2004), increasing bromide concentrations in swimming pools will increase the formation of Br-DBPs, an undesired consequence. The predominance of the Br-DBPs despite the lower concentrations of bromine than chlorine is likely due to the higher halogenation reactivity of bromine compared to chlorine, i.e., bromine is incorporated into organic matter at a faster rate (Cowman and Singer, 1996). As discussed in **Section 2.4**, Br-THMs were generally more abundant in seawater filled swimming pools compared to those filled with freshwater, for pools where treatment methods and location were comparable (Manasfi et al., 2016), consistent with the higher concentrations of bromide entering in the seawater filled pools. Other Br-DBPs, e.g. HAAs (Parinet et al., 2012), have been found in higher concentrations in pools filled with seawater compared to those filled with freshwater. Whilst this section has focused on the input of bromide originating from filling waters, bromide may also be introduced by bromine based disinfectants, which is discussed further in **Section 2.5.2**. Further discussion on the impact of halide ions on DBP formation is provided in **Section 2.6.2**.

2.5.2. Disinfectants

Many studies have investigated the effect of different disinfectants on DBP formation, however these studies are often performed at conditions more reflective of drinking water and may not be a true representation of the chemistry that would occur in swimming pool waters. For example, although the desired outcome is shared, protection against the microbial disease risk, disinfectants are generally added to drinking waters in individual doses (e.g. at the end of the treatment process or the outlet of the reservoir) whereas due to constant bather load, rapid loss of disinfectant and the inefficiency of manual treatment (Nnaji et al., 2011), disinfectants in swimming pools are often continually added by means of automatic dosing systems, with oxidant residuals often much higher than those found in drinking waters. Whilst all studies have led to a better understanding of the chemistry of disinfectants, this review only discusses studies carried out under conditions applicable to the swimming pool environment. Furthermore, a recent review by Ilyas et al. (2018) discusses

the impact of disinfectants on a limited range of DBPs (THMs, HAAs, HANs, trihaloacetaldehydes and chloramines).

Swimming pool disinfectants are produced on a large industrial scale and hence may not be 100% pure. Specific impurities, such as bromate, chlorite and chlorate, have been reported in feed stocks of sodium hypochlorite in median concentrations of 1022, 2646 and 20 462 mg L^{-1}, respectively, as well as in calcium hypochlorite pellets (median concentrations 240, 695 and 9516 mg kg^{-1}, respectively) (Garcia-Villanova et al., 2010). Similarly, chloride is commonly found as an impurity in sodium bromide (Chlorine Chemistry Council and Canadian Chlorine Coordinating Committee, 2003; PWTAG, 1999). Fillers are often added to solid disinfectants (e.g. BCDMH or hypochlorite pellets) and can potentially remain as a residue in waters and possibly lead to the formation of DBPs. Further studies on the impact of fillers on DBP formation are recommended. Naturally, disinfectants themselves can result in DBPs as they are reduced. For example, ClO_2 as a disinfectant introduces chlorite, chlorate and chloride due to the reaction and hydrolysis products of chlorine dioxide (Gordon et al., 1972). Bromide and chloride are introduced by bromine and chlorine based disinfectants, respectively, and can have an effect on DBP formation as discussed in **Section 2.6.2**. The occurrence of a range of inorganic anions in swimming pools and spas was discussed **Section 2.4.1.10**.

As presented throughout **Section 2.4**, Br-DBPs are generally detected at higher concentrations in pools treated with bromine based disinfectants compared to their chlorinated counterparts, whilst Cl-DBPs dominate in pools disinfected with chlorine. For example, Kelsall and Sim (2001) investigated THMs in three pools treated with chlorine, chlorine in combination with ozone (Cl_2/Ozone) and sodium bromide in combination with ozone (Br_2/Ozone), in order to assess the disinfectant impact on THM formation. Chloroform was the dominant THM in the chlorinated pools (up to 85 μg L^{-1}) but was not detected in the Br_2/ozone treated pool, due to the absence of chlorine, Similarly, bromoform was the dominant THM in the Br_2/ozone treated pool, but was below detection in the chlorinated pools investigated (Kelsall and Sim, 2001). These observations may be explained by the minimal formation of HOCl or HOBr, and therefore minimal formation of the chloro- or bromo-THMs, in the brominated and chlorinated pools, respectively. Daiber et al (2016) compared the DBPs detected in a number of swimming pools and spas employing either bromination or chlorination, reporting the dominance of Cl-DBPs and Br-DBPs in pools treated by chlorination and bromination, respectively. Additionally, on a molar basis, the total DBP concentrations were higher in pools where chlorination (predominantly hypochlorite) was employed, when compared to those where bromination (BCDMH) was employed, although in spas similar total DBP molar concentrations were observed regardless

of disinfectant (Daiber et al., 2016). These observations can be explained by the slower dissolution and formation (and hence availability) of free chlorine equivalents from BCDMH compared to the readily available hypochlorite, where the dissolution rate from BCDMH is increased in the spas due to the higher operating temperature. Lourencetti et al. (2012) also reported the dominance of Br-THMs and Cl-THMs in the ambient air at swimming pool sites treated with bromine and chlorine based disinfectants, respectively, with Richardson et al. (2010) reporting a similar finding. Additionally, Richardson et al. (2010) reported a lower maximum dichloramine concentration (<10 μg L^{-1}) in a pool treated with BCDMH compared to one where chlorination was employed (650 μg L^{-1}).

The use of EGMO was demonstrated to be a suitable replacement to traditional chlorination methods in a study of synthetic and real swimming pool waters, however, while the authors demonstrated the ability to reduce pathogens, no investigation into the impact of DBPs was performed (Naji et al., 2018). Lee et al. (2009) reported both higher TTHM and Br-THM concentrations in pools treated with EGMO compared to pools where chlorination was employed, although this comparison was of mass concentrations. It is important to note that to accurately compare concentrations of groups of compounds, such as TTHMs, Br-THMs or HAA9, it is crucial to use molar concentrations. The use of mass or molar concentrations for comparison has been noted along with each study throughout this review. In a subsequent study, Lee et al. (2010) further investigated the effect of these treatment methods, by studying a wider range of DBPs: THMs, HAAs, HANs and CH. Considering average concentrations by mass, total THMs and total HANs were higher in pools where EGMO was employed compared to chlorinated pools, which is likely due to the higher abundance of brominated THMs and HANs detected in the EGMO treated pools. Although higher concentrations of Br-HAAs were detected in the pools treated with EGMO, Lee et al. (2010) reported a higher HAA9 concentration in pools treated with chlorination compared to those treated by EGMO. Whilst concentrations varied greatly, compared to where chlorination was employed, on average CH was detected at slightly lower concentrations in the EGMO treated pools (Lee et al., 2010). Zhang et al. (2015) reported substantially lower DBP concentrations in pools treated with chlorine dioxide and TCICA compared to pools treated by chlorination, when comparing molar totals of the sum of TTHMs, HAA9, CH, HAN-4, 1,1-DCP, 1,1,1-TCP and TCNM. Pools treated by chlorination in combination with ozone were also investigated by Lee et al. (2009, 2010) and Zhang et al. (2015), and these are discussed in **Section 2.6.1**.

In addition to comparing DBPs detected in real pools, several studies have been conducted on the laboratory scale in order to better understand the impact of disinfectants on DBPs in the swimming pool environment. Pu et al. (2013) investigated the effect of

bromination versus chlorination on DBP formation, by comparing TTHM and total HAN molar concentrations resulting from the oxidation of algae solutions under conditions comparable to pool waters. An increase in total molar HAN concentrations was observed when bromination was employed over that observed during chlorination. TTHM molar concentrations were comparable between chlorination and bromination (Pu et al., 2013). Similarly, Judd and Jeffrey (1995) reported a 74% greater THM formation with the use of bromine (HOBr) as a disinfectant, compared to when chlorine (HOCl) was used under the same conditions, in their investigation carried out under conditions similar to that of real swimming pool waters.

More recently, Yang et al. (2016) investigated a range of disinfectants commonly used in swimming pools (BCDMH, sodium hypochlorite and TCICA), analysing oxidant decay, reaction kinetics and DBP formation in laboratory scale studies of modelled swimming pool waters. Mainly brominated DBPs were formed when BCDMH was employed, whilst mainly Cl-DBPs formed in experiments where sodium hypochlorite and TCICA were used. Oxidant residuals were similar for BCDMH and sodium hypochlorite, although a higher residual was observed in waters treated with TCICA. Although comparison of molar concentrations would be more accurate, on a mass basis, slightly lower total DBP formation was reported for waters treated with TCICA compared to those treated with sodium hypochlorite, whilst the use of BCDMH produced up to twice the concentration of DBPs compared to the other disinfectants (Yang et al., 2016). Consistent findings were reported in real pools by Wang et al. (2014), where, on a molar basis, lower HAA5 concentrations were observed in pools treated by TCICA compared to those treated by chlorination. These observations were explained by (i) the slower release of chlorine, and hence its availability to form DBPs, for TCICA and (ii) the slow dissolution and fast consumption of HOBr for BCDMH (Yang et al., 2016). In a study of DBPs in a model swimming pool at the laboratory scale, the use of chlorine dioxide in conjunction with chlorine was found to produce less THMs compared to when chlorine alone was used, although the authors reported an increase in both HAAs and chlorate concentrations over the duration (4 weeks) of their investigation (Kim et al., 2017). While the decrease of some DBPs (e.g. THMs, HAAs, HANs, HNMs and chlorate) was observed in several of their simplified experiments, Kim et al. (2017) highlighted the complexity of DBP formation in the swimming pool environment and the difficulty of modelling this DBP formation at the laboratory scale.

2.5.3. Bather Load and Human Input

Excluding disinfectants, bather load is the largest chemical input and oxidant consumer in swimming pool waters (Keuten et al., 2012) and can be divided into two main categories: human body excretions and personal care products. Although various studies

exist in a more general sense, the following subsections will only present a review of literature discussing bather load and human input in relation to the swimming pool environment and will focus mainly on the impact on DBP formation.

2.5.3.1. Human Body Excretions

As the name suggests, human body excretions are comprised of any human derived input generally introduced via sweat, urine, saliva, hair or skin cells, and, although differing from person to person, can include urea, ammonia, uric acid, creatinine, creatine, lactic acid, citric acid, hippuric acid, uracil, ornithine, chloride, sulfate, cations such as K^+, Na^+, Ca^{2+}, Mg^{2+} and Zn^{2+}, and amino acids, such as histidine, glycine, cysteine, asparagine, lysine, arginine and guanine (Hirokawa et al., 2007; Montain et al., 2007; Mosher, 1933).

Although not inclusive, bulk parameters such as TN (the total nitrogen content) or TON (the organic nitrogen fraction), which are summarised in **Table A1-14**, can be used as an indication of contamination of human origin, as many human inputs contain nitrogenous compounds. In terms of TN, chlorinated pools located indoors have higher reported concentrations than those located outdoors (<0.1 to 12 and 0.6 to 8.4 mg L^{-1}, respectively) (Chowdhury, 2016; Yeh et al., 2014; Zhang et al., 2015), although Yang et al. (2018) reported TN concentrations up to 35 mg L^{-1} in their study of 35 outdoor swimming pools treated with TCICA. Additionally, TON concentrations followed the same trend, with reported concentrations of 0.2 to 11 and 0.09 to 1.3 mg L^{-1} for indoor and outdoor pools, respectively (Yeh et al., 2014; Zhang et al., 2015). The generally higher concentrations of TN and TON observed in indoor pools (compared to outdoor pools) may be due to (i) indoor pools are often used by a larger number of babies and children (**Section 2.6.4**), and (ii) indoor pools are generally operated at higher temperatures (**Section 2.6.5**), both of which would see an increased release of nitrogen containing anthropogenic chemicals and hence a higher nitrogen content. To the best of our knowledge, only two studies have reported TN in seawater filled swimming pools, where concentrations of 0.7 to 7.7 mg L^{-1} were reported (Parinet et al., 2012; Manasfi et al., 2017b). It should be noted that human inputs differ upon swimmer activity and water temperature, and these aspects are addressed in **Sections 2.6.4** and **2.6.5**, respectively.

The quantity of contamination due to human input has been investigated in various studies. The release of chemicals from swimmers was investigated by Keuten et al. (2014), who found that on average a person released 250, 77, 37 and 10 mg of non-purgeable organic carbon, TN, urea and ammonium, respectively, during a 30 minute swim time. Urea, a component of urine and sweat (Mosher, 1933), has been detected in a range of pools in concentrations up to 17 mg L^{-1} (De Laat et al., 2011; Parrat et al., 2012; Schmalz et al.,

2011a, 2011b; Tachikawa et al., 2005; Weng and Blatchley, 2011; Yang et al., 2018), with a full summary provided in **Table A1-14**. Afifi and Blatchley (2016) also investigated urea in swimming pools, reporting that it was correlated to the number of swimmers. While the use of PARAFAC to monitor bather load has been suggested (Seredyńska-Sobecka et al., 2011), perhaps the most indicative marker of human input is the monitoring of a urinary marker acesulfame-K, as proposed by Jmaiff Blackstock et al. (2017). Acesulfame-k, also referred to as ACE, is a stable artificial sweetener that remains un-metabolised and almost fully excreted in urine, rendering it an ideal urinary marker. Jmaiff Blackstock et al. (2017) provide details regarding a suitable analytical method for the analysis of ACE in swimming pool and spa waters, and this is the only known study to report the occurrence of ACE in these water matrices, where concentrations between 30 to 2110 ng L^{-1} in pools and between 70 to 7110 ng L^{-1} in spas were reported. Nitrate concentrations were found to vary, with no trends observed for swimming pool type (indoor or outdoor) or treatment method. Only two studies have examined nitrate in outdoor chlorinated swimming pools, with concentrations of 13 to 88 mg L^{-1} found (Beech et al., 1980; Zhang et al., 2015). Concentrations varied greatly between pools located indoors, with nitrate levels reported between 2.2 to 129, 4.2 to 208, 1.2 to 26 and 11 to 49 mg L^{-1} for pools treated with sodium hypochlorite, chlorine dioxide, chlorination in combination with ozone and EGMO, respectively (E et al., 2016; Lee et al., 2010; Michalski and Mathews, 2007; Spiliotopoulou et al., 2015; Zhang et al., 2015).

The potential of DBP formation from human body derived precursors has been investigated at the laboratory scale, with studies investigating either individual precursors or body fluid analogue (BFA), a synthetic mixture containing the main components of bodily fluids, under conditions commonly reported in swimming pool waters.

The BFA (or precursor) to chlorine ratio has been shown to be the major factor affecting the amount of DBPs formed (Hansen et al., 2012a, 2013a; Kanan, 2010; Schmalz et al., 2011a), with the precursor source having some effect. Judd and Bullock (2003) compared the formation of THMs and chloramines upon chlorination of BFA alone and BFA with a standard humic acid sample (as a soil analogue) in a model pool, reporting eight times higher concentration of THMs was produced when the humic acid was present. Small increases in humic acid saw little change to chloramine concentrations, however concentrations doubled upon doubling the humic acid concentration (Judd and Bullock, 2003). This study highlights the importance of humic substances on DBP formation in pools, which can be minimised with correct swimmer hygiene. THMs were produced at a lower rate than HAAs upon chlorination of BFA, with HNMs produced at the lowest rate (Kanan and Karanfil, 2011). In the same study, individual BFA components at a concentration of 1 mg L^{-1} carbon were investigated for their potential DBP formation, with almost all components forming varying

concentrations of chloroform, DCAA and TCAA and TCNM, with citric acid leading to the highest formation (based on mass concentration) in almost all cases (Kanan and Karanfil, 2011). Uric acid, citric acid and hippuric acid have been shown to be the components of BFA most responsible for HAA formation upon chlorination (Yang et al., 2016).

Additionally, a range of DBPs including halo(nitro)phenols were detected in pool waters, which were later confirmed to form from the chlorination of human derived precursors, particularly urine (Xiao et al., 2012). Although the formation of HAAs, THMs and HANs were observed upon chlorination of BFA (Hansen et al., 2012a), formation was dependent on pH. This is discussed in more detail in **Section 2.6.5**.

A mixture including hair, saliva, skin, urine and moisturising body lotion, as well as the individual components, was investigated for potential formation of chloroform, bromodichloromethane, CH, DCAN and 1,1,1-TCP upon chlorination in a study by Kim et al. (2002). Chloroform was the most abundant DBP (on a mass concentration basis) in all cases, with DCAN formation higher upon chlorination of components of human origin, which is likely due to the formation of nitrogen containing degradation products which enhance DCAN formation (Kim et al., 2002). Additionally, the chlorination of skin specimens by Xiao et al. (2012) led to the formation of HAAs and THMs, with Br-DBPs increasing with increasing bromide concentrations.

In almost all samples analysed, saliva, urine, gastric juice, blood and faeces were found to contain several secondary amine precursors, dimethylamine, pyrrolidine and piperidine (Tricker et al., 1992), which may lead to *N*-nitrosamine formation. Additionally, Carter et al. (2015) demonstrated the formation of NDMA from chloramination of synthetic urine, which was likely due to several mechanism pathways involving dimethylamine and nitrate (Masuda et al., 2000; Mitch and Sedlak, 2001). In another study, urea, ammonium ions, amino acids and creatinine were identified as the main precursors to trichloramine formation, with urea responsible for 76% of the total trichloramine formation observed (Schmalz et al., 2011a). The degradation rate of urea was reported to be 1% per hour at chlorine concentrations equivalent to those found in swimming pools, and its likely degradation products were suggested to be chlorinated urea and trichloramine (De Laat et al., 2011). At a pH value similar to that expected in pools, formation of trichloramine was reported to be favoured over the monosubstituted or disubstituted analogues (Schmalz et al., 2011a).

UV treatment and chlorination of three amino acids, L-arginine, L-histidine, and L-glycine, led to the formation of chloramines and cyanogen chloride (Weng and Blatchley, 2013). The formation of chloramines was suggested to be due to rapid *N*-chlorination, with

UV irradiation and hydrolysis then promoting cleavage and subsequent formation of ammonia, which formed chloramines upon further chlorination. The formation of CNCl was proposed to occur through a similar pathway of *N*-chlorination followed by UV promoted hydrolysis, where reactions and by-products were found to be dependent on both the chlorine to precursor ratio (Cl/P) and UV dose (Weng and Blatchley, 2013),. Lian et al. (2014) reported the formation of CNCl from the chlorination of uric acid, with reactions found to be not only dependant on the Cl/P ratio, but also on pH and temperature. Additionally, at Cl/P ratios greater than 1 (i.e. conditions reflective of real swimming pools), the formation of other intermediates and their subsequent DBPs (due to ring cleavage and subsequent chlorination) were observed, which was likely due to the lower stability of these products promoting decarboxylation or hydrolysis reactions (Lian et al., 2014). CNCl was also the major product observed upon chlorination of uric acid in a study by Li and Blatchley (2007). For all these studies of CNCl formation (Li and Blatchley, 2007; Lian et al., 2014; Weng and Blatchley, 2013), CNCl concentrations were found to decrease at higher chlorine doses. Li and Blatchley (2007) reported the formation of cyanogen chloride upon chlorination of L-histidine, also observing the formation of other DBPs. Creatinine, urea, L-histidine and L-arginine all produced trichloramine upon chlorination, with DCAN and dichloromethylamine observed in some cases (Weng and Blatchley, 2013). Complex mechanisms were proposed for all compounds, and hypothesised to involve several chlorine substitution, hydrolysis, and/or decarboxylation reactions, with several intermediate species (Li and Blatchley, 2007). Similarly, the chlorination of creatinine was demonstrated to involve several multistep and multipathway mechanisms in which a range of intermediate and secondary by-products (e.g. chlorocreatinine, trichloromethane, 1,1,1-TCP, DCAN and TCNM) were identified (Zhang et al., 2018). In a study of the reaction mechanism of the chlorination of urea in a swimming pool context, molecular chlorine, Cl_2, was found to be the chlorine species involved in the rate-determining first step of *N*-chlorination of urea, with HOCl being the chlorine species involved in the subsequent steps to ultimately form trichloramine and nitrate (Blatchley and Cheng, 2010).

Chlorination of six nitrogen containing precursors, glycine, asparagine, uracil, cytosine, guanine and cysteine, all led to the formation of cyanogen chloride, with its concentration again found to be highly dependent on the chlorine to precursor ratio (Shang et al., 2000). Although an overall mechanism was not provided, the tentative identification of several other DBPs (DCAN, chloroform, acetone, *N*,*N*-dichloroaminoacetonitrile and *N*-chloroformamide) in this study suggested several mechanistic pathways and therefore intermediate species are likely (Shang et al., 2000). Wlodyka-Bergier and Bergier (2016) investigated urea, creatinine, glycine, histidine and arginine for their potential to form a

series of DBPs (chloroform, CAA, DCA, TCAA, TCAN, 1,1-DCP, 1,1-TCP, CH and TCNM) upon chlorination and chlorination in combination with UV treatment. Although all investigated precursors showed a potential to form all investigated DBPs, chloroform formation was highest from creatinine and glycine, HK formation was highest for creatinine and histidine, CH, HAAs and HANs showed highest formation from histidine, whilst all precursors showed similar formation potentials for TCNM. The impact of UV treatment had a significant effect on the DBP formation potential of the different precursors. For all precursors, HAAs and TCNM concentrations increased when UV treatment was applied. Excluding glycine, CH formation increased for all precursors, whilst only creatinine showed a decreased formation potential for HANs, when UV was applied. For HKs, an increased formation was observed for urea and arginine, with other precursors demonstrating a decreased formation potential when UV was applied. Although a large increase in chloroform formation was observed from urea and histidine, the effect of UV treatment was somewhat ambiguous for other precursors investigated (Wlodyka-Bergier and Bergier, 2016).

2.5.3.2. Pharmaceuticals and Personal Care Products

Although recent reviews by Bottoni et al. (2014), Sharifan et al. (2016) and Haman et al. (2015) discuss some potential issues of pharmaceuticals and personal care products (PPCPs), or more specifically their components such as metal ions, metal oxides or nanoparticles (Sharma et al., 2017), in aquatic environments, this review will present studies of PPCPs applicable to swimming pools (especially UV filters, antifungal agents and parabens that are commonly added to sunscreens and other cosmetic products), with a particular focus on the potential for DBP formation. Although known by various chemical and trade names, for the purpose of this review, some commonly reported PPCPs will be abbreviated as per **Table 2-3**, with full lists of names provided in **Table A1-15**.

A recent, and arguably the most comprehensive, study of pharmaceuticals in pools is that by Fantuzzi et al. (2018), who investigated the occurrence of over forty eight pharmaceuticals (including antibiotics, analgesics, diuretics, estrogens, lipid regulators, and a range of anti-cancer, anti-inflammatory, antihypertensive, bronchodilator, cardiovascular, central nervous system, gastrointestinal and hypoglycemic medications), as well as several illicit drugs (including cocaine, amphetamine like substances, new psychoactive substances, opioids and cannabinoids) and/or their respective metabolites, in ten indoor swimming pools treated by chlorination. While the investigated opioids, amphetamines and cannabis derivatives were never detected, cocaine and its metabolites were measured in 90% of pool water samples, at concentrations between 0.1 and 49 ng L^{-1}. Ibuprofen was the most commonly detected pharmaceutical compound, measured in all pools between 16 to 197

ng L^{-1}, with ketoprofen, valsartan, carbamazepine and its derivative, 10,1-dihydro-10-hydroxycarbamazepine, detected in at least 80% of pool samples, where concentrations of up to 127 ng L^{-1} were reported. Lower concentrations (0.03 to 2.6 ng L^{-1}) of several other pharmaceuticals were detected in at least one of the investigated pools, namely atenolol, enalapril, paracetamol, hydrochlorothiazide, irbesartan and dehydroerythromycin.

Table 2-3: Commonly used names and abbreviations (Abbr.) for selected components commonly used in personal care products.

Antifungal Agents		**Parabens**			
Common Name	**Abbr.**	**Common Name**	**Abbr.**	**Common Name**	**Abbr.**
dichlorophene	dichlorophen	methylparaben	MeP	isobutylparaben	iBuP
5-chloro-(2,4-dichlorophenoxy)phenol	2,4-DCPh	ethylparaben	EtP	pentylparaben	PeP
2,4,6-trichlorophenol	2,4,6-TCPh	propylparaben	PrP	heptaparaben	HeP
Butylated hydroxytoluene	BHT	isopropylparaben	iPrP	octylparaben	OcP
Triclocarban	TCC	butylparaben	BuP	benzylparaben	BzP
UV Filters					
Common Name	**Abbr.**	**Common Name**			**Abbr.**
isoamyl 4-methoxycinnamate	Amiloxate	4-hydroxybenzophenone			4-HB
avobenzone	Avobenzone	3,3,5-trimethylcyclohexyl-2-hydroxybenzoate			Homosalate
2,4-dihydroxybenzophenone	BP-1	4-methylbenzylidene camphor			4-MBC
2,2′,4,4′-tetrahydroxybenzophenone	BP-2	octocrylene			OCR
benzophenone-3	BP-3	octyldimethyl-para-aminobenzoic acid			OD-PABA
2,2′-dihydroxy-4-methoxybenzophenone	BP-8	octylmethoxycinnamate			OMC
benzyl salicylate	BzS	2-phenyl-3H-benzimidazole-5-sulfonic acid			PBS
4, 4′-dihydroxybenzophenone	4-DHB	*p*-hydroxybenzoic acid			PHBA
5,6-dimethyl-1H-benzotriazole monohydrate	DMeBT	phenyl salicylate			PS
ethyl 4-aminobenzoate	Et-PABA	2,3,4–trihydroxybenzophenone			THB
1H-benzotriazole	1-HBT				
Other					
5-methyl-1H-benzotriazole	5-MeBT	5-Cl-1H-benzotriazole			5CBT

Lu et al. (2017) investigated twenty two target PPCPs, including parabens, UV filters, anticorrosion agents and antimicrobials, in thirty five outdoor swimming pools over five locations throughout China. Five of the target compounds (MeP, EtP, PrP, 2,4-DCPh and 1-HBT) were detected in all pool water samples (<0.1 to 6.0 µg L^{-1}), while eleven others (PHBA, i-PrP, Butyl-PBS (the sum of BuP and i-BuP), 4-HB, BP-1, BP-3, TCC, 5-MeBT, 5-CBT and DMeBT) were measured in at least one of the investigated pools at concentrations up to 0.4 µg L^{-1} (Lu et al., 2017). Thirty pharmaceuticals were investigated in seawater filled and freshwater pools by Teo et al. (2016a), with only caffeine (16 to 1540

ng L^{-1}) and ibuprofen (16 to 83 ng L^{-1}) detected in twelve and eight of the fifteen freshwater pools investigated, respectively. All thirty pharmaceuticals investigated were below detection limits in the seawater filled pools (Teo et al., 2016a). Of thirty-two PPCPs, *N*,*N*-diethyl-*m*-toluamide, caffeine and tri(2-chloroethyl)phosphate were the only detectable PPCPs in swimming pool waters investigated by Weng et al. (2014), who also showed the potential of PPCPs to form chlorinated by-products. Similarly, the occurrence of thirty-two pharmaceuticals and fourteen UV filter compounds were investigated over a range of swimming pools, with over 88% of the pools containing pharmaceuticals and over 94% containing UV filters (Ekowati et al., 2016). Only ten pharmaceuticals (atenolol, carbamazepine, hydrochlorothiazide, metronidazole, ofloxacin, sulfamethoxazole, acetaminophen, ibuprofen, ketoprofen and phenazone) and eleven UV filters (BP-1, BP-2, BP-3, BP-8, THB, 4-DHB, 4-MBC, OD-PABA, 1-HBT, 5-MeBT and DMeBT) were detected, with maximum concentrations of 904 and 69 ng L^{-1}, respectively. Generally, spas had higher concentrations than pools and, whilst pharmaceuticals were lower in pools treated with sodium hypochlorite, UV filters were lower in pools with EGMO/UV treatment (Ekowati et al., 2016). A recent study also reported the presence of caffeine, carbamazepine and BP-3 in five Polish swimming pools, where concentrations of up to 13.6, 176 and 13.6 ng L^{-1} were reported, respectively (Lempart et al., 2018a).

A range of UV filters (BP-3, OMC, PBS, 4-MBC and OCR) were present in up to ten times higher concentrations in a pool used exclusively by babies, compared to concentrations in a pool used by adults, with maximum concentrations of 40 µg L^{-1} reported (Zwiener et al., 2006). Two UV filters (BP-3 and BP-8) and an antioxidant (BHT) were investigated in fifteen swimming pools, including sports pools, jacuzzis, waterslides and leisure pools, where concentrations of 19 to 1179, 50 to 227 and 3.8 to 5.5 ng L^{-1} were reported, respectively (Lempart et al., 2018b). Cuderman and Heath (2007) investigated a range of UV filters (4-MBC, OCR, OMC, BP-3, homosalate and avobenzone) and two antifungal agents (2,4-DCPh and dichlorophen) in two individual swimming pools. 4-MBC (330 ng L^{-1}), OCR (17 ng L^{-1}) and OMC (15 ng L^{-1}) were detected in one pool and BP-3 (103 and 400 ng L^{-1}) was detected in both pools. Homosalate, avobenzone, 2,4-DCPh and dichlorophen, were not detected in any of the investigated swimming pools (Cuderman and Heath, 2007). Similarly, avobenzone was not detected in a swimming pool investigated by Giokas et al. (2004), however BP-3 (5.7 ng L^{-1}), 4-MBC (5.4 ng L^{-1}) and OMC (3.0 ng L^{-1}) were all detected. Higher concentrations (2400 to 3300 ng L^{-1}) of BP-3 were reported in a swimming pool in an earlier study by Lambropoulou et al. (2002), who also reported finding OP-PABA in concentrations of below detection (<600) to 2100 ng L^{-1}. Vidal et al. (2010) compared the concentrations of six UV filters (BP-3, amiloxate, 4-MBC, OCR, OD-PABA and OMC) in

private and public pools. Amiloxate was detected in the public pool (700 ng L^{-1}) and, although below the limit of quantification (60 ng L^{-1}), 4-MBC was also detected. All other UV filters were below their respective detection limits (60 to 3000 ng L^{-1}) in the public pools, with no UV filters detected in the private pool (Vidal et al., 2010).

Parabens are used as preservatives in some PPCPs (such as sunscreen) and have been investigated in both pool waters and at the laboratory scale. Whilst none of the investigated parabens (BuP and BzP) were detected in the actual pool water samples, the addition of sunscreen (200 µL) to pool water resulted in both parabens being detected: 29 µg L^{-1} of BuP and 43 µg L^{-1} of BzP (López-Darias et al., 2010). Additionally, whilst the pool water was found to have no detectable levels of several endocrine disruptor chemicals which are suspected to negatively affect reproductive function, increase risks of some cancers and result in abnormal growth and neurodevelopment in children (UNEP and WHO, 2013), namely six polycyclic aromatic hydrocarbons (naphthalene, acenaphthene, phenanthrene, anthracene, 9-methylanthracene and fluoranthene) and six alkylphenols (4-*tert*-butyl-, 4-*tert*-octylphenol, 4-octylphenol, 4-cumylphenol, 4-n-nonylphenol and bisphenol A), an increase in 4-n-nonylphenol (16 µg L^{-1}) was detected after the addition of sunscreen to the swimming pool water (López-Darias et al., 2010). This study provides evidence that sunscreens are a source of PPCPs in swimming pool waters.

The occurrence of MeP, EtP, PrP, BuP, 2,4,6-TCPh and 2,4-DCPh in swimming pool waters was investigated in two individual studies by Regueiro et al. (2009a, 2009b). In one pool water sample, PrP (32 ng L^{-1}) and BuP (78 ng L^{-1}) were quantified, MeP, EtP and 2,4,6-TCPh were detected, and 2,4-DCPh was below the limit of detection (<21 ng L^{-1}) (Regueiro et al., 2009a). In their later investigation of swimming pool waters, BuP (14 ng L^{-1}) was quantified, MeP, EtP, PrP and 2,4-DCPh were detected, and 2,4-DCPh was again below the detection limit (Regueiro et al., 2009b). PrP (900 ng L^{-1}) was the only paraben detected in a swimming pool investigated by Almeida and Nogueira (2014), where MeP, EtP and BuP were below the detection limits (<100 ng L^{-1}).

A few studies have reported the formation of halogenated by-products from parabens in swimming pool waters. Terasaki and Makino (2008) investigated seven parabens (MeP, EtP, PrP, iPrP, BuP, iBuP and BzP) and their monochlorinated and dichlorinated by-products in two indoor and four outdoor chlorinated swimming pools. Only one indoor and one outdoor pool showed detectable levels of the investigated parabens or their chlorinated by-products. iPrP-Cl2 and BzP were quantified (25 and 28 ng L^{-1}, respectively), with MeP-Cl2 and BzP-Cl1 detected for the indoor pool, whilst iPrP-Cl2, BzP and BzP-Cl1 were detected in the outdoor pool. All other compounds were below their respective limits of

detection (5 to 15 ng L^{-1}) (Terasaki and Makino, 2008). Li et al. (2015b) investigated a range of parabens (MeP, EtP, PrP, BuP, PeP, HeP, OcP and BzP), some chlorinated by-products (MeP-Cl1, MeP-Cl2, EtP-Cl1 and EtP-Cl2) and their main hydrolysis product, *p*-hydroxybenzoic acid (PHBA) in a range of pools treated by either chlorination or chlorination in combination with ozone. Of the detected parabens, MeP and PrP dominated and accounted for over 91% of the total paraben concentrations on a molar basis. Considering the summed concentrations of the investigated parabens and their chlorinated derivatives, indoor pools had an approximately twenty times higher average concentration than pools located outdoors (144 and 6.8 ng L^{-1}, respectively), which the authors suggest is likely due to (i) the lower paraben loading of outdoor pools as outdoor pools often have shorter opening times and (ii) the increased degradation of parabens in outdoor pools via UV due to the prolonged exposure to sunlight. Additionally, paraben concentrations were reported to be higher on weekends compared to weekdays, which is likely due to the higher bather loads during weekends (Li et al., 2015b). Consistent with the authors' suggestions, parabens have been shown to degrade in the presence of ozone and UV treatment (Cuerda-Correa et al., 2016), which may explain the observations of Li et al. (2015b) as both ozone (via treatment) and UV (via sunlight) were present in some pools. Further investigation into the degradation and transformation products of parabens, particularly under conditions applicable to swimming pool waters, is therefore warranted.

Although only limited studies exist, a range of other DBPs likely introduced via PPCPs have been investigated in pools. Swimming pool water is suggested to increase the leaching of nanoparticles (TiO_2 and ZnO) during swimming (Virkutyte et al., 2012), which have the potential to accumulate in swimming pools (Jeon et al., 2016). A range of aliphatic and aromatic aldehydes (glyoxal, methylglyoxal, 2,5-dihydroxybenzaldehyde, butyraldehyde, propionaldehyde, acetaldehyde, 3-hydroxybenzaldehyde, benzaldehyde, formaldehyde, valeraldehyde, 3-methylbenzaldehyde, 2-ethylbenzaldehyde and 2,5-dimethylbenzaldehyde) have been detected in both indoor and outdoor swimming pools in concentrations up to 12 µg L^{-1} (Fernandez-Molina and Silva, 2013; Serrano et al., 2013). Halobenzoquinones (2,6-dichloro benzoquinone, 2,3,6-trichloro benzoquinone, 2,3-dibromo benzoquinone, 5,6-dimethyl benzoquinone and 2,6-dibromo-1,4-benzoquinone) have also been detected in swimming pool waters, at concentrations up to 299, 11, 0.7 and 3.9 ng L^{-1}, respectively (Wang et al., 2013). Only 3-chloroanaline, 4-chloroanaline and 2,4,5-trichloroaniline were detected in swimming pool waters (160, 200 and 40 ng L^{-1}, respectively) in an investigation of twenty seven amines, being aliphatic amines, anilines and *N*-nitrosamines (Jurado-Sánchez et al., 2009). Daiber et al. (2016) detected several halogenated DBPs previously not reported in swimming pool waters, including 4,5-dibromo-

imidazole, 1-methyl-1H-imidazole and 2,4,5-tribromo-1-methylimidazole, which likely result from the use of BCDMH as a disinfectant in these waters. Although found to be unlikely to pose a health risk, organophosphate flame retardants (tributylphosphate, tris(2-chloroethyl)phosphate, tris(1-chloro-2-propyl)phosphate, tris(1.3-dichloro-2-propyl)phosphate and triphenylphosphate) were detected in a range of indoor and outdoor pools (treated by chlorination or UV in combination with chlorine) at concentrations between 5 and 1180 ng L^{-1} (Teo et al., 2016b). The investigated organophosphate flame retardants were generally measured at higher concentrations in the indoor swimming pools compared to the concentrations measured in the outdoor pools, and were found to leach from swimsuits in laboratory studies (Teo et al., 2016b).

Some studies have investigated the possible DBP formation from the aforementioned PPCPs, by carrying out laboratory studies under swimming pool conditions. Various PCPs, as well as pharmaceuticals, were subject to chlorination in a series of laboratory-scale studies, in which chloroform was produced in all cases (Rose, 2014). Pharmaceuticals containing amine groups were the centre of a study by Shen and Andrews (2011) who reported all pharmaceuticals investigated produced NDMA upon chloramination. Based on molar concentrations, ranitidine led to the highest NDMA formation, with NDEA detected in some cases (Shen and Andrews, 2011). Two salicylates commonly found in several personal care products, benzyl salicylate and phenyl salicylate, were found to produce monochloro and dichloro substituted by-products upon their chlorination (de Oliveira e Sá et al., 2014).

Twenty-five possible by-products of the most commonly used UV filter, avobenzone, were identified by Trebše et al. (2016), upon treatment with UV and chlorination under conditions similar to that of swimming pools. Additionally, avobenzone was shown to only partially degrade upon UV/chlorination treatment, and may persist in swimming pool waters, potentially leading to a high formation of by-products over time (Trebše et al., 2016). Similarly, a range of chlorinated by-product intermediates were detected by Nakajima et al. (2009), who treated two UV filters commonly found in sunscreens (OD-PABA and OMC) with sodium hypochlorite at a pH reflective of swimming pools. The extent of the reactions was shown to be dependent on a range of parameters including pH and chlorine dose. The toxicities of these by-products were evaluated and found to pose no significant health risk (Nakajima et al., 2009). A similar investigation by Zhang et al. (2016) reported the formation of a range of intermediate by-products (trichloromethoxyphenol and monochlorinated and dichlorinated oxybenzone), as well as final (trichloromethane, DCAA, TCAA and CH) by-products, upon the chlorination of BP-3 under conditions common to swimming pools. In addition to demonstrating the degradation of several UV filters (BP-3, BP-4 and PBS) via advanced oxidation processes, Celeiro et al. (2018) identified the formation of several DBPs,

i.e., 2,4-di-*tert*-butylphenol, 2,2'-dihydroxy-4-methoxybenzophenone, BP-1 and (2-chlorophenyl)-(2-hydroxy-6-methoxyphenyl)-methanone. Manasfi et al. (2015) investigated the degradation of BP-3 under conditions comparable to seawater filled swimming pools treated by chlorination. The proposed degradation mechanism included ten different by-products, with final products of bromoform and tribromoacetaldehyde, which were found to increase with increasing chlorine dose and temperature (Manasfi et al., 2015). In further work, Manasfi et al. (2017a) proposed degradation of four UV filters (BP-3, BP-8, OMC and avobenzone) under conditions like those employed in seawater filled swimming pools, also demonstrating the stability of OCR under these conditions. While several brominated intermediate degradation products were identified, tribromoacetaldehyde (bromal) and tribromomethane were the key final degredation products identified (Manasfi et al., 2017a).

Although the aforementioned studies have provided some insight to the possible transformation by-products of both PPCPs and human body excretions, much is still unknown. Controlled laboratory and real pool investigations of human body excretions and PPCPs are required in order to fully understand their impact on DBP formation in the swimming pool environment. Human body excretions have been shown to be a major source of DBP formation in swimming pools, with TON reported to be the main precursor of N-DBPs (Shah and Mitch, 2011), and, as such, human body excretions in pools should be minimised.

2.6. Disinfection By-Products: Other Factors to Consider

2.6.1. Secondary Treatment

Treatments such as ozone and UV are also employed to treat swimming pool waters, being used in addition to chlorination and bromination. Although many studies have evaluated the use of UV or ozone on DBP formation, this review will focus only on those studies carried out under conditions similar to those used to treat swimming pool waters. It is noted that Ilyas et al. (2018) have recently reviewed the effects of secondary treatment processes, but on a more limited set of DBPs (THMs, HAAs, HANs, trihaloacetaldehydes and chloramines).

UV, like many other treatment methods, has advantages and disadvantages. The addition of chlorine prior to UV treatment is undesired, as although some contaminants are decreased by UV, so is the disinfectant residual (Rand and Gagnon, 2008). Due to this, a chlorination step post UV treatment is commonly adopted in the treatment of swimming pool waters. UV treatment is known to degrade chloramines (Cimetiere and De Laat, 2014; Soltermann et al., 2014), however many factors, particularly turn-over rate, affect the

efficiency of this degradation. In addition, post-chlorination is reported to increase trichloramine stability (Soltermann et al., 2014). In a study of *N*-nitrosamine formation and degradation during UV treatment of pool water, UV treatment of monochloramine and chlorinated dimethylamine was found to lead to a substantial increase in NDMA formation, proposed to occur through reaction of nitric oxide or peroxynitrite with the dimethylaminyl radical, species produced by UV photolysis of monochloramine and chlorinated dimethylamine, respectively (Soltermann et al., 2013). Despite the problematic NDMA being generally efficiently degraded by UV treatment, in the swimming pool environment where high levels of nitrogen containing NDMA precursors exist, the rate of formation of NDMA outweighed that of its degradation, resulting in a net increase in NDMA concentration (Soltermann et al., 2013). Removal of *N*-nitrosamines from swimming pool waters requires UV doses over thirty times those currently employed at swimming pool sites (Soltermann et al., 2013) . Soltermann et al. (2013) concluded that UV treatment would only be useful for reduction in *N*-nitrosamine concentrations if the pool water contained high *N*-nitrosamine concentrations compared to the concentrations of chloramines and chlorinated secondary amines.

The effect of UV treatment on DBPs has been reported, where many studies focused on volatile DBPs. In a laboratory based study by Hansen et al. (2013b), solutions containing the following DBPs, trichloromethane, bromodichloromethane, dibromochloromethane and tribromomethane, TCAN, DBAN, BCAN, DCAN, CH, 1,1,1-TCP, 1,1-DCP and TCNM, were exposed to medium pressure UV treatment with DBP concentrations measured over time. Generally, Br-DBPs were degraded faster than their chlorinated counterparts, although the order of degradation (listed from fastest to slowest) was found to be TCNM, tribromomethane, dibromochloromethane, DBAN, TCAN, BCAN, CH, bromodichloromethane, DCAN, 1,1,1-TCP, trichloromethane and 1,1-DCP (Hansen et al., 2013b). In experiments where chlorine was added prior to UV treatment, no increase in DBP degradation was observed, indicating that DBPs are not degraded by chlorine radicals (Hansen et al., 2013b). Whist this study showed the degradation rates of the investigated DBPs due to UV, it does not truly represent conditions of real swimming pools where chlorination generally occurs post-UV treatment.

A later study by Spiliotopoulou et al. (2015) expanded the work by Hansen et al. (2013b) by evaluating changes in DBP concentrations in samples of swimming pool waters treated by UV and UV-post chlorination, although TCNM, BCAN, DBAN and TCAN were excluded in this study. DCAN, 1,1,1-TCP and 1,1-DCP were all found to increase in waters treated with UV-post chlorination, although with extended UV exposure, concentrations decreased, as observed by Hansen et al. (2013b). Similar results were reported for THMs,

although with longer UV exposure, more Br-THMs and less Cl-THMs were detected. These observations suggest that (i) DBPs are not formed in the UV reactor but in the subsequent chlorination stage and (ii) that bromide released from the photodecay of Br-DBPs within the UV reactor leads to the formation of HOBr upon addition of chlorine, which subsequently induces Br-DBP formation (Spiliotopoulou et al., 2015).

Cimetiere and De Laat (2014) also investigated the effect of UV-post chlorination on a range of DBPs (HAAs, THMs, DCAN, 1,1,1-TCP, TCNM and TOX) by exposing swimming pool water samples to medium pressure UV, followed by chlorination. Results suggest that UV-post chlorination had little effect on HAAs, slightly increased the concentrations of TOX and CH, but significantly increased the concentrations of THMs (particularly bromodichloromethane and dibromochloromethane), DCAN, 1,1,1-TCP and TCNM (Cimetiere and De Laat, 2014). Whilst tribromomethane was found to decrease in this study, the increase of other Br-THMs is consistent with the model proposed by Spiliotopoulou et al. (2015).

To the best of our knowledge, only one study has investigated the effect of UV treatment on DBPs in seawater filled swimming pools. Cheema et al. (2017b) investigated the concentration changes for HAAs, THMs and HANs in real seawater filled swimming pool water samples when exposed to medium pressure UV followed by chlorination. While a decrease in concentrations of brominated HAAs (e.g. TBAA and DBAA) was observed, concentrations of brominated THMs (tribromomethane and dibromochloromethane) and HANs (e.g. BCAN and DBAN) were found to increase. This study suggests that UV treatment applied to seawater filled swimming pools may increase the occurrence of some brominated DBPs (Cheema et al., 2017b).

Perhaps the best representation of UV treatment in pools is that by Afifi and Blatchley (2016), who compared concentrations of DBPs (cyanogen chloride, cyanogen bromide, DCAN, dichloromethylamine, monochloramine, dichloramine and trichloramine, and trichloromethane, tribromomethane and dibromochloromethane) in a single chlorinated swimming pool at times where (i) no UV was employed, (ii) medium pressure UV-post chlorination was employed and (iii) low pressure UV-post chlorination was employed. Whilst some differences were observed between the two UV treatments, regardless of the UV type and in comparison to where only chlorination was present, trichloromethane, tribromomethane, cyanogen bromide, dichloromethylamine and the inorganic chloramines were all detected at lower concentrations, whilst increases in DCAN and dibromochloromethane were observed. No change was observed for cyanogen chloride. Although some finding are supported by the aforementioned studies (Cimetiere and De Laat,

2014; Hansen et al., 2013b; Spiliotopoulou et al., 2015), this is the first in-depth investigation of the impact of UV-post chlorination treatment in a real swimming pool. Although here we will summarise findings regarding UV treatment, another investigation by Tardif et al. (2016b) investigated the effects of different treatment procedures (UV, air stripping and extraction (carried out in the balance tank) and coagulation) on DBP occurrence by systematically changing these processes and monitoring DBPs (namely TTHMs, HAAs, 1,1,1-TCP, TCNM, NDMA and HANs in water and THMs and chloramines in air) in a real operating swimming pool. The use of UV treatment saw an increase in TCNM, 1,1,1-TCP and TTHMs in the pool water but saw a decrease of NDMA in pool water and chloramines in pool air (Tardif et al., 2016b), observations which are generally similar to observations reported by Zare Afifi and Blatchley (2016), the only other investigation to be carried out on this scale. These differing outcomes highlight the uncertainty of the effects of UV treatment on DBPs in the swimming pool environment, warranting further investigation into DBP chemistry after UV-post chlorination treatment. Future work should follow the work by Afifi and Blatchley (2016) and assess this chemistry on a larger scale, i.e. continual precursor input and treatment, which is more reflective of swimming pool waters. Whilst the studies presented here focused generally on swimming pools, the effect of UV treatment on suspected DBP precursors introduced via bather load was discussed in **Section 2.5.3**.

Like UV treatment, ozonation is often employed prior to chlorination, as a secondary treatment in swimming pools. As discussed in **Section 2.4**, swimming pools treated with ozone generally contained lower concentrations of the investigated DBPs than pools where ozone was not employed. For example, TTHM concentrations were lowest in swimming pools where ozone/chlorination was used, compared to those treated exclusively by chlorination, which the authors attribute to the oxidation of long chained organic molecules by hydroxide ions (introduced by use of sodium hypochlorite) at the relatively high pH (up to 8.5) found in these pools, leading to a higher formation of THMs compared to that observed in the ozone/chlorine pools where ozone is the more dominant oxidant and hence oxidation by hydroxide ions would be less prevalent (Lee et al., 2009). Similar results were reported by Kelsall and Sim (2001), where lower TTHMs (13 to 24 μg L^{-1}) were detected in swimming pools treated by chlorination in combination with ozone than pools treated only by chlorination (21 to 87 μg L^{-1}).

In a laboratory scale study, Hansen et al. (2016) investigated the effect of ozone treatment on the formation of a range of DBPs (trichloromethane, bromodichloromethane, dibromochloromethane and tribromomethane, DCAN, BCAN, DBAN, TCAN, 1,1,1-TCP, 1,1-DCP and TCNM) in tap water, swimming pool water and swimming pool water where BFA was added. Initial ozone dose was found to react directly with the added BFA

pollutants, reducing their reactivity with chlorine and hence a lower THM formation was observed. However, upon subsequent ozone treatments, an increase in THMs was observed, which the authors explained by the increased half-life of ozone (as no functional groups remained for reaction), in which ozone decomposed to radicals which reacted with organic precursors and made them more susceptible to reaction with chlorine. Other DBPs, DCAN, 1,1,1-TCP, TCNM, were also found to increase with subsequent treatments (Hansen et al., 2016). This study suggested that, although ozone treatment has the potential to reduce DBPs in swimming pools, it must be carefully employed, as DBP formation can also be enhanced under periods of low precursor input (e.g. overnight).

The use of an ozone-bromine treatment, the formation of HOBr by oxidation of bromide by ozone, for pools has been suggested by Hoffmann et al. (2015), who successfully applied this treatment method to a hydrotherapy pool for three years, in which microbiological parameters were found to meet guideline values. In waters rich in bromide, ozone has the potential to form bromate, but bromate formation was controlled by pH in the pool (Hoffmann, 2015). Although this study demonstrated the potential use of an ozone-bromine treatment in pools, DBP formation was not closely examined and future work should assess the impact of ozone-bromine treatment on the formation of DBPs, particularly Br-DBPs.

Cheema et al. (2017a) investigated the effect of a combined treatment method of UV followed by ozonation and chlorination on a range of DBPs (trichloromethane, bromodichloromethane and dibromochloromethane, DCAN, BCAN 1,1-DCP, 1,1,1-TCP and TCNM) by exposing real swimming pool waters to one-off and repeated treatments. With the exception of TCNM, all DBPs were found in lower concentrations after an initial treatment than in the pre-treated water. Although an increase in TCNM was observed upon the initial combined treatment, an overall decrease was observed after subsequent repeated treatments (Cheema et al., 2017a). In a later study, Cheema et al. (2018) reported similar results in their expanded investigation of real pool waters repeatedly treated with UV followed by chlorine, ozone followed by chlorine, or the combined treatment of UV followed by ozone and chlorine. A gradual increase in the concentrations of all investigated DBPs (trichloromethane, bromodichloromethane, DCAN, BCAN, TCNM, 1,1-DCP and 1,1,1-TCP) were observed for pool waters exposed to repeated UV/chlorine treatments, with several DBPs (trichloromethane, DCAN, TCNM and 1,1-DCP) also observed to increase in pool waters repeatedly treated with ozone/chlorine. Interestingly, a decrease in concentrations for most DBPs was observed in pool waters exposed to repeat combined UV/ozone/chlorine treatments, which the authors suggest is likely due to the removal of DBPs formed during post ozone chlorination by UV photolysis during the following

treatment cycle (Cheema et al., 2018). As swimming pools are continually treated, these studies demonstrate that a combined UV, ozonation and chlorination treatment method may help reduce DBPs in swimming pool waters, however further studies should assess the impact of continual precursor input on this combined treatment method, which would be more reflective of real swimming pools.

In addition, pools where UV treatment was combined with chlorination are reported to be less toxic, with up to 3x less cytotoxicity observed (Liviac et al., 2010b; Plewa et al., 2011). Whilst secondary treatments have been shown to increase the overall quality of swimming pool water, further studies are required to fully understand the chemistry underpinning secondary treatment methods under conditions more reflective of swimming pools, e.g. continual chlorine residual and continual precursor input. Further studies should investigate a wider range of DBPs under these conditions, in both laboratory and swimming pool studies.

2.6.2. Halide Anions: Bromide and Chloride

One major impact of the disinfectant is the introduction of halide ions, which in turn can affect the formation of DBPs. As previously discussed in **Sections 2.3.1** and **2.6.2**, after oxidation reactions in the pool, chlorine based disinfectants introduce chloride (Cl^-), whilst bromine based disinfectants introduce bromide (Br^-), and these ions can often accumulate due to the continual recirculation in pools. Highlighting the impact of the disinfectant on the ionic composition of pool waters, chloride has been reported at concentrations up to 3233 mg L^{-1} for freshwater chlorinated swimming pools (E et al., 2016) and up to 2920 mg L^{-1} in seawater filled and chlorinated pools (Boudenne et al., 2017).

E et al. (2016) presented a linear correlation of the concentrations of three volatile DBPs, trichloramine, trichloromethane and DCAN, with chloride concentrations, in both bench scale experiments and real swimming pool waters. The authors attributed this relationship to chloride promoting speciation shifts of free chlorine from HOCl to the more reactive Cl_2 (Voudrias and Reinhard, 1988), hence a higher formation of these chlorinated DBPs (E et al., 2016). Additionally, oxidant consumption was shown to increase with increasing chloride levels (E et al., 2016).

2.6.3. Swimming Pool Location

The location of a swimming pool, whether indoor or outdoor, may also affect the formation and occurrence of DBPs. Although bound by similar constraints, the contaminants found in indoor swimming pools can differ greatly to those found in pools located outdoors. Intuitively, the occurrence of sunscreens and their components is likely to be greater in outdoor swimming pools compared to those located indoors. Similarly, contaminants, such

as plant material, insects, pesticides, fertilizers, bird droppings and possibly even animals, are more likely to be found in outdoor swimming pools (Simard et al., 2013). This difference in contaminants and their subsequent reactions will result in the occurrence of different DBPs in outdoor pools compared to indoor pools.

One distinct difference between pools located indoors and outdoors is that outdoor pools are subject to natural UV irradiation and, although this is a less energetic radiation source than that typically used as secondary treatment, DBPs have been shown to decrease in sunlight exposure (Chen et al., 2010). DBP formation from sunscreen agents has been demonstrated (in laboratory based studies) to occur upon exposure to irradiation similar to that of sunlight (Sakkas et al., 2003), which is discussed in **Section 2.5.3.2**. Disinfectant residual is also known to degrade by solar photolysis, which may affect the formation of DBPs where more disinfectant is added to maintain the desired oxidant residual. Solar irradiation is likely to have a lesser impact on DBP occurrence and formation in swimming pools compared to UV based secondary treatments (**Section 2.6.1**) due to the less energetic nature of the irradiation. Additional work is required to assess the impact of solar irradiation on DBPs in the swimming pool environment.

Indoor swimming pools are often operated at higher temperatures than those located outdoors and, as discussed in **Section 2.6.5**, the increased temperature can have several effects on DBP formation. Factors such as higher reaction rates and increased volatilisation of some DBPs will impact their occurrence in indoor swimming pools. On the one hand, higher volatilisation would lead to lower concentrations of DBPs in the water but would increase their concentration in the ambient air, but on the other hand, DBPs in the ambient air of indoor pools may become trapped and hence be observed at higher concentrations, compared to outdoor pools where volatile DBPs can easily disperse.

As suggested above, the volatile THMs and chloramines were detected at lower concentrations in indoor pools compared to those located outdoors in a study by Zwiener et al. (2006). However, other studies have reported the opposite, such as the study by Simard et al. (2013) where higher THM concentrations were observed in outdoor swimming pools compared to those indoor. Higher total inorganic chloramine concentrations (up to 1723 $\mu g\ L^{-1}$) were reported for the indoor swimming pools, with lower concentrations (up to 845 $\mu g\ L^{-1}$) reported in the outdoor pools (Simard et al., 2013). In contrast, Li and Blatchley (2007) found maximum trichloramine concentrations were higher in an outdoor swimming pool (up to 160 $\mu g\ L^{-1}$) compared to an indoor swimming pool (100 $\mu g\ L^{-1}$). Natural UV treatment may help to explain the lower concentrations of *N*-nitrosamines detected in outdoor pools compared to indoor pools (Walse and Mitch, 2008).

Although swimming pool location cannot be directly correlated to DBP formation, for the reasons stated above, pool location may assist in the explanation of differences in DBP occurrence, where other parameters are comparable.

2.6.4. Swimmers: Activity and Usage

The water quality, and hence DBP formation, of swimming pools is dependent on both the number of swimmers and type of activity undertaken. Based on studies by Keuten et al. (2012), athletic swimmers (those who swim for exercise) are more likely to introduce more DBP precursors from sweat, whilst recreational swimmers (those who swim for leisure) are more likely to introduce more DBP precursors via urine. Hence, in terms of DBPs and water quality, swimming pools used mainly by athletic swimmers, such as lap pools, competition pools and pools designated for exercise (e.g. water aerobics/aquafitness), would differ from those used mainly by recreational swimmers, e.g. leisure pools, paddling pools. Additionally, although more reflective of bather load input (**Section 2.5.3**), pools used by specific people (e.g. babies, toddlers or children) may have different water chemistry and hence DBP formation.

In a very early study, Goshorn (1922) used the concentrations of nitrites, nitrates, urea and free ammonia as a measure of contamination in five swimming pools located in Philadelphia, USA, in order to investigate anthropogenic input based on gender. Swimming pools used exclusively by women were found to have the highest contamination, with pools used exclusively by men exhibiting the lowest (Goshorn, 1922). Similarly, Yeh et al. (2014) reported higher chlorinated HAA concentrations in a swimming pool used mainly by babies and mothers compared to concentrations found in other investigated pools, and, although only indicative, proposed that baby swimming pools contain higher concentrations of other DBPs due to the likely higher anthropogenic input.

Although no DBPs were studied, increased usage (due to it being the holiday period) was suggested as one reason for the high variability in chlorine (free and total chlorine equivalents) and cyanuric acid concentrations in the forty-four Turkish swimming pools investigated over three summer months, where many concentrations exceeded their respective local guidelines (Uysal et al., 2017). The effect of heavy use was investigated by Weng and Blatchley (2011), who studied an indoor chlorinated swimming pool during a swimming competition. Trichloramine was found to double in concentration over the first day and increase over the time of the competition. Similarly, DCAN and dichloromethylamine were both found to increase over the time of the swim competition. Urea concentrations significantly increased during the day, however concentrations decreased overnight. Weng and Blatchley (2011) suggested that the observed urea decrease

overnight is likely a result of surface water mixing with deeper parts of the swimming pool (resulting in a lower urea concentration at the surface where samples were collected), rather than reactions with chlorine, as the urea-chlorine reactions have shown to be quite slow (De Laat et al., 2011).

Swimmers and water activity can have a twofold effect on DBPs in the swimming pool environment. Whilst bather load increases the DBP precursors in swimming pool waters, swimming activity is known to increase volatilisation of some volatile DBPs, mainly THMs and chloramines, and hence decrease their concentrations in the swimming pool water and increase their concentrations in the ambient air. Although no correlation was observed for other DBPs (HAAs, HANs, HKs or TCNM), this may explain why THMs showed no correlation with the number of swimmers in a study by Hang et al. (2016). Supportive results were reported by Aggazzotti et al. (1995), where an increase in trichloromethane concentrations in the ambient air of a swimming pool was linked to the number of swimmers at the time of sampling, with similar results reported by Chen et al. (2016). Daiber et al. (2016) also reported an increase of non-volatile DBPs (HAA9) and decrease of volatile DBPs (TTHMs) with an increasing number of swimmers and water activity. In addition, Aggazzotti et al. (1998) reported an increase in THM concentrations in swimming pool air during water activity, when compared to those measured in the air above still waters. Similarly, a strong correlation between water jets, swimmer activity and THM removal from water has been found (Kristensen et al., 2010; Marco et al., 2015). The trichloramine concentrations in the ambient air was found to increase (0.11 to 0.36 mg m^{-3}) when school children entered a pool compared to when no swimmers were present, which was suggested to be a result of the increased mass transfer coefficient due to increased water agitation (Zwiener and Schmalz, 2015). An earlier study showed increase in mass transfer coefficients of trichloramine by swimming activity, ($1.8x10^{-3}$ to $7x10^{-3}$ g h^{-1} m^{-2}), and by splashing or water jets (up to $12.6x10^{-3}$ g h^{-1} m^{-2}) (Schmalz et al., 2011a). This trend is likely to extend to other volatile DBP classes, as demonstrated for other potentially harmful compounds (Tolis et al., 2018), with agitation leading to decreased concentrations in swimming pool waters, and increased concentrations in swimming pool air. This transfer of volatile DBPs to the gas phase is of high importance due to the inhalation uptake mechanism and further studies are required to fully understand the water-to-air relationship in terms of volatile DBPs and water activity.

Keuten et al. (2012) investigated the anthropogenic chemical release from a 60 second shower by following three parameters: TOC, TN and intracellular adenosine triphosphate, where an average release of 211, 46 and 1.6 mg per person was found for the three parameters, respectively. These studies show that a pre-swim shower will help to minimise

the anthropogenic input from bathers into pool waters. Although showering before swimming is mandatory in some countries, studies have shown that many swimmers are still unaware of the impact their swimming habits can have. Surveys of swimmers in countries where pre-swim showering is encouraged found as much as 50% of swimmers were unware of the correct reasoning behind pre-swim showering (Pasquarella et al., 2013, 2014). Whilst most swimmers (50.5%) gave the correct reasoning, "to wash oneself", many (44.3%) believed pre-swim showers are encouraged to "get you used to the temperature of the water", with a few (5.2%) indicating both reasons (Pasquarella et al., 2013, 2014). A key study highlighting the importance of swimmer education is that by Galle et al. (2016), who conducted a self-administering survey of 184 adults and 184 children in regards to five unhealthy behaviours common to swimming pools: (i) lack of pre-bathing shower, (ii) lack of pre-bathing footbath, (iii) no use of proper footwear, (iv) no use of proper swimming cap and (v) consumption of food in swimming pool environment. Although approximately 83% of children and 80% of adults stated they were aware of the rules, only 2% of people could correctly identify why the rules were in place. Additionally, results suggest that there is no correlation between viewing regulations and adopting the healthy behaviour, although an adoption of healthy behaviour (or decrease in unhealthy behaviour) was observed to increase with awareness and education level (Gallè et al., 2016). These studies show that more attention to swimmer education is required in order to decrease swimmer input to pools, which would minimise DBP formation and generally increase the quality of the swimming pool environment.

2.6.5. Temperature and pH

As mentioned in throughout this review, swimming pool water temperature has been shown to affect (i) the release of human derived input, (ii) volatilisation rates of volatile DBPs and (iii) reaction rates of DBP formation, all of which are interrelated. In addition, pools operated at elevated temperatures are required to maintain a higher disinfectant residual, which may also affect the formation of DBPs.

Heated waters were shown to promote the release of bather load derived DBP precursors, particularly those that contain nitrogen, as perspiration rate increased with increasing water temperature (Keuten et al., 2014). The increased concentrations of DBP precursors, combined with the higher disinfectant residual required in waters at elevated temperatures, likely result in an increase in DBP formation in heated swimming pools and spas. In a study of two outdoor swimming pools, Simard et al. (2013) reported higher concentrations of THMs and HAAs in the heated swimming pool. Formation of HAAs were shown to significantly increase with temperature in a laboratory scale study by Kanan (2010) where DBP formation in waters at 26 °C and 40 °C was compared. The same study also

reported that HNM formation in waters at 40 °C was twice as high as in waters at 26 °C (Kanan, 2010). NDMA concentrations were reported to be up to 10 times higher in heated spas compared to swimming pools at lower temperatures (Walse and Mitch, 2008).

Many studies have investigated the effect of pH on DBP formation in drinking waters, however few studies have investigated its effect in the swimming pool environment. The effect of pH (6 to 8) on the formation of THMs, HAAs, HANs and trichloramine was investigated by Hansen et al. (2012a) via a series of experiments involving chlorination of BFA at different pH values. Although no significant change in HAA concentrations was observed at any pH within the range investigated, THM concentrations were found to increase with increasing pH, whilst concentrations of HANs were found to decrease. In particular, one order of magnitude difference was observed in trichloramine concentrations at pH 6 compared to 7.5, with higher concentrations being evident at the lower pH values, confirming results previously reported by Schmalz et al. (2011a). A second laboratory study by Hansen et al. (2013a) reported a negligible genotoxicity effect at pH values between 6.8 and 7.5, however a significant increase in genotoxicity was observed below pH 6. Trichloromethane concentrations were observed to increase when the pH was above 7.2, similarly HANs increased at pH values below 6, and for these reasons, Hansen et al (2013a) suggest swimming pools operate at a pH range of 7 to 7.2 in order to minimise DBP formation. Swimming pool filter particles collected from a hot tub filter bed in Denmark were investigated by Hansen et al. (2012b), where chlorination of these filter particles under swimming pool conditions (pH 6 to 8, 25 °C and in the presence of constant free chlorine residual) produced similar trends to the previous studies where chlorination of BFA was performed (Hansen et al., 2012a, 2013a), i.e. the THMs increased, whilst HANs decreased, with increasing pH. However, where no change in HAA concentration was observed in the previous studies of BFA, in this study of swimming pool filter particles, concentrations of HAAs were found to increase with increasing pH (Hansen et al., 2012b). Both genotoxicity and cytotoxicity were also found to increase significantly with decreasing pH, which was reported to be likely due to the increased formation of HANs

Although knowledge has been gained from these studies, the difference in laboratory to real pool studies highlights the need for further investigation at both the laboratory scale and real pool scale. Future laboratory studies should encompass a wider range of DBPs and be conducted at conditions more suited to swimming pool waters. Additionally, the impact of temperature should be assessed for all DBP classes, in both laboratory and full scale studies, to provide a better understanding of the role of temperature on (i) reaction and formation rates of DBPs and (ii) the partitioning of DBPs from water to the air phase.

2.7. Disinfection By-Products: The Health Impacts

While the potential health impacts of DBPs have been more generally discussed in recent reviews, e.g. Richardson et al. (2007) and Cortés and Marcos (2018), this review will focus on investigations of potential health effects of DBPs specific to the swimming pool environment. Swimming pool waters have shown increased genomic DNA damage effects on Chinese hamster ovary cells compared to the corresponding filling water (Liviac et al., 2010b), an increase which is likely due to more than one mutagen (Honer et al., 1980). Short term exposure to chlorinated swimming pools saw changes in gene and microRNA expression which were indicative of an increased risk to bladder and colon cancer (Espín-Pérez et al., 2018). Changes in gene expression due to short term exposure to chlorinated swimming pools were also reported by Salas et al. (2017) in their study of adult swimmers over a forty minute swim session. Although found not to be linked to level of physical activity, exposure to chlorinated swimming pools resulted in a decrease of the concentrations of several immune markers (L-8, CCL22, CCL11, CRP and CXCL10) in serum I, as well as a significant increase in the IL-1RA concentrations in swimmers, when comparing levels measured pre and post forty minute swim session (Vlaanderen et al., 2017). In a similar study, while the uptake of several DBPs (e.g. THMs) and changes in metabolic profiles were observed, no link between the parameters could be demonstrated, nor with physical activity (van Veldhoven et al., 2018). Swimming pools treated exclusively with chlorine were found to be more toxic than those treated in combination with ozone (Fernandez-Luna et al., 2009) or UV (Liviac et al., 2010b; Plewa et al., 2011), which was attributed to the lower DBP formation when these secondary treatments were employed, compared to that when chlorination was used alone. In a more recent study, however, 320 recreational and 53 competitive swimmers reported a lower occurrence of cough and irritation to the eyes, nose or throat after modification of the swimming pool treatment method: NaOCl in conjunction with HCl was altered to include salt electrolysis and UV treatment, while replacing HCl addition with the addition of CO_2. This positive outcome was likely due to the levels of irritating oxidants (particularly trichloramine) in the air of the indoor facility, which were greatly reduced (up to 75%) after treatment modification (Gomà et al., 2017). Reported cases of contact dermatitis were much higher in swimming pools where chlorine gas was employed as the disinfectant compared to those that employed TCICA, BCDMH, calcium hypochlorite or sodium hypochlorite, which was proposed to be due to the more aggressive environment produced by (i) an increased demand (and use) of gaseous disinfectant due to the higher ability of chlorine gas to oxidise organic nitrogen, and (ii) the reduction in pH when gaseous chlorine is employed (Pardo et al., 2007). Treatment type was investigated for perceived health effects (eye or skin irritation, respiratory problems or skin dryness) in a self-reported survey of 1001 users across twenty indoor pools (Fernandez-Luna et al., 2015).

Pools treated by chlorine based disinfectants had the highest reports of health problems, with slightly lower reports for pools treated by bromine based disinfectants. Pools where secondary treatment, ozone or UV, was employed in addition to chlorine or bromine generally had lower reported health problems than pools treated by the corresponding disinfectant alone. EGMO treated pools had the lowest reported health problems of all pools investigated (Fernandez-Luna et al., 2015). The authors proposed that the higher perceived health problems in chlorinated pools can be explained by higher DBP formation (particularly chloramines and THMs) in these pools compared to pools employing additional secondary treatment or EGMO, although did not provide evidence to support this claim. The authors also acknowledged that factors other than DBPs, particularly the number of swimmers and the oxidising power of the different disinfectants and hence their ability to destroy DBPs, may also be involved (Fernandez-Luna et al., 2015).

A study from 'source to pool' by Daiber et al., (2016) showed swimming pools disinfected by bromine based disinfectants were 1.8x more mutagenic than comparable pools treated by chlorine. In comparison to their respective filling waters, pools were found to be 2.4x more mutagenic, whilst spas were found to be 4.1x more mutagenic, with spas being 1.7x more mutagenic than pools. Mutagenicity was correlated to Br-HAAs (r^2=0.98) and N-DBPs (r^2=0.97) for the chlorine treated waters, with an increase in correlation with Br-DBPs (r^2=0.82) observed in bromine treated waters. Bromine incorporation into DBPs was proposed to increase mutagenicity, although the DBP class must also be considered (Daiber et al., 2016).

A recent study by Li et al. (2015a) investigated the behaviour and appearance of rats over a 12 week swimming program, where participants were exposed to waters with similar conditions to real swimming pools (free chlorine: 1.4 to 1.6 mg L^{-1}, pH 6.5 to 7.0 and water temperature 25 to 30 °C) once a day for five days, with two days rest, for a total of 12 weeks. Some disease symptoms were induced: bloody eyes, bloody noses, loss of hair; decreased training effects (rats in chlorinated water reached exhaustion significantly faster than the control group), and deterioration of key organs (liver and lungs); all of which were proposed to be likely due to the chlorinated DBPs, trichloromethane (0.7 µg L^{-1}) and trichloramine (1.1 mg L^{-1}), also measured in the study. The intensity and frequency of training, as well as water choking, may be the primary cause of the lung damage observed in the rats (Li et al., 2015a). A similar study investigated the effect of exposure to volatile DBPs in pool air on spinal development, by exposing pulp mice to air collected from a lab scale, model swimming pool daily for several hours during early stages of life (Mcmaster et al., 2018). Results showed that, compared to the control counterparts, mice exposed to the volatile DBPs, namely trichloromethane, trichloramine and cyanogen chloride, developed

hyperkyphosis in the sagittal plane of the spine, that is, had an increase in the normal thoracic kyphotic spinal angle. Mcmaster et al. (2018) also report a period (six weeks) of latency where no negative effect was observed, such that the authors highlighted that exposure to volatile DBPs, like those reported in swimming pool air, may have a delayed negative effect on subsequent spinal development.

