University of Nevada, Reno

Reduction of Disinfection Byproduct-associated Toxicity by Adjustment of Distribution System pH

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science in Civil and Environmental Engineering

by

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Abstract

Nitrogenous disinfection byproducts (N-DBPs) such as Haloacetonitriles (HANs) and haloacetamides (HAMs) have come under scrutiny in recent years due to their high toxicity compared to currently regulated carbonaceous disinfection byproducts (C-DBPs). Lowering concentrations of N-DBPs in drinking water is an important step for mitigating toxicity exposure to the public. A possible mitigation strategy may be to distribute water at increased pH, resulting in increased HAN hydrolysis to HAMs and HAM hydrolysis to haloacetic acids (HAAs). Because the ranked toxicity of these groups is HAN > HAM > HAA, such a hydrolysis scheme would result in reduced sample toxicity. The formation and degradation of HANs and HAMs was examined in conventionally treated surface water and in Milli-Q water spiked with two natural organic matter (NOM) isolates using formation potential (FP) tests at varying pH. HAN concentrations in finished drinking water at pH 6, 7.5, and 9 at the end of the 5-day experimental period were 12.5, 7.1, and 1.9 µg/L, respectively, equivalent to a 77% (pH 6 to 9) and 45% (pH 7.5 to 9) decrease in summed calculated toxicity from the DBPs measured. Similar conclusions followed from the FP tests with NOM isolates. Increasing distribution system pH is an effective method to reduce HAN-associated toxicity.

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Chapter 1: Introduction and Structure of Thesis

1.1 Introduction, Objective, and Justification of Thesis

As human population and potable water consumption continue to grow as the 21st century progresses, new processes for potable reuse, water-saving technologies, and treatment methods for mitigating drinking water pollutant exposure to the public will be important for advancing national and global water security. Advances in these processes will be of utmost importance in arid regions and densely populated cities, including much of the western U.S. To ensure excess demand from population growth is met and water systems are truly robust, many water purveyors have invested in aquifer storage and recovery (ASR) infrastructure as key components for ensuring the resiliency of their systems. Aquifer storage and recovery is a method of treating and injecting surface water into wells to recharge a local aquifer for later use in times of drought. ASR programs are typically operated in areas where seasonal variations in water supply lead to an excess or deficit of water availability at different times of the year or interannually. ASR programs are common throughout many southwestern and western states due to large population centers, intensive agriculture, and limited surface water availability. Such is the case in Reno, Nevada. During spring snowmelt, excess water is taken from the Truckee River, treated, and injected into aquifers for recovery in late summer and early fall, when river flows are low, but demand is high. Residual chlorine from disinfection in many cases reacts with organic matter in the aquifer and drinking water distribution systems, and results in the formation of disinfection byproducts (DBPs).[1] DBPs have been linked to

liver, kidney, and central nervous system malfunction.[2] DBPs of highest abundance by mass include trihalomethanes (THMs) and HAAs.[3] THMs and HAAs are some of the most widely studied DBPs to date and were regulated by the U.S. EPA soon after their discovery in the 1970s.[4] However, hundreds of DBPs have been discovered since then, many of which have been found to be more geno- and cytotoxic at concentrations orders of magnitude lower than THMs and HAAs. Such is the case for HANs and HAMs; nitrogenous DBPs that form as a result of reactions of chlorine and chloramine disinfectants with organic matter in drinking water treatment and distribution systems. Interest in these compounds has grown in recent years due to the widespread prevalence of N-DBPs in potable water supplies and their high toxicity at low levels, many having notable geno- and cyto-toxic effects even at sub-ppb levels.

Due to their high toxicity at relatively low concentrations, HANs are considered to be the main drivers of DBP-associated cytotoxicity in many finished drinking waters. Figure 1.1 shows example data from a drinking water treatment plant indicating the relatively low concentration of HANs and large contribution to DBP-associated cytotoxicity in treated drinking water.[5] While HANs constituted only 2% of TOX by mass, the contribution to DBP-associated cytotoxicity was 67%, the largest of any DBP class measured.

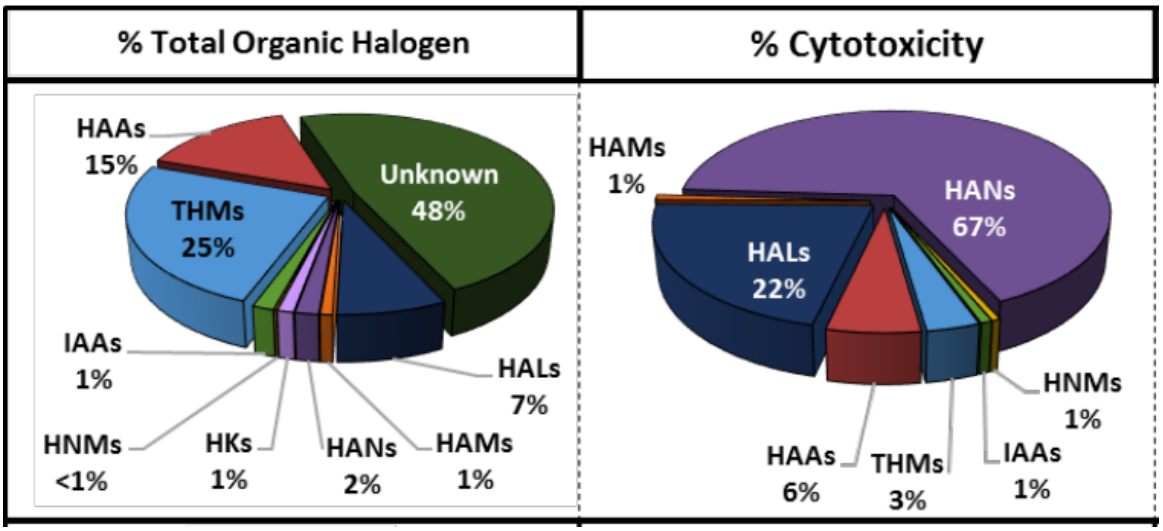

Figure 1.1: Breakdown of Total Organic Halogen (TOX) and cytotoxicity contribution by each DBP class measured from a drinking water treatment plant utilizing pre-oxidation and granular activated carbon (GAC) treatment.

Important precursors for HANs are nitrogenous compounds including those present in wastewater and agricultural effluents such as amino acids, proteinaceous material, nucleic acids and inorganic nitrogen such as ammonia, nitrite, and nitrate. Eutrophicated source waters may be impacted by algal organic matter that also contains nitrogenous precursors leading to high levels of HAN formation upon disinfection.[6] Formation potential experiments were conducted using water collected from a treatment plant utilizing pre-oxidation and GAC filtration and DBP classes by mass and contribution to cytotoxicity are shown in figure 1.1. The treatment plant was reported to have minimal algae, wastewater, and agricultural influence on the source water, indicating that even in the absence of highly impacted source waters, enough nitrogenous precursors are available in naturally present organic matter to form significant concentrations of HANs upon disinfection.

The goal of this research was to provide a novel method for the reduction of toxicity associated with HANs in finished drinking water samples through laboratory simulated distribution system experiments and understand DBP formation during point-of-use (POU) water treatment.

1.2 Structure of Thesis

A brief introduction to the research topic as well as objective and justification of the thesis is provided in Chapter 1. Chapter 2 contains a draft publication which includes a literature review, description of experimental methodologies, and demonstrates the potential for small changes in distribution system pH to reduce exposure to N-DBPs. Chapter 3 details formation of DBPs during POU water treatment experiments. Analytical methods were developed to quantify DBPs in drinking water using Gas Chromatography Electron Capture Detection (GC-ECD). Information on GC-ECD method development, parameters, and retention time of compounds can be found in Appendix A.

Chapter 2: Reduction of Disinfection Byproduct-associated Toxicity by Adjustment of Distribution System pH

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Keywords

Disinfection Byproducts, Nitrogenous DBPs, Haloacetonitriles, Haloacetamides, Calculated Toxicity

2.1 Abstract

Haloacetonitriles (HANs) and haloacetamides (HAMs) have come under scrutiny in recent years due to their high toxicity compared to currently regulated carbonaceous disinfection byproducts. Lowering concentrations of N-DBPs in drinking water is an important step for mitigating toxicity exposure to the public. HANs and HAMs have been shown to be susceptible to base catalyzed hydrolysis, resulting in formation of HAAs and THMs, hydrolysis products of lower toxicity. A possible mitigation strategy may be to distribute water at increased pH, resulting in increased HAN hydrolysis to HAMs and HAM hydrolysis to HAAs. Because the ranked toxicity of these groups is HAN > HAM > HAA, such a hydrolysis scheme would result in reduced sample toxicity. The formation and degradation of HANs and HAMs was examined in conventionally treated surface water and in Milli-Q water spiked with two natural organic matter (NOM) isolates using formation potential (FP) tests at varying pH. HAN concentrations in finished drinking water at pH 6, 7.5, and 9 at the end of the 5-day experimental period were 12.5, 7.1, and 1.9 µg/L, respectively, equivalent to a 77% (pH 6 to 9) and 45% (pH 7.5 to 9) decrease in summed calculated toxicity from the DBPs measured. Similar conclusions followed from the FP tests with NOM isolates. Increasing distribution system pH is an effective method to reduce HAN-associated toxicity.

2.2 Introduction

Total trihalomethanes (THMs, sum of chloroform, bromodichloromethane, dibromochloromethane, and bromoform) and five haloacetic acids (HAA_5, sum of mono-,

di-, and trichloroacetic acid, and mono-, and dibromoacetic acid) are currently regulated in drinking water by the United States Environmental Protection Agency at 80 µg/L and 60 µg/L respectively.[4] The US EPA is currently considering whether to regulate DBPs further, and as of July 2021 the draft contaminant candidate list (CCL) 5 has been published and includes dichloroacetonitrile (DCAN), and dibromoacetonitrile (DBAN) as possible candidates for regulation under the Disinfection Byproducts Group. With the possible future regulation of HANs, treatment strategies may be necessary to meet regulatory requirements.

Many DBPs induce cyto- and genotoxic responses and the toxicity of many individual DBPs has been assessed by various *in-vitro* and *in-vivo* assays.[7-14] A common method for comparing DBP toxicity is to compare their lethality (LC_{50}) with Chinese hamster ovary (CHO) cells.[13] Further, the drivers in the overall toxicity of a sample can be determined by weighting DBP concentrations by their lethality (i.e., the concentration of an individual disinfection byproduct is divided by its corresponding LC_{50}). Summing the weighted concentrations results in a total toxicity of the sample, often referred to as the "summed calculated toxicity" of a sample. The summed calculated toxicity can be compared against itself after a proposed treatment to assess whether a treatment had a net positive or negative impact on the quality of the water, assuming the majority of the toxic substances have been measured and weighted by their lethality.[15] A similar approach for genotoxic agents is to use a 50% tail DNA moment (i.e., measure of DNA strand breaks) as the toxicity weighting factor.[13]

Although HANs account for less than 2% of total organic halides (TOX) by mass in chlorinated waters,[16] their contribution to calculated toxicity, compared with currently regulated DBPs, suggests that they are the main drivers of DBP-associated toxicity of the known and characterized byproducts.[17,18] To date, many treatment methods have been proposed for the control of HANs in drinking water. These include pre-ozonation,[19] direct UV photolysis, persulfate treatment, UV/H_2O_2, and UV/persulfate processes,[20] several strategies involving nanomaterial catalytic treatment,[21,22] point-of-use heat treatment,[23] and natural organic matter removal.[24] However, these treatment methods involve energy intensive and costly equipment or are not easily implementable at existing water treatment plants (WTPs).

