Optimal pH in chlorinated swimming pools – balancing formation of by-products

Kamilla M. S. Hansen, Hans-Jørgen Albrechtsen and Henrik R. Andersen

ABSTRACT

In order to identify the optimal pH range for chlorinated swimming pools, the formation of trihalomethanes, haloacetonitriles and trichloramine was investigated in the pH-range 6.5–7.5 in batch experiments. An artificial body fluid analogue was used to simulate bather load as the precursor for by-products. The chlorine-to-precursor ratio used in the batch experiments influenced the amounts of by-products formed, but regardless of the ratio the same trends in the effect of pH were observed. Trihalomethane formation was reduced by decreasing pH, but haloacetonitrile and trichloramine formation increased. To evaluate the significance of the increase and decrease of the investigated organic by-products at the different pH values, the genotoxicity was calculated based on literature values. The calculated genotoxicity was approximately at the same level in the pH range 6.8–7.5 and increased when pH was 6.7 or lower. An optimal pH range for by-products formation in swimming pools was identified at pH 7.0–7.2. In the wider pH range (pH 6.8–7.5), the effect on by-product formation was negligible. Swimming pools should never be maintained at lower pH than 6.8 since formation of both haloacetonitriles and trichloramine increase significantly below this value.

Key words | disinfection by-products, haloacetonitrile, pH, swimming pool, trichloramine, trihalomethane

Kamilla M. S. Hansen
Hans-Jørgen Albrechtsen
Henrik R. Andersen (corresponding author)
Department of Environmental Engineering,
Technical University of Denmark,
Building 113,
Miljøvej,
2800 Kgs. Lyngby,
Denmark
E-mail: *henrik@ndersen.net*

INTRODUCTION

Swimming pools are used for recreation, rehabilitation purposes and physical activity and it is therefore imperative that the water and air quality are safe for the health of bathers. Chlorination is the most frequently applied method for controlling pool water quality and preventing spreading of pathogenic diseases among bathers. Chlorine exhibits a pH- and temperature-dependent equilibrium between the hypochlorous acid (HOCl) and the hypochlorite ion (OCl^-), with the sum of the two commonly known as free chlorine (White 1992). Hypochlorous acid is significantly more effective than hypochlorite as a bactericide and in inactivating viruses, cysts and spores. Therefore it is crucial to closely monitor and control pool water pH to ensure disinfection effectiveness (White 1992).

In addition to pathogens, swimming pool water is polluted by organic matter deposited by the bathers, such as saliva, urine and sweat, hair, moisturizing lotions and sunscreens, which together are known as bather load (Judd & Bullock 2003; Zwiener *et al.* 2007). Since chlorine is a strong oxidant, it oxidizes the organic matter from the bather load and the natural organic matter (NOM) in the source water and a minor fraction of this forms chlorinated organic compounds commonly known as disinfection by-products (DBPs). A recent study identified over 100 DBPs in pool water and reported a higher fraction of nitrogen-containing DBPs than typically found in chlorinated drinking water with several of the chemicals not identified in drinking water (Richardson *et al.* 2010). Nitrogen-containing DBPs are generally regarded as more toxic than carbon-based DPBs (Plewa *et al.* 2008).

The DBPs may affect human health. Since some of the swimming pool DBPs are also found in chlorinated drinking

doi: 10.2166/wh.2013.156

water, their genotoxicity, carcinogenicity and health effect risks have been studied. However, swimming pool waters are substantially more genotoxic than their source tap water (Liviac *et al.* 2010) which is likely due to the nitrogen-rich precursors from the bather load. Furthermore, a recent study on bladder cancer found a clear increased risk associated with chlorination by-products in drinking water and indicated use of swimming pools further increased the risk (Villanueva *et al.* 2007).

However, due to the lack of alternatives, the continued use of chlorine as a disinfectant for public swimming pools is the most realistic immediate future scenario. There is therefore a need for methods to minimize the level of DPBs in chlorinated swimming pools.

