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The Fate of the Haloacetates in Drinking Water—Chemical Kinetics in Aqueous Solution

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Contents

I. Introduction

Chloroacetates are formed during the disinfection of water supplies when oxidizing (active) chlorine compounds are used, e.g., hypochlorous acid, hypochlorite, or dichlorine. When bromide ion is present in the source water, hypobromous acid is formed upon addition of active chlorine. In this fashion, bromoacetates and bromochloroacetates are also formed. The formation of these species due to the action of halogen oxidants upon natural organic matter (NOM) has been the focus of many studies.[1−5] In particular, a number of compounds that degrade to give chloroform have been identified.[2] However, trichloroacetate was not listed among them.

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The fate and transformation of the haloacetates is a concern for two reasons. First, the haloacetates in and of themselves exhibit a degree of toxicity.[6−9] In addition to exposure through chlorinated drinking water, these compounds are metabolites of chlorinated solvents and can be found in urine after exposure to trichloroethene and/or tetrachloroethene. As a Group B2 probable human carcinogen, dichloroacetate has a maximum contaminant level goal (MCLG) of zero. Trichloroacetate has an MCLG of 300 μg L^{-1}. In the United States, the haloacetic acids have come under regulation as part of the Environmental Protection Agency's Stage 1 Disinfection Byproducts Rule.[10] Five of these, known as HAA5,

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Table 1. Acid Dissociation Constants of the Homohaloacetic Acids[a]

acid	pK_a
chloroacetic	2.867
dichloroacetic	1.26
trichloroacetic	0.52
bromoacetic	2.902
dibromoacetic	1.39
tribromoacetic	−0.147

[a] Values were taken from Dean, J. A., Ed. *Lange's Handbook of Chemistry*,13th ed. McGraw-Hill: New York, NY, 1985; p 5•18.

are regulated as part of the rule: mono-, di-, and trichloroacetate and mono- and dibromoacetate. HAA5 is expressed as the sum of the concentrations in micrograms per liter (ppb) and is limited to 60 μg L^{-1}. Additional details on the regulation and analytical chemistry of these DBPs can be found elsewhere.[11,12] Second, haloacetates can be converted to trihalomethanes, which have been regulated in drinking water since the 1980s. Some attempts have been made to model the formation of the haloacetates when a source water is chlorinated.[13] Unfortunately, these models are limited by the completeness of the data used to generate them as has been discussed.[13] In addition to predicting the concentration of haloacetates with time, it is desirable to know whether these species are appreciably converted to trihalomethanes so that the fates of these DBPs might also be effectively modeled.

Lest we think that haloacetates are found only as a result of human endeavors (e.g., potable water disinfection), it is worth taking note that these species are produced by natural processes. Trichloroacetate has been identified in soil. Organisms that possess chloroperoxidases can produce hypochlorous acid (HOCl), which reacts with NOM to form haloacetates and other chlorinated compounds.[14] Biological processes reflect an additional source for these compounds. Consequently, some raw water sources may contain them prior to disinfection. Moreover, if microbes with similar metabolic processes can exist in a biofilm, there may be an additional source in the finished water distribution system.

It is unfortunate that these DBPs have traditionally been named as the haloacetic acids (HAAs) in both U.S. national primary drinking water regulations (NPDWRs) and the literature. In this report, such nomenclature will be abandoned from this point forward as it is inaccurate and misleading. Haloacetic acids are moderately strong; consequently, they are more than 99.99% dissociated to the carboxylate anions under potable water conditions, i.e., dilute concentrations (<50 nM) and pH ≥ 6. This is evident immediately upon considering the acid dissociation constants in Table 1. Moreover, the observable chemistry of the haloacetates is profoundly influenced by their ionic nature. This includes their resistance to extraction and relatively low volatility. Referring to these species as the parent acids emphasizes properties that are not observed and diverts attention from their actual behavior. Accordingly, this report will emphasize the ionic nature of the species by choice of nomenclature.

Halocarboxylates can be transformed into other species in essentially two ways: (1) decarboxylation, which involves the splitting of a carbon−carbon σ-bond and loss of a molecule of CO_2 (or an ion of HCO_3^- or CO_3^{2-}) as in eq 1 where (R may be Cl, Br, and/or H), or (2) nucleophilic substitution, in which a nucleophile displaces a halide ion (X = Cl or Br) on the α-carbon atom. Because water is the most abundant nucleophile in the potable water system, this second transformation is often regarded as a hydrolysis (eq 2) but it can occur directly by hydroxide attack (eq 3).

$$CR_3CO_2^- + H_2O \rightarrow CR_3H + HCO_3^- \quad (1)$$

$$CXR_2CO_2^- + H_2O \rightarrow C(OH)R_2CO_2^- + X^- + H^+ \quad (2)$$

$$CXR_2CO_2^- + OH^- \rightarrow C(OH)R_2CO_2^- + X^- \quad (3)$$

The products that would be predicted from either nucleophilic substitution or decarboxylation of haloacetates are summarized in Table 2. Alternate decomposition reactions (most notably elimination of HX) have been reported in the gas phase[15] but will not be addressed herein.

II. Decarboxylation

Decarboxylation is the spontaneous loss of carbon dioxide from a carboxylic acid moiety. In the case of either the haloacetic acids or their conjugate base anions, the haloacetates, decarboxylation results in the net formation of a halomethane (eqs 4 and 5)

$$X_3C-CO_2H \rightarrow CHX_3 + CO_2 \quad (4)$$

$$X_3C-CO_2\text{-} + H_2O \rightarrow CHX_3 + HCO_3^- \quad (5)$$

where X = Cl, Br, or H in most potable water supplies (but possibly I in some cases). Some carboxylic acids are especially susceptible to decarboxylation, including α-oxo, β-oxo-, α-aryl, α-cyano-, α-nitro-, and of course α,α,α-trihalospecies.[16,17] Understanding the rate and mechanism of decarboxylation is important not only for predicting the fate of these species in potable water supplies, but also for improving understanding of metabolism/detoxification by bacteria, where the decarboxylation is thought to play a significant role in the interaction with dehalogenases.[18]

A. Trichloroacetate

Many of the kinetics studies on the decarboxylation of trichloroacetate were conducted in solvents other than water;[19−28] thus, the results cannot be directly applied here. However, fundamental information about the molecularity of the reaction can be deduced from these studies. For example, Pawlak et al. reported that the protonated form, i.e., trichloroacetic acid itself, did not undergo decarboxylation in acetonitrile.[25] Atkins et al. observed suppression in the presence of HCl, consistent with inertness of the parent acid, when the reaction was studied in dimethyl sulfoxide (DMSO). A few studies have shown

