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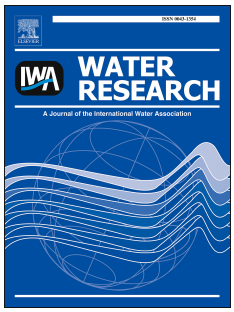

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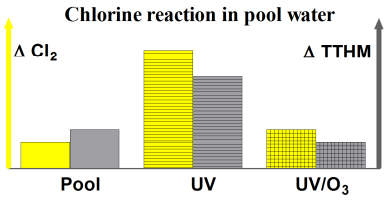

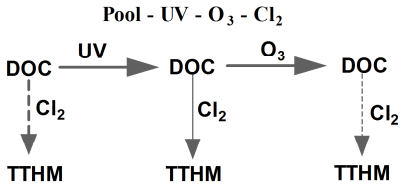

Combined UV treatment and ozonation for the removal of by-product precursors in swimming pool water

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Abstract

Both UV treatment and ozonation are used to reduce different types of disinfection by-products (DBPs) in swimming pools. UV treatment is the most common approach, as it is particularly efficient at removing combined chlorine. However, the UV treatment of pool water increases chlorine reactivity and the formation of chloro-organic DBPs such as trihalomethanes. Based on the similar selective reactivity of ozone and chlorine, we hypothesised that the created reactivity to chlorine, as a result of the UV treatment of dissolved organic matter in swimming pool water, might also be expressed as increased reactivity to ozone. Moreover, ozonation might saturate the chlorine reactivity created by UV treatment and mitigate increased formation of a range of volatile DBPs. We found that UV treatment makes pool water highly reactive to ozone. The subsequent reactivity to chlorine decreases with increasing ozone dosage prior to contact with chlorine. Furthermore, ozone had a half-life of 5 min in non-UV treated pool water whereas complete consumption of ozone was obtained in less than 2 min in UV treated pool water. The ozonation of UV-treated pool water induced the formation of some DBPs that are not commonly reported in this medium, in particular trichloronitromethane, which is noteworthy for its genotoxicity, though this issue was removed by UV treatment when repeated combined UV/ozone treatment interchanging with chlorination was conducted over a 24-hour period. The discovered reaction could form the basis for a new treatment method for swimming pools.

Keywords: Ozone, UV, swimming pool, trihalomethane, disinfection by-products

1 Introduction

Swimming pools are used for recreational activities, and it is necessary to disinfect swimming pool water in order to protect against infection by microbiological pathogens. Chlorine is the most commonly used disinfectant in swimming pool water. However, a general problem with maintaining chlorine concentrations for hygiene reasons is that the chlorine reacts continuously with organic matter in the water to form chloramines (combined chlorine) and chloro-organic by-products. A general concern about chloro-organic disinfection by-product (DBP) formation is the effect on human health, because some are carcinogenic (Richardson et al., 2007). There has been identified more than 100 DBPs in pool water (Richardson et al., 2010) where the most frequently investigated DBPs are chloramines, haloacetonitriles (HANs), haloacetic acids (HAAs) trihalomethanes (THMs), chloral hydrates and nitrosamines (Chowdhury et al., 2014; World Health Organisation, 2006). Both types of by-product can be reduced through water exchanges or different treatment methods. Combined chlorine concentration can be reduced with UV treatment via direct photolysis (PWTAG, 2009). It is an efficient way of removing chloramines to photolyse them with UV treatment in the return flow. Soltermann et al. (2014) reported that trichloroamine is the easiest of the combined chlorine species to be removed by UV.

A reduction in the combined chlorine level via medium pressure UV treatment has been reported by several full-scale studies (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 trihalomethane (THM) formation. An increase (Cassan et al., 2006) and decrease (Beyer et al., 2004) of THM formation has been reported in short-term full-scale studies. In contrast, Kristensen et al. (2009) observed no effect on THM levels in a swimming pool treated with UV in a long-term full-scale study. However, Liviac et al. (2010) illustrated that UV treatment might be beneficial for the reduction of genotoxicity and cytotoxicity in chlorinated swimming pool water. Hansen et al. (2013b) reported that photolysis is less important than volatilization for some volatile DBPs e.g. chloroform. Moreover, Zare Afifi and Blatchley. (2016)

demonstrated that concentration of most volatile DBPs decreased with both MP and LP UV treatment. A recent laboratory study (Spiliotopoulou et al., 2015) reported that UV treatment appears to break down relative non-reactive organic molecules into smaller molecules which react quickly with chlorine and accelerated DBP formation but did not clearly increase the total amount formed. Ozone is difficult to use for pool water treatment, as there is a lack of a good reliable sensor for ozone detection in water and ozone cannot be allowed in the pool due to toxicity to swimmers. Different authors have mentioned ozone dosage of 1 ppm (Eichelsdörfer and Jandik, 1985), 0.8-1.2 ppm (Eichelsdörfer and Jandik, 1988) and 1.6 ppm (Hamil, 2011) for swimming pool water treatment. There is limited literature on the effect of ozonation on formation of chlorination DBPs in recirculated water, but knowledge about ozone and its kinetics can be found in the drinking water and wastewater ozonation literature (von Gunten, 2003). It has been found that the most common DBPs, along with nitrogen compounds and chloramine, react very slowly with ozone (Eichelsdörfer and Jandik, 1985); however, according to DIN standards for swimming pool water ozonation, a decrease (34-48%) in chloroform formation potential can be achieved, depending on ozone contact time (Eichelsdörfer and Jandik, 1988). Alternatively, Glauner et al. (2005) achieved 12% absorbable organohalogen (AOX) reduction and 3% reduction of total trihalomethane (TTHM) formation potential after 10 minutes of ozone oxidation compared with untreated pool water. An investigation of several pools (Lee et al., 2010) found that ozone/chlorine-treated swimming pools had lower levels of DBPs than chlorinated pools. A laboratory study (Hansen et al., 2016) reported that ozone reacts well with freshly added organic matter but slowly with organic matter that remains after extended chlorination. Additionally, it was reported that reaction with fresh organic matter decreases formation of volatile chlorination by-products, while a slow reaction with already chlorinated organic matter produces more volatile by-products with further chlorination.