Alternatively, several kinetic studies have been published in recent years demonstrating predictable hydrolysis of HANs to HAMs, and then HAMs to HAAs.[25-27] Notably, this sequential hydrolysis pathway (Figure 1) is in order of decreasing toxicity (excepting bromoacetonitrile, which is slightly lower in toxicity than the corresponding bromoacetamide). Higher pH and chlorine residual lead to increased hydrolysis rates presumably through nucleophilic attack by hydroxide and hypochlorite ions at the nitrile and carbonyl carbons of HANs and HAMs, respectively.[27]

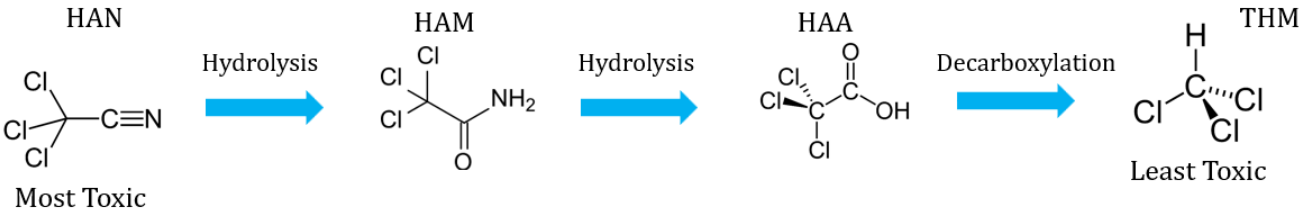

Figure 2.1: Hydrolysis of HANs to HAMs, HAMs to HAAs, and decarboxylation of HAAs to THMs.

Taking advantage of base-catalyzed hydrolysis of HANs is a simple, economically viable, and easily implementable treatment method for lowering N-DBP-associated toxicity in delivered drinking water. The rate of HAN hydrolysis is increased at higher pH, pushing HANs to products of lower toxicity. To achieve this, a distribution system was simulated by holding treated drinking water or Milli-Q water spiked with NOM isolates and sodium hypochlorite at varying pH and comparing the resulting DBP-associated toxicity.

2.3 Materials and Methods

1. Sample Collection

Finished drinking water was collected in October 2021 from a conventional drinking water treatment plant in Reno, NV utilizing coagulation, flocculation, sedimentation, filtration, and free chlorine disinfection. The sample was immediately transported to the laboratories at the University of Nevada, Reno. Suwannee River NOM (2R101N) and Upper Mississippi River NOM (1R110N) reverse osmosis (RO) isolates were purchased from the International Humic Substances Society.

2. Experimental Methods

The finished drinking water sample had a free chlorine residual of 1.7 mg Cl_2/L which was not further supplemented. Samples were buffered using a 10mM phosphate buffer at three different pH: 6, 7.5, and 9, confirmed using a pH probe. For experiments with NOM, 3 mg C/L was spiked of either SRNOM or UMRNOM. The samples were buffered the same as the drinking water samples and sodium hypochlorite stock solution

was added, resulting in an initial concentration of 9 mg Cl_2/L. Chlorine was quenched within one hour after chlorination at the treatment plant for the drinking water sample or immediately after the chlorine spike in the NOM tests to produce the initial data points. Treatment groups were partitioned to headspace-free 70 mL reaction vials for the allotted reaction times (up to 5 days) at room temperature (22°C). 36% of the data was produced via experimental triplicates (initial, 1-hour, and 24-hour samples). After the planned reaction duration, free chlorine was quenched with 50 µL of 0.5 M ascorbic acid. Samples were then acidified using 150 µL of 12 N HCl to preserve the DBPs and stored at 5°C for up to two weeks before extraction.

3. Analytical Methods

Four THMs, six HANs, and six HAMs were extracted and quantified using liquid-liquid extraction (LLE) with methyl tert-butyl ether (MTBE, Fisher HPLC grade) and gas chromatography with a HP-5 column (Agilent, 30m length, 0.32mm diameter, 0.25µm film) and electron capture detection (GC-ECD, Agilent 8860) similar to EPA Method 551. Specific oven programs can be found in the supplemental information. In brief, 30 mL of sample was pipetted into a pre-ashed 40 mL vial. 3 mL of MTBE containing 1 µg/mL of 1,2-dibromopropane and 10 grams of granular, anhydrous sodium sulfate (Fisher, 99%) were added to the vials. Samples were shaken at 250 rpm for 30 minutes using an orbital shaker. Approximately 1 mL of the upper organic layer of each extract was transferred to GC vials and stored at -20°C for up to 14 days until GC-ECD analysis.

Nine HAAs were extracted using LLE, esterified, and quantified using GC-ECD with DB-1 column (Agilent, 30m length, 0.25mm diameter, 1µm film) based on EPA Method

552. Briefly, 30 mL of sample was transferred to a pre-ashed 40 mL vial. 20 µL of 20 µg/mL 2-bromobutyric acid (surrogate standard), 1.5 mL concentrated sulfuric acid, 3 mL of MTBE containing 1 µg/mL 1,2,3-trichloropropane (internal standard), and 10 grams of anhydrous sodium sulfate were then added to each vial. Samples were then shaken at 250 rpm for 30 minutes using an orbital shaker. 1 mL of MTBE extract was then transferred to a new clean and baked 20 mL vial containing 2 mL of acidic methanol (5% H_2SO_4). The extract and acidic methanol mixture was placed in a water bath at 50°C for 2 hours. Upon completion of the esterification, vials were cooled for approximately 5 minutes, and 5 mL of saturated sodium bicarbonate solution (96 g/L) was added to each vial for pH adjustment. 1 mL of MTBE was then added to each sample and vials were shaken for 2 minutes. 1 mL of final extract was transferred to GC vials and stored at -20°C for up to 14 days until GC-ECD analysis.

Free chlorine was measured using a Hach DR 6000 spectrophotometer with the standard N,N Diethyl-1,4 Phennylenediamine (DPD) reagent method. Total organic carbon (TOC) was quantified using a Shimadzu TOC analyzer using standard methods.[28] pH was measured with a Fischer Scientific Accumet 13-620-299B pH probe. Error associated with calculated toxicity sums includes error propagated from experimental/analytical uncertainty from the DBP formation and measurement and a 12% assumed error from the published LC_{50} values.[15,29]

4. Reagents

EPA 501/601 2,000 µg/mL trihalomethanes calibration mix containing chloroform (99.9%), bromodichloromethane (BDCM, 97.2%), dibromochlormethane (DBCM,

96.5%), and bromoform (96.1%) and EPA 552.2 2,000 µg/mL haloacetic acids mix (HAA_9) containing chloroacetic acid (CAA, 98.1%), dichloroacetic acid (DCAA, 98.5%), bromoacetic acid (BAA, 98.1%), trichloroacetic acid (TCAA, 99.9%), bromochloroacetic acid (BCAA, 98.3%), bromodichloroacetic acid (BDCAA, 99.6%), dibromoacetic acid (DBAA, 100%), chlorodibromoacetic acid (CDBAA, 98%), and tribromoacetic acid (TBAA, 99.7%) were purchased from Sigma Aldrich. 5,000 µg/mL disinfection byproducts standard containing bromochloroacetonitrile (BCAN), trichloronitromethane (TCNM), dibromoacetonitrile (DBAN), dichloroacetonitrile (DCAN), 1,1-dichloroacetone (1,1-DCA), trichloroacetonitrile (TCAN), 1,1,1-trichloroacetone (1,1,1-TCA) was purchased from Agilent. Bromoacetonitrile (BAN, 97%), chloroacetonitrile (CAN, 98%), chloroacetamide (CAM, 98%), bromoacetamide (BAM, 98%), dichloroacetamide (DCAM, 98%), trichloroacetamide (TCAM, 98%), 2-bromobutyric acid (99%), 1,2-dibromopropane (98%), and 1,2,3-trichloropropane (98%) were purchased from Sigma Aldrich. Dibromoacetamide (DBAM, 99%), and bromochloroacetamide (BCAM, 99%) were purchased from Toronto Research Chemicals.

2.4 Results and Discussion

DBP Formation at Circumneutral pH

Initial experiments utilized finished drinking water collected from a regional drinking water treatment plant in Reno, NV, and confirmed the conclusions using Milli-Q water spiked with NOM and sodium hypochlorite and buffered at varying pH. Figure 2.2 shows example data of the formation of individual DBPs in the finished drinking water at

circumneutral pH stacked by common functional group. Data for other pH are provided in Figures B-1 and B-2. THMs, HAAs, HAMs, and HANs were formed at detectable concentrations in all pH groups tested. The greatest species by mass for each DBP class was chloroform, bromochloroacetic acid, bromodichloroacetic acid, dichloroacetamide, and trichloroacetonitrile, and this was similar over time. HAN concentrations reached a maximum of 10.2 µg/L at pH 7.5, and HAMs reached a maximum concentration of 4.3 µg/L at pH 7.5.

At pH 7.5 THMs reached a high of 81.7 µg/L, HAA_5 reached a maximum of 29 µg/L, and HAA_9 reached a maximum concentration of 101.6 µg/L. Notably, in finished drinking water, the concentration of THMs exceeded the U.S. EPA MCL at pH 7.5. Overall, at circumneutral pH, HAAs were present at the highest concentration by mass, followed by THMs, HANs, and HAMs.

