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Impact of water heating on disinfection byproducts concentration

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IMPACT OF WATER HEATERING ON DISINFECTION BYPRODUCT CONCENTRATIONS

A Dissertation Presented

by

BONING LIU

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DEDICATION

To the memory of my beloved father, Zhongchen Liu

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ABSTRACT

IMPACT OF WATER HEATING ON DBP CONCENTRATIONS

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Drinking water disinfection byproducts (DBP) are a group of inorganic and organic compounds formed during water disinfection. Epidemiologic studies suggest an association between rectal, and colon cancer and exposure to DBPs in chlorinated surface water. Therefore, DBPs are a growing public health concern; one that has been mitigated by multiple regulations of US Environmental Protection Agency (EPA) including the Stage 2 Disinfectants and Disinfection Byproducts Rule (Stage 2 DBPR). Tremendous efforts and cost have been spent on controlling DBPs in drinking water; however, human exposure has been poorly characterized. In addition to ingestion exposure, inhalation and dermal absorption during showering for example could also be significant exposure pathways. This dissertation focuses on investigating DBP formation and degradation in heated water (~50^{o}C) in both lab simulated tests and field studies.

The first objective of this dissertation was to investigate the temporal variability of regulated DBPs and non-regulated DBPs in cold and hot tap water at a residential home, in a water plant and in a simulated distribution system test. The results showed that

the residence time of water in hot water tanks plays an important role on the formation and degradation of DBPs in the hot water plumbing. There was no obvious difference between the concentrations of TCAA (trichloroacetic acid) in long-heated hot tap water and cold tap water. The terminal DBPs for cold and hot tap water were measured and compared to the instantaneous DBP formation in cold and hot tap water. The heating of tap water in the water tank was found to increase the extent of THM formation.

The second objective of this dissertation was to investigate the impact of heating scenarios on the formation and degradation of DBPs. A field study involving homes equipped with either tankless heaters or tank heaters was conducted. The concentrations of DBPs were measured for cold and hot tap water of each home. A lab-controlled heating test was later on set up to investigate the formation and degradation of DBPs in short term and long term heating to understand the difference in DBP concentrations in the hot tap water out of different types of water heaters. The results from the field study revealed that the differences in DBP levels in the hot tap water out of the two types of heaters were statistically significant for chlorine residual, total trihalomethanes (TTHMs), dichloroacetic acids (DCAA), dichloroacetonitrile (DCAN), trichloroproprane (TCP) and chloropicrin (CP). Bench scale heating tests showed that long term heating changed the concentrations of DBPs significantly.

The third objective of this dissertation was to investigate the thermal formation and degradation in various conditions. Especially, the impact of water age on DBP formation and degradation in cold and heated water was investigated. The results of this study demonstrate that DBP concentration profiles in heated water were quite different

from the DBP concentrations in the cold tap water. Chloroform concentrations in the heated water remained constant or even decreased slightly with increasing distribution system water age, despite the fact that its levels always increased with water age in the cold water.

The final objective of this dissertation was to propose a method to model chlorine decay, not only in the distribution system, but also applicable to home heating scenarios. A robust two-site chlorine decay model of combined effects of pH, temperature in water distribution system and heating condition was proposed. A single set of readily interpretable parameters were estimated by stochastic search using differential evolution.

TABLE OF CONTENTS

Page

LIST OF TABLES

LIST OF FIGURES

CHAPTER 1

INTRODUCTION

1.1 Background

Disinfection is a water treatment process designed to combat waterborne microbial diseases. This process along with other treatment process such as filtration or coagulation-flocculation-sedimentation provides effective removal or inactivation of microorganisms in drinking water. However, the use of disinfectants such as chlorine, ozone, chloramines or chlorine dioxide, while reducing microbial risks, creates new possible health risks because potentially harmful compounds known as disinfection by-products (DBPs) are formed during the disinfection process. In sight of this rapidly growing public health concern, the Environmental Protection Agency (EPA) in the United States implemented multiple regulations including the Stage 2 Disinfectants and Disinfection Byproducts Rule (Stage 2 DBPR). Tremendous efforts and cost have been spent on controlling DBPs in drinking water for public water systems for compliance purposes.

The vast majority of research on disinfection byproducts exposure for human beings focuses on the direct ingestion pathway, and most of this research was conducted on “cold water” collected by flushing a cold-water tap with the intent of sampling water from the main. However, it is not uncommon for people to use water from a “hot water” tap directly in the home (e.g., drinking, cooking, brewing tea or coffee, washing and showering). Inhalation and dermal adsorption pathways involving hot water such as showering and bathing were found to be very important exposure routes and could generate or release higher concentrations of DBPs (Maxwell et al., 1991, King et al.,

2004, Whitaker et al., 2003; Nuckols et al., 2005). It brings into question the efforts and costs that water utilities take to meet current DBP regulations and if they really help to reduce human DBP exposure. Little research has been done to relate DBP levels in the water main to actual concentrations of these compounds in the water that people are exposed to in the home.

The concentrations of DBPs at the point of use may also be affected by the residence time of water within the household (distribution system, heating tank etc.). Very little research has been conducted on the impact of temperature or water heating on DBP concentrations at the point of use. The general research question posed by this dissertation is "how do actual DBP concentrations in domestic water compare to water collected for compliance purpose"? In other words, to what extent, does heating and exposure to hot tap water in the home change concentrations of regulated and non-regulated DBPs?

1.1.1 Chlorination

Disinfection is the process of inactivating harmful and objectionable bacteria, cysts and other pathogenic microorganisms in water treatment by various agents such as chemicals (e.g. Cl_2, ClO_2, ozone, and chloramine), heat, ultraviolet light, ultrasonic waves, or radiation. It is one of the most important measures to safeguard public health against waterborne diseases. As one of the most common disinfectants of large volumes of water, chlorine was added to drinking water since early late 1800 and chlorination became a drinking water standard in 1904 (White, 1999). Chlorine can provide residual disinfection and therefore public water distribution systems are required to maintain

chlorine residuals in pipelines to prevent microbial re-growth and help protect treated water throughout the distribution system up to the point of consumption. To address continuing concerns about waterborne disease, the U.S. Environmental Protection Agency (EPA) promulgated the Surface Water Treatment Rule (SWTR) in 1989. The Public water systems are required to achieve an overall minimum 3-log removal or inactivation of Giardia cysts and a 4-log removal or inactivation of viruses (U.S.EPA 1989a). Accordingly, to ensure the microbial quality of finished water, the SWTR mandates all systems to maintain a minimum disinfectant residual of at least 0.2 mg/l at the entry point to the distribution system, and a detectable residual in at least 95% of the measurements taken throughout the distribution system.

The maximum residual disinfectant level (MRDL) set by EPA is 4 mg/l for chlorine whether chlorine is used as a primary disinfectant or not. Although chlorine levels are usually found to be significantly lower in tap water than the MRDLs, those levels as high as the MRDL should not pose risk of adverse health effects but allow for an adequate margin of safety (U.S. EPA, 1998a).

Although chlorination is recognized as a cheap and reliable disinfection method to effectively disinfect microbes, some disadvantages of chlorination are present (Kirmeyer et. al., 2004; Deborde and Von Gunten, 2008; White, 2009).

- The effectiveness of chlorination is depended on water pH. pH impacts the speciation of chlorine and different species of chlorine show different reactivity with microbes and micro-pollutants in water.
- Chlorination leads to some potential harmful byproducts such as trihalomethanes (THMs) and haloacetic acid (HAAs) in drinking water.

- Chlorination causes odor and bad taste for drinking water. Both hypochlorous acid and hypochlorite ion have strong smells, and therefore chlorine residuals in the distribution system bring an unpleasant favor and odor to drinking water. Furthermore, odor and taste problems could be caused by some DBPs with lower threshold limits than for the disinfectant itself.

1.1.2 The chemistry of chlorine

Either chlorine gas (Cl_2) or a salt of hypochlorous acid (NaOCl) are commonly used in chlorination. When adding chlorine gas to water, it rapidly hydrolyzes to chloride (Cl^-), hydrogen ion (H^+), and hypochlorous acid (HOCl) spontaneously according to the following reaction (Fair et al., 1948):

$$Cl_2 + H_2O \longleftrightarrow HOCl + HCl \quad (K_h = 1 \times 10^{-4}) \tag{1.1}$$

The equilibrium of the above reaction favors the formation of HOCl as the hydrolysis constant $K_h \ll 1$. Hypochlorous acid salt can also produce HOCl through hydrolysis as follows:

$$NaOCl \longrightarrow OCl^- + Na^+ \tag{1.2}$$

$$OCl^- + H_2O \rightarrow HOCl + OH^- \tag{1.3}$$

Hypochlorous acid is a weak acid and it partially dissociates in water with a dissociation constant pk_a of 7.54 at 25°C (Benjamin, 2002):

$$HOCl \leftrightarrow H^+ + OCl^- \tag{1.4}$$

The distributions of hypochlorite ion and hypochlorous acid are highly dependent on pH. Hypochlorous acid is a more reactive oxidant and effective disinfectant than

hypochlorite ion and it is the dominant species at pH values lower than 7.54. The sum of hypochlorite ion and hypochorous acid is defined as free available chlorine.

1.1.3 Disinfection byproducts

As briefly discussed earlier, disinfection byproducts are formed when chemicals (disinfectants) used in disinfection process react with bromide and/or natural organic matter such as humic and fulvic acids present in the source water. Equation 1.5 generalizes the formation of THMs and other halogenated DBPs:

$$HOCl + Br + NOM \rightarrow \text{THMs and Other Halogenated DBPs} \qquad (1.5)$$

Trihalomethanes were the first class of halogenated DBPs discovered in finished drinking water (Rook, 1974). The National Organics Reconnaissance Survey by the U.S. EPA found that chloroform was ubiquitous in all chlorinated drinking water; thereby the presence of chloroform was linked to chlorination (Symons et al., 1975). Shortly after the discovery of THMs in drinking water, di-and trichloroacetic acids were identified as a second major class of DBPs in chlorinated water (Quimby et al. 1980; Miller and Uden 1983; Christman et al. 1983; Reckhow and Singer 1984; Krasner et al., 1989). Typical HAA concentrations were found to be about the same level to or even higher than the THMs (Norwood et al., 1980). Other frequently identified halogenated DBPs include haloacetontriles, haloketones, chloropicrin, cyanogen chloride, and chloral hydrate and are found at lower concentrations (Krasner et al., 1989). Hundreds of halogenated DBPs have been identified to date (Stevens et al., 1989). However, these identified DBPs only account for no more than 50% of the total organic halide (TOX) concentrations in

drinking water, the rest are unknown(Christman et al., 1983, Reckhow and Singer 1984; Singer and Chang 1989).

In the last thirty years, the potential health risk of DBPs in drinking water has gained a lot of attention. Several epidemiological and laboratory studies have shown that some of these DBPs may pose cancer risks as well as other acute and chronic effects to human health (King and Marrett, 1996, King et al., 2000 Waller et al., 1998). To protect humans from exposure to DBPs, the U.S. EPA imposed regulations to limit the formation of some known DBPs in drinking waters. Trihalomethanes (THMs) and haloacetic acids (HAAs) are two groups of the identified disinfection byproducts for which regulations have been established. The regulated THMs consist of the four chlorine and bromine containing trihalomethanes; chloroform, bromodichloromethane, dibromochloromethane, and bromoform. In the current Stage 2 Disinfectants/Disinfection Byproducts (D/DBP) Rule, the MCL (Maximum Contaminant Level) for the sum of the four THMs is set at 80 μg/L based on a locational running annual average (U.S.EPA, 2006). The most recently published Stage2 D/DBP Rule sets an MCL for five HAAs (HAA_5) at 60 μg/L based on a locational running annual average (U.S.EPA, 2006). The five HAAs include mono-, di, and trichloroacetic acids and mono- and dibromoacetic acid. In the presence of bromide, bromochloroacetic acid, bromodichloroacetic acid, dibromochloroacetic acid, and tribromoacetic acid are also formed, however, these bromine containing haloacetic acids are not regulated in the current DBP rules.

Emerging DBPs beyond those that are currently regulated are becoming important. Recent research has suggested that certain nitrogenous DBPs (e.g., haloacetonitriles) and certain non-regulated carbonaceous DBPs (e.g., haloacetaldehydes) may be of greater

health concern than the regulated carbonaceous DBPs (i.e., THMs, HAAs) (Muellner et al., 2007; Plewa et al., 2008). In general, brominated DBPs are now recognized as having special toxicological importance because tests have shown that brominated DBPs may be more carcinogenic than their chlorinated analogs (WHO, 2000). In addition, preliminary studies are indicating that iodinated compounds may be even more toxic than the brominated analogs (Plewa et al., 2004). Early evidence in epidemiologic studies also gives indication that brominated DBPs may be associated with adverse reproductive and developmental effects (Waller et al., 2001), as well as cancer endpoints. Specific DBPs that are of current interest include iodo-acids, bromonitromethanes, iodo-THMs, brominated forms of MX (MX is 3-chloro-4-(dichloromethyl)-5-hydroxy-2(5H)-furanone), bromoamides, a bromopyrrole, and nitrosamine (which is not halogenated, but is classified as a probable carcinogen) (Weinberg et al., 2002).

The factors influencing the formation of halogenated DBPs include (Singer, 1993; Singer, 1994):

- pH
- Contact time
- Temperature and season
- Nature and concentration of NOM
- Chlorine dose and residual
- Bromide concentration

The key points of the impact of the above factors are summarized briefly below (Singer, 1994):

- The formation of HAA is known to increase with decreasing pH, compared to THM formation, which increases with increasing pH. Additionally, overall TOX formation decreases with increasing pH because of the hydrolysis of halogenated DBPs at alkaline pH-values.
- While the formation of THMs and HAA continues to increase with increasing contact time in the distribution system as long as there is a presence of free chlorine, some of the other halogenated DBPs such as haloacetonitriles and haloketones form rapidly initially upon chlorination but then decay due to the hydrolysis and further reaction with chlorine residuals. Therefore, the exposure of consumers to these compounds depends upon their proximity to the point of chlorination at the water treatment plant.
- Seasonal impact of DBPs in the distribution system could be explained by temperature considerations partially as reaction kinetics are faster in the warmer season and slower in the colder season. Additionally, the nature of DBP precursors as well as bromide concentration could be seasonally or weather (dry or wet) related.
- The characteristics of DBP precursors impact DBP formation. Halogenated DBP's formation increases with the increasing activated aromatic content of NOM (Reckhow et al., 1990).
- Chlorine dose and free chlorine residual impact the rate, extent and distribution of DBPs. While the formation of THM and HAA stops as soon as the chlorine residual are depleted, some other DBPs continue to form through hydrolysis reactions. The trihalogenated HAAs are favored over THMs, dihalogenated

HAAs, brominated DBP respectively when the chlorine dose and free chlorine residual are high.

- Bromine incorporation into DBPs increases with increasing bromide concentration.

1.1.4 Human exposures to DBPs

The US regulatory approach to minimizing human exposure to DBPs has been to use THMs and HAAs as surrogates with associated required compliance for THMs and HAAs at selected locations in the distribution system. However, DBPs constitute a complex mixture of hundreds of compounds with heterogeneous physical and chemical properties, different mutagenic and carcinogenic potentials, and with substantial temporal and spatial variability (Symanski et al., 2004, Villanueva et al., 2004). Therefore, the current regulatory approach may not be the best means of minimizing human exposure to DBPs.

An important issue for epidemiological study of DBPs in public water supplies is the determination of exposure. By definition, exposure is "an event consisting of contact at a boundary between a human and the environment at a specific concentration for a specific interval of time" (National Research Council, 1994). There are three basic routes of exposure: ingestion, inhalation and dermal absorption. Although ingestion has been thought to be the most significant route of exposure to DBPs, the high volatility and dermal permeability of certain DBPs suggest the potential contribution of the inhalation and dermal absorption pathways. In most of the previous epidemiological studies, only the route of water ingestion was used as a surrogate for exposure by all means. It may be

a good surrogate for exposure to non-volatile compounds such as HAAs; however, for volatile compounds such as THMs, both inhalation and dermal exposure must be taken into consideration. It is predicted that over a lifetime, the inhalation and dermal doses (ng/kg) of THMs could be as high as 5.7 and 1.8 times, respectively, the dose received by ingestion (Maxwell et al., 1991).

Exposure depends on the type of DBP and the exposure route, such as drinking water or beverages made with tap water, consuming food prepared with tap water, showering, bathing, swimming in chlorinated pools, and to a lesser extent engaging in other water related activities such as washing dishes, cooking, and clothes washing (Lin et al., 2000; Kaur et al., 2004; Aggazzotti et al.; Jo et al., 1990). Human exposure pathways for DBPs are illustrated in **Figure 1.1**.

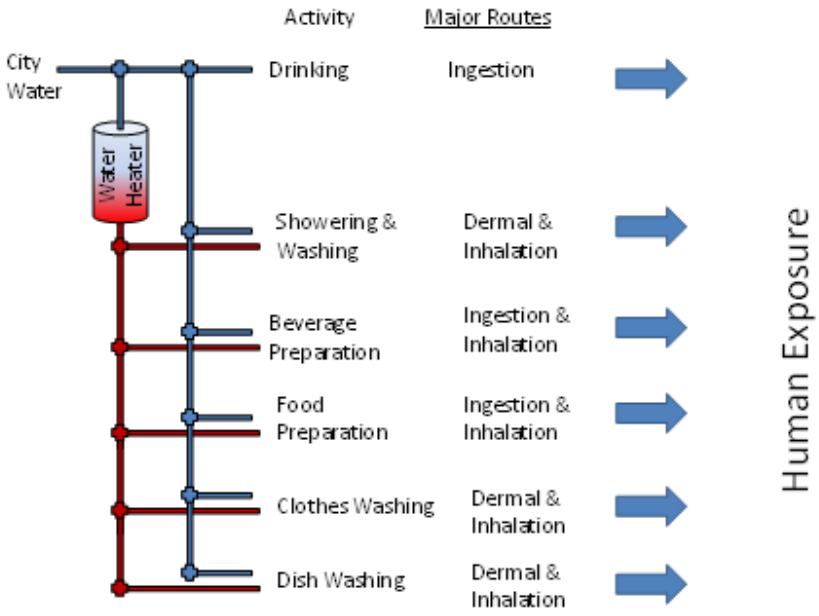

Figure 1.1 DBPs exposure routes in the home

Recent studies show that inhalation and dermal absorption are important routes of population exposure to THMs (King et al., 2004, Whitaker et al., 2003 and Nuckols et al., 2005). Inhalation exposure occurs when water contaminants are volatilized or aerosolized and subsequently inhaled. Volatile compounds are present in both the droplet and vapor phase, while nonvolatile compounds with low Henry's law constants tend to remain in the droplets. For the less volatile compounds, ingestion and dermal contact play more significant roles in exposure and uptake. Inhaled and dermally absorbed THMs reach the bloodstream without being metabolized, requiring separate consideration. Toxicological studies in animals and experimental studies in humans have shown that the most prevalent compounds (THMs) are absorbed primarily through the lungs or the skin (Nuckols et al., 2005, Whitaker et al., 2003). Inhalation or dermal absorption may lead to a higher delivery directly to target organs (e.g., kidney, bladder, or colon), bypassing efficient detoxification steps in the liver that occur upon ingestion (Landi et al., 2003).

Gordon et al., (2006) studied the changes in breath trihalomethane levels resulting from 12 common household water-use activities. Although showering (for 10 minutes) and bathing (for 14 minutes), as well as machine washing of clothes and opening mechanical dishwashers at the end of the cycle resulted in substantial increases in indoor air chloroform concentrations, only showering and bathing caused significant increases in breath chloroform levels. In the case of bromodichloromethane (BDCM), only bathing yielded significantly higher air levels in relation to the pre-exposure concentration. For chloroform from showering, strong correlations were observed for indoor air and exhaled breath, blood and exhaled breath, indoor air and blood, and tap water and blood. Only water and breath, and blood and breath were significantly associated for chloroform from

bathing. For BDCM, significant correlations were obtained for blood and air, and blood and water from showering. Neither dibromochloromethane nor bromoform gave measurable breath concentrations for any of the activities investigated because of their much lower tap-water concentration. It should be noted that all the breath THM concentration were normalized to the THM concentrations in the water associated with the water use activities with a units of (μg/m3)/(μg/L). According to the data provided by Nuckols et al., (2005) for the same set of experiments, the concentration of THMs in the water used for all these activities was close to that of baseline case (cold tap water samples). So the comparison of the normalized data is meaningful. Water temperature was not found to be correlated with the THM concentrations. This study confirmed that activities using hot water are important exposure routes. The differences between the air samples in shower, bath and other water use activities could be attributed to the fact that water used in the shower and bath is heated, which needs to be further studied. Also there was no detailed description of the way they prepared hot beverages, such as temperatures used, the method of heating and the residual chlorine concentration in the tap water which could lead to further DBP formation and transport. As this series of studies didn't include analysis of HAAs for the hot water use activities, studies of other hot water use activities are still necessary.

1.1.5 DBP fate in the thermal processes

Performing total DBP exposure studies for humans could be a challenging task. Tap water concentrations are likely to be accurate for a given system, however not necessarily accurate for individual exposure. There are also a lot of variables that need to

be included in these studies, such as ingested volume, water use habits (filter, boiling), and the assessment of DBP exposure in showers, bathing, etc. There is a need for more defined studies, other than those based on the self-reported information. Most of the epidemiological and exposure research on DBPs has focused on THMs and HAAs as surrogates for overall human DBP exposure from showering, bathing and swimming. However, the ingestion and inhalation of DBPs in prepared food, and cooked food has not been deeply explored as of yet. Also, it is not uncommon in cooking, beverage and food preparation to use tap water that is heated and boiled prior to ingestion. This seems to call for more research on the effect of temperature on DBP exposure via these routes. It is well established that THM concentrations of water for the residential water heaters are generally much higher than in tap water from the utility distribution system (Nuckols et al., 2005). The population's exposure to DBPs is more significant in residential water system than from the municipal distribution system. However, the current regulations concerning drinking water only apply to the latter.

Weisel and Chen (1994) investigated the impact of heating drinking water on THM concentration during laboratory tests (water temperature comparable to those of a water heater). They observed that THMs increased significantly when water containing 0.7-0.8 mg/L residual chlorine was heated to a temperature of 65^{o}C. Chloroform was found to double while a 50% increase in $CHBrCl_2$ and $CHBr_2Cl$ was observed during the heating. They reported that most of this increase occurred within 0.5 hr and was essentially complete within 1 hr. If THM concentrations do stabilize in a residential water heater, obtaining measurements of temperature and THM concentration in separate hot

and cold water samples during an epidemiology study could help improve exposure assessment.

Betterman et al. (2000) studied volatilization losses from water served in an ordinary pitcher, glass, and mug. They found minor (20%) volatilization losses of THMs under conditions of low temperature (below 30^{o}C). Rapidly heating of the water to 60 or 80^{o}C also was not expected to result in significant volatilization. However, volatilization losses approached 75% when water was boiled even for brief periods and reached 90% when boiled water was poured and served.

Wu et al. (2001) conducted thermal treatment of halogenated DBPs in drinking water. They used Seattle tap water, chlorinated synthesized water containing HPOA (hydrophobic acid) isolate and high purity water spiked with DBPs of interests. For heating in the system which allows mass transfer to the atmosphere, 95% of the THMs were removed after boiling for 5 minutes. The DBPs in spiked high purity water showed more THM loss than the other two parallel experiments using chlorinated synthetic water and tap water. This indicates that some additional THMs were generated in the tap water and HPOA-containing solutions when the temperature was elevated. Their hypothesis was confirmed by experiments in closed vials, in which the THM concentrations increased steadily with increasing temperature. The fractional increase in the samples heated to 95^{o}C was approximately 60% for $CHC1_3$ and 33% for $CHCl_2Br$ and $CHCIBr_2$. The mechanism of THM generation was believed to be the cleavage of larger halogenated intermediate species in the HPOA containing solution, and could have been at least partly attributed to decomposition of HAAs from the heated solutions. These compounds are known to decompose by decarboxylation leading to the corresponding

THM. (e.g., TCAA goes to chloroform, bromodichloroacetic acid goes to bromodichloromethane) as reported by several researchers (Christensen et al., 1988; Cammann and Hubner, 1993; Koch and Voelker, 1996). They also found that volatilization is negligible for HAAs, and the formation and destruction of HAAs is of equal importance. The decrease of the majority of TOX (total organic halides) was attributed to the labile nature of other DBPs, although at least 1, 1-Dichloro-2-propanone and chloral hydrate can also be generated by thermal decomposition of other molecules at a temperature of 65^{o}C.

Krasner and Wright (2005) investigated the impact of boiling on DBP concentration using chloraminated and chlorinated waters containing bromide. The experiments consisted of 1-, 2-, and 5-minutes boiling times. Less net removal of chloroform was detected in the chlorinated water (34%) compared to the chloraminated water (75%) for 1 min of boiling, which indicated that simultaneous formation and volatilization of chloroform was occurring. However, the removal ratios for trihalomethanes were similar upon 5 minutes boiling. No significant concentration changes for dihalogenated haloacetic acids (DXAAs) were noted upon boiling of chloraminated water, whereas an increase of DXAAs was detected in chlorinated water. A decrease of trihalogenated haloacetic acids (TXAAs) was detected over time in the chloramined water. Trihaloacetic acid concentration was unchanged after boiling chlorinated water for 1 min, but a 30% reduction was observed after 5 minutes of boiling. The bromodichloroacetic acid concentration decreased by more than half upon boiling for 1 minute, and was completely removed after 2 minute of boiling, whereas dibromochloroacetic acid was removed after boiling chlorinated water for 1 minute.

These studies improved the knowledge of the impact of heating on the variations of DBPs in drinking water. However, the heating time is short, and it is based on lab-controlled condition, not real conditions at home. Dion-Fortier et al. (2009) studied the impact of electric water heaters on THM and HAA levels in Canada. Water samples from three systems were investigated, which include one that had always been in compliance with Quebec TTHM (total trihalomethanes) standards (annual average of 80 μg/L); one that often exceeded the standards and one that had relatively high concentration of bromide ion. For each system, which includes two households each, water samples from the distribution system in the evening after the whole day water use, samples at entry to the residence, cold tap water samples, and hot tap water samples early in the morning and in the late afternoon were collected for TTHM and HAAs analysis. The results show that a residence time of water in the hot water tank of several hours had a very important effect on DBP levels. Increases in chlorinated DBPs were much more important during the residence in the hot water tank compared with what was observed during stagnation in the cold water pipes. This increase was attributed to the dramatic temperature increase in the water heater, which accelerates the reactions of chlorine in water. The results also indicate that temperature increase promotes the formation of brominated THMs, but not much brominated HAAs. The water consumption pattern was found to have little impact on DBP concentration in the hot water tank while sustained use of water in the residence was found to prevent stagnation in the pipes, thereby reducing the additional formation of DBPs in water. This study confirmed the change of DBP levels in water heaters and indicated the importance of studying human exposure to DBPs from residential tap water other than just the distribution system. It may be interesting to compare water systems

with different disinfection schemes (chlorination, chloramination etc.). It is also interesting to compare the impact of heaters with tanks and tankless heaters.

Aside from impacting DBP concentrations, temperature may also have an important impact on absorption across human skin. Gordon et al. (1998) reported a strong impact of bath water temperature on dermal absorption of chloroform, and it is likely this impact would hold for other hot water use activities with respect to dermal contact. It might be possible and promising to estimate air THM concentrations for specific water use activities based on the hot and cold water THM concentrations. This could be done by using air to water THM concentration ratios for each activity to construct "confidence intervals" for predictions of air THM concentrations from specific water use activities. A limitation to this approach is that these ratios could vary by activity as a function of room volume, ventilation, and other factors (Nuckols et al., 2005).

Although changes and reductions of some DBPs upon boiling are possible (Krasner et al., 1989), Bove Jr. et al. (2007) found no difference between the effects of hot water tap consumption and cold water tap consumption on rectal cancer risks in their study; they thought that may be due in part to a sample size in the study which may have been too small to measure such an effect. It may also be the case that bromoform is serving as a surrogate for exposure to some of the most mutagenic brominated forms of other DBPs such as MX that are not affected by volatilization such as BMX-1, BMX-2, BMX-3 (McDonald et al., 2005; Suzuki et al.,1995).

Rahman et al. 2011 found a significant reduction of THMs in kettle-boiled water, instant boiled water, jug-filtered water and instant boiled-filtered water and a large decrease for all HAA species in jug-filtered, instant boiled-filtered water. In a population

that mainly drinks bottled water (e.g., pregnant women), individual THM uptakes are dominated by inhalation and dermal absorption during, showering, swimming in pools, and bathing (Villanueva et al., 2007).

These studies provided important information for the changes and reductions of regulated DBPs under different temperature conditions; however, more research is needed on the transformation of DBPs other than simple reduction for cold and hot water use as a decrease of the concentration of regulated DBPs in the water phase does not guarantee a decrease of DBP exposure. Other DBPs are transformed from THMs and HAAs, and the volatilization of the regulated species will enter into the gas phase at home, which could enter the human body by inhalation exposure.

1.2 Research Objectives

Objective of this research were: (1) Investigate the temporal variability and extent of regulated DBP and non-regulated DBP formation and degradation out of water heaters, (2) Investigate the impact of water heaters and heating scenarios on regulated DBP and non-regulated DBPs profiles, (3) Investigate the relationship between water age and DBP profiles in heated water and assess the corresponding impact of pH, heating time, dose, incubation temperature, (4) Build a mechanistic model to describe chlorine decay in the water with or without heating with a single set of readily interpreted parameters.

