

3 Halogen-based disinfectants

3.1 Introduction

There are a number of chlorine-based, and to a lesser extent bromine-based, disinfectants available for use both in municipal water treatment and private or municipal swimming pools. Chlorine gas itself is a very effective disinfectant but imposes a significant chemical hazard. It is a more concentrated and purer source of chlorine than hypochlorite salts since the latter contain a lower proportion of active chlorine as well as higher levels of suspended solids and other impurities (such as bromide in hypochlorite). In municipal swimming pools chlorine-based disinfectants are normally used in the main pool, but bromine-based ones may be used in spas and whirlpools due to the lower volatility and odour of bromine and its by-products, as previously mentioned. Both chlorine and bromine-based disinfectants are relatively cheap to use and can be handled safely by plant/pool operatives. Chlorine can be added to a pool either as a hypochlorite salt, stabilised isocyanurate, or pressurised liquid, or else can be generated electrochemically in situ from a brine solution.

3.2 Basic chlorine water chemistry

Chlorine (Cl_2) dissolves in water to form hypochlorous acid (HOCl) (Equation 4) an almost instantaneous reaction (Shilov and Soludushenkov, 1936). HOCl dissociates to form hypochlorite (OCl^-) (Equation 5) at pH values above 6:



Hence:

$$K_A = [\text{H}^+][\text{OCl}^-]/[\text{HOCl}] \sim 7.5 \quad \text{Equation 6}$$

or $\log [\text{OCl}^-] - \log [\text{HOCl}] = \text{pH} - \text{p}K_A$. The quantity of Cl_2 , HOCl and OCl^- present is known as the '*free available chlorine*'. The distribution between HOCl and OCl^- being a function of pH; at pH values less than $\text{p}K_A$, HOCl predominates. The $\text{p}K_A$ ranges from 7.47 to 7.83 over a temperature range of 33°C to 0°C (Morris, 1986).

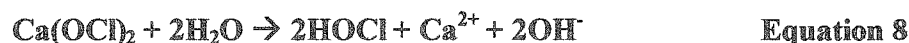
Since HOCl is more powerful an oxidant and biocide than OCl⁻, operation at a lower pH increases biocidal efficiency. However, this decreases at pH values lower than 6 as significant degradation of HOCl to molecular oxygen (O₂) takes place:



3.3 Chlorine based disinfectants

3.3.1 Na and Ca hypochlorite

Hypochlorous acid can be stabilised by either a calcium (Ca) or sodium (Na) salt (Ca(OCl)₂ and NaOCl). When mixed with water, these salts dissolve to liberate hypochlorous acid at low-neutral pH values.



Both sodium and calcium hypochlorite are routinely used in pool water treatment since they are relatively easy to use, imposing a much reduced hazard compared with liquefied chlorine.

3.3.2 Stabilised chlorine disinfectants

Chlorine disinfectants can be stabilised further to form chlorinated isocyanurates. These are organic compounds that decompose in water to produce cyanuric acid (H₃Cy) and chlorine hydrolysis products (Equation 9). The two types of isocyanurates that are used commonly in pools are:

- 1) sodium dichloroisocyanurate or 'dichlor' (C₃N₃O₃NaCl₂) and
- 2) trichloroisocyanuric acid 'trichlor' (C₃N₃O₃Cl₃)



Where x=1 for monchlor, x=2 for dichlor and x=3 for trichlor

Dichlor delivers two molecules of hypochlorous acid and the isocyanuric acid acts to stabilise the free hypochlorous acid released (Equation 5). Such stabilised forms of hypochlorous acid may degrade four times slower in direct sunlight than non-stabilised forms and therefore find use in outdoor pools in hot climates.

3.3.3 On Site Chlorine Generation

Sodium hypochlorite can be generated by electrolysis of high concentrations of sodium chloride. This liberates hypochlorite at the anode and hydroxide ions at the cathode with net production of hypochlorous acid according to Equation 4. The H₂ gas generated is potentially explosive and must be carefully managed. Also, the solution in the region of the electrodes is highly acidic (at the anode) and basic (at the cathode).

3.3.4 Chlorine dioxide

Chlorine dioxide (ClO₂) is an unstable gas and therefore is produced on site, normally by mixing a solution of sodium chloride with a strong chlorine solution:



ClO₂ possesses an oxidising power 25 times greater than that of chlorine. It does not react with humic acid to produce THMs and does not react directly with ammonia to produce chloramines. However, ClO₂ is not very stable, degrading to chlorate and chlorite:



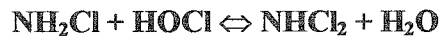
This very fast decomposition rate is catalysed by carbonate and by chloride yielding dissolved chlorine as an intermediate. The health impacts of chlorate and chlorite mean that chlorine dioxide is not an appropriate disinfectant for pools.

3.4 *Reaction of chlorine with ammonia*

3.4.1 Chloramine formation and breakpoint chlorination

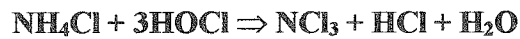
Ammonia reacts with chlorine (as hypochlorite) to form monochloramine and dichloramine:





Equation 13

Nitrogen trichloride (NCl_3) will also form in solutions with excess chlorine, and at low pH, though only arises at low levels (Chapin, 1931; Saguinsin 1975; Dokter, 1985).



Equation 14

The total concentration of monochloramine (NH_2Cl), dichloramine (NHCl_2) and nitrogen trichloride (trichloramine) (NCl_3) is known as the 'combined chlorine' level. Whilst chloramine formation takes place at low Cl:N ratios, at higher ratios further oxidation takes place to yield a classic 'N' shaped plot when the total chlorine residual is plotted against the chlorine dose (Figure 3.1). At a certain point there is a sudden decrease in the ammoniacal nitrogen concentration at the same time as the decrease in the residual chlorine curve. This is due to the decomposition of chloramines in excess chlorine (i.e. where all of the free ammonia has reacted with Cl) where nitrogen products of N_2O , NO_2^- , N_2 and NO_3^- are formed (Table 3.1), with much of the chlorine forming chloride. After the so-called breakpoint, when all the combined ammoniacal-N has been oxidised, all of the chlorine added forms a free chlorine residual.