Table 2. Products Expected or Known to Be Formed by Either Decarboxylation or Hydroxide (nucleophilic) Substitution (hydrolysis) of Haloacetates

haloacetate[a]	halomethane after decarboxylation	oxygenated hydrocarbon after nucleophilic hydrolysis[b]
monohaloacetate $CH_2XCO_2^-$	monohalomethane (methyl halide) CH_3X	hydroxyethanoate (glycolate) $CH_2(OH)CO_2^-$
dihaloacetate $CHX_2CO_2^-$	dihalomethane (methylene halide) CH_2X_2	oxoethanoate (glyoxylate)[c] $CH(O)CO_2^-$
trihaloacetate $CX_3CO_2^-$	trihalomethane (haloform) CHX_3	ethanedioate (oxalate)[d] $^-O_2CCO_2^-$ ($C_2O_4^{2-}$)

[a] Of the halogens, X = Cl, Br, or less commonly I. [b] Nucleophilic substitution (hydrolysis) must occur without concurrent decarboxylation for these products to form. [c] Oxoethanoic acid occurs as a polymeric form; see: Urbansky, E. T. *J. Chem. Educ.* **2000**, *77*, 1644−1647. [d] It is reported that decomposition results in the formation of methanoate (formate) via a decarboxylation step, which may be base-assisted. Note that methanoate results from the complete (alkaline) hydrolysis of a trihalomethane.

rate acceleration due to cyclodextrins.[29,30] Compounds with functional groups similar to these might be found among the constituents of NOM.

In 1905, Eichloff reported that trichloroacetic acid and tribromoacetic acid were stable to decarboxylation even in boiling water but that the sodium salts were not.[31] This instability has been taken advantage of for analyzing urine; after heating the sample at 90 °C, trichloracetate is assayed as its degradate (chloroform).[32,33] Decarboxylation can also lead to false high values of chloroform in swimming pool water. When a sample is subjected to headspace analysis, the higher temperatures used to promote volatilization of the analytes also promote the decarboxylation of $CCl_3CO_2^-$, thereby producing more $CHCl_3$.[34] Johnson and Moelwyn-Hughes studied the decarboxylation of both trichloroacetic and tribromoacetic acids.[35] Although they did not account for the degree of dissociation in analyzing their data, they did point out that the anion might be the reactive species rather than the un-ionized acid. Verhoek found that the rate of trichloroacetate decarboxylation was unaffected by the choice of cation when the decarboxylation was studied in aqueous solution.[36] Such behavior is expected due to the high degree of association of the cation and anion with the solvent water and their relative independence from each other. In less polar or nonpolar solvents, differences in rate constants (and therefore activation energies) have been attributed to ion pairing and solvation.[26−28] It is important to point out that Verhoek never varied pH;[36] consequently, his study could not show decarboxylation to be independent of pH. Similar results were obtained by Fairclough,[37] thereby confirming Verhoek's work on trichloroacetate, but again without varying pH. Under high pressure and temperatures (275 bar, 100−260 °C) in a titanium vessel, slower rates were observed for the acids relative to the carboxylate anions, apparently confirming Eichloff's results.[38]

B. Tribromoacetate

As noted above, Eichloff studied both trichloroacetic and tribromoacetic acids.[31] The decarboxylation of tribromoacetate was also studied by de Groote, who followed the reaction by evolution of CO_2 and loss of conductivity; he suggested that the decarboxylation experienced specific base (hydroxide) catalysis.[39] Upon the addition of base, he reported rapid conversion of tribromoacetate to tribromomethane and carbonate. Fairclough also studied tribromoacetate.[37] In contrast to de Groote, Fairclough argued that the decarboxylation was not catalyzed by base. Like Verhoek, Fairclough did not vary the pH; accordingly, there is no experimental evidence for his assertion that the decarboxylation is not base-catalyzed. Fairclough assumed that addition of base would have resulted in immediate nucleophilic substitution of the halide as is known to occur with the monohaloacetates, which he did not observe. He then intuited that the activation energy for the nucleophilic substitution would necessarily be smaller than that for the cleavage of the C−C bond. While this may have appeared reasonable at the time, Hine and Dowell later showed that the C−C bond in trichloroacetate cleaves readily.[40] Moreover, the trichloromethide anion is quite stable (vide infra). Johnson and Moelwyn-Hughes also studied the decarboxylation of sodium tribromoacetate.[35] They obtained an activation energy about 40% larger than that obtained by Fairclough.[37] As Johnson and Moelwyn-Hughes point out, resolving the concomitant hydrolysis from the decarboxylation markedly complicates the data analysis. However, they concluded that nearly all the reaction was carried by the carboxylation anion based on the values of the rate constants for solutions containing the same formalities of either the acid or its sodium salt.

C. Dibromochloroacetate

Sutherland and Aston studied the decarboxylation of dibromochloroacetate.[41] Unlike their predecessors, Sutherland and Aston actually varied the hydroxide concentration. While they observed an increase in nucleophilic substitution due to the base, they observed no change in the rate of the decarboxylation of dibromochloroacetate. For bromochlorofluoroacetate, however, they did observe the expected rate acceleration upon addition of base. This suggests that the cleavage of the carbon−carbon bond is influenced by the stability of the resulting carbanion. In the case of fluorinated acetates, the fluorine atom does not have low-energy d-orbitals to accept electron density back from the carbon atom as is proposed for tribromoacetate, trichloroacetate, and dibromochloroacetate (vide infra).

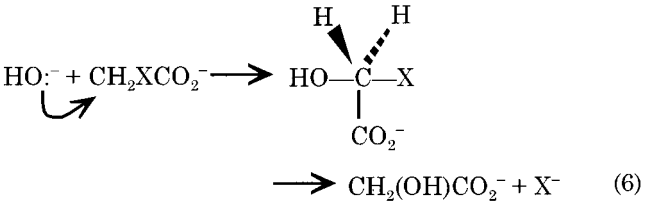
D. Other Haloacetates

Decarboxylation of the dihaloacetates and monohaloacetates has not been studied much. Silberstein showed that chloroacetate could decarboxylate upon heating when dissolved in *N,N*-dimethylaniline, but he did not investigate the reaction in water.[19] Pre-

sumably, by analogy with acetic acid and the acetate anion, the dihaloacetates and trihaloacetates do not readily undergo decarboxylation, except possibly under pyrolytic or other forcing conditions. Dissolution in a tertiary organic alkali, such as *N*,*N*-dimethylaniline, may be sufficient to accelerate decarboxylation owing to the nucleophilic character of the base. Note that tertiary bases cannot usually act as substitutable nucleophiles because such a reaction forms a quaternary ammonium ion. The paucity of investigations into decarboxylation of the dihalo- and trihalospecies may represent a deficiency in the literature but more likely indicates a lack of reactivity under typical experimental conditions.

