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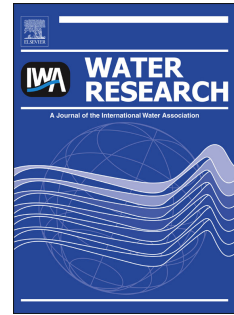Authors: Cynthia M.M. Bougeard, Emma H. Goslan, Bruce Jefferson, Simon A. Parsons

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1 **Comparison of the disinfection by-product formation potential of treated waters**

2 **exposed to chlorine and monochloramine**

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8

9 **Abstract**

10 The formation of disinfection by-products (DBPs) from chlorination and

11 monochloramination of treated drinking waters was determined. Samples were collected

12 after treatment at 11 water treatment works but before exposure to chlorine or

13 monochloramine. Formation potential tests were carried out to determine the DBPs

14 formed by chlorination and monochloramination. DBPs measured were trihalomethanes

15 (THMs), haloacetic acids (HAAs), halonitromethanes, haloacetonitriles, haloketones

16 and iodo-THMs. All waters had the potential to form significant levels of all the DBPs

17 measured. Compared to chlorine, monochloramination generally resulted in lower

18 concentrations of DBPs with the exception of 1,1-dichloropropanone. The

19 concentrations of THMs correlated well with the HAAs formed. The impact of bromine

20 on the speciation of the DBPs was determined. The literature findings that higher

21 bromide levels lead to higher concentrations of brominated DBPS were confirmed.

22

23 **Key words**: disinfection by-products, trihalomethanes, haloacetic acids,

24 haloacetonitriles, monochloramination, semi-volatile DBPs

25

26 **Abbreviations**

27 1,1-DCP – 1,1- dichloropropanone

28 1,1,1-TCP – 1,1,1- trichloropropanone

29 BCAA – bromochloroacetic acid

30 BCIM – bromochloroiodomethane

31 BDCAA – bromodichloroacetic acid

32 BDCM - bromodichloromethane

33   BIF – bromine incorporation factor

34   CHO – Chinese hamster ovary cells

35   DBAA – dibromoacetic acid

36   DBAN –dibromoacetonitrile

37   DBCAA – dibromochloroacetic acid

38   DBCM – dibromochloromethane

39   DBIM – dibromoiodomethane

40   DBNM – dibromonitromethane

41   DBPs – disinfection by-products

42   DCA – dichloroacetaldehyde

43   DCAA – dichloroacetic acid

44   DCAN - dichloroacetonitrile

45   DCBM – dichlorobromomethane

46   DCIM – dichloroiodomethane

47   DXAA – dihalogenated acetic acids

48   FP – formation potential

49   GC-ECD – gas chromatography electron capture detection

50   HAs - haloaldehydes

51   HAAs – haloacetic acids

52   HANs – haloacetonitriles

53   HKs – haloketones

54   HNMs – halonitromethanes

55   HOBr/OBr⁻ – hypobromous acid

56   HOI – hypoiodous acid

57   ICP/MS – inductively coupled plasma/mass spectrometry

58   IO_3^- - iodate

59   i-THMs – iodo THMs

60   MXAA – monohalogenated acetic acids

61   MtBE – methyl tert butyl ether

62   NaOCl – hypochlorous acid

63   NOM – natural organic matter

64   NPOC – non-purgeable organic carbon

65   SUVA – specific ultraviolet absorbance

66   TBAA – tribromoacetic acid

67   TBM – tribromomethane

68   TCA – trichloroacetaldehyde

69   TCAA – trichloroacetic acid

70   TCAN – trichloroacetonitrile

71   TCM – trichloromethane

72   TCNM – trichloronitromethane

73   TXAA – trihalogenated acetic acids

74   THMs – trihalomethanes

75   US EPA – United States Environmental Protection Agency

76   UV – ultraviolet absorbance

77

78   **Introduction**

79   Drinking water disinfection by-products (DBPs) result from the reaction of

80   disinfectants, such as chlorine or chloramines, with natural organic matter (NOM)

81   and/or bromide/iodide present in drinking water supplies (Rook et al., 1974).

82   Trihalomethanes are the only regulated DBP in the UK and it is required by law that the

83   sum of four THMs does not exceed 100 µg L^{-1} with a frequency of sampling dependent

84   on the population size. Haloacetic acids (HAAs) are often found to be as prevalent as

85   THMs but are currently not regulated in the UK. However, the European Union is

86   considering regulating the nine HAAs at 80 µg L^{-1} (Cortvriend, 2008) and as such there

87   is growing interest in the levels of these compounds found in UK drinking waters and

88   how best to control them . In order to comply with these proposed regulations, there

89   has been an increasing interest in using monochloramine as a secondary disinfectant

90   because of reduced DBP formation and its ability to provide residuals in water

91   distribution systems. Monochloramine is known to only form trace amounts of THMs

92   and HAAs, but the formation of dihalogenated HAAs (DXAAs), although generally

93   lower than with chlorine, can still reach significant levels depending on the dose,

94   chlorine to ammonia ratio, pH and other conditions (Diehl et al., 2000; Hua and

95   Reckhow, 2007). The use of monochloramine may also lead to an increase in other

96   DBPs such as haloacetonitriles (HANs) and iodo- THMs (i-THMs) (Krasner et al.,

97   1989; Bichsel and Von Gunten, 2000). HANs and i-THMs are two unregulated classes

98   of semi-volatile DBPs also present in disinfected waters alongside other unregulated

99   DBPs including halonitromethanes (HNMs), haloaldehydes (HAs) and haloketones

100   (HKs) (Krasner et al., 2006). These semi-volatile DBPs are of interest because of their

101   toxicity. HANs have been reported to be genotoxic and potentially carcinogenic for

102   human health and HKs exerted carcinogenic or mutagenic effects in mice (Bull and

103   Robinson, 1986; Daniel et al., 1986). Plewa et al. (2004) found HNMs to be toxic in

104   chinese hamster ovary cells (CHO) and Richardson (2003) suggested than i-THMs

105   could be more toxic than their brominated and chlorinated analogues. Despite their

106   potential health effects, there is no UK or US regulatory limit for these compounds, but

107   the WHO has suggested guideline values of 20 µg/L for DCAN, 70 µg/L for DBAN and

108   10 µg/L for TCA (WHO, 2006).

109   Past research has established that levels of HAAs and THMs in chlorinated waters vary

110   according to the levels of their precursors. High NOM concentrations have generally

111   been associated with high HAA and THM concentrations (Liang and Singer, 2003;

112   Sharp et al., 2006) and nitrogenous precursors from algae or effluent organic matter

113   (EfOM) have been related to nitrogenous DBPs, such as HANs (Oliver et al., 1983).

114   The presence of bromide in water will also affect the concentration of DBPs as will

115   other factors such as the disinfectant dose applied, the pH, the temperature of the water

116   samples and the reaction time of disinfectant in water (Singer et al., 2002). To better

117   control and understand the formation of DBPs in water samples, the use of formation

118   potential (FP) tests have been widely used (Zhang et al., 2000; Liang and Singer, 2003;

119   Ates et al., 2007; Krasner et al., 2007). FP tests are usually conducted at bench scale

120   with controlled pH, controlled temperature and relatively high chlorine concentration

121   dosed for a long contact time in order to maximise DBPs formation (Krasner et al.,

122   2007).