One approach to decrease DBP formation could be to reduce the chlorine concentration and the pH value, so that the HOCl concentration, and thus the disinfection efficiency, remains constant because HOCl is a stronger disinfectant than OCl^-. This was supported by a Danish full scale study on a public indoor pool, where the DBP formation at 0.4 mg chlorine/L at pH = 6.7, was compared with 1.5 mg/L of chlorine at pH = 7.3. At the lower pH, the trihalomethanes (THMs), absorbable organic halogen (AOX) and combined chlorine decreased while microbiological quality was maintained at the lower pH (Kristensen *et al.* 2007). However, in previous studies we found that at decreased pH the formation of the more toxic DBP group, haloacetonitriles (HANs) increased, when organic matter from pool water (Hansen *et al.* 2012a) or particles from a swimming pool filter (Hansen *et al.* 2012b) reacted with chlorine in a laboratory study. Increased formation of NCl_3 at lower pH in pools has been known for decades (Palin 1950; Schmalz *et al.* 2011; Hansen *et al.* 2012a).

Our previous studies (Hansen *et al.* 2012a, b) were conducted with intervals of 0.5 pH unit from 6.0 to 8.0 and clearly demonstrated a marked pH effect on the formation of DBPs groups. However, due to the wide steps between the tested pH values it is difficult to identify the optimal pH value where a minimal formation can be expected of both THMs and haloacetic acids, the formation of which is favoured at high pH and HANs, and trichloramine, the formation of which is favoured at low pH. Another limitation with the published laboratory studies by Hansen *et al.* (2012a, b) is that the reported laboratory experiments were all performed with a fixed ratio between chlorine and precursors. This ratio varies greatly in swimming pools with actual bathing load and the formation of DBPs is known to depend on the ratio between chlorine concentration and organic matter (Kanan 2010; Schmalz *et al.* 2011; Hansen *et al.* 2012a).

To address the importance of the latter-mentioned lack of exploration of the effect of the ratio between chlorine and precursors in the previous studies and to obtain an estimation of the optimal range for pH in swimming pools in terms of minimizing DBP formation, this investigation was carried out. Thus the first aim of this study was to investigate the effect of the ratio between chlorine and precursors in the batch experiment design and to perform an experiment to clarify the effect of adding the chlorine as one initial high dose compared with adding chlorine several times in smaller doses. Further, the second aim for the study was to identify the optimal pH in swimming pool waters for minimizing the formation of DBPs and therefore the effect of pH on the formation of DBPs was investigated in the pH-range 6.5–7.5.

To make experiments repeatable and comparable to previous studies, an artificial mixture of precursors that simulates sweat and urine was used as the by-products' precursor in all the experiments. In order to compare the different concentration of DBPs at each pH value investigated, the predicted genotoxicity was calculated in the assay water by calculation based on the genotoxic potency of each measured DBP.

METHODS

Chemicals

All chemicals and standard solutions were purchased from Sigma-Aldrich.

Analysis

Free and combined chlorine

The residual chlorine and the pH were measured at the beginning and the end of each experiment. The hypochlorite

stock solution (~10% w/w, Sigma-Aldrich) and the free and total chlorine of the samples were measured with a photometer (DR 2800, Hach Lange) using the diethyl-*p*-phenylenediamine method from a cell test kit (LCK 310, Lange).

Volatile organic DBPs

The THMs (chloroform, bromodichloromethane, dibromochloromethane, and bromoform) and HANs (trichloroacetonitrile, dichloroacetonitrile, bromochloroacetonitrile, and dibromoacetonitrile) were analysed as previously described (Hansen *et al*. 2012b, 2013). In short, free chlorine in THM and HAN samples was quenched by adding ammonium chloride (Munch & Hautman 1995; American Public Health Association [APHA] 2005) to 40 mL borosilicate glass vials before they were filled head-space-free with the sample. The samples were analysed the same day by Purge and Trap (Velocity XPT Purge and Trap Sample Concentrator, Teledyne Tekmar, with autosampler AQUATek 70, Teledyne Tekmar) coupled to a gas chromatograph with mass spectroscopy detection (GC-MS) (HP 6890 Series GC System – 5973 Mass selective detector, Hewlett Packard). This method was also used for the analysis of trichloronitromethane, dichloropropanone, and trichloropropanone.

Trichloramine

Trichloramine was measured with a method for air analysis described by Hery *et al*. (1995) with modifications described by Lützenkirchen & Breuer (2006) (for details see Hansen *et al*. 2012a). Trichloramine was stripped from the water by aerating the sample for 20 min while the trichloramine in the off air was trapped on a filter where it was reduced to chloride by reaction with arsenic trioxide. The filter was equipped with a protecting column consisting of silica gel impregnated with sulphamic acid, which blocks airborne water droplets containing chloride, as well as monochloramine and dichloramine reaching the filter. The purge time for samples for trichloramine analysis was checked by purging selected samples with high trichloramine content, a second time for another 20 min. These samples gave results similar to the blank values. Chloride was subsequently measured by ion chromatography (Dionex, ICS-1500 with Ion Pac AS 14A column). For more details see the supplementary material of Hansen *et al*. (2012a).