Gaining an understanding of UV treatment followed by ozonation in swimming pools could help in designing more efficient treatment systems to minimise the occurrence of disinfection by-products. Thus, the aim of the current study is to investigate the effect of a combined

treatment system on DBP formation. As both ozone and chlorine preferably react with electrophilic groups in compounds (von Sonntag and von Gunten, 2012; White, 1992), we hypothesise that reactivity to chlorine, created by the UV treatment of dissolved organic matter in pool water, might also mean that there is increased reactivity to ozone and that ozonation might remove the chlorine reactivity created by UV treatment. Therefore, we first performed an experiment to range-find the effect of swimming pool water UV activation on chlorine reactivity. Second, an experiment was carried out to characterise the effect of adding various doses of ozone to pool water, with or without UV pre-treatment, before chlorination to study the effect on chlorine reactivity and the formation of chlorination by-products. Finally, the possible effect on chlorination by-product formation was investigated by a repeated, combined UV-ozone treatment interchanged with chlorination (repeated cycles of UV followed by ozone with subsequent chlorination). Toxicity estimation was used to evaluate water quality.

2 Material and methods

2.1 Reagents and standard analysis

All chemicals and reagents were purchased from Sigma-Aldrich, Denmark. The experimental set-up for ozonation was based on a 20 g/h ozone generator from O3-Technology AB (Vellinge, Sweden) which was supplied with dry oxygen gas. Generated ozone was dispersed through a diffuser in a collection bottle containing ultra-pure water, which was immersed in an ice bath so that ozone solubility would be maximised. To increase further the solubility of ozone, a manometer and valve were placed after the collection bottle, and a pressure of 1.4 barG was applied. Based on these experimental conditions, the concentration of ozone achieved in the stock solution was between 80 and 100 mg/L.

Ozone was quantified via a colorimetric method using indigotrisulfonate (Bader and Hoigné, 1981). Reagents used were 0.5 M phosphate buffer at pH 2 and 1.00 g/L potassium indigotrisulfonate dissolved in 20 mM phosphoric acid and further description can be found in Hansen et al. (2016). Free and total chlorine in the collected pool water samples were measured

using the colorimetric method based on the oxidation of diethyl-p-phenylenediamine (DPD), with and without addition of iodide, while residual chlorine during the experiment was determined by employing the colorimetric method, using 2, 2-azino-bis (3-ethylbenzothiazoline)-6-sulfonic acid-diammoniumsalt (ABTS), as described by Pinkernell et al. (2000). Non-volatile organic carbon in the pool water samples was quantified with a Shimadzu ASI-V UVC/Persulphate analyser with a sample injection volume of 3 mL. A calibration curve was formed by using potassium hydrogen phthalate standards, with concentrations ranging from 50 to 2000 µg/L (R^2 = 0.9994). The method quantification limit was 50 µg/L. Non-volatile organic carbon is referred to herein as 'dissolved organic carbon' (DOC).

2.2 Pool water

Pool water samples were collected from a public swimming pool and used for experiments on the day of collection. The pool for water collection was the main practice basin in Gladsaxe (Denmark). It is a typical public pool (temperature 26ºC, sand filter with flocculation and a side stream activated carbon filter) with a hydraulic retention time (HRT) of 4 hours. The water in the pool is not replaced besides the amount of water which is added due to evaporation and loss during backwash of filters. Water for filling the pool is obtained from the public distribution network, which comprises non-chlorinated groundwater. The pH was measured immediately upon arrival to the laboratory and it was 7.2 ± 0.1.

2.3 Disinfection by-products

Samples were analysed 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), and the analyses were conducted as described by Hansen et al. (2012a).

The employed method detects the following compounds: chloroform, bromodichloromethane, dibromochloromethane, dichloroacetonitrile, bromochloroacetonitrile, trichloropropanone,

dichloropropanone and trichloronitromethane. The limit of detection (LOD) and limit of quantification (LOQ) were expressed as LOD = $X_{b1} + 3S_{b1}$ and LOQ = $X_{b1} + 10S_{b1}$, where 'X_{b1}' is the mean concentration of the blank and S_{b1} is the standard deviation of the blank. The LOQ values for all compounds were: chloroform (0.6 µg/L), bromodichloromethane (0.6 µg/L), dibromochloromethane (0.4 µg/L), dichloroacetonitrile (0.6 µg/L), bromochloroacetonitrile (0.2 µg/L), trichloropropanone (1.0 µg/L), dichloropropanone (1.0 µg/L), and trichloronitromethane (0.6 µg/L).

