Evaluation of byproduct formation and toxicity change during drinking water treatment with UV/H_2O_2 and UV/Chlorine using *in vitro* bioassays combined with chemical analysis

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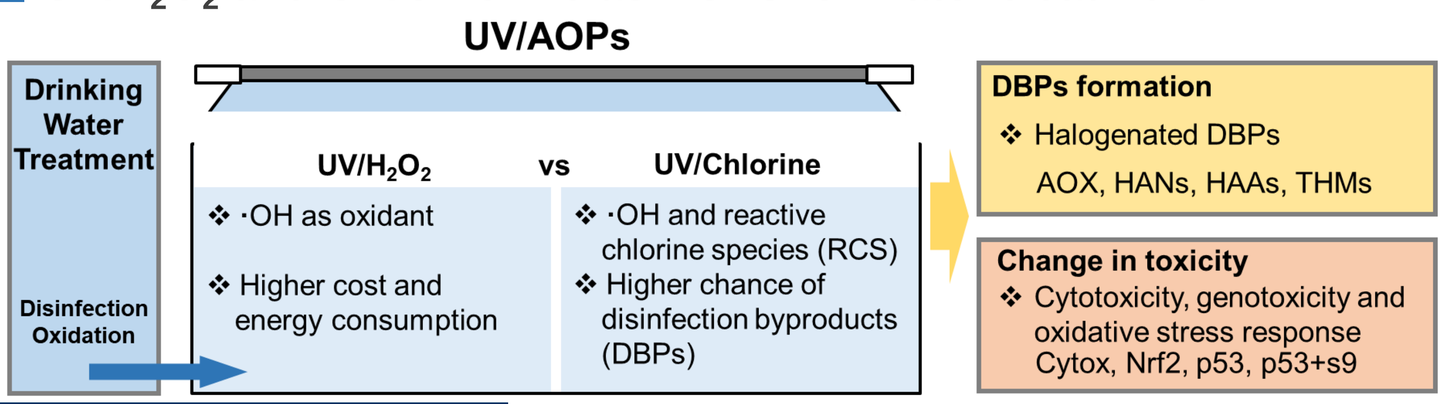
Introduction

