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ARTICLE

Comparison of the impact of ozone, chlorine dioxide, ferrate and permanganate pre-oxidation on organic disinfection byproduct formation during post-chlorination

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Pre-oxidation is commonly used to mitigate the formation of byproducts during post-disinfection. A comparative study of the impact of four pre-oxidants, ozone (O₃), chlorine dioxide (ClO₂), permanganate (Mn(VII)) and ferrate (Fe(VI)), on the formation of trihalomethanes (THMs), haloacetonitriles (HANs) and adsorbable organic halogens (AOX) in chlorinated synthetic and real waters was conducted. The pre-oxidant doses were based on their impact on natural organic matter reactivity measured by the electron donating capacity before chlorination. The influence of pH (6.5–8.1) and bromide (0–500 µg L⁻¹) was evaluated in terms of disinfection byproduct (DBP) formation and theoretical toxicity assessment (based on THM and HAN formation). All oxidants were efficient in mitigating chlorinated DBPs, except Mn(VII) which had little impact on THM formation. O₃ was generally more efficient than the other oxidants in mitigating AOX and THM formation, all pre-oxidants were readily reducing the formation of HANs (> 45% reduction at high dose). pH depression improved AOX mitigation by O₃ and Fe(VI) but diminished Mn(VII) efficiency for all DBPs. Pre-oxidation was less efficient in mitigating brominated DBPs and generally enhanced the bromine substitution factor. Although HANs were formed at low concentrations compared to THMs, they dominated the calculated toxicity, particularly the brominated HANs. The increased dibromoacetonitrile formation after pre-oxidation was a major factor counteracting the benefit of the overall DBP mitigation. In presence of bromide, the pre-oxidant dose should be optimized to decrease the reactivity of the matrix while controlling the toxicity induced by formation of brominated DBPs, notably the brominated HANs.

1. Introduction

Chlorine is worldwide the most widely used disinfectant in drinking water treatment. However, the formation of disinfection byproducts (DBPs) has been an on-going problem of concern.¹ Pre-oxidation has been successfully applied to reduce the formation of DBPs during post-chlorination.^{2–4} Potential pre-oxidants such as ozone (O₃), ferrate (Fe(VI)) or permanganate (Mn(VII)) are chlorine-free, and therefore are not producing chlorinated DBPs (Cl-DBPs) while degrading their precursors.^{2, 3} Chlorine dioxide (ClO₂) is not directly able to

halogenate natural organic matter (NOM) but was recently shown to produce chlorine *in situ*, which can in turn form Cl-DBPs.^{5, 6} Nonetheless, the formation of halogenated DBPs after ClO₂ treatment is much lower compared to chlorination. Together with the reduction of the water matrix reactivity, the use of pre-oxidation can have complementary benefits such as a degradation of contaminants,^{7, 8} disinfection (except for Mn(VII)),^{9, 10} or coagulation.^{10–12}

DBP reduction has been achieved by pre-oxidizing various water matrices with ClO₂,^{4, 13, 14} O₃,^{2, 15, 16} Fe(VI),^{14, 17–19} and Mn(VII).^{18, 20} However, only a few studies compared the efficiency of different pre-oxidants. In a previous study ClO₂ and Fe(VI) efficiency on DBP formation during chlorination of 12 waters has been compared.¹⁴ It was found that 20 mgFe L⁻¹ as Fe(VI) was more efficient to mitigate HAN formation and less efficient for THM mitigation compared to 1 mg L⁻¹ of ClO₂. Even though there are effects of both oxidants on DBPs, the very different doses don't allow a direct comparison (see discussion below). In two other comparative studies, equivalent mass concentrations of Fe(VI) and O₃ gave similar THM, haloacetic acid and HAN abatement in different real waters.^{3, 21} Another study showed a generally better DBP abatement by Fe(VI) pre-oxidation compared to Mn(VII) pre-oxidation, based on equivalent mass concentrations.¹⁸ The reactivity of the water matrix (described by dissolved organic carbon, DOC, and UV absorbance at 254 nm, UVA₂₅₄), the pH as well as the presence

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of halide ions such as bromide or iodide will impact the formation of DBPs and their mitigation.^{22–24} For instance, the presence of bromide leads to the formation of brominated DBPs (Br-DBPs) during chlorination,²² which are generally more toxic than their chlorinated analogues.²⁵ Applying ClO_2 and O_3 pre-oxidation has been shown to impact the speciation of DBPs in bromide-containing waters.^{2, 13, 15, 16, 26} Furthermore, the fate of bromide during pre-oxidation also needs to be considered. O_3 is known to moderately react with bromide to form bromine (HOBr/OBr^-), leading to the formation of organic Br-DBPs and potentially bromate.²⁷ However, HOBr is in competition with O_3 for some highly reactive NOM moieties.²⁶ Therefore, the formation of Br-DBPs and bromate depends on the O_3 and hydroxyl radical exposure, the bromide concentration and the NOM characteristics. ClO_2 is unreactive toward bromide,²⁸ but may form HOBr through the *in situ* formation of chlorine (HOCl/OCl^-),^{5, 6} while Fe(VI) was recently shown to slowly react with bromide, leading to HOBr and bromate in absence of phosphate.²⁹ The pH is also an important parameter affecting the formation and the speciation of DBPs during chlorination.²² Typically, increasing the pH will enhance the formation of trihalomethanes (THMs) and lowering the pH will lead to higher haloacetonitriles (HANs) concentrations.^{30, 31} The pH may also impact the stability of the pre-oxidants. O_3 and Fe(VI) are known to self-decompose in water. The self-decays of O_3 (and concomitant hydroxyl radical formation) and Fe(VI) increases or decreases, respectively, with increasing pH.^{32, 33} In comparison, ClO_2 and Mn(VII) are stable across the pH range relevant to water treatment.³⁴ The reactivity of the pre-oxidants toward DBP precursors will also be affected by pH. For instance, the reactivity of phenol toward Fe(VI) tends to decrease with increasing pH while it increases with Mn(VII), O_3 and ClO_2 .^{7, 28, 35, 36} Furthermore, the reaction or degradation products of O_3 , ClO_2 , Mn(VII) and Fe(VI) can participate in the overall oxidation processes.^{5, 32, 37, 38}

It is challenging to make general conclusions on the impact of pre-oxidation treatment on DBP formation due to the variability of experimental conditions across studies. Furthermore, supplementary treatments after pre-oxidation, such as coagulation, (biological) activated carbon or sand filtration, are likely to affect the NOM characteristics and therefore the formation of DBPs during subsequent chlorination.^{17, 39–41} In previous studies, the choice of the pre-oxidant doses has usually been based on molar concentrations, mass concentrations or doses commonly used in drinking water treatment plants. Recently, it was proposed to compare the efficiency of pre-oxidants based on their impact on NOM characteristics.⁴² The recently introduced electron donating capacity (EDC) is a proxy for activated aromatic compounds,^{43, 44} generally decreasing more readily than the UVA_{254} upon oxidative treatments.^{42, 45, 46} Therefore, it is an interesting surrogate to monitor changes in NOM characteristics related to oxidative treatment.