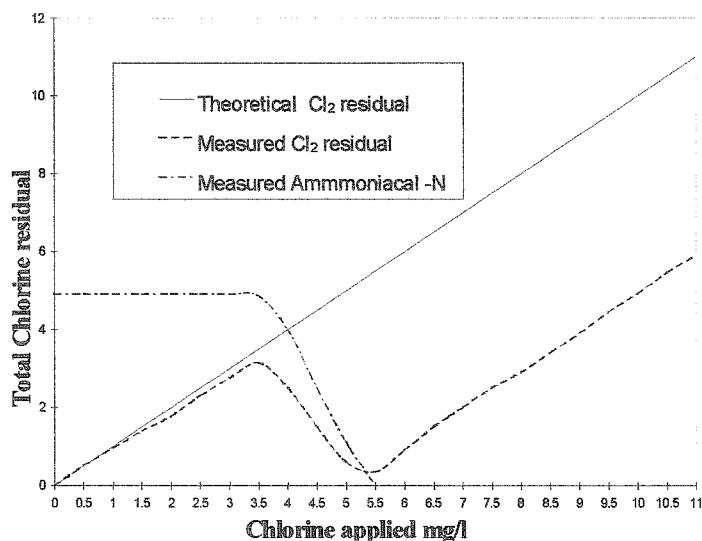


Figure 3.1 Breakpoint curve after 24 hours, pH 7 (from Griffin & Chamberlin (1941))

Table 3.1 Ratio of Cl:N needed for breakpointing from various reactions

Molar Cl:N ratio	Weight C:N ratio	Reaction	Products
3:2	3.8:1	$2\text{NH}_3 + 3\text{HOCl}$	$\text{N}_2 + 3\text{H}^+ + 3\text{Cl}^- + 3\text{H}_2\text{O}$
7:4	4.4:1	$4\text{NH}_2\text{Cl} + 3\text{Cl}_2 + \text{H}_2\text{O}$	$\text{N}_2 + \text{N}_2\text{O} + 10\text{H}^+ + 10\text{Cl}^-$
4:1	10.2:1	$\text{NH}_4^+ + 4\text{HOCl}$	$\text{NO}_3^- + 6\text{H}^+ + 4\text{Cl}^- + \text{H}_2\text{O}$

In the 1930's and 40's much research was carried out to establish the impact of water quality on the Cl/N ratio (Table 3.2). Data indicate that of the whole range of reaction stoichiometries indicated in Table 3.2 that are possible for achieving breakpoint, the ratios determined experimentally are generally much higher and only yield nitrate as the minor product.

Table 3.2 Experimentally-derived Cl/N dose needed to achieve breakpoint

Reference	Starting NH_3 -N concentration	Cl/N ratio (by weight) initially present to achieve breakpoint
Stoichiometrically calculated	-	7.6 : 1
Chapin (1931)	>70mg/l	8.6 : 1 to 7.6 : 1
Weber <i>et al</i> (1940)	15mg/l	8.5 : 1
Norman (1936)		8.2 : 1
Harvill <i>et al</i> (1942)	1mg/l	7.6 : 1
Moore <i>et al</i> (1943)	0-5 mg/l	9 : 1
Calvert (1940)	-	7.5 : 1
Griffin & Chamberlin (1941)	0.5mg/l	10 : 1

Work by Weil and Morris (1949) revealed the rate of chloramine formation to be determined by the concentration of HOCl and NH_3 , these species having pK values of ~7.4 and 9.2 respectively, such that the maximum rate occurred at a pH of around 8.3 $[(\text{pK}_a \text{ NH}_3 + \text{pK}_a \text{ HOCl})/2]$. Both Weil and Morris (1949) and Dennie *et al* (1978) noted the rate of reaction to increase by a factor of 5~15 (depending on the pH), on increasing the temperature from 6 to 37 °C. The proportion of NCl_3 over the mono- and dichloramine levels also increased with temperature. The effect of pH and Cl:N ratio on chloramine speciation at equilibrium has been presented by McKee (1960)

and can be seen in Figure 3.2. It can be seen that at a higher pH, there is a more marked effect on chloramine speciation at a ratio of >1 . This is because at a higher pH the $\text{HOCl}:\text{OCl}^-$ ratio is lower which reduces the rate of chloramine formation attributed to Equation 12.

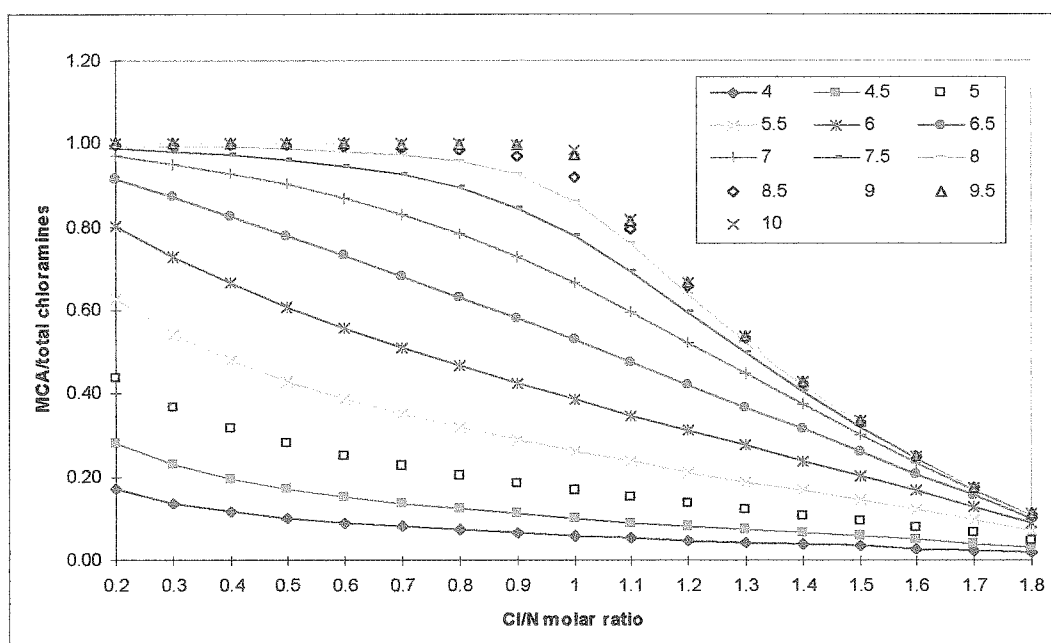


Figure 3.2 DCA/MCA ratio against Cl/N ratio for pH range of 4 to 10 (calculated from McKee, 1960)

A complete kinetic analysis performed by Jafvert and Valentine (1992) resulted in the generation of a model able to predict breakpoint kinetics at chlorine to nitrogen ratios greater than 1.6, as well as the slow oxidation of ammonia to chloramines at $\text{Cl}/\text{N} < 1$ and the transition region at Cl/N 1-1.6 where rapid initial decay results in chloramine species radicals (Table 3.3). According to these authors, unlike the rapid redox reactions that occur under breakpoint conditions, the oxidation of ammonia by combined chlorine is very slow with oxidant half lives generally in the order of days.

