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Secondary formation of disinfection by-products by UV treatment of swimming pool water

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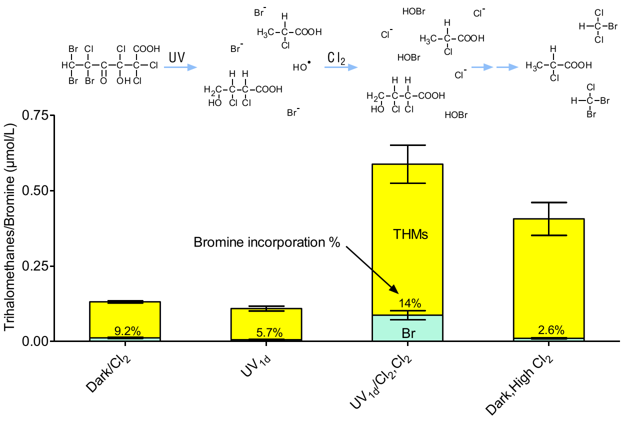
HIGHLIGHTS

- Trihalomethanes were only induced in pool water by post-UV chlorination.
- Increased chlorine exposure induced trihalomethanes similar to post-UV chlorination.
- No trihalomethane induction was caused by UV in continuously UV treated pool water.
- Brominated trihalomethanes formed by post-UV chlorination increased with UV dose.
- UV accelerates trihalomethane formation in batch test, but does not increase concentration.

GRAPHICAL ABSTRACT



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ABSTRACT

Formation of disinfection by-products (DBPs) during experimental UV treatment of pool water has previously been reported with little concurrence between laboratory studies, field studies and research groups. In the current study, changes in concentration of seven out of eleven investigated volatile DBPs were observed in experiments using medium pressure UV treatment, with and without chlorine and after post-UV chlorination.

Results showed that post-UV chlorine consumption increased, dose-dependently, with UV treatment dose. A clear absence of trihalomethane formation by UV and UV with chlorine was observed, while small yet statistically significant increases in dichloroacetonitrile and dichloropropanone concentrations were detected. Results indicate that post-UV chlorination clearly induced secondary formation of several DBPs. However, the formation of total trihalomethanes was no greater than what could be replicated by performing the DBP formation assay with higher chlorine concentrations to simulate extended chlorination. Post-UV chlorination of water from a swimming pool that continuously uses UV treatment to control combined chlorine could not induce secondary formation for most DBPs. Concurrence for induction of trihalomethanes was identified between post-UV chlorination treatments and simulated extended chlorination time treatment. Trihalomethanes could not be induced by UV treatment of water from a continuously UV treated pool. This indicates that literature reports of experimentally induced trihalomethane formation by UV may be a result of kinetic increase in formation by UV. However, this does not imply that higher trihalomethane concentrations would occur in pools that apply continuous UV treatment.

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The bromine fraction of halogens in formed trihalomethanes increased with UV dose. This indicates that UV removes bromine atoms from larger molecules that participate in trihalomethane production during post-UV chlorination.

Additionally, no significant effect on DBP formation was observed due to photo-inducible radical forming molecules NO_3^- (potentially present in high concentrations in pool water) and H_2O_2 (added as part of commercially employed DBP reducing practices).

1. Introduction

Chlorine is the most common disinfectant used in swimming pools due to its rapid killing rate of bacteria and viruses. However, it is well known that chlorine reacts with organic and inorganic matter to form chlorinated disinfection by-products (DBPs). The most common DBPs identified in pool water are combined chlorine species (organic and inorganic chloramines), haloacetic acids (HAAs), and trihalomethanes (THMs) (Chowdhury et al., 2014; World Health Organization (WHO), 2006). The general concern regarding the formation of DBPs is their effect on human health since some are genotoxic and thus might be carcinogenic (Richardson et al., 2007). Others, mainly the chloramines, cause irritation of eyes and the respiratory tract (Florentin et al., 2011). Thus, there is a need for techniques to ensure water quality of an acceptable level is maintained in public swimming pools. DBP formation is affected by numerous factors such as; bather load, filling water quality, temperature, pH, filtration, and disinfectant used (Hansen et al., 2012a, 2012b; Keuten et al., 2012; Lee et al., 2010; Pool Water Treatment Advisory Group (PWTAG), 2009; WHO, 2006).

An established method to reduce combined chlorine concentrations in swimming pools is direct photolysis by UV treatment (PWTAG, 2009). Several full scale studies have reported reduction in combined chlorine levels when treating with medium pressure UV (MP UV) lamps (Beyer et al., 2004; Cassan et al., 2011, 2006; Kristensen et al., 2009).

However, these studies do not agree regarding the effect of UV treatment on THM formation. For example, a short-term full-scale study by Cassan et al. (2006) found increased THM levels in pool water, while a similar study by Beyer et al. (2004) reported a decrease. In further contrast, Kristensen et al. (2009) observed no effect on THM levels in a swimming pool treated with UV over several weeks compared to reference periods in a full-scale study using real time THM measurements and meticulous monitoring of bather load. Clearly a better understanding of the effect of UV treatment on the formation of DBPs is needed and thus laboratory studies have also been conducted. A recent study has shown that trichloramine is the easiest of the combined chlorine species to be removed by UV followed by dichloramine and monochloramine (Soltermann et al., 2014). Another laboratory study reports that low-pressure UV (LP UV) exposure, followed by post-UV chlorination, increased concentration of some DBPs (e.g. THMs and dichloroacetonitrile), while haloacetic acids were not affected (Cimetiere and De Laat, 2014). It should be noted however that most laboratory based research has been performed using LP UV lamps and/or synthetic pool water (Cimetiere and De Laat, 2014; Weng et al., 2012). Consequently as MP UV is the most widely used in swimming pools for combined chlorine control, LP UV studies have little relevance for understanding processes occurring in real swimming pools. Thus our study was conducted using a MP UV lamp.

Induction of increased THM formation from existing organic matter in swimming pools has previously been reported by Glauner et al. (2005) during an investigation concerning two different ozone based advanced oxidation methods (AOPs) for swimming pool water treatment. Results showed that reaction of hydroxyl radicals with organic matter dissolved in pool water resulted in evolution of activated compounds, which produced significantly more THM when subsequently chlorinated (Glauner et al., 2005).