Rosenman et al. (2015) found a positive correlation with swimming pool attendance and several health issues, particularly asthma. Similarly, Fitch et al. (2008) reported that exposure to chlorinated pools may irritate the airways, with extended exposure likely to increase the risk of developing asthma. Several studies have suggested asthma is likely due to chlorinated volatile DBPs such as chloramines (Andersson et al., 2018; Bernard et al., 2006; Ferrari et al., 2011; Jacobs et al., 2007; Kaydos-Daniels et al., 2008; Rosenman et al., 2015; Seys et al., 2015; Uyan et al., 2009), with one study reporting a direct link between trichloramine in the air of indoor swimming pool complexes and asthma in young children (Bernard et al., 2006) and another laboratory based study reporting a causal effect of trichloramine on lung cells (Schmalz et al., 2011a). A swimming pool located indoors at a hotel in the USA was found to induce negative health effects on hundreds of occupants, the most severe case resulting in the hospitalisation of a child, which was likely due to exposure to toxic levels of chloramines in the air of the swimming pool complex (CDC, 2007).Similarly, an outbreak of health issues (e.g. cough, dyspnoea, nausea, tearing or red eyes and blocked or runny nose) were reported by several members of a swimming club (twenty-two competitive swimmers and six coaches), where an in-depth investigation (including pulmonary function testing, spirometry, measurement of fraction of exhaled nitric oxide and histamine provocation) found increased trichloramine concentrations in the ambient air of the indoor swimming pool facility were a likely cause (Seys et al., 2015). Although determined to be non-significant, a slightly higher occurrence of self-reported eye and/or throat related symptoms was also recorded for swimming pool workers compared to a control group in a more recent study (Westerlund et al., 2018). A significant increase in at least one ocular symptom was linked to trichloramine exposure in the workers, with trichloramine suggested to be responsible for the increase in the average fraction of exhaled nitric oxide in pool workers compared to the control group (Westerlund et al., 2018). Competitive and regular swimmers have been reported to have higher cases of asthma and other respiratory issues than any other type of professional sports person (Nemery et al., 2002). Considering showering, bathing, water ingestion and swimming, Font-Ribera et al. (2010a) estimated the daily THM uptake, based on THM blood levels and using published uptake algorithm factors, for children, and estimated that children who swam in indoor pools treated with chlorine or bromine would have up to four times higher THM uptake than those

who did not swim in pools, with swimming pools estimated to be the main pathway of THM exposure. This is likely due to the inhalation of THMs during swimming, as the breathing zone for swimmers is the water-air interface, where high concentrations of volatilised THMs have been reported (Catto et al., 2012a). THM concentrations in alveolar air were reportedly significantly higher for almost all adult swimmers after a forty minute swim session in a chlorinated indoor swimming pool, where increases were linked to their level of physical activity throughout the swim session (Salas et al., 2017).

Higher respiratory problems were reported in those who attended swimming pools compared to the general population (Jacobs et al., 2007), with asthma found to be higher in swimmers compared to those who did not swim (Ferrari et al., 2011). Similar results were reported by Kaydos-Daniels et al. (2008), where, in a survey of 32 swimmers, the most reported illnesses were found to be cough (84%), eye irritation (78%) and rash (34%). A survey of lifeguards who regularly work at indoor swimming pools found 55% suffered from respiratory and other health issues (Boskabady et al., 2014). THM concentrations in alveolar air were greatest in those who worked poolside compared to those who worked in reception or café areas of an indoor swimming pool complex (Fantuzzi et al., 2010). Uyan et al. (2009) suggested that lifeguards are at risk of developing eye, nose and throat issues, where the risk increases upon longer term exposure. Although in agreement that asthma is more commonly found in those who swim regularly, Goodman and Hays (2008) suggested that "it is premature to draw conclusions about the causal link between swimming and asthma".

No significant change in lung function was observed in a study by Font-Ribera et al. (2010b), who investigated the effect of swimming at an indoor swimming pool complex on respiratory health. Lung damage, as measured by changes in serum surfactant-associated protein A, was found to be negligible in a study of twenty swimmers who completed a single 40 minute session of aerobic swimming at indoor swimming pools, two treated by chlorination and one treated by chlorination in combination with UV (Llana-Belloch et al., 2016). Despite the increase of total chloramines in the air with swimmers activity (hence exposure via inhalation), no lung epithelial damage or oxidative stress was observed. Although the authors acknowledge the limitations of the study (a single swim session and relatively low free chlorine in some pools (below detection to 0.3 mg L^{-1} for chlorine pools and 1 to 1.3 mg L^{-1} for chlorine/UV pools)), they concluded that short term exercise in a pool was not correlated to lung damage (Llana-Belloch et al., 2016). A recent study by Westerlund et al. (2018) also reported no significant change in lung function of 24 swimming pool workers, when comparing lung function measured before and after shifts.

Agopain et al. (2013) reportedly showed no link exists between attendance at indoor chlorinated swimming pools and birth defects, in their study of maternal swimming pool use during early pregnancy. Similarly, no adverse health effects were observed in a study investigating swimming pool attendance and asthma (Fitch et al., 1976). Font-Ribera et al. (2009) reported lower health issues (asthma, current rhinitis and allergic rhinitis symptoms) in children who attended swimming pool complexes before the age of 2, compared to those who attended after the age of 4; however, an increase in eczema was found in children of all ages (Font-Ribera et al., 2009). Respiratory issues (lower respiratory tract infections, wheeze and otitis) were found to be higher in children who attended baby swim classes in their first six months and may be related to later respiratory issues up to 18 months of age (Nystad et al., 2008). Similarly, children exposed to pools at a young age were reported to be at a higher risk of developing asthma later in life, where a dose-response relationship was reported (Andersson et al., 2018). Inflammation of the airways and immunoglobulin E (IgE) sensitization to house dust mites were also found to be higher in children who attended swimming pool complexes at an early age (Voisin et al., 2014). Additionally, children who did not swim until a later age reportedly had lower cases of ear infections (Schoefer et al., 2008). In a study of 858 school children, categorised as either current swimmers, past swimmers or non-swimmers, Rufo et al. (2018) investigated whether swimming pool attendance influenced lung and autonomic function in children. Significantly lower maximum and average pupil restriction velocities (used to evaluate autonomic nervous function) were reported in current swimmers compared to past or non-swimmers, with current swimmers also found to have significantly higher levels of exhaled nitric oxide and a higher affinity to the beta-2 agonist compared to other groups. These findings suggest a link between swimming pool use and changes in autonomic function may exist. Furthermore, the authors concluded that, although minor and likely reversible, continual swimming pool attendance will likely increase these symptoms causing parasympathetic dysautonomia, consequently leading to increased baseline airway smooth muscle contriction (Rufo et al., 2018). Valeriani et al. (2017) recently performed a systematic review and meta-analysis of the reported epidemiology studies linking DBP exposure in swimming pools to negative health effects in children, where asthma was a particular focus. Although their review excluded studies where in vivo, in vitro or professional and accidental exposure were investigated, Valeriani et al. (2017) concluded that their review suggests “swimming in childhood does not increase the likelihood of doctor-diagnosed asthma”, further stating “the association of the disease (asthma) with indoor pool attendance is still unclear”, in agreement with the earlier conclusions of Goodman and Hayes (2008).

Villanueva et al. (2007) investigated the bladder cancer risk associated with exposure to THMs, by examining several exposure routes: ingesting of chlorinated drinking water, as well as inhalation and dermal absorption during bathing, showering and swimming in chlorinated pools. Several factors (e.g. age, type of activity, frequency and duration of swim) were evaluated for swimmers and odds ratios were determined. The study reported that exposure to THMs via swimming may be associated with the formation of bladder cancer and was the first study to demonstrate that inhalation and dermal absorption are additional exposure routes to THMs, where previous reports considered only ingestion (Villanueva et al., 2007). A later study by Lee et al. (2009) used their measured THM concentrations in 183 indoor swimming pools (treated by either chlorine, chlorine in combination with ozone or EGMO) to estimate the associated lifetime cancer risk posed to swimmers. Results showed that the cancer risk via inhalation was up to three times higher than the negligible risk factor (defined by the US EPA), whilst the risk factor from ingestion or dermal absorption was negligible in almost all cases. Although exposure to THMs via inhalation was not considered, the lifetime cancer risk associated with exposure to THMs via ingestion was reported to be lower than that associated with exposure via dermal absorption, where risks were calculated based on a multipathway model encompassing THM concentrations measured in the investigated pools treated by a combination of chlorine and ozone (Abbasnia et al., 2018). Dermal absorption was also associated with an increased risk factor in pools treated with EGMO, which was suggested to be due to the higher concentrations of brominated THMs (bromodichloro- and dibromochloro-methane) measured in these pools compared to those treated by chlorine or chlorine in combination with ozone (Lee et al., 2009). Additionally, brominated THMs have been shown to increase the genotoxicity effect (Kogevinas et al., 2010), demonstrating the high importance of minimisation of the formation of brominated THMs. Short term exposure to chlorinated swimming pools has been reported to be linked to increased risk of colon and bladder cancers, suggested to be a result of exposure to THMs (Espín-Pérez et al., 2018).

Similarly, brominated HAAs have been shown to be more toxic than their chlorinated counterparts (Liviac et al., 2010a; Plewa et al., 2008b). DeAngelo and McMillan (1990) found both DCAA and TCAA produced liver cancer in mice, with DCAA more potent than TCAA. Yeh et al. (2014) suggests that HAAs may be the decomposition products of other compounds but has shown that HAAs degrade to the equally toxic THMs. Despite HAAs having low skin permeability (Xu et al., 2002), they are still of high importance due to the transformations suggested by Yeh et al. (2014) giving rise to a wider variety of uptake mechanisms and therefore a wider range of health issues.

CH is a genotoxic and carcinogenic DBP that can be formed from a wide range of precursors evident in swimming pool waters, and has been found to decompose to chloroform and TCAA, two other potentially toxic DBPs (Barrott, 2004). HANs are another genotoxic and cytotoxic class of DBP (Plewa et al., 2008b) and are often reported to be responsible for the majority of the cytotoxicity in swimming pool waters (Hansen et al., 2012a; Pu et al., 2013). Limited data exists on the health effects of HKs, however their skin permeability has been found to triple with increasing temperature (Xu et al., 2002), therefore HKs should be of high importance in the absorption uptake mechanism, particularly in heated swimming pools and spas. Chronic cytotoxicity and genomic DNA damage have been shown in hamsters that were exposed to HNMs, with brominated NMs showing higher toxicity than their chlorinated analogues (Plewa et al., 2004). Even at low concentrations, HAAms are of high importance as they have reportedly shown much higher toxicity than many other classes of DBPs (Plewa et al., 2008a).

Nitrosamines, particularly NDMA, have been found to have several negative health effects, as summarised by CDPH (2007). Not only are nitrosamines carcinogenic in animals, they are probable carcinogens in humans, rendering them important in the swimming pool environment, even at the nanogram per litre level.

While exposure to low levels (ng L^{-1} range) of pharmaceuticals via drinking waters has been suggested to be unlikely to pose any risk to humans (e.g. Houtman et al., 2014), limited studies have investigated the risk associated with exposure to PPCPs in swimming pools. Fantuzzi et al. (2018) is the only known study to evaluate the risk associated with exposure to PPCPs in pools, where the risk (individual and cumulative) of exposure to several pharmaceuticals was reported to be negligible. Although Fantuzzi and co-workers provide an insight to this unexplored area, their study evaluated the risk associated with oral exposure (ingestion) to pharmaceuticals at the levels measured in their study. With the type and concentrations of PPCPs found to vary among pools (**Section 2.5.3.2**), and with dermal absorption and inhalation demonstrated to be important routes of exposure in the swimming pool environment, further studies are essential to fully evaluate, if any, the risk associated with exposure to PPCPs in pools.

Despite the many studies of the health impacts of swimming pools, as summarised by Lubick (2007) and Richardson et al. (2010), no definitive answers can yet be drawn in regards to the potential health effects. Many studies are only suggestive, reporting health issues that may be correlated with attending swimming pools, particularly those that are indoors and disinfected with chlorine. The lack of certainty and conflicting reports suggest that further investigation into the health impacts of swimming pools is warranted.

2.8. Conclusions

Disinfection is required to minimise the significant microbial disease risk in pools, however, leads to the unwanted formation of DBPs. Studies of DBPs in swimming pool waters have increased in recent years, focusing not only on the well documented THM and HAA DBP classes, but preliminary studies have expanded to other DBP classes, such as *N*-nitrosamines, HANs, HKs, haloacetaldehydes, halonitromethanes and haloacetamides. HAAs are generally more prevalent than THMs in swimming pool waters, which is likely due to the volatile nature of THMs, decreasing their concentration in swimming pool water but increasing their concentration in swimming pool air, as well as their rates of formation. THMs, along with other volatile DBPs, such as chloramines, are suggested to be responsible for many of the respiratory health issues potentially associated with indoor swimming pools. Other volatile or semi-volatile DBPs, such as cyanogen halides, may also have a negative impact on respiratory health, however further investigation is required to fully understand their effects.

Various factors affect DBP formation in pools, including the filling water, type of disinfectant and treatment method, numbers of swimmers and particularly input from swimmers (bather load). High use has been correlated with increasing concentrations of some DBPs, such as THMs, and TOC and mutagenicity. Volatilisation of THMs increases during swimmer activity, resulting in an initial decrease in THM concentration in the pool, with increasing concentrations observed during periods of low use (swimming pool closed). Similar effects are seen in waters with elevated temperatures, such as heated spas. These types of pools are still of high importance due to the dominant inhalation uptake mechanism demonstrated in the swimming pool environment.

Limited knowledge exists on the transformation of PPCPs in the swimming pool environment. Due to the high occurrence of nitrogen containing components, PPCPs likely result in the formation of N-DBPs, which may be more detrimental to human health than those that are entirely carbonaceous. Further studies on N-DBPs are required to fully understand their formation in the swimming pool environment. Cyanogen chloride and cyanogen bromide should be of high interest, since not only are they highly toxic DBPs, they are also intermediate products in a series of DBP formation reactions. Further knowledge of the role of these cyanogen halides may help in understanding the chemistry of swimming pool waters.

Initial studies show the presence of bromide is correlated with an increase in brominated DBPs, which are more detrimental to human health than the chlorinated analogues. Further studies are required to fully understand the role of bromide in the

swimming pool environment, particularly in seawater filled swimming pools and those that use bromine based disinfectants, where bromide/bromine concentrations are higher.

Whilst swimming pools have been correlated to respiratory health effects, such as asthma, the health effects of many DBPs at the concentrations reported in swimming pool waters and under swimming pool exposure conditions are yet to be defined. Apart from Germany and Denmark, no known swimming pool specific guidelines exist for DBPs worldwide. While of the same order of magnitude as their drinking water THM guideline value (10 $\mu g\ L^{-1}$) (TrinkwV, 2001), the German (German Institute for Standardization, 2012) and Danish (Lovtidende, 2012) swimming pool guideline values for THMs are approximately five times lower than that recommended by the World Health Organisation for THMs in drinking waters (WHO, 2011), demonstrating the need for swimming pool specific guidelines. Further investigation into DBPs and anthropogenic chemicals within the swimming pool environment should aim to support development of swimming pool specific guidelines in the future.

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2.10. References

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CHAPTER 3.1

SIMULTANEOUS ANALYSIS OF HALOACETONITRILES, HALOACETAMIDES AND HALONITROMETHANES IN CHLORINATED WATERS BY GAS CHROMATOGRAPHY-MASS SPECTROMETRY

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Statement of Contribution to Co-authored Published Paper

This Chapter includes the co-authored paper '*Simultaneous analysis of haloacetonitriles, haloacetamides and halonitromethanes in chlorinated waters by gas chromatography-mass spectrometry*', published in Chemosphere. The bibliographic details of the co-authored paper, including all authors are:

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I, Rhys Aaron Ainsley Carter, contributed to the publication by: undertaking a review of the current literature in this field, undertaking all field work, laboratory experiments and data analysis, being the primary writer (including creating figures and tables), and editing and finalising the manuscript.

I, as a Co-Author, endorse that this level of contribution by the candidate indicated above is appropriate.

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3.1.1. Abstract

Nitrogenous classes of disinfection by-products (DBPs), such as haloacetamides (HAAms), haloacetonitriles (HANs) and halonitromethanes (HNMs), while generally present at lower concentrations in disinfected waters than carbonaceous DBPs, such as trihalomethanes or haloacetic acids, have been shown to be more detrimental to human health. While several methods have been shown to be suitable for the analysis of some nitrogenous DBPS (N-DBPs) in disinfected waters, many are unable to quantify HAAms, the most detrimental to health of these three N-DBP classes. Here, we report the first method for the simultaneous analysis of twenty-five N-DBPs (nine HANs, nine HNMs and seven HAAms) in disinfected waters using liquid-liquid extraction followed by gas chromatography-mass spectrometry. The use of a programmable temperature vaporiser injector minimises degradation of the thermally labile HNMs, while avoiding the concomitant decreases in HANs and HAAms which occur when using lower injector temperatures. Extraction parameters, including sample pH, solvent volume, salt addition and sample pre-concentration, were investigated to determine the optimal conditions across all target N-DBPs. Good detection limits were achieved for all analytes (0.8 to 1.7 μg L^{-1}) and both laboratory and instrumental runtimes were significantly reduced compared to previous methods. The method was validated for the analysis of N-DBPs in drinking, swimming pool and spa waters, and concentrations of up to 41 μg L^{-1} of some N-DBPs were measured in some pools.

3.1.2. Introduction

Water quality is of great importance as untreated waters contain a wide variety of microbiological pathogens which can lead to a variety of illnesses and, in severe cases, death (Montgomery, 1985). Producing water that is safe for human use is therefore of high importance and can be easily achieved by disinfection. In disinfection, a disinfectant (an oxidising compound) is applied to a body of water, inactivating pathogens and thus improving the quality of the water. While pathogen inactivation is a desired outcome of the disinfection process, the formation of disinfection by-products (DBPs) is not. DBP formation results from reactions between the disinfectant and organic matter contained within the water.

Although over 700 individual DBPs have been identified in drinking water, recycled water and wastewater, this represents less than half the measured total organic halogen concentration and many DBPs are yet to be identified (Plewa and Richardson, 2017). Of the fraction of known DBPs, many have been demonstrated to be cytotoxic, neurotoxic and/or genotoxic, with several additionally demonstrating mutagenic, carcinogenic and/or teratogenic properties (Richardson et al., 2007), and/or developmental toxicity and growth inhibition (e.g. Liu and Zhang, 2014). Over 100 DBPs have been identified in swimming pool waters, where

a prevalence of nitrogenous DBPs (N-DBPs), e.g. haloacetamides (HAAms), haloacetonitriles (HANs), haloanilines, haloanisoles, and halonitromethanes (HNMs), have been observed (Richardson et al., 2010). N-DBPs are generally detected at much lower concentrations than carbonaceous DBPs (C-DBPs) (Krasner et al., 2006; Shah and Mitch, 2011), however, N-DBPs are still of high importance as they are often more detrimental to human health than C-DBPs (Liew et al., 2012a; Plewa et al., 2008a; Yin et al., 2018). Due to their detrimental health effects and relatively low concentrations, the ability to detect and quantify N-DBPs in waters is critical, warranting reliable N-DBP analytical methods. Several reviews of reported analytical methods for the analysis of DBPs have recently been published (Kinani et al., 2016a, 2016b; Yang and Zhang, 2016), with another focusing on N-DBPs (Ding and Chu, 2017). The existing methods for analysis of HANs, HNMs and/or HAAms, the target N-DBP classes of this study, are summarised in **Table 3.1-1**, where details of the extraction, separation and detection are provided.

Several extraction techniques, including closed loop stripping (Kampioti and Stephanou, 1999), purge and trap (Nikolaou et al., 2002), and the various methods of headspace analysis (direct injection (Montesinos and Gallego, 2012a, 2013; Nikolaou et al., 2002), solid-phase microextraction (Kermani et al., 2013; Kristiana et al., 2012; Martínez et al., 2014), single-drop microextraction (Montesinos et al., 2011)), take advantage of the volatile nature of some N-DBPs, allowing their extraction, and in some cases, pre-concentration directly from water samples. While these methods are often automated and lead to low detection limits, they require the use of specialised and somewhat costly equipment. Furthermore, such extraction techniques have been shown to be unsuitable for the extraction of HAAms (Carter et al., 2015; Quinn, 2009), which is likely due to the higher molecular weight and lower volatility of HAAms in comparison to other N-DBP classes. Solid-phase extraction methods have been shown to be suitable for the analysis of N-DBPs (Chen et al., 2002; Chinn et al., 2013; Chu et al., 2012; Hladik et al., 2014), however these methods can be time intensive and require large sample volumes to achieve the low detection limits required to detect N-DBPs in some waters. The most common extraction technique reported is liquid-liquid extraction (LLE) (Chen et al., 2015; Huang et al., 2013; Liew et al., 2012b; Ma et al., 2014; Nikolaou et al., 2002; Oliver, 1983; Plewa et al., 2008b; US EPA, 1995), with a micro-LLE (Montesinos and Gallego, 2012b) method also reported. While LLE requires the use of an organic solvent, it is currently the most suitable technique to analyse the three target N-DBP classes as (i) due to their differing physio-chemical properties it is the only extraction technique able to extract all three target classes, and (ii) LLE can easily be adopted into most analytical laboratories without purchasing additional specialised equipment.

Table 3.1-1: Reported analytical methods for the analysis of haloacetamides, haloacetonitriles and/or halonitromethanes. Detection limits refer to the reported limits of detection and correspond to the target N-DBPs analysed by each given method (those shown in **bold**). Analysis time represents the minimum time required to analyse each sample (calculated based on information supplied) and is displayed as "time of extraction/chromatographic runtime". Analysis time does not include general laboratory procedures (e.g. measuring of sample, addition of reagents) but does include sample vortex/mix/shaking times during liquid-liquid extraction (LLE) procedures.

Analytes	Detection Limits (ng L^{-1})	Water Matrix	Extraction	Separation (Column Type)	Detection	Analysis Time (min)	Reference
4 HANs, TCNM, 5 HMs, 2 HKs & 2 HAAs	1.07-1.15	Drinking	CLS	HP-5MS	ECD	120/39	(Kampioti and Stephanou, 1999)
4 HANs, 4 THMs & 3 HKs	1000-20000	Drinking	HS	HPVOC	MS	40/12	(Nikolaou et al., 2002)
9 HNMs	30-600	Drinking, Pool	HS	HP-5MS	MS	6/ 12	(Montesinos and Gallego, 2012a)
6 HANs, 6 HNMs & 4 THMs	10-200	Drinking, Pool	HS	HP-5MS	MS	20/21	(Montesinos and Gallego, 2013)
9 HNMs	50-100	Drinking, Pool	HS SDME	TRB-5	MS	22/12	(Montesinos et al., 2011)
6 HANs & 2 long chain HNs	0.9-80	Drinking	HS SPME	ZB-5MS	MS	15/53	(Kristiana et al., 2012)
4 HANs, TCNM & 2 HKs	4-200	Drinking	HS SPME	RTX-5	MS	20/10	(Kermani et al., 2013)
4 HANs, 2 HNMs, 5 THMs, 4 HKs, 3 halogenated alkanes, CIM & TBAL	3-10	RO Treated[(c)]	HS SPME	TRB-5MS	MS	15/27	(Martínez et al., 2014)
3 HANs	50-300[(a)]	Drinking	LLE	10% Squalane on chromosorb P 80/100 mesh	ECD	60/9	(Oliver, 1983)

Table 3.1-1 continued

Analytes	Detection Limits (ng L^{-1})	Water Matrix	Extraction	Separation (Column Type)	Detection	Analysis Time (min)	Reference
4 HANs, TCNM, 4 THMs, 2 HKs, CH, 8 chlorinated solvents & 17 halogenated herbicides/pesticides	1-3	Drinking	LLE	DB-1	ECD	10/40	(US EPA, 1995)
6 HANs, 4 THMs, CH & 4 HKs	7-70	Drinking	LLE	DB-1	ECD	NR/12	(Nikolaou et al., 2002)
4 HANs, 4 THMs, CH & 4 HKs	7-70	Drinking	LLE	HPVOC	MS	NR/ 36	(Nikolaou et al., 2002)
Chloro-, bromo-, iodo- & mixed haloacetamides	NR	Drinking	LLE	DB-5	MS	NR/65	(Plewa et al., 2008b)
6 HNMs & 5 HAAms	80-6000	Drinking	LLE	ZB-5MS	MS	6/41	(Liew et al., 2012b)
9 HNMs	17-217	Drinking	LLE	HP-5	ECD	NR/15	(Huang et al., 2013)
7 HANs	0.4-13.2	Drinking	LLE	RTX-5MS	MS	4/19	(Ma et al., 2014)
5 HAAms	10-100	Drinking, Pool	LLE	DB5-MS	MS	6/ 20	(Chen et al., 2015)
9 HNMs & 4 THMs	9-400	Drinking, Pool	Micro-LLE	SLB-5MS	MS	3/28	(Montesinos and Gallego, 2012b)
4 HANs, 4 THMs & 2 HKs	500-10000	Drinking	PT	HPVOC	MS	22/36	(Nikolaou et al., 2002)
4 HNMs, **TBAN**, TBAL, & 2 degradation products	NR	Drinking	SPE	DB-1	ECD	NR/40	(Chen et al., 2002)
4 HNMs, TBAN, TBAL & 2 degradation products	NR	Drinking	SPE	DB-5	MS	NR/37	(Chen et al., 2002)

Table 3.1-1 continued

Analytes	Detection Limits (ng L^{-1})	Water Matrix	Extraction	Separation (Column Type)	Detection	Analysis Time (min)	Reference
13 HAAms	7.9-19.7	Drinking	SPE	C18 Reversed Phase[(b)]	tq-MS	6[(e)]/ 9	(Chu et al., 2012)
2 HANs, 4 HNMs, 10 THMs, 1 HKs & 5 HALs	1000	Drinking	SPE	DB-5/DB-1	ECD	10/57	(Chinn et al., 2013)
8 HANs, 8 HNMs, 10 THMs, 2 HKs & 7 HALs	1000-2500	Drinking	SPE	RTX-1	MS	10/ 57	(Chinn et al., 2013)
8 HANs, 10 THMs, 1 HKs & 2 HALs	20-200	Wastewater, River[(d)]	SPE	HP-1MS	MS	215/40	(Hladik et al., 2014)

Note: All methods employed gas chromatography unless otherwise indicated. **(a)**: mg L^{-1}. **(b)**: Liquid chromatography was used. **(c)**: Includes influents and effluents of tertiary advanced reverse osmosis membrane treatment of several water types (wastewater, brackish water and sea water). **(d)**: Includes wastewater effluents and river waters upstream and downstream of wastewater treatment plants. **NR**: Not reported. **(e)**: Full details regarding SPE were not provided. **CH**: Chloral hydrate (Trichloroacetaldehyde monohydrate). **CIM**: Chloroiodomethane. **HAAms**: Haloacetamides. **HAAs**: Haloacetic acids. **HALs**: Haloacetaldehydes. **HANs**: Haloacetonitriles. **HKs**: Haloketones. **HMs**: Halomethanes. **HNMs**: Halonitromethanes. **HNs**: Halonitriles. **TCNM**: Trichloronitromethane. **TBAL**: Tribromoacetaldehyde**. TBAN:** Tribromoacetonitrile. **THMs**: Trihalomethanes. **RO**: Reverse osmosis. **CLSA**: Closed loop stripping. **HS**: Head-space. **LLE**: Liquid-liquid extraction. **SDME**: Single-drop microextraction. **SPE**: Solid-phase extraction. **SPME**: Solid-phase microextraction. **ECD**: Electron capture detector. **MS**: Mass spectrometry. **tq**: Triple quadrupole

While several analytical methods can analyse several N-DBPs of different classes, usually in combination with other DBPs, such as trihalomethanes or haloketones (Chen et al., 2002; Chinn et al., 2013; Kampioti and Stephanou, 1999; Kermani et al., 2013; Liew et al., 2012b; Martínez et al., 2014; Montesinos and Gallego, 2013; US EPA, 1995), chosen optimal conditions generally favour particular DBPs over others.

In the current study, a single LLE gas chromatography-mass spectrometry (GC-MS) method, employing a programmable temperature vaporiser (PTV) inlet, has been developed for the simultaneous analysis of twenty-five N-DBPs from three different N-DBP classes: i.e. seven HAAms, nine HANs and nine HNMs, in water samples. To the best of our knowledge, this is the first report of an analytical method developed for the simultaneous analysis of these three classes of N-DBPs. The method has been validated for drinking, swimming pool and spa waters.

3.1.3. Methodology

3.1.3.1. Analytical Standards and Reagents

All reagents were of analytical grade purity (>97%). Bromoacetonitrile (BAN), chloroacetonitrile (CAN), dibromoacetonitrile (DBAN), dichloroacetonitrile (DCAN), 1,1,2,2-tetrachloroethane-*d2* (TCE-*d2*), methyl *tert*-butyl ether (MTBE) 2,2,2-trichloroacetamide (TCAAm), trichloroacetonitrile (TCAN), acetone, and ammonium chloride were purchased from Sigma-Aldrich (Sydney, Australia). 2,2-Bromochloroacetamide (BCAAm), 2,2-dichloroacetamide (DCAAm), bromochloronitromethane (BCNM), 2,2,2-bromodichloroacetamide (BDCAAm), bromodichloroacetonitrile (BDCAN), bromodichloronitromethane (BDCNM), 2,2-dibromoacetamide (DBAAm), chloronitromethane (CNM), 2,2,2-dibromochloroacetamide (DBCAAm), dibromochloroacetonitrile (DBCAN), dibromochloronitromethane (DBCNM), dibromonitromethane (DBNM), dichloronitromethane (DCNM), 2,2,2-tribromoacetamide (TBAAm), tribromoacetonitrile (TBAN) and tribromonitromethane (bromopicrin, TBNM) were purchased from CanSyn Chem. Corp. (Ontario, Canada). 1,2-Dibromopropane-*d6* (1,2-DBP-*d6*) was purchased from CDN Isotopes (Quebec, Canada). Bromonitromethane (BNM), bromochloroacetonitrile (BCAN) and trichloronitromethane (chloropicrin, TCNM) were purchased from AccuStandard Inc. (Connecticut, USA), with BCAN and TCNM purchased as individual solutions (5 g L^{-1} in acetone). Hydrochloric acid, magnesium sulfate and sodium sulfate were purchased from Ajax Finechem. (Sydney, Australia). Ultrapure water, purified by an ELGA PURELAB Ultra purification system (18.2 MΩ cm^{-1} resistivity), was used in all experiments.

3.1.3.2. Preparation of Standard Solutions and Calibration Standards

Separate HAN, HNM and HAAm stock solutions (1 g L^{-1} of each of the nine HANs, nine HNMs and seven HAAms, respectively), as well as separate surrogate (1,2-DBP-*d6*) and internal (TCE-*d2*) standard solutions (1 g L^{-1}), were prepared in acetone. These surrogate and internal standards were selected because their use in analytical methods for N-DBPs has previously been reported (e.g. Kristiana et al., 2012; Liew et al., 2012b). Similarly, the use of acetone and MTBE as solvents has been previously reported (e.g. US EPA, 1995). Two combined analyte working solutions (containing all 25 N-DBPs at 10 and 100 mg L^{-1}) and a surrogate standard working solutions (100 mg L^{-1}) were prepared by dilution of relevant stock solutions into acetone. Similarly, an internal standard working solution (10 mg L^{-1}) was prepared by dilution of its stock solution into MTBE. Stock and working solutions were prepared each month and week, respectively. Calibration standard solutions in water containing all 25 N-DBPs (0.5 to 200 µg L^{-1}) and the surrogate standard (20 µg L^{-1}) were prepared by adding an aliquot of each of the working solutions to ultrapure water (50 mL).

3.1.3.3. Optimised Extraction Process

Water samples (50 mL, contained in 60 mL glass vials) were adjusted to pH 2 by addition of concentrated hydrochloric acid. Surrogate standard working solution (10 µL, 100 mg L^{-1}) was added, followed by MTBE (5 mL) and sodium sulfate (20 g, 40% wt/vol; previously heated at 400 °C for 24 hrs). Samples were shaken by hand (4 min) and phases were allowed to separate (5 min). The organic phase was collected and passed through magnesium sulfate which had been pre-washed with MTBE. The organic extract was pre-concentrated (to approximately 200 µL) under nitrogen by use of a Ratek dryblock heater (40 °C), transferred to a GC micro vial and internal standard working solution (2 µL, 10 mg L^{-1}) was added. GC vials were capped and analysed within 12 hours.

3.1.3.4. Optimisation of the Extraction Process

MTBE is commonly used as a LLE solvent, and its applicability as an extractant for N-DBPs has been previously demonstrated (summarised by Ding and Chu, (2017)), with several studies reporting MTBE to have equal or greater selectivity compared to other solvents (e.g. hexane, pentane or ethyl acetate) (Chen et al., 2015; Montesinos and Gallego, 2013, 2012b). MTBE was confirmed (data not presented) to be a suitable LLE solvent considering all target analytes in the current study. A range of extraction parameters, including solvent volume, pH and salt addition, were investigated by systematically changing these conditions during the extraction process and comparing the chromatographic response of each analyte, corrected by normalisation with the surrogate standard response, from the analysis of a N-DBP

calibration standard solution. Briefly, samples containing all 25 N-DBPs (20 μg L^{-1}) in ultrapure water (50 mL) were adjusted to pH 2 to 8 by addition of hydrochloric acid/sodium hydroxide solution and surrogate standard solution (10 μL, 100 mg L^{-1}) was added. Sodium sulfate (5 to 25 g; 10 to 50% wt/vol) and MTBE (1 to 5 mL) were added and samples shaken (4 min). The whole MTBE extract was concentrated (to approximately 200 μL) under a nitrogen flow by use of a dryblock heater (40 °C) and fortified with internal standard (10 μL, 10 mg L^{-1}) prior to GC-MS analysis (**Section 3.1.3.5**). The highest response ratios (analyte response/surrogate standard response) when considering all analytes were used to select the optimal condition for each extraction parameter.

3.1.3.5. Optimised Gas Chromatograph-Mass Spectrometer Conditions

The optimised N-DBP analytical method utilises GC-MS analysis on an Agilent 6890N gas chromatograph coupled with a 5975 mass selective detector (MSD) running in electron ionisation (EI) mode (70 eV) under the following conditions: MS Quad: 150 °C; MS source: 230 °C; and MSD transfer: 225 °C. Injection in splitless mode was carried out using a programmed temperature vaporiser (PTV) inlet under the following conditions: injection volume: 1 μL; purge time: 1.5 min; purge flow rate: 100 mL min^{-1}; and under the following inlet temperature conditions: 40 °C held for 1 min, heated to 160 °C at 5 °C s^{-1} and held for 5 min, heated to 270 °C at 5 °C s^{-1} and held at 270 °C for 5 min. GC separation was carried out on a Phenomenex ZB-5MS column (30 m x 250 μm i.d. and 1 μm film thickness) with helium as the carrier gas (flow rate: 1.1 mL min^{-1}). The oven temperature conditions were as follows: 40 °C held for 6 min, heated to 100 °C at 5 °C min^{-1}, heated to 130 °C at 15 °C min^{-1}, heated to 300 °C at 20 °C min^{-1} and held at 300 °C for 5 min; where the final heating from 130 °C to 300 °C occurred after elution of all target analytes with the purpose of conditioning the GC column. The total instrumental runtime was 33.5 min. Selective ion monitoring (SIM) was used for analyte identification and quantification using mass-to-charge (m/z) ratios provided in **Table A2-1.**

3.1.3.6. Optimisation of the Gas Chromatograph-Mass Spectrometer Conditions

A range of analytical parameters, including injection temperature, oven temperature program, purge flow rate and purge time, were investigated by systematically changing conditions and comparing the chromatographic response of each analyte corrected by normalisation with the surrogate standard (analyte/surrogate standard response), as well as chromatographic resolution, from the analysis of an MTBE solution containing all 25 N-DBPs (100 μg L^{-1}), the surrogate standard (100 μg L^{-1}) and the internal standard (100 μg L^{-1}). The highest resolution and normalised response over all analytes were used to select the optimal condition for each analytical parameter.

3.1.3.7. Collection of Water Samples

All water samples were collected in amber glass bottles (500 mL), filled to leave no headspace, from various locations around Perth, Western Australia. Drinking water (tap water) samples were collected from Curtin University. Swimming pool and spa water samples were collected from one facility where disinfection was achieved by use of chlorine gas combined with ultraviolet irradiation (UV) as secondary treatment. Compared to unpreserved samples, no statistically significant changes were reported to be observed in concentrations of HNMs or HAAms (Liew et al., 2012b), or HANs (Kristiana et al., 2014), in waters containing these DBPs when treated with ammonium chloride. Considering these reports, although not exclusively examined here, ammonium chloride was selected as a preservation agent, and was added to all samples at the time of collection (in 110% excess (by mass) of the measured chlorine equivalent residual). All samples were analysed within 12 hours of collection.

3.1.4. Results and Discussion

3.1.4.1. Optimisation of the Gas Chromatograph-Mass Spectrometer Conditions

Basic GC parameters, e.g. injector, column, oven and detector parameters, were initially selected based on values previously reported to be optimal (Liew et al., 2012b; Luo et al., 2014), although these studies investigated a smaller suite of N-DBPs. As such, several investigations using a solution of the 25 N-DBPs, the surrogate standard and the internal standard, each at a concentration of 1 g L^{-1} in MTBE, were undertaken in order to confirm whether initially selected values were optimal when the full suite of 25 N-DBPs were considered. Initially selected column type and detector conditions were found to be suitable across the full suite of target analytes and were chosen as optimal (**Section 3.1.3.5**).

Under these initially selected conditions, however, peak co-elution between (i) TCAN and CAN, (ii) TCE-d2 (the internal standard) and DBAN, and (iii) TBNM and BCAAm was observed. To improve peak separation between these compounds, while also considering analyte response, overall instrumental runtimes and column lifetime, changes in oven heating and hold temperatures (5 to 20 °C min^{-1} and ± 20 °C to those initially chosen, respectively) were systematically performed and chromatographic response monitored. Oven conditions that produced the greatest peak separation of the aforementioned analytes were considered optimal. **Figure 3.1-1** shows a chromatogram for the analysis a standard solution containing all 25 N-DBPs, surrogate and internal standards obtained under the chosen optimal conditions, where a total instrumental run time of less than 34 minutes was achieved. While avoiding any significant decrease in the normalised response of other target analytes, an almost complete chromatographic separation of all 25 target analytes was achieved under the selected optimal

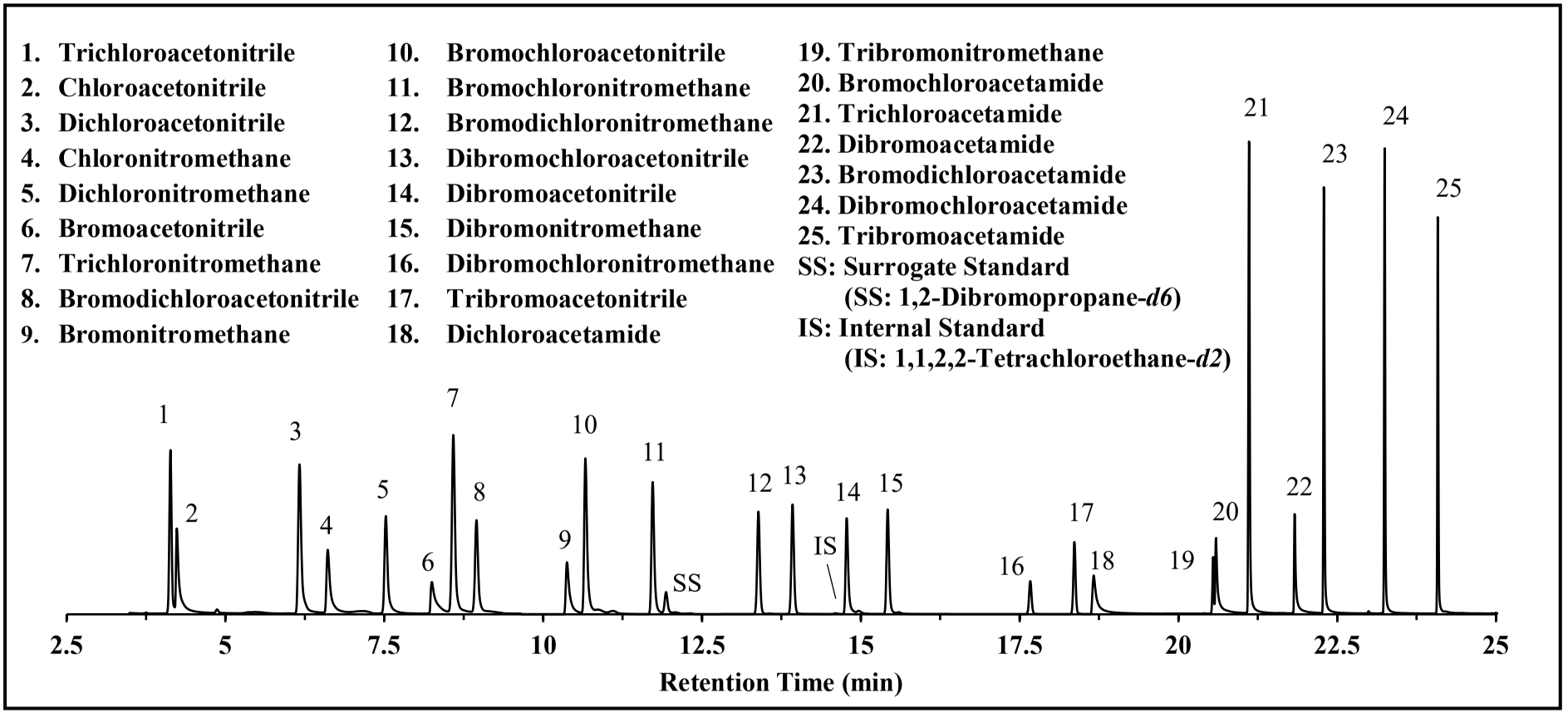

Figure 3.1-1: Chromatographic separation of 25 nitrogenous disinfection by-products (N-DBPs), (9 haloacetonitriles (HANs), 9 halonitromethanes (HNMs) and 7 haloacetamides (HAAms)), using the developed liquid-liquid extraction gas chromatography-mass spectrometry (LLE GC-MS) analytical method. N-DBPs: 100 µg L^{-1}; surrogate standard: 100 µg L^{-1}: and internal standard: 100 µg L^{-1}. Chromatogram collected in selected ion monitoring (SIM) mode on a ZB-5MS column (30 m x 250 µm i.d. and 1 µm film thickness) under conditions described in **Section 3.1.2.5**.

conditions (**Table 3.1-2**), although peak overlap is still observed between TBNM and BCAAm (**Figure 3.1-1,** peaks 19 and 20, respectively). Although further adjustment in oven temperature programming may have led to a more complete chromatographic separation of these compounds, employing such conditions would result in impractical and unrealistic instrumental runtimes. While all efforts were made to reduce the peak co-elution between TBNM and BCAAm, its effect on the ability to quantify these N-DBPs was minimised by carrying out analysis in SIM mode. The selection of quantification ions unique to each target analyte (**Table A2-1**) resulted in complete baseline separation between these two N-DBPs and, hence, any impact of peak co-elution was avoided.

Table 3.1-2: Investigated parameters and their chosen optimal values during method optimisation. Temperatures are presents as "Initial or target; heating rate; (hold time)".

Parameter	Range Investigated	Chosen as Optimum
Gas Chromatograph-Mass Spectrometer		
Oven Temperature Programming	Initial: 30 or 40 °C; (1 to 6 min) Ramp 1: 90 or 100 °C; 5 to 20 °C min^{-1} Ramp 2: 110 or 130 °C; 5 to 20 °C min^{-1} Ramp 3: 300 °C; 5 to 20 °C min^{-1}	40 °C; (6 min) 100 °C; 5 °C min^{-1} 130 °C; 15 °C min^{-1} 300 °C; 20 °C min^{-1}
Split/Splitless Injector Temperature	120 to 220 °C	160 °C
PTV Injector Temperature	-	Initial: 40 °C; (1 min) Ramp 1: 160 °C; 5 °C s^{-1}; (5 min) Ramp 2: 270 °C; 5 °C s^{-1}; (5 min)
Liquid-Liquid Extraction		
Sample pH	2 to 10	2
Acid Type	Hydrochloric or Sulfuric	Hydrochloric
Solvent Volume	1 to 5 mL	5 mL
Extract Pre-concentration	Pre-concentrate or Aliquot	Pre-concentrate
Salt Quantity	5 to 25 g (0 to 50% wt/vol)	20 g (40% wt/vol)
Salt Type	Sodium Sulfate or Sodium Chloride	Sodium Sulfate

Higher injection port temperatures (170 to 250 °C) have been shown to be optimal for the analysis of HANs (Kampioti and Stephanou, 1999; Kristiana et al., 2012; US EPA, 1995) and HAAms (Plewa et al., 2008b), however, high injection temperatures (>140 °C) have been shown to cause decreases in the response of HNMs due to their thermal degradation (Chen et al., 2002). In order to select optimal injector conditions across the full suite of 25 N-DBP target analytes, injection port temperatures were systematically altered (120 to 220 °C) and analyte response (normalised by surrogate standard) monitored. Consistent with a previous study (Chen et al., 2002), higher responses for HNMs were observed at lower injection temperatures,

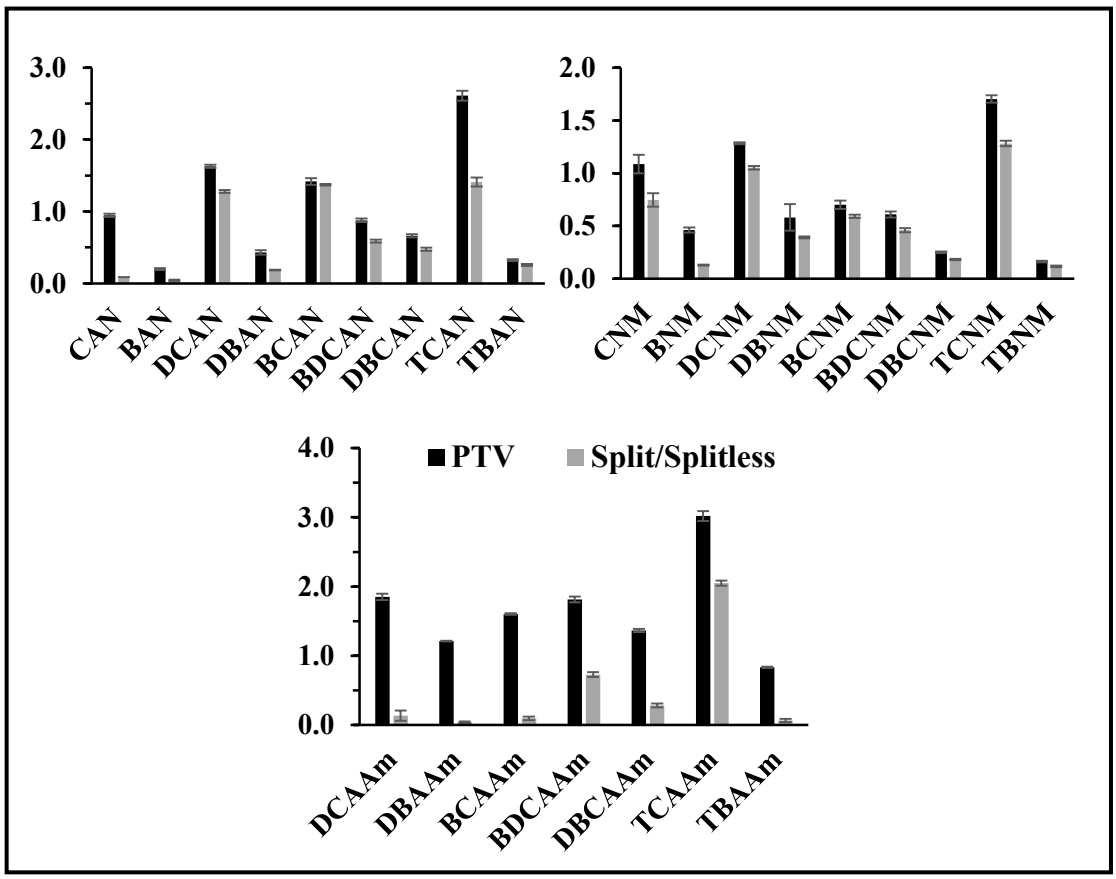

Figure 3.1-2: Comparison of normalised analyte response (analyte response/surrogate standard response) observed when employing a program temperature vaporiser (PTV) inlet to those observed when employing a split/splitless injector.

while responses of HAAms generally increased with increasing injection port temperatures. In order to avoid thermal degradation of HNMs, an injection port temperature of 160 °C was chosen. As observed in the current study, lower injection temperatures have been reported to be optimal in the analysis of HNMs despite a concomitant decrease in the response of HAAms (Liew et al., 2012b). Furthermore, regular operation at low injector temperatures leads to the potential for injector contamination and the need for more regular injector maintenance (Carter, 2014), particularly in the analysis of more complex matrices such as swimming pool waters. Fortunately, this compromise in response is likely to be easily overcome by use of a PTV inlet, resulting in an increase in method performance and lower detection limits, and the ramping capabilities of a PTV inlet can be employed as a cleaning mechanism between sample injections to minimise the impact of sample contamination. As low initial PTV temperatures (40 to 45 °C) have previously been shown to be optimal for the analysis of HANs (Ma et al., 2014) and HNMs (Montesinos and Gallego, 2012b), an initial temperature of 40 °C was chosen in the current method optimisation. Similarly, a low PTV temperature ramp rate (5 °C s^{-1}) to the previously demonstrated optimal hold temperature (160 °C) was selected in order to minimise HNM degradation. Finally, a second inlet temperature increase was employed as a

cleaning mechanism. The final PTV temperature conditions were as follows: 40 °C held for 1 min, heated to 160 °C at 5 °C s^{-1} and held for 5 min, heated to 270 °C at 5 °C s^{-1} and held at 270 °C for 5 min. Significantly higher responses for all target N-DBPs were observed when a PTV inlet was employed using the aforementioned conditions, compared to those observed when a split/splitless injector at the previously optimised temperature (160 °C) was used (**Figure 3.1-2**). As such, the use of a PTV inlet was selected.

3.1.4.2. Optimisation of the Liquid-Liquid Extraction Procedure

3.1.4.2.1. The Effect of Organic Extract Pre-Concentration and Liquid-liquid Extraction Solvent Volume

In order to determine the detection limits of the target N-DBPs, two standard solutions each containing all 25 N-DBPs (20 and 100 µg L^{-1}), the surrogate standard (20 µg L^{-1}) and the internal standard (20 µg L^{-1}) in MTBE were analysed. While most N-DBPs were not observed in the lower concentration standard solution (20 µg L^{-1}), some N-DBPs (BDCAN, DBCAN, TBAN, BDCNM, DBCNM, DCAAm and DBAAm) were not observed even for the high concentration (100 µg L^{-1}) standard solution. To increase analyte response and detection, and thus decrease analyte detection limits, the effect of organic extract pre-concentration was investigated for all analytes by the analysis of pre-concentrated and non-pre-concentrated organic extracts. Briefly, samples containing all 25 N-DBPs (20 µg L^{-1}) in ultrapure water (50 mL) were adjusted to pH 4 by addition of hydrochloric acid and surrogate standard solution (10 µL, 100 mg L^{-1}) was added. Sodium sulfate (20 g, 40% wt/vol) and MTBE (5 mL) were added and the mixtures were shaken (4 min). The MTBE extracts were collected and dried. To prepare pre-concentrated extracts, the whole MTBE extract was concentrated (to approximately 200 µL) under a nitrogen flow by use of a dryblock heater (40 °C), while for non-pre-concentrated extracts, an aliquot of the MTBE extract (1 mL) was analysed. Both extracts were fortified with internal standard added at the same concentration prior to GC-MS analysis. With organic extract pre-concentration, all 25 N-DBPs were observed in the GC-MS analysis, demonstrating the significant improvement in analyte detection limits. In addition, the analyte responses, normalised by the internal standard, for the pre-concentrated and non-pre-concentrated organic extracts were compared. As expected, most analytes showed an increased response (2 to 343%) when organic extract pre-concentration was employed (**Figure 3.1-3(a)**). Generally, higher increases in response were observed for the HAAms (152 to 343%) and the HANs (2 to 275%) compared to those observed for the HNMs (28 to 120%). CAN, TCAN, TCNM and TBNM showed decreases in response (19, 52, 7 and 95%, respectively) when organic extract pre-concentration was employed, which is likely due to

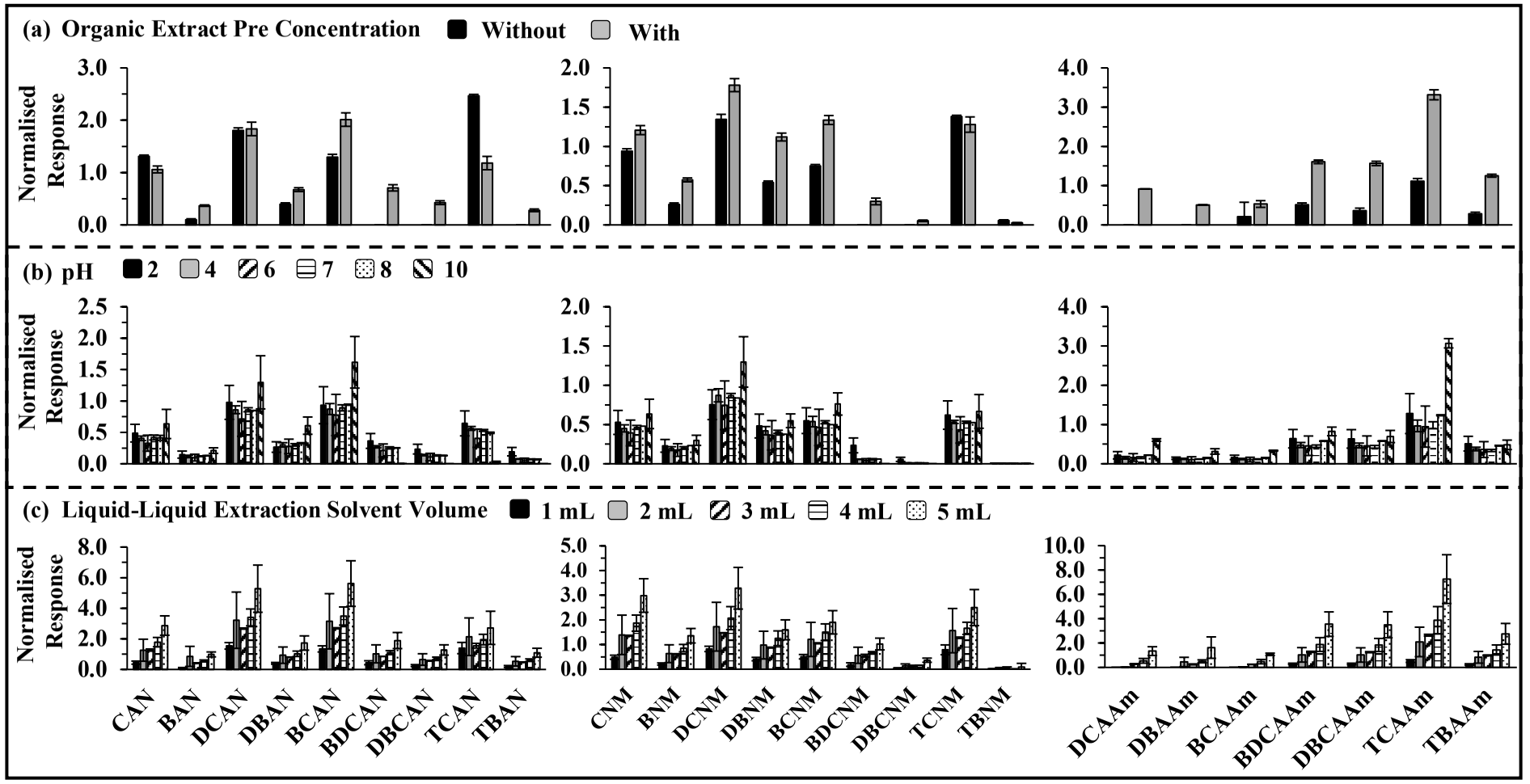

Figure 3.1-3: The effect of **(a)** organic extract pre-concentration, **(b)** sample pH and **(c)** liquid-liquid extraction solvent volume during the extraction process on normalised analyte response (analyte response/surrogate standard response).

their highly volatile nature (Nikolaou, 2003). While some loss of these few analytes was observed, this is overcome by the use of a surrogate standard added at the start of the extraction process. As N-DBPs are likely to be detected in trace amounts in real water samples (Carter and Joll, 2017; Liew et al., 2012a), lowering analyte detection limits is key for application of this method to real water samples. For these reasons, organic extract pre-concentration after LLE was adopted for this analytical method.

The effect of LLE solvent volume on analyte response was also investigated by altering the amount of solvent (1 to 5 mL) employed during the LLE. Extractions were performed as per **Section 3.1.3.4**, where a sample pH of 4 and a sodium sulfate concentration of 40% (wt/vol) was employed. Normalised analyte responses (**Figure 3.1-3(c)**) for differing LLE solvent volumes were used to assess the impact of solvent volume. Solvent volumes of 1 mL were problematic during extraction as visual separation of layers and physical separation of the organic layer for analysis was difficult. For volumes between 2 to 5 mL, generally, an increase in solvent volume led to an increase in response across all analytes. In development of an earlier analytical method for HAAms and HNMs, we have previously reported lower response ratios of HAAms and HNMs when a lower MTBE volume (2 mL) was used compared to when a larger volume (5 mL) was employed (Liew et al., 2012b). Although solvent volumes greater than 5 mL may show a further increase in response, these larger volumes would lead to excess solvent usage and become impractical for operation, i.e. total sample volume would exceed the size of the sample vial used. Therefore, a solvent volume of 5 mL was chosen.

3.1.4.2.2. The Effect of Sample pH

The effect of pH on analyte response was investigated by adjusting the sample pH (2 to 10), using HCl or NaOH solutions, during the LLE. Extractions were performed as per **Section 3.1.3.4**, where a solvent volume of 5 mL and a concentration of 40% (wt/vol) of sodium sulfate was employed. Normalised responses (analyte response/surrogate standard response) for each investigated pH value were used to assess the impact of sample pH on analyte extraction (**Figure 3.1-3(b)**). While maximum response was observed at pH 10 for some analytes, tri-HANs and mixed Br/Cl-tri-HNMs were not detected at this pH, which is likely due to their susceptibility to base-catalysed degradation, as previously observed for TBNM, TCNM and TBAN (Chen et al., 2002). Only slight differences in analyte response ratios were observed between pH 2 to 8 for all analytes (N-DBPs). As equal or greater response ratios were observed at pH 2 for the majority of the analytes (all but DBAN and DCNM), a pH value of 2 was identified as optimal. This optimal pH value is consistent with lower responses of HNMs being reported outside the pH range of 2 to 4 (Montesinos et al., 2011;

Montesinos and Gallego, 2012b, 2012a). As observed in the current study, little or no change in responses for HNMs (Huang et al., 2013; Liew et al., 2012b), HAAms (Liew et al., 2012b) or HANs (Ma et al., 2014) over the pH ranges 3.5 to 8, 3.5 to 5 and 2 to 7, respectively have also been reported. The type of acid used for pH adjustment was also investigated by comparing normalised analyte responses from samples where the pH was adjusted (to pH 2) using concentrated hydrochloric or sulfuric acid (**Figure A2-1(a)**). Similar or slightly higher responses were observed for all analytes when hydrochloric acid was used for pH adjustment compared to the use of sulfuric acid, hence hydrochloric acid was chosen for pH adjustment.

3.1.4.2.3. The Effect of Salt Addition

The addition of salt during extraction has previously been shown to increase the extraction of N-DBPs (Huang et al., 2013; Kampioti and Stephanou, 1999; Kristiana et al., 2012; Ma et al., 2014; Montesinos et al., 2011; Montesinos and Gallego, 2012a, 2012b), although one study also reported a decrease for some HNMs investigated (Huang et al., 2013). As the effect of salt addition on N-DBP response is still unclear and to determine the concentration of salt to be used in the current method to produce the optimal extraction across all 25 N-DBPs, the normalised analyte responses were compared between samples where different concentrations of sodium sulfate (10 to 50% (wt/vol)) were employed. Extractions were performed as per **Section 3.1.3.4**, where a solvent volume of 5 mL and a sample pH of 2 were employed. Normalised analyte response was observed to generally increase with increasing salt addition up to 40% (wt/vol) (**Figure A2-1(c)**), although a decrease in response for all analytes was observed at 50% (wt/vol). Similar results have previously been reported (Ma et al., 2014), where an increase in sodium sulfate concentration (0 to 30% wt/vol investigated) saw an increase in the response of several HANs, although a slight decrease (or constant) response of TCAN was observed for salt concentrations greater than 20% (wt/vol). The authors suggested this may be attributed to the lower polarity of TCAN compared to other HANs. Additionally, the study noted that their investigations did not study the impact of salt addition above the point at which saturation occurred (> 30% wt/vol), which may explain why a decrease in analyte response was observed at 50% (wt/vol) in the current study. Furthermore, the sorption of HANs to undissolved sodium sulfate particles was proposed as an explanation to the observed decrease in response of some HANs at salt concentrations greater than 30% (wt/vol) (Kristiana et al., 2012), and although this was observed during the development of a solid-phase microextraction (SPME)/GC-MS method, it may aid in explaining the decrease in N-DBP responses observed at 50% (wt/vol) in the current study. Since the response for all analytes was highest for a sodium sulfate concentration of 40% (wt/vol), this concentration was considered optimal.

The type of salt employed was also investigated in this work, by comparing normalised analyte responses from the use of sodium sulfate and sodium chloride for salt addition. For all target N-DBPs, significantly higher responses were observed when sodium sulfate was employed compared to responses observed when sodium chloride was used (**Figure A2-1(b)**). This is consistent with previous reports (Liew et al., 2012b; Montesinos et al., 2011; Montesinos and Gallego, 2012b), where sodium chloride was found to be less efficient compared to sodium sulfate in the extraction of N-DBPs, therefore, the use of sodium sulfate (40% wt/vol) was chosen for salt addition in this method.

3.1.4.3. Method Validation

3.1.4.3.1. Linearity, Limits of Detection and Precision

In order to validate the developed analytical method, linearity, detection limits and precision were evaluated for each analyte using external calibration. Linearity was investigated by assessing the correlation coefficient (r^2) resulting from the analysis of N-DBPs in ultrapure water extracted under the defined optimal conditions (**Sections 3.1.3.3** and **3.1.3.5**), across the concentration range of 0.5 to 200 µg L^{-1}, as this encompasses the ranges reported for the N-DBPs in treated waters (e.g. Bond et al., 2011; Carter and Joll, 2017; Liew et al., 2012a). As summarised in **Table 3.1-3**, most analytes showed almost perfect linearity ($r^2 \geq 0.99$), with the other analytes, DCAAm, BCAAm, TBAAm, BAN, TBAN, BNM, BCNM, DBNM and TBNM, still demonstrating excellent linearity over the investigated range ($r^2 \geq 0.98$). Method detection limits were calculated as defined by the United States Environmental Protection Agency (US EPA, 2014). Briefly, the standard deviation in response ratios of replicates (n = 10) of a low concentration standard (5 µg L^{-1}) was multiplied by 2.764 (the appropriate t-value at the 99th percentile with n-1 degrees of freedom). Calculated detection limits were found to range from 0.8 µg L^{-1} for DBCAN to 1.7 µg L^{-1} for DCNM and TBAN (**Table 3.1-3**), which are comparable to most other analytical methods employing LLE (**Table 3.1-1**). Due to the higher sensitivity of SPME compared to LLE, lower detection limits were reached for methods employing SPME (**Table 3.1-1**), however, SPME has been shown to be unsuitable for the analysis of HAAms (Quinn, 2009).

The precision of the developed method was assessed by evaluating the repeatability and reproducibility in the analysis of ultrapure water samples containing all 25 N-DBPs (20 µg L^{-1}). Repeatability was evaluated by the analysis of 10 samples within one day, while reproducibility was evaluated by the analysis of 30 samples on 3 different days (within 1 week), where samples were prepared and extracted on each given day. Excellent repeatability was observed for all three classes of N-DBPs (2 to 11% RSD). Good reproducibility (3 to 15% RSD) was observed for most analytes, however reproducibility for

DBCNM, TBNM, DBCAAm and TBAAm was slightly poorer (17 to 31% RSD), which is likely due to the lower stability of these compounds (Liew et al., 2012b).

Table 3.1-3: Method validation data for the current analytical method.

Analyte	Correlation Coefficient (r^2)	Detection Limit (µg L^{-1})	Repeatability (%) / Reproducibility (%)	Recovery (%)		
				Tap Water	Pool Water	Spa Water
Haloacetamides (HAAms)						
Dichloroacetamide	0.988	1.1	11/11	112±14	124±7	93±22
Dibromoacetamide	0.990	1.2	9/14	109±10	125±8	121±19
Bromochloroacetamide	0.986	1.2	11/12	115±12	118±1	128±11
Bromodichloroacetamide	0.994	1.1	8/14	99±7	95±1	95±7
Dibromochloroacetamide	0.992	1.2	9/21	101±7	96±1	87±4
Trichloroacetamide	0.997	1.4	7/8	96±6	100±2	113±19
Tribromoacetamide	0.988	1.0	11/31	104±8	99±2	98±6
Haloacetonitriles (HANs)						
Chloroacetonitrile	0.996	1.6	3/4	98±11	97±1	94±2
Bromoacetonitrile	0.986	1.3	10/12	115±7	112±7	114±9
Dichloroacetonitrile	0.999	1.6	3/4	94±6	90±1	85±1
Dibromoacetonitrile	0.996	1.1	5/11	107±8	95±1	87±3
Bromochloroacetonitrile	0.998	1.3	4/5	98±7	89±2	82±1
Bromodichloroacetonitrile	0.996	0.9	2/15	92±9	89±4	79±8
Dibromochloroacetonitrile	0.994	0.8	4/12	98±7	87±1	80±13
Trichloroacetonitrile	0.999	1.5	7/10	75±8	79±2	79±1
Tribromoacetonitrile	0.989	0.7	6/17	108±9	96±1	87±18
Halonitromethanes (HNMs)						
Chloronitromethane	0.996	1.3	3/7	98±8	88±1	104±9
Bromonitromethane	0.989	1.1	6/8	105±7	102±1	100±4
Dichloronitromethane	0.999	1.7	3/3	74±2	69±5	77±2
Dibromonitromethane	0.997	1.1	5/10	70±5	66±3	76±2
Bromochloronitromethane	0.998	1.2	4/5	63±14	58±1	78±1
Bromodichloronitromethane	0.992	1.2	4/9	116±3	127±10	86±12
Dibromochloronitromethane	0.986	0.5	7/17	110±5	119±13	130±22
Trichloronitromethane	0.999	1.3	2/3	93±16	97±1	79±3
Tribromonitromethane	0.980	0.4	10/19	108±4	120±2	107±22

3.1.4.3.2. Bias of Different Water Matrices

Tap, pool and spa water samples fortified with all 25 N-DBPs (20 µg L^{-1}) were analysed to assess the applicability of the method for these different water matrices. Good to excellent recoveries (58 to 128%; **Table 3.1-3**) were observed for almost all analytes over all water types investigated. Larger ranges in recoveries were observed in pool (58 to 127%) and spa (68 to 128%) waters compared to that observed in tap water (63 to 116%), which is likely due to the more complex matrices of the pool and spa waters. Furthermore, the lower recoveries (49 to 77%) observed for the di-halogenated NMs (DCNM, BCNM and DBNM) are likely due to their partial conversion to their corresponding tri-halogenated nitromethane (i.e. TCNM, BDCNM and DBCNM, respectively) by reaction with the low concentration of chlorine (in equilibrium with the chloramine the fortified water matrices). This is consistent with the rapid transformation of HNMs observed in our previous work (Carter, 2014), and further aligns with the higher recoveries observed for BDCNM and DBCNM (**Table 3.1-3**) for most water matrices investigated. To minimise this issue in real water samples, samples should be immediately quenched and then analysed for HNMs as soon as practicable. Overall, the results show that this method is applicable for the analysis of N-DBPs in tap, pool and spa waters.

3.1.4.3.3. Analysis of Swimming Pool Waters

The optimised method was used to investigate the occurrence of N-DBPs in several swimming pools (a 10 lane (25 m) lap pool, a 3 lane leisure and walk pool and a heated spa) located at one swimming pool facility in Perth, Western Australia. All pools were treated independently by disinfection using chlorine gas, with UV employed as a secondary treatment. Eight of the 25 N-DBPs were measured in at least one of the investigated swimming pools (**Table A2-2**). DCAN was the only HAN to be detected in all pools, being measured at 16, 41 and 27 µg L^{-1} in the lap, leisure and spa pools, respectively. CAN and BDCAN were also detected in some of the investigated pools, up to 1.9 and 1.5 µg L^{-1}, respectively. TBNM was detected in the spa pool (2.8 µg L^{-1}), while TCNM was measured in the leisure pool (1.4 µg L^{-1}). BCAAm and TCAAm were detected in all investigated pools: 2.6 to 3.4 and 2.7 to 3.3 µg L^{-1}, respectively; with DCAAm only measured in the spa pool: 3.5 µg L^{-1}. Excluding DCAN, where several studies of swimming pool waters have reported significantly higher concentrations (up to 206 µg L^{-1} (Hang et al., 2016; Manasfi et al., 2016; Weaver et al., 2009)), N-DBPs measured in this study were generally similar to those reported in the literature (Carter and Joll, 2017).

3.1.5. Conclusions

This study presents the first method for the simultaneous analysis of these three N-DBP classes, including 9 HANs, 9 HNMs and 7 HAAms. In comparison to previous LLE methods, which require multiple methods to analyse all twenty five N-DBPs proposed in this work, the combination of these 25 N-DBPs in one simple LLE followed by GC-MS method provides a significant decrease in both laboratory and instrumental runtimes. The use of a PTV inlet enhances the detection of the thermally labile HNMs, while minimising instrumental maintenance that would usually be necessary with constant operation at lower injection port temperatures, particularly with more complex matrices such as pool and spa waters. The method provides excellent linearity and repeatability, and achieves detection limits less than 2 $\mu g\ L^{-1}$ for all 25 N-DBPs, which are comparable to previously reported detection limits for LLE methods for these analytes. Furthermore, the method has been shown to be suitable for the analysis of tap, swimming pool and spa waters and the occurrence of a number of N-DBPs in these water types has been demonstrated.

3.1.6. Acknowledgements

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CHAPTER 3.2

AN ANALYTICAL METHOD FOR THE ANALYSIS OF TRIHALOMETHANES IN AMBIENT AIR USING SOLID-PHASE MICROEXTRACTION GAS CHROMATOGRAPHY-MASS SPECTROMETRY: AN APPLICATION TO INDOOR SWIMMING POOL COMPLEXES

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I, Rhys Aaron Ainsley Carter, contributed to the publication by: undertaking a review of the current literature in this field, undertaking all field work, laboratory experiments and data analysis, being the primary writer (including creating figures and tables), and editing and finalising the manuscript.

I, as a Co-Author, endorse that this level of contribution by the candidate indicated above is appropriate.

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3.2.1. Abstract

A simple method for the collection and analysis of the four brominated and chlorinated trihalomethanes (THMs) in air samples is described. Ambient air samples were collected in pre-prepared glass vials, with THM analysis performed using solid-phase microextraction gas chromatography-mass spectrometry, where the need for chemical reagents is minimised. Analytical parameters including oven temperature program, solvent volume, incubation time, vial agitation, extraction time and temperature, as well as desorption time and temperature, were evaluated to ensure optimal method performance. The developed method allows for point-in-time quantification (compared to an average concentration measured over extended periods of time), with detection limits between 0.7 to 2.6 μg m^{-3}. Excellent linearity (r^2>0.99), repeatability (3 to 11% RSD) and reproducibility (3 to 16% RSD) were demonstrated over a concentration range from 2 to 5000 μg m^{-3}. The method was validated for the analysis of THMs in indoor swimming pool air and was used to investigate the occurrence of THMs in the air above fifteen indoor swimming pools. This is the first study to report the occurrence of THMs in swimming pool air in Australia and concentrations higher than those previously reported in other countries were measured.

3.2.2. Introduction

Trihalomethanes (THMs), particularly the chloro-, bromo-, and mixed bromochloro-trihalogenated species, are a commonly reported class of disinfection by-product (DBP) in swimming pool waters (Carter and Joll, 2017), although iodinated THMs have also been reported (Carter et al., 2015; Yeh et al., 2014). THMs are a volatile class of DBP, and as such, can partition from the water into the ambient air of swimming pool complexes, where concentrations have been shown to increase with water agitation either by swimmers or water jets (Chen et al., 2016; Font-Ribera et al., 2016; Kristensen et al., 2010; Lévesque et al., 2015; Llana-Belloch et al., 2016). Many studies have reported the occurrence of THMs in the air above swimming pool and spa waters (Carter and Joll, 2017), and THMs have also been reported in passageways, offices/reception areas, café areas, plant/engine rooms and change-rooms in pool complexes (Aprea et al., 2010; Fantuzzi et al., 2010, 2001; Tardif et al., 2015).