In this study, a comparison of the impacts of four pre-oxidants, i.e., ClO_2 , O_3 , Fe(VI) and Mn(VII), on the formation of AOX, THMs and HANs during post-chlorination is presented. The impact of the pre-oxidation treatments is presented based

on doses of oxidants inducing equivalent effects on the EDC of NOM. The impact of pre-oxidant dose, bromide concentration and pH on the reduction of the concentration and the speciation of DBPs is evaluated. The benefits of pre-oxidation are also discussed in terms of calculated toxicity, based on THM and HAN formation. This study does not aim to reproduce real water treatment conditions since no coagulation or (bio)filtration step was applied after pre-oxidation.

2. Materials and methods

2.1. Chemicals and reagents.

Sodium chlorite (80%) and a sodium hypochlorite solution (10–15%) were purchased from Sigma Aldrich. The impurities in the sodium chlorite and sodium hypochlorite were mostly chloride and traces of chlorate ($< 0.01 \mu\text{M}$ of ClO_3^- per μM of NaOCl or NaClO_2). All other chemicals were of analytical grade purity ($\geq 98\%$). Solutions were prepared with ultrapure water (Purelab Ultra, Elga, UK). Suwannee River NOM extract was purchased from the International Humic Substances Society (Cat. No. 2R101N).

2.2. Preparation of oxidant solutions.

Chlorine stock solutions were prepared from a sodium hypochlorite solution standardized by direct UV measurement at 292 nm ($\epsilon_{292} = 362 \text{ M}^{-1} \text{ cm}^{-1}$).⁴⁷ A ClO_2 stock solution was produced by reaction of sodium persulfate (40 g L^{-1}) and sodium chlorite (80 g L^{-1}) under nitrogen bubbling for about 1 h.^{48, 49} The ClO_2 was retrieved in chilled ultrapure water ($T < 10^\circ\text{C}$) and standardized by direct UV measurement at 359 nm ($\epsilon_{359} = 1230 \text{ M}^{-1} \text{ cm}^{-1}$).⁴⁷ An O_3 stock solution was prepared by bubbling an ozone-oxygen gas mixture produced by an ozone generator (American Ozone System Inc.) in chilled ultrapure water ($T < 5^\circ\text{C}$). The O_3 stock solution ($0.6\text{--}0.9 \text{ mM}$) was standardized by direct UV measurement at 260 nm ($\epsilon_{260} = 3200 \text{ M}^{-1} \text{ cm}^{-1}$).³² Potassium ferrate (K_2FeO_4) was prepared by oxidation of 0.186 mol of ferric nitrate nonahydrate with 0.845 mol of potassium hypochlorite in a concentrated potassium hydroxide solution.⁵⁰ The final product purity was 43% and was stable over the duration of this study. The rest of the solid was potassium hydroxide or potassium chloride. Mn(VII) and Fe(VI) solutions were prepared by dissolution of KMnO_4 in ultrapure water for Mn(VII), or of K_2FeO_4 in a phosphate/borate buffer at pH 9.5 ($5 \text{ mM} / 1 \text{ mM}$) for Fe(VI). The stock solutions ($1\text{--}5 \text{ mM}$) were filtered through $0.22 \mu\text{m}$ (Polyethersulfone, Merck Millipore) and standardized by direct UV measurements at 525 nm ($\epsilon_{525} = 2430 \text{ M}^{-1} \text{ cm}^{-1}$)⁵¹ for Mn(VII) or 510 nm ($\epsilon_{510} = 1150 \text{ M}^{-1} \text{ cm}^{-1}$)⁵² for Fe(VI).

2.3. Pre-oxidation and chlorination experiments.

The synthetic waters were prepared with 3.0 mgC L^{-1} of a Suwannee River NOM extract (SRNOM) buffered at pH 6.5 (40 mM phosphate) or pH 8 (40 mM borate) with or without bromide ($0, 150$ or $500 \mu\text{g L}^{-1}$). The synthetic waters (500 mL) were spiked with O_3 (with 5 mM of tert-butanol, *t*-BuOH), ClO_2 , KMnO_4 or K_2FeO_4 ($1\text{--}94.3 \mu\text{M}$, Table 1), kept without headspace

(< 10% of total volume) and protected from light until complete depletion of the oxidants. For medium and high O₃ doses, the significant volume of O₃ stock solution added was taken into account by concentrating the matrix accordingly. *t*-BuOH was added as a hydroxyl radical scavenger to account for direct O₃ reactions only, at a dose based on a previous study.⁴⁵ All the samples were then filtered through 0.22 µm (Polyethersulfone, Merck Millipore). Around 200 mL of the sample was used for the analyses of electron donating capacity (EDC), anions (bromide, bromate, chlorite and chlorate), AOX, THMs and HANs, and 200 mL was chlorinated with 85 µM of sodium hypochlorite (6.0 mgCl₂ L⁻¹) for 72 h, without headspace (< 10% of total volume) and protected from light. The chlorine dose was sufficient to ensure an oxidant residual of at least 1.4 mgCl₂ L⁻¹ after 72 h at pH 8, without bromide and without pre-treatment. The oxidant residual was quenched by sodium sulfite (10% molar excess relative to the chlorine residual) for AOX, bromide and bromate measurements, or by a 5 times molar excess (relative to the chlorine residual) of ascorbic acid for THMs and HANs. All experiments with SRNOM were duplicated, except for the highest doses of Fe(VI) and Mn(VII). A similar experiment was conducted with water from a shallow groundwater (W20) with 10–30 µM pre-oxidant (Table 1) and 155 µM chlorine (12.4 mgCl₂ L⁻¹). The chlorine dose was chosen based on a chlorine demand experiment allowing a chlorine residual of about 1.5 mgCl₂ L⁻¹ after 72 h. The main characteristics of the real and synthetic waters are given in Table 2.

2.4. Analytical methods.

THMs (trichloro-, bromodichloro-, dibromochloro- and tribromomethane) and HANs (chloro-, bromo-, dichloro-, bromochloro-, dibromo- and trichloroacetonitrile) were analyzed by headspace GC-MS with a method adapted from a previous publication (details are given in Text S1).^{53, 54} Abbreviations used for individual THMs and HANs are shown in Table S1 (Electronic Supporting Information, ESI). The AOX was measured by ion chromatography after combustion of the activated carbon with the adsorbed samples.⁵⁵ The limit of quantification (LOQ) for AOCl and AOBr were 0.5 µM (as Cl⁻) and 0.1 µM (as Br⁻), respectively. Standard deviations for AOCl and AOBr typically ranged between 5–15%. The chlorine residual was measured by ABTS (2,2'-azino-bis(3-ethylbenzothiazoline-sulfonic acid) diammonium salt).⁵⁶ Bromide (LOQ = 0.05 µM), bromate (LOQ = 0.02 µM), chlorite (LOQ = 0.08 µM) and chlorate (LOQ = 0.06 µM) were measured by ion chromatography using an AS10-HC column with a Na₂CO₃ eluent (9 mM, 1 mL min⁻¹).⁵⁷ The electron donating capacity (EDC), a proxy for antioxidant capacity of NOM,^{43, 45} was measured on an Agilent 1100 series system using a size-exclusion chromatography (SEC) followed by a post-column reaction with the ABTS radical. This method was adapted from previous studies,^{58, 59} and is described elsewhere.⁴² The EDC is presented as percentage of reduction compared to the untreated sample, or relative to a 3 mgC L⁻¹ unbuffered SRNOM sample.

