



# Impact of UV disinfection on the potential of model organic-nitrogen precursors to form chlorination by-products in swimming pool water

Agnieszka Włodyka-Bergier\*, Tomasz Bergier

Faculty of Mining Surveying and Environmental Engineering, Department of Environmental Management and Protection, AGH University of Science and Technology, al. Mickiewicza 30, 30-059 Kraków, Poland, Tel. +48 12 617 47 57; Fax: +48 12 617 50 76; email: wlodyka@agh.edu.pl (A. Włodyka-Bergier)

Received 14 July 2014; Accepted 8 March 2015

#### ABSTRACT

The article presents the results of experiments on the potential of model solutions of urea, creatinine, glycine, histidine, and arginine to form several organic chlorination by-products. These precursors are the main organic-nitrogen compounds, whose sources in swimming pool water are sweat and urine of bathers. In the article, the formation potential of the following by-products has been presented and discussed: trichloromethane; monochloroacetic acid; dichloroacetic acid; trichloroacetic acid; trichloroacetonitrile; dichloroacetonitrile; 1,1-dichloropropanone; 1,1,1-trichloropropanone; chloral hydrate; and chloropicrin. The test on by-products formation potential was applied to conduct the experiments, and 24-h incubation time was applied for the swimming pool water samples, disinfected in three variants of treatment: (1) chlorination alone; (2) chlorination and Ultraviolet (UV) irradiation (cumulative dose 23.5 kJ/m<sup>2</sup>); and (3) chlorination and UV irradiation (cumulative dose 47 kJ/m<sup>2</sup>). The low-pressure UV lamp by Heraeus was used to irradiate the samples. The results of experiments have been discussed and analyzed to study the influence of UV radiation on the reactivity of individual model precursors and their potential to form halogenated organic chlorination by-products.

Keywords: Disinfection by-products; UV disinfection; Swimming pool water

## 1. Introduction

Ultraviolet (UV) disinfection is being increasingly used in water treatment. This process is highly effective at inactivating waterborne pathogens which are resistant to chlorine, such as *Cryptosporidium* [1]. UV irradiation in swimming pool water treatment is also used to reduce chloramine compounds concentration [2–4]. UV treatment has to be used in a sequence with chemical disinfectants to eliminate the secondary water contamination. In Poland, chlorine is a primary disinfectant to prevent microbial regrowth in swimming pool water [5]. However, using chlorine compounds is integrally connected with their reactivity and forming halogenated disinfection by-products (DBPs). The main organic DBPs identified in swimming pool water are trihalomethanes (THMs),

Presented at the 12th Scientific Conference on Microcontaminants in Human Environment 25–27 September 2014, Czestochowa, Poland

1944-3994/1944-3986 © 2015 Balaban Desalination Publications. All rights reserved.

<sup>\*</sup>Corresponding author.

haloacetic acids (HAAs), haloacetonitriles (HANs), haloketones (HKs), haloaldehydes (including chloral hydrate (CH)), halonitromethanes (HNMs) (including chloropicrin (CP)) [6–9]. These compounds are of scientific interest, due to the health effects they may have on human beings [10–16].

In swimming pools, the majority of DBPs formation is attributable to particles consisting mainly of hair and skin cells, dirt, cosmetics, soap residues, and organic-nitrogen (organic-N) precursors which are introduced into swimming pool water by bathers with human body fluids [2,17–22]. Among organic-N precursors, which are carried to swimming pool water with sweat and urine, urea is the main contaminant [17]. The others, such as: creatinine, arginine, histidine, and glycine occur in lower concentrations [23,2,18,19], Most organic-N precursors are carried to pool water with urine—one bather releases on average 25–30 mL of urine into swimming pools [18]. Typical composition of organic-N precursors in human urine and sweat is presented in Table 1.

In the case of drinking water treatment, UV irradiation alone at doses commonly used for disinfection does not appear to generate halogenated DBPs [24,1]; however, UV irradiation combined with chlorination can promote formation of several chlorinated DBPs (THMs, CH, HNMs) and can also increase the rate of free chlorine consumption [1,25–27].

In swimming pool water treatment, UV light sequenced with chlorination can lead an increase in the concentration of THMs (particularly trichloromethane (TCM)), CH, dichloroacetonitrile (DCAN), 1,1,1-trichloropropanone (1,1,1-TCP), and CP [3,28].