Table 3.3 Summary of rate constants for the chlorination of ammonia (adapted from Jafvert & Valentine, 1992)

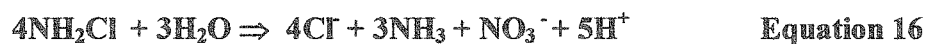
Type of reaction	Reaction stoichiometry	Rate constant at 25°C
Substitution reactions involving HOCl	$\text{HOCl} + \text{NH}_3 \rightarrow \text{NH}_2\text{Cl} + \text{H}_2\text{O}$	$1.5 * 10^{10} \text{ M}^{-1} \text{ h}^{-1}$
	$\text{HOCl} + \text{NH}_2\text{Cl} \rightarrow \text{NHCl}_2 + \text{H}_2\text{O}$	$1 * 10^6 \text{ M}^{-1} \text{ h}^{-1}$
Disproportionation reactions	$\text{NH}_2\text{Cl} + \text{NH}_2\text{Cl} \rightarrow \text{NHCl}_2 + \text{NH}_3$	$k_{\text{H}}[\text{H}^+] + k_{\text{H2P}}[\text{H}_2\text{PO}_4^-] + k_{\text{H3P}}[\text{H}_3\text{PO}_4]$
	$\text{NHCl}_2 + \text{NH}_3 \rightarrow \text{NH}_2\text{Cl} + \text{NH}_2\text{Cl}$	$2.16 * 10^8 \text{ M}^{-2} \text{ h}^{-1}$
NHCl ₂ decomposition in excess ammonia	$\text{NHCl}_2 + \text{H}_2\text{O} \rightarrow \text{I}^1$	$4 * 10^5 \text{ M}^{-1} \text{ h}^{-1}$
	$\text{I} + \text{NHCl}_2 \rightarrow \text{HOCl} + \text{products}^2$	$1 * 10^8 \text{ M}^{-1} \text{ h}^{-1}$
	$\text{I} + \text{NH}_2\text{Cl} \rightarrow \text{products}$	$3 * 10^7 \text{ M}^{-1} \text{ h}^{-1}$
	$\text{NH}_2\text{Cl} + \text{NHCl}_2 \rightarrow \text{products}$	$55.0 \text{ M}^{-1} \text{ h}^{-1}$
NHCl ₂ decomposition in HOCl and OCl ⁻	$\text{HOCl} + \text{NHCl}_2 \rightarrow \text{NCl}_3 + \text{H}_2\text{O}$	$K_{\text{HP}} [\text{HPO}_4^{2-}] + k_{\text{OCl}} [\text{OCl}^-] + k_{\text{OH}} [\text{OH}^-]$
	$\text{NHCl}_2 + \text{NCl}_3 + 2\text{H}_2\text{O} \rightarrow 2\text{HOCl} + \text{products}$	$2 * 10^{14} \text{ M}^{-2} \text{ h}^{-1}$
	$\text{NH}_2\text{Cl} + \text{NCl}_3 + \text{H}_2\text{O} \rightarrow \text{HOCl} + \text{products}$	$5 * 10^{12} \text{ M}^{-2} \text{ h}^{-1}$
	$\text{NHCl}_2 + 2\text{HOCl} + \text{H}_2\text{O} \rightarrow \text{NO}_3^- + 5\text{H}^+ + 4\text{Cl}^-$	$8.3 * 10^5 \text{ M}^{-1} \text{ h}^{-1}$

¹I = Intermediate

²Products = may include N₂, H₂O, Cl⁻, H⁺, NO₃⁻ and unidentified reaction products

3.4.2 Chloramine decomposition

The redox reaction involved in the decomposition of monochloramines in excess chlorine can take hours to days with the net effect being to increase the free ammonia concentration (Ozekin, 1996; Vikesland *et al*, 1998). During decomposition monochloramine decomposes to NH₃ (47% ± 3.4) and N₂ (43% ± 7.9), with smaller amounts of NO₃⁻ (1.2% ± 0.66) and other unidentified products:



Natural organic matter (NOM) has been found to increase the rate of monochloramine decay (Vikesland *et al* 1998). When NH₂Cl decomposes in the presence of NOM, the product speciation changes so that less molecular nitrogen gas and higher concentrations of NO₃⁻ and NH₃ are produced. Vikesland *et al* (1998) reported that the promotion of the NO₃⁻ decomposition route decreases the amount of N₂ by

between 50 and 80 %. This is thought to be due to the catalysis by NOM of the second order reaction:



Dichloramine decomposition experiments conducted by Jafvert and Valentine (1987) led to the postulation of equations of the form:



where 'I' is a short-lived intermediate, presumed to contain N, and 'Products' are stable end products such as H^+ and Cl^- depending on the structure of 'I' (shown in Table 3.3). The rate of this reaction, k_1 , is slow and is thought to be OH^- catalysed. Although 'I' is important in the decomposition of dichloramine, it must be at a low concentration as there is no experimental evidence found for its existence. The intermediate reacts further with monochloramine:



or



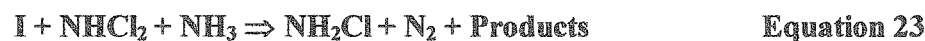
where Equations 19 and 20 are relatively fast. Equations 18 and 19 can be combined to produce:



and Equations 19 and 20 can be also combined:



Equation 19 can be further modified to show the production of monochloramine from dichloramine in excess ammonia:



Many other reactions involving dichloramine are thought to occur under these conditions. However, most of them are considered to be insignificant as they are much slower; the reaction of HOCl with NHCl_2 , for example, is 700 times slower than HOCl with NH_3 at pH 9.3.

3.4.3 Unidentified disinfection by-products

During breakpoint chlorination the formation of 'intermediates' or 'unidentified products' (UPs) is proposed to explain discrepancies evident both in the kinetic data and mass balance calculations already identified. The existence of an unidentified product was first identified by Leao (1981), whilst trying to determine combined chlorine speciation. This UP was also detected by Hand and Margerum (1983) in a study of dichloramine in the presence of ammonia. Evidence of its existence comes from spectrophotometric data which shows the presence of an unaccounted for compound with a maximum absorbance of 243 nm.

The concentration of this UP was determined by Leung and Valentine (1994) from the difference between the measured and predicted chloramine concentrations, and its formation was found to be related to the level of nitrate. It was postulated that nitrate formation involved a series of reactions including the formation of the unidentified product, or parallel reactions with some common intermediate. Although nitrite is thought to be involved in the formation of nitrate, it was not present in the chloramine decomposition experiments. This suggests that nitrite must react rapidly with free chlorine and can therefore be viewed as being pseudo steady state. If this is true, then the nitrate production would be equal to the nitrite production.

In experiments by Leung and Valentine (1994), where nitrite was added to the chloramine decay experiment, the concentration of the UP increased by up to 200%, although the UP formation rate was not linearly related to nitrate addition. During photolysis the UP disappeared and the concentration of chloride, nitrate and nitrite increased. Through measurement of the concentrations of these products it was surmised that the UP contained a 1:1 ratio of chlorine: nitrogen, although as 100% recovery was not possible the exact quantity could not be calculated. Excess ammonia decreased the UP formation slightly, possibly due to the extra ammonia shifting the chemical equilibrium, lowering the HOCl in the reacting solution. Jafvert and Valentine (1987), however, have shown that excess ammonia does not affect the rate of dichloramine decomposition (and therefore the rate of N production). It was

postulated by these authors that the production of a UP is not directly related to the ammonia concentration. Mass balances calculated for these experiments suggested that the UP might have accounted for more than 6.7 % of the total initial chlorine.

