B−46 Change of Haloacetic Acids formation potentials during UV and UV/H_2O_2 treatment

○Hiroshi Sakai[1]* ・ Olivier Autin[2] ・ Simon Parsons[3]

[1]Depart ment of Urban Engineering, The University of Tokyo（7-3-1, Hongo, Bunkyo-ku, Tokyo, 143-0022）

[2]Cranfield Water Science Institute, Cranfield University（Cranfield University, College Road, Cranfield, Bedfordshire, MK43 0AL）

[3]Scottish Water（Castle House, 6 Castle Drive, Carnegie Campus, Dunfermline, KY11 8GG）

＊ E-mail: *h_sakai@envt.u-tokyo.ac.jp*

1. Introduction

Disinfection byproduct (DBPs) is one of the serious concerns in drinking water treatment. It was first reported in 1974 (1), since then a number of studies were made on DBPs. In 1977, resorcinol was found to be a major Trihalomethanes (THMs) precursors. THMs and Haloacetic acids (HAAs) are considered to be two major classes of DBPs, and known to be human caricinogens (2, 3). A regulation by US EPA is 60 μg L^{-1} for total of five HAAs, namely monochloroacetic acid (MCAA), dichloroacetic acid (DCAA), trichloroacetic acid (TCAA), monobromoacetic acid (MBAA), and dibromoacetic acid (DBAA). Japanese drinking water standard has regulation values, 20μg L^{-1} for MCAA, 40 μg L^{-1} for DCAA, and 200 μg L^{-1} for TCAA.

UV or UV/H_2O_2 treatment is thought to be one of the promising ways against DBPs. UV energy is absorbed into chemical bonds to dissociate. Conventional UV lamps have an emission peak around 254 nm. Carbon-carbon double bond could be a primary target for such physicochemical dissociation, because its absorption peak also lies around 254 nm. UV/H_2O_2 treatment primarily produces OH radical from the reaction between UV and H_2O_2. OH radical is a highly reactive radical species; degradation of chemicals would take place by OH radical, due to its high oxidation potentials. With these reaction mechanisms, UV or UV/H_2O_2 treatments are expected to degrade NOMs in water, finally to reduce DBP formation potentials.

Recent study (4) found noteworthy phenomena; HAA formation potential could rise after chemical and biological treatments of NOMs. HAA formation potential from leucine and glutamic acid was clearly increased after 48,000 mJ cm^{-2} of Vacuum UV treatment, 186,000 mJ cm^{-2} of UV treatment, and 47,000 mJ cm^{-2} of UV/H_2O_2 treatment. This result showed degradation of NOMs did not always lead to reduction of DBP formation potentials, contrary to conventional expectations. Then, it became our primary concern if this phenomena impacts at more practical UV irradiance ranges. It was our major interest about mechanisms which underlies HAA formation potential increase during these treatments.

Three model organic compounds, resorcinol, leucine, and serine were selected in reference to previous work (4). These three compounds were exposed to 254 nm low pressure UV lamp with or without H_2O_2. Degradation of model compounds and associated HAA formation potentials were examined during the course of treatment. Then, relations between HAA formation potential changes and degradation of model organic compounds were discussed. Further discussions were made to reveal underlying phenomena, which contributed to HAA formation potential increase.

2. Materials and Methods

Three model organic compounds, resorcinol, leucine and serine, were selected from the lists of surrogates in previous researches (5). Those compounds were dissolved into deionized water at initial concentrations of about 1 mg C L^{-1} and then exposed to UV by collimated beam UV apparatus (ITT-Wedeco). UV irradiance was determined to be 2.26 mW cm^{-2}, by ferrioxalate actinometry (6). H_2O_2 was added at 1 mM. Soon after UV exposure, H_2O_2 was quenched by 10% methanol for UPLC-MS/MS analysis and by catalase for TOC and HAA analysis. It was previously confirmed catalase quenching did not affect HAA and TOC analysis (4, 5). TOC was measured by TOC machine (Shimadzu, TOC 5000A). Resorcinol, leucine and serine concentrations were measured by UPLC-MS/MS (Quattro Premier XE, Waters). Chlorination procedures were referred to previous study (5). Briefly, surrogates were chlorinated at 35 M (Cl_2)/M (Compound), for 24 h,

pH 7, at room temperature. Chlorination was duplicated for single exposure condition. HAAs were measured according to a previous study (5), which based on US EPA Method 552.3. Briefly, they were extracted by MTBE, then methylated by sulfuric acid-methanol. After final extraction into MTBE, samples were measured by GC-ECD (HP-6890, Agilent).