III. Hydrolytic Nucleophilic Substitution

Nucleophilic substitution of alkyl halides is well-known in organic chemistry, and the reaction mechanisms (S_N1 and S_N2) have been widely accepted since the 1930s.[42] Nucleophilic hydroxide substitution of a monohaloacetate to give glycolate (hydroxyacetate) proceeds according to the S_N2 process illustrated by eq 6, where X = Cl or Br.



$$HO:^- + CH_2XCO_2^- \longrightarrow HO{-}C(H)(H){-}C(X){-}CO_2^- \longrightarrow CH_2(OH)CO_2^- + X^- \quad (6)$$

A. Chloroacetate

The late 19th century saw the first careful study of the hydrolysis of bromoacetate and chloroacetate conducted by Kastle and Keiser in 1893.[43] They observed the loss of the salts of the monohaloacetates by the action of water. One of the earliest works to show the profound influence of base on the rate of chloroacetate substitution took place at the beginning of the 20th century.[44] This has been confirmed for the bromoacetate and chloroacetate.[45−48] For chloroacetate, the nucleophilic substitution by hydroxide has a rate given by eq 7 at 43 °C:

$$-\mathrm{d}[CH_2ClCO_2^-]/\mathrm{d}t = (7.6 \times 10^{-6}\ M^{-1}\ s^{-1})[CH_2ClCO_2^-][OH^-] \quad (7)$$

There can be no question that hydrolytic nucleophilic substitution is faster and more favorable under alkaline conditions. Although water can act as a nucleophile, the reaction involves the net consumption of a hydroxide ion and release of a chloride ion. Therefore, the hydrolysis results in a net gain of hydrogen ion. In the case of chloroacetate, nucleophilic substitution proceeds via a straightforward S_N2 process as expected.[45,49] Combining the rate constants from these two papers, values for the Arrhenius equation parameters for the second-order reaction between chloroacetate and hydroxide are calculated to be as follows: activation energy $E_a = 93.9$ kJ mol^{-1} and preexponential factor $A = 2.68 \times 10^{10}$ s^{-1}.

B. Bromoacetate

The chemistry of bromoacetate is more complicated. Senter's first work suggested that the entire hydrolysis was carried by the direct reaction of bromoacetate ion and water.[44,50] Subsequent papers showed surprising effects from higher concentrations of the sodium salt,[50] but the interpretation of the data is debatable. Senter also studied the effect of silver cation.[51] His lab's later work suggested that the reaction was accelerated by acetate and formate[52] or even by unreacted bromoacetate itself.[53] The ability for other carboxylates to exert a catalytic effect has also been extended to include the substitution product, glycolate.[46,47] The impact of glycolate on the bromoacetate hydrolysis reaction was first observed by Kastle and Keiser,[43] who did not see a similar effect for the chloroacetate reaction. The nucleophilic behavior of glycolate appears to have been thoroughly investigated and confirmed.[48] Even at the time, this behavior was considered unusual as evinced by its inclusion in an article dedicated to anomolous behavior of halogenated compounds.[54] While the nucleophilic behavior of glycolate was observable in kinetics studies, it is unlikely that it could be observed in a potable water system because of the low concentrations of all the reactants relative to the concentration of water. Any contribution of these side pathways should be negligible; consequently, the simple hydrolysis is the only reaction that shall be considered further here. For bromoacetate, the water path has a rate at 25 °C given by eq 8:[48]

$$-\mathrm{d}[CH_2BrCO_2^-]/\mathrm{d}t = (4.1 \times 10^{-8}\ s^{-1})[CH_2BrCO_2^-] \quad (8)$$

Brooke and Dawson did not attempt to discern whether the glycolate acted as a reactant or catalyst in terms of acceleration, but they did propose that formation of acetoxyacetates was responsible for the rate increase.[48] If we plot their rate data, we see a result consistent with an equilibrium-type catalytic (enzyme-like) process (Figure 1). At zero added glycolate, the rate is considerably slower. Linear regression of the data for 0.20−0.50 M glycolate gives a *y*-intercept of 16.5 M s^{-1}. In this glycolate concentration region, the kinetics are nearly first order in excess glycolate concentration. For example, the ratio $[\mathrm{rate}_{(0.40\ M)} - 16.5\ M\ s^{-1}] \div [\mathrm{rate}_{(0.20\ M)} - 16.5\ M\ s^{-1}] = 2.2$, which is close to the value of 2 expected for a first-order system. There are not enough data to confirm this interpretation or to construct a Lineweaver−Burk double-reciprocal plot since only one concentration of bromoacetate was tested. We can suppose that the curve would lie between the two lines drawn on the graph and furthermore would be concave down between 0 and 0.20 M glycolate, but this is little more than a guess without experimental rate data.

C. Dihaloacetates and Trihaloacetates

Whether a first-order (S_N1) process is possible for the dihaloacetates or trihaloacetates depends on the formation of a stable carbocation (actually an ylid

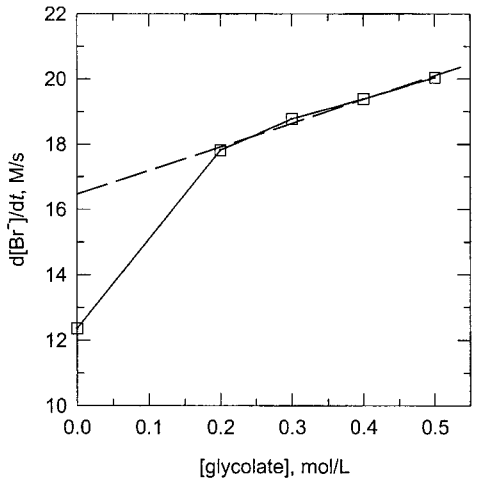


Figure 1. Influence of the concentration of the product (glycolate) on the rate of hydrolysis (nucleophilic substitution) of bromoacetate, based on the data of Brooke and Dawson.[48] The dashed line is constructed from the least-squares regression when the zero concentration point is excluded.

here due to the adjacent anionic carboxylate), the likelihood of which seems dubious at best

$$CXY_2{-}CO_2^- \xrightarrow{-X^-} [{}^+CY_2{-}CO_2^-]^{\ddagger} \xrightarrow{OH^-} CY_2(OH){-}CO_2^- \quad (9)$$