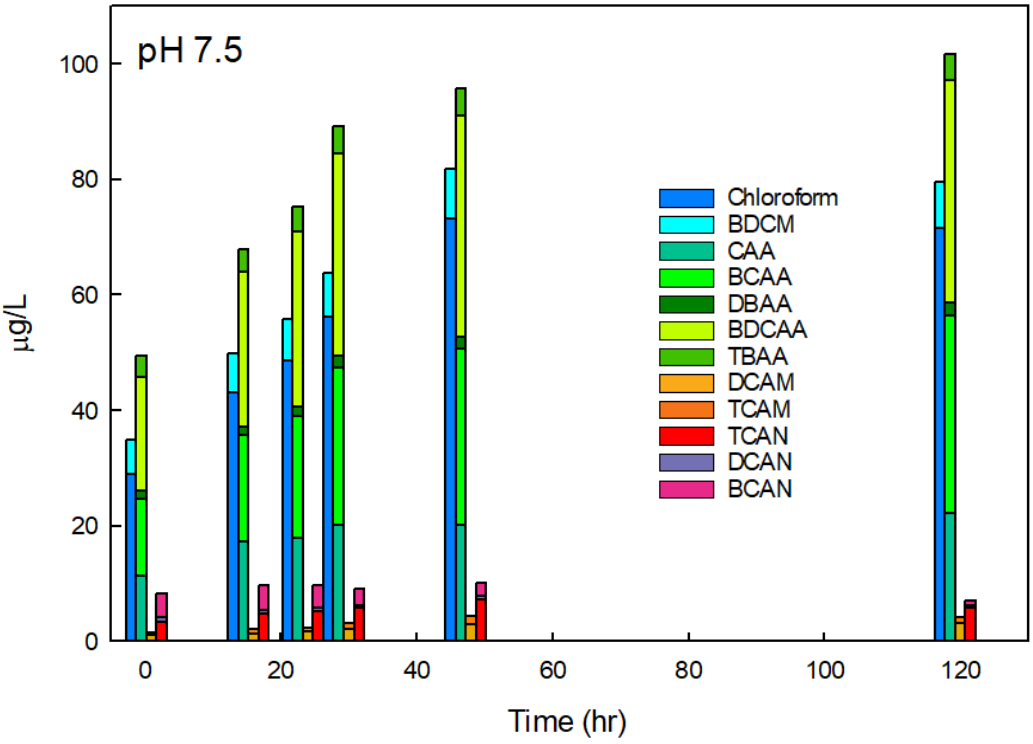

Figure 2.2: DBPs in finished drinking water at pH 7.5 at varying reaction durations. DBPs which were not present or present below the detection limits are not shown. The initial free chlorine concentration was 1.7 mg Cl_2/L. Data for 0-, 1-, and 24-hr time intervals were conducted in experimental triplicate, others are from single experiments. For experiments conducted in replicate, the coefficient of variation for the summation of THMs was always less than 0.03, always less than 0.17 for HAAs, always less than 0.22 for HAMs, and always less than 0.03 for HANs, accounting for error propagation of the individual DBPs measured in the replicates.

Effect of pH on DBP Formation and Degradation

HANs formed rapidly in the first hour following chlorine treatment; in all three pH groups, the 1-hour samples had HAN concentrations of approximately 8 µg/L. Comparing formation of DBPs across varying pH, there was a generally positive correlation between pH and formation of THMs and HAMs (Figure 2.3), but pH and total HANs were negatively correlated (Pearson correlation coefficients between pH and formation of different DBP classes are presented in Table B-2).

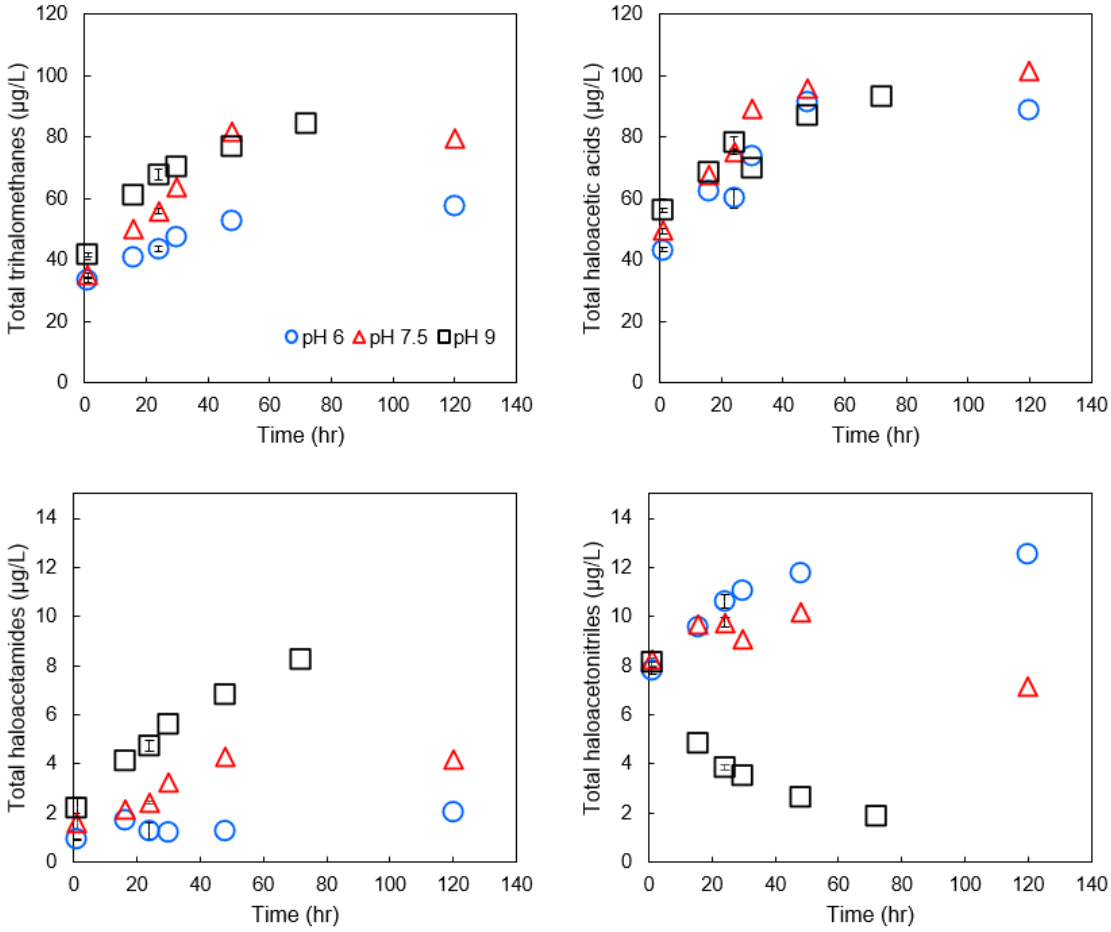

Figure 2.3: DBP formation across time by class at pH 6, 7.5, and 9 in finished drinking water. Error bars represent one standard deviation above and below the mean for samples measured in triplicate.

The greatest levels of THMs at pH 6 and 9 were 57.6 and 84.1 µg/L, respectively, and the latter would violate the EPA MCL for THMs. HAA_5 concentrations did not exceed the respective MCL at any pH. HAAs tended to be minimally impacted by pH with differences in formation across the three pH being generally <10% under the current experimental conditions. Maximum concentrations of total HAMs were 2.0 and 8.3 µg/L at pH 6 and 9. It is not clear why lower pH minimized net formation of HAMs, but one possibility is that the observed "formation" of HAMs is substantially influence by hydrolysis of HANs to HAMs. For example, at pH 9 total HANs were 8.2 µg/L after 1-hour, decreasing to 1.9 µg/L after 72 hours. At pH 6, the formation of HANs approximated the formation curves of other DBPs, thus hydrolysis likely plays a minimal role in acidic conditions. Although many HAMs are reported to have basic hydrolysis rate constants on the same order of magnitude as their HAN counterparts,[30,31] HAMs were not, on net, degraded in this study. Converting the difference in HAN concentration from the 1-hour sample to the 72-hour sample at pH 9 results in a loss of 2.38 µg/L (0.0165 µM) TCAN, 0.16 µg/L (0.0014 µM) DCAN, and 3.74 µg/L (0.0242 µM) BCAN. Considering the degradation mechanism where one mole of HAN hydrolyzes to 1 mole of HAM, the hydrolyzed HANs would form approximately 2.67 µg/L TCAM, 0.20 µg/L DCAM, and 7.03 µg/L BCAM. Formation of HAMs by the end of the experimental period included 1.22 µg/L TCAM and 7.06 µg/L DCAM. No BCAM was detected. The disparity between the theoretical and observed yield of HAMs may be due to differences in the degradation rate constants or formation rates of HANs and HAMs. While they have similar hydrolysis rate constants, the rate of formation of HAMs is not well examined and these results may indicate that HAMs have higher observed formation rates than

corresponding HANs at increased pH, offsetting the observed degradation in treated drinking waters over the current experimental timescale. HANs may also be degraded by the hypochlorite ion in a mechanism similar to hydrolysis, resulting in the formation of an N-chloro-HAM instead of a 2-chloro-HAM.[27] This separate pathway (Figure 2.4) may explain why the observed formation and degradation of HAMs deviates from conclusions drawn from models utilizing solely the hydroxide catalyzed degradation pathway, as only 2-chloro-HAMs were measured in this study.

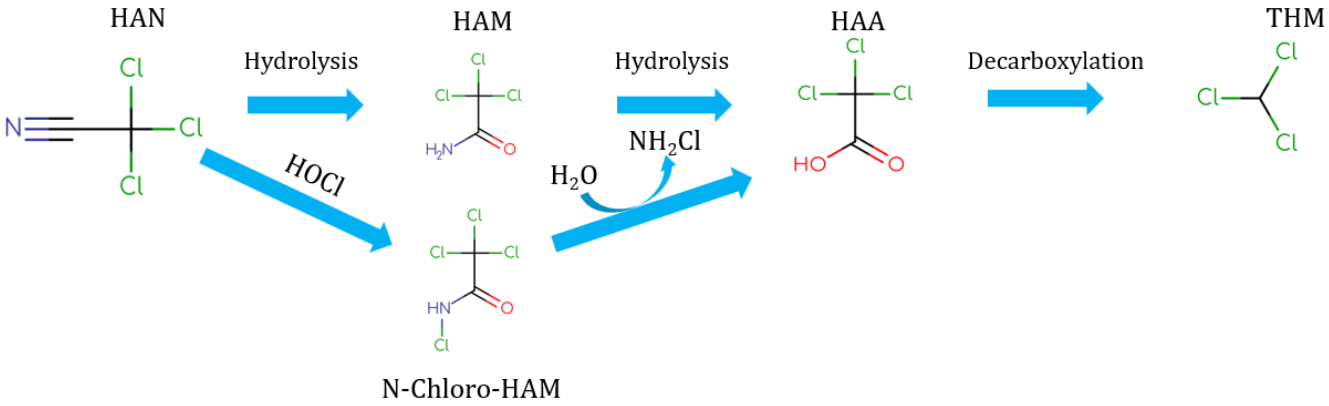

Figure 2.4: Proposed hydrolysis pathway including the hypochlorous acid chlorination pathway creating N-chloro-HAM as an intermediate to the HAA degradation product.

Degradation of HANs exceed formation substantially at increased pH, the only class of DBPs to do so under the pH and chlorine contact time investigated. Yu and Reckhow[27] and others[26] demonstrated that HANs are subject to nucleophilic attack by hydroxide ion at the nitrile carbon leading to hydrolysis and formation of the corresponding HAM and, upon further hydrolysis, a haloacetic acid. It is also possible that the HAA may further undergo decarboxylation to form a halomethane analogue.[32-34] While the chemical kinetic

model and rate constants proposed by Yu and Reckhow are useful for predicting hydrolysis of HANs in waters with fixed starting concentrations, a model incorporating formation kinetics in addition to degradation kinetics may be necessary for quantitative predictions of HAN concentrations in drinking water treatment and distribution systems.