Assuming hydroxyl radicals cause observed increases in DBP formation through a UV induced process, one can consider several processes during UV treatment of water that cause the formation of radicals. For example, photolysis of water will occur under exposure to UV at wavelengths <200 nm, and hydroxyl radicals formed (Bolton, 2010). Also in chlorinated water, direct photolysis of hypochlorous acid (HOCl) can produce hydroxyl and chlorine radicals (Nowell and Hoigne, 1992; Watts and Linden, 2007). Additionally it has been shown that the UV/chlorine system is usable as an AOP to remove contaminants (Sichel et al., 2011). Furthermore, swimming pool water can contain nitrate in the range of less than 1 mg/L to 46 mg/L (Beech et al., 1980; Cimetiere and De Laat, 2014; Lee et al., 2010; Wang et al., 1998). Nitrate adsorbs light in the UV range (<240 nm) and produces hydroxyl radicals according to Eq. (1) (Lyon et al., 2012):

$$NO_3^- + H_2O \overset{hv}{\rightarrow} NO_2^- + 2^{\cdot}OH. \quad (1)$$

Once returned to the pool, nitrite will be oxidized by chlorine and form nitrate again (Lyon et al., 2012).

A Dutch company (Pool Water Treatment, Rotterdam, Netherlands) claims to prevent chloroform formation when UV is used in pools by adding hydrogen peroxide to the UV reactor, thereby reducing chlorine and producing radicals that degrade organic matter (Appel, 2013). It is well known that hydrogen peroxide is photolyzed by UV light to form hydroxyl radicals. This AOP can be used to remove organic pollutants from drinking water and wastewater (Hansen and Andersen, 2012; Sichel et al., 2011).

In the present study the effect of UV treatment and different radical initiators on DBP formation was studied using laboratory experiments. Water from two different swimming pools (without UV treatment installed) was exposed to light from a medium pressure UV lamp under controlled conditions in a collimated beam setup. The DBPs (chloroform, bromodichloromethane, dibromochloromethane, bromoform, dichloroacetonitrile, bromochloroacetonitrile, dibromoacetonitrile, trichloroacetonitrile trichloropropanone, dichloropropanone, and trichloronitromethane) were measured before and after UV exposure and again after (post-UV) chlorination to examine when DBPs were formed. The effect of chlorine, nitrate, and hydrogen peroxide as radical initiators was investigated via addition prior to UV irradiation. DBP levels measured after UV and post-UV chlorination were compared to a control sample not subjected to UV exposure. Additionally, water from a third swimming pool employing continuous UV treatment was exposed to UV in the laboratory to investigate whether further irradiation could induce DBP formation.

2. Material and methods

2.1. Reagents and standard analysis

All chemicals and standard solutions were purchased from Sigma-Aldrich. Colorimetric reactions were used to quantify chlorine, hydrogen peroxide, nitrate, and nitrite. Characterization of the pool water was performed using a cell test kit (LCK 310, Hach Lange, Germany) to measure free and total chlorine. During the experiments, residual chlorine was determined by 2,2-azino-bis(3-ethylbenzothiazoline)-6-

sulfonic acid-diammonium salt (ABTS), as described by Pinkernell et al. (2000). Hydrogen peroxide was determined using potassium titanium oxide oxalate (Antoniou and Andersen, 2015), while test kits (NO_3^- 09713 and NO_2^- 114776, Merck, Germany) were used to determined nitrate and nitrite. Further information can be found in the Supplementary information (SI Section 1).

A Shimadzu ASI-V UVC/Persulfate analyzer was utilized for the quantification of the remaining total organic carbon (TOC) in the samples. The sample injection volume was 3.00 mL and a calibration curve was formed using standards with concentrations ranging from 50–2000 μg/L ($R^2 = 0.9994$). The method quantification limit is 50 μg/L.

2.2. Pool water

Pool water was collected from public swimming pools and used for experiments on the day of collection. The pools for water collection were the main practice basin in Lyngby and the main practice basin and the hot water therapy basin in Gladsaxe (Denmark). The two practice basins are typical public pools (temperature 26 °C, sand filter with flocculation, and a side stream activated carbon filter) both with a hydraulic retention time (HRT) of 4 h. The hot water therapy basin has a much higher bather load and faster combined chlorine development, due to the higher temperature (34 °C). Hence, it is operated with three 400 W medium pressure mercury UV lamps in a side stream of about 20% of the circulation, in order to effectively control the combined chlorine concentration. Furthermore, the hot water pool has sand filters and a HRT of only 0.5 h. Filling water for the swimming pools is obtained from the public distribution network, which is comprised of non-chlorinated ground water. Water samples were collected during regular operating hours and analyzed for pH, NO_3^-, free and combined chlorine.

2.3. Analysis of DBPs

Analyses were performed as previously published by Hansen et al. (2012a). In brief, free chlorine was quenched by adding 50 mg/L ammonium chloride solution to the vials before they were filled, head-space-free, with sample. Samples were analyzed the same day by purge and trap (purge temperature = 30 °C, Velocity XPT Purge and Trap Sample Concentrator, Teledyne Tekmar, with auto-sampler: AQUATek 70, Teledyne Tekmar) coupled with a GC–MS (HP 6890 Series GC System, 5973 Mass selective detector, Hewlett Packard).

The method employed detects for the following compounds: chloroform, bromodichloromethane, dibromochloromethane, bromoform, dichloroacetonitrile, bromochloroacetonitrile, dibromoacetonitrile, trichloroacetonitrile, trichloropropanone, dichloropropanone, and trichloronitromethane. The limit of quantification (LOQ) is based on the lowest standard of the linear calibration curve and was found to be 0.6 μl/L for THMs and dichloroacetonitrile and 1.0 μg/L for dichloropropanone, trichloropropanone, trichloroacetonitrile, bromochloroacetonitrile, dibromoacetonitrile and trichloronitromethane.

2.4. UV treatment

UV treatment was conducted using a quasi-collimated beam apparatus with a doped, medium pressure lamp (P = 700 W, ScanResearch, Denmark). The lamp employed produces an enhanced output in the lower range (200–240 nm), compared to a standard mercury medium pressure lamp, as described by Hansen et al. (2013b). A comparison of spectra from low and medium pressure lamps is shown in Figure S1.