The influence of individual organic-N precursors on DBPs formation has been studied and described only in a few publications [2,19]. As Weng et al. [2] reported, chlorocreatinine was more sensitive to UV irradiation (254 nm) than creatinine. UV irradiation of chlorocreatinine leads to opening of the ring structure and such formed intermediates are more susceptible to the reaction with free chlorine and DBPs formation. In Weng's study, inorganic chloramines (NH<sub>2</sub>Cl, NHCl<sub>2</sub>, NCl<sub>3</sub>), organic chloramine (CH<sub>3</sub>NCl<sub>2</sub>) and a cyanogen chloride (CNCl) were examined. In other studies [19], it was reported that DCAN formation from L-histidine and L-arginine, CNCl from L-histidine, L-arginine and glycine were promoted by monochromatic UV light provided that chlorine was presented in solution.

As the above literature review has shown, our knowledge of the influence of UV-chlorine sequences on organic halogenated DBPs formation potential from organic-N precursors is very limited.

In this paper, the influence of UV-chlorine sequence disinfection on organic DBPs formation potential is examined. The experiments were conducted for the following organic-N precursors: urea, creatinine, glycine, histidine, and arginine, which are introduced into swimming pool water by bathers with human body fluids. Their potential to form: TCM, monochloroacetic acid (MCAA), dichloroacetic acid (DCAA), trichloroacetic acid (TCAA), trichloroacetonitrile (TCA), DCAN, 1,1dichloropropanone (1,1-DCP), 1,1,1-trichloropropanone (1,1,1-TCP), CH, and CP were analyzed.

## 2. Materials and methods

### 2.1. Preparation of organic-N precursors solutions

A stock solution of urea (POCh) and creatinine, glycine, histidine, and arginine (ACROS) was prepared by dissolving these compounds in ultra-pure water from the Micro Pure Water Purification System (Thermo), to which phosphate buffer was added. The concentration of all solutions  $(1.8 \times 10^{-5} \text{ mol/L})$  was adopted from the work of Weng et al. [19].

## 2.2. UV/chlorination experiment

The procedure of identifying the DBPs formation potentials of individual organic-N precursors in swimming pool water disinfected with UV-chlorine

Table 1 Organic-N precursors in human body fluids [18,19]

Compound	Sweat		Urine				
	WHO [18], (mg/L)	Weng et al. [19], (mg/L)	WHO [18], (mg/L)	Weng et al. [19], (mg/L)			
Urea	680	1,177.2	10,240	16,697.7			
Creatinine	7	1.2	640	1,210.4			
Glycine	N.A. <sup>a</sup>	N.A. <sup>a</sup>	N.A. <sup>a</sup>	129.1			
Histidine	N.A. <sup>a</sup>	N.A. <sup>a</sup>	N.A. <sup>a</sup>	119.0			
Arginine	N.A. <sup>a</sup>	N.A. <sup>a</sup>	N.A. <sup>a</sup>	2.9			

<sup>a</sup>Data not available.

sequence was adopted from the work of Cimetiere and Laat [3]. In the method, proposed by these authors, water is irradiated with cumulative UV doses  $(23.5-47.0 \text{ kJ/m}^2)$ . Both before and after each irradiation, free chlorine concentration was adjusted to 3 mg/L. In the experiment, monochromatic UV lamp TNN 15/32 by Heraeus was used. The procedure has been adapted to a photoreactor, in which experiments were conducted (the details are presented in Fig. 1).

For each individual organic-N precursor, 3 samples were taken: (1)  $Cl_2$ —water only chlorinated; (2) Cl<sub>2</sub> + UV1-water chlorinated and treated with UV dose 23.5 kJ/m<sup>2</sup> and chlorinated again; (3)  $Cl_2 + UV2$ —water chlorinated, treated with UV dose  $23.5 \text{ kJ/m}^2$ , chlorinated, treated with UV dose 23.5 kJ/m<sup>2</sup> (cumulative UV dose  $47.0 \text{ kJ/m}^2$ ), and chlorinated. On all stages of the experiment, the free chlorine concentration was adjusted to  $3.0 \pm 0.2 \text{ mg/L}$ , using sodium hypochlorite. The free chlorine concentration was determined using the DPD (N,N-diethylphenylendiamine) method (according to the Polish Standard PN-ISO 7393-2). The free chlorine concentration was measured using the Aurius 2021 UV-vis spectrophotometer (Cecil Instruments). The detection limit of the method was 0.03 mg/L.