1.3 Research Hypotheses

The major research hypothesis is that the stage 2 disinfectant and disinfection byproduct rule and the corresponding DBP control strategies could not significantly

reduce human exposure to disinfection byproducts. There are also some more specific research hypotheses which include:

(1) Heating of tap water containing chlorine could lead to the further formation of DBPs until the free chlorine is exhausted, (2) A tankless heater has less impact on DBP profiles of hot tap water out of the heater than conventional tank heater because of its instant heating of the cold tap water, (3) chlorine depletion is due to be concurrent 2^{nd} order reactions with multiple aqueous constituents.

1.4 Scope of Work

As first part of this dissertation, Chapter 2 "Disparity in DBP Concentrations between Hot and Cold Tap Water" focused on temporal variability and ultimate disinfection byproducts formation and degradation in cold and hot tap water in a household and also in a distribution system. The results help validate the adoption of lab simulation tests to study the DBP formation and degradation in heated water out of a water tank.

Also as a part of this dissertation, cold and hot water samples were collected in homes equipped with tankless and tank heater within a cohousing community. The concentration of DBPs in the cold and hot tap water samples were compared statistically for each type of water heater. Bench scale tests were conducted to simulate short term heating and long term heating of the cold tap water in order to investigate the thermal formation and degradation of DBPs. The results are presented in Chapter 3 "Impact of water heaters on the formation of disinfection byproducts in a residential plumbing system".

In Chapter 4 “DBP formation in hot and cold water across a simulated distribution system--Effect of incubation time, heating time, pH, dose, and incubation temperature”, natural water was selected and treated with chlorine to investigate the thermal formation and degradation in various conditions. Most importantly, the impact of water age on the DBP formation and degradation in cold and heated water was investigated.

Since the chlorine residual plays an important role in the further formation of DBPs in the heated water, a study for modeling the chlorine residual during heating process is necessary and important. Therefore, in Chapter 5, “A two-sites chlorine decay model of combined effects of pH, temperature in water distribution system and heating condition using differential evolution ”, a model that could incorporate temperature and pH effects were explored and demonstrated with data from heating scenarios.

CHAPTER 2

DISPARITY IN DBP CONCENTRATION BETWEEN HOT AND COLD TAP WATER

(*Prepared manuscript for submission to "Water Research"*)

2.1 Abstract

The quality of water entering a distribution system may differ substantially from the quality at the point of exposure to the consumer. This is true even when raw water quality and treatment operations are time-invariable and water quality at the point of entry exhibits little or no variability. The reasons for changing water quality include continuing reaction of residual chlorine during distribution, reactions with pipe walls, and accelerated reactions due to in-home water heating. This study investigated temporal variations in the levels of regulated and non-regulated Disinfection Byproducts (DBPs) in cold and hot tap water in a home on a medium-sized municipal water system. In addition, samples were collected directly from the water plant with some being held in accordance with a simulated distribution system test protocol. The location for this work was a system in western Massachusetts, USA that uses free chlorine as a final disinfectant. Very little short term variability of DBPs at the Point of Entry (POE) was observed. The concentration of DBPs in the simulated distribution system (SDS) test was similar to concentrations in the cold water tap. For most DBPs, the concentrations continued to increase as the cold water tap sample was held for the SDS incubation period. However, the impact of heating on DBP levels was quite compound specific. For example, the concentrations of trihalomethanes (THMs), dichloroacetic acid (DCAA) and chloropicrin

(CP) were significantly higher in the hot water tap than in the cold water SDS samples. In contrast, the concentration of trichloroacetic acid (TCAA) was lower in the heated hot tap water, but about equal to that observed in the cold tap water. The situation was more pronounced for dichloroacetonitrile (DCAN), bromodichloroacetic acid (BDCAA), bromochloroacetic acid (BCAA) and 1,1,1-trichloropropanone (TCP), which all showed lower concentrations in the hot water then in either of the cold water samples (instantaneous or SDS). The latter was viewed as a clear indication of thermally-induced decomposition. The ratio of unknown total organic halide (UTOX) to TOX was substantially lower in the hot tap water as the THM to TOX ratio became correspondingly larger. The results of this study show that DBP exposure in the home is not well represented by concentrations measured in cold water taps where most compliance monitoring is done.

Keywords

Disinfection byproducts, water heater, premise plumbing, tap water

2.2 Introduction

Disinfection byproducts (DBPs) are a concern in municipal water systems because of their potential health risks and because of current federal and state regulations limiting their concentrations. Assessing human exposure to DBPs is a challenging task because of their high degree of temporal and spatial variability and the multiple routes of exposure. In additional to ingestion of cold tap water, inhalation and dermal absorption are likely to be important exposure pathways (Maxwell et al., 1991, King et al., 2004, Whitaker et al., 2003, Nuckols et al., 2005). For example, hot water use was found

to have an important impact on people's exposure to trihalomethanes (THMs) (Gordon et al., 1998; Nuckols et al., 2005).

Many studies of hot water exposure have focused on the relationship of hot water DBP concentrations and concentrations in the exposed individual's breath and blood (Nuckols et al., 2005). For research applied to exposure through ingestion, the temperatures selected were either at the boiling point (Betterman et al, 2000; Wu et al, 2001, Krasner and Wright, 2005) or seasonally elevated temperature (i.e., <40^{o}C; Arora et al., 1997). In these studies, the elevated temperatures were held constant throughout the incubation period (e.g. Zhang et al., 2013). However, none of published studies were designed to examine DBP profiles in domestic hot water systems, where temperatures become elevated midway through the reaction of chlorination with NOM (natural organic matter).

Very little research has been conducted on the temporal variability of disinfection byproduct concentrations in household plumbing systems especially when including the impact of water heaters. Dion-Fortier et al. (2009) studied the effect of electric water heaters and plumbing on THM and HAA levels. They observed considerable increases in THMs when water stagnated in pipes and hot water tanks, and they noted that HAAs increased as well but less significantly. Yamamoto et al., (2007) evaluated the effect of storage time in building plumbing systems on the variation of trihalomethane levels. They found the total THM levels to be higher in building plumbing systems than the city water entering the home, and additional incubation of the city samples produced levels that approached the levels of THMs in the building stored water. While they did not explicitly examine the effect of heating; they did report that the average daily air

temperature was positively correlated with the total THM level. Eyring et al. 2008 collected hot and cold water samples throughout the Philadelphia system in 2006. Philadelphia uses chloramines as a final disinfectant with average residuals of about 1.5 mg/L across the system, and average treated DOC levels of about 1.5 mg/L as well. THM levels in hot tap water were found to be about double the amount in cold tap water. However, hot water HAA values were found not to differ as much from the corresponding cold water values. Li and Sun (2001) investigated the enhanced formation and volatilization of trihalomethanes in water heaters. However, their experimental design (quickly heating to the boiling point with large air spaces) may not have been appropriate to home water heaters.

Home processing of potable water (boiled, filtered, refrigerated) is very common in the US, which may serve to reduce exposure to DBPs by ingestion. As a result, exposure to DBPs during hot water use activities (showering, bathing) may become more significant. In order to improve the accuracy of DBP exposure assessments, the concentrations of DBPs in water coming from home water heaters needs to further study. In a companion paper (Liu et al., 2013) the concentrations of DBPs under simulated water heating conditions were found to exhibit substantially different behavior compared to non-heated water, and these differences changed with increasing distribution system water age. In the current study, we document the detailed temporal variation of DBPs at cold and hot tap water from an actual water heater at a single-family home.

2.3 Distribution System Background

One average-sized single-family residence in the City of Northampton, MA was selected for this study. This house used a conventional water tank for heating up the water. The size of its water tank is about 60 gallons. The tap water of the residential property selected for this study was supplied by the City of Northampton's Department of Public Works-Water division through 36 inch ductile iron and 20 inch cast iron transmission mains which branch off to smaller diameter residential piping. The Northampton water treatment plant, with a design flow of 6.5 million gallons per day (MGD), delivers 2.9 MGD on an average day to its 28,000 customers. Approximately 90% of Northampton's drinking water comes from three surface water reservoirs: the Francis P. Ryan, and the West Whately Reservoirs, located in the town of Whately, and the Mountain Street Reservoir located in Williamsburg and Hatfield. The remaining 10% comes from its two groundwater wells in Florence. Since 2008, the operation of its water filtration plant, which is comprised of an upflow roughing filter (adsorption clarifier) and a granular activated carbon filter, has significantly reduced the amount of total organic carbon and DBPs in the system. As a result, the city's DBP levels are well below the maximum contaminant levels (MCLs) set by the Stage 2 Disinfectants/Disinfection Byproducts (D/DBP) Rule. Sodium hypochlorite is added to the filtered water prior to entering a 4 million gallon storage tank. As the water leaves the 4 million gallon storage tank, sodium carbonate is added for pH adjustment and corrosion control. Zinc orthophosphate is added as a corrosion inhibitor.

2.4 Experimental Design

Water samples were collected from the entry point of the system (POE) at the effluent of the Northampton water treatment facility and at a residential distribution location over a 40 hours period. The total transit time from the POE to the residential sampling site was about 18-30 hours based on the flow conditions as impacted by the community water use during the sampling period. At the water treatment plant, bulk effluent samples were collected at 3 pm on Monday afternoon and were incubated in a temperature-controlled water bath in the lab at the actual system temperature (7~8^{o}C) to simulate the DBP formation profile expected during transit from the POE to the residential sampling site (i.e., the "Simulated DS" tests). The incubated samples were analyzed for DBPs starting at 6:30 pm on Monday and analysis continued over a 40 hours period.

The effect of varying plant effluent quality was determined by monitoring the instantaneous DBP levels in POE samples. In addition, the levels of DBPs in unquenched POE water samples (i.e., terminal) were measured after holding the samples for 28 days at 4ºC. These are intended to represent levels one might expect in areas of the city's distribution system with the highest water ages. These POE samples were collected starting at 7am on Tuesday and collection continued every 2 hours until 7pm.

At the residential sampling location, cold tap water and hot tap water samples were collected over a period of 40 hours. Cold water samples were collected after running the tap until the water temperature stabilized. For this reason, these are believed to be representative of the distribution system and little affected by the premise plumbing. Flushing was kept at a minimum for collection of hot tap water samples, with the

exception of one period which is discussed later. For each residential sampling event, both quenched and unquenched water samples were simultaneously collected for each type of water. The DBPs in the quenched samples represented the instantaneous DBP levels at the sampling time, while the DBPs in the unquenched samples continued to form during the additional incubation time (28 days at 4ºC) and represented the terminal DBP levels for that particular sample.

2.5 Material and Methods

Twenty-liter finished water samples were collected from the effluent of the clear well at the Northampton Water Treatment Facility on 3pm, February 22nd, 2010, which is considered as the point of entry (POE) to the distribution system. The TOC of the finished water was 1.73 mg/L in this sample. The effluent water (POE) was partitioned to 12 biochemical oxygen demand (BOD) bottles without headspace. The BOD bottles were placed in a temperature-controlled water bath and incubated at the system temperature (7~8^{o}C). Periodically, samples were removed for chlorine residual analysis and measurement of DBPs. The samples destined for DBP analysis were transferred to BOD bottles containing ascorbic acid, sodium azide and sulfuric acid (2 drops of 6M H_2SO_4) and mixed thoroughly. Plant effluent (POE) samples were also periodically collected in BOD bottles, some with preservative (instantaneous) and some without (terminal).

For sampling at the residential location, the aerator was replaced with a threaded union and a barbed connector with attached tygon tube that could be submerged into the sampling bottle. This allowed samples to be collected without introduction of air. Cold tap water samples were collected in parallel BOD bottles with and without preservatives

and either incubated or stored for later analysis. At the time of each sampling event, chlorine residual and temperature was measured. All hot tap water samples were collected after 2L of hot water had been flushed through the tap. One half liter of hot water was collected in 500-mL serum "piston bottles" (as described by Liu and Reckhow, 2013 and shown in Figure 2.1). Hot water samples with and without preservative were collected. If the chlorine residual was completely gone, only preserved samples were collected. These bottles were cooled down to room temperature for an hour avoiding exposure to light. All samples were transported to the environmental engineering laboratories at the University of Massachusetts in coolers. They were then partitioned to smaller bottles for specific DBP analysis (40-mL vial for THMs and HAAs, and 125-mL amber bottles for TOX), kept in a refrigerator at 4^{o}C and analyzed within 14 days. Plant effluent water samples were transported to the UMass lab and held at the incubation temperature.

Chlorine residuals in both the cold and hot water samples were analyzed at the time of collection by the DPD ferrous titrimetric method (APHA, 1998). All samples were analyzed for the four chlorine and bromine containing THMs (THM4), dihaloacetonitriles (DHAN), chloropicrin (CP), trichloropropanone (TCP) and dichloropropane (DCP), which were quantified by liquid/liquid extraction with pentane followed by gas chromatography and electron capture detection (GC/ECD) according to U.S. Environmental Protection Agency (U.S.EPA) method 551.1. Nine haloacetic acids were analyzed by liquid/liquid extraction with methyl-tertbutyl-ether (MTBE), followed by derivatization with acidic methanol and GC/ECD based on USEPA method 552.2. Total organic chlorine and bromine (TOCl and TOBr) were analyzed by a high-

temperature combustion and off-line ion chromatographic method (Hua and Reckhow, 2006). Total organic halide (TOX) was calculated as the sum of the TOCl and TOBr (TOI was not detected in the samples). Unknown TOCl (UTOCl) and unknown TOBr (UTOBr) were calculated as the difference between the measured TOCl and TOBr, and the amount of corresponding halogen-equivalents in the specific DBPs.

2.6 Results

2.6.1 Temporal variability of temperature in cold and hot tap water

The temperature for the cold and hot tap water samples collected was closely monitored and recorded. The temporal cold and hot water temperatures are shown in Figure 2.2. The cold water temperature was relatively constant with an average temperature at 7.6^{o}C. On the other hand, the hot water temperature varied, and had the lowest temperature at 24^{o}C when the whole water tank was flushed with a large amount of cold tap water filled in because of high water usage. The temperature finally stabilized at around 55^{o}C.

2.6.2 Temporal variability of chlorine residual in cold and hot tap water

After about 24 hours of reaction, the chlorine residuals of the samples from simulated distributed system tests had decreased to the level observed in the cold water of the residential sampling location (Figure 2.3). This agrees well with expectations as the estimated water age at the residential location was 24 hours. There were no measureable chlorine residuals in the hot tap water samples except those collected when the water tank was flushed. The high temperature drove the reaction between NOM and chlorine more

rapidly, and the consumption of chlorine was complete within the residence time of the tank. Immediately after the hot water tank was flushed, but before the temperature had started to increase, the chlorine residual in the hot tap water sample was roughly the same as the chlorine residual in the cold tap water samples. After that, the chlorine residuals decreased gradually. The chlorine in the hot water tank was entirely depleted in less than 10 h.

2.6.3 Temporal variability of DBP concentrations in residential cold tap water, plant effluent, and simulated distribution system tests

Accurate assessment of human exposure to DBPs in chlorinated drinking water requires estimating the concentration of DBPs present in the consumer's tap water. The simulated distribution system test used in this study, like most SDS testing protocols, is quite simple, and does not consider other factors that affect DBP concentrations such as biodegradation, reactions with pipe materials and flow conditions (Rossman et al., 2001). Nevertheless, SDS testing produced concentrations that were remarkably similar to those from the residential tap (Figure 2.4). This is especially true considering that the estimated water age for the residential site is about 24 hours. Figure 2.4 shows that the SDS and cold tap lines merge at just about that reaction time. The THM levels for the unquenched plant effluent samples varied and were much higher. Note that bromoform ($CHBr_3$) was not detected in any of the samples due to the low bromide concentration in the source water. The THMs levels in the cold tap water samples were slightly higher than that in the plant effluent samples as expected based on the small additional formation believed to occur in transit to the residential location. Thus the simulated

distribution system test seemed to do a good job of estimating THMs in the cold tap water.

The temporal concentration profiles for HAA species are shown in Figure 2.5. It was observed that the concentrations of DCAA, BCAA, TCAA and BDCAA in the simulated distribution system were close to the levels in residential tap water at the 24 h holding time and higher than the levels in the plant effluent. The unquenched HAA levels were significantly higher than the quenched HAA levels except BDCAA. The lower levels of BDCAA in the terminal (unquenched) plant sample suggest that this compound may not be favored by higher temperatures and longer reaction times.

Dichloroacetonitrile (DCAN) exhibited a profile similar to the THMs (Figure 2.6). While this compound is known to be subject to decomposition, there was no obvious sign of DCAN loss during this experiment. Much like the THMs, DCAN shows clear signs of formation across the distribution system. Chloropicrin (CP) was present at low levels in the terminal plant effluent samples and below detection limit in the others (Figure 2.7). Trichloropropanone (TCP) showed clear signs of increasing across the system, and this increase was a bit higher on a percentage basis than the THMs (Figure 2.8). The unquenched plant effluent sample exhibited even further increases, despite the fact that this compound is known to slowly decompose to chloroform and dichloroacetic acid (Reckhow & Singer, 1985).

2.6.4 Temporal variability of DBP concentration in cold and hot tap water

2.6.4.1 Trihalomethanes

Figure 2.9 shows the instantaneous and terminal THMs from samples collected of both the cold and hot tap water. The three THM species exhibited similar behavior. The THMs in cold tap samples were nearly constant over time. The terminal THMs in the cold tap water samples were also relatively consistent and about three times that of the instantaneous values. In each case, the terminal cold water tap values were similar to the terminal values of the samples collected at the plant (compare with Figure 2.4). This supports the assertion that the DBP and precursor content of the cold tap water is the same as the plant effluent after being held for about 24 hours, regardless of whether it was held in the distribution system or in a borosilicate glass bottle. The amount of THMs in hot tap water was about three to four times higher than in cold tap water except during the period when the hot water tank was flushed. This is not surprising as the high temperature of the hot water samples would be expected to drive the reaction between THM precursors and chlorine residual very quickly to its endpoint and thus generate more THMs. The role of temperature in accelerating these reactions is well documented (e.g., Englerholm and Amy, 1983; Garcia, 2005). However higher temperatures may also accelerate some DBP forming pathways more than others, thereby shifting the DBP speciation. Furthermore, some DBPs are known to be unstable at high temperature and there are unidentified halogenated intermediates (e.g., Peters et al., 1980; Reckhow et al., 1990; Wu et al., 2001; Hua et al., 2012) that could undergo accelerated decomposition. It is our opinion that all three may be important to varying extents depending on the temperature and the specific DBP. In the case of the THMs, we note that Weisel and

Chen (1994) showed that higher levels were formed only when a chlorine residual existed at the time of heating. This suggests that accelerated reaction between chlorine and precursors, as well as shifting pathways were the cause rather than thermal decomposition of halogenated intermediates.

2.6.4.2 Haloacetic acids

Concentration profiles for the four major HAA species (Dichloroacetic acid (DCAA), trichloroacetic acid (TCAA), bromochloroacetic acid (BCAA) and dichlorobromoacetic acid (DBCAA) showed some similarities to the THMs and some differences (Figure 2.10). Although BCAA and BDCAA are not currently regulated by US EPA, they were investigated in this study as there is evidence for brominated DBPs being more harmful than their chlorinated analogs (e.g., Plewa et al., 2004).

As shown in Figure 2.10, the level of DCAA in hot tap water was more than 2 times higher than that of DCAA in cold tap water except when the water tank was flushed. Although the degradation of DCAN could contribute to the increase of DCAA upon heating, the majority of this increase must be attributable to either a more rapid formation, a shift in pathways or decomposition of intermediates other than DCAN (e.g., Hua and Reckhow, 2012). After the water tank was flushed, the level of DCAAs quickly dropped to about the level of DCAAs in the cold tap water, and then gradually increased to its prior levels and above as the temperature in the water exceeded 50ºC Wu et al., (2001) observed increases in DCAA of 15% and 20% at 45^{o}C and 65^{o}C respectively for chlorinated water containing hydrophobic acid (HPOA) fraction of NOM after depletion of chlorine. This suggests that the decomposition of halogenated precursors which are

capable of forming DCAA occurs at high temperatures as well as at high pHs (e.g., Hua and Reckhow, 2012).The level of terminal DCAAs in the cold tap water was about twice of the level of instantaneous DCAAs in cold tap water, yet it was still a bit lower than the level in the hot tap water. The BCAA level in cold tap water was higher than that of hot tap water before the water tank was flushed. Immediately after the water tank was flushed, the BCAA level increased and became higher than the BCAA in the cold tap before elevated temperatures in the hot tap water seemed to drive it down again. However, considerable reduction was observed after the chlorine was depleted and the tap water continued to be heated in the water tank. Thermal hydrolysis by decarboxylation cannot be more important under these conditions as BCAA hydrolysis has a first order rate constant of $1.59\times10^{-6}s^{-1}$ at $50^{o}C$, which corresponds to a half-life of about 120 hours (Lifongo et al., 2010). Unlike THM species and DCAA, further BCAA formation was observed in the unquenched hot tap water after collection (i.e., the terminal sample).

In contrast to the THMs and DCAA, trichloroacetic acid exhibited little or no increase upon heating. One possible explanation is that TCAA was decomposing via decarboxylation (Zhang and Minear, 2002) at a rate similar to the formation rate, giving the appearance of little net change. However, the decomposition rate constant of $6.18\times10^{-7}s^{-1}$(Verhoek, 1934) at $50^{o}C$ corresponds to a half-life of about 300 hours, which is too slow to be of importance. Wu and coworkers (2001) noted TCAA losses at 85ºC, but not at 65ºC. Little or no increase in TCAA has been observed in a laboratory study (Liu and Reckhow 2013) and in water collected from water heaters under field conditions (Dion-Fortier et al., 2009). Recent research by Zhang et al. (2013) found the highest yield of TCAA for chlorinated humic precursors-containing water was at $50^{o}C$ among all the

heating temperatures (4°C, 15°C, 25°C, 35°C, 50°C). It should be noted that the higher yield at high temperature was attributed to the fact that the heating was applied at the beginning of the chlorination process in the presence of high chlorine residuals. This could reflect the fact that the kinetics of TCAA formation are favored by higher chlorine concentrations (Rossman, 2001). The high temperature and rapid chlorine depletion favored the formation of THMs over TCAA. This result supports the mechanisms proposed by Liu and Reckhow, 2013.

Figure 2.10 indicates BDCAA decreased at elevated temperatures in the water heater. Except for a period after flushing, BDCAA concentration in the cold tap water was significantly higher than that in the hot tap water, suggesting thermal decomposition. Decomposition of BDCAA forms BDCM via decarboxylation with a rate constant of 0.234 day^{-1} at 50°C which corresponds to a half-life of about 70 hours (Zhang and Minear, 2002). Thus, BDCAA is expected to degrade measurably only when the temperature drifts above 50ºC and when the water sits for many hours in the tank. The terminal BDCAA level for cold tap water was quite similar to the instantaneous BDCAA levels in cold tap water. After the water tank was flushed, the BDCAA concentration in the hot water sample quickly increased as the tank was filled with newly added cold tap water which contained higher amounts of BDCAA. The continued incubation in the hot water tank lead to the gradual degradation of BDCAA. The terminal BDCAA level in hot tap water was lower than the BDCAA level in both the cold tap water and hot tap water.

2.6.4.3 Dichloroacetonitrile, chloropicrin and trichloropropanone

Dichloroacetonitrile (DCAN) is the only member of the dihaloacetonitrile (DHAN) group that was detected in the water samples. Instantaneous DCAN levels in the cold tap water were quite constant at about 0.7 μg/L throughout the monitoring period (Figure 2.11). The terminal DCAN in the cold tap water was roughly double the amount in the instantaneous samples. The DCAN concentration in the hot tap water was near or below the detection limit except during the water tank flushing process, when the DCAN concentration peaked and reached the same level as in the cold tap water. The DCAN level dropped with increasing time in the hot water tank because at temperatures above 35^{o}C decomposition of DCAN occurs much faster than formation (Zhang et al., 2013). Estimates of the base and hypochlorite-catalyzed decomposition rate of DCAN under these conditions (pH 7.5; 50ºC) range from 0.035 hr^{-1} upon entry to the tank (0.7 mg/L chlorine residual) to 0.011 hr^{-1} upon exit (no residual), which correspond to half-lives of 2-6 hours (Reckhow et al., 2001; Yu & Reckhow, 2014). The DCAN degradation results in a small contribution to DCAA levels in the hot water.

Chloropicrin (CP) is the most commonly found halonitromethane in chlorinated drinking water and it was detected in this study at levels below 1 μg/L (Figure 2.12). The CP level in the terminal samples was found to be in the range of 0.1-0.25 μg/L and this was close to the level observed in the hot tap water. The cold tap water as well as the hot water right after flushing the tank all showed levels below the detection limit. No apparent degradation of CP was observed in the hot tap water. Based on data from Zhang and coworkers (2013), we expect that we might have observed CP degradation had the residence time in the hot water tank exceeded 24 hours.

Trichloropropanone (TCP) concentrations in the cold tap water were quite consistent at about 1.4 μg/L throughout the study period (Figure 2.13). This compound is known to undergo base and hypochlorite catalyzed degradation (Reckhow & Singer, 1985) and that degradation occurs at rates on the order of 0.2 hr^{-1} at 50ºC (Zhang et al., 2013). Therefore, substantial loss of TCP during storage in a hot water tank is not surprising. The terminal TCP level in the cold tap was about 40% higher than that of instantaneous value, indicating the rate of continued formation exceeds the rate of degradation at low temperatures. Very low levels of TCP were also detected in the hot water during tank flush and in the terminal hot tap water samples prior to reaching the final set point temperature.

2.6.4.4 Total organic halide

Total organic halide (TOX) concentrations in the instantaneous cold and hot tap water sample were relatively consistent at about 40 μg/L and 70 μg/L during the monitoring period, respectively (Figure 2.14). The increase of about 75% upon heating can only be attributed to further reaction between NOM and the chlorine residual. In fact, the actual increase was probably greater as evidenced by the peak in TOX after tank flushing but before the tank had reached its maximum temperature. This point (91 μg/L) probably represents the accelerated TOX formation at a temperature where dehalogenation reactions are not yet occurring at a very high rate. According to Wu et al. (2001), a substantial fraction of the Cl incorporated into organic compounds (i.e., the TOX) was apparently released as inorganic chloride when the solutions were heated. The

hot tap water TOX level finally stabilized and was about the same level compared to the level before the water tank was flushed.

The distributions of TOX in the cold and hot tap water are shown in Figure 2.15. Unidentified TOX (UTOX) is calculated by subtracting the halogen concentration of detected DBPs from the TOX. For the cold tap water, THMs, HAA, UTOX accounted for about 28%, 26% and 42% respectively of the chlorinated TOX, whereas other measured DBPs accounted for 2.7%. This composition is typical for drinking water. For hot tap water, THMs, HAA, UTOX accounted for about 46%, 23% and 30% respectively whereas the other measured DBPs accounted for 0.2%. The results indicate that the TOX distribution has shifted in the hot tap water to more THMs, less UTOX and less of the minor DBPs (haloacetonitriles, halopropanones). One important exception is the increase in concentration of chloropicrin.

2.7 Summary

In this particular drinking water system, DBP levels were found to have very little temporal variability over a period of 40 hours, spanning 3 weekdays. The concentrations of DBPs and residual precursors were little changed from plant to residential cold tap water, except for conversion of precursors to DBPs as expected for the calculated water age. This suggests that there were little or no reactions with residue on pipe walls or reactions with pipe materials in the study system.

Results of this study indicate that the disinfection byproducts in hot tap water of residential users show a different profile as compared to the disinfection byproducts in cold tap water and in the system point of entry. The impact of heating in a typical

residential tank water heater was to completely deplete the chlorine residual and substantially elevate the concentrations of dichloroacetic acid, trichloromethanes and chloropicrin. Further formation of dichloroacetic acid was observed even when chlorine was depleted suggesting decomposition of halogenated intermediates. In contrast, other disinfection byproducts, such as bromodichloroacetic acid, dichloroacetonitrile, bromochloroacetic acid and trichloropropanone were substantially reduced in concentration due to accelerated decomposition at high temperature. The end result was that the TOX profile in the hot water was substantially different from that in the cold water. These differences are important as home exposure to DBPs occurs from hot water as well as cold water, whereas only concentrations in cold water are regularly monitored and regulated.

2.8 Implication for Drinking Water Treatment

Drinking water regulations have historically have been applied to water in distribution systems including plant effluent and distribution system sampling locations but not to water in household plumbing systems. The use of detailed field monitoring in this study revealed the need to clarify the impacts of water heating on DBP profiles and the relationship between hot water DBP concentrations and those in the cold water which is used for assessing compliance. It seems that, for a home with a typical level of chlorine residual, significant elevation of some of the regulated THMs, DCAA and non-regulated DBPs, such as chloropicrin is expected to occur in a domestic hot water tank. Since toxicological research has indicated that halonitromethanes can be much more cyto- and genotoxic than their haloacetic acid analogues (Plewa et al., 2004), the impact

of storage in a hot water tank may be of public health concern. As exposure pathways such as inhalation and dermal absorption in hot water use are increasingly under scrutiny, the distinct DBP profiles of home hot water should be considered in exposure assessments.

The observed increase in DBPs in hot tap water can be attributed to the chlorine residual in the cold tap water that enters the tank. That suggests that households closer to the system point of entry (e.g., those having a lower water age) might have the highest hot water THM levels, whereas those furthest from the point of entry often have the highest cold water THM levels. This is challenging our current practice in controlling distribution system DBPs. Therefore, reducing exposure to THMs may be best achieved by minimizing areas of high chlorine residual rather than focusing on areas of high water age. In other words, consumers in areas of low water age may be at risk for the highest THM exposure in any given system. For unrelated reasons (i.e., biodegradation at long water ages), this can also be the case for HAA exposure. For some metastable non-regulated DBPs, the exposure risk may be higher for households in areas of low water age because of rapid decomposition that will occur at longer contact times and elevated temperatures.