Experiments performed

Volatile organic DBPs

The experiments were performed using phosphate-buffered MilliQ water (50 mM). Sodium hydroxide and sulphuric acid were used to achieve pH at 6.5, 6.7, 6.8, 6.9, 7.0, 7.2 and 7.5.

The body fluid analogue (BFA) used for experiments consisted of ammonia (2.00 g/L), urea (14.8 g/L), creatinine (1.80 g/L), histidine (1.21 g/L), hippuric acid (1.71 g/L), uric acid (0.49 g/L) and citric acid (0.64 g/L) (Judd & Bullock 2003). Based on the concentration of each compound and their carbon content, the total theoretical organic carbon (TOC) was determined to be 5.73 g/L.

The chlorination experiments were carried out as batch experiments. Freshly produced reversed osmosis water was buffered with phosphate buffer (50 mM) at pH 6.5, 6.7, 6.8, 6.9, 7.0, 7.2 and 7.5, followed by the addition of BFA corresponding to 1.0 mg/L (71 μmol/L) TOC. The chlorine was added as an initial dose of 35 and 10 mg/L or as a dose of 10 mg/L divided over three additions (3×3.33 mg/L). Since the chlorine is basic, an equivalent amount of sulphuric acid was added each time chlorine was added. The experiments were performed in head-space-free 40 mL borosilicate glass bottles sealed with a polytetrafluoroethylene (PTFE) seal and each combination of chlorine and pH level was performed as triplicates. At pH 7.0, control experiments were conducted with addition of either BFA or chlorine (35 mg/L). All bottles were kept at 28 °C for 48 h. Afterwards, the pH and concentration of chlorine, combined chlorine, THMs and HANs were measured.

Trichloramine

The experiments with formation of trichloramine were conducted as described above expect for the size of the bottles. For pH 6.5, 6.7 and 6.8 bottles of 500 mL were used, whereas for pH 6.9, 7.0 and 7.2 bottles of 1,000 mL were used. Furthermore, the chlorine was either added as an initial dose of 35 mg/L or 10 mg/L and reaction time of

24 h at 28 °C was applied. Each combination of pH and chlorine dose was performed as duplicates.

Estimation of genotoxicity

Based on the measured concentration of the different DBPs, the genotoxicity was 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 the EC_{50} values used were from the *in vitro* cellular assay based on Chinese hamster ovary cells in which the genotoxicity was measured by single cell gel electrophoresis (Plewa *et al.* 2002, 2008; Muellner *et al.* 2007). All the measured compounds in this study have been tested in this assay, except dichloropropanone and trichloropropanone which were not detected in the experiments. The EC_{50} values used for the calculations are summarized in Table S2 in the supplementary material of Hansen *et al.* (2012a).

RESULTS AND DISCUSSION

Reaction of chlorine

In the experiment where the chlorine was added as 3 times 3.33 mg/L, the chlorine level after the first addition was measured and the next chlorine dose was added when the measured concentration was under 1 mg/L. Thus the chlorine was added in the beginning, after 5 h and again after 23 h. The addition of chlorine as one initial dose of 10 mg/L and the divided dosage (3×3.33 mg/L) resulted in different chlorine concentration profiles as shown in Figure 1.

The initial consumption of chlorine was high when adding chlorine as one initial dose of 10 mg/L in the beginning (Figure 1) and the chlorine concentration was reduced by half after 5 h. Then the consumption was reduced and another reduction by half was obtained after an additional 18 hours. In the last 25 h, there was almost no consumption