The lamp was turned on at least 20 min before the beginning of the experiment, in order to ensure a constant spectra and emission output. The distance between the center of the lamp and the sample was 24 cm. Petri-dishes (d = 8 cm and h = 4.5 cm) were used as reaction vessels with the exposed volume of sample to be approximately 180 mL. Samples were maintained headspace free and covered by a disc of quartz glass in order to limit volatilization of treated sample and losses of volatile DBPs. During irradiation, the sample was gently mixed with a stirrer in order to ensure homogeneity.

The UV dose was determined according to a method described by Hansen et al. (2013b). In summary, the UV exposure in the collimated beam setup was correlated to a real flow through system on a pool, using the removal of combined chlorine. The UV system needs 1.0 kWh/m^3 to remove 90% of the combined chlorine. For the collimated beam setup, required radiation time to remove 90% of combined chlorine from pool water was 12.3 min. In order to compare the experimental UV dose to a realistic treatment level, the UV system in the hot water basin in Gladsaxe swimming pool was used. This system consists of 4 UV lamps using a total of 2800 W which operate 24 h per day on a total pool volume of 50 m^3 (Kristensen et al., 2010, 2009). Therefore the applied electrical energy dose from UV was 1.34 $kWh/(m^3 \cdot d)$, thus it can be calculated that the dose equivalent to 1 day of treatment is achieved by 17 min radiation.

To test the stability of the UV system, the removal efficiency of monochloramine was determined in the collimated beam setup for each experiment.

2.5. DBP formation with chlorine

In the current study the formation of DBPs from chlorination was investigated. Similar tests have been used in other studies that investigate the potential for the formation of NCl_3 (Schmalz et al., 2011), THM and HAA in swimming pool water (Kanan and Karanfil, 2011), and THM, HAN and HAA from synthetic body fluid (Hansen et al., 2012a).

After UV exposure, chlorine was added to samples (post-UV chlorination) based on chlorine consumption measured by ABTS. Chlorine consumption was determined in pre-experimental tests. The aim was to achieve a chlorine residual after 24 h of either 1–3 mg/L (typical chlorine levels) or 31–33 mg/L (high chlorine levels). This relatively high chlorine level was used to simulate formation over an extended reaction time. After chlorine addition pH was adjusted to 7.10 and samples were stored at 25 °C. After 24 h residual chlorine was determined and samples were analyzed the same day for DBPs.

2.6. Experiments

In the current study laboratory batch experiments were employed in order to ensure controlled experimental conditions. The general methodology was for a sample of pool water to be spiked with a radical initiator (Cl_2, NO_3^- or H_2O_2) and then UV irradiated, as described in Section 2.4. The irradiation time varied from 8 to 34 min, equivalent to a 0.5–2 day dose of UV in a real treatment situation. After exposure, the sample was either analyzed for DBPs (Section 2.3) or chlorine was added (post-UV chlorination, Section 2.5), and allowed to react for 24 h before DBP analysis.

Water samples were analyzed for DBPs before UV exposure (in order to establish a baseline), after UV exposure (to determine dosage impact), and after post-UV chlorination (to investigate whether DBPs are formed during UV irradiation or after reaction with chlorine).

Chlorine concentrations were seen to vary in sampled pool water, therefore chlorine was added to ensure that a homogenous concentration of 3 mg/L was present before UV exposure. A few experiments were conducted without chlorine addition to investigate the effect of high and typical chlorine concentrations.

To examine the effect of nitrate, some samples were spiked with 11.1 mg/L NO_3^--N (50 mg/L NO_3^-) prior to UV exposure.

Hydrogen peroxide was added until the concentration was maintained at 1.0 mg/L after reaction with chlorine residual from the pool. Radiation time was 58 and 65 min (3.4 and 3.8 days of UV dose) for Lyngby and Gladsaxe, respectively. This prolonged exposure time was necessary to achieve 80% removal of H_2O_2 through photolysis, as applied by the commercial company Pool Water Treatment (Rotterdam, Netherlands) in real treatment situations (Appel, 2013).