Figure 2.1 "Piston Bottle" for minimization of headspace during heating and cooling

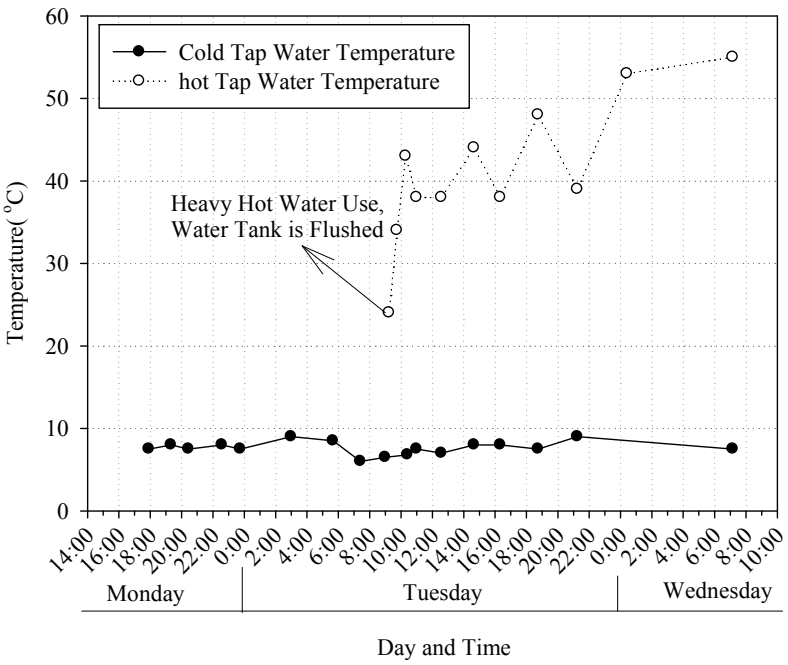

Figure 2.2 Cold and Hot Tap Water Temperature

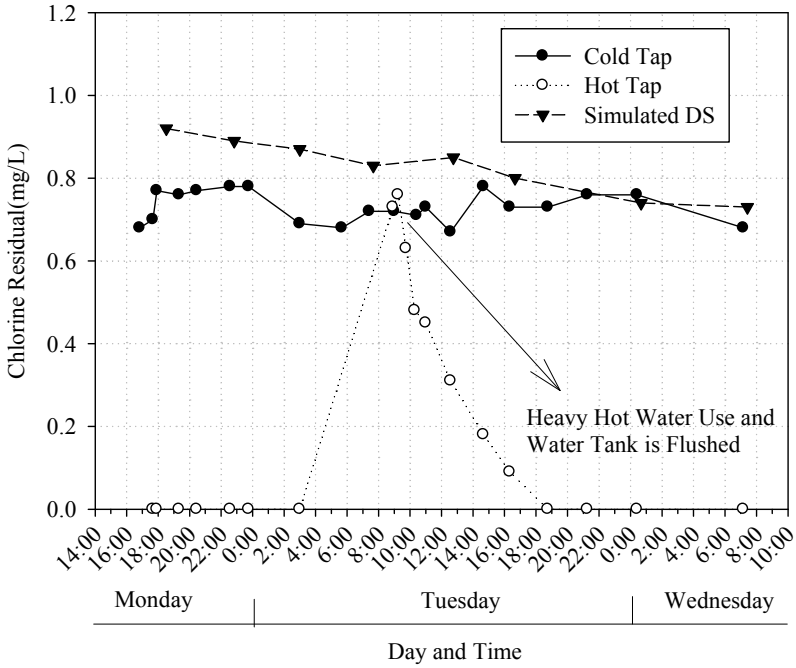

Figure 2.3 The temporal cold and hot water chlorine residuals at residential location and simulated distribution system

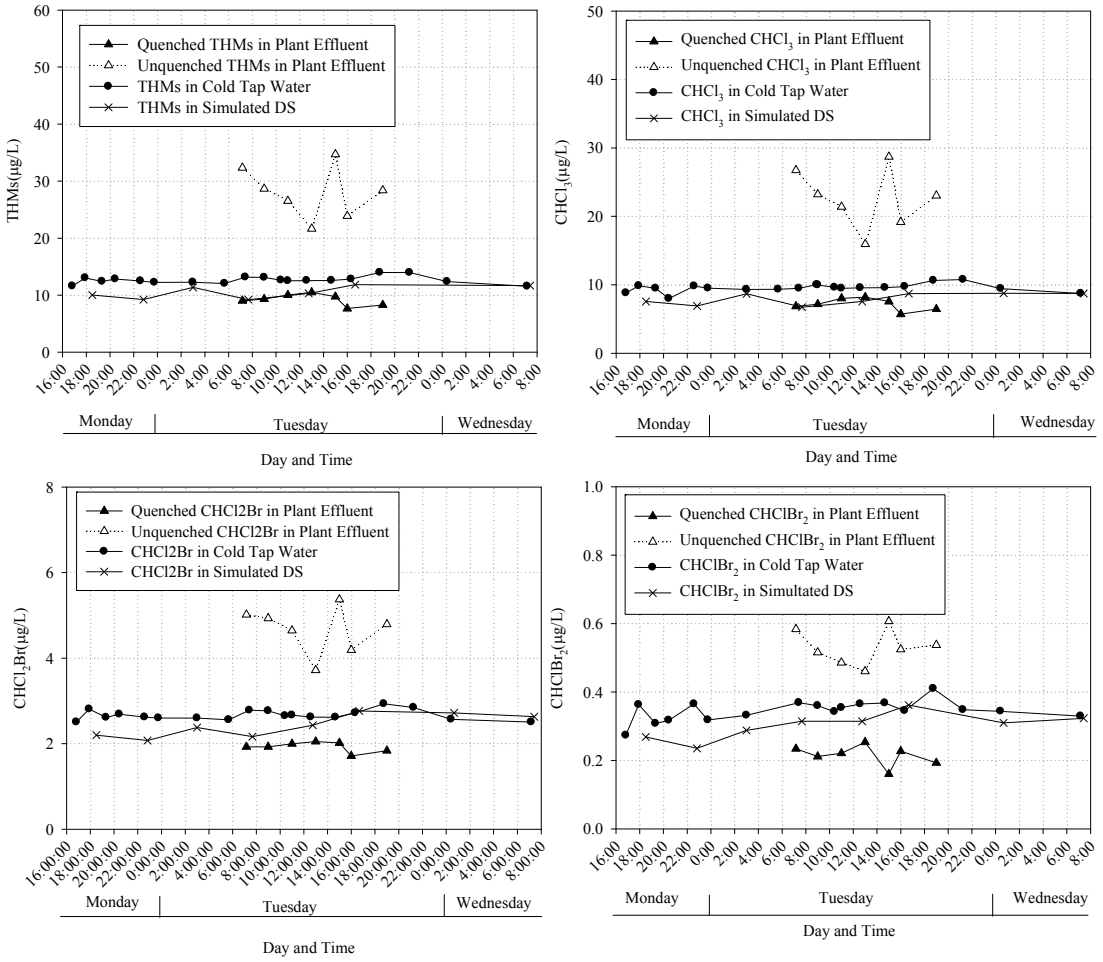

Figure 2.4 THMs in residential cold tap water, plant effluent and simulated distribution system

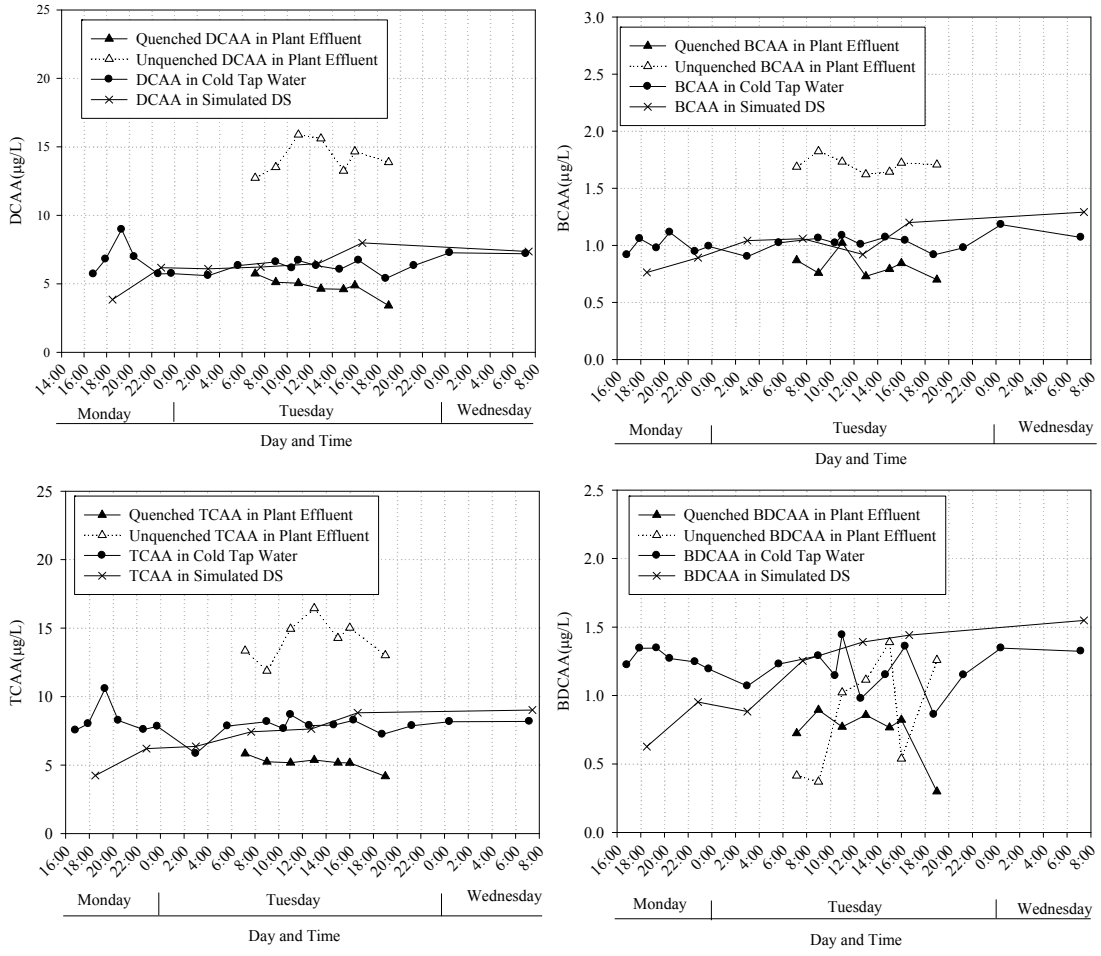

Figure 2.5 HAAs in residential cold tap water, plant effluent and simulated distribution system

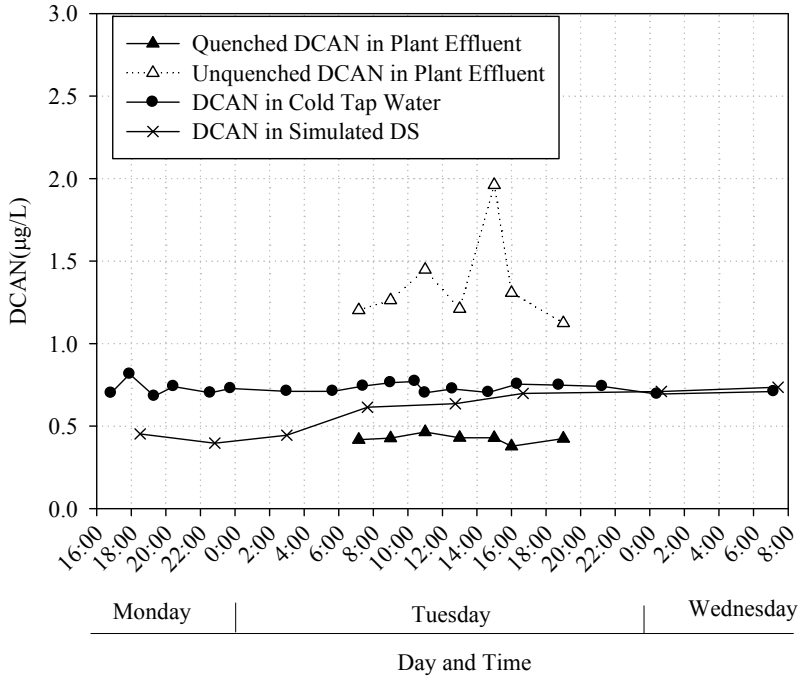

Figure 2.6 DCAN in residential cold tap water, plant effluent and simulated distribution system

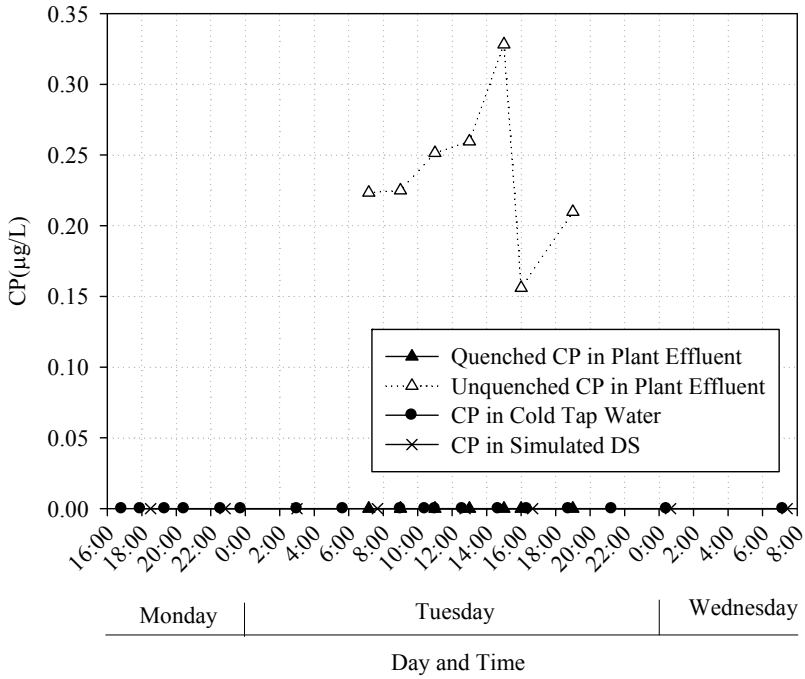

Figure 2.7 CP in residential cold tap water, plant effluent and simulated distribution system

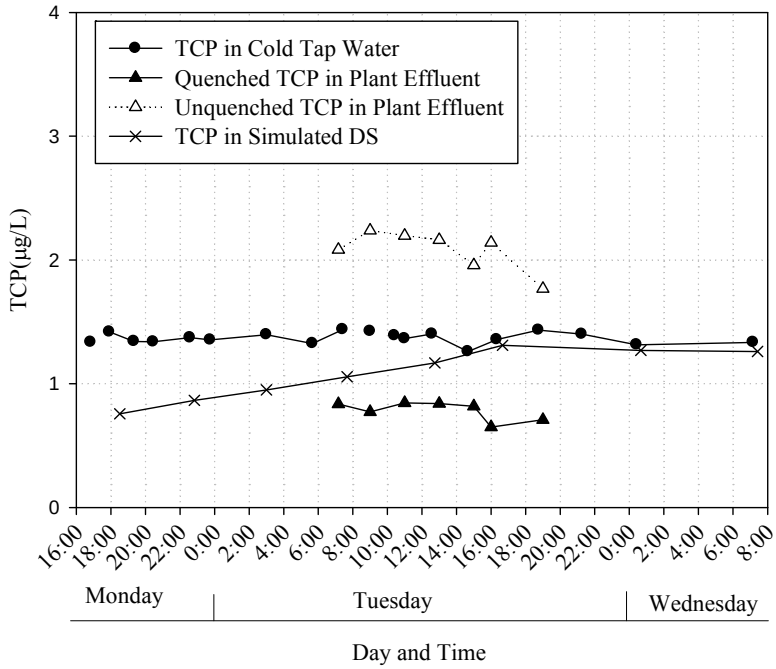

Figure 2.8 TCP in residential cold tap water, plant effluent and simulated distribution system

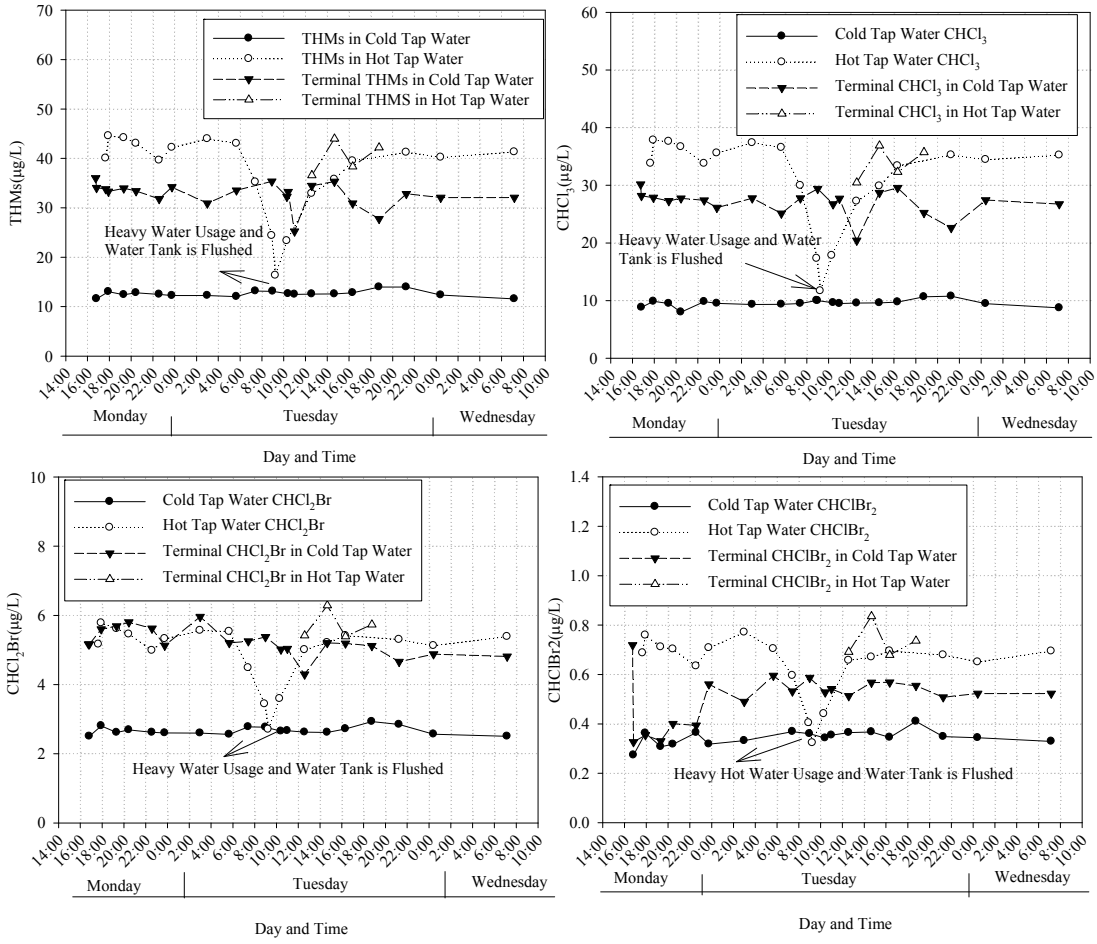

Figure 2.9 THMs in cold and hot tap water in the residential location

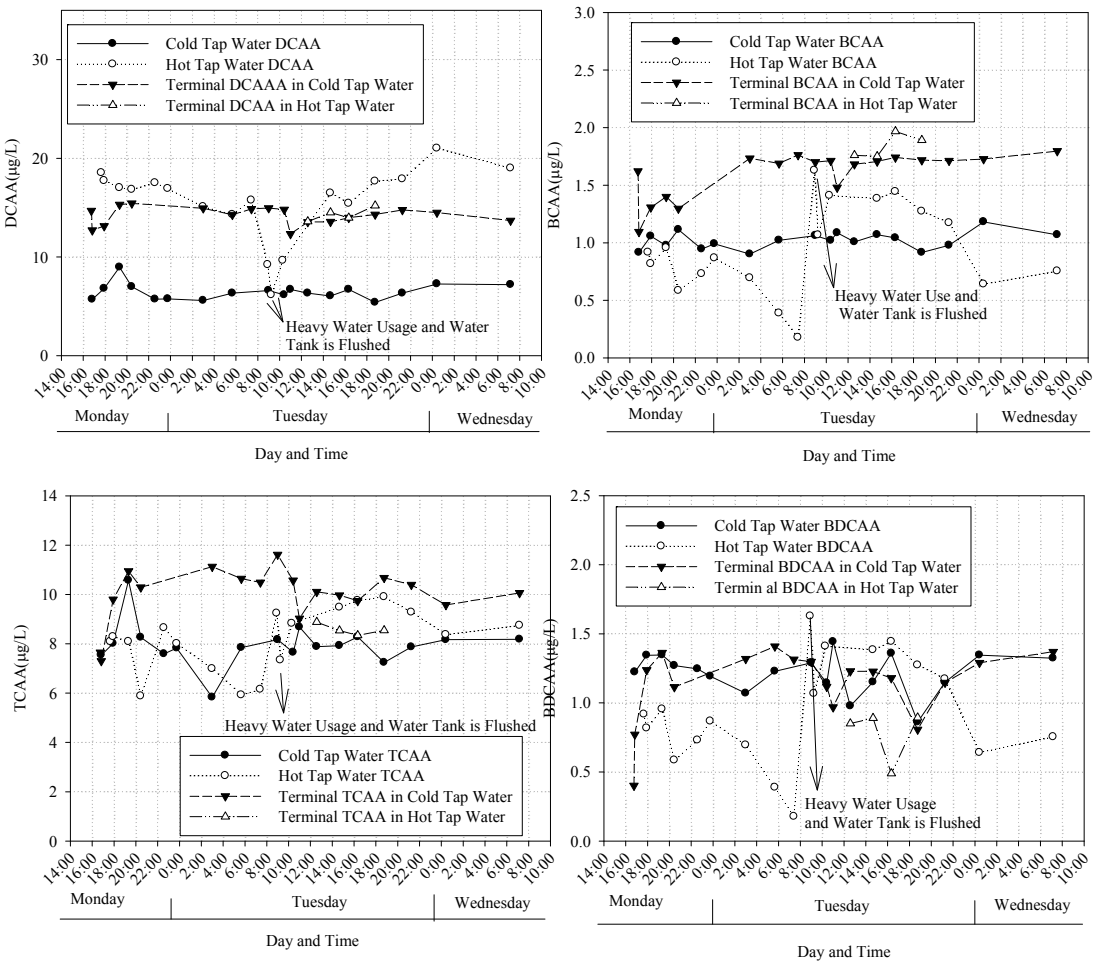

Figure 2.10 HAAs in cold and hot tap water in the residential location

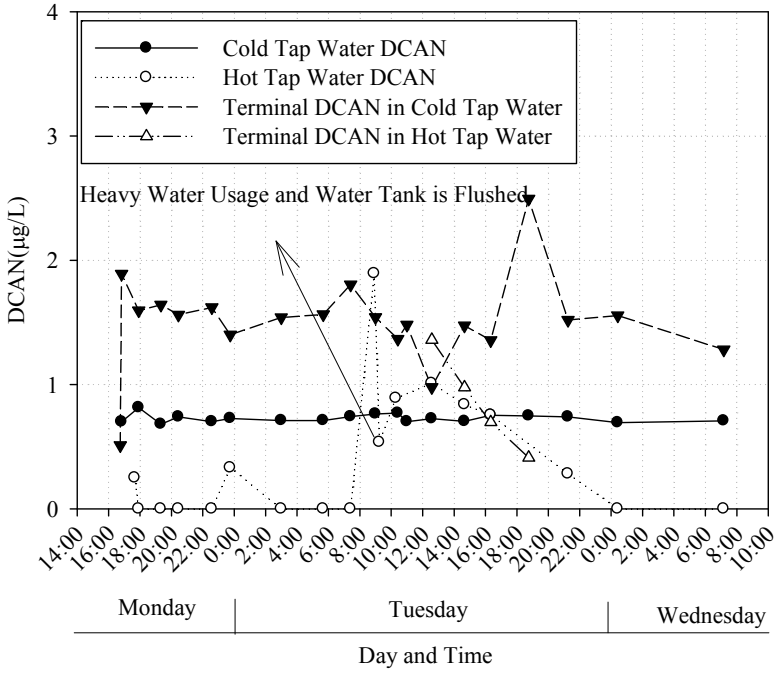

Figure 2.11 DCAN in cold and hot tap water at the residential location

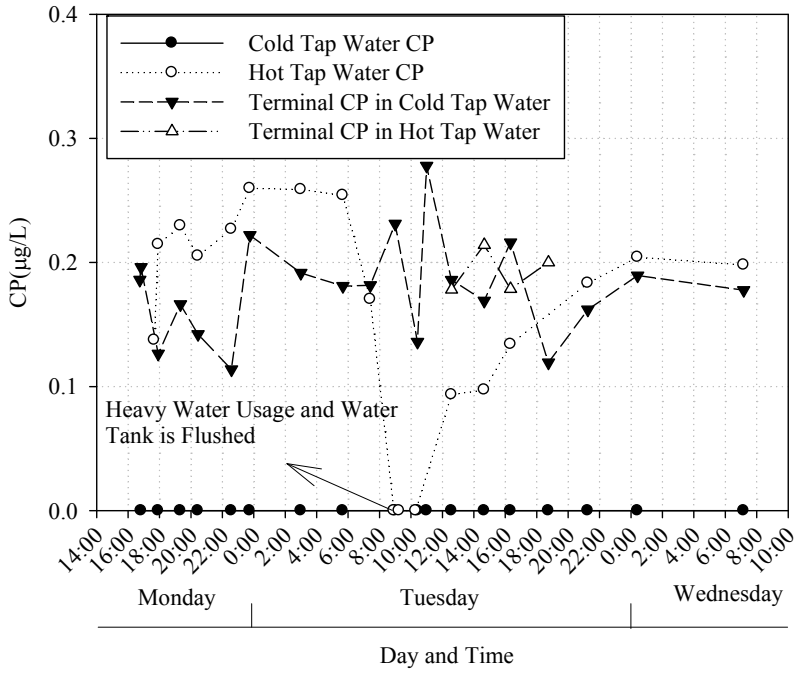

Figure 2.12 CP in cold and hot tap water at the residential location

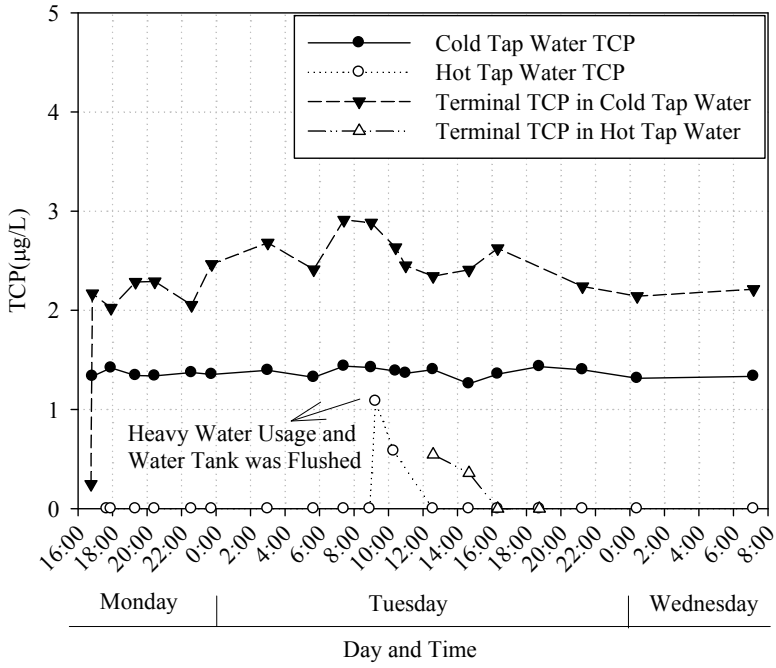

Figure 2.13 TCP in cold and hot tap water at the residential location

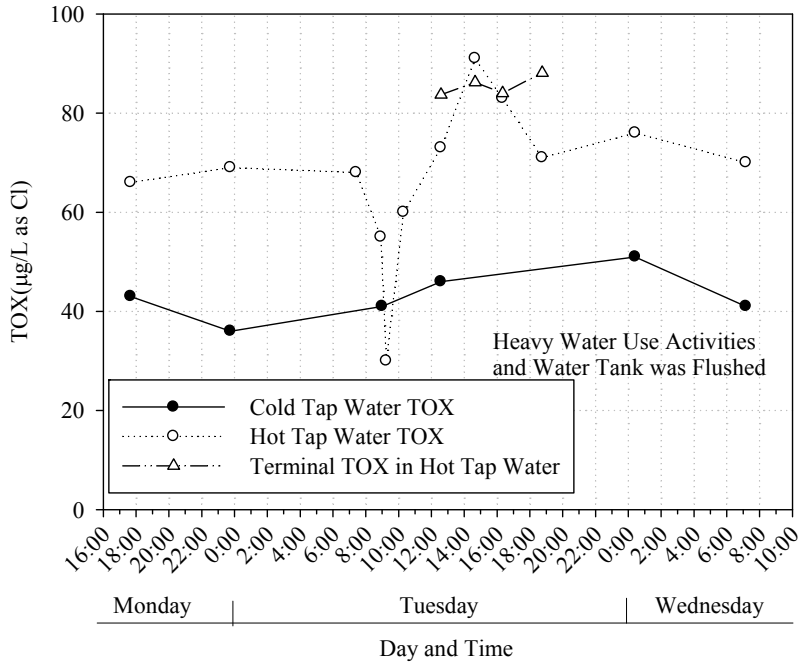

Figure 2.14 TOX in cold and hot tap water at the residential location

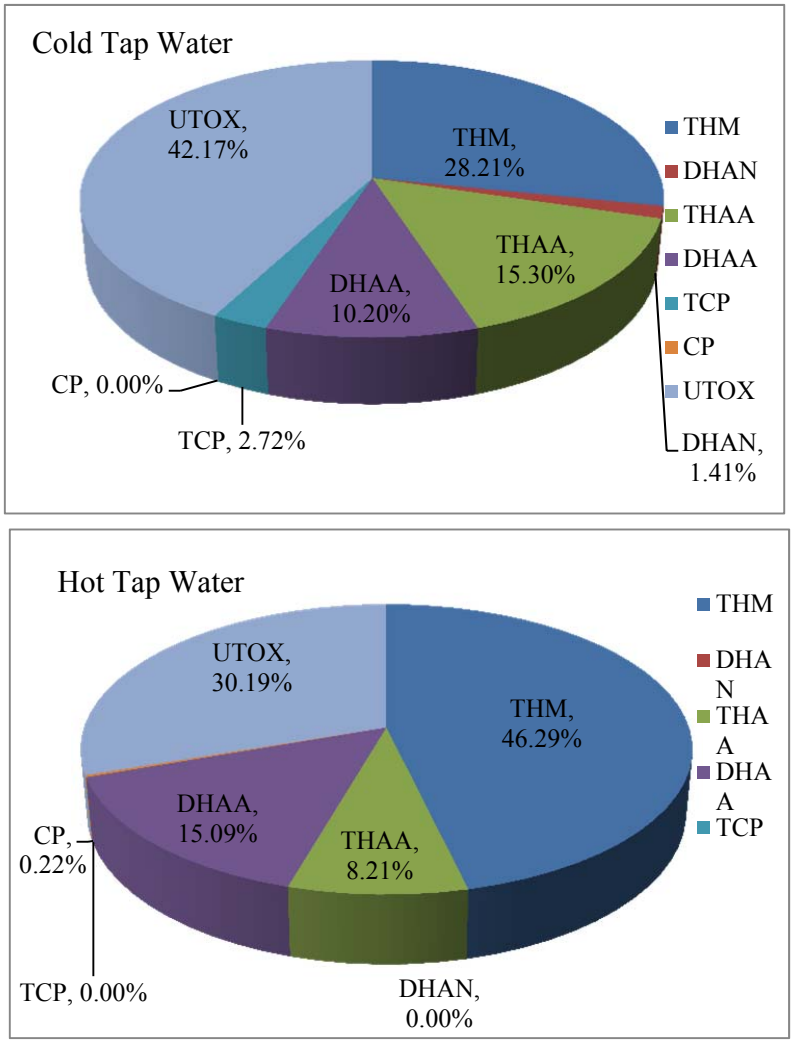

Figure 2.15 TOX distribution for cold and hot tap water samples*

*The pie chart is based on the data of hot and cold water samples collected on Monday 11:45pm.

