The Formation of Halogen-Specific TOX from Chlorination and Chloramination of Natural Organic Matter Isolates

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The formation of disinfection by-products (DBPs) is a public health concern. An important way to evaluate the presence of DBPs is in terms of the total organic halogen (TOX), which can be further specified into total organic chlorine (TOCl), bromine (TOBr), and iodine (TOI). The formation and distribution of halogen-specific TOX during chlorination and chloramination of natural organic matter (NOM) isolates in the presence of bromide and iodide ions were studied. As expected, chloramination produced significantly less TOX than chlorination. TOCl was the dominant species formed in both chlorination and chloramination. TOI was always produced in chloramination, but not in chlorination when high chlorine dose was used, due to the limited presence of HOI in chlorination as a result of the oxidation of iodide to iodate in the presence of excess chlorine. The formation of TOI during chloramination increased as the initial iodide ion concentration increased, with a maximum of ~60% of the initial iodide ion becoming incorporated into NOM. Iodine incorporation in NOM was consistently higher than bromine incorporation, demonstrating that the competitive reactions between bromine and iodine species in chloramination favoured the formation of HOI and thus TOI, rather than TOBr. Correlations between the aromatic character of the NOM isolates (SUVA$_{254}$ and % aromatic C) and the concentrations of overall TOX and halogen-specific TOX in chloramination were observed. This indicates that the aromatic moieties in NOM, as indicated by SUVA$_{254}$ and % aromatic C, play an important role in the formation of overall TOX and halogen-specific TOX in chloramination. THMs comprised only a fraction of TOX, up to 7% in chloramination and up to 47% in chlorination. Although chloramine produces less TOX than chlorine, it formed proportionally more non-THM DBPs than chlorine. These non-THM DBPs are mostly unknown, corresponding to unknown health risks. Considering the higher potential for formation of iodinated DBPs and
unknown DBPs associated with the use of chloramine, water utilities need to carefully balance the risks and benefits of using chloramine as an alternative disinfectant to chlorine in order to satisfy guideline values for THMs.

**Keywords:** Total organic halogen, iodide, bromide, disinfection by-products, trihalomethanes, chlorination, chloramination, natural organic matter

### 1 Introduction

Disinfection is an integral part of drinking water treatment due to its crucial role in preventing the spread of water-borne diseases. However, as well as inactivating microorganisms in the water, chemical disinfectants also react with natural organic matter (NOM) and inorganic materials in the water, producing disinfection by-products (DBPs). The formation of DBPs from disinfection practices is a public health concern and a drinking water treatment problem (Richardson *et al.*, 2002).

Organic DBPs can be quantified as individual species, *e.g.* trihalomethanes (THMs), haloacetic acids (HAAs), and haloacetonitriles (HANs), or as total organic halogen (TOX). TOX is a group parameter that is used to characterise the incorporation of halogen into organic molecules, regardless of their identity. Chlorine and chloramine are the two major disinfectants that produce significant amounts of TOX. In chlorinated samples, only about 50% of TOX can reportedly be assigned to individual species, while, in chloraminated samples, the corresponding value is less
than 20% (Richardson, 2003; Li et al., 2002). TOX therefore provides a measure of the overall organic halogenated DBP formation which cannot be provided by analysis of specific DBPs. It is also an alternative to the measurement of individual DBPs, which cannot be done routinely due to the diversity and the large range of molecular weights of the DBPs formed.

In the presence of bromide and iodide ions, chlorinated organic compounds (TOCl), as well as brominated (TOBr) and iodinated (TOI) organic compounds, are formed. TOX analysis can thus be further specified into TOCl, TOBr, and TOI. The knowledge of the proportions of TOBr and TOI is important for the evaluation of the health risks of DBPs. Brominated and iodinated DBPs are now being recognised as toxicologically important. Many brominated DBPs have been shown to be more carcinogenic than their chlorinated analogues, while iodinated compounds may be more toxic than their brominated analogues (Plewa et al., 2004; Li et al., 2002).

The conventional method for TOX measurement combines carbon adsorption, pyrolysis, and measurement of hydrogen halide by microcoulometry. The method cannot differentiate between the different halides as it measures all halogens as chloride equivalents. In order to differentiate the halogen constituents in TOX, a different method of halide detection is required. Ion chromatography has been the popular choice for the differential analysis of TOCl, TOBr, and TOI (Hua and Reckhow, 2006; Echigo et al., 2000; Oleksy-Frenzel et al., 2000). Using ion chromatography, chloride, bromide, and iodide ions contained in the halide mixture can be individually measured.

There have been many studies reporting the formation and behaviour of TOX from chlorination of water samples and NOM isolates (e.g. Li et al., 2002; Baribeau et al., 2001; Pourmoghaddas...
and Stevens, 1995). However, there are only a few studies on the formation of TOX from chloramination (Wu et al., 2003; Diehl et al., 2000; Symons et al., 1998), and those that compare the formation of TOX from chlorination and chloramination of water samples and NOM isolates (Hua and Reckhow, 2007; Wu et al., 2003; Zhang et al., 2000). The extent to which bromide and iodide ions can influence the formation of TOX is also not well known. Most of the previously published studies only measured TOX, not the halogen-specific fractions of TOX (TOCl, TOBr, and TOI). Echigo et al. (2000) compared the concentrations of TOCl and TOBr in water samples treated with chlorine, chloramine, and chlorine dioxide, while Oleksy-Frenzel et al. (2000) measured the concentrations of TOCl, TOBr, and TOI in drinking water and wastewater samples. Recently, the formation of all three species of TOX (TOCl, TOBr, and TOI) from chlorination of natural waters in the presence of bromide and iodide ions was reported (Hua and Reckhow, 2006; Hua et al., 2006).