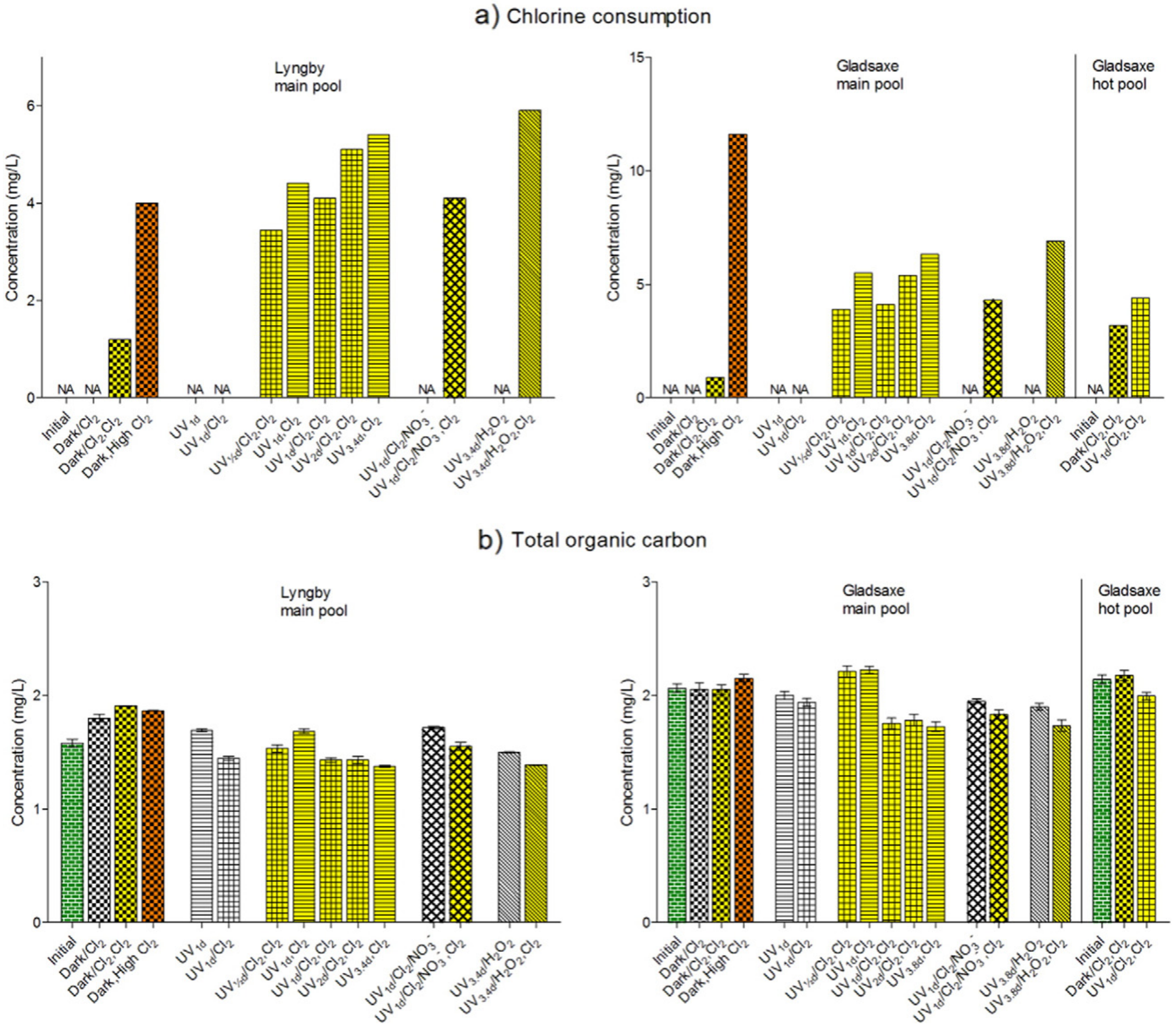


Fig. 1. a) Chlorine consumption in 24 h at 25 °C and b) content of total organic carbon at the different experimental treatments for the three investigated swimming pools. NA = not applicable, indicates that there is no incubation with chlorine and therefore no consumption is determined. The error bars in TOC analysis represent the standard deviation of analytical duplicates.

Water from the hot water therapy basin in Gladsaxe was only used in selected experiments to determine potential to induce the formation of DBPs in pool water already treated with UV light.

Three samples were not exposed to UV light (i.e. were kept dark by covering with cardboard) however were subjected to the same experimental conditions as other samples (temperature, retention time, stirring etc.). One of the non-UV samples was used as a control for the handling of samples during UV exposure. This control sample was not post-UV chlorinated, but directly analyzed for DBPs. The remaining non-UV samples were post-UV chlorinated and acted as controls to determine the formation potential of pool water without UV exposure.

Selected experiments were repeated on different days with different waters for range finding but the results given in this paper originate from a single day where all treatments were performed using water collected at that time from the swimming pool.

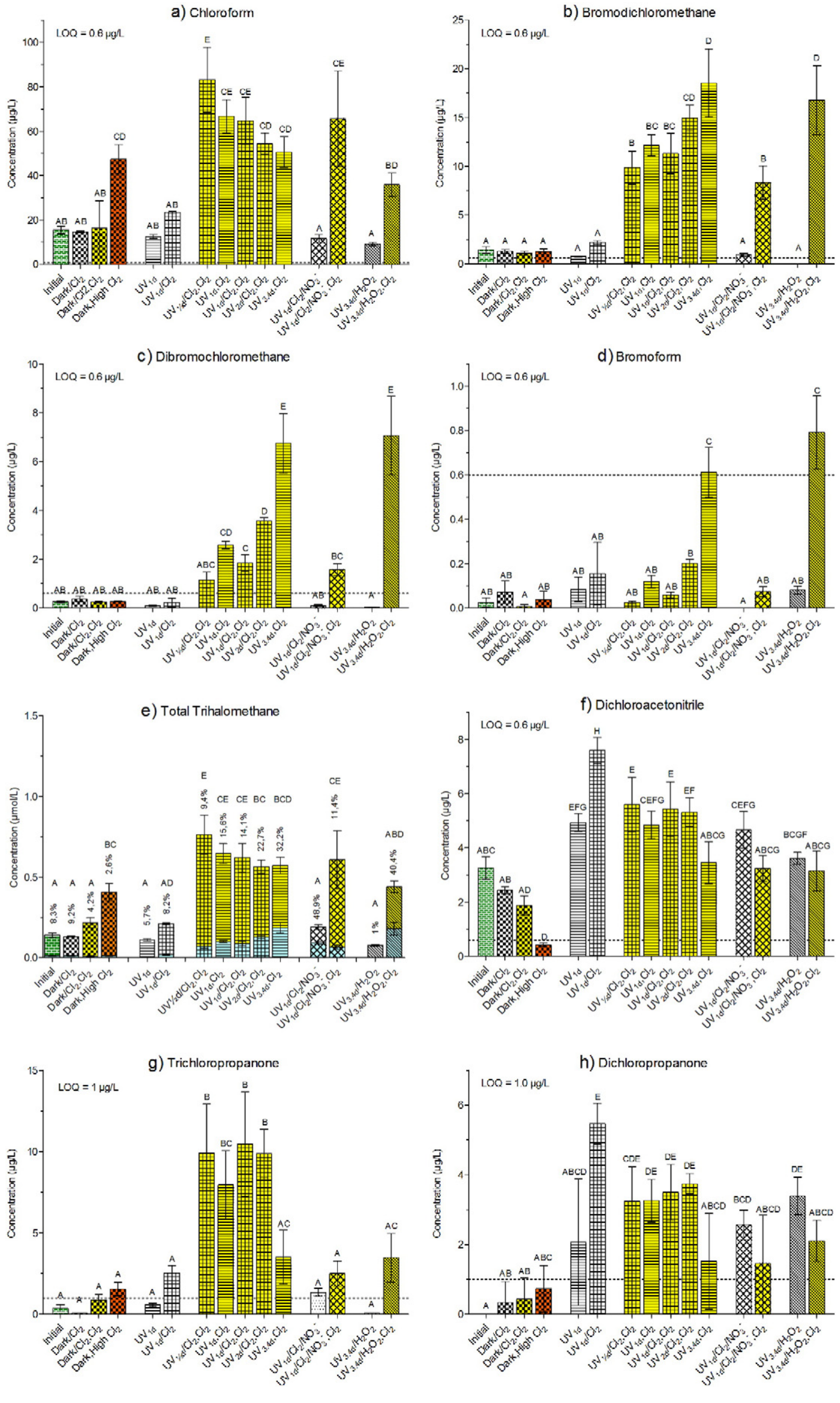
In most cases, experiments were performed in quadruplicate with three samples used for DBP analysis and one for the determination of residual chlorine (single analysis) and TOC (duplicate analysis). In a few cases ($UV_{1d}/Cl_2,Cl_2$ and $Dark/Cl_2,Cl_2$) experiments were performed with 6 replicates, five for DBP analysis and one for residual chlorine and TOC.