CHAPTER 3

IMPACT OF WATER HEATERS ON THE FORMATION OF DISINFECTION BYPRODUCTS IN THE RESIDENTIAL PLUMBING SYSTEM*

(Prepared manuscript for submission to "Water Research")*

3.1 Abstract

The objective of this study is to examine the effect of water heaters and home heating scenarios on the formation and decomposition of disinfection byproducts (DBPs). This study investigated residential disinfection byproduct concentrations in cold and hot tap water from 18 houses equipped with either conventional water heaters or on-demand tankless heaters in a western Massachusetts water system. The system selected for this work uses conventional treatment with GAC filtration and free chlorine as the final disinfectant. As a companion study, lab incubation and different heating scenarios (short term heating & long term heating) were tested to understand the impact of stagnation and high temperature on DBP concentrations. The results from the field study revealed that the difference in water quality in hot tap water out of the two types of heaters was statistically significant for chlorine residual, total trihalomethanes (TTHMs), dichloroacetic acids (DCAA), dichloroacetonitrile (DCAN), trichloropropane (TCP) and chloropicrin (CP). The conditions in the conventional heater tended to cause consumption of more chlorine and led to more TTHMs, DCAA, CP formation and substantial decomposition of DCAN and TCP. The results from bench scale tests indicated that the concentrations of DBPs in the heated solution depended on the relative rate of DBP formation and decomposition. Little change in TTHM, HAA, and CP concentrations was

noted after short term heating, but substantial reductions of DCAN and TCP were observed in the bench-scale test. The long term heating yielded more TTHMs, DCAA, TCAA and CP, but led to lower levels of BCAA, BDCAA and significant reduction of DCAN and TCP.

Keywords

Disinfection byproducts, water heater, premise plumbing, degradation

3.2 Introduction

Disinfection byproducts (DBPs) are formed during disinfection of potable water through the reaction between natural organic matter (NOM) and chemical disinfectant. These compounds include the regulated DBPs, such as trihalomethanes (THMs), haloacetic acids (HAAs) and nonregulated DBPs, such as nitrosamines, haloacetonitriles (HANs), haloketones (HKs) and many more (Singer et al., 1994; USEPA, 2006; Richardson et al., 2007). The existence of the DBPs in water creates a health concern because many are potential carcinogens (Singer et al., 1994). In the United States, Maximum Contaminant Levels (MCLs) have been established for total trihalomethanes (TTHMs) and five haloacetic acids (HAAs) at 80 μg/L and 60 μg/L respectively. However, assessing human exposure to DBPs is a challenging task because of the high level of temporal and spatial variability and the multiple routes of exposure. In addition to ingestion of cold tap water, inhalation and dermal absorption during hot water use activities (such as showering, bathing, and washing dishes) are likely to be significant exposure pathways (Maxwell et al., 1991, King et al., 2004, Whitaker et al., 2003; Nuckols et al., 2005). Therefore, the occurrence level of DBPs in hot tap water needs to

be considered in assessing exposure. Bench scale heating studies have demonstrated significant changes in the levels of THMs, HAAs and some emerging DBPs at temperatures equal to or above 50^{o}C in closed systems (Weisel and Chen, 1994; Wu et al., 2001; Zhang et al., 2013). However, there remain many unanswered questions on the impacts of home heating as currently practiced on the full range of DBPs. The study by Weisel and Chen (1994) did not include HAAs, and they used a higher temperature (65^{o}C) than the temperature (55^{o}C) recommended by the World Health Organization (WHO) for pathogen control without excessive risk of scalding (WHO, 2007). Wu et al. 2001 used an open heating design and temperatures at the boiling point for tap water studies and a closed heating system for chlorinated synthetic water, but without chlorine residual. Zhang et al. 2013 used uniform formation conditions for different temperatures, but without combining low and high temperature incubation as normally occurs in the home.

Recent research has shown an increase in the concentration of THMs and HAAs from the distribution system to the consumer's hot tap, and this has been attributed to the long residence times in traditional hot water systems (Dion-Fortier et al., 2009; Chowdhury et al., 2011; Eyring et al., 2008) and simulated distribution system(Liu and Reckhow, 2013). The extended incubation of water that occurs in many domestic hot water systems may accelerate the reactions between chlorine and NOM (Saiq and Rodriguez, 2004). Therefore, unheated water samples collected in the distribution system or from premise plumbing may not represent the water that is important to assessing human exposure during showering and related hot water activities.

There are relatively few published studies regarding disinfection byproduct occurrence in premise plumbing and none has looked at the different types of home water

heaters regarding their impact on water quality. Tankless water heaters have been gaining popularity in the United States. Instead of having a storage tank like a conventional tank heater, tankless water heaters operate by heating and delivering hot water only as needed. As soon as the hot tap is opened, the water flow detector delivers a signal which leads to ignition of the burner (many of these are powered by natural gas). The water circulates through the heat exchange coil and it reaches the set temperature in about 5 seconds. Bagh et al., (2004) found substantial chlorine decay and bacterial growth in a recirculating hot water system that had a long period of high-temperature incubation. Brazeau (2012) compared the sustainability of three different hot water heaters, standard tank heaters with and without hot water recirculation, and an on-demand tankless water heater without hot water recirculation. He found that the disinfectant (monochloramine) decay was negligible in the on-demand tankless water heater, a bit higher in the tank heater without recirculation but very high in the tank heater with recirculation. However, no research has been conducted on the impact of different types of heaters on the full spectrum of DBPs (regulated and non-regulated). In this study, both cold and hot water samples were collected from homes using the two major types of water heaters in a recently constructed housing development in western Massachusetts. Bench scale heating experiments were also conducted as a companion study.

3.3 Material and Methods

3.3.1 Field sampling

The Rocky Hill cohousing community is located in the City of Northampton, MA, USA. The community has 28 dwellings on 6.5 acres of a 27.5 acre site off Florence road

in Northampton (Figure 3.1). Most of the units are duplexes built about the same time with similar infrastructure. Their drinking water is supplied from the Northampton filtration plant located in the neighboring town of Williamsburg. The Northampton water treatment plant has a design flow of 6.5 million gallons per day (MGD), and delivers 2.9 MGD on an average day to its 28,000 customers. Approximately 90% of Northampton's drinking water comes from three surface water reservoirs, and the remaining 10% comes from two groundwater sources. Since 2008, the operation of its water filtration plant, which is comprised of an upflow roughing filter (adsorption clarifier) and a granular activated carbon filter, has significantly reduced the amount of total organic carbon and DBPs in the system. As a result, the City's DBP levels are well below the maximum contaminant level (MCL) set by the Stage 2 Disinfectants/Disinfection Byproducts (D/DBP) Rule. Sodium hypochlorite is added to the filtered water prior to entering a 4 million gallon storage tank. As the water leaves the 4 million gallon storage tank, sodium carbonate is added for pH adjustment and corrosion control. Zinc orthophosphate is added as corrosion inhibitor. The free chlorine residual of the finished water from the water treatment plant was in the range of 1.1 to 1.3 mg/L at that time of the study.

Eighteen houses from the Rocky Hill community were selected in this study. Eight of these houses are equipped with "whole-house" tankless gas water heaters, and the remaining ten houses use conventional tank heaters. Hot and cold tap water samples were collected from each house over a two day period during the summer season. Each house was sampled once during that period, and the sampling was conducted early in the day, before any heavy hot water usage. Before sampling, the faucet aerator was removed and replaced with a custom-made threaded connector with attached tubing. This was

done to minimize volatilization of the THMs and other DBPs. In each case the cold tap water was sampled after being flushed for a period of 10 minutes and when the temperature of the water had stabilized. Cold water samples were collected in 300 mL chlorine-demand-free and glass-stopped bottles containing ascorbic acid and sodium azide. Just prior to capping, 2 drops of 6M sulfuric acid were added at the top of the liquid phase of the sample and the sample was capped and mixed thoroughly. Hot water samples were collected as soon as the temperature stabilized to avoid substantially depleting the hot water from the water tank. Eyring et al. 2008 reported the problem of bubble formation in their hot tap water sample container; therefore, in the current study, hot tap water samples were collected in serum "piston bottles" (described in Liu and Reckhow, 2013). All quenched water samples were placed in a cooler and transported to the UMass laboratory for DBP analysis. Temperature, chlorine residual and pH were measured in the field from both taps at the time of collection.

3.3.2 Bench scale test

Complementary bench-scale testing was conducted to evaluate the impact of heating on the DBP concentrations in the Northampton water. Drinking water was first chlorinated and incubated for different contact time at 20^{o}C to produce samples that could mimic cold tap water samples at different locations of the distribution system. Then those incubated samples were heated to 55^{o}C for either short term heating (10 minutes) or long term heating (24 hours), representing tankless and tank water heating systems, respectively.

A large sample of filter effluent from the Northampton water treatment plant was collected and transported on ice to UMass. Tests were performed on a 4-L sample of the water that was transferred to a 20°C incubator and equilibrated for 8 hours. The sample was then buffered at pH 8.0±0.2 with monopotassium phosphate and sodium hydroxide. A stock solution of sodium hypochlorite (Fisher Scientific, Pittsburgh, PA) was used for chlorination and it was standardized by the DPD ferrous titrimetric method (method 4500-Cl F; APHA et al.,1998). The chlorine dose was set at 5mg/L to reach a target chlorine residual of 0.5 mg/L after 24 h of heating.

The 4-L borosilicate bottles containing chlorinated samples were incubated at 20°C for different contact times (6 h, 24 h, 48 h, 72 h, and 96 h). At the end of each time period a "no heating" subsample was collected by transferring 300 mL to a chlorine-demand-free, glass-stopped bottle. A portion of this was used for the measurement of chlorine residual. The remaining portion was partitioned into glass vials containing ascorbic acid. A drop of concentrated sulfuric acid was added to each vial. The glass vials were sealed headspace-free for subsequent analysis of DBPs. After collection of these first "no heating" subsamples, the remaining volume was carefully partitioned into piston bottles, headspace-free with the piston in place at the fully depressed level so that it could rise as the water sample expanded during incubation in the warm water bath. Each bottle was sealed tightly and placed in the water bath pre-equilibrated to 55°C. It took about 30 minutes for each bottle to reach 55°C. At each of the two defined heating contact times (10 minutes and 24 hours); one of the "piston bottles" was withdrawn from the 55°C water bath, and were placed in the cold water bath (4°C) immediately for cooling down. The chlorine residuals were measured as soon as the samples were collected, and the

remaining samples were quenched and preserved as done for those first samples in preparation for subsequent analysis of DBPs.

3.3.3 Analytical method

In the field, chlorine residuals were measured by the N, N-diethyl-p-phenylenediamine (DPD) colorimetric method using a Hach field kit (Model CN-70). Temperature and pH were measured using a Fisher Scientific portable meter (AP85). In the lab, four chlorine and bromine containing THMs (THM4), dihaloacetonitriles (DHAN), chloropicrin (CP) and 1,1,1-trichloropropanone (TCP) were quantified by liquid/liquid extraction with pentane and by gas chromatography with electron capture detection (GC/ECD) according to U.S. Environmental Protection Agency (USEPA) method 551.1. Nine haloacetic acids (HAA9) were analyzed by liquid/liquid extraction with methyl-tertbutyl-ether (MTBE), followed by derivatization with acidic methanol and by GC/ECD based on USEPA method 552.2. Total organic carbon analysis was performed by the high temperature combustion method using a commercial TOC Analyzer (TOC-VCPH, Shimadzu).

3.4 Results and Discussions

3.4.1 Field sampling

The temperatures of the cold tap water samples were in the range of 18 to 22^{o}C, which is typical of the Northampton water during the sampling period (July, 2010). The water temperature exiting the tankless water heaters was factory set at 55^{o}C in order to prevent scalding, and on-site measurements showed it to be in the range of 50 to 60^{o}C.

The pipes exiting the tankless water heater were cool to ambient, whereas the pipes of exiting the standard tank heater were warm because of heat conduction from the hot tank. Nevertheless, there was no significant temperature difference between the water collected from the different types of heaters. Little variations of TOC, pH, UV-254 were found for cold and hot tap water. The water quality of the finished water and the tap water is summarized in Table 3.1.

3.4.2 Chlorine residual

In tap water, chlorine decay is mainly attributed to reactions with natural organic matter. The water age, presence of dissolved reactive substances (e.g., natural organic matter, sulfide, and ferrous ion) and reactive pipe materials could also affect the chlorine decay in a distribution system (Roseman et al., 1994, Mutoti et al., 2007). The water ages from the plant to the sampled homes at the Rocky Hill Cohousing community are essentially identical and in the range of 24 h to 96 h. The chlorine residuals in the cold tap water were in the range of 0.23 to 0.52 mg/L for the sampled homes. As shown in Figure 3.2, the hot water results indicate a significant decrease in the chlorine residual for those homes using a tank water heater, but only a marginal decrease for homes with a tankless heater.

The loss in chlorine residual upon heating is unlikely to be caused by thermal decomposition (i.e., reaction with water or disproportionation) as sodium hypochlorite was found to be stable around 50^{o}C (Gambarini G et al., 1998). Rather, it seems mostly likely to be caused by a higher rate of reaction with NOM and other reduced solutes in the water. The longer exposure to the high temperatures makes the standard tank heating

system more vulnerable to loss of chlorine residual. There was one home with a tank heater that had a relative high chlorine residual in the hot water and this seems to have been the result of heavy use of the hot water just prior to sample collection.

3.4.3 Trihalomethanes

Since the organic content of the finished water was low, the yield of total trihalomethanes (TTHMs) in the consumer's cold tap water was well below the MCL established by the Stage 1 Disinfectant/Disinfection Byproducts Rule (Figure 3.3). The concentration of TTHMs was in the range of 17.6 to 27.5 µg/L for the cold tap water samples whereas the concentration of TTHMs was in the range of 21.9 to 42.5 µg/L for the hot tap water samples, exceeding those of the cold tap water up to 120%. All of the significant differences in TTHMs between hot and cold water were observed in homes that have traditional tank water heating systems. Little TTHM variations between cold and hot tap water samples were observed in homes that had tankless water heaters. The individual trihalomethane exhibited very similar trends as the TTHMs; therefore the individual trihalomethane species are not discussed.

3.4.4 Other neutral DBPs (DCAN, CP, TCP)

The occurrence of three other neutral DBPs, dichloroacetonitrile (DCAN), chloropicrin (CP) and 1,1,1-trichloropropanone (TCP) in the cold and hot tap water are shown in Figure 3.4. The concentrations of dichloroacetonitrile, the only chlorinated haloacetonitrile detected, were comparable for all cold water samples as well as for hot water samples from tankless systems. There was a large decrease in the DCAN concentration for hot tap water coming from traditional tank water heating systems. A

very slight increase in DCAN concentration in the hot tap water out of the tankless water heater was observed. Dichloroacetonitrile is known to undergo a base-catalyzed decomposition (Trehy and Bieber, 1981; Reckhow et al., 2001). In the presence of a chlorine residual, the concentration of DCAN was found to increase initially and then decreased because of auto-decomposition (Zhang et al., 2013). Exner et al. (1973) suggested that DCAN would transform to dichloroacetamide (DCAM) which would undergo rapid hydrolysis to form dichloroacetic acid (DCAA) in the absence of free chlorine. Therefore, the decomposition of DCAN could also contribute to the DCAA increase observed in the hot tap water, albeit by a rather small amount. Chloropicrin was not detected in neither the cold tap water nor in the hot water from the tankless heaters. The presence of chloropicrin in the hot tap water samples from conventional tank water heating systems suggests that high temperatures enhances the formation of chloropicrin more than it's degradation. The case for trichloropropanone was much like that for DCAN. There was a slight decrease in TCP in the hot water samples out of the tankless water heaters. In contrast, significant loss of TCP was observed in the hot water samples out of the conventional tank water heating systems.

3.4.5 Haloacetic acids

Low precursor levels in this water kept the HAA5 levels well below the MCL of 60 μg/L set by USEPA (Figure 3.5). Among the HAA9 species, only DCAA and TCAA were detected for the samples collected at the cohousing community. The concentration of DCAA was clearly higher in the hot tap water coming from conventional hot water tank systems as compared to the cold tap water at the same sampling locations. In

contrast, the difference was minor in most of the sampled homes equipped with tankless heating systems. Conversely, regardless of the water heater types, TCAA concentrations in cold and hot taps water were comparable in most of the sampling locations. Although bromochloroacetic acid (BCAA) and bromodichloroacetic acid (BDCAA) are not currently regulated by US EPA, they were detected and are discussed as they are believed to be more genotoxic than their chlorinated analogs (Plewa et al., 2004). There were very minor differences in BDCAA concentration between the cold and hot tap water samples for homes equipped with tankless heaters (Figure 3.5). However, significant reduction of BDCAA was observed in the hot tap water samples for homes equipped with traditional water heaters. Thermal decarboxylation of BDCAA was found to occur faster than with TCAA, forming bromodichloromethane with a first-order rate constant of 0.234 day-1 at 50^{o}C (Zhang and Minear, 2002). Therefore, the net drop in BDCAA level in tank water systems may be attributed to accelerated decomposition superimposed over little change for the trihaloacetic acids as a group (i.e., the effect noted for the more stable member, TCAA). As shown in Figure 3.5, the concentration of BCAA in the cold tap water was comparable for all the sampled homes. The tiny increase in BCAA concentration noted in the hot water of home with tank systems may mirror a similar relative increase seen for the more abundant DCAA.

Of the four HAAs reported in this study only the dihaloacetic acids (DCAA, and possibly BCAA) exhibited behavior that was similar to the THMs (i.e., higher levels in hot water from tank systems), whereas the trihaloacetic acids (TCAA and BDCAA) showed the opposite behavior. Differences between these two groups extend from differences in precursor origins to differences in formation and degradation kinetics (e.g.

Reckhow & Singer, 2010). Others have found the concentration of HAA5 to be significant higher in hot tap water than in cold tap water (Chowdhury et al., 2011). Eyring et al., 2008 found that HAA5 tended to increase only a minor amount in a distribution system that used chloramine as the final disinfectant (winter-spring seasons). Note that these authors only reported on the sum of haloacetic acids whereas the individual haloacetic acid are compared in this work. It was found that the precursors of DCAA are overall less hydrophobic than TCAA precursors and their formation kinetics are quite different (Liang and Singer, 2003). Therefore, TCAA and DCAA results may exhibit significant differences in occurrence. Also, chlorine decay in hot water tanks could lead to the growth of thermophilic bacteria in these tanks and the associated plumbing (Bagh et al., 2004). Bacteria collected from water distribution systems were capable of degrading haloacetic acids (Williams et al., 1996). A thermophilic dehalogenase enzyme (L-HADST) was found to be active and capable of degrading haloacids at temperatures above 50 ^{o}C (Bachas-Daunert et al., 2009). Nevertheless, there are no reports of organisms identified in home hot water systems that have been shown to be responsible for biodegradation of DBPs in these systems. Additionally, TCAA was found to be not readily biodegradable by different enrichment culture (McRae et al., 2004; Zhang et al., 2009). Therefore, it is unlikely that a large amount of TCAA could have been biologically degraded in the hot water tank. The existence of competitive formation pathways between TCAA and THM formation may be a more reasonable explanation for the small difference of TCAA between cold and hot tap water samples. The work of Reckhow and Singer (1985) suggests that the substantial THM and THAA formation

occur through competitive pathways including hydrolysis and oxidation. The low level of chlorine residual may restrict the formation of TCAA.

3.4.6 Significant testing of field data

In order to assess the statistical significance of the impact of water heaters on water quality parameters, paired t tests were conducted on the chlorine residual and DBP concentrations in hot and cold tap water for the two types of water heating systems. The results were listed are Table 3.1. A p-value above 0.05 is considered to be an indication of no significant difference. The paired t-test confirmed that the chlorine residual, DCAA, TTHMs, CP, TCP, DCAN concentration changes between the hot water from conventional water heaters and cold tap water were significantly different than those changes for tankless heater. The concentration changes for TCAA, BCAA, and BDCAA were not considered significant.

3.4.7 Bench scale testing

The collected water used for bench scale testing was the finished water without chlorine addition at the plant. As shown in Table 3.1, the Northampton finished water was very low in organic carbon and had a low specific UV-254 value (SUVA), which is indicative of NOM with a hydrophilic character, not heavily impacted by condensed tannins or lignin.

The various ambient temperature incubation times were intended to represent different water ages in the distribution system. The short term heating (10 min) and the long term heating (24 h) are used to represent the heating processes for the two types of hot water heating systems, tankless on-demand systems and conventional tank systems,

respectively. Figure 3.6 shows the TTHM formation and chlorine residual profile during the incubation and the heating process. There were substantial chlorine residuals throughout the 4 day 20 ^{o}C incubation time. The short term heating caused a small increase in the consumption of chlorine at each ambient incubation time. After 24 h heating of the samples, the chlorine residuals had dropped to 0.6 mg/L or below. The TTHM formation increased with increasing ambient reaction time during the 20 ^{o}C incubation. After 10 minutes of heating, the TTHM concentrations increased a very small amount. However, after 24 hours of heating, the TTHM levels increased by a factor of 2.5 to 4.9 compared to the samples without heating.

The rapid formation of TTHMs at the early stage of chlorination is often attributed to fast reactive NOM sites (Amy, 1998). Zhang et al. (2013) demonstrated that the formation of chloroform increased rapidly in the early stages regardless of temperature. It is generally accepted that reaction rates increase with increasing temperature. However, since heating was initiated midway through the overall chlorine contact time, the short term (10 min) heating did not significantly change the formation of trihalomethanes as the fast reactive NOM sites had already been consumed. After 24 h of heating, further formation of TTHMs in the water samples (by reaction with slower NOM sites) increased with increasing incubation time. However, less additional formation of TTHMs with increasing reaction time was expected as the chlorine residual decreased with increasing incubation time. There might be some unknown halogenated byproducts that accumulate from ambient-temperature incubation that can react with the chlorine residual to form additional trihalomethanes at a greater temperature. Simultaneous thermal decarboxylation of trichloroacetic acids could also contribute to the

increase in THMs (Zhang and Minear, 2002; Verhoek, 1934), but this might be a minor contribution.

Figure 3.7 shows that the DCAA and TCAA concentrations increased with increasing incubation time, whereas the BCAA and BDCAA reached a maximum at about 48 h. Since the water contained a low bromide concentration, the concentrations of BCAA and BDCAA were quite low. There was little change in DCAA and TCAA concentrations in the water after the short term heating. After 24 h of heating, the DCAA concentration tripled for samples with 24 h and 48 h ambient incubation time, whereas it doubled for samples with 72 h and 96 h ambient incubation time. The increase in TCAA concentration after 24 h heating was in the range of 31% to 66%, significantly lower than the increase of TTHMs and DCAA. This is a bit different from the field results where no significant difference in TCAA concentrations was noted between cold and hot tap water. This may be attributed to the low chlorine residual in the field; and the alkaline condition favors the chloroform formation pathway (Reckhow and Singer, 1985, Liu and Reckhow, 2013). Noticeable reductions of BDCAA and BCAA were observed after 24 h heating. The first order decomposition rate constants at 50^{o}C for BDCAA and BCAA were found to be 2.7×10-6 s^{-1} and 1.6×10-6 s^{-1} respectively (Zhang and Minear, 2002; Lifongo et al., 2010). The reduction appeared to be smaller for the shorter ambient incubation as continuing formation was probably occurring at the shorter incubation times.

The effect of incubation time on the formation of CP, DCAN, and TCP is shown in Figure 3.8. The concentration of CP in the ambient temperature incubated water was quite low starting at 0.04 µg/L and slowly increasing to 0.12 µg/L. The 10 min heating time caused a modest increase in the concentration of CP. However, the 24 h heating time

resulted in very substantial increases in the concentration of CP, elevating the final concentration by a factor of 3.7 to 7.8. This is not consistent with previous research done by Weisel and Chen, 1994, who found that the CP concentration in the heated solution was unchanged during the 8 h heating. However our bench results were consistent with our field study, which found a significant amount of CP in the hot tap water, whereas it was below the detection limit for the cold samples. In many drinking waters, the concentration of CP is low and at or near the detection limit (typically ~0.16 µg/L; Krasner et al., 1989; Yang et al., 2007). The observation that long term heating could significantly increase the levels of CP is troubling, as this family of compounds is considered among the more toxic of the DBPs (e.g., Plewa et al., 2004). The concentration of DCAN increased with increasing incubation time and leveled off at 48 h. Short term of heating led to an increase in DCAN concentration for samples with shorter ambient incubation times, but it decreased the DCAN concentration for samples with longer ambient incubation times. Dichloroacetonitrile is not stable in aqueous solution and can decompose, especially under alkaline conditions (Stevens et al., 1989; Reckhow et al., 2001). Increasing the temperature also enhanced the decomposition rate of DCAN (Nikolaou et al., 2000). Therefore, the occurrence of DCAN depends on the relative rates of formation and decomposition. After 24 h of heating, a substantial amount of DCAN was lost. Trichloropropanone decomposed quickly with time under pH 8 after its fast initial formation. The short term heating accelerated the decomposition of TCP with a reduction of 40% to 67%. After 24 h of heating, only very low levels of TCP were detected.

3.5 Conclusions

The results of this study showed that the DBPs could change significantly during in-home heating of drinking water. On-demand heating without long-term storage of hot water resulted in little or no change in the DBP formation profile. In contrast, the longer-term storage of hot water (as typically occurs with conventional tank water heating systems) does yield more trihalomethanes and haloacetic acids when the influent water has a chlorine residual. In addition to the high-temperature incubation time, the increase of TCAA in the water heater also depends on the chlorine residual. A significant increase of TCAA after incubation in a water heater may not occur if the cold tap water contains a low chlorine residual. The incubation of tap water in a tank water heater could also lead to a significant increase in CP. The concentration of DCAN is the result of a competition between formation and decomposition. An increase in DCAN concentration after short term heating was observed whereas a substantial decrease in DCAN was the outcome after long term heating. The TCP concentration was found to decrease with both the incubation time and higher temperature. Overall, the different types of heaters and different heating scenarios could lead to different DBP formation profiles. Therefore, as tankless water heaters are becoming popular, their specifics should be considered by researchers when the assessments of human exposure to DBPs are conducted.