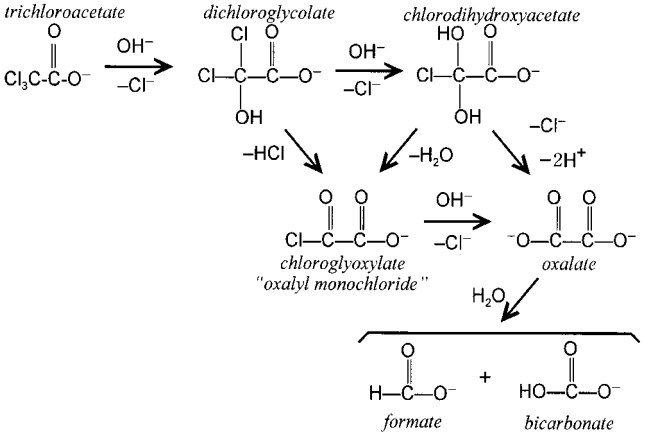
where X = Cl or Br and Y = Cl, Br, or H. Such an ylid should be extremely unstable. Accordingly, it seems reasonable to accept that all nucleophilic substitutions must proceed through concerted S_N2 mechanisms.

Destruction of halogenated hydrocarbons by base is well known and has even been applied as a remediative strategy.[55] Recall that, as early as 1905, Eichloff demonstrated that trichloroacetate and tribromoacetate were attacked by sodium hydroxide and converted entirely to species containing no halogen atoms.[31] Such forcing conditions are not encountered in drinking water; nonetheless, oxalate has been identified in chlorinated drinking water.[5] The mechanism for its formation has not been determined. As noted in Table 2, oxalate is the final product expected from complete hydrolysis of trihaloacetates.

The observed behavior of chloroacetate offers some credence to the mechanism proposed in Scheme 1 for the loss of trichloroacetate. Chloroglyoxylate and dichloroglycolate may be proposed as intermediates in this process but have not been observed. That they have not been observed suggests these species must be susceptible to rapid hydrolysis. This assertion is reasonable because chloroglyoxylate can be viewed as "oxalyl monochloride," the partial hydrolysis product of oxalyl (di)chloride, $(CO_2Cl)_2$. Oxalyl chloride is in fact known to undergo rapid, exothermic hydrolysis to oxalate. Glycolate (hydroxyacetate) has been observed as a product in the base-catalyzed hydrolysis of trichloroethene ($Cl_2C{=}CHCl$) and is therefore a reasonable intermediate here.[55]

In the case of bromodichloroacetate, additional support is found in toxicology and pharmacokinetics studies, where more oxalate is found in urine than

Scheme 1[a]



[a] Possible routes of halide ion loss are shown for the conversion of trichloroacetate to oxygenated species by nucleophilic substitution. The final hydrolysis to formate and bicarbonate is a disproportionation. Although it represents a nucleophilic substitution of sorts, all of the halogen atoms have already been displaced. The reaction rate is expected to be dependent on hydroxide concentration; therefore, the mechanism is shown in terms of hydroxide attack. Nevertheless, the reaction can also be written in terms of the acids undergoing reaction with water and producing hydrogen ion. Presumably, each subsequent step is faster than the preceding step, so that the intermediates are not observed (up to oxalate, for which the hydrolysis is exceedingly slow). Such steps may be postulated under neutral and dilute conditions encountered in chlorinated potable water supplies but have not been studied. Alternate mechanisms include simultaneous decarboxylations and substitutions as explained later in the text.

can be accounted for by known metabolic processes.[8] However, caution must be exercised to avoid drawing incorrect conclusions about fundamental chemistry by extrapolating results from studies in living organisms, where physiobiochemically mediated processes abound. Sutherland and Aston estimated the loss due to hydrolysis during their studies of decarboxylation.[41] If we analyze their rate data in terms of hydroxide concentration (Figure 2), we obtain the following differential rate expression for the nucleophilic substitution of bromodichloroacetate

$$-\mathrm{d}[BrCl_2CCO_2^-]/\mathrm{d}t = (k_0 + k_{OH}[OH^-])[BrCl_2CCO_2^-] \quad (10)$$

where the (uncatalyzed) water path has $k_0 = (1.6 \pm 0.5) \times 10^{-6}$ s^{-1} and the specific base-assisted path has $k_{OH} = (2.4 \pm 0.5) \times 10^{-4}$ M^{-1} s^{-1} at 61 °C. This is a fairly rough approximation due to the estimations made by the original authors. Nonetheless, $R^2 = 0.90$ for 3 degrees of freedom, and the value is probably in the right order of magnitude. The hydroxide-assisted path would not be a major contributor in most potable water supplies; for the hydroxide path rate to reach 10% of water path rate, the pH of the solution would have to be ~10.9.

IV. Combined (Perhaps Inseparable) Modes of Decomposition for the Trihaloacetates

More recent work has suggested that the nucleophilic substitution of trichloroacetate proceeds first by decarboxylation unlike chloroacetate.[56] The net reaction is shown in eq 11. It is possible—even likely—

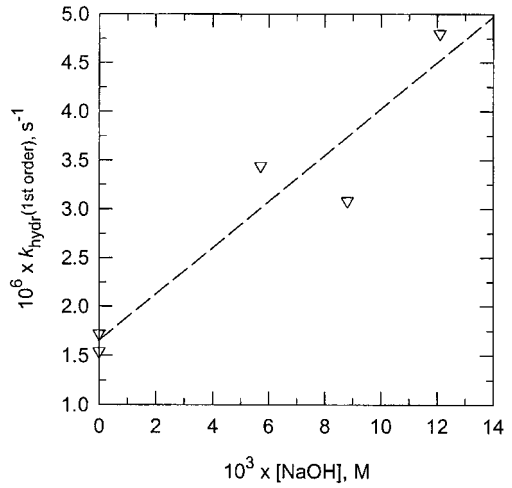


Figure 2. Influence of hydroxide on the rate of nucleophilic substitution of dibromochloroacetate, based on the rate data of Sutherland and Aston.[41] The kinetic data suggest a first-order dependence on [OH^-], consistent with an S_N2 reaction that involves a haloacetate ion and a hydroxide ion (concerted addition−elimination).

that these processes are neither kinetically nor mechanistically distinguishable, both occurring simultaneously. Accordingly, simple kinetic models are perhaps not capable of describing the behavior of these species, especially in high concentrations of strong base or other nucleophiles.