Inhalation has been shown to be the major route of exposure for THMs in the swimming pool environment (Aprea et al., 2010; Chen et al., 2011; Erdinger et al., 2004). Higher exposure to THMs was reported for employees who worked pool-side compared to those working in reception or plant room areas of the same facility (Caro and Gallego, 2008; Fantuzzi et al., 2001). Although irritants, such as chloramines and other chlorinated products, in swimming pool air have been suggested to promote asthma and other respiratory issues (e.g. cough, wheeze or lower respiratory tract irritation) in swimming pool attendees (Parrat et al.,

2012; Rosenman et al., 2015; Schmalz et al., 2011), particularly in young children/adolescents (Bernard et al., 2009, 2006; Kaydos-Daniels et al., 2008; Nystad et al., 2008; Voisin et al., 2014), and swimming pool workers (Boskabady et al., 2014; Jacobs et al., 2007; Uyan et al., 2009), several studies have suggested that exposure to swimming pool air does not promote respiratory issues (Fitch et al., 1976; Font-Ribera et al., 2009; Fornander et al., 2013; Llana-Belloch et al., 2016; Schoefer et al., 2008). Considering THMs specifically, Font-Ribera et al. (2010) found no significant changes in lung function (surfactant protein D, 8-isoprostane, eight cytokines or vascular endothelial growth factor) and only a slight change (3.3% increase) to median serum CC16 (a marker of lung epithelium permeability), suggesting that any impact of THMs on lung function is short-lived (Font-Ribera et al., 2010). With the potential health effects of exposure to THMs in swimming pool ambient air unclear, further investigations into their occurrence, human exposure and the potential health effects are warranted, requiring robust and validated analytical methods.

A comprehensive review of the reported analytical methods for analysis of THMs in the ambient air of swimming pool complexes indicated that most studies employ methods based on those previously developed, that is, few studies have fully developed or validated their analytical method since their adaptation. Furthermore, while adapted methods were employed for the analysis of the four common THMs (trichloromethane, bromodichloromethane, dibromochloromethane and tribromomethane), the original methods were only validated for trichloromethane, and tribromomethane in some cases; not for the analysis of bromodichloromethane or dibromochloromethane. Fully developed and validated methods include those by Aggozzotti et al (1990), NIOSH Standard Method 1003 (NIOSH, 2003), Van Den Velde (2007), Sa et al. (2011) and US EPA Standard Methods TO-14 (US EPA, 1999a), while a summary of the various adaptions of these validated methods is provided in **Table A3-1**. Each analytical method can be divided into two distinct parts: (i) air sample collection and (ii) air sample analysis. In all cases, analysis was carried out by gas chromatography (GC) with various detectors: electron capture detection (ECD), flame ionisation detection (FID) or mass spectrometry (MS).

Sample collection can be achieved by collecting air as a whole or by trapping target compounds by the use of adsorbent materials. Whole air samples are generally collected by the use of Summa canisters, as per EPA Standard Methods TO-14 (US EPA, 1999a), although other collection methods employing Tedlar or aluminised bags, or glass vials, have been employed (**Table A3-1**). Due to the large volumes collected (5 to 20 L) and pressurisation of Summa canisters, water condensation, and hence loss of water soluble analytes, has been shown to be a potential issue with Summa canisters (US EPA, 1999a), an effect which is likely to be more profound for humid samples, such as swimming pool air. Although reusable,

Summa canisters require extensive preparation, and thorough cleaning, usually with harsh chemicals, and can be costly to purchase. While the use of Tedlar or aluminised bags can be less expensive compared to Summa canisters, they are limited to a one-time use. Alternatively, target compounds may be trapped on sorbent materials, including Tenax, activated carbon and Chromosorb (**Table A3-1**). The use of adsorbents allows greater selectivity compared to whole air samples, as different adsorbents can trap different compounds. While the issues of moisture can be avoided by selection of a hydrophobic sorbent or the use of moisture traps at the time of sample collection, contamination from adsorbents and artifact formation have been shown to occur (US EPA, 1999b). Furthermore, adsorbent preparation can involve the use of harsh chemicals. With the exception of Summa canisters, where negative pressure may be used as a driving force, other containers, including those containing adsorbent materials, require the use of a pump.

While pumps allow automation of sample collection, flow rates must be calibrated in order to collect desired volumes and minimise analyte breakthrough (US EPA, 1999b). Analyte breakthrough can also be minimised by the use of lower flow rates and additional sorbent materials, although this often leads to extended sampling times (12 to 24 hours). One advantage of extended sampling times is the possibility of lower detection limits. However, while sampling can be performed unattended in some situations making extended sampling times practical, they are impractical in some field settings such as public swimming pools as the sampling equipment must remain poolside, and as such, may pose a safety issue and/or create anxiety amongst swimming pool patrons. Furthermore, sample collection performed over extended periods of time (e.g. up to several hours; **Table A3-1**) produces responses that represent an average over the collection period, rather than those at one specific point in time. The averaging of concentration responses can lead to less accurate results, particularly when calculating risk factors, as swimmers may be exposed to higher than these average concentrations during their swim session, particularly as THM concentrations in pool air have been shown to be highly variable over short periods (Chen et al., 2016). Due to the uncertainty surrounding the health effects of volatile organic compounds, e.g. THMs, in swimming pool air, rapid analytical methods allowing point-in-time quantification to provide more representative results are essential to fully understand the relationship between THMs in swimming pool air and associated health risks.

Depending on the sample collection technique employed, various options exist for sample extraction. While direct injection of whole air samples is possible from canisters and bags (Aggazzotti et al., 1990), this often leads to lower detection limits and, as such, the use of a cryogenically cooled trap is employed to trap and concentrate target analytes (US EPA, 1999a). For samples collected on adsorbent materials, target analytes are desorbed either

thermally (Van Den Velde et al., 2007) or by the use of chemicals (NIOSH, 2003), with methods generally involving the use of a cryogenically cooled trap. While validated for the analysis of some THMs, many methods involve expensive specialised equipment (e.g. cryogenic trap or thermal desorption unit) and/or the use of harsh chemicals for solvent desorption (e.g. carbon disulfide, pentane or 3-phenoxybenzyl alcohol). Overcoming many of the issues involved with other techniques, one method combines sampling and sample preparation into one step by employing solid-phase microextraction (SPME) to automatically extract, concentrate and desorb target analytes (Sa et al., 2011).

Here, we describe a simple SPME GC-MS analytical method for the analysis of THMs (trichloromethane, bromodichloromethane, dibromochloromethane and tribromomethane) in ambient air which has been validated for swimming pool air. Improvements over previous methods include point-in-time sampling, providing more representative results, minimisation of sample contamination and the use of specialised sampling equipment, and elimination of the need for harsh chemicals. Furthermore, in the new method, a significant decrease in instrumental runtime was achieved, compared to previous methods. Other improvements were achieved by employing a surrogate standard to correct for analyte losses and variation in chromatographic performance; minimising the impact of residual solvent during extraction; and employing a more optimal extraction temperature.

3.2.3. Methodology

3.2.3.1. Analytical Standards and Reagents

All reagents were of analytical grade purity (>98%). Trichloromethane (chloroform), bromodichloromethane, dibromochloromethane, tribromomethane (bromoform) and 1,2-dibromopropane (1,2-DBP) were purchased as neat compounds from Sigma-Aldrich (Sydney Australia). 1,2-Dibromopropane-*d6* (1,2-DBP-*d6*) was purchased as a neat compound from CDN Isotopes (Quebec, Canada). Methanol was purchased from Sigma-Aldrich (Sydney, Australia).

3.2.3.2. Preparation of Standard Solutions and Air Calibration Standards

Separate THM (containing all four THMs), internal standard (1,2-DBP) and surrogate standard (1,2-DBP-*d6*) stock solutions (1 g L^{-1}) were prepared in methanol. Several working solutions containing both THMs (varying concentrations) and surrogate standard (25 µg L^{-1}) were prepared by dilution of stock solutions into methanol. Stock and working solutions were prepared each month and week, respectively. Calibration standards in air containing the four THMs (2 to 5000 µg m^{-3}) and the surrogate standard (25 µg m^{-3}) were prepared by adding the appropriate working solution (1 µL) into a capped, nitrogen flushed amber vial (20 mL) via

injection through the septum. Samples/standards were fortified with internal standard (final concentration of 25 µg m^{-3}, added as 1 µL in methanol) prior to GC analysis. The advantages and applicability of the standard addition method for calibration for air analysis using glass vials and SPME has previously been demonstrated (Baimatova et al., 2016).

3.2.3.3. Optimised Analytical Method

All analyses were performed on an Agilent 6890N gas chromatograph coupled with a 5975 mass selective detector (MSD) running in electron ionisation (EI) mode (70 eV) under the following conditions: MS Quad: 150 °C; MS source: 230 °C; and MSD transfer: 230 °C. Extraction and desorption of samples were carried out by SPME using a Gerstel MPS2 automatic sampler fitted with a 2 cm 50/30 µm divinylbenzene/Carboxen/polydimethylsiloxane (DVB/CAR/PDMS) fibre (Sigma-Aldrich). Samples for analysis of THMs in air were incubated (6 mins at 70 °C) with agitation (750 rpm) to ensure complete volatilisation of the calibration solution containing the four THMs and the surrogate standard, prior to SPME extraction for 15 mins at 70 °C. Target analytes were desorbed from the SPME fibre in splitless mode under the following conditions: desorption time: 10 mins; desorption temperature: 190 °C; purge time: 1 min; purge flow rate: 55 mL min^{-1}. GC separation was carried out on a Phenomex ZB-5MS column (30 m x 250 µm i.d. and 1 µm film thickness) with helium as the carrier gas (flow rate: 1.1 mL min^{-1}). The oven temperature conditions were as follows: 40 °C held for 5 mins, heated to 160 °C at 8 °C min^{-1}, heated to 300 °C at 25 °C min^{-1} and held at 300 °C for 5 mins (total time 26 mins). Selective ion monitoring (SIM) was used for analyte identification and quantification using mass-to-charge (m/z) ratios provided in **Table A3-2**.

3.2.3.4. Optimisation of the Analytical Method

A range of analytical parameters, including oven temperature program, solvent volume, incubation time, vial agitation, extraction time and temperature, as well as desorption time and temperature, were investigated. This was achieved by systematically changing conditions and comparing the chromatographic response of each analyte corrected by normalisation with the surrogate standard response (analyte peak area/surrogate standard peak area). A THM calibration standard (1 µL in methanol) containing each of the four THMs and the surrogate standard was added to a capped, nitrogen flushed amber vial to achieve a concentration of 20 and 25 µg m^{-3} for each THM and the surrogate standard, respectively. Solvent volume was investigated by adding additional methanol. The highest resolution chromatography (baseline separation) and normalised response ratios when considering all analytes were used to select the optimum condition for each analytical parameter.

3.2.3.5. Pool Air Sample Collection

Swimming pool air samples were collected, (on one occasion, in duplicate), 30 cm above the surface of the water and 50 cm from the edge of the pool, from a range fifteen indoor swimming pools located in Perth, Western Australia. Further details are provided in **Section 3.2.4.3**. A similar sampling approach to that of Baimatova et al. (2016), who validated sampling and calibration procedures using SPME glass vials, was adopted. In the current work, air samples were collected in capped, nitrogen flushed amber vials (glass, 20 mL) by flushing ambient air (3 x 60 mL) through the septum (18 mm, polytetrafluoroethylene (PTFE)/silicone) using a 60 mL disposable plastic syringe (**Figure 3.2-1**), allowing for point-in-time quantification while minimising sample cross-contamination. Needles (23 ga; 70 x 0.63 mm) were of a size that allowed the septum to reseal after it had been pierced, avoiding leakage of the collected air sample. A surrogate standard (final concentration of 25 μg m^{-3} added as 1 μL in methanol) was added at the time of sample collection using a glass syringe, while an internal standard (final concentration of 25 μg m^{-3} added as 1 μL in methanol) was added just prior to GC analysis upon return to the laboratory. No outliers among the replicate measurements were evident, and as such, all obtained data was included in the occurrence study. To be consistent with previous studies, data was not corrected to standard dry air concentrations in this study.

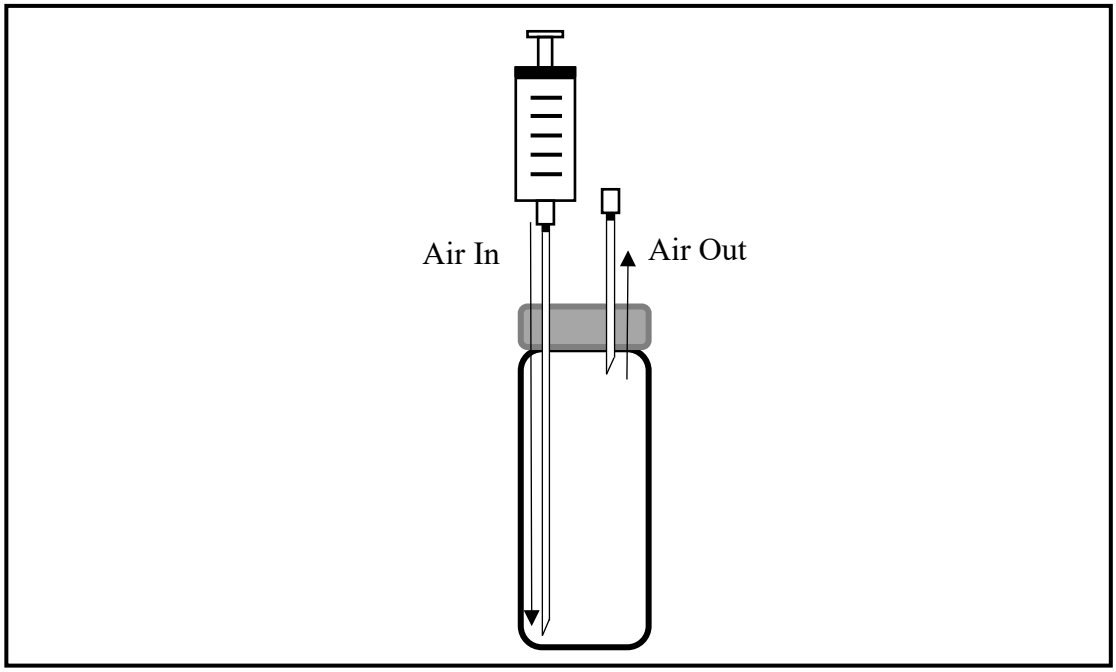

Figure 3.2-1: Sample collection apparatus. Amber vial (20 mL) fitted with an 18 mm, polytetrafluoroethylene (PTFE)/silicone septum and pre-flushed with nitrogen. Input needle, 23 ga (70 x 0.63 mm); output needle, 23 ga (10 x 0.63 mm). Disposable plastic syringe (60 mL) used for air collection/displacement.

3.2.4 Results and Discussion

3.2.4.1 Preservation of Analytes

The loss of THMs between sampling and analysis has previously been reported to be a significant problem in analysis of THMs in air using SPME (Sa et al., 2011). In the present work, the effect of storage time on the loss of THMs was therefore investigated periodically over 7 days by the use of nitrogen flushed vials fortified with THMs (100 μg m^{-3}) and stored at room temperature, which were fortified with internal standard (1,2-DBP, 25 μg m^{-3}) just prior to GC-MS analysis. THM response ratios (analyte peak area/internal standard peak area) obtained from the periodic analysis at any given time (t = n, where n= 1 to 168 hours (7 days)) were compared to initial (t = 0) THM response ratios. Decreasing response ratios were observed for all THMs with increasing storage time (**Figure A3-1**), with further decreasing response ratios also observed with increasing bromine substitution. A significant decrease (45 to 92%) in response ratios was observed for all THMs even after only 24 hours of storage, with dibromochloromethane and tribromomethane not detected after 2 days of storage. Minimal decreases in response ratios (6 to 8%) were observed for most THMs after 1 hour of storage, although a larger decrease (37%) in response ratios was observed for tribromomethane. Consistent with previous reports (Sa et al., 2011), these results suggest the analysis of THMs in air samples should be performed as soon as possible after sample collection.

The effect of storage temperature was investigated with the goal to find the optimal storage temperature to minimise loss of THMs. Here, nitrogen flushed vials fortified with THMs (100 μg m^{-3}) were stored at one of three storage temperatures, -25, 4 or 25 °C, and were periodically analysed over 7 days. Prior to GC-MS analysis, samples were fortified with internal standard (1,2-DBP, 25 μg m^{-3}) and, as before, THM response ratios measured were compared to those initially observed. For most THMs, higher response ratios (3 to 48%) were observed for samples stored at room temperature (25 °C) compared to those stored at 4 or -25 °C, at any given time (**Figure A3-2**). Unlike the trends observed for the other THMs, response ratios for tribromomethane, however, were generally higher (4 to 37%) for samples stored at -25 °C compared to the other storage temperatures, at any given time. As higher response ratios were observed for more THMs for samples stored at room temperature (25 °C), it is suggested that samples should be stored at room temperature. However, we also highlight that storage time was shown to have significant impact on the loss of THMs, and therefore analysis of air samples should be performed as soon as possible after collection.

While the decrease of THMs could not be minimised by adjusting storage temperature, the use of a surrogate standard was evaluated. A surrogate standard should have

similar chemical and physical properties to the target analyte(s), and mimic the behaviour of the target analyte(s) in the analytical. Here, a surrogate standard, 1,2-dibromopropane-*d6* (1,2-DBP-*d6*), was evaluated to correct for THM loss over sample holding time, i.e., the time between sample collection and GC-MS analysis. It is expected that the response of 1,2-DBP-*d6* would decrease in a similar way to the decrease in THM response observed, and hence, normalising the THM response with the response of the surrogate standard (if added at time of sample collection) would account for any THM analyte loss during holding time. To investigate, nitrogen flushed vials fortified with THMs and 1,2-DBP-*d6* (100 µg m^{-3}) stored at room temperature (25 °C) were periodically analysed over 7 days. Prior to GC-MS analysis, samples were fortified with internal standard (1,2-DBP, 25 µg m^{-3}) and THM/internal standard response ratios were compared to those initially observed. Response ratios for 1,2-DBP-*d6* were observed to decrease in a similar fashion to the response ratios of the THM analytes (**Figure A3-1**), hence, the use of 1,2-DBP-*d6* as a surrogate standard to be added at the time of sample collection minimises the effect of THM analyte loss over sample holding times on quantification. This use of a surrogate standard is a significant improvement over other SPME methods for analysis of THMs in air (Sa et al., 2011) where no efforts were made to account for analyte loss over holding times, hence, the developed method offers a significant improvement in accurate analyte quantification.

3.2.4.2. Optimisation of the Analytical Method

3.2.4.2.1. Chromatographic Performance

GC-MS parameters (MS Quadrupole, MS source, and MSD transfer line), as well as oven temperature programming, were selected based on those previously demonstrated to be suitable for the analysis of THMs in water (Allard et al., 2012). Initial tests (data not shown) reconfirmed their suitability for the analysis of THMs in air, with final conditions summarised in **Section 3.2.3.3**. These tests also indicated that residual solvent volume (methanol): i.e. the solvent remaining in vials after the addition of surrogate standard, internal standard or THM analyte working solutions (**Section 3.2.3.2**); may impact the recovery of analytes so this was investigated further. Flushed vials fortified with surrogate standard (1,2-DBP; final concentration of 25 µg m^{-3} added as 1 µL in methanol), with sequential volumes of methanol added (0 to 50 µL), were employed to test the impact of solvent volume on analyte response. These experiments showed that solvent volume had a significant impact on the response of the surrogate standard (**Figure A3-3**), which was found to decrease with increasing solvent volumes. For consistency, and to maximise analyte response, residual solvent volumes should be minimised and kept consistent between calibration standards and samples, hence a total volume of 2 µL (1 µL each for the addition of the surrogate standard and internal standard)

was chosen. Although the Sa et al. (2011) did not explicitly investigate the effect of solvent volume, limiting the impact of solvent volume in the current method is an improvement on the previous method (Sa et al., 2011), where larger and varying (5 to 50 μL) solvent volumes were employed.

Figure A3-4 shows the chromatographic response of the THMs, the surrogate standard and the internal standard recorded by employing the developed SPME GC-MS method. While all THM peaks were easily resolved from each other, co-elution exists between the internal and the surrogate standards. While use of a surrogate standard that has a different retention time to the analytes would result in a 'cleaner' TIC chromatogram, the surrogate and internal standards were selected for several strategic reasons. The inclusion of these standards did not add additional 'target ions' for monitoring during data acquisition, as these ions are also those monitored for THM analytes. The inclusion of additional 'target ions' different to those of THM analytes (which would occur when selecting a different internal/surrogate standard) would result in an overall lower sensitivity, which would consequently see a decrease in chromatographic and quantification performance, with an increase in detection limits also likely. Furthermore, the use of selective ion monitoring (SIM) mode in the developed method allows target ions unique to the surrogate and internal standards (at the given retention time) to be monitored. While peak overlap of the surrogate and internal standards is evident in the overall TIC chromatogram (**Figure A3-4(a)**), no overlap is observed for the corresponding target ions (those used for quantification), and quantification of THMs was therefore not hindered. As no negative effects were evident, method performance was prioritised over aesthetics with the choice of the surrogate standard. Slight tailing occurred for all analyte peaks, as well as for the peaks corresponding to the surrogate and internal standards. Peak tailing was also observed in the chromatograms obtained for the analysis of THMs in water (data not shown), although it was more pronounced in the chromatograms from air samples which is likely due to (i) the lower background noise observed in chromatograms from air samples compared to those from water samples, and (ii) the significant increase in analyte response ratios from air samples compared to from water samples due to the fewer equilibrium mechanisms involved in air (sample/fibre) compared to water (sample/headspace plus headspace/fibre) analysis. These observations are consistent with a previous study where peak tailing was observed in the chromatograms obtained from the analysis of THMs in water and air, employing another SPME method (Sa et al., 2011). In the current study, peak shape was found to have minimal impact on quantification of target analytes. Furthermore, the use of a surrogate standard minimises the impact of peak tailing on quantification. Hence, the peak shape has minimal negative effects on the performance of the developed method.

3.2.4.2.2. Fibre Selection and Extraction Conditions

Extraction conditions were evaluated to determine the optimum conditions for the analysis of THMs in air samples, including: fibre type, incubation time (pre-warming of vial), sample agitation, extraction time and extraction temperature. Although maximum analyte response ratios were observed once equilibrium is reached, it is possible to quantify analytes at non-equilibrium conditions due to the proportional relationship that exists between the analyte concentration in the sample and that extracted by the SPME fibre, at any given time (Ai, 1997). For this reason, optimum conditions were chosen which gave satisfactory analyte response ratios for all four THMs, largely under non-equilibrium conditions, and which minimised overall instrumental run time.

While the authors offered no explanation, compared to those obtained when a 100 µm-PDMS fibre was employed, greater responses for all THMs were reported when a 75 µm-CAR/PDMS fibre was used, for the analysis of THMs in ambient air (Sa et al., 2011). These differences are potentially due to the low THM sorption capacity of the non-polar PDMS coating, as previously reported (Allard et al., 2012). A DVB/CAR/PDMS coated fibre has previously been demonstrated to be more suitable (compared to a CAR/PDMS coated fibre) for the analysis of THMs in water by SPME (Allard et al., 2012) and was chosen for this investigation. Extractions of nitrogen flushed vials containing THMs (20 µg m^{-3}) and surrogate standard (25 µg m^{-3}) were carried out using both 1 and 2 cm DVB/CAR/PDMS fibres. Higher analyte response ratios were observed (data not presented) for a fibre length of 2 cm compared to 1 cm, due to the higher sorption capacity of the 2 cm fibre. To minimise the effects of competitive adsorption and hence increase response ratios, hence a 2 cm DVB/CAR/PDMS was chosen.

Increasing sample incubation time resulted in an increase in response ratios for most THMs (**Figure 3.2-2(a)**), which was likely due to the equilibrium transfer of analytes from the air phase to the fibre phase being more complete, although a decrease in response ratios was observed for tribromomethane, which increased with increasing incubation time. To minimise overall instrumental runtime and increase the sensitivity of the method towards tribromomethane, as lower concentrations of tribromomethane are expected in real samples (Carter and Joll, 2017), an intermediate incubation time of 6 minutes was chosen.

Sample agitation primarily affects the transportation of analytes from the water-to-headspace by increasing the rate of transfer from water to headspace (Kolb, 2006; Pawliszyn, 1997). Although this is not applicable in the analysis of air samples, an increase in the response ratios for all THMs was observed with increasing agitation rate (**Figure 3.2-2(b)**). This may be due to the equilibrium transportation of THM analytes to the headspace from the residual

solvent (used to fortify test samples), where an increase in agitation rate should lead to an increase in THM transfer. For the analysis of real samples, this equilibrium exists between the residual solvent from the added surrogate standard and the sample, and so the highest agitation rate of 700 rpm was chosen to maximise response ratios of THMs.

Increasing extraction temperature was shown to increase the extraction efficiency of higher molecular weight analytes (such as tribromomethane) in the analysis of water samples, as increasing temperature generally increases the volatility and hence transfer of these compounds to the headspace (Kolb, 2006; Pawliszyn, 1997). In the developed method, extraction temperature was found to impact the response ratios for all THMs (**Figure 3.2-2(c)**). Response ratios for all THMs were observed to increase with increasing extraction temperatures (30 to 70 °C), but a noticeable decrease in response ratios was observed at temperatures above 70 °C. These results suggest that, although no water-headspace equilibrium exists, extraction temperature still influences the extraction efficiency of THMs in air samples. In a previous study of analysis of THMs in the ambient air of indoor swimming pools, Sa et al. (2011) used an extraction temperature of 30 °C (without investigating a range of extraction temperatures) because it was the ambient temperature at the site of the pool. However, an extraction temperature of 30 °C has been found in the current study to afford the poorest analyte response ratios. Our study has shown that extraction temperature is in fact an important parameter that needs to be optimised and controlled in the SPME analysis of air samples. In order to maximise analyte response ratios, an extraction temperature of 70 °C was chosen for this method.

Increasing extraction time resulted in an increase in analyte response ratios for trichloromethane, bromodichloromethane and dibromochloromethane (**Figure 3.2-2(d)**), which is likely to be due to a more complete equilibrium being reached. Unlike the other THMs where linear increases in response were observed, no significant change in response was observed for tribromomethane for increasing extraction time. This is likely due to a combination of (i) the limited sorption capacity of the fibre coating, and (ii) the larger molecular size average diameter of tribromomethane compared to the other THMs. While an increase in extraction time led to an increase in response for most THMs (more complete equilibrium), a compromise is required between attainment of equilibrium and analysis time. As complete equilibrium is not required (Ai, 1997) and sufficient response ratios were observed for all analytes, an intermediate extraction time of 15 minutes was chosen. The choice of this extraction time allows for a significantly higher analysis rate (over 3x faster) compared to the method of Sa et al. (2011) where an extraction time of 50 minutes was used. Minimisation of analysis time is particularly important considering the rapid loss of THMs over time observed in this study (**Section 3.2.4.1**).

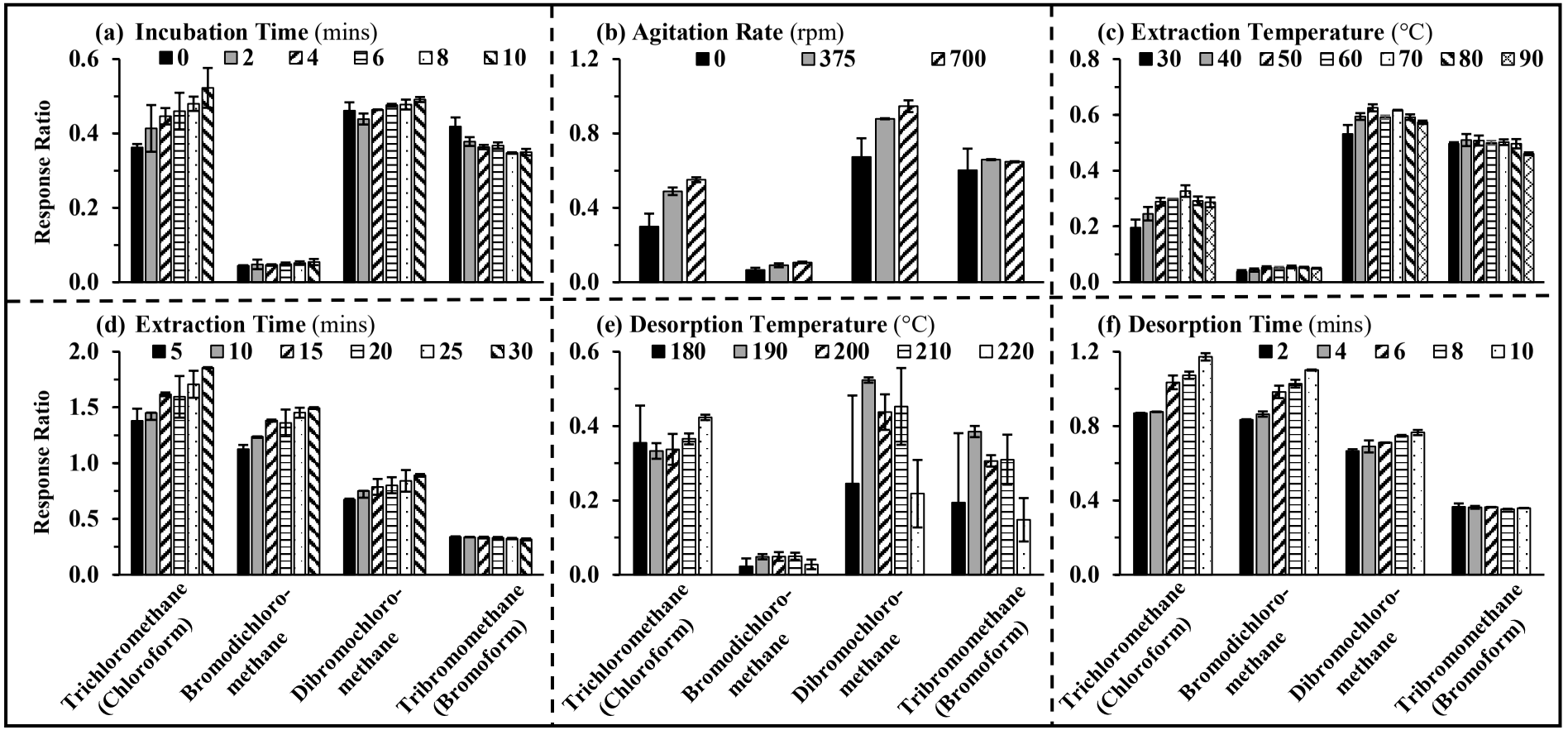

Figure 3.2-2: Summary of results obtained during optimisation of extraction (**(a)** to (**d)**) and desorption (**(e)** and (**f)**) conditions. Samples were prepared in vials (20 mL) that were pre-flushed with nitrogen, containing all four THMs and surrogate standard (final concentration of 200 µg m^{-3} each, added as 1 µL in methanol), and internal standard (final concentration of 200 µg m^{-3}, added as 1 µL in methanol). Conditions were evaluated by systematically altering the tested condition and comparing response ratios (analyte response/surrogate standard response).

3.2.4.2.3. Desorption Conditions

In SPME analysis, analytes are thermally desorbed from the SPME fibre into the GC injection port, in which they volatilise and are separated in the GC column (Kolb, 2006; Pawliszyn, 1997). As with extraction, phase transportation equilibria exist during desorption: mainly the fibre-to-injection port, which is driven by temperature. Furthermore, desorption time and sample volatilisation is critical to achieve maximum sensitivity and ensure analytes are fully desorbed from the fibre. However, excessive desorption temperatures can result in thermal decomposition of the analyte, especially for thermally labile analytes.

Desorption temperatures between 180 °C and 220 °C were chosen for evaluation, as these have been used in previously published methods (**Figure A3-1**). Excluding trichloromethane, THM response ratios generally decreased with increasing desorption temperatures above 190 °C (**Figure 3.2-2(e)**). The uncertainties associated with the response ratios for the four THMs in the 180, 210 and 220 °C desorption temperature tests were significant. The reasons for these large uncertainties are unknown, however the uncertainties associated with the response ratios for the four THMs in the 190 and 200 °C tests were much smaller, indicating repeatable analysis for each of the THMs. Based on the observations in **Figure 3.2-2**, of all the variables tested, desorption temperature appeared to have the greatest influence on the repeatability of the analysis, and so 190 °C was chosen as the desorption temperature to ensure maximum repeatability and operational longevity. While only one other SPME method for the analysis of THMs in air has been reported, a method which uses a slightly higher optimum desorption temperature of 200 °C (Sa et al., 2011), the selected desorption temperature for the developed method (190 °C) is comparable to injector temperatures reported for other techniques, 150 to 220 °C (Batjer et al., 1980; Catto et al., 2012; Chen et al., 2016; Lindstrom et al., 1997).

Desorption times of 2 to 10 minutes were investigated. Although similar response ratios were observed for tribromomethane over all desorption times, an increase in desorption time led to an increase in response ratios for the other THMs (**Figure 3.2-2(f)**). To maximise response ratios and minimise contamination via carryover (i.e., to ensure analytes were completely removed from the fibre), a desorption time of 10 minutes was chosen. A desorption time of 10 minutes was also selected in the only other known SPME method for THM analysis in air (Sa et al., 2011).

3.2.4.3. Method Validation

3.2.4.3.1. Linearity, Limits of Detection and Precision

Table 3.2-1: Calibration and method validation data for the developed analytical method. Limits of detection were calculated based on ten replicates (n=10) of a low standard (2 μg m^{-3}), as defined by the Environmental Protection Agency (US EPA, 2014). Bias was determined by the analysis of swimming pool air samples (n=3) fortified with THMs (20 μg m^{-3}) and is expressed as the average recoveries obtained. Repeatability was evaluated by calculating the percent relative standard deviation (%RSD) for five samples analysed over one day, while reproducibility was evaluated by calculating the %RSD for fifteen samples analysed over three days (within one week). All errors are reported as standard deviations. All quantification was via external calibration.

Analyte	Correlation Coefficient (r^2)	Detection Limit (μg m^{-3})	Repeatability RSD (%)	Reproducibility RSD (%)	Recovery (%)
Trichloromethane	0.9941	2.65	11	16	112 ± 22
Bromodichloromethane	0.9963	0.71	11	13	120 ± 13
Dibromochloromethane	0.9961	0.66	3	3	85 ± 5
Tribromomethane	0.9995	0.91	3	3	86 ± 3

The developed method was validated by evaluating the linearity, detection limits and precision for each THM (**Table 3.2-1**). Linearity was investigated by assessing the Pearson correlation coefficient (r^2) for a concentration range between 2 to 5000 μg m^{-3}, as concentrations within this range have previously been reported (Carter and Joll, 2017). All THMs showed excellent linearity (r^2=0.994 to 0.999) over the range investigated. Detection limits (calculated based on ten replicates (n=10) of a low standard (2 μg m^{-3}), as defined by the Environmental Protection Agency (US EPA, 2014)) were slightly higher for trichloromethane (2.6 μg m^{-3}) compared to the other THMs (0.7 to 0.9 μg m^{-3}); the detection limits for the latter three THMs are within the range achieved by other methods for the analysis of THMs in air samples, but similar to or less than those achieved by another SPME method for the analysis of THMs in air samples (1.3 to 2.5 μg m^{-3} (Sa et al., 2011)). The precision of the developed method was evaluated by determining the reproducibility and repeatability of the method. Repeatability was evaluated by calculating the percent relative standard deviation (%RSD) for five samples analysed over one day, while reproducibility was evaluated by calculating the %RSD for fifteen samples analysed over three days (within one week). Excellent repeatability and reproducibility were achieved for dibromochloromethane and tribromomethane (RSD=3%), with good repeatability and reproducibility achieved for bromodichloromethane and trichloromethane (RSD=11 to 16%; **Table 3.2-1**). In comparison to another reported SPME method (Sa et al., 2011), the developed method has demonstrated similar or better repeatability (RSD=5 to 10%, (Sa et al., 2011)) and significantly better

reproducibility (RSD=15 to 25%, (Sa et al., 2011)). The good to excellent precision of the current method demonstrates its excellent performance.

3.2.4.3.2. Validation of Method for Swimming Pool Air

Swimming pool air samples fortified with THMs (20 µg m^{-3}) were evaluated to assess the applicability (bias) of the method to the swimming pool environment, with chromatograms obtained for swimming pool air and fortified swimming pool air displayed in **Figures A3-4(b)** and **(c)**. Good recoveries (85 to 120%; **Table 3.2-1**) were achieved for all THMs, indicating the method is reliable and free from interferences (matrix effects) for this application.

3.2.4.4. Occurrence of Trihalomethanes in the Ambient Air of Indoor Swimming Pool Complexes

This study is the first known report of THMs in the ambient air above swimming pool waters in Australia. A total of fifteen pools (3 leisure pools, 5 lap pools (3 x 25m and 2 x 50m), 4 spa pools, 2 hydrotherapy pools and a pool for emergency evacuation training) across five indoor swimming pool facilities were investigated. Fourteen pools were treated with chlorine (chlorine gas or sodium hypochlorite), with ten of these also employing ultraviolet irradiation (UV) secondary treatment. The emergency evacuation training pool was treated with bromochlorodimethylhydantoin (BCDMH). The occurrence of THMs in the ambient air above these pools is summarised in **Figure 3.2-3**. While some studies may have reported higher concentrations (summarised by Carter and Joll (2017)), for the purpose of this paper, comparisons here are drawn only between studies investigating pools of similar characteristics, i.e. filled with fresh water, and studies that do not provide all details (pool type and/or treatment method) have been excluded.

In the air above most investigated pools, trichloromethane was measured at concentrations between 28 and 368 µg m^{-3}, which is consistent with concentrations reported in most previous studies (12 to 320 µg m^{-3}, as summarised by Carter and Joll (2017)), but generally higher than those reported in several other studies (12 to 81 µg m^{-3}) (Font-Ribera et al., 2010; Lourencetti et al., 2012; Richardson et al., 2010; Thiriat et al., 2009; Westerlund et al., 2018). While some studies have reported higher trichloromethane concentrations in the ambient air of indoor pool complexes (maximum concentrations between 477 and 853 µg m^{-3}) (Aggazzotti et al., 1995, 1990; Benoit and Jackson, 1987), in some cases even higher trichloromethane concentrations (up to 6490 µg m^{-3}) were measured in the air above three pools at one facility in the current study. Although not specifically investigated, these unusually high trichloromethane concentrations were likely due to the fact that these pools had recently been covered by pool blankets. Compared to the other investigated pools, higher concentrations (31 to 174 µg m^{-3}) of bromodichloromethane were also reported above these

three pools at this facility, with only a few other studies reporting comparable concentrations (39 to 155 μg m^{-3}) (Benoit and Jackson, 1987; Nitter et al., 2017; Tardif et al., 2016a, 2015).

In the air above other chlorinated pools in this study, bromodichloromethane was measured at concentrations of 3.7 to 15 μg m^{-3}, which are generally similar to those reported in most other studies, up to 26 μg m^{-3} (as summarised by Carter and Joll (2017)). Dibromochloromethane was the only THM not detected above all pools investigated, where concentrations of 0.8 to 8.5 μg m^{-3} were measured above 12 of the 14 chlorinated pools, but dibromochloromethane was below its detection limit (i.e. <0.7 μg m^{-3}) in the air above one spa and one lap pool located at different facilities. While significantly higher concentrations (95 and 205 μg m^{-3}) have been previously reported, (Lahl et al., 1981; Tardif et al., 2015) dibromochloromethane concentrations measured in this study are generally similar to most other reported studies (Aggazzotti et al., 1998; Caro and Gallego, 2008; Catto et al., 2012; Font-Ribera et al., 2010; Lourencetti et al., 2012; Richardson et al., 2010; Tardif et al., 2016b). Tribromomethane was detected in the air above all chlorinated pools at concentrations of 3.4 to 36 μg m^{-3}, which are similar to most other studies, as summarised by Carter and Joll (2017), although two studies (Nitter et al., 2017; Tardif et al., 2016a) have reported significantly higher tribromomethane concentrations (103 and 319 μg m^{-3}) in the air above chlorinated pools, with another study reporting tribromomethane at concentrations up to 1910 μg m^{-3} above a brominated spa (Benoit and Jackson, 1987).

Generally, concentrations of the more bromine substituted THMs (e.g. tribromomethane and dibromochloromethane) were higher in the ambient air above the training pool (treated with BCDMH) compared to those measured in the ambient air above the other investigated pools (treated with chlorine), which likely reflects the predominant availability of bromine over chlorine, and hence formation of brominated THMs, associated with the use of BCDMH as a disinfectant. All THMs were detected in the air above the BCDMH treated evacuation training pool, where concentrations of 28, 5.0, 4.9 and 53 μg m^{-3} were measured for trichloro-, bromodichloro-, dibromochloro- and tribromo-methane, respectively. Very limited data exists for THMs in the air above pools treated with bromine (i.e. BCDMH). Excluding trichloromethane where significantly higher concentrations were measured here compared to those previously reported (1.7 to 9.4 μg m^{-3}) (Lourencetti et al., 2012; Richardson et al., 2010). THM concentrations measured in the current study were either similar to, or lower than concentrations previously reported in the air above bromine treated pools, up to 4.8, 9.7 and 101 μg m^{-3} for bromodichloro-, dibromochloro- and tribromochloro-methane, respectively (Lourencetti et al., 2012; Richardson et al., 2010). Significantly higher tribromomethane concentrations, up to 1910 μg m^{-3}, have been reported (Benoit and Jackson, 1987), although this was in the air above a brominated spa.

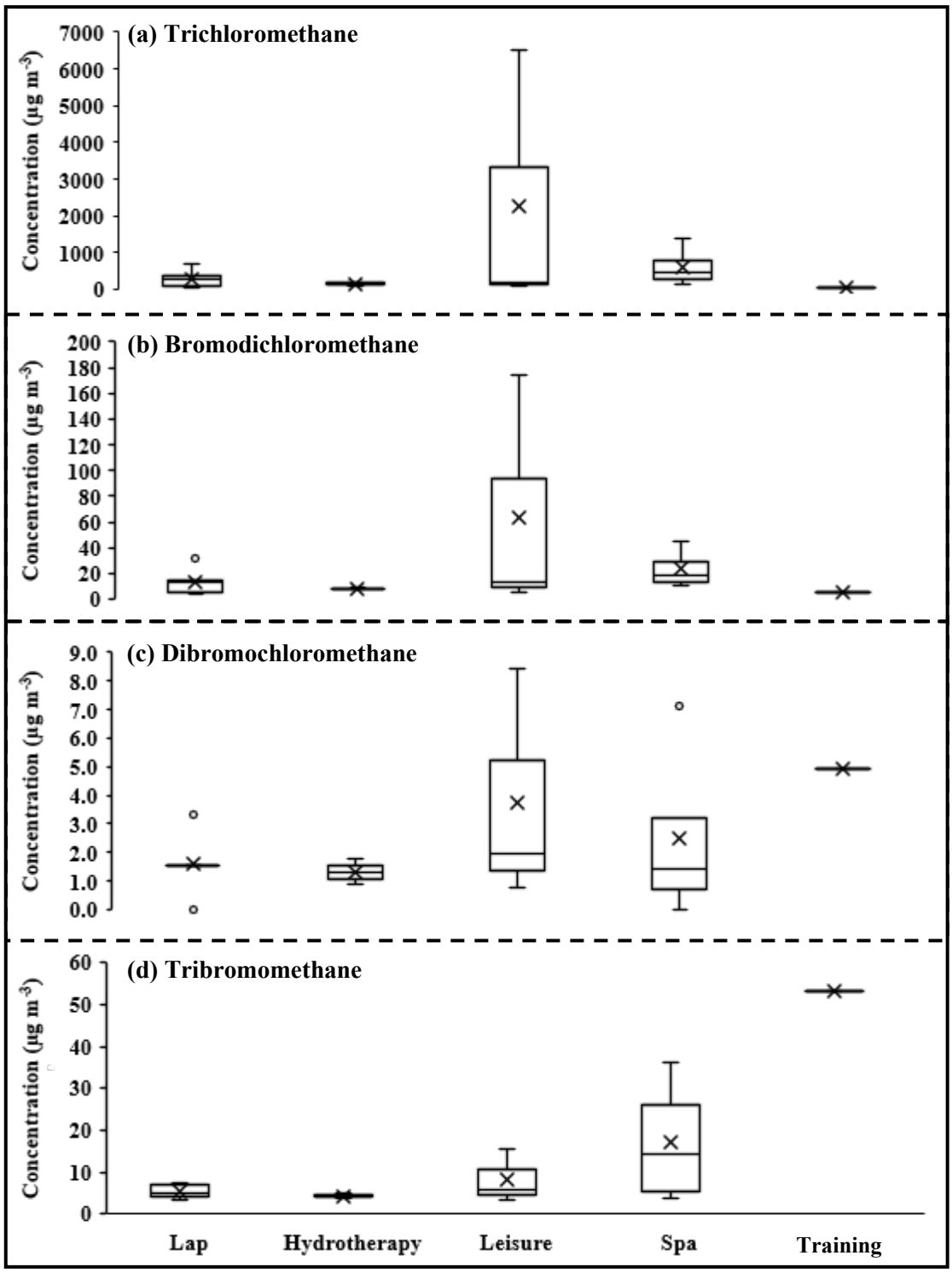

Figure 3.2-3: Occurrence of **(a)** trichloromethane, **(b)** bromodichloromethane, **(c)** dibromochloromethane and **(d)** tribromomethane in the ambient air above various pools at several indoor swimming pool complexes. Fourteen chlorinated pools (5 lap pools (3 x 25m and 2 x 50m), 2 hydrotherapy pools, 3 leisure pools and 4 spa pools) and a pool for emergency evacuation training using bromochlorodimethylhydantoin (BCDMH) for disinfection (training pool), across five indoor swimming pool facilities were investigated. Results are an average of duplicate analyses (n=2).

Concentrations of the THMs in the ambient air above some pools in this study are higher than those previously reported in other countries (Carter and Joll, 2017), which may be due to the relatively high disinfectant residuals employed in pools in Australia (minimum 2 to 4 mg L^{-1} chlorine). Although definite conclusions regarding the health impacts of exposure to THMs in the ambient air above pools are yet to be drawn, THMs in the ambient air at concentrations comparable (or lower) to those measured in the current study have been reported to induce negative health effects, e.g. cough, nose and throat irritation, and other respiratory issues (Carter and Joll, 2017). With the health effects associated with exposure to THMs in pool air still largely unclear, reliable detection and quantification of THMs in pool air is key, highlighting the importance of the developed analytical method.

3.2.5. Conclusions

A simple method that minimises chemical usage for the analysis of THMs in air has been demonstrated. Samples are instantly collected, via air displacement, directly into GC-compatible vials, allowing point-in-time quantification and minimising sample contamination. Storage time was found to have a significant impact on the loss of THMs in air samples, while the impact of storage temperature was found to be less significant. The use of a surrogate standard added at the time of sample collection is employed in order to minimise any negative effect of, and account for, analyte loss in real samples over the time between collection and analysis. The SPME GC-MS parameters were optimised including incubation (6 mins at 70 °C), extraction (15 mins at 70 °C with a 2 cm DVB/CAR/PDMS fibre) and desorption conditions (10 mins at 190 °C). The developed method achieves low detection limits (0.7 to 2.6 μg m^{-3}) and allows quantification of THMs at concentrations up to approximately 5000 μg m^{-3}, typical of concentrations in real-world samples. Excellent repeatability (3 to 11% RSD) and reproducibility (3 to 16% RSD) was demonstrated and the method has been validated for swimming pool air. The developed method was used to investigate the occurrence of THMs in the air above fifteen swimming pools, where concentrations of up to 6500 μg m^{-3} were measured for trichloromethane. The developed method can therefore be used to quantify THMs in swimming pool air more broadly, allowing improved understanding of exposure and health risks associated with THMs in the air above swimming pools.

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CHAPTER 4

OCCURRENCE OF DISINFECTION BY-PRODUCTS IN SWIMMING POOLS AND THE ESTIMATED RESULTING CYTOTOXICITY

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I, Rhys Aaron Ainsley Carter, contributed to the publication by: undertaking a review of the current literature in this field, undertaking all field work, laboratory experiments and data analysis, being the primary writer (including creating figures and tables), and editing and finalising the manuscript.

I, as a Co-Author, endorse that this level of contribution by the candidate indicated above is appropriate.

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4.1. Abstract

Swimming pools are disinfected to protect against the risk of microbial disease, however, the formation of disinfection by-products (DBPs) is an unwanted consequence. While many studies have reported the occurrence of commonly investigated DBPs (trihalomethanes and haloacetic acids) in pools, few studies have investigated more emerging DBP classes, such as the haloketones or haloacetaldehydes, nor the nitrogenous DBP classes of the haloacetamides, halonitromethanes, haloacetonitriles and *N*-nitrosamines. This study investigated the occurrence of sixty-four DBPs from the eight aforementioned DBP classes, in various types of swimming pools employing different treatment methods. Approximately 70% of the investigated DBPs were detected in at least one of the investigated pools, where most concentrations in this study were equal to or greater than those previously reported. Chloral hydrate (trichloroacetaldehyde) was one of many DBPs detected in all chlorinated waters (202 to 1313 μg L^{-1}), and, on a molar basis, was the predominant DBP measured in this study. Several other DBPs, namely chloroacetic acid, dichloroacetic acid, trichloroacetic acid, dichloroacetamide, dibromoacetamide, dibromochloroacetamide and trichloroacetamide, and many of the investigated *N*-nitrosamines were measured at concentrations greater than previously reported: up to 200 to 479 μg L^{-1} for the haloacetic acids, 56 to 736 μg L^{-1} for the haloacetamides and up to 1093 ng L^{-1} for some *N*-nitrosamines. The higher disinfectant residuals required to be employed in Australian pools, and poor pool management (e.g. of chlorine residual and pH) are likely factors contributing to these relatively high DBP concentrations. Where possible, the cytotoxicity values of the investigated DBPs were evaluated, resulting in chloral hydrate representing over 90% of the total chronic cytotoxicity despite only representing up to 64% of the total molar DBP concentration. This study is the first known report of bromodichloroacetaldehyde and bromochloroacetaldehyde in swimming pools and is the first known investigation of *N*-nitrosamines in a brominated pool. Furthermore, this work aids in understanding DBPs in both chlorine and bromine treated pools, the latter being the subject of only limited previous studies.

4.2. Introduction

While disinfection is essential to protect against the risk of microbial disease, the reaction between added disinfectants and organic matter contained in water leads to the unwanted formation of disinfection by-products (DBPs). Although over 700 DBPs have been identified in drinking, waste and recycled waters (Plewa and Richardson, 2017), less is known about DBPs in the swimming pool environment. Two recent reviews (Carter and Joll, 2017; Manasfi et al., 2017) provide a thorough summary of DBPs in swimming pools, summarising the occurrence, formation and potential health impacts of DBPs in this unique environment.

These reviews highlight two important trends in relation to research on DBPs in pools: (i) that most studies have focused on investigating trihalomethanes (THMs), haloacetic acids (HAAs) or inorganic halamines, with fewer studies of haloacetonitriles (HANs), and (ii) very limited information exists for several other classes of DBPs, e.g. the halonitromethanes (HNMs), haloacetamides (HAAms), haloketones (HKs), haloacetaldehydes (HALs), cyanogen halides and *N*-nitrosamines. Richardson et al. (2010) identified over 100 DBPs previously not reported in either drinking or swimming pool waters in a comprehensive investigation of several chlorinated and brominated swimming pools, where many of these DBPs were found to be nitrogenous. Furthermore, Daiber et al. (2016) identified over 100 DBPs in their study of several pools and spas where disinfection was achieved by either bromine or chlorine. Such studies highlight significant knowledge gaps regarding DBPs in swimming pool waters, warranting further investigation. In the same study, Daiber et al. (2016) reported the increased concentrations of DBPs in swimming pool and spa waters, where, in comparison to those measured in the respective filling waters, a 610% and 900% increase in total DBP molar concentration was observed for pools and spas, respectively. This increase is likely due to the higher availability of disinfectants (continually dosed) and organic matter (continual input from swimmers) in pools compared to filling waters, highlighting that DBP formation in swimming pools can be a magnified issue compared to other disinfected waters (e.g. drinking waters).

The most commonly used disinfectant in swimming pools is chlorine, either in gas (Cl_2) or solid (sodium/calcium hypochlorite) form. Other pool disinfectants include chloroisocyanurates (and their acid counterparts), chlorine dioxide, bromine (usually sodium bromide in combination with an oxidant such as ozone), bromochlorodimethylhydantoin (BCDMH) and electrochemically generated mixed oxidant (Montgomery, 1985). Although not exclusive, the use of chlorine based or bromine based disinfectants generally lead to the dominance of chlorinated and brominated DBPs, respectively. With many of the existing studies primarily focused on chlorinated pools, less is known about DBPs in those treated with bromine, where only six known studies exist (Daiber et al., 2016; Kelsall and Sim, 2001; Lourencetti et al., 2012; Norin and Renberg, 1980; Plewa et al., 2011; Richardson et al., 2010). Ultraviolet irradiation (UV) is commonly employed in swimming pools as a secondary treatment, and while conflicting results have been reported (e.g. as summarised by Carter and Joll, 2017), its use has been shown to impact DBP formation (Cheema et al., 2017; Hansen et al., 2013b; Spiliotopoulou et al., 2015). Furthermore, in laboratory studies modelling pools, disinfectant dose has been shown to impact DBP formation, where a higher disinfectant concentration has led to a higher formation of DBPs (e.g. Li and Blatchley, 2007; Manasfi et al., 2015). While studies have investigated the impact of disinfectants and/or secondary

treatments on DBPs in laboratory experiments (Carter and Joll, 2017), further investigations in real swimming pools are required, particularly for those treated with bromine or where higher disinfectant residuals are employed (e.g. in Australia or in the USA).

Many DBPs have been demonstrated to be cytotoxic, neurotoxic, genotoxic, with several additionally demonstrating mutagenic, carcinogenic and/or teratogenic nature (Richardson et al., 2007). Furthermore, nitrogenous DBPs (N-DBPs) have been shown to be generally more genotoxic (to Chinese hamster ovary cells) compared to most other DBPs (Richardson et al., 2015). While these studies provide an insight to the health impact of DBPs, they are generally based on the risk associated with ingestion of drinking waters. As (i) quantity and frequency of ingesting swimming pool water is significantly less than that for drinking waters, and (ii) additional uptake mechanisms such as inhalation and dermal absorption are significant in pools (Carter and Joll, 2017), these health impact studies are therefore not directly applicable to assessing the risk associated with DBPs in the swimming pool environment. In comparison to their filling waters, swimming pool waters have shown increased genomic DNA damage effects to CHO cells (Liviac et al., 2010). Links between exposure to swimming pool waters and several health issues, e.g. kidney and liver issues, as well as cancer of the bladder, have been suggested (Villanueva et al., 2007; Villanueva and Font-Ribera, 2012). Exposure to DBPs in swimming pool air has been linked to several respiratory issues (Jacobs et al., 2007; Kaydos-Daniels et al., 2008), although conflicting results exist (Goodman and Hays, 2008).

This study expands on the limited knowledge of DBPs in the swimming pool environment, by investigating the occurrence of sixty-four individual DBPs (from eight different DBP classes), as well as several other general water quality parameters, in fifteen pools across six locations in Perth, the capital city of Western Australia. A range of pool types (lap, leisure, spa and hydrotherapy) and treatment methods (chlorine gas (Cl_2), chlorine gas in combination with UV (Cl_2/UV), sodium hypochlorite (NaOCl) and BCDMH) were investigated in order to assess the impact of pool type and treatment methods on DBP occurrence. Filling waters were investigated to assess their impact on the formation of DBPs in the pools. Correlations between DBP classes and other general water quality and/or operational parameters were examined. The chronic cytotoxicity was estimated via calculation for most of the investigated DBPs to indicate if any trends exist between pool type and/or treatment method, and DBP derived cytotoxic nature of pools.

4.3. Methodology

4.3.1. Analytical Standards and Reagents

All chemicals and reagents used were of analytical grade purity (>97%) and purchased from a range of suppliers including Sigma Aldrich (Sydney, Australia), CanSyn Chemical Corporation (Ontario, Canada), AccuStandard (Connecticut, USA), Thermo Fisher (Victoria, Australia) and CDN isotopes (Quebec, Canada). Ultrapure water, purified by an ELGA PURELAB Ultra purification system (18.2 MΩ-cm resistivity), was used in all experiments.

4.3.2. Preparation of Standard Solutions and Calibration Standards

DBP standard stock solutions (1 g L^{-1}) were prepared by weighing neat compounds into acetone. Solutions were prepared per DBP class, that is, one stock solution containing all individual DBPs of a particular class were prepared. Secondary solutions were prepared by dilution of relevant stock solution(s) into acetone. Calibration standards were prepared by fortifying ultrapure water samples with the desired DBPs, surrogate standard and internal standard as per individual method requirements (**Section 4.3.3**).

4.3.3. Analytical Methods and Quantification

Table A4-1 summarises the analytical methods employed in this study, where all analysis was performed within five days from sample collection. Free and total chlorine equivalent concentrations were measured using a Pocket Colorimeter (HACH; 5870000). Temperature, pH, conductivity and dissolved oxygen were measured using a portable multi-meter (HACH; HQ40D). DBPs (THMs including iodinated-THMs (I-THMs, 10), HAAs (9), HKs (5), HALs (7), HNMs (9), HANs (9), HAAms (7) and *N*-nitrosamines (8)) were analysed (after quenching the oxidant residual in the samples) using various extraction methods followed by gas chromatography-mass spectrometry (GC-MS). Non-purgeable organic carbon (NPOC) and total nitrogen (TN) were measured using high temperature catalytic combustion with non-dispersive infrared detection using a Shimadzu total organic carbon analyser (TOC-L) equipped with a total nitrogen measuring unit (TNM-L). Bromide (determined only in filling waters) was determined using ion chromatography after quenching the oxidant residual using sodium sulfite. Due to analytical issues, while detection of tribromoacetic acid was possible, quantification could not be carried out. Surrogate and internal standards were employed where possible, with DBP quantification based on response ratios (analyte response/surrogate standard response). All quantification was via external calibration(s) which were analysed in conjunction with corresponding samples. Excluding *N*-nitrosamines where a single analysis was performed, all samples were analysed in duplicate (n=2) with reported concentrations representing the average of the two results.

4.3.4. Water Samples

Samples from a range of pool water types (e.g. lap pool, leisure pool, hydrotherapy and spa), as well as their filling water (disinfected distributed drinking water used to fill the pools), were collected from six different indoor pool facilities located in Perth, Western Australia, with permission granted by the Department of Health (Western Australia). At any given facility, all pools were treated independently, that is, all pools existed as individual water bodies with independent circulation and treatment systems. In order to ensure confidentiality, samples have been de-identified by applying codes: letters (A to F) to represent the facility, followed by a number to represent the particular pool or water type (as per **Table A4-2**). Furthermore, samples A5, B3, C2, D3, E4 and F4 represent the filling water at the given facility. While the study aimed to investigate several pools of the same type/treatment method, only one facility using BCDMH was known to be available for sampling. Samples were collected at the centre of the longest poolside, from approximately 50 cm from the edge and 20 to 30 cm below the water's surface. Samples were collected directly into amber bottles so as to leave no headspace, the oxidant residual quenched at 110% of the total chlorine equivalent concentration (as per **Table A4-1**) and stored at 4 °C until analysis (within one to five days).

4.3.5. Cytotoxicity Evaluation

The chronic cytotoxicity of the investigated DBPs was evaluated based upon published C_{50} values (a measure of the minimum concentration of a particular compound that induces a 50% reduction in density of Chinese hamster ovary cells after 72 hours) (Wagner and Plewa, 2017). Following previously published calculations (e.g. Allard et al., 2015; Smith et al., 2010), the concentration (M) of each DBP was divided by its C_{50} value (M), resulting in a dimensionless cytotoxicity value. Finally, these results were multiplied by 10^6 to make the cytotoxicity value more readable. The purpose of this evaluation was not to provide a direct assessment of the human health impact of DBPs in pools, rather to allow a comparison between the unitless DBP-associated chronic cytotoxicity of pools differing in type and and/or treatment method, to indicate if any trends may exist between these parameters and DBP derived cytotoxic nature of pools. HKs, bromodichloroacetonitrile, dibromochloroacetonitrile and tribromoacetonitrile, as well as most of the investigated *N*-nitrosamines (excluding *N*-nitrosomorpholine; NMOR), were excluded from cytotoxicity evaluation as C_{50} values do not currently exist for these compounds.

4.3.6. Statistical Evaluation

Statistical analysis was performed using SPSS Statistics version 24 software (IBM, Armonk, New York). Spearman's rank correlation coefficient was used to evaluate, if any, correlations that may exist amongst the DBPs and other water quality parameters of the investigated swimming pools.

4.4. Results and Discussion

4.4.1. General Water Quality Parameters of Pools

Table 4-1 summarises some general water quality and operational parameters of the investigated swimming pool waters, with additional details provided in **Tables A4-2** and **A4-3**, respectively. In Western Australia, swimming pools and spa/hydrotherapy pools have different operating guidelines, which are also dependent on whether the disinfectant employed is chlorine or bromine based (Western Australian Department of Health, 2013). Slightly less than half (47%, 7/15) of the waters investigated did not meet their operational guidelines for pH: 7.2 to 7.8 and 7.2 to 8.0 for waters treated with chlorine and bromine, respectively. Free chlorine equivalent concentrations were found to vary greatly amongst the investigated waters (1.5 to 5.7 mg L^{-1}), where only half of the chlorinated pools/spas investigated met their local guidelines for free chlorine equivalents, a minimum of 2 and 3 mg L^{-1} for chlorinated pools and spas/hydrotherapy pools, respectively. Furthermore, the free chlorine equivalent concentration (1.5 mg L^{-1}) measured in the BCDMH treated pool (sample C1) was significantly lower than specified in local guidelines, where a minimum concentration of 4 mg L^{-1} should be maintained. One third (5/15) of the pools/spas contained combined chlorine levels at concentrations greater than the accepted guideline (must not exceed 30% of the measured free chlorine), with combined chlorine concentrations of up to 2.6 mg L^{-1} observed.

Studies reporting measured general water quality and operational parameters (e.g. disinfectant residuals and pH) in Australian swimming pools are currently limited. Of the five known Australian pool studies (Carter et al., 2015; Kelsall and Sim, 2001; Teo et al., 2016a, 2016b; Yeh et al., 2014), only three have reported such parameters (Carter et al., 2015; Kelsall and Sim, 2001; Yeh et al., 2014). In a study of twelve chlorinated Queensland pools, where multiple samples (52 in total) were analysed, Yeh et al. (2014) reported that "most pools met these (local guideline) requirements" for free chlorine equivalents, although 4 of the 12 pools were reported to contain, on average, concentrations lower than the recommended local guideline. In a Victorian study, Kelsall and Sim (2001) investigated three pools (distinguished by treatment type: bromine/ozone, chlorine/ozone and chlorine) over six weeks, where samples were collected weekly. While all samples collected from the bromine/ozone pool met

local guidelines for free chlorine equivalent concentrations, only half of the samples collected form the chlorine pool contained free chlorine equivalent concentrations that met their local guidelines, with one sample collected from the chlorine/ozone pool also below the recommended guideline value (Kelsall and Sim, 2001). The only other known study of pools to be conducted in Western Australia is our previous study (Carter et al., 2015), where of the four pools investigated (1 sample per pool), all met their local guideline for free chlorine equivalent concentrations. Of these known Australian studies (Carter et al., 2015; Kelsall and Sim, 2001; Yeh et al., 2014), combined chlorine concentrations were only available in our previous study (Carter et al., 2015), where only half of the pools (4 pools, sampled once) contained concentrations outside their local guidelines. While pH was not reported by Yeh et al. (2014), the pH measured in all samples of all pools investigated by Kelsall and Sim (2001) met local guidelines, however, in our previous study (Carter et al., 2015), pH values were outside local guidelines in 75% of samples analysed (4 pools, sampled once).

For comparison to Australian pools, three international studies have been selected for discussion here (Yang et al., 2018; Zare Afifi and Blatchley, 2015; Zhang et al., 2015). In their investigation of fourteen pools in China, Zhang et al. (2015) reported that only four met their local guideline for free chlorine equivalent concentrations, where five pools contained concentrations below, and five others above, this local guideline. Although all other pools were within acceptable limits, more than half (8/14) were found to have pH values higher than their local guideline (Zhang et al., 2015). Similar results were reported in a more recent Chinese study of 35 pools, where only five met local guidelines for free chlorine equivalent concentrations, with 20 and 10 pools found to contain concentrations lower and higher than the local guideline, respectively (Yang et al., 2018). Unlike Zhang et al. (2015), of the investigated pools, most (31/35) were found to meet their local pH guidelines (Yang et al., 2018). In a fourteen month study of a chlorinated pool in the USA, although both pH and combined chlorine concentrations were reported to "regularly exceed the recommended limit" (Zare Afifi and Blatchley, 2015), free chlorine equivalent concentrations were generally within acceptable limits, where only 23 samples (of the ~200 collected) contained concentrations outside their local guidelines.

While it is evident that pool operation and management, particularly regarding pH and disinfectant residuals, could be improved for the investigated facilities, results of this study suggest that Western Australian pools are operated similarly to both other Australian pools and those located internationally. This does not however indicate that poor pool management is acceptable, rather it highlights that an improvement in pool management worldwide is required, which would likely see an overall improvement in the chemical water quality of swimming pools.

Table 4-1: Summary of general water quality and operational parameters of the swimming pools and spas investigated. Values are presented as "measured value (facilities target value)". Values presented in *italics* represent those outside their local operational guidelines.

Pool Code	Pool Type	Disinfectant	Secondary Treatment	Chlorine Equivalent Concentrations		pH	Temperature (°C)	NPOC (mg L^{-1})	TN (mg L^{-1})
				Free (mg L^{-1})	Combined (mg L^{-1})				
A1	25 m Lap	Chlorine gas	UV	*1.7* (3.5)	0.4	*7.0*	26 (28)	4.2	9.8
A2	Hydrotherapy	Chlorine gas	UV	*2.7* (4.0)	0.7	*6.8*	33 (34)	6.9	2.6
A3	Leisure	Chlorine gas	UV	3.3 (3.5)	0.3	7.2	29 (31)	4.3	4.7
A4	Spa	Chlorine gas	UV	3.0 (4.0)	0.2	7.3	34 (34)	3.1	2.7
B1	Hydrotherapy	Chlorine gas	UV	*2.8* (4.0)	0.5	7.4	32 (34)	8.9	16
B2	25 m Lap	Chlorine gas	-	3.1 (3.5)	*1.1*	*6.9*	28 (31)	10	-*
C1	Training Pool	BCDMH	-	*1.5* (4.0)	0.2	7.4	27 (28)	87	40
D1	50 m Lap	Chlorine gas	UV	*1.8* (2.2-5)	*1.5*	7.2	27 (27)	15	11
D2	Spa	Chlorine gas	UV	*1.5* (2.2-5)	2.0	*6.5*	33 (36)	28	8.6
E1	50 m Lap	Chlorine gas	-	5.2 (3.5)	0.8	7.4	28 (28)	7.4	1.7
E2	Leisure	Chlorine gas	-	5.7 (3.5)	*2.1*	7.7	30 (32)	14	2.5
E3	Spa	Chlorine gas	-	3.5 (4.0)	*2.6*	*8.0*	34 (36)	29	4.0
F1	25 m Lap	Sodium Hypochlorite	UV	2.9 (3.0)	0.5	7.5	26 (30)	6.9	2.9
F2	Leisure	Sodium Hypochlorite	UV	2.7 (3.0)	0.4	7.4	29 (31)	6.6	5.2
F3	Spa	Sodium Hypochlorite	UV	*2.7* (3.5)	0.3	7.2	29 (37)	4.5	1.3

*No result available for this sample. **BCDMH**: Bromochlorodimethylhydantoin. **NPOC**: Non-purgeable organic carbon. **TN**: Total nitrogen. **UV**: Ultraviolet irradiation. Additional details are provided in **Tables A4-2** and **A4-3**.

Excluding the pool treated by BCDMH, NPOC and TN concentrations were 3.1 to 29 and 1.3 to 16 mg L^{-1}, respectively, concentrations which are generally similar to, or only slightly higher than, concentrations reported previously in pools (4.2 to 29 and 0.6 to 12 mg L^{-1} for NPOC and TN, respectively) (Carter and Joll, 2017 and refernces therein). Significantly higher TN (40 mg L^{-1}) and NPOC (87 mg L^{-1}) concentrations were measured in the BCDMH treated pool (C1). While TN concentrations have not previously been reported in a pool treated with BCDMH, the NPOC concentration measured in the BCDMH treated pool (C1) of the current study was considerably lower than that reported in an indoor pool treated with BCDMH (125 mg L^{-1}, (Plewa et al., 2011)), and lower than NPOC concentrations (5 to 345 mg L^{-1}) measured in most BCDMH treated whirlpool spas investigated by Benoit and Jackson (1987). The TN and NPOC concentrations were likely elevated due to the presence of dimethylhydantoin (DMH), a molecule contributing both nitrogen and carbon, which remains after release of chlorine and bromine from BCDMH.

4.4.2. Occurrence of Disinfection By-products in Pools

Of the 64 investigated DBPs, only 20 (~31%) were not detected in any of the pools investigated (**Tables A4-4** to **A4-10**). Dibromochloroacetaldehyde, tribromoacetamide, bromodichloroacetonitrile, dibromochloroacetonitrile, trichloroacetonitrile and tribromoacetonitrile, all HNMs excluding bromonitromethane and trichloronitromethane and all I-THMs were all below their respective limits of detection (<0.1 to 1.3 µg L^{-1}), despite their occurrence in pools being reported in previous studies (Carter and Joll, 2017). The dominance of chlorine over bromine (via bromide oxidation) in pools may help to explain the absence of the subset of these DBPs which contain bromine in the waters investigated. Similarly, the absence of I-THMs is likely due to the low input of iodide and, hence low availability of iodine, to form I-THMs. Although surprising, the absence of trichloroacetonitrile is likely due to its susceptibility to base catalysed hydrolysis, which has been shown to occur at pH values greater than 5.5 (Croue and Reckhow, 1989), the operating pH range of these pools (7.2 to 7.8).

A more detailed summary of THM concentrations in all waters is provided in **Table A4-4**. Excluding the BCDMH treated pool (C1), trichloromethane was detected in all chlorinated pools and was the predominant THM, with concentrations of 7.7 to 96 µg L^{-1} being measured. Trichloromethane concentrations in this study were generally comparable to previously reported concentrations, as summarised by Carter and Joll (2017), although some studies have reported significantly higher concentrations, e.g. up to 980 µg L^{-1} (Lahl et al., 1981). Although present in significantly lower concentrations (0.2 to 5.4 µg L^{-1}) than trichloromethane, bromodichloromethane, dibromochloromethane and tribromomethane were also found in some of the chlorinated pools. As observed for trichloromethane, these other

THM concentrations were generally similar to previous reports, as summarised by Carter and Joll (2017), although some studies reported significantly higher concentrations in chlorinated pools (e.g. up to 133, 223 and 318 μg L^{-1} for tribromomethane, dibromochloromethane and bromodichloromethane, respectively; Hang et al., 2016). Despite being measured in some previous studies of bromine treated pools at concentrations up to 0.7 μg L^{-1} (Daiber et al., 2016; Kelsall and Sim, 2001; Lourencetti et al., 2012; Richardson et al., 2010), trichloromethane and bromodichloromethane were below detection (0.2 to 0.5 μg L^{-1}) in the investigated BCDMH treated pool (C1) in this study. However, both dibromochloromethane and tribromomethane were measured in this pool at 2.4 and 132 μg L^{-1}, respectively, concentrations generally similar to those previously reported for bromine treated pools (Daiber et al., 2016; Kelsall and Sim, 2001; Lourencetti et al., 2012; Richardson et al., 2010), although Norin et al. (1980) reported a significantly higher concentration of tribromomethane (400 μg L^{-1}) in their study of a brominated pool.

Based on molar concentrations, trichloromethane accounted for 64 to 98% of the total THMs in chlorinated pools, while tribromomethane represented 98% of the measured THMs in the BCDMH treated pool. While no guidelines for THMs in pools currently exist in Australia, several European countries impose a guideline for the total THM concentration (calculated as trichloromethane equivalents of the molar sum of trichloromethane, bromodichloromethane, dibromochloromethane and tribromomethane). For example, the German guidelines state that the THM concentrations in pools should not exceed 20 μg L^{-1} (German Institute for Standardization, 2012). In the current study, 73% (11/15) of the investigated pools exceeded this German guideline, where total THM concentrations of 24 to 99 μg L^{-1} (as trichloromethane equivalents) were present. It should be noted however, that pools exceeding this German guideline should not be considered unsafe. Individual guidelines for THMs in pools differ between countries, regions, states, cities and local councils, and the most relevant guideline should be considered when assessing if THMs in pools are at levels considered to be acceptable.