3. Results and Discussions

(1) Degradation of surrogate NOMs

Degradation kinetics were studied by total organic carbon (TOC) and UPLC-MS/MS. TOC was not changed during the course of UV or UV/H_2O_2 treatments. Even though TOC was stable during the treatment, model compounds concentrations were changed. In case of UV only treatment, model compounds seemed to remain stable, while all three model compounds were degraded in UV/H_2O_2 treatment. Its rate was the fastest for resorcinol, second fastest for leucine, and third for Serine. Reaction rate constant was calculated for all three compounds, with an assumption of pseudo-first order reaction. Calculated values were 2.7 cm^2 J^{-1} for leucine, 0.5 cm^2 J^{-1} for serine, 9.4 cm^2 J^{-1} for resorcinol. As they were not degraded by UV alone, their degradations were likely to be done by hydroxyl radicals. Reaction rate constants with hydroxyl radicals were previously reported for these NOM surrogates (7). They were 1.2×10^{10} for resorcinol, 3.2×10^{8} for serine, and 1.7×10^{9} for leucine. Rate constants with hydroxyl radicals were the highest in resorcinol, followed by leucine and serine, same rank order as that of degradation reaction in this study. Those results also supported hydroxyl radical played main role in degradation of these NOM surrogates.

(2) Dichloro acetic acid (DCAA) and Trichloro acetic acid (TCAA)

DCAA and TCAA were focused among nine HAAs, because Bromo-species generally appeared at low concentrations. DCAA and TCAA formation potentials were increased along UV/H_2O_2 treatment. DCAA was initially 25 μg L^{-1} at 0 mJ cm^{-2} of UV. DCAA was linearly increased as UV irradiance increased, and it became 88 μg L^{-1} after 2000 mJ cm^{-2} of UV. Similar phenomenon was observed in TCAA as well. It was 4.1 μg L^{-1} and became 19.4 μg L^{-1} at 0 and 2000 mJ cm^{-2} UV, respectively. In case of UV-only treatment of leucine, clear trend was not observed compared with UV/H_2O_2 case. DCAA was 63 μg L^{-1} and became 45 μg L^{-1}, TCAA was 7.8 μg L^{-1} and became 12.1 μg L^{-1}, both at 0 and 2000 mJ cm^{-2}, respectively. From these results, it was found that HAAs precursors could be produced from leucine by UV/H_2O_2 treatment, which agreed with previous report (4). HAAs formation potentials were not increased by UV treatment, which differs from previous research (4). It should be noted there was about twenty five times difference in UV irradiance range in this study and previous study (4). It was between 0 to 2000 mJ cm^{-2} in this research, and 47,000 mJ cm^{-2} in previous research. Thus, it could be pointed out further treatment would reduce HAA formation potential in turn.

Contrary to leucine, change was not distinct in either UV or UV/H_2O_2 treatment in Serine. In UV treatment, DCAA was 53 μg L^{-1} at 0 mJ cm^{-2} and remained 50 μg L^{-1} at 2000 mJ cm^{-2}. TCAA was initially 7.3 μg L^{-1} and stayed 10.9 μg L^{-1} after 2000 mJ cm^{-2}. Even with H_2O_2 addition, changes were slight. DCAA and TCAA were 29 and 3.6 μg L^{-1} at 0 mJ cm^{-2} and still 36 and 4.8 μg L^{-1} at 2000 mJ cm^{-2}. This result well agreed with previous report (4), which did not show increse of HAAs formation potentials after VUV, UV, or UV/H_2O_2 treatment. Thus, it could be confirmed degradation products from serine would not likely to be HAAs precursors, regardless of chlorination condition.

Resorcinol seemed to be more susceptible to be UV or UV/H_2O_2 treatment. DCAA and TCAA were greatly reduced especially at UV/H_2O_2 treatment. They were initially 120 and 610 μg L^{-1}, which were reduced to 68 and 22 μg L^{-1} after 2000 mJ cm^{-2}. Substantial increase of DCAA or TCAA was observed only by the addition of H_2O_2, without UV. It might be assumed resorcinol was partially degraded only by H_2O_2 addition, which may have resulted in DCAA or TCAA formation.

(3) Relations between DCAA, TCAA-formation potentials and Surrogate degradation

Relations between DCAA, TCAA production and NOM surrogate degradation are shown in Figure 1. Horizontal axis is amount of degraded NOM surrogates in μg C L^{-1} unit. Vertical axis is amount of produced DCAA or TCAA, also in μg L^{-1} unit. There was a distinct relation in case of UV/H_2O_2 treatment of leucine. The more leucine is degraded, the more DCAA or TCAA is produced. It could be easily assumed that HAA precursors were produced along with UV/H_2O_2 treatment of leucine. An interpretation may be added about non-linearity of the relation between leucine degradation and HAA formation potential increase. The way of DCAA or TCAA increase as a function of degraded leucine, seemed like an exponential increase rather than linear increase. It could be said that primary degradation product would not be the precursor of HAA, from kinetics point of view. It would be more likely that degradation compounds at the latter half of overall degradation pathway had contributed to HAA production. It is generally said β-keto acid compounds or β-carbonyl compounds are main precursors of HAAs (4, 8). Then, it was assumed that compounds which have a similar structure with those known precursors, were produced during the course of UV/H_2O_2 treatment of leucine. Previous reports showed H atom abstraction from β or further CH groups from backbone could be the primary reaction of OH radical with leucine (9). It could be assumed

subsequent reaction shortened carbon chain of aliphatic amino acids, which might have led to production of β-keto or β-carbonyl structure. It would be required several further steps from H abstraction to production of β-keto or β-carbonyl structure. This assumption well agrees with the "exponential increase" of HAA formation potential observed in this study.