123   To have a better understanding of HAAs, THMs and semi-volatile DBPs in treated

124   waters, their formation was evaluated under controlled conditions. Here 11 water

125   treatment works selected from across England and Wales have been surveyed to allow

126   us to determine the potential for formation, relative distribution and speciation of DBPs

127   as well as identify any relationships between water sources. The DBPs selected include

128   THM_4 (trichloromethane   (TCM),   bromodichloromethane   (BDCM),

129   dibromochloromethane (DBCM) and tribromomethane (TBM)), HAA_9 (MCAA,

130   MBAA, DCAA, TCAA, bromochloroacetic acid (BCAA), DBAA, bromodichloroacetic

131   acid (BDCAA), dibromochloroacetic acid (DBCAA), and tribromomethane (TBAA))

132   plus four HANs (DCAN, trichloroacetonitrile (TCAN), bromochloroacetonitrile

133   (BCAN) and DBAN), two HKs (1,1-dichloropropanone (1,1-DCP) and 1,1,1-

134   trichloropropanone (1,1,1-TCP)), two HAs (dichloroacetaldehyde (DCA) and TCA),

135   two HNMs (trichlornitromethane (TCNM) and dibromonitromethane (DBNM)) and two

136   i-THMs (dichloroiodomethane (DCIM) and bromochloroiodomethane (BCIM)). This is

137   the first study that has reported the potential for formation of HAAs, THMs and a range

138   of semi-volatile DBPs in drinking water in England and Wales. It is also the first

139   European study to directly assess what impact the switch from chlorine to

140   monochloramine would have on the concentrations of the DBPs found.

141

142

143   **Materials and methods**

144   **Water samples**

145   Treated water samples were collected in July 2008 from 11 water treatment works,

146   spread geographically across England and Wales (Table 1). Samples were collected

147   prior to disinfection in polyethylene or glass 1L bottles and shipped to Cranfield

148   laboratory. These were then analysed for pH, non-purgeable organic matter (NPOC)

149   using a TOC 5000 Analyser (Shimadzu, Milton Keynes, UK), ultraviolet (UV)

150   absorbance at 254 nm, and specific ultraviolet absorbance (SUVA), which was

151   calculated as the ratio of UV absorbance at 254 nm (m^{-1}) to NPOC (mg C L^{-1}). NPOC

152   was used rather than DOC/TOC as the level of inorganic carbon in some samples was

153   too high to enable accurate DOC/TOC measurement. Bromide and iodine were

154   measured using inductively coupled plasma/mass spectrometry (ICP/MS) (Elan 9000,

155   Perkin Elmer, UK). Total bromine and iodine measured were assumed to be primarily

156   bromide and iodide.

157   **Sample preparation**

158   Chlorine and monochloramine solutions were prepared following the 4500-Cl B.

159   Iodometric method I and 4500-Cl F. DPD Ferrous Titrimetric Method respectively

160   (APHA, 1992). For the chlorinated samples, a 100 mL bottle was partly filled with the

161   water sample, the buffer at pH 7.2 and the chlorine solution (chlorine:NPOC ratio was

162   3:1 on a weight basis). The bottle was filled completely and capped headspace free with

163   a PTFE-lined cap. Samples were incubated for 24 hours at 20°C in the dark. For the

164   monochloraminated samples, a chlorine to nitrogen mass ratio of 3:1 was used in all

165   samples and addition of monochloramine was based on the NPOC level, with combined

166   chlorine:NPOC ratio of 3:1 by weight. The procedure of monochloraminated samples

167   was the same as that for chlorinated samples.

168   Ammonium chloride at a concentration of 100 mg L^{-1} was used to quench chlorine and

169   monochloramine residual while not degrading HAAs, in particular HAA_3 (BDCAA,

170   DBCAA and TBAA) (Singer et al., 2002). Ascorbic acid at a concentration of 35 mg L^{-1}

171   was used to quench chlorine and monochloramine residual in THM and semi-volatile

172   DBP samples. The choice is based on the fact that ascorbic acid has been shown not to

173   degrade any of these 16 DBPs (Chinn et al., 2007).

174

175   **DBP analytical methods**

176   HAA_9 were extracted with a modified version of the US EPA Method 552.2 reported by

177   Tung et al. (2006). The HAAs were converted to their methyl esters and quantified

178   using gas chromatography coupled with an electron capture detector (GC/ECD)

179   (Agilent 6890). THM$_4$, four HANs, two HKs, two HAs, two HNMs and two i-THMs

180   were extracted with an adapted method from Krasner et al. (2001). Standards for THM$_4$,

181   for halogenated volatiles as a mixture of DBPs (DCAN, TCAN, BCAN, DBAN, 1,1-

182   DCP, 1,1,1-TCP and TCNM) and for TCA were available from Sigma-Aldrich Ltd

183   (UK). DCA standard was provided by TCI Europe (Belgium); DBNM, DCIM and

184   BCIM were obtained from Helix Biotech (Canada). A 30 mL sample was transferred to

185   a 60 ml glass vial, then adjusted to a pH of 3.5 or less and extracted with 3 mL of MtBE

186   containing an internal standard. The solvent phase containing the DBPs was separated

187   from the aqueous phase by addition of 10 g of sodium sulphate and 1 g copper sulphate.

188   Then the sample was shaken manually for 3 to 5 minutes. Once settled the top layer was

189   finally transferred to an autosampler vial and analysed with GC/ECD (Agilent 6890).

190   The instrument conditions were as follows. A volume of 1 µL was injected splitless

191   with the detector set at 200°C. Separation was performed by a ZB-1ms column (30 m ×

192   0.25 mm × 0.25 µm) with a helium carrier gas at a column flow rate of 1.0 mL/min. The

193   initial oven temperature was 35°C and held for 22 minutes followed by a 10°C per

194   minute temperature ramp to 145°C and held for 2 minutes and a final ramp of 20°C per

195   minute ramp to 225°C and held for 10 minutes. The total run time was 49 minutes. The

196   detector temperature was 290°C and the data were collected with a rate of 20 Hz.

197   Quality assurance undertaken showed good reproducibility of the method and limits of

198   detection were typically in the low µg L^{-1} range (Table 2).

199

200   **Results and discussion**

201   **Water characterisation**

202   Samples of treated waters collected from drinking water treatment works across

203   England and Wales were analysed for pH, NPOC, UV, bromine and iodine. These

204   results are presented below along with calculated SUVA values (Table 1). The average

205   NPOC concentration was 1.6 mg L^{-1} with the highest value (3.7 mg L^{-1}) found in LR

206   and the lowest concentration (0.2 mg L^{-1}) in B1. The NPOC concentration of the

207   lowland rivers (mean of 1.7 mg L^{-1}) was similar to that measured in the upland

208   reservoirs (mean of 1.5 mg L^{-1}). SUVA values calculated here ranged from 1.5 m^{-1} L

209   mg^{-1} C (B1) to 5.4 m^{-1} L mg^{-1} C (UR3). L1 and UR3, with low NPOC values (1.2 and

210   1.1 mg L^{-1} respectively), had high SUVA values of 4.6 and 5.4 m^{-1} L mg^{-1} C

211   respectively, which indicate that the NOM was hydrophobic in character. No specific

212   trends were observed between the water treatment processes used and the treated water

213   SUVA values. The two waters with the highest SUVA (L1 and UR3) were treated with

214   direct filtration, not coagulation which is more effective towards removal of

215   hydrophobic material (Sharp et al., 2006).

216   The level of bromine, which we have assumed here to be mainly bromide, ranged from

217   14 to 310 µg L^{-1} (Table 1), with an average concentration of 105 µg L^{-1}. This is in

218   agreement with the concentrations of bromide in natural waters reported by Amy et al.

219   (1994) ranging from 30 to 200 µg L^{-1}, with an average of 100 µg L^{-1}. The highest

220   concentrations were found in B1, LR, BR2 and BR3 and it is expected here that these

221   waters with levels of bromide > 100 µg L^{-1} would form primarily brominated DBPs

222   (Singer et al., 2002).

223   The level of iodine found during this survey varied between 0.9 and 16.7 µg L^{-1} (Table

224   1) and is in line with the findings of Fuge et al. (1986) who reported total iodine in

225   water sources ranging between 0.5 and 20 µg L^{-1}. Interestingly the ratio of bromine to

226   iodine here varied considerably between 1 and 22%, which indicates no specific trend

227   between the level of bromine and iodine in the water sources.