3.4.4 Chloramine precursors in pool water

Early research by Lomas (1965) showed the chloramine level in pools to rise after breakpointing, causing the free chlorine level to drop. When chlorinating samples of urine, dichloramine levels and formation trends identical to those found in pool water were observed, suggesting urine to be a major source of pollution in pool water: adding more chlorine to the sample after breakpointing increased the level of dichloramine. Further lab-based experiments by Lomas (1967) showed that it was the presence of various nitrogenous materials in pool water that were resistant to oxidation by chlorine which were forming the apparent excess of dichloramine following breakpointing. Lomas postulated that this was probably due to the formation of a chlorine substitution product by the oxidation resistant materials which simulated dichloramine in the standard FAS/DPD wet chemical analysis (see below).

Chlorination of urea and hippuric acid beyond the breakpoint did not result in the residual dichloramine reading after breakpointing. Chlorination of creatinine, on the other hand, showed a very stable level of apparent dichloramine in the presence of excess chlorine. As this product, named chlorocreatinine, was stable it was postulated (by Lomas) that it could build up in swimming pool water, giving false positive readings for dichloramine.

By comparing the DPD/FAS method for determining chloramines with Membrane Induction Mass Spectrometry (MIMS) it has been shown possible to identify the level of 'false' dichloramine measured by the DPD method (Shang *et al*, 2000). The DPD method results in much higher levels of chloramines than the MIMS method (which is regarded as being more accurate). DPD reacts with free chlorine to produce a red colour that can either be measured spectrophotometrically or titrated. If iodide is added, then chloramines and chloramine simulants react with the iodide to produce

iodine which in turn oxidizes more DPD to give a red colour. The MIMs method is considered to be more accurate as it does not rely on the reaction of chloramine with a reagent, but instead measures the species directly. The apparent chloramine recorded by Shang *et al* (2000) in the DPD test was postulated to be organic compounds such as monochloroglycine or dichloroglycine.

Urine contains around 70 different compounds exceeding a concentration of 10mg/l (Putnam/NASA, 1971), and around 20 exceeding 40 mg/l (Table 3.4). The total concentration of solutes in urine is 36,700 – 46,700 mg/l of which urea accounts for 25–50%. The large variation in concentrations of some constituents, such as creatinine, arises due to differences in diet, age and gender.

Table 3.4 Main constituents of human urine over 40 mg/l (Putnam/NASA, 1971)

Constituent	Range, mg/l	Constituent	Range, mg/l
Urea	9300 – 23,300	Uric acid	40 – 670
Chloride	1870 – 8400	Uropepsin	70 – 560
Sodium	1170 – 4390	Bicarbonate	20 – 560
Potassium	750 – 2610	Creatine	0 – 530
Creatinine	670 – 2150	Organic Sulphur	77 – 470
Inorganic Sulphur	103 – 1800	Glycine	90 – 450
Hippuric acid	50 – 1670	Phenols	130 – 420
Phosphorus	470 – 1070	Lactic acid	30 – 400
Citric acid	90 – 930	Calcium	30 – 390
Glucuronic acid	70 – 880	Histidine	40 – 330
Ammonia	200 – 730		

3.5 THM formation

3.5.1 THM precursors

THM formation from NOM is thought to occur through two main pathways: the haloform reaction and a second mechanism involving the reaction of HOCl with the aromatic ring in certain aromatic compounds. The formation of THMs through the haloform reaction is complex and involves the reaction of methyl ketones (RCOCH_3) and HOCl:



Equation 24

The reaction proceeds through alternate hydrolysis and halogenation steps (Figure 3.3). The first step, reaction with hydroxide, is the rate-determining step. The chromophores in humic acid (HA), which forms a significant proportion of NOM, are thought to be conjugated double bonds that react readily with HOCl to ultimately form THMs (Rook, 1976; Ram *et al*, 1986; Trussel and Umphres, 1978). At a pH range of 5 to 9, and at the NOM concentrations arising in natural water, the haloform reaction has been calculated to be too slow to account for the levels of THMs found in water treatment plants (WTPs) and it can thus be concluded that THMs are formed by a combination of routes (Sykes, 1990).

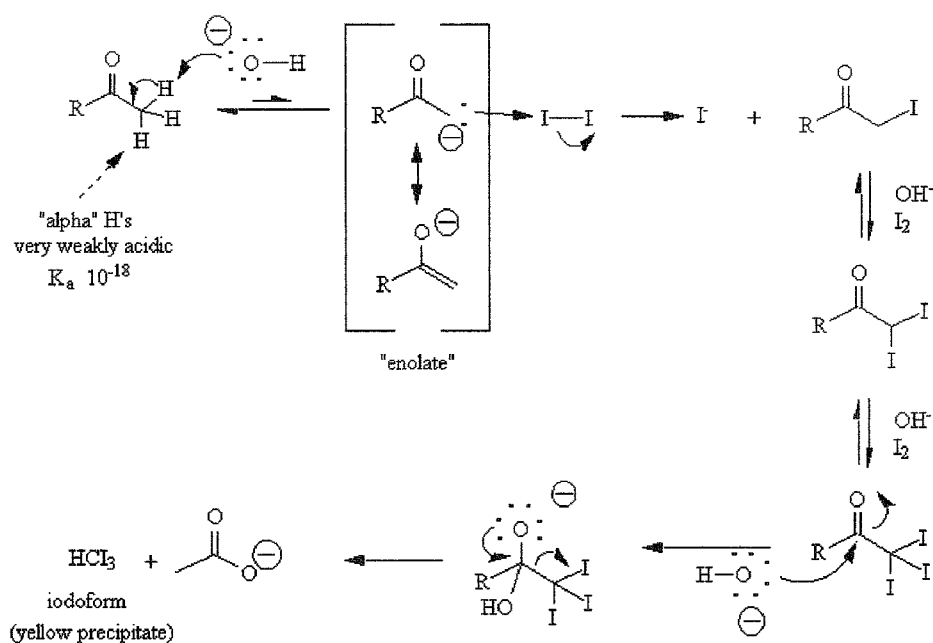


Figure 3.3 The haloform reaction resulting in the formation of iodoform (a test for the reaction)

Adin *et al* (1991) reported patterns of THM production from HA chlorination that are not in accordance with those proposed by the haloform reaction, where THM

formation is dependent on chlorine concentration. From previous studies (Rook, 1977; Babcock and Singer, 1979) it has been reported that 85% to 95% of THMs are produced within 24 hours of initial chlorination, and that peak THM formation occurs just before chlorine is depleted. From this point on, the THM concentration significantly decreases in the presence of excess HA.

The application of varying levels of chlorine in experiments by Adin *et al* (1991) generated similarly shaped THM production curves. However, the time at which the 'peak' of each curve occurred was shifted depended on the HA concentration. This suggested that there are two pathways for THM production: a direct route, and one via organochlorine intermediates. As the conversion of these intermediates to THMs is of a higher order with respect to chlorine, they accumulate at low levels of chlorine in excess HA, resulting in a decrease in THM levels. A lag in THM formation was also noted at the start of low-chlorine level experiments, the time lag increasing in length with decreasing chlorine concentration. This again infers that an insufficient chlorine concentration to react with excess HA gives rise to organochlorine intermediates rather than THMs.