2.4 Treatments

2.4.1 UV treatment

Treatment was conducted using a quasi-collimated beam apparatus with a doped, medium pressure lamp (P = 700 W, ScanResearch, Denmark). To ensure constant spectra and emission output, the lamp was turned on half an hour before the experiment. Petri dishes (350 mL) were used as reaction vessels, while samples were maintained headspace-free and covered by a disc of quartz glass, to limit the volatilisation of the treated sample. To ensure homogeneity during irradiation, samples were mixed gently with a stirrer. The UV dose was determined according to a method described by Hansen et al. (2013b). In summary, UV exposure in the collimated beam set-up 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 set-up, required radiation time to remove 90% of the combined chlorine from the pool water was 12.3 mins. In order to compare the experimental UV dose to a realistic treatment level, the UV system in the Gladsaxe swimming pool's hot water basin was used. This system consists of 4 UV lamps using a total of 2800W and operating 24 hours 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), and so it can be calculated that the dose equivalent to 1 day of treatment is achieved after 19 minutes of radiation. To test the stability of the UV system, the removal efficiency of monochloramine was determined in the collimated beam set-up for each experiment. As monochloramine was used as an actinometer, the UV dose was

159 recalculated for our system to correspond to 250 mJ/cm^2 UV dose delivered per minute from a
160 low pressure UV lamp based on the data published in Li and Blatchley (2009).

161 **2.4.2 Ozonation**

162 Ozonation was achieved by adding an amount of ozone stock solution to a water sample which
163 resulted in maximum 10% dilution of the sample and the concentrations were back calculated
164 according to actual dilution. Ozone dosage was determined by adding a sufficient amount of
165 potassium indigotrisulfonate and a phosphate buffer to a separate ultra-pure water sample and
166 measuring the absorbance of the unreacted indigotrisulfonate. A detailed description can be
167 found in Hansen et al. (2016).

168 **2.4.3 Chlorination and chlorine consumption**

169 The formation of DBPs as a result of chlorination was investigated using a standardised DBP
170 formation assay. Similar tests have been used in other studies investigating the potential for the
171 formation of NCl_3 (Schmalz et al., 2011), THM and HAA in swimming pool water (Kanan,
172 2010), THM, HAN and HAA from synthetic body fluid (Hansen et al., 2012a) and particles
173 from pools (Hansen et al., 2012b). The effect of chlorine concentration in the assay was also
174 recently investigated by Hansen et al. (2013a). In the current study, the same approach was used
175 to simulate chlorination in the pool after the return of UV/ozone-treated water.

176 Water samples were transferred to 40 mL glass vials after treatment in which chlorine and boric
177 acid were added based on the chlorine consumption determined in pre-experimental tests. The
178 aim was to have 1 ± 0.3 mg Cl_2/L after 24 hours at 25ºC (measured by ABTS). Chlorination was
179 performed in quintuplicate, with three samples used for DBP analysis and two for the
180 determination of residual chlorine. Samples for DBP analyses were dosed with ammonium
181 chloride solution (50 mg/L), to quench free chlorine which neither affects the already formed
182 DBP (Kristiana et al., 2014) nor increases N-DBP formation (Hua et al., 2014). The samples
183 were analysed the same day.

2.5 Experiments

In the current study, laboratory batch experiments were employed, to ensure controlled experimental conditions. The control samples were chlorinated directly for DBP analysis, to analyse the formation potential of pool water without UV and ozone treatment. Control samples for UV treatment were kept in the dark by covering them with cardboard, and thus they were not exposed to UV light – thereby ensuring the same experimental conditions (temperature, retention time, stirring). Samples of the same pool water were collected on different days (between 10 and 11 am) and used for experiments no later than 3 hours after collection. In the figures that accompany this study, the notation comma "," separates an action; for example, UV_{2d}, 2 ppm O_3,Cl_2 represents a sample treated with a UV dose of two days (9.5 J/cm^2), subsequently ozonated with a 2 ppm dosage and then finally chlorinated for 24 hours.

2.5.1 UV treatment

Samples of pool water were UV irradiated for times varying between 9 and 38 minutes, which is equivalent to a half-day (2.1 J/cm^2) to two-day dose (9.5 J/cm^2) of UV in a real treatment situation. After UV treatment, the samples were chlorinated according to Section 2.4.3.

2.5.2 Ozonation

Different ozone dosages were used for the range-finding experiments. Pool samples were divided into three equal subsamples which were then ozonated with 1, 2 and 4 ppm dosages and left for at least 30 min to allow ozone reactions to proceed until completion. After ozonation, the samples were chlorinated according to Section 2.4.3.

2.5.3 Combined treatment

The pool samples were divided into seven subsamples. One sample out of seven was taken for the control and transferred to four 40 mL glass vials (one for TOC and three replicates for DBPs), while the remaining six samples were UV-irradiated with a dose corresponding to two days of UV dose (9.5 J/cm^2). One sample was immediately taken for DBP analysis while the others were ozonated with range of different dosages (1, 2, 4, 7, 10 ppm) and left for at least 30

min to allow ozone reactions to proceed until completion. After ozonation, samples were chlorinated according to Section 2.4.3.

2.5.4 Repeated treatment cycle

Pool water samples were divided into nine subsamples. The treatment cycle consisted of UV treatment followed by ozonation and subsequent chlorination. To begin with, all samples were UV-irradiated with a UV dose (38 min, 9.5 J/cm^2) corresponding to the average dose the water get during two days. Thereafter, one sample was taken for analysis while the rest were treated with an ozone dosage of 7 ppm and left for 30 minutes to allow ozone reactions to proceed until completion. After ozonation, another sample was taken for analysis while the rest were chlorinated according to Section 2.4.3. Then, a third sample was taken for analysis, which completed the first cycle. The remaining six samples were treated in the same order, to complete two more cycles.

2.6 Estimation of toxicity

Toxicity was estimated as reported by Hansen et al. (2012a). Based on the measured concentration of the different DBPs, cyto- and genotoxicity were estimated as the sum of the concentration of each compound divided by its EC_{50} (Equation (1)):

$$\text{Toxicity} = \sum_1^i \frac{C_i}{EC_{50,i}} \quad (1)$$

All EC_{50} values were used as reported in the literature (Muellner et al., 2007; Plewa et al., 2008).