228

229   **DBP levels from different water sources**

230   *HAAs*

231   The concentrations of nine HAAs from the 11 treated waters were quantified after

232   exposure to chlorine and monochloramine (Figure 1). In Figure 1, chlorine data are

233   represented as the treatment work reference only (e.g. B1) and the monochloramine data

234   are shown as NH_2Cl-work reference (e.g. NH_2Cl-B1). It is clear that using

235   monochloramine produced significantly less HAAs (average reduction of 77%) when

236   compared to chlorine. These findings compare well with previous studies that have

237   looked at HAA formation when using preformed monochloramine, typically a 90 to

238   95% reduction was observed (Cowman and Singer, 1996; Guay et al., 2005).

239   When chlorine was used as the disinfectant (Figure 1), considerable variation was

240   observed between the individual waters with HAA levels ranging from 5.0 to 69 µg L^{-1},

241   with an average value of 37 µg L^{-1}. This is the first HAAFP data set published that we

242   are aware of for England and Wales, although, Malliarou et al. (2005), earlier reported

243   finished waters from three regions in England and Wales water and found means of 35,

244   52 and 95 µg L^{-1}.

245   Across the chlorinated water samples, the major species formed were TCAA (ranging

246   from 1.0 to 40 µg L^{-1}) and DCAA (ranging from 2.5 to 22 µg L^{-1}). Sérodes et al. (2003)

247   also found TCAA and DCAA to be the major species formed in treated waters from

248   Quebec exposed to FP tests using chlorine. On a mass basis, DCAA and TCAA were

249   followed here by BDCAA, BCAA, MCAA and DBCAA. The brominated HAAs

250 MBAA, DBAA and TBAA were found at the lowest concentration and of these TBAA

251 was not always detected.

252 The ratio of TCAA:DCAA varied across the chlorinated samples, with TCAA being

253 predominant in six of the treated waters (B2, L2, UR1, UR2, UR3 and BR1), and

254 DCAA for the remaining waters (B1, L1, LR, BR2 and BR3). Similar variations were

255 also observed by Sérodes et al. (2003) and the excess chlorine used during FP tests as

256 well as the bromine concentration is believed to be the cause. When the bromine

257 concentration was ≤ 75 µg L^{-1}, TCAA was predominantly formed whilst when a high

258 concentration of bromine (> 100 µg L^{-1}) (water samples B1, LR, BR2 and BR3) and an

259 excess of chlorine were present, it is believed that bromide reacted to form

260 hypobromous acid (HOBr/OBr$^-$), which is known to react with NOM faster than

261 aqueous chlorine (Westerhoff et al., 2004). Consequently, the NaOCl to the NPOC

262 ratio (NaOCl:NPOC), on a mass basis, decreased as the bromine increased. Miller and

263 Uden (1983) amongst others found that at lower NaOCl:NPOC, the relative amount of

264 DCAA formed was higher than that of TCAA, which was observed here. For example

265 BR1, with a bromine concentration of 14 µg L^{-1}, formed 22 µg L^{-1} of DCAA and 40 µg

266 L^{-1} of TCAA, whereas LR, with a bromine concentration of 209 µg L^{-1} formed 16 µg L^{-1}

267 ^{1} of DCAA and 12 µg L^{-1} of TCAA.

268 When monochloramine was used as the disinfectant the highest concentration of HAAs

269 formed was 14 µg L^{-1} (L2, LR and BR1) and the average concentration 8.2 µg L^{-1}

270 (Figure 1). DXAAs, and in particular DCAA, were the predominant HAAs formed,

271 comprising at least 60% of the total HAA formation. This is expected as Karanfil et al.

272 (2008) and Cowman and Singer (1996) both reported DXAA to be the main HAA

273 species when using monochloramine and, in their studies, constituted 80 and 65%

12

274   respectively of the total HAA formed. Monohalogenated HAAs (MXAA) were always

275   the minor HAAs formed and did not contribute more than 20%.

276   The difference in HAA concentrations obtained with chlorine and monochloramine is

277   believed to be due to different formation routes. When using chlorine, it was concluded

278   that its reaction with NOM preferentially forms TCAA in low bromine-containing

279   waters. However, the formation mechanism with monochloramine is more complex and

280   different models have been proposed in the literature. Karanfil et al. (2007) and Hong et

281   al. (2007) both showed that the direct reaction between preformed monochloramine and

282   NOM is responsible for about 80% of HAA formation and that the remaining HAA

283   formation was attributed to the dissociation of monochloramine to chlorine. Duirk and

284   Valentine (2006) attributed the formation of DXAA to be mostly from the reaction

285   between NOM and chlorine in equilibrium with monochloramine. The presence of

286   bromide in the samples complicates the chemistry of the system because bromide reacts

287   with free chlorine and/or monochloramine to form $HOBr/OBr^-$, bromamines and

288   bromochloramine (Diehl et al., 2000). Here, the concentration of TXAA, and especially

289   TCAA remains high in many of the monochloraminated samples, such as B1, L2, LR,

290   BR2 and BR3, whilst in others, such as UR1 or UR2, the main species was DCAA,

291   making it unclear as to which mechanism is predominant.

292   ***Bromine incorporation***

293   To assess the extent of bromine substitution in HAA when using chlorine and

294   monochloramine, the bromine incorporation factor (BIF) was calculated (Symons et al.,

295   1993):

296    $$BIF = \frac{HAABr_9 (\mu mol\,L^{-1})}{HAA_9 (\mu mol\,L^{-1})},$$ Equation 1

13

297   where HAABr$_9$ is the sum of the molar concentrations of bromine incorporated in the

298   nine HAA species and HAA$_9$ represents the sum of molar concentrations of all nine

299   HAAs. The value BIF can range from zero to three. Calculated BIF values were plotted

300   against the bromine concentration (Figure 2) and it was found that the correlation

301   between BIF and bromine was better in water exposed to monochloramine ($R^2 = 0.72$)

302   than to chlorine ($R^2 = 0.39$).

303   Overall the results show that BIF increased with increasing bromine concentrations,

304   leading to more brominated HAAs. Also, BIF is higher in chlorinated waters than in

305   monochloraminated waters. Chlorine is a more powerful oxidant and its reaction with

306   bromine to form HOBr and then the formation of brominated HAAs will be faster and

307   more predominant than with monochloramine (Deborde and Von Gunten, 2008).

308   ***THMs and i-THMs***

309   As with the HAAs, shifting from chlorine to monochloramine produced significantly

310   less THMs and the average reduction was 92% (Figure 3).   While using chlorine there

311   was considerable variation in THM levels across the 11 waters with concentrations

312   ranging from 2.6 to 66 µg L^{-1}. The average concentration was 30 µg L^{-1}, which is

313   similar to the value observed for the HAAs (average of 37 µg L^{-1}). The lowest

314   concentration of THMs was found in L1 and the highest in LR, followed by L2. These

315   results are similar to those for the HAAs, and specifically, the concentration of TCM

316   was similar to that of TCAA in many samples, indicating possible common precursors.

317   For example, in B2, TCM was 13 µg L^{-1} and TCAA was 11 µg L^{-1}, in UR1, both TCM

318   and TCAA were at concentrations of 25 µg L^{-1} and in BR1, TCM was 35 µg L^{-1} and

319   TCAA was 40 µg L^{-1}. It was observed that L1 had a lower concentration of THMs than

320   L2 (2.6 and 47 µg L^{-1} respectively), both waters having the same NPOC values, but L1

321   having a greater SUVA value than L2, which indicates that neither NPOC, nor SUVA

14

322   were effective surrogates for these two treated waters.   In all the chlorinated waters

323   with bromine < 50 µg L^{-1}, TCM was found to be the major THM species, whereas in

324   those waters with bromine ≥ 75 µg L^{-1} brominated THMs became the major group.

325   When using monochloramine the concentrations of THMs were mostly below 1 µg L^{-1},

326   aside from B1, LR and BR2. Interestingly, BR2, which had the highest concentration of

327   bromine (310 µg L^{-1}) could form brominated THMs (13 µg L^{-1}) even when using

328   monochloramine as a disinfectant.