$$Cl_3CCO_2^- + 4OH^- \rightarrow HCO_2^- + HCO_3^- + 3Cl^- + H_2O \quad (11)$$

Nucleophilic substitution occurs on the subsequent products of the decarboxylation rather than on the trichloroacetate ion itself as was suggested in Scheme 1. The authors suggested that trichloromethide was a stable enough carbanion to allow a first-order reaction (eq 12). They hypothesized that stability was imparted by C(pπ) → Cl(dπ) back-donation and resonance, which was consistent with views common at that time.[40,57] However, this explanation has since been contradicted by modern ab initio calculations; CCl_3^- stability is now attributed primarily to the inductive electron-withdrawing ability of the halogen substituent.[58] They offered the mechanism shown in Scheme 2, which proceeds through a singlet dichlorocarbene (dichloromethylene) intermediate.[56]

Scheme 2. Mechanism Proposed by Diefallah and Ghoniam[56] for the Alkaline Decarboxylation of Trichloroacetate[a]

$$Cl_3C{-}CO_2^- \rightarrow :CCl_3^- + CO_2 \quad (12)$$

$$:CCl_3^- \rightarrow :CCl_2 + Cl^- \quad (13)$$

$$:CCl_2 + H_2O \rightarrow [H_2O^{(+)}{\rightarrow}^{(-)}CCl_2] \rightarrow [HOCCl_2]^- + H^+ \quad (14)$$

$$[HOCCl_2]^- \rightarrow :C{\equiv}O: + H^+ + 2Cl^- \quad (15)$$

$$:C{\equiv}O: + OH^- \rightarrow HCO_2^- \quad (16)$$

[a] The first step is the decarboxylation. This is followed by dissociation and then attack by water.

Curiously, the authors wrote all of the steps as irreversible forward reactions rather than treating some as equilibria. There is little doubt that trichloroacetate experiences a high degree of hydration, so that there is a close association between individual water molecules and portions of the reactant. Even with solvent stabilization, it is difficult to imagine reaction 12 as irreversible. Due to a solvent cage that contains the encounter complex, we would expect eq 12 to occur either (1) as a reversible process with individual rate constants that produce a steady state in trichloromethide concentration or (2) as a rapid preequilibrium. The same logic applies to eq 13. There is no discussion of these possibilities in the text. In the case of trichloromethane hydrolysis, Hine and Dowell did in fact consider both reaction 13 and the preceding deprotonation ($CHCl_3 \rightleftharpoons CCl_3^- + H^+$) to be rapid reversible equilibria.[40]

Diefallah and Ghonaim reported that the decarboxylation rate was unaffected by the base concentration, but they varied the hydroxide concentration by a factor of only 2 (0.40 and 0.80 M).[56] Furthermore, no data are presented to support this assertion. First, reaction 12 purportedly represents the rate-limiting elementary step. Second, it produces a Lewis acid, carbon dioxide. Therefore, it is hard to believe that the reaction rate is invariant to hydroxide concentration. A suitable leaving group (CO_2 here) is required. As Clark points out, there is a 1000-fold change in rate constant when the reaction is conducted in solvents of varying nucleophilicity.[59] This is consistent with a bimolecular reaction involving the solvent—specifically, general base assistance in attacking the carboxylate carbon atom—rather than a unimolecular reaction. However, when the solvent acts in a bimolecular reaction, factors other than acid/base assistance may also be involved. It is unfortunate that the reaction has not been conducted in a variety of buffered systems. Concerted deprotonation is required to obtain the leaving group, but this requirement is not sufficient to negate the proposed mechanism. The problem with all the decarboxylation studies is that pH was never varied. Thus, the roles of specific and general acid/base catalyses remain unstudied. Many carboxylates are resistant to decarboxylation because the alkide ion thus produced is intrinsically unstable; however, trichloromethide is considerably stable. As a result, invariance to hydroxide concentration for assorted aliphatic carboxylates or for malonic acids, where a cyclic mechanism is well established,[60] cannot be used for trihaloacetates. Moreover, as Richardson and O'Neal observed, acid-catalyzed decarboxylations are known, and the mechanism of unimolecular decarboxylation cannot be applied generally.[61] Bimolecular reactions involving the solvent are not precluded kinetically,[36,37] and furthermore, trichloroacetic acid does not decarboxylate readily in nonbasic solvents.[36,59] This led Verhoek to conclude that the reaction was probably not the simple unimolecular decarboxylation previously assumed.[36] Whether the observed effects are due to general base assistance remains open to debate. A 1951 review emphasized this uncertainty: "No unambiguous evidence has been

Scheme 3[a]

Acidic/neutral path:

$$Cl_3C{-}CO_2^- \overset{k_{1a}/k_{-1a}}{\rightleftharpoons} :CCl_3^- + CO_2 \quad (17)$$

Basic path(?):

$$Cl_3C{-}CO_2^- + OH^- \overset{k_{1b}/k_{-1b}}{\rightleftharpoons} :CCl_3^- + HCO_3^- \quad (18)$$

$$:CCl_3^- \overset{K_2}{\rightleftharpoons} :CCl_2 + Cl^- \quad (19)$$

Acidic/neutral path:

$$:CCl_2 + H_2O \overset{k_{3a}}{\longrightarrow} HO{-}CCl_2 + H^+ \quad (20)$$

Basic path:

$$:CCl_2 + OH^- \overset{k_{3b}}{\longrightarrow} HO{-}CCl_2 \quad (21)$$

$$HO{-}CCl_2 + 2OH^- \overset{\text{fast}}{\longrightarrow} HCO_2^- + 2Cl^- + H_2O \quad (22)$$

[a] This mechanism is proposed in place of Scheme 2, taking into account a speculated impact of hydroxide. Note that eq 22 is not intended to represent an elementary process but some combination of steps that occur rapidly and allow the mechanism to sum to the overall reaction stoichiometry.

recorded to indicate the decomposition of an ordinary acid molecule by a unimolecular mechanism."[62] In a 1969 review, Clark argued convincingly that the formation of a nucleophilic adduct with the carbonyl carbon (e.g., by the solvent) is essential to decarboxylation.[59] In acetonitrile, a correlation has been observed between increasing strength of an added amine base and increasing rate of trichloroacetate decarboxylation.[25] Linear free energy relationships, such as Brønsted−Pedersen acid/base catalysis, are now well-known in chemical kinetics.[63,64] If an assistance mechanism exists for the haloacetates, it is probably nucleophilic in nature rather than relating to proton transfer and therefore represents a special case of general base assistance.[64,65]

If we modify the proposed mechanism to include rapid reversible reactions—as in eqs 17 and 18—it is clear how a hydroxide dependence could be overlooked, especially given the high concentration of base used. The concentration of trichloromethide could be a steady-state species at drinking water pHs, but eqs 17 and 18 could become essentially irreversible in terms of the kinetics governing the reaction if the product $k_{3b}[OH^-]$ becomes large enough, perhaps when $[OH^-]$ exceeds 0.1 M. It is therefore possible that, at the high concentrations of NaOH used by Diefallah and Ghonaim,[56] the formation of trichloromethide is rate-limiting. In other words, we cannot assume that the behavior at >0.10 M base (pH ≥ 13) represents what would be observed under drinking water conditions, i.e., pH ≲ 9, Scheme 3.