3 Results and discussion

Water samples from Gladsaxe swimming pool were tested, to evaluate the effect of treatment with UV followed by ozone on swimming pool water chemistry. Eleven DBPs which are usually found in swimming pool water (Chowdhury et al., 2014) were examined. However, bromoform, dibromoacetonitrile and trichloroacetonitrile were not detected, and hence they are not reported in the results. Batch experiments were conducted in the laboratory, so DBP

235 formation results may differ from those observed over longer time scales, where pool water is
236 treated continually with UV followed by ozonation. In a real system, water does not receive UV
237 doses equivalent to several days of treatment at once, so reactions can take place between
238 chlorine and photolysis products after ozonation and when the water enters the UV chamber for
239 the second time.

240 **3.1 Effect of UV on reaction with chlorine**

241 Residual chlorine was measured after 24 hours' incubation, following which chlorine
242 consumption was calculated (Figure 1a). Samples treated with UV irradiation exhibited higher
243 chlorine consumption than the non UV-treated samples (dark control). Furthermore, chlorine
244 consumption increased dose-dependently following UV exposure. A similar trend was observed
245 in a recent paper (Spiliotopoulou et al., 2015). Moreover, increase in chlorine consumption due
246 to UV irradiation of pool water have been reported by Cimetiere and De Laat (2014) and Weng
247 et al. (2012).

248 The chlorinated samples were analysed for DBPs and trends were observed for the formation of
249 DBPs when the pool water was treated with different UV doses. For some DBPs, formation
250 increased initially with the lowest dose exposure, but then it did not change significantly with
251 higher doses, e.g. dichloroacetonitrile (Figure 1b), bromodichloromethane (Figure 1e) and
252 dichloropropanone (Figure 1g). Weng et al. (2012) also reported an increase in
253 dichloroacetonitrile formation due to UV irradiation and chlorination. Furthermore, the
254 formation of chlorinated nitriles involves cleavage of N-Cl bonds (Li and Blatchley, 2007;
255 Weng et al., 2012) and UV irradiation has been effective for cleavage of N-Cl bonds (Li and
256 Blatchley, 2009; Weng and Blatchley, 2013; Weng et al., 2013, 2012). However, for other
257 DBPs formation increased dose-dependently, similar to chlorine demand, e.g.
258 dibromochloromethane (Figure 1f) and trichloropropanone (Figure 1h), where formation
259 increased following higher UV doses but then decreased when exposed to the highest dose
260 (UV_{10d}). Another pattern was also observed in chloroform (Figure 1d) and

bromochloroacetonitrile, where formation increased almost threefold with the lowest UV dose ($UV_{1/2d}$) but then did not change with a further increase in UV dose. However, it decreased when treated with the highest dose (UV_{10d}). An explanation for this decrease in formation during post UV chlorination could likely be due to decrease in DOC level by oxidation at very high UV dose (Figure S3) and thus lower amount of precursor was available for reaction.. The DOC level in UV_{10d} decreased by 37% compared to the initial value (Figure S3). Trichloronitromethane increased almost threefold with chlorination following treatment with the lowest UV dose, but then trichloronitromethane decreased with a higher UV dose and fell to its minimum level at the highest applied UV dose.

The amount of bromide incorporated in THM increased in the UV-treated samples compared to the dark control. Brominated DBP formation increased significantly with the lowest UV dose, and formation increased further with higher UV doses. Spiliotopoulou et al. (2015) have reported similar results and suggested that UV treatment breaks down Br-carbon bond in large molecules (DOC), which results in brominated DBPs, as the released bromide is oxidised to HOBr by HOCl which then reacts with DOC. This is supported by that brominated THMs absorbs UV irradiation more effectively than chlorinated THM (Nicole et al., 1991) which results in faster removal of brominated compounds than chlorinated compounds during UV irradiation (Hansen et al., 2013b). Calculated cytotoxicity (Figure 1k), which was mainly attributed to dichloroacetonitrile (as this was the largest addend in the calculation according to Equation 1), increased in the samples treated with UV followed by chlorination, but the increase was not dose-dependent, whereas genotoxicity (Figure 1l), which derives mainly from trichloronitromethane (contributing generally with the largest addend in the summation according to Equation 1), was highest when treated with the lowest UV dose and then decreased dose-dependently.

3.2 Effect of ozonation

There was a significant increase in chlorine consumption, due to ozone exposure to the pool water (Figure 2a). Consumption was almost twice the amount compared to the control (DC) with the lowest ozone dosage (1 mg/L), and it increased further with higher ozone exposure. A similar increase in chlorine consumption has been reported by Hansen et al. (2016) during the chlorination of ozonated pool water. The increase is likely due to radical oxidation of precursor which can be observed during long life time of ozone where most of the ozone is removed by decomposition to radicals (Hansen et al., 2016).

There was a dosage-dependent effect observed in concentrations of most of the DBPs, in accordance with the trend in chlorine consumption. Regarding THMs, chloroform formation increased almost twofold with the lowest ozone dosage (1 mg/L of ozone) exposure, and it increased further with higher ozone dosages (Figure 2d). However, the formation of bromodichloromethane (Figure 2e) and dibromochloromethane (Figure 2f) increased with the initial dosage, but their concentrations remained unchanged with further increases in dosage. These results contradict the small decrease in TTHM formation potential after the ozonation of pool water reported by Glauner et al. (2005). A recent study (Hansen et al., 2016) observed that the effect of ozone on THM formation during subsequent chlorination is dependent on the characteristics of the DOC. If the DOC is mainly fresh pollutant from bathers, then ozone is consumed quickly and THM formation decreases following ozone treatment. Conversely, if the DOC is mainly “old” pollutant which has been exposed to chlorine for a long period, the DOC is less reactive with ozone, and a longer ozone lifetime and increased THM formation are observed following ozone treatment. We observed the lifetime of 2 mg/L ozone to be more than 20 mins (Figure S1, SI), which indicates that the DOC in the pool water reacted only very slowly with ozone. This fits with the increase in THM formation observed following ozone treatment.