329   The concentrations of two i-THMs were also evaluated (Figure 4). The maximum

330   concentration found here was 0.73 µg L^{-1} and most concentrations were below the MRL

331   of 0.58 µg L^{-1}. Cancho et al. (2000) reported average levels lower than 1 µg L^{-1} for three

332   species (DCIM, BCIM and DBIM) in sand filters and ozonated waters, and Krasner et

333   al., (2006) reported a maximum of 19 µg L^{-1} for six i-THMs with DCIM and BCIM

334   being the prevalent species. Overall the concentration of i-THMs formed was low when

335   compared to THMs (Figure 4), with the ratio of the i-THMs to THMs being 1% on an

336   average basis and 0.4% on a median basis. Krasner et al. (2006) reported a median ratio

337   of 2% for six i-THMs and it known that chlorine can oxidise iodide through to iodate

338   (IO$_3^-$) and, hence, minimises any potential for i-THM formation (Bichsel and Von

339   Gunten, 1999). In the formation potential tests reported here chlorine is largely in

340   excess and hence we would expect the formation of IO$_3^-$ which is the likely reason for

341   the low level of i-THMs and the lack of any correlation between the i-THMs and the

342   iodine level in the water sources.

343   The formation of i-THMs is favoured by monochloramine because monochloramine,

344   unlike chlorine, is unable to oxidise hypoiodus acid (HOI) to IO$_3^-$ meaning that HOI has

345   a longer lifetime with monochloramine and can react with NOM to form i-THMs

346   (Bichsel and Von Gunten, 1999). Here, it was found that levels of i-THMs after

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347   monochloramine were between not detected to 0.89 µg L^{-1} (Figure 4), with five water

348   samples (B1, B2, L1, L2 and BR2) having greater concentrations of i-THMs than after

349   exposure to chlorine, whereas the contrary was observed in LR, UR1, UR2, UR3 and

350   BR3.

351   *HANs*

352   When using chlorine, HANs were detected in all waters and their concentrations were

353   typically an order of magnitude lower than the concentrations of THMs and HAAs

354   (Figure 5). Total HAN concentrations ranged between 0.023 and 5.5 µg L^{-1}, which is in

355   line with the findings of Krasner et al. (2007), who reported levels of dihalogenated

356   HANs between approximately 0.80 µg L^{-1} and 6.2 µg L^{-1} when using FP tests. DCAN

357   was the major HAN formed and contributed up to 56% of the total HAN, followed by

358   BCAN (27%), DBAN (16%) and TCAN (2%). Dihalogenated HANs are reported to be

359   more stable than the trihalogenated HANs by a number of authors (Peters et al., 1990;

360   Singer et al., 1995). In addition, TCAN can undergo base-catalysed hydrolysis at pH

361   higher than 5.5 (here, the pH was 7.2) which is likely to explain why it was rarely

362   detected in this study (Croué and Reckhow, 1989).

363   DCAN was the most abundant species found in chlorinated waters containing levels of

364   bromine < 50 µg L^{-1}. In the waters with bromine ≥ 75 µg L^{-1} the brominated HANs

365   (BCAN and DBAN) were dominant (67% total HAN). Peters et al. (1990) reported a

366   similar value with the brominated dihalogenated HANs accounting for 60% of the total

367   HAN in Dutch surface waters with bromide concentrations ≥ 500 µg L^{-1}. Here the

368   lowland waters L1, BR2 and BR3 produced more HANs which is expected as these

369   sources are more likely to contain dissolved organic nitrogen, the main precursor for

370   HANs (Oliver et al., 1983).   The speciation observed was again dependent on the

371   presence of bromine. For example BR2, which contains 310 µg L^{-1} of bromine, formed

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372   mainly BCAN and DBAN (0.31 and 0.39 µg L^{-1} respectively), whereas UR2 with a

373   bromine concentration of 18 µg L^{-1} formed 0.013 and 0.014 µg L^{-1} for both BCAN and

374   DBAN, but 0.26 µg L^{-1} of DCAN.

375   Changing from chlorine to monochloramine decreased the concentration of HANs by

376   81% (Figure 5). Hua and Reckhow (2007) also found that concentrations of HAN were

377   reduced by between 93% and 100% when using monochloramine and little

378   dihalogenated HANs (<1 µg L^{-1}) were formed.

379

380   *HKs, HAs and HNMs*

381   The concentrations of the two HKs formed following exposure to chlorine and

382   monochloramine are presented (Figure 6). HKs were detected in all the treated waters

383   exposed to chlorine (Figure 6), with concentrations ranging from 0.37 to 3.9 µg L^{-1},

384   with a mean value of 1.8 µg L^{-1}. The highest concentration was observed in BR1,

385   whereas the lowest concentration was observed in L1 and B1.   1,1,1-TCP was the

386   major HK formed in B2, L2, LR, UR1, UR2, UR3, BR1, BR2 and BR3. The greater

387   formation of 1,1,1-TCP in the samples is believed to be the result of the excess chlorine

388   used in FP tests, involving the oxidation of 1,1-DCP to 1,1,1-TCP (Gurol *et al.,* 1983).

389   The use of monochloramine resulted on average in a decrease of 70% in the total HK

390   compared to the of chlorine (Figure 6). No 1,1,1-TCP was detected which, given

391   that monochloramine does not provide enough free chlorine to push further substitution

392   into 1,1-DCP, was expected (Yang et al. (2007)

393   HAs were present in all samples after 24 hours contact time with chlorine (Figure 7).

394   The minimum value was 0.92 µg L^{-1} for L1 and the maximum value was 9.5 µg L^{-1} for

395   BR1. The average of HAs formed was 4.4 µg L^{-1} and this group of DBPs represented

396   the third major class of halogenated DBPs formed (on a weight basis) after HAAs and

397   THMs. The major HA detected was TCA (also called chloral hydrate) and Williams et

398   al. (1997) also found TCA to be the most prevalent DBP after HAAs and THMs.

399   Koudjonou et al. (2008) also reported TCA in drinking water made up 60% of the total

400   HA. Ozonation is known to increase the levels of DCA and TCA (Weinberg et al.,

401   1993) and here if we consider the two boreholes B1 and B2 they have different

402   concentrations of HKs, with B2, the pre-ozonated site, having a greater formation

403   potential for DCA (0.62 µg L^{-1}) and TCA (2.4 µg L^{-1}) than B1 (0.31 and 0.61 µg L^{-1}

404   respectively), which has no ozone.   The use of monochloramine resulted on average in

405   a 90% decrease in the total HA concentration (Figure 7).

406   The total concentration of HNMs measured after exposure to chlorine ranged from not

407   detected to 3.4 µg L^{-1} (Figure 8). The predominant HNM was TCNM and the

408   concentrations are in agreement with Krasner et al. (2001) who reported TCNM

409   concentrations of up to 2.0 µg L^{-1}. DBNM was detected here in B1, B2, L2, LR, BR2

410   and BR3, with the highest concentration found in BR2. Although other researchers have

411   shown that pre-ozonation can increase the formation of TCNM (Hoigné and Bader,

412   1988) or other HNMs (Plewa et al., 2004), it was not possible to see this trend here. The

413   highest concentration of HNMs was observed in BR1, a lowland river, followed by

414   UR1, which is an upland reservoir. On average the concentration of HNM was reduced

415   by 81% when using monochloramine and agrees with the recent findings of Hua and

416   Reckhow (2007) who showed that when using monochloramine only traces

417   concentrations of TCNM and those of Zhang et al. (2000) who reported a decrease of

418   58% with monochloramine in comparison to chlorine.