Diefallah and Ghonaim liken the decarboxylation of trichloroacetate to the alkaline hydrolysis of chloroform because both proceed through the formation of trichloromethide anion. Their reported rate constant ($k = 1.34 \times 10^{-8}$ s^{-1}) is about 1/10^4 the value reported for the base-catalyzed hydrolysis of chloroform by Hine and Dowell ($k \approx 2 \times 10^{-4}$ M^{-1} s^{-1}).[40] In 0.20 M NaOH, the first-order rate constant for the loss of chloroform would be 4×10^{-5} s^{-1}. Diefallah and Ghonaim's rate constant is therefore consistent with some process prior to the formation of the trichloromethide ion acting as the rate-determining step because subsequent steps must be the same as for the chloroform hydrolysis once the trichloromethide anion forms. Hine and Dowell concluded that the deprotonation of chloroform was a rapid preequilibrium and that the dissociation of trichloromethide to dichlorocarbene and chloride limited the rate of the chloroform reaction. Unlike Hine and Dowell, Diefallah and Ghonaim wrote the first step, that is, the decarboxylation, as irreversible. That notwithstanding, it is possible for an elementary process to be reversible and still to influence the rate through the formation of a steady-state species.

Because drinking water has an intrinsic buffer capacity due to dissolved minerals or additives (silicates, carbonates, phosphates),[66] no change in pH would occur due to decomposition of chloroacetates, which are formed at nanomolar levels during chlorination. Thus, there is no limiting reagent problem. If Diefallah and Ghonaim's supposition were true (that the reaction rate is invariant to hydroxide concentration), the haloacetates would be as kinetically inert to decay at high pH as they are at low pH. Such is not the case. Loss of chloroacetates occurs at higher pH and is the justification for preserving samples by acidification despite the increased volatility of the acids over the anions.[12] Moreover, recall that both tribromoacetate and trichloroacetate are destroyed by NaOH, but the parent (tribromoacetic and trichloroacetic) acids do not decompose.[31] When these observations are considered along with those of Clark[59] and Verhoek,[36] it is obvious that the disagreement over the roles of Brønsted−Lowry bases and nucleophiles will not be resolved readily.

Unlike the hydroxide concentrations in many of these experiments, those in drinking water are low, usually $<10^{-6}$ M. Consequently, water is more apt to act as a nucleophile than hydroxide. Under these far less forcing conditions, it is not clear what mechanisms prevail. None of the kinetic data in the literature preclude a shift in the predominant reaction pathways as a function of pH. At neutral and acidic conditions, where moderate concentrations of bicarbonate exist in most potable water supplies, reaction 20 would be expected to govern the kinetics. However, in experiments conducted at moderately high concentrations of base (>1 mM), hydroxide can become a significant reactant. If the decarboxylation of trihaloacetates is in fact unimolecular (or bimolecular but with only solvent interaction), then the opportunity for nucleophilic attack by hydroxide is increased with moderate increases in pH, thereby leading to the products suggested in Scheme 1.

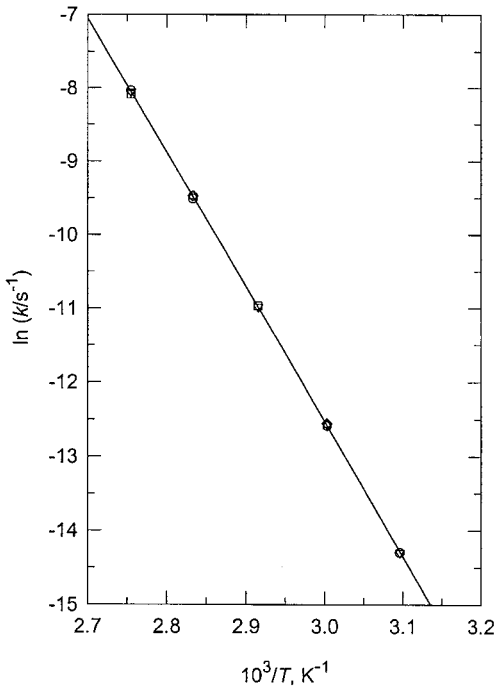


Figure 3. Arrhenius plot for the decarboxylation of trichloroacetate, demonstrating the effect of temperature upon the rate constant, showing the data of Verhoek.[36] Compare with Table 3.

V. Application of Fundamental Kinetics to Modeling DBP Concentrations in Potable Water

One of the concerns mentioned in the Introduction was the potential for the conversion of haloacetates to halomethanes. The decarboxylation of the trihaloacetates to produce trihalomethanes is of special interest. The prior exposition should suffice to demonstrate that there is considerable debate over what is observed and how those observations should be interpreted. Nevertheless, if the decarboxylation rate constants are taken at face value, it is possible to make some predictions about the rate at which trichloroacetate, tribromoacetate, and dibromochloroacetate are transformed into chloroform, bromoform, and dibromochloromethane, respectively. Using preexponential factors and activation energies calculated from the rate data reported in the literature, we can predict the rate constants as a function of temperature and thus the half-lives. A sample Arrhenius plot is shown for trichloroacetate in Figure 3. In general, the original investigators neither plotted their data in this fashion nor analyzed them by least-squares linear regression. Even if the resulting values are within an order of magnitude of the true values, the exercise is instructive. These results are summarized in Tables 3−5.

Casual inspection of the half-lives in Tables 3−5 suggests that trihaloacetate decarboxylation cannot be a significant source of trihalomethanes in drinking water *if* the reported rate constants are even approximately correct. Only at the elevated temperatures encountered in cooking—and to a lesser degree in water heaters—are the decarboxylations sufficiently facile to result in complete destruction (as well as some volatilization). Consequently, the continued formation of trihalomethanes must be due to the direct reaction of halogen oxidants with remaining NOM or with other partly oxidized DBPs that are as yet unidentified. After all, ~62% of the post-disinfec-

Table 3. Effect of Temperature on Decarboxylative Loss of Trichloroacetate[a]

T, °C	k, s^{-1}	$t_{1/2}$, s	$5t_{1/2}$ (≳97% loss) units vary for clarity
0	2.0×10^{-11}	3.4×10^{10}	5400 years
5	6.7×10^{-11}	1.0×10^{10}	1600 years
20	1.9×10^{-9}	3.6×10^{8}	57 years
25	5.5×10^{-9}	1.3×10^{8}	20 years
37[b]	5.9×10^{-6}	1.2×10^{7}	1.9 years
49[b]	5.2×10^{-7}	1.3×10^{6}	77 days
60[b]	3.4×10^{-6}	2.0×10^{5}	12 days
100	1.2×10^{-3}	570	47 min

[a] Arrhenius equation parameters: $E_a = 152$ kJ mol^{-1} K^{-1} and $A = 2.13 \times 10^{18}$ s^{-1} were computed from the data of Verhoek[36] and used to determine the rate constants. Implicit is an assumption that the preexponential factor A is reasonably invariant to changes in temperature. Compare with Verhoek's actual rate data plotted in Figure 3. [b] A temperature of 37 °C corresponds to human body temperature, while 49 and 60 °C correspond to water heater settings of 120 and 140 °F, respectively.