This work aimed to bridge the knowledge gaps in studies of TOX formation. The formation and distribution of halogen-specific TOX, namely TOCl, TOBr, and TOI, upon chlorination and chloramination of natural organic matter (NOM) isolates in the presence of bromide and iodide ions were studied. This is the first study to investigate the influence of the characteristics of NOM and the concentrations of bromide and iodide ions on the formation of all three species of halogen-specific TOX in chloramination.

2 Materials and Methods

2.1 NOM Samples
NOM samples were collected from the following sources: Loire River and Ribou Reservoir in France; and Suwannee River, Colorado River, and South Platte River in the United States. The NOM in raw water samples from these sources was fractionated using XAD resins. Details of the fractionation procedure have been presented elsewhere (Croué et al., 2000). The hydrophobic (HPO) fraction refers to the NOM fraction recovered from the XAD-8 resin by elution with an acetonitrile/water mixture; the hydrophobic acid (HPOA) fraction refers to the NOM fraction recovered from the XAD-8 resin by elution with NaOH solution; the transphilic (TPI) fraction refers to the NOM fraction recovered from the XAD-4 resin by elution with an acetonitrile/water mixture. Solid samples of the NOM fractions were obtained from the respective eluents by freeze-drying.

The NOM fractions were characterised by determination of their specific UV$_{254}$ values (SUVA$_{254}$ = UV$_{254}$/mg/L of dissolved organic carbon (DOC)) and % aromatic carbon content. The aromaticity of the samples was determined using solid state $^{13}$C nuclear magnetic resonance (NMR) spectroscopy. Details of the procedures used to characterise the NOM fractions have been presented elsewhere (Croué et al., 2000). Details of the NOM fractions used in this work and their relevant characteristics are given in Table 1. For the disinfection experiments, these fractions were individually dissolved in MilliQ (MQ) water at concentrations of 5 mg solid NOM fraction per litre. The DOC concentrations of these solutions were determined by the UV/persulfate oxidation method, using a Shimadzu TOC Analyser TOC-VWS.

2.2 Disinfection Experiments

The hydrophobic fraction from the Loire River (LR HPO) (5 mg L$^{-1}$ of NOM sample in MQ water, corresponding to 2.62 mg L$^{-1}$ DOC) was subjected to chlorination (10 mg L$^{-1}$ Cl$_2$) and
chloramination (preformed chloramine, mass ratio of Cl$_2$ : N = 4 : 1, 10 mg L$^{-1}$ as total Cl$_2$) in the presence of added bromide (300 µg L$^{-1}$) and iodide (50 µg L$^{-1}$) ions. Relatively high chlorine and chloramine doses were used in these experiments to ensure that the disinfectant is always in excess, which will result in significant TOX formation. The experiments were carried out at 20$^\circ$C and 50$^\circ$C, for 48 hours, at pH 7 for chlorination and pH 8 for chloramination, using phosphate buffer (10 mM). Chlorination experiments were carried out at pH 7 to simulate the commonly used pH in chlorination practices (White, 1999), and since it is the pH used in the Standard Method for determination of trihalomethane formation potential (Clesceri et al., 1998). A higher pH was used in the chloramination experiments to ensure that monochloramine was the active species during chloramination, and to minimise the decomposition of monochloramine (Symons et al., 1998). The expected difference in the formation of disinfection by-products between chlorination and chloramination would have been enhanced if pH 8 was used in chlorination experiments (Reckhow and Singer, 1985). Since the comparison of the two oxidants did not represent the major objective of this work, conducting chlorination at pH 8 was not considered.

In each experiment, at various times up to 48 hr, the residual chlorine in a subsample of the reaction solution was quenched with aqueous sodium sulfite solution, and the sample was then analysed for halogen-specific TOX and THMs.

A separate set of chloramination experiments was carried out, using all 9 NOM samples, in which the concentration of iodide ion was varied from 50 to 300 µg L$^{-1}$ (0.39 to 2.36 µM), while the concentration of bromide ion was kept constant at 300 µg L$^{-1}$ (3.75 µM). Another set of chloramination experiments was carried out, using 7 of the 9 NOM samples, in which the concentration of bromide ion was varied from 32 to 190 µg L$^{-1}$ (0.40 to 2.40 µM i.e. equal molar
concentrations to 50 – 300 µg L\(^{-1}\) iodide ion), while the concentration of iodide ion was kept constant at 475 µg L\(^{-1}\) (3.74 µM i.e. equal molar concentration to 300 µg L\(^{-1}\) bromide ion). These chloramination experiments were all performed at 20°C and pH 8. Relatively high concentrations of bromide and iodide ions were used in these experiments to represent source waters that contain high concentrations of these ions, where significant formation of brominated and iodinated DBPs are expected (Richardson, 2007). Sampling for the analysis of halogen-specific TOX in these experiments was carried out only at the end of the 48-hour experimental period, after quenching with sodium sulfite.