Table 3.1 Water quality for finished water of Northampton water supply system

	Finished Water	**Cold Tap Water**	**Hot Tap Water**
TOC, mg/L	0.96	0.89(0.12)*	1.01(0.11)*
DOC, mg/L	0.78	-	-
UV-254, cm^{-1}	0.006	-	-
SUVA, L/mg/m	0.8	-	-
pH	7.52	7.56(0.017)*	7.55(0.027)*
Temperature, ^{o}C	17	20.3(0.97)*	53.3(2.97)*
		Significant test results of Delta value $^{\Delta}$ for the difference between two types of water heaters	
		P value	Significant or not
Cl_2 residuals, mg/L	1.02	0.00002	Yes
DCAA, µg/L	7.23	0.0018	Yes
TCAA, µg/L	6.76	0.398	No
BCAA, µg/L	1.52	0.0639	No
BDCAA, µg/L	0.55	0.22742	No
TTHMs, µg/L	20.57	0.04	Yes
CP, µg/L	BDL$^{\Delta}$	0.00775	Yes
TCP, µg/L	0.36	0.003449	Yes
DCAN, µg/L	2.36	0.0000002135	Yes

*: The value in the bracket is the standard deviations. Δ: BDL: Below detection limit

$^{\Delta:}$ Cl_2 o DBPs difference between the cold and hot tap water in the field

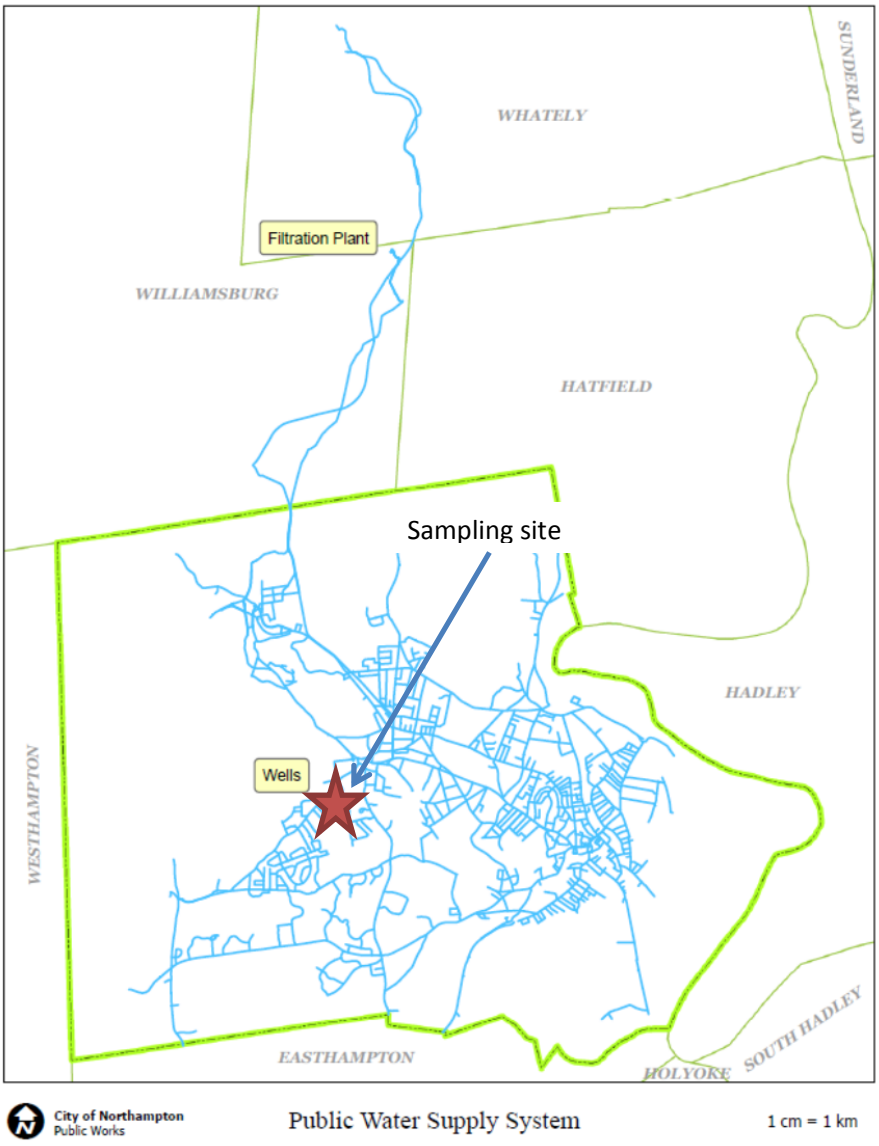

Figure 3.1 The location of Rocky Hill Cohousing Community

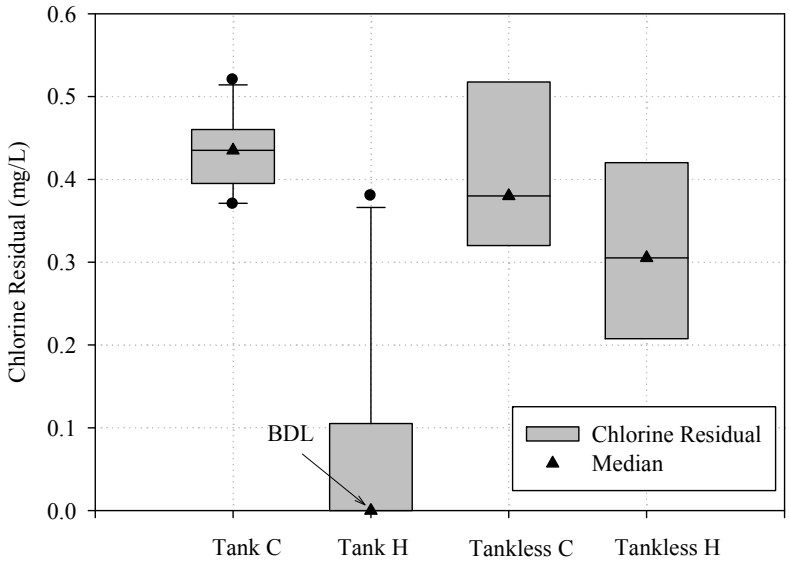

Figure 3.2 Chlorine residual in the cold (C) and hot (H) tap water from homes with Tank and Tankless water heating systems

BDL: Below detection limit (detection limit for chlorine residual is 0.02mg/L);

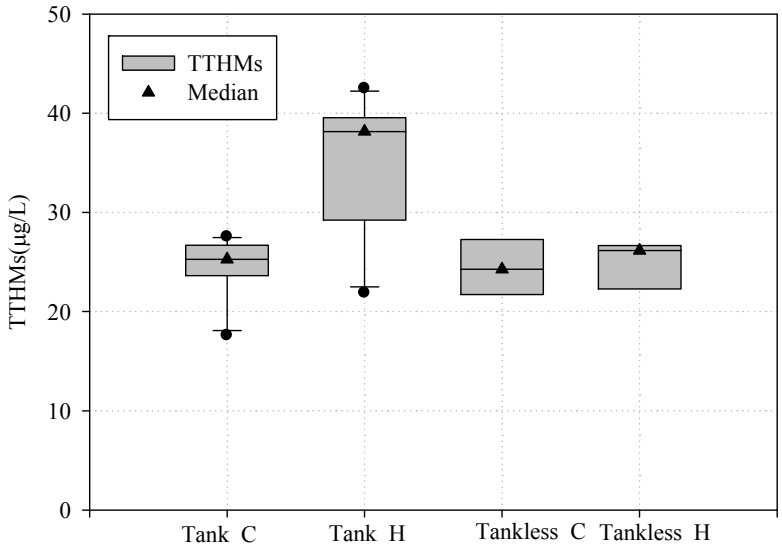

Figure 3.3 TTHMs in the cold (C) and hot (H) tap water from homes with Tank and Tankless water heating systems

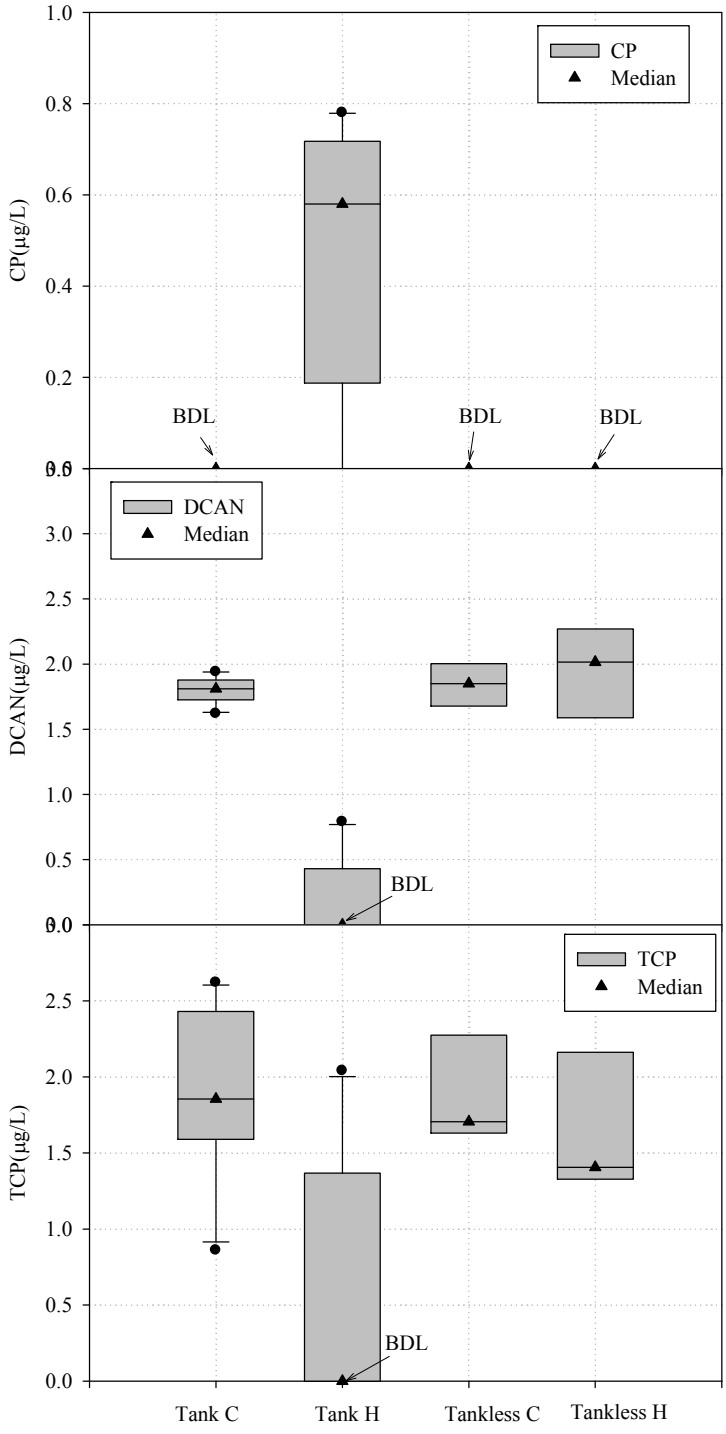

Figure 3.4 CP, DCAN and TCP in the cold (C) and hot (H) tap water from homes with Tank and Tankless water heating systems

BDL: Below detection limit Dection limit for DCAN, CP, and TCP are 0.06, 0.02 and 0.10 µg/L respectively.

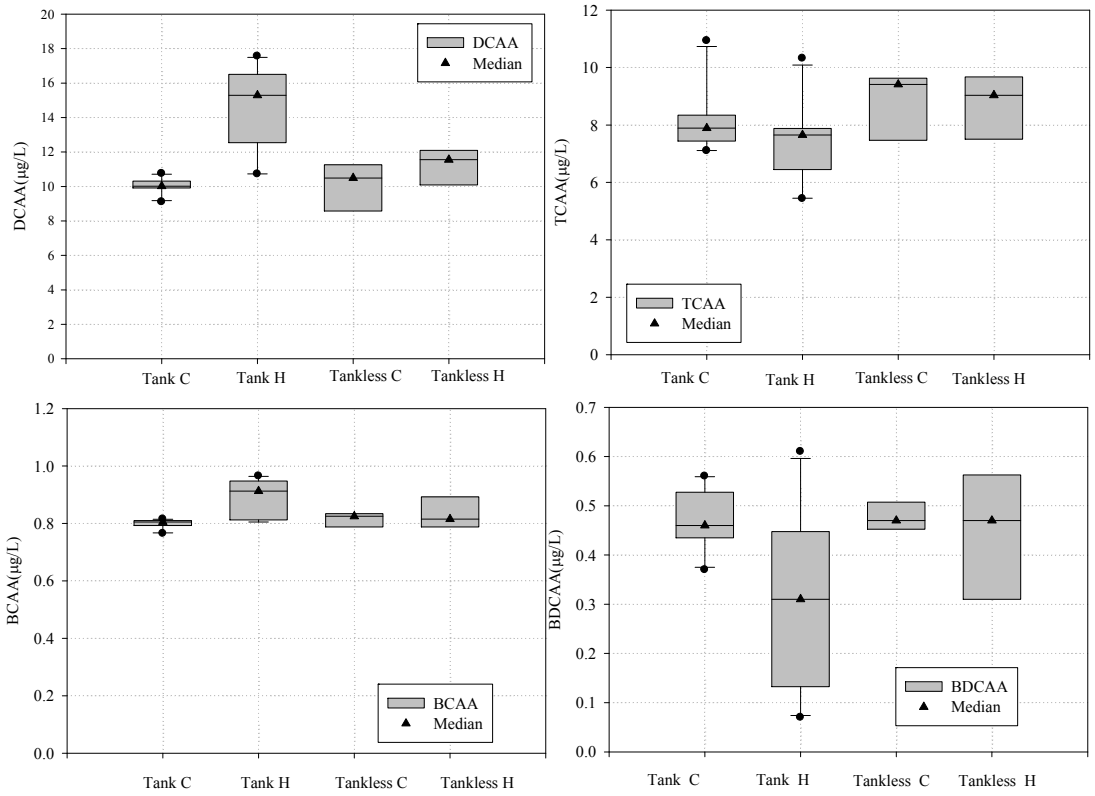

Figure 3.5 Haloacetic acids in the cold (C) and hot (H) tap water from homes with Tank and Tankless water heating systems

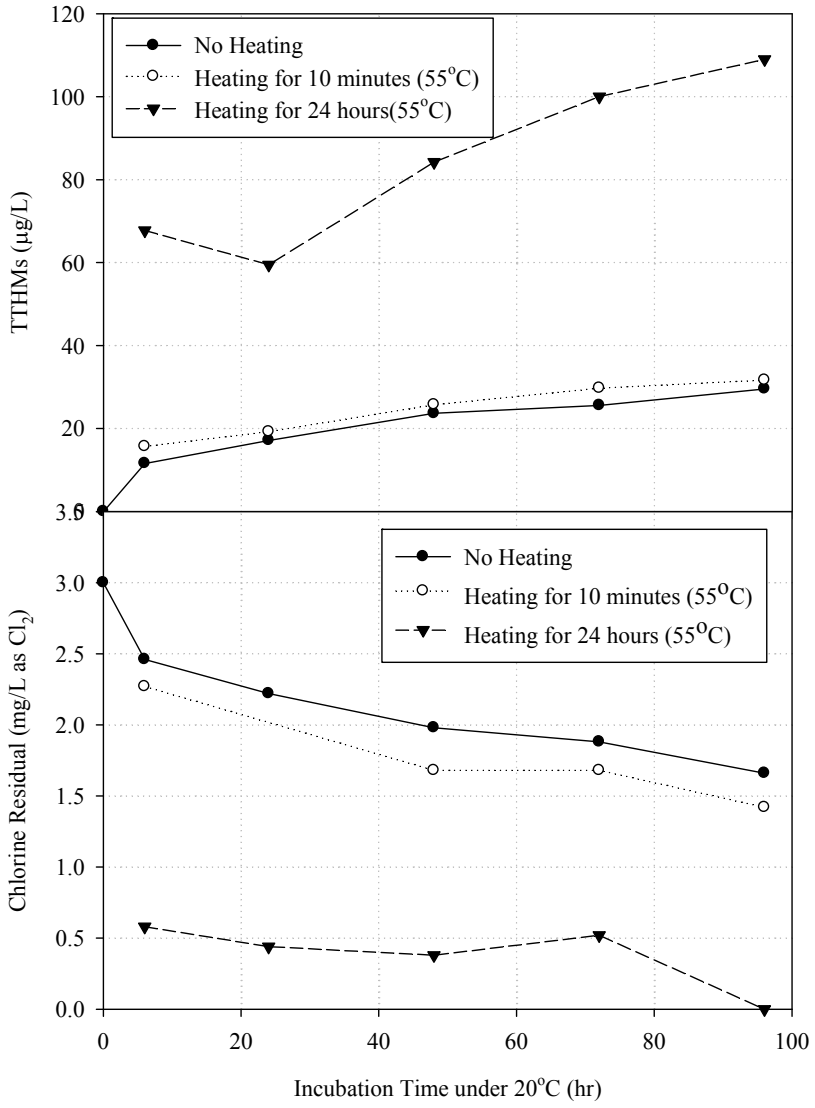

Figure 3.6 Time dependent formation of TTHMs during the heating process (pH=8± 0.2)

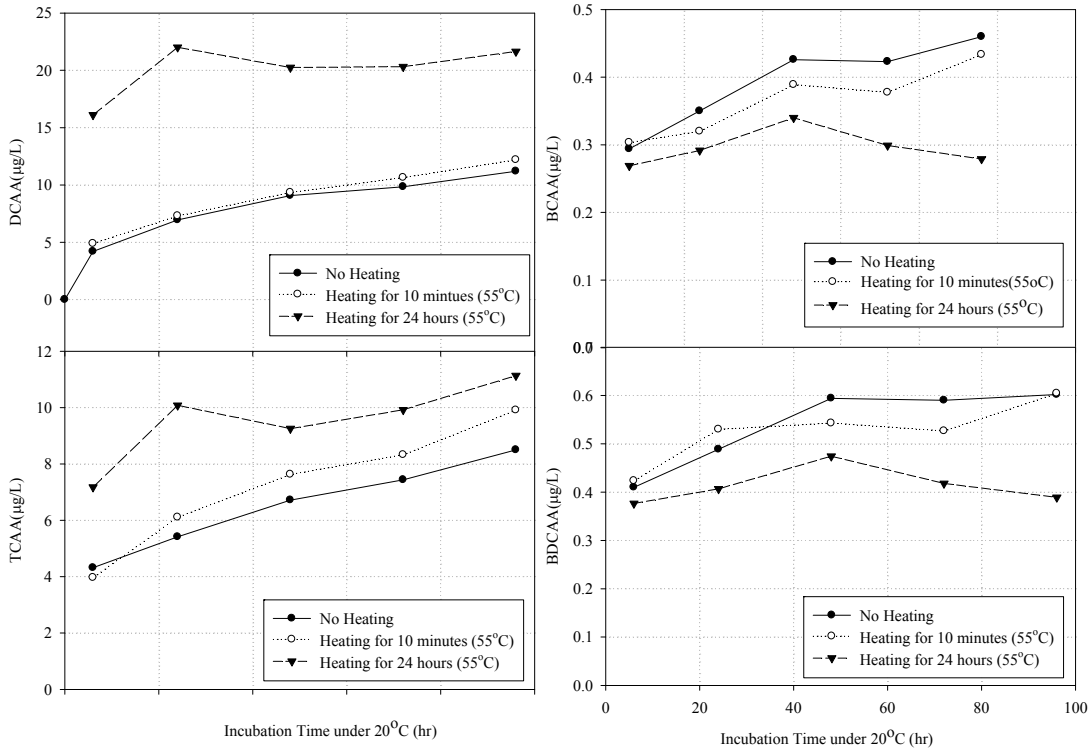

Figure 3.7 Time dependent formation of haloacetic acids during the heating process (pH=8± 0.2)

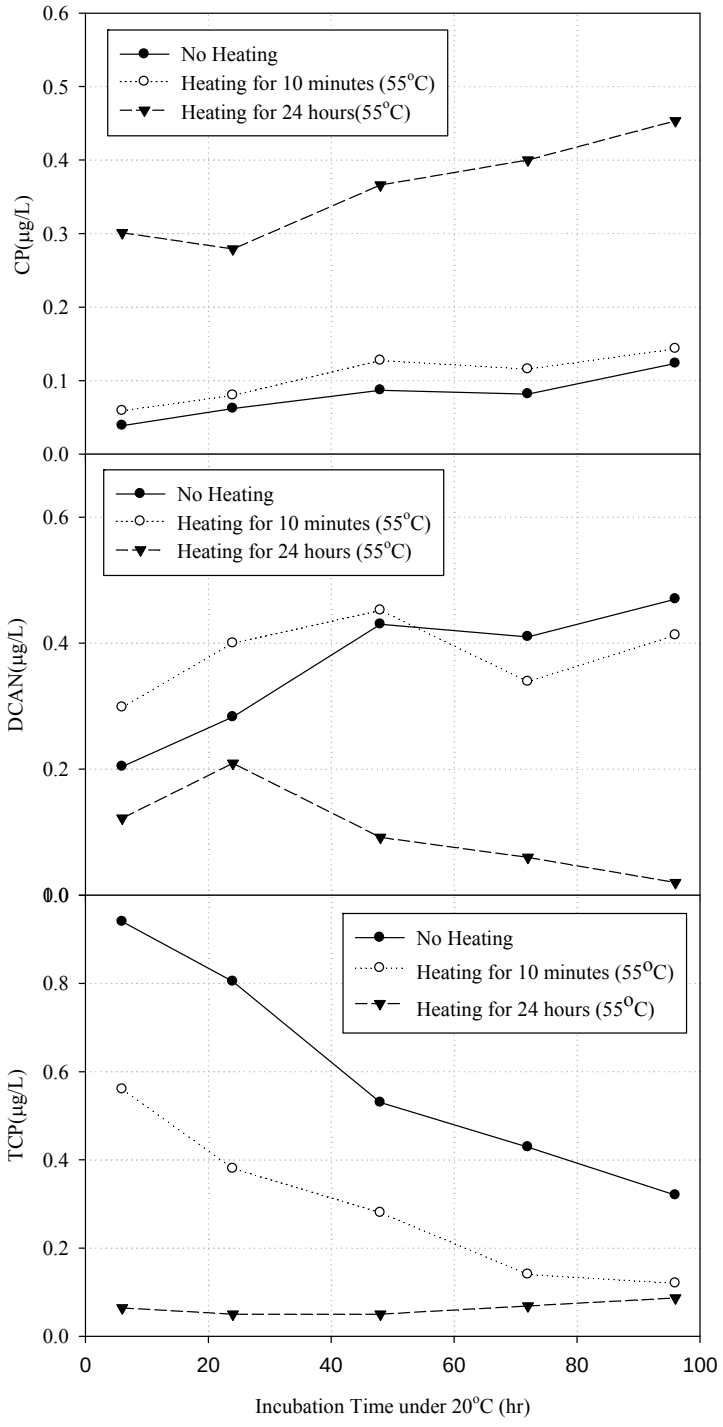

Figure 3.8 Time dependent formation of CP, DCAN, TCP, DCP during the heating process (pH=8± 0.2)

CHAPTER 4

DBP FORMATION IN HOT AND COLD WATER ACROSS A SIMULATED DISTRIBUTION SYSTEM: EFFECT OF INCUBATION TIME, HEATING TIME, PH, DOSE, AND INCUBATION TEMPERATURE

(an extended version of the paper published in *Environmental Science and Technology**)

4.1 Abstract

Despite extensive research on DBP formation and occurrence in tap water, limited research has been conducted on DBP concentrations in home water heaters. This is especially important as health effects research on DBPs has increasingly recognized the importance of home exposures other than ingestion of cold water. In this study, we investigated DBP formation in heated water across a range of simulated distribution system water ages. The primary objective of this study was to evaluate the impacts of the distribution system water age, in-home heating time, pH, chlorine dose and temperature on DBP concentrations. The results of this study demonstrate that DBP concentration profiles in heated water were quite different from the DBP concentrations in the cold tap water. Chloroform concentrations in the heated water remained constant or even decreased slightly with increasing distribution system water age, despite the fact that its levels always increased with water age in the cold water. The amount of dichloroacetic acid was much higher in the heated water than in the cold water; however, the maximum

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*: Liu, B.; Reckhow, D. A. DBP formation in hot and cold water across a simulated distribution system--Effect of incubation time, heating time, pH, chlorine dose, and incubation temperature. *Environ. Sci. Technol*. **2013**, 47 (20), 11584–11591.

levels in heated water with different distribution system water ages did not differ substantially. The levels of TCAA in the heated water were similar to the TCAA levels in 1-1-DCP and TCP) were observed after the tap water was heated for 24 h. For tap water with lower water ages, there were significant increases in DCAN, CP and DCP after a short period of heating. Regardless of water age, loss of TCP in the heated water was observed throughout the entire heating period. Heating of the tap water with low pH led to a more significant increase of chloroform and a more significant short term increase of DCAN. High pH accelerated the loss of the non-regulated DBPs in the heated water. The results indicated that as the chlorine doses increased, levels of chloroform and DCAA in the heated water increased significantly. However, for TCAA, the thermally induced increase in concentration was only notable for chlorinated water with very high chlorine dose. Heating may lead to higher DBP concentrations in chlorinated water with lower distribution system temperatures.

Keywords

Disinfection byproducts, heating, distribution system, degradation, formation

4.2 Introduction

Disinfection byproducts (DBPs) are compounds formed during the disinfection of potable water and a direct result of the reaction between natural organic matter (NOM) and the disinfectant. Upon the discovery of DBPs in 1974, considerable attention has been given to balancing the risks between microbial pathogens and health effects related to DBPs. The concentration of DBPs can be highly variable throughout a distribution system and can also vary over time at the same distribution system location (Singer, 2001;

Pereira et al., 2004). Currently, four trihalomethanes (THM4) and five haloacetic acids (HAA5) are regulated by the United States Environmental Protection Agency (USEPA) with maximum contaminant levels (MCLs) of 80 and 60 μg/L, respectively. The cost of implementing the Stage 2 Disinfectants and Disinfection Byproduct Rules is estimated to be $79 million annually (USEPA, 2013). Despite such regulatory scrutiny, overall exposure to DBPs is still poorly characterized. Current regulatory oversight considers DBPs formed in the water plant and across distribution systems. However, exposure to DBPs cannot be accurately assessed from concentrations in samples collected for regulatory purposes. Rather exposure is also affected by physical and chemical affects inherent in household water use (e.g., showering, bathing, beverage preparation). Additionally, dermal and inhalation exposure during household water use activities have been recognized as significant contributors to human exposure to DBPs in the water (Maxwel et al., 1991; King et al., 2004; Whitaker et al., 2003, Nuckols et al., 2005). Dermal and inhalation exposure during showering may result in higher elevated THMs levels in the blood as compared to other water use activities (Backer et al., 2000; Gordon et al., 2006). Therefore, the concentration of DBPs in water heaters may be of equal or greater importance than from the cold tap.

Some researchers have examined changes in DBP concentrations upon heating in well-controlled laboratory studies (Weisel and Chen, 1994, Wu et al., 2001, Zhang et al., 2013). These have generally shown increases in some DBPs (e.g., THMs) in with increasing temperature, and decreases in others (e.g., HAAs). Other researchers have investigated the concentrations of DBPs in home water heaters and compared them with levels in the cold water taps (Dion-Fortier et al., 2009, Chowdhury et al., 2011; Eyring et

al., 2008). However, these studies only focus on the general comparison between the DBP levels in cold and hot tap water for randomly selected homes. While providing valuable insight, the studies to date have been of limited scope, as they have not considered such important factors as the interactions between water quality entering the home and final concentrations in the heated water.

Numerous epidemiological studies on the DBP exposure during showering have focused on DBPs in breath and blood samples. These have shown the importance of exposure to hot water and water vapor. Nevertheless, these researchers have been unable to determine DBP exposure accurately because of the limited information on the concentration of DBPs formed under different heating scenarios, and the complex chemical interrelationships between DBPs and other parameters within a distribution system (Arbuckle et al., 2002). In this paper, we present a systematic laboratory study that considers a broad range of conditions, including distribution system water age (e.g, reaction time at ambient temperature) and in-home heating conditions.

The DBP levels in the water leaving a water heater are the sum of the initial level in the cold tap water and the net affect caused by heating. Historically, Stage 1 and Stage 2 Disinfectant and DBP rules recognize the relationship between DBP occurrence and water age (USEPA, 1994; USEPA, 2006). For the THMs, increasing water age is generally associated with elevated DBP levels. At the same time, chlorine residual, which contributes to further formation in the heating process, decreases gradually with increasing water age in water distribution systems. This introduces a level of complexity that is not widely recognized. In this study, the concentration of THMs and HAAs, as well as non-regulated DBPs including dichloroacetonitrile (DCAN),

trichloronitromethane (chloropicrin or CP), 1,1-dichloropropanone (1,1-DCP), 1,1,1-trichloropropanone (TCP) were investigated under controlled simulated distribution system and heating scenarios.

4.3 Experimental Methods

4.3.1 Test water

Water for these experiments was collected from the water treatment plant in Springfield, MA. We used a blend of slow sand filter effluent and rapid sand filter effluent at a volume ratio of 3:1 intended to simulate the actual water at the point of disinfection in the Springfield system. These water samples were transported to the University of Massachusetts laboratory on ice and stored at 4 ^{o}C until use. The key organic characteristics of this water are listed in Table 4.1.

4.3.2 Chlorination and lab-controlled heating

Figure 4.1 illustrates the general procedure used for chlorination and lab-controlled heating tests in this study. A stock solution of sodium hypochlorite was used for chlorination and standardized by the DPD ferrous titrimetric method, 4500-C1 F (APHA, 1998). The water was first brought to 20 ^{o}C in an incubator for an hour before chlorination. Buffers (pH 6, 7, and 8) were prepared with monopotassium phosphate and varying amounts of sodium hydroxide such that the final phosphate concentration was 10 mM. To prepare the final test solutions, the requisite volume of the stock buffer was transferred to a 4 L borosilicate bottle which was then filled with water samples each time. The pH of this diluted buffer was measured. Variable-volume, headspace-free

"piston bottles" were prepared. Chlorine was added to the buffered water samples using stock sodium hypochlorite solution in order to achieve a dose of 5 $mg \cdot L^{-1}$. The 4 L borosilicate bottles containing chlorinated samples were incubated at 20 ^{o}C for different contact times representing a range of water ages typical of many distribution systems (6 h., 1 day, 2 days, 3 days, and 4 days). At the end of each prescribed contact time, the chorine residual was measured and a set of samples were partitioned to glass vials containing sodium sulfite quenching agent (400 μL of 1 $g \cdot L^{-1}$ in a 40 mL glass vial) and a drop of concentrated sulfuric acid was added on the top of each vial. The glass vials were sealed headspace-free for subsequent analysis of DBPs within one hour of quenching. After collection of these first samples, the remaining volume was carefully partitioned into the piston bottles, headspace-free with the piston in place at the fully depressed position so that it could rise as the water sample expanded during incubation in the hot water bath, and each was sealed tightly and placed in the water bath pre-equilibrated to 55 ^{o}C. We chose 55 ^{o}C as the target temperature because it is the optimal temperature recommended by World Health Organization (WHO) for pathogen control and scalding prevention purpose (WHO, 2007; Feldman, 1983). At each defined heating contact time (0.53, 6, and 24 h), the piston bottles were withdrawn from the 55 ^{o}C water bath, and placed in a cold water bath (4 ^{o}C) for rapid cooling. The chlorine residuals were then measured as soon as the samples were collected, and the remaining samples were quenched. Samples were extracted for analysis of DBPs within 1 hour of quenching.