In Figs. 1–3 condensed notation is used where forward slash "/" refers to treatments applied simultaneously and comma "," separates an action e.g. $UV_{1d}/Cl_2,Cl_2$ represents a chlorinated sample treated with a UV dose similar to 1 day which afterwards was chlorinated for 24 h.

A detailed description of each treatment together with a schematic overview of the experiments performed is given in SI (Section S3 and Figure S2).

2.7. Data analysis

Data analysis was conducted using MS Excel and Prism Graph Pad. Results for different treatments were subsequently assessed using a one-way ANOVA with Tukey's Multiple Comparison Test (95% confidence level). Treatments were grouped according to significant difference ($P < 0.05$). All treatments with insignificant difference were given the same letter (full explanation and examples given in SI Sections 5.1 and 5.2). Error bars in the figures indicate standard deviation among the replicates.


a) Chloroform
LOQ = 0.6 µg/L
Concentration (µg/L)
b) Bromodichloromethane
LOQ = 0.6 µg/L
c) Dibromochloromethane
LOQ = 0.6 µg/L
d) Bromoform
LOQ = 0.6 µg/L
e) Total Trihalomethane
Concentration (µmol/L)
8,3%
9,2%
4,2%
2,6%
5,7%
8,2%
9,4%
15,8%
14,1%
22,7%
32,2%
48,9%
11,4%
1%
40,4%
f) Dichloroacetonitrile
LOQ = 0.6 µg/L
g) Trichloropropanone
LOQ = 1 µg/L
h) Dichloropropanone
LOQ = 1.0 µg/L
Initial
Dark/Cl2
Dark/Cl2,Cl2
Dark,High Cl2
UV 1d
UV 1d/Cl2
UV ½d/Cl2,Cl2
UV 1d,Cl2
UV 1d/Cl2,Cl2
UV 2d/Cl2,Cl2
UV 3,4d,Cl2
UV 1d/Cl2/NO3-
UV 1d/Cl2/NO3-,Cl2
UV 3,4d/H2O2
UV 3,4d/H2O2,Cl2

3. Results and discussion

Water samples from 3 indoor swimming pool facilities were tested to evaluate UV-induced effects on swimming pool water chemistry. Eleven DBPs, which are usually found in swimming pool water, (Chowdhury et al., 2014) were examined. However, bromochloroacetonitrile, dibromoacetonitrile, trichloroacetonitrile and trichloronitromethane were not detected and are consequently omitted in the following discussion. The remaining seven investigated DBPs (chloroform, bromodichloromethane, dibromochloromethane, bromoform, dichloroacetonitrile, trichloropropanone, and dichloropropanone) were all detected in the collected pool water, indicating that UV exposure is not responsible for the creation of the total amount of these by-products. Experiments were conducted in batch format, thus DBP formation may differ from observed over longer time scales where pool water is treated continually with UV light. In a real system water does not receive a dose equivalent to several days of treatment at once so there can be reactions between chlorine and the photolysis products from when the first UV dose is delivered until water enters the UV chamber a second time. For example, in the hot water basin UV system in Gladsaxe swimming pool (see Section 2.4) the actual UV dose delivered in a single pass through the UV reactor is 2800 W/25 m^3/h (=0.112 kWh/m^3) which can be compared to the daily UV dose of 1.34 kWh/m^3 to show that in this system water is treated about 12-times per day.

3.1. Aqueous chemistry of pool water

For the 3 pool water samples used in this study, the initial pH ranged from 7.10 to 7.24. TOC, free chlorine, and combined chlorine concentrations ranged from 1.58 to 2.14 mg/L, 0.44 to 1.37 mg/L, and 0.17 to 0.36 mg/L, respectively (Table S1). Nitrate analysis for untreated water samples exhibited initial concentrations ranging from 2.2 to 6.1 mg/L NO_3^--N.

3.2. Chlorine consumption

Treatments that included chlorination were analyzed for residual chlorine after 24 h incubation as shown in Fig. 1a. Chlorine consumption was seen to vary greatly among treatments. The lowest chlorine consumption is observed in solely chlorinated samples (Dark/Cl_2,Cl_2: Fig. 1a). Chlorine consumption tends to increase with increasing chlorine concentration (Dark/Cl_2,Cl_2 vs. Dark,High Cl_2). The highest chlorine consumption was observed in post-UV chlorinated samples and appears proportional to exposure time ($UV_{½d}$/Cl_2,Cl_2 vs. UV_{1d}/Cl_2,Cl_2 vs. UV_{2d}/Cl_2,Cl_2). Similar to this finding, UV treatment of drinking water increased the fraction of low molecular organic matter which increased the chlorine demand (Choi and Choi, 2010).

According to Weng et al. (2012), the concentration of nitrite in irradiated water samples increases after depletion of chlorine, due to nitrate photolysis. Nitrite formation may increase chlorine consumption since nitrite is oxidized by chlorine to form nitrate (Diyamandoglu et al., 1990). However, in this study no change was detected in chlorine consumption in experiments where nitrate was added (UV_{1d}/Cl_2,Cl_2 vs UV_{1d}/Cl_2/NO_3^-,Cl_2).

For the experiment studying the addition of hydrogen peroxide as a radical initiator, chlorine consumption was observed to be similar to the samples subjected to prolonged UV exposure ($UV_{3.4d}$/H_2O_2,Cl_2 vs $UV_{3.4d}$,Cl_2).

3.3. Total organic carbon

Observed levels of total organic carbon varied only slightly between experimental treatments thus it is difficult to conclude any effect on this analyte caused by the varying conditions.

3.4. Formation of volatile by-products

3.4.1. Effect of UV on trihalomethanes

Trihalomethanes in this study refer only to chlorinated and brominated trihalomethanes, i.e. chloroform, bromodichloromethane, dibromochloromethane and bromoform. We hypothesize that these compounds are formed from the same precursor with any variation observed between different species being due to the ratio of bromine to chlorine.