The effect of decreasing pH was to shift the THM peak towards high HA concentrations, probably due to the higher percentage of chlorine as HOCl at a lower pH. Increasing pH resulted in a larger THM peak, reflecting THM yield. The peak size is mainly dependent on the last step of the THM pathway, which is base catalysed (like the haloform reaction), thus increasing pH would be expected to increase THM yield. Chlorination experiments performed with 3 HA model compounds (i.e. aniline derivatives) by Adin *et al* (1991) resulted in similar THM production to experiments performed with HA. The maximum production for all 3 compounds was similar and occurred at identical Cl₂: organic ratios. This indicates that THM formation proceeds via chlorine attack at the benzene ring rather than at the alkyl side chain.

Chambon (1983) selectively extracted and chlorinated different compounds in urine attempting to identify specific THM precursors in urine. Carboxylic acids, phenols, and aliphatic and alicyclic alcohols, were extracted into an acidic phase, whilst nitrogenous bases were extracted into an alkaline phase before being chlorinated. The acidic phase produced concentrations of THMs 25 times higher than those of the alkaline phase.

Chlorination of a 10mg/l solution of urea (Figure 3.4) by Ueno (1995) with 10mg of Cl_2 resulted in a yield of 23 ppb chloroform. Chlorination of histidine (Figure 3.8) and glycine in the same way yielded much lower levels (1ppb and 2ppb respectively). It has also been shown by Larsen *et al* (1979) and Rice (1980) that chloroform is produced from the chlorination of citric acid. All of these organic compounds are constituents of urine (Table 3.4).

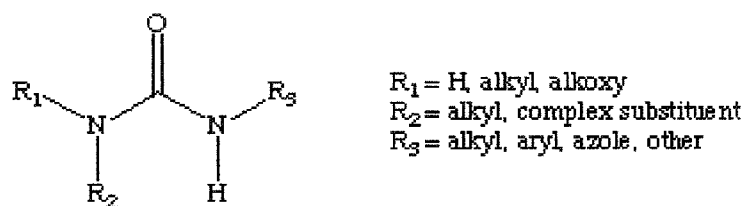


Figure 3.4 Basic structure of urea

Although some of the nitrogenous compounds in urine are known to form THMs, the levels at which they are being added in an average pool (Section 3.5.3) are unlikely to be enough to produce the levels of THMs seen in some pools (Table 3.7). There must therefore be a source of contamination to the pool with a higher propensity to form THMs than urine. Other candidate contaminants include: bacteria, sweat, mucous, skin particles, dirt, various chemicals from cosmetics, and wind blown debris in outside pools (Weil and Eischeldorfer, 1990; Powick 1989; Chambon *et al* 1983).

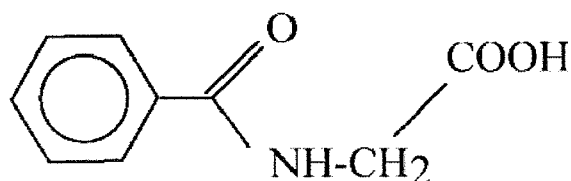


Figure 3.5 Structure of Hippuric Acid

Studies on naturally coloured water used for drinking water have shown that high levels of THMs can be generated from relatively low levels of carbon. Levels as high as 200 µg of CHCl_3 per mg C have been reported (Oliver and Thurman, 1986; Rook, 1977) – two orders of magnitude greater than those reported for urea (Ueno, 1995) - with the two main precursors being humic and fulvic acid (Rock, 1976; Alawi *et al*, 1994). Tests on the propensity of humic and fulvic acids to form THMs show that humic acid consumes 75% more chlorine and produces 117% more CHCl_3 per unit of TOC than fulvic acid (Trussel and Umphres 1978). As humic and fulvic acids constitute over 80% by weight of all organic matter in surface water (Babcock and Singer, 1979) it seems reasonable to suppose that it forms a large constituent of the ‘dirt’ introduced to pools by bathers. Moreover, since ordinary tap water (municipal drinking water) is used to fill swimming pools, it is likely that the make up water provides a low-level source of humic acid (and also chloramine) contamination (McGuire and Meadow, 1988).

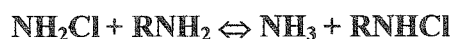
3.5.2 Other DBPs

3.5.2.1 Reaction of chlorine with other nitrogenous compounds

In addition to ammonia, hypochlorous acid also reacts with organic amines to form organic chloramines (Yoon and Jenson, 1993):



Organic chloramines can also form by direct transfer of chlorine from inorganic chloramines, although this is much slower:



Equation 26

Amino acids (Figure 3.6) often contribute to NOM and thus arise in low levels in drinking water supplies and so in pool water. Natural amino acids are varied in structure with different ratios of N, H, C and O. Specific amino acids possessing reactive side groups such as amino nitrogen, sulphur, or activated aromatic rings have been identified as being the main consumer of chlorine (Hureiki *et al*, 1994). The classical mechanism of the reaction of chlorine with the primary amine group proposed in the literature reveals that 2 moles of chlorine are consumed in the first step to form organic monochloramine and dichloramine (Figure 3.7).

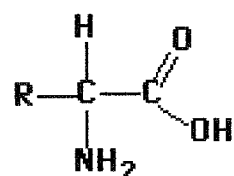


Figure 3.6 General formula of an amino acid

Alouini and Seux (1987), reported that the reaction between hypochlorous acid and α amino acids is analogous to that with amines, forming α (N-chloro) and α (N, N dichloro) amino acids. This reaction is rapid at the initial chlorination stage. The chloroamino acids formed then decompose spontaneously by first order kinetics to form the corresponding aldehydes or ketones, ammonia, carbonic acid and chloride. The rate constant for the reaction shows a dependence on pH that arises as a consequence of the various species that are generated from addition or removal of protons from the amino and carboxyl groups of the molecule.

Alouini *et al* (1986), found that the development of chlorine demand for an aqueous solution of α amino acids differs little from that in swimming pool water. Histidine (Figure 3.8) in particular only displays a regular chlorine demand when large stoichiometric excesses of chlorine are added. Alouni *et al* (1983) and Hureiki *et al*

(1994) both postulated this as being attributable to reactions involving the nitrogen atoms in the heterocyclic ring.