Fig. 1. Experimental procedures scheme.

After each stage of the experiment, water samples were put into dark-glass bottles of volume 250 mL, with the silicon sealing covered with PTFE. The bottles were completely filled to avoid air bubbles. The chlorinated water samples were incubated at  $25 \pm 2$ °C. After 24 h, these samples were dechlorinated and DBPs were analyzed.

### 2.3. DBPs analysis

After 24-h incubation time, the concentration of TCM; MCAA; DCAA; TCAA; TCAN; DCAN; 1,1-DCP; 1,1,1-TCP; CH; and CP was analyzed in all samples. Prior to the analysis of HAAs, the samples were dechlorinated with sodium sulfite, while prior to the analysis of the rest of volatile DBPs, the samples were dechlorinated with ascorbic acids.

The DBPs concentrations were analyzed using a gas chromatograph with a Trace Ultra DSQII GC-MS mass spectrometer (Thermo Scientific). Helium was used as a carrier gas. The Rxi<sup>TM</sup>-5 ms capillary column (Restek) was used (film thickness  $0.5 \,\mu$ m, column length 30 m, column diameter  $0.25 \,\text{mm}$ ). The TCM, HANs, HKs, CH, and CP were extracted using the liquid–liquid extraction method with methyl tert-butyl ether. The HAAs concentrations were analyzed using an acidic methanol esterification method. Details of the analytical procedure of these individual compounds were presented in the article [29].

# 3. Results and discussion

The concentrations of individual DBPs in each of organic-N solutions have been presented in Table 2. Fig. 2 presents the DBPs formation potential of individual organic-N precursors for TCM (Fig. 2(A)); for sum of TCAN and DCAN (Fig. 2(B)); for the sum of 1,1-DCP and 1,1,1-TCP (Fig. 2(C)); for CH (Fig. 2(D)); for CP (Fig. 2(E)); and for the sum of MCAA, DCAA, and TCAA (Fig. 2(F)).

# 3.1. Trichloromethane

As it can be observed in Fig. 2(A), TCM only in chlorinated samples (Cl<sub>2</sub>) was formed in a low amount by all organic-N precursors. In this case, TCM concentration was 0.97  $\mu$ g/L for urea, 2.41  $\mu$ g/L for creatinine and 2.42  $\mu$ g/L for glycine. In the samples irradiated with the UV dose of 23.5 kJ/m<sup>2</sup> (Cl<sub>2</sub> + UV1), an increase in TCM concentration was observed for urea (to the level of 2.17  $\mu$ g/L, thus the concentration increased by123%), glycine (to 2.82  $\mu$ g/L, 16%) and histidine (to 7.26  $\mu$ g/L, 243%). In the solution of

Table 2

DBPs concentration	in	solution	of	urea,	creatinine,	glycine,	histidine,	and	arginine	treated	with	chlorine	and/or	UV
radiation														