419

420

421 **Relative toxicity of DBPs measured**

422 Although, HAAs and THMs were more significant in regards to the mass concentration

423 than the semi-volatile DBPs, it should be noted that the toxicity of some of the semi-

424 volatile DBPs is much higher than the toxicity for HAAs and THMs. As shown in

425 Figure 9, Plewa et al. (2008) reported that the genotoxicity and the cytotoxicity were

426 much higher for the nitrogen-containing compounds HANs and HNMs than the THMs

427 and HAAs. Furthermore, the same authors reported that the iodo- and bromo-DBPs

428 were more cytotoxic and genotoxic than their chlorinated counterparts, and this is

429 because iodine and bromine are better leaving groups than chlorine due to their greater

430 polarisable bondings (Woo et al., 2002). Therefore, here, despite their lower

431 concentrations, some of the semi-volatile DBPs are more significant than HAAs and

432 THMs from a health standpoint.

433

434 **Relationships between HAAs, THMs and other DBPs**

435 Here the correlation between HAAs and THMs was investigated (Figure 10) and it was

436 found that for the waters evaluated that THMs were generally a good surrogate for

437 HAAs when chlorine was used (coefficient of correlation $R^2 = 0.82$). The slope of this

438 correlation was 1.21, which suggests that there is slightly more than one microgram of

439 HAA formed for one microgram of THM. No correlation could be found between

440 THM and HAAs when using monochloramine. Malliarou et al. (2005) also reported a

441 good relationship between THM and HAAs in final waters from two geographically

442 different regions in England and Wales ($R^2 = 0.82$ and 0.90), whereas they found a poor

443 correlation in the waters of their third region investigated and suggested that total THM

444 could not be assumed to be a good indicator for HAA levels. Moderate relationships

445   were also found between the total THM and the sum of the semi-volatile DBPs (HAN,

446   HA, HK, i-THM and HNM) measured after exposure to chlorine (Figure 10). The R^2

447   obtained for the collated semi-volatile DBPs was 0.68, which is in line with a previous

448   correlation (R^2 = 0.76) found between total THM and non-THM DBPs in drinking

449   waters (Krasner et al., 1989). This correlation suggests that the control of THM

450   precursors is closely linked to the control of other DBP precursors. As explained by

451   Krasner et al. (1989) this trend is valid for the sum of the measured halogenated DBPs

452   but it does not give similar trends for individual compounds; e.g. comparing total THMs

453   to HNMs yields an R^2 of only 0.08. In terms of regulation, it is interesting to note that

454   the regulatory limit of 100 µg L^{-1} for the THM_4 would fail a regulation of 80 µg L^{-1} for

455   the nine HAAs, currently under consideration by the European Union (Cortvriend,

456   2008). Indeed from the correlation found here, if 100 µg L^{-1} of THM_4 would be formed,

457   it would be expected to form 121 µg L^{-1} of HAA_9. In the specific waters investigated

458   here, to achieve a concentration of 80 µg L^{-1} for HAA_9, THM_4 should be no higher that

459   65 µg L^{-1}.

460

461   **Relationships between NPOC, UV and SUVA with DBPs**

462   Relationships between NPOC, UV and SUVA with HAAs, THMs and the semi-volatile

463   DBPs were investigated with chlorine FP test data. NPOC, UV and SUVA have been

464   used previously as surrogates for measuring DBPs as they are easier, cheaper and faster

465   to measure than DBPs (Goslan et al., 2002; Parsons et al., 2005; Ates et al., 2007).

466   Firstly, HAAs, THMs and semi-volatile DBPs have been correlated against NPOC and

467   correlation (R^2 values) between NPOC and HAAs, THMs and the semi-volatile DBPs

468   (collated together) were moderate (0.51, 0.63 and 0.56 respectively).   Although

469   stronger correlations have been reported, such as White et al. (2003), who found an R^2

20

470   of 0.86 and 0.87 for HAAs and THMs respectively to NPOC, it is likely that

471   correlations observed in a single sample are better than correlations observed from a

472   range of water sources. In terms of semi-volatile DBPs as separate species it was

473   observed that NPOC correlated well with HANs (R^2 = 0.82) and moderately with HAs

474   (R^2 = 0.52) (Table 3). However, no correlations were found between NPOC with i-

475   THMs, HNMs and HKs. No correlations were found between either SUVA or UV_{254}

476   and DBPs was also investigated (Table 3).

477

478   **Conclusions**

479   • The results have shown how all the waters have the potential to form significant

480   levels of all the DBPs monitored for and that in general a decrease in

481   concentration was been observed when shifting from chlorine to

482   monochloramine, the one exception being 1,1-DCP.

483   • In general the concentrations of THMs correlated well with HAAs, and in

484   particular the levels of TCM were similar to the levels of TCAA supporting the

485   hypothesis that they share similar precursor material.

486   • The impact of bromide on the formation of DBPs is well documented in the

487   literature and here the data reaffirmed that more bromide species are formed in

488   high bromide-containing waters. For HAAs, brominated species are

489   predominant when bromine was > 100 µg L^{-1}, whereas the brominated THMs

490   dominated when bromine was ≥ 75 µg L^{-1}.

491   • The use of FP test was found here to be unsuitable for the quantification of i-

492   THMs as the high chlorine levels are likely to limit formation.

493

501

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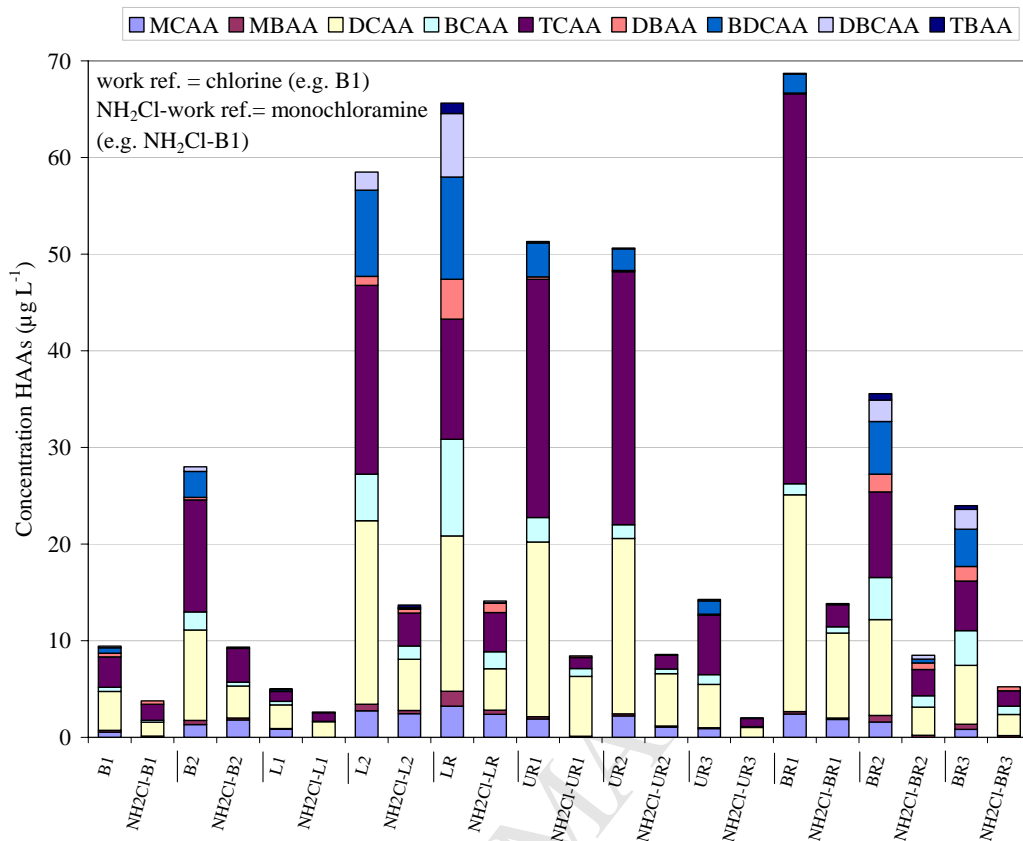663   District of Columbia, US, p. 299-314.