2.3 Chlorine and Chloramine Measurements

Chlorine and chloramine (NH\(_2\)Cl) were analysed using the N,N-diethylphenylene-1,4-diamine (DPD) colorimetric method (Ventresque et al., 1990). For free chlorine determination, 250 µL of a pH 6.8 phosphate buffer solution (24 g L\(^{-1}\) Na\(_2\)HPO\(_4\) and 46 g L\(^{-1}\) KH\(_2\)PO\(_4\), with added 0.8 g L\(^{-1}\) disodium EDTA and 0.02 g L\(^{-1}\) HgCl\(_2\)) was mixed with 250 µL of aqueous DPD solution (2 mL concentrated H\(_2\)SO\(_4\); 0.2 g L\(^{-1}\) disodium EDTA; 1.1 g L\(^{-1}\) anhydrous N,N-diethylphenylene-1,4-diamine) and 5 mL of sample. The absorbance of the resulting solution was then measured at 510 nm using a SAFAS 320 spectrophotometer. For total chlorine (chloramine) determination, a similar procedure was followed, except that potassium iodide (~50 mg per sample) was added to the solution prior to measurement of its absorbance.

2.4 Halogen-Specific TOX Analysis

The analysis of halogen-specific TOX was based on the method developed by Hua and Reckhow (2006) with minor modifications. The sample for TOX analysis was firstly acidified to pH 2 and
enriched through adsorption onto an activated carbon column using a Dohrmann® AD-3 Adsorption Module. The activated carbon was then placed in a quartz sample boat and introduced into the combustion chamber of a Dohrmann® DX20 TOX Analyser. The activated carbon sample was combusted in the presence of oxygen for 10 minutes at 1000°C. The hydrogen halide gases produced were collected in MQ water by way of a custom-made absorber. The MQ water was then analysed for Cl⁻, Br⁻, and I⁻ using a Dionex® DX400 ion chromatograph with conductimetric detection and an ASRS Ultra II Anion Self-Regenerating Suppressor. For the analysis of Cl⁻ and Br⁻, an AS9-HC column (Dionex®) was used with 9 mM Na₂CO₃ solution as mobile phase. For the analysis of I⁻, an AS11 column (Dionex®) was used with 20 mM NaOH solution as mobile phase. The concentrations of Cl⁻, Br⁻, and I⁻ (in µg L⁻¹) obtained from the ion chromatographic (IC) analysis were used to calculate the sample concentration of TOCl (as µg L⁻¹ Cl⁻), TOBr (as µg L⁻¹ Br⁻), and TOI (as µg L⁻¹ I⁻), respectively, taking into account the concentration factors from the initial sample to the absorber solution. Where the concentration of TOX is given as a Cl equivalent concentration (µg L⁻¹ Cl), it refers to the sum of the molar concentrations of TOCl, TOBr, and TOI, multiplied by the atomic mass of Cl.

2.5 THMs Analysis

THMs were extracted from the samples by headspace extraction (Dani HSS 3950 headspace sampler), and analysed using gas chromatography (Varian 3300) with an electron capture detection (HS/GC-ECD) (Hureiki et al., 1994). GC separation of THMs was carried out using high purity nitrogen as the carrier gas and a DB-624 megabore capillary column (J&W; 30 m x 0.53 mm, film thickness 0.25 µm).
3 Results and Discussion

3.1 Validation of the Halogen-Specific TOX Analytical Method

The analysis of halogen-specific TOX was carried out according to the method developed by Hua and Reckhow (2006), with minor modifications to adapt to the instrument and material availability in our laboratory. The recovery of the method was evaluated by determination of the recoveries of several model compounds containing chlorine, bromine, and iodine. These model compounds were bromoform, dichloroacetonitrile, trichloroacetic acid, 2,4,6-tribromophenol, 4-iodophenol and iodoacetic acid. The recoveries of these compounds were studied in a concentration range of 20 to 100 µg L\(^{-1}\) in the aqueous samples, which corresponds to a concentration range of 100 to 500 µg L\(^{-1}\) in the IC samples, due to the five fold concentration factor in the method. All analyses were conducted in duplicate. Good recoveries, between 85 to 109%, were obtained in the present study, comparing well with analytical recoveries reported for halogen-specific TOX by other researchers (Hua and Reckhow, 2006; Echigo et al., 2000; Oleksy-Frenzel et al., 2000).

Matrix effects were not observed in the analysis of halogen-specific TOX using the modified method. Good precision of the method was demonstrated, with % RSD (% Relative Standard Deviation = 100 x [standard deviation / mean]) values of five replicates analysis ranging from 2 – 9%. Low detection limits for the ion chromatographic determination of halides were obtained. Detection limits of 5 µg L\(^{-1}\), 2 µg L\(^{-1}\), and 2 µg L\(^{-1}\) were achieved for the analysis of chloride, bromide, and iodide, respectively.

3.2 The Formation of DBPs in Chlorination vs. Chloramination
A hydrophobic fraction isolated from the Loire River (LR HPO) was subjected to chlorination (10 mg L\(^{-1}\); pH 7, DOC = 2.62 mg C L\(^{-1}\)) and chloramination (10 mg L\(^{-1}\) as total Cl\(_2\); pH 8) in the presence of bromide (300 µg L\(^{-1}\)) and iodide (50 µg L\(^{-1}\)) ions for 48 hours. The concentrations of halogen-specific TOX and THMs in these samples were measured. Figures 1 and 2 show the production of halogen-specific TOX (as µmol L\(^{-1}\) halide) at 20°C over the experimental period in the chlorination and chloramination experiments, respectively, while Figure 3 shows the production of THMs in both experiments. Table 2 presents the specific yield of halogen-specific TOX and the disinfectant demand after 48 hours contact time for both chloramination and chlorination, as well as the ratio of TOX to disinfectant demand. Separate chlorination and chloramination experiments in the presence of iodide ion only (200 µg L\(^{-1}\)) were carried out using SR HPOA, in order to study the effect of chlorine and chloramine dose on the formation of TOI. Various doses of chlorine and chloramine between 1 and 10 mg Cl\(_2\) L\(^{-1}\) were used, and only the concentration of TOI was measured in these samples. Table 3 gives the TOI formation at 48-hour contact time for this set of experiments.