The investigated swimming pools were chlorinated with the source of aqueous bromine mainly originating from the low concentrations present in the source water. Consequently chloroform was the most pronounced of the THMs detected (Figs. 2 and 3). Values on the figures are also given in Table S2–4 in IS. Furthermore, an overview including detailed description of experimental conditions applied to the pool water samples can be found in IS (Section S3).

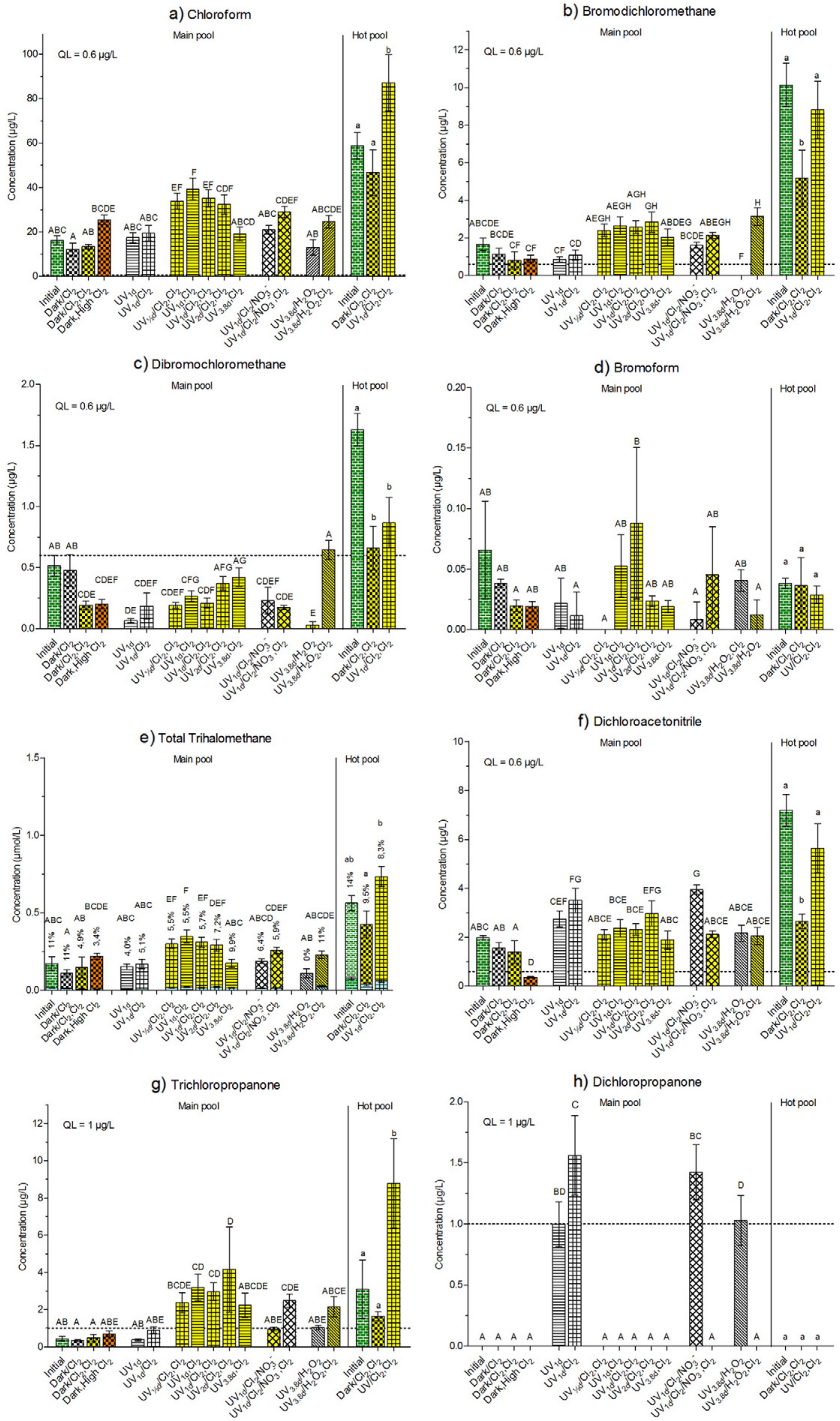
UV treatment of swimming pools has been suspected of creating THMs directly in the UV chamber. However, when comparing initial concentrations with UV treated only samples, it was seen that concentrations of the four different THMs were not elevated. Thus, THMs were not formed directly during the UV irradiation. Interestingly, for the brominated THMs, a statistically insignificant trend of reduction was observed for experimental conditions which exclusively involved UV irradiation (UV_{1d}), compared to initial levels (Figs. 2b, c, d and 3b, c, d). Hansen et al. (2013b) previously found that direct photolysis of THMs increased with an increasing amount of bromine incorporated. Therefore, bromoform photolyzed most readily, while chloroform was recalcitrant (Hansen et al., 2013b). Levels of the brominated THMs in the pool water were close to or under the quantification limit. Similar results were observed in the current study.

In the present study THMs were not formed directly by exposure to UV. However, in post-UV chlorinated samples, an increased amount of THM was found (UV_{1d}/Cl_2 vs $UV_{½d}$/Cl_2,Cl_2; UV_{1d}/Cl_2,Cl_2; and UV_{2d}/Cl_2,Cl_2). A likely explanation for this is that UV treatment increases the reactivity of organic compounds in the pool water towards chlorine thus acting as precursors for THM formation. Therefore when this activated organic matter subsequently interacts with chlorine THMs are formed. Cimetiere and De Laat (2014) observed a similar increase in THM formation when chlorinating pool water after UV exposure by low pressure lamp. Furthermore, UV treatment of drinking water increases the fraction of low molecular organic matter and increased chlorine demand together with THM formation (Choi and Choi, 2010). Thus, UV treatment of pool water could also contribute to the activation of organic matter, making it more reactive.

In the present study increasing UV exposure time ($UV_{½d}$/Cl_2,Cl_2; UV_{1d}/Cl_2,Cl_2; UV_{2d}/Cl_2,Cl_2; $UV_{3.4d}$,Cl_2) was seen to decrease chloroform formation while increasing the formation of brominated THMs (Figs. 2 and 3). This infers that the activated organic matter was further photodegradable. The difference in bromine incorporation for the two pools studied is likely due to different bromine levels in their source water.