Figure 1. Distribution of HAAs after 24 hours bench scale exposure to chlorine and monochloramine for 11 treated waters
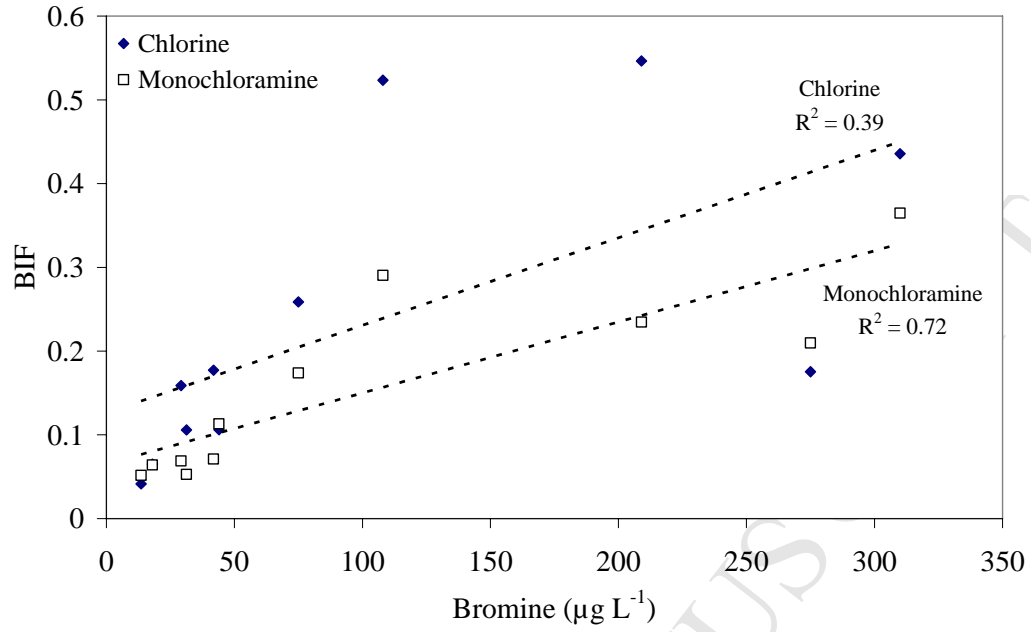
Figure 2. BIF in chlorinated and monochloraminated samples versus bromine concentration
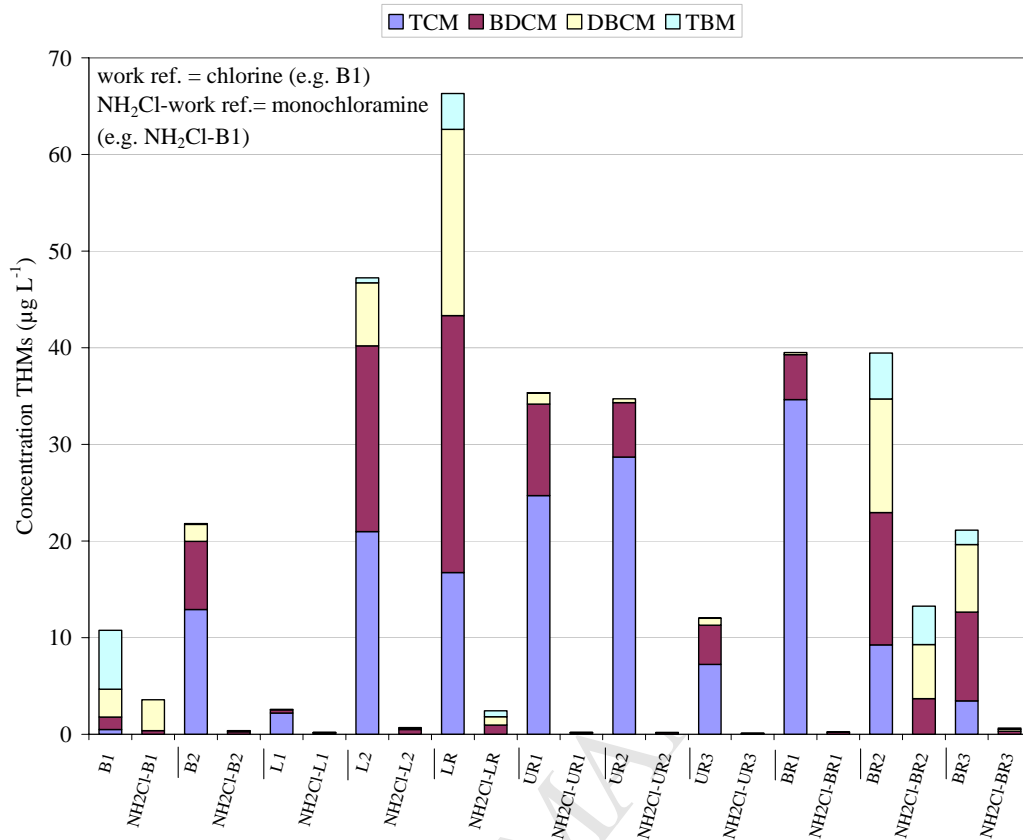
Figure 3. Distribution of THMs after 24 hours bench scale exposure to chlorine and monochloramine for 11 treated waters
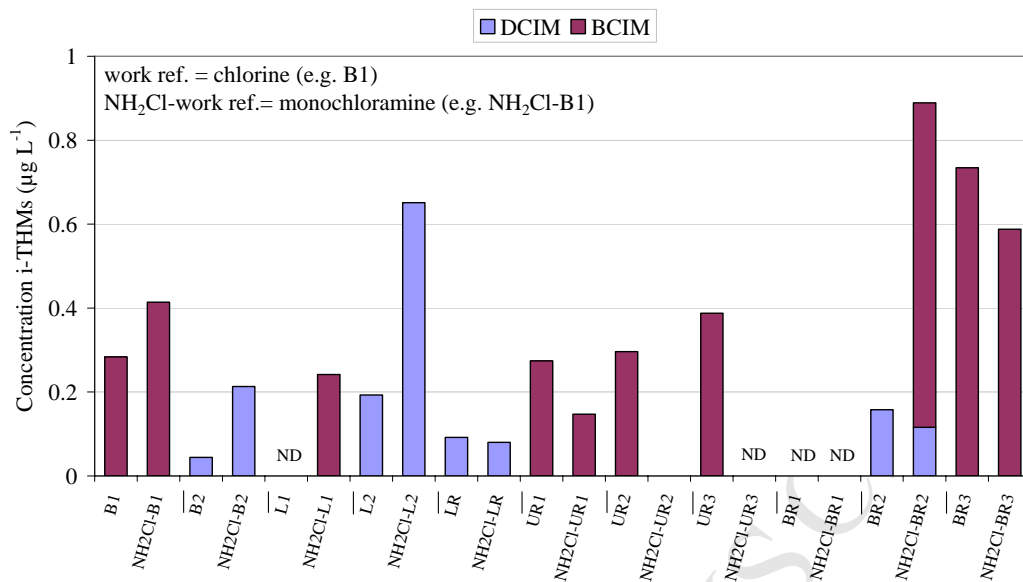
Figure 4. Distribution of i-THMs after 24 hours bench exposure to chlorine and monochloramine for 11 treated waters (ND – not detected)
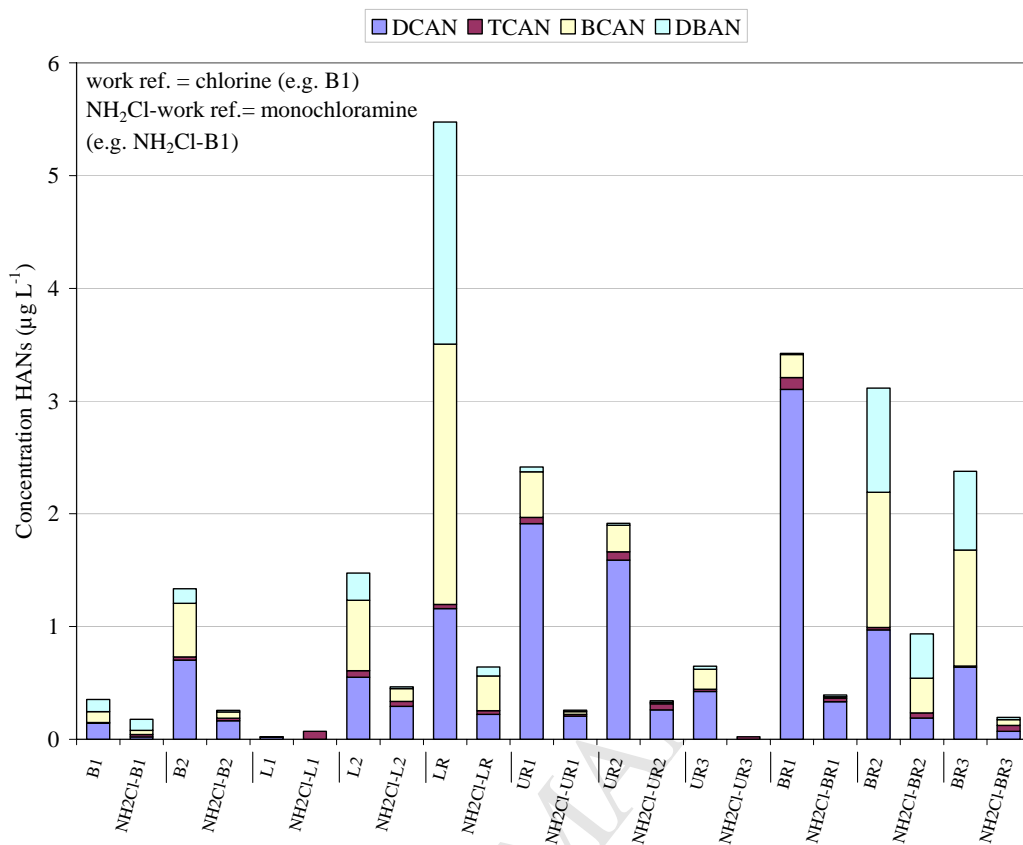
Figure 5. Distribution of HANs after 24 hours bench scale exposure to chlorine and monochloramine for 11 treated waters
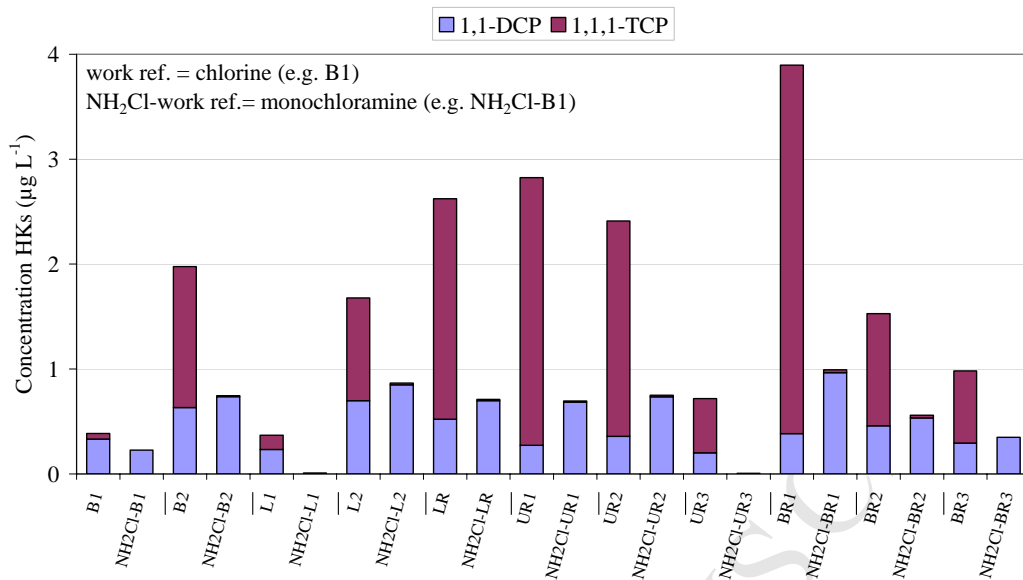
Figure 6. Distribution of HKs after 24 hours bench scale exposure to chlorine and monochloramine for 11 treated waters
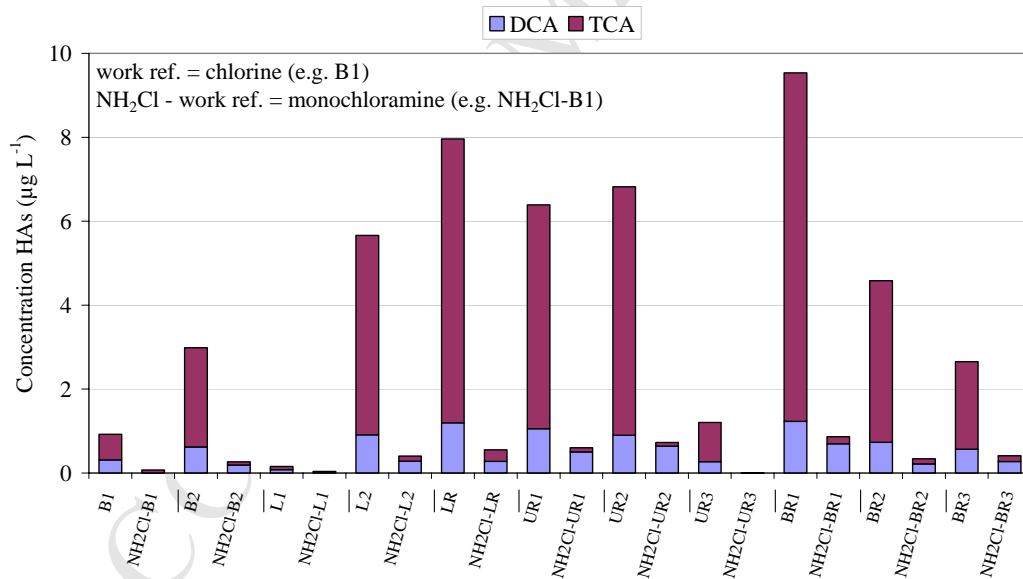