Regarding HANs, the only increase was observed following a higher ozone dosage (4 mg/L), so with lower ozone dosage (1 mg/L, 2 mg/L) exposure there was almost no effect on the formation of either dichloroacetonitrile (Figure 2b) or bromochloroacetonitrile (Figure 2c). For other DBPs, the formed concentration of dichloropropanone was under the detection limit (Figure 2g), while the formation pattern was quite similar to the one observed in chloroform for trichloropropanone (Figure 2h) and trichloronitromethane (Figure 2i). Increase in trichloronitromethane formation during ozonation has previously been reported for pool water treatment (Hansen et al., 2016) and drinking water treatment (Hoigne and Bader, 1988; Merlet et al., 1985). Cytotoxicity and genotoxicity increased dosage dependently. Hence, toxicity of following the ozonated swimming pool water increased. However, brominated DBP formation increased minimally with the lowest ozone dosage, but it did not increase further with higher dosages.

3.3 Combined treatment

Chlorine consumption decreased when UV-treated pool water samples were exposed to the lowest ozone dosage (1 mg/L). Consumption decreased further dose-dependently and was lowest when treated with the highest ozone dosage (10 mg/L). A likely explanation for this is that the UV treatment of pool water made the DOC more reactive to chlorine (as seen in the previous section) which then reacts with ozone. Thus, when ozone reacts with the reactive DOC, reactivity is removed and lower chlorine consumption is observed.

The chlorination of UV-treated pool water samples produced the highest formation of THMs (Figure 2). However, this formation decreased when the UV-treated samples received added ozone at a low dosage (1 mg/L of ozone). In addition, the formation of chloroform reduced significantly with a lower ozone dosage added to UV-treated pool water, while the decrease was less significant with higher ozone dosages. The formation of brominated THMs (bromodichloromethane, dibromochloromethane) also decreased in line with increasing ozone dosage. However, for the brominated THMs the reduction in formation was lowest at low ozone

dosages and highest with high ozone dosages. At the highest ozone dosage (10 mg/L) THM formation was below the limit of quantification. The reason for this contradiction in the effect of ozone dosage should be found in the lifetime of ozone. For the low ozone dosage, ozone was consumed quickly, as it reacts with the reactive DOC induced by UV (Figure S1, SI). Bromate formation is not expected with short lifetime of ozone as bromide requires ozone contact time or radical exposure from decomposition of ozone to form bromate (Antoniou and Andersen, 2012). Thus bromide can react with chlorine to form hypobromous acid which then forms brominated DBPs (Hansen et al., 2016; Spiliotopoulou et al., 2015). At high ozone dosages, ozone saturated the DOC with high reactivity to ozone, and hence a longer ozone lifetime is expected – as seen in previous research (Hansen et al., 2016) which results in the oxidation of bromide to bromate.

A similar trend in THM formation was observed in HANs (dichloroacetonitrile, bromochloroacetonitrile) and dichloropropanone. However, trichloropropanone formation could not be reduced, even at the highest ozone dosage. A previous study reports that trichloropropanone did not form directly following the UV treatment of pool water; rather, precursor formation for trichloropropanone occurs (Spiliotopoulou et al., 2015). Based on our results it appears that ozone does not react with the precursor for trichloropropanone once it is formed during the UV treatment of pool water.

The trichloronitromethane trend was different from other DPBs, where lower ozone dosages had a negligible effect on formation; however, formation increased significantly with higher ozone dosages. The formation of trichloronitromethane during ozonation followed by chlorination is known in drinking and pool water treatment (Hansen et al., 2016; Hoigne and Bader, 1988; Merlet et al., 1985). A recent study identified primary and secondary amines as being the most dominant trichloronitromethane precursors in natural water during ozonation followed by chlorination (McCurry et al., 2016). In general, ozone reacts slowly with nitrogen-containing compounds (Rice, 1995), which explains the lack of effect of ozone on trichloronitromethane formation at lower ozone dosages, due to the very short ozone lifetime. Calculated cytotoxicity,

362 which was mainly attributable to HANs, e.g. dichloroacetonitrile and bromochloroacetonitrile,
363 reduced significantly during the combined treatment. However, genotoxicity, which was mainly
364 caused by trichloronitromethane, increased with the combined treatment. The ozonation of UV-
365 treated pool water removed the formation of most of the DBPs except for trichloronitromethane,
366 which is the main contributor to the calculated genotoxicity of water.

367 **3.4 Repeated treatment cycle**

368 The increase in genotoxicity, due to an increase in trichloronitromethane, seems to be a problem
369 at this stage of the combined treatment, but in the literature it has also been reported that UV
370 treatment can photolyse trichloronitromethane (Hansen et al., 2013b). Therefore, a combined
371 treatment experiment was performed in cycles to investigate the effect of continued treatment
372 that would occur in a swimming pool. The experiment with repeated treatment cycles was
373 performed with high treatment levels (two-day UV dose and 7 mg ozone/L). This level of
374 treatment is not realistic in a pool, but it is used herein to investigate trends in DBP formation
375 during repeated treatment.