Impact of pH on DBP-associated Summed Calculated Toxicity

To assess the relative impact of pH on DBP-associated toxicity, individual DBP concentrations were divided by their measured cytotoxicity and the results summed (i.e., calculated toxicity, Figure 2.5). At pH 6, calculated toxicity increases steadily throughout the entire experimental period. At pH 7.5, calculated toxicity increases for the first 16 to 24 hours then decreases steadily throughout the remainder of the experimental period. Holding water at pH 9 results in hydrolysis of HANs and a swift decrease in calculated toxicity, reaching its lowest point approximately 24 hours after treatment.

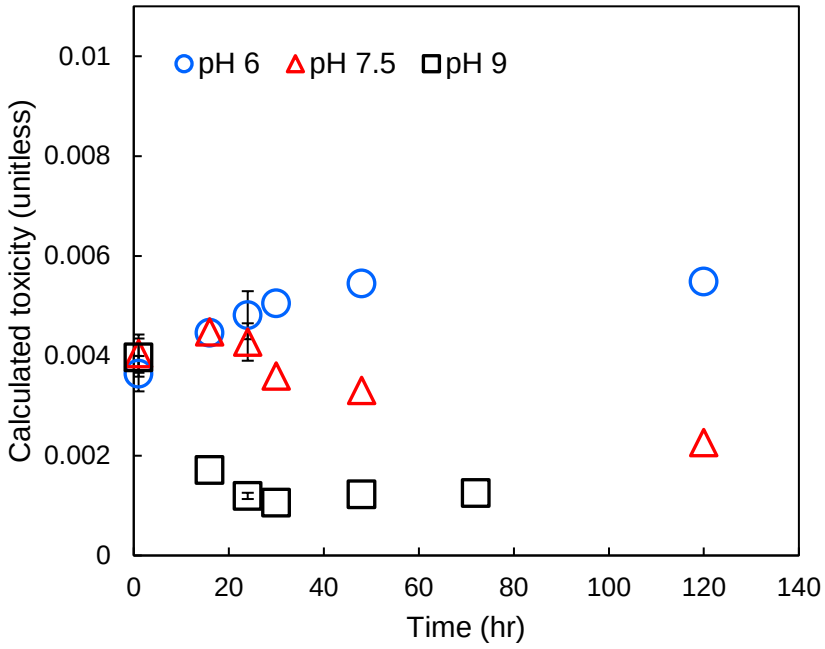

Figure 2.5: Calculated toxicity in finished drinking water at pH 6, 7.5 and 9. Errors bars show 1 standard deviation above and below the mean including propagated error from both analytical uncertainty and an assumed 12% uncertainty in the LC_{50} values.[13,27]

To understand which DBPs drive the observed changes in calculated toxicity at varying pH, the contribution to calculated toxicity by DBP class is shown in Figure 2.6. HANs were the primary drivers of DBP-associated toxicity at pH 6 and 7.5 in finished drinking water, accounting for as much as 85% of summed calculated toxicity. At pH 9 HANs were rapidly hydrolyzed to HAMs and HAAs, with HANs accounting for approximately 82% of initial calculated toxicity, then decreasing to 10% of calculated toxicity by the 72-hour sample. HAAs were the primary drivers of toxicity following HAN hydrolysis.

Although HAMs may be up to an order of magnitude more toxic than similarly substituted HAAs, HAMs accounted for only 0.1% to 2.6% of summed calculated

toxicity, attributable to their relatively low concentration at all pH (≤4% of total DBPs by mass). THMs accounted for up to 47% of total DBPs by mass in finished drinking water, but only contributed 0.7% to 5.6% to calculated toxicity across all time steps, representative of their relatively low toxicity. HAAs accounted for up to 58% of total DBPs by mass and 15% to 82% of calculated toxicity. The greatest contribution by HAAs was at pH 9 after the majority of HANs were hydrolyzed. Similarly, the lowest contribution by HANs was at pH 9 after the majority were hydrolyzed. HANs accounted for up to 9% of total DBPs by mass and 10% to 85% of summed calculated toxicity.

HANs represent the highest contribution to DBP-associated toxicity directly following treatment in finished drinking waters at circumneutral pH. Since HANs and other DBPs continue to form as water is in contact with chlorine disinfectant, methods that reduce formation of HANs throughout the entire distribution system, such as pH adjustment, will be most effective at lowering public exposure to DBP-associated toxicity.

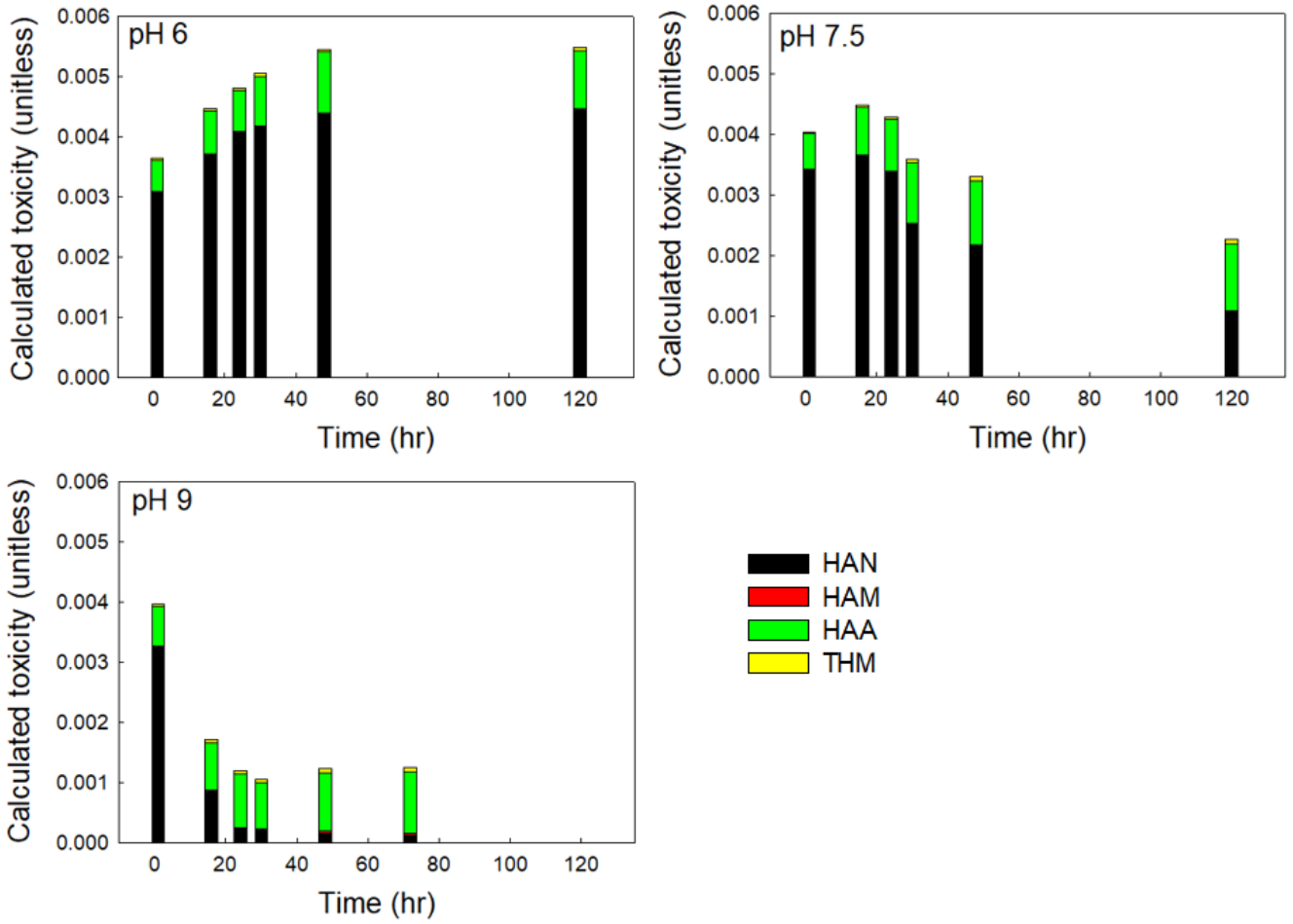

Figure 2.6: Toxicity contribution of each DBP class in finished drinking water at pH 6, 7.5, and 9. (CV always less than 0.1).

Confirmation of conclusions with NOM Isolates

To verify trends in DBP formation and calculated toxicity across varying NOM sources, formation potential experiments were conducted using IHSS NOM standards spiked at 3 mg C/L to pH buffered Milli-Q water. The spiked NOM was exposed to an initial addition of 9 mg Cl_2/L free chlorine. Figure 2.7 shows calculated toxicity over time from these experiments.

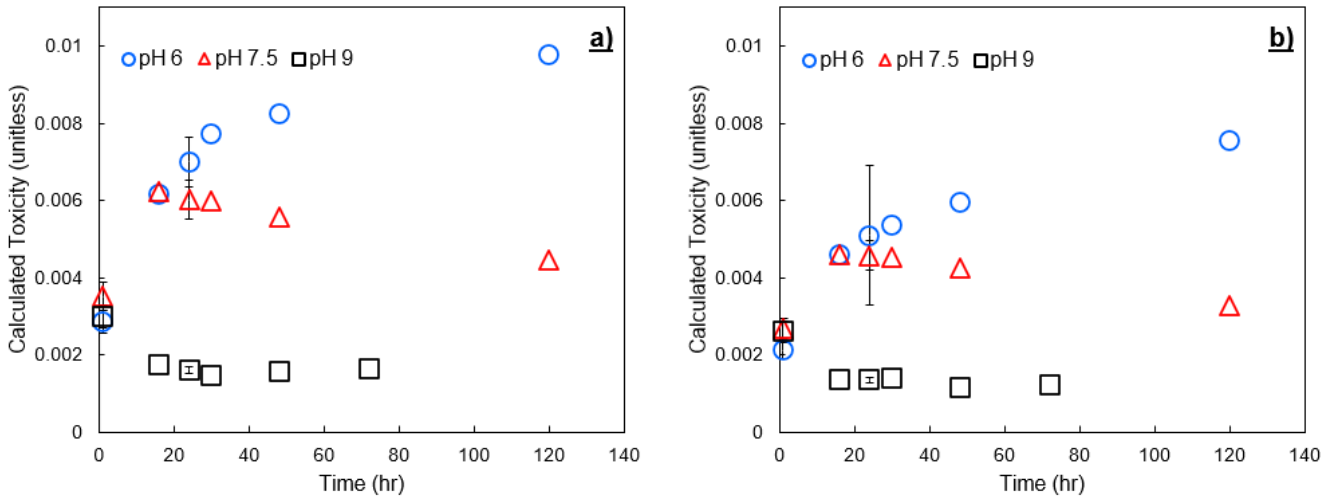

Figure 2.7: Calculated toxicity from formed DBPs at pH 6, 7.5, and 9 after chlorination of a) Suwannee River NOM FP Test, and b) Upper Mississippi River NOM FP. The chlorine dose was 9 mg Cl_2/L. Error bars show 1 standard deviation above and below the mean including propagated error from both analytical uncertainty and an assumed 12% uncertainty in the LC_{50} values.[15,29]

DBP-associated calculated toxicity from chlorination of NOM isolates had similar trends to that of finished drinking water; rapidly decreasing toxicity at pH 9 following HAN degradation closely. In all experiments, calculated toxicity at circumneutral pH increased for the first 16 to 24 hours following initial chlorine contact, then declined steadily throughout the remaining sampling period. pH 6 resulted in the greatest calculated toxicity at all time points due to the increased stability of HANs at low pH (i.e., decreased hydrolysis). Although increased pH reduced the calculated toxicity, the EPA MCL for THMs was again exceeded for SRNOM and UMRNOM. These samples have not been subject to traditional drinking water treatment methods and are therefore not representative of drinking water samples or subject to the MCL, but notably there is a

tradeoff between decreased calculated toxicity and increased potential to violate MCLs for regulated DBPs.