3. Results and discussion

3.1. Impact of pre-oxidation on the EDC of SRNOM.

Different pre-oxidant doses were spiked to solutions containing 3 mgC L⁻¹ of SRNOM. Three doses were applied, low, medium and high, corresponding to an approximate relative EDC abatement of 20, 50 and 70% at pH 8, respectively (Table 1). As previously observed, very low doses of Mn(VII) led to large relative EDC abatements,⁴² resulting in inaccuracies for the low dose experiment. Therefore, only medium and high doses of Mn(VII) were applied in this study.

3.2. DBP formation after pre-oxidation.

After pre-oxidation and in absence of bromide, only ClO₂ produced low concentrations of Cl-DBPs through the *in situ* formation of hypochlorous acid (HOCl) (Figs. S1a,b, Electronic Supporting Information, ESI).^{5, 6} A maximum of 2.1 ± 0.8 µM of AOCl and 10 ± 0.7 nM of trichloromethane (TCM) were formed, consistent with a previous study.⁵ Along with organic DBPs, up to 25 µM of chlorite (1.7 mg L⁻¹) and up to 2.7 µM of chlorate (0.2 mg L⁻¹) were formed after ClO₂ pre-oxidation (Fig. S2, ESI).

In presence of bromide, both ClO₂ (through the *in situ* formation of HOCl) and O₃-*t*-BuOH (through direct oxidation of bromide to HOBr) formed Br-DBPs. As a result, a significant decrease of bromide (7–22%) was observed after pre-oxidation at high doses of ClO₂ and O₃-*t*-BuOH (Fig. S3, ESI). Consequently, up to 1.8 ± 0.3 µM and 1.1 ± 0.1 µM of AOBr was formed after pre-oxidation with high doses of ClO₂ and O₃-*t*-BuOH, respectively (Figs. S1a,c, ESI). Traces of all four THMs were produced after ClO₂ pre-oxidation (total THMs < 10 nM) while only tribromomethane (TBM) was formed at high dose of O₃-*t*-BuOH (up to 69 ± 11 nM) (Figs. S1b,d, ESI).

The bromide concentration was not decreasing after Fe(VI) and Mn(VII) pre-oxidation (< 5% decrease, Fig. S3, ESI) and consequently no Br-DBPs were formed.²⁹ No bromate (< 3 nM) was detected after O₃-*t*-BuOH and Fe(VI) pre-oxidation. As mentioned in the introduction, Fe(VI) slowly reacts with bromide.²⁹ In borate buffer at pH 7.5 and without NOM, 50 µM of Fe(VI) oxidizes only about 5% of bromide.²⁹ Both a higher pH and the presence of NOM are likely mitigating the oxidation of bromide by Fe(VI), which is consistent with the apparent lack of reactivity between bromide and Fe(VI) (Fig. S3, ESI) and with the lack of formation of Br-DBPs or bromate.

3.3. Pre-oxidation efficiency on DBP mitigation during final chlorination of SRNOM in absence of bromide.

3.3.1. Chlorination alone.

Without pre-oxidation and in absence of bromide, 16.6 ± 1.5 µM of AOCl (Fig. 1a), 2.8 ± 0.3 µM of THMs (TCM only, Fig. 1d) and 50 ± 8 nM of HANs (95% dichloroacetonitrile (DCAN) and 5% trichloroacetonitrile (TCAN), Fig. 1g) were formed after 72 h of chlorination of SRNOM. These results are consistent with a previous study carried out with a 3 mgC L⁻¹ Suwannee River Fulvic Acids extract, which produced 16 µM and 2.5 µM of AOCl and TCM, respectively, after 5 days of chlorination at pH 7.5.⁶⁰ In the previous study, HANs were not

detected, probably because of the longer reaction time, promoting HAN hydrolysis.³⁰

3.3.2. Pre-oxidation followed by chlorination.

When pre-oxidation was applied, the formation of AOCl, TCM and HANs were mitigated to various extents (Figs. 1a, d, g). The mitigation generally increased with increasing pre-oxidant doses. For the highest pre-oxidant dose, AOCl formation was reduced by $30 \pm 3\%$, $53 \pm 5\%$, $21 \pm 2\%$ and $20 \pm 2\%$ for ClO_2 , O_3 -*t*-BuOH, Fe(VI) and Mn(VII), respectively, compared to chlorination alone (Fig. 1a). It was previously shown that for a similar EDC abatement, an equivalent AOX mitigation was obtained for ClO_2 , Fe(VI) and Mn(VII).⁴² The slightly higher mitigation of AOCl observed for ClO_2 compared to Fe(VI) and Mn(VII) could be explained by the slightly higher EDC abatement (77% versus 68–69%, Table 1). In comparison, O_3 -*t*-BuOH was more efficient to mitigate AOX than the other pre-oxidants probably due to Criegee-type reactions which lead to aromatic ring cleavage, resulting in a reduction of halogenation reactions (Fig. 1a).^{61, 62}

High doses of ClO_2 , O_3 -*t*-BuOH and Fe(VI) also reduced THM formation by $29 \pm 3\%$, $30 \pm 1\%$ and $26 \pm 3\%$, respectively (Fig. 1d). In contrast, Mn(VII) was not efficient in mitigating THM formation ($\leq 10\%$), which is consistent with previous studies.^{18, 20, 40, 63} While the THMs represented 49–54% of the AOX (in moles of halogen) in samples without pre-treatment, this fraction was substantially increased by O_3 -*t*-BuOH (up to 74%) and Mn(VII) (up to 62%) while it remained unaffected by ClO_2 (47–55%) and Fe(VI) (42–50%) (Fig. S4, ESI). This increase in the THM fraction may be explained by a transformation of activated aromatic compounds to aliphatic β -keto-acid and β -diketone structures which lead to less halogenation but a higher yield of THMs compared to phenol.⁶¹

In comparison to the AOX and THMs, the HAN formation was much more efficiently mitigated by all oxidants with $66 \pm 4\%$, $68 \pm 7\%$, $45 \pm 4\%$ and $53 \pm 12\%$ reduction of their formation at high doses of ClO_2 , O_3 -*t*-BuOH, Fe(VI) and Mn(VII), respectively (Fig. 1g). A generally efficient HAN mitigation by pre-oxidation has been previously observed by all four oxidants.^{13, 14, 16, 18, 21}

3.4. Pre-oxidation efficiency on DBP mitigation during final chlorination of SRNOM: impact of bromide.

3.4.1. Total DBPs.

During chlorination in presence of 150 or 500 $\mu\text{g L}^{-1}$ bromide and without pre-oxidation, $15.8 \pm 1.5 \mu\text{M}$ and $15.6 \pm 1.6 \mu\text{M}$ of AOX were produced, respectively, which was not significantly different from the AOX formation without bromide (Figs. 1a–c). Similarly, the total THM (2.8 – $2.9 \pm 0.4 \mu\text{M}$) and the total HAN (50 – 59 ± 9 – 18 nM) formations in presence of bromide were not significantly different from the sample without bromide (Figs. 1d–f and Figs. 1g–i). The AOX and THM formation was consistent with a previous study using a Suwannee River Fulvic Acid extract (3 mgC L^{-1}) in presence of $104 \mu\text{g L}^{-1}$ bromide, where $16.1 \mu\text{M}$ of AOX and $2.0 \mu\text{M}$ of THMs were found.⁶⁰