376 Chlorine consumption was measured after each treatment cycle (Figure 3a) and was found to
377 decrease gradually in each cycle. This indicates that the remaining DOC becomes less reactive.
378 In general, when chlorine consumption decreases, the formation of DBPs also decreases, which
379 was also observed for the investigated DBP except for trichloropropanone. Based on the results
380 presented in Figures 1 and 2, which are summarised in Table 1, both UV and ozone may
381 increase the formation of trichloropropanone, and ozone dosage does not remove the precursor
382 when added as post-UV treatment. However, trichloropropanone was removed by UV in the
383 next treatment cycle (Figure 3h), which is in accordance with the findings in Hansen et al.
384 (2013b). Nonetheless, it should be noted that UV removal was not enough to decrease the
385 concentration during the three treatment cycles.

386 Chloroform, bromodichloromethane, dibromochloromethane and bromochloroacetonitrile all
387 show similar patterns during the repeated treatment cycle. UV treatment increases formation

potential, and the following ozone treatment decreases it. During UV treatment in the next treatment cycle, bromodichloromethane, dibromochloromethane and bromochloroacetonitrile decrease (Figure 3), as expected, since they previously have been found to be photolysed by UV (Hansen et al., 2013b). Dichloroacetonitrile and dichloropropanone behave a little differently (Figure 3b and 3g), in that their formation also increased following UV treatment and decreased again after ozone exposure (Table 1), but both compounds were formed during UV treatment and seemed to be removed by ozone.

Trichloronitromethane exhibited a different pattern. As for the other DBPs, UV increased the formation of trichloromethane during chlorination, but ozone increased it further, and thus a relatively high concentration of trichloronitromethane was found after chlorination at the end of a cycle. However, UV can easily photodegrade trichloronitromethane (Hansen et al., 2013b), which was also observed in the following treatment cycle. For each completed treatment cycle, the level of trichloronitromethane measured at the end decreased.

Bromine-containing DBPs were photolysed during UV treatment, and bromide was liberated into the water. The following ozone mainly reacted with reactive DOC and not with the bromide; thus, the bromide was oxidized by chlorine and new brominated DBPs were formed. However, based on the measured brominated THMs and bromochloroacetonitrile, the results indicated that fewer brominated DBPs were formed after a few repeated treatment cycles. Consequently, the genotoxicity of pool water should also decrease, as brominated DBPs in general are more genotoxic than their chlorinated counterparts (Muellner et al., 2007; Plewa et al., 2008). As trichloronitromethane was the main contributor to the calculated genotoxicity, genotoxicity follows the same pattern as trichloronitromethane and thus decreased after a few repeated treatment cycles. These results indicate that continuous treatment with UV, followed by ozone, could be a possible solution to reducing the amount of DBPs and thereby improving water quality in swimming pools.

4 Conclusions

The treatment of swimming pool water by means of UV irradiation increased chlorine demand. Furthermore, the ozonation of pre-treated UV-irradiated pool water subsequently removed chlorine demand and decreased DBP formation. Combined treatment effectively reduced the level of disinfection by-products in pool water except for trichloronitromethane where an increase was observed. Trichloronitromethane was reduced after repeated treatment cycles and thus UV/ozone treatment is predicted to improve swimming pool water quality.

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Figure 1. Effect of the UV treatment of swimming pool water on chlorine consumption, DBP formation potential and toxicity. j) The red area and the % indicate the amount of bromine incorporated in the total trihalomethane. The dotted line indicates the limit of quantification (LOQ), whereas the error bar indicates the range of measured values.

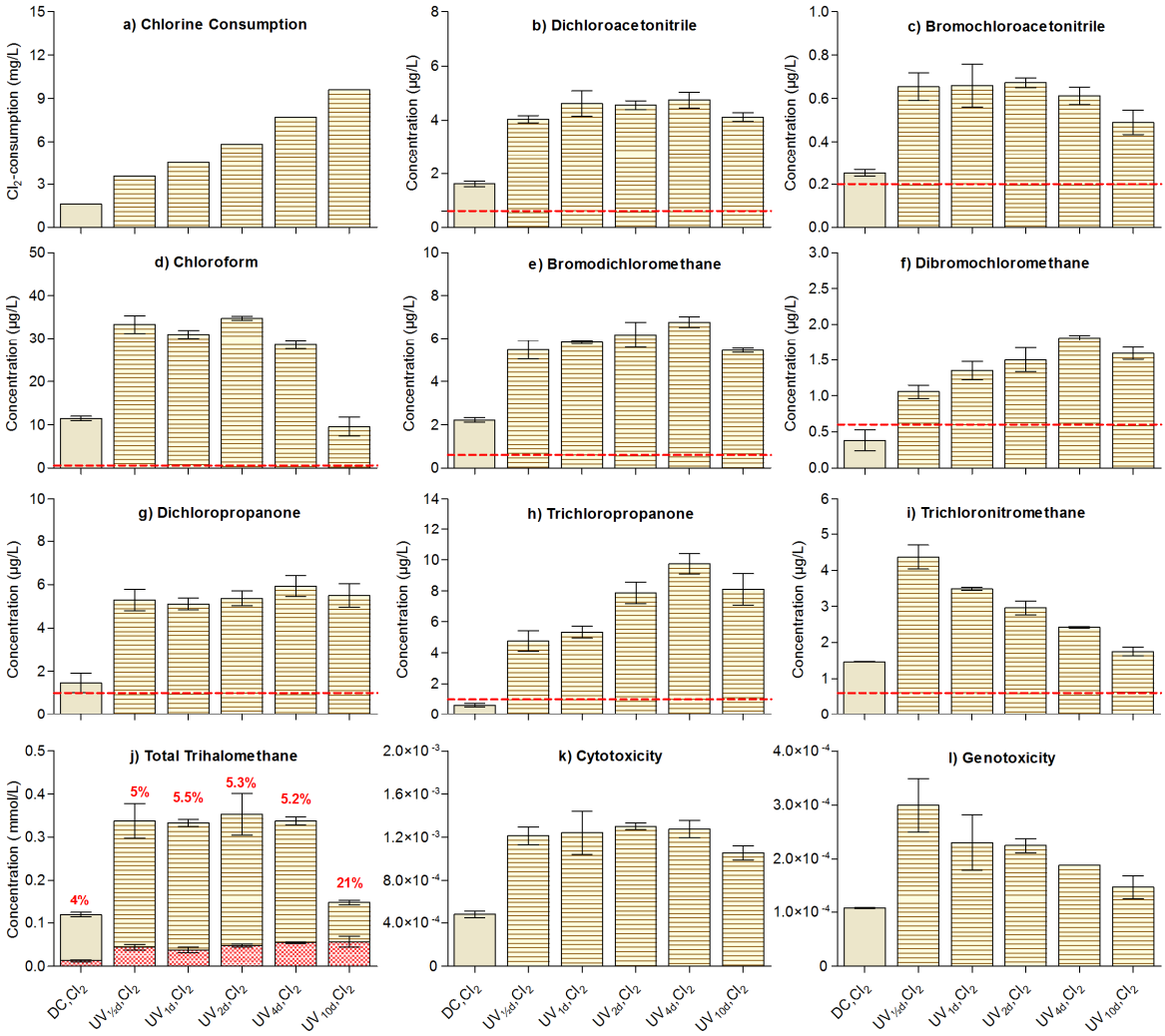
Figure 2. Effect of ozonation and combined treatment of swimming pool water on chlorine consumption, DBP formation potential and toxicity. j) The red area and the % indicate the amount of bromine incorporated in the total trihalomethane. The dotted line indicates the limit of quantification (LOQ), whereas the error bar indicates the range of measured values.