Total HAA concentrations (the sum of all HAAs excluding tribromoacetic acid, which could be detected but not quantified) were 125 to 861 μg L^{-1} for chlorinated pools, with a significantly lower concentration (15 μg L^{-1}) in the BCDMH treated pool (C1). A more detailed summary of HAA concentrations is provided in **Table A4-5**. Based on molar concentrations, Cl-HAAs (sum of chloroacetic acid, dichloroacetic acid and trichloroacetic acid) accounted for 87 to 99% of the total HAA concentrations in chlorinated waters, where concentrations of up to 266, 200 and 479 μg L^{-1} were measured for chloroacetic acid, dichloroacetic acid and trichloroacetic acid, respectively. These concentrations are generally higher than most other studies of chlorinated pools, as summarised by Carter and Joll (2017),

although several studies have reported significantly higher concentrations, up to 6787 μg L^{-1} (Carter et al., 2015; Dehghani et al., 2018; Hang et al., 2016; Kanan, 2010; Lee et al., 2010; Simard et al., 2013; Tardif et al., 2015; Wang et al., 2014; Yeh et al., 2014). Compared to most previous studies (e.g. Manasfi et al., 2016), similar concentrations of bromoacetic acid, bromochloroacetic acid, bromodichloroacetic acid and dibromochloroacetic acid were also detected in some of the chlorinated pools in this study, where concentrations of 4.8 to 12, 2.9 to 16, 0.3 to 19 and 4.2 to 18 μg L^{-1} were measured, respectively. Dibromoacetic acid (6.2 μg L^{-1}) and tribromoacetic acid (identified only) were only detected in the BCDMH treated pool (C1), where other HAAs were also measured, 0.3 to 7.7 μg L^{-1} (**Table A4-5**). The only other known study of HAAs in brominated pools is by Daiber et al (2016), where generally higher HAA concentrations compared to those in the current study were reported, particularly for dibromoacetic acid, trichloroacetic acid and tribromoacetic acid, where concentrations up to 131 μg L^{-1} were reported.

Table A4-6 summarises the concentrations of HKs in all waters. None of the five investigated HKs were detected in the BCDMH treated pool (C1), which is consistent with the results of Daiber et al. (2016) who reported 1,1-dichloropropanone and 1,1,1-trichloropropanone (1,1-DCP and 1,1,1-TCP, respectively) as being below detection limits (<1 μg L^{-1}) in all pools and spas treated with BCDMH. For the chlorinated waters, 1,1,1-TCP was detected in all waters (2.2 to 13 μg L^{-1}) and represented 11 to 89% of the total HKs on a molar basis. Previous studies have reported similar concentrations (0.4 to 14 μg L^{-1}) of 1,1,1-TCP in chlorinated pools or spas (Carter et al., 2015; Font-Ribera et al., 2016; Manasfi et al., 2016; Serrano et al., 2014; Tardif et al., 2016). However, higher concentrations (up to 180 μg L^{-1}) were reported for some pools investigated by Hang et al. (2016). Chloropropanone was detected in all but one of the chlorinated pools (2.3 to 10 μg L^{-1}, 25 to 82% of the total HK concentration) but it has only been reported in two other studies at concentrations of 1.2 to 4.0 μg L^{-1} (Carter et al., 2015; Serrano et al., 2014). 1,1-DCP and 1,3-dichloropropanone were detected (up to 4.3 μg L^{-1}) in some of the chlorinated pools. These concentrations were similar to the concentrations (0.2 to 11 μg L^{-1}) reported in most previous studies: (Carter et al., 2015; Daiber et al., 2016; Hang et al., 2016; Serrano et al., 2014; Yeh et al., 2014), although concentrations up to 21 μg L^{-1} for 1,1-DCP have been reported (Manasfi et al., 2016). 1,1,3,3-Tetrachloropropanone was measured in most chlorinated pools in concentrations ranging from 0.2 to 1.6 μg L^{-1}, similar to the concentrations (0.1 to 2.7 μg L^{-1}) reported by Serrano et al. (2014), the only other study to report this ketone in swimming pools.

HALs were the predominant DBP class in chlorinated pools, accounting for 28 to 67% of the total DBPs in these waters. **Table A4-7** summarises the occurrence of HALs in the investigated waters. Chloral hydrate (the monohydrate of trichloroacetaldehyde) was detected

in all chlorinated waters (202 to 1313 μg L^{-1}), accounting for 86 to 97% of the total HALs measured. Furthermore, chloral hydrate, on a molar basis, was the predominant DBP in this study, representing up to 64% of the total DBP concentrations. The concentrations of chloral hydrate in this study were generally higher than the concentrations (5 to 340 μg L^{-1}) reported previously in chlorinated pools or spas (Daiber et al., 2016; Lee et al., 2010; Zhang et al., 2015). Although Yeh et al. (2014) reported significantly lower concentrations (19 to 24 μg L^{-1}) of chloral hydrate in some Australian pools compared to those measured in the current study, similarly high concentrations (up to 405 μg L^{-1}) were reported in our previous study (Carter et al., 2015), the only other known Australian study. These higher concentrations reported in Australian pools are likely a reflection of the higher chlorine residuals employed in Australia (i.e. those observed in our current (**Table A4-2**) and previous study (Carter et al., 2015)) compared to other countries (e.g. Europe). The lower chloral hydrate concentrations reported in Australian pools by Yeh et al. (2014) are likely a result of these pools not meeting the minimum chlorine residual requirements, since chlorine residuals of 1.2 ± 0.7 mg L^{-1} were reported in the majority (>60%) of the investigated pools. Dichloroacetaldehyde was measured in all chlorinated pools (2.7 to 35 μg L^{-1}) and has only been reported in one other study (at 23 μg L^{-1}) (Serrano et al., 2011). The present study is the first known report of bromodichloroacetaldehyde (found in all chlorinated pools between 9.5 and 46 μg L^{-1}) and bromochloroacetaldehyde (found in two of the investigated waters up to 3.0 μg L^{-1}) in swimming pools. In the BCDMH treated pool (C1), however, dibromoacetaldehyde and tribromoacetaldehyde were the only HALs detected, in concentrations of 2.5 and 20 μg L^{-1}, respectively, which is consistent with the findings of Daiber et al. (2016) who reported the absence of chloral hydrate in several pools treated by BCDMH. Similarly, to the situation observed for chloral hydrate in chlorinated pools, tribromoacetaldehyde contributed 85% of the total HALs in the BCDMH pool (C1, based on molar concentrations), however, unlike chloral hydrate, it only accounted for less than 1% of the total DBP concentrations in this pool.

Table A4-8 summarises the concentrations of HANs in this study. Of the investigated chlorinated pools, chloroacetonitrile, dichloroacetonitrile and bromochloroacetonitrile were detected in all pools (0.4 to 1.9, 4.1 to 38 and 0.4 to 2.3 μg L^{-1}, respectively), with bromoacetonitrile and dibromoacetonitrile detected only in a few pools (up to 1.4 μg L^{-1}). For the chlorinated pools, dichloroacetonitrile accounted for 70 to 93% of the total HAN concentrations, however, dibromoacetonitrile (detected at 7.9 μg L^{-1}) only represented 41% of the total HAN concentration measured in the BCDMH treated pool (C1), where all comparisons were based on molar concentrations. Bromoacetonitrile was the only other HAN measured in the BCDMH treated pool (C1) (6.7 μg L^{-1}). Concentrations in this study are similar to most previous studies, summarised by Carter and Joll (2017), although some studies

have reported higher dichloroacetonitrile and dibromoacetonitrile in chlorinated and brominated pools, respectively (Hang et al., 2016; Weaver et al., 2009).

Of the pools investigated in this study, only two contained a HNM at a detectable concentration. Although below detection (<0.5 μg L^{-1}) in all other pools, trichloronitromethane (1.6 μg L^{-1}) was measured in one of the hydrotherapy pools (B1), which is similar to concentrations (<1 to 5 μg L^{-1}) reported for other chlorinated pools (Hang et al., 2016; Manasfi et al., 2016; Tardif et al., 2016). Although tribromonitromethane, bromochloronitromethane and bromonitromethane have been reported in other chlorinated pools (1.2 to 11 μg L^{-1}; Kanan, 2010; Yeh et al., 2014), these HNMs were below detection limits (0.7, 0.2 and 0.4 μg L^{-1}, respectively) in all pools of the current study. Furthermore, while this study is the first known investigation of several other HNMs (chloronitromethane, dichloronitromethane, dibromonitromethane, bromodichloronitromethane and dibromochloronitromethane) in pools, these HNMs were below their respective detection limits (0.2 to 0.7 μg L^{-1}) in all pools. Bromonitromethane was below detection (0.2 μg L^{-1}) in all pools except the BCDMH treated pool (C1) where it was found at a concentration of 0.8 μg L^{-1}, which is the first detection of a HNM in a BCDMH treated pool. Only one other study is known to have investigated HNMs (trichloronitromethane only) in this pool type, where concentrations were below the detection limit (<1 μg L^{-1}) in both a BCDMH treated pool and spa (Daiber et al., 2016).

A significantly higher concentration (907 μg L^{-1}, 4.0 μM) of total HAAms was measured in the BCDMH treated pool (C1) compared to the concentrations measured in chlorinated pools (13 to 88 μg L^{-1}, 0.09 to 0.58 μM) (**Table A4-9**). Dibromoacetamide (736 μg L^{-1}; representing 83% of the total HAAm concentration) was the predominant HAAm in the BCDMH treated pool (C1), although the other brominated HAAms were also detected. In the chlorinated pools, dichloroacetamide and trichloroacetamide were detected in all cases (up to 65 μg L^{-1}), contributing 14 to 97 and 3 to 78% of total HAAms, respectively. The mixed bromo/chloro HAAms were detected (0.2 to 5.2 μg L^{-1}) in some of the chlorinated pools. There have only been two previous studies of the concentrations of HAAms in pools (Carter et al., 2015; Yeh et al., 2014). HAAm concentrations in the current study were often significantly higher than concentrations in the earlier two studies (up to 3.1 μg L^{-1}) with most HAAms below detection limits (<0.1 μg L^{-1}) in the study by Yeh et al. (2014).

All *N*-nitrosamines were detected in at least one of the pools (**Table A4-10)**, with this study the first to report *N*-nitrosamines in a brominated pool. *N*-Nitrosodi-*n*-butylamine (NDBA) was the only *N*-nitrosamine detected in all pools, representing up to 87% of the total *N*-nitrosamine concentration (molar basis), where concentrations of 11 to 386 ng L^{-1} were

measured in pools and even higher concentrations (up to 1093 ng L^{-1}) measured in some spas. These concentrations are considerably higher than those reported in our previous study (up to 33 ng L^{-1}) (Carter et al., 2015), and generally higher than most pools investigated by Wang (2011), although concentrations up to 403 ng L^{-1} were reported in some of the chlorinated pools in this study (Wang, 2011). NDBA could not be detected in a chlorinated spa in our previous study (Carter et al., 2015), and was detected but not quantified in a chlorinated spa by Walse and Mitch (2008). *N*-Nitrosodimethylamine (NDMA) was found in all chlorinated pools at concentrations between 0.5 and 65 ng L^{-1}. Similar or lower concentrations (0.7 to 65 ng L^{-1}) of NDMA have been reported in other chlorinated pools (Carter et al., 2015; Font-Ribera et al., 2016; Fu et al., 2012; Kim and Han, 2011; Walse and Mitch, 2008), although higher concentrations have also been reported in some pools (up to 208 ng L^{-1}; Fu et al., 2012; Kanan, 2010; Kim and Han, 2011; Tardif et al., 2015) and chlorinated spas (up to 429 ng L^{-1}; Walse and Mitch, 2008). NMOR was also measured in all chlorinated pools in the current study (5.3 to 30 ng L^{-1}), in similar concentrations to those previously reported (0.3 to 34 ng L^{-1}) (Carter et al., 2015; Kim and Han, 2011). Despite being detected in all chlorinated pools, both NDMA and NMOR were below detection limits (1.9 and 0.9 ng L^{-1}, respectively) in the BCDMH pool (C1) in this study. Significantly higher concentrations (21 ng L^{-1}) of *N*-nitrosopyrrolidine (NPYR) were measured in the BCDMH treated pool (C1) compared to concentrations (up to 2.9 ng L^{-1}) in the 4 chlorinated pools (B2, D2, E3 and F2) where NPYR was detected. In our previous study, NPYR was below the limit of detection in all pools and the spa investigated (Carter et al., 2015), although NPYR has been reported in other pool studies (4.5 to 127 ng L^{-1}) (Jurado-Sánchez et al., 2010; Pozzi et al., 2011). *N*-Nitrosopiperidine (NPIP) was detected in all but one of the investigated pools (1.8 to 5.9 ng L^{-1}), despite being below detection in the only other known study of this DBP (Carter et al., 2015). *N*-Nitrosodipropylamine (NDPA) was only detected (2.2 to 7.7 ng L^{-1}) in 10 of the 15 pools investigated. In the only previous study of this DBP, we found NDPA at 2.3 ng L^{-1} in a chlorinated pool (Carter et al., 2015). *N*-Nitrosoethylmethylamine (NEMA) was only detected in sample C1 (the BCDMH treated pool) (24 ng L^{-1}), as was *N*-nitrosodiethylamine (NDEA, 9.2 ng L^{-1}), but NDEA was also detected in one chlorinated spa at 4.5 ng L^{-1}. NEMA was below the detection limit in all pools in our previous study (Carter et al., 2015), but was detected (1.7 ng L^{-1}) in one of the two pools investigated by Fu et al. (2012). Similar concentrations of NDEA have been previously reported for chlorinated pools (1.1 to 9.0 ng L^{-1}; Carter et al., 2015; Fu et al., 2012; Jurado-Sánchez et al., 2010), although one study reported much higher concentrations (18 to 53 ng L^{-1}; Kim and Han, 2011).

4.4.3. Contribution of Filling Waters

Mains water (disinfected distributed drinking water) is used to fill and regularly top-up the pools and spas at each facility. Mains water was therefore investigated in order to evaluate its impact, if any, on the occurrence of DBPs in the pools. Samples A5, B3, C2, D3, E4 and F4 represent the mains water at the given facility. **Table A4-2** presents the general water quality parameters of the six mains waters investigated, while **Tables A4-4** to **A4-10** report the concentrations of DBPs. It should be noted that unusually low chlorine residuals (<0.1 mg L^{-1}) were measured in sample A5. The lower chlorine residuals indicate that the occurrence of DBPs in this sample may be lower than that typically expected. Due to its outlying nature, where noted, sample A5 has been excluded from comparisons in this section.

Tribromomethane and dibromochloromethane were the predominant THMs in all of the filling waters (based on molar concentrations), being present at concentrations between 3.5 to 35 and 1.9 to 49 µg L^{-1}, respectively **(Table A4-4)**. Bromodichloromethane was measured in 4 of the 5 filling waters (1.7 to 27 µg L^{-1}), while trichloromethane was only measured in two filling waters (5.6 and 12 µg L^{-1} in samples D3 and F4, respectively). These THMs were present in the filling waters in significantly lower concentrations than the concentrations in most pools of this study. I-THMs were below detection limits (0.4 to 0.9 µg L^{-1}) in all filling waters. Predominantly brominated HAAs (dibromoacetic acid, bromodichloroacetic acid and dibromochloroacetic acid) were detected in most filling waters (up to 6.6, 4.9 and 31 µg L^{-1}, respectively), although some filling waters contained bromochloroacetic acid, dichloroacetic acid and/or trichloroacetic acid (<9.3 µg L^{-1}, **Table A4-5**). Chloroacetic acid and bromoacetic acid were below detection limits (6.4 and 4.4 µg L^{-1}, respectively) in all filling waters.

Generally, N-DBPs were measured at lower concentrations in the filling waters compared to THMs and HAAs. HNMs were below detection limits (0.1 to 0.7 µg L^{-1}) in all filling waters. Dibromoacetonitrile (1.1 to 3.4 µg L^{-1}) and dibromoacetamide (2.6 to 9.6 µg L^{-1}) were the predominant HAN and HAAm, respectively, in filling waters (molar basis), being detected in all but one (A5) of the five filling waters (**Tables A4-8 and A4-9**). Other HANs (dichloroacetonitrile and dibromoacetonitrile) and HAAms (dichloroacetamide and bromochloroacetamide) were measured (0.9 to 4.3 µg L^{-1}) in some of the filling waters investigated, with these concentrations being generally lower than those in the corresponding pools. NPIP and NMOR were detected in all filling waters (1.4 to 4.2 and 1.3 to 2.2 ng L^{-1}, respectively), while NDBA (3.4 to 16 ng L^{-1}) was detected in 4 of the 5 filling waters (**Table A4-10**). Other *N*-nitrosamines (NDEA, NDPA and NPYR) were only detected (2.1 to 9.9 ng L^{-1}) in several of the filling waters, while NDMA and NEMA were below detection

limits (1.9 and 1.1 ng L^{-1}, respectively) in all five filling waters. All HKs and HALs were below detection limits (0.2 to 8.3 µg L^{-1}) in four of the five filling waters, although 1,1,1-TCP (0.68 µg L^{-1}) and three HALs (2.5 to 11 µg L^{-1}) were detected in one of the filling waters (F4).

In some cases, however, the filling water contained equal or greater concentrations of some DBPs, mainly brominated species (e.g. tribromomethane), compared to those measured in the pools. The presence of bromide in the drinking water source waters, measured in filling waters after quenching the oxidant residual at concentrations of 37 to 291 mg L^{-1}, which is not removed in conventional drinking water treatment processes, can lead to bromine formation upon disinfection (chlor(am)ine), where DBP formation via bromination would be greater than that via chlor(am)ination, hence the higher brominated DBPs observed in the drinking waters. While the formation of brominated DBPs can occur in pools filled with distributed drinking waters, this is considered a 'secondary pathway' of DBP formation as, in comparison to bromine concentrations, a significantly higher level of chlorine is expected in chlorinated pools, leading to much higher concentrations of chlorinated DBPs in comparison to brominated DBPs. In the case of the BCDMH treated pool (C1), however, bromination remains the major DBP formation pathway, as concentrations of bromine are significantly greater than those of chlorine and bromine is faster reacting than chlorine. As expected, for this pool (C1), brominated DBPs were generally higher than those found in the filling water (C2), a trend which is discussed further in **Section 4.4.4**.

All pool waters contained a higher molar concentration of total DBPs compared to their corresponding filling waters. For the purpose of this comparison and for reasons mentioned earlier in this section, sample A5 has been excluded. All other pools contained between 3 and 26 times higher molar concentrations of the investigated DBPs compared to their corresponding filling waters, with total N-DBP concentrations (molar sum of HANs, HNMs, HAAms and *N*-nitrosamines) also considerably less in the filling waters (0.08 to 0.23 µM) compared to those in the pools (0.40 to 8.63 µM). These concentration differences suggest that filling water is not a significant contributor of these DBPs in swimming pool waters, particularly N-DBPs. To more fully assess the impact of filling waters on DBPs in swimming pools, the DBP formation potential of filling waters could be quantified by exposing filling waters to conditions similar to those of swimming pools (e.g. chlorine concentrations, pH, and water temperature) under controlled laboratory conditions.

4.4.4. Comparison of the Swimming Pools

Swimming pool type is known to impact the formation and hence occurrence of DBPs, as summarised in two recent reviews (Carter and Joll, 2017; Manasfi et al., 2017). Keuten et al. (2012) has shown the amount of sweat, urine and loose dirt released from swimmers is

dependent on the type of swimmer. Of the organic input from athletic swimmers, i.e. those who swim for exercise, 40% resulted from sweat, 30% from urine and 30% from loose dirt. However, of the organic input released by recreational swimmers, i.e. those who swim for leisure, 5 to 15% was found to originate from sweat, 5 to 45% from urine, with 30 to 40% originating from loose dirt. This study highlights that the type of activity (e.g. exercise, aquaerobics, or leisure swimming) and therefore pool type (e.g. lap, spa or leisure pool) can have a significant impact on the organic input, and hence DBP formation, in these waters. Therefore, the occurrence of DBPs based on pool type (lap, leisure, spa and hydrotherapy pools) was compared.

Figure 4-1 depicts the occurrence of the investigated DBPs classified by pool type. For almost all DBP classes investigated, spas showed the highest variability (greatest range) in concentrations, which is likely a result of (i) some spas having been recently emptied and refilled with fresh water prior to sample collection, and (ii) the generally higher operational temperature of spas compared to other pool types resulting in an increase in the release of anthropogenic chemicals and DBP formation rates. Furthermore, high outliers can be observed for several DBP classes for lap pools, corresponding to pools D1 and E1, suggesting these pools were significantly different in DBP concentration to the other lap pools investigated. Considering total DBP molar concentrations (**Figure 4-1(a)**) on average (based on median values), hydrotherapy and spa pools contained higher concentrations of DBPs compared to lap and leisure pools. This may be due to the higher operating temperatures of these waters (32.5 °C on average) compared to those of leisure and lap pools (on average 29.4 and 26.9 °C, respectively), increasing the rate of DBP formation. The higher DBP concentrations in these waters may also be reflective of their water volumes. Spa and hydrotherapy pools are often smaller in size, and therefore smaller in water volumes, compared to lap and leisure pools, and, as such, DBPs can become more easily concentrated in these waters. This, however, would be dependent on many factors, e.g. particularly DBP precursor input from bather load, including swim duration, DBP formation rates, pool dilution and water replacement are additional factors which may be involved.

HAAs (**Figure 4-1(b)**) and HALs (**Figure 4-1(c)**) were, based on median values, generally higher in spa and hydrotherapy pools, suggesting these waters contain higher levels of HAA and HAL precursors, compared to lap and leisure pools. Concentrations of HKs (**Figure 4-1(d)**) were generally similar across all investigated pools, suggesting that pool type is not a major factor in the formation of HKs. In addition to temperature, water agitation via swimmers and water jets has been shown to increase the volatilisation rate (and hence decrease water concentrations) of THMs (Kristensen et al., 2010; Marco et al., 2015), which may

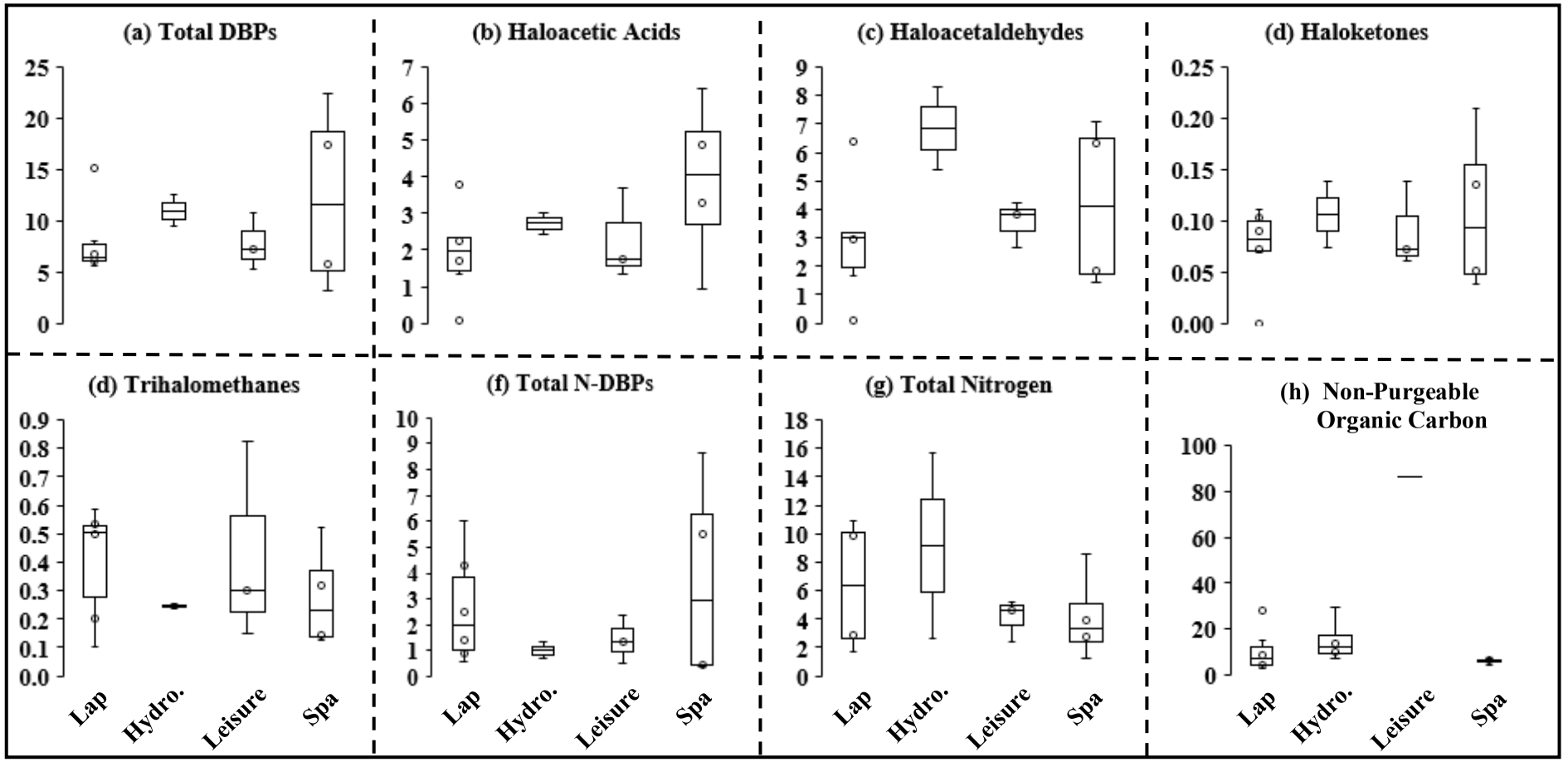

Figure 4-1: Comparison of the occurrence of disinfection by-products (DBPs) based on pool type. Y-axis represents the following: Figures **(a)** to **(f)** – the total concentration (µM), representing the molar sum of each DBP of a given class; Figures **(g)** and **(h)** – concentration (mg L^{-1}). Total N-DBPs refers to the molar sum of all detected haloacetonitriles, halonitromethanes, haloacetamides and *N*-nitrosamines. **Hydro.**: Hydrotherapy.

explain why, on average (based on median values), spa pools had lower concentrations of THMs compared to lap pools (**Figure 4-1(e)**).

Considering molar averages, similar concentrations of N-DBPs were measured amongst the different pool types (**Figure 4-1(f)**), although some spa and lap pools contained higher concentrations. Lap pools, where more strenuous swimmer activity is likely, and spa pools, where higher operating temperatures are found, likely see a higher release of N-DBP precursors (via an increase in sweat) which may assist in explaining the higher observed N-DBP concentrations in these two pool types.

Considering concentration ranges and median values, generally similar NPOC concentrations were observed (**Figure 4-1(g)**) across most pool types, although a significantly larger range and higher median value were observed for NPOC concentrations in spa pools. Although lowest for leisure pools (**Figure 4-1(g)**), TN concentrations largely varied between pools of a given type, with spa and leisure pools generally observed to have lower median and range values compared to lap or hydrotherapy pools. Considering concentration ranges and median values, generally similar NPOC concentrations were observed (**Figure 4-1(g)**) across most pool types, although a significantly larger range and higher median value were observed for NPOC concentrations in spas pools. It should be noted that NPOC and TN concentrations in pool C1 were excluded from **Figure 4-1**, for reasons discussed later in this section. Organic carbon was reported to reach steady state via mineralization in 200 to 500 hours in a model swimming pool for organic carbon concentrations of between 6.5 to 28 mg L^{-1} (Judd and Bullock, 2003). This provides a possible explanation for the fairly consistent NPOC concentrations in most pools during this study, as these pools had not been emptied for some time. Total nitrogen concentrations, likely a consequence of nitrogen rich bodily fluids (e.g. sweat, urine) which can be introduced via bathers (Keuten et al., 2014, 2012), were generally higher in lap and hydrotherapy pools compared to concentrations in the leisure and spa pools. As previously discussed, for lap pools, higher total nitrogen concentrations can be explained by the higher activity level, hence higher release of organic nitrogen (e.g. via sweat), of swimmers who frequent lap pools. Although driven by water temperature as opposed to swimmer activity level, higher levels of nitrogen rich compounds are likely released from swimmers in hydrotherapy pools, consistent with the higher TN concentrations in these pools. Although not observed in this study (due to likely recent re-filling of some of the spas), a similar trend is expected for spa pools.

In addition to pool type, disinfectants and secondary treatment are known to impact the formation and hence occurrence of DBPs in swimming pools (Carter and Joll, 2017). Generally, chlorine and bromine based disinfectants lead to the formation of predominantly

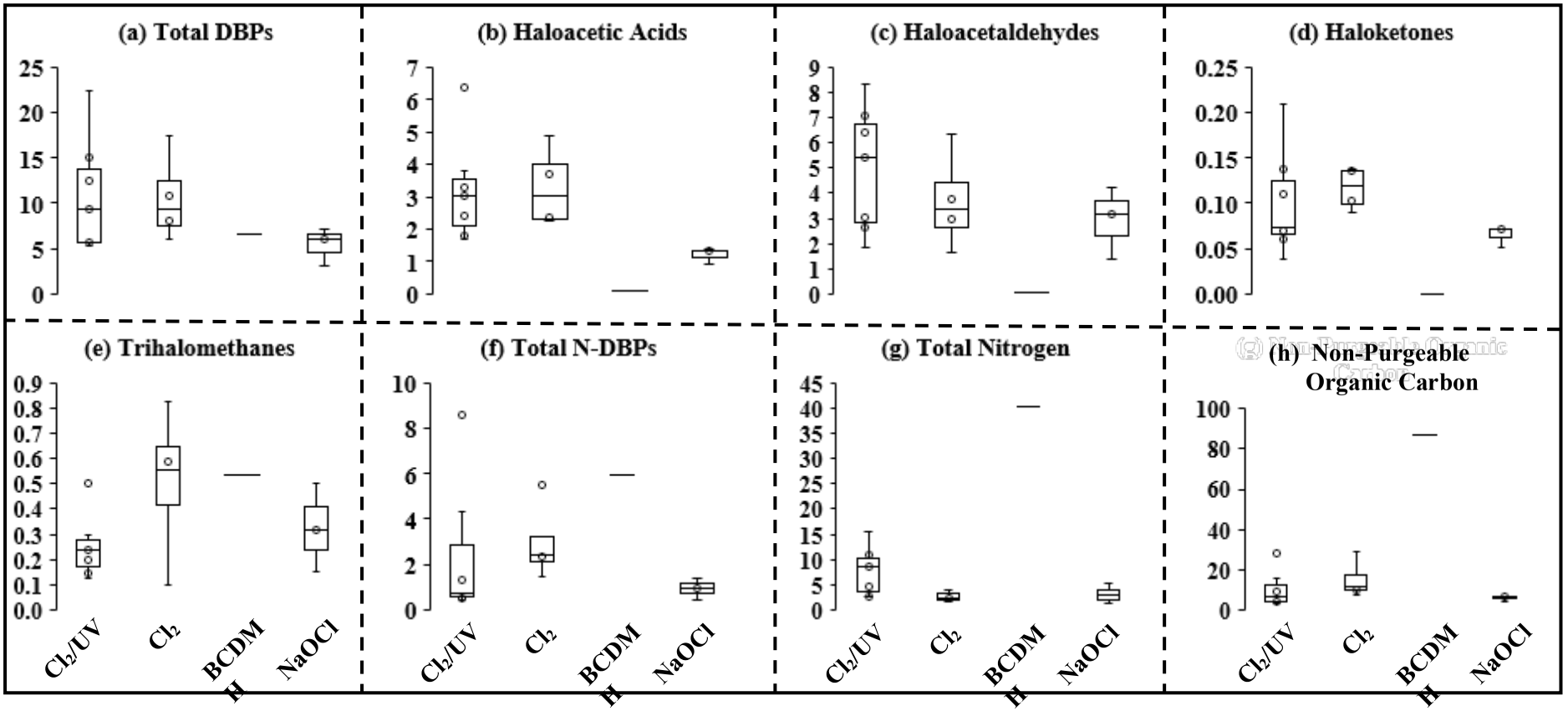

Figure 4-2: Comparison of the occurrence of disinfection by-products (DBPs) based on treatment type. Y-axis represents the following: Figures **(a)** to **(f)** – the total concentration (µM), representing the molar sum of each DBP of a given class; Figures **(g)** and **(h)** – concentration (mg L^{-1}). Total N-DBPs refers to the molar sum of all detected haloacetonitriles, halonitromethanes, haloacetamides and *N*-nitrosamines. **BCDMH**: Bromochlorodimethylhydantoin. **Cl_2**: Chlorine gas. **Cl_2/UV**: Chlorine gas combined with ultraviolet irradiation. **NaOCl**: Sodium hypochlorite.

chlorinated and brominated DBPs, respectively. For chlorine based disinfectants, although the predominant species is HOCl, more reactive species such as Cl_2 have been shown to play an important role in DBP formation (De La Mare et al., 1975; Sivey and Roberts, 2012). Similarly, while UV treatment has been shown to decrease the concentration of some DBPs (Zare Afifi and Blatchley, 2016), other studies have reported the opposite, particularly for THMs (Cimetiere and De Laat, 2014; Spiliotopoulou et al., 2015). As with pool type, a comparison of the DBP concentrations for pools with differing disinfectants and secondary treatments (chlorine gas (Cl_2), chlorine gas with ultraviolet irradiation (Cl_2/UV); sodium hypochlorite (NaOCl) and BCDMH) is provided (**Figure 4-2**).

As expected, higher concentrations of brominated DBPs were measured in the BCDMH treated pool (C1) compared to those pools employing chlorine disinfectants. On a molar basis, however, generally a higher concentration (0.5 μM) of tribromomethane was measured in the BCDMH treated pool (C1) compared to the concentrations (0.1 to 0.5 μM) of trichloromethane measured in most chlorinated pools, although higher levels (up to 0.8 μM) of trichloromethane were measured in some chlorinated pools (E1 and E2). This is likely a result of the higher reactivity of BCDMH, mainly the released bromine, compared to chlorine (Hunter and Jiang, 2010). Several high outliers were observed for pools treated by Cl_2/UV for some DBP classes and NPOC. These outliers were found to originate from one facility (Facility D), indicating that while the treatment method of Cl_2/UV can have an influence on the chemical water quality of pools, other factors related to facility (e.g. management or bather load) also have an impact.

Considering total molar DBP concentrations (**Figure 4-2(a)**), similar concentrations (median and range values) were found for most treatment types, although slightly lower median concentrations were observed for pools disinfected with NaOCl. The more reactive species (Cl_2 electrophiles and bromine) have been shown to be present in pools treated with Cl_2 and BCDMH (Carter and Joll, 2017 and references therein), but are absent in the NaOCl treated pools, potentially explaining the lower molar DBP concentrations in these pools.

Considering median values, HAA concentrations were similar in pools treated with Cl_2 and Cl_2/UV, which were approximately 3 times higher than the HAA concentrations in NaOCl treated pools (**Figure 4-2(b)**). Lowest HAA concentrations were measured in the BCDMH treated pool, where trichloroacetic acid was the only chlorinated HAA detected. This is likely due to the inability to quantify tribromoacetic acid (due to analytical issues), which has been reported to be the dominant HAA in BCDMH treated pools (Daiber et al., 2016). THMs were generally higher in Cl_2 treated pools compared to Cl_2/UV (**Figure 4-2(c)**), suggesting that UV may decrease the formation of THMs, as previously reported in some studies (Zare Afifi and Blatchley, 2016), and/or degrade the THMs formed. THM concentrations in pools treated with

NaOCl were, for the majority of pools, higher than those measured in pools treated with Cl_2/UV, but lower than in pools treated with Cl_2. Despite THM concentrations measured in the BCDMH treated pool being similar to those in the Cl_2 treated pools, the THM species were predominantly brominated and chlorinated, respectively.

Only slight differences in the concentrations of HALs and HKs were observed in most chlorinated pools (**Figures 4-2(c)** and **4-2(d)**, respectively), although a larger range was observed for Cl_2/UV treated pools. No HKs, or HALs (excluding dibromoacetaldehyde or tribromoacetaldehyde) were measured in the BCDMH treated pool (C1). THMs were generally higher in Cl_2 treated pools compared to Cl_2/UV (**Figure 4-2(e)**), suggesting that UV may decrease the formation of THMs, as previously reported in some studies (Zare Afifi and Blatchley, 2016), or degrade the THMs formed. THM concentrations in pools treated with NaOCl were, for the majority of pools, higher than those measured in pools treated with Cl_2/UV, but lower than in pools treated with Cl_2. Despite THM concentrations measured in the BCDMH treated pool being similar to those in the Cl_2 treated pools, the THM species were predominantly brominated and chlorinated, respectively.

While disinfectants are likely to play only a minor role in the formation of N-DBPs compared to other parameters (e.g. bather load and temperature), generally the pools treated with Cl_2/UV contained lower concentrations of N-DBPs compared to those treated solely with Cl_2, although pools treated by NaOCl generally contained the lowest N-DBP concentrations (**Figure 4-2(f)**). The lower concentrations of N-DBPs in pools treated with Cl_2/UV compared to Cl_2 are potentially due to the reduction of chloramines in the pools by UV (Cimetiere and De Laat, 2014; Soltermann et al., 2014), since chloramines have been shown to play an important role in the formation of some N-DBPs (Soltermann et al., 2013; Yang et al., 2012). The BCDMH treated pool (C1) contained significant concentrations of N-DBPs compared to the other pools (**Figure 4-2(f)**), which is most likely a reflection of the high TN content (40 mg L^{-1}) of this water. As discussed earlier in this section for total molar DBP concentrations, the lower concentrations of N-DBPs in pools treated with NaOCl may also be a result of the absence of the more reactive species present only in the pools treated with Cl_2 or BCDMH.

TN concentrations were similar for Cl_2 and NaOCl treated pools (**Figure 4-2(g)**), while pools treated by Cl_2/UV generally contained higher TN concentrations. While human input is likely the major contributor of TN in the chlorinated pools, the major input of TN in the BCDMH treated pool is likely to be the disinfectant itself. As discussed in **Section 4.4.1**, the use of BCDMH results in the build-up of DMH, a source of both organic nitrogen and carbon, likely explaining the elevated TN and NPOC concentrations in this pool. For the other pools,

NPOC concentrations (**Figure 4-2(h)**) were generally similar regardless of treatment type, which as discussed earlier in this section, is likely due to NPOC reaching steady state via mineralisation, despite its continual input.

4.4.5. Estimation of Cytotoxicity

The cytotoxicity of the pool waters was estimated based on measured DBP concentrations and their reported C_{50} values (Wagner and Plewa, 2017). An example calculation and summary of calculated cytotoxicity values are presented in **Figure A4-1** and **Table A4-11**, respectively. **Figure 4-3** shows the average contribution of each DBP class to the total calculated cytotoxicity for pools and their filling waters.

At the concentrations measured, HALs were found to contribute, on average, 93% of the total calculated cytotoxicity of swimming pool waters, despite only being responsible for, on average, 7.7% of the total calculated cytotoxicity in filling waters. Excluding the pool treated with BCDMH, the calculated HAL cytotoxicity component was predominantly due to chloral hydrate, which was found to represent 96 to 99% of the associated calculated cytotoxicity of HALs, but represented almost 99% of the overall calculated cytotoxicity in chlorinated pools. For filling waters, however, chloral hydrate was either below detection (1.3 $\mu g\ L^{-1}$) or at significantly lower concentrations than in the pools, which aids in explaining the observed difference in the calculated cytotoxicity contribution between pools and their filling waters.

HAAms were the second largest DBP class contributing to the calculated cytotoxicity for both pools and filling waters, although they accounted for a larger fraction (29%) in filling waters, compared to pools (6.2%). This is likely a reflection of HAAms relatively higher concentrations (in comparison to other DBP classes) in the filling waters compared to the pools. While HANs did not significantly contribute (<1%) to the total calculated cytotoxicity in swimming pools, HANs were calculated to represent 42% of the total estimated cytotoxicity of the filling waters. This is likely due to the absence (or significantly lower concentrations) of other more (or equally) cytotoxic DBPs (e.g. chloral hydrate) in the filling waters compared to the pools.

While HAAs and THMs were generally the predominant (considering molar concentrations) DBP classes in the filling waters, they were only found to contribute up to 19 and 1.9% of the total calculated cytotoxicity, respectively. A similar trend was observed for pools, where HAAs and THMs combined were calculated to only represent <0.3% of the total calculated cytotoxicity. For pools, other DBPs (e.g. HNMs and NMOR) were found to

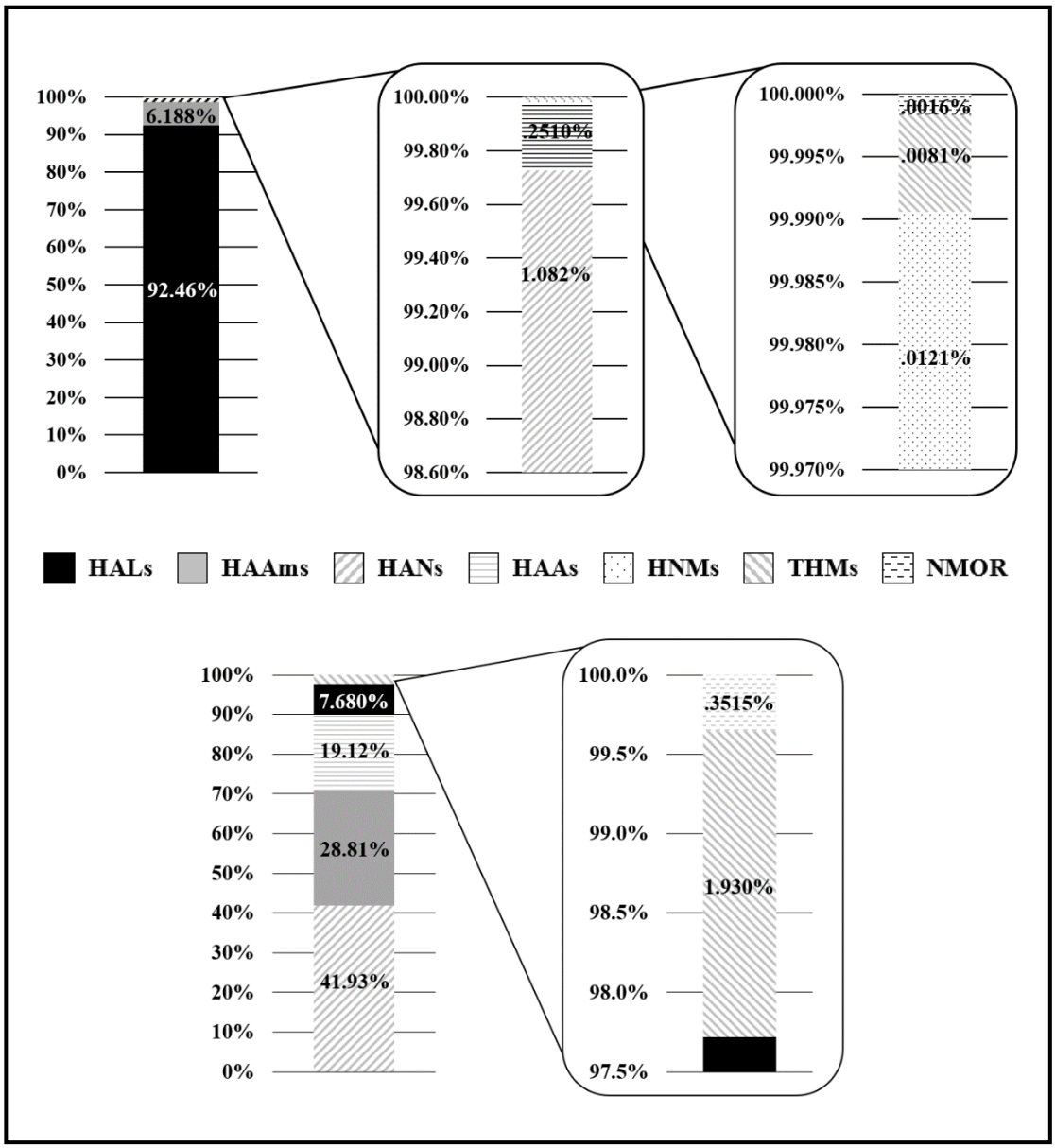

Figure 4-3: Contribution of disinfection by-product class to the calculated relative cytotoxicity of pool (top) and filling (bottom) waters. Values represent an average across all pool or filling waters investigated, determined by summing the relative cytotoxicity calculated for each detected DBP of a given class. **HAAms**: Haloacetamides. **HAAs**: Haloacetic acids. **HALs**: Haloacetaldehydes. **HANs**: Haloacetonitriles. **HNMs**: Halonitromethanes. **NMOR**: *N*-Nitrosomorpholine. **THMs**: Trihalomethanes.

contribute equal or greater calculated cytotoxicity (0.002 to 0.01%) than THMs (0.008%), despite THMs being measured at significantly higher concentrations. This study highlights that (i) the concentration of DBPs alone, particularly the commonly reported HAAs or THMs, is an insufficient method to assess the chemical water quality of swimming pool waters; and (ii) despite their relatively lower concentrations, other DBPs (e.g. HANs, HNMs or *N*-nitrosamines) may contribute significantly to the cytotoxicity of pool waters.

Although we acknowledge that DBP speciation should be considered, due to the generally higher total molar DBP concentrations measured in these waters (**Figure 4-2(a)**), spa and hydrotherapy pools were expected to be higher in calculated cytotoxicity compared to other pool types. Considering pool type (**Figure 4-4(a)**), and based on the average calculated cytotoxicity, hydrotherapy pool waters were found to be the most cytotoxic, followed by (in order of decreasing estimated cytotoxicity), those of spa, leisure, lap and training pool(s). Based on average calculated cytotoxicity values, hydrotherapy pools were estimated to be approximately twice as cytotoxic as lap, leisure and spa pools, and three times more cytotoxic than the investigated training pool. The difference in calculated cytotoxicity between the pool types is a reflection of DBP concentrations, particularly chloral hydrate, which was generally highest in hydrotherapy pools. Chloral hydrate has been shown to form from the chlorination of several anthropogenic constituents, such as skin, saliva, hair and urine (Kim et al., 2002), and amino acids, such as creatinine, urea, histidine, arginine and glycine (Wlodyka-Bergier and Bergier, 2016). As discussed in **Section 4.4.4**, these constituents are likely to be found at greater concentrations in spa and hydrotherapy pools, consistent with the higher cytotoxicity observed in hydrotherapy pools and most spas of this study.

Calculated cytotoxicity also differed with pool treatment methods. Considering the average calculated cytotoxicity (**Figure 4-4(b)**), pools treated with Cl_2/UV were slightly more cytotoxic than those treated with Cl_2, NaOCl and BCDMH, all of which were found to have similar calculated cytotoxicity. As with swimming pool type, these results are likely a reflection of the chloral hydrate concentrations and may not be directly correlated with treatment type. The potential impact of UV on DBPs was discussed in further detail in **Section 4.4.4**. A slight increase in the formation of chloral hydrate was observed in a lab scale study where real pool water samples were exposed to low pressure UV (Cimetiere and De Laat, 2014), although other studies have reported lower concentrations of chloral hydrate with UV treatment (e.g. Hansen et al., 2013). Wlodyka-Bergier and Bergier (2016) reported an increase in chloral hydrate formation during the chlorination of several amino acids when UV was applied, compared to non UV irradiated samples. Spiliotopoulou et al. (2015) reported that post-UV chlorination induced the secondary formation of several DBPs in their study of real pool waters exposed to UV irradiation and chlorination. They also reported that, while

total DBP concentrations were generally unchanged, UV irradiation was found to increase the proportion of brominated DBPs (Spiliotopoulou et al., 2015), which, in addition to the formation of chloral hydrate, may help to explain the slightly higher cytotoxicity of the UV treated pools.

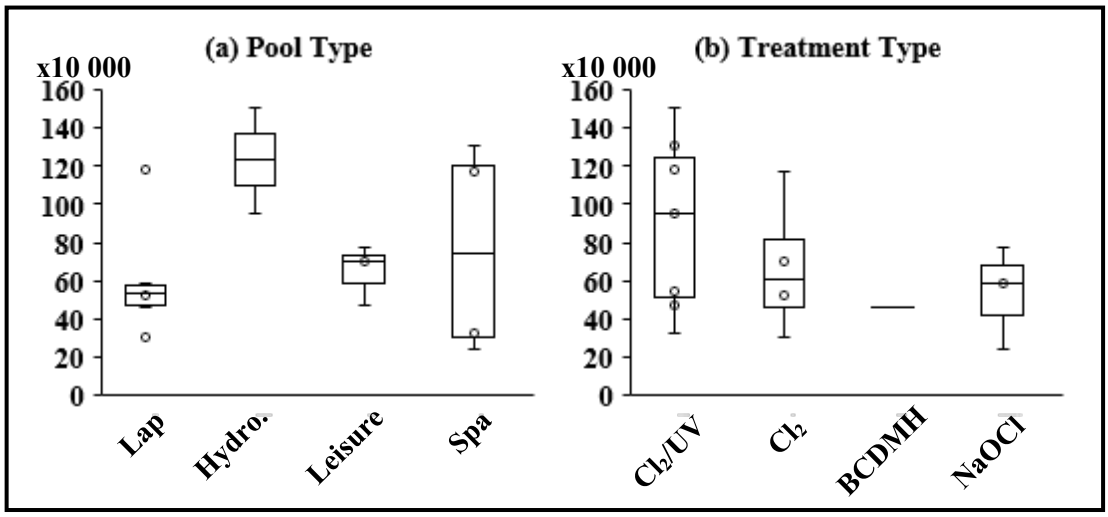

Figure 4-4: Comparison of the estimated cytotoxicity of pools based on **(a)** pool type and **(b)** treatment type. Y-axis represents an arbitrary number obtained by calculation (**Section 4.3.5**). **BCDMH**: Bromochlorodimethylhydantoin. **Cl_2**: Chlorine gas. **Cl_2/UV**: Chlorine gas combined with ultraviolet irradiation. **Hydro.**: Hydrotherapy. **NaOCl**: Sodium hypochlorite.

It was expected that the pool treated with BCDMH (C1) would exhibit the highest level of calculated cytotoxicity due to the higher occurrence and concentration of brominated DBPs, which are reported to be generally more cytotoxic than their corresponding chlorinated analogues (Wagner and Plewa, 2017). However, this trend was not observed (**Figure 4-4(b)**), possibly due to (i) the inability to quantify tribromoacetic acid despite it appearing to be present in high concentration in the BCDMH treated pool, and (ii) the lower concentration (0.07 µM) of tribromoacetaldehyde (bromal hydrate) in the BCDMH treated pool (C1) compared to the higher concentrations (1.2 to 7.9 µM) of chloral hydrate in the chlorinated pools, and (iii) the limited sample size of pools treated with bromine. In comparison to chloral hydrate, bromal hydrate has been shown to be less stable under typical drinking water conditions (pH 7.2 and 8.2, ambient temperature), degrading to afford tribromomethane (Koudjonou and LeBel, 2006). While this study was conducted under typical drinking water conditions, the detection (0.07 µM) of tribromomethane in the BCDMH treated pool (C1) suggests the lower than expected cytotoxicity in this pool is, at least in part, due to the degradation of bromal hydrate to tribromomethane, which is less cytotoxic in nature (Wagner and Plewa, 2017).

DBP derived cytotoxicity calculated for the pools was compared to that calculated for the filling water. In this context, comparisons between calculated cytotoxicity of pools at Facility A (A1 to A4) and that calculated for the corresponding filling water (A5) must be considered outliers due to the unusually low chlorine residuals measured in the filling water (A5), which has likely led to an unusually low DBP occurrence and hence a low estimate of the filling waters' cytotoxicity. Excluding Facility A, as expected, due to the higher prevalence of DBPs, all pools were found to be higher in calculated cytotoxicity than their corresponding filling waters. Considering only pools at Facilities B to F, all pools were calculated to be between 4 and 342 times higher (98 times higher on average) in calculated cytotoxicity than their corresponding filling waters. The hydrotherapy pool showed the greatest increase (343 times higher), which as discussed earlier in this section, is likely a result of the greater release of several anthropogenic constituents and subsequent chloral hydrate formation in hydrotherapy pools. As these anthropogenic constituents are also generally higher in spa pools, we expected a similarly greater cytotoxicity in spa pools in comparison to their filling waters. However, in this study, spa pools were calculated to be between 12 and 114 times higher in cytotoxicity than their filling waters, which is more similar to the difference observed for lap pools (29 to 118 times higher). This unexpected finding may be a combined result of (i) the larger variability of DBPs (and subsequent cytotoxicity) of spa waters, and (ii) the hydrotherapy pool sample may have been a high outlier in terms of cytotoxicity. Similarly, the significantly lower increase in cytotoxicity calculated for leisure pools (45 to 67 times higher than corresponding filling waters) may also be a reflection of the small sample size used (two pools).

Similarly, as DBP concentrations were generally greater in Cl_2/UV treated pools, it was expected that compared to filling waters, these pools would show the greatest increase in calculated cytotoxicity among the different treatment methods. In almost all cases, Cl_2/UV treated pools had the highest increase in cytotoxicity, 103 to 342 times higher than corresponding filling waters, compared to pools treated by other means. Pools treated with BCDMH and Cl_2 had similar increases in cytotoxicity compared to filling waters, where an average increase of 111 and 81 times higher was calculated, respectively. Pools treated with NaOCl were calculated to have the smallest increase (compared to filling waters) of estimated cytotoxicity, 14 to 45 times higher. These difference in increased cytotoxicity between pools of different treatment types is likely largely due to the occurrence (and speciation) of DBPs present, as discussed earlier in this section, as well as in **Section 4.4.4**.

Although the exclusion of results from Facility A aimed to minimise the effect of outliers in this comparison, it inadvertently weakens the ability to assess trends in this context by decreasing the sample size of pools of different types and/or treatment methods. While the

results presented here provide a general indication of the differences in cytotoxic nature of pools of differing types and/or treatment methods, future work should investigate a larger number of pools of any given type and/or treatment method before any definite conclusions can be drawn.

4.4.6. Correlations between Disinfection By-Products and other Swimming Pool Parameters

Table 4-2 summarises the correlations between the investigated DBP classes (a molar sum of the individual DBPs of a given class) and (i) other investigated DBP classes, (ii) general water quality parameters of the pools and (iii) the estimated cytotoxicity of the pools.

HAAs were strongly significantly correlated to HKs ($p=0.002$) and HALs ($p=0.007$) which is in agreement with Zhang et al. (2015), who observed a correlation between HAAs and chloral hydrate in their study of 14 pools treated with various chlorine based disinfectants (NaOCl, NaOCl plus ozone, chlorine dioxide and trichloroisocyanuric acid). No correlation, however, was observed between HAAs and chloral hydrate for chlorinated pools investigated by Lee et al. (2010). In the current study, HAAs also showed a moderate significant correlation with HANs ($p=0.028$) and *N*-nitrosamines ($p=0.043$), although no correlations between HAAs and HANs have previously been reported (Lee et al., 2010). HKs were strongly significantly correlated to HALs ($p<0.001$), HANs ($p=0.004$) and *N*-nitrosamines ($p=0.008$). A strong significant correlation was observed between HALs and HANs ($p<0.001$), as well as between HAAms and *N*-nitrosamines ($p=0.003$). Lee et al. (2010) also reported a correlation between HALs and HANs in their study of 84 indoor swimming pools treated by various disinfectants.

Although THMs have previously been demonstrated to be correlated to HAAs, HANs and chloral hydrate in swimming pools (Lee et al., 2010; Zhang et al., 2015), in the current study, THMs were the only investigated DBP class that was not significantly correlated ($p>0.05$) with any other DBP. This may be due to the volatile nature of THMs, and hence their lower concentrations in pools. The significant correlations between several classes of DBPs in this study are indeed consistent with the origin of most DBPs being organic precursors released from swimmers (bather load).

Table 4-2: Evaluation of correlations amongst the different investigated disinfection by-product classes, as well as between the investigated disinfection by-product classes and general water quality parameters and estimated cytotoxicity. Values are displayed as "Spearmans correlation rank (p-value)" and rounded to 3 significant figures. Values shown in **BOLD** were found to be moderately significantly correlated ($p<0.05$), while values including a '*' indicate a significantly strong correlation ($p<0.01$) was observed.

	Total DBPs	Trihalomethanes	Haloacetic Acids	Haloketones	Haloacetaldehydes	Haloacetontriles	Halonitromethanes	Haloacetamides	*N*-Nitrosamines
Temperature	0.399 (0.141)	-0.173 (0.537)	**0.559 (0.031)**	0.248 (0.372)	0.327 (0.234)	0.470 (0.077)	0.060 (0.831)	**0.002 (0.995)**	0.095 (0.737)
pH	0.032 (0.909)	**0.710 (0.003)***	-0.114 (0.685)	0.021 (0.940)	-0.123 (0.661)	-0.161 (0.567)	0.199 (0.476)	0.538 (0.039)	0.127 (0.652)
UV_{254}	**0.750 (0.001)***	0.425 (0.114)	0.454 (0.089)	**0.604 (0.017)**	0.311 (0.260)	0.246 (0.376)	0.121 (0.668)	**0.761 (0.001)***	**0.904 (<0.001)***
$SUVA_{254}$	0.407 (0.132)	0.218 (0.435)	**0.521 (0.046)**	**0.586 (0.022)**	0.168 (0.550)	-0.021 (0.940)	-0.441 (0.100)	0.250 (0.369)	**0.514 (0.050)**
Total Chlorine	0.420 (0.119)	0.272 (0.327)	**0.597 (0.019)**	**0.631 (0.012)**	0.259 (0.351)	0.216 (0.439)	-0.393 (0.148)	0.055 (0.845)	0.409 (0.13)
Non-Purgeable Organic Carbon	**0.886 (<0.001)***	0.354 (0.215)	**0.675 (0.008)***	**0.908 (<0.001)***	**0.635 (0.015)**	0.477 (0.085)	0.103 (0.726)	**0.727 (0.003)***	**0.916 (<0.001)***
Total Nitrogen	0.318 (0.248)	-0.211 (0.451)	0.349 (0.203)	0.384 (0.157)	**0.735 (0.002)***	**0.622 (0.013)**	0.063 (0.822)	0.089 (0.751)	0.055 (0.845)
Cytotoxicity	**0.836 (<0.001)***	-0.104 (0.713)	**0.600 (0.018)**	**0.764 (0.001)***	**0.979 (<0.001)***	**0.900 (<0.001)***	0.169 (0.547)	0.393 (0.147)	**0.525 (0.044)**

Table 4-2 continued

	Total DBPs	Trihalomethanes	Haloacetic Acids	Haloketones	Haloacetaldehydes	Haloacetontriles	Halonitromethanes	Haloacetamides	*N*-Nitrosamines
Trihalomethanes	0.068 (0.810)								
Haloacetic Acids	**0.743 (0.002)***	-0.061 (0.830)							
Haloketones	**0.839 (<0.001)***	0.079 (0.781)	**0.732 (0.002)***						
Haloacetaldehydes	**0.800 (<0.001)***	-0.154 (0.585)	**0.661 (0.007)***	**0.804 (<0.001)***					
Haloacetonitriles	**0.714 (0.003)***	-0.157 (0.576)	**0.564 (0.028)**	**0.696 (0.004)***	**0.879 (<0.001)***				
Halonitromethanes	0.151 (0.591)	0.163 (0.562)	-0.248 (0.374)	-0.006 (0.983)	0.042 (0.881)	0.169 (0.547)			
Haloacetamides	**0.557 (0.031)**	0.436 (0.104)	0.050 (0.860)	0.318 (0.248)	0.279 (0.315)	0.236 (0.398)	0.392 (0.148)		
N-Nitrosamines	**0.843 (<0.001)***	0.175 (0.533)	**0.529 (0.043)**	**0.654 (0.008)***	0.454 (0.089)	0.368 (0.177)	0.078 (0.781)	**0.707 (0.003)***	

HAAs, HKs, HALs, HAAms and *N*-nitrosamines were significantly correlated ($p<0.001$ to 0.015) with NPOC, suggesting, as expected, that NPOC is a key precursor for the formation of these DBPs in pool waters. No correlation between HAAs and total organic carbon (TOC) was observed by Wang et al. (2014) in their study of nine swimming pools and three spas, and none of the investigated DBPs (four THMs, nine HAAs, chloral hydrate, four HANs, two HKs and trichloronitromethane) were found to correlate with TOC in a study of 14 swimming pools by Zhang et al. (2015). Although not observed in the current study, TOC levels in pools have previously been correlated to THM concentrations (Chu and Nieuwenhuijsen, 2002; Peng et al., 2015), where a time delay in correlation of up to 2 days was observed (Peng et al., 2015). Total DBP concentrations (the molar sum of THMs, HAAs, HANs and chloral hydrate) were correlated to both TOC and nitrate levels in a study of 84 swimming pools by Lee et al. (2010), with similar results reported in a controlled laboratory study where the chlorination of model compounds of human origin (hair, saliva, skin, urine and lotion) was carried out in a model swimming pool (Kim et al., 2002). In the current study, HALs and HANs were significantly correlated with TN (p values 0.002 and 0.013, respectively), indicating the potential formation of these DBPs from nitrogen containing precursors.

HAAms, *N*-nitrosamines and HKs were the only DBP classes that showed a significant correlation ($p<0.001$ to 0.017) with UV_{254}, suggesting that organic matter containing conjugated double bonds and aromatic moieties may act as precursors to these DBPs in swimming pool waters. While the current study was mostly in agreement with Zhang et al. (2015) in terms of the lack of correlation between THMs, HAAs, HANs and trichloronitromethane with UV_{254}, Zhang et al. (2015) reported no correlation between HKs and UV_{254}. This difference is likely due to the fact that the current study investigated a larger number of HKs. Although temperature was only significantly correlated with HAAs ($p=0.031$), temperature still likely has a major impact on the formation of other DBP classes. The absence of correlations observed between temperature and other DBP classes (such as THMs or HALs) is likely due to the volatile nature of these DBPs, where higher temperatures would increase their evaporation rate and hence decrease their occurrence in the swimming pool waters. THMs, however, were strongly correlated with pH ($p=0.003$), suggesting that pH has a major impact on the formation of THMs in swimming pools. This is consistent with several laboratory based studies of Hansen et al. (2012a, 2012b, 2013a), where decreasing formation of THMs with decreasing pH values was reported. With only just over half of the pools investigated in the current study meeting their operational guidelines for pH, these findings highlight the importance of correct pH control in pools as one method to mitigate THM formation.

HAA and HK concentrations were found to be moderately correlated with total chlorine equivalent concentrations ($p=0.019$ and 0.012, respectively), similar to the findings of Zhang et al. (2015), who reported correlations between both HAA and THM concentrations and total chlorine equivalent concentrations. Although no correlation was observed for chloral hydrate, HAA and THM concentrations were also found to correlate with free chlorine equivalent concentrations (Zhang et al., 2015). In the current study, however, no significant correlation ($p>0.05$) was observed between any of the investigated DBP classes and free chlorine equivalent concentrations (**Table A4-12**). This absence of correlations between free chlorine equivalent concentrations may be due to the higher free chlorine equivalent concentrations compared to other studies, as THM and HAA concentrations have been shown to be linearly correlated to free chlorine concentrations at low free chlorine levels (Kanan, 2010). The dynamic nature of free chlorine equivalent concentrations in pools, along with the limited sampling events in this study, may also be factors contributing to the absence of the expected correlations. Additionally, poor pool management, as evidenced by many pools having free and total chlorine equivalent concentrations and/or pH values outside operational guidelines (**Section 4.4.1**), may have hindered the possibility to observe the correlation(s) between free chlorine equivalent concentrations and DBPs that are theoretically expected. Furthermore, no significant correlations ($p>0.05$) were observed between conductivity, dissolved oxygen, time of sampling, pool volume or water turnover rate with any of the investigated DBP classes in this study (**Table A4-12**).

Correlations between each DBP class and the calculated chronic cytotoxicity of the pool water were evaluated. Of the eight investigated DBP classes, three (THMs, HNMs and HAAms) did not show any significant correlation ($p>0.05$) with the calculated cytotoxicity, which is likely a combination of their lower concentrations and/or lower cytotoxicity in comparison to other DBP classes. Despite their high concentrations in pools, HAAs were only moderately significantly correlated ($p=0.018$) with cytotoxicity, as were *N*-nitrosamines ($p=0.044$). HKs and HANs were strongly significantly correlated with cytotoxicity ($p<0.001$), while HALs showed the strongest correlation ($p<0.001$). This study shows that while THMs and HAAs are often used as an indication of chemical water quality in pools, at the concentrations measured in pools, other DBP classes, particularly HALs, HANs and HKs, must be considered when assessing the health impact.

4.5. Conclusions

The current study provides a comprehensive DBP analysis of various pool types (lap, leisure, spa and hydrotherapy) employing several different treatment methods (Cl_2, Cl_2/UV, NaOCl and BCDMH), where the occurrence of 64 DBPs and several general water quality

parameters were measured. The concentrations of DBPs were generally equal to or greater than those previously reported, which is likely due to the higher disinfectant residuals required to be employed in Australian pools, although poor pool management, in particular control of chlorine residuals and pH, is a key factor in the occurrence of DBPs in pools. Of the 64 DBPs investigated, approximately 70% were measured in at least one pool, with 17 (26%) DBPs measured in all chlorinated waters. Considering average molar concentrations across all pools, the predominant DBP class was the HALs (representing 41% of the total DBP concentration), followed by HAAs (28%), *N*-nitrosamines (18%), HAAms (6%), THMs (4%) and HANs (2%), with HKs and HNMs together representing less than 1% of the total DBP molar concentration. Chloral hydrate was the predominant DBP in all chlorinated waters (202 to 1313 μg L^{-1}), while tribromomethane was the predominant DBP in the BCDMH treated pool (132 μg L^{-1}).

Filling waters (disinfected distributed drinking waters) were found to be an insignificant source of most DBPs in pools, with all pools found to contain almost 100 times higher total molar DBP concentrations than their corresponding filling waters. HAAs, HKs, HALs, HAAms and *N*-nitrosamines were significantly correlated ($p<0.001$ to 0.015) with NPOC, suggesting that NPOC is a determinant factor for the formation of these DBPs in swimming pool waters. Significant correlations were also observed between several of the investigated DBP classes and TN and/or $SUVA_{254}$.

THMs, HNMs and HAAms were found to not significantly contribute to the total estimated chronic cytotoxicity, which is likely due to their lower concentrations (e.g. due to lower rate of formation or loss via volatilisation) and/or lower cytotoxic nature compared to the other DBPs investigated. HALs, mainly chloral hydrate, were calculated to be the greatest contributor of cytotoxicity in chlorinated pools, representing on average 92.5% (HALs) and up to 98.7% (chloral hydrate) of the overall total calculated chronic cytotoxicity. Considering average calculated cytotoxicity values, hydrotherapy pools were found to be the most cytotoxic compared to other pool types, followed in order by spa, leisure and lap pools. Pools treated by Cl_2/UV were calculated to be slightly more cytotoxic than those employing other treatment methods (Cl_2, NaOCl or BCDMH), however this is likely due to the higher chloral hydrate concentrations in Cl_2/UV treated pools rather than a reflection of treatment methods.

This study is the first investigation of several HNMs (chloronitromethane, dichloronitromethane, dibromonitromethane bromodichloronitromethane and dibromochloronitromethane) and the first report of bromodichloroacetaldehyde (46 μg L^{-1}) and bromochloroacetaldehyde (3 μg L^{-1}) in swimming pools. The detection of a HNM (bromonitromethane, 0.8 μg L^{-1}) in a BCDMH treated pool has previously not been achieved,

with this study also the first known investigation of *N*-nitrosamines in a pool treated with BCDMH. Furthermore, this study significantly adds to the limited existing knowledge of DBPs in both Australian and BCDMH treated pools.

4.6. Acknowledgements

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CHAPTER 5

500 DAYS OF SWIMMERS: THE CHEMICAL WATER QUALITY OF SWIMMING POOL WATERS FROM THE BEGINNING

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This Chapter includes the co-authored paper '*500 Days of swimmers: The chemical water quality of swimming pool waters from the beginning*', which has been accepted for publication in *Environmental Science and Pollution Research*. The bibliographic details of the co-authored paper, including all authors are:

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I, Rhys Aaron Ainsley Carter, contributed to the publication by: undertaking a review of the current literature in this field, undertaking all field work, laboratory experiments and data analysis, being the primary writer (including creating figures and tables), and editing and finalising the manuscript.

I, as a Co-Author, endorse that this level of contribution by the candidate indicated above is appropriate.

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5.1. Abstract

Many studies of disinfection by-products (DBPs) in pools have focused on haloacetic acids, trihalomethanes and chloramines, with less studies investigating the occurrence of other DBPs, such as haloketones, haloacetaldehydes, haloacetontiriles, halonitromethanes and haloacetamides. Furthermore, while many studies have achieved a broadscreen analysis across several pools, fewer studies have followed the water quality of pools over time, with information regarding the production and fate of DBPs in pools over extended periods (e.g. >1 year) being limited. This study reports the occurrence of 39 DBPs and several general water quality parameters in two newly built and filled swimming pools over fifteen months, where investigations began prior to opening. DBP concentrations measured in this study were generally similar to or higher than those previously reported in chlorinated pools, with concentrations of chloroacetic acid, dichloroacetic acid, trichloroacetic acid and chloral hydrate (trichloroacetaldehyde) in some samples being higher than previously reported maximum concentrations. Considering both pools, lower concentrations of DBPs were measured in the pool where a steady state non-purgeable organic carbon concentration was achieved, highlighting the importance of the establishment of a steady state balance of mineralisation versus addition of organic carbon to reduce precursors for DBP formation in pools. Pools were found to exhibit significantly higher estimated cytotoxicity than their filling water, which reflects the significantly higher concentrations of DBPs measured in the pools in comparison to the filling water. Chloral hydrate accounted for up to 99% the total estimated cytotoxicity and was found to be correlated to the number of pool entries, suggesting that swimmers may be a potential source of chloral hydrate precursors in pools. The presence and subsequent peak of non-purgeable organic carbon and DBPs prior to, and soon after, opening suggest that the building process and/or new pool infrastructure may have had a significant impact on the chemical water quality, particularly on DBP formation. This study includes the first quantification of bromochloroacetaldehyde, bromodichloroacetaldehyde, bromochloronitromethane and dichloronitromethane in chlorinated swimming pools, and provides important new knowledge on the long-term trends of DBPs in pools.

5.2. Introduction

While the aim of water disinfection is to kill pathogens and minimise microbial disease risk, it can also lead to the formation of disinfection by-products (DBPs). Many DBPs have been reported to be potentially genotoxic, neurotoxic and/or cytotoxic, with several DBPs also exhibiting a potentially carcinogenic, teratogenic and/or mutagenic nature (Richardson et al., 2007). As such, disinfection should be controlled in order to minimise DBP formation while maintaining significant protection against the microbial risk, which is generally much greater

than that posed by DBPs. While more is known about DBPs in drinking, waste and recycled waters, where over 700 DBPs have been identified (Plewa and Richardson, 2017), less is understood about DBPs in the swimming pool environment. Due to the continual input of organic matter (e.g. via filling water, human body excretions, personal care products and pharmaceuticals) and continual availability of disinfectants (e.g. chlorine), swimming pools are a unique environment compared to other water matrices, particularly in terms of DBPs. Furthermore, over 100 new DBPs have been identified exclusively in swimming pools or spas (Daiber et al., 2016; Richardson et al., 2010), highlighting the unique nature of these waters.

As with other water types, many investigations of DBPs in swimming pool waters have focused on the occurrence and/or formation of trihalomethanes (THMs), haloacetic acids (HAAs) and inorganic chloramines (particularly trichloramine), with fewer studies including haloacetonitriles (HANs), particularly dichloroacetonitrile, and chloral hydrate, the hydrated analogue of trichloroacetaldehyde. Very few studies have investigated the occurrence and/or formation of halonitromethanes (HNMs), haloacetamides (HAAms), haloketones (HKs) or other haloacetaldehydes (HALs) in swimming pool waters. Furthermore, very limited studies of DBPs in Australian swimming pools have been reported (Carter et al., 2015; Kelsall and Sim, 2001; Teo et al., 2016a, 2016b; Yeh et al., 2014), where higher disinfectant residuals (2 to 4 mg L^{-1} as Cl_2) are employed.

Many studies reported the occurrence of DBPs in pools by carrying out a broadscreen analysis, that is, the analysis of several pools for a suite of DBPs, where different pool types or those employing different treatment methods were investigated (Carter and Joll, 2017). While these studies have led to a better understanding of DBPs in pools, they provide minimal information regarding Long term trends of these DBPs and their fate in swimming pools. While some studies have followed DBPs over a matter of days (Gérardin et al., 2015; Judd and Black, 2000; Peng et al., 2015; Weng and Blatchley, 2011) or months (Golfinopoulos, 2000; Kanan and Karanfil, 2011; Lahl et al., 1981; Yeh et al., 2014), limited investigations exist for longer periods (e.g. >1 year) (Kristensen et al., 2010; Simard et al., 2013; Zare Afifi and Blatchley, 2015). Of these aforementioned time based studies, most have limited their focus to the occurrence of THMs and/or chloramines (Gérardin et al., 2015; Golfinopoulos, 2000; Judd and Black, 2000; Kristensen et al., 2010; Lahl et al., 1981; Peng et al., 2015; Weng and Blatchley, 2011; Yeh et al., 2014), with only a few also investigating other DBPs, including HAAs, HNMs, HANs, *N*-nitrosodimethylamine (NDMA) and/or cyanogen halides (Kanan and Karanfil, 2011; Simard et al., 2013; Zare Afifi and Blatchley, 2015). With fewer studies encompassing a large number of DBP classes, knowledge of DBP trends over a large time period is limited.