TOCl and TOBr were detected and quantified in both chloramination and chlorination experiments (Figures 1 and 2, Table 2). In contrast, TOI was always formed in chloramination experiments (see Tables 2 and 3), but only formed in chlorination experiments where the chlorine doses were less than 10 mg L\(^{-1}\) (Table 3). The results presented in Table 3 demonstrate that the concentration of chlorine significantly affects the formation of TOI, whereas the concentration of chloramine has little effect on the concentration of TOI. In this experiment, at high chlorine concentration of 10 mg L\(^{-1}\), where the initial molar ratio of chlorine to iodide was 90, excess chlorine was likely to have oxidised all iodide ion to HOI, and then further oxidised the HOI to
iodate. Unlike chlorine, chloramine is only able to oxidise iodide ion to HOI, which is then available for reactions with NOM (Bichsel and von Gunten, 1999) to form TOI. In this case, since chloramine was always in excess compared to iodide, the chloramine concentration had no effect on the amount of TOI formed (Table 3).

In both chlorination and chloramination, TOCl was the dominant TOX produced (Table 2). In the chlorination experiments, TOCl accounted for 77% of TOX on a molar concentration basis, while the corresponding value for chloramination was 88%. The high proportion of TOCl formed is associated with the high disinfectant dose used in these experiments relative to the typical chlorine doses used in the field. With higher iodide ion concentrations, chloramination was found to result in TOI formation higher than TOCl formation (see Section 3.4).

Chloramination produced significantly less TOX and THMs than chlorination (Table 2, Figure 3). For a 48-hour contact time, chloramination resulted in a 72% lower concentration of TOCl and a 93% lower concentration of TOBr than chlorination (Table 2), which corresponds to an overall TOX (TOCl + TOBr only) reduction of 67% upon chloramination. The final concentration of total THMs was reduced by 95% upon chloramination (Figure 3). These results are consistent with other reported studies (e.g. Richardson, 2003; Wu et al., 2003; Richardson et al., 2002), where chloramine has been found to produce less DBPs than chlorine, due to the lower reactivity of chloramine.

Numerous studies have reported that identified DBPs, such as THMs and HAAs, account for only a fraction of TOX (e.g. Richardson, 2003; Zhang et al., 2000; Symons et al., 1998). This was also observed in the chlorination and chloramination of LR HPOA. In the chloramination experiments
at 20°C, THMs represented only 7% of TOX, while, in chlorination, 47% of TOX could be accounted for by THMs. The proportions of THMs in TOX were lower in the chloramination experiments due to the relative oxidising strengths of chlorine versus chloramine. As a stronger oxidant, chlorine, in the form of HOCl, reacts with NOM mainly through oxidation reactions, producing cleavage by-products such as THMs (Li et al., 2002). Chloramine, however, is a weaker oxidant than chlorine. The formation of DBPs in chloramination is thought to be a result of reactions between NOM and small amounts of HOCl present in equilibrium with chloramine (Duirk et al., 2002; Cowman and Singer, 1996), as well as through direct reaction of chloramine with NOM (Duirk et al., 2002). Since there are less reactive species in a chloraminated system, the formation of cleavage by-products, such as THMs, is less favoured, and the formation of by-products of higher molecular weight is preferred (Johnson and Jensen, 1986). Therefore, the THMs form only a small percentage of the TOX measured in chloraminated samples.

The relative oxidizing strength and the reactivity of chlorine and chloramine were also reflected in the kinetics of the formation of TOX and THMs. In chlorination, 35% of TOX produced after 48 hours was formed in the first 30 minutes, while the corresponding value for THM formation at the same time was 30%. In chloramination, 55% of the total TOX produced was formed in the first 30 minutes, and the corresponding value for THM formation was 15%. These results demonstrate that chloramination favours the formation of non-THMs halogenated by-products, and that the formation of THMs during chloramination would only be the result of reactions between NOM and residual free chlorine (HOCl) that is present in equilibrium with chloramine (NH₂Cl).
3.3 The Effect of Temperature on the Formation of TOX in Chlorination and Chloramination

In order to evaluate the influence of temperature on the formation of halogen-specific TOX, chlorination and chloramination experiments with LR HPO NOM were performed at 20°C and 50°C. The concentrations of halogen-specific TOX formed at these temperatures are presented in Table 2. In both chlorination and chloramination, higher concentrations of halogen-specific TOX were formed at the higher temperature, which was expected, since an increase in temperature has been associated with a higher rate and a greater extent of formation of DBPs (e.g. Carlson and Hardy, 1998; Engerholm and Amy, 1983). Further data observation showed that increasing the temperature from 20°C to 50°C did not have a significant effect on the proportions of TOCl and TOBr in both chlorination and chloramination experiments. Therefore, the proportions of TOX in chlorination and chloramination are not influenced by temperature, rather, they appear to be primarily determined by the relative amounts of the halogen species in the system.

Furthermore, although the concentrations of halogen-specific TOX increased with increasing temperature, the amount of halogen-specific TOX produced per mg of disinfectant consumed was found to be higher at 20°C than at 50°C, for both chlorination and chloramination experiments (Table 2). This demonstrates that the proportion of the disinfectant used in non-TOX forming reactions, e.g. oxidation reactions forming non-halogenated DBPs, increases when the temperature increases.