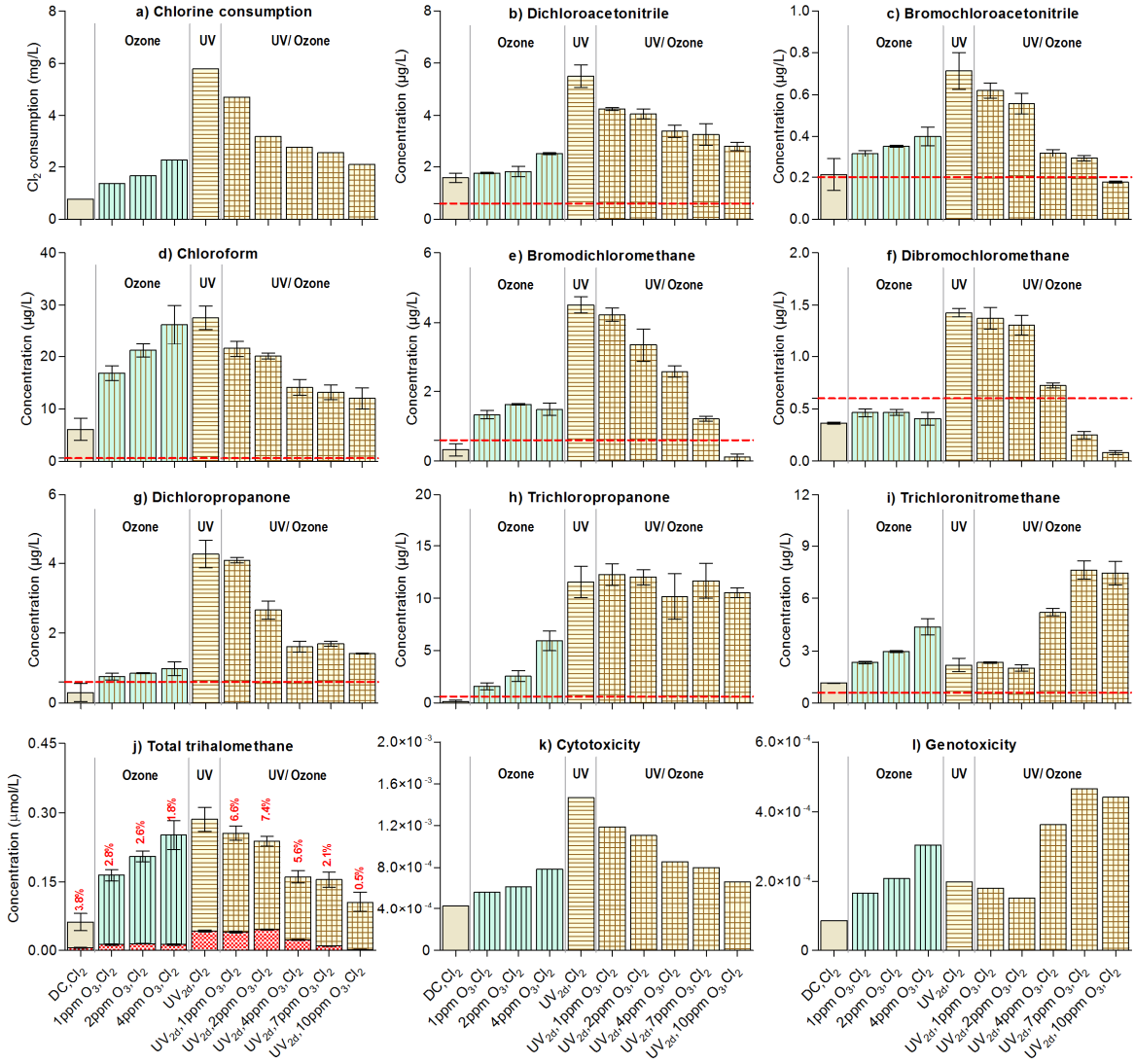
Figure 3. Effect of repeated combined treatment of swimming pool water in a cycle on chlorine consumption, DBPs formation potential and toxicity. j) The red area and the % indicate the amount of bromine incorporated in the total trihalomethane. The dotted line indicates the limit of quantification (LOQ) whereas the error bar indicates the range of measured values.