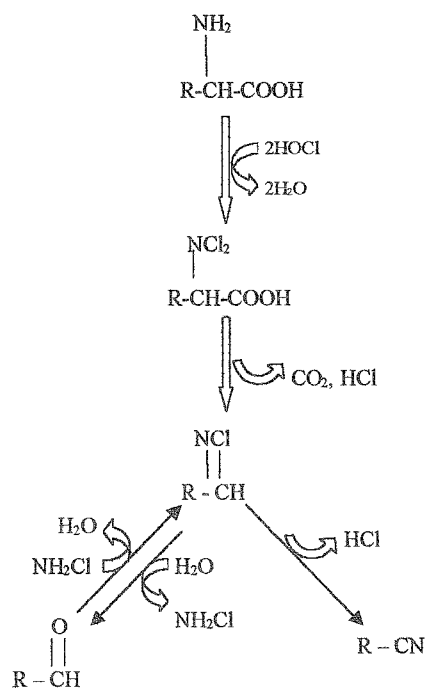


Figure 3.7 Classical mechanism of the reaction of chlorine with amino acids
from, Hureiki *et al* (1994)

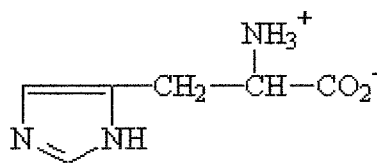


Figure 3.8 Structure of Histidine

Research by Froese *et al* (1999) has suggested that the presence of amino acids in raw waters may be the main cause of off flavours and odours in drinking water supplies. If amino acids are not fully oxidised by chlorination to inert end products such as N_2 , then the formation of aldehydes may generate such problems.

3.5.3 Estimating bather load

Nitrate levels in pool water can be used to estimate the amount of pollution being added to a pool by swimmers since chlorination of the nitrogenous compounds found in both urine and sweat form measurable amounts of nitrate by breakpointing (Section 3.4). The total nitrogen content of sweat is around 1 g/l and of urine around 12g/l, which is sourced mainly from urea, ammonia, amino acids and creatinine (Jandik, 1977). The concentration of potassium in pool water may also be used to estimate bather load, as it is unaffected by chlorine and should give a direct indicator of urine concentration (Erdinger, 1997). Using this method, Erdinger estimated a bather load of 77.5 ml of urine per bather per swim. A summary of estimated bather load according to various authors is given in Table 3.5

Table 3.5 Estimated bather loads of urine.

Reference	Estimated (urine) bather load, ml
Warren and Ridgeway (1978)	25-50
Erdinger (1997)	77.5
Gunkel and Jessen (1988)	25-30
Seux <i>et al</i> (1985)	25-60
Jurion (1982)	25-100

Kuno (1956), estimated that the average amount of sweat produced by an adult is 1 l/hour, for an active swimmer, with an average swimmer producing only 10% of this figure but with an additional load of 50 ml of urine. This equates to a bather load of 1340 mg of urea for a two hour swim. Assuming:

- a) 0.5 mole of nitrate per mole of urea,
- b) a pool volume of 500m³
- c) a user rate of 2000 swimmers per week, and
- d) a 30% loss in pool water volume through splashing and backwashing,

then the nitrate concentration at the end of a 14 week season would be expected to be around 27mg/l NO₃⁻. Assuming the average bather load is ~50 ml urine the concentration of N in urine is ~12g/l, and around ~10% of this will be break-pointed

to nitrate (Palin (1950) then each bather contributes around 0.06mg of nitrate per swim (Warren and Ridegway, 1978).

3.6 *Reported concentrations of DBPs in pool water*

3.6.1 Chloramines

Results of a survey of 14 pools by Lomas in 1967 in which the pH, N level (from free ammonia and chloramines) and the free and combined chlorine residuals were measured (Table 3.6) revealed the latter to be predominantly dichloramine. Concentrations of nitrogen trichloride (NCl₃), the most powerfully lachrymatory of the chloramines and therefore the most problematic, (in swimming pool atmospheres) have been measured between 0.1 – 0.55 as mg/m³ NCl₃ for pool atmospheres with no extras (i.e. baths, spas) and 0.19 – 1.49 for pools with extras (Hery *et al*, 1995). Agitation of pool water by swimmers, baths, slides and spas, and air recycling has been found to increase atmospheric levels. Operating bubble baths within the pool complex for example, can increase the concentration of NCl₃ in the atmosphere by 2-3 times (Hery *et al* 1995).

Table 3.6 Measured concentrations of DBPs in pools Lomas (1967)

Parameter	Range
pH	6.9 - 8.35
Total N, mg/l ¹	0.03 - 0.8
Free chlorine, mg/l	0 - 1.4
Monochloramine, mg/l	0.06 – 0.8
Dichloramine, mg/l	0.18 – 2.6

¹From free NH₃ and chloramines

3.6.2 Measured concentrations of THMs in pool water and air

In a study by Beech 1980(a) concentrations of THMs in swimming pools were shown to be at least 5 times the recommended level for drinking water at that time (100µg/l total THM according to the USEPA, 1979). This led to an interest in swimming pool DBP concentrations and the first extensive study of THM levels in swimming pools was undertaken by Lahl *et al* (1981). These authors measured THM concentrations in pool water and air in 8 pools in Bremen, Germany. Results showed THM levels to vary considerably between different pools, and also within each pool, with the total range of THM concentrations measured in all pool water being 43 – 980 µg/l. Aggazzotti *et al* (1986) found levels ranging from 70 to 177.4 µg/l in 10 pools in Modena, Italy. Other studies (Table 3.7) have revealed similarly varied levels, the lowest level being <4 µg/l (Armstrong and Golden, 1986). Results have also indicated CHCl₃ as being the predominant THM in both air and water.

Based on a reported Henry's Law value of 0.175 for CHCl₃ (Holzwarth, 1984) and pool water results reported by Aggazzotti *et al* (1993) then the concentration in pool air would be expected to range between 3412 µg/m³ to 16,975µg/m³, assuming equilibrium conditions (see Table 3.7). In fact, the reported pool air concentration is only around ~1.5% of that predicted by Henry's Law. This % is similar to that calculated from results reported by Jo (1994) where less than 1% of the CHCl₃ in the pool water is accounted for in air sampling measurements. This significant deviation from Henry's Law suggests that assumptions made by Judd and Black (2000) in carrying out a mass balance across the pool for carbon, nitrogen and chlorine, are inappropriate. These authors assumed that a significant loss of organic matter arose from volatilization in accordance with Henry's Law.

Table 3.7 Summary of THM levels recorded in water and air of real pools

Reference	Disinfectant	No. of pools	THM concentration	
			WATER ($\mu\text{g/l}$)	AIR ($\mu\text{g/m}^3$)
Lahl, <i>et al</i> (1981)	N/S	8	1) R=43-980	
Chambon, <i>et al</i> (1983)	Cl ₂	10	1) M=271, R=83-665	
	NaOCl	14	1) M=163, R=43-543	
	Chloro-Isocyanurate	4	1) M=333, R=280-372	
Faust (1993)	N/S	N/S		1) 31-170, 2) 3.4-7.2, 3) 0.6-1.1, 4) 0.3-4.9
Aggazzotti <i>et al</i> (1993)	N/S	N/S	1) MR = 19.5 - 97	1) M=140, R=49-280
Jo. (1994)	Ozone and NaOCl	3	1) R = 19.5-31.1	Indoor=18-33.6 Outdoor=0.16-0.41
Aiking, <i>et al</i> (1994)	N/S	1	1) M=27.3, R=25.6-28.7	
Aggazzotti <i>et al</i> (1995)	N/S	12	5) R= 9-179	5) R = 16 - 853
Aggazzotti <i>et al</i> (1998)	Various	1	1) M=33.7 R=25-43 2) M=2.3, R=1.8-2.8 3) M=0.8, R=0.5 - 10 4) M=0.1	Before swim 1) M=91.7, R = 69 - 103. After swim 1) M=169.7, R=135 -195
Armstrong and Golden (1986)	Various	5 outside	1) MR=4-402	1) R=0.1 - 260
		5 inside	1) MR= 5 - 580	
Levesque <i>et al</i> (2000)	N/S	N/S	1) R=18 - 80	1) R= 78 - 329

1) CHCl₃ 2) CHCl₂Br 3) CHClBr 4) CHBr₃ 5) total THM

M = mean R =

range

'MR' is used when more than one pool is sampled, and when each of these pools was then sampled more than once to give a mean for that pool.