DCAA, TCAA formation potential increase was not observed in Serine. As far as OH radical reaction played a main role, degradation would start from abstraction of H atom from β CH group. Although further pathway was not identified, it might be speculated that any further degradation product could not be long enough to have a β carbon. Then, it would become impossible for degradation products to have a β-keto or β-carbonyl structure, which resulted in stableness of DCAA or TCAA formation potential during treatment.

DCAA, TCAA formation potential was reduced during treatment of resorcinol. It was reported that OH radical attacked benzene ring of resorcinol, led to opening of the ring. Comparison of 20 amino acids in previous research mentioned possible contribution of benzene ring to HAA formation potential (10). Thus, it might be assumed that hydroxyl radical attacked benzene ring of resorcinol, which caused opening of the ring, finally resulted in reduction of HAA formation potential.

Effect of UV or UV/H_2O_2 treatment on HAA formation potential was experimentally investigated. It was clearly shown degradation intermediates of leucine contributed to increase of HAA formation potential. It could be speculated that OH radical shortened carbon chain of leucine, which resulted in β-keto or β-carbonyl structure. From this assumption, it could also be speculated that longer aliphatic amino acid could increase HAA formation potential during the course of treatment. It might be speculated that way of increase might be different from different length of aliphatic amino acids. Further investigation employing different length of aliphatic amino acid chain might be useful for further identification of underlying mechanisms.

4. References

1) Rook, J. J. *Water Treat. Exam.* **1974**, 23 (2), 234–243.
2) Muellner, M. G.; Wagner, E. D.; McCalla, K.; Richardson, S. D.; Woo, Y.-T.; Plewa, M. J. *Environ. Sci. Technol.* **2007**, 41 (2), 645–651. 2332
3) Nieuwenhuijsen, M. J.; Toledano, M. B.; Eaton, N. E.; Fawell, J.; Elliott, P. *Environ. Med.* **2000**, 57, 73–85. 2375
4) Bond, T.; Goslan, E. H.; Jefferson, B.; Roddick, F.; Fan, L.; Parsons, S. A. *Water Res.* **2009**, 43 (10), 2615–2622.
5) Bond, T.; Henriet, O.; Goslan, E. H.; Parsons, S.A.; Jefferson, B. *Environ. Sci. Technol.* **2009**, 43, 5982–5989.
6) Hatchard, C. G.; Parker, C. A. *Proc. R. Soc. London.* **1956**, 235, 518–536.
7) Buxton, G. V.; Greenstock, C. L.; Helman, W. P.; Ross, A. B. *J. Phys. Chem. Ref. Data.* **1988**, 17(2), 513–886.
8) Dickenson, E. R. V.; Summers, R. S.; Croue´, J.-P.; Gallard, H. *Environ. Sci. Technol.* **2008**, 42 (9), 3226–3233.
9) Scheiner, S.; Kar, T. *J. Am. Chem. Soc.* **2010**, 132, 16450–16459.
10) Hong, H. C.; Wong, M. H.; Liang, Y. *Arch. Environ. Con. Tox.*, **2009**, 56, 638–645.

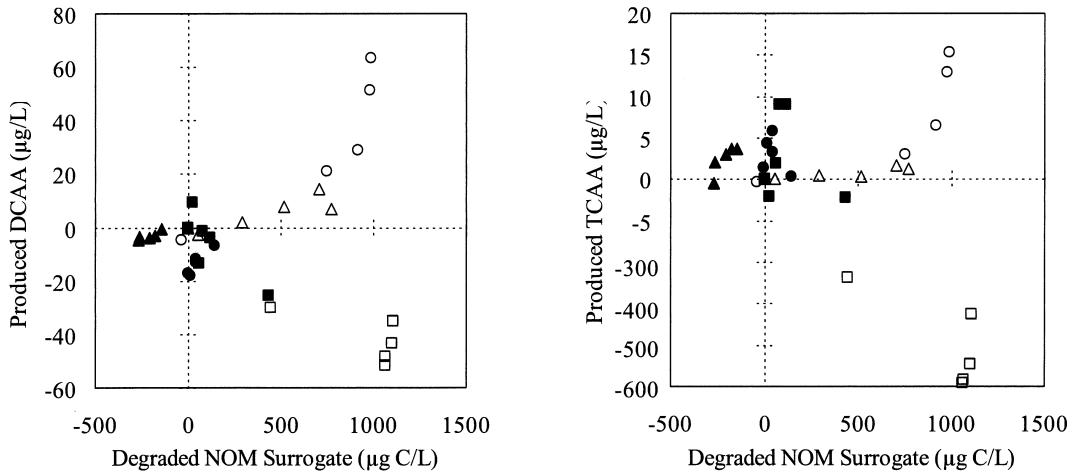

Figure 1 Relations between degradation of NOM surrogates and DCAA (left) or TCAA (right) formation. NOM surrogates are Leucine (●), Serine(▲), and Resorcinol(■) for UV-only treatment, and Leucine (○), Serine(△), and Resorcinol(□) for UV/H_2O_2 treatment