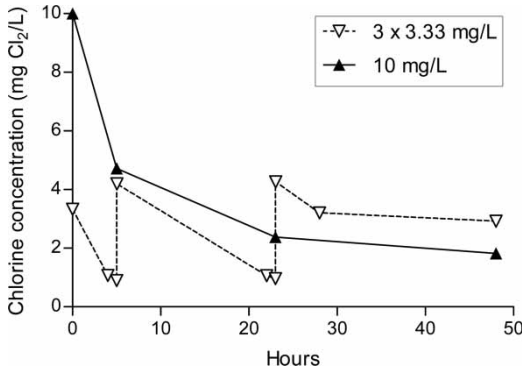

Figure 1 | Profiles of chlorine concentration when adding chlorine as one initial dose of 10 mg/L and divided dosing (3×3.33 mg/L) at pH 6.9. Experimental conditions: BFA = 1 mg/L as TOC, temperature = 28 °C and reaction time = 48 h.

of chlorine. When adding the chlorine as 3×3.33 mg/L, the initial consumption was high as well and the concentration decreased to below 1 mg/L within 5 h. After the next addition of chlorine, the consumption was lower and it took 18 h before the concentration was decreased to below 1 mg/L again. After the last addition of chlorine, the chlorine concentration did not decrease to 1 mg/L before the end of the experiment which was after 48 h. The final concentrations of chlorine after 48 h were very similar regardless of how the chlorine was added (10 mg/L vs. 3×3.33 mg/L).

In the experiments with formation of volatile organic DBPs, the concentration of chlorine was measured at the end. In the experiment where 35 mg/L of chlorine was added, only 20–30% of the initial chlorine was consumed and there was no correlation between pH and chlorine consumption (Figure 2). The experiment with 10 mg Cl_2/L had chlorine consumption at approximately 70% regardless of whether the chlorine was added as a single dose or divided over three smaller doses. Thus the addition of chlorine as an initial high dose or several small doses has a relatively small effect on the reaction of chlorine. The average consumption of chlorine in the experiments with 10 mg/L was 7.0 ± 0.3 mg/L for 3×3.33 mg/L and 7.1 ± 0.1 for 10 mg/L, while the average consumption for addition of 35 mg/L was 8.3 ± 1.5 mg/L. Consequently, the chlorine consumption was similar regardless of the chlorine-to-precursor ratio in which chlorine was added. This supports that a simple experimental design for reaction of BFA with chlorine in water where a single dose of chlorine is added in

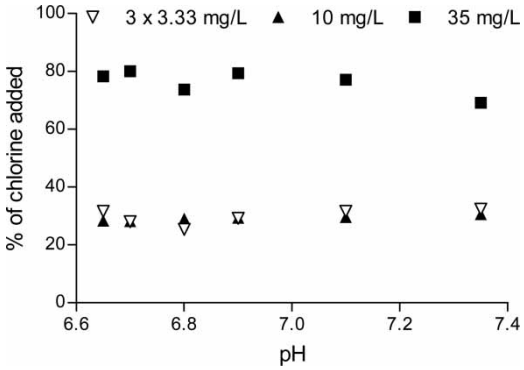

Figure 2 | Fraction of free chlorine remaining after 48 h as % of the added chlorine. Experimental conditions: BFA = 1.0 mg/L as TOC, temperature = 28 °C and reaction time = 48 h.

the beginning of the experiment, can give valid results similar to more advanced experimental designs where the chlorine is added continuously or in smaller portions during the reaction period in order to avoid a high initial chlorine concentration.

Effect of chlorine-to-BFA ratio on DBP formation

Recent studies have used similar batch tests to investigate DBP formation related to chlorination of swimming pool waters (Kanan 2010; Schmalz *et al*. 2011; Hansen *et al*. 2012a, 2012b) and have found that the chlorine-to-precursor ratio will affect the formation of DBPs (Kanan 2010; Schmalz *et al*. 2011; Hansen *et al*. 2012a).

To investigate the effect of different chlorine-to-precursor ratios and pH on the DBP formation, experiments with 0–5 mg C/L to 35 mg Cl_2/L were carried out at pH 6.7 and pH 7.5. The results are shown in Figure 3.

Experiments with 0 mg/L BFA and 35 mg Cl_2/L were considered as experimental blanks. Dichloroacetonitrile was detected at low levels, while trichloroacetonitrile was detected in most cases at a concentration below the formal limit of quantification (LOQ).

The formation of the volatile organic DBPs generally increased with BFA to chlorine. In all the different chlorine-to-precursor ratios the chloroform formation was higher at pH 7.5 than 6.7, whereas for the haloacetonitriles the formation was highest at the lowest pH. Thus the same trend in the effect of pH on the DBP formation was observed regardless of the ratio of chlorine-to-precursor.