The pre-oxidation efficiency on the total formation of AOX, THMs and HANs was generally reduced by the presence of bromide as illustrated in Fig. 2 and Fig. S5 (ESI) (DBP mitigation relative to the experiment without pre-oxidation). At the highest doses of ClO_2 , O_3 -*t*-BuOH and Mn(VII), the addition of $500 \mu\text{g L}^{-1}$ bromide decreased the HAN mitigation efficiency from $66 \pm 4\%$, $68 \pm 7\%$ and $53 \pm 12\%$ without bromide to $42 \pm 7\%$, $63 \pm 26\%$ and $14 \pm 5\%$, respectively (Fig. 2). Similarly, AOX mitigation decreased from $30 \pm 3\%$, $53 \pm 5\%$ and $20 \pm 2\%$ to $23 \pm 3\%$, $42 \pm 9\%$ and $16 \pm 1\%$, in presence of $500 \mu\text{g L}^{-1}$ bromide, at the highest doses of ClO_2 , O_3 -*t*-BuOH and Mn(VII), respectively (Fig. S5a, ESI). For THMs, the addition of $500 \mu\text{g L}^{-1}$ bromide led to a decrease of the mitigation efficiency from $29 \pm 3\%$, $30 \pm 1\%$ and $26 \pm 3\%$ to $25 \pm 3\%$, $14 \pm 2\%$ and $3 \pm 1\%$ at the highest doses of ClO_2 , O_3 -*t*-BuOH and Fe(VI), respectively (Fig. S5b, ESI).

Conversely to the general trend, the experiment carried out with Fe(VI) and $500 \mu\text{g L}^{-1}$ bromide showed an improved mitigation of AOX (Fig. S5a, ESI). However, the presence of $150 \mu\text{g L}^{-1}$ bromide still decreased the AOX mitigation efficiency (Fig. S5a, ESI).

3.4.2. DBP Speciation.

Although the total DBP concentration was not affected by the presence of bromide in samples without pre-oxidation of SRNOM, Br-DBPs were formed. At 150 and $500 \mu\text{g L}^{-1}$ bromide, 1.6 ± 0.1 and $5.8 \pm 0.3 \mu\text{M}$ of AOBBr were produced, respectively, representing 85 – $93 \pm 5\%$ of the initial bromide (Figs. 1b,c). About half of the bromide (49–57%) was recovered in THMs as TBM, dibromochloromethane (DBCM) and dichlorobromomethane (DCBM) (Figs. 1e,f), while 0.7 – 1.2% was incorporated in the HANs as dibromoacetoneitrile (DBAN) and bromochloroacetoneitrile (BCAN) (Figs. 1h,i). Increasing the bromide concentration increased the bromine substitution factor (BSF). The BSF is the fraction of bromine relative to the total halogens of a specific group of DBPs,⁶⁴ and is calculated as follows (eq. 1):

$$BSF = \frac{[Br-DBPs]}{[X-DBPs]} \times 100 \quad (1)$$

Without pre-oxidation, the addition of $150 \mu\text{g L}^{-1}$ bromide led to a BSF of $11 \pm 1\%$, $12 \pm 0.5\%$ and $13 \pm 3\%$ in AOX, THMs and HANs, respectively (Fig. 3). Increasing the bromide concentration to $500 \mu\text{g L}^{-1}$ increased the BSF in AOX, THMs and HANs to $39 \pm 2\%$, $38 \pm 2\%$ and $51 \pm 25\%$, respectively (Fig. S6, ESI). Interestingly, the BSF for HAN increased to a higher extent than for the AOX or the THM when increasing the bromide concentration. A higher stability of brominated HANs compared to their chlorinated analogues may partly explain this trend.³⁰

Overall, the pre-oxidation more efficiently mitigated the Cl-DBPs than the Br-DBPs (Figs. 3 and S5, ESI), consistent with previous studies.^{13, 21} For example an increase of BSF following the order O_3 -*t*-BuOH (+ 11%) > ClO_2 / Fe(VI) (+ 5%) > Mn(VII) (+ 2%) for THMs and $150 \mu\text{g L}^{-1}$ bromide was observed (Fig. 3b). The only exception was a reduction of the BSF by Mn(VII) for the HANs at $150 \mu\text{g L}^{-1}$ bromide (Fig. 3c), although the variations of the data were high. While the AOBBr formation was not significantly impacted by any pre-oxidant at 150 or $500 \mu\text{g L}^{-1}$

bromide ($\leq 10\%$ mitigation, Figs. 1b,c), pre-oxidation could enhance the concentration of brominated THMs and HANs. At $150 \mu\text{g L}^{-1}$ bromide, the sum of BDCM and DBCM concentrations increased with increasing dose for all the pre-oxidants (Fig. 1e). For $500 \mu\text{g L}^{-1}$ bromide, a similar trend was observed for DBCM and TBM concentrations (Fig. 1f). For the HANs, the main increase in concentration was observed for DBAN upon pre-oxidation (Figs. 1h, i). More specifically, the mitigation efficiency of a specific DBP was anticorrelated to the number of bromine atoms in the compound. In presence of $500 \mu\text{g L}^{-1}$ bromide, TCM and DBCM were mitigated by 19–85% and 8–37%, respectively, while DBCM and TBM concentrations were enhanced by 4–12% (Mn(VII) was still mitigating DBCM formation by 5%) and 7–195%, respectively (Fig. 4, THM mitigation is calculated based on the experiment without pre-oxidation). Similar patterns were observed for the THMs at $150 \mu\text{g L}^{-1}$ bromide (Fig. S7, ESI) and with HANs (Fig. S8, ESI).

3.4.3. Discussion.

In presence of chlorine, bromide is rapidly oxidized to HOBr,⁶⁵ which in turn reacts with NOM 1–2 orders of magnitude faster than chlorine.⁶⁶ About 20% of the HOBr will react through electrophilic aromatic substitutions, i.e., forming AOBBr, while the rest will be reduced back to bromide via electron transfer reactions.⁶⁷ When pre-oxidation was applied, the decrease of AOCl and the limited impact observed on AOBBr can be explained by four factors. (i) A reduction of the number of HOX-reactive sites. (ii) An excess of NOM sites reactive with HOX (HOCl or HOBr) after pre-oxidation treatment relative to the available HOBr. (iii) A re-oxidation of the bromide released in solution after electron transfer reactions by the excess chlorine until its complete incorporation in NOM.²³ (iv) A faster reaction of HOBr with NOM moieties compared to HOCl.^{66, 68} Therefore, even after pre-oxidation, bromine was fully incorporated in NOM while the remaining fraction of AOX precursors available to react with the excess of HOCl was limited, leading to a mitigation of AOCl (Figs. 1b–c). Furthermore, it was previously shown that bromine could substitute chlorine, which is already incorporated in NOM.²³

The decrease of reactive sites after pre-oxidation also favoured multiple bromination of the same site and the formation of di- and tri-Br-DBPs (Figs. 1e,f,h,i and Fig. 4). In contrast, the pre-oxidation did not enhance multiple chlorination since an excess of chlorine and a long reaction time already ensured multiple halogenation with or without pre-oxidation. Overall, for chlorine, the number of reactive sites is restrained, leading to a limited concentration of chlorinated DBPs.