An explanation for why increased UV exposure resulted in increasing formation of brominated THMs and less chloroform after chlorination (Figs. 2a–d and 3a–d) might be that UV irradiation cleaved the bonds between larger organic compounds and bromine (Fig. 4), so that bromide was liberated into solution. It is well known that the

Fig. 2. The concentration of the investigated DBPs at different experimental treatments of water from the main basin in Lyngby. e) The light green area and the % indicate the amount of bromine incorporated in the total trihalomethane. The dotted line indicates the limit of quantification (LOQ). The letters illustrate the significant difference between means (95% confidence level). The error bars indicate the standard deviation among 3–5 replicates (see IS Table 2–4 for details).



a) Chloroform

b) Bromodichloromethane

c) Dibromochloromethane

d) Bromoform

e) Total Trihalomethane

f) Dichloroacetonitrile

g) Trichloropropanone

h) Dichloropropanone

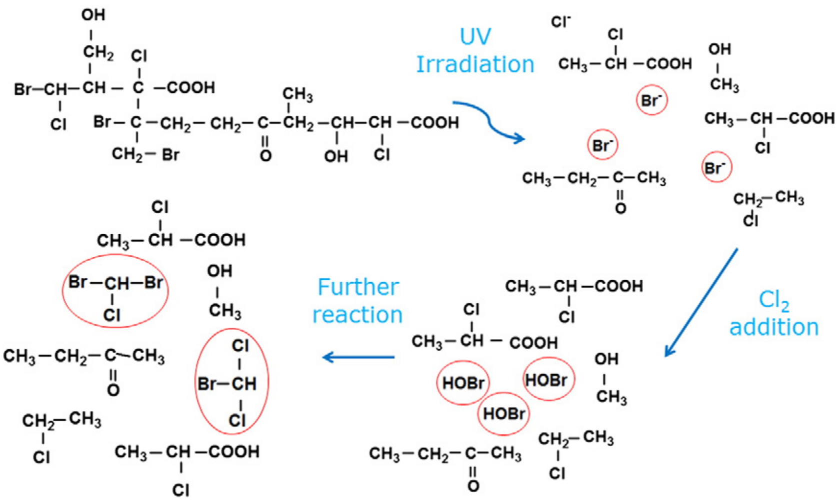


Fig. 4. Schematic representation of brominated species formation.

carbon–bromine (C–Br) bond (280 kJ/mol) is weaker than the carbon–chlorine (C–Cl) bond (397 kJ/mol) (White, 1992; Xiao et al., 2014). Furthermore, the C–Br bond tend to give a higher absorbance in the UV range than the C–Cl bond which is e.g. seen from the increased absorbance of dichlorobromomethane compared to chloroform (Nicole et al., 1991) and further increase with increasing bromine substitution. The first law of photochemistry states that light must be absorbed by a compound in order for a photochemical reaction to take place and the third law states that the energy of an absorbed photon must be equal to or greater than the energy of the weakest bond in the molecule. Thus the chance that a molecule with C–Cl and C–Br bonds absorbs a photon in the UV range (200 nm (=598 kJ/mol) to 400 nm (=299 kJ/mol)) with energy higher than the energy of the C–Br is greater than the chance of absorbing a photon with energy higher than C–Cl bond. In laboratory experiments with photodegradation of THMs by the same UV lamp as used in this study, bromoform was found to degrade fastest of the THMs and the degradation speed decreased with substitution of bromine to chlorine (bromoform > dibromochloromethane > bromodichloromethane > chloroform) (Hansen et al., 2013b).

When chlorine was added post-UV exposure, bromide should be oxidized to hypobromous acid as hypochlorous acid is a stronger oxidant (White, 1992). Hypobromous acid in turn reacted with the newly formed THM precursors in competition with chlorine, to form Br-Cl-DBPs. The rate constants for hypobromous acid reactions with organic molecules are up to three orders of magnitude higher than for chlorine (Heeb et al., 2014). Thus, bromine can be transferred from the larger brominated molecules to smaller volatile compounds like bromodichloromethane, dibromochloromethane and bromoform as illustrated in Fig. 4.

It has been hypothesized that UV irradiation only accelerates chloroform formation and that over time the amount of chloroform formed in a swimming pool is the same with or without UV treatment (Kaas and Andersen, 2007). This is a result of the amount of precursor being limited and the main removal processes for these contaminants being oxidation and mineralization by chlorine (Judd and Bullock, 2003). In the present study, this was examined by adding a higher chlorine dose in non-irradiated samples (Dark, High Cl_2) to simulate long-term residence time of pool water in a basin. In most cases, a significant difference was not found in chloroform levels for post-UV chlorinated samples ($UV_{½d}/Cl_2,Cl_2$; $UV_{1d}/Cl_2,Cl_2$; $UV_{2d}/Cl_2,Cl_2$; $UV_{3.4d},Cl_2$ and $UV_{3.8d},Cl_2$) and for samples with simulated long residence times (Dark, High Cl_2: Figs. 2a and 3a). Thus, our obtained data supports this hypothesis. Furthermore, when examining THMs as a sum of the four THM species (total trihalomethane; TTHM), it was seen that post-UV chlorinated samples ($UV_{1d}/Cl_2,Cl_2$) were not significantly different from samples subjected to simulated long residence times (Dark,High Cl_2: Figs. 2e and 3e).

The experimental treatment of water from the hot therapy pool in Gladsaxe demonstrated that post-UV chlorination induced a slight elevation in the levels of TTHMs observed compared to the initial levels, however this was not statistically significant. The hot water therapy pool employed UV as part of its water treatment system in order to decrease combined chlorine levels. Our results support the hypothesis that UV accelerates, but does not increase THM formation.

3.4.2. Effect of UV on miscellaneous DBPs

Dichloroacetonitrile was unstable in the presence of free chlorine, and an increasing concentration of chlorine results in decreasing levels of dichloroacetonitrile when reacting for 24 h (Initial vs. Dark/Cl_2,Cl_2 vs. Dark,High Cl_2; Figs. 2f and 3f). Other studies reporting instability of haloacetonitriles in chlorinated water can be found in the literature (Hansen et al., 2013a; Kristiana et al., 2014). In the present study it was found that dichloroacetonitrile was formed directly by UV (UV_{1d}/Cl_2 vs. Initial). Furthermore, elevated or at least equivalent levels of dichloroacetonitrile as found in the initial samples were found in post-UV chlorinated samples. UV irradiation did not affect the amount of dichloroacetonitrile found after post-UV chlorination ($UV_{½d}/Cl_2,Cl_2$; UV_{1d},Cl_2; $UV_{1d}/Cl_2,Cl_2$; $UV_{2d}/Cl_2,Cl_2$). However, in samples with prolonged UV exposure ($UV_{3.4d},Cl_2$ and $UV_{3.8d},Cl_2$), there was a tendency for decreased dichloroacetonitrile concentrations. In the water from the hot water therapy pool, no significant increase in dichloroacetonitrile was observed in the post-UV chlorinated samples compared to the initial samples. This suggests that any increased formation might be transient.