Effect of pH on formation of organic DBPs

The formation of volatile organic DBPs by reaction of different chlorine-to-precursor ratios with 35 mg/L of chlorine is depicted in Figure 4. Only chlorinated DBPs were observed

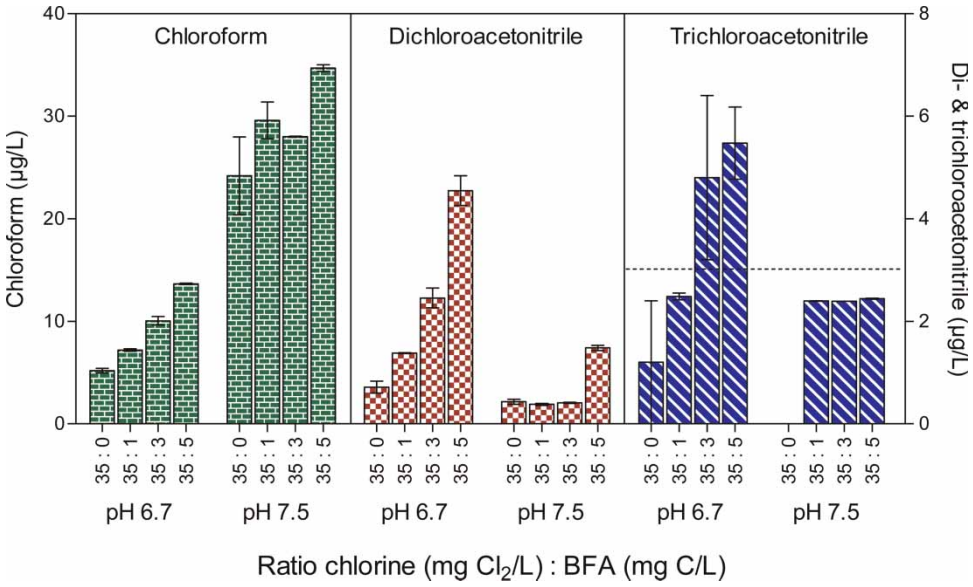

Figure 3 | Effect of reaction ratio between chlorine and organic matter on formation of DBPs at high and low pH. The punctuated line indicates the LOQ and the T-bars indicate the standard error of the mean ($n = 2$).