Figure 7. Distribution of HAs after 24 hours bench scale exposure to chlorine and monochloramine for 11 treated waters
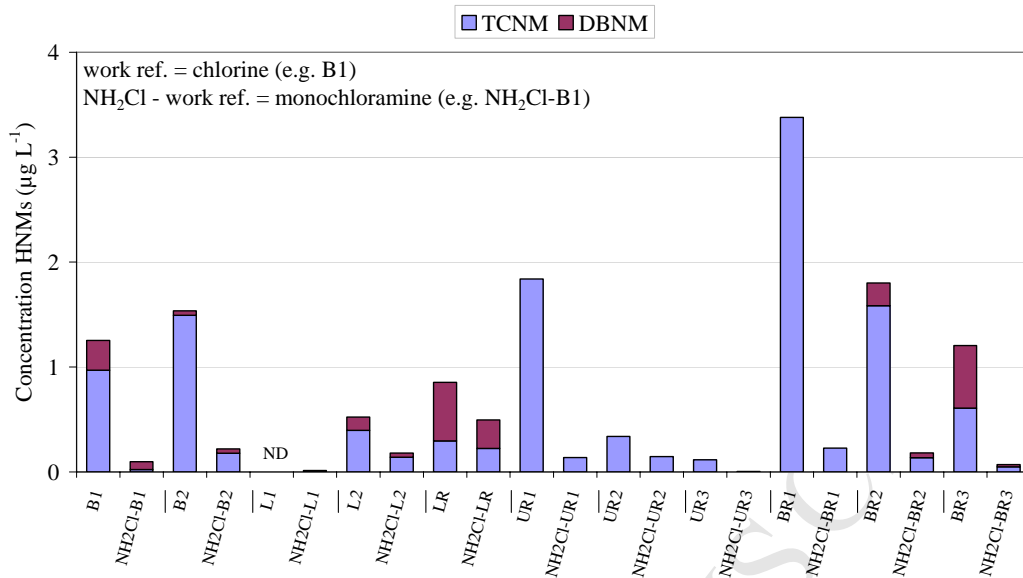
Figure 8. Distribution of HNMs after 24 hours bench scale exposure to chlorine and monochloramine for 11 treated waters (ND – not detected)
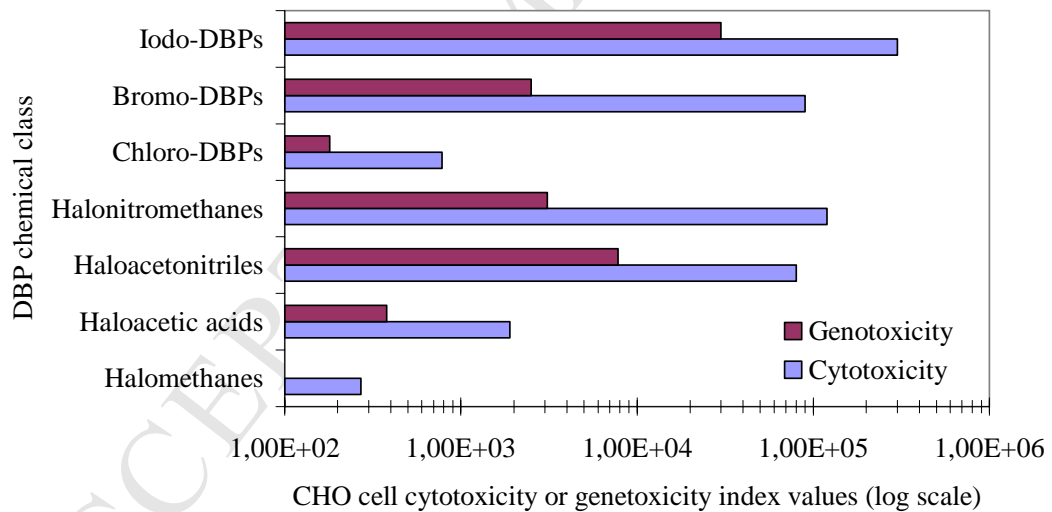