3.4 The Effect of Iodide Ion and Bromide Ion on the Distribution of TOX in Chloramination
The effect of iodide ion and bromide ion concentrations on the formation and distribution of halogen-specific TOX upon chloramination of a selection of NOM isolates was investigated. NOM fractions were separately subjected to chloramination (10 mg L\(^{-1}\) as total Cl\(_2\); pH 8) for 48 hours and for different concentrations of iodide and bromide ions: a constant iodide ion concentration of 3.74 µM (475 µg L\(^{-1}\)), with initial concentrations of bromide ion varying from 0.40 µM to 2.40 µM (32 to 190 µg L\(^{-1}\)); and a constant bromide ion concentration of 3.75 µM (300 µg L\(^{-1}\)), with initial concentrations of iodide ion varying from 0.39 to 2.36 µM (50 to 300 µg L\(^{-1}\)). Figure 4 shows, as an example, a typical halogen-specific TOX distribution obtained from the LR HPOA isolate, illustrating the effect of varying bromide ion concentrations (Figure 4a) and iodide ion concentrations (Figure 4b).

In these experiments, for all conditions, TOCl and TOI were the dominant TOX produced, while TOBr was produced at concentrations more than ten-fold lower than those of TOCl and TOI. The average concentration of TOBr formed was approximately 1.5 µg eq Cl / mg C when the initial bromide ion concentration was 300 µg L\(^{-1}\). Although the concentrations of TOBr increased slightly with increasing initial bromide ion concentration, the bromine incorporation (percentage of the initial bromide ion incorporated into TOBr i.e. \(\frac{[TOBr]}{[Br^{-]}_{\text{initial}} \times 100}\)) decreased, for all NOM isolates (Table 4). The maximum bromine incorporation was 18%, which was achieved in the chloramination of SPR HPOA with an initial bromide ion concentration of 32 µg L\(^{-1}\). The change in the initial concentration of bromide ion was found to have no significant effect on the concentrations of TOCl, TOI, and TOX.
High linear correlations were observed between the production of TOI and the initial iodide ion concentration ($r^2 = 0.95 – 0.99$) (see Figure 4b for results from LR HPOA). As a result of the increase in TOI concentration with increasing initial iodide ion concentration, the overall TOX concentration also increased. Unlike the observations from varying the initial bromide ion concentration, iodine incorporation (percentage of the initial iodide ion incorporated into TOI i.e. $\frac{[\text{TOI}]}{[\Gamma]_{\text{initial}} \times 100}$) was found to increase as the initial iodide ion concentration increased, varying from 8 to 64%, depending on the NOM isolate and the initial iodide ion concentration (Table 4). At constant bromide ion concentration (300 $\mu$g L$^{-1}$), TOBr production was not significantly affected by the increase in initial iodide ion concentration (Figure 4b).

The observed lower proportion of TOBr compared to TOI, and the lower bromine incorporation compared to iodine incorporation in chloramination can be explained by a low concentration of HOBr in the system. In chloramination, a variety of bromine species can be formed, but not all contribute to DBP formation. $\text{NH}_2\text{Cl}$ reacts with bromide ion to form bromochloramine as shown by the following equation (Gazda et al., 1993):

$$\text{NH}_2\text{Cl} + \text{Br}^- \rightleftharpoons \text{NHBrCl}$$

Small amounts of HOBr could be produced by the reaction between bromide ion and HOC1, present in equilibrium with $\text{NH}_2\text{Cl}$. The HOBr formed could then react with $\text{NH}_2\text{Cl}$, as well as $\text{NH}_3$ that results from chloramine decay, to produce $\text{NH}_2\text{Br}$ and other bromamine species, as shown by the following equations (Gazda et al., 1993; Wajon and Morris, 1980):

$$\text{HOBr} + \text{NH}_2\text{Cl} \rightleftharpoons \text{NHBrCl and NBr}_2\text{Cl}$$
$$\text{HOBr} + \text{NH}_3 \rightleftharpoons \text{NH}_2\text{Br} + \text{H}_2\text{O}$$
$$\text{NH}_2\text{Cl} + \text{NHBr}_2 \rightleftharpoons \text{NHBrCl and NBr}_2\text{Cl}$$
$$\text{NH}_2\text{Cl} + \text{NH}_2\text{Br} \rightleftharpoons \text{NHBrCl}$$
In addition, HOBr could also be consumed in reactions with iodide and HOI, which would reduce
its availability to react with NOM. HOBr could induce the oxidation of iodide ion in the same
manner as HOCl (Hua et al., 2006), and it has been reported to oxidise iodide to HOI through an
IBr intermediate (Troy and Margerum, 1991).

Bromamines and bromochloramine do not react with NOM as readily as HOBr, and their
reactivities in the formation of DBPs are largely unknown (Symons et al., 1998). Therefore, the
likely formation of bromamines and bromochloramine in chloramination would limit the steady
state concentration of active bromine species, and thus the formation of TOBr.

In contrast to bromide, NH$_2$Cl oxidizes iodide ion into hypoiodous acid, which is not further
oxidized by NH$_2$Cl (Bichsel and von Gunten, 1999). HOI is then available for fast reactions with
NOM, leading to iodosated DBPs (Bichsel and von Gunten, 2000). Even though HOI is also
involved in oxidation reactions with formation of iodide, iodide is continuously reoxidised into
HOI, which favours the substitution reactions and explains the high incorporation of iodide into
NOM as TOI.