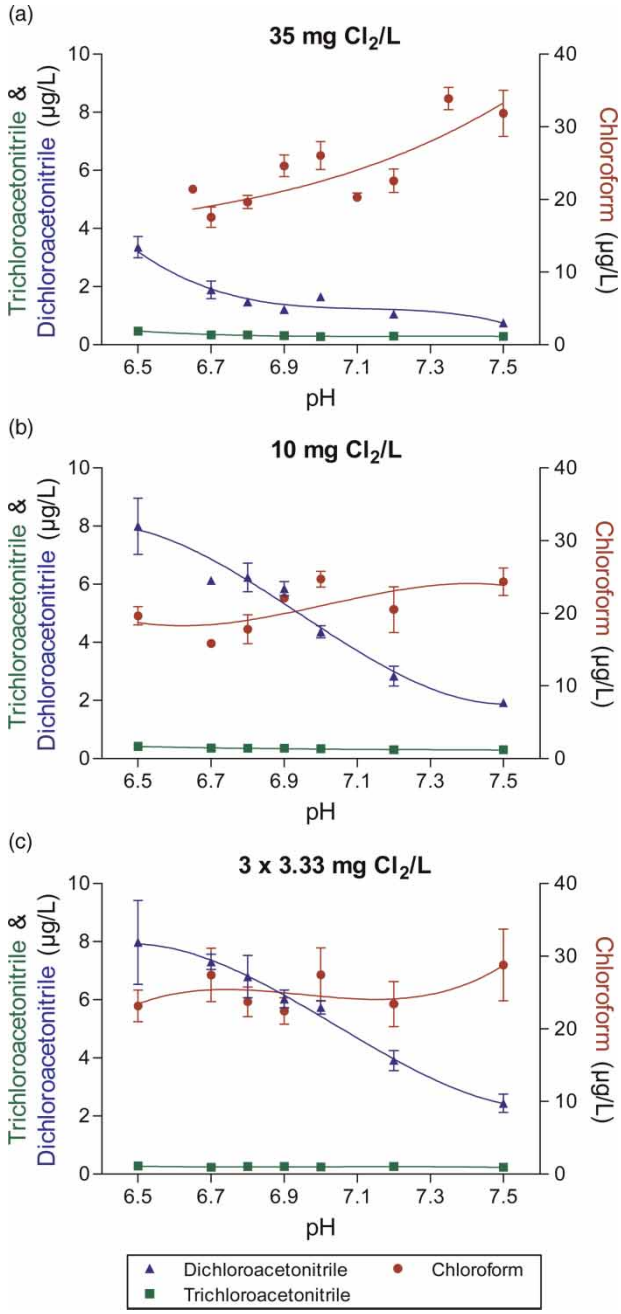

Figure 4 | Formation of chloroform, di- and trichloroacetonitrile at different chlorination levels. Experimental conditions: BFA = 1.0 mg/L as TOC, temperature = 28 °C and reaction time = 48 h. T-bars indicate the standard error of the mean (n = 3–6). Trendlines are based on least square methods fitting of a cubic polynomial to the data. This is done to aid the readability of the graphs and there is no direct theory that dictates this curve shape should fit the data.

since bromide was not present in the experiments. Further trichloronitromethane, dichloropropanone, and trichloropropanone were not detectable in any samples. The pH effect on the formation of THMs and HANs is as expected from previous studies (Kanan 2010; Hansen *et al.* 2012a). The formation of chloroform increased with increasing pH whereas the dichloroacetonitrile decreased. In the experiment with 35 mg/L chlorine (Figure 4(a)), the formation of dichloroacetonitrile was almost stable within the pH interval of 6.8–7.2, but at pH 6.7 and lower the formation increased considerably. The concentration of trichloroacetonitrile is close to the limit of quantification for the analysis and no conclusion on the effect of pH can be drawn.

The results for the volatile organic DBPs from adding the chlorine as one initial dose are similar to those from adding chlorine as three smaller doses (Figures 4(b) and 4(c)). Since chlorine consumption and DBP formation are similar for both experiments (10 mg/L vs. 3×3.33 mg/L).

It is well known that changing the ratio between chlorine and precursors will affect the formation of DBPs. That was also the case in the experiments performed. In general, higher chloroform formation was found in the experiments with 35 mg/L compared with 10 mg/L. For the dichloroacetonitrile it was opposite and the highest formation was found in the experiments with 10 mg/L. The decrease in dichloroacetonitrile and increase in chloroform at 35 mg/L could be explained by the fact that the high chlorine dose results in further oxidation of the haloacetonitriles to chloroform or other DBPs. The same relation between chlorine and formation of haloacetonitriles and chloroform was found in a study by Hansen *et al.* (2012a).

Effect of pH on formation of trichloramine

The experiments with 35 mg Cl_2/L revealed a strong relation between pH and trichloramine formation. For pH < 7.2, an increased formation of trichloramine was found (Figure 5). For urea, Schmalz *et al.* (2011) found increased trichloramine formation at pH 7.1 (76%) compared with pH 7.7 (24%). Besides the effect of pH, the formation is also affected by the chlorine-to-nitrogen ratio (Schmalz *et al.* 2011). When the chlorine-to-nitrogen ratio decreased, the formation of trichloramine decreased (Figure 5) which is in accordance with the findings of Schmalz *et al.* (2011). Furthermore, at 10 mg Cl_2/L the increase at lower pH was not as large as

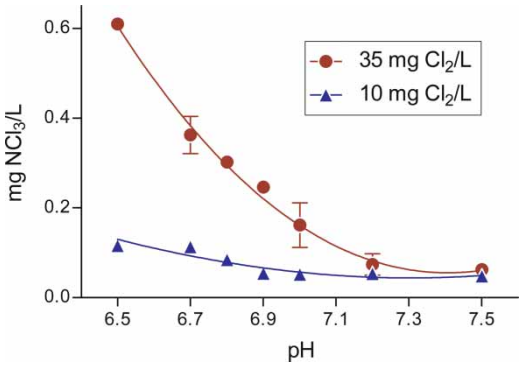

Figure 5 | Formation of trichloramine at different pH values. Experimental conditions: BFA = 1.0 mg/L as TOC, temperature = 28 °C and reaction time = 24 h. T-bars indicate the standard error of the mean ($n = 2$) except when the interval is smaller than the symbol used to indicate the mean. Trendlines are based on least square methods fitting of a cubic polynomial to the data. This is done to aid the readability of the graphs and there is no direct theory that dictates this curve shape should fit the data.

for the experiments at 35 mg/L and an increase in the formation was found at pH ≤ 6.8.