This study expands on the knowledge of DBP occurrence in swimming pools, particularly in Australian conditions, by reporting the occurrence of 39 DBPs (across seven different DBP classes), as well as several general water quality parameters, in two chlorinated swimming pools. This work follows the concentrations of these investigated DBPs over fifteen months, providing information to assess any weekly, monthly or seasonal trends, an area where knowledge is lacking. While Yeh et al., (2014) investigated limited parameters in a pool from a complete water replacement, the current study is the first investigation of the water quality and occurrence of DBPs in newly built and filled swimming pools, where investigations began prior to the opening of the facility. The filling water for the pools was investigated concurrently to assess its impact, if any, on DBP occurrence. Statistical analysis between DBPs, general water quality and/or operational parameters was performed to assess any correlations between these parameters. Furthermore, the chronic cytotoxicity of the pool water samples was estimated based on calculation, in order to provide an idea of the health impact these DBPs may pose, at the concentrations measured.

5.3. Methodology

5.3.1. Analytical Standards and Reagents

All chemicals and reagents used were of analytical grade purity (>98%) and purchased from a range of suppliers including AccuStandard (Connecticut, USA), CanSyn Chemical Corporation (Ontario, Canada), CDN isotopes (Quebec, Canada), Sigma Aldrich (Sydney, Australia), Thermo Fisher (Victoria, Australia). Ultrapure water, purified by an ELGA PURELAB Ultra purification system (18.2 MΩ-cm resistivity), was used in all experiments.

5.3.2. Preparation of Standard, Working and Calibration Solutions

DBP standard stock solutions (1 g L^{-1} of each DBP) in acetone were prepared by DBP class, i.e., one stock solution containing each individual DBP of a given class. Bromochloroacetonitrile (BCAN), trichloronitromethane (TCNM) and 1,1,1-trichloropropanone (1,1,1-TCP) were added to relevant DBP standard stock solutions by dilution of individual purchased solutions (5 g L^{-1} in acetone). Similarly, haloacetic acid working solutions were prepared by dilution of a purchased stock solution containing all nine HAAs (2 g L^{-1} in methyl *tert*-butyl ether (MTBE)). Surrogate standard and internal standard stock solutions were prepared by weighing neat compound(s) into acetone. Working DBP, surrogate standard and internal standard solutions were prepared by dilution of appropriate stock solution(s) into acetone. Calibration standards were prepared by fortifying ultrapure water samples with the desired DBP standard, surrogate standard and internal standard working solution(s), as per individual method requirements (**Section 5.3.3**).

5.3.3. Analytical Methods

Analytical methods employed in this study are summarised in **Table A5-1**. Free and total chlorine equivalent concentrations, pH, conductivity, dissolved oxygen and temperature were measured at time of collection using a Pocket Colorimeter (HACH; 5870000) or a portable multimeter (HACH; HQ40D). Non-purgeable organic carbon (NPOC) and total nitrogen (TN) were analysed using high temperature catalytic combustion with non-dispersive infrared detection on a Shimadzu total organic carbon analyser (TOC-L) equipped with a total nitrogen measuring unit (TNM-L). The THMs (trichloromethane (chloroform), bromodichloromethane, dibromochloromethane and tribromomethane (bromoform)) were analysed by headspace solid-phase microextraction (HS SPME) gas chromatography-mass spectrometry (GC-MS) using a simplified version of the method of Allard et al. (2012). HKs (chloropropanone, 1,1-dichloropropanone, 1,3-dichloropropanone and 1,1,1-trichloropropanone (CP, 1,1-DCP, 1,3-DCP and 1,1,1-TCP, respectively)) and HALs (dibromoacetaldehyde, bromochlorocetaldehyde, bromodichlorocetaldehyde, dibromochlorocetaldehyde, trichlorocetaldehyde and tribromoacetaldehyde (DBAL, BCAL, BDCAL, DBCAL, TCAL (chloral hydrate; CH) and TBAL, respectively)) were analysed simultaneously by liquid-liquid extraction (LLE) followed by GC-MS by an adaption of Standard Method 551.1 (US EPA, 1995). HAAs (chloroacetic acid, bromoacetic acid, dichloroacetic acid, dibromoacetic acid, bromochloroacetic acid, bromodichloroacetic acid, dibromochloroacetic acid, trichloroacetic acid and tribromoacetic acid (CAA, BAA, DCAA, DBAA, BCAA, BDCAA, DBCAA, TCAA and TBAA, respectively)) were analysed as their methyl esters by LLE derivatisation GC-MS as per Standard Method 552.2 (US EPA, 2003). HANs (chloroacetonitrile, bromoacetonitrile, dichloroacetonitrile, dibromoacetonitrile, bromochloroacetonitrile and trichloroactetonitrile (CAN, BAN, DCAN, DBAN, BCAN and TCAN, respectively)), HNMs (dichloronitromethane, dibromonitromethane, bromochloronitromethane, bromodichloronitromethane, dibromochloronitromethane, trichloronitromethane and tribromonitromethane (DCNM, DBNM, BCNM, BDCNM, DBCNM, TCNM (chloropicrin) and TBNM (bromopicrin), respectively)), and HAAms (dichloroacetamide, dibromoacetamide and trichloroacetamide (DCAAm, DBAAm and TCAAm, respectively)) were analysed simultaneously by LLE GC-MS, using a simplified version of the method of (Carter et al., 2019). All analyses were performed in duplicate with average results presented. For GC-MS analysis, selective ion monitoring (SIM) was used for quantification with results normalised by the use of internal and/or surrogate standards where appropriate.

5.3.4. Water Samples

Two newly built and filled public swimming pools in Perth, Western Australia were investigated from prior to opening (November 2015) until March 2017, with permission granted by the Department of Health (Western Australia). In order to ensure confidentiality, pools have been de-identified and coded as Pool A and Pool B. Pool A was a 20 m (4 lane) outdoor/covered leisure pool (300 kL), disinfected by chorine gas and equipped with ultraviolet (UV) treatment, with a target operational temperature of 30 °C. Pool B was a 50 m (10 lane) outdoor lap pool (1.86 ML), disinfected by chlorine gas and employing cyanuric acid (20 to 50 mg L^{-1}) as a chlorine stabiliser, with a target operational temperature of 27 °C. Both pools were filtered (~1 μm) via individual diatomaceous earth filters and the pools were operated/treated independently from one another. The target pH of both pools was 7.2 to 7.8, with free chlorine equivalent residual concentration targets of 2.5 to 3 and 3 to 3.5 mg L^{-1} for Pool A and B, respectively. Additional information regarding the operation of the pools is presented in **Tables A5-2 and A5-3**.

Samples were collected at the centre of the longest side of each pool, from approximately 20 to 30 cm below the water's surface and 50 cm from the pools edge. Samples were collected directly into amber bottles with no headspace and the oxidant residual quenched (110% of the measured total chlorine equivalent molar concentration as per **Table A5-1**), before storage at 4 °C until analysis (24 to 72 hours). Where possible, water samples were collected at the same time of day (5 to 6 am) on each sampling occasion. Initially, samples were collected twice daily (morning and night), then when sampling frequency decreased, they were collected daily, weekly, biweekly and finally monthly for the duration, totalling thirty-three sampling events. The pools' filling water, disinfected distributed drinking water, was collected regularly for analysis.

5.3.5. Cytotoxicity Evaluation

The chronic cytotoxicity for most investigated DBPs was evaluated based upon C_{50} values available in the literature (Wagner and Plewa, 2017). HKs were excluded from cytotoxicity evaluation as C_{50} values do not currently exist for these compounds. In these calculations, the concentration (M) of each DBP measured was divided by its C_{50} value (M), resulting in a dimensionless number. Finally, these results were multiplied by 10^6 to make the numbers more readable.

5.3.6. Statistical Evaluation

Spearman's rank correlation coefficient was used to evaluate any correlations that may exist amongst the DBPs and other water quality parameters of the investigated swimming

pools. Statistical analysis was performed using SPSS Statistics version 24 software (IBM, Armonk, New York).

5.4. Results and Discussion

5.4.1. General Water Quality Parameters

A summary of several general water quality and operational parameters, free and total chorine equivalent residual concentrations, NPOC and TN concentrations, pH, temperature, dissolved oxygen and conductivity, measured in each pool is presented in **Table A5-4**. Free chlorine equivalent concentrations were, on average, 3.4 and 3.3 mg L^{-1} in Pools A and B, respectively, although concentrations up to 6.6 mg L^{-1} were measured. Minimum free chlorine equivalent concentrations that met local regulations (greater than 3 and 2 mg L^{-1} for stabilised and non-stabilised pools, respectively (Standards Australia, 2002)) were measured in only 88% and 54% of samples taken from Pools A and B, respectively. Total chlorine equivalent concentrations, measured between 1.8 to 7.1 mg L^{-1} across both pools, were outside the local guideline (must not exceed 30% of the measured free chlorine (Standards Australia, 2002)) on three occasions (<11% of the time), in which all cases were limited to Pool A. It should be noted, however, that due to technical issues with the chlorine dosing unit, unusually high free and total chlorine equivalent concentrations (15 to 26 and 15 to 29 mg L^{-1}, respectively) were measured in Pool A on three successive days soon after opening (day 1.5, 2 and 3). These concentrations were excluded in the subsequent statistical analysis, as well as in the determination of minimum, maximum and average values for free and total chlorine equivalent concentrations. pH levels in Pools A (6.6 to 7.7) and B (6.8 to 7.8) met local regulations (7.2 to 7.8 (Standards Australia, 2002)) in only 73 and 76% of the samples, respectively. NPOC concentrations ranged between 2.8 to 30 mg L^{-1} in Pool A and between 1.7 to 21 mg L^{-1} in Pool B, while TN concentration between 0.1 to 16 and 4.5 to 21 mg L^{-1} (for Pools A and B, respectively) were measured. It should be noted that the use of isocyanuric acid in Pool B contributed to the concentrations of NPOC and TN measured in Pool B.

5.4.2. Occurrence of Disinfection By-Products in the Swimming Pool Waters

Of the thirty-nine investigated DBPs, only thirteen were not detected in any samples of either Pool A or B. Despite being measured in previous pool water studies at concentrations up to 53 µg L^{-1} (Carter et al., 2015; Daiber et al., 2016; Hang et al., 2016; Kanan, 2010; Manasfi et al., 2016; Tardif et al., 2016, 2015; Yeh et al., 2014), BAA, DBCAA, TBAA, BAN, DBAL, TBAL, DBAAm, DCAAm, TCAAm and TBNM were all below their respective limits of detection (0.2 to 1.1 µg L^{-1}) in all pool samples investigated in the current study. DBCNM, DBNM and BDCNM were below their respective limits of detection (0.7 µg L^{-1}) in all pool

samples in the current study, consistent with other studies of chlorinated pools as these DBPs have not previously been reported. The majority of these DBPs are brominated and their absence in the investigated pools may be attributed to the lower availability of bromine (via bromide oxidation) compared to that of chlorine, and hence lower formation of brominated DBPs. In addition, HNMs, particularly those that are trihalogenated, have been shown to be unstable in chlorinated waters (Liew et al., 2012), which may explain the absence of DBCNM and BDCNM to date in swimming pool waters and, although reported at concentrations up to 1.2 μg L^{-1} by Yeh et al. (2014), the absence of TBNM in all pools investigated by Manasfi et al. (2016). In the presence of free chlorine, HAAms are rapidly degraded, presumably due to their conversion to HAAs (Chu et al., 2010), the most likely reason for their absence in the current study.

A detailed summary of the concentrations of DBPs measured in Pools A and B is provided in **Table 5-1**. Furthermore, **Table 5-2** summarises the contribution of each DBP class to the total concentration of DBPs measured, on any given day in each pool, where concentrations were compared on a molar basis.

Haloacetic acids were generally the predominant class of DBP measured in both pools, where total HAA concentrations (also referred to as HAA9; the sum of CAA, BAA, DCAA, DBAA, BCAA, BDCAA, DBCAA, TCAA and TBAA concentrations) represented between 34 to 99% and between 58 to 97% of the total measured DBP molar concentrations in any sample, of Pool A and B, respectively. DCAA and TCAA were detected in all pool samples at concentrations significantly higher than any other HAA measured in this study, up to 26 and 11 mg L^{-1}, respectively. These concentrations are generally higher than those previously reported for chlorinated swimming pools (as summarised by Carter and Joll (2017)), and were up to 4x and 11x higher than the maximum previously reported concentrations for DCAA and TCAA, respectively (Yeh et al., 2014). TCAA and DCAA are known degradation products of CH in waters containing chlorine (Barrott, 2004). As the pools in this study were found to contain significant CH concentrations (discussed in detail below), chlorine degradation of CH may be a significant formation pathway for the high DCAA and TCAA concentrations measured in this study.

While Wang et al. (2011) reported a higher CAA concentration (up to 300 μg L^{-1}) in some of the chlorinated pools in their study, CAA concentrations measured in Pool B (3.3 to 180 μg L^{-1}) were similar to, or only slightly higher than, those reported in most other studies, <0.5 to 120 μg L^{-1} (Berg et al., 2000; Cardador and Gallego, 2011; Carter et al., 2015; Hang et al., 2016; Sa et al., 2012; Tardif et al., 2016; Yeh et al., 2014). In Pool A, however, CAA concentrations were generally higher than the previously reported concentrations,

Table 5-1: Detection frequency (%) and concentrations (µg L^{-1}) of disinfection by-products (DBPs) in the investigated swimming pools and filling water. Presented as: "detection frequency | average (minimum-maximum) concentrations".

Disinfection By-Product	Acronym	Limit of Detection (µg L^{-1})	Pool A		Pool B		Filling Water	
Haloacetaldehydes (HALs)								
Bromochloroacetaldehyde	BCAL	1.0	8	2.1 (1.1-3.2)	0	-	0	-
Bromodichloroacetaldehyde	BDCAL	0.3	48	8 (1.9-31)	36	3.5 (1.6-8.8)	0	-
Dibromoacetaldehyde	DBAL	0.7	0	-	0	-	0	-
Dibromochloroacetaldehyde	DBCAL	0.2	4	1.7 (1.7-1.7)	0	-	0	-
Tribromoacetaldehyde	TBAL	0.2	0	-	0	-	0	-
Trichloracetaldehyde	TCAL	1.0	100	1536 (2434-3202)	100	52 (2.7-151)	0	-
Haloacetamides (HAAms)								
Dibromoacetamide	DBAAm	1.1	0	-	0	-	13	2.4 (1.6-3.2)
Dichloroacetamide	DCAAm	0.6	0	-	0	-	0	-
Trichloroacetamide	TCAAm	0.3	0	-	0	-	0	-
Haloacetic Acids (HAAs)								
Bromoacetic Acid	BAA	0.4	0	-	0	-	0	-
Bromochloroacetic Acid	BCAA	0.6	97	74 (11-187)	90	14 (0.7-86)	88	4.2 (1.2-7.5)
Bromodichloroacetic Acid	BDCAA	1.0	71	12 (4.2-18)	81	10 (5.0-43)	31	1.9 (0.8-3.8)
Chloroacetic Acid	CAA	0.5	100	2454 (93-6092)	16	49 (3.3-180)	0	-
Dibromoacetic Acid	DBAA	0.8	71	3.6 (0.9-6.2)	61	2.78 (1.2-8.3)	81	7.6 (2.3-14)
Dibromochloroacetic Acid	DBCAA	0.4	0	-	0	-	31	6.1 (0.5-20)
Dichloroacetic Acid	DCAA	0.4	100	12847 (167-25977)	100	151 (26-804)	44	3.1 (1.5-4.5)
Tribromoacetic Acid	TBAA	1.1	0	-	0	-	13	4.3 (1.3-7.4)
Trichloroacetic Acid	TCAA	0.5	100	2564 (114-11283)	100	689 (52-4347)	63	3.3 (1.2-6.8)

Table 5-1 continued

Disinfection By-Product	Acronym	Limit of Detection (µg L^{-1})	Pool A		Pool B		Filling Water	
Haloacetonitriles (HANs)								
Bromoacetonitrile	BAN	0.5	0	-	0	-	7	0.6 (0.6-0.6)
Bromochloroacetonitrile	BCAN	0.3	54	3.8 (1.1-12)	75	4.5 (1.5-16)	31	1.2 (0.4-2.6)
Chloroacetonitrile	CAN	0.2	55	3.3 (0.4-9.1)	14	1.8 (0.3-6.12)	0	-
Dibromoacetonitrile	DBAN	0.4	38	2.0 (0.4-6.7)	17	1.4 (0.8-1.9)	53	5.2 (0.5-22)
Dichloroacetonitrile	DCAN	0.2	100	43 (2.0-263)	100	38 (0.5-148)	13	3.1 (2.2-4.1)
Trichloroacetonitrile	TCAN	0.1	31	0.5 (0.2-1.0)	21	0.4 (0.2-0.9)	0	-
Haloketones (HKs)								
Chloropropanone	CP	0.4	8	1.6 (0.7-2.5)	0	-	0	-
1,1-Dichloropropanone	1,1-DCP	0.2	56	1.3 (0.2-3.4)	16	0.9 (0.5-1.1)	0	-
1,3-Dichloropropanone	1,3-DCP	0.3	36	10 (0.4-27)	0	-	0	-
1,1,1-Trichloropropanone	1,1,1-TCP	0.2	96	22 (1.1-140)	96	12 (3.8-33)	0	-
Halonitromethanes (HNMs)								
Bromochloronitromethane	BCNM	0.6	21	2.9 (0.8-5.3)	17	5.3 (1.0-9.2)	0	-
Bromodichloronitromethane	BDCNM	0.7	0	-	0	-	0	-
Dibromochloronitromethane	DBCNM	0.7	0	-	0	-	0	-
Dibromonitromethane	DBNM	0.7	0	-	0	-	0	-
Dichloronitromethane	DCNM	0.2	21	2.5 (0.2-5.9)	21	1.1 (0.2-2.9)	0	-
Tribromonitromethane	TBNM	0.7	0	-	0	-	0	-
Trichloronitromethane	TCNM	0.1	79	1.6 (0.2-6.2)	59	1.6 (0.4-4.4)	27	3.1 (0.7-9.9)
Trihalomethanes (THMs)								
Bromodichlromethane	-	0.1	100	2.6 (0.3-8.2)	100	3.2 (0.4-7.7)	100	7.1 (0.8-16)
Dibromochloromethane	-	0.1	29	0.6 (0.4-0.9)	42	0.7 (0.3-1.5)	100	22 (1.5-62)
Tribromomethane	-	0.1	26	0.5 (0.2-0.7)	23	0.5 (0.2-0.9)	94	33 (17-76)
Trichloromethane	-	0.2	100	419 (22-4400)	100	39 (7.5-92)	100	1.8 (1.0-4.3)

Table 5-2: Contribution (%) of disinfection by-product (DBP) class to measured (i) total molar DBP concentration (molar sum of haloketones (HKs), haloacetaldehydes (HALs), haloacetonitriles (HANs), haloacetamides (HAAms), trihalomethanes (THMs) and haloacetic acids (HAAs)) and (ii) total calculated cytotoxicity. Values are reported as percentages and were calculated as follows; For concentration: values were obtained by dividing the total molar concentration of one DBP class by the total molar concentration of all DBPs and multiplying by 100; For cytotoxicity: values were calculated by dividing the calculated cytotoxicity value of each DBP class by the total calculated cytotoxicity and multiplying by 100. Only samples where all DBP classes were analysed are included.

	Pool A				Pool B				Filling Water			
	Concentration		Cytotoxicity		Concentration		Cytotoxicity		Concentration		Cytotoxicity	
	min	max	min	max	min	max	min	max	min	max	min	max
Haloketones (HKs)	0.0	1.1	Not Calculated		0.0	4.6	Not Calculated		Not Detected		Not Calculated	
Haloacetaldehydes (HALs)	1.0	62	85	99.8	0.5	20	52	98	Not Detected		0.0	0
Haloacetonitriles (HANs)	0.0	0.9	0.1	1.4	0.5	23	1.3	47	0.0	10	0.0	96
Halonitromethanes (HNMs)	0.0	0.1	0.0	0.1	0.0	2.4	0.0	7.8	0.0	1.3	0.0	13
Haloacetamides (HAAms)	Not Detected		Not Calculated		Not Detected		Not Calculated		0.0	3.3	0.0	97
Trihalomethanes (THMs)	0.1	18	0.0	0.4	1.5	18	0.1	0.3	51	97	0.4	85
Haloacetic Acids (HAAs)	34	99	0.1	14	57	97	0.6	24	2.7	49	0.8	88

with CAA being measured at concentrations of 93 to 6092 μg L^{-1} in Pool A. As observed with DCAA and TCAA, maximum concentrations of CAA measured in Pool A were significantly higher (approximately 6x greater) than any previous study, where a maximum concentration of 300 μg L^{-1} has been reported (Wang, 2011). A significantly higher CAA concentration (1000 μg L^{-1}) has been reported by Loos and Bacelo (2001), however this study was not included for comparison as no information regarding pool type or treatment method was provided.

BCAA and BDCAA were also detected in some of the Pool A and B samples, where concentrations of up to 187 and 43 μg L^{-1} were measured, respectively. BCAA concentrations in this study were generally higher than most previously reported concentrations (e.g. Daiber et al., 2016; Tardif et al., 2016; Wang, 2011), although up to 1353 μg L^{-1} has been reported (Hang et al., 2016). Only two studies have reported higher concentrations of BDCAA, up to 912 and 110 μg L^{-1} (Kanan, 2010; Loos and Barceló, 2001), with concentrations measured in this study (4.2 to 43 μg L^{-1}) found to be similar to, or only slightly higher than, the other previously reported studies (e.g. Kanan, 2010; Yeh et al., 2014). Compared to other HAAs, significantly lower concentrations (0.9 to 8.3 μg L^{-1}) of DBAA were measured in some pool samples in this study. Although higher DBAA concentrations have been reported, up to 88 μg L^{-1} (Wang, 2011), concentrations measured in this study were generally similar to those reported for other chlorinated pools, 1 to 28 μg L^{-1} (e.g. Carter et al., 2015; Daiber et al., 2016; Hang et al., 2016). The high NPOC content of the pools may help explain the occurrence of HAAs, as HAA formation from the chlorination of filling water organic matter and body fluid analogue has been demonstrated (Kanan and Karanfil, 2011).

Based on molar concentrations, HALs represented 1 to 62% and 0.5 to 20% of the total measured DBP concentrations in Pools A and B, respectively (**Table 5-2**). CH was the only HAL detected in all samples of each pool, where concentrations up to 3202 and 151 μg L^{-1} were measured in Pools A and B, respectively (**Table 5-1)**. While CH concentrations measured in Pool B were generally similar or lower to those previously reported, 17 to 301 μg L^{-1} (Carter et al., 2015; Daiber et al., 2016; Lee et al., 2010; Manasfi et al., 2016; Serrano et al., 2011; Yeh et al., 2014; Zhang et al., 2015), concentrations measured in Pool A (2434 to 3202 μg L^{-1}) are the highest ever reported in chlorinated swimming pools, where a previous maximum concentration of 400 μg L^{-1} was reported in our previous study (Carter et al., 2015). CH is known to form from the chlorination of both humic and fulvic substances, as well as amino acids, as summarised by Barrott (2004). The high NPOC concentrations (up to 30 mg L^{-1}) measured in the pools may, in part, explain the elevated CH concentrations measured over this study, as amino acids are a likely contributor to the organic carbon (OC) content in pools. Although not in all samples, only one other HAL, BDCAL, was detected in both pools, where

concentrations of 1.6 to 31 μg L^{-1} were measured. BCAL and DBCAL were detected in some samples of Pool A (up to 3.2 and 1.7 μg L^{-1}, respectively), only slightly higher than the DBCAL concentrations (0.3 μg L^{-1}) in our previous study of swimming pool water (Carter et al., 2015). This is the first known quantification of BDCAL and BCAL in swimming pool waters. As mentioned for CH, the occurrence of other HALs in the current study may also be attributed to the high NPOC concentrations, suggesting the potential presence of HAL precursors. Furthermore, as discussed for CH (Barrott, 2004), the degradation of these HALs to their corresponding HAAs is consistent with the significant concentrations of most of the corresponding HAAs measured in this study.

THMs represented between 0.1 to 18% and between 1.6 to 18% of the total molar DBP concentrations measured in Pools A and B, respectively (**Table 5-1**). Trichloromethane and bromodichloromethane were the only THMs detected in all samples of both pools. Trichloromethane was measured at concentrations of up to 4400 and 92 μg L^{-1} in Pools A and B, while bromodichloromethane was found at concentrations up to 8.2 and 7.7 μg L^{-1}, respectively. Although some studies have reported higher concentrations of bromodichloromethane in chlorinated pools (e.g. up to 318 μg L^{-1}, Hang et al. (2016)), concentrations measured in this study were generally similar to those observed in most other studies of chlorinated pools (Carter and Joll, 2017 and references therein). Similarly, the majority of the trichloromethane concentrations measured in this study (0.3 to 194 μg L^{-1}) were generally similar to those reported previously (e.g. Hang et al., 2016; Yeh et al., 2014), although elevated concentrations (1.7 to 4.4 mg L^{-1}) were observed in Pool A at the beginning of this study. The trichloromethane concentrations measured at the beginning of this study were significantly higher than those reported in any other study, where a previous maximum of 980 μg L^{-1} was reported in an indoor chlorinated pool (Lahl et al., 1981). Trichloromethane has been shown to be a degradation product of CH and TCAA (Barrott, 2004; Zhang and Minear, 2002), and, as previously discussed, these DBPs were observed at elevated concentrations, potentially accounting for the elevated trichloromethane concentrations also observed. Although not detected in all samples, dibromochloromethane and tribromomethane were measured in both Pool A and B, up to 1.5 and 0.9 μg L^{-1}, respectively, concentrations which were generally similar to those previously reported in chlorinated pools (as summarised by Carter and Joll, 2017).

Only three of the five HNMs investigated were detected in the pools, although not in all samples. BCNM, DCNM and TCNM were found at concentrations up to 5.3, 5.9 and 6.2 μg L^{-1} in Pool A, and up to 9.2, 2.9 and 4.4 μg L^{-1} in Pool B, respectively. Concentrations of TCNM measured throughout this study were generally similar to concentrations previously reported in other chlorinated pools, e.g. up to 5 μg L^{-1} (Carter and Joll, 2017; Tardif et al., 2015). Only

one other study has identified BCNM in pools, at concentrations between 0.8 to 11 μg L^{-1} (Kanan, 2010), which generally reflect those observed in the pools in this study. Furthermore, we report here the first known quantification of DCNM in swimming pools, where concentrations of between 0.2 and 5.9 μg L^{-1} were measured.

Although not in all samples, all HKs investigated (1,1-DCP, 1,3-DCP, CP and 1,1,1-TCP) were detected in Pool A, while only 1,1-DCP and 1,1,1-TCP were detected in Pool B. 1,1,1-TCP was the predominant HK in both pools, routinely measured at concentrations of 1.1 to 45 μg L^{-1}, which are generally similar to, or only slightly higher than, most previous studies (Carter et al., 2015; Daiber et al., 2016; Font-Ribera et al., 2016; Hang et al., 2016; Manasfi et al., 2016; Spiliotopoulou et al., 2015; Tardif et al., 2016, 2015; Yeh et al., 2014; Zhang et al., 2015). It should be noted, however, that 1,1,1-TCP concentrations in several samples of Pool A soon after opening were significantly higher (91 to 140 μg L^{-1}) than the routine measurements. Higher concentrations (up to180 μg L^{-1}) have also been reported in one other study (Hang et al., 2016). These previous studies of 1,1,1-TCP also reported the occurrence of 1,1-DCP, where concentrations were similar to those observed in the current study (0.2 to 3.4 μg L^{-1}). Our previous study is the only other known quantification of both CP and 1,3-DCP in pool water (1.9 and 0.8 μg L^{-1}, respectively) (Carter et al., 2015). While only measured in Pool A of the current study, concentrations were generally similar for CP (0.7 to 2.5 μg L^{-1}), but were generally higher for 1,3-DCP (0.4 to 27 μg L^{-1}), in comparison to our previous study (Carter et al., 2015).

BCAN, DBAN and TCAN were detected (up to 12 μg L^{-1}) in some samples from each pool, at concentrations generally comparable to those previously reported (Carter and Joll, 2017 and references therein). These previous studies also reported the occurrence of DCAN in chlorinated pools, at concentrations generally similar to those in the current study (2 to 263 and 0.5 to 148 μg L^{-1} in Pools A and B, respectively). Furthermore, several samples in the current study were found to contain DCAN at concentrations higher than the previously reported maximum, 206 μg L^{-1} (Hang et al., 2016). CAN was also observed (0.4 to 9.1 μg L^{-1}) in the current study, which for some samples was higher than any previously reported concentration (3 μg L^{-1}) (Carter et al., 2015; Kanan, 2010). In swimming pools, HAN formation has been reported to occur from human derived compounds high in nitrogen, such as urea or hair proteins (Kim et al., 2002). Hypochlorite has also been shown to oxidise HANs, resulting in the formation of HAAms and HAAs (Glezer et al., 1999; Yu and Reckhow, 2015), which is consistent with the low HAN and high HAA concentrations observed in the current study.

5.4.3. Contribution of Filling Water

The filling water, i.e. distributed disinfected drinking water, used to fill and regularly top up the swimming pools, was also investigated. Detection frequency and concentrations of the investigated DBPs measured in the filling water are presented in **Table 5-1**, with details of several general water quality parameters also presented in **Table A5-4**.

Considering average molar concentrations, the different classes of DBPs in order of highest to lowest concentrations measured in the filling water were THMs > HAAs > HANs > HNMs > HAAms > HKs/HALs. Generally, the brominated DBPs were detected at significantly higher concentrations in the filling water compared to the pool waters which is likely due to (i) the transformation of brominated DBPs to mixed bromo-chloro-DBPs due to the constant availability of free chlorine in the pool waters; and (ii) the faster degradation of brominated DBPs (compared to chlorinated DBPs) by UV (Hansen et al., 2013b). The presence of bromide/bromine in the filling water, measured at ~0.2 mg L^{-1} bromide after quenching the oxidant residual, may lead to the formation of brominated DBPs in the pool waters. If the filling water contains residual disinfectant, bromide will be present in its oxidised form, bromine, which can react with organic matter in pools. Alternatively, if no residual disinfectant is present in the filling water, the bromide can be oxidised to bromine via chlorine in pools (Hunter and Jiang, 2010). For these reasons, the filling water is likely the major source of brominated DBPs in swimming pool waters. As mentioned, neither Pool A nor Pool B contained detectable concentrations of DBAAm, DBCAA, TBAA or BAN, despite their detection in the filling water. Thirteen DBPs (BCAL, BDCAL, DBAL, CH, CAA, CAN, TCAN, BCNM, DCNM and the four HKs) were detected in at least one of the swimming pools but were not detected in the filling water, and as such, the filling water can be eliminated as a source of these DBPs in the swimming pool waters. The remaining thirteen DBPs were detected in both the filling water and at least one of the swimming pools. Of these, the chlorinated DBPs were generally at much higher concentrations in the pools compared to the filling water and hence the filling water is not considered a major source of these chlorinated DBPs in the pool waters. NPOC concentrations measured prior to opening (~16 and 9 mg L^{-1} for Pool A and B, respectively) were significantly higher than those observed in the filling water (~2 mg L^{-1}). While the filling water contributed a small portion of the NPOC measured prior to opening, it is clear that filling water was not the major source of NPOC in these pools. Similarly, the total DBP concentrations measured prior to opening (104 and 2.9 μM for Pools A and B, respectively) were significantly higher (408 and 18 times higher) than those measured in the filing water (0.1 to 0.4 μM). As pools contained DBPs at much higher concentrations than their filling water, it can be concluded that the filling water was generally an insignificant source of DBPs in the investigated pools.

5.4.4. Comparison of the Swimming Pools

Table 5-1 summarises the concentrations of DBPs measured in Pools A and B, while **Table 5-2** summarises the contribution of each DBP class to the total DBP concentrations measured (based on molar concentrations), for both Pool A and Pool B. Considering average molar concentrations, the different classes of DBPs in order of highest to lowest concentrations were found to be HAAs > HALs > THMs > HANs > HKs > HNMs > HAAms for Pool A and HAAs > HANs > THMs > HALs > HKs > HNMs > HAAms for Pool B. HAAs were also found to be the dominant species by Lee et al. (2010) in their investigation of 30 chlorinated pools (representing 73%), followed by THMs (14%), CH (10%) and HANs (3%), in terms of total average molar concentrations. Although some differences can be observed in the order of dominant DBP classes between Pool A and Pool B, these are likely due to a range of factors including bather load, water recirculation and DBP volatilisation which naturally differ between the pools, also noted by Lee et al. (2010).

On average, total molar DBP concentrations were approximately 23x higher in Pool A compared to Pool B. Excluding the first two days where concentrations measured in Pool A were up to 38 and 190 times higher than Pool B, for HKs and THMs, respectively, concentrations of these DBPs were generally similar between the pools. Concentrations of HAAs were roughly one order of magnitude higher in Pool A compared to those observed in Pool B, possibly due to the higher NPOC concentrations measured in Pool A, as HAA formation from organic matter from filling waters and body fluid analogue has been reported (Kanan and Karanfil, 2011). HAN concentrations measured in Pool A were approximately double those measured in Pool B, while HALs were approximately 20 times higher in Pool A compared to Pool B. Although similar concentrations were observed in some cases, HNMs measured in Pool A were generally higher (up to 8x) than those measured in Pool B. As discussed below, the higher concentrations of nitrogenous DBPs (N-DBPs) observed in Pool A compared to Pool B are potentially due to higher release of anthropogenic compounds (e.g. sweat) which are nitrogen rich. As HALs have been shown to convert to HAAs (Barrott, 2004), HAA precursors may also act as precursors to HALs, which therefore may help explain the higher concentrations of HALs in Pool A compared to Pool B.

While not explicitly investigated in this study, the main anion contributing to conductivity is presumably chloride, a by-product of chlorination. Chloride has been shown to promote the formation of some DBPs, attributed to its effect on oxidant speciation, where a shift from HOCl to the more reactive Cl_2 was observed to increase with increasing chloride concentration (E et al., 2016). Consistent with observations of this study, the higher levels of DBPs measured in Pool A compared to those in Pool B may be a reflection of the higher

conductivity measured in Pool A compared to Pool B, 1.4 to 4.5 and 0.5 to 2.7 mS cm^{-1}, respectively.

The generally higher concentrations of DBPs observed in Pool A may also result from a range of operational factors including swimming pool size, bather load, swimmer activities and treatment. Due to its smaller size (Pool A is approximately six times smaller than Pool B when comparing total water volumes), and assuming the number of swimmers and hence bather load inputs are comparable between both pools, a higher concentration of bather derived inputs would be observed in Pool A, which may lead to higher DBP formation in comparison to Pool B. Similarly, the higher operating temperature of Pool A (30 °C) compared to Pool B (27 °C) may have increased the formation of some DBPs, as observed in previous studies (Kanan, 2010; Simard et al., 2013). The higher temperature in Pool A may also have slightly increased the release rate of bather load derived precursors (Keuten et al., 2012). The use of UV treatment in Pool A may also be a contributing factor for the higher concentrations observed for some DBPs in Pool A, as UV treatment in swimming pools has been shown to increase the formation of DCAN, 1,1,1-TCP, 1,1-DCP, CH, THMs and TCNM (Cimetiere and De Laat, 2014; Hansen et al., 2013b; Spiliotopoulou et al., 2015).

5.4.5. Potential Health Effects and Estimation of Cytotoxicity

Although no swimming pool specific guidelines exist for these DBPs, DCAA, TCAA and CH concentrations were greater than their respective Australian Drinking Water Guideline (ADWG) values (100 µg L^{-1}; (ADWG, 2011)) in all samples taken from Pool A, and in 61, 97 and 16% of samples taken from Pool B, respectively. DCAA and TCAA were also measured at concentrations greater than their respective World Health Organisation (WHO) drinking water guidelines (50 and 200 µg L^{-1} for DCAA and TCAA, respectively; (WHO, 2011)) in 100 and 90% of samples taken from Pool A and in 90 and 97% of samples taken from Pool B. Although not detected in all samples, CAA was greater than both the ADWG and WHO values (150 and 20 µg L^{-1}, respectively; (ADWG, 2011; WHO, 2011)) in 97 and 100% of samples from Pool A, as well as in 20 and 40% of samples from Pool B. Although DCAN is currently not regulated in Australian drinking water, concentrations measured in 48 and 34 % of samples taken from Pools A and Pool B, respectively, were higher than the WHO guideline value (20 µg L^{-1}; (WHO, 2011)). Total THMs were the only other DBPs to exceed their ADWG value (250 µg L^{-1}; (ADWG, 2011), although this only occurred in Pool A within the first two days from opening, where concentrations of 1.7 to 4.4 mg L^{-1} were measured. However, considering the WHO value (80 µg L^{-1}; (WHO, 2011)), total THM concentrations exceeded this drinking water guideline in 48 and 6% of samples taken from Pool A and Pool B, respectively. Germany have imposed a swimming pool specific guideline for total THMs, being 20 µg L^{-1} as

trichloromethane equivalents (Deutsches Institut für Normung e. V. (German Institute for Standardization), 2012). Concentrations of total THMs measured in all samples of Pool A and in 94% of samples from Pool B exceeded this German swimming pool guideline.

As few swimming pool specific guidelines exist and guidelines for drinking waters are unlikely to represent the true risk associated with swimming pools as more than one uptake mechanism is viable in pools, the chronic cytotoxicity of the swimming pool waters was estimated (via calculation) to indicate the potential health effect of these DBPs at the concentrations measured in this study. **Table 5-2** summarises the contribution of each DBP class to the overall theoretically calculated chronic cytotoxicity.

Excluding the sample prior to opening for Pool B, for all pool samples, the estimated cytotoxicity was significantly higher than that estimated for the filling water, being between 108 to over 46000 and 20 to over 2300 times higher for Pools A and B, respectively. Furthermore, Pool A demonstrated a consistently higher (between 7 and 113 times) level of estimated cytotoxicity than Pool B, when considering total estimated cytotoxicity values.

Compared to all investigated DBP classes, HALs were found to contribute the most to the estimated cytotoxicity in pools, representing up to 98 and >99% of the total calculated cytotoxicity in some samples of Pools A and B, respectively. This cytotoxicity was found to be predominantly due to CH, which represented over 93% of the estimated cytotoxicity associated with HALs. Considering HALs only represented 0.5 to 62% of the total measured DBP molar concentration, HALs, more specifically CH, were found to pose the highest health risk (in terms of cytotoxicity) in the investigated pools. Furthermore, as CH was below its detection limit (1.0 $\mu g\ L^{-1}$) in all filling water samples, the increase in CH concentration in the pools is likely to account for the significant increase in the estimated cytotoxicity in both pools.

While HAAs represented up to 99% of the total measured DBP concentrations (molar), they were found to only represent a maximum of 24% of the total estimated cytotoxicity in some pool samples. Similarly, while N-DBPs (such as HANs or HNMs) were generally detected at lower concentrations, accounting for up to 23% of total molar concentrations, they were found to represent almost half the estimated cytotoxicity (up to 47% in some samples). Furthermore, while it was observed that THMs accounted for a similar portion of the total measured DBP concentrations (up to 18% in some samples) compared to HANs (up to 23% in some samples), THMs only accounted for less than 0.4% of the estimated cytotoxicity. These observations highlight that DBPs measured at higher concentrations, e.g. HAAs, may not be as significant as those detected in lower concentrations (e.g. HANs), when considering health effects of DBPs in swimming pools.

5.4.6. Trends Over Time

General water quality parameters NPOC and TN can be easily used to assess overall trends in swimming pool water quality. **Figure 5-1** presents the concentrations of NPOC and TN for Pools A and B, both newly built and filled, measured over the duration of this study. NPOC concentrations measured prior to opening were much higher in the pools than in the filling water, being 16, 7 and <1 mg L^{-1} for Pool A, Pool B and the filling water, respectively.

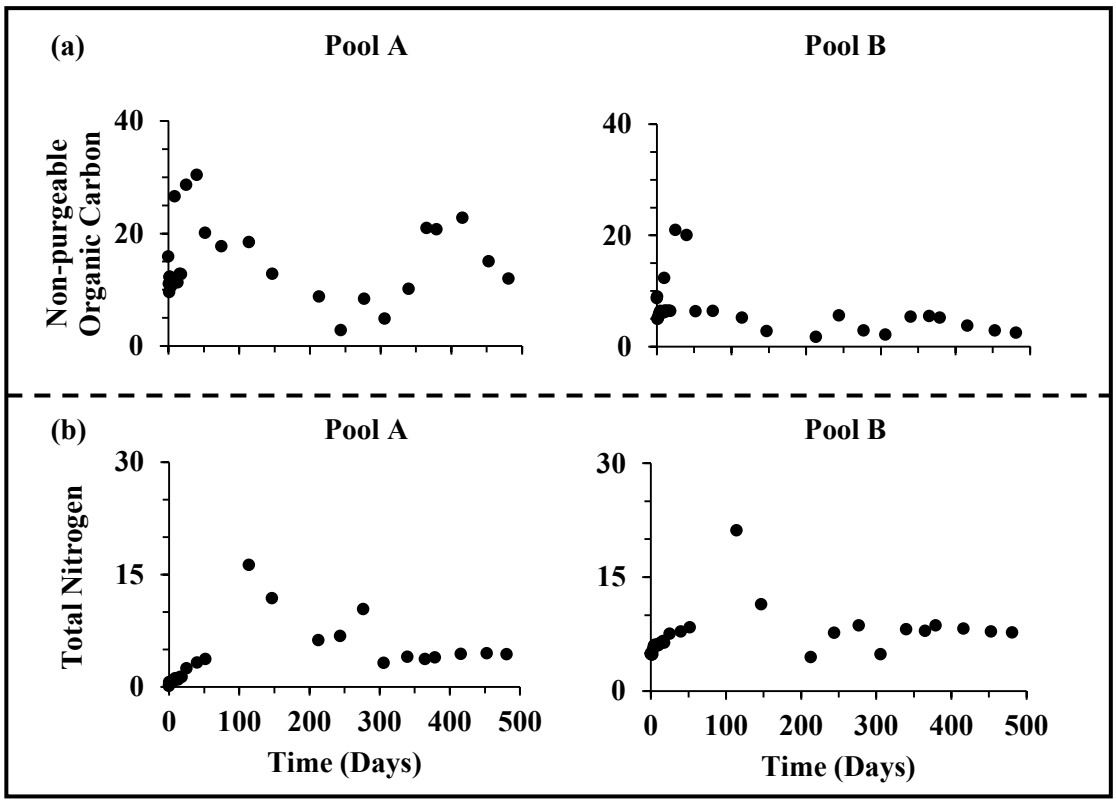

Figure 5-1: Concentrations of **(a)** non-purgeable organic carbon (NPOC) and **(b)** total nitrogen (TN) measured in Pool A (left) and Pool B (right). Y-axis represents concentration (mg L^{-1}). X-axis represents time (days), where t=0 represents initial samples collected prior to the opening of the pools.

For Pool A, NPOC concentrations generally increased soon after opening (summer 2015), after which they gradually decreased until approximately day 250 (winter 2016). A gradual increase was then observed for the duration of the study, until summer 2017. These trends in measured NPOC concentrations suggest NPOC follows a seasonal trend in Pool A, that is, highest during times of peak periods (summer) and lowest at times of minimal bather loads (winter). While an initial increase (and subsequent maximum) in NPOC concentration was also observed for Pool B (containing cyanuric acid which contributes to the NPOC concentration), after this, NPOC concentrations were found to be relatively constant in Pool B. In a previous six month study of a newly re-filled and chlorinated swimming pool, total

organic carbon (TOC) was also reported to be fairly uniform (3.1 to 3.9 mg L^{-1}) (Yeh et al., 2014), which the authors attributed to the TOC reaching steady state due to mineralisation of TOC, presumably due to chlorine oxidation, offsetting continual TOC input. Similarly, in a model pool operated under conditions reflective of those of full-scale swimming pools, concentrations of TOC were reported to reach steady state after 200 to 500 hours, again attributed to the mineralisation of organic carbon (OC) (Judd and Bullock, 2003). Consistent with these previous two studies, the relatively constant NPOC concentrations measured after around 50 days in Pool B are indicative of the NPOC reaching a steady state due to mineralisation (i.e. oxidation) processes balancing the addition of fresh OC. Judd and Bullock (2003) highlighted the importance of the establishment of a steady state balance of mineralisation versus addition of OC in pools to reduce precursors for DBP formation. A steady state for NPOC was not observed in Pool A, possibly due to a) the higher NPOC concentrations, such that a longer time was required to achieve steady state levels, and b) seasonal trends in NPOC input. Unfortunately, the shorter duration of the Yeh et al. (2014) study (6 months, over summer period) limits observation of any seasonal trends in NPOC concentration that may have been present, as observed in Pool A. The generally lower concentrations of DBPs in Pool B than in Pool A are consistent with the likely establishment of mineralisation balancing addition of OC in Pool B, which was not observed in Pool A.

Interestingly, the initial peak in NPOC concentration (16 and 7 mg L^{-1} in Pools A and B, respectively) in both pools of the current study was not observed in the study by Yeh et al. (2014), where the initial TOC concentration was 3.5 mg L^{-1}. As similar TOC concentrations were measured in the filling water and the pool investigated by Yeh et al. (2014), and with both swimmers and filling water excluded as a major source of NPOC in the current study, these observations suggest that newly built and filled pools may, at least initially, differ significantly in water quality compared to those simply re-filled, presumably as a result of the pool building process and/or new pool infrastructure.

TN may be used in swimming pools as an indication of bather load derived chemical input, as many bather load compounds are high in nitrogen content (Keuten et al., 2012). In relation to initial concentrations measured prior to opening, TN concentrations gradually increased, with maximum concentrations being observed at day 114 (16 and 21 mg L^{-1} for Pool A and Pool B (containing cyanuric acid), respectively) after which a gradual decrease (until day 200) was observed. This increase is likely due to the input of swimmers, where perhaps the decrease may be attributed to the TN reaching steady state levels, as observed for NPOC. Fairly constant TN concentrations were measured for the remainder of the study for both pools.

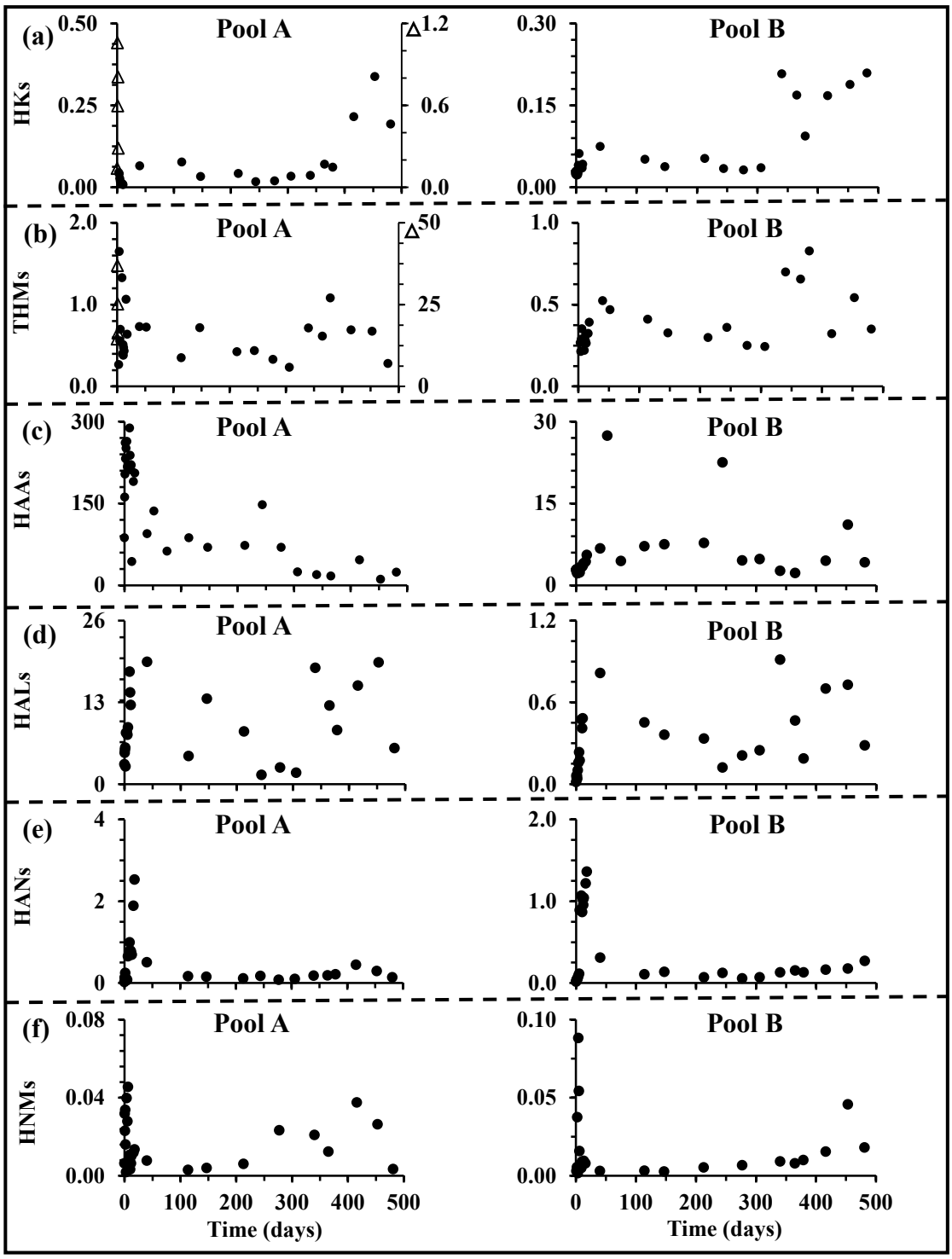

Figure 5-2: Concentration of **(a)** haloketones (HKs), **(b)** trihalomethanes (THMs), **(c)** haloacetic acids (HAAs), **(d)** haloacetaldehydes (HALs), **(e)** haloacetonitriles (HANs) and **(f)** halonitromethanes (HNMs) measured in Pool A (left) and Pool B (right). Data is presented as total concentration by DBP class. Y-axis represents concentration (μM) and, where required (**(a)** and **(b)** for Pool A), a secondary y-axis with a scale change has been included with its data represented by triangles. X-axis represents time (days), where t=0 represents initial samples collected prior to the opening of the pools.

Yeh et al. (2014) reported a similar trend for TN, where a somewhat linear increase was observed in the newly re-filled pool of their study, where input of swimmers was reported to be the major source of TN. Unlike the current study, however, no subsequent decrease or constant concentrations of TN were reported, which, as with NPOC, is likely a consequence of the shorter duration of their study.

One key finding of this study is the occurrence of many DBPs at high concentration soon after the opening of the pool facility. Most DBPs showed a significant increase in concentration during these initial days (up to day 52, **Figure 5-2**), however, significant concentrations were also measured prior to opening, hence, bather load is not the only contributor to the observed DBP formation. Using Pool B as an example, in comparison to its concentration measured in the filling water (< 4 µg L^{-1} (LOD)), higher levels of TCAA were measured both prior to and soon after opening: 345 and 4347 µg L^{-1} at t=0 and t=52 days, respectively. The significantly higher NPOC concentrations measured prior to the opening of the facility (16 and 7 mg L^{-1} for Pool A and Pool B, respectively), i.e. no contribution from swimmers, compared to that measured in the filling water (<1 mg L^{-1}) suggest relatively large amounts of potential DBP precursors were present even prior to the opening of the pools. The high levels of NPOC (and subsequent DBPs) observed prior to opening (and their subsequent increase soon after) appear to have been generated from the pool building process and/or new pool infrastructure.

Spearman's rank coefficients (summarised in **Tables A5-5 and A5-6**) were used to assess any correlation(s) that exists between: (i) the thirty-nine measured DBPs (on a molar basis) and (ii) the measured general water quality parameters, for each of the investigated pools. Parameters that resulted in a rank coefficient between 0.01 and 0.05 ($0.01<p<0.05$) were said to exhibit a moderate significant correlation, while parameters resulting in a rank coefficient of 0.01 or less ($p<0.01$) were said to have a significantly strong correlation.

While some correlations differed between Pool A and Pool B, correlations between several parameters and/or DBPs were observed for both pools. Residual free chlorine equivalent concentrations were also found to be negatively weakly correlated with HANs in Pool A (r^2=-0.44), which is consistent with the findings of Yu and Reckhow (2001), where the instability of HANs in waters containing residual hypochlorite was demonstrated. This negatively weak correlation differs to other studies where no significant correlation (Lee et al., 2010), and a significant positive correlation (Yang et al., 2018), between HANs and free residual chlorine was reported. No other DBP class was found to correlate with either free or total chlorine equivalent residual concentrations in either pool, which is consistent with Lee et al. (2010) who, in addition to HANs, reported no correlation was observed between chlorine

residual concentrations and the concentrations of THMs, HAAs, or CH in their studied pools. While Zhang et al. (2015) reported no correlation between chlorine residual concentrations and CH in their study of 14 swimming pools, a correlation was reported for chlorine residual concentrations and both HAAs and THMs (r^2=0.31 to 0.40), with THMs and TCNM also previously reported to be positively correlated to free chlorine residual concentrations (Yang et al., 2018). Zhang et al. (2015) attributed these unusual correlations to the level of chlorine concentrations measured, suggesting that correlations between chlorine concentrations and DBPs are dependent on the residual concentration employed. Consistent with correlations being suggested to be more evident for pools where lower residuals are employed (<2.2 mg L^{-1}; (Yang et al., 2018; Zhang et al., 2015)), correlations between chlorine residual concentration and levels of DBPs were not observed for the pools of the current study as relatively high chlorine residuals were employed (target free chlorine residuals of 2.5 to 3.5 mg L^{-1}). Both free and total chlorine equivalent concentrations were however found to weakly correlate with conductivity in Pool A (r^2=0.42 to 0.46), suggesting that residual chloride, a by-product from the reaction of chlorine with organic matter, is likely responsible for much of the conductivity measured. Conductivity (presumably mostly chloride) was also found to be moderately to strongly correlated with TN and HKs for both pools (r^2=0.53 to 0.97) and weakly correlated with THMs in Pool B (r^2=0.46). While HKs and TN were not target parameters of their study, E et al. (2016) also reported a significant relationship between chloride and several DBPs (e.g. trichloromethane and DCAN; r^2=0.62 to 0.98), in both bench scale studies and swimming pool waters. As chlorine is continually added into the pools, the chloride concentration increases and more DBPs are formed, likely resulting in the apparent correlation between conductivity and some DBP concentrations.

NPOC concentrations were found to weakly correlate with HANs in both pools (r^2=0.45 to 0.46) and moderately with HALs in Pool A (r^2=0.51), although no other correlations of NPOC with any other DBP class were observed. While Lee et al, (2010) also reported TOC to be correlated to CH (the monohydrate of trichloroacetaldehyde; r^2=0.68), HANs and TOC were not found to correlate in their study of chlorinated pools. No correlation between HANs and TOC, nor THMs or TCNM and TOC, were reported by Yang et al. (2018) in their recent study of 35 outdoor chlorinated pools. Zhang et al. (2015) reported no correlation to exist between TOC and CH in their study of 14 chlorinated pools, which is consistent with observations with Pool B but opposite to those for Pool A in the current study. Zhang et al. (2015) did, however, report no correlation between TOC and either THMs or HAAs, consistent with the current study. Furthermore, consistent with this study is the correlation between HANs and UV_{254} (an indicator of TOC) observed by Hang et al (2016), although unlike this study, a correlation between THMs was also reported. THMs have also been reported to be

correlated with either dissolved organic carbon (DOC) or TOC (Chu and Nieuwenhuijsen, 2002; Peng et al., 2015), although Peng et al. (2015) report a time delay of 2 days.

TN concentrations were found to be moderately correlated to HAAs in both pools (r^2=0.49 to 0.64), which is likely why a weak to moderate correlation between TN and total DBPs was also observed (r^2=0.38 to 0.66) in these pools, as HAAs accounted for a significant portion of the total DBP molar concentrations (34 to 99%) across both pools. While no correlation between TN and THMs was observed by Yang et al. (2018) in their study of 35 outdoor chlorinated pools, and although only observed for Pool B, TN concentrations were found to strongly correlate with HKs, HALs and THMs in the current study. These correlations observed in the current study suggest that nitrogenous compounds may act as precursors to these DBPs, although due to conflicting previously published results, further investigations under more controlled conditions (e.g. bench scale) are required to confirm these correlations. A weak correlation between TN concentrations and number of pool entries (r^2=0.45) was also only observed in Pool B, which is likely due to the release of nitrogen-rich compounds from swimmers (Keuten et al., 2014, 2012).

Number of pool entries were found to weakly to moderately correlate with HKs, HALs and NPOC in Pool A (r^2=0.40 to 0.61), and weakly to moderately correlate with THMs and TN in Pool B (r^2=0.55 and 0.45, respectively), however, no significant correlation between number of pool entries and either HAAs, HNMs or HANs was observed in either pool. While a correlation (r^2=0.50) between TOC and number of swimmers was reported by Chu and Nieuwenhuijsen (2002), no significant correlation was observed between number of swimmers and DOC concentrations in other studies (Hang et al., 2016; Peng et al., 2015), with the differing observations also seen between the two pools of this study. Similarly, while both HAAs and THMs have been correlated (r^2=0.70 to 0.72) to number of swimmers (Chowdhury et al., 2016; Chu and Nieuwenhuijsen, 2002), negative or no significant correlations have also been reported (Chowdhury et al., 2016; Hang et al., 2016; Peng et al., 2015). Consistent with the current study, Hang et al. (2016) reported no significant correlation between number of swimmers with HNMs and HANs, however, no significant correlation with number of swimmers and both HAAs, HKs and HALs was also reported. These reports are neither in agreement nor disagreement with the current study, as these correlations were observed in one of the pools, whilst being absent in the other. The differing correlations reported suggest that the number of swimmers may not be a reliable indication of DBP levels in pools, although swimmers habits (e.g. pre-swim showering or urinating while swimming) and their activity (e.g. water agitation and splashing) have been demonstrated to have a significant impact on DBPs in pools, as discussed in more detail elsewhere (e.g. Carter and Joll, 2017).

A weak negative correlation between pH and number of pool entries was observed in Pool A (r^2=-0.43), which can potentially be explained by the likely release of bodily fluids (e.g. sweat and urine), which are generally acidic, pH 4.5 to 7 (Rose et al., 2015). This release is presumably higher for Pool A (compared to Pool B) as Pool A is designed for use by children and babies. HANs were found to be weakly and negatively correlated with pH in Pool A (r^2=-0.42), which is consistent with several other studies (Lee et al., 2010; Yang et al., 2018) who also reported a negative correlation between pH and HANs, which is presumably due to a higher pH supressing their formation (Hansen et al., 2013a). Although not observed in the current study and an opposite observation was reported by Kanan (2010), a negative correlation between TCNM and pH was reported by Yang et al. (2018), which the authors attribute to the more complicated precursors that exist in real swimming pools. No other significant correlations between pH and other DBP classes were observed in this study. This is consistent with other studies (Chu and Nieuwenhuijsen, 2002; Lee et al., 2010), where no significant correlation between pH and THMs, HAAs, CH or TOC were also reported, with Yang et al. (2018) also reporting no correlation to exist between THMs and pH in their study of chlorinated pools.

Water temperature was weak to moderately correlated with HANs for both pools (r^2=0.38 to 0.52) and weakly with both HKs and HALs in Pool B (r^2=0.41 to 0.43). These correlations suggest higher temperatures lead to an increase in the formation of these DBPs, as reported for THMs and HAAs (Kanan and Karanfil, 2011). The absence of any correlation between THMs and water temperature, particularly for Pool A, is potentially, in part, due to the operating temperatures of the pools (24 to 32 °C). While the higher operating temperatures likely increased the formation rate of THMs (as reported by Kanan and Karanfil (2011)), they likely also increased their volatilisation, resulting in an overall decrease in THM concentrations in the pools. The loss of volatile DBPs is supported further as a weak negative correlation between total molar DBP concentrations and temperature in Pool A was also observed (r^2=-0.36). Furthermore, the relatively high operating temperature of Pool A (30 °C) is likely to promote the release of sweat from swimmers, which in addition to supporting the negative correlation observed with pH and number of pool entries, is consistent with the weak correlation between temperature and TN concentration observed in Pool A (r^2=0.46). While not observed in this study, THMs were found to correlate with temperature (r^2=0.50) in a study of chlorinated pools (Chu and Nieuwenhuijsen, 2002), although as summarised by Carter and Joll (2017), it has been suggested that THM correlations in general are highly dependent on both swimmer activity and water agitation, both of which affect THM volatilisation (hence THM water concentrations) and inherently any correlation with other parameters. Chu and Nieuwenhuijsen (2002) did, however, report a correlation between temperature and TOC

(r^2=0.40), for which a moderate correlation was also observed for Pool B in the current study (r^2=0.53).

As expected, due to their high dominance in each pool, HAAs were found to be strongly significantly correlated (r^2=0.94 to 0.98) to the total DBP concentrations for both pools, which is consistent with other studies (Hang et al., 2016; Lee et al., 2010). Furthermore, the dominance of both HALs and HANs in Pool B (each up to 23% of the total molar DBP concentration) is likely the reason they were observed to weakly to moderately correlated to the total DBP concentrations measured in Pool B (r^2=0.43 to 0.59). While some results differ for each pool in the current study, Lee et al. (2010) also reported correlations between both THMs and CH to total DBP concentrations (r^2=0.51 to 0.58), although HANs were not found to be correlated.

Between the investigated DBP classes, only HANs and HALs were found to be correlated to one another for both pools, where a significantly strong correlation (r^2=0.68 to 0.71) was observed. These results are similar to those of Lee et al. (2010), where a significant correlation (r^2=0.67) between HANs and CH was reported in their study of 30 chlorinated pools. This correlation is potentially a result of the formation of nitriles as transformation products of aldehydes, via reactions involving monochloramine and two intermediate species, *N*-chloramino alcohols and *N*-chloraldimines, as we have previously demonstrated for valine in model compound studies (How et al., 2017). The only other correlation observed in Pool A was that between THMs and HNMs (r^2=0.49), likely a consequence of their similar chemical structures and hence similar precursors, with the lower HNM concentrations measured in Pool B a possible reason for the absence of this correlation in Pool B. Yang et al. (2018) also reported a correlation (r^2=0.76) between THMs and TCNM in their study of 35 chlorinated pools, however unlike the current study, correlations between THMs and HANs, as well as between HANs and TCNM, were also reported. Although only observed for Pool B, a moderate to strong correlation was observed between HKs and HANs, HALs and THMs (r^2= 0.62, 0.76 and 0.58, respectively). Hang et al. (2016) also reported a correlation between HKs and THMs in their study of 13 chlorinated pools, although no correlation between HKs and HANs was observed.

The observed DBP correlations in the current study suggest a potential relationship between HANs, HKs, HALs and THMs. Methyl ketones (i.e. the HKs investigated in this study) can be converted to THMs via the haloform reaction, supporting the observed correlation between HKs and THMs. Although not a direct decomposition product (it is suggested to be a result of a secondary reaction when organic matter is present), CH (i.e. a HAL) has been observed as a result of the decomposition of 1,1,1-TCP (i.e. a HK) (Nikolaou

et al., 2001). While this study was limited to the relationship between CH and 1,1,1-TCP, it is reasonable to suggest that a similar relationship may exist between other HALs and their corresponding HKs, supporting the observed correlation between HKs and HALs in the current study. How et al. (2018) provide a multi-pathway reaction scheme for the formation of several DBPs from the chlorination of amino acids, summarising and linking the findings of several earlier studies (How et al., 2017; Kimura et al., 2015; Ueno et al., 1996; Yu and Reckhow, 2015). Here, aldehydes have been shown to be transformed to their corresponding nitriles via several reaction steps involving monochloramine. Although demonstrated for isobutyraldehyde (a chlorination by-product of valine) by detection of several of its corresponding transformation products (e.g.1-(chloroamino)-2-methylpropan-1-ol, *N*-chloroisobutyraldimine and isobutyronitrile) (How et al., 2017), this pathway may be applicable to other compounds, e.g. HALs such as CH. The potential conversion of HKs to their corresponding HANs via the pathways proposed by Nikolaou et al. (2001) and How et al. (2017) supports the correlation observed between HKs and HANs in the current study.

No further significant correlations were observed between any of the investigated DBP classes in either of the pools investigated, which is consistent with most observations of Lee et al. (2010), who reported no significant correlations existed between HANs and either HAAs or THMs, or between CH and HAAs. In contrast to the current study, Lee et al. (2010) reported correlations to exist between THMs and both HAAs and CH (r^2=0.49 and 0.42, respectively), as did Zhang et al. (2015), r^2=0.35 to 0.55, who also reported a correlation to exist between HAAs and CH (r^2=0.42). As discussed, the absence of correlations between THMs and other DBP classes may be due to the volatilisation of THMs, as suggested to occur for other volatile DBPs (e.g. Schmalz et al., 2011; Zwiener and Schmalz, 2015), likely to be more pronounced in Pool A due to the higher operating temperature and/or the higher splashing potential (leisure pool).

5.5. Conclusions

This study is the first investigation of the water quality and occurrence of DBPs in newly built and filled swimming pools, where investigations occurred for 500 days and began prior to the opening of the facility. A range of DBPs (THMS, HAAs, HANs, HNMs, HKs and HALs) were detected throughout the duration of the study, where many of the DBPs were generally measured at higher concentrations than previously reported for chlorinated swimming pools. The maximum concentrations of CAA, DCAA, TCAA and CH were significantly greater than any previously reported concentrations. HAAs were the dominant class (based on molar concentrations) for both pools, followed by HALs, THMs, HANs, HKs and HNMs for Pool A, and by HANs, THMs, HALs, HKs and HNMs for Pool B. HAAms

were not detected in either pool. This study is the first known quantification of four DBPs (BCAL, BDCAL, BCNM and DCNM) in swimming pools.

Considering total molar concentrations, on average, Pool A contained 23x higher levels of DBPs compared to Pool B, with both pools found to contain significantly higher total molar concentrations than their filling water. In most cases, similar concentrations of THMs and HKs were found in both pools, although HANs, HNMs, HAAs and HALs were generally higher (on average 2, 8, 10 and 20 times higher, respectively) in Pool A compared to Pool B. These differences are likely due to the NPOC concentration measured prior to opening, the potential organic input from bather load, as well as operational parameters such as water temperature and chlorine residual, all of which were higher in Pool A compared to Pool B. The lower concentrations of DBPs in Pool B, where a steady state NPOC concentration was achieved, highlight the importance of the establishment of this steady state balance of mineralisation versus addition of OC to reduce precursors for DBP formation.

Filling waters were found to be the major source of brominated DBPs in the pools, but were an insignificant source of other DBPs, NPOC and TN, while swimmers were found to be the major source of TN in the pools. Significant concentrations of NPOC were measured prior to opening. Furthermore, compared to the filling water, a significant concentration of DBPs were measured in both pools prior to opening, suggesting that DBP precursors (encompassed in NPOC concentrations) existed prior to the opening of the facility. Almost all DBPs and NPOC significantly increased soon after opening, where maximum concentrations were generally observed at approximately fifty days after opening. The pool building process and/or new pool infrastructure appears to have had a major impact on the chemical water quality of the pools, particularly with regard to the significant concentrations of NPOC and DBPs prior to, and after, opening of the facility.

Pool A exhibited higher estimated cytotoxicity compared to Pool B and, in almost all cases, pool water samples exhibited higher cytotoxicity than their filling water. HALs were found to contribute the most to the total estimated cytotoxicity, predominantly due to CH. With correlations between number of pool entries and HALs also observed, findings suggest that swimmers may be a potential source of HAL precursors and in turn may have significant impact on the cytotoxicity of pool waters. While HAAs were found to contribute significantly to the total molar DBP concentrations, they only accounted for up to 24% of the total estimated cytotoxicity. Furthermore, other DBP classes (e.g. N-DBPs), while measured at lower concentrations, were found to account for almost half the total estimated cytotoxicity. These observations highlight that the predominant DBPs (e.g. HAAs or THMs) are not necessarily the significant DBPs in terms of potential health effects from swimming pools.

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5.7. References

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CHAPTER 6

IMPACT OF BUILDING MATERIALS ON THE CHEMICAL WATER QUALITY OF SWIMMING POOLS

Statement of Contribution to Co-authored Submitted Paper

This Chapter includes the co-authored manuscript '*Impact of building materials on the chemical water quality of swimming pools*', currently under review for publication in Journal of Hazardous Material. The bibliographic details of the co-authored paper, including all authors are:

Carter, R.A.A., and Joll, C.A., Impact of building materials on the chemical water quality of swimming pools. Submitted to Journal of Hazardous Materials.

I, Rhys Aaron Ainsley Carter, contributed to the publication by: undertaking a review of the current literature in this field, undertaking all laboratory experiments and data analysis, being the primary writer (including creating figures and tables), and editing and finalising the manuscript.

I, as a Co-Author, endorse that this level of contribution by the candidate indicated above is appropriate.

Cynthia Joll

6.1. Abstract

This study investigates the potential of two commercial building materials to be sources of organic materials in pools and assesses their potential to lead to disinfection by-product (DBP) formation. A concrete product and a latex additive were demonstrated to leach significant concentrations of non-purgeable organic carbon under conditions commonly used in pools. The leachates from the concrete product and the latex additive produced trichloromethane, dichloroacetic acid and trichloroacetic acid upon chlorination. Furthermore, while the concrete product did not form chloral hydrate (CH), the latex additive was identified as a precursor of CH. Styrene, a monomer of the styrene-butadiene co-polymer: being a major constituent of the commercial latex additive; led to the formation of trichloromethane, trichloroacetic acid and CH upon chlorination. Styrene monomer potentially remaining in the co-polymer within the latex additive is therefore a possible precursor for formation of these DBPs from the latex additive. The potential of building materials to not only act as a source of organic carbon in pools, but to lead to formation of DBPs, was demonstrated. For the first time, the potential impact of building materials on the chemical water quality of pools, particularly pools which are newly constructed or have recently undergone maintenance, is highlighted.

6.2. Introduction

Although disinfection of water leads to a reduction in the microbial disease risk, it can also lead to the unwanted formation of disinfection by-products (DBPs) via reactions between organic matter and the added disinfectant. Over 700 DBPs have been identified in treated drinking, waste and recycled waters (Plewa and Richardson, 2017), where many have demonstrated negative health effects (Richardson et al., 2007). Although investigated to a lesser extent than other disinfected water types, DBPs have been reported in swimming pools and spas, as summarised by two recent reviews (Carter and Joll, 2017; Manasfi et al., 2017).

Swimming pools and spas are likely to have a higher input of organic compounds compared to drinking waters, where the largest input of organic compounds is natural organic matter (NOM) introduced via source waters. For swimming pools and spas, however, in addition to low concentrations of NOM introduced by regular pool top-ups using distributed drinking water, organic input also includes bather load, including human body excretions (e.g. sweat, urine and saliva), as well as pharmaceuticals and personal care products (PPCPs; e.g. analgesics, antibiotics, sunscreens, lotions and cosmetics). This wider variety (and often greater concentration) of organic materials in pools, in addition to the usually higher disinfectant residuals employed, likely lead to a greater formation of DBPs in pools compared to drinking water distribution systems. As summarised in two recent reviews

(Carter and Joll, 2017; Manasfi et al., 2017), additional factors specific to pools have also been shown to affect the formation and occurrence of DBPs in pools, including the use of secondary treatments (e.g. UV or ozone), bromide concentration, swimmer activity and pool usage, temperature and pH. Additionally, these reviews summarise a variety of studies investigating the potential of body fluid analogue (and/or its individual components) and PPCPs to act as DBP precursors, where laboratory scale chlorination was conducted under conditions reflective of pools. While these studies have provided an insight to DBP precursors and formation in the swimming pool environment, much remains unexplored.

Recent reviews by Richardson and Kimura (2016) and Richardson and Ternes (2018) discuss building materials, polymers and/or plastics as emerging contaminants in water (mainly drinking) and highlight the need for further investigation of these products regarding their potential to lead to DBP formation. Additionally, Tomboulian et al. (2004) provide a critical in-depth summary of the impact of building materials used in drinking water systems on the quality of the water. While their review is focused on studies involving the production of taste and odour compounds, it identifies several categories of building materials found to leach under drinking water conditions, including pipes and liners (e.g. cement/concrete, polyvinylchloride (PVC)/chlorinated-PVC, polyethylene, polyurethane coatings and liners, epoxy coatings and liners), joining and sealing materials (e.g. adhesives, caulk and flux), gaskets and O-rings (e.g. nitrile-butadiene rubber and styrene butadiene rubber (SBR)), lubricants (e.g. grease, silicones, primers and sealants), solder and thread compound. While leaching of building materials were observed in almost all studies summarised by Tomboulian et al. (2004), the review highlights that product leaching should be regarded on a case-by-case situation as "unusual chemical contamination may arise from certain conditions related to the surface application of coatings and adhesives, or from non-compliance issues (e.g. addition of solvents or other chemicals not in the approved formulation)".