### 3.5 The Influence of NOM Characteristics on the Formation of TOX in Chloramination

In chlorination, aromatic structures within NOM have been reported to be especially reactive
with chlorine in producing DBPs, and significant correlations have been observed between
aromaticity (as indicated by UV$_{254}$ or SUVA$_{254}$, and % aromatic C) and DBP formation (Croué et
al., 2000; Wu et al., 2000; Reckhow et al., 1990). In light of this, correlations between the
production of organohalides (TOX as µg L$^{-1}$ Cl, TOCl as µg L$^{-1}$ Cl, TOBr as µg L$^{-1}$ Br, and TOI
as µg L⁻¹ I) in chloramination and the characteristics of NOM (SUVA₂₅₄ and % aromatic C) were examined in the present study. Data from the chloramination experiments using 300 µg L⁻¹ bromide ion and 50 µg L⁻¹ iodide ion was used for this purpose. The correlation coefficient values obtained for the different linear correlations evaluated in the present study are presented in Table 5. Good linear correlations were observed for TOCl and TOBr with SUVA₂₅₄ and % aromatic C, indicating that aromatic moieties play a significant role in the formation of TOCl and TOBr. Similar correlations were also obtained for TOX since TOCl was the major contributor to TOX. Other researchers have reported that a linear correlation exists between SUVA₂₅₄ and the formation of TOX in chlorination (Rostad et al., 2000; Krasner et al., 1996) and chloramination (Wu et al., 2003), and also between TOX and % aromatic C in chloramination (Wu et al., 2003). Wu et al. (2003) reported r² = 0.69 for the correlation between aromatic C and TOX formation from chloramination of humic substance samples. Rostad et al. (2000) reported r² = 0.67 for the correlation of 7-day TOX formation potential and SUVA₂₅₄ in the chlorination of tertiary treated wastewaters from Arizona, USA. Krasner et al. (1996) obtained r² = 0.93 for the correlation of specific yield of TOX and SUVA₂₅₄ in the chlorination of NOM fractions from Apremont reservoir, France. This data indicates that the fraction of NOM represented by SUVA₂₅₄ and % aromatic C plays an important role in the formation of overall TOX, in both chlorination and chloramination.

For a low iodide ion concentration of 50 µg L⁻¹ (i.e. 0.39 µM), TOI formation was low for all NOM isolates, and no linear correlation was observed between TOI and SUVA₂₅₄ or % aromatic C (Table 5). For this condition, the character of NOM was not a predominant factor influencing the formation of TOI. As it was shown previously in Section 3.4, the concentrations of TOI
linearly increased with increasing iodide ion concentration \textit{i.e.} with increasing concentration of reactive iodine species. Linear regression was fitted to graphs of [TOI] \textit{vs.} initial [I] for all NOM isolates. Different values of the slope for this correlation were obtained and the following trend was observed: the higher the SUVA$_{254}$ value, the higher the reactivity of NOM and the higher the value of [TOI] \textit{vs.} [I] slope. Fitting a logarithmic regression to the data points showed a relationship between the slope of TOI formation and SUVA$_{254}$ values, with a significant $r^2$ value of 0.875 (Figure 5). Furthermore, a good linear correlation was also obtained between TOI concentrations and SUVA$_{254}$ values ($r^2 = 0.983$, results not shown) for the experiments conducted using high initial iodide ion concentration of 475 µg L$^{-1}$ (\textit{i.e.} 3.74 µM) and various initial bromide ion concentrations. This means that aromatic moieties in NOM, as indicated by SUVA$_{254}$, are major reactive sites for the production of TOI in chloramination of iodide-containing waters, which is in accordance with the general findings of NOM reactivity with halogenated oxidants. Bichsel and von Gunten (2000) showed that phenolic moieties (but not carbonyl moieties) could account for the observed reactivity of HOI with NOM. The present study further demonstrates that the aromaticity of NOM significantly affects the formation of DBPs in general, and halogen-specific TOX (TOCl, TOBr, and TOI) in particular.

4 Conclusions

The formation of halogen-specific TOX upon chlorination and chloramination of NOM isolates in the presence of bromide and iodide ions was studied and the following conclusions were made:

- Chloramination produced significantly less TOX and THMs than chlorination.
- In both chlorination and chloramination, TOCl was the dominant TOX species produced, as a result of the high ratios of Cl$_2$/Br$^-$ and Cl$_2$/I$^-$ used in these experiments. A higher proportion
of TOCl was observed in chloraminated samples, indicating limited formation of TOBr in chloramination due to the formation of bromamines instead of HOBBr.

• TOI was always produced during chloramination, which is in agreement with the known chemistry of iodine in water treatment. In chlorination, chlorine oxidises iodide through to iodate, limiting the presence of HOI for reactions with NOM to produce TOI. A suitable chlorine dose, sufficient to completely oxidise iodide to iodate, should be utilised for source waters containing iodide, since it will limit the formation of TOI, which is considered to be more harmful than TOCl and TOBr.

• In chlorination, THMs constituted 47% of TOX, while THMs comprised only 7% of TOX in chloramination. Although chloramine produced less TOX than chlorine, it formed proportionally more non-THM DBPs. These non-THM DBPs are mostly unknown, corresponding to unknown health risks. The results of this study highlight that better understanding and estimation of the health risks associated with chloramination DBPs are needed.