Predicted genotoxicity

To evaluate the significance of the changes in the formation of the investigated organic DBPs at the different pH, the genotoxicity was calculated for each sample. Taking the genotoxicity at 7.2 as the reference, it is seen that for pH ≥ 6.8 the genotoxicity was approximately stable while for pH ≤ 6.7 the toxicity increased (Figure 6). This is in accordance with previous results where it was found that comparable genotoxicity was estimated for pH 7.0, 7.5, and 8.0, while pH 6.5 had increased toxicity (Hansen *et al.* 2012a).

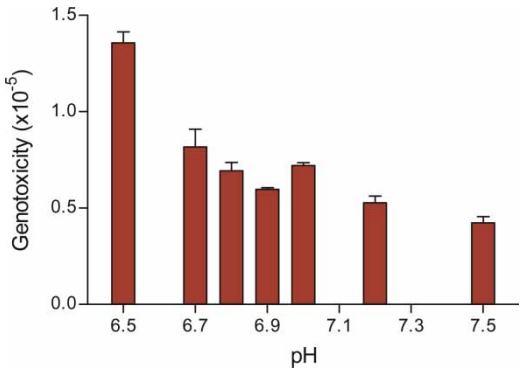

Figure 6 | The estimated genotoxicity of chlorinated BFA solution at 6.5 ≤ pH ≤ 7.5. T-bars indicate the standard error of the mean ($n = 2$).

CONCLUSION

- Whether the chlorine was added as an initial high dose or several small doses had relatively little effect on the DBP formation and the consumption of chlorine.
- The chlorine-to-precursor ratio influenced the amounts of DBPs formed, but regardless of the chlorine-to-precursor ratio the same trends in the effect of pH on DBP formation were observed.
- An opposite effect of pH was found for the formation of chloroform which was highest at high pH while the formation of the nitrogen-containing DBPs haloacetonitriles and trichloramine were high at low pH.
- The formation of chloroform increased above pH 7.2 whereas the formation of chloroform was low and independent of pH at lower pH-values.
- The haloacetonitrile formation, particular dichloroacetonitrile, increased with decreasing pH, but the increase is more pronounced at pH < 6.8.
- Swimming pools should be operated with pH not lower than 6.8 in order to limit the genotoxicity and trichloramine exposure and not higher than 7.2 in order to limit THM formation.

ACKNOWLEDGEMENTS

This study was partly funded by the Danish Nature Agency (Naturstyrelsen) from the Danish Ministry of the Environment. The authors recognise the technical assistance of Sabrina Nedell and Christina Hagberg.

REFERENCES

APHA 2005 *Standard Methods for the Examination of Water & Wastewater*. American Public Health Association, American Water Works Association and Water Environment Federation, Washington, DC.

Hansen, K. M. S., Willach, S., Antoniou, M. G., Mosbæk, H., Albrechtsen, H.-J. & Andersen, H. R. 2012a Effect of pH on the formation of disinfection byproducts in swimming pool water – Is less THM better? *Wat. Res.* **46** (19), 6399–6409.

Hansen, K. M. S., Willach, S., Mosbæk, H. & Andersen, H. R. 2012b Particles in swimming pool filters – does pH determine the DBP formation? *Chemosphere* **87** (3), 241–247.

Hansen, K. M. S., Zortea, R., Piketty, A., Vega, S. R. & Andersen, H. R. 2013 Photolytic removal of DBPs by medium pressure UV in swimming pool water. *Sci. Total Environ.* **443**, 850–856.

Hery, M., Hecht, G., Gerber, J. M., Gendre, J. C., Hubert, G. & Rebuffaud, J. 1995 Exposure to chloramines in the atmosphere of indoor swimming pools. *Ann. Occup. Hyg.* **39** (4), 427–439.

Judd, S. J. & Bullock, G. 2003 The fate of chlorine and organic materials in swimming pools. *Chemosphere* **51** (9), 869–879.

Kanan, A. 2010 Occurrence and formation of disinfection by-products in indoor swimming pools water. Dissertation, Environmental Engineering, Clemson University, South Carolina, USA.