Limited studies investigating the effect of disinfectants (e.g. chlorine or chloramine) on building materials exist (e.g. Heim and Dietrich, 2007a, 2007b, Nagisetty et al., 2014, 2010; Schoenbaechler, 2007). Although not investigated under conditions analogous to swimming pools, a key study by Nagisetty et al. (2014) investigated the impact of several building material components (including natural rubber, styrene-butadiene-rubber (also referred to as styrene-butadiene co-polymer; SBR) and sulfur-cured ethylene-propylene-diene monomer) on chemical water quality when exposed to chloramine over time. While several reported outcomes of this study (e.g. an increase in turbidity and detection of a range of organic compounds were observed for all building materials investigated) demonstrate the potential of building materials to impact chemical quality of

real waters, perhaps the most interesting finding by Nagisetty et al. (2014) is the change of organic compounds detected over the duration of their study. The authors propose that different organic compounds are released or formed at different times, which is likely influenced by the material matrix of the building material(s)/compound(s), particularly the degradation of the material(s) and the penetration of oxidant into the material(s) matrix. Reflective of their results, the authors theorise that the release of organic compounds contained within a material matrix (e.g. additives or fillers) will occur initially, while organic compounds formed as by-products (i.e. DBPs) will be formed over longer periods of time as further material degradation and leaching occurs (Nagisetty et al., 2014). As also noted by Tomboulian et al. (2004), Nagisetty et al. (2014) further suggest that building materials are likely to exhibit greater leaching after new installation or maintenance and theorise that leaching chemicals, while differing over time, will decrease in concentration due to flushing (e.g. in drinking water treatment systems).

Although these ideas of Tomboulian et al. (2004) and Nagisetty et al. (2014) are based on studies of building materials under conditions of drinking water distribution systems, it is reasonable to hypothesise that similar phenomena may also be observed for building materials in swimming pools and spas. In fact, we hypothesise that, compared to drinking water distribution systems, pools and spas would result in (i) an enhanced leaching of organic compounds from building materials, due to the generally higher oxidant residuals employed, and (ii) an increase in concentration of any leached organic compounds over time (and subsequently formed DBPs), as a result of the continual recirculation and minimal fresh water input (flushing or dilution) in pools, particularly as many pools seldom undergo a complete water renewal.

In our previous study of the chemical water quality of two newly built, filled and opened pools (**Chapter 5**), high concentrations of non-purgeable organic carbon (NPOC) and DBPs were measured both before and after the pools were opened. Consistent with the ideas of Tomboulian et al. (Tomboulian et al., 2004) and Nagisetty et al. (Nagisetty et al., 2014), the building process and/or new pool infrastructure was proposed to have had a significant impact on the water quality in these pools. In the current study, we provide evidence for building materials as an additional potential source of organic compounds and DBP precursors in swimming pool waters, by investigating a variety of commercial building materials, as well as one of their individual components, under conditions representative of swimming pools.

6.3. Methodology

6.3.1. Analytical Standards and Reagents

Excluding commercial building material samples which were provided directly from relevant suppliers, all chemicals and reagents were of analytical grade (>98%) and purchased from a range of suppliers including AccuStandard (Connecticut, USA), CanSyn Chemical Corporation (Ontario, Canada), CDN isotopes (Quebec, Canada), Sigma Aldrich (Sydney, Australia) and Thermo Fisher (Victoria, Australia). Where applicable, experiments used ultrapure water (18.2 MΩ-cm resistivity) which was purified by an ELGA PURELAB Ultra purification system.

6.3.2. Selection of Commercial Products and Model Compound as Precursors

A comprehensive investigation of the composition of building materials used during construction of the two newly built pools (**Chapter 5**) was undertaken in partnership with the architect, builder and pool management. Two key products, and a likely component of one of these products, were identified as potential DBP precursors based on their chemical composition. These two products are approved and certified for use in swimming pools in Australia. These products/component included a commercial concrete powder used for the construction of pool walls, flooring and/or grouting of tiles, and a commercial latex additive which was added to the concrete. The liquid latex additive was comprised of a styrene-butadiene copolymer (SBR, 17 to 20% by weight) in water. In addition to the commercial building products, styrene (an individual monomer of the SBR) was investigated.

6.3.3. Preparation of Standard Solutions and Model Compound Solutions

DBP, NPOC and total organic chlorine (TOCl) standard stock solutions (1 g L^{-1}) were prepared by weighing out neat compounds into acetone, methanol or ultrapure water, as per individual method requirements, with secondary solutions prepared by further dilution. Calibration standards were prepared by fortifying ultrapure water samples with target DBP/analyte secondary solutions, and relevant internal and surrogate standards where applicable, as per individual method requirements.

Commercial building material products were prepared as per the manufacturer's instructions and presumably as done during the construction of the earlier investigated pools (**Chapter 5**). Briefly, concrete was prepared by mixing the commercial powdered concrete with ultrapure water (4:1), while the latex samples were prepared by mixing commercial powdered concrete with the latex additive (4:1). Resulting mixtures were poured into individual silicone stick moulds (approx. 1.5 x 1.7 x 7.5 cm) and, as per the manufacturer's

instructions, were allowed to cure for at least 14 days prior to contact with water. Concentrated latex additive (solid) was prepared by allowing commercial latex additive (liquid) to naturally evaporate, affording a white solid.

6.3.4. Analytical Methods

Free and total chlorine equivalents were measured by a DPD colorimetric method using a pocket colorimeter (HACH; 5870000). NPOC was analysed by high temperature combustion with non-dispersive infrared detection using a Shimadzu total organic carbon analyser (TOC-L). TOCl, also referred to as adsorbable organic chlorine (AOCl), was analysed using activated carbon adsorption-high temperature combustion-ion chromatography (Kristiana et al., 2015), the instrument being comprised of a TOX sample preparator (TXA-03, Mitsubishi Chemical Analytech, Japan), an automatic quick furnace (AQF-100, Mitsubishi Chemical Analytech, Japan) and an on-line ion chromatography system with conductivity detection (ICS-3000, Dionex, USA). Individual DBPs were analysed by an Agilent 6890N gas chromatograph coupled with a 5975 mass selective detector (MSD) running in electron ionisation (EI) mode (70 eV) using respective methods. Trihalomethanes (THMs; trichloromethane, bromodichloromethane, dibromochloromethane and tribromomethane) were analysed by headspace solid-phase microextraction (HS-SPME) GC-MS using a simplified version of the method of Allard et al. (2012). Here, a lower quantity of sodium sulfate (1.67 g) was used, and cryogenic cooling of the GC oven temperature was not employed. Haloacetic acids (HAAs; chloroacetic acid (CAA), dichloroacetic acid (DCAA) and trichloroacetic acid (TCAA) were analysed as their corresponding methyl esters by derivatisation (using dimethyl sulfate) HS-SPME GC-MS (Sa et al., 2012; Sarrión et al., 2000). Trichloroacetaldehyde (chloral hydrate, CH) was analysed by liquid-liquid extraction (LLE) GC-MS (US EPA, 1995). All analyses were performed within 12 hours of sample collection which included quenching the oxidant residual with sodium sulfite (10% molar excess). All quantification was via external calibration and made use of surrogate and internal standards where possible (THMs: 1,2-dibromopropane; HAAs: 2-bromo- and 2,2-dichloro-propanoic acid; CH: 1,2-dibromopropane-d6 and 1,1,2,2-tetrachloroethane-d2), that is, quantification was based on response ratios (analyte response/surrogate standard response). Samples were analysed in duplicate with reported concentrations representing the calculated average.

6.3.5. Disinfection By-Product Formation Potential Studies Under Swimming Pool Conditions

Ultrapure water samples buffered at pH 7.5 with phosphate buffer (20 mM) were used in all experiments. Individual concrete and latex 'models' were prepared by adding relevant sticks (5 per 1 L) and sample volumes were adjusted so as to leave no headspace in sealed reaction flasks. HOCl solution was added to achieve an initial target chlorine residual of 4 mg L^{-1} (56 μM), which was selected as it is the upper minimum legal limit of chlorine concentrations employed in Australian pools (NHMRC, 2008; Western Australian Department of Health, 2013). Reaction flasks were stored at room temperature (24 °C), in the absence of light, and mixed manually by repeatedly inverting reaction flasks (1 minute, twice daily) for the duration of the experiments. To represent swimming pools where a constant oxidant residual is maintained, chlorine concentrations were measured daily (where free chlorine concentrations were observed to decay, on average, 43 and 45% for concrete and latex models, respectively), with additional chlorine added to return the residual concentration to the target concentration of 4 mg L^{-1} (56 μM). Upon sample collection for analysis of NPOC and trichloromethane, additional buffer solution was added to reaction flasks in order to maintain constant volume and return vessels to headspace free conditions, with dilution factors applied during quantification. Furthermore, standardisation for mass of reactant (sticks) was conducted where possible.

6.3.6. Disinfection By-Product Formation Potential Studies Under Laboratory Conditions

Chlorination of the several possible DBP precursors either (i) identified as components of commercial building materials (leachates of concrete and latex models (those used in **Section 6.3.5**), commercial latex product (as supplied, liquid) and concentrated latex product (solid)) or (ii) selected as a model compound (styrene monomer) was carried out. All experiments were performed using ultrapure water (500 mL) buffered at pH 7.5 with phosphate buffer (20 mM) and were performed at room temperature (24 °C), in the absence of light and without headspace. As a known carbon content was available (either via NPOC measurement or calculation) for all potential DBP precursors, all models were standardised based on molar carbon content. While a precursor concentration of 150 μM C was selected for all models, due to the low mass (36 mg) required to achieve the target concentration for the solid latex concentrate model, a more easily measured product mass of 0.5 g was selected for the solid latex concentrate model. HOCl solution was added to all reaction flasks to achieve an initial target chlorine residual of 53 mg L^{-1} (750 μM). While this chlorine concentration is significantly higher than those typically employed in real pools, this concentration was selected to observe maximum DBP formation. Reaction flasks were continually shaken mechanically (200 rpm) for a total reaction time of 36 hours. As a higher

precursor concentration was used for the concentrated latex model (i.e. >2000 μM C compared to 150 μM C), to easily compare DBP formation across all investigated models, measured DBP concentrations for this model were converted to numerically equivalent concentrations by multiplying by the concentration factor of 0.072 (the product mass required to achieve 150 μM C (36 mg) divided by the product mass actually employed (0.5 mg)).

6.4. Results and Discussion

6.4.1. Leaching of Non-Purgeable Organic Carbon from Commercial Building Materials Under Swimming Pool Conditions

In order to investigate whether selected building materials used in swimming pools could leach organic compounds into water, NPOC concentrations were followed over a period of 21 days in concrete and latex models under swimming pool conditions (**Section 6.3.5**). The impact of chlorine on the leaching of these building materials was investigated by comparing NPOC concentrations in concrete and latex models containing chlorine (4 mg L^{-1}, 53 μM) to NPOC concentrations in corresponding models where no chlorine was added.

Comparing NPOC concentrations standardised by mass of product, NPOC continued to leach from the latex model over the 21-day experiment, while the NPOC concentration quickly reached a constant value in the concrete model, such that a significantly higher level of NPOC was measured in the latex model (**Figure 6-1(a)**) compared to the concrete model (**Figure 6-1(b)**) after 21 days. Over the 21 days, the NPOC concentrations in the concrete model were in the range of 0.02 to 0.04 mg C g^{-1} (measured as 2.3 to 5.8 mg L^{-1}), while concentrations of 0.02 to 0.17 mg C g^{-1} (measured as 3.0 to 23 mg L^{-1}) were observed for the latex model. Significant leaching of organic carbon from both materials, but particularly from the latex additive, can therefore occur under conditions commonly used in swimming pools. This may at least partly explain the occurrence of relatively high concentrations of NPOC in pools in our previous investigation (**Chapter 5**): where concentrations of up to 16 mg L^{-1} were measured in pools prior to the opening of the facility; which, as discussed in greater detail in **Chapter 5**, are generally higher than NPOC concentrations reported in other pools.

In the concrete models, and the latex models up until day 12, higher levels of NPOC were measured in the chlorinated models compared to those measured in models where no chlorine was added, suggesting that chlorine may enhance the leaching of NPOC under these conditions. After day 12, similar or lower concentrations of NPOC were measured in the chlorinated latex model compared to those in the model where no chlorine was added. This may be due to the formation of volatile DBPs (e.g. trichloromethane), from the reaction of

leached NPOC and chorine, and loss of these volatile DBPs in the NPOC analytical method, since volatile compounds have been shown to be partly lost during purging in the NPOC analysis and may therefore not be fully recovered in the NPOC measurement (**Figure A6-1**).

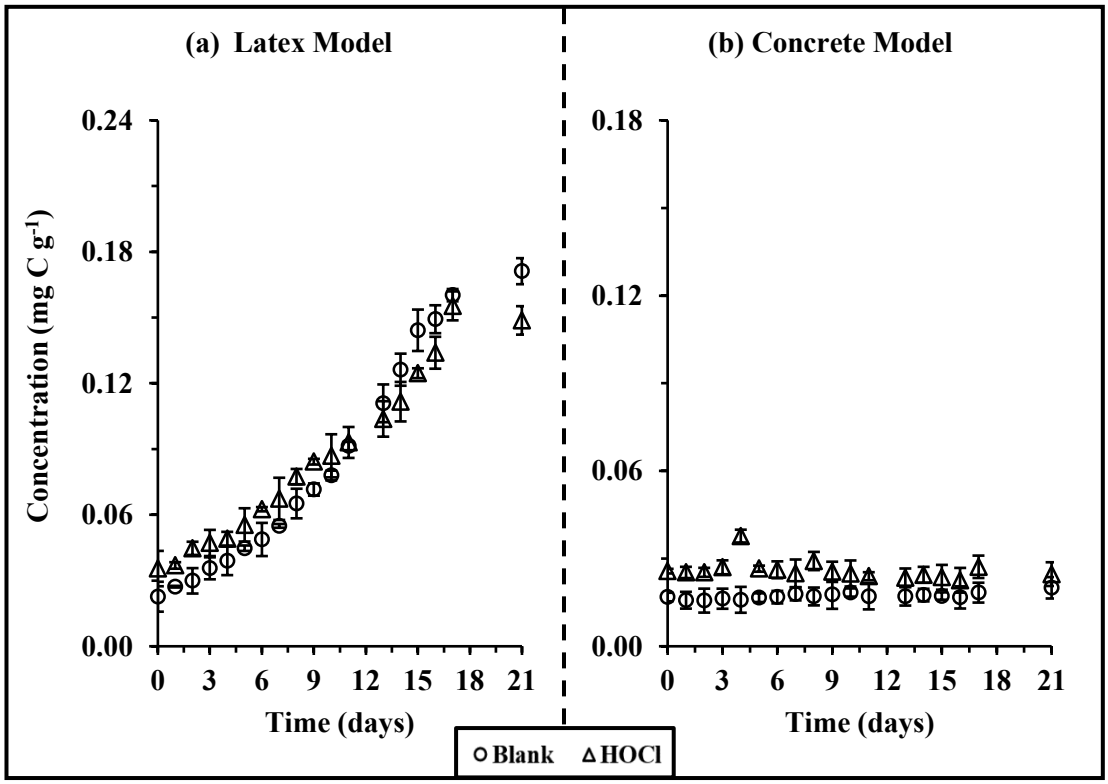

Figure 6-1: Concentrations of non-purgeable organic carbon (NPOC) leaching from (**a**) latex model and (**b**) concrete model under swimming pool conditions. All values were corrected for dilution and standardised by mass of product. Values represent the average of replicates and are presented as mg carbon per g product.

6.4.2. Disinfection By-Product Formation from Commercial Building Materials Under Swimming Pool Conditions

To investigate whether DBPs can be formed from commercial concrete or latex additive products and/or their leachates under swimming pool conditions, chlorination models over 34 days were performed (**Section 6.3.5**). Briefly, concrete and latex models were prepared in ultrapure water (4 L) containing concrete or latex (20 sticks each). Models were buffered with phosphate (pH 7.5, 20 mM) and the initial concentration of chlorine (4 mg L^{-1}, 53 μM) was maintained by daily addition. Models were analysed periodically over 34 days for NPOC and trichloromethane concentrations. To minimise the impact of dilution, a total of 10 samples from each model was taken over the duration of the investigation. Trichloromethane was selected as an indicator of DBP formation due to its common occurrence in swimming pools (Carter and Joll, 2017; Manasfi et al., 2017).

Following similar trends as the smaller scale 21-day study (**Section 6.4.1**), NPOC continued to leach in the latex model, while the NPOC concentrations remained fairly constant in the concrete model, in the larger scale 34-day investigation (**Figure 6-2(a)**). Again, in the 34-day experiment, significantly higher NPOC concentrations (0.01 to 0.08 μM C g^{-1}) were measured in the latex model compared to concentrations (0.004 to 0.01 μM C g^{-1}) measured in the concrete model (**Figure 6-2(a)**). It is important to note that significantly less (less than 50%) NPOC leached in the two models in the 34-day experiment than in the 21-day experiment. While the scale of these two experiments was different, another factor which was different was the curing time of the building material 'sticks'. All commercial building material 'sticks' were prepared concurrently as per the manufacturer's recommendations (**Section 6.3.3**, i.e. left to cure for at least 14 days prior to contact with water). As a result, sticks used during the 21-day study had a total curing time of 2 weeks, while those used in the 34-day study had a significantly greater curing time (6 weeks). It is possible that the greater curing time may have resulted in more loss of volatile organic components from the sticks used in the 34-day study, resulting in lower NPOC concentrations compared to those in the 21-day investigation. The potential importance of maximising curing time of building materials used in pools before filling the pools is thus highlighted. While current Australian manufacturers' procedures and regulations for pre-treatment of building materials are largely designed to ensure correct structural integrity of the products, future consideration should be given to the effect of procedures on pool water quality.

Trichloromethane was detected in all chlorinated models (**Figure 6-2(b)**), where a linear increase in concentration was observed (r^2 = 0.79 and 0.91 for concrete and latex models, respectively). Trichloromethane concentrations were also reported to increase during the chlorination of styrene-butadiene rubber (SBR), as well as other building materials, in the study by Nagisetty (2014), where SBR is the main constituent of the latex product investigated here. While similar trends were observed in each model in the current study, significantly and consistently greater (59 to 80%) concentrations of trichloromethane were measured in the latex model compared to the concrete model, suggesting that latex is a more active trichloromethane precursor than concrete. Overall, both the commercial concrete and latex additive products leach NPOC when exposed to water and can lead to DBP formation under conditions commonly employed in swimming pools.

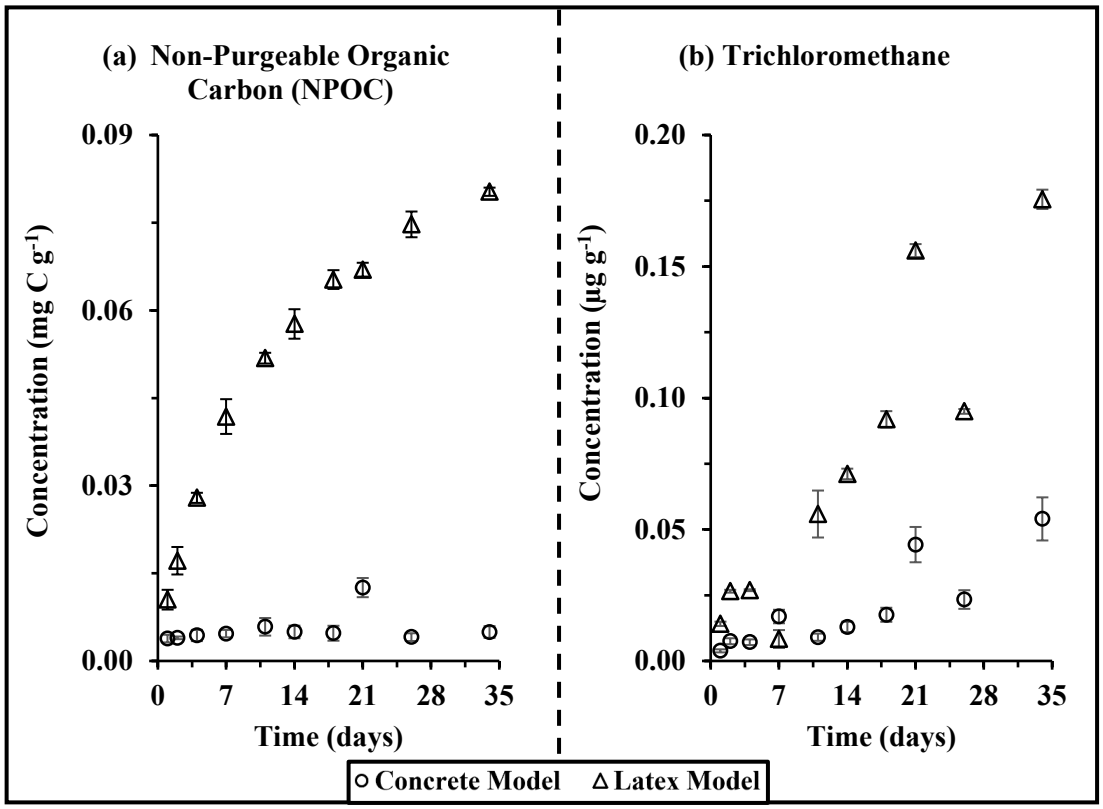

Figure 6-2: Concentrations of **(a)** non-purgeable organic carbon (NPOC) leaching and **(b)** trichloromethane formed from commercial building materials under swimming pool conditions over the 34-day study. All values represent the average of replicates, have been corrected for dilution and standardised by mass of product.

6.4.3. Disinfection By-Product Formation from Commercial Building Materials Under Laboratory Conditions

With the potential for DBP formation from commercial building materials under conditions commonly employed in swimming pools demonstrated, further studies were conducted in an attempt to broaden the knowledge in this area and to greater understand the potential of DBP formation from these commercial products. Here, the broader suite of chlorinated organic DBPs, measured as TOCl, was investigated by chlorination under laboratory conditions of models of several commercial building materials: leachates of concrete and latex (obtained by filtration of non-chlorinated models after 34 days), latex product (as the supplied liquid: 17 to 20% SBR (by weight) in water) and the latex concentrate (solid) (**Section 6.3.6**). Briefly, models were prepared by adding relevant products (150 µM C, except latex concentrate where 0.5 g was added) to ultrapure water (500 mL) buffered with phosphate buffer (pH = 7.5, 20 mM) in headspace free reaction flasks. Each model was chlorinated for 36 hours at an initial chlorine concentration of 53 mg L^{-1} (750 µM) HOCl, where chlorination occurred at room temperature (24 °C), in the absence of light and with continual shaking (200 rpm).

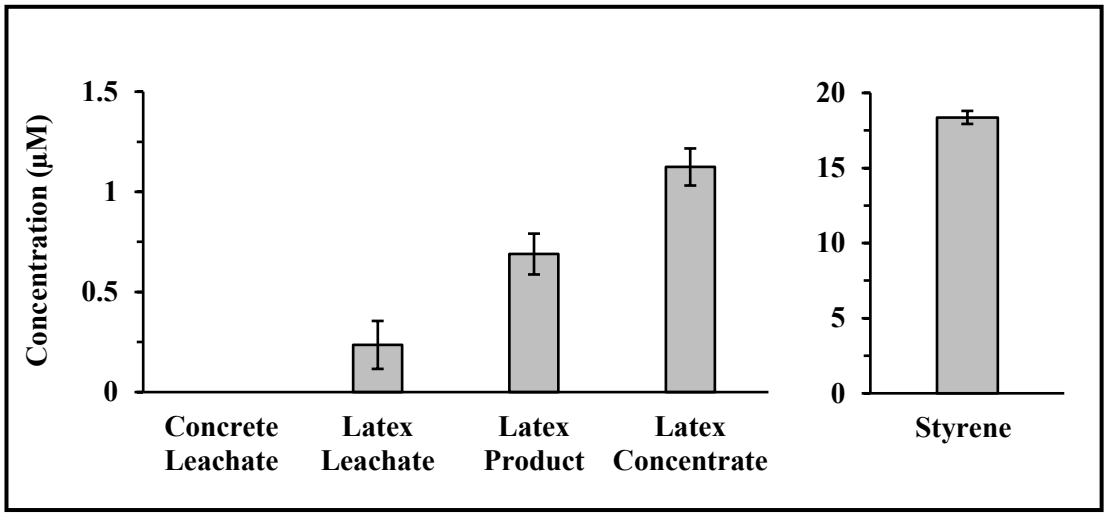

Figure 6-3: Total organic chlorine (TOCl) measured in chlorinated models of commercial building materials and styrene.

Concentrations of TOCl measured in each model of commercial building material are presented in **Figure 6-3**. With the exception of the concrete model, where concentrations were below the method limit of detection (LOD; 0.14 µM, 5 µg L^{-1} as Cl^-), all other models contained significant concentrations of TOCl. Lowest concentrations of TOCl were observed in the latex leachate model (0.24 µM), followed by the latex product and latex concentrate models, 0.70 and 1.2 µM, respectively. While Nagisetty et al. (2014) did not measure TOCl concentrations, they did report the formation of trichloromethane (which is measured as a component of the TOCl measurement (Kristiana et al., 2015)) as a result of the chlorination of a range of building material components including SBR, a major constituent of the latex additive investigated here. The increasing TOCl concentrations observed across these models in the current study (**Figure 6-3**, i.e. latex leachate < latex product < latex concentrate) may be a result of the increasing proportion of SBR in these models. The absence of measurable TOCl in the concrete model may also be reflective of the absence of SBR in the concrete model. The lower reaction time of this study (36 hrs) compared to that used in previous models (up to 34 days) may also assist in explaining the absence of TOCl observed in the concrete model here, despite the detection of trichloromethane (and hence TOCl) in the concrete model under swimming pool conditions (**Section 6.4.2**). Dilution of the concrete leachate (approx. 18x to achieve the target starting concentration of 150 µM C), and a consequential reduction in other potential DBP precursors, may also give rise to the absence of any detectable TOCl in the concrete model here. While conditions of this laboratory DBP formation study may not be analogous to those employed in swimming pools (e.g. NPOC (potential precursors) or chlorine concentrations), this study demonstrates that commercial building materials, particularly the latex additive, have the potential to lead to the formation of chlorinated DBPs (TOCl), and may be a cause of the elevated concentrations of some DBPs in pools in our previous study (**Chapter 5**).

6.4.4. Disinfection By-Product formation from Styrene Under Laboratory Conditions

In order to gain an understanding of DBP formation from commercial building materials, styrene (a component of the SBR of the latex additive) was chlorinated under laboratory conditions and TOCl was analysed (**Section 6.3.6**). An individual model containing the styrene monomer (150 μM C) was prepared in ultrapure water (500 mL) buffered with phosphate buffer (pH = 7.5, 20 mM). Chlorination (53 mg L^{-1} (750 μM) HOCl) occurred at room temperature (24 °C), in the absence of light and with constant shaking (200 rpm) over 36 hours.

TOCl (18 μM) was measured under the conditions tested (**Figure 6-3**). This concentration is significantly greater than those measured in the previous models (up to 1.2 μM, **Section 6.4.3**), suggesting styrene is a more active DBP precursor. While styrene is not listed as an ingredient of the commercial latex product, it is a monomer of the SBR used during latex preparation. As styrene monomer, in addition to fifteen other compounds, has been reported as a contaminant detected in commercial SBR (Tomboulian et al., 2004), it is therefore reasonable to suggest that styrene, at least in part, can account for the DBP formation observed in the latex models. The formation of several chlorinated DBPs (e.g. trichloromethane and *N*-chloro-1-phenyl-2-chloroethylamine and *N*,*N*-dichloro-1-phenyl-2-chloroethylamine (the latter two in the presence of ammonium ions)) from the chlorination of styrene has previously been reported (Chaidou et al., 1999; Nojima et al., 1994), consistent with the observed formation of TOCl from styrene in the current study.

6.4.5. Identification of Specific Disinfection By-Products

More in-depth studies were carried out to determine if these commercial building materials and their specific component(s) could lead to the formation of specific DBPs. Here, concrete leachate, latex leachate, latex product, latex concentrate and styrene models (as in **Sections 6.4.3** and **6.4.4**) were employed to investigate their potential to form DCAA, TCAA, CH and trichloromethane upon chlorination, as these DBPs were measured at elevated concentrations in the pools in our previous study (**Chapter 5**).

At least one of the investigated DBPs was detected after chlorination in all models (**Figure 6-4**). CH was the only DBP measured in the latex product model (0.005 μM, 0.9 μg L^{-1}). The absence of other DBPs in the latex product model is likely a reflection of the conditions employed: e.g. significant dilution of active precursors to achieve target initial carbon concentrations, the solid nature of the precursor which required additional steps such as leaching, and/or the short reaction time of the models.

While not detected after chlorination in the concrete model (LOD=0.004 μM, 0.65 μg L^{-1}), CH was measured at concentrations of 0.006 to 0.151 μM (0.90 to 25 μg L^{-1}) in all models containing the latex additive, as well as in the styrene model (0.022 μM, 3.7 μg L^{-1}). These results suggest that while the commercial concrete product did not act as a precursor to CH, precursors to CH were present in the commercial latex additive. Although not detected in the latex product model (LOD=0.007 μM, 0.85 μg L^{-1}), trichloromethane was the dominant DBP in all other models, where higher concentrations were measured in models containing the commercial latex product or its constituent (i.e. the latex leachate, latex concentrate and styrene models) compared to that measured in the concrete model: 0.14 to 0.25 μM (17 to 30 μg L^{-1}) compared to 0.12 μM (14 μg L^{-1}), respectively. These concentration differences suggest that while the commercial concrete product can act as a precursor to trichloromethane, the latex additive is more active as a precursor in comparison, where SBR (the main constituent of the latex additive investigated) has previously been demonstrated to be a precursor to trichloromethane (Nagisetty et al., 2014). Excluding the latex product model where concentrations were below detection limits (LOD = 0.003 μM, 0.50 μg L^{-1}), TCAA was detected in all other building material models at concentrations of 0.004 to 0.062 μM (0.6 to 10.4 μg L^{-1}). DCAA was only detected in the concrete, latex leachate and latex concentrate models, where concentrations of up to 0.206 μM (27 μg L^{-1}) were measured. In a similar trend to that observed for trichloromethane, the higher concentrations of DCAA and TCAA formed in the latex containing models compared to that measured in the concrete model suggest that the latex additive is a more active precursor for DCAA and TCAA.

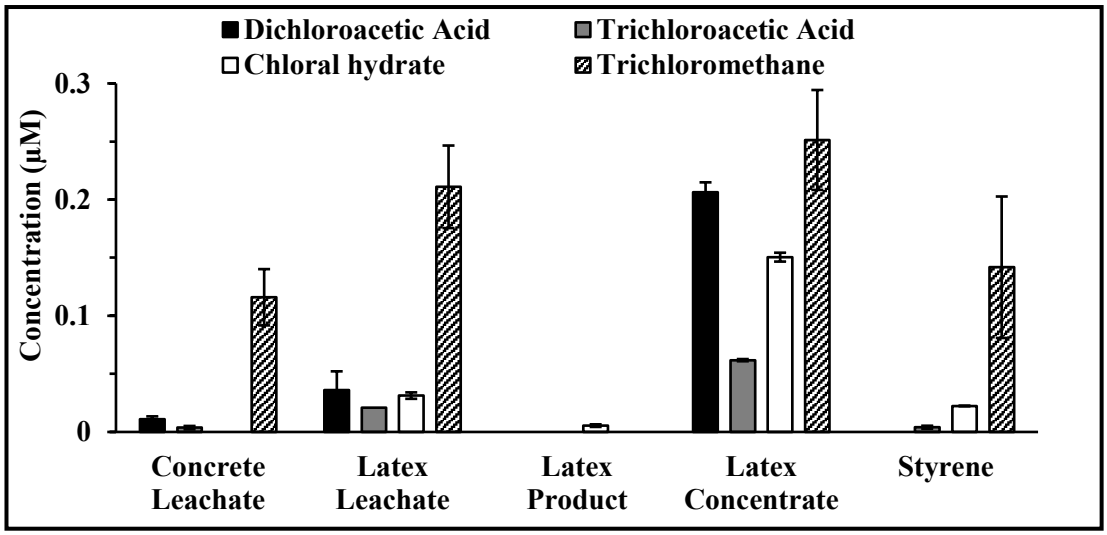

Figure 6-4: Concentrations (μM) of disinfection by-products (DBPs) measured in chlorinated models of commercial building materials and a model compound.

Chaidou et al. (Chaidou et al., 1999) have also reported the formation of trichloromethane from the chlorination of styrene. The occurrence of these three DBPs in the latex containing models can therefore be attributed, at least in part, to potential styrene monomer remaining in the SBR co-polymer within the latex additive. The similar concentrations of CH measured in the latex leachate and styrene models are consistent with styrene monomer being the predominant CH precursor leaching from the latex additive. In contrast, the lower concentrations of TCAA and trichloromethane in the styrene model compared to those in the latex leachate model demonstrate that styrene alone cannot fully account for the total concentrations of these DBPs formed. We consider it possible that i other impurities, e.g. potentially butadiene (the other monomer of the SBR co-polymer), which may also act as precursors to TCAA and trichloromethane, leached from the latex additive.

While the commercial concrete product was identified as a precursor to DCAA, TCAA and trichloromethane, the commercial latex product was found to be more active as a precursor to all four investigated DBPs. Since these commercial building materials were used during the construction of two pools in our previous study (**Chapter 5**), these materials are likely to be partly responsible for the elevated concentrations of these four key DBPs in the two pools. CH was detected in all models containing the latex additive, as well as in the styrene model, but not in the concrete model. The commercial latex product is therefore potentially partly responsible for the high concentrations of CH in the two pools.

6.5. Conclusions

This study introduces the concept of building materials as an additional source of organic input to swimming pool waters, and investigates the potential impact of these materials on chemical water quality. Controlled laboratory scale models were prepared under conditions (i.e. pH and chlorine concentrations) reflective of Australian swimming pools, in order to investigate the leaching and DBP formation potential of two commercial building materials.

Over a 21-day study, NPOC continued to leach from the latex model, while the NPOC concentration quickly reached a constant value in the concrete model, where concentrations ranged from 3.0 to 23 and 2.5 to 5.8 mg L^{-1}, respectively. Both of the investigated commercial products therefore have the potential to introduce organic carbon into swimming pools, and the leaching of organic carbon appeared to be enhanced in the presence of HOCl. Chlorination of several forms of the two building materials resulted in the formation of chlorinated organic compounds (i.e. TOCl: 0.24 to 1.2 μM) for almost all models tested. While chlorination here was performed at a higher concentration than employed in

swimming pools, the potential of commercial building materials, particularly the commercial latex additive, to form chlorinated DBPs was demonstrated.

While TOCl was used as an overall indicator of DBP formation, the formation of several individual DBPs, namely DCAA, TCAA, trichloromethane and CH, from the laboratory scale chlorination of five building material models was also investigated. All four DBPs were measured in almost all models containing the latex additive, indicating that the latex additive contains precursors to these DBPs. Trichloromethane, DCAA and TCAA concentrations were higher in the latex models compared to those measured in the concrete model, demonstrating that while both the commercial concrete and latex additive products can act as a precursor to these DBPs, the latex additive is a more active precursor. CH was detected in all models containing the latex additive, but not in the concrete model, suggesting that only the commercial latex product contains CH precursors.

Chlorination of styrene (a monomer of SBR; SBR being a major constituent of the commercial latex additive) led to the formation of chlorinated DBPs (measured as TOCl) with trichloromethane, TCAA and CH identified, demonstrating its ability to act as a precursor to these DBPs. The occurrence of these three DBPs in the latex containing models may therefore be attributed to potential styrene monomer remaining in the SBR co-polymer within the latex additive, although future investigations should confirm the presence of styrene in the latex additive. Commercial building materials should therefore be considered as an additional source of organic material and resultant DBPs in swimming pool waters, with results of this study suggesting more stringent regulations on building materials may be required.

While the current study provides evidence that commercial building materials and/or their components can lead to the formation of DBPs under conditions reflective of real swimming pools, further work should be carried out before drawing more definitive conclusions. A comprehensive characterisation of building material leachates is strongly recommended, where the identification of potential DBP precursors should be targeted. While chlorine concentrations employed in the models under swimming pool conditions (i.e. those equal to the Australian upper legal limit; 4 mg L^{-1}) provide an indication of what may be observed in Australian swimming pools, these conditions may not be reflective of possible conditions in pools in other countries, particularly as many countries employ significantly lower chlorine concentrations. For comparison, and to provide results more applicable to the international pool community, similar investigations should be performed under conditions (e.g. pH and chlorine concentrations) reflective of those employed in pools of other countries. Furthermore, while DBP formation (e.g. HAAs, CH, TOCl) was

demonstrated under the laboratory conditions selected here (i.e. Cl_2:P = 5, chlorine dose = 53 mg L^{-1}), additional controlled laboratory studies should be conducted to investigate a wider range of conditions (e.g. different Cl_2:P ratios and chlorine doses), with a focus on including not only laboratory conditions, but also those reflective of real swimming pools. Such investigations would provide further information regarding resulting DBP formation under a wider range of conditions, allowing more definite conclusions to be drawn. Future investigations should also focus on assessing the potential leaching of organic material from a larger range of commercial building materials (e.g. piping or filter components) and any resultant DBP formation, where the current study will provide the groundwork for these future investigations.

6.6. Acknowledgements

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CHAPTER 7

CONCLUSIONS AND RECOMMENDATIONS

A comprehensive survey of disinfection by-products (DBPs) from eight DBP classes (haloacetic acids (HAAs), haloacetaldehydes (HALs), trihalomethanes (THMs), haloketones (HKs), haloacetonitriles (HANs), halonitromethanes (HNMs), haloacetamides (HAAms) and *N*-nitrosamines) in fifteen public swimming pools of differing types and treatment methods across six facilities was conducted. An additional in-depth investigation of the chemical water quality of two newly built, filled and opened, public swimming pools was performed, focusing on the occurrence of a range of DBPs and several other general water quality parameters in these pools over fifteen months. Controlled laboratory scale studies were performed to further understand several observations of these two investigations of real pool waters.

This Thesis highlights that the occurrence and formation of DBPs in swimming pools may be greater than other disinfected waters (e.g. drinking waters). During both the comprehensive survey and the in-depth investigation, almost all samples of pools were found to contain DBPs at significantly higher (2 to 1941 times higher) concentrations than those measured in their respective filling waters (based on total molar concentrations), suggesting that filling waters are likely an insignificant source of many of the investigated DBPs in pool waters. Furthermore, the estimated chronic cytotoxicity of the pools was calculated to be significantly and continually greater than respective filling waters, indicating the potentially increased risk(s) of pool water compared to filling waters (here, drinking waters). While a significant finding, these results are only indicative of the potential health impact of these DBPs, as these results were derived from calculation. Further studies must be conducted performing laboratory based toxicity studies on real pool waters. Furthermore, all three uptake mechanisms (ingestion, absorption and inhalation) must be considered during future studies assessing the health impact of DBPs in the pool environment.

Considering pools in the comprehensive survey, of the sixty-four investigated DBPs, 26% were measured in all pool water samples, with 76% detected in at least one of the investigated pools. Based on average molar concentrations across all pools, HALs were the dominant DBP class, followed by (in order of decreasing contribution) *N*-nitrosamines, HAAms, THMs, HANs, HKs and HNMs. Chloral hydrate (CH) was identified as a significant DBP in chlorinated pool waters since, in addition to being measured in all chlorinated pools (202 to 1313 $\mu g\ L^{-1}$), it was the dominant DBP (based on molar concentrations). Across all pools, several other DBPs (HAAs, HAAms and *N*-nitrosamines) are also noted to be of potential concern due to their detection at concentrations greater than previously reported in pools, particularly chloroacetic acid (up to 266 $\mu g\ L^{-1}$), dichloroacetic acid (DCAA; up to 200 $\mu g\ L^{-1}$), trichloroacetic acid (TCAA; 479 $\mu g\ L^{-1}$), dichloroacetamide (up to 56 $\mu g\ L^{-1}$), dibromoacetamide (up to 736 $\mu g\ L^{-1}$), dibromochloroacetamide (up to 145 $\mu g\ L^{-1}$), trichloroacetamide (up to 65 $\mu g\ L^{-1}$), *N*-nitrosodimethylamine (up to 456 $ng\ L^{-1}$),

N-nitrosopiperidine (up to 5.9 ng L^{-1}), *N*-nitrosodipropylamine (up to 7.7 ng L^{-1}) and *N*-nitrosodibutylamine (up to 1093 ng L^{-1}). The relatively higher disinfectant residuals required to be employed in Australian pools, and poor pool management (particularly disinfectant residuals and pH control), were identified as possible reasons for these high DBP concentrations. Non-purgeable organic carbon (NPOC) was identified as a determining factor for HALs, HAAms, HAAs, HKs and *N*-nitrosamines, as their occurrence was correlated to measured NPOC concentrations in the investigated pools.

Of the pool types investigated during the comprehensive survey, hydrotherapy and spa pools are potentially of greater concern due to the higher total DBP molar concentrations measured. Several factors that may affect DBP concentrations in these pool types are suggested, e.g. water temperature, total pool water volume and DBP precursor input. While some slight differences in DBP concentrations were observed between pool treatment methods, the different pool treatment methods were suggested to have a less significant impact in terms of DBPs compared to other factors such as pool type or swimmer input.

This Thesis adds to the limited knowledge of DBPs in bromine treated pools, particularly for *N*-nitrosamines, as no known study has investigated *N*-nitrosamines in bromine treated pools. Furthermore, this Thesis is the first known report of total nitrogen in bromine treated pools, where 40 mg L^{-1} was measured in a training pool treated with bromochlorodimethylhydantoin (BCDMH). On a molar basis, while tribromomethane was the dominant DBP measured (0.5 μM, 132 μg L^{-1}), the significantly higher concentrations of HAAms measured in this training pool (4.1 μM) compared to those measured in chlorinated pools (up to 0.6 μM) are of particular concern due to the increased health risk of HAAms compared to some other DBPs (e.g. THMs or HAAs).

This Thesis is the first investigation of the water quality and occurrence of DBPs in newly built and filled swimming pools, and also adds to the limited information of the fate of DBPs in pools over extended periods (e.g. >1 year). Concentrations of chloroacetic acid, DCAA, TCAA and CH (up to 6100, 26000, 11300 and 3200 μg L^{-1}, respectively) in some samples of the pools were higher than previously reported maximum concentrations, where CH concentrations in one of the pools were almost always higher than any previously reported concentration. Considering total molar DBP concentrations, higher levels of DBPs were generally measured in one of the two pools, where several factors including NPOC concentration, potential organic input from bather load, and operational parameters (e.g. water temperature and chlorine residual) were suggested to have had impact on DBP concentrations. An example, using real pools, which highlights the importance of the establishment of a steady

state balance of mineralisation of organic carbon versus addition of organic carbon to reduce precursors for DBP formation, is provided.

In all real pool investigations, where possible, the total chronic cytotoxicity values of the measured DBPs was evaluated by calculation. Of all pools of the comprehensive survey, spa and hydrotherapy pools had significantly higher calculated cytotoxicity levels compared to those observed for lap or leisure pools, suggesting hydrotherapy and spa pools may potentially be of greater health concern. For all chlorinated pools of the comprehensive survey, HALs were identified as the greatest contributor to the estimated cytotoxicity, representing up to 99% of the total cytotoxicity. In particular, and based on average concentrations, CH was calculated to contribute up to 99% of the overall total cytotoxicity in these pools. HALs were also significant DBPs in the pools of the in-depth study, representing up to 62% of the total DBP molar concentration and up to over 99% of the total estimated cytotoxicity. Furthermore, CH represented up to 97% of the total molar DBP concentrations measured, and in some cases, contributed to over 99.8% of the total estimated cytotoxicity. Even when HALs represented only 1% of the total DBP molar concentration, they were calculated to contribute 85% of the total estimated cytotoxicity, where 99% of these contributions were due to CH. Over the in-depth study, HALs (e.g. CH) were found to be correlated to number of swimmers, suggesting that swimmers may potentially be a significant source of HAL precursors, and, in turn, a significant contributor to DBP derived cytotoxicity. These results highlight the need for further investigations into the input of DBP precursors from swimmers, which should focus on the formation and potential health effects of DBPs, particularly CH.

From the in-depth study of new pools, the presence and subsequent increase of NPOC and some DBPs (e.g. DCAA, TCAA, CH and trichloromethane) prior to, and soon after, opening appears to have been generated from the pool building process and/or new pool infrastructure. Two commercial building materials, a concrete and a latex additive used during the construction of these pools, were investigated for their potential to impact chemical water quality. These materials, upon treatment under conditions commonly employed in Australian pools, were observed to leach significant concentrations of NPOC (up to 23 mg L^{-1} over 21 days) and lead to the formation of chlorinated DBPs (up to 1.2 μM; measured as total organic chlorine (TOCl)). Both commercial products were found to lead to the formation of several DBPs (CH, DCAA, TCAA and trichloromethane), with the latex additive observed to be a more active precursor. Styrene, a monomer of styrene-butadiene rubber (SBR: the main component of the commercial latex additive), was identified as a significant precursor for CH, TCAA and chloroform. Styrene monomer potentially remaining in the co-polymer within the latex additive is therefore a possible precursor for formation of these DBPs from the latex

additive. These results suggest that building materials may be a cause of the elevated concentrations of NPOC and some DBPs in the newly built pools of the in-depth investigation.

For the first time, this Thesis demonstrates the potential of commercial building materials, which have been approved for use in Australian swimming pools, to not only act as a source of organic compounds in pools, but also lead to the formation of DBPs under conditions commonly used in Australian swimming pools. Building materials should therefore be considered as an additional source of organic material and resultant DBPs in swimming pool waters, particularly those that are newly constructed or have recently undergone maintenance. More stringent regulations on building materials may be required. As studies in this area are limited, further investigations into the impact of building materials on the chemical water quality of pools are therefore warranted. Such investigations should assess the impact of a larger range of commercial building materials and/or their components (e.g. cements, piping, filter components, adhesives, glues and sealants) on chemical water quality of pools, with a particular focus on their potential to lead to DBP formation under conditions commonly employed in pools (e.g. comparable pH, oxidant concentrations, water recirculation).

In summary, this Thesis reports the occurrence of a range of DBPs in pool waters, where many of the DBPs were measured at concentrations greater than those previously reported in pools. This Thesis is the first reported quantification of several of these DBPs, the first investigation of several HNMs, and for bromine treated pools, the first investigation of *N*-nitrosamines and detection of a HNM. This Thesis estimated the potential impact of these DBPs at the measured concentrations by calculating the chronic cytotoxicity, finding pool waters were consistently and significantly higher in estimated chronic cytotoxicity than their corresponding filling waters. HALs, particularly CH, were identified as a significant contributor to DBP derived cytotoxicity, with swimmers identified as a potential source of HAL precursors. In laboratory studies, several commercial building materials, which have been approved for use in Australian swimming pools, were demonstrated to lead to DBP formation under conditions similar to those commonly employed in Australian swimming pools. This Thesis provides evidence that further investigations into DBPs in pools are essential. Future studies should further investigate the potential precursors, formation pathways and potential health impacts of CH, investigate DBPs in bromine treated pools, as well as investigate commercial building materials and their impact on DBPs in pools, with this Thesis providing the groundwork for future studies.

APPENDIX 1

Table A1-1: Reported occurrence of trihalomethanes in swimming pool and spa waters.

Country	Pool(s) Type	Disinfection Method	Trihalomethane Concentration in Swimming Pool Water (µg L^{-1}); mean (min-max). Total THMs (TTHMs)	Trichloromethane (Chloroform)	Bromodichloromethane	Dibromochloromethane	Tribromomethane (Bromoform)	Analytical Method	Reference
USA	Outdoor* Outdoor Outdoor	Cl Cl Cl		6 (NR-21) 106 (NR-271) 103 (NR-386)	5 (NR-19) 34 (NR-117) 13 (NR-98)	27 (NR-102) 15 (NR-83) 3 (NR-38)	651 (NR-1166) 2 (NR-8) <1 (NR-6)	LLE GC-ECD	(Beech et al., 1980)
Sweden	NR NR	Cl Br		(50-100)			 (NR-400)	LLE GC-ECD	(Norin and Renberg, 1980)
Germany	Covered	Cl	174 (59-1224)	198 (43-980)	23 (0.1-150)	11 (0.1-140)	4 (nd-88)	LLE GC-ECD	(Lahl et al., 1981)
France	NR NR	Cl Br		256 (43-665) 32 (18-45)			5.4 (1-14) 289 (177-600)	PT GC-ECD	(Chambon et al., 1983)
Italy	NR	Cl		115 (62-179)	8 (6-10)	1.4 (0.8-2)	nd	HS GC-ECD	(Aggazzotti and Predieri, 1986)
Canada	Spa Spa	Cl Br		154 (5-750)		 1253 (37-3600)		PT GC-MS	(Benoit and Jackson, 1987)
Germany	Indoor Indoor	NR NR		95 (41-118) 81 (44-170)	0.5 (0.2-1.5) 9 (6-20)	0.1 (0.05-0.3) 1.5 (1.2-2.2)		GC	(Puchert et al., 1989)
Italy	Indoor	NR		274 (142-349)				HS GC-ECD	(Aggazzotti et al., 1990)
Canada	Indoor	NR		365 (159-568)				HS GC-ECD	(Lévesque et al., 1994)
Italy	Indoor	NR		62 (3-179)				HS GC-ECD	(Aggazzotti et al., 1995)
Germany	Indoor	Cl		(3-28)	(0.7-5.6)	(0.03-6.5)	(nd-2.3)	HS GC-ECD	(Cammann and Hübner, 1995)
Italy	Indoor	Cl		34 (25-43)	2.3 (1.8-2.8)	0.8 (0.5-10)	0.1 (0.1-0.1)	HS GC-ECD	(Aggazzotti et al., 1998)
Greece	Indoor	Cl		8.7 (4-26)	2.7 (0.3-7)	1.2 (0.5-3)	0.3 (0.07-0.9)	PT GC-ECD	(Golfinopoulos, 2000)
Canada	Indoor	Cl		(18-80)				HS GC-ECD	(Lévesque et al., 2000)
Australia	Indoor Indoor Indoor	Cl/Oz Cl Br/Oz	(13-24) (21-87) (107-158)	(13-24) (20-85) nd	(0.1-0.9) (0.2-2) (0.3-0.5)	nd nd (0.8-1.2)	nd nd (106-157)	NR	(Kelsall and Sim, 2001)
Italy	Indoor	Cl	40	33	4.3	1.9	0.4	HS GC-ECD	(Fantuzzi et al., 2001)
UK	Indoor	Cl	132 (57-223)	121 (45-212)	8.3 (2.5-23)	2.7 (0.7-7)	0.9 (0.7-2)	HS GC-ECD	(Chu and Nieuwenhuijsen, 2002)
Germany	Indoor	Cl		7.1-25				PT GC-ECD	(Erdinger et al., 2004)

Table A1-1 continued

Country	Pool(s) Type	Disinfection Method	Trihalomethane Concentration in Swimming Pool Water (µg L^{-1}); mean (min-max). Total THMs (TTHMs)	Trichloromethane (Chloroform)	Bromodichloromethane	Dibromochloromethane	Tribromomethane (Bromoform)	Analytical Method	Reference
Germany	Indoor Outdoor	Cl Cl	21 (35-47)					PT GC-ECD	(Glauner et al., 2005)
Poland	Indoor	Cl		(10-41)	(0.7-5.7)	(0.4-1.6)		TL-HS-DAI GC–ECD	(Kozlowska et al., 2006)
USA	Indoor Outdoor	NR NR		0.1 (nd-0.14) (0.08-0.13)				MIMS	(Li and Blatchley, 2007)
Spain	Indoor	Cl		110 (95-120)	2.2 (2-2.3)			HS0 GC-MS	(Caro and Gallego, 2007)
Spain	Indoor	Cl	80 (63-98)	67 (47-82)	9.3 (5.1-12)	3.2 (1.4-4.6)	1.4 (1-1.9)	HS GC-MS	(Villanueva et al., 2007)
Italy	Indoor	Cl	(27-98)					HS GC-MS	(Aggazzotti et al., 2007)
Spain	Indoor	Cl		127 (85-155)	2 (1.8-2.2)			HS GC-MS	(Caro and Gallego, 2008)
Thailand	Outdoor	Cl	47 (26-65)	20 (9.5-37)	13 (8.9-18)	10 (5.2-23)	3 (nd-6.6)	HS GC-ECD	(Panyakapo et al., 2008)
Taiwan	Indoor	NR		56 (44-74)				PT GC-MS	(Hsu et al., 2009)
Korea	Indoor Indoor Indoor	Cl Cl/Oz EGMO		41 (0.2-102) 29 (0.2-65) 27 (6.8-56)	3 (nd-11) 2.4 (nd-5.7) 9.8 (1.6-27)	0.5 (nd-5.6) 0.2 (nd-3.4) 9.1 (nd-30)	nd nd 19 (nd-36)	PT GC-MS	(Lee et al., 2009)
USA	Indoor	Cl	88 (3.3-311)	73 (nd-298)	45 (nd-150)	6.7 (nd-55)	7.3 (nd-68)	MIMS	(Weaver et al., 2009)
USA	Indoor	Cl	63 (26-213)	62 (25-207)	2 (1-28)	2 (<1-4)	1 (nd-1)	LLE GC-ECD	(Kanan, 2010)
Italy	Indoor	CIC	41 (7-134)					SHS GC-ECD	(Fantuzzi et al., 2010)
Spain	Indoor	Cl	45 (35-75)	16 (8.5-21)	12 (9.3-23)	11 (6.5-23)	6.1 (3-16)	PT GC-MS	(Font-Ribera et al., 2010c)
Spain	Indoor	Cl	45					PT GC-MS	(Kogevinas, 2010)
Korea	Indoor Indoor Indoor	Cl Cl/Oz EGMO		21 (nd-46) 7.4 (nd-21) 15 (nd-40)	2.1 (nd-7) 1.1 (nd-2.5) 10 (nd-34)	nd nd 8.9 (nd-32)	nd nd 4.1 (nd-18)	PT GC-MS	(Lee et al., 2010)

Table A1-1 continued

Country	Pool(s) Type	Disinfection Method	Trihalomethane Concentration in Swimming Pool Water (µg L^{-1}); mean (min-max). Total THMs (TTHMs)	Trichloromethane (Chloroform)	Bromodichloromethane	Dibromochloromethane	Tribromomethane (Bromoform)	Analytical Method	Reference
Italy	Indoor	SDCIC		86 (36-127)	1.9 (1.6-2)	nd	nd	PT GC-MS	(Aprea et al., 2010)
	Indoor	Cl		12 (10-14)	18 (16-19)	18 (15-20)	4.6 (3.9-5.9)		
	Indoor	SDCIC		23 (11-41)	2.3 (nd-3.3)	1 (0.5-1.5)	0.1 (0.1-0.5)		
Spain	Indoor	NR	43					PT GC-MS	(Font-Ribera et al., 2010a)
	Outdoor	NR	151						
Spain	Indoor	Cl	92 (29-247)	77 (22-217)	11 (3.6-25)	2.4 (0.4-5.3)	1.5 (0.2-2.8)	PT GC-MS	(Font-Ribera et al., 2010b)
	Indoor	Br	110 (82-150)	1.1 (0.5-2.2)	1 (0.3-2.5)	2.6 (1.2-5.1)	105 (79-147)		
Spain	Indoor	Cl		15 (8.4-21)	14 (9.3-27)	13 (6.5-23)	7.2 (3-16.5)	PT GC-MS	(Richardson et al., 2010)
	Indoor	BCDMH		0.2 (0.1-0.3)	0.4 (0.2-0.7)	2.4 (2.1-2.7)	57 (52-64)		
USA	Indoor	Cl		(20-30)		(0.5-2.5)		MIMS	(Weng and Blatchley, 2011)
France	Indoor	Cl	26 (4.8-81)	22 (3.5-73)	2.6 (0.6-15)	0.8 (0.3-3.8)	0.4 (0.3-2.2)	LLE GC-MS	(Bessonneau et al., 2011)
Canada	Indoor	Cl		83 (30-160)	112 (39-187)	17 (4.6-38)	4.0 (nd-7.3)	LLE GC-ECD	(Wang, 2011)
	Outdoor	Cl		556 (170-882)	125 (34-315)	10 (6.3-13)	nd		
Taiwan	Indoor	NR		9.8 (8-12)				PT GC-MS	(Chen et al., 2011)
Germany	Indoor	Cl		(6-7.6)				HS GC-ECD	(Schmalz et al., 2011b)
Portugal	Indoor	Cl	(22-577)	(2-520)				HS-SPME GC-ECD	(Sa et al., 2011)
Italy	Indoor	Cl		15 (8.5-20)	14 (9.4-25)	13 (6.7-23)	7.2 (3.1-16)	PT GC-MS	(Lourencetti et al., 2012)
	Indoor	Br		0.2 (0.1-0.3)	0.4 (0.2-0.6)	2.4 (2.1-2.6)	60 (52-61)		
France	Indoor*	Cl	408 (233-996)	0.1 (0.01-0.2)	0.3 (0.05-1.1)	25 (14-64)	383 (220-931)	HS GC-MS	(Parinet et al., 2012)
	Indoor*	DCICA	(32-78)	(0.2-0.3)	(0.3-0.7)	(3-3.2)	(29-74)		
Canada	Indoor	Cl	26 (10-46)					HS-SPME GC-ITMS	(Catto et al., 2012)
Switzerland	Indoor	Various Cl	30 (15-110)					HS GC-MS	(Parrat et al., 2012)
Portugal	Indoor	Cl	61 (nd-155)	(6.3-123)	(1-22)	(1-9.8)	(1-5.9)	HS-SPME GC-ECD	(Silva et al., 2012)
Canada	Indoor	Cl	44 (18-114)					LLE GC-MS	(Simard et al., 2013)
	Outdoor	Cl	98 (12-311)						

Table A1-1 continued

Country	Pool(s) Type	Disinfection Method	Trihalomethane Concentration in Swimming Pool Water (μg L^{-1}); mean (min-max). Total THMs (TTHMs)	Trichloromethane (Chloroform)	Bromodichloromethane	Dibromochloromethane	Tribromomethane (Bromoform)	Analytical Method	Reference
USA	Indoor	Cl		81 (12-282)	2 (nd-10)		1.4 (nd-32)	MIMS	(Zare Afifi and Blatchley, 2015)
Australia	Outdoor	Cl		76 (65-84)	2.3 (2-2.6)	0.3 (0.3-0.4)	<0.1	LLE GC-ECD	(Yeh et al., 2014)
Portugal	Indoor	Cl		(17-407)				HS-SPME GC-ECD	(Maia et al., 2014)
Italy	Indoor	Various Cl	37 (6.8-134)	29 (2.5-122)	5.5 (1.4-18)	2.3 (0.2-12)	0.4 (<0.1-3.6)	HS GC-ECD	(Righi et al., 2014)
	Indoor	Cl	32 (6.8-98)						
	Indoor	DCICA	54 (14-134)						
	Indoor	TCICA	32 (12-53)						
China	Outdoor	Cl	55 (27-74)					LLE GC-ECD	(Zhang et al., 2015)
	Indoor	Cl/Oz	31 (7.6-57)						
Denmark	NR	Cl		30 (15-59)	4.4 (1.4-10)	0.8 (0.3-1.6)	0.04 (0.03-0.07)	PT GC-MS	(Spiliotopoulou et al., 2015)
Taiwan	Indoor	Cl	25 (13-47)					PT-HS GC-ECD	(Peng et al., 2015)
Australia	Indoor	Cl		47 (39-50)	3.1 (2.7-3.9)	0.5 (0.2-0.7)	0.3 (0.02-0.5)	HS-SPME GC-MS	(Carter et al., 2015)
	Spa	Cl		19	0.1	0.2	0.1		
Canada	Indoor	Cl	84 (29-140)	63 (22-100)	9.9 (1.2-38)	21 (nd-56)	13 (nd-25)	HS-SPME GC-ITMS	(Tardif et al., 2015)
Saudi Arabia	Indoor*	Cl	61 (29-96)	<5[a]	<5[a]	<5[a]	50 (43-58)	LLE GC-MS	(Chowdhury et al., 2016)
USA	Spa	Br/TCICA		nd	nd	14	253	MIMS	(Daiber et al., 2016)
	Indoor	BCDMH		nd	nd	2.5 (2.4-2.6)	152 (118-186)		
	Spa	Cl/Oz		(nd-31)	nd	nd	nd		
	Spa	BCDMH		1.6 (nd-2.0)	2.9 (nd-2.9)	4.7 (3.0-7.1)	182 (168-198)		
	Indoor	Cl		19 (13-25)	6.2 (1.3-11)	17 (nd-28)	21 (nd-22)		
Spain	Indoor	Cl	49 (30-75)	37 (24-62)	7.1 (3.8-13)	2.0 (0.9-4.7)	0.9 (0.2-1.9)	HS GC-MS	(Font-Ribera et al., 2016)

Table A1-1 continued

Country	Pool(s) Type	Disinfection Method	Trihalomethane Concentration in Swimming Pool Water (µg L^{-1}); mean (min-max). Total THMs (TTHMs)	Trichloromethane (Chloroform)	Bromodichloromethane	Dibromochloromethane	Tribromomethane (Bromoform)	Analytical Method	Reference
China	Indoor Indoor	Cl Cl/Oz		243 (46-467) 141 (96-213)	167 (9.9-318) 106 (85-141)	13 (nd-226) 2.0 (1.5-4.9)	44 (1.9-133) 47 (38-59)	LLE GC-MS	(Hang et al., 2016)
France	Outdoor Indoor*	Cl Cl	80 70 (50-92)	70 nd	7.9 nd	1.9 3.6 (1.6-5.2)	0.6 66 (49-87)	LLE GC-ECD	(Manasfi et al., 2016)
China	Outdoor	Cl	56					LLE GC-ECD	(Tang and Xie, 2016)
Canada	Indoor	Cl	65 (21-132)	38 (6.7-127)	9.7 (nd-30)	11 (nd-51)	6.6 (nd-46)	HS-SPME GC-ITMS	(Tardif et al., 2016a)
Canada	Indoor	Cl	35 (14-44)					HS-SPME GC-ITMS	(Tardif et al., 2016b)
China	Outdoor	Cl	90 (32-170)					HS GC-MS	(Yang et al., 2016)
Saudi Arabia	Indoor*	Cl	50 (31-77)	<5.0	<5.0	<5.0	40	LLE GC-MS	(Chowdhury, 2016)
France	Indoor*	Cl		0.1 (NR-0.9)	0.3 (NR-2.2)	19 (NR-81)	300 (NR-1029)	HS GC-MS	(Boudenne et al., 2017)
France	Indoor*	Cl	83 (52-106)	nd	nd	4.1 (2.1-5.5)	79 (50-101)	LLE GC-ECD	(Manasfi et al., 2017)
Iran	Indoor	Cl/Oz		138 (5.5-540)	55 (nd-171)	0.5 (nd-12)	0.9 (nd-16)	LLE GC-FID	(Abbasnia et al., 2018)
Poland	Outdoor	Cl	203 (15-551)	132 (15-372)	6.2 (<0.1-16)	1.0 (<0.1-2)	64 (<0.1-351)	LLE GC-ECD	(Klosok-Bazan et al., 2018)
Italy	Indoor	Various Cl	39 (23-62)	(19-55)	(2.8-7.9)	(0.3-3.7)	nd	SHS GC-ECD	(Fantuzzi et al., 2018)
China	Outdoor	TCICA	21 (3.2-61)	19 (2.6-59)	0.7 (nd-3.4)	0.3 (nd-2.0)	0.5 (nd-3.4)	LLE GC-ECD	(Zhang et al., 2018)

*Seawater filled. **a:** Specific values not reported. **nd**: Not detected. **NR**: Not Reported. **BCDMH**: Bromochlorodimethylhydantoin. **Br**: Bromine based (NaBr in combination with an oxidiser or Br_2). **CIC**: Chloroisocyanurate. **Cl**: Chlorine based (NaOCl, $Ca(OCl)_2$ or Cl_2). **DCICA**: Dichloroisocyanuric acid. **EGMO**: Electrochemically-generated mixed-oxidant. **Oz**: Ozone. **SDCIC**: Sodium dichloroisocyanurate. **TCICA**: Trichloroisocyanuric acid. **Various Cl**: Refers to any of the following individually or in combination: Cl Based, SDCIC, CIC, DCICA and/or TCICA. **DAI**: Direct aqueous injection. **ECD**: Electron capture detector. **FID**: Flame ionisation detector. **GC**: Gas chromatography. **HS**: Headspace. **ITMS**: Ion trap mass spectrometry. **LLE**: Liquid-liquid extraction. **MIMS**: Membrane-inlet mass spectrometry. **MS**: Mass spectrometry. **PT**: Purge and trap. **SHS**: Static headspace. **SPME**: Solid-phase microextraction. **TL**: Thin layer.

Table A1-2: Reported occurrence of haloacetic acids in swimming pool and spa waters.

Country	Pool(s) Type	Disinfection Method	Haloacetic Acid Concentration in Swimming Pool Water (µg L^{-1}); mean (min-max). THAA	TCAA	DCAA	CAA	BAA	DBAA	TBAA	BDCAA	DBCAA	BCAA	Analytical Method	Reference
Spain	NR	Cl		42	69	25	7.1	15					SPE CZE	(Martínez et al., 1999)
Switzerland	NR	Cl		45 (17-95)	76 (0.9-240)	47 (11-117)							LLE GC-MS	(Berg et al., 2000)
Spain	NR	Cl	330	155	45	4.2	nd	2.8	19	61	33	11	HS–SPME GC–ITMS	(Sarrión et al., 2000)
Portugal	NR	NR	2333 (1300-3200)	1400 (1000-1700)		378 (15-1000)			15	533 (208-912)	62		SPE–LC ESI–MS	(Loos and Barceló, 2001)
Italy	Indoor	Cl	(109-387)[a]										IC-MS	(Aggazzotti et al., 2007)
USA	Indoor	Cl	960 (172-9005)	241 (76-1925)	504 (52-6787)	nd	2 (<1-5)	4.5 (<1-25)	nd	22 (8-110)	3 (<1-32)	5 (1-176)	LLE GC-ECD	(Kanan, 2010)
Korea	Indoor	Cl		156 (20-636)	68 (14-246)								LLE GC-ECD	(Lee et al., 2010)
	Indoor	Cl/Oz		17 (1-86)	12 (nd-32)									
	Indoor	EGMO		97 (1-413)	34 (1.5-96)									
Spain	NR	NR	(201-363)	(55-195)	(94-130)	(34-42)		(1.4-1.6)		(<1-5)			SBME GC-MS	(Cardador and Gallego, 2010)
Canada	Indoor	Cl	427 (201-700)	116 (17-234)	173 (49-384)	24 (9.8-46)	12 (3.8-27)	28 (5.4-88)	5.6 (0.2-10)	18 (0.4-33)	9.1 (0.3-21)	46 (7.1-106)	LLE GC-ECD	(Wang, 2011)
	Outdoor	Cl	1039 (144-2777)	382 (43-961)	540 (71-1517)	110 (18-300)	nd	nd	nd	2.7 (2.3-3.3)	nd	8.3 (2-12)		
Spain	Indoor	Cl		110 (85-166)	77 (60-109)	23 (8.5-36)							HS GC-MS	(Cardador and Gallego, 2011)
	Outdoor	Cl		120 (99-146)	151 (130-170)	26 (19-34)								
France	Indoor*	Cl	757 (323-2233)	27 (2.9-87)	4.8 (2.2-8.7)	120 (99-146)	55 (8.2-155)	307 (132-1089)	164 (49-428)	13 (5-20)	103 (36-243)	65 (27-216)	LLE GC-ECD	(Parinet et al., 2012)
	Indoor*	DCICA	(84-123)	(4.6-15)	(1.4-2.1)	(1.3-1.5)	(4.3-6.5)	(11-17)	(4-5)	(1.2-2.3)	(50-67)	(5.1-5.3)		

Table A1-2 continued

Country	Pool(s) Type	Disinfection Method	Haloacetic Acid Concentration in Swimming Pool Water (µg L^{-1}); mean (min-max). THAA	TCAA	DCAA	CAA	BAA	DBAA	TBAA	BDCAA	DBCAA	BCAA	Analytical Method	Reference
Canada	Indoor	Cl	238 (111-391)	118 (54-201)	103 (48-192)					15 (nd-24)		1.8 (0.4-3)	LLE GC-ECD	(Catto et al., 2012)
Portugal	Indoor	Cl	(10-183)	(0.5-73)	(0.4-54)	(0.6-13)		(0.1-12)[b]	(0.4-0.9)	(0.1-12)[b]	(0.2-0.9)	(0.4-25)	HS-SPME GC-ECD	(Sa et al., 2012)
Portugal	NR	NR	106 (76-154)[c]	54 (29-76)	51 (29-84)	2.4 (nd-2.7)	nd	0.6 (0.3-0.7)					HPLC ESI-tq-MS	(Prieto-Blanco et al., 2012)
Canada	Indoor	Cl	364 (104-1195)										PT GC-ECD	(Simard et al., 2013)
	Outdoor	Cl	808 (155-2224)											
Saudi Arabia	NR	NR		(nd-13)	(11-35)	(46-49)	(8.6-25)	(16-17)				(6.8-7.1)	SPME UPLC–UV	(Nsubuga and Basheer, 2013)
China	Indoor	NR	95 (13-133)[c]	53 (6-90)	34 (5-60)								LLE GC-ECD	(Wang et al., 2014)
	Outdoor	NR	156 (89-332)[c]	53 (33-98)	81 (44-195)									
USA	Indoor	NR	1613 (70-3980)[c]	890 (20-2970)	727 (50-750)								LLE GC-ECD	(Wang et al., 2014)
	Outdoor	NR	1442 (800-2430)[c]	680 (370-1140)	700 (310-1330)									
	Spa	NR	1067 (690-1360)[c]	330 (40-530)	450 (50-750)									
Australia	Indoor	Various Cl		893 (110-1700)	983 (230-2100)	43 (32-64)	<0.5	<0.5		17 (<0.5-22)	<0.5	<0.5	LLE GC-ECD	(Yeh et al., 2014)
	Outdoor	Various Cl		978 (650-1300)	856 (480-1400)	73 (<0.5-120)	<0.5	<0.5		8.8 (7-12)	<0.5	<0.5		
	Baby	Various Cl		(1600-2600)	(400-2400)	(<0.5-110)	<0.5	<0.5		(14-16)	<0.5	<0.5		

Table A1-2 continued

Country	Pool(s) Type	Disinfection Method	Haloacetic Acid Concentration in Swimming Pool Water (µg L^{-1}); mean (min-max). THAA	TCAA	DCAA	CAA	BAA	DBAA	TBAA	BDCAA	DBCAA	BCAA	Analytical Method	Reference
Italy	Indoor	Various Cl	164 (11-403)	53 (<1-291)	11 (<1-65)								IC-MS	(Righi et al., 2014)
Australia	Indoor	Cl		NQ	409 (113-656)	80	nd	3.0	nd	NQ	NQ	32	LLE GC-MS	(Carter et al., 2015)
	Spa	Cl		NQ	668	NQ	nd	nd	nd	NQ	NQ	nd		
Canada	Indoor	Cl	303 (123-606)[d]	139 (23-289)	127 (32-281)	15 (4.4-33)	4 (nd-4.7)	11 (nd-25)				16 (1.8-35)	LLE GC-ECD	(Tardif et al., 2015)
China	Outdoor	Cl	168 (95-351)										LLE GC-ECD	(Zhang et al., 2015)
	Indoor	Cl/Oz	102 (14-161)											
Saudi Arabia	Indoor*	Cl		2.6 (0.5-4.7)	1.7 (0.8-2.6)	1.25 (0.04-2.2)	3.8 (1.4-6.9)	16 (5.9-28)	186 (5.8-73)	2.1 (0.3-4.4)	25 (7.3-48)	5.2 (1.5-8.7)	LLE GC-MS	(Chowdhury et al., 2016)
USA	Indoor	BCDMH		64 (52-77)	2.2 (1.1-3.3)	nd	4.7 (4.2-5.2)	123 (115-131)	72 (50-93)	8.9 (6.3-12)	4.1 (2.5-5.6)	2.2 (nd-2.2)	LLE GC-MS	(Daiber et al., 2016)
	Indoor	Cl		158 (65-249)	163 (89-201)	13 (8.3-19)	6.6 (1.2-12)	14 (nd-19)	nd	15 (8.6-21)	5.0 (nd-6.1)	25 (11-39)		
	Spa	BCDMH		37 (28-49)	27 (23-32)	3.9 (3.8-4.0)	62 (26-90)	337 (91-506)	97 (26-175)	10 (6.7-14)	4.4 (2.9-6.3)	13 (7.7-17)		
	Spa	Br/TCICA		13	89	nd	47	1795	74	117	14	294		
	Spa	Cl/Oz		(nd-1865)	(nd-343)	(nd-31)	nd	nd	nd	(nd-12)	nd	(nd-2.6)		
Spain	Indoor	Cl	111 (73-144)	63 (39-84)	30 (15-52)			1.0 (0.5-3.1)		12 (4.8-24)		4.9 (2.4-8.8)	LLE GC-MS	(Font-Ribera et al., 2016)
China	Indoor	Cl		19 (nd-43)	365 (nd-2435)	10 (nd-94)	2.1 (nd-27)	nd	nd	nd	nd	510 (nd-1353)	LLE GC-MS	(Hang et al., 2016)
	Indoor	Cl/Oz		21 (nd-49)	200 (36-536)	41 (nd-475)	16 (nd-103)	nd	8.1 (nd-122)	nd	1.2 (nd-18)	425 (190-657)		
France	Indoor	Cl	116 (107-132)	nd	nd	nd	nd	66 (63-72)	43 (36-53)	nd	3.1 (2.7-3.5)	4.3 (3.5-4.8)	LLE GC-ECD	(Manasfi et al., 2016)
	Outdoor	Cl	498	461	23	nd	nd	1.7	nd	7.3	2.7	2.4		
China	Outdoor	Cl	1364										LLE GC-ECD	(Tang and Xie, 2016)

Table A1-2 continued

Country	Pool(s) Type	Disinfection Method	Haloacetic Acid Concentration in Swimming Pool Water (µg L^{-1}); mean (min-max). THAA	TCAA	DCAA	CAA	BAA	DBAA	TBAA	BDCAA	DBCAA	BCAA	Analytical Method	Reference
Canada	Indoor	Cl	295 (109-886)[c]	107 (24-250)	134 (27-500)	17 (2.1-78)	3.8 (nd-15)	17 (nd-70)				31 (1.2-118)	LLE GC-ECD	(Tardif et al., 2016a)
Canada	Indoor	Cl	228 (160-290)[c]										LLE GC-ECD	(Tardif et al., 2016b)
Saudi Arabia	Indoor*	Cl	87 (58-129)					15	34		22		LLE GC-MS	(Chowdhury, 2016)
China	Outdoor	Cl	798 (191-1906)	492	462								LLE GC-MS	(Yang et al., 2016)
France	Indoor*	Cl	151 (68-216)	nd	nd	nd	nd	73 (30-104)	60 (28-83)	2.0 (1.1-3.3)	10 (4.5-16)	6.1 (3.4-8.7)	LLE GC-ECD	(Manasfi et al., 2017)
Iran	Indoor	Cl	1045 (148-3488)[d]	501 (72-1709)	368 (1.6-1256)	113 (16-377)	37 (5.9-114)	18 (3.4-56)					LLE GC-MS	(Dehghani et al., 2018)

*Seawater filled. **a**: HAA3; Sum of CAA, DCAA and TCAA. **b**: The range values refer to the sum (DBAA+BDCAA). **c**: HAA6; Sum of CAA, DCAA, TCAA, BAA, DBAA and BCAA. **d**: HAA5; Sum of CAA, DCAA, TCAA, BAA and DBAA. **nd**: Not detected. **NR**: Not reported. **NQ**: Detected but not quantifiable. **BAA**: Bromoacetic acid. **BCAA**: Bromochloroacetic acid. **BDCAA**: Bromodichloroacetic acid. **CAA**: Chloroacetic acid. **DBAA**: Dibromoacetic acid. **DBCAA**: Dibromochloroacetic acid. **DCAA**: Dichloroacetic acid. **TBAA**: Tribromoacetic acid. **TCAA**: Trichloroacetic acid. **THAA**: Total haloacetic acids: Sum of CAA, DCAA, TCAA, BAA, DBAA, TBAA, BCAA, CDBAA and BDCAA. **BCDMH**: Bromochlorodimethylhydantoin. **Br**: Bromine based (NaBr in combination with an oxidiser or Br_2). **Cl**: Chlorine based (NaOCl, $Ca(OCl)_2$ or Cl_2). **Oz**: Ozone. **TCICA**: Trichloroisocyanuric acid. **Various Cl**: Refers to any of the following individually or in combination: Cl Based, sodium dichloroisocyanurate (SDCIC), chloroisocyanurate (CIC), dichloroisocyanuric acid (DCICA) and/or TCICA. **CZE**: Capillary zone electrophoresis. **ECD**: Electron capture detector. **ESI**: Electrospray ionisation. **GC**: Gas chromatography. **HPLC**: High performance liquid chromatography. **HS**: Headspace. **IC**: Ion chromatography. **ITMS**: Ion trap mass spectrometry. **LLE**: Liquid-liquid extraction. **MS**: Mass spectrometry. **SBME**: Solvent bar microextraction. **SPE**: Solid-phase extraction. **SPME**: Solid-phase microextraction. **tq**: Triple quadrupole. **UPLC:** Ultra performance liquid chromatography. **UVD**: Ultraviolet detection.