3.6.3 Estimation of body burden of THMs by swimmers

A method to estimate the potential uptake of THMs by bathers from swimming pools based on concentrations of THMs in air and pool water has been formulated by Beech (1980 b). This method takes into account uptake of THMs via: 1) absorption through the skin; 2) oral absorption (ingestion), 3) buccal and sublingual absorption (through the mouth) and 4) orbital and nasal absorption. Equation 27 can be used to calculate the body burden from dermal absorption, and Equation 28 to calculate the dose from buccal and sublingual routes.

$$D_p = P_c * C_w * T * A_b * 10^{-3}, \quad \text{Equation 27}$$

D_p – dermal dose from penetration of chlorination into epidermal tissue, µg

P_c – permeability constant for CHCl₃ in aqueous solution ($125 * 10^{-3}$ cm/hr. Beech, 1980a)

C_w – mean water CHCl₃ conc. (24.8 µg/l)

T – swim duration (1 hour)

A_b – Adult body area, 18,000cm² for reference adult: (Brown *et al* 1984)

$$D_{bs} = W_p * T * E_{bs} * C_w \quad \text{Equation 28}$$

D_{bs} – dermal dose by buccal and sublingual routes

W_p – volume of pool water passed through mouth, in 1hr swim (5.5 l: Beech, 1980)

E_{bs} – absorption efficiency of chloroform (0.01)

The estimated total dose of chloroform that a 6 year old boy would receive from a 3 hour swim in a pool containing 500µg/l chloroform, is 0.129mg of CHCl₃ per kg of body weight, which is 14 times higher than the estimated dose received from drinking 2 litres of water containing 100µg/l CHCl₃. The estimated body burden of THMs of bathers has been investigated by Lahl *et al* (1991) using the Beech Method and from measurements of real pool water. This resulted in an estimated body burden of 0.77µg THMs per kg of body weight for a one hour swim (Table 3.8).

Measurement of the *actual* uptake of THMs of bathers and lifeguards was attempted by Aggazzotti *et al* (1990). This research focussed on plasma chloroform levels, the basis of which was to assess the correlation between the following factors:

- | | |
|----------------------------------|------------------------------------|
| A. type of swimmer, | D. levels of THMs in air |
| B. number of swimmers, | E. time spent swimming |
| C. levels of THMs in pool water, | F. plasma chloroform concentration |

THM concentrations in plasma (F) were found to be significantly related to: C (r^2 value of 0.478), E (0.573), B (0.322) and most significantly, D (0.739). Further work also revealed a correlation between levels in pool air and those found in bathers by measuring levels of THMs in the alveolar air of swimmers and bath attendants (Aggazzotti *et al*, 1993; Jo, 1994). The correlation measured by Levesque (1994) between aqueous CHCl_3 and alveolar air was 0.75 for a 35 minute swim and 0.8 for an 80 minute swim. A lesser correlation was found between air concentration and alveolar air: 0.58 for 35 minutes swimming and 0.63 for 55 minutes. The significance of exposure to chloroform from dermal absorption versus absorption from inhalation has also been investigated (Levesque *et al*, 1974). 24% of body burden from THM was as a result of dermal absorption alone. Jo (1994), however, estimated that the dermal dose of chloroform was 2.5 times *higher* than the dose from inhalation (Table 3.8).

The concentration in alveolar air in competitive athletes shows a strong correlation to intensity of training and ambient air levels. In groups of leisure swimmers, the only positive correlation reported was between alveolar and ambient air (Levesque *et al*, 2000). Total uptake of CHCl_3 using measurements of CHCl_3 metabolites bound to hepatic and renal macromolecules in the same test subjects gave a total estimated concentration of 0.22 μg of CHCl_3 /kg of tissue. However, this level is at least 10,000 times lower than the smallest no observed effect for liver tumors in animals (Jorgensen *et al*, 1985).

Table 3.8 Calculation of body burden of THMs

Reference	Parameters of investigation			Route of exposure				Total Per Swim Session
	Swim time, min.	Body weight, kg	Average level in pool $\mu\text{g/l}$ and air $\mu\text{g/m}^3$	Amount ingested, ml	Dermal	Oral	Inhaled	
Beech (1980)	180	22	W=500	50ml	1.65 mg	0.079 mg	0.866 mg	B = 0.075 mg O = 0.07 mg Aural = 0.075 mg 0.129 mg/kg (CHCl ₃)
Lahl, <i>et al</i> (1981)	60	70	W=200	50ml	N/S	0.07 $\mu\text{g/kg}$	50 μg	0.77 $\mu\text{g/kg}$ (total THM)
Shatkin and Brown (1991)	20	N/S	N/S	N/S	4.8 mg	N/S	N/S	N/S
Jo (1994)	60	70	W=4.8 A=30.8	50	55.8 μg	0.8 $\mu\text{g/kg}$	23.77 $\mu\text{g/ml}$	O = 1.3 μg N = 1.3 μg 1.18 $\mu\text{g/kg}$ CHCl ₃)
Kaas and Rudiengaard (1987/88)	60	N/S	Water = 150	N/S	700 μg	200 μg	12.9 g (2hr)	N/S

W= water, A = Air, B = buccal and sublingual, O = orbital and nasal

3.7 Health effects of exposure to DBPs

3.7.1 Disinfection by-product regulations

By-products of chlorination that may build up in pool water may be harmful to swimmers and pool operatives and their concentrations are therefore regulated. WHO (2000) recommend a guideline value of no more than 3mg/l for monochloramine in pool water; a level that is based on toxicity data from animals (Thomas *et al*, 1987). The Pool Water Treatment Advisory Group (PWTAG) however, recommends a level of 0.5mg/l, based on the impact of chloramines on odour and eye irritations. There are no recommended levels for either dichloramine or trichloramine in either drinking water or pool water.