• The formation of TOI in chloramination increased as the initial iodide ion concentration increased. A maximum of ~60% of the initial iodide ion was incorporated into NOM and measured as TOI. Iodine incorporation into NOM was consistently higher than bromine incorporation, although the molar concentrations of initial iodide ion were lower than bromide ion. Competitive reactions between bromine and iodine species were found to favour the formation of HOI and thus TOI, rather than TOBr. NH$_2$Cl reacts with iodide to produce only HOI, which then reacts with NOM to produce TOI; while NH$_2$Cl reacts with bromide forming HOBBr, as well as other bromine species such as bromamine and bromochloramine, which have little contribution to the formation of TOBr, and HOBBr could
also be consumed in reactions with iodide to produce HOI, rather than in reactions with
NOM to produce TOBr. This study shows that the presence of bromide and iodide ions
significantly affected the extent of TOX formation, as well as the distribution of TOX
species. Therefore, inorganic precursors of DBPs also need to be seriously considered in
efforts to minimise DBP formation and risks.

- This study demonstrates that high concentrations of TOI could be formed, and that the
  formation of TOI is favoured, in chloramination. Recent research has shown that iodinated
  DBPs are more toxic and pose a greater health risk than chlorinated and brominated DBPs.
  Since chloramine also has the potential to form proportionally higher unknown DBPs, water
  utilities need to carefully balance the risks and benefits of using chloramine as an alternative
disinfectant to satisfy guideline values for THMs.

- Good linear correlations between the aromatic character of the NOM isolates (SUVA \textsubscript{254} and
  % aromatic C) and the formation of TOX, TOCl, and TOBr in chloramination were observed,
  indicating that aromatic moieties play an important role in the formation of TOX, TOCl, and
  TOBr in chloramination. For all NOM isolates, the formation of TOI linearly increased with
  increasing initial iodide ion concentration and the slopes of linear regression of $[\text{TOI}]$ vs.
  initial $[\text{I}^-]$ were well correlated to the aromatic character of NOM (SUVA \textsubscript{254} and % aromatic
  C). Hence, the aromaticity of NOM also affected the formation of TOI in chloramination.

Based on these results, it is recommended that removal of the aromatic fraction of NOM
during water treatment is maximised, in order to limit the formation of TOX in chlorination
and chloramination.
Acknowledgements

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References


**Tables and Figures**

**Table 1:** NOM fractions and their characteristics

<table>
<thead>
<tr>
<th>NOM Source</th>
<th>Fraction</th>
<th>Abbreviation</th>
<th>SUVA_{254} (L mg^{-1} m^{-1})</th>
<th>% Aromatic Carbon</th>
</tr>
</thead>
<tbody>
<tr>
<td>Loire River</td>
<td>Hydrophobic Acid</td>
<td>LR HPOA</td>
<td>3.1</td>
<td>15</td>
</tr>
<tr>
<td>Loire River</td>
<td>Hydrophobic (HPO)</td>
<td>LR HPO</td>
<td>2.9</td>
<td>18</td>
</tr>
<tr>
<td>Loire River</td>
<td>Transphilic (TPI)</td>
<td>LR TPI</td>
<td>2.0</td>
<td>11</td>
</tr>
<tr>
<td>Suwannee River</td>
<td>HPOA</td>
<td>SR HPOA</td>
<td>4.6</td>
<td>26</td>
</tr>
<tr>
<td>Ribou Reservoir</td>
<td>HPO</td>
<td>RR HPO</td>
<td>3.4</td>
<td>16</td>
</tr>
<tr>
<td>Ribou Reservoir</td>
<td>TPI</td>
<td>RR TPI</td>
<td>2.1</td>
<td>9</td>
</tr>
<tr>
<td>Colorado River</td>
<td>HPO</td>
<td>CR HPO</td>
<td>1.8</td>
<td>14</td>
</tr>
<tr>
<td>Colorado River</td>
<td>TPI</td>
<td>CR TPI</td>
<td>1.4</td>
<td>10</td>
</tr>
<tr>
<td>South Platte River</td>
<td>HPOA</td>
<td>SPR HPOA</td>
<td>2.9</td>
<td>12</td>
</tr>
</tbody>
</table>

**Table 2:** Specific yield of halogen-specific TOX (µg as Cl/mg C) from chlorination (Cl₂ dose 10 mg L^{-1}, pH 7) and chloramination (NH₂Cl dose 10 mg L^{-1}, pH 8) of LR HPO at 20°C and 50°C ([Br⁻] = 300 µg L^{-1}; [I⁻] = 50 µg L^{-1}; 48 hours contact time)

<table>
<thead>
<tr>
<th></th>
<th>20°C</th>
<th>50°C</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CHLORINATION (pH 7, 10 mg L^{-1} Cl₂)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TOCl</td>
<td>76</td>
<td>89</td>
</tr>
<tr>
<td>TOBr</td>
<td>23</td>
<td>34</td>
</tr>
<tr>
<td>TOI</td>
<td></td>
<td>not detected</td>
</tr>
<tr>
<td>TOX</td>
<td>99</td>
<td>123</td>
</tr>
<tr>
<td>Cl₂ demand (mg L^{-1})</td>
<td>3.7</td>
<td>7.8</td>
</tr>
<tr>
<td>TOX in µg Cl/mg Cl₂</td>
<td>26.8</td>
<td>15.8</td>
</tr>
<tr>
<td><strong>CHLORAMINATION (pH 8, 10 mg L^{-1} NH₂Cl)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TOCl</td>
<td>22</td>
<td>33</td>
</tr>
<tr>
<td>TOBr</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>TOI</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>TOX</td>
<td>25</td>
<td>37</td>
</tr>
<tr>
<td>NH₂Cl demand (mg L^{-1})</td>
<td>0.7</td>
<td>3.3</td>
</tr>
<tr>
<td>TOX in µg Cl/mg NHCl₂</td>
<td>35.7</td>
<td>11.2</td>
</tr>
</tbody>
</table>
Table 3: TOI formation in chlorination (Cl₂ dose 10 mg L⁻¹, pH 7) and chloramination (NH₂Cl dose 10 mg L⁻¹, pH 8) of SR HPOA at various disinfectant doses (DOC = 2.9 mg C L⁻¹; [I⁻] = 200 µg L⁻¹, 20°C, 48 hours contact time)