Table A1-3: Reported occurrence of inorganic chloramines in swimming pool and spa waters.

Country	Pool(s) Type	Disinfection Method	Chloramine Concentration in Swimming Pool Water (µg L^{-1}); mean (min-max). Total Inorganic Chloramines	Trichloramine (NCl_3)	Dichloramine ($NHCl_2$)	Monochloramine (NH_2Cl)	Analytical Method	Reference
Japan	Outdoor	NR			(40-120)	(110-190)	DPD/KI Colorimetric	(Tachikawa et al., 2005)
USA	Indoor	NR		90 (70-100)			MIMS	(Li and Blatchley, 2007)
	Outdoor	NR		(70-160)				
USA	Indoor	Cl	513 (nd-2070)	94 (nd-3412)	121 (nd-417)	311 (nd-1880)	MIMS	(Weaver et al., 2009)
Spain	Indoor	Cl			430 (160-650)		DPD/KI Colorimetric	(Font-Ribera et al., 2010c)
Spain	Indoor	Cl		<100	380 (<10-650)	290 (100-640)	DPD/KI Colorimetric	(Richardson et al., 2010)
	Indoor	BCDMH		<100	<10	270 (240-300)		
USA	Indoor	Cl		(100-780)	(180-750)	(180-300)	MIMS	(Weng and Blatchley, 2011)
Canada	Indoor	Cl				40 (10-60)	DPD/KI Colorimetric	(Wang, 2011)
	Outdoor	Cl				10 (9-11)		
Canada	Indoor	Cl	689 (376-981)	341 (nd-650)	25 (nd-593)	323 (188-434)	DPD/KI Colorimetric	(Catto et al., 2012)
	Indoor	Cl	527 (268-802)	232 (nd-557)	11 (nd-70)	284 (nd-450)		
Canada	Indoor	Cl	736 (311-1723)				DPD/KI Colorimetric	(Simard et al., 2013)
	Outdoor	Cl	142 (8-845)					
USA	Indoor	Cl		420 (nd-2190)	65 (nd-250)	89 (nd-620)	MIMS	(Zare Afifi and Blatchley, 2015)
Switzerland	Indoor/Outdoor	Various[a]		29 (2.4-58)			MIMS	(Soltermann et al., 2014)
China	Indoor	Cl		7 (5-11)			MIMS	(Lian et al., 2014)
Canada	Indoor	Cl		600 (400-800)			DPD/KI Colorimetric	(Lévesque et al., 2015)
Saudi Arabia	Indoor*	Cl		70 (nd-110)	220 (10-490)	220 (70-450)	DPD/KI Colorimetric	(Chowdhury et al., 2016)
USA	Indoor	Cl		319 (66-527)	41 (nd-55)	58 (43-71)	MIMS	(Daiber et al., 2016)
	Indoor	BCDMH		12 (5.6-18)	51 (45-56)	67 (nd-67)		
	Spa	BCDMH		183 (3.7-363)	40 (39-41)	48 (43-52)		
	Spa	Br/TCICA		91	142	205		
Spain	Indoor	Cl		1500 (nd-1600)	300 (nd-700)	200 (nd-700)	DPD/KI Colorimetric	(Font-Ribera et al., 2016)

Table A1-3 continued

Country	Pool(s) Type	Disinfection Method	Chloramine Concentration in Swimming Pool Water (µg L^{-1}); mean (min-max).				Analytical Method	Reference
			Total Inorganic Chloramines	Trichloramine (NCl_3)	Dichloramine ($NHCl_2$)	Monochloramine (NH_2Cl)		
Spain	Indoor	Cl		50 (nd-300)	300 (180-400)	180 (100-200)	DPD/KI Colorimetric	(Llana-Belloch et al., 2016)
	Indoor	Cl		50 (nd-300)	350 (300-400)	320 (300-400)		
	Indoor	UV only		nd	nd	nd		

*Seawater filled. **nd**: Not detected. **NR**: Not reported. **a**: Cl in conjunction with UV or Oz. **BCDMH**: Bromochlorodimethylhydantoin. **Cl**: Chlorine based (NaOCl, $Ca(OCl)_2$ or Cl_2). **Oz**: Ozone. **UV:** Ultraviolet irradiation. **DPD**: Diethyl-*p*-phenylenediamine. **KI**: Potassium iodide. **MIMS**: Membrane-inlet mass spectrometry.

Table A1-4: Reported occurrence of haloacetonitriles in swimming pool and spa waters.

Country	Pool(s) Type	Disinfection Method	Haloacetonitrile Concentration in Swimming Pool Water (µg L^{-1}); mean (min-max). BAN	CAN	BCAN	DBAN	DCAN	TCAN	THAN	Analytical Method	Reference
USA	Indoor Outdoor	NR NR					100 (100-100) (20-30)			MIMS	(Li and Blatchley, 2007)
USA	Indoor	Cl					15 (0.6-87)			MIMS	(Weaver et al., 2009)
USA	Indoor	Cl	1.0 (nd-1.0)	1.2 (nd-3.0)	2.7 (nd-13)	1.8 (nd-5.0)	15 (4-47)	(nd-1.0)	16 (5.0-53)	LLE GC-ECD	(Kanan, 2010)
Korea	Indoor Indoor Indoor	Cl/Oz EGMO Cl			0.4 (nd-0.6) 3.5 (nd-8.0) 0.8 (nd-1.9)	0.4 (nd-0.8) 2.6 (nd-6.8) 0.5 (nd-0.9)	1.3 (0.2-3.2) 3.8 (nd-9.0) 3.9 (0.5-12)	nd nd nd		LLE GC-ECD	(Lee et al., 2010)
USA	Indoor	Cl					(100-200)			MIMS	(Weng and Blatchley, 2011)
USA	Indoor	Cl					8.6 (0.7-31)			MIMS	(Zare Afifi and Blatchley, 2015)
Australia	Outdoor	Cl			0.6 (0.5-0.8)	0.3 (nd-0.3)	7.1 (4.9-8.9)	0.3 (nd-0.3)		LLE GC-ECD	(Yeh et al., 2014)
Australia	Indoor Spa	Cl Cl	nd	2.4	nd	0.2	8.9 (3.9-12) 9.0	0.4		LLE GC-MS	(Carter et al., 2015)
Denmark	NR	Cl					4.2 (1.9-7.2)			PT GC-MS	(Spiliotopoulou et al., 2015)
Canada	Indoor	Cl			3.2 (0.5-11)	2.9 (nd-15)	12 (4.-24)	0.2 (nd-1.1)		LLE GC-ECD	(Tardif et al., 2015)
China	Indoor Outdoor	TCICA Cl							5.0 (1.3-13)[a] 3.6 (0.8-8.3)[a]	LLE GC-ECD	(Zhang et al., 2015)
USA	Spa Indoor Spa Spa Indoor	Br/TCICA BCDMH Cl/Oz BCDMH Cl			5.6 1.8 (nd-1.8) nd 1.8 (nd-1.8) 5.6 (nd-7.4)	219 37 (35-39) nd 80 (47-98) nd	nd nd (nd-14) nd 4.9 (1.8-9.4)	nd nd nd nd nd		MIMS	(Daiber et al., 2016)
Spain	Indoor	Cl			3.0 (1.8-4.7)	1.3 (1.1-3.6)	7.3 (3.8-12)			HS GC-MS	(Font-Ribera et al., 2016)
China	Indoor Indoor	Cl/Oz Cl			nd 5.6 (nd-89)	nd 3.1 (nd-34)	5.3 (4.2-8.5) 17 (nd-206)	nd 0.1 (nd-0.5)		LLE GC-MS	(Hang et al., 2016)
France	Indoor* Outdoor	Cl Cl			0.9 (0.9-1.0) 9.2	19 (13-28) 2.5	nd 75	nd 1.2		LLE GC-ECD	(Manasfi et al., 2016)
Canada	Indoor	Cl			5.8 (0.3-30)	5.8 (nd-31)	9.8 (2.3-23)	0.03 (nd-0.1)		LLE GC-ECD	(Tardif et al., 2016a)
Canada	Indoor	Cl							26 (22-34)[a]	LLE GC-ECD	(Tardif et al., 2016b)
France	Indoor*	Cl			nd	27 (18-35)	nd			LLE GC-ECD	(Manasfi et al., 2017)
China	Outdoor	TCICA			0.6 (nd-1.0)	0.1 (nd-0.8)	1.3 (0.3-6.3)	0.2 (nd-0.3)	2.1 (0.7-7.2)[a]	LLE GC-ECD	(Yang et al., 2018)

*Sea water filled. **a**: Refers to HAN-4; Sum of TCAN, BCAN, DBAN and DCAN. **BAN**: Bromoacetonitrile. **CAN**: Chloroacetonitrile. **BCAN**: Bromochloroacetonitrile. **DBAN**: Dibromoacetonitrile. **DCAN**: Dichloroacetonitrile. **TCAN**: Trichlorocetonitrile. **THAN**: Total haloacetonitrile: Sum of BAN, CAN, BCAN, DBAN, DCAN and TCAN. **BCDMH**: Bromochlorodimethylhydantoin. **Br**: Bromine Based (NaBr in combination with an oxidiser or Br_2). **Cl**: Chlorine based (NaOCl, $Ca(OCl)_2$ or Cl_2). **EGMO**: Electrochemically-generated mixed-oxidant. **Oz**: Ozone. **TCICA**: Trichloroisocyanuric acid. **ECD**: Electron capture detector. **GC**: Gas chromatography. **HS**: Headspace. **LLE**: Liquid-liquid extraction. **MIMS**: Membrane-inlet mass spectrometry. **MS**: Mass spectrometry. **PT**: Purge and trap.

Table A1-5: Reported occurrence of *N*-nitrosamines in swimming pool and spa waters.

Country	Pool(s) Type	Disinfection Method	*N*-Nitrosamine Concentration in Swimming Pool Water (ng L^{-1}); mean (min-max). NDMA	NDEA	NEMA	NDBA	NMOR	NPIP	NDPA	NPYR	Analytical Method	Reference
USA	Indoor	Cl	32 (NR-42)			NQ		NQ			SPE GC-MS	(Walse and Mitch, 2008)
	Outdoor	Cl	5.3 (NR-6.9)			NQ		NQ				
	Spa	Cl	313 (NR-429)			NQ		NQ				
Spain	NR	NR	5.5 (5.0-5.9)	1.2 (1.1-1.4)						4.5	SPE GC-MS	(Jurado-Sánchez et al., 2010)
USA	Indoor	Cl	17 (2-83)								SPE GC-MS	(Kanan, 2010)
Korea	Indoor	Cl	52 (0.7-208)	31 (1.4-53)			3.1 (0.3-34)				SPE HPLC-FLD	(Kim and Han, 2011)
Italy	NR	NR								77 (53-127)	SPE GC-MS	(Pozzi et al., 2011)
Canada	Indoor	Cl	5.2 (1.0-9.8)	14.8 (5.9-53)	7.1 (2.6-26)	15 (6.8-22)	15 (12-18)	4.4 (3.2-5.5)	nd	nd	SPE GC-MS	(Wang, 2011)
	Outdoor	Cl	6.6 (3.1-15)	35 (3.5-72)	16 (15-17)	141 (1.6-403)	5.9 (5.8-6.0)	nd	nd	nd		
Taiwan	Indoor	NR	(7.2-100)	(1.4-3.7)	(nd-1.7)						SPE GC-MS	(Fu et al., 2012)
	Outdoor	NR	(nd-4.7)	(nd-9.0)	nd							
Australia	Indoor	Cl	34 (31-38)	3.3 (3.2-3.4)	nd	24 (15-33)	26 (26-27)	nd	nd	nd	SPE GC-MS	(Carter et al., 2015)
	Spa	Cl	5.5	nd	nd	nd	nd	nd	nd	nd		
Canada	Indoor	Cl	43 (2.4-105)								LLE GC-MS-MS	(Tardif et al., 2015)
Spain	Indoor	Cl	11 (8.0-14)								SPE GC-MS-MS	(Font-Ribera et al., 2016)
Canada	Indoor	Cl	43 (2.8-105)								LLE GC-MS	(Tardif et al., 2016a)
Canada	Indoor	Cl	10 (nd-13)								LLE GC-MS	(Tardif et al., 2016b)

nd: Not detected. **NQ**: Detected but not quantifiable. **NR**: Not reported. **NDBA**: *N*-Nitrosodi-*n*-butylamine. **NDEA**: *N*-Nitrosodiethylamine. **NDMA**: *N*-Nitrosodimethylamine. **NDPA**: *N*-Nitrosodipropylamine. **NEMA**: *N*-Nitrosoethylmethylamine. **NMOR**: *N*-Nitrosomorpholine. **NPIP**: *N*-Nitrosopiperidine. **NPYR**: *N*-Nitrosopyrrolidine. **Cl**: Chlorine based (NaOCl, $Ca(OCl)_2$ or Cl_2). **FLD**: Fluorescence detection. **GC**: Gas chromatography. **HPLC**: High performance liquid chromatography. **LLE**: Liquid-liquid extraction. **MS**: Mass spectrometry. **SPE**: Solid-phase extraction.

Table A1-6: Reported occurrence of haloacetaldehydes in swimming pool and spa waters.

Country	Pool(s) Type	Disinfection Method	Haloacetaldehyde Concentration in Swimming Pool Water (µg L^{-1}); mean (min-max).					Analytical Method	Reference
			CH	DCAL	DBAL	DBCAL	TBAL		
Korea	Indoor	EGMO	10 (nd-23)					LLE GC-ECD	(Lee et al., 2010)
	Indoor	Cl/Oz	3.6 (nd-10)						
	Indoor	Cl	17 (5.1-35)						
Spain	NR	NR	(53-340)	(1.8-23)				MLLE LVI-PTV-GC-MS	(Serrano et al., 2011)
Australia	Outdoor	Cl	21 (19-24)					LLE GC-ECD	(Yeh et al., 2014)
China	Outdoor	Cl	58 (16-156)					LLE GC-ECD	(Zhang et al., 2015)
	Indoor	Cl	47 (6.0-132)						
Australia	Indoor	Cl	301 (177-400)		2.4	0.3		LLE GC-MS	(Carter et al., 2015)
	Spa	Cl	405						
France	Outdoor*	Cl	190					LLE GC-ECD	(Manasfi et al., 2016)
	Indoor*	Cl	nd				(0.4-2.2)		
USA	Indoor	Cl	120 (68-165)					MIMS	(Daiber et al., 2016)
	Indoor	BCDMH	nd						
	Spa	Cl/Oz	(nd-101)						
	Spa	Br	2.9 (1.3-3.9)						
France	Indoor*	Cl	nd				8.4 (10-13)	LLE GC-ECD	(Manasfi et al., 2017)

*Seawater filled. **nd**: Not detected. **NR**: Not reported. **CH**: Chloral hydrate (trichloroacetaldehyde). **DBAL**: Dibromoacetaldehyde. **DBCAL**: Dibromochloroacetaldehyde. **DCAL**: Dichloroacetaldehyde. **TBAL**: Tribromoacetaldehyde. **BCDMH**: Bromochlorodimethylhydantoin. **Br**: Bromine based (NaBr in combination with an oxidiser or Br_2). **Cl**: Chlorine based (NaOCl, $Ca(OCl)_2$ or Cl_2). **Oz**: Ozone. **ECD**: Electron capture detector. **GC**: Gas chromatography. **LLE**: Liquid-liquid extraction. **LVI**: Large volume injection. **MIMS**: Membrane-inlet mass spectrometry. **MLLE**: Micro-liquid-liquid extraction. **MS**: Mass spectrometry. **PTV**: Program temperature vaporiser.

Table A1-7: Reported occurrence of haloketones in swimming pool and spa waters.

Country	Pool(s) Type	Disinfection Method	Haloketone Concentration in Swimming Pool Water (µg L^{-1}); mean (min-max). 1,1-Dichloropropanone (1,1-DCP)	1,1,1-Trichloropropanone (1,1,1-TCP)	1, 2-Dichloropropanone (1,2-DCA)	Chloropropanone	Analytical Method	Reference
Australia	Outdoor	Cl	0.4 (0.3-0.5)	6.9 (3.6-9.6)			LLE GC-ECD	(Yeh et al., 2014)
Australia	Indoor	Cl	0.7	5.8	0.8	1.9	LLE GC-MS	(Carter et al., 2015)
Denmark	NR	Cl		1.3 (0.4-3.1)			PT GC-MS	(Spiliotopoulou et al., 2015)
Canada	Indoor	Cl		3.1 (0.4-11)			LLE GC-ECD	(Tardif et al., 2015)
China	Indoor	Cl		1.8 (0.2-7.4)[a]			LLE GC-ECD	(Zhang et al., 2015)
	Outdoor	Cl		2.4 (0.9-6.3)[a]				
USA	Spa	Br/TCICA	nd	nd			MIMS	(Daiber et al., 2016)
	Indoor	BCDMH	nd	nd				
	Spa	Cl/Oz	nd	(nd-9.7)				
	Spa	BCDMH	nd	nd				
	Indoor	Cl	1.4 (nd-1.4)	1.8 (nd-2.4)				
Spain	Indoor	Cl	2.1 (1.4-5.8)				LLE GC-MS	(Font-Ribera et al., 2016)
China	Indoor	Cl/Oz	nd	11 (7.1-15)			LLE GC-MS	(Hang et al., 2016)
	Indoor	Cl	0.7 (nd-7.7)	46 (1.9-180)				
France	Indoor*	Cl	nd	nd			LLE GC-ECD	(Manasfi et al., 2016)
	Outdoor	Cl	21	72				
Canada	Indoor	Cl		1.9 (0.3-7.3)			LLE GC-MS	(Tardif et al., 2016a)
Canada	Indoor	Cl	nd	2.5 (1.8-3.4)			LLE GC-MS	(Tardif et al., 2016b)

*Seawater filled. **a**: Refers to the sum of 1,1-di- and 1,1,1-tri-chloropropanone. **nd**: Not detected. **NR**: Not reported. **BCDMH**: Bromochlorodimethylhydantoin. **Br**: Bromine based (NaBr in combination with an oxidiser or Br_2). **Cl**: Chlorine based (NaOCl, $Ca(OCl)_2$ or Cl_2). **Oz**: Ozone. **TCICA**: Trichloroisocyanuric acid. **ECD**: Electron capture detector. **GC**: Gas chromatography. **LLE**: Liquid-liquid extraction. **MIMS**: Membrane-inlet mass spectrometry. **MS**: Mass spectrometry. **PT**: Purge and trap.

Table A1-8: Reported occurrence of halonitromethanes in swimming pool and spa waters.

Country	Pool(s) Type	Disinfection Method	Halonitromethane Concentration in Swimming Pool Water (µg L^{-1}); mean (min-max).					Analytical Method	Reference
			THNMs	TCNM	TBNM	BCNM	BNM		
Germany	NR	NR		0.2 (nd-0.8)				NR	(Puchert et al., 1989)
USA	Indoor	Cl	4.8 (1.4-13)	1.1 (nd-2.3)		4.0 (0.8-11)	1.5 (nd-2.2)	LLE GC-ECD	(Kanan, 2010)
Spain	NR	NR		1.2 (0.4-1.9)				HS SPME GC-MS	(Montesinos and Gallego, 2012)
Australia	Outdoor	Cl		1.2 (1.2-1.3)	1.2 (nd-1.2)			LLE GC-ECD	(Yeh et al., 2014)
Canada	Indoor	Cl		0.9 (nd-5.0)				LLE GC-ECD	(Tardif et al., 2015)
China	Indoor Outdoor	Cl Cl		0.1 (nd-0.1) nd				LLE GC-ECD	(Zhang et al., 2015)
USA	Indoor Indoor Spa Spa Spa	BCDMH Cl NR BCDMH Cl/Oz		nd nd nd nd nd				MIMS	(Daiber et al., 2016)
China	Indoor Indoor	Cl Cl/Oz		1.0 (nd-4.5) 0.4 (nd-2.1)				LLE GC-ECD	(Hang et al., 2016)
France	Indoor Outdoor	Cl Cl		nd 4.5	nd			LLE GC-ECD	(Manasfi et al., 2016)
Canada	Indoor	Cl		0.3 (0.02-3.7)				LLE GC-ECD	(Tardif et al., 2016a)
Canada	Indoor	Cl		0.3 (0.2-0.9)				LLE GC-MS	(Tardif et al., 2016b)
China	Outdoor	TCICA		0.9 (nd-2.7)				LLE GC-ECD	(Yang et al., 2018)

*Seawater filled. **nd**: Not detected. **NR**: Not reported. **BNM**: Bromonitromethane. **BCNM**: Bromochloronitromethane. **TBNM**: Tribromonitromethane. **TCNM**: Trichloronitromethane. **THNMs**: Total halonitromethanes. **BCDMH**: Bromochlorodimethylhydantoin. **Cl**: Chlorine based (NaOCl, $Ca(OCl)_2$ or Cl_2). **Oz**: Ozone. **ECD**: Electron capture detector. **GC**: Gas chromatography. **HS**: Headspace. **LLE**: Liquid-Liquid Extraction. **MIMS**: Membrane-inlet mass spectrometry. **MS**: Mass spectrometry. **SPME**: Solid-phase microextraction. **TCICA**: Trichloroisocyanuric acid.

Table A1-9: Reported occurrence of haloacetamides in swimming pool and spa waters.

Country	Pool(s) Type	Disinfection Method	Haloacetamide Concentration in Swimming Pool Water (µg L^{-1}); mean (min-max).										Analytical Method	Reference
			TCAAm	DCAAm	TBAAm	DBAAm	BCAAm	BDCAAm	DBCAAm	BIAAm	CIAAm	DIAAm		
Australia	Outdoor	Cl	2.7 (2.4-3.1)	nd	nd	1.9 (nd-2.0)	nd	nd	nd	nd	nd	nd	LLE GC-ECD	(Yeh et al., 2014)
Australia	Indoor	Cl	2.0	1.5		0.6							LLE GC-MS	(Carter et al., 2015)

nd: Not detected. **TCAAm**: Trichloroacetamide. **DCAAm**: Dichloroacetamide. **TBAAm**: Tribromoacetamide. **DBAAm**: Dibromoacetamide. **BCAAm**: Bromochloroacetamide. **BDCAAm**: Bromodichloroacetamide. **DBCAAm**: Dibromochloroacetamide. **BIAAm**: Bromoiodoacetamide. **CIAAm**: Chloroiodoacetamide. **DIAAm**: Diiodoacetamide. **Cl**: Chlorine based (NaOCl, $Ca(OCl)_2$ or Cl_2). **ECD**: Electron capture detector. **GC**: Gas chromatography. **LLE**: Liquid-liquid extraction. **MS**: Mass spectrometry.

Table A1-10: Reported occurrence of inorganic anions in swimming pool and spa waters.

Country	Pool(s) Type	Disinfection Method	Inorganic Anions in Swimming Pool Water (µg L^{-1}); mean (min-max).					Reference
			Bromide (Br^-)	Bromate (BrO_3^-)	Chlorite (ClO_2^-)	Chlorate (ClO_3^-)	Nitrate (NO_3^-)	
USA	Outdoor	NR				16000 (NR-124000)	8600 (NR-54000)	(Beech et al., 1980)
Australia	Indoor	Cl		<10		(<20-15000)		(Kelsall and Sim, 2001)
	Indoor	Cl/Oz		(<10 -80)		(<20-110000)		
	Indoor	Br/Oz		(<10-900)		<20		
Italy	Indoor	Cl				(190-12500)		(Aggazzotti et al., 2007)
Thailand	NR	NR	2200 (nd-3900)					(Panyakapo et al., 2008)
Korea	Indoor	Cl					11000 (6600-24000)	(Lee et al., 2010)
	Indoor	Cl/Oz					13000 (1200-22000)	
	Indoor	EGMO					23000 (11000-49000)	
Spain	Indoor	Cl	<100					(Cardador and Gallego, 2011)
	Outdoor	Cl	<200					
Portugal	Indoor	Various[a]			nd	(25-270)		(Riberio et al., 2011)
France	Indoor*	Cl	86000 (73000-107000)					(Parinet et al., 2012)
	Indoor*	DCICA	(68000-70000)					
China	Indoor	Cl	<2					(Xiao et al., 2012)
	Outdoor	Cl	<2					
Australia	Outdoor	Cl	<5					(Yeh et al., 2014)
Italy	Indoor	Various Cl		3 (<2-48)	(<20-22)	3700 (<5-20000)		(Righi et al., 2014)
	Indoor	Cl				2000 (100-20000)		
	Indoor	SDCIC				40 (5-60)		
	Indoor	TCICA				1700 (200-4500)		
Denmark	NR	Cl					4.0 (2.2-6.1)	(Spiliotopoulou et al., 2015)

Table A1-10 continued

Country	Pool(s) Type	Disinfection Method	Inorganic Anions in Swimming Pool Water (µg L^{-1}); mean (min-max).					Reference
			Bromide (Br^-)	Bromate (BrO_3^-)	Chlorite (ClO_2^-)	Chlorate (ClO_3^-)	Nitrate (NO_3^-)	
China	Indoor	ClO_2					51950 (13550-207610)	(Zhang et al., 2015)
	Outdoor	Cl					37680 (12930-88350)	
Saudi Arabia	Indoor*	Cl	560 (160-1090)					(Chowdhury et al., 2016)
China	Indoor	Cl	nd				63000 (18000-129000)	(E et al., 2016)
France	Indoor	Cl	78870 (72000-90500)					(Manasfi et al., 2016)
	Outdoor	Cl	200					
France	Indoor*	Cl	81000 (75000-89000)					(Manasfi et al., 2017)

*Seawater filled. **a**: Eight of the 54 pools investigated were sea water filled. **nd**: Not detected. **NR**: Not reported. **Br**: Bromine based (NaBr in combination with an oxidiser or Br_2). **Cl**: Chlorine based (NaOCl, $Ca(OCl)_2$ or Cl_2). **ClO_2**: Chlorine dioxide. **DCICA**: Dichloroisocyanuric acid. **EGMO**: Electrochemically-generated mixed-oxidant. **Oz**: Ozone. **SDCIC**: Sodium dichloroisocyanurate. **TCICA**: Trichloroisocyanuric acid. **Various**: Refers to any of the following individually or in combination: Cl Based, SDCIC, chloroisocyanurate (CIC), DCICA, TCICA, Br based (NaBr or Br_2) and/or ultraviolet irradiation (UV).**Various Cl**: Refers to any of the following individually or in combination: Cl Based, SDCIC, chloroisocyanurate (CIC), DCICA, TCICA and/or ultraviolet irradiation (UV).

Table A1-11: Reported occurrence of total organic halogen in swimming pool and spa waters.

Country	Pool(s) Type	Disinfection Method	Total Organic Halogen in Swimming Pool Water (µg L^{-1}); mean (min-max). TOX	TOCl	TOBr	TOI	Reference
Australia	Indoor	Cl/Oz	(880-1080)				(Kelsall and Sim, 2001)
	Indoor	Cl	(930-1380)				
	Indoor	Br/Oz	(810-970)				
Germany	Indoor	Cl	235				(Glauner et al., 2005)
	Outdoor	Cl	(161-177)				
Germany	Indoor	Cl		(124-136)			(Schmalz et al., 2011b)
China	Indoor	Cl		246	4		(Xiao et al., 2012)
	Outdoor	Cl		213	4		
Australia	Indoor	Cl	1508	1490	15	3.2	(Yeh et al., 2014)
	Outdoor	Cl	699 (194-1150)	680 (193-1117)	18 (1.6-32)	0.6 (nd-1.3)	
	Indoor	EGMO	1538	1524	12	1.3	
	Outdoor	EGMO	1049	1039	8	2.5	
	Baby	EGMO	(2894-3015)	(2825-2907)	(69-107)	(nd-1.3)	
USA	Indoor	Cl		3682 (1428-4828)	200 (137-280)		(Daiber et al., 2016)
	Indoor	BCDMH		1337 (1162-1511)	4897 (4106-5688)		
	Spa	BCDMH		1213 (950-1394)	4197 (2198-5444)		
	Spa	Br/TCICA		13860	18239		
	Spa	Cl/Oz		(1081-9512)	(53-84)		
Spain	Indoor	Cl	480 (420-570)	450 (390-550)	600 (500-800)		(Font-Ribera et al., 2016)
France	Indoor*	Cl		707 (510-920)*			(Manasfi et al., 2017)

nd: Not detected. * measured as extractable organic chlorine (EOCl). **TOBr**: Total organic bromine. **TOCl**: Total organic chlorine. **TOI**: Total organic iodine. **TOX**: Total organically bound halogen. **BCDMH**: Bromochlorodimethylhydantoin. **Br**: Bromine based (NaBr in combination with an oxidiser or Br_2). **Cl**: Chlorine based (NaOCl, $Ca(OCl)_2$ or Cl_2). **EGMO**: Electrochemically-generated mixed-oxidant. **Oz**: Ozone. **TCICA**: Trichloroisocyanuric acid.

Table A1-12: Reported occurrence of trihalomethanes in the ambient air of indoor swimming pool complexes.

Country	Disinfection Method	Trihalomethane Concentration in Swimming Pool Air (µg m^{-3}); mean (min-max). Total THMs (TTHMs)	Trichloro-methane (Chloroform)	Bromodichloro-methane	Dibromochloro-methane	Tribromo-methane (Bromoform)	Collection height above water's surface (cm)	Collection Method	Analytical Method	Reference
Germany	Cl		117 (10-384)	9.5 (0.1-39)			Directly	XAD-2 Resin	LD GC-MS/ECD	(Lahl et al., 1981)
Canada	Cl [a] Br [a]		154 (4-750)			747 (nd-1910)	10-20	Tenax	PT GC-MS	(Benoit and Jackson, 1987)
Italy	NR		214 (66-650)				150	Glass Vial	DI GC-MS	(Aggazzotti et al., 1990)
Canada	NR		1252 (507-1630)				150	Glass Vial	DI GC-MS	(Lévesque et al., 1994)
Germany	Cl		(7.8-191)	(nd-22.4)	(nd-2.9)		NR	NR	HS GC-ECD	(Cammann and Hübner, 1995)
Italy	NR		222 (16-853)				150	Glass Vial	DI GC-MS	(Aggazzotti et al., 1995)
Italy	NR		92 (69-103)[a] 170 (135-195)[b]	11 (7-14)[b] 20 (16-24)[c]	5.2 (4-7)[b] 11 (9-14)[c]	0.2[b] 0.2[c]	150	Glass Vial	DI CG-MS	(Aggazzotti et al., 1998)
Canada	Cl		(78-239)				Directly	Aluminized Bags	HS GC-EDC	(Lévesque et al., 2000)
Italy	NR	58	46	8.7	3.1	0.8	Directly	Tedlar Bags	DI GC-MS	(Fantuzzi et al., 2001)
Germany	NR		(85-235)				150	Activated Carbon	HS GC-ECD	(Erdinger et al., 2004)
Italy	Cl	(39-119)					NR	NR	HS GC-MS	(Aggazzotti et al., 2007)
Spain	Cl		242 (92-340)	9.1 (4.3-12)			50	Chromosorb 102	ATD GC-MS	(Caro et al., 2007)
Taiwan	NR		3510 (46-13000)				20-250	Summa Canisters	GC-MS	(Hsu et al., 2009)
France	Cl		39 (17-81)				100	Tenax	ATD GC-MS	(Thiriat et al., 2009)

Table A1-12 continued

Country	Disinfection Method	Trihalomethane Concentration in Swimming Pool Air (µg m^{-3}); mean (min-max). Total THMs (TTHMs)	Trichloro-methane (Chloroform)	Bromodichloro-methane	Dibromochloro-methane	Tribromo-methane (Bromoform)	Collection height above water's surface (cm)	Collection Method	Analytical Method	Reference
Italy	Cl		85 (21-182)[d] 52 (12-127)[e]				150	Activated Carbon	LLE GC-MS	(Aprea et al., 2010)
Italy	Cl	81 (36-127)					NR	Tedlar Bags	DI GC-MS	(Fantuzzi et al., 2010)
Spain	Cl	74					NR	Tenax	PT GC-MS	(Kogevinas, 2010)
Spain	Cl	74 (44-125)	35 (19-62)	15 (7.5-24)	13 (6-26)	11 (4-23)	60	Tenax	PT GC-MS	(Font-Ribera et al., 2010c)
Spain	Cl BCDMH		32 (12-62) 4.4 (1.7-9.4)	15 (7.5-23) 2.9 (1.7-4.8)	14 (6.1-26) 7.3 (6.1-9.7)	11 (4.4-23) 75 (53-101)	60	Tenax	ATD GC-MS	(Richardson et al., 2010)
France	Cl		75 (1.5-793)				NR	Tenax	ATD GC-MS	(Bessonneau et al., 2011)
Portugal	Cl	(98-1225) (51-519)					5 150	Glass Vial	HS-SPME GC-ECD	(Sa et al., 2011)
Spain	Cl Br		32 (18-61) 4.5 (1.8-6.9	15 (8.2-23) 3 (1.9-4.2)	14 (6.4-22) 7.3 (6.4-8.7)	6.4 (5.9-22) 75 (55-92)	60	Tenax	ATD GC-ECD	(Lourencetti et al., 2012)
Canada	Cl Cl	130 (47-311) 90 (34-180)	129 (46-307) 89 (34-178)	1.6 (nd-4.3) 1.1 (nd-2.6)			30 and 150	Activated Carbon	LD-USH GC-ECD	(Catto et al., 2012)
Portugal	Cl		(45-373)				30	Activated Carbon	HS-SPME GC-ECD	(Silva et al., 2012)
Taiwan	Cl		36 (13-182)				150	-	OP-FTIR	(Chen et al., 2016)
Canada	Cl	195 (117-320) 60 (2.9-140)[f]	148 (54-241) 53 (2.7-134)[f]	25 (3.8-86) 4.9 (0.1-16)[f]	16 (0.2-95) 1.9 (nd-4.8)[f]	9.6 (nd-36) 1.2 (nd-1.8)[f]	150 -	Activated Carbon	LD GC-ECD	(Tardif et al., 2015)

Table A1-12 continued

Country	Disinfection Method	Trihalomethane Concentration in Swimming Pool Air (µg m^{-3}); mean (min-max). Total THMs (TTHMs)	Trichloro-methane (Chloroform)	Bromodichloro-methane	Dibromochloro-methane	Tribromo-methane (Bromoform)	Collection height above water's surface (cm)	Collection Method	Analytical Method	Reference
Saudi Arabia	Cl*	83 (36-134)					60	Tenax	ATD GC-ECD	(Chowdhury et al., 2016)
Canada	Cl	191 (58-552)	119 (20-320)	31 (1.3-155)	27 (nd-205)	14 (nd-103)	150	Activated Carbon	LD GC-ECD	(Tardif et al., 2016a)
Saudi Arabia	Cl*	69 (46-106)	5.0 (1.5-9.2)	5.7 (1.3-13)	6.6 (2.8-12)	52 (26-88)	60	Tenax	TD GC-MS	(Chowdhury, 2016)
Canada	Cl	241 (232-250)					150	Activated Carbon	ATD GC-ECD	(Tardif et al., 2016b)
France	Cl*		3.4 (NR-29)	2.5 (NR-19)	14 (NR-150)	266 (NR-1600)	20	Charcoal	LD GC-ECD	(Boudenne et al., 2017)
France	Cl*	146 (25-261)	nd	nd	13 (3.6-21)	133 (22-240)	100	Canister	PTR ToF-MS	(Manasfi et al., 2017)
Norway	Cl[g]	297 (89-782)	213 (89-477)	29 (nd-94)	16 (nd-38)	97 (nd-319)	5-150	Tenax	TD GC-MS	(Nitter et al., 2017)
Sweden	Cl[h]	NR	12	NR	NR	NR	150	Activated Carbon	ATD GC-ECD	(Westerlund et al., 2018)

***** Seawater filled**. a**: Spa. **b**: No water activity. **c**: During water activities – swimmers. **d**: Sampling over 9 hours. **e**: Sampling over 24 hours. **f**: Offices**. g**: Some pools partly filled with seawater**. h**: 70 and 10% of pools were equipped with ultraviolet irradiation (UV) and ozone as secondary treatments, respectively. **nd**: Not detected. **NR**: Not reported. **BCDMH**: Bromochlorodimethylhydantoin. **Br**: Bromine based (NaBr in combination with an oxidiser or Br_2). **Cl**: Chlorine based (NaOCl, $Ca(OCl)_2$ or Cl_2). **ATD**: Automatic thermal desorption. **DI**: Direct injection. **ECD**: Electron capture detector. **FTIR**: Fourier tranform infrared spectroscopy. **GC**: Gas chromatography. **HS**: Headspace. **LD**: Liquid-desorption. **LLE**: Liquid-liquid extraction. **MS**: Mass spectrometry. **OP**: Open path. **PT**: Purge and trap. **PTR**: Proton-transfer-reaction. **SPME**: Solid-phase microextraction. **ToF**: Time of flight. **USH**: Ultrasound heating.

Table A1-13: Reported occurrence of trichloramine in the ambient air of indoor swimming pool complexes.

Country	Disinfection Method	Trichloramine (NCl_3) Concentration in Swimming Pool Air (µg m^{-3})	Collection height above water's surface (cm)	Collection Method	Analytical Method	Reference
Belgium	Cl	(250-450)	NR	NR	NR	(Bernard et al., 2006)
Netherlands	Cl/Oz [a]	560 (130-1340) 660 (380-1100)[b]	30 150	Quartz Fibre[c]	LD IC-MS	(Jacobs et al., 2007)
Spain	Cl BCDMH	290 (170-430) 80 (70 -100)	60 60	Quartz Fibre[c]	LD IC-MS	(Richardson et al., 2010)
France	Cl	190 (20-1260)	NR	Quartz Fibre[c]	LD IC-MS	(Bessonneau et al., 2011)
Germany	Cl	(160-190)	NR	NR	NR	(Schmalz et al., 2011a)
Canada	Cl	180 (80-350)	150	Activated Carbon	LD-USH GC-ECD	(Catto et al., 2012)
Switzerland	Various Cl	110 (1-890)	NR	Quartz Fibre[c]	LD IC-MS	(Parrat et al., 2012)
Italy	NR	637 (204-1020)	150	Glass Vial	DPD/KI Colorimetric	(Predieri and Giacobazzi, 2012)
Taiwan	NR	(20-150)	100	Quartz Fibre[c]	LD IC-MS	(Chu et al., 2013)
Sweden	NR	200 (40-360)	130	Quartz Fibre[c]	LD IC-MS	(Fornander et al., 2013)
USA	Cl	150 (nd-620)	NR	Glass Vial	DPD/KI Colorimetric	(Zare Afifi and Blatchley, 2015)
Germany	NR	(150-300)	20	Direct Analysis	IMS	(Zwiener and Schmalz, 2015)
Canada	Cl	380 (110-700)	30	Pallflex Tissuquartz[d]	IC	(Lévesque et al., 2015)
Canada	Cl	270 (60-450)[b]	150	Teflon[e]	LD IC-MS	(Tardif et al., 2015)
Sweden	Cl	130 (20-290)	130	NR[e]	IC	(Johannesson et al., 2016)
Canada	Cl	230 (nd-560)[b]	150	Teflon[e]	LD IC-MS	(Tardif et al., 2016a)
Spain	Cl	150 (70-320)[f] 473 (249-858)	150	Quartz Fibre[c]	LD IC-MS	(Font-Ribera et al., 2016)
Canada	Cl	582 (420-820)[b]	150	Teflon[e]	LD IC-MS	(Tardif et al., 2016b)
Belgium	NR	526 (200-1280)	150	Teflon[e]	LD IC-MS	(Jmaiff Blackstock et al., 2017)
Spain	Cl	620 (290-960)	5	NR	CE	(Gomà et al., 2017)
Sweden	Cl[g]	23 (1-140)	150	Glass Fibree	LD IC	(Westerlund et al., 2018)
Sweden	Cl	150 (20-550)	30	NR[e]	LD IC	(Andersson et al., 2018)

a: Number of pools treated with chlorine (76%), salt electrolysis (11%) chlorine/salt electrolysis (5%) and ozone/chlorine (8%). **b**: Refers to total chloramines (sum of mono-, di- and tri-chloramine). **c:** Injected with diarsenic-trioxide, sodium carbonate and glycerol. **d**: Cellulose filter impregnated with sodium carbonate. **e**: Filter media impregnated with sodium carbonate and arsenic trioxide. **f**: Refers to monochloramine. **g**: 70 and 10% of pools were equipped with ultraviolet irradiation (UV) and ozone as secondary treatments, respectively. **NR**: Not reported. **BCDMH**: Bromochlorodimethylhydantoin. **Cl**: Chlorine based (NaOCl, $Ca(OCl)_2$ or Cl_2). **Oz**: Ozone. **Various Cl**: Refers to any of the following individually or in combination: Cl based, sodium dichloroisocyanurate (SDCIC), chloroisocyanurate (CIC), dichloroisocyanuric acid (DCICA) and/or trichloroisocyanuric acid (TCICA). **CE**: Capillary electrophoresis. **DPD**: Diethyl-*p*-phenylenediamine. **ECD**: Electron capture detector. **GC**: Gas chromatography. **IC**: Ion chromatography**. IMS:** Ion mobility spectrometer. **KI**: Potassium iodide. **LD**: Liquid-desorption**. MS**: Mass spectrometry. **USH**: Ultrasound heating.

Table A1-14: Reported occurrence of total organic carbon, total nitrogen and urea in swimming pool and spa waters.

Country	Pool(s) Type	Disinfection Method	Total Carbon, Total Nitrogen and Urea in Swimming Pool Water (mg L^{-1}); mean (min-max).			Reference
			Total Organic Carbon (TOC)	Total Nitrogen (TN)	Urea	
Canada	Spa	Br	111 (5-345)			(Benoit and Jackson 1987)
	Spa	Cl	36 (1-155)			
England	Indoor	NR	6.3 (3.3-13)			(Chu and Nieuwenhuijsen 2002)
Japan	Outdoor	NR			(0.2-0.7)	(Tachikawa et al., 2005)
Germany	Indoor	Cl	1.7			(Glauner et al. 2005)
	Outdoor	Cl	(1.6-2)			
Thailand	NR	NR	1.1 (0.6-1.5) 0.9 (0.6-1.5)[a]			(Panyakapo et al. 2008)
Korea	Indoor	Cl	4.4 (0.2-71)			(Lee et al. 2009)
	Indoor	Cl/Oz	3.7 (0.2-82)			
	Indoor	EGMO	3.5 (0.4-12)			
USA	Indoor	Cl	7.1 (3-24)	3.6 (0.8-12)		(Kanan 2010)
Korea	Indoor	Cl	2.3 (0.5-7)			(Lee et al. 2010)
	Indoor	Cl/Oz	1.7 (0.7-3)			
	Indoor	EGMO	3.2 (1.9-5.8)			
France	Indoor	Cl	3.5 (1.6-7.3)		1.1 (0.1-3.7)	(De Laat et al. 2011)
France	Indoor	Cl	3.1 (1.8-7.3)			(Bessonneau et al. 2011)
Germany	Indoor	Cl	1.3[a]		0.8	(Schmalz et al., 2011b)
Germany	Various	NR			1.3 (0.5-2.1)	(Schmalz et al., 2011a)
USA	Indoor	Cl/UV	(5.2-18)			(Plewa et al., 2011)
	Indoor	BCDMH	125			
	Indoor	Cl	23 (13-33)			
	Outdoor	Cl	33			
	Spa	Cl	12			
Portugal	Indoor	Cl	4 (1.13-6.7)			(Sa et al., 2011)
Canada	Indoor	Cl	2.1 (0.02-4.4)			(Wang, 2011)
	Outdoor	Cl	6.2 (0.02-16			
USA	Indoor	Cl			(<0.1-0.3)	(Weng and Blatchley, 2011)
France	Indoor	Cl	4.8 (3.6-8.6)	3 (0.7-7.7)		(Parinet et al., 2012)
	Indoor	DCICA	(2.8-3.3)	(1.3-2.7)		

Table A1-14 continued

Country	Pool(s) Type	Disinfection Method	Total Carbon, Total Nitrogen and Urea in Swimming Pool Water (mg L^{-1}); mean (min-max).			Reference
			Total Organic Carbon (TOC)	Total Nitrogen (TN)	Urea	
China	Indoor Outdoor	Cl Cl	3.2 2.8			(Xiao et al., 2012)
Portugal	NR	NR	5.4 (2.4-7.4)			(Prieto-Blanco et al., 2012)
Switzerland	Indoor	NR			0.3 (nd-2)	(Parrat et al., 2012)
Canada	Indoor Indoor	Cl/UV Cl	12 (10-15) 7.3 (4.9-9.5 [a] 13 (11-16) 7.9 (5.9-11)[a]			(Wang et al., 2013)
Australia	Outdoor	Cl	3.4 (3.1-3.9)	2.9 (0.6-4.6) 0.4 (0.1-0.7)[b]		(Yeh et al., 2014)
Switzerland	Various	Various			0.3 (<0.1-0.6)	(Soltermann et al., 2014)
USA	Indoor	Cl			0.1	(Zare Afifi and Blatchley, 2015)
Portugal	Indoor	NR	7.2 (7.1-7.3)			(Maia et al., 2014)
USA	Indoor Outdoor Spa	NR NR NR	4.7 (1.3-8.4) 2.5 (0.9-8.5) 8.1 (3.7-11)			(Wang et al., 2014)
China	Indoor Outdoor	NR NR	11 (2.7-27) 9.5 (nd-13)			(Wang et al., 2014)
Australia	Indoor Spa	Cl Cl	5.7 (3.6-7.2) 12			(Carter et al., 2015)
Denmark	NR	Cl	1.9 (1.6-2.2)			(Spiliotopoulou et al., 2015)
China	Indoor Outdoor	Cl/Oz Cl	8.9 (2.5-27) 9.5 (3.2-13)	2.2 (0.2-11)[b] 0.9 (0.2-1.3)[b]		(Zhang et al., 2015)
Saudi Arabia	Indoor*	Cl	2.1 (1.3-3.9) 1.9 (1.0-3.6)[a]			(Chowdhury et al., 2016)
Spain	Indoor	Cl	2.4 (1.8-10)			(Font-Ribera et al., 2016)
China	Indoor Indoor	Cl/Oz Cl	13 (8.0-25)[a] 22 (4.2-39)[a]			(Hang et al., 2016)
France	Indoor Outdoor	Cl Cl	11 (10-12) 11			(Manasfi et al., 2016)

Table A1-14 continued

Country	Pool(s) Type	Disinfection Method	Total Carbon, Total Nitrogen and Urea in Swimming Pool Water (mg L^{-1}); mean (min-max).			Reference
			Total Organic Carbon (TOC)	Total Nitrogen (TN)	Urea	
China	Outdoor	Cl	1.1 (NR)			(Tang and Xie, 2016)
Canada	Indoor	Cl	1.0 (1.4-10)[a]			(Tardif et al., 2016a)
China	Outdoor	Cl	4.5 (1.0-6.5)	5.8 (4.1-8.4)		(Yang et al., 2016)
Saudi Arabia	Indoor*	Cl	2.4 (1.4-3.6)[a]	0.2 (<0.1-0.4)		(Chowdhury, 2016)
France	Indoor*	Cl	20 (14-29)			(Boudenne et al., 2017)
Canada	NR	Cl	9.5 (6.7-14)[a]			(Jmaiff Blackstock et al., 2017)
	Spa	Cl	19 (7.4-41)[a]			
France	Indoor*	Cl	3.2 (2.7-3.9)	1.8 (1.4-2.3)		(Manasfi et al., 2017)
China	Outdoor	TCICA	13 (2-39)	15 (nd-35)	6.1 (nd-17)	(Yang et al., 2018)
Iran	Indoor	Cl	7.1 (1.1-17)			(Dehghani et al., 2018)
Poland	Outdoor	Cl	3.2 (1.1-4.4)			(Klosok-Bazan et al., 2018)
Poland	NR	NR	10 (1.1-17)			(Lempart et al., 2018a)
Poland	NR	Cl	5.0 (0.7-28)			(Lempart et al., 2018b)

*Sea water filled. **a:** Reported as dissolved organic carbon (DOC). **b:** Reported as total organic nitrogen (TON). **nd**: Not detected. **NR**: Not reported. **Br**: Bromine based (NaBr in combination with an oxidiser or Br_2). **Cl**: Chlorine based (NaOCl, $Ca(OCl)_2$ or Cl_2). **BCDMH**: Bromochlorodimethylhydantoin. **DCICA**: Dichloroisocyanuric acid. **EGMO**: Electrochemically-generated mixed-oxidant. **TCICA**: Trichloroisocyanuric acid. **Various**: Refers to any of the following individually or in combination: Cl based, Br based.

Table A1-15: Common and other names of selected personal care products.

Antifungal Agents	
Dichlorophen	Dichlorophene; 4-chloro-2-[(5-chloro-2-hydroxyphenyl)methyl]phenol; eptiphene; *o*-benzyl-*p*-chlorophenol; *ortho*-benzyl-*p*-chlorophenol; benzylchlorophenol; 5-chloro-2-hydroxydiphenylmethane; 4-chloro-alpha-phenyl-*o*-cresol; chlorofene; 4-chloro-2-(phenylmethyl)phenol; benzyl-*p*-chlorophenol; 4-chloro-alpha-phenylcresol; 2-benzyl-4-chlorophenol; 4-chloro-2-(phenylmethyl)phenol; 5-chloro-2-hydroxydiphenylmethane; *o*-benzyl*para*chlorophenol; *p*-chloro-*o*-benzylphenol; ketolin-h; santophen 1; neosobenil; sentiphene clorofenum
2,4-DCPh	5-chloro-(2,4-dichlorophenoxy)phenol; 2,4,4'-trichloro-2'-hydroxydiphenyl ether; trichloro-2'-hydroxydiphenyl ether; triclosan; CH-3565, Lexol 300, Irgasan DP 300, Ster-Zac
BTH	2,6-di-*tert*-butyl-*p*-cresol; 3,5-di-*tert*-butyl-4-hydroxytoluene; DBPC; BHT; AO-29; Avox BHT; Additin RC 7110; dibutylated hydroxytoluene; 4-methyl-2,6-di-*tert*-butyl phenol
TCC	Triclocarban; Trichlorocarbanilide; Solubacter; 3-(4-Chlorophenyl)-1-(3,4-dichlorophenyl)urea
2,4,6-TCPh	2,4,6-trichlorophenol; phenaclor; Dowicide 2S; Dowcide 2S; omal
Parabens	
PHBA	*p*-hydroxybenzoic acid; 4-hydroxybenzoic acid; para-hydroxybenzoic acid; 4-hydroxybenzoate
MeP	methylparaben; methyl-4-hydroxybenzoate; methyl *p*-hydroxybenzoate; methyl parahydroxybenzoate; Nipagin M; E218; Tegosept; Mycocten
EtP	ethylparaben; ethyl-4-hydroxybenzoate; ethyl *para*hydroxybenzoate; ethyl *para*-hydroxybenzoate; ethyl-*p*-hydroxybenzoate; 4-hydroxybenzoic acid ethyl ester; E214
PrP	propylparaben, propyl-4-hydroxybenzoate; 4-hydroxybenzoesäurepropylester; propyl-*p*-hydroxybenzoate; propyl *para*hydroxybenzoate; nipasol; E216
iPrP	isopropylparaben
BuP	butylparaben; butyl-4-hydroxybenzoate; butyl-*para*hydroxybenzoate; butyl-*p*-hydroxybenzoate
iBuP	isobutylparaben
PeP	pentylparaben; pentyl-4-hydroxybenzoate; amyl-4-hydroxybenzoate; pentyl-*p*-hydroxybenzoate; n-pentyl-4-hydroxybenzoate
HeP	heptaparaben; heptyl-*p*-hydroxybenzoate; heptyl-4-hydroxybenzoate; n-heptyl-4-hydroxybenzoate; n-heptyl-*p*-hydroxybenzoate; heptyl-*para*-hydroxybenzoate; nipaheptyl; E209
OcP	octylparaben; octyl-4-hydroxybenzoate; *n*-octyl-4-hydroxybenzoate; octyl-*p*-hydroxybenzoate
BzP	benzylparaben; benzyl-4-hydroxybenzoate; benzyl *p*-hydroxybenzoate; benzyl *para*hydroxybenzoate; phenylmethyl 4-hydroxybenzoate; nipabenzyl; parosept; benzyl parasept
UV-Filters	
Amiloxate	isoamyl 4-methoxycinnamate; 3-methylbutyl-(2*E*)-3-(4-methoxyphenyl)acrylate; isopentyl 4-methoxycinnamate; isoamyl-*p*-methoxycinnamate
Avobenzone	avobenzone; 1-(4-methoxyphenyl)-3-(4-*tert*-butylphenyl)propane-1,3-dione; butylmethoxydibenzoylmethane; 4-*tert*-butyl-4'-methoxydibenzoylmethane; Eusolex 9020; Parsol 1789; Milestab 1789; Escalol 517; Neo Heliopan 357
BP-1	2,4-dihydroxybenzophenone
BP-2	2,2′,4,4′-tetrahydroxybenzophenone
BP-3	benzophenone-3; oxybenzone; (2-hydroxy-4-methoxyphenyl)-phenylmethanone; 2-hydroxy-4-methoxybenzophenone; Eusolex 4360; Milestab 9; Escalol 567; KAHSCREEN BZ-3
BP-8	2,2′-dihydroxy-4-methoxybenzophenone
BzS	benzyl salicylate; benzyl 2-hydroxybenzoate
4-DHB	4, 4′-dihydroxybenzophenone
DMeBT	5,6-dimethyl-1*H*-benzotriazole monohydrate
Et-PABA	ethyl 4-aminobenzoate

Table A1-15 continued

UV-Filters	
4-HB	4-hydroxybenzophenone
1HBT	1*H*-benzotriazole
Homosalate	3,3,5-trimethylcyclohexyl-2-hydroxybenzoate; Eusolex HMS
4-MBC	4-methylbenzylidene camphor; enzacamene; (3*E*)-1,7,7-trimethyl-3-[(4-methylphenyl)methylene]-2-norbornanone; 3-(4-methylbenzylidene)bornan-2-one; 3-(4-methylbenzylidene)-dl-camphore; Eusolex 6300
OCR	octocrylene; 2-ethylhexyl-2-cyano-3,3-diphenyl-2-propenoate; 2-ethylhexyl-2-cyano-3,3-diphenylacrylate; Eusolex OCR
OD-PABA	octyldimethyl-para-aminobenzoic acid; 2-ethylhexyl-4-(dimethylamino)benzoate; Padimate O; Escalol 507; Sundown
OMC	octylmethoxycinnamate; (*RS*)-2-ethylhexyl-(2*E*)-3-(4-methoxyphenyl)prop-2-enoate; ethylhexyl-methoxycinnamate; (*E*)-3-(4-methoxyphenyl)-prop-2-enoic acid; 2-ethylhexyl ester; octinoxate; Eusolex 2292; Uvinul MC80
PBS	2-phenyl-3*H*-benzimidazole-5-sulfonic acid; Ensulizole
PS	phenyl salicylate; phenyl 2-hydroxybenzoate
THB	2 ,3 ,4 –trihydroxybenzophenone
Other	
5-MeBT	5-methyl-1H-benzotriazole; 5-tolyltriazole
5-CBT	5-Cl-1H-benzotriazole; 5-chloro-1H-benzotriazole

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APPENDIX 2

Table A2-1: Ions (mass-to charge-ratio; m/z) used for identification and quantification of haloacetamides, haloacetonitriles and halonitromethanes in selective ion monitoring (SIM) mode.

Analyte	Abbreviation	Quantification Ion (m/z)	Additional Identification Ions (m/z)
Haloacetamides (HAAms)			
Dichloroacetamide	DCAAm	44	127
Dibromoacetamide	DBAAm	44	217, 172
Bromochloroacetamide	BCAAm	44	173
Bromodichloroacetamide	BDCAAm	44	82
Dibromochloroacetamide	DBCAAm	44	128
Trichloroacetamide	TCAAm	44	82
Tribromoacetamide	TBAAm	44	172
Haloacetonitriles (HANs)			
Chloroacetonitrile	CAN	75	77
Bromoacetonitrile	BAN	119	121
Bromochloroacetonitrile	BCAN	74	82
Dichloroacetonitrile	DCAN	74	82
Dibromoacetonitrile	DBAN	120	118, 93
Bromodichloroacetonitrile	BDCAN	163	161
Dibromochloroacetonitrile	DBCAN	154	152
Trichloroacetonitrile	TCAN	108	110, 82
Tribromoacetonitrile	TBAN	209	205
Halonitromethanes (HNMs)			
Chloronitromethane	CNM	49	51
Bromonitromethane	BNM	93	95
Dichloronitromethane	DCNM	83	85
Dibromonitromethane	DBNM	173	175
Bromochloronitromethane	BCNM	129	127, 131
Bromodichloronitromethane	BDCNM	163	161
Dibromochloronitromethane	DBCNM	209	205
Trichloronitromethane	TCNM	117	119, 121,82
Tribromonitromethane	TBNM	251	253

Table A2-2: Concentrations of nitrogenous disinfection by-products (N-DBPs) measured in lap, leisure and spa pools. Concentrations are displayed in µg L^{-1} and represent the average of duplicate analyses.

Analyte	Abbreviation	Limit of Detection (µg L^{-1})	Lap Pool	Leisure Pool	Spa Pool
Haloacetamides (HAAms)					
Dichloroacetamide	DCAAm	1.1	<LOD	<LOD	3.5
Dibromoacetamide	DBAAm	1.2	<LOD	<LOD	<LOD
Bromochloroacetamide	BCAAm	1.2	3.4	3.0	2.6
Bromodichloroacetamide	BDCAAm	1.1	<LOD	<LOD	<LOD
Dibromochloroacetamide	DBCAAm	1.2	<LOD	<LOD	<LOD
Trichloroacetamide	TCAAm	1.4	3.3	3.0	2.7
Tribromoacetamide	TBAAm	1.0	<LOD	<LOD	<LOD
Haloacetonitriles (HANs)					
Chloroacetonitrile	CAN	1.6	<LOD	1.8	1.9
Bromoacetonitrile	BAN	1.3	<LOD	<LOD	<LOD
Dichloroacetonitrile	BCAN	1.6	16	41	27
Dibromoacetonitrile	DCAN	1.1	<LOD	<LOD	<LOD
Bromochloroacetonitrile	DBAN	1.3	<LOD	<LOD	<LOD
Bromodichloroacetonitrile	BDCAN	0.9	<LOD	<LOD	1.5
Dibromochloroacetonitrile	DBCAN	0.8	<LOD	<LOD	<LOD
Trichloroacetonitrile	TCAN	1.5	<LOD	<LOD	<LOD
Tribromoacetonitrile	TBAN	1.7	<LOD	<LOD	<LOD
Halonitromethanes (HNMs)					
Chloronitromethane	CNM	1.3	<LOD	<LOD	<LOD
Bromonitromethane	BNM	1.1	<LOD	<LOD	<LOD
Dichloronitromethane	DCNM	1.7	<LOD	<LOD	<LOD
Dibromonitromethane	DBNM	1.1	<LOD	<LOD	<LOD
Bromochloronitromethane	BCNM	1.2	<LOD	<LOD	<LOD
Bromodichloronitromethane	BDCNM	1.2	<LOD	<LOD	<LOD
Dibromochloronitromethane	DBCNM	1.5	<LOD	<LOD	<LOD
Trichloronitromethane	TCNM	1.3	<LOD	1.4	<LOD
Tribromonitromethane	TBNM	1.4	<LOD	<LOD	2.8

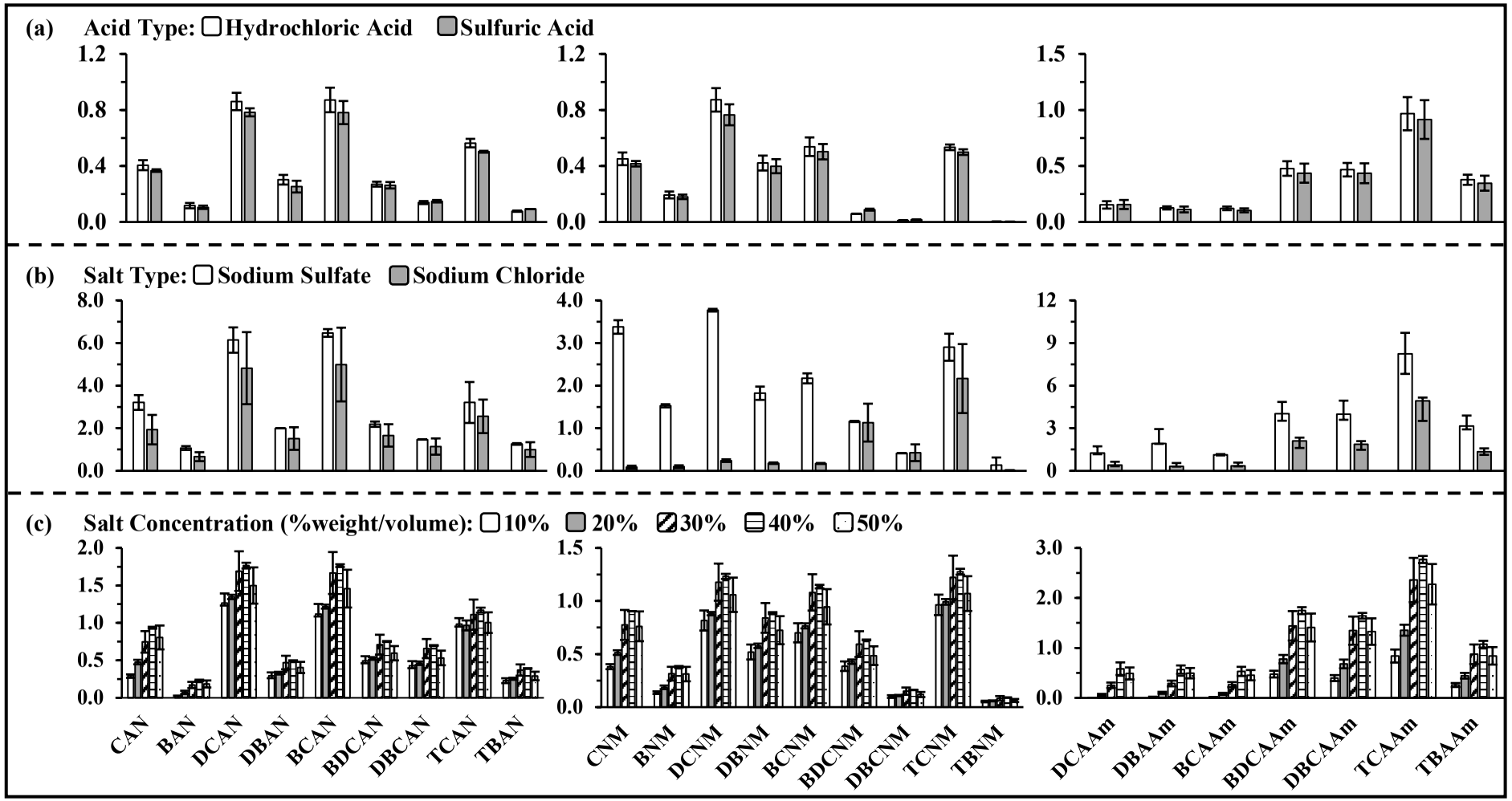

Figure A2-1: The effect of **(a)** acid type, **(b)** salt type and **(c)** salt concentration on the response ratio of haloacetonitriles (HANs; left), halonitromethanes (HNMs; middle) and haloacetamides (HAAms; right). For analyte abbreviations see **Table A2-2**.

APPENDIX 3

Table A3-1: Reported methods for the analysis of trihalomethanes (THMs) in the ambient air of swimming pool complexes.

Target Analytes	Sample Collection			Preparation and Injection			Separation			Detection		Reference	Secondary Reference
	Collection Method	Height Above Water (cm)	Pump Conditions	Sample Extraction	Sample Introduction	Injection Conditions	Column	Flow	Oven Temperature Program	Conditions	Limit of Detection ($\mu g\ m^{-3}$)		
THM-4	XAD_2-Resin filter (0 °C)	10-200		LD (pentane)	Auto, Liquid		50 m capillary			MS/ECD	0.03-0.1 ($\mu g\ L^{-1}$)	(Lahl et al., 1981)	(Batjer et al., 1980)
Chloroform Bromoform	Tenax	10-20	15 mins @ 617 mL min^{-1}	Purge & Trap		220 °C	glass column (2 m x 2 mm) packed with 1% SPI000 on Carbopack B (mesh 60/80)	He, 24 mL min^{-1}	70 °C (4 min) to 220 °C @ 12 °C min^{-1} (24 min)	MS	1.00	(Benoit and Jackson, 1987)	-
THM-4	Activated Carbon	150	10 L (air)	LD (3-phenoxy-benzyl-alcohol)	Auto, Liquid		Optima 624 (25 m x 0.32 mm x 1.4 µm)	He, 30 cm s^{-1}	40 °C (10 min) to 90 °C @ 5 °C min^{-1} (10 min) to 200 °C @ 10 °C min^{-1} (10 min)	ECD	0.04-0.08 ($\mu g\ L^{-1}$)	(Erdinger et al., 2004)	-
Chloroform	Summa Canisters	20-250	6 L							MS		(Hsu et al., 2009)	-
THM-4	Activated Carbon*	150	540 mins @ 100 mL min^{-1}	LD (CS_2, 30 mins)	Auto, Liquid						0.1 (μg sample^{-1})	(Aprea et al., 2010)	-
THM-4	Tenax (300 mg)		10 mL min^{-1}	ATD						MS	0.2-0.5	(Bessonneau et al., 2011)	-
THM-4	Activated Carbon (20/40), 100/50 mg	30 & 150	95 mins @ 165 mL min^{-1}	LD (CS_2, 1 mL; 30 mins) ultrasound heating	Auto, Liquid		VF5ms (30m x 0.35 mm x 0.25 µm)	He, 1 mL min^{-1}		ECD	0.095-0.690	(Catto et al., 2012)	-
Chloroform	Glass Vial (40 mL)	150		Gas Tight Syringe	DI, Air	150 °C	VOCOL (30 m x 0.53 mm x 3.0 µm)	He, 8 mL min^{-1}	50 °C (1 min) to 100 °C @ 6 °C min^{-1} (7 min)	ECD (260 °C)	1	(Aggazzotti et al., 1990)	
Chloroform	Glass Vial (40 mL)	>30		Gas Tight Syringe	DI, Air		DB-1 (30 m x 0.25 mm x 1 µm)	He, 14 psi		ECD	49	(Lévesque et al., 1994)	(Aggazzotti et al., 1990)
THM-4	Glass Vial (40 mL)	150		Gas Tight Syringe	DI, Air					ECD	0.1	(Aggazzotti et al., 1998)	(Aggazzotti et al., 1990)
Chloroform	Glass Vial (40 mL)	150		Gas Tight Syringe	DI, Air					ECD	1	(Aggazzotti et al., 1995)	(Aggazzotti et al., 1990)

Table A3-1 continued

Target Analytes	Sample Collection			Preparation and Injection			Separation			Detection		Reference	Secondary Reference
	Collection Method	Height Above Water (cm)	Pump Conditions	Sample Extraction	Sample Introduction	Injection Conditions	Column	Flow	Oven Temperature Program	Conditions	Limit of Detection (µg m^{-3})		
Chloroform	Aluminised Bag (5 L)	Directly	1000 mL min^{-1}	Gas Tight Syringe	DI, Air		DB-1 (30 m x 0.25 mm x 1 µm)			ECD	50	(Lévesque et al., 2000)	(Lévesque et al., 1994)
THM-4	Tedlar Bag (2 L)	Directly	120 mins @ 15 mL min^{-1}	Gas Tight Syringe	DI, Air							(Fantuzzi et al., 2001)	(Aggazzotti et al., 1990)
Chloroform	Tedlar Bag (2 L)	150	120 mins @ 15 ml/min	Gas Tight Syringe	DI, Air					MS	1	(Fantuzzi et al., 2010)	(Aggazzotti et al., 1995)
THM-4	Glass Vial (40 mL)	5 & 150	1 min @ 1000 mL min^{-1}	SPME (CAR/PDMS) 50 min, 30 °C	SPME, 10 mins	200 °C	VB-624 (30 m x 0.53 mm x 3.0 µm)	N_2, 1.1 mL min^{-1}	80 °C (5 mins) to 150 °C @ 10 °C min^{-1} (6 mins)	ECD (300 °C)	1.25-2.5	(Sa et al., 2011)	-
Chloroform	Activated Carbon	30	120 mins @ 200 mL min^{-1}							FID		(Silva et al., 2012)	(NIOSH, 2003)
Chloroform	Online	150				200 °C (10 mins) 300 °C (3 mins)				mercury–cadmium–telluride		(Chen et al., 2016)	(US EPA, 1999)
THM-4	Chromosorb 102 (200 mg)	50	15 mins @ 200 mL min^{-1}	ATD	Cold Trap (-10 °C, N_2)		HP-5MS (30 m x 0.25 mm x 0.25 µm)	He, 10 psi	-40 °C (1 min) to 180 °C @ 4 °C min^{-1} (0.1 mins) to 300 °C @ 30 °C min^{-1} (0.25 mins)	MS	0.01	(Caro and Gallego, 2008)	(Van Den Velde et al., 2007)
THM-4	PWAS & Summa Canister (1 L)	30		50 mL aliquot air	Cold Trap (-165 °C, primary; -190 °C, precolumn, (2 m x 0.53 mm)).	Precolumn heated to 150 °C	XTI-5 (30 m x 0.25 mm x 1 µm)		-50 °C (2 mins) to 220 °C @ 10 °C min^{-1} (8 min)	MS	0.39-1.7	(Lindstrom et al., 1997)	(US EPA, 1999)

ATD: Automatic thermal desorption. **DI**: Direct injection. **ECD**: Electron capture detector. **FID**: Flame ionisation detector. **LD**: Liquid desorption. **MS**: Mass spectrometry. **PWAS**: Personal whole air sampler. **SPME**: Solid-phase microextraction. **THM-4**: Refers to trichloromethane, bromodichloromethane, dibromochloromethane and tribromomethane.