UV/H_2O_2 and UV/Chlorine as AOPs for water treatment



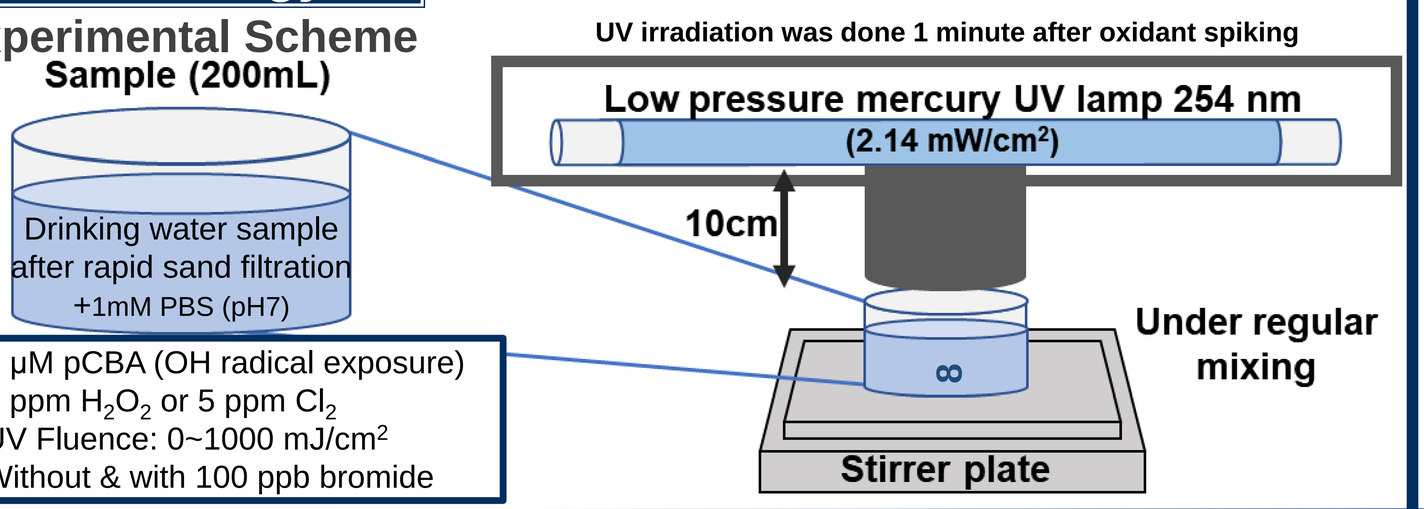
Research Goals

❖ Comparing the performance of UV/H_2O_2 and UV/Chlorine by assessing DBPs formation via chemical analysis and toxicity change using *in vitro* bioassays

❖ Providing detailed comparison of UV-AOPs which could further act as a guide for their appropriate applications in various scenarios in drinking water treatment

Methodology

Experimental Scheme



- 1 μM pCBA (OH radical exposure)
- 5 ppm H_2O_2 or 5 ppm Cl_2
- UV Fluence: 0~1000 mJ/cm^2
- Without & with 100 ppb bromide

Determination of OH radical exposure

❖ pCBA degradation was monitored as a basis to determine oxidant dosage yielding the same oxidation capacity

- (5 ppm H_2O_2/4 ppm Cl_2) + 0~500 mJ/cm^2 UV dose

UV-AOP treatment conditions

❖ 5 ppm H_2O_2 or 5 ppm Cl_2 added 1 min before UV irradiation
❖ UV fluence = 0, 300, 600, 1000 mJ/cm^2
❖ Bromide = 0 and 100 ppb, to see impact on DBPs

Chemical Analysis

Table 1. Summary of chemical analysis

Compound	Instrument	Model	Reference
pCBA	HPLC/UV	Dionex, U3000, USA	
THM			
HAA	GC/uECD	Agilent, 6890N, USA	USEPA 552.3
HAN			USEPA 551.1
AOX	AOX analyzer	Euroglas, ECS1600, GER	

❖ Analytes for THMs, HAAs and AOX were quenched by sodium sulfite before analysis

❖ Analytes for HANs were quenched by H_2O_2 for residual chlorine, and catalase for excess H_2O_2 before analysis

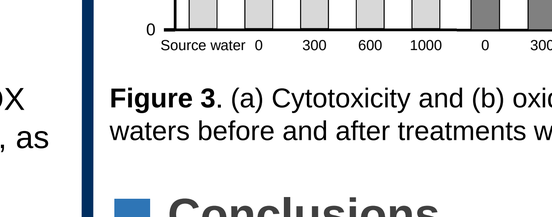
❖ THM: $CHCl_3$, $CHCl_2Br$, $CHBr_2$, $CHBr_3$
❖ HAA: MCAA, MBAA, DCAA, TCAA, BCAA, BDCAA, DBAA, DBCAA, TBAA
❖ HAN: DBAN, BCAN, DCAN, TCAN

In vitro Bioassay (volatile DBPs excluded)

Table 2. Summary of bioassays

CALUX Assay	End point	Reference compound	Limit of quantification (LOQ)
Nrf2	Oxidative stress	Curcumin (Cur)	13 ng/L
p53	Genotoxicity	Actinomycin D (Act)	0.65 ng/L
Cytox	cytotoxicity	Tributylyin (TBT)	36 ng/L

Enrichment of sample with SPE → Used in bioassay (CALUX) → Sample treatment



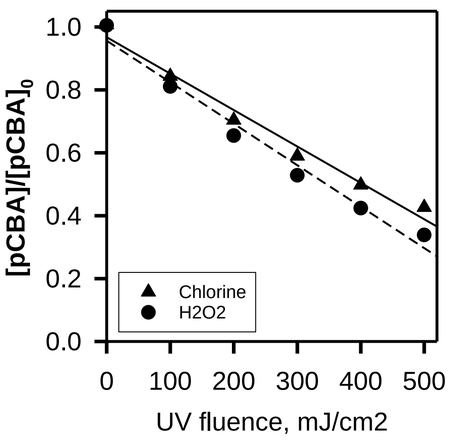
❖ Analytes went through SPE to be enriched by 300-fold before bioassay