Kristensen, G. H., Klausen, M. M. & Janning, K. 2007 Forsøgsdrift af varmtvandsbassin i Gladsaxe Svømmehal ved lavt indhold af frit klor og reduceret pH (Experimental Operation of a Warm Water Pool in Glasaxe Swimming Pool at Low Chlorine Level and pH; in Danish). *Report from DHI*, Denmark. www.dhigroup.com.

Liviac, D., Wagner, E. D., Mitch, W. A., Altonji, M. J. & Plewa, M. J. 2010 Genotoxicity of water concentrates from recreational pools after various disinfection methods. *Environ. Sci. Technol.* **44** (9), 3527–3532.

Lützenkirchen, C. & Breuer, D. 2006 *Stickstofftrichlorid* (*Kennzahl 8623*) (*Trichloramine, no. 8623; In German*) *In: BGIA - Arbeitsmappe Messung von Gefahrstoffen* (*BGIA - Institut für Arbeitsschutz, ed.*). Erich Schmidt Verlag, Berlin, Germany.

Muellner, M. G., Wagner, E. D., McCalla, K., Richardson, S. D., Woo, Y. T. & Plewa, M. J. 2007 Haloacetonitriles vs. regulated haloacetic acids: Are nitrogen-containing DBPs more toxic?. *Environ. Sci. Technol.* **41** (2), 645–651.

Munch, D. J. & Hautman, D. P. 1995 Determination of Chlorination Disinfection Byproducts, Chlorinated Solvents, and Halogenated Pesticides/Herbicides in Drinking Water by Liquid-Liquid Extraction and Gas Chromatography with Electron-Capture Detection - Method 551.1. Report from Environmental Protection Agency, Cincinnati, Ohio.

Palin, A. T. 1950 Chemical aspects of swimming bath treatment. *The Baths Service: J. Nat. Assoc. Bath Superintendents* **9**, 12–20.

Plewa, M. J., Kargalioglu, Y., Vankerk, D., Minear, R. A. & Wagner, E. D. 2002 Mammalian cell cytotoxicity and genotoxicity analysis of drinking water disinfection by-products. *Environ. Mol. Mutagen.* **40** (2), 134–142.

Plewa, M. J., Wagner, E. D., Muellner, M. G., Hsu, K. M. & Richardson, S. D. 2008 Comparative Mammalian Cell Toxicity of N-DBPs and C-DBPs. In: *Disinfection By-Products in Drinking Water: Occurrence, Formation, Health Effects, and Control* (T. Karanfil, S. W. Krasner & Y. Xie, eds.). American Chemical Society, Washington, DC, USA, pp. 36–50.

Richardson, S. D., DeMarini, D. M., Kogevinas, M., Fernandez, P., Marco, E., Lourencetti, C., Ballesté, C., Heederik, D., Meliefste, K., McKague, A. B., Marcos, R., Font-Ribera, L., Grimalt, J. O. & Villanueva, C. M. 2010 What's in the pool? A comprehensive identification of disinfection by-products and assessment of mutagenicity of chlorinated and brominated swimming pool water. *Environ. Health Persp.* **118** (11), 1523–1530.

Schmalz, C., Frimmel, F. H. & Zwiener, C. 2011 Trichloramine in swimming pools – formation and mass transfer. *Wat. Res.* **45** (8), 2681–2690.

Villanueva, C. M., Cantor, K. P., Grimalt, J. O., Malats, N., Silverman, D., Tardon, A., Garcia-Closas, R., Serra, C., Carrato, A., Castaño-Vinyals, G., Marcos, R., Rothman, N., Real, F. X., Dosemeci, M. & Kogevinas, M. 2007 Bladder cancer and exposure to water disinfection by-products through ingestion, bathing, showering, and swimming in pools. *Am. J. Epidemiol.* **165** (2), 148–156.

White, G. C. 1992 *Handbook of Chlorination and Alternative Disinfectants*. Van Nostrand Reinhold, New York, USA.

Zwiener, C., Richardson, S. D., DeMarini, D. M., Grummt, T., Glauner, T. & Frimmel, F. H. 2007 Drowning in disinfection byproducts? Assessing swimming pool water. *Environ. Sci. Technol.* **41** (2), 363–372.

First received 24 March 2013; accepted in revised form 5 June 2013. Available online 5 July 2013