<table>
<thead>
<tr>
<th>Disinfectant dose</th>
<th>Chlorination</th>
<th>Chloramination</th>
</tr>
</thead>
<tbody>
<tr>
<td>mg Cl₂ L⁻¹</td>
<td>Cl₂/I molar ratio</td>
<td>µg TOI as Cl/mg C</td>
</tr>
<tr>
<td>1</td>
<td>9</td>
<td>9</td>
</tr>
<tr>
<td>2</td>
<td>18</td>
<td>10</td>
</tr>
<tr>
<td>5</td>
<td>45</td>
<td>2</td>
</tr>
<tr>
<td>10</td>
<td>90</td>
<td>not detected</td>
</tr>
</tbody>
</table>

Table 4: Iodine and bromine incorporation into NOM in chloramination experiments (5 mg L⁻¹ NOM, NH₂Cl dose 10 mg L⁻¹, pH 8, 20°C, 48 hours contact time)

<table>
<thead>
<tr>
<th>Sample</th>
<th>% Iodine incorporation at various initial [I⁻]</th>
<th>% Bromine incorporation at various initial [Br⁻]</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>50 µg L⁻¹</td>
<td>100 µg L⁻¹</td>
</tr>
<tr>
<td>LR HPOA</td>
<td>30</td>
<td>40</td>
</tr>
<tr>
<td>LR HPO</td>
<td>11</td>
<td>24</td>
</tr>
<tr>
<td>LR TPI</td>
<td>24</td>
<td>28</td>
</tr>
<tr>
<td>SR HPOA</td>
<td>12</td>
<td>24</td>
</tr>
<tr>
<td>RR HPOA</td>
<td>40</td>
<td>32</td>
</tr>
<tr>
<td>RR TPI</td>
<td>28</td>
<td>26</td>
</tr>
<tr>
<td>CR HPOA</td>
<td>10</td>
<td>19</td>
</tr>
<tr>
<td>CR TPI</td>
<td>8</td>
<td>13</td>
</tr>
<tr>
<td>SPR HPOA</td>
<td>28</td>
<td>28</td>
</tr>
</tbody>
</table>

nd = not determined

Table 5: Linear correlation coefficient (r²) values for the correlation between overall and specific TOX concentrations and SUVA₂₅⁴ and % aromatic C (NH₂Cl dose 10 mg L⁻¹, 5 mg L⁻¹ NOM, 50 µg L⁻¹ iodide ion, 300 µg L⁻¹ bromide ion, pH 8, contact time 48 hours, 20°C)

<table>
<thead>
<tr>
<th>Linear Correlation</th>
<th>r²</th>
</tr>
</thead>
<tbody>
<tr>
<td>SUVA₂₅⁴ – TOX</td>
<td>0.711</td>
</tr>
<tr>
<td>SUVA₂₅⁴ – TOCl</td>
<td>0.700</td>
</tr>
<tr>
<td>SUVA₂₅⁴ – TOBr</td>
<td>0.723</td>
</tr>
<tr>
<td>SUVA₂₅⁴ – TOI</td>
<td>0.035</td>
</tr>
<tr>
<td>% aromatic C – TOX</td>
<td>0.792</td>
</tr>
<tr>
<td>% aromatic C – TOCl</td>
<td>0.792</td>
</tr>
<tr>
<td>% aromatic C – TOBr</td>
<td>0.758</td>
</tr>
<tr>
<td>% aromatic C – TOI</td>
<td>0.008</td>
</tr>
</tbody>
</table>
Figure 1: Formation of TOCl and TOBr in the chlorination of LR HPO (2.6 mg L\(^{-1}\) DOC, 10 mg L\(^{-1}\) Cl\(_2\), pH 7, 300 µg L\(^{-1}\) bromide ion, 50 µg L\(^{-1}\) iodide ion, 20\(^\circ\)C). TOI was not detected in all samples.

Figure 2: Formation of TOCl, TOBr and TOI in the chloramination of LR HPO (2.6 mg L\(^{-1}\) DOC, 10 mg L\(^{-1}\) NH\(_2\)Cl, pH 8, 300 µg L\(^{-1}\) bromide ion, 50 µg L\(^{-1}\) iodide ion, 20\(^\circ\)C)
Figure 3: Formation of total THMs in the chlorination and chloramination of LR HPO (2.6 mg L\(^{-1}\) DOC, 300 µg L\(^{-1}\) bromide ion, 50 µg L\(^{-1}\) iodide ion, 20ºC, chlorination: 10 mg L\(^{-1}\) Cl\(_2\) at pH 7, chloramination: 10 mg L\(^{-1}\) NH\(_2\)Cl at pH 8)
Figure 4: TOX distribution after chloramination of LR HPOA, with varying initial a) bromide ion and b) iodide ion concentration (2.5 mg L\(^{-1}\) DOC, NH\(_2\)Cl dose 10 mg L\(^{-1}\), pH 8, 20\(^\circ\)C)
Figure 5: Logarithmic function of SUVA\textsubscript{254} values and the slopes of the linear regressions of [TOI] and initial [I] for all NOM isolates