❖ Based on the responses, bioactivities were normalized to bioanalytical equivalent concentration (BEQ)

$$BEQ = \frac{EC_{10\,(reference\ compound)}}{EC_{10\,(sample)}} = \frac{EC_{IR1.5\,(reference\ compound)}}{EC_{IR1.5\,(sample)}}$$

Results & Discussion

Comparison of OH radical exposure



❖ The degradation rates of pCBA during UV/Chlorine and UV/H_2O_2 were comparable as a function of UV fluence in the tested oxidant conditions (4 ppm Cl_2 and 5 ppm H2O2).

❖ The result indicate that comparable levels of OH radical exposure can be achieved with UV/Chlorine and UV/H2O2 processes when applying the same mass-based oxidant concentration.

◀**Figure 1**. Relative residual pCBA concentration as a function of UV fluence during UV/H_2O_2 and UV/Chlorine treatment

Formation of DBPs

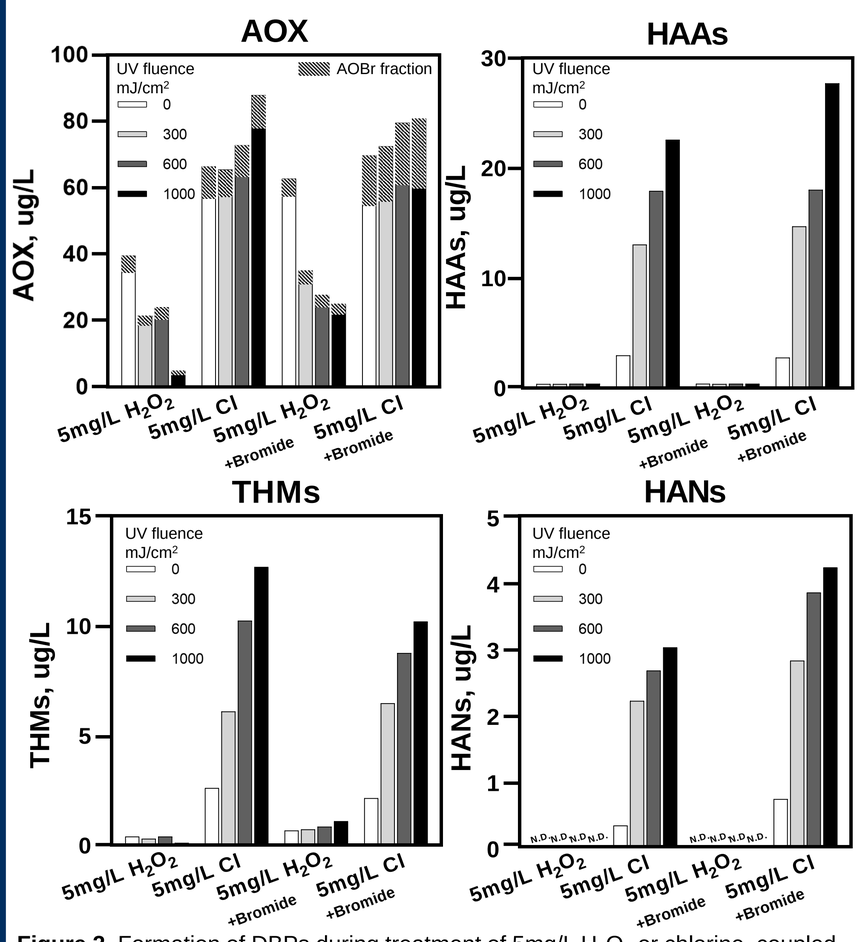


Figure 2. Formation of DBPs during treatment of 5mg/L H_2O_2 or chlorine, coupled with 0~1000 mJ/cm^2 UV dose with and without 100ppb bromide spiking