At pH 9, calculated toxicity was similar at the end of the experimental period for all samples in this research; 0.0013, 0.0012, and 0.0016 for finished drinking water, UMRNOM FP test, and SRNOM FP test, respectively. However, at the end of the experimental period, calculated toxicity at pH 7.5 increased across the same samples; 0.0023, 0.0033, and 0.0044, respectively. At pH 6, this phenomenon was more pronounced; 0.0055, 0.0075, and 0.0098, respectively. The notable similarity in calculated toxicity across different NOM sources at pH 9 suggests that DBP-associated toxicity at the tap may be similar, regardless of source water quality or chlorine dose, when higher distribution pH is utilized.

Overall, concentrations of HANs decreased with increasing pH across all source waters and chlorine doses, leading to drastic decreases in DBP-associated toxicity at early, middle, and late-stage distribution time intervals, regardless of simultaneous changes in concentrations of THMs, HAAs, and HAMs.

2.5 Conclusions

HANs form rapidly (i.e., hours) following chlorine addition, and pH is a crucial factor in determining subsequent persistence in treated drinking waters. While lower pH creates an environment where HANs are stable, increasing distribution system pH may quickly lead to degradation of these compounds through based catalyzed hydrolysis. At pH 9, the

ability of hydroxide ions to quickly destroy HANs in finished drinking water was demonstrated. However, any increase in pH above 7.5 will cause HAN hydrolysis to supersede formation. In turn, higher levels of HAMs, HAAs, and THMs are created as the products of this degradation, resulting in an overall decrease in DBP-associated toxicity. One caveat of this method is that limits for THMs and HAA_5 may be violated as a consequence of driving HANs, an unregulated but highly toxic class of DBPs, to lower hydrolysis products that are regulated by the US EPA, even though overall toxicity is decreased as a result. Another caveat inherent in this approach is that only measured compounds are accounted for, if other known or unknown DBPs are not included, conclusions from the calculated toxicity method could be skewed.[15]

Previously published kinetic models do not consider simultaneous formation and degradation of HANs that occurs in real treated drinking water. Therefore, further research into the kinetic formation and degradation of HANs is necessary before quantitative predictions may be applied in drinking water treatment and distribution systems.

2.6 Practical Implications and Recommendations

Reduction of DBP-associated toxicity by increasing distribution system pH comes with several inherent caveats. These include the increased likelihood of violating the EPA MCL for THMs at higher pH, concerns of scaling in the drinking water distribution system, and effective chlorine disinfection at higher pH. Concerns for corrosivity or scaling may be addressed using the Langelier Saturation Index (LSI). Important

parameters for the LSI include pH, total dissolved solids (TDS), temperature, calcium, and alkalinity.

$$LSI = pH - pH_s$$

Where, $$pH_s = (9.3 + A + B) - (C + D)$$

And, $$A = (\log[TDS] - 1)/10$$

$$B = -13.12 * \log(temperature) + 34.55$$

$$C = \log[Ca^{2+}] - 0.4$$

$$D = \log[alkalinity]$$

Equation 2.1: Equation used to calculate the Langelier Saturation Index. Units of TDS are mg/L, temperature in degrees Kelvin, Ca^{2+} and alkalinity in mg/L as $CaCO_3$.

The Langelier Saturation Index is an equilibrium model that may be used as an indicator of the saturation of water with respect to calcium carbonate. To avoid scaling or corrosivity in drinking water distribution systems, water should be near an LSI of 0, with generally acceptable values being between -0.3 and 0.3. If the LSI is positive, the distributed water will tend to deposit calcium carbonate in the piping (scaling), and if the LSI is negative, the water is undersaturated and will tend to be corrosive, taking up carbonate from the distribution system.

Averaging water quality parameters from a local drinking water quality database gives an average TDS of 207.3 mg/L, an average temperature of 14.6 °C (287.6 Kelvin), an average [Ca^{2+}] of 28.6 mg/L as $CaCO_3$, and an average alkalinity of 90.6 mg/L as

$CaCO_3$. A higher pH generally leads to the tendency of water to precipitate calcium carbonate in the distribution system, assuming other parameters are held constant. To achieve an LSI value between -0.3 and 0.3 using the water quality parameter averages above, distribution pH should be between 8.4 and 9. Since the local source water in Reno, NV is only moderately hard, it may be possible to distribute water at an increased pH of 8.4-9 to quickly rid the system of harmful HANs without the drawback of scaling in the distribution system. However, in systems with harder source water containing higher concentrations of Ca^{2+} and alkalinity, particularly those deriving their water from groundwater sources, a softening step may be needed in the treatment train to utilize an increased distribution pH for lowering of HANs through base-catalyzed hydrolysis.

In order to meet disinfection requirements recommended by the US EPA, it is advised to only adjust pH after disinfection in the clear well. If pH is adjusted prior to chlorine contact at the treatment plant, a higher chlorine dose or a longer contact time may be necessary to achieve the desired concentration*time (CT) value. As the pka of HOCl is 7.53, OCl^-, a less potent disinfectant, would dominate at increased pH. Post-chlorination pH adjustment could achieve HAN mitigation without negatively impacting disinfection at the treatment plant.

Another caveat inherent in increasing distribution system pH to reduce HAN levels in delivered drinking water is the tradeoff between decreased HAN levels and increased tendency for THM concentrations to exceed the 80 µg/L EPA MCL. Unless the EPA moves to a toxicity-based system for regulating DBPs instead of the current concentration-based system, treatment plants with higher organic matter loadings in their

source waters may have trouble meeting EPA regulations for THMs if utilizing an increased distribution pH to lower overall DBP-associated toxicity. To solve this problem, such treatment plants could use a number of processes in addition to increasing distribution pH such as granular activated carbon (GAC) filtration prior to disinfection for lowering of dissolved organic matter (DOC) concentrations, changing disinfection processes including the addition of ultraviolet (UV) treatment or switching to chloramine disinfection, utilizing a lower initial chlorine residual coupled with chlorine booster stations throughout the distribution system, or use of aeration systems at strategic points throughout the distribution system for reduction of THMs. Drinking water regulations require a minimum of 1.0 mg/L combined chlorine residual or 0.2 mg/L of free chlorine residual maintained throughout the entire distribution system. Utilizing a lower initial chlorine concentration while still meeting the CT requirements for disinfection and boosting chlorine in the system to meet requirements for disinfectant residual later may lead to lower levels of DBPs formed throughout the entire system. Utilizing UV disinfection prior to chlorination may help mitigate DBP formation post-chlorination by allowing the plant to utilize a lower concentration of chlorine for chemical disinfection while still gaining sufficient inactivation credits for *Cryptosporidium* and *Giardia lamblia*. If a treatment plant intends to switch from chlorine to chloramine disinfection to mitigate THM and HAA formation, preliminary research should be undertaken to ensure that NDMA precursors are not present in the source water and that nitrification will not be a significant issue in the treatment and distribution system with added chloramines. Using aeration systems throughout the distribution system at strategic points such as storage tanks with long residence times may also aid in lowering concentrations of

regulated DBPs as THMs are low molecular weight, relatively hydrophobic compounds that may be removed by sparging large amounts of gas through a water column or storage tank.

2.7 Acknowledgements

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Chapter 3: Formation of Nitrogenous Disinfection Byproducts During Point-of-Use Water Treatment

3.1 Background and Justification of Research

Point-of-use (POU) water treatment includes any water treatment devices, technologies, or chemical processes that treat source water directly at the time and location of use. POU water treatments are decentralized and are generally used in applications where there is no centralized water treatment system available such as during outdoor recreational trips (backpacking, cycle touring, etc.), in rural areas or developing nations, temporary housing situations such as refugee camps, and emergency response situations. POU water treatment may include treatment with a pre-packaged oxidative disinfectant, filtration, water softening, or pH adjustment, among others. Pre-packaged oxidative disinfectants are used to disinfect source water in many emergency response situations due to their low cost, chemical stability, light weight, and long shelf life.[35]

While the formation of DBPs has been studied extensively in centralized drinking water treatment and distribution systems, the scientific literature is greatly lacking data on the formation of DBPs, particularly N-DBPs, with regards to POU treatment using pre-packaged oxidative disinfectants. Understanding how different pre-packaged oxidative POU water treatments lead to DBP formation is important for mitigating toxicity exposure to consumers, many times those who are in emergency situations and have no other options.

This chapter describes experimentation partially conducted by an undergraduate student, Jackie Kittridge, who completed approximately 50% of the laboratory work.

3.2 Methods

To understand how different pre-packaged oxidative POU water treatments contribute to formation of DBPs, an experimental plan was devised to compare four commercially available disinfectant tablets. The treatment tablets include *Aquatabs* (sodium dichloroisocyanurate (NaDCC) 17.4%), *Katadyn Micropur MP1* (sodium chlorite 6.4%, NaDCC 1%), *Aquamira* (chlorine dioxide 2%, phosphoric acid 5%), and *Potable Aqua* (tetraglycine hydroperiodide 16.7%).

	Aquatabs	Katadyn MP1	Aquamira	Potable aqua
Control (treatment but no NOM)	✓	✗	✗	✗
SRNOM 2.5 mg C/L	✓	✗	✗	✗
Low TOC Alpine Lake/Stream	✗	✗	✗	✗

Table 3.1: Experimental plan for comparison of DBP formation using different POU water treatments. Checks indicate completion of experiment while crosses indicate the experiment is recommended for future research but has not been completed.