3.5. Pre-oxidation efficiency on DBP mitigation during final chlorination of SRNOM: impact of pH

Experiments with 3 mgC L^{-1} of SRNOM were carried out at pH 6.5 (40 mM phosphate) without and with bromide ($150 \mu\text{g L}^{-1}$). The AOX, THM and HAN formations at pH 6.5 are given in Fig. S9 (ESI) and were compared to the corresponding experiments at pH 8 (40 mM borate, Fig. 1).

3.5.1. Chlorination alone.

Without pre-oxidation, during chlorination of SRNOM higher concentrations of AOX and HANs were produced at pH 6.5 ($17.6\text{--}18.2 \mu\text{M}$ of AOX, $74\text{--}87 \text{ nM}$ of HANs) (Figs. S9a, b, e, f, ESI) compared to pH 8 ($15.7\text{--}16.6 \mu\text{M}$ of AOX and $45\text{--}50 \text{ nM}$ of HANs) (Figs. 1a, b, g, h). However, THM formation was lower at pH 6.5 ($1.9\text{--}2.1 \mu\text{M}$, Figs. S9c, d, ESI) compared to pH 8 ($2.8\text{--}2.9 \mu\text{M}$, Figs. 1d, e). Therefore, at pH 6.5, THMs accounted for a significantly lower relative contribution to AOX compared to pH 8 (32–35% vs 49–54%). The lower HAN and higher THM formations at higher pH are consistent with their base-catalyzed degradation and formation, respectively.^{30, 31} However, the brominated HANs were not significantly affected by the pH, in agreement with the lower impact of pH on their stability compared to the Cl-HANs.³⁰

3.5.2. Pre-oxidation followed by chlorination.

3 mgC L^{-1} of SRNOM buffered at pH 6.5 (40 mM phosphate) was pre-treated with low and medium doses (medium and high for Mn(VII)) of each oxidant, in absence or presence of bromide ($150 \mu\text{g L}^{-1}$). To compare the impact of pH on the pre-oxidation efficiency, the DBP formation after post-chlorination was normalized based on the samples without pre-oxidation (Fig. 5, Figs. S10, S11, ESI).

For a medium oxidant dose, a larger fraction of the AOX was mitigated at pH 6.5 for Fe(VI) and $\text{O}_3\text{-t-BuOH}$ with about 15–20% and 60% AOX mitigation, respectively, compared to only < 2% and 40% at pH 8 (Fig. 5). As shown in Fig. 5, to achieve a relative AOX mitigation of 15–20%, Fe(VI) doses of about 15 or $50 \mu\text{M}$ were needed at pH 6.5 or 8, respectively. Similarly, to achieve 60% of relative AOX mitigation a $\text{O}_3\text{-t-BuOH}$ dose 3 times higher was needed at pH 8 compared to pH 6.5. The relative mitigation of THMs also increased when decreasing the pH after $\text{O}_3\text{-t-BuOH}$ pre-oxidation (Fig. S11, ESI). The improved mitigation of THMs but not HANs at lower pH by $\text{O}_3\text{-t-BuOH}$ is consistent with a previous study (conducted without hydroxyl radical scavenger and at a fixed chlorination pH), where increasing the ozonation pH from 6 to 9 decreased THM abatement from 35% to 21% while the formation of HAN was abated by about 44% across the pH range.¹⁶ In contrast, lowering the pH, decreased the relative reduction of AOX, THM and HAN formation by Mn(VII) (Figs. S10 to S12, ESI). No significant impact of the pH was observed on the ClO_2 mitigation efficiency (Figs. S10 to S12, ESI).

3.5.3. Discussion.

Based on the relative mitigation of DBPs, the positive effect of a lower pH during pre-oxidation increased in the order Mn(VII) < ClO_2 < Fe(VI) < $\text{O}_3\text{-t-BuOH}$. The better performance of $\text{O}_3\text{-t-BuOH}$ at lower pH can be attributed to a higher stability of O_3 with a higher ozone exposure, leading to an enhanced cleavage of aromatic rings.^{32, 46} In contrast to O_3 , the stability of Fe(VI) decreases with decreasing pH.³³ However, the Fe(VI) reactivity towards a large selection of organic NOM moieties also increases with decreasing pH, while the O_3 reactivity tends to decrease with decreasing pH.^{36, 69–71} For example, when

decreasing the pH from 8 to 6.5, the apparent second order rate constants for the reactions of Fe(VI) with phenol or 4-methylphenol increase by about 2–4 times, respectively, whereas they decrease about 30 times for the reactions with O_3 .^{36, 69} The improved mitigation of AOX at lower pH for Fe(VI) and O_3 -*t*-BuOH suggests that the higher reactivity of Fe(VI), and/or of its degradation products, towards AOX precursors at lower pH counterbalanced its enhanced decomposition, while the higher stability of O_3 at lower pH counterbalanced its lower reactivity towards AOX precursors.

It has to be noted that Fe(VI) self-decay is known to be affected by the type of buffer (by complexation of Fe(III)). However, no significant differences were observed between phosphate and borate at the high buffer concentrations used in our experiments (40 mM).^{72, 73} This was supported by the absence of precipitates at both pHs.

The self-decays of ClO_2 and Mn(VII) are slow and not affected by the pH, hence, not affecting the oxidation efficiency.³⁴ The lower efficiency of Mn(VII) pre-oxidation at lower pH could not be explained by its known chemistry (Figs. S10 to S12, ESI). It is important to state that the results are presented as relative abatement and that a higher formation of AOX and HANs was observed at pH 6.5 compared to pH 8 in samples without pre-oxidation.

Therefore, using a low pH during Fe(VI) or O_3 pre-treatment to enhance the degradation of DBP precursors and a higher pH for disinfection might further reduce the overall DBP formation. This strategy has previously been tested for O_3 pre-oxidation in a laboratory study and was shown to efficiently abate THMs while having no effect on HAN formation during post-chlorination.¹⁶

3.6. Pre-oxidation efficiency on DBP mitigation during final chlorination of SRNOM: calculated toxicity.

3.6.1. Chlorination alone.

Chronic cytotoxicity (cell density reduction over 72 h exposure) and acute genotoxicity (DNA damage over 4 h exposure) are two toxicity indicators which have been assessed for a wide range of DBPs.²⁵ The total relative cytotoxicity has previously been used to evaluate the impact of different treatment options on the DBP-induced toxicity potency.^{24, 74, 75} The total relative cytotoxicity is estimated by dividing the concentration of each DBP (C) by its specific cytotoxicity concentration (C_{50}). C_{50} is the concentration of a specific DBP resulting in a 50% reduction of Chinese hamster ovary cell densities.²⁵ The same approach can be applied for the specific genotoxicity of each DBP, represented by the migration of DNA from a Chinese hamster ovary nucleus, to calculate a total relative genotoxicity.²⁵ The relative cytotoxicity results will be presented and compared to the relative genotoxicity. In absence of other important classes of DBPs such as haloacetic acids, haloacetaldehydes, halonitromethanes or haloacetamides, the obtained evolution presented below is not an exhaustive representation of the DBP-induced estimated toxicity, but provides a conceptual trend analysis.