	DBPs concentration in organic-N precursor solution, ( $\mu$ g/L)										
Compound	Urea	Creatinine	Glycine	Histidine	Arginine						
Cl <sub>2</sub>											
TCM	0.97	2.41	2.42	2.12	1.64						
TCAN	0.05	0.01	0.02	0.21	0.07						
DCAN	0.26	0.36	0.70	36.07	0.42						
1,1-DCP	0.89	1.53	1.15	2.09	0.59						
1,1,1-TCP	0.68	0.80	0.81	0.25	0.70						
CH	0.38	0.90	2.70	22.47	1.20						
СР	0.19	0.25	0.18	0.14	0.17						
MCAA	0.39	0.50	3.24	2.78	1.03						
DCAA	1.59	2.09	1.64	11.45	2.25						
TCAA	2.47	2.39	2.41	5.84	3.67						
$Cl_2 + UV1$											
TCM	2.17	1.31	2.82	7.26	1.59						
TCAN	0.02	0.01	0.01	0.27	0.06						
DCAN	0.32	0.26	0.84	45.19	2.69						
1,1-DCP	1.41	1.03	1.07	0.77	1.13						
1,1,1-TCP	0.74	0.11	0.68	0.30	1.19						
СН	1.27	1.11	1.51	94.67	13.87						
СР	0.42	27.96	4.49	1.39	0.34						
MCAA	0.82	0.74	1.68	3.59	1.83						
DCAA	1.84	3.91	3.11	30.38	8.83						
TCAA	2.91	4.75	4.67	16.95	5.60						
$Cl_2 + UV2$											
TCM	10.22	3.64	2.34	8.24	2.06						
TCAN	0.02	0.06	0.03	0.14	0.03						
DCAN	0.44	0.20	0.48	42.32	7.33						
1,1-DCP	1.47	0.76	0.43	0.44	2.32						
1,1,1-TCP	0.83	0.42	1.10	0.52	1.55						
СН	1.94	2.35	2.19	101.47	24.88						
СР	0.85	41.45	1.49	0.68	0.50						
MCAA	1.17	0.97	1.35	10.30	1.18						
DCAA	6.62	3.77	4.76	35.81	13.99						
TCAA	6.49	3.97	5.31	20.10	6.06						

arginine, a minor decrease in the concentration of TCM was observed (to  $1.59 \ \mu g/L$ , -3%), as well as of creatinine (to  $2.41 \ \mu g/L$ , -45%). In the samples irritated with the cumulative UV dose  $47 \ \text{kJ/m}^2$  (Cl<sub>2</sub> + UV2) for all organic-N precursors (except glycine), an increase in TCM concentration was noticed. For urea, the concentration of TCM increased more than 10 times when compared to the solution, which was only chlorinated (to  $10.22 \ \mu g/L$ , 950%), as well as for creatinine (to  $3.64 \ \mu g/L$ , 51%), histidine (to  $8.24 \ \mu g/L$ , 289%), and arginine (to  $2.06 \ \mu g/L$ , 26%). While for glycine, a minor decrease in TCM concentration was observed (to  $2.34 \ \mu g/L$ , -3%). In other experiments, conducted on water with the higher concentration of urine and free residual chlorine, the

more intensive formation of TCM was reported [7]. In the experiments presented in this article, the significant influence of urine compounds on TCM formation was not observed. However, in the conditions of real swimming pool, the higher concentration of urine is usually correlated with the presence of other contaminants carried with bathers such as particles consisting hair and skin cells, which could also play some role in TCM formation precursors [21]. The presented results proved that UV radiation had a significant influence on the formation of TCM from the substances carried with human body fluids to swimming pool water. Among the organic-N precursors, the highest importance in TCM formation in water treated with UV-chlorine sequence has urea (which has a high



Fig. 2. DBPs formation potential of organic-N precursors: (A) TCM, (B) haloacetonitriles, (C) HKs, (D) CH, (E) CP, and (F) HAAs (graphs B, D, and E are presented in a logarithmic scale).

share in human urine) and histidine (low share in human urine) [18,19] (see Table 1).