Figure 9. Cytotoxicity and genetoxicity indices for different classes of DBPs and for chloro-, bromo- and iodo-DBPs (Adapted from Plewa et al., 2008)
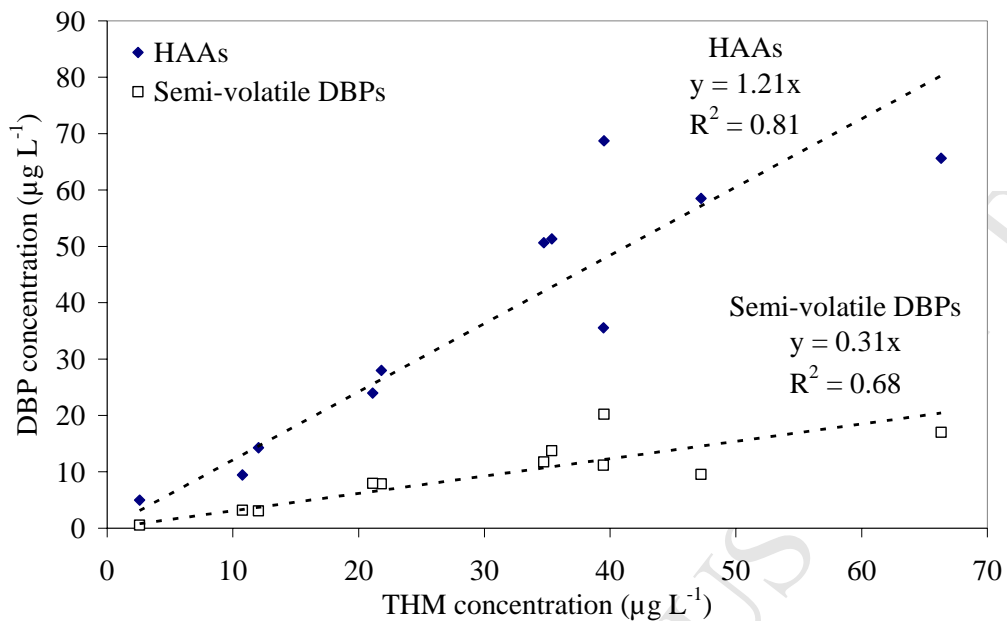
Figure 10. Correlation between THMs with HAAs and the semi-volatile DBPs (chlorine FP tests)

Table 1. List of water treatment works, sources and water characteristics

Work ref.	Work description	pH	NPOC (mg L^{-1})	UV$_{254}$ (m^{-1})	SUVA$_{254}$ (m^{-1}. L mg^{-1} C)	Bromine (µg L^{-1})	Iodine (µg L^{-1})
	BOREHOLE (B)						
B1	*Sampling point:* Post filter *Main process:* Filtration	7.8	0.2	0.4	1.5	275	3.5
B2	*Sampling point:* Post membrane prior to superchlorination *Main process:* Membrane filtration with pre-oxidation	7.2	1.2	2.2	1.8	42	6.9
	LAKE (L)						
L1	*Sampling point:* Post membrane *Main process:* Membrane filtration	5.9	1.2	5.5	4.6	31	1.3
L2	*Sampling point:* Post filter *Main process:* Coagulation/Direct filtration	6.8	1.2	3.4	2.7	75	16.7
	LOWLAND RESERVOIR (LR)						
LR	*Sampling point:* Post GAC *Main process:* Ozone/coagulation /GAC	7.8	3.7	5.8	1.6	209	8.9
	UPLAND RESERVOIR (UR)						
UR1	*Sampling point:* Post sand filtration *Main process:* Coagulation	7.4	1.6	4.2	2.6	44	0.9
UR2	*Sampling point:* Post filter *Main process:* Coagulation/filtration	8.9	1.7	4.1	2.4	18	0.9
UR3	*Sampling point:* Post slow sand filter *Main process:* Direct filtration	6.2	1.1	5.9	5.4	29	0.9
	LOWLAND RIVER (BR)						
BR1	*Sampling point:* Post GAC *Main process:* Coagulation/GAC	5.5	2.2	5.3	2.4	14	0.9
BR2	*Sampling point:* Post GAC *Main process:* Coagulation/GAC	7.5	1.6	2.9	1.8	310	6.3
BR3	*Sampling point:* Post GAC *Main process:* Coagulation/GAC	7.2	1.4	2.4	1.7	108	3.0

ref. – reference

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Table 2. Method performance

Compound	Average tr[a] (min)	RSD[b] (%)	Detection Limit[c] (µg/L)	MRL[d] (µg/L)
TCM	7.72	0.058	0.088	0.264
DCA	7.95	0.052	0.124	0.371
TCAN	9.99	0.042	0.020	0.061
DCAN	11.17	0.060	0.019	0.057
BDCM	11.24	0.063	0.036	0.108
TCA	12.02	0.035	0.029	0.086
1,1-DCP	13.12	0.030	0.029	0.086
TCNM	18.53	0.059	0.039	0.117
BDCM	19.10	0.045	0.049	0.148
BCAN	20.01	0.042	0.023	0.070
DCIM	22.64	0.067	0.086	0.257
1,1,1-TCP	25.28	0.013	0.089	0.268
TBM	27.10	0.037	0.095	0.284
DBAN	27.71	0.009	0.014	0.041
BCIM	28.28	0.015	0.108	0.324
DBNM	28.81	0.012	0.059	0.178

[a] The average retention time corresponds to the average of seven injections; [b] Corresponds to the relative standard deviation and must be less than 15% according to US EPA Method 551.1 (1995a); [c] Fortified waters were extracted and analysed over 3 days for seven replicates; [d] Corresponds to the minimum reporting level and is the threshold expected for accurate quantification in an unknown sample. It has to be at least three times the limit of detection.

Table 3. Correlation between DBPs and water characteristics

DBPs (µg L^{-1})	Coefficient of correlation (R^2)			
	NPOC (mg L^{-1})	UV (m^{-1})		SUVA (m^{-1}. L mg^{-1} C)
		All data	Coagulated waters	
HAAs	0.51	0.11	0.78	0.15
THMs	0.63	0.06	0.49	0.23
i-THMs	0.07	0.09	0.50	0.003
HANs	0.82	0.09	0.45	0.27
HKs	0.42	0.11	0.72	0.12
HAs	0.52	0.11	0.86	0.16
HNMs	0.03	0.03	0.06	0.25

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