Fig. 6 shows the relative cytotoxicity of THMs and HANs (bars) compared to the sum of THM and HAN concentrations (triangles), without and with bromide ($150 \mu g L^{-1}$). The results for $500 \mu g L^{-1}$ bromide and for pH 6.5 are presented in Fig. S14 and Fig. S15 (ESI), respectively. Although the HAN concentrations were very low compared to the THMs, they are more potent compounds ($C_{50} = 10^{-3}$ – 10^{-2} M for THMs vs 10^{-6} – 10^{-4} M for HANs).⁷⁷ At pH 8 and without bromide, THM concentrations represented $\geq 98\%$ of the sum of the THMs and HANs concentrations, but accounted for $\leq 25\%$ of the relative cytotoxicity (Fig. 6a). The contribution of THMs to the relative cytotoxicity was lower in presence of bromide ($\leq 11\%$ and $\leq 5\%$ in presence of 150 and $500 \mu g L^{-1}$ bromide, respectively) (Fig. 6b, Fig. S14, ESI). If other potent DBPs were considered, THM contribution would be even lower. Since Br-DBPs are more potent toxicants than their chlorinated analogues, the total relative cytotoxicity increased with increasing bromide concentration. In presence of 150 and $500 \mu g L^{-1}$ bromide and at pH 8, the relative cytotoxicity increased by a factor 2.4 or 8.8, respectively, compared to samples without bromide, while the sum of the THM and HAN concentrations remained constant ($2.8 \pm 0.1 \mu M$, Fig. 6 and Fig. S14, ESI).

In comparison to pH 8, more HANs and less THMs were formed at pH 6.5 (Figs. 1d, e, g, h and Figs. S9c–f, ESI), hence increasing the total relative cytotoxicity by 50% in absence of bromide (compare data in Fig. 6a with Fig. S15a, ESI). It was previously shown that the brominated HANs were not increasing with decreasing pH (Fig. 1h and Fig. S9f, ESI). Therefore, no significant difference in total relative cytotoxicity was observed between pH 6.5 and 8 in presence of $150 \mu g L^{-1}$ bromide and without pre-oxidation (compare Fig. 6b and Fig. S15b, ESI). Although THMs are cytotoxic, their genotoxicity is not quantifiable.⁷⁷ Furthermore, the increase in genotoxicity when incorporating bromine in HANs is more pronounced compared to the cytotoxicity. DBAN cytotoxicity and genotoxicity are 20 and 58 times higher, respectively, compared to DCAN.²⁵ Therefore, the increase in total relative genotoxicity was more pronounced when adding 150 and $500 \mu g L^{-1}$ bromide (4 and 25 times higher, respectively, compared to samples without bromide, Fig S16, ESI).

3.6.2. Pre-oxidation followed by chlorination.

In absence of bromide and at pH 6.5 and 8, both TCM and DCAN were mitigated by pre-oxidation (Figs. 1d, g and Figs. S9c, e, ESI). Therefore, the calculated total relative cytotoxicity decreased (Fig. 6a and Fig. S15a, ESI).

In presence of $150 \mu g L^{-1}$ bromide and at pH 8, DCAN, BCAN and TCM formations were mitigated while TBM, DBCM and DBAN concentrations were enhanced (Figs. 1e, h). Although DBAN levels were very low (< 2 nM without pre-oxidation), their increase can overcome, in some cases, the benefit of DCAN and BCAN mitigation in terms of total relative cytotoxicity (Fig. 6b, the impact of THMs was negligible). The detrimental effect of pre-oxidation treatment on the total relative cytotoxicity is emphasised for O_3 -*t*-BuOH for solutions containing $150 \mu g L^{-1}$ bromide. The lowest total relative cytotoxicity was found for the lowest O_3 dose (Fig. 6b). While higher doses further decreased

DCAN, they also further enhanced DBAN formation. In addition, BCAN mitigation was less efficient at medium and high dose compared to low dose (Fig. 6b). Overall, the total relative cytotoxicity was reduced by 31% at the low O₃ dose but only by 5% at the high O₃ dose, whereas a continuous decrease of total DBP concentration was observed with increasing O₃ dose (Fig. 6b). In presence of bromide, the cytotoxicity therefore contrasted with the continuous decrease of total DBP concentration. Similar effects were observed at pH 6.5 and 8 for all the oxidants (Fig. 6b, Fig. S15b, ESI).

The detrimental effect of pre-oxidation due to the enhanced formation of brominated HANs was more pronounced for total relative genotoxicity, especially in presence of 150 µg L⁻¹ bromide for which ClO₂, Fe(VI) and O₃-*t*-BuOH pre-oxidation could enhance the total relative genotoxicity by up to 13, 16 and 50%, respectively (Fig. S16b, ESI). Nevertheless, a medium dose of ClO₂ reduced DCAN and BCAN formation without enhancing DBAN formation at both pH 6.5 and 8, leading to an efficient reduction in both cytotoxicity and genotoxicity (5–47%, Fig. 6b, Fig. S15b and Figs. S16b-c, ESI).

3.6.3. Discussion.

Other compounds, e.g., haloacetic acids, haloacetaldehydes, halonitromethanes or haloacetamides, were not monitored in this study and are likely to impact the cytotoxicity and genotoxicity. The formation of these compounds may be affected by pre-oxidation differently compared to HANs. In particular, DBPs such as halonitromethanes,^{13, 16, 40} chloral hydrate or halopropanones can be enhanced by pre-oxidation.^{13, 16, 21, 40}

As brominated DBPs are generally more toxic than their chlorinated analogues, the increased relative cytotoxicity or genotoxicity with increasing bromide observed in Fig. 6 and Fig. S14-S16 (ESI) is likely to extend to other emerging DBPs.^{25, 75}

The impact of the pH on the formation of other DBPs may vary. For example, halopropanone and halonitromethane formation will decrease and increase, respectively, with increasing pH.^{78, 79} Therefore, the overall impact of pH on cytotoxicity/genotoxicity would require a more extended DBP monitoring.

Although not exhaustive, the toxicity calculations presented here suggest that (i) THMs have a limited significance in terms of toxicity, and that they are not necessarily a good surrogate for the mitigation of other DBPs (here HANs) and (ii) in presence of bromide, the formation of brominated emerging DBPs needs to be considered, because they may heavily impact the overall toxicity.

3.7. Pre-oxidation efficiency for DBP mitigation in a real groundwater.

The different pre-oxidants were tested in a real groundwater, W20, (main characteristics are given in Table 2). O₃ was also tested without *t*-BuOH to account for the impact of hydroxyl radical formation. The doses of pre-oxidants were chosen based on a 50% reduction of the EDC for W20 and are shown in Table 1.