To assess the impact of dose on the occurrence of DBPs in the heated water, another set of similar experiments were conducted at 8 ^{o}C, and pH 8 with chlorine doses

of 1.5 $mg \cdot L^{-1}$, 2.5 $mg \cdot L^{-1}$ and 4 $mg \cdot L^{-1}$. This temperature and pH were selected as they represent typical winter conditions for the plant.

Quality control tests were conducted on every 10 samples for each type of analysis which include spike-recovery samples. The spike recoveries ranged from 92 to 104%.

4.3.3 Analytical methods

Chlorine residuals were analyzed at the time of sampling by the DPD ferrous titrimetric method. Four chlorine and bromine containing THMs, DCAN, CP, 1,1-DCP and TCP were quantified by liquid/liquid extraction with pentane followed by gas chromatography and electron capture detection (GC/ECD) according to U.S. Environmental Protection Agency (USEPA) method 551.1. HAA9 compounds were analyzed by liquid/liquid extraction with methyl-tertbutyl-ether (MTBE), followed by derivatization with acidic methanol and by GC/ECD based on USEPA method 552.2.

4.4 Results and Discussion

4.4.1 The concentration of THMs and HAAs in the heating process

Figure 4.2 presents the chlorine decay during ambient (20 ^{o}C) incubation and during subsequent heating. The chlorine residuals in the treated water after an ambient incubation time of 6, 24, 48, 72, 96 h were 1.09, 0.62, 0.28, 0.11 and <0.05 $mg \cdot L^{-1}$ as Cl_2 respectively. In each case the chlorine residuals were rapidly depleted after the water was heated up to 55 ^{o}C. For chlorinated water with an ambient reaction time of 6 h and 24 h, the levels dropped below detection limit within 3 h. For chlorinated water allowed to

react longer than 24 h at ambient temperature, chlorine depletion during subsequent heating occurred within half an hour.

Although THM4 and HAA5 are regulated as groups by the USEPA, we present data on individual THM and HAA species as this provides a more targeted understanding of the impacts of temperature on their formation and decay. Springfield water has a relatively low bromide level and as a result chloroform, trichloroacetic acid and dichloroacetic acid were always the most abundant DBPs. Because of the qualitative similarity between the concentrations profiles for chloroform and other detected brominated THMs, and the relatively low concentrations (Figure 4.3, Figure 4.4), we focused our discussion of THMs on chloroform.

As expected, chloroform concentrations increased with increasing ambient reaction time (Figure 4.5), reaching a maximum concentration of about 75 $\mu g \cdot L^{-1}$ after 4 days (96 h). Rapid heating of each sample to 55 ^{o}C caused a similar rapid rise in chloroform for the samples with >0.5 $mg \cdot L^{-1}$ chlorine residual as Cl_2 (i.e., the 6 and 24 h water ages). For example, the concentration of chloroform doubled after only half an hour of heating of the sample with a water age of 6 h. The increase in the chloroform became slower until it reached a maximum concentration slightly above 100 $\mu g \cdot L^{-1}$ as the residual chlorine dropped to undetectable levels. Longer ambient reaction times (corresponding to longer water ages in a distribution system) resulted in less pronounced increases upon heating at 55^{o}C. Paradoxically, the end result after heating was a slightly lower chloroform level in the samples with the longer water ages (83 $\mu g \cdot L^{-1}$ for 96 h vs. 100 $\mu g \cdot L^{-1}$ for 6 h). This implies that heating to 55 ^{o}C does not simply accelerate reactions that would otherwise occur at 20 ^{o}C over longer times. Rather, it must alter

DBP product yields, possibly due to differences in the activation energies of key steps in the various DBP forming pathways. It is significant that chloroform increased in concentration when 96 h samples, devoid of any measurable chlorine residual, were heated. This may be attributed to THM formation from accelerated hydrolysis of trihalogenated precursors.

The regulated HAAs are dominated by dihaloacetic acids (e.g., dichloroacetic acid or DCAA) and trihaloacetic acids (e.g., trichloroacetic acid or TCAA). Although grouped together for analytical and regulatory purposes, these two are distinct from the standpoint of formation pathways (Reckhow et al., 1990) and precursor characteristics (Liang and Singer, 2003). Therefore, impacts of heating on TCAA and DCAA will be dealt with separately. The profile of DCAA concentration over ambient and elevated temperatures (Figure 4.6) bears many similarities to the chloroform profile. There are at least two differences that merit attention. First the ultimate formation seems to be a fixed value (~41 $\mu g \cdot L^{-1}$) and independent of the water age or time of reaction at ambient temperature. This would suggest that elevated temperatures may simply accelerate the reaction, but not change the pathways as it did for chloroform. Second, the increase in DCAA upon heating in the absence of a chlorine residual (i.e., at 96 h) was relatively large and immediate. This implies the presence of a large amount of gem-dichloro intermediates (for example, see Reckhow et al., 1990) with high activation energy for hydrolysis in the pathway that leads to DCAA.

It is clear that the concentration profile for TCAA (Figure 4.7) was substantially different from the pattern exhibited by DCAA and chloroform. Although the TCAA concentration showed a monotonic increase with increasing ambient incubation time, its

behavior during subsequent heating was more complex. Under conditions typical of a home water heater, the TCAA showed a slight initial increase followed by a gradual decrease. In contrast to the tripling and doubling of the chloroform and DCAA levels for some of the samples, heating never resulted in more than a 26% increase in TCAA. We interpret this behavior as the net result of accelerated formation and decomposition. Based on a first order rate constant for TCAA decomposition of 1.47×10^{-6} s^{-1} (Zhang et al., 2002; Verhoek, 1934) one would expect about 12% decomposition of TCAA over 24 h of heating at 55 ^{o}C. This is close to the drops seen in Figure 4.7 from each heated water maximum to its minimum about 24 h later.

The next most abundant brominated HAAs are bromodichloroacetic acid (BDCAA) and bromochloroacetic acid (BCAA). The formation of BDCAA at ambient temperature is rapid nearing maximum yield in less than 6 h (Figure 4.8). This is typical of many brominated DBPs and it is thought to reflect the rapid incorporation of bromine under low Br/TOC conditions. Heating of the chlorinated water did not apparently lead to any increases in BDCAA; but instead, this haloacid shows substantial losses. Based on a first order decomposition rate of 5.45×10^{-6} s^{-1} (24) we would have expected about a 37% drop after 24 h at 55 ^{o}C. However, the sample with 96 h water age exhibited the highest relative loss which was about 65%. As with chloroform, increasing water age resulted in slightly lower hot water concentrations of BDCAA. The concentration profile for BCAA is similar to DCAA, and the graph is shown in Figure 4.9.

The contrasting behavior of DCAA, TCAA and chloroform during the heating process supports prior evidence that their formation mechanisms or precursors are normally different. Figure 4.10 shows a simplified mechanism for DCAA, TCAA and

chloroform based on the conceptual model by Reckhow and Singer (1985). The underlying concept is that in the presence of a high free chlorine residual, formation of a trihalomethyl intermediate is favored. This type of intermediate can then lead to either TCAA or chloroform (and, indeed, brominated forms if bromide is present). The formation of TCAA requires an oxidative cleavage which can be assisted by chlorine residual. Chloroform can arise from a simple alkaline hydrolysis, with a rate-determining step that is strongly favored at elevated temperature. While this conceptual model was originally developed to explain data from low temperature tests, it also seems to fit the higher temperature data in this paper. Chloroform formation is strongly enhanced at 55 ^{o}C. Also at this temperature, chlorine residual was rapidly depleted thereby limiting TCAA formation. The further formation of DCAA and chloroform after the chlorine depletion could be attributed to hydrolysis of DCAA and chloroform intermediates (Hua and Reckhow, 2012).

4.4.2 The concentration of 1,1-DCP, CP, 1,1,1-TCP and DCAN in the heating process

Many of the non-regulated DBPs are of interest because of their greater genotoxicity (Richardson et al., 2007). In this study, 1,1-DCP, CP, 1,1,1-TCP and DCAN were measured in the chlorinated water and shown in Figure 4.11 to Figure 4.14.

As shown in Figure 4.11, the concentration of CP was low with a concentration of 0.32-0.44 $\mu g \cdot L^{-1}$ and increasing modestly with increasing simulated water age. These low levels are typical of systems not using preozonation or medium pressure UV (Weinberg et al., 2002; Reckhow et al., 2010). After heating, short term increases in CP levels were observed for the samples with lower water age and higher chlorine residual.

Continued heating led to a substantial reduction of CP in the chlorinated water. Nevertheless, after 24 h of heating, there was still more than 50% of the original CP present in the chlorinated water. Both formation and loss of CP during heating has been noted by others (Weisel and Chen, 1994; Zhang et al., 2013).

Regardless of the water age, 3 h of heating led to a significant increase in the 1,1-DCP concentration (up to 5x) after which its concentration diminished. It should be noted that the large increases for 1,1-DCP were not only observed in samples with high chlorine residual but also in the samples with undetectable residuals. Weisel and Chen (1994) also noted increases in 1,1-DCP followed by a decrease upon heating to 65^{o}C. It seems likely that 1,1-DCP concentrations remain low in systems maintaining free chlorine residual through slow formation that is balanced by degradation to 1,1,1-TCP and other products. The high-temperature formation and loss seen in Figure 4.12 must be due to hydrolysis reactions as chlorine residuals are essentially nonexistent.

The concentration of TCP increased rapidly in the first 6 h, reaching a maximum at 24 h under ambient temperature conditions (Figure 4.13). Heating for as little as 30 min resulted in substantial loss of the TCP, and samples with longer water ages showed lower 1,1,1-TCP levels. However, regardless of the ambient reaction time, the 1,1,1-TCP level in the heated water was below detection limit within 3 h of heating. Trichloropropanone is known to undergo hydrolysis and chlorine-assisted degradation forming chloroform and DCAA (26), although the kinetics of 1, 1,1-TCP degradation at 55 ^{o}C have not been explored. Dichloropropanone exhibited a low, yet consistent concentration of about 0.3-0.5 $\mu g \cdot L^{-1}$ almost regardless of the simulated distribution

system water age. This behavior is typical of systems using free chlorine (Weinberg et al., 2002; Reckhow et al., 2013).

The concentration of DCAN exhibited a similar trend as with chloropicrin. After 30 min of heating, the DCAN concentration in the lowest water age sample (6 h) increased 60% from 0.63 to 1.00 $\mu g \cdot L^{-1}$. For samples with longer water ages, decomposition dominated and, the net was a loss of DCAN. Although the ambient-temperature DCAN concentration was highest for the sample with the highest water age (96 h), after 24 h of heating, its DCAN level was the lowest and was near the detection limit. The decomposition of DCAN has been extensively studied at 20 ^{o}C (Reckhow et al., 2001) and to a lesser extent at higher temperatures (Weisel and Chen, 1994, Nikolaou et al., 2000). Recent work indicates that DCAN has an apparent first order rate constant of 0.21 h^{-1} at 55 ^{o}C in buffered water at pH 7 (Reckhow et al., 2013). This nearly accounts for the loss rate seen in Figure 4.14.

4.4.3 Factors that affect the concentration of DBPs during heating

The concentration of DBPs in chlorinated water depends largely on source water characteristics and specific disinfection conditions. Factors, such as pH, temperature, dose and reaction time could significantly affect the rate, extent and distribution of the DBPs in drinking water systems (Yang et al., 2007; Hua and Reckhow, 2008). These factors could also affect the concentration profiles of DBPs in heated tap water.

4.4.3.1 Effect of pH

Concerning the regulated HAAs, our results indicated that pH did not substantially impact the DCAA and TCAA concentration profiles in heated water as similar trends were observed for pH 6 to pH 8 (Figures 4.15 to 4.18) as compared to pH 7.

However, THMs, especially chloroform, did seem to show different behavior under different pH conditions. Figure 4.19 presents the chloroform concentration profiles at pH 6 and pH 8. The results for pH 7 have been previously shown in Figure 4.5. As expected from prior studies (Liang and Singer, 2003; Engerholm and Amy, 1983), chloroform concentration increased at ambient temperature with increasing pH and reaction time (i.e., simulated water age). After 96 h, the chloroform concentrations were 55.4, 75.3 and 93.5 $\mu g \cdot L^{-1}$ for pH6, pH7 and pH8 respectively. It has been proposed that base-catalyzed reactions play a significant role in THM formation (Reckhow et al., 1990; Peters et al., 1980; Trussell and Umphres, 1978). Heating caused substantial increases at all pHs when the water age was low. Because the increase was greater for the lower pHs, heating had the effect of almost leveling the ultimate chloroform yield across the pHs. For example, heating the 6 h water age sample lead to an increase of about 250%, 180% and 100% of chloroform for pH6, pH7 and pH8 respectively. Heating of the 96 h water age samples lead to an increase of 36%, 11% and 2% of chloroform for pH 6, pH 7 and pH 8 respectively. The large heating-induced increase in chloroform at pH 6 for longer water ages (i.e., with little or no chlorine residual) supports the idea of a large reservoir of halogenated intermediates present at all pHs that undergoes slow hydrolysis to form THMs. The implication is that high pH induces elevated THM concentrations largely due to faster conversion of these intermediates and not faster halogenation. It's also

significant to note that water age has little effect on the hot water chloroform levels (6-24 hr of heating) and the relationship might even be the inverse of that for the cold water (i.e., longer water ages lead to slightly lower chloroform levels).

Previous research has shown that DCAN, 1,1,1-TCP, and 1,1-DCP are unstable in aqueous solution and can decompose quickly in alkaline conditions (Reckhow and Singer, 1985; Reckhow et al, 2013; Krasner et al., 1989; Stevens et al., 1989). Figure 4.21 shows the levels of DCAN in the heated water at pH 6 and pH 8, whereas results for pH 7 were shown in Figure 4.14 previously. At pH 6 (Figure 4.21), 3 h of heating lead to significant increases in DCAN concentration regardless of prior water age. In each case, longer heating times led to a net loss of DCAN. In contrast, decomposition was clearly dominant pH 8 (Figure 4.22). For the other non-regulated DBPs, the thermal stability generally decreased with increasing pH (Figures 4.23-4.28, Figure 4.11-4.13). Under differing pHs, 1,1-DCP displayed nearly the same concentration profile except that at lower pH the maximum 1,1-DCP levels in the heated water were significantly higher. The maximum 1,1-DCP levels in the heated water at pH 6 was more than twice the level in the heated water at pH 8. It was observed that 1,1,1-TCP decomposed quickly with time at high pH under both 20 ^{o}C and 55 ^{o}C conditions. At pH 8, 30 min of heating removed most of the TCP in the chlorinated water regardless of ambient reaction time. Although Croué and Reckhow (1989)'s short term study of CP (<3 h) did not detect any base-catalyzed hydrolysis, our results indicated high pH leads to significantly higher decomposition rate of this compound.

4.4.3.2 Effect of Dose and incubation temperature on the concentration of regulated DBPs

Different chlorine doses (1.5, 2.5 and 4 $mg \cdot L^{-1}$ as Cl_2) were applied to the water before the incubated chlorinated water was heated from 8 ^oC to 55 ^oC and remained under the temperature of 55 ^oC for 24 h. The chlorine residuals after the incubation time of 96 h were 0, 0.8 and 1. 86 $mg \cdot L^{-1}$ as Cl_2 respectively. 24 h of heating depleted the chlorine residuals for the doses of 1.5 and 2.5 $mg \cdot L^{-1}$ and lead to a low level of chlorine residual for a dose of 4 $mg \cdot L^{-1}$ (< 0.3 $mg \cdot L^{-1}$ as Cl_2). Figure 4.29 illustrated the occurrence of the regulated DBPs in the heated chlorinated water with different starting chlorine doses. The concentration of chloroform, DCAA, TCAA increased with increasing chlorine dose. The results indicated that as the chlorine dose increased, the chloroform in the heated water increased significantly. A similar trend was observed for DCAA. However, for TCAA, the additional thermal increase was only significant for the chlorinated water with the dose of 4 $mg \cdot L^{-1}$ as Cl_2. Moreover, the initial temperature of 8 ^oC and chlorine dose of 4 $mg \cdot L^{-1}$ as Cl_2 lead to less chloroform formation compared to the chlorinated water with incubation temperature of 20 ^oC and chlorine dose of 5 $mg \cdot L^{-1}$ as Cl_2 (Figure 4.5 and Figure 4.29), however, after 24 h of heating, the additional temperature-induced increase and final chloroform levels were higher for the chlorinated water with a lower initial temperature and a lower chlorine dose. Nevertheless, the results revealed the impact of incubation temperature. Heating may lead to higher DBPs for chlorinated water with a lower initial incubation temperature. This may be caused by the lower reaction rate and higher chlorine residual, which yields more DBPs under the same heating conditions.

4.4.4 Implication in the control of DBPs in drinking water treatment process

This study shows that the water quality in hot water can be substantially different from that in cold water. In addition the DBP levels in hot water from home to home across a distribution system may not be correlated with the corresponding cold water DBP levels. This is problematic as both regulations (U.S.EPA, 2013) and health effect studies (Nuckols et al., 2005; Gordon et al., 2006) have tended to focus on concentrations in the main or cold water tap as an indicator of health risk.

Of the US utilities that are struggling to meet the Stage 2 DBP standards, most have a greater risk of exceeding the TTHM4 than the HAA5 standards. Because THMs accumulate with water age and do not normally degrade, it is often the highest water age sites that are most problematic. This research indicates that efforts to reduce THMs at compliance points by reducing water age may not be helpful. If dermal and inhalational contact to hot water is the most important of the household exposure routes, reducing water age would not reduce the consumer's exposure, and it could even increase it.

For the more thermally liable DBPs, such as BDCAA, DCAN, CP, 1,1-DCP, and 1,1,1-TCP, concentrations in the heated tap water are the net result of simultaneous formation and decomposition. Our results indicate that not only water age but also the heating time strongly impacts the levels of these DBPs. Long term heating (i.e., 24 h) eventually destroys most of these DBPs. However, locations with lower water ages and a sufficient level of chlorine residual tend to have elevated levels of these thermally liable DBPs.

pH adjustment is one of the DBP control strategies that is frequently cited. However, if the objective is to control THMs in the hot water, our results indicate that low pH may be beneficial to homes with high chlorine residual, but not very beneficial for locations with low chlorine residuals. High pH, on the other hand, could help destroy many of the non-regulated DBPs especially in the heated water.

Seasonal variations of DBPs in the tap water are widely observed. Many studies reported maximum THM formation in summer because of the higher system temperature and higher initial chlorine doses (Rodriguez et al., 2004; Toroz and Uyak, 2005). Eying et al. (2008) found small changes in THMs in premise plumbing from the levels in the cold tap water in summer-fall season, whereas the THMs changed significantly during the winter-spring season. Concerning the effect of incubation temperature, our results indicate that the impact of heating on DBP formation may be more significant in colder seasons than warmer seasons.

Table 4.1 Inorganic and organic characteristics of the Springfield water

Parameter	**TOC ($mg \cdot L^{-1}$-C)**	**DOC ($mg \cdot L^{-1}$-C)**	**UV-254 (cm^{-1})**	**SUVA* ($L \cdot mg^{-1}m^{-1}$)**	**NH_4-N($mg \cdot L^{-1}$)**	**Br^- ($\mu g \cdot L^{-1}$)**
Value	2.50	2.41	0.065	2.7	0.06	8

* SUVA (specific ultraviolet absorbance) was calculated from ultraviolet absorbance at 254 nm (UVB254) divided by the dissolved organic carbon (DOC).

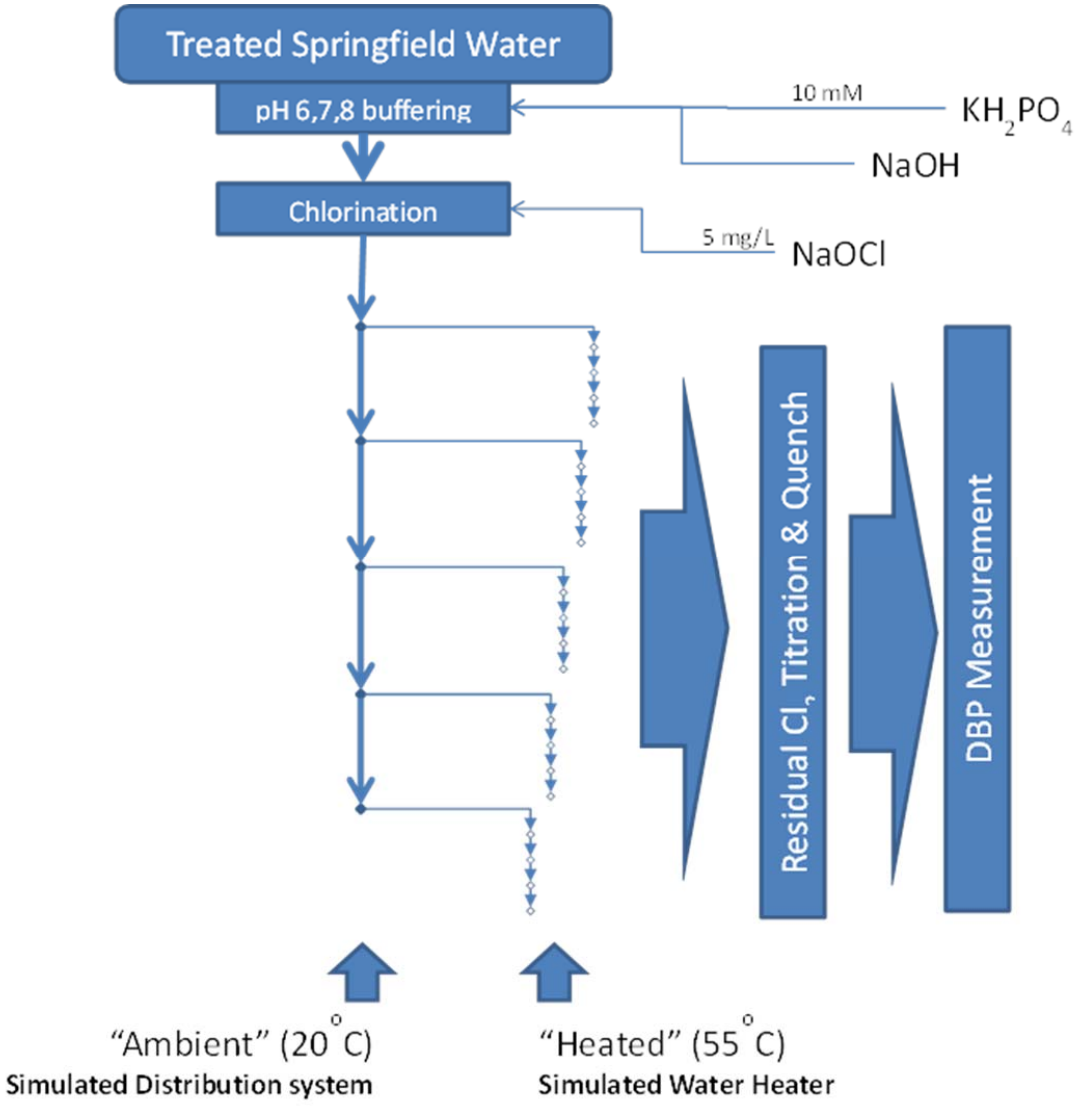

Figure 4.1 Chlorination and lab-controlled heating

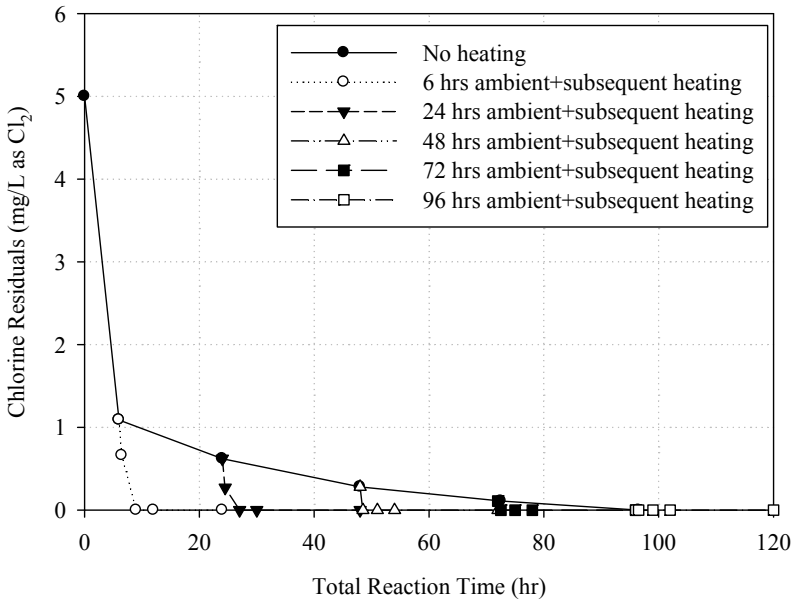

Figure 4.2 Chlorine residual during ambient reaction and subsequent heating (reaction conditions: pH 7, incubation temperature: 20 ^{o}C, heating temperature 55 ^{o}C)

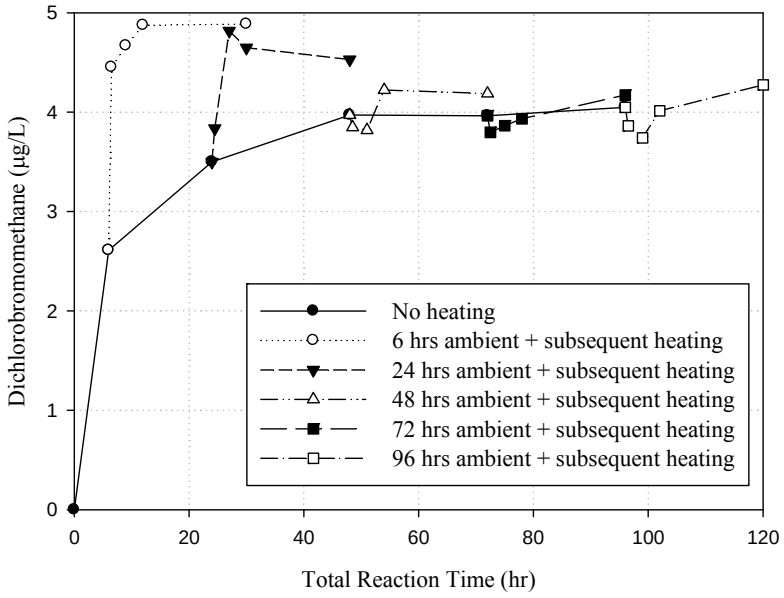

Figure 4.3 Concentration of dichlorobromethane during the ambient reaction and subsequent heating (reaction conditions: pH 7, ambient incubation temperature: 20 ^{o}C, heating temperature 55 ^{o}C)

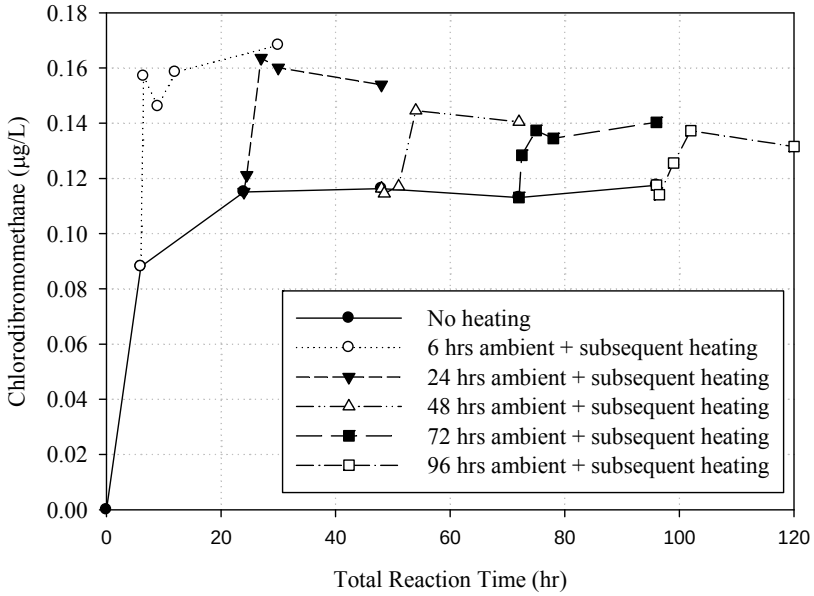

Figure 4.4 Concentration of chlorodibromomethane during the ambient reaction and subsequent heating **(**reaction conditions: pH 7, ambient incubation temperature: 20 ^{o}C, heating temperature 55 ^{o}C)