Table 4. Effect of Temperature on Decarboxylative Loss of Dibromochloroacetate[a]

T, °C	k, s^{-1}	$t_{1/2}$, s	$5t_{1/2}$ (≳97% loss) units vary for clarity
0	1.4×10^{-9}	5.1×10^{8}	80 years
5	4.1×10^{-9}	1.7×10^{8}	27 years
20	8.7×10^{-8}	8.0×10^{6}	1.3 years
25	2.2×10^{-7}	3.1×10^{6}	180 days
37[b]	1.9×10^{-6}	3.6×10^{5}	21 days
49[b]	1.4×10^{-5}	5.0×10^{4}	2.9 days
60[b]	7.8×10^{-5}	8900	12 h
100	1.6×10^{-2}	43	3.6 min

[a] Arrhenius equation parameters: $E_a = 138$ kJ mol^{-1} K^{-1} and $A = 3.32 \times 10^{17}$ s^{-1} were computed from the data of Sutherland and Aston[41] and used to determine the rate constants. Implicit is an assumption that the preexponential factor A is reasonably invariant to changes in temperature. [b] A temperature of 37 °C corresponds to human body temperature, while 49 and 60 °C correspond to water heater settings of 120 and 140 °F, respectively.

Table 5. Effect of Temperature on Decarboxylative Loss of Tribromoacetate[a]

T, °C	k, s^{-1}	$t_{1/2}$, s	$5t_{1/2}$ (≳97% loss) units vary for clarity
0	6.7×10^{-8}	1.0×10^{7}	1.6 years
5	1.5×10^{-7}	4.6×10^{6}	265 days
20	1.5×10^{-6}	4.8×10^{5}	27 days
25	3.0×10^{-6}	2.4×10^{5}	14 days
37[b]	1.5×10^{-5}	4.7×10^{4}	2.7 days
49[b]	6.3×10^{-5}	1.1×10^{4}	15 h
60[b]	2.3×10^{-4}	3100	4.2 h
100	1.2×10^{-2}	58	4.8 min

[a] Arrhenius equation parameters: $E_a = 102$ kJ mol^{-1} K^{-1} and $A = 2.54 \times 10^{12}$ s^{-1} were computed from the data of Fairclough[37] and used to determine the rate constants. Contrast Fairclough's activation energy with 142 kJ mol^{-1} K^{-1} obtained by Johnson and Moelwyn-Hughes.[35] Implicit in the calculation of rate constants is an assumption that the preexponential factor A is reasonably invariant to changes in temperature. [b] A temperature of 37 °C corresponds to human body temperature, while 49 and 60 °C correspond to water heater settings of 120 and 140 °F, respectively.

tion halogenated organic matter detectable by adsorption to activated carbon is unknown in composition.[67] Certainly, raw and finished potable water supplies are more complex matrixes than the deionized water or salt solutions used for these kinetics studies. Therefore, additional confirmation of these rate constants and mechanisms is necessary before these values can be applied directly to predict DBP concentration profiles. Whether species other than hydroxide may act as catalysts is an important issue to be resolved. Cramer and Kampe argued that cyclodextrins could engage in bifunctional (general acid/base) catalysis by simultaneously providing a proton to the anion and a nucleophile to the carboxylate carbon, thereby promoting the decarboxylation.[30] Given the range of compounds that make up NOM, it is likely that compounds with the same moieties and shapes as cyclodextrins can be found dissolved in water supplies. Verhoek concluded that metal cations did not influence the rate in aqueous solution.[36] On the other hand, more recent work has suggested that the surface of the reaction chamber (e.g., a pipe wall) might speed up the reaction. Specifically, high-pressure studies conducted in a titanium cell showed faster decarboxylation than those in a cell constructed of 316 stainless steel.[38] Even more interesting, the activation energies and preexponential factors are such that Arrhenius plots (ln k versus T^{-1}) for the acid and the anion cross over each other. In other words, the decarboxylation of the acid is faster than than the anion under some temperatures and slower under others.[38] The complicated combined reactions that appear to be responsible for the net decomposition of the trihaloacetates require further elucidation, especially to determine which pathways predominate under typical potable water conditions. Even if decarboxylation occurs stoichiometrically to produce trihalomethanes, typical haloacetate concentrations would normally augment the trihalomethane concentrations by <15%. Therefore, the primary focus should be on the loss of the trihaloacetates as opposed to a possible gain of trihalomethanes.

Rate constants and mechanisms for the decomposition of the dihaloacetates are largely unexplored. It is possible that two halogen atoms are sufficient to offer stability to a dihalomethide anion, CHX_2^-, especially if one is bromine. Whether the dihaloacetates behave more like the monohaloacetates or the trihaloacetates remains unknown. More effort has been directed in the past to trihalo species over dihaloacetates or dihalomethanes. Presumably, the dihalomethanes do not represent a substantial portion of post-disinfection halogenated matter. Unquestionably, the behavior of these compounds is ripe for study. As an interesting complication, bromochloroacetate is the only chiral haloacetate [(*R*) and (*S*) enantiomers] routinely encountered in finished potable water. Influences of chirality on biological activity are only beginning to be studied.

The monohaloacetates have been adequately studied, and there is a satisfactory foundation which could be further built upon with a modest effort. The monohaloacetates behave in a manner similar to simple alkyl halides, undergoing nucleophilic substitution (eqs 7 and 8), albeit at a rate too slow to have practical consequences. Given the behavior of acetic acid itself toward decarboxylation, it is likely that monohaloacetates do not undergo decarboxylation under any condition to which drinking water would normally be exposed, including boiling. Additional study of the monohaloacetates is probably not necessary from a practical standpoint, but might afford a better understanding of haloacetates overall, and may be important in explaining microbiologically mediated detoxification processes as described earlier.