Without pre-oxidation, chlorination of W20 with a dose of 12.4 mg L⁻¹ (72 h) produced 23.8 ± 0.9 µM AOX, 2.4 ± 0.1 µM THMs and 143 ± 16 nM HANs (Figs. 7a-c). Similarly to SRNOM at pH 6.5 (Figs. S9b, d, ESI), THMs and HANs represented 30% and 1.2% of the AOX, respectively (Figs. 7a-c). The presence of 144 µg L⁻¹ bromide led to the formation of Br-DBPs.

Under our experimental conditions, the AOX and HAN formation was reduced by pre-oxidation while THM formation was slightly enhanced by all pre-oxidants. AOX mitigation efficiency ranked in the order O₃-*t*-BuOH (33%) > Fe(VI) (25%) > O₃ (14%) > Mn(VII) (7%) > ClO₂ (5%) (Fig. 7a). HAN mitigation efficiency ranked in the order Fe(VI) (72%) > O₃-*t*-BuOH (60%) > O₃ (56%) > Mn(VII) (46%) > ClO₂ (41%) (Fig. 7c). THM formation after pre-oxidation was found to increase after all pre-oxidation treatments, in the order O₃-*t*-BuOH (+7%) < ClO₂ (+13%) < Fe(VI) (+15%) < Mn(VII) (+16%) < O₃ (+22%) (Fig. 7b). The increase in THM formation observed in the real water was a major difference with the results observed in the SRNOM extract (Figs. S9b, d, ESI) and contrasted with the decrease in AOX (Fig. 7a). This increase in THM formation may be explained by the difference in NOM characteristics possibly leading to a higher transformation of activated aromatic compounds to aliphatic β-keto-acid and β-diketone structures which lead to less halogenation (AOX decrease) but a higher yield of THMs.⁶¹ Higher pre-oxidant dosage may be necessary to efficiently oxidize THM precursors.

Similar to SRNOM, the AOB_r formation was not significantly affected by pre-oxidation (≤ 10% reduction, Fig. 7a) due to the excess of chlorine, re-oxidising bromide until complete incorporation of bromine in NOM. The formation of brominated and chlorinated THMs was enhanced to a similar extent by ClO₂, Fe(VI) and Mn(VII), while O₃ (with and without *t*-BuOH) enhanced the brominated THMs to a slightly higher extent compared to the chlorinated THMs (Fig. 7b). This was illustrated by the increase of the BSF from 9% to 15% and 13% after pre-oxidation by O₃-*t*-BuOH and O₃, respectively (Fig. 7b). The sum of brominated HANs was efficiently mitigated by ClO₂ and Fe(VI) (38–43%) while O₃-*t*-BuOH, O₃ and Mn(VII) had little impact (7–12%) (Fig. 7c). Furthermore, O₃-*t*-BuOH and O₃ strongly enhanced the formation of DBAN (+ 333–362%, Fig. 7c), for which the concentrations were still low (about 6 nM). Overall, ClO₂ was the only oxidant that mitigated the formation of chlorinated and brominated HANs to a similar extent while O₃-*t*-BuOH, O₃, Fe(VI) and Mn(VII) increased the BSF from 7% to 21%, 19%, 16% and 12%, respectively (Fig. 7c). In comparison to O₃ alone, the presence of *t*-BuOH increased the O₃ exposure and thus improved the mitigation of AOX and HANs while the increase of THMs was limited (Fig. 7).

Finally, for the real water, the efficiency of pre-oxidation on the calculated reduction of the relative cytotoxicity ranked in the order Fe(VI) (54%) > ClO₂ (39%) > Mn(VII) (29%) > O₃-*t*-BuOH (11%) > O₃ (9%) (Fig. S17a, ESI). The same ranking was observed for the total relative genotoxicity, although the enhancement of DBAN by O₃ pre-oxidation (with and without *t*-BuOH) led to an increase of the total relative genotoxicity (Fig. S17b, ESI). The main difference compared to the DBP mitigation in SRNOM (pH 6.5, 150 µg L⁻¹ bromide, medium doses) was the higher

efficiency of Fe(VI) and Mn(VII), relatively to the other oxidants, on HAN mitigation, and consequently on the cumulative estimated toxicity, in W20.

Conclusions

Pre-oxidation has been shown to be a viable option to reduce DBP formation during post-chlorination. Based on an equivalent EDC reduction, O₃-*t*-BuOH was generally the most efficient oxidant to mitigate total AOX, total THMs and total HANs while Mn(VII) was often the least efficient option, particularly for THMs.

The doses of O₃ (2.0–94.3 μM, 0.1–4.5 mg L⁻¹), Fe(VI) (2.5–50 μM, 0.1–2.8 mg Fe L⁻¹) and Mn(VI) (1–8.6 μM, 0.05–0.5 mg Mn L⁻¹) were within the range of concentrations typically used in drinking water treatment, although Fe(VI) and Mn(VI) doses can be significantly higher.^{20, 80, 81} In practice, the ClO₂ dose is limited to about 15 μM (1 mg L⁻¹) due to the regulation of chlorite,⁸² which is lower than the highest dose used in this study (39 μM, 2.8 mg L⁻¹). However, chlorite can be removed in presence of GAC,⁸³ ferrous ions,⁸⁴ or oxidized during post-chlorination. During pre-oxidation, O₃ decomposition leads to the formation of hydroxyl radicals which reduces its exposure, and decreases its efficiency,¹⁶ as confirmed by experiments (with and without *t*-BuOH) in W20 (Figure 7).

Decreasing the pH increased the concentration of AOX and HANs while decreasing that of THMs after chlorination. Compared to samples without pre-oxidation, a lower pH improved the relative AOX mitigation by O₃-*t*-BuOH and Fe(VI) pre-oxidation and the relative THM mitigation by O₃-*t*-BuOH pre-oxidation. However, a lower pH was decreasing the relative mitigation of all DBPs by Mn(VII). To minimize the concentration of the AOX and HANs after post-chlorination, pre-oxidation with O₃ or Fe(VI) should be carried out at lower pH, while raising the pH before post-chlorination. Other DBP classes would need to be evaluated as pH can impact their formation differently.^{78, 79}

While Cl-DBPs were efficiently mitigated, in presence of bromide the mitigation efficiency of Br-DBPs was much lower. The extent of specific DBP mitigation was anticorrelated to the number of bromine atoms in the compounds. In many cases, highly brominated DBPs were enhanced by increasing the pre-oxidant doses, with O₃-*t*-BuOH generally having the highest detrimental effect.

The assessment of toxicity based on individual relative cytotoxicity of THMs and HANs suggests that regulated THMs are not significantly affecting the DBP-induced toxicity, especially in presence of bromide, and are not a good surrogate for the mitigation of more potent DBPs such as HANs. Conversely, the impact of pre-oxidation on HAN formation is likely to be an important factor for toxicity assessment, and the enhancement of DBAN was a major factor counteracting the efficiency of pre-oxidation in terms of relative toxicity for all oxidants, particularly O₃. Rather than a decreasing relative toxicity with increasing doses of oxidants, optimal conditions were often observed at low or medium doses. Such conditions would also be desired to avoid excessive formation of bromate after O₃ pre-oxidation of bromide-containing waters,⁸⁵ or of

chlorite after ClO₂ pre-oxidation. The formation of other DBPs such as the widespread haloacetic acids or other very potent DBPs such as haloacetamide, haloketones, halonitromethanes and iodinated DBPs, should heavily influence the relative toxicity,⁷⁷ and their formation may be impacted very differently upon pre-oxidation.^{14, 86} Given the large pool of DBPs that may affect the toxicity, toxicity assays should be performed to fully evaluate the efficiency of the different pre-oxidation strategies.