## 3.2. Haloacetonitriles

Some other researchers reported the more intensive formation of DCAN in swimming pool water caused by the presence of hair, lotion, saliva, skin, and urine, which are carried to water by bathers [7,30]. There are also some reports on the significant increase in DCAN concentration in swimming pool water as an effect of UV irradiation [3]. The experiments conducted by Weng et al. [19] showed that UV irradiation (254 nm) promoted DCAN formation from histidine and arginine, as long as free chlorine was present in the solution. The experiments described in this article also showed the high potential of histidine and arginine to form compounds from HANs group (Fig. 2(B)). TCAN was formed in a very low quantity (<0.1  $\mu$ g/L), and UV radiation had no significant influence on this process. Only in the case of histidine slightly higher concentration of TCAN was observed-it was 0.21, 0.27 and 0.14  $\mu$ g/L for water, respectively, only chlorinated, irradiated in a variant  $Cl_2 + UV1$  and  $Cl_2 + UV2$ . But even in this case TCAN concentration was low. DCAN concentration for urea, creatinine and glycine was also low-ranged from 0.20 µg/L for creatinine solution irradiated in  $Cl_2 + UV2$  variant to 0.84  $\mu g/L$ for glycine solution irradiated in Cl<sub>2</sub> + UV1 variant. For arginine solution, a significant influence of UV radiation on DCAN formation was observed. DCAN concentration after 24-h incubation was 0.42, 2.69 (increased by 540%) and 7.33  $\mu$ g/L (1,645%) in the samples, respectively: only chlorinated, irradiated in Cl<sub>2</sub> + UV1 and Cl<sub>2</sub> + UV2 variants. Histidine showed the highest potential to form DCAN. In the case of this organic-N precursor, a strong influence of UV radiation on DCAN formation was also observed. DCAN concentration in only chlorinated sample was  $36.07 \,\mu g/L$ , while after irradiation with a dose UV1 and UV2, DCAN concentration increased, respectively, to 45.19  $\mu$ g/L (25%) and 42.32  $\mu$ g/L (17%). During the experiments, the additional chlorination was conducted to adjust the free chlorine concentration to 3 mg/L, after each irradiation of the organic-N precursors solution. After the irradiation of water, in which the free chlorine concentration was 3 mg/L, with UV dose of UV 23.5 kJ/m<sup>2</sup>, due to dechlorination free chlorine concentration decreased to the level of about 1.5 mg/L. Thus, this higher dose of chlorine in treated with UV samples is a possible reason why the concentration of DCAN (and some other tested DBPS) increased after UV irradiation. However, some research reports (e.g. Weng et al. [19]) show that UV irradiation of organic-N precursors: creatinine, glycine, histidine and arginine increases the chlorine consumption in water. Histidine constitutes a very low percentage among the urine components, therefore-during the presented experiments-it was impossible to observe high concentration of DCAN nor the strong influence of UV radiation on its formation, despite such a high potential of this substance to form DCAN in swimming pool water.

#### 3.3. Haloketones

The formation of compounds from HKs group (1,1-DCP and 1,1,1-TCP) in chlorinated water ranged from 1.29  $\mu$ g/L for arginine to 2.33  $\mu$ g/L and 2.35  $\mu$ g/L for

creatinine and histidine. The influence of UV radiation on HKs concentration was observed in urea and arginine solutions. This effect was stronger for arginine —HKs concentration increase was  $1.03 \,\mu\text{g/L}$  (80%) for  $Cl_2 + UV1$  variant and 2.58 µg/L (201%) for  $Cl_2 + UV2$ , while for urea, it was 0.58 (37%) and 0.73  $\mu$ g/L (46%), respectively. The irradiation of creatinine, glycine, and histidine solutions resulted in the decrease in HKs concentration—for creatinine, it was  $1.19 \,\mu\text{g/L} (-51\%)$ in  $Cl_2 + UV1$  variant and  $1.15 \,\mu g/L$  (-49%) in  $Cl_2 + UV2$ , for glycine—respectively, 0.21 µg/L (-11%) and  $0.42 \,\mu\text{g/L}$  (-22%), and for histidine  $1.28 \,\mu\text{g/L}$ (-55%) and  $1.38 \mu g/L$  (-59%). The compounds from this group are relatively rarely examined and studied by other authors. Cimetiere and De Laat [3] in their laboratory experiments on swimming pool water proved that UV treatment significantly increased the concentration of 1,1,1-TCP; however, this effect was not observed for 1,1-DCP.

## 3.4. Chloral hydrate

CH is one of the most abundant DBPs in swimming pool water, and UV treatment can increase its formation [3]. The conducted experiments proved that urea, creatinine, glycine, and arginine had no significant influence on CH formation in swimming pool water, which was only chlorinated (concentration from  $0.38 \,\mu\text{g/L}$  for urea to  $2.70 \,\mu\text{g/L}$  for glycine), and CH concentration in chlorinated histidine solution reached the level of 22.47  $\mu$ g/L (Fig. 2(D)). For urea, creatinine, and glycine, no significant influence of UV radiation on CH formation was observed, while in the case of histidine and arginine, the higher dose of UV caused the formation of CH in higher quantity. Especially, the high increase in CH concentration was observed for histidine-it was 72.20  $\mu$ g/L (321%) in Cl<sub>2</sub> + UV1 variant and 79.00  $\mu$ g/L (352%) in Cl<sub>2</sub> + UV2. CH concentration reached the value of 94.67  $\mu$ g/L for Cl<sub>2</sub> + UV1 variant and 101.47  $\mu$ g/L for Cl<sub>2</sub> + UV2. For arginine, a very strong influence of UV radiation on CH formation was observed-in Cl<sub>2</sub> + UV1 variant CH concentration raised from 1.20 to 13.87  $\mu$ g/L (1,058%), in  $Cl_2 + UV2$  it reached the value of 24.88  $\mu$ g/L (1,975%). Similar to the case of DCAN, an increase in CH concentration in the samples irradiated with UV may be caused by the higher dose of chlorine. Similar to DCAN, such a high potential of histidine and arginine to form CH will not significantly influence CH concentration in real swimming pools due to the low share of these precursors in human body fluids.