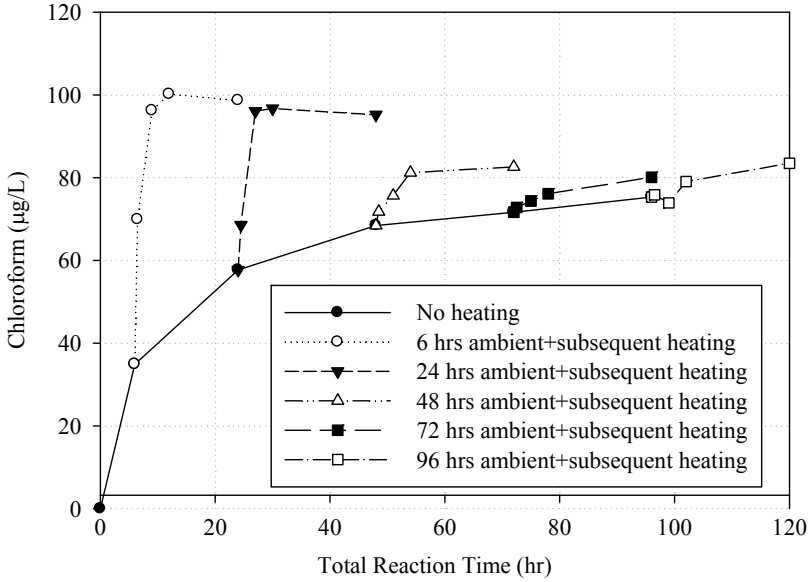

Figure 4.5 Concentration of chloroform during ambient reaction and subsequent heating (reaction conditions: pH 7, ambient incubation temperature: 20 ^{o}C, heating temperature 55 ^{o}C)

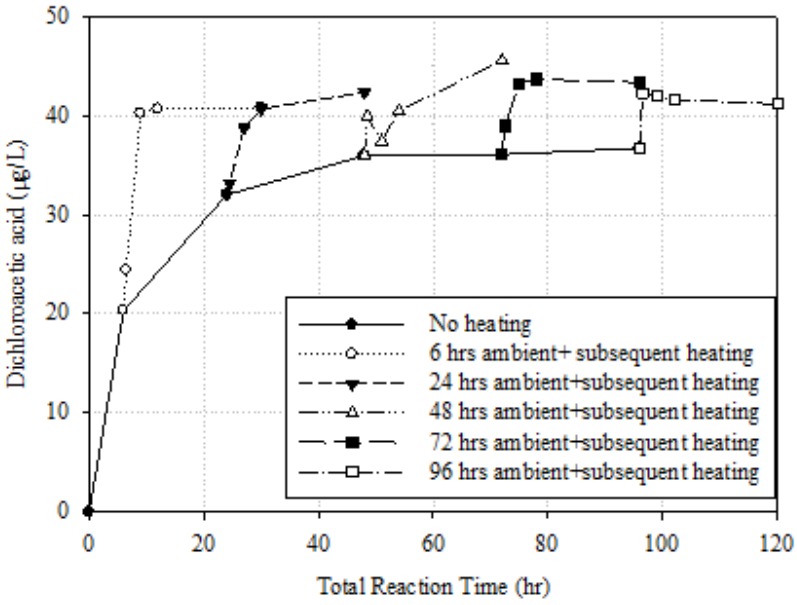

Figure 4.6 Concentration of dichloroacetic acid during ambient reaction and subsequent heating (reaction conditions: pH 7, ambient incubation temperature: 20 ^{o} C, heating temperature 55 ^{o}C)

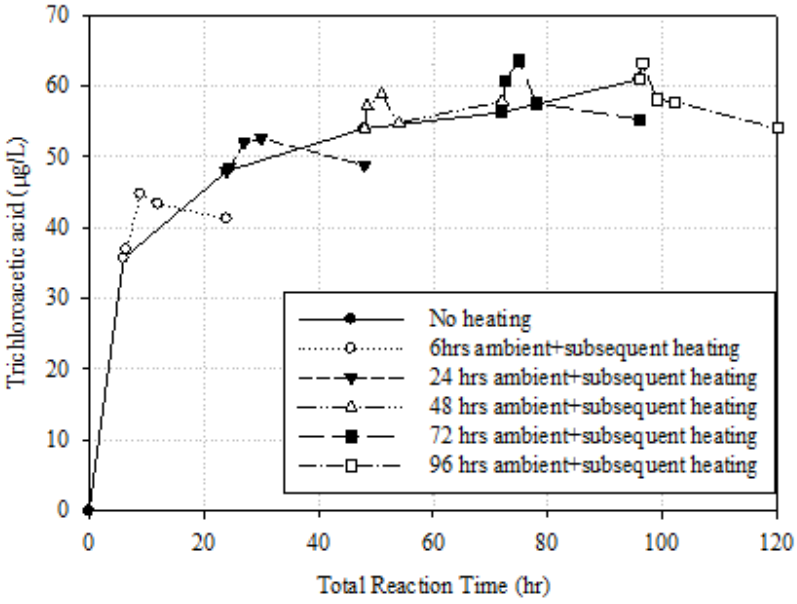

Figure 4.7 Concentration of trichloroacetic acid during the ambient reaction and subsequent heating (reaction conditions: pH 7, ambient incubation temperature: 20 ^{o}C, heating temperature 55 ^{o}C)

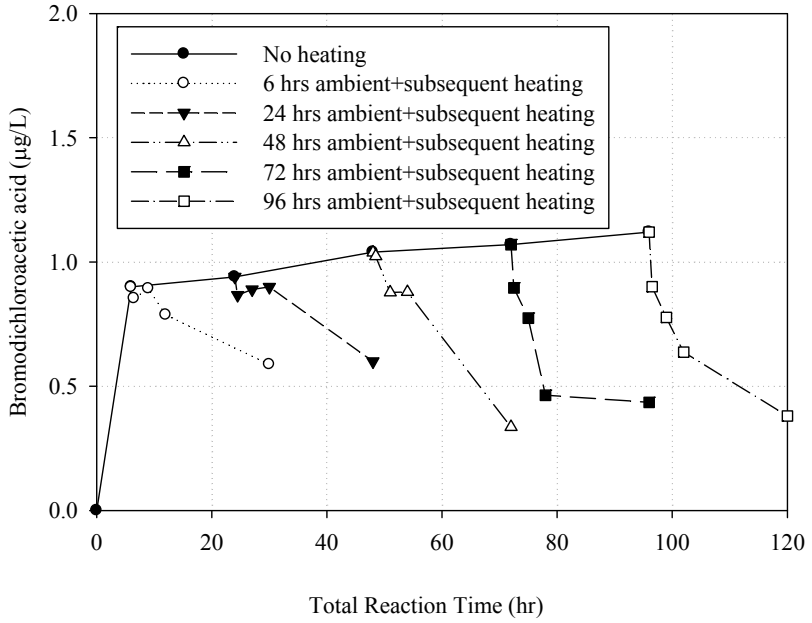

Figure 4.8 Concentration of bromodichloroacetic acid during the ambient reaction and subsequent heating (reaction conditions: pH 7, ambient incubation temperature: 20 ^{o}C, heating temperature 55 ^{o}C)

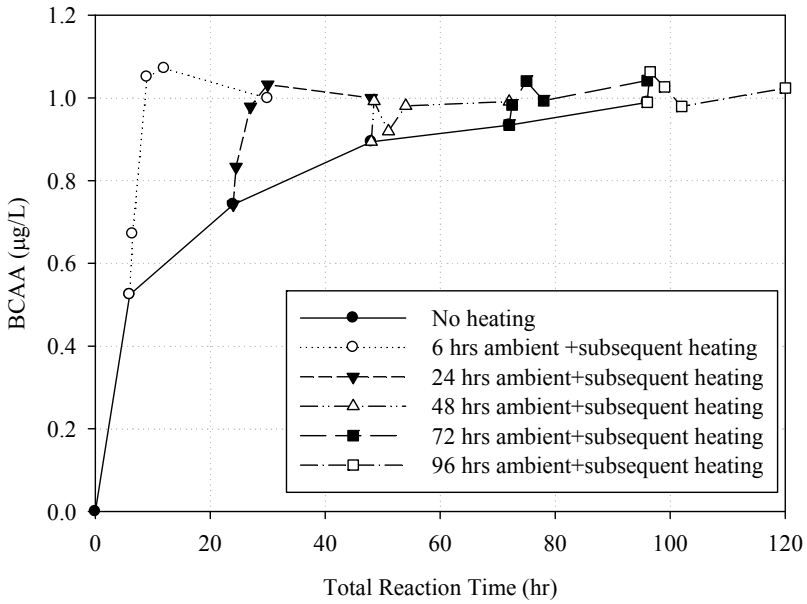

Figure 4.9 Concentration of bromochloroacetic acid during the ambient reaction and subsequent heating (reaction conditions: pH 7, ambient incubation temperature: 20 ^{o}C, heating temperature 55 ^{o}C)

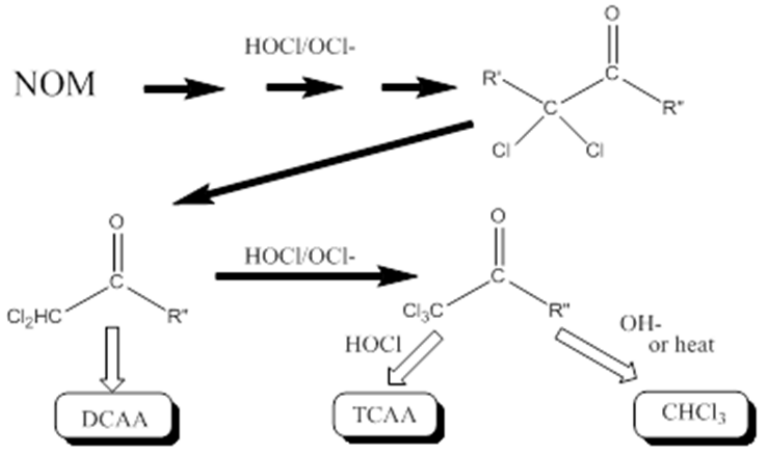

Figure 4.10 Conceptual mechanisms for the formation of DCAA, TCAA and Chloroform during chlorination (Simplified from Reckhow and Singer, 1985)

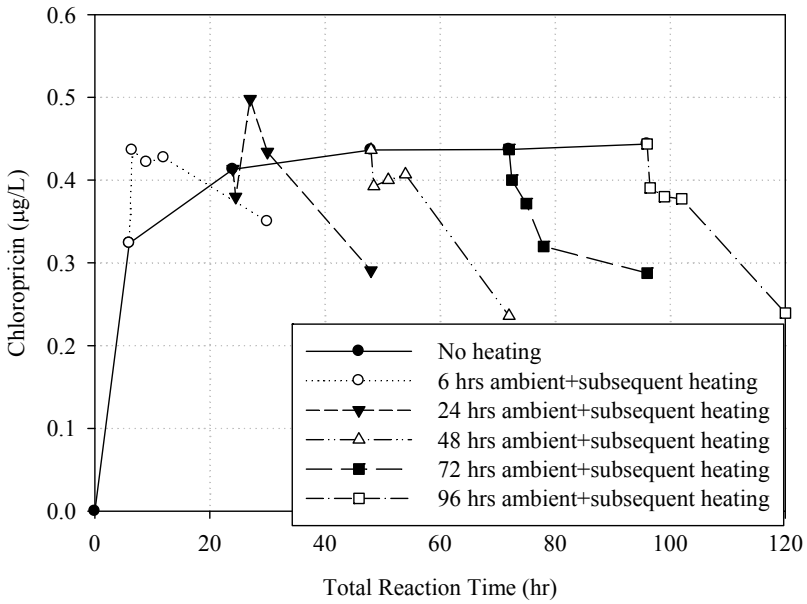

Figure 4.11 Concentration of chloropicrin during ambient reaction and subsequent heating (reaction conditions: pH 7, ambient incubation temperature: 20 ^{o}C, heating temperature 55 ^{o}C)

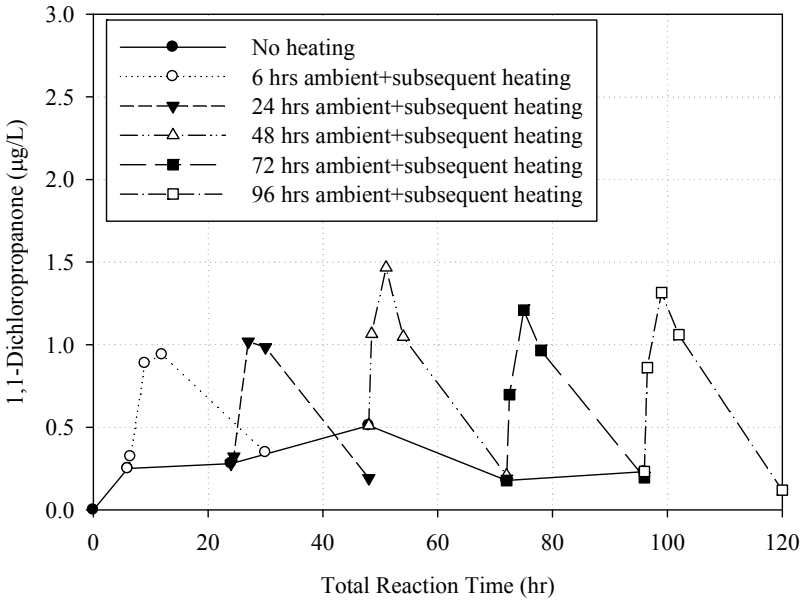

Figure 4.12 Concentration of 1,1-DCP during ambient reaction and subsequent heating (reaction conditions: pH 7, ambient incubation temperature: 20 ^{o}C, heating temperature 55 ^{o}C)

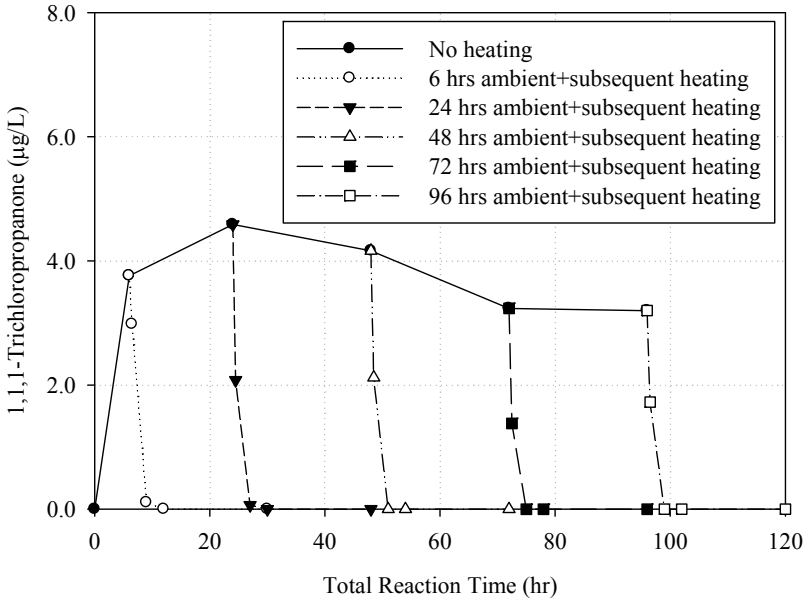

Figure 4.13 Concentration of 1,1,1-TCP during ambient reaction and subsequent heating (reaction conditions: pH 7, ambient incubation temperature: 20 ^{o}C, heating temperature 55 ^{o}C)

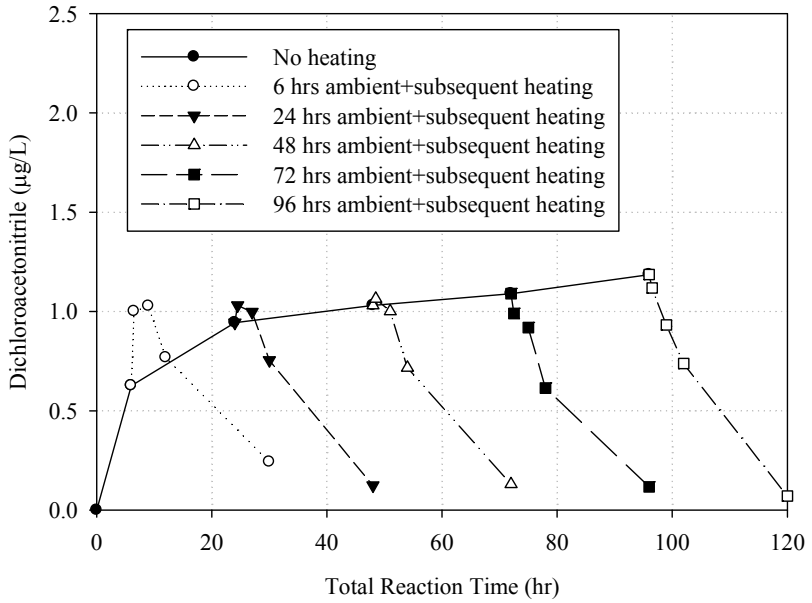

Figure 4.14 Concentration of dichloroacetonitrile during ambient reaction and subsequent heating (reaction conditions: pH 7, ambient incubation temperature: 20 ^{o}C, heating temperature 55 ^{o}C)

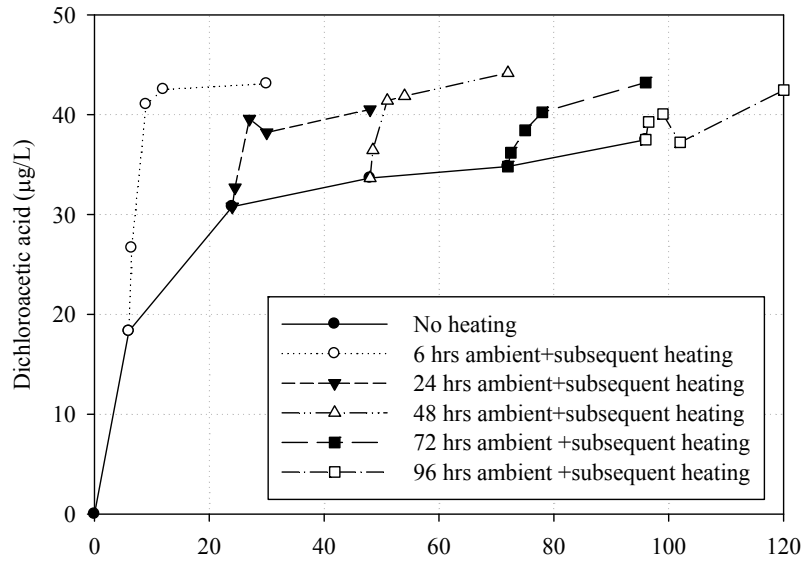

Figure 4.15 Concentration of dichloroacetic acid during the ambient reaction and subsequent heating (reaction conditions: pH 6, ambient incubation temperature: 20 ^{o}C, heating temperature 55 ^{o}C)

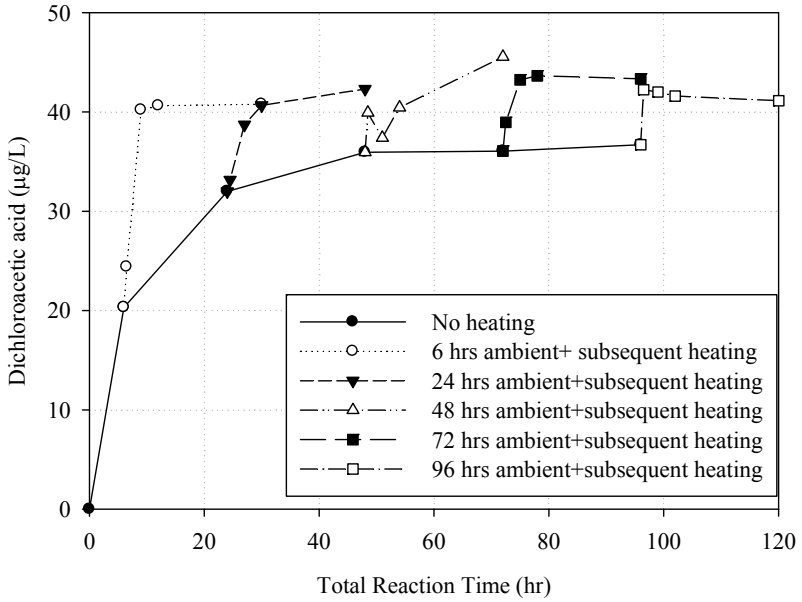

Figure 4.16 Concentration of dichloroacetic acid during the ambient reaction and subsequent heating (reaction conditions: pH 8, ambient incubation temperature: 20 ^{o}C, heating temperature 55 ^{o}C)

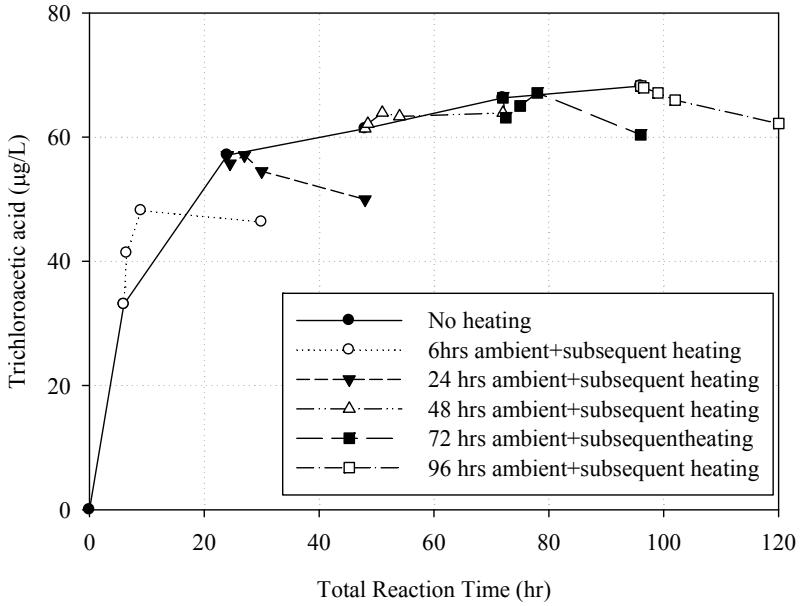

Figure 4.17 Concentration of trichloroacetric acid during the ambient reaction and subsequent heating (reaction conditions: pH 6, ambient incubation temperature: 20 ^{o}C, heating temperature 55 ^{o}C)

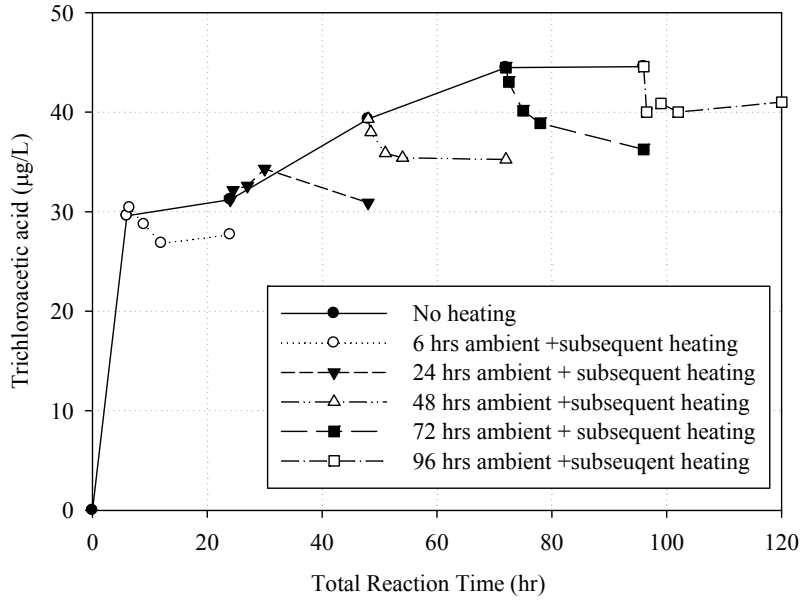

Figure 4.18 Concentration of trichloroacetic acid during the ambient reaction and subsequent heating (reaction conditions: pH 8, ambient incubation temperature: 20 ^{o}C, heating temperature 55 ^{o}C)

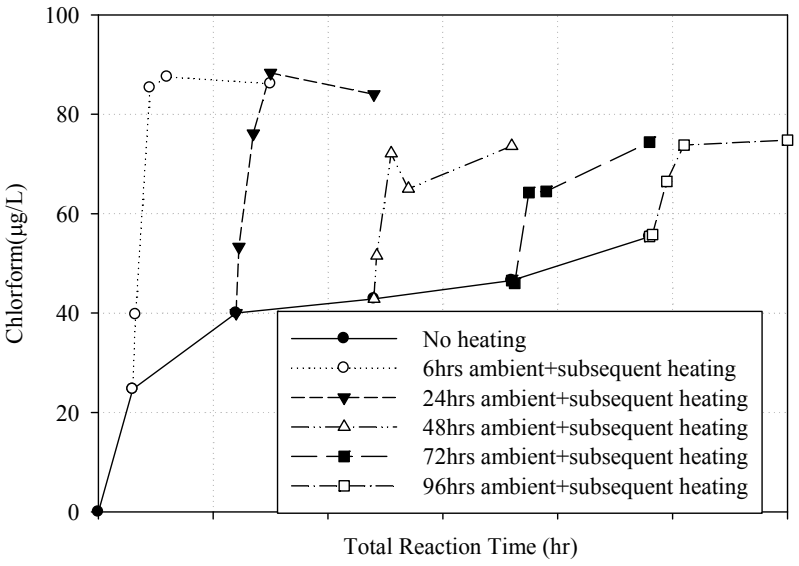

Figure 4.19 Concentration of chloroform during the ambient reaction and subsequent heating (reaction conditions: pH 6, ambient incubation temperature: 20 ^{o}C, heating temperature 55 ^{o}C)

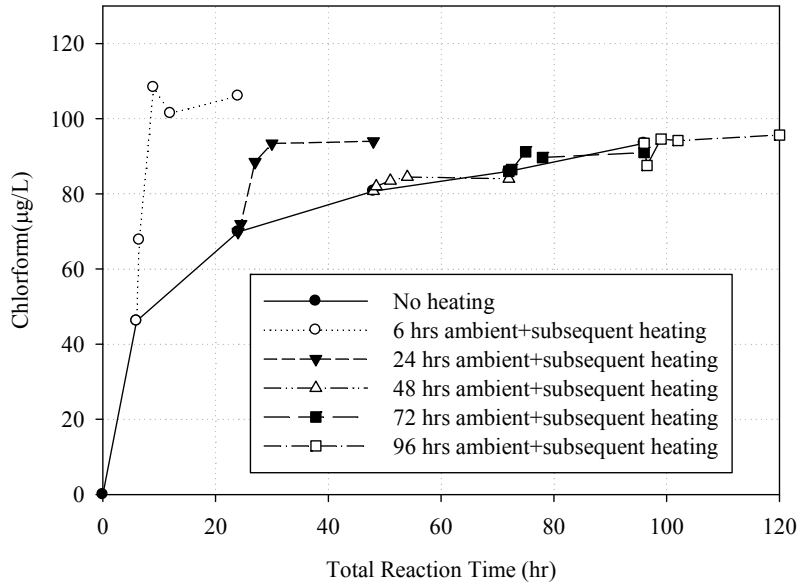

Figure 4.20 Concentration of chloroform during the ambient reaction and subsequent heating (reaction conditions: pH 8, ambient incubation temperature: 20 ^{o}C, heating temperature 55 ^{o}C)

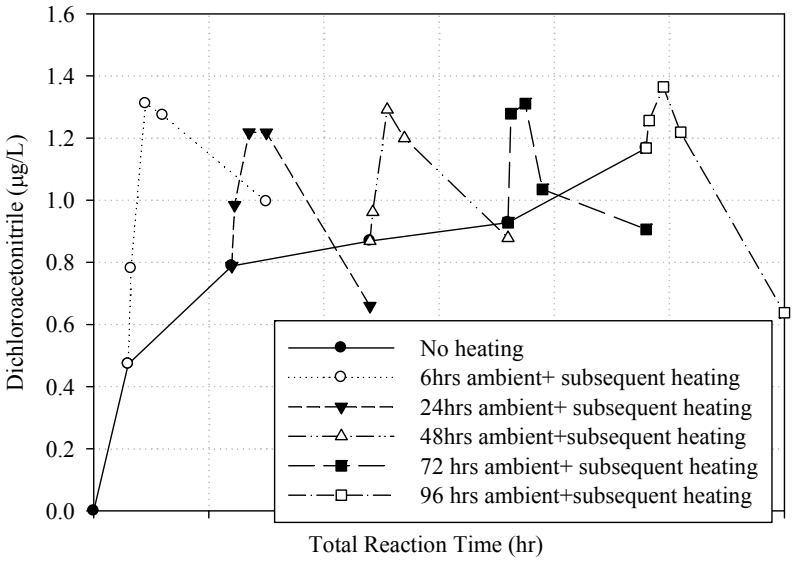

Figure 4.21 Concentration of dichlroacetonitrile during the ambient reaction and subsequent heating (reaction conditions: pH 6, ambient incubation temperature: 20 ^{o}C, heating temperature 55 ^{o}C)

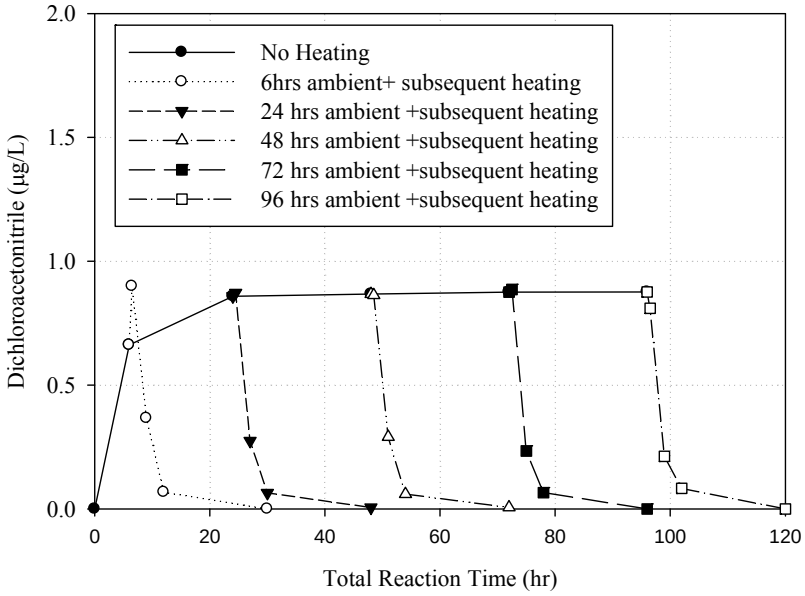

Figure 4.22 Concentration of dichloroacetonitrile during the ambient reaction and subsequent heating (reaction conditions: pH 8, ambient incubation temperature: 20 ^{o}C, heating temperature 55 ^{o}C)