The present study compares the effects of oxidative pre-treatments on DBP precursors. The same chlorine dose was used with and without pre-oxidation, which led to chlorine residuals much higher than 1.5 mgCl₂ L⁻¹ in pre-oxidized samples (Fig. S18, ESI). In practice, the chlorine dose could therefore be optimized, which will reduce the overall formation of DBPs. Furthermore, supplementary treatments are likely to be applied between pre-oxidation and disinfection such as coagulation-sedimentation-filtration or the use of biofiltration to remove the assimilable organic carbon and/or DBP precursors that may arise from pre-oxidation, notably from O₃, Fe(VI) and Mn(VII) treatment.⁵¹

Conflicts of interest

There are no conflicts to declare.

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Table 1 Doses of pre-oxidants applied, and corresponding EDC reduction at pH 8. The range of data represents triplicate experiments.

Oxidant	Applied dose (μM)				% EDC reduction			
	Low	SRNOM Medium	High	W20 Medium	Low	SRNOM Medium	High	W20 Medium
ClO_2	3.5	15.1	39.3	10	27 ± 3	56 ± 2	77 ± 1	50
O_3 - <i>t</i> -BuOH	2.0	36.1	94.3	30	9 ± 2	61 ± 2	74 ± 3	59
O_3				30				50
Fe(VI)	2.5	14.7	50	24	20 ± 2	49 ± 3	68 ± 2	50
Mn(VII)		1	8.6	10		36 ± 2	69 ± 1	42

Table 2 Main characteristics of the real and synthetic waters. The EDC is presented relative to a 3 mgC L^{-1} unbuffered SRNOM sample.

NOM	pH	DOC (mgC L^{-1})	SUVA ₂₅₄ ($\text{L mgC}^{-1} \text{ cm}^{-1}$)	Bromide ($\mu\text{g L}^{-1}$)	Relative EDC
SRNOM	6.5 or 8 (40 mM phosphate or borate)	3	4.0	0, 150 or 500	1.04
W20	6.5 (natural water)	4.5	3.4	144	2.04

Figure 1 Formation of (a–c) AOX, (d–f) THMs and (g–i) HANs after chlorination of SRNOM at pH 8, with and without (blank) pre-oxidation. For each pre-oxidant, the first bar represents the blank and the following bars the samples pre-oxidized with a low, medium and high doses. Pre-oxidant doses are given in Table 1. Chlorine dose = 85 µM (6.0 mgCl₂ L⁻¹), [SRNOM] = 3 mgC L⁻¹, pH 8 (40 mM borate), [Br⁻] = 0–500 µg L⁻¹, 72 h. Error bars represent the range of analytical results for duplicate experiments (except the high dose of Fe(VI) and Mn(VII) for which error bars represent duplicate analysis).

Figure 2 Impact of bromide on the total HAN mitigation by pre-oxidation of SRNOM with ClO_2 , $\text{O}_3\text{-t-BuOH}$, Fe(VI) and Mn(VII) . The % mitigation is presented relative to the HAN formation without pre-oxidation. A negative mitigation represents an increase in HAN formation. Pre-oxidant doses are given in Table 1. Chlorine dose = $85 \mu\text{M}$ ($6.0 \text{ mgCl}_2 \text{ L}^{-1}$), $[\text{SRNOM}] = 3 \text{ mgC L}^{-1}$, pH 8, 72 h. Error bars represent the range of analytical results for duplicate experiments (except the high dose of Fe(VI) and Mn(VII) for which error bars represent duplicate analysis).

Figure 3 Bromine Substitution Factor (BSF) in (a) AOX, (b) THMs and (c) HANs after chlorination of SRNOM in presence of $150 \mu\text{g L}^{-1}$ bromide, with and without pre-oxidation. Pre-oxidant doses are given in Table 1. Chlorine dose = $85 \mu\text{M}$ ($6.0 \text{ mgCl}_2 \text{ L}^{-1}$), $[\text{SRNOM}] = 3 \text{ mgC L}^{-1}$, pH 8, 72 h. Error bars represent the range of analytical results for duplicate experiments (except the high dose of Fe(VI) and Mn(VII) for which error bars represent duplicate analysis).

Figure 4 Chlorination of SRNOM: Mitigation of individual THMs by a high dose of pre-oxidants in presence of $500 \mu\text{g L}^{-1}$ bromide. The % mitigation is presented relatively to the THM formation without pre-oxidation. A negative mitigation represents an increase in THM formation. Pre-oxidant doses: $[\text{ClO}_2] = 39.3 \mu\text{M}$, $[\text{O}_3\text{-t-BuOH}] = 94.3 \mu\text{M}$, $[\text{Fe(VI)}] = 50 \mu\text{M}$, $[\text{Mn(VII)}] = 8.6 \mu\text{M}$. Chlorine dose = $85 \mu\text{M}$ ($6.0 \text{ mgCl}_2 \text{ L}^{-1}$), $[\text{SRNOM}] = 3 \text{ mgC L}^{-1}$, pH 8 (40 mM borate), 72 h. Error bars represent the range of duplicate analysis for one experiment.

Figure 5 Chlorination of SRNOM: Relative AOX formation after O_3 -t-BuOH (orange symbols) and Fe(VI) (green symbols) pre-oxidation at pH 6.5 (crosses) and 8 (circles). Chlorine dose = $85 \mu\text{M}$ ($6.0 \text{ mgCl}_2 \text{ L}^{-1}$), $[\text{SRNOM}] = 3 \text{ mgC L}^{-1}$, $[\text{Br}^-] = 0\text{--}150 \mu\text{g L}^{-1}$, 40 mM phosphate (pH 6.5) or borate (pH 8), 72 h. Error bars represent the range of duplicate analysis for one experiment.

Figure 6 Calculated relative cytotoxicity (bars) compared to DBP formation (triangles) after chlorination of SRNOM and without/with pre-oxidation (a) without bromide and (b) with $150 \mu\text{g L}^{-1}$ bromide. Pre-oxidant doses are given in Table 1. Chlorine dose = $85 \mu\text{M}$ ($6.0 \text{ mgCl}_2 \text{ L}^{-1}$), $[\text{SRNOM}] = 3 \text{ mgC L}^{-1}$, pH 8 (40 mM borate), 72 h. For more clarity, error bars are not shown.

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Figure 7 Chlorination of a real water (W20): Formation of (a) AOX, (b) THMs and (c) HANs without and with pre-oxidation. The bromine substitution factors (BSF) are also shown for THMs and HANs. Chlorine dose = 155 μM (12.4 $\text{mgCl}_2 \text{ L}^{-1}$), 72 h. The pre-oxidant doses and the water characteristics are given in Table 1 and Table 2, respectively. Error bars represent the range of duplicate analysis for one experiment.