#### 3.5. Chloropicrin

So far, CP precursors have not been identified in swimming pool water by any authors. In the research of Cimetiere and De Laat [3], conducted on the samples of water taken from swimming pools and irradiated with UV in laboratory conditions, it was proved that UV-chlorine sequence increased CP concentration. In the experiments described in this article, the CP concentration in the samples only chlorinated was low —from 0.14  $\mu$ g/L in histidine solution to 0.25  $\mu$ g/L in creatinine one. Irradiation of samples with UV1 dose caused an increase in CP concentration in all analyzed samples of organic-N precursors. The highest increase of CP concentration was observed for creatinine (to the level of 27.96  $\mu$ g/L, thus it increased by 11,080%). The relatively high increase was also observed for glycine (from 0.18 to 4.49  $\mu$ g/L, 2,374%) and for histidine (from 0.14 to 1.39  $\mu$ g/L, 920%). The second irradiation caused an increase in CP concentration in the urea sample—from  $0.42 \mu g/L$  (Cl<sub>2</sub> + UV1) to  $0.85 \mu g/L$  $(Cl_2 + UV2)$  (increased by 227%)—and in the arginine one-from 0.34 to 0.50 µg/L, respectively, (96%). However, the highest increase was observed in creatinine solution, in which CP concentration increased to the level of  $41.45 \,\mu g/L$  (Cl<sub>2</sub> + UV2), which is 16,476% higher than in the sample only chlorinated. In other samples after the second irradiation, a decrease in CP concentration was observed-it was 3.00 µg/L for glycine and 0.71 µg/L for histidine. Therefore, the strong influence of UV radiation on CP formation from creatinine was proved in these experiments. Creatinine is present in human body fluids in the relatively high concentration; therefore, UV irradiation of swimming pool water can be a cause of an increase in CP concentration. In the experiments by Weng'a et al. [2], chlorocreatine was shown to be more sensitive to UV irradiation than creatinine, and formed intermediates (with N-Cl bonds) which are more susceptible to free chlorine impact and to the formation of DBPs (such as chloramines and cyanogen chloride). Perhaps the same mechanism caused that during the disinfection of creatinine solution in the sequence chlorine-UV-chlorine more CP was formed.

## 3.6. Haloacetic acids

In swimming pool water, HAAs are the most numerous fraction among all DBPs and are proved to be formed from human body fluids [7,20]. In the experiments conducted on the samples of swimming pool water and treated with UV in lab-scale study, the influence of UV irradiation on HAAs formation was not proved [3]. The concentration of individual HAAs obtained in the experiments described in this article is presented in Table 2 and their sum-in Fig. 2(F). The influence of UV irradiation on HAAs concentration was observed in each organic-N precursor solution. The highest HAAs' formation potential was observed for histidine-in that case HAAs' concentration was  $20.07 \,\mu g/L$  only in the chlorinated sample, and it rose to 50.92  $\mu$ g/L (154%) after Cl<sub>2</sub> + UV1 treatment, and to 66.20  $\mu$ g/L (230%) after Cl<sub>2</sub> + UV2. The relatively high potential to form HAAs was also observed for arginine-the concentration of the sum of MCAA, DCAA, and TCAA was 6.95  $\mu$ g/L, 16.26  $\mu$ g/L (an increase by 134%), and 21.22  $\mu$ g/L (205%) in the samples, respectively, only chlorinated, treated in Cl<sub>2</sub> + UV1 variant and Cl<sub>2</sub> + UV2. The lowest HAAs formation potential was observed for creatinine. HAAs concentration was  $4.98 \,\mu g/L$  for chlorination alone, it increased by 4.42  $\mu$ g/L (89%) to the level of 9.40  $\mu$ g/L after irradiation with a lower UV dose, and as a result of the second irradiation, it decreased to  $8.71 \,\mu g/L$  (75% higher than in the chlorinated sample). The dynamics of by-products formation driven by the sequence of UV irradiation and chlorination has not been fully studied. Thus, it is difficult to define the reason of HAAs a decrease in creatinine solution after the second Cl<sub>2</sub> + UV2 treatment. However, one of possible explanations is the fact that other by-products, especially TCM and CP, were formed more intensively.