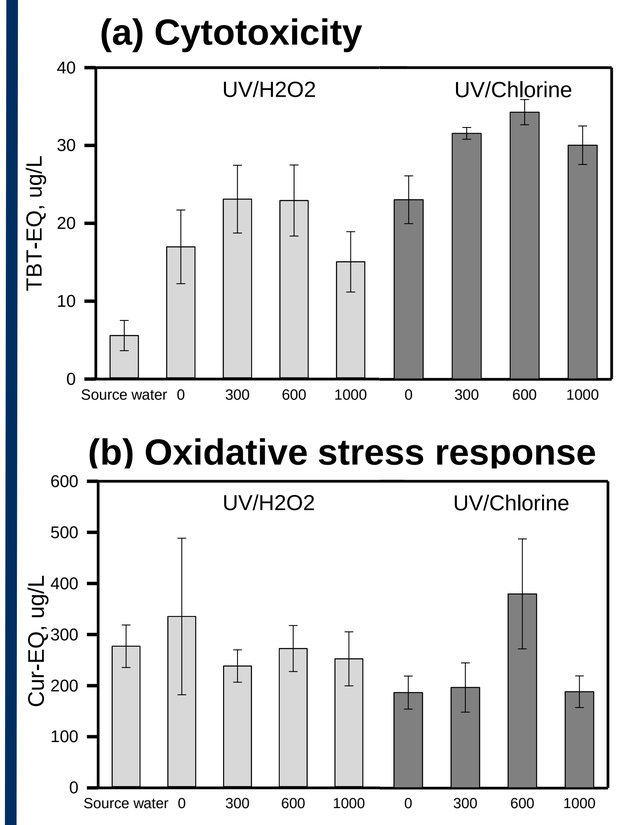
❖ **AOX**

- The AOX formation was higher for UV/Chlorine compared to UV/H2O2
- For UV/Chlorine, the majority of AOX formation occurred during the first minute of dark chlorination, whereas only a minor increase in AOX formation was observed during chlorine photolysis
- UV/H_2O_2 showed a reduction in AOX that was pre-existing in the sample, as the UV dose increased
- The proportion of AOBr increased following the addition of 100 ppb of bromide

❖ **THMs, HAAs, HANs**

- Formation of THMs, HAAs, and HANs was significantly higher for UV/Chlorine compared to UV/H_2O_2
- For UV/Chlorine, the DBP formation increased with increasing UV dose
- Addition of bromide had minor effect on the total formation of halogenated DBPs

Changes of biological activities



❖ **Cytotoxicity**

- Cytotoxicity increased with increasing UV fluence in both UV/Chlorine and UV/H_2O_2 treatments
- UV/Chlorine showed higher levels of cytotoxicity than UV/H_2O_2
- Genotoxicity was below LOQ for all cases

❖ **Oxidative stress response**

- Oxidative stress response remained stable with increasing UV fluence for both UV/Chlorine and UV/H_2O_2 treatments and was slightly higher for UV/H_2O_2 than UV/Chlorine

Figure 3. (a) Cytotoxicity and (b) oxidative stress response of the sand-filtered waters before and after treatments with UV/Chlorine and UV/H_2O_2

Conclusions

- Formation of halogenated DBPs (AOX, THMs, HAAs, HANs) during UV/Chlorine treatment was significantly higher compared to UV/H_2O_2 treatment, consistent with previous reports
- Assessment with *in vitro* bioassays, covering cytotoxicity, genotoxicity, and oxidative stress response, exhibited insignificant differences between UV/Chlorine and UV/H_2O_2 treatments. This outcome contrasted with the significant difference observed in the chemical DBP analyses
- Research is on-going to assess the changes of overall toxicity during UV/Chlorine and UV/H_2O_2 treatments by considering both volatile and non-volatile DBP mixtures

Acknowledgement

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