The role of biodegradation may now be seen as potentially more important in light of a study monitoring the concentrations of the haloacetates in a distribution system.[68] Let us consider the fate of trichloroacetate in this study. During the warm season, when the water temperature was 20−25 °C, the trichloroacetate concentration after 3 days in the distribution system was 0.030−0.061 μM (5−10 μg L^{-1}) lower than it had been after 1 day, i.e., 0.03 μM $\leq \Delta[Cl_3CCO_2^-]_{1-3d} \leq$ 0.06 μM. Meanwhile, the chloroform concentration was reasonably constant throughout this same period: 0.25 μM $\leq [CHCl_3] \leq$ 0.33 μM (30−40 μg L^{-1}). The (molar) stoichiometry between trichloroacetate and chloroform is 1:1; however, $\Delta[CHCl_3] \leq$ 0.06 μM (= 7 μg L^{-1}) would be difficult to distinguish from experimental error. It is unfortunate that limitations in the $[CHCl_3]$ data preclude evaluation of the mass balance. Regardless, the decarboxylation reaction could not possibly be responsible for the loss of trichloroacetate since the half-life predicted at 25 °C is 20 years (see Table 3). Dichloroacetate and dibromoacetate showed a precipitous drop between 1 day and 3 days out. In fact, these two species were often undetectable after 3 days. Even at colder temperatures (≤15 °C), loss of these homodihaloacetates was significant and far more than could be expected from simple inorganic (abiotic) decarboxylation and/or nucleophilic substitution (hydrolysis).

Regulations regarding trihalomethanes have been predicated on the assumption that trihalomethane concentration profiles are strictly increasing functions, which appears to be borne out by the data for many potable water systems. In other words, $[CHX_3]_{max}$ occurs at the longest residence times in the distribution system (residence time essentially corresponds to reaction time). On the other hand, this appears not to be the case for the haloacetates; the concentration maxima occur nearer to the point of disinfection (i.e., at the utility plant or at the head of the distribution system). Moreover, conversion of trihaloacetates to trihalomethanes (decarboxylation) cannot be responsible for the loss of the trihaloacetates if the kinetics are even close to correct. Several investigators have suggested that biodegradation is responsible for this drop in haloacetate concentration in the distribution system.[69−72] If the phenomena are biochemically or biologically mediated (such as via a dehalogenase enzyme), then models based strictly on inorganic chemical kinetics will never be able to predict the concentrations.

Nonetheless, it is still necessary to know what limits on reaction rates are imposed by the inorganic chemistry alone if we are to invoke an alternative explanation. Microbial life varies from system to system; therefore, wider variation in rates of haloacetate loss can be expected than would otherwise be predicted on purely chemical grounds. This suggests that models developed for some systems may not apply at all to others.

VI. Research Needs

Clearly, there is disagreement over the nature of the decomposition reactions for the haloacetates. The nature and existence of specific or general acid/base catalyses remain in question. It is reasonable to believe that specific (if not general) base catalyses occur. Unfortunately, the experimental design of most studies of haloacetate decomposition was such that it is not possible to assess acid/base effects. The assertion that the decarboxylation reactions are necessarily unimolecular rather than bimolecular (involving a water molecule or hydroxide ion) has not been fully tested.

A systematic study of the pH-dependence of decarboxylation and subsequent/simultaneous hydrolyses or nucleophilic substitutions has not been done. Designing experiments to determine the magnitude of specific or general base assistance should be fairly straightforward, but conducting these experiments may be more complicated in actual practice. Research needs are best presented as a series of questions.

(1) Is decarboxylation of haloacetates (especially trihaloacetates) assisted by bases or acids? If so, can this phenomenon be observed under a reasonable range of drinking water pHs, e.g., 6−9? Can the measured rate constants reported in the literature be confirmed using modern techniques at more dilute concentrations?

(2) In addition to acid/base assistance, can other types of homogeneous catalysis occur, given the range of chemical species commonly found in a potable water supply? Does heterogeneous catalysis play a role in the degradation of haloacetates via interaction with the surfaces of a metal pipe wall, mineral deposits (scales) on the pipe wall, or particulates suspended in the water stream itself?

(3) Are there conditions where decarboxylation occurs with a minimum of nucleophilic substitution? Do all haloacetates undergo decarboxylation to halomethanes the way the trihaloacetates do? Are monohalomethanes (methyl halides) or dihalomethanes (methylene halides) observed as DBPs or as degradates of other DBPs? In finished water supplies, are the haloacetates transformed to haloalkanes by decarboxylation to any appreciable extent?

(4) Can all haloacetates undergo nucleophilic substitution without decarboxylation to give the full range of oxygenated products: hydroxyacetate (glycolate), dihydroxyacetate, oxoethanoate (glyoxylate), ethanedioate (oxalate). Alternately, is posthydrolysis decarboxylation to give methanoate (formate), hydrogen carbonate, and carbonate possible?

(5) How do decarboxylation and nucleophilic substitution combine to give the net distribution of end products at drinking water conditions? What is the interplay between the rates of parallel or consecutive reactions? Are the processes kinetically and/or mechanistically distinguishable?

(6) Are the activation energies for these reactions in the realm where decomposition can be expected to occur rapidly when water is heated to physiological, cooking, or bathing temperatures?

(7) Are biologically mediated processes entirely responsible for the loss of haloacetates with increased residence time? Are the rates of strictly chemical processes so slow as to require an enzyme rather than a nonbiological catalyst (cf. question 2)?

(8) If biodegradative processes are the primary means of haloacetate loss, are the consortia of microbes sufficiently similar among water distribution systems to allow the development of a generalized model of haloacetate decay?

(9) Do systems with higher residual disinfectant show reduced rates of haloacetate decomposition? If pilot-scale distribution systems are constructed and run under conditions that inhibit biodegradation, can they obtain haloacetate concentration profiles consistent with the reaction kinetics reported in the chemical literature, that is, no observable loss? Suppose a utility plant finished water experiences haloacetate degradation when sampled after residence in the distribution system. If the same finished water is instead collected at the plant and filtered to remove microbes, will the haloacetate concentration profiles remain essentially constant, thereby demonstrating that all degradation occurs from some phenomena that take place specifically within the distribution system?

(10) Is the rate of haloacetate loss equal to the rate of biodegradation or does it reflect a net rate that includes both a rate of production (from the reaction of residual disinfectant and organic matter) and a rate of simultaneous destruction by living organisms?

(11) Are the same organisms and/or enzymes responsible for the destruction of mono-, di-, and trihaloacetates? Do decarboxylation and nucleophilic substitution play important roles in the overall process as well? What are the roles of dehalogenases? Are there radical and peroxo intermediates in microbiodegradation as have been postulated in eukaryotic peroxisomes?

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