# 4. Conclusions

The results of experiments on the influence of UV irradiation on the formation of DBPs from organicnitrogen precursors allowed one to draw the following conclusions:

- TCM was formed by all analyzed precursors; however, a significant influence of UV radiation on trichlorometane formation was observed for urea and histidine;
- a significant influence of histidine on the formation of haloacetonitriles (especially DCAN) was observed, as well as the influence of UV radiation on the formation of haloacetonitriles from histidine and arginine;
- the compounds from the group of HKs were formed by all organic-nitrogen precursors, and the UV irradiation of the samples with urea and arginine caused an increase in the concentration of HKs and a decrease in the samples of creatinine, glycine, and histidine;
- histidine showed the highest potential to form CH, and for histidine and arginine, a significant

influence of UV radiation on the concentration of CH in solution was observed;

- the highest potential to form CP was observed for creatinine, and for this precursor, a strong influence of UV radiation on CP concentration was also proved;
- HAAs were formed by all organic-nitrogen precursors, and for all of them, some influence of UV radiation on the concentration of HAAs was observed; however, histidine showed the highest potential to form these compounds.

The conducted research experiments proved that UV-chlorination sequence could increase the potential of organic-nitrogen precursors to form some water chlorination by-products. UV radiation is commonly treated as a safe disinfection method; however, in the case of swimming pool water, to which some quantity of organic-nitrogen compounds can be introduced by bathers with sweat and urine, the application of UV-chlorine sequence should be carefully controlled.

## Acknowledgments

The work was completed under AGH University of Science and Technology statutory research for the Department of Environmental Management and Protection.

### References

- B. Lyon, A. Dotson, K. Linden, H. Weinberg, The effect of inorganic precursors on disinfection byproduct formation during UV-chlorine/chloramine drinking water treatment, Water Res. 46 (2012) 4653–4664.
- [2] S. Weng, J. Li, K. Wood, H. Kenttämaa, P. Williams, L. Amundson, E. Blatchley III, UV-induced effects on chlorination of creatinine, Water Res. 47 (2013) 4948–4956.
- [3] N. Cimetiere, J. De Laat, Effects of UV-dechloramination of swimming pool water on the formation of disinfection by-products: A lab-scale study, Microchem. J. 112 (2014) 34–41.
- [4] K. Hansen, R. Zortea, A. Piketty, S. Vega, H. Andersen, Photolytic removal of DBPs by medium pressure UV in swimming pool water, Sci. Total Environ. 443 (2013) 850–856.
- [5] J. Wyczarska-Kokot, Swimming pool water disinfection system aided by UV irradiation, Install. Mark. 12 (2009) 96–99 (in Polish).
- [6] S. Richardson, D. DeMarini, M. Kogevinas, 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 Perspect. 118 (2010) 1523–1530.