For the experiment involving *Aquatabs* and Suwannee River NOM (SRNOM), 2.5 mg C/L of SRNOM was spiked into Milli-Q water and directions were followed on the packaging to treat approximately 3 liters of water. Upon completion of the treatment step (10 minutes mixing, 30 minutes disinfection time), the treated water was transferred

to headspace free reaction vials and the 0-hour sample quenched and preserved for later extraction and quantification of DBPs. The remaining were allowed to react for intervals of 1-, 19-, 24-, 48-, and 96-hours. Results are shown in section 3.3.

3.3 POU Water Treatment Preliminary Results and Discussion

Preliminary results from formation potential experiments using *Aquatabs* (NaDCC 17.4%) show generation of various C- and N-DBPs including chloroform, bromodichloromethane, chloroacetic acid, bromochloroacetic acid, bromodichloroacetic acid, trichloroacetamide, dichloroacetamide, trichloroacetonitrile, dichloroacetonitrile, and bromochloroacetonitrile. Figure 3.1 shows formation of DBPs by class. The highest and second highest individual DBPs by mass were bromodichloroacetic acid and chloroform at 110 µg/L and 95 µg/L, respectively, after 96 hours of reaction time. The highest concentration of an individual haloacetonitrile was bromochloroacetonitrile at 8.9 µg/L at the 48-hour sample interval. Trichloroacetamide and dichloroacetamide reached maximum concentrations of 1.5 µg/L and 6.1 µg/L, respectively. Upon completion of the recommended disinfection time, the initial free chlorine concentration was 12.3 mg Cl_2/L. With conventional drinking water treatment generally containing a chlorine residual between 1-2 mg Cl_2/L following disinfection, a concentration of free chlorine approximately 6-12 times this amount during POU treatment led to relatively high concentrations of each class of DBPs. The high initial free chlorine concentration during this POU treatment may be because POU oxidative disinfectants are manufactured for a broad range of source waters and treatment situations. While the high chlorine dose

would disinfect some of the most pathogen laden source water, the risk for formation of high levels of DBPs is increased dramatically. One possible solution to this problem is to treat small batches of water more often, consuming water within hours after treatment, thus reducing the reaction period in which DBPs may form.

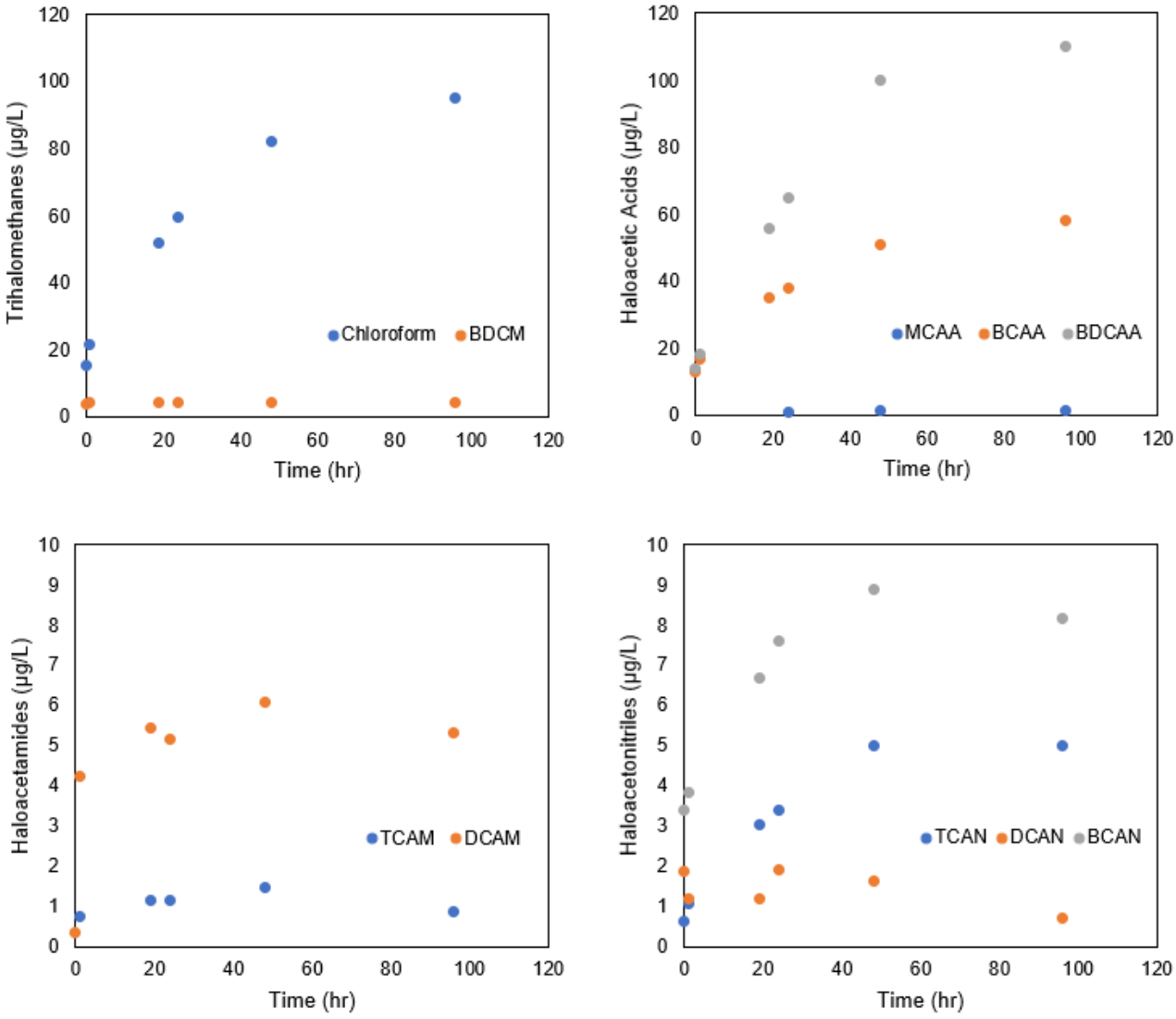

Figure 3.1: Speciation of DBPs by class during the 4-day formation potential experiment using *Aquatabs* (NaDCC 17.4%) and 2.5 mg C/L Suwannee River NOM isolate. Initial free chlorine concentration was 12.3 mg Cl_2/L.

Recommendations for future research

To quantitatively compare the differences between POU water treatments and corresponding DBP formation potentials, the experimental plan outlined in Table 3.1, or a similar set of experiments should be completed using experimental triplicates to ensure accuracy, and care should be taken to replicate and record water quality parameters across all POU treatments. Water quality parameters of interest include TOC, residual free chlorine concentration, temperature, and bromide ion concentration. pH is of particular importance as haloacetonitriles and other DBPs are sensitive to changes in pH (described in detail in the chapter 2). Because of this sensitivity, it is recommended to buffer all experimental waters to the same pH prior to treatment with POU disinfectants.

Chapter 4: Summary, Conclusions, and Recommendations for Future Work

4.1 Summary and Conclusions

This thesis includes work encompassing several components relevant to environmental engineering including drinking water quality and distribution problems, aquifer storage and recovery, multivariate modeling of DBP formation, and a novel method for treatment of N-DBPs of priority concern; two HANs being included in the draft CCL 5 published by the EPA in July 2021.

It was found that holding finished drinking water at pH 9 decreased the DBP-associated toxicity by 45% compared to pH 7.5, and 77% compared to pH 6. This decrease in toxicity at increased pH is largely due to hydrolysis of haloacetonitriles, an unregulated but highly toxic class of N-DBPs. This work has proven a simple method for reducing public exposure to N-DBPs; distributing water above pH 7.5 results in net degradation of HANs within 24 hours. Distributing water at approximately pH 9 would result in net degradation of HANs within hours, providing even consumers in very close proximity to the treatment plant water with lowered concentrations of HANs.

In Chapter 3, research with the commercially available POU water treatment tablets *Aquatabs*, with experimental support from Jackie Kittridge, found that high levels of all measured classes of DBPs were formed, most likely due to the high concentration of free chlorine resulting from POU treatment. It is notable that the EPA limit for THMs in a conventional water treatment and distribution scenario would have been violated as a

result of this treatment. However, POU water treatments do not fall under the category of public water systems and thus DBPs produced during these processes are not currently regulated under the disinfection byproducts group by the U.S. EPA national primary drinking water regulations.[4]

4.2 Recommendations for Future Work

To understand how the results found in Chapter 2 may be affected by different treatment processes, it is advised to conduct similar HAN hydrolysis experiments across a range of source waters with differing treatments to understand how unit processes other than the conventional coagulation, flocculation, sedimentation, filtration, and chlorine disinfection influence precursors and other water quality factors relevant to HAN formation and degradation. Additionally, other DBPs of increasing interest and high toxicity such as nitrosamines and iodinated trihalomethanes may be included in future studies and compared with the influence of HANs in weighted DBP-associated calculated toxicity.

To prepare a manuscript for publication comparing the DBP formation and DBP-associated toxicity between different POU water treatments the research plan outlined in Table 3.1 should be completed using experimental triplicates for all sampling intervals to ensure accuracy and understand analytical error. Source waters should be buffered to circumneutral pH prior to treatment with the POU oxidants for accurate comparison since the formation and degradation of many DBPs are sensitive to changes in pH. In addition to the 28 compounds for which quantification methods using GC-ECD were developed,

measuring iodinated DBPs would be ideal for this experimentation, since one of the suggested POU treatments (*Potable Aqua*) includes tetraglycine hydroperiodide as the active ingredient.

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Appendix A: GC-ECD Analytical Method Development

Quantification methods for halogenated DBPs using the GC-ECD were developed during the Spring of 2021. For THMs and HAAs, the extraction, derivatization, and quantification protocols are based on US EPA methods 551 and 552. The extraction and quantification of HAMs and HANs is based on EPA method 551, with additional trial and error of different GC columns, oven temperature programs, ramp rates, flow rates, and extraction solvents for separation of chromatographic peaks and optimization of the quantification methods for use in water quality analysis at UNR. The final methods are described below along with tables indicating the approximate retention times of the 28 compounds for which quantification methods were developed. Method detection limits (MDLs) for individual THMs and HAAs are approximately 1 µg/L and MDLs for HAMs and HANs are approximately 0.25 µg/L.

Trihalomethane, Haloacetonitrile, and Haloacetamide Extraction and Quantification Protocol

Adapted from EPA Method 551.1

Reagent and IS Prep:

1. Free Chlorine Quenching Solution: 0.5 M Ascorbic Acid (~88 g/L)
2. Internal Standard/Extraction Solvent: Prepare 1 ug/mL 1,2-dibromopropane in MTBE.
3. Calibration Standards: Prepare 1-100 ppm for THMs (usually 1, 5, 10, 25, 50, and 100 ppm), 0.25 to 25 ppm for HANs and HAMs (0.25, 0.5, 1, 5, 10, 25 ppm) in methanol. These standards will be diluted 1000x in water to ppb level.
4. Bake all vials and glassware associated with THM/HAN experiments and extraction process. (Don't bake volumetric glassware - wash, and rinse well).