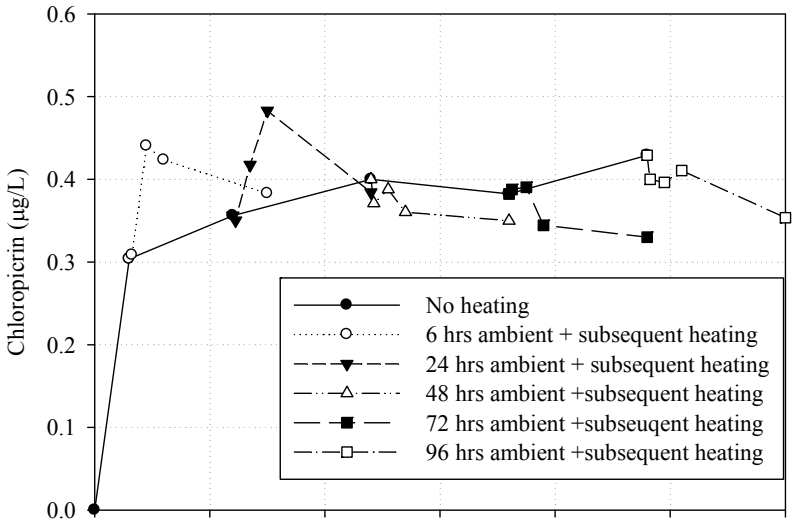

Figure 4.23 Concentration of chloropicrin during the ambient reaction and subsequent heating (reaction conditions: pH 6, ambient incubation temperature: 20 ^{o}C, heating temperature 55 ^{o}C)

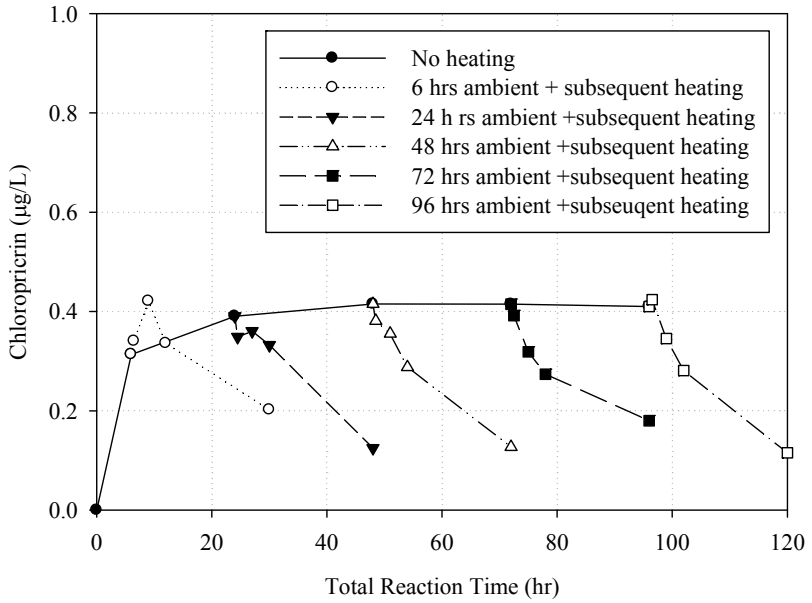

Figure 4.24 Concentration of chloropicrin during the ambient reaction and subsequent heating (Reaction Conditions: pH 8, ambient incubation temperature: 20 ^{o}C, heating temperature 55 ^{o}C)

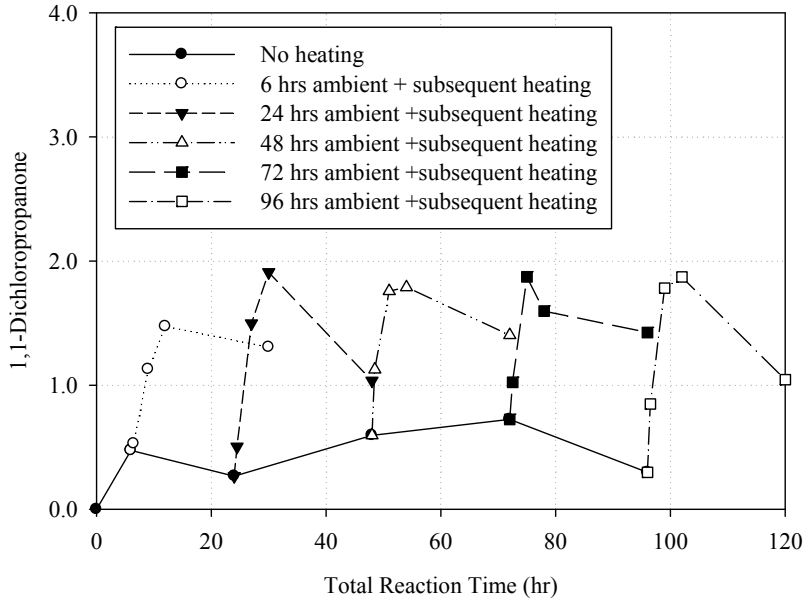

Figure 4.25 Concentration of 1,1-DCP during the ambient reaction and subsequent heating (reaction conditions: pH 6, ambient incubation temperature: 20 ^{o}C, heating temperature 55 ^{o}C)

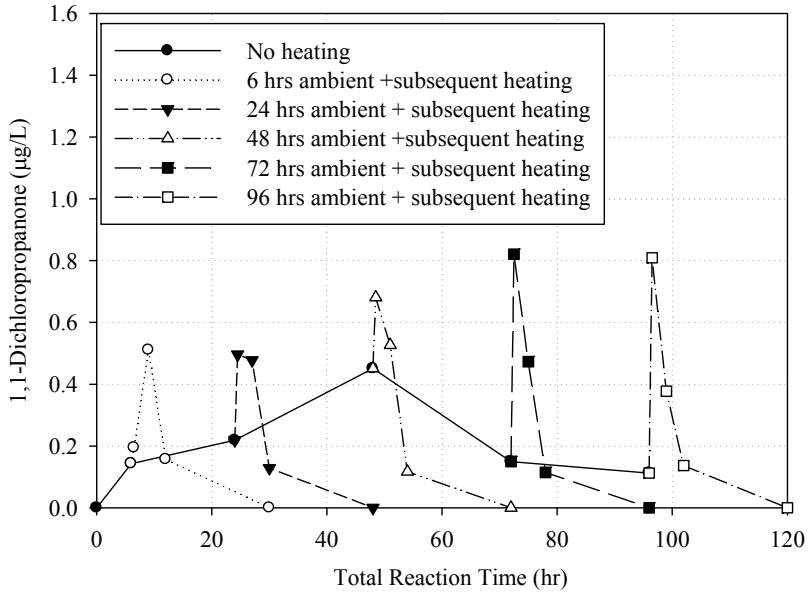

Figure 4.26 Concentration of 1,1-DCP during the ambient reaction and subsequent heating (reaction conditions: pH 8, ambient incubation temperature: 20 ^{o}C, heating temperature 55 ^{o}C

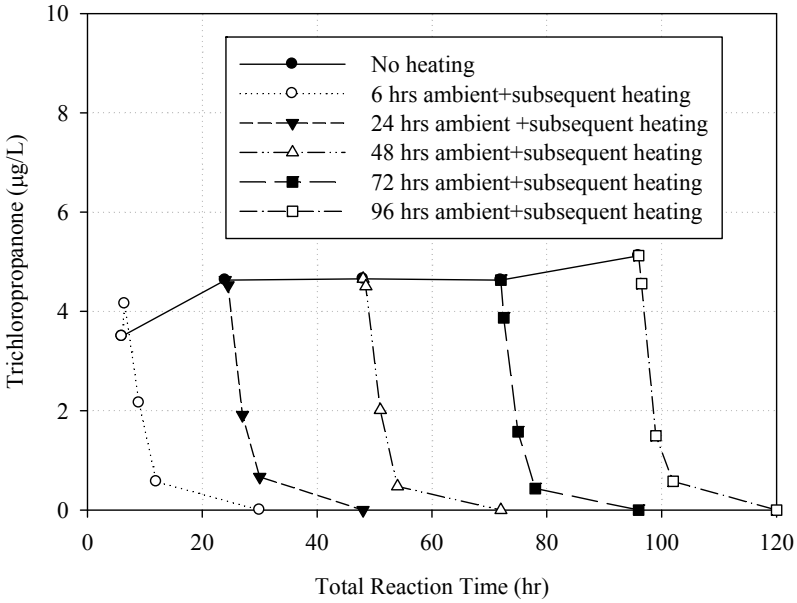

Figure 4.27 Concentration of 1,1,1-TCP during the ambient reaction and subsequent heating (reaction conditions: pH 6, ambient incubation temperature: 20 ^{o}C, heating temperature 55 ^{o}C)

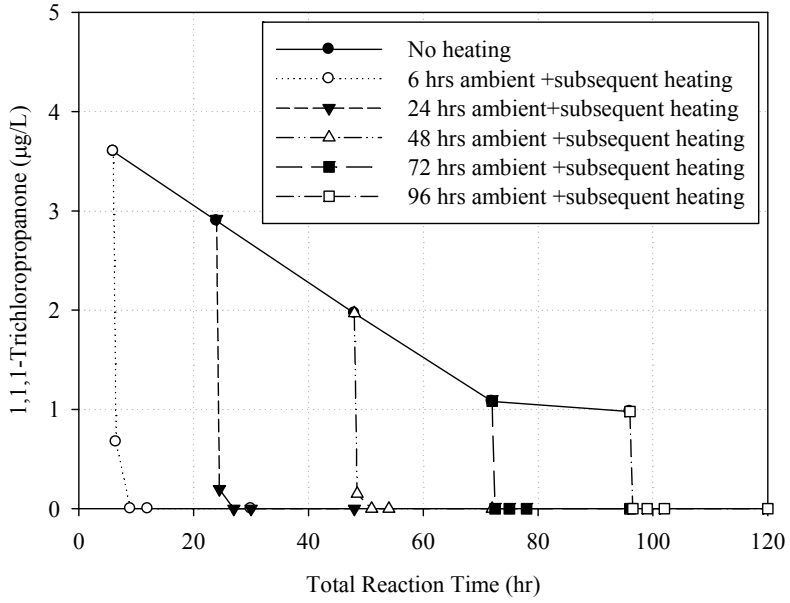

Figure 4.28 Concentration of 1, 1,1-TCP during the ambient reaction and subsequent heating (reaction conditions: pH 8, ambient incubation temperature: 20 ^{o}C, heating temperature 55 ^{o}C)

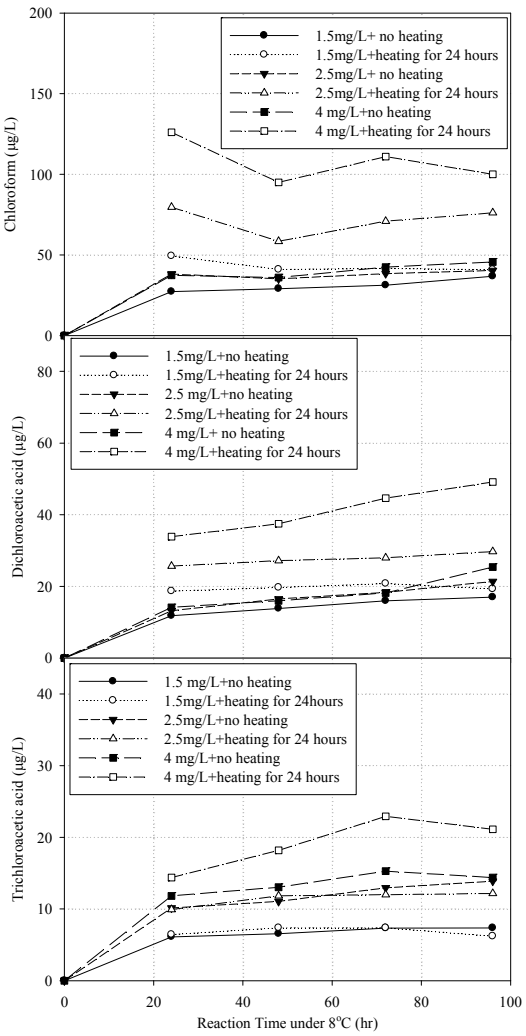

Figure 4.29 The effect of chlorine doses on the concentration of DBPs during incubation and heating(reaction conditions: incubation temperature: 8 ^{o}C, heating temperature 55 ^{o}C)

CHAPTER 5

A TWO-SITES CHLORINE DECAY MODEL OF COMBINED EFFECTS OF PH, TEMPERATURE IN WATER DISTRIBUTION SYSTEM AND UNDER HEATING CONDITION USING DIFFERENTIAL EVOLUTION

(an extended version of the paper published in *Water Research**)

5.1 Abstract

A general framework for modeling the bulk chlorine decay that accommodates effects of pH, temperature in water distribution system and in-home heating profiles is developed. With a single set of readily interpreted parameters, and various fictive concentrations of reactive constituents in the water, chlorine decay for the different water systems could be simultaneously modeled. Differential Evolution is employed to estimate the parameters stochastically. By using Bayesian Information Criterion, it is shown that a model consisting of two reactive species is preferred over models that consist of one or three reactive species. The flexibility and power of the framework is demonstrated with a case study of both types of effects.

Keyword: Chlorine decay, mechanistic modeling, differential evolution, heating

5.2 Introduction

Chlorination is widely used in drinking water treatment for pathogen control.

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*: Liu, B.; Reckhow, D. A.; Li, Y. "A two-site chlorine decay model for the combined effects of pH, water distribution temperature and in-home heating profiles using differential evolution". *Water Res*. **2014**, 53, 47-57.

However, the application of chlorination and the presence of chlorine residuals lead to the formation of potentially harmful disinfection byproducts (DBPs) in the treated water. As a result water utilities have worked hard to balance the benefits and potential risks. A certain level of chlorine residual has to be maintained in distribution systems to inhibit bacterial growth in the pipes, as required by the US Safe Drinking Water Act (SDWA). Chlorine decays in water because of its reactions with inorganic and organic solutes that impose chlorine demands. It is a challenging task for water treatment plant operators to determine the optimal chlorine dose necessary to achieve the required chlorine residual in their water distribution systems while minimizing the consumers' odor/taste complaints and ensuring compliance with the DBP regulations. Most recently, dermal and inhalation exposure during household water use activities have been recognized as significant contributors to human exposure to DBPs (e.g. Maxwell et al., 1991). Recent research by Liu and Reckhow (2013) suggested that the presence of a chlorine residual in residential plumbing could contribute to a significant increase in trihalomethanes (THMs) and haloacetic acids (HAAs) for waters that have been heated for home use. This underscores the need to expand existing chlorine decay models to include in-home heating conditions in support of our efforts to control DBP concentrations at points of exposure.

It is generally accepted that the loss of chlorine residual through distribution systems includes two separate mechanisms: homogeneous decay due to reaction between chlorine and reactants in the bulk water (bulk decay) and wall-decay from the reaction between chlorine and pipe materials, biofilm etc. If the modeling of bulk decay is sufficiently accurate, the wall-decay of chlorine can be reliably quantified by difference from in-system residual data.

First-order chlorine decay models assume the concentration of reactants other than chlorine is in excess. Under those conditions the bulk decay rate is proportional to the remaining chlorine residual concentration. It is reasonable to assume first order kinetics for bulk decay in the network modeling where freshly-chlorinated water samples are not involved (Powell et al., 2000a). However, it is problematic to assume that all organic compounds in the bulk water have the same reactivity with chlorine, and the assumption of excess reactant will not always hold especially for high initial chlorine doses and rechlorination conditions (Boccelli et al., 1997). Temporal variability is not included in these types of models; therefore, their application is limited. In addition, first-order chlorine decay models display inaccuracies when reaction time is extended (Kastl et al., 1999).

Clark and Sivaganesan (1998) proposed a second order (first order with respect to both chlorine residual and first order with a reactive component) chlorine decay model and an empirical equation for the model parameters using measurable water quality parameters (Initial chlorine residual, TOC, pH, temperature etc.). The model is essentially the same as suggested by Jadas-Hécart (1992) which has a simplified stoichiometry. Powell et al. (2000a) concluded that the second-order model (Clark and Sivaganesan, 1998; Jadas-Hécart, 1992) provides more stable model parameters, and it is better or as good as first-order chlorine decay models for samples not freshly chlorinated. Boccelli et al. (2003) extended the second-order reactive model proposed by Clark and Sivaganesan (1998) to account for discontinuities associated with rechlorination events. Huang and McBean (2007) used Bayesian statistics to estimate the coefficient based on Clark and Sivaganesan's two-component, second -order model. Although they proposed a

new approach to estimate chlorine decay model parameters, the analytical solution they derived may be questionable owing to the lack of proper boundary conditions as other researchers have pointed out (Fisher et al., 2010).

Neither the simple first-order rate model nor a second-order model with a single rate term and fixed coefficient could describe the chlorine bulk decay accurately (Jonkergouw et al., 2008; Fisher, 2009) in freshly chlorinated samples over the long term. Several studies adopted multi-stage chlorine decay processes in their modeling work to describe both rapid and slow chlorine decay. Chlorine decay was described to include initial rapid chlorine loss in minutes or hours and subsequent slower loss processes throughout the remainder of the reaction. These models include: a parallel first-order decay model for raw and alum-treated water (Gang et al., 2003); a three stage "Water Treatment Plant Model" (Malcolm, 1992); a second-order short-term model describing chlorine decay reacting with fulvic acid (Qualls and Johnson, 1983); a pseudo first-order model after initial chlorine demand (Zhang et al., 1992); a second-order chlorine decay model (Clark and Sivaganesan, 2002); and a parallel second-order model after initial chlorine demand (Jadas-Hécart et al., 1992). While providing valuable insights, these models are difficult to implement in complicated urban distribution systems. The transition time between decay stages are arbitrarily adopted (Qualls and Johnson, 1983; Malcolm, 1992; Zhang et al., 1992; Jadas-Hécart et al., 1992). Additionally, the parallel allocation of chlorine residuals between the two conceptual components (Gang et al., 2003; Clark and Sivaganesan, 2002) is not fundamentally valid, as the same amount of chlorine residual should be available to the two components as the reaction proceeds. Jonkergouw et al. (2009) used a four-parameter, second-order, variable-rate coefficient

(VRC) chlorine decay model. However, the application of the model is limited because of the arbitrary calibration for the initial concentration of the reactant and the introduction of parameter α, which has no physical meaning. Kastl et al. (1999) evaluated five simplified reaction schemes including first-order, one-reactant-site second-order, the combination of first-order and one-reactant-site second-order, parallel two-reactant-site second-order and serial reaction of chlorine with one reactant site. They found that the simplified two-site (fast and slow reactant sites), second-order chlorine decay model provides the best fit for the experimental data. Kohpaei and Sathasivan (2011) tried to derive an analytical solution for the model proposed by Kastl et al. (1999) to approximate the solution of these differential equations. It demonstrates that it is valid to assume that the fast reaction is dominant until the fast reactant site is consumed and good accuracy is maintained by modeling the fast and slow chlorine decay processes sequentially.

5.2.1 The effect of temperature on chlorine decay

Temperature has been shown to impact chlorine decay significantly (Powell et al., 2000b), which should be considered in chlorine decay modeling because of the large seasonal variability common in many distribution systems. Vasconcelos et al. (1996) proposed an empirical expression of the first order chlorine bulk decay rate constant:

$$k = \alpha * TOC * \exp\left[\frac{-E_a}{R(T+273)}\right] \quad (5.1)$$

where T is temperature (^oC), TOC is total organic carbon (mg-C L^{-1}), α is an empirical coefficient for a specific water and E_a/R is the Arrhenius ratio of activation energy to the ideal gas constant (K). The derived α and E_a/R were determined to be 1.8×10^6 $L\cdot mg^{-1}$ hr^{-1}

and 6050 K respectively. Assuming first order for the chlorine decay kinetics, Powell et al. (2000 a) adopted the classic Arrhenius equation (equation 5.2) to investigate the impact of temperature on chlorine decay rate constant for the laboratory decay tests. They simultaneously tested three different temperatures and selected 7300 K as the best fixed value of E_a/R to reduce the high variability of F (Frequency Factor, h^{-1})

$$k_T = F * \exp[\frac{-E_a}{RT}] \quad (5.2)$$

The range of 6050 to 7300 (K) corresponds to a 2.1 to 2.5 fold rise in the first order decay constant between 10 to 20 ^oC. A similar approach has been adopted by other researchers (Hallam et al, 2002.; Hua et al., 1999; Jadas-Hécart et al., 1992); however, the relationship between the chlorine decay rate constant and temperature for their data was not found to follow the classic Arrhenius model because of the oversimplification of reaction kinetics. Additionally, these types of models could not be used for data with a large variability of initial chlorine concentration.

Although the impact of temperature on chlorine decay was investigated by these researchers, the effect of temperature was rarely integrated into the chlorine decay model.

Fisher et al. (2012) extended their previous model (Kastl et al., 1999) by including a temperature effect for the two-site, second-order chlorine decay model:

$$k_T = k_{20} * \exp\left[\frac{-E_a(20-T)}{R(273+20)(273+T)}\right] \quad (5.3)$$

where k_T is the rate coefficient at temperature T, k_{20} is the rate coefficient at 20 ^oC.

They used the same E_a/R for the fast and slow reactions between reactants and chlorine and defined k_T as a function of k_{20} in order to remove F. Their chlorine decay data are from three surface waters with a temperature range of 3.5 ^oC to 28 ^oC. The ratios of E_a/R derived are in the range of 5440 to 11800 K. McClellan (2000) used a series of ODEs (ordinary differential equations) to model chlorine decay, THMs and HAAs simultaneously for surface water with a temperature range of 2 ^oC to 25 ^oC. The proposed model integrated the effect of temperature by using a standard engineering representation of Arrhenius expression for the second order chlorine decay rate constants:

$$k_T = k_{20} * \theta^{T-20} \tag{5.4}$$

where k_T is the rate coefficient at temperature T, k_{20} is the rate coefficient at 20 ^oC, θ is the temperature correction factor. By plotting the ratio of reaction rate at temperature T to reaction rate at 20 ^oC (k_T/k_{20}) as a function of temperature and activation coefficient (Ea/R), the θ value (1.0899) for the fast reactant site optimized by McClellan (2000) is equivalent to an E_a/R of 7600K (refer to Figure 5.5). Additionally, he did not provide the θ value for the slow reactive site (θ_{22}) with respect to the chlorine decay.

Based on modeling efforts in the literature which include temperature effects, it seems that a two-reactant second-order model is the most appropriate (Fisher et al., 2012). We believe that if the temperature is integrated in the two-reactant model, different values for E_a/R should be assigned for the fast reactant site and slow reactant site. Additionally, it is possible to have a single set of these E_a/R values optimized for different waters. Kohpaei et al. (2011) used separate E_a/R values for fast and slow reactant sites in a parallel second-order model for chlorine residual decay. Their dataset

spanned temperatures ranging from 15 ^{o}C to 50 ^{o}C for one water, and they achieved good fit from the model.

This study explores the modeling of chlorine residuals under in-home heating conditions, where temperatures become elevated midway through the reaction of chlorination with NOM (natural organic matter) in the water. Few of models in the literature have included data for chlorine decay under high temperatures (>30 ^{o}C), and none for in-home heating systems. The impact of temperature on the auto-decomposition of hypochlorite is not considered in this study as sodium hypochlorite solutions were found to be stable at temperatures as high as 50 ^{o}C (Gambarini et al., 1998).

5.2.2 The effect of pH on chlorine decay

The pH of water primarily determines the speciation of aqueous chlorine and the protonated form; hypochlorous acid (HOCl) is known to be a stronger oxidant than the deprotonated form, hypochlorite (OCl^{-}). The reaction kinetics for chlorine decay is therefore expected to vary for different pH values. Zhang and Andrews (2012) found no statistical difference in chlorine decay rates among samples treated at three pH values (6.6, 7.6 and 8.6) with pseudo-first-order decay constants of 0.0018, 0.0022, and 0.0022 h^{-1} respectively. In short term tests of chlorine consumption by fulvic acids isolated from surface, Qualls and Johnson (1983) failed to observe expected differences in the water across a pH range of 6.5-8.0. The authors attributed this to the offset of the change of fulvic acid reactivity. No significant relationship between pH and first-order chlorine decay constants were observed over the range of pH from 6.8 to 8.3 (Powell et al., 2000b). This non-correlation may explain the tendency to ignore pH effects in

chlorine decay models. Additionally, it is common for water utilities to add buffers to treated waters to reduce pipe corrosion which have the effect of narrowing pH range. This is another reason why researchers may be tempted to ignore pH effects.

McClellan (2000) included different rate constants and pH dependent distribution factors for the reaction of the fast reactant site with HOCl and OCl^-. In order to simplify the solution of the ODE; several assumptions were applied, which were, however, only valid at near-neutral pH. For a single reactant, Liu and Qi (2010) represent the loss of total free chlorine as:

$$-\frac{d\{[OCl^-]+[HOCl]\}}{dt} = \{k_{HOCl}[HOCl] + k_{OCl^-}[OCl^-]\}[S] \quad (5.5)$$

where k_{HOCl} and k_{OCl^-} are the second order rate constants for the reaction between the reactant with $[HOCl]$ and $[OCl^-]$, respectively. S is the general form of the reactant.

If a bulk or conditional rate constant (k_S) for the total free chlorine ($[HOCl] + [OCl^-]$) was assigned as:

$$-\frac{d\{[OCl^-]+[HOCl]\}}{dt} = k_S\{[HOCl] + [OCl^-]\}[S] \quad (5.6)$$

And based on the molar ratio of $[HOCl]$ and $[OCl^-]$

$$\frac{[HOCl]}{[OCl^-]} = 10^{pK_a - pH} \quad (5.7)$$

The conditional rate constant therefore can be determined from equation 5.8

$$k_s = \frac{k_{HOCl} + k_{OCl^-} * 10^{pH - pK_a}}{1 + 10^{pH - pK_a}} \quad (5.8)$$

The effect of temperature on the pK_a of hypochlorous acid was ignored in this model, and pK_a at 25°C (Dean, 1992) was used.

Morris (1966) proposed an equation to relate the pK_a to temperature:

$$pK_a = \frac{3000}{T} - 10.0686 + 0.0253T \quad (5.9)$$

which yields a range of 7.82 to 7.39 over a temperature range of 0 to 50 °C. The temperature impact on pK_a could be neglected as the impact of temperature on the species-specific rate constants is more significant in the typical pH range of distribution systems than is the impact of temperature on the extent of dissociation of HOCl.

5.3 Model Development

The objective of this study is to set up a model for chlorine decay that could bring both temperature and pH effects into the model. Most importantly, the model should be applicable to the complex tap water chlorine decay process in home heating scenarios.

It is assumed the chlorine depletion was due to concurrent reactions with multiple aqueous constituents, each of which can be expressed as:

$$Cl + S_i \xrightarrow{k_{s_i}} P_i \quad (5.10)$$

Where Cl represents the chlorine component, S_i is the i^{th} reactive constituent, P_i is the corresponding primary product and k_{s_i} is the rate constant.

Although the models used to describe chlorine decay historically involve one or two reactive constituents, for this study, a model that involves three constituents may be necessary because of the introduction of heating midway through the chlorine contact period. Therefore, single-constituent models, two-constituent models and three-constituent models are compared in order to select the best alternative.

Based on second order kinetics, the chlorine reactions with multiple constituents are represented by the following reactions:

$$\frac{d[Cl_F]}{dt} = \sum_{i=1}^{n} -k_{s_i}[S_i][Cl_F] \tag{5.11}$$

$$\frac{d[S_i]}{dt} = -k_{s_i}[S_i][Cl_F] \tag{5.12}$$

Where $[Cl_F]$ is the free chlorine concentration in mg·L^{-1}, $[S_i]$ is the concentration of the ith reactive constituent in mg Cl-equiv·L^{-1}. The rate constant (k_{s_i}) is represented by equation 5.13 if the two forms of chlorine rate constants are incorporated based on equation 5.8

$$k_{s_i} = \frac{k_{S_i,HOCl} + k_{s_i,OCl} * 10^{pH-pK_a}}{1+10^{pH-pK_a}} \tag{5.13}$$

Each of the rate constants is a function of temperature, which is described by:

$$k_{S_i,HOCl} = k_{S_i,HOCl,20} e^{\frac{-\frac{E\alpha_{i,HOCl}}{R} * (20-T)}{(273+T)(20+273)}} \tag{5.14}$$

$$k_{S_i,OCl} = k_{S_i,OCl,20} * e^{\frac{-\frac{E\alpha_{i,OCl}}{R} * (20-T)}{(273+T)(20+273)}} \tag{5.15}$$

where $k_{S_i,HOCl}$ and $k_{S_i,OCl}$are the rate constants for reactions of the two forms of free chlorine with the ith reactive constituents respectively. $k_{S_i,HOCl,20}$ and $k_{S_i,OCl,20}$ are the

corresponding rate constants at 20 ^{o}C. $E_{\alpha_{i,HOCl}}/R$ and $E_{\alpha_{i,OCl}}/R$ are the Arrhenius ratios of each reaction's activation energy to the ideal gas constant.

In order to model chlorine decay accurately for different scenarios, the fictive concentrations of the reactants in the water are treated as variables that need to be explicitly determined. Their values are dependent on the source of water and time of collection. The rate constants for the interaction between chlorine and reactive constituents are independent of the initial and rechlorination doses and the concentration of reactive constituents in the water but dependent on pH and temperature. Although, in our approach the rate constant is highly dependent on the nature of reactive constituents, the second order rate constants for