- [7] S. Chowdhury, K. Alhooshani, T. Karanfil, Disinfection byproducts in swimming pool: Occurrences, implications and future needs, Water Res. 53 (2014) 68–109.
- [8] E. Righi, G. Fantuzzi, G. Predieri, G. Aggazzotti, Bromate, chlorite, chlorate, haloacetic acids, and trihalomethanes occurrence in indoor swimming pool waters in Italy, Microchem. J. 113 (2014) 23–29.
- [9] S. Simard, R. Tardif, M. Rodriguez, Variability of chlorination by-product occurrence in water of indoor and outdoor swimming pools, Water Res. 47 (2013) 1763–1772.
- [10] A. Florentin, A. Hautemanière, P. Hartemann, Health effects of disinfection by-products in chlorinated swimming pools, Int. J. Hygiene Environ. Health 2011 (2014) 461–469.
- [11] S. Richardson, M. Plewa, E. Wagner, R. Schoeny, D. Demarini, Occurrence, genotoxicity, and carcinogenicity of regulated and emerging disinfection byproducts in drinking water: A review and roadmap for research, Mutat. Res. Rev. Mutat. Res. 636 (2007) 178–242.
- [12] M. Panyakapo, S. Soontornchai, P. Paopuree, Cancer risk assessment from exposure to trihalomethanes in tap water and swimming pool water, J. Environ. Sci. 20 (2008) 372–378.
- [13] M. Chen, C. Lin, J. Duh, W. Chou, H. Hsu, Development of a multi-pathway probabilistic health risk assessment model for swimmers exposed to chloroform in indoor swimming pools, J. Hazard. Mater. 185 (2011) 1037–1044.
- [14] C. Lourencetti, J. Grimalt, E. Marco, P. Fernandez, L. Font-Ribera, Trihalomethanes in chlorine and bromine disinfected swimming pools: Air-water distributions and human exposure, Environ. Int. 45 (2012) 59–67.
- [15] I. Zimoch, A. Stolarczyk, Raman spectroscopy in estimating THM formation potential in water pipe network, Environ. Protect. Eng. 36 (2010) 55–64.
- [16] I. Zimoch, Assessing spatial variations of water quality in water-pipe network by analysis of THM formation, Environ. Pollut. Control, 29 (2007) 49–52 (in Polish).
- [17] J. De Laat, W. Feng, D. Freyfer, F. Dossier-Berne, Concentration levels of urea in swimming pool water and reactivity of chlorine with urea, Water Res. 45 (2011) 1139–1146.
- [18] World Health Organization, Guidelines for Safe Recreational Water Environments, Swimming Pools, Spas and Similar Environments, vol. 2, World Health Organization, Geneva, 2006.
- [19] S. Weng, J. Li, E. Blatchley III, Effects of UV254 irradiation on residual chlorine and DBPs in chlorination of model organic-N precursors in swimming pools, Water Res. 46 (2012) 2674–2682.
- [20] A. Kanan, T. Karanfil, Formation of disinfection by-products in indoor swimming pool water: The contribution from filling water natural organic matter and swimmer body fluids, Water Res. 45 (2011) 926–932.
- [21] K. Hansen, S. Willach, H. Mosbæk, H. Andersen, Particles in swimming pool filters—Does pH determine the DBP formation? Chemosphere 87 (2012) 241–247.
- [22] M. Keuten, F. Schets, J. Schijven, J. Verberk, J. van Dijk, Definition and quantification of initial anthropogenic

pollutant release in swimming pools, Water Res. 46 (2012) 3682–3692.

- [23] J. Li, E. Blatchley III, Volatile disinfection byproduct formation resulting from chlorination of organicnitrogen precursors in swimming pools, Environ. Sci. Technol. 41 (2007) 6732–6739.
- [24] W. Liu, L. Cheung, X. Yang, C. Shang, THM, HAA and CNCl formation from UV irradiation and chlor (am)ination of selected organic waters, Water Res. 40 (2006) 2033–2043.
- [25] Y. Choi, Y-j Choi, The effects of UV disinfection on drinking water quality in distribution systems, Water Res. 44 (2010) 115–122.
- [26] Water Research Foundation and EPA, Impact of UV Location and Sequence on By-product Formation, Water Research Foundation, Denver, CO, 2012.

- [27] A. Włodyka-Bergier and T. Bergier, Influence of UV disinfection on halogen water chlorination by-product formation potential in water distribution system, Environ. Polluti. Control 35(3) (2013) 53–57 (in Polish).
- [28] D. Cassan, B. Mercier, F. Castex, A. Rambaud, Effects of medium-pressure UV lamps radiation on water quality in a chlorinated indoor swimming pool, Chemosphere 62 (2006) 1507–1513.
- [29] A. Włodyka-Bergier, M. Rajca and T. Bergier, Removal of halogenated by-products precursors in photocatalysis process enhanced with membrane filtration, Desalin. Water Treat. 52 (2014) 3698–3707.
- [30] H. Kim, J. Shim, S. Lee, Formation of disinfection by-products in chlorinated swimming pool water, Chemosphere 46 (2002) 123–130.