Calibration, Sample Extraction, and Analysis:

Calibration:

1. Add 30 mL reagent water into 40 mL vial.
2. Add 75 µL 0.5 M ascorbic acid and 100 µL 37% (12 N) HCl to each.
3. Add 30 µL of appropriate ppm standard (1, 5, 10, 25, 50, 100 ppm).
4. Follow procedure for sample extraction. New calibration curves to be run biweekly to monthly on GC-ECD. Standard checks to be run every 10 samples to ensure detector response remains constant.

Sample Extraction:

1. Use 50 mL graduated cylinder to measure 30 mL of sample and pour into 40 mL extraction vial. Rinse graduated cylinder with MilliQ water in between each sample.
2. Add 3 mL MTBE with internal standard.
3. Add 10 g Na_2SO_4, cap quickly and shake. Place vials horizontally to prevent Na_2SO_4 from clumping on bottom of vial.
4. Shake for 30 min at 250 rpm on shake table.
5. Let samples sit ~2 minutes upright until MTBE layer is completely separated from aqueous layer.
6. Use 1 mL airtight syringe and needle to remove ~1 mL of upper organic layer from vial through the septa. Take care not to touch or take up water from lower aqueous layer. Do not remove the cap during this step as THMs could be lost due to volatility. Move ~1 mL MTBE from extraction vial into 2 mL GC vials. Run on GC-ECD – ECD method: HP5 THM HAN HAM

   <u>GC-ECD Method Parameters:</u>

   1. Inlet:

      200°C

      Split Mode – Split Ratio 10:1, Split Flow 9 mL/min

      Gas Saver: On – 20 mL/min after 3 min
   2. Column: HP-5, 30 m x 320 µm x 0.25 µm

      Flow: 0.9 mL/min He

      Avg Velocity: 16.647 cm/sec

      Constant Flow

Post Run: 2.3922 mL/min

3. Oven:

   Equilibration Time: 0.5 min

   Max Oven Temp: 325°C

   Oven Program:

   Initial 35°C, Hold 9 min

   Ramp to 40°C at 1°C/min and hold for 3 min

   Ramp to 120°C at 6°C/min and hold for 3 min

   Ramp to 250°C at 30°C/min and hold for 3 min

4. Detector:

   ECD, 290°C

   Makeup flow 25 mL/min N_2

   Constant Makeup Flow

   Total run time: 40.7 min + oven cooldown (~50 min total)

Compound	Approximate Retention Time (min)
chloroform	4.76
chloroacetonitrile	6.12
bromodichloromethane	6.97
trichloroacetonitrile	7.46
dichloroacetonitrile	8.12
bromoacetonitrile	10.45
1,1-dichloroacetone	10.95
chlorodibromomethane	11.83
trichloronitromethane	13.67
bromochloroacetonitrile	16.25
1,2-dibromopropane (IS)	17.15
bromoform	19.74
1,1,1-trichloroacetone	21.88
2-chloroacetamide	22.1
dibromoacetonitrile	22.76
2-bromoacetamide	25.63
2,2-dichloroacetamide	27.195
2,2-dibromoacetamide	29.9
trichloroacetamide	31.06
bromochloroacetamide	32.5

Table A-1: List of compounds quantified on GC-ECD using HP-5 column and their retention times.

Haloacetic Acid Extraction, Derivatization, and Quantification Protocol

Adapted from EPA Method 552.3

Reagent and IS Prep:

1. Dechlorinating solution of 0.5 M (88g /L) ascorbic acid in reagent water
2. 20 ug/mL 2-bromobuyric acid in MTBE (Surrogate Standard)
3. 1 ug/mL 1,2,3-trichloropropane in MTBE (Internal Standard in Extraction Solvent)
4. 5% sulfuric acid in methanol
5. Saturated sodium bicarbonate solution (~96 g/L $NaHCO_3$)
6. Prepare 1-100 ppm calibration standards of 9 Haloacetic Acids mix in MTBE.
7. Wash and bake all vials and glassware associated with HAA extraction and derivatization.

Calibration, Sample Extraction, and Analysis:

Calibration:

1. Add 30 mL reagent water into 40 mL vial.
2. Add 75 µL 0.5 M ascorbic acid and 100 µL 37% (12 N) HCl to each.
3. Add 30 µL of appropriate ppm standard (1, 5, 10, 25, 50, 100 ppm).
4. Follow procedure for sample extraction. New calibration curves to be run biweekly to monthly on GC-ECD. Standard checks to be run every 10 samples to ensure detector response remains constant.

Sample Extraction:

5. Use 50 mL graduated cylinder to measure 30 mL of sample and pour into 40 mL extraction vial. Rinse graduated cylinder with MilliQ water in between each sample.
6. Add 20 µL surrogate stock solution to each 40mL extraction vial containing 30 mL of sample.
7. Add 1.5 mL concentrated H_2SO_4 to each vial.
8. Add 3 mL MTBE with internal standard.
9. Add 10 g Na_2SO_4, cap vials quickly and shake, lay horizontally until all samples complete to prevent Na_2SO_4 from clumping on bottom of vial.
10. Shake for 30 min at 250 rpm on shake table.
11. Make acidic methanol solution (5% v/v H_2SO_4). Place 2 mL in 20 mL vials (TOC-sized vials).
12. Once done shaking, remove 1 mL MTBE from 40 mL vials using 1000 µL pipette and place into 20 mL vials containing acidic methanol and cap tightly. Ensure none of the lower aqueous layer is transferred to acidic methanol vials.
13. Place in water bath at 50°C for 2 hours (esterification step).
14. Make saturated sol. of $NaHCO_3$. Once samples are done in water bath, allow to cool and add 5 mL $NaHCO_3$ solution to each vial.
15. Add 1 mL pure MTBE (No IS) to each vial. Shake for 2 minutes.
16. Remove ~1 mL top organic layer and pipette into GC vials and analyze on GC-ECD. Take care not to touch or take up any of the lower aqueous layer.

    GC-ECD Method: HAA Method for DB-1 column Nikolaou et al 2002

GC-ECD Method Parameters:

1. Inlet:

   175°C

   Split Mode – Splitless, Pure Flow to Split Vent: 30 mL/min at 0.75 min

   Gas Saver: On – 20 mL/min after 3 min

2. Column: DB-1, 30 m x 250 µm x 1 µm

   Flow: 1.6 mL/min He

   Avg Velocity: 35.053 cm/sec

   Constant Flow

   Post Run: 2.3922 mL/min

3. Oven:

   Equilibration Time: 0.5 min

   Max Oven Temp: 325°C

   Oven Program:

   Initial 35°C, Hold 10 min

   Ramp to 40°C at 1°C/min and hold for 5 min

   Ramp to 110°C at 2.5°C/min and hold for 3 min

   Ramp to 220°C at 20°C/min and hold for 2 min

4. Detector:

   ECD, 300°C

   Makeup flow 25 mL/min N_2

   Constant Makeup Flow

   Total run time: 58.5 min + oven cooldown (~67 min total)

Compound	Approximate Retention Time (min)
MCAA	21.57
MBAA	31.54
DCAA	34.46
BCAA	35.9
TCAA	39.91
Trichloropropane (IS)	42.49
DBAA	42.852
BDCAA	43.34
2-Bromobutyric Acid (SS)	45.52
CDBAA	48.94
TBAA	50.2

Table A-2: List of haloacetic acids quantified on GC-ECD using DB-1 column and their retention times.

Appendix B: Supplementary Information for Reduction of Disinfection Byproduct-associated Toxicity by Adjustment of Distribution System pH

NTU	0.02
TSS (mg/L)	BDL
pH	7.9
Alkalinity (mg/L as $CaCO_3$)	59.3
TOC (mg/L)	1.61
ORP (mV)	618
FCl_2 (mg/L Cl_2)	1.72
TCl_2 (mg/L Cl_2)	1.8
NH_2Cl (mg/L Cl_2)	BDL
NH_3 (mg/L N)	BDL
NO_2^- (mg/L N)	BDL
NO_3^- (mg/L N)	0.764
Br^- (mg/L)	BDL
Chloride (mg/L)	20.48
Sulfate (mg/L)	6.60
TN (mg/L)	0.157

Table B-1: Water quality parameters for finished drinking water.

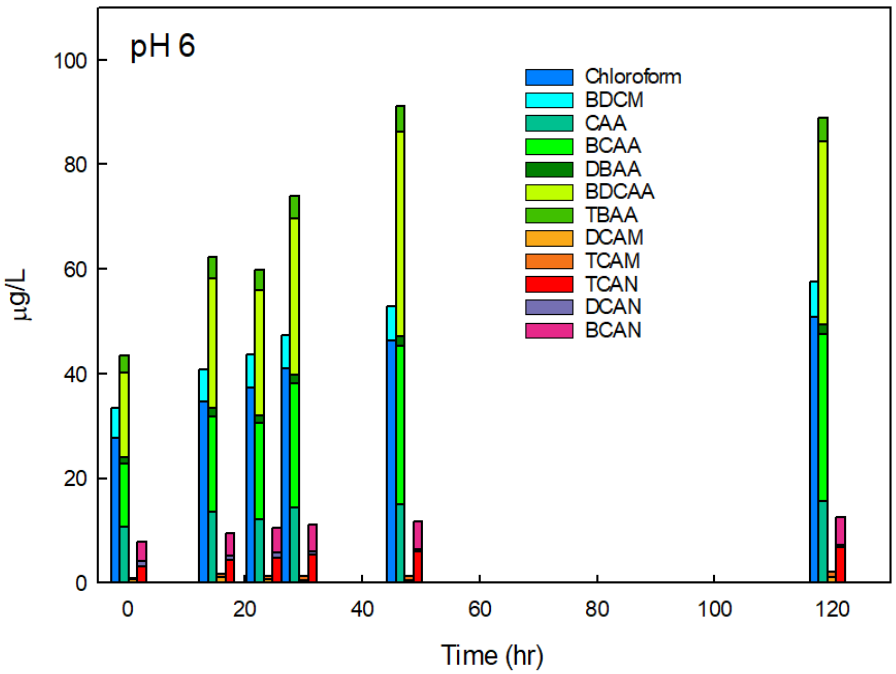

Figure B-1: DBPs in finished drinking water at pH 6 stacked by common functional group.