Table A3-2: Ions (mass-to-charge ratio; m/z) used for identification and quantification of trihalomethanes (THMs), surrogate standard (1,2-dibromopropane; 1-2,DBP) and internal standard (1,2-dibromopropane-*d6*; 1,2-DBP) in selective ion monitoring (SIM) mode.

Analyte	Quantification Ion (m/z)	Additional Identification Ions (m/z)
Trichloromethane (Chloroform)	83	85
Bromodichloromethane	129	83, 127
Dibromochloromethane	129	127, 131
Tribromomethane (Bromoform)	173	171, 175, 256
1,2-Dibromopropane-*d6* (internal)	127	129
1,2-Dibromopropane (surrogate)	121	123

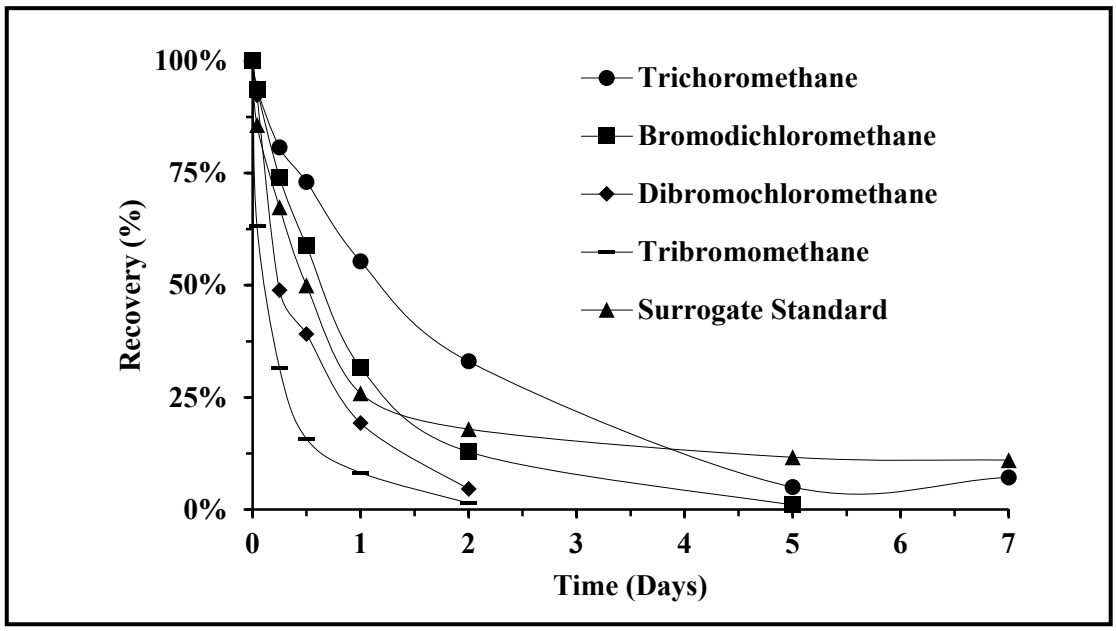

Figure A3-1: Effect of storage time on the loss of trihalomethanes (THMs) and surrogate standard (1,2-dibromopropane-*d6*, 1,2-DBP-d6) in ambient air samples. Data is presented as a percentage (response ratio at t=n/response ratio at t=0) where the response ratio refers to the peak area of a THM or surrogate standard divided by the peak area of the internal standard (1,2-dibromopropane, 25 µg m^{-3}) added prior to gas chromatography-mass spectrometry (GC-MS) analysis. Samples were prepared in flushed vials, fortified with each trihalomethane or surrogate standard (100 µg m^{-3}) and stored at room temperature (25 °C) over 7 days.

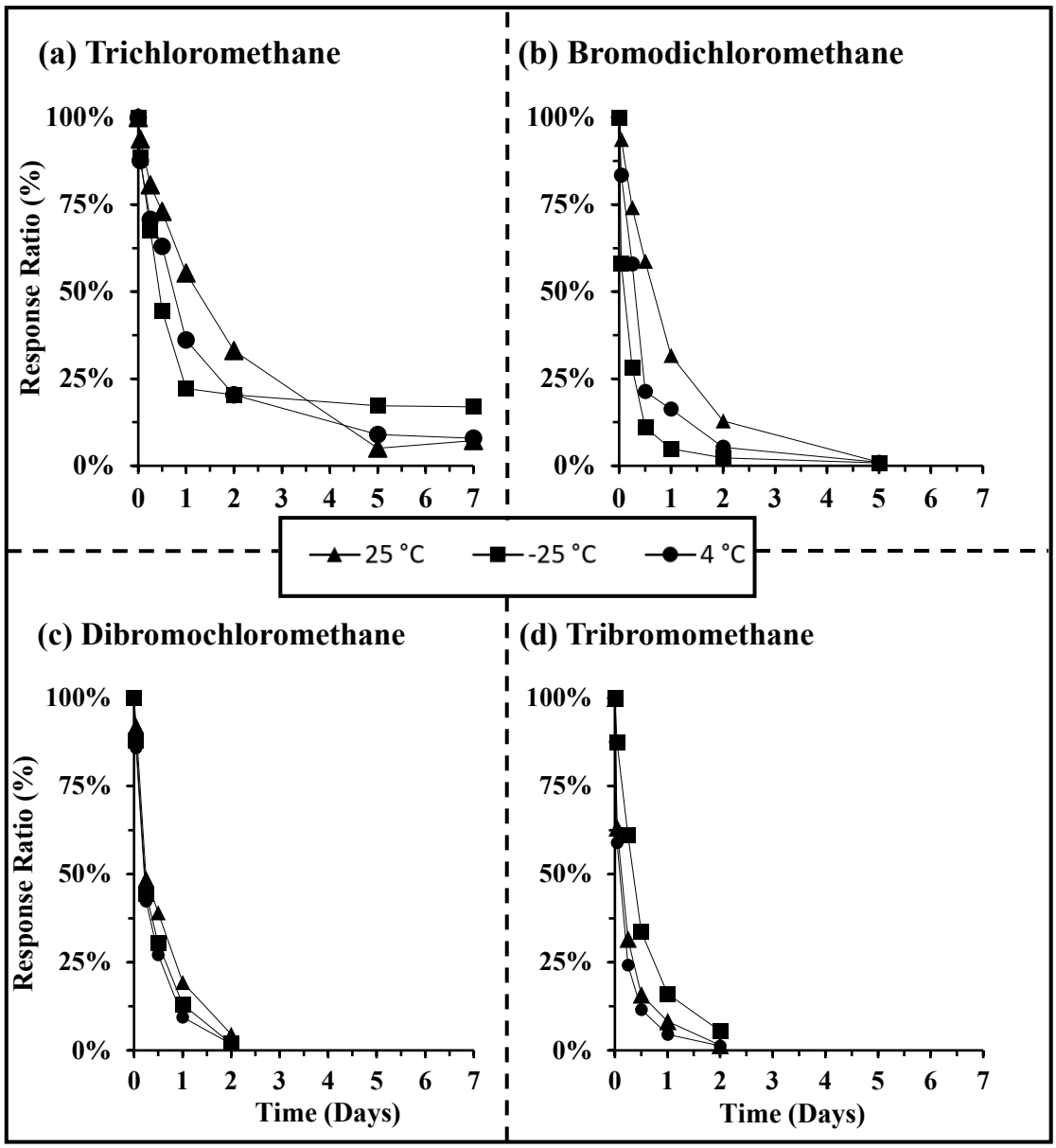

Figure A3-2: Effect of storage temperature on the loss of **(a)** trichloromethane, **(b)** bromodichloromethane, **(c)** dibromochloromethane and **(d)** tribromomethane in ambient air samples. Data is presented as a percentage (response ratio at t=n/response ratio at t=0) where the response ratio refers to the peak area of a THM divided by the peak area of the internal standard (1,2-dibromopropane, 25 µg m^{-3}) added prior to analysis. Samples were prepared in flushed vials, fortified with each trihalomethane (THM, 100 µg m^{-3}) and stored at various temperatures over time.

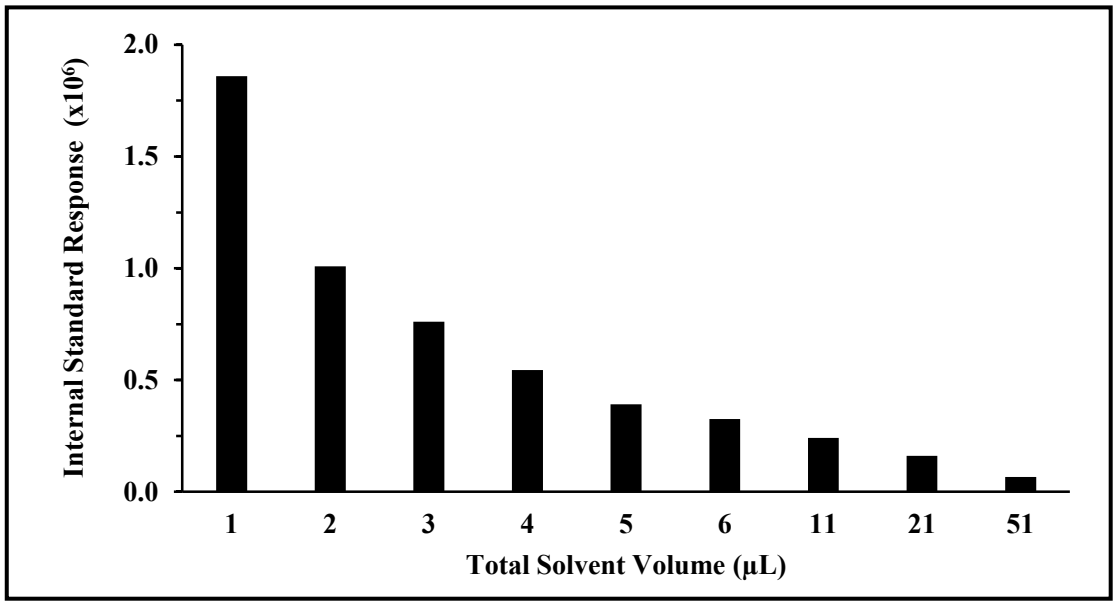

Figure A3-3: Effect of residual solvent volume on analyte response (peak area). Internal standard (1,2-dibromopropane; 1,2-DBP) was used as a representative of trihalomethanes (THMs); samples contained internal standard (final concentration of 25 µg m^{-3}) added as 1 µL with additional methanol (0 to 50 µL) added.

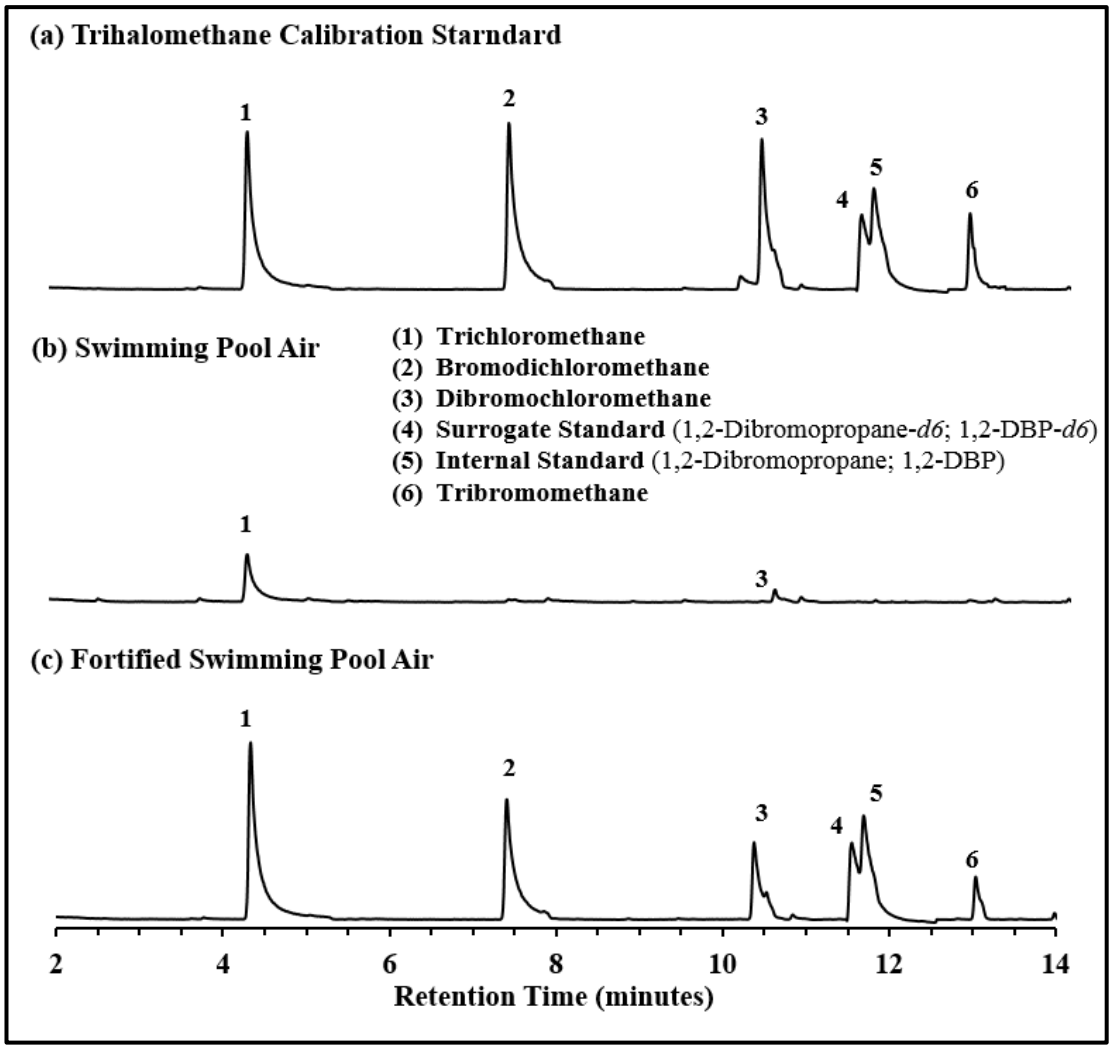

Figure A3-4: Chromatographic separation of four trihalomethanes (THMs), the surrogate standard (1,2-dibromopropane-d6) and internal standard (1,2-dibromopropane) for samples of **(a)** flushed vial fortified with THMs, surrogate standard and internal standard (final concentration of 100 µg m^{-3} each, added as 1 µL in methanol), **(b)** swimming pool air, and **(c)** swimming pool air fortified with THMs, surrogate standard and internal standard (final concentration of 100 µg m^{-3} each, added as 1 µL in methanol). Total ion chromatograms were collected in selected ion monitoring (SIM) mode on a ZB-5MS column under conditions described in **Section 3.2.3.3**.

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APPENDIX 4

Table A4-1: Analytical methods for the analysis of disinfection by-products (DBPs) and general water quality parameters employed in this study.

Parameter or DBP Class	Analytical Method	Quenching Agent	Reference
Trihalomethanes (including Iodo-THMs)	HS-SPME GC-EI-MS	Sodium Sulfite	(Allard et al., 2012)
Haloacetic Acids	HS-SPME GC-EI-MS	Sodium Sulfite	(Sa et al., 2012; Sarrión et al., 2000)
Haloketones Haloacetaldehydes	LLE GC-EI-MS	Sodium Sulfite	(US EPA, 1995)
Halonitromethanes Haloacetonitriles Haloacetamides	LLE GC-EI-MS	Ammonium Chloride	(Carter et al., 2019)
N-Nitrosamines	SPE GC-CI-MS	Ascorbic Acid	(Charrois et al., 2004)
Total Nitrogen Non-Purgeable Organic Carbon	High Temperature Combustion-NDIR	Sodium Sulfite	(APHA, 1988)
Free & Total Chlorine Equivalent Concentrations	DPD Colorimetric	N/A	(HACH, 2017)
pH Temperature Conductivity Dissolved Oxygen	HQ40D Portable Multi Meter	N/A	(HACH, 2015a, 2015b, 2015c)
$SUVA_{254}$	US EPA 415.3*	N/A	(US EPA, 2009)
Bromide	IC	Sodium Sulfite	(Salhi and Von Gunten, 1999)

*Calculated based on non-purgeable organic carbon. **CI**: Chemical Ionisation. **DPD**: *N, N*-Diethyl-*p*-phenylenediamine. **EI**: Electron ionisation. **GC**: Gas chromatography. **HS**: Headspace. **IC**: Ion chromatography. **LLE**: Liquid-liquid extraction. **MS**: Mass spectrometry. **N/A**: Not applicable. **NDIR:** Non-dispersive infrared. **SPE**: Solid-phase extraction. **SPME**: Solid-phase micro-extraction. **$SUVA_{254}$**: Specific ultraviolet absorbance at 254 nm.

Table A4-2: Summary of general water quality parameters of all waters investigated. Number of swimmers refers to those present at the time of water collection. Samples A5, B3, C2, D3, E4 and F4 represent the mains water at the given facility.

Sample Code	Collection Time	Number of Swimmers	Temp.	pH	Free Chlorine	Total Chlorine	Conductivity	Dissolved Oxygen	NPOC	Total Nitrogen	$SUVA_{254}$
	-	# people	°C	-	mg L^{-1}	mg L^{-1}	µS cm^{-1}	mg L^{-1}	mg L^{-1}	mg L^{-1}	L mgC^{-1} m^{-1}
A1	10.00	42	26.3	7.03	1.68	2.06	6130	7.72	4.22	9.84	0.15
A2	10.20	11	32.8	6.77	2.70	3.40	1447	6.43	6.88	2.59	0.16
A3	10.40	20	29.4	7.18	3.28	3.58	2590	6.93	4.26	4.65	0.19
A4	10.50	3	33.8	7.29	2.98	3.22	1179	6.54	3.05	2.72	0.28
A5	11.15	-	20.8	8.06	0.07	0.09	218	4.97	0.41	<LOD	1.36
B1	13.20	2	32.2	7.36	2.78	3.28	9350	6.38	8.94	15.63	0.23
B2	12.55	2	27.5	6.85	3.08	4.18	6490	7.34	10.40	26.00	0.55
B3	13.30	-	20.5	8.23	0.31	0.34	598	8.72	0.75	0.33	1.72
C1	14.20	8	28.6	7.39	1.50	1.74	2380	6.84	86.51	40.17	0.08
C2	14.30	-	22.8	8.30	0.48	0.55	374	8.60	0.99	0.32	0.69
D1	16.55	32	27.0	7.19	1.82	3.34	1178	7.87	15.43	10.87	0.45
D2	17.05	2	33.1	6.50	1.46	3.42	1380	6.59	27.83	8.60	0.42
D3	17.30	-	25.0	7.79	0.93	1.00	725	7.24	1.62	0.47	1.35
E1	18.25	12	27.5	7.43	5.16	5.96	1680	7.65	7.38	1.71	0.46
E2	18.05	7	29.8	7.71	5.68	7.76	2560	6.84	13.59	2.45	0.58
E3	18.50	5	34.0	7.99	3.52	6.16	2270	6.51	29.31	3.97	0.52
E4	18.40	-	26.0	7.90	0.75	0.92	723	7.62	1.63	0.37	1.49
F1	20.10	0	26.0	7.50	2.92	3.40	2930	7.26	6.91	2.92	0.33
F2	20.15	0	29.0	7.36	2.68	3.12	4750	6.84	6.62	5.21	0.27
F3	20.20	0	28.9	7.24	2.66	2.98	1092	6.69	4.51	1.25	0.39
F4	20.25	-	23.0	7.48	0.84	1.01	691	8.37	1.75	0.42	1.67

LOD: Limit of detection. **NPOC**: Non-purgeable organic carbon. **$SUVA_{254}$**: Specific ultraviolet absorbance at 254 nm (calculated based on NPOC). **Temp.**: Temperature.

Table A4-3: Summary of operational parameters of the swimming pools and spas investigated. All pools/spas were filled with disinfected distributed drinking water (mains water) and were independently treated. Turnover Rate is displayed as "L sec^{-1} (hours taken for one complete turnover)".

Pool Code	Pool Type	Disinfectant	Secondary Treatment	Water Volume (kL)		Filter Media	Turnover Rate	Additional Chemicals Used
				Swimmer Zone	Total			
A1	25 m Lap	Chlorine gas	UV	678	750	Sand	139 (1.5)	Soda ash, $NaHCO_3$, $CaCl_2$, HCl
A2	Hydrotherapy	Chlorine gas	UV	70	80	Perlite	22 (1.0)	Soda ash, $NaHCO_3$, $CaCl_2$, HCl
A3	Leisure	Chlorine gas	UV	615	1000	Perlite	159 (1.75)	Soda ash, $NaHCO_3$, $CaCl_2$, HCl
A4	Spa	Chlorine gas	UV	10	20	Perlite	22 (0.25)	Soda ash, $NaHCO_3$, $CaCl_2$, HCl
B1	Hydrotherapy	Chlorine gas	UV	63	110	Sand	115 (0.27)	Soda ash
B2	25 m Lap	Chlorine gas	-	550	941	Sand	95 (2.75)	Soda ash
C1	Training Pool	BCDMH	-	980	Unknown	Sand	Unknown	Soda ash, $NaHCO_3$, HCl
D1	50m Lap	Chlorine gas	UV	812.5	850	Perlite	94 (2.5)	Soda ash, $NaHCO_3$, HCl
D2	Spa	Chlorine gas	UV	10	10	Perlite	69 (2.4)	Soda ash, $NaHCO_3$, HCl
E1	50m Lap	Chlorine gas	-	NP	884	Sand	NP	$Al_2(SO_4)_3$, $NaHCO_3$, HCl
E2	Leisure	Chlorine gas	-	NP	197.5	Sand	NP	$Al_2(SO_4)_3$, $NaHCO_3$, HCl
E3	Spa	Chlorine gas	-	NP	5.7	Cartridge	NP	$Al_2(SO_4)_3$, $NaHCO_3$, HCl
F1	25m Lap	Sodium Hypochlorite	UV	411.25	456	Sand	93 (1.36)	HCl
F2	Leisure	Sodium Hypochlorite	UV	100	125	Sand	56 (0.62)	HCl
F3	Spa	Sodium Hypochlorite	UV	5.3	6.3	Sand	14 (0.13)	-

BCDMH: Bromochlorodimethylhydantoin. **NP**: Not provided. **Soda Ash**: Sodium carbonate. **UV**: Ultraviolet irradiation.

Table A4-4: Concentrations of trihalomethanes (THMs) measured in all waters investigated. Concentrations are displayed in "µg L^{-1} (µM)". Samples A5, B3, C2, D3, E4 and F4 represent the mains water at the given facility.

	Trichloromethane	Bromodichloromethane	Dibromochloromethane	Tribromomethane
Limit of Detection	0.2 (0.001)	0.5 (0.003)	0.5, (0.002)	0.2, (0.001)
A1	24 (0.194)	1.1 (0.006)	<LOD	<LOD
A2	27 (0.229)	1.4 (0.008)	<LOD	<LOD
A3	33 (0.280)	3.2 (0.020)	<LOD	<LOD
A4	14 (0.118)	1.2 (0.007)	<LOD	<LOD
A5	<LOD	<LOD	1.9 (0.009)	3.5 (0.014)
B1	27 (0.230)	3.6 (0.022)	<LOD	<LOD
B2	7.7 (0.064)	5.4 (0.033)	0.7 (0.004)	<LOD
B3	<LOD	1.7 (0.011)	8.6 (0.041)	30 (0.119)
C1	<LOD	<LOD	2.4 (0.011)	132 (0.523)
C2	<LOD	3.4 (0.021)	12 (0.058)	26 (0.103)
D1	58 (0.487)	2.2 (0.013)	0.4 (0.002)	0.3 (0.001)
D2	17 (0.145)	<LOD	<LOD	<LOD
D3	5.6 (0.047)	13 (0.076)	36 (0.171)	30 (0.120)
E1	67 (0.561)	3.3 (0.020)	0.7 (0.003)	0.3 (0.001)
E2	96 (0.807)	2.9 (0.018)	0.4 (0.002)	<LOD
E3	60 (0.503)	2.9 (0.018)	0.4 (0.002)	0.2 (0.001)
E4	<LOD	12 (0.073)	35 (0.167)	35 (0.140)
F1	56 (0.465)	5.0 (0.030)	1.1 (0.005)	<LOD
F2	15 (0.124)	3.8 (0.023)	0.6 (0.003)	<LOD
F3	35 (0.289)	4.3 (0.027)	1.1 (0.005)	<LOD
F4	10 (0.086)	27 (0.168)	49 (0.234)	17 (0.067)

Table A4-5: Concentrations of haloacetic acids (HAAs) measured in all waters investigated. Concentrations are displayed in "µg L^{-1}, (µM)". Samples A5, B3, C2, D3, E4 and F4 represent the mains water at the given facility.

	CAA	BAA	DCAA	TCAA	BCAA	DBAA	BDCAA	DBCAA	TBAA
LOD	6.4 (0.068)	4.4 (0.032)	2.90 (0.023)	0.1 (0.001)	2.6 (0.015)	2.4 (0.011)	0.1 (0.011)	0.2 (0.001)	-
A1	17 (0.176)	<LOD	99 (0.770)	120 (0.734)	<LOD	<LOD	0.34 (0.002)	6.3 (0.025)	-
A2	33 (0.344)	<LOD	183 (1.417)	200 (1.22)	3.3 (0.019)	<LOD	1.3 (0.006)	4.5 (0.018)	-
A3	20 (0.211)	<LOD	109 (0.844)	113 (0.691)	<LOD	<LOD	1.0 (0.005)	5.3 (0.021)	-
A4	143 (1.510)	<LOD	154 (1.192)	87 (0.531)	4.6 (0.026)	<LOD	1.7 (0.008)	4.2 (0.017)	-
A5	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD	3.0 (0.012)	-
B1	46 (0.487)	<LOD	135 (1.043)	142 (0.869)	<LOD	<LOD	0.6 (0.003)	7.1 (0.028)	-
B2	37 (0.389)	<LOD	139 (1.074)	104 (0.635)	17 (0.095)	<LOD	19 (0.092)	16 (0.064)	-
B3	<LOD	<LOD	<LOD	0.28 (0.002)	<LOD	3.5 (0.016)	0.6 (0.003)	13 (0.053)	-
C1	<LOD	<LOD	<LOD	0.3 (0.002)	<LOD	6.2 (0.029)	0.5 (0.003)	7.7 (0.031)	-
C2	<LOD	<LOD	<LOD	0.3 (0.002)	<LOD	2.8 (0.013)	0.5 (0.002)	10 (0.0400)	-
D1	37 (0.395)	<LOD	123 (0.950)	383, (2.34)	4.1 (0.024)	<LOD	9.8 (0.047)	8.0 (0.032)	-
D2	191 (2.016)	12 (0.085)	170 (1.316)	479 (2.93)	3.7 (0.021)	<LOD	6.3 (0.030)	<LOD	-
D3	<LOD	<LOD	9.3 (0.072)	5.0 (0.031)	4.6 (0.026)	6.2 (0.028)	4.2 (0.020)	31 (0.124)	-
E1	47 (0.500)	<LOD	131 (1.012)	95 (0.582)	9.8 (0.057)	<LOD	13 (0.0600)	12 (0.047)	-
E2	86 (0.905)	<LOD	200 (1.553)	168 (1.02)	12 (0.067)	<LOD	17 (0.082)	18 (0.0700)	-
E3	266 (2.818)	<LOD	173 (1.338)	93 (0.568)	9.5 (0.055)	<LOD	6.5 (0.031)	17 (0.068)	-
E4	<LOD	<LOD	<LOD	0.5 (0.003)	<LOD	4.1 (0.019)	0.80 (0.004)	16 (0.065)	-
F1	39 (0.416)	4.8 (0.035)	79 (0.609)	39 (0.240)	4.7 (0.027)	<LOD	1.7 (0.008)	5.6 (0.022)	-
F2	42.33 (0.448)	6.7 (0.048)	72 (0.556)	41 (0.251)	2.9 (0.017)	<LOD	0.97 (0.005)	5.9 (0.024)	-
F3	24 (0.248)	<LOD	53 (0.408)	23 (0.142)	10 (0.057)	<LOD	4.2 (0.020)	12 (0.046)	-
F4	<LOD	<LOD	3.9 (0.030)	1.3 (0.008)	5.2 (0.030)	6.6 (0.031)	5.0 (0.024)	16 (0.064)	-

LOD: Limit of detection. **CAA**: Chloroacetic acid. **BAA**: Bromoacetic acid. **DCAA**: Dichloroacetic acid. **DBAA**: Dibromoacetic acid. **BCAA**: Bromochloroacetic acid. **BDCAA**: Bromodichloroacetic acid. **DBCAA**: Dibromochloroacetic acid. **TCAA**: Trichloroacetic acid. **TBAA**: Tribromoacetic acid.

Table A4-6: Concentrations of haloketones (HKs) measured in all waters investigated. Concentrations are displayed in "µg L^{-1}, (µM)". Samples A5, B3, C2, D3, E4 and F4 represent the mains water at the given facility.

	Chloropropanone	1,1-Dichloropropanone	1,1,1-Trichloropropanone	1,3-Dichloropropanone	1,1,3,3-Tetrachloropropanone
LOD	2.28 (0.0247)	0.7 (0.005)	0.2 (0.001)	2.5 (0.020)	0.2 (0.001)
A1	<LOD	0.8 (0.006)	9.9 (0.062)	<LOD	0.3 (0.002)
A2	2.3 (0.025)	0.7 (0.006)	6.7 (0.042)	<LOD	0.3 (0.002)
A3	2.3 (0.025)	<LOD	5.5 (0.034)	<LOD	0.2 (0.001)
A4	2.3 (0.025)	<LOD	2.3 (0.014)	<LOD	<LOD
A5	<LOD	<LOD	<LOD	<LOD	<LOD
B1	3.2 (0.035)	1.0 (0.008)	10 (0.062)	4.0 (0.032)	0.4 (0.002)
B2	3.0 (0.032)	0.9 (0.007)	5.7 (0.035)	3.3 (0.026)	0.4 (0.002)
B3	<LOD	<LOD	<LOD	<LOD	<LOD
C1	<LOD	<LOD	<LOD	<LOD	<LOD
C2	<LOD	<LOD	<LOD	<LOD	<LOD
D1	3.5 (0.038)	0.8 (0.006)	6.5 (0.040)	3.0 (0.024)	0.5 (0.003)
D2	7.0 (0.075)	1.8 (0.014)	13 (0.079)	4.3 (0.034)	1.6 (0.008)
D3	<LOD	<LOD	<LOD	<LOD	<LOD
E1	6.9 (0.075)	<LOD	2.4 (0.015)	<LOD	0.2 (0.001)
E2	9.2 (0.099)	0.9 (0.007)	4.8 (0.030)	<LOD	0.5 (0.002)
E3	10 (0.108)	1.1 (0.009)	2.4 (0.015)	<LOD	0.8 (0.004)
E4	<LOD	<LOD	<LOD	<LOD	<LOD
F1	3.8 (0.042)	<LOD	4.8 (0.030)	<LOD	0.2 (0.001)
F2	2.4 (0.026)	<LOD	7.1 (0.044)	<LOD	0.3 (0.002)
F3	3.2 (0.035)	<LOD	2.8 (0.018)	<LOD	<LOD
F4	<LOD	<LOD	0.7 (0.004)	<LOD	<LOD

LOD: Limit of detection.

Table A4-7: Concentrations of haloacetaldehydes (HALs) measured in all waters investigated. Concentrations are displayed in "µg L^{-1}, (µM)". Samples A5, B3, C2, D3, E4 and F4 represent the mains water at the given facility.

	DCAL	DBAL	BCAL	BDCAL	DBCAL	TCAL	TBAL
LOD	2.1 (0.019)	1.5 (0.008)	1.5 (0.009)	3.0 (0.016)	1.3 (0.006)	1.3 (0.008)	8.3 (0.030)
A1	19 (0.168)	<LOD	<LOD	9.5 (0.050)	<LOD	471 (2.85)	<LOD
A2	35 (0.313)	<LOD	<LOD	16 (0.085)	<LOD	824 (4.99)	<LOD
A3	18 (0.164)	<LOD	<LOD	15 (0.076)	<LOD	400 (2.42)	<LOD
A4	13 (0.115)	<LOD	<LOD	12 (0.065)	<LOD	278 (1.68)	<LOD
A5	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD
B1	33 (0.293)	<LOD	<LOD	19 (0.101)	<LOD	1313 (7.94)	<LOD
B2	9.5 (0.085)	<LOD	<LOD	46 (0.241)	<LOD	437 (2.64)	<LOD
B3	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD
C1	<LOD	2.5 (0.013)	<LOD	<LOD	<LOD	<LOD	20, (0.069)
C2	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD
D1	16 (0.140)	<LOD	<LOD	13 (0.070)	<LOD	1026 (6.21)	<LOD
D2	12 (0.104)	<LOD	2.3, (0.0146)	15 (0.080)	<LOD	1136 (6.87)	<LOD
D3	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD
E1	2.7 (0.024)	<LOD	<LOD	13 (0.067)	<LOD	260 (1.57)	<LOD
E2	7.6 (0.068)	<LOD	<LOD	19 (0.097)	<LOD	600 (3.70)	<LOD
E3	12 (0.110)	<LOD	3.0 (0.019)	23 (0.117)	<LOD	1008 (6.10)	<LOD
E4	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD
F1	6.2 (0.055)	<LOD	<LOD	19 (0.098)	<LOD	504 (3.05)	<LOD
F2	13 (0.112)	<LOD	<LOD	26 (0.134)	<LOD	662 (4.01)	<LOD
F3	3.9 (0.035)	<LOD	<LOD	30 (0.159)	<LOD	202 (1.22)	<LOD
F4	<LOD	<LOD	<LOD	11 (0.058)	3.0 (0.013)	2.5 (0.015)	<LOD

LOD: Limit of detection. **DCAL**: Dichloroacetaldehyde. **DBAL**: Dibromoacetaldehyde. **BCAL**: Bromochloroacetaldehyde. **BDCAL**: Bromodichloroacetaldehyde. **DBCAL**: Dibromochloroacetaldehyde. **TCAL**: Trichloroacetaldehyde. **TBAL**: Tribromoacetaldehyde.

Table A4-8: Concentrations of haloacetonitriles (HANs) measured in all waters investigated. Concentrations are displayed in "µg L^{-1}, (µM)". Samples A5, B3, C2, D3, E4 and F4 represent the mains water at the given facility.

	CAN	BAN	DCAN	DBAN	BCAN	BDCAN	DBCAN	TCAN	TBAN
LOD	0.3 (0.004)	0.6 (0.005)	0.5 (0.004)	0.4 (0.002)	0.3 (0.002)	0.3 (0.002)	0.3 (0.001)	0.5 (0.003)	0.2 (0.001)
A1	0.9 (0.012)	<LOD	20 (0.182)	<LOD	0.5, (0.003)	<LOD	<LOD	<LOD	<LOD
A2	1.1 (0.014)	<LOD	24.85 (0.226)	<LOD	0.9 (0.006)	<LOD	<LOD	<LOD	<LOD
A3	0.7 (0.009)	1.4 (0.011)	15 (0.138)	<LOD	0.8 (0.005)	<LOD	<LOD	<LOD	<LOD
A4	0.4 (0.005)	<LOD	7.1 (0.065)	<LOD	01.0 (0.006)	<LOD	<LOD	<LOD	<LOD
A5	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD
B1	1.9 (0.025)	<LOD	38 (0.349)	<LOD	1.3 (0.008)	<LOD	<LOD	<LOD	<LOD
B2	0.7 (0.009)	<LOD	14 (0.126)	0.4 (0.002)	2.3 (0.015)	<LOD	<LOD	<LOD	<LOD
B3	<LOD	<LOD	<LOD	1.5 (0.008)	<LOD	<LOD	<LOD	<LOD	<LOD
C1	<LOD	6.7 (0.056)	<LOD	7.9 (0.040)	<LOD	<LOD	<LOD	<LOD	<LOD
C2	<LOD	<LOD	<LOD	1.1 (0.006)	<LOD	<LOD	<LOD	<LOD	<LOD
D1	1.2 (0.016)	<LOD	16 (0.148)	<LOD	0.6 (0.004)	<LOD	<LOD	<LOD	<LOD
D2	1.8 (0.024)	<LOD	30 (0.269)	<LOD	0.5 (0.003)	<LOD	<LOD	<LOD	<LOD
D3	<LOD	<LOD	<LOD	3.1 (0.016)	0.6 (0.004)	<LOD	<LOD	<LOD	<LOD
E1	0.7 (0.009)	<LOD	4.1 (0.037)	<LOD	0.4 (0.002)	<LOD	<LOD	<LOD	<LOD
E2	1.5 (0.020)	<LOD	19 (0.173)	<LOD	1.2 (0.008)	<LOD	<LOD	<LOD	<LOD
E3	1.9 (0.025)	<LOD	24 (0.215)	<LOD	1.0 (0.007)	<LOD	<LOD	<LOD	<LOD
E4	<LOD	<LOD	<LOD	2.6 (0.013)	0.5 (0.003)	<LOD	<LOD	<LOD	<LOD
F1	1.1 (0.014)	<LOD	14 (0.128)	<LOD	0.9 (0.006)	<LOD	<LOD	<LOD	<LOD
F2	1.4 (0.019)	<LOD	19 (0.170)	<LOD	1.2 (0.008)	<LOD	<LOD	<LOD	<LOD
F3	0.4 (0.006)	<LOD	5.8 (0.053)	0.8 (0.004)	1.9 (0.013)	<LOD	<LOD	<LOD	<LOD
F4	<LOD	<LOD	0.9 (0.008)	3.4 (0.017)	2.0 (0.013)	<LOD	<LOD	<LOD	<LOD

LOD: Limit of detection. **CAN**: Chloroacetonitrile. **BAN**: Bromoacetonitrile. **BCAN**: Bromochloroacetonitrile. **DCAN**: Dichloroacetonitrile. **DBAN**: Dibromoacetonitrile. **BDCAN**: Bromodichloroacetonitrile. **DBCAN**: Dibromochloroacetonitrile. **TCAN**: Trichloroacetonitrile. **TBAN**: Tribromoacetonitrile.

Table A4-9: Concentrations of haloacetamides (HAAms) measured in all waters investigated. Concentrations are displayed in "µg L^{-1}, (µM)". Samples A5, B3, C2, D3, E4 and F4 represent the mains water at the given facility.

	DCAAm	DBAAm	BCAAm	BDCAAm	DBCAAm	TCAAm	TBAAm
LOD	1.0 (0.008)	3.5 (0.016)	4.1 (0.024)	0.2 (0.001)	0.3 (0.001)	0.3 (0.002)	0.4 (0.002)
A1	12 (0.094)	<LOD	5.0 (0.029)	<LOD	<LOD	0.9 (0.006)	<LOD
A2	10 (0.078)	<LOD	<LOD	0.3 (0.001)	<LOD	2.5 (0.016)	<LOD
A3	17 (0.132)	<LOD	<LOD	<LOD	<LOD	1.4 (0.009)	<LOD
A4	13 (0.104)	<LOD	<LOD	<LOD	<LOD	0.5 (0.003)	<LOD
A5	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD	<LOD
B1	56 (0.440)	<LOD	<LOD	<LOD	<LOD	2.4 (0.015)	<LOD
B2	7.1 (0.055)	<LOD	<LOD	5.2 (0.025)	1.1 (0.004)	49 (0.299)	<LOD
B3	<LOD	3.7 (0.017)	<LOD	<LOD	<LOD	<LOD	<LOD
C1	<LOD	736 (3.39)	2.3 (0.013)	<LOD	145 (0.579)	<LOD	24 (0.080)
C2	<LOD	4.9 (0.023)	<LOD	<LOD	<LOD	<LOD	<LOD
D1	15 (0.117)	<LOD	<LOD	3.5 (0.017)	0.4 (0.002)	65 (0.401)	<LOD
D2	17 (0.134)	<LOD	<LOD	3.2 (0.016)	0.5 (0.002)	46 (0.282)	<LOD
D3	1.4 (0.011)	9.3 (0.043)	3.6 (0.021)	<LOD	<LOD	<LOD	<LOD
E1	8.9 (0.069)	<LOD	<LOD	3.0 (0.014)	0.9 (0.004)	27 (0.165)	<LOD
E2	37 (0.293)	<LOD	2.8 (0.016)	3.7 (0.018)	1.1 (0.005)	25 (0.156)	<LOD
E3	28 (0.218)	<LOD	2.7 (0.016)	3.8 (0.018)	1.7 (0.007)	52 (0.318)	<LOD
E4	1.3 (0.011)	9.6 (0.045)	4.3 (0.025)	<LOD	<LOD	<LOD	<LOD
F1	43 (0.334)	<LOD	2.9 (0.017)	0.3 (0.002)	<LOD	5.1 (0.031)	<LOD
F2	46 (0.356)	<LOD	2.9 (0.017)	3.3 (0.016)	1.0 (0.004)	26 (0.158)	<LOD
F3	23 (0.181)	<LOD	4.3 (0.025)	0.2 (0.001)	<LOD	1.0 (0.006)	<LOD
F4	<LOD	2.6 (0.012)	<LOD	<LOD	<LOD	<LOD	<LOD

LOD: Limit of detection. **DCAAm**: Dichloroacetamide. **DBCAAm**: Dibromochloroacetamide. **BCAAm**: Bromochloroacetamide. **BDCAAm**: Bromodichloroacetamide. **DBCAAm**: Dibromochloroacetamide. **TCAAm**: Trichloroacetamide. **TBAAm**: Tribromoacetamide.

Table A4-10: Concentrations of *N*-nitrosamines measured in all waters investigated. Concentrations are displayed in "ng L^{-1}, (nM)". Samples A5, B3, C2, D3, E4 and F4 represent the mains water at the given facility.

	NDMA	NEMA	NDEA	NDPA	NDBA	NPIP	NPYR	NMOR
LOD	1.9 (0.025)	1.1 (0.013)	3.9 (0.038)	2.0 (0.016)	2.7 (0.017)	1.9 (0.017)	2.2 (0.021)	0.9, (0.008)
A1	0.9 (0.012)	<LOD	<LOD	2.2 (0.017)	11 (0.072)	5.4 (0.047)	<LOD	8.9 (0.077)
A2	2.5 (0.034)	<LOD	<LOD	<LOD	32 (0.203)	2.9 (0.025)	<LOD	13 (0.109)
A3	1.4 (0.019)	<LOD	<LOD	2.4 (0.019)	13 (0.081)	3.6 (0.032)	<LOD	7.8 (0.068)
A4	0.5 (0.007)	<LOD	<LOD	2.7 (0.020)	22 (0.142)	<LOD	<LOD	5.7 (0.049)
A5	<LOD	<LOD	<LOD	<LOD	16 (0.104)	4.2 (0.037)	<LOD	1.7, (0.014)
B1	2.4 (0.032)	<LOD	<LOD	<LOD	39 (0.247)	5.7 (0.050)	<LOD	18 (0.154)
B2	49 (0.655)	<LOD	<LOD	3.1 (0.024)	172 (1.09)	3.0 (0.026)	2.3 (0.023)	16 (0.141)
B3	<LOD	<LOD	9.9 (0.097)	2.1 (0.016)	3.4 (0.022)	2.1 (0.018)	2.2 (0.022)	2.2 (0.019)
C1	<LOD	24 (0.268)	9.2 (0.090)	2.1 (0.016)	201 (1.27)	2.1 (0.018)	21 (0.209)	<LOD
C2	<LOD	<LOD	<LOD	<LOD	<LOD	3.9 (0.034)	<LOD	2.0 (0.018)
D1	65 (0.879)	<LOD	<LOD	<LOD	386 (2.44)	4.2 (0.036)	<LOD	30 (0.254)
D2	456 (0.618)	<LOD	4.5 (0.045)	7.7 (0.059)	1093 (6.91)	5.9 (0.052)	2.9 (0.029)	22 (0.193)
D3	<LOD	<LOD	<LOD	<LOD	4.3 (0.027)	1.4 (0.012)	<LOD	1.7 (0.015)
E1	27 (0.362)	<LOD	<LOD	4.4 (0.034)	99 (0.627)	2.0 (0.017)	<LOD	12 (0.103)
E2	25 (0.341)	<LOD	<LOD	<LOD	179 (1.13)	1.8 (0.016)	<LOD	19 (0.165)
E3	52 (0.697)	<LOD	<LOD	<LOD	583 (3.68)	2.4 (0.021)	2.3 (0.023)	29 (0.251)
E4	<LOD	<LOD	4.9 (0.048)	2.7 (0.021)	4.1 (0.026)	3.1 (0.027)	<LOD	2.0 (0.017)
F1	5.1 (0.068)	<LOD	<LOD	4.8 (0.037)	11 (0.068)	2.7 (0.023)	<LOD	21 (0.179)
F2	26 (0.347)	<LOD	<LOD	2.4 (0.018)	18 (0.115)	3. (0.026)	2.4 (0.024)	9.1 (0.078)
F3	1.0 (0.013)	<LOD	<LOD	4.0 (0.031)	9.9 (0.062)	1.9 (0.016)	<LOD	5.2 (0.045)
F4	<LOD	<LOD	<LOD	2.1 (0.016)	3.5 (0.022)	1.4 (0.012)	<LOD	1.3 (0.011)

LOD: Limit of detection. **NDBA**: *N*-Nitrosodi-*n*-butylamine. **NDMA**: *N*-Nitrosodimethylamine. **NDPA**: *N*-Nitrosodipropylamine. **NDEA**: *N*-Nitrosodiethylamine. **NEMA**: *N*-Nitrosoethylmethylamine. **NMOR**: *N*-Nitrosomorpholine. **NPIP**: *N*-Nitrosopiperidine. **NPYR**: *N*-Nitrosopyrrolidine.

Table A4-11: Summary of calculated cytotoxicity values of investigated waters based on the measured disinfection by-product concentrations and their associated C_{50} values. All haloketones, bromodichloroacetonitrile, dibromochloroacetonitrile, tribromoacetonitrile, as well as most of the investigated *N*-nitrosamines (excluding *N*-nitrosomorpholine), were excluded from cytotoxicity evaluation as C_{50} values do not currently exist for these compounds. Samples A5, B3, C2, D3, E4 and F4 represent the mains water at the given facility.

	Total	THMs	HAAs	HALs	HANs	HNMs	HAAms	NMOR
A1	548102	21	754	541868	3698	-	1754	6.9
A2	954767	24	1248	948475	4817	-	193	9.8
A3	469694	31	774	462192	6618	-	73	6.1
A4	325955	13	2378	321574	1931	-	55	4.4
A5	65	5.1	58	-	-	-	-	1.3
B1	1510637	26	1249	1501670	7424	18	236	14
B2	519287	10	1466	509009	4828	-	3961	13
B3	4414	39	292	-	2684	-	1397	1.7
C1	455230	134	205	22007	31351	823	400710	-
C2	4091	38	223	-	1974	-	1854	1.6
D1	1177326	52	1849	1169641	3232	-	2528	23
D2	1307714	15	3962	1295872	5412	-	2436	17
D3	11436	74	747	-	5867	-	4748	1.3
E1	303328	61	1392	298324	1044	-	2498	9.3
E2	697677	86	2308	686828	4250	-	4191	15
E3	1168259	54	4351	1154198	4915	-	4719	23
E4	10447	73	359	-	4922	-	5091	1.6
F1	582773	52	853	577383	3152	-	1317	16
F2	769161	15	879	760186	4131	-	3944	7.1
F3	243392	33	754	237064	3883	-	1654	4.1
F4	17271	84	451	8145	7613	-	977	1.0

HAAms: Haloacetamides. **HAAs:** Haloacetic acids. **HALs:** Haloacetaldehydes. **HANs:** Haloacetonitriles. **HNMs:** Halonitromethanes. **NMOR:** *N*-Nitrosomorpholine. **THMs:** Trihalomethanes.

Table A4-12: Evaluation of correlations between the investigated disinfection by-product classes and operating, general water quality or sampling parameters. Values are displayed as "Spearman's correlation rank (p-value)" and rounded to 3 significant figures.

	Total DBPs	Trihalomethanes	Haloacetic Acids	Haloketones	Haloacetaldehydes	Haloacetontriles	Halonitromethanes	Haloacetamides	*N*-Nitrosamines
Time of Sampling	0.093 (0.742)	0.396 (0.143)	-0.157 (0.576)	0.136 (0.63)	-0.007 (0.98)	-0.204 (0.467)	-0.139 (0.622)	0.525 (0.054)	0.221 (0.428)
Conductivity	-0.021 (0.940)	-0.186 (0.508)	-0.289 (0.296)	0.136 (0.630)	0.143 (0.612)	0.296 (0.283)	0.338 (0.218)	0.143 (0.612)	-0.007 (0.98)
Dissolved Oxygen	-0.297 (0.282)	0.168 (0.548)	-0.265 (0.339)	-0.158 (0.575)	-0.312 (0.258)	-0.452 (0.091)	-0.339 (0.216)	0.004 (0.99)	0.079 (0.780)
Free Chlorine Equivalent Concentration	-0.043 (0.879)	0.318 (0.248)	0.214 (0.443)	0.189 (0.499)	-0.118 (0.676)	-0.125 (0.657)	-0.254 (0.362)	-0.093 (0.742)	-0.061 (0.830)
Total Volume	-0.064 (0.853)	0.136 (0.689)	-0.082 (0.811)	0.064 (0.853)	-0.036 (0.915)	-0.018 (0.958)	-0.100 (0.770)	0.118 (0.729)	0.136 (0.689)
Turnover Rate	0.027 (0.936)	0.091 (0.79)	0.041 (0.905)	0.246 (0.466)	0.191 (0.573)	0.333 (0.318)	0.301 (0.369)	0.141 (0.679)	0.137 (0.689)

Cytotoxicity formula:

$$\text{Calculated Cytotoxicity} = \frac{\text{Concentration Measured (M)}}{C_{50}\text{ Value (M)}} * 10^6$$

Example: Using trichloromethane detected in pool A1 (0.19427 µM) and its reported C_{50} value (0.00962 M) (Wagner and Plewa, 2017).

$$\text{Calculated Cytotoxicity}_{\text{Trichloromethane}} = \frac{0.19427 * 10^{-6}}{0.00962} * 10^6 = 20.19$$

Figure A4-1: Formula used for cytotoxicity evaluation and example calculation, based on previously published methods (e.g. Allard et al., 2015; Smith et al., 2010).

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APPENDIX 5

Table A5-1: Analytical methods for the analysis of disinfection by-products (DBPs) and general water quality parameters employed in this study.

Parameter or DBP Class	Analytical Method	Quenching Agent	Reference
Trihalomethanes	HS-SPME GC-EI-MS	Sodium Sulfite	(Allard et al., 2012)
Haloacetic Acids	LLE Derivatisation GC-EI-MS	Sodium Sulfite	(US EPA, 2003)
Haloketones Haloacetaldehydes	LLE GC-EI-MS	Sodium Sulfite	(US EPA, 1995)
Halonitromethanes Haloacetonitriles Haloacetamides	LLE GC-EI-MS	Ammonium Chloride	(Carter et al., 2019)
Total Nitrogen Non-Purgeable Organic Carbon	High Temperature Combustion-NDIR	Sodium Sulfite	(APHA, 1988)
pH Temperature Conductivity Dissolved Oxygen	HQ40D Portable Multi Meter	N/A	(HACH, 2015a, 2015b, 2015c)
Free & Total Chlorine Equivalent Concentrations	DPD Colorimetric	N/A	(HACH, 2017)

DPD: *N,N*-Diethyl-*p*-phenylenediamine. **EI**: Electron ionisation. **GC**: Gas chromatography. **HS**: Headspace. **IC**: Ion chromatography. **LLE**: Liquid-liquid extraction. **MS**: Mass spectrometry. **N/A**: Not applicable. **NDIR:** Non-dispersive infrared. **SPE**: Solid-phase extraction. **SPME**: Solid-phase microextraction.

Table A5-2: General parameters and target operating conditions of Pools A and B.

Parameter	Units	Pool A	Pool B
Type	-	20 m (4 lane) outdoor/covered leisure pool	50 m (10 lane) outdoor 'lap' pool
Temperature	°C	30	27
pH	-	7.2-7.8	7.2-7.8
Free Chlorine	mg L^{-1}	2.5-3	3-3.5
Cyanuric Acid	mg L^{-1}	NOT USED	20-50
Pool Volume (Swimming Volume)	L	275 000	1 800 000
Total Pool Volume (including pipes/tanks)	L	300 000	1 860 000
Turnover rate (hours)	hrs	1.5	3.5
Water through UV	%	100%	No UV
Backwash Volume*	L	4500	9000

*Backwashes are performed every 5 to 10 days in summer and every 3 weeks in winter.

Table A5-3: Frequency and purpose of addition of the chemicals used in Pools A and B.

Chemical	Pool A	Pool B	Purpose
Chlorine Gas	Daily*	Daily*	Disinfectant
Soda Ash (Sodium Carbonate)	Daily*	Daily*	Increase pH
Bicarb (Sodium Bicarbonate)	Daily*	Daily*	Increase pH
Calcium Chloride	Weekly	Weekly	Increases hardness
Hydrochloric Acid (15%)	Twice Weekly	Twice Weekly	Clean pipes/lines from carbonate build-up
Calcium Hypochlorite	When Required	When Required	Increase disinfectant
Isocyanuric Acid	NOT USED	Needs basis (monthly)	Chlorine stabiliser
Lanthanum Chloride	One off	One off	Phosphate remover

*Added Automatically.

Table A5-4: Summary of measured water parameters as investigated over the duration of the study in Pool A, Pool B and the filling water. Values presented as: "average (minimum-maximum)".

Parameter	Units	Pool A	Pool B	Filling Water
pH	-	7.3 (6.6-7.7)	7.3 (6.8-7.8)	7.6 (7.1-8.0)
Free Chlorine Equivalent Concentration	mg L^{-1}	3.5 (1.2-6.2)*	3.3 (1.8-6.6)	0.1 (nd-0.4)
Total Chlorine Equivalent Concentration	mg L^{-1}	4.1 (1.8-6.7)*	3.5 (1.9-7.1)	0.1 (nd-0.5)
Temperature	°C	29 (26-32)	27 (24-30)	21 (14-32)
Dissolved Oxygen Concentration	mg L^{-1}	7.8 (7.3-8.8)	8.1 (7.6-8.6)	8.3 (7.3-9.9)
Conductivity	mS cm^{-1}	2.6 (1.4-4.5)	1.6 (0.-2.7)	0.4 (0.3-0.5)
Non-Purgeable Organic Carbon Concentration	mg L^{-1}	14 (2.8-30)	6.3 (1.7-21)	2.8 (0.6-15)
Total Nitrogen Concentration	mg L^{-1}	3.4 (0.1-16)	7.3 (4.5-21)	0.1 (nd-0.4)

nd: not detected. ***** Excludes three concentrations measured on three successive occasions (days 1.5, 2 and 3) just after opening as the automated disinfectant dosage system on Pool A experienced technical issues. Concentrations of 26, 20 and 15 mg L^{-1} for free chlorine equivalents and 29, 22, 15 mg L^{-1} for total chlorine equivalents were measured on days 1.5, 2 and 3, respectively. **UV**: Ultraviolet irradiation.

Table A5-5: Summary of Spearman's correlation rank between: (i) the thirty-nine measured disinfection by-products (DBPs; on a molar basis), (ii) the measured general water parameters, and (iii) theoretical chronic cytotoxicity, for Pool A. Values are displayed as "Spearman's correlation rank (p-value)" and rounded to 3 significant figures. Values shown in **BOLD** were found to be moderately significantly correlated ($p<0.05$), while values including a '*' indicate a significantly strong correlation ($p<0.01$) was observed.

	HKs	HALs	HANs	HNMs	THMs	HAAs	Total DBPs	NPOC	TN	DO	Cond.	Temp.	pH	Free Chlorine	Total Chlorine
HALs	-0.158 (0.449)														
HANs	-0.124 (0.563)	**0.680 (<0.001)***													
HNMs	0.166 (0.471)	0.029 (0.902)	-0.104 (0.612)												
THMs	0.295 (0.152)	0.272 (0.189)	0.120 (0.535)	**0.494 (0.010)**											
HAAs	-0.223 (0.296)	-0.061 (0.778)	0.113 (0.568)	0.055 (0.789)	0.172 (0.362)										
Total DBPS	-0.152 (0.470)	-0.015 (0.945)	0.090 (0.644)	0.116 (0.573)	0.207 (0.264)	**0.982 (<0.001)***									
NPOC	0.268 (0.194)	**0.513 (0.009)***	**0.445 (0.015)**	-0.191 (0.350)	0.312 (0.088)	-0.081 (0.666)	-0.135 (0.462)								
TN	-0.240 (0.248)	0.172 (0.412)	0.134 (0.488)	-0.247 (0.233)	-0.278 (0.129)	**0.636 (<0.001)***	**0.664 (<0.001)***	0.144 (0.431)							
DO	-0.263 (0.324)	0.007 (0.978)	**0.583 (0.006)***	-0.284 (0.254)	-0.396 (0.068)	0.350 (0.111)	0.260 (0.232)	-0.274 (0.195)	-0.004 (0.986)						
Cond.	**0.526 (0.036)**	0.015 (0.957)	-0.171 (0.457)	-0.216 (0.390)	-0.416 (0.054)	-0.382 (0.079)	-0.380 (0.073)	0.023 (0.913)	**0.974 (<0.001)***	0.024 (0.910)					
Temp.	-0.261 (0.208)	0.384 (0.058)	**0.383 (0.040)**	0.153 (0.456)	-0.105 (0.575)	0.340 (0.061)	**-0.358 (0.044)**	0.109 (0.544)	**0.461 (0.008)**	0.016 (0.942)	0.163 (0.446)				
pH	-0.369 (0.070)	0.011 (0.959)	**-0.423 (0.022)**	-0.227 (0.264)	-0.216 (0.242)	0.022 (0.907)	0.044 (0.811)	-0.157 (0.382)	0.035 (0.849)	-0.115 (0.594)	0.197 (0.356)	-0.021 (0.910)			

Table A5-5 continued

	HKs	HALs	HANs	HNMs	THMs	HAAs	Total DBPs	NPOC	TN	DO	Cond.	Temp.	pH	Free Chlorine	Total Chlorine
Free Chlorine	0.341 (0.096)	-0.360 (0.077)	**-0.436 (0.018)**	-0.162 (0.428)	-0.047 (0.804)	0.130 (0.486)	0.126 (0.492)	0.026 (0.886)	-0.030 (0.869)	0.026 (0.904)	**0.423 (0.040)**	**-0.499 (0.003)**	-0.032 (0.862)		
Total Chlorine	0.395 (0.051)	-0.260 (0.209)	-0.336 (0.074)	-0.186 (0.362)	-0.048 (0.799)	0.030 (0.873)	0.001 (0.994)	0.181 (0.314)	0.059 (0.747)	0.003 (0.987)	**0.457 (0.025)**	**-0.393 (0.024)**	-0.126 (0.484)	**0.958 (<0.001)***	
Pool Entries	**0.522 (0.007)***	**0.398 (0.049)**	0.352 (0.061)	0.076 (0.712)	0.093 (0.618)	-0.354 (0.051)	-0.334 (0.062)	**0.614 (<0.001)***	0.256 (0.158)	-0.144 (0.502)	0.150 (0.483)	0.153 (0.395)	**-0.427 (0.013)**	0.173 (0.336)	0.303 (0.086)

Cond.: Conductivity. **DO**: Dissolved oxygen. **HAAs**: Haloacetic acids. **HALs**: Haloacetaldehydes. **HANs**: Haloacetonitriles. **HNMs**: Halonitromethanes. **NPOC**: Non-purgeable organic carbon. **Temp**.: Temperature. **THMs**: Trihalomethanes. **TN**: Total nitrogen. **Total DBPs**: Sum of all DBPs (on a molar basis).

Table A-6: Summary of Spearman's correlation rank between; (i) the thirty-nine measured disinfection by-products (DBPs; on a molar basis), (ii) the measured general water parameters, and (iii) the theoretical chronic cytotoxicity, for Pool B. Values are displayed as "Spearman's correlation rank (p-value)" and rounded to 3 significant figures. Values shown in **BOLD** were found to be moderately significantly correlated ($p<0.05$), while values including a '*' indicate a significantly strong correlation ($p<0.01$) was observed.

	HKs	HALs	HANs	HNMs	THMs	HAAs	Total DBPs	NPOC	TN	DO	Cond.	Temp.	pH	Free Chlorine	Total Chlorine
HALs	**0.762 (<0.001)***														
HANs	**0.619 (0.002)***	**0.710 (<0.001)***													
HNMs	0.385 (0.093)	-0.038 (0.872)	0.150 (0.506)												
THMs	**0.584 (0.003)***	0.378 (0.068)	0.264 (0.174)	-0.049 (0.828)											
HAAs	0.309 (0.151)	0.405 (0.055)	0.252 (0.205)	-0.144 (0.533)	0.285 (0.134)										
Total DBPS	0.380 (0.067)	**0.590 (0.002)**	**0.425 (0.024)**	-0.132 (0.559)	0.309 (0.097)	**0.937 (<0.001)***									
NPOC	-0.155 (0.593)	0.032 (0.881)	**0.463 (0.013)**	0.090 (0.013)	-0.161 (0.395)	-0.176 (0.353)	-0.192 (0.292)								
TN	**0.577 (0.003)***	**0.572 (0.003)***	0.363 (0.057)	-0.049 (0.830)	**0.613 (<0.001)***	**0.487 (0.007)***	**0.384 (0.033)**	-0.063 (0.738)							
DO	0.295 (0.286)	0.304 (0.271)	0.336 (0.147)	-0.053 (0.858)	0.080 (0.731)	0.104 (0.654)	0.317 (0.141)	-0.129 (0.559)	-0.173 (0.440)						
Cond.	**0.689 (0.004)***	0.404 (0.136)	-0.044 (0.855)	0.475 (0.086)	**0.456 (0.038)**	0.408 (0.067)	0.308 (0.152)	**-0.585 (0.003)***	**0.616 (0.002)***	-0.006 (0.979)					
Temp.	**0.425 (0.039)**	**0.409 (0.047)**	**0.516 (0.005)***	0.293 (0.186)	0.070 (0.714)	0.085 (0.657)	0.116 (0.526)	**0.527 (0.002)***	0.254 (0.168)	-0.297 (0.169)	-0.207 (0.344)				
pH	-0.245 (0.249)	0.097 (0.651)	-0.137 (0.485)	-0.032 (0.887)	-0.244 (0.193)	0.127 (0.504)	0.164 (0.369)	-0.089 (0.630)	-0.258 (0.161)	0.193 (0.379)	0.127 (0.565)	-0.141 (0.442)			

Table A5-6 continued

	HKs	HALs	HANs	HNMs	THMs	HAAs	Total DBPs	NPOC	TN	DO	Cond.	Temp.	pH	Free Chlorine	Total Chlorine
Free Chlorine	0.121 (0.574)	-0.023 (0.915)	0.208 (0.289)	0.055 (0.809)	0.291 (0.119)	0.339 (0.066)	0.190 (0.298)	0.094 (0.608)	0.236 (0.202)	-0.112 (0.612)	0.059 (0.788)	-0.004 (0.984)	0.133 (0.468)		
Total Chlorine	0.059 (0.785)	-0.074 (0.730)	0.158 (0.423)	0.034 (0.881)	0.273 (0.144)	0.316 (0.089)	0.152 (0.405)	0.104 (0.570)	0.203 (0.273)	-0.101 (0.647)	-0.044 (0.842)	-0.038 (0.835)	0.148 (0.417)	**0.987 (<0.001)***	
Pool Entries	0.377 (0.069)	0.360 (0.084)	0.113 (0.568)	0.057 (0.799)	**0.550 (0.002)***	0.095 (0.618)	0.035 (0.848)	-0.001 (0.998)	**0.454 (0.010)**	-0.338 (0.114)	0.378 (0.075)	0.145 (0.427)	-0.231 (0.204)	-0.041 (0.823)	-0.017 (0.925)

Cond.: Conductivity. **DO**: Dissolved oxygen. **HAAs**: Haloacetic acids. **HALs**: Haloacetaldehydes. **HANs**: Haloacetonitriles. **HNMs**: Halonitromethanes. **NPOC**: Non-purgeable organic carbon. **Temp**.: Temperature. **THMs**: Trihalomethanes. **TN**: Total nitrogen. **Total DBPs**: Sum of all DBPs (on a molar basis).

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APPENDIX 6

Figure A6-1: Loss of organic carbon content due to purging of trihalomethanes (THMs) during the analysis of non-purgeable organic carbon (NPOC). An ultrapure water sample fortified with each of the four trihalomethanes (500 µg L^{-1} each) was analysed pre- and post-NPOC analysis. Data is presented as normalised response: peak area of a THM divided by the peak area of the internal standard (1,2-dibromopropane) added prior to gas chromatography-mass spectrometry analysis.

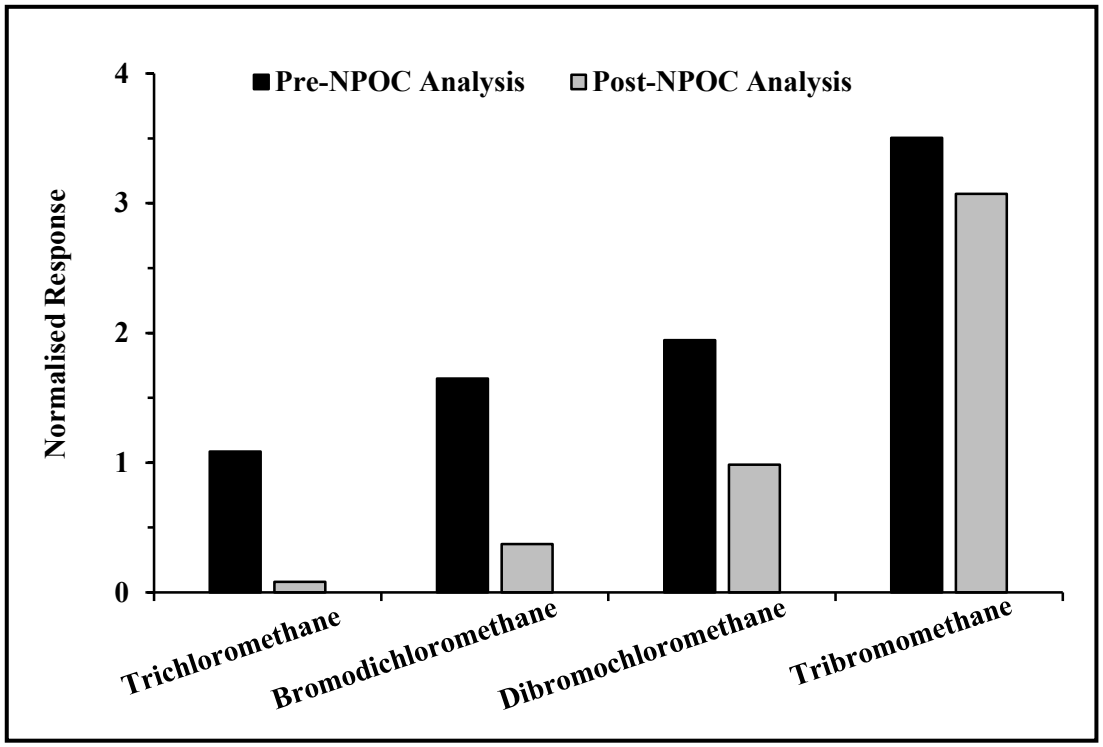
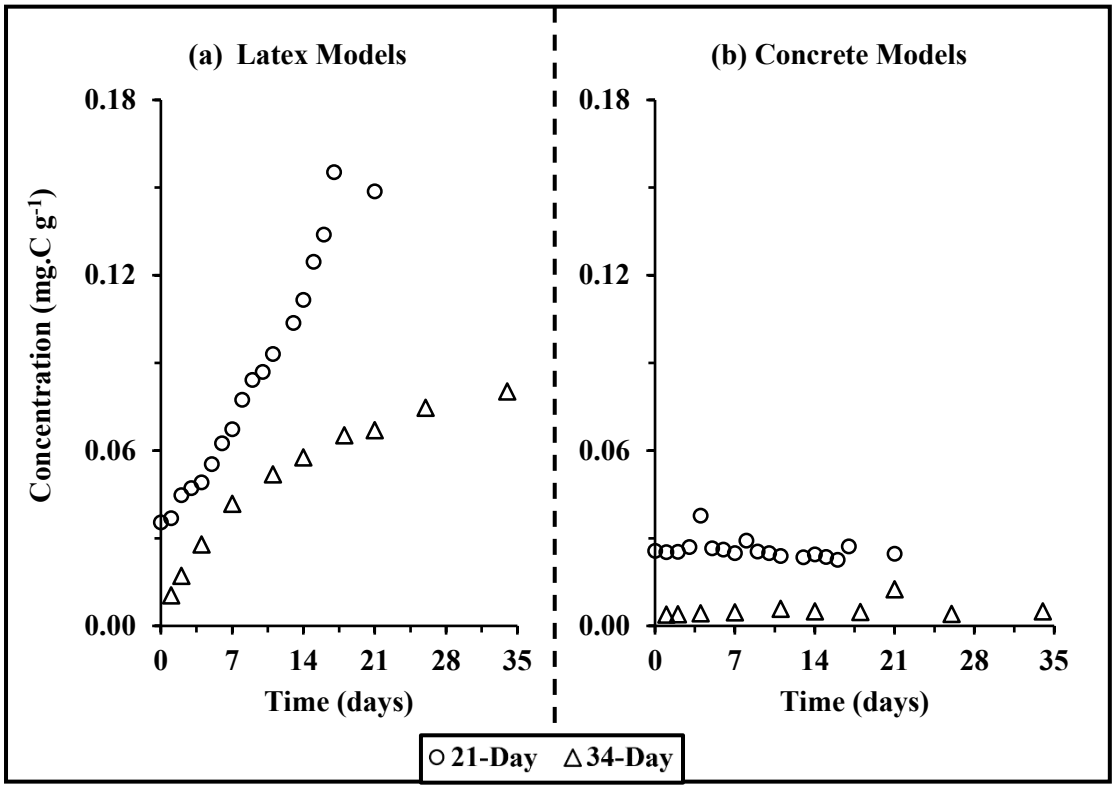


Figure A6-2: Comparison of concentrations of non-purgeable organic carbon (NPOC) leaching from **(a)** latex models and **(b)** concrete models measured during the 21-day and 34-day investigations. Data presented is that obtained from chlorinated models. All values were corrected for dilution and standardised by mass of product. Values represent the average of replicates and are presented as mg carbon per g product.



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