Although there are no legal levels set for THMs in swimming pools, the Federation Internationale de Nage Athletique (FINA) recommend a level of no more than 20 µg/l total THM for competitive pools when using chlorine based disinfectants. Some European countries, such as Denmark, have stricter guidelines based on the principle that swimming pool water should be of the same quality as drinking water. The tolerable daily intake of chloroform set by WHO (2000) is 10 µg/kg. Pool water is regularly ingested by swimmers and other research has suggested that chloroform may also be absorbed from pool water through the skin (Section 3.6.3). Prior to 1998, the Occupational Safety and Health Administration (OSHA) permissible exposure limit for chloroform was 50ppm (240 mg/m³, Federal Register 1976), implying that the concentration in the air should not exceed 50ppm at any time. This was revised in 1998 to 2ppm (9.78 mg/m³) as a 60 minute short term exposure limit based on recommendations by the National Institute for Occupational Health and Safety (NIOHS, 1977).

3.7.2 Chloramines

Toxicity data available for chloramines (NH_{3-x}Cl_x) is scant. WHO (2000b) have reported a No Observed Adverse Effect Level (NOAEL) of 94 µg per kg body weight of monochloramine (NH₂Cl) and 150 µg/kg for free chlorine from long-term bioassays in rats (Table 3.9); the maximum limit for monochloramine in drinking water is 3mg/l (WHO, 2000a). All chloramines, but in particular trichloramine (NCl₃),

are powerfully lachrymatory (Mood *et al*, 1951; Rylander *et al*, 1971; Gagnaire *et al* 1994). A survey of lifeguards exposed to NCl_3 from indoor pools revealed them to be at risk of developing irritant eye, nose and throat symptoms from chloramines (Massin, 1998). In another survey, Hery *et al* (1995) found that complaints of eye irritation from non-swimmers at the poolside began at atmospheric concentrations of $\sim 0.5 \text{ mg/m}^3 \text{ NCl}_3$. This concentration is an average of various experiments conducted on a test group; subjects who are 'sensitised' to chloramines can demonstrate adverse reactions at much lower concentrations (Thickett *et al*, 2002). It is thus recommended in the UK that the residual of combined chlorine should be no more than half the free chlorine level (PWTAG, 1999).

Table 3.9 Summary of measured chloramine toxicity

Reference	DBP	Exposure route	Toxicity investigated	Level needed to 'cause harm' or NOAEL level
Eaton <i>et al</i> , 1973	CA	blood/dialysis	Methaemoglobin-aemia in humans	1 mg/l CA in blood
Daniel <i>et al</i> 1990, 1991	MCA	ingestion	Toxicity in 1) rats 2) mice	NOAEL = 1) 5.8 and 2) 8.6 mg/kg body weight
Robinson <i>et al</i> , 1986	MCA	dermal	hyperplasia (increased skin cell growth) in mice	No effect immersing in 1g/l for 10 minutes a day, for 4 days
Abdel-Rahman <i>et al</i> , 1982 and Carlton <i>et al</i> 1986	MCA	ingestion	reproductive or developmental toxicity in 'lab animals'	No effect up to 15mg/kg body weight
NTP, 1992	CA	ingestion	Carcinogenicity in mice	>100 mg/l dose shows positive response

3.7.3 THMs

Much attention has been focused on trichloromethane (CHCl_3), a class 2B carcinogen (IARC, 1999), and its level in pools has been measured by a number of researchers. As previously noted, The NIOSH recommend an exposure limit for CHCl_3 of 2 mg/l for a short-term exposure. Although this is much higher than the upper limit of reported pool water and atmosphere levels, it has been postulated that the long-term exposure of lifeguards and professional swimmers to these lower concentrations may be harmful (Thickett *et al*, 2002). Analyses performed on swimmers and lifeguards, along with models estimating the 'worst case body burden', confirm that pools may

be a major route for uptake of THMs. Research by Aggazzotti *et al* (1990 and 1993) showed a direct correlation between the CHCl_3 concentration in the blood plasma and alveolar air of swimmers and attendants and that in pool atmospheres, and a similar conclusion was reached from experiments involving rats (Islam *et al*, 1996). Shatkin and Brown (1991) estimated that for a 20 minute swim in an average pool, the dermal absorption of CHCl_3 could be as much as 4.8 mg, and Kaas and Rudiengaard (1988) calculated a dermal absorption of 700 μg and an oral intake of 200 μg for a 1 hour swim in a pool containing 150 $\mu\text{g/l}$ CHCl_3 . On the other hand, experiments on lab animals have demonstrated that serious health impacts of chloroform, such as kidney and liver cancer in rats and mice, only occur at doses considerably higher than those arising from a well-run pool (Table 3.10). For example, Templin *et al* (1996b) reported that the NOAEL for CHCl_3 in mice was 50 mg/m^3 at exposure times and frequencies similar to those that may be experienced by lifeguards (6hrs/day and 7 days/week). This dose is 50-500 times greater than the concentrations regularly found in pool atmospheres.

Recent publications by the USEPA (2003) have expressed concern over the occurrence of brominated DBPs in particular. This is because, on a weight-for-weight basis, they are considerably more toxic than non-brominated THMs. Previous research has concentrated on non-brominated DBPs as they are known to form at much higher levels, especially in water where bromide levels are low. This latest research has shown that they may be occurring at unacceptable levels in drinking water, especially where secondary treatments such as ozonation is used (Section 4.1).

Table 3.10 Summary of results of measured chloroform toxicity.

Reference	Exposure route	Toxicity investigated	Level needed to 'cause harm' or NOAEL level
Keegan <i>et al</i> 1998	ingestion	liver damage in mice	>60g/kg body weight, NOAEL = 30g/kg
Templin <i>et al</i> 1996 (a) & (b)	inhalation	Hepatic (liver cell) growth in 1) mice, 2) rats	1) <446 mg/m^3 CHCl_3 for 6 hrs/7 days a week. 2) NOAEL = 50 mg/m^3
Jaimison <i>et al</i> 1996	inhalation	hepatic growth in rats	1490 mg/m^3 for 90 days gave positive response

Most research conducted on the effects of CHCl_3 in humans has focused on epidemiological studies involving chlorinated water supplies (WHO 2000b). However, in a recent study on the acute effects of CHCl_3 (Harada *et al*, 1997) it was reported that accidental exposure to massive doses of CHCl_3 produces heart failure in humans. This finding is consistent with those of Templin *et al* (1996a) who demonstrated doses of 149 and 446 mg/m^3 to be lethal in rats, yielding 40% and 80% mortality respectively after two weeks. Some evidence of increased rates of cancer have been reported in chlorinated water supplies when compared to non-chlorinated sources (Doyle *et al*, 1997), and also in surface water supplies when compared to ground water by Bean *et al* (1982). These studies, however, are often influenced by socio-economic differences in the surveyed population, and therefore often cannot properly quantify the increased risk. In analyses conducted by Tuthill and Moore (1980) and Savin and Cohn (1996) it was concluded that little credence can be placed on such epidemiological findings: when ethnicity, migration patterns and age are taken into account there is little correlation. Having said this, some health risks of prolonged exposure to low levels of DBPs by more vulnerable recipients, such as pregnant women (Nieuwenhuijsen *et al*, 2000), have only recently been identified.