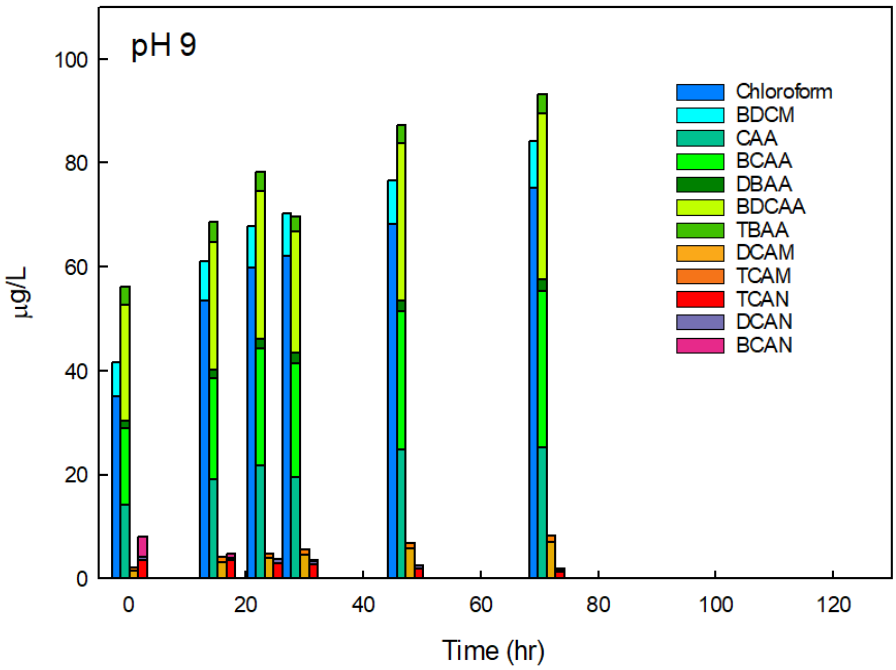

Figure B-2: DBPs in finished drinking water at pH 9 stacked by common functional group.

1-hour

pH	THM	HAA	HAM	HAN
6	33.52	43.36	0.95	7.77
7.5	34.88	49.39	1.57	8.23
9	41.61	56.14	2.22	8.15
Pearson R	**0.93**	**1.00**	**1.00**	**0.77**

16-hour

pH	THM	HAA	HAM	HAN
6	40.87	62.21	1.70	9.57
7.5	49.86	67.79	2.14	9.69
9	61.16	68.56	4.14	4.83
Pearson R	**1.00**	**0.92**	**0.94**	**-0.85**

24-hour

pH	THM	HAA	HAM	HAN
6	43.59	59.96	1.27	10.61
7.5	55.77	75.14	2.42	9.75
9	67.79	78.27	4.74	3.86
Pearson R	**1.00**	**0.93**	**0.98**	**-0.92**

30-hour

pH	THM	HAA	HAM	HAN
6	47.35	73.86	1.21	11.07
7.5	63.66	89.16	3.20	9.09
9	70.34	69.62	5.60	3.49
Pearson R	**0.97**	**-0.21**	**1.00**	**-0.96**

48-hour

pH	THM	HAA	HAM	HAN
6	52.98	91.18	1.25	11.79
7.5	81.70	95.65	4.30	10.15
9	76.65	87.32	6.83	2.63
Pearson R	**0.77**	**-0.46**	**1.00**	**-0.94**

Table B-2: Pearson correlations between pH and different classes of DBPs in finished drinking water.

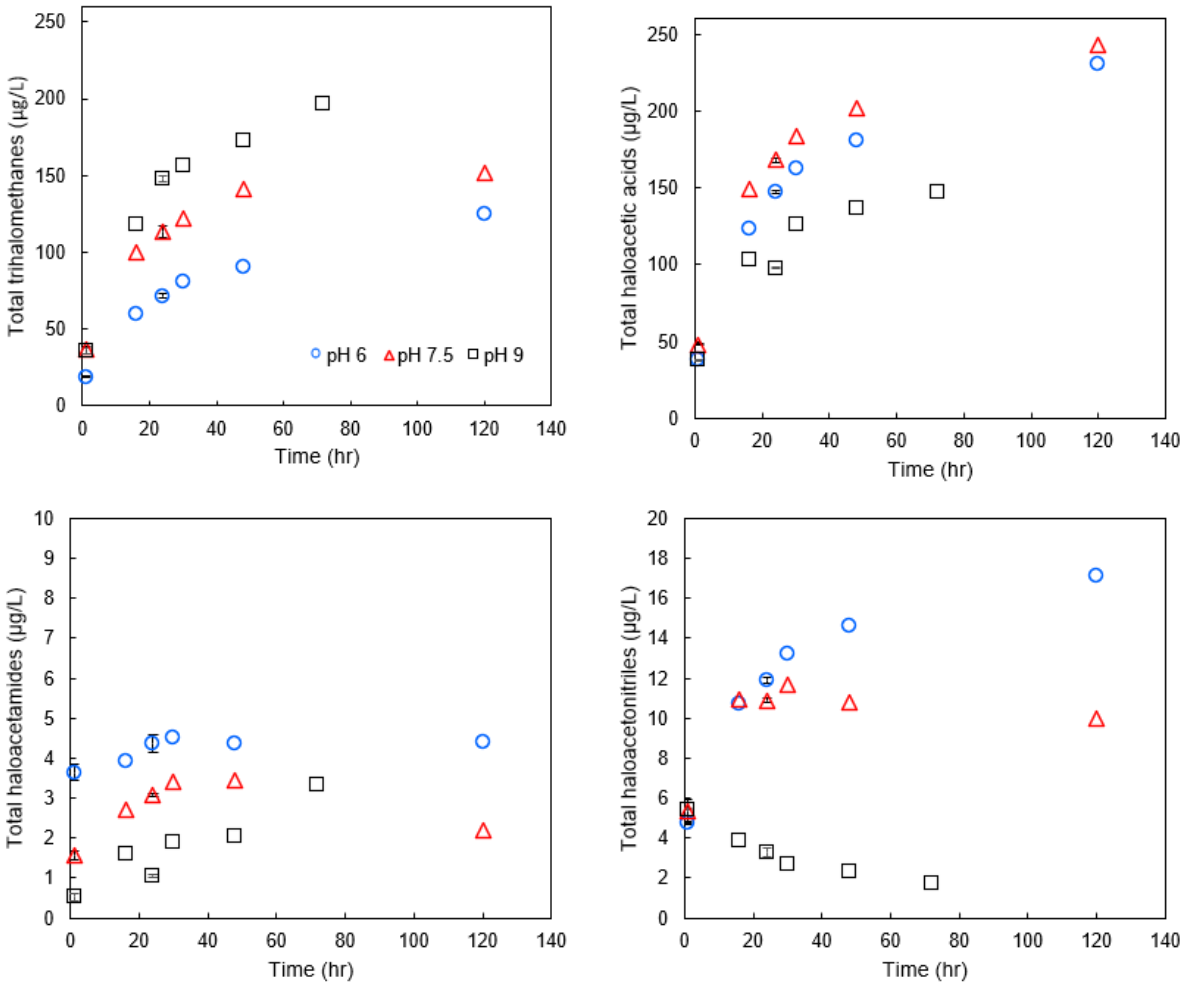

Figure B-3: DBP formation over time by class at pH 6, 7.5, and 9 for Suwannee River NOM FP Test.

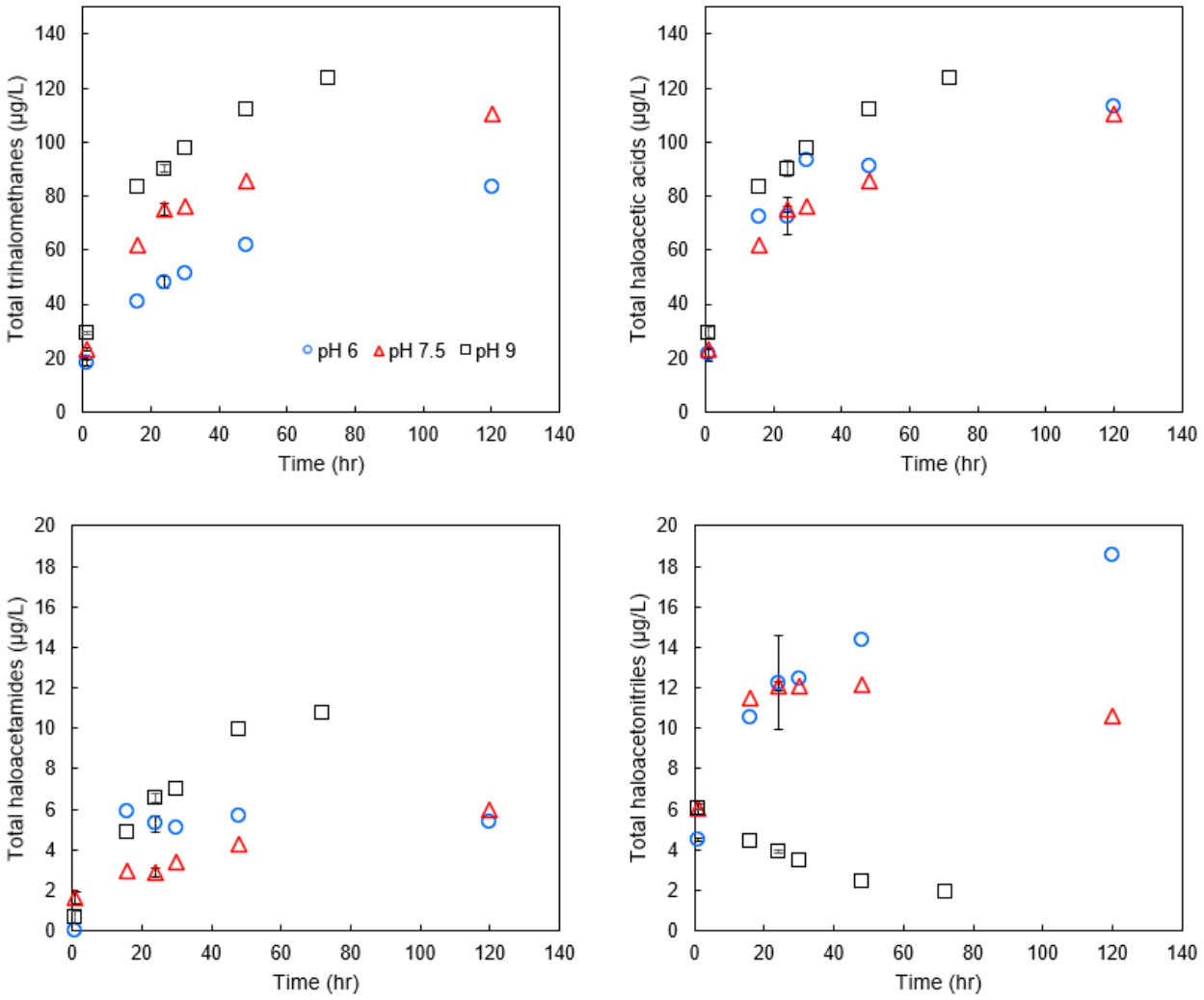

Figure B-4: DBP formation over time by class at pH 6, 7.5, and 9 for Upper Mississippi River NOM FP Test.