Di- and trichloropropanone might be formed directly by UV. However, for trichloropropanone, detected concentrations were close to the quantification limit (Figs. 2g and 3g). For dichloropropanone, greater concentrations were observed in the UV exposed sample than the levels observed in water from Lyngby (Initial vs UV_{1d}/Cl_2; Fig. 2h). Meanwhile water from the main pool in Gladsaxe was seen to have concentrations close to the quantification limit (Fig. 3h). Nevertheless, an increased

Fig. 3. The concentration of the investigated DBPs at different experimental treatments of water from the main basin and hot water therapy basin in Gladsaxe. e) The light green area and the % indicate the amount of bromine incorporated in the total trihalomethane. The dotted line indicates the limit of quantification (LOQ). The letters illustrate the significant difference between means (95% confidence level). The error bars indicate the standard deviation among 3–5 replicates (see IS Table 2–4 for details).

formation of trichloropropanone was found in the post-UV chlorinated samples for both main pools. Dichloropropanone however increased in concentration in the water from Lyngby, but was not detected in the water from Gladsaxe.

3.5. Effects of radicals on DBP formation

Hydroxyl radicals from ozone based AOPs have previously been shown to activate organic matter in swimming pool water, forming increased concentrations of THM when subsequently chlorinated (Glauner et al., 2005). During UV treatment, hydroxyl radicals can be formed by several processes such as photolysis of chlorine, hydrogen peroxide, or nitrate.

To investigate the effect of radicals on DBP formation, experiments with the addition of nitrate, hydrogen peroxide or chlorine were performed. Increasing nitrate concentrations to 50 mg/L (11 mg N/L) had no significant effect on DBP formation, concentrations of TTHM or dichloroacetonitrile. However, a decrease in trichloropropanone was observed while the effect on trichloropropanone was uncertain (Figs. 2 and 3). After UV exposure, nitrate spiked samples were analyzed for nitrite. Nitrite concentrations were 0.45 and 0.20 mg N/L in water from Lyngby and Gladsaxe, respectively. Only a low fraction of nitrate was therefore converted to nitrite and in that process produced hydroxyl radicals. However, chlorine will oxidize nitrite to nitrate (Lyon et al., 2012) during irradiation until all chlorine is photolyzed. The low conversion of nitrate to nitrite could explain that no statistically significant change was seen for TTHM.

Activation of hydrogen peroxide via UV irradiation did not affect TTHM formation significantly with formation magnitude seen to be similar in samples irradiated with or without hydrogen peroxide (UV$_{3.4d}$,Cl$_2$ vs. UV$_{3.4d}$/H$_2$O$_2$,Cl$_2$; Figs. 2 and 3). This finding is clearly contradictory to the commercially claimed process (Appel, 2013). However, results are based on the treatment of water from just two pools so it cannot be concluded that the treatment does not work in general. The presence of hydrogen peroxide during UV treatment contributed to a negligible decrease in dichloroacetonitrile concentrations (UV$_{3.4d}$,Cl$_2$ vs. UV$_{3.4d}$/H$_2$O$_2$,Cl$_2$; Figs. 2 and 3). However, a significant decrease was observed in trichloropropanone formation (Figs. 2 and 3).

Insignificant changes in the concentration of investigated DBPs were observed for direct photolysis (UV$_{1d}$ vs. UV$_{1d}$/Cl$_2$). For post-UV chlorinated samples the addition of chlorine prior to UV treatment did not significantly affect the formation of the investigated DBPs (UV$_{1d}$,Cl$_2$ vs. UV$_{1d}$/Cl$_2$,Cl$_2$; Figs. 2 and 3). Thus, direct formation of DBPs during UV irradiation by reaction with chlorine radicals appears unimportant with regard to water quality.

Glauner et al. (2005) observed that treatment of swimming pool water with two other radical forming methods (i.e. H$_2$O$_2$/O$_3$ and O$_3$/UV) increased THM formation potential. Consequently it was expected that greater levels of THM formation would be observed in the present study for experiments employing chlorination after irradiation in the presence of hydrogen peroxide and nitrate, which produced hydroxyl radicals. However, our results show no enhanced stimulation of DBP formation beyond what can be achieved by UV followed by chlorination (UV$_{1d}$/Cl$_2$,Cl$_2$ vs. UV$_{1d}$/Cl$_2$/NO$_3^-$,Cl$_2$ and UV$_{3.4d}$,Cl$_2$ vs. UV$_{3.4d}$/H$_2$O$_2$,Cl$_2$). A possible reason could be that post-UV chlorination increases THM formation by two- to tenfold, while increases described by Glauner et al. (2005) are comparably small showing only 10 and 20% increase in THM formation for H$_2$O$_2$/O$_3$ and O$_3$/UV treatments following chlorination, respectively. Thus, the effect of radicals on THM formation in the present study might be masked by the much larger stimulation of THM formation observed which is caused by the UV treatment.

4. Conclusions

The findings can be summarized thus:

- DBPs are not formed in the UV reactor but in secondary reactions that occur after chlorine addition;
- UV treatment followed by chlorination amplified DBP formation;
- Radicals do not significantly affect DBP formation;
- UV treatment amplified the fraction of brominated THM observed; and
- UV irradiation accelerates DBP formation but does not clearly increase the total amount formed.

Moreover, the observations that chloroform is not formed in the UV reactor but via a secondary reaction and that UV irradiation does not increase the total amount of DBPs formed must be considered in the regulation of the use of UV in pools (e.g. Circulaire DGS/EA4 2008-6 from Ministère de la santé, de la jeunesse et des sports, France).

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Appendix A. Supplementary data

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.scitotenv.2015.03.044.

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