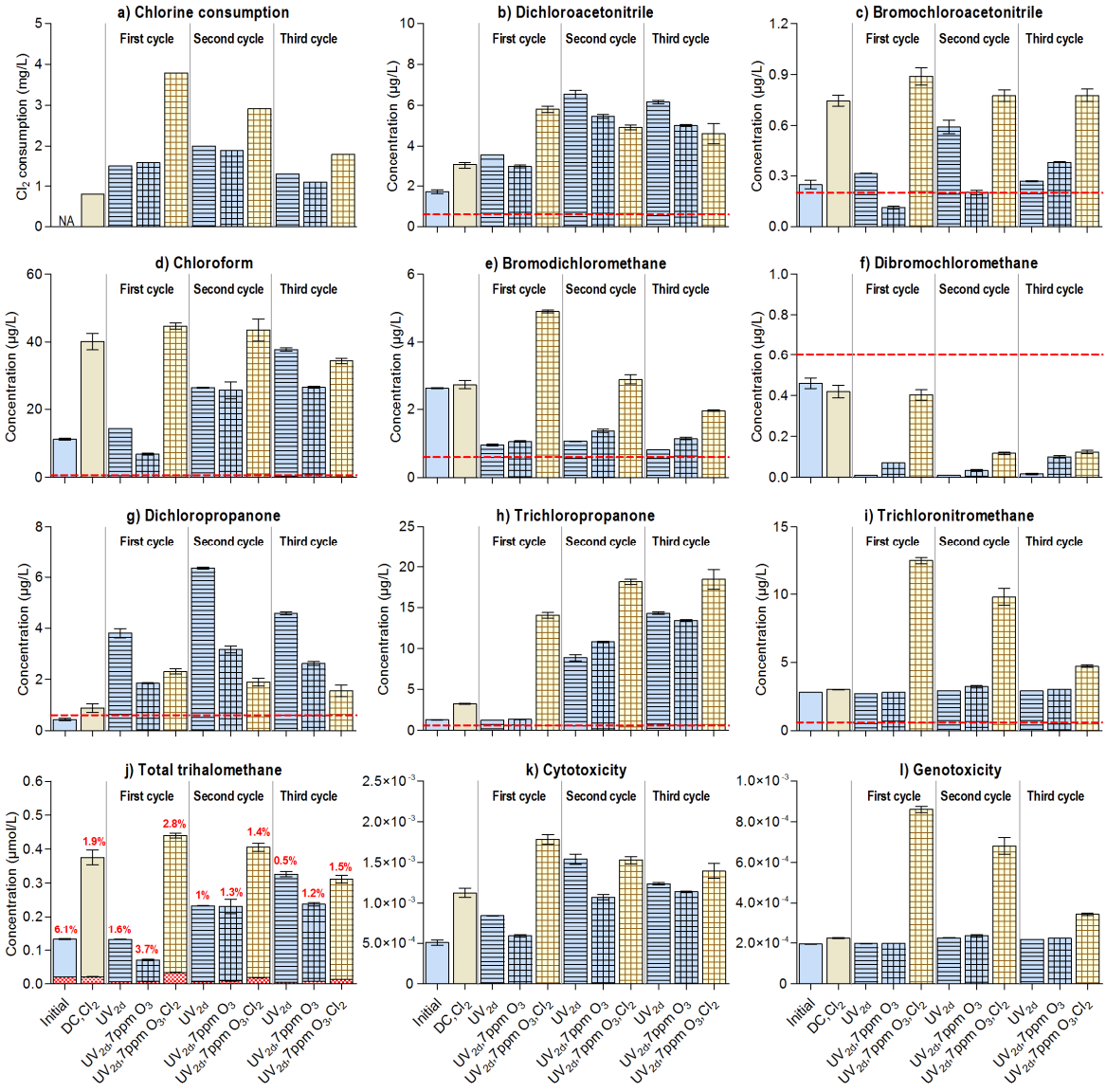
Table 1. Summary of the effect of ozone and UV on the formation of DPB during chlorination and on the DBPs themselves. Bold indicates a high effect.

DBP	DBP formation during chlorination after treatment		Effect on the DBPs themselves	
	Increase	Decrease	Increase	Decrease
Dichloroacetonitrile	**UV**, O_3	O_3	UV*	O_3
Bromochloroacetonitrile	**UV**, O_3	O_3	-	UV
Chloroform	**UV**, O_3	O_3	-	-
Bromodichloromethane	**UV**, O_3	O_3	-	UV
Dibromochloromethane	**UV**, O_3	O_3	-	**UV**
Dichloropropanone	**UV**, O_3	O_3	UV*	O_3
Trichloropropanone	**UV**, O_3	-	-	UV
Trichloronitromethane	UV, **O_3**	-	-	**UV**

* Confirmed in Spiliotopoulou et al. (2015)







Highlights

- UV treatment increased the reactivity of pool water to both chlorine and ozone
- Ozonation of UV-treated water decrease chlorine reactivity
- Genotoxic trichloronitromethane formed by ozonation was removed with UV treatment
- Continuous UV/ozone treatment decreases chlorine by-product formation
- Continuous UV/ozone treatment predicted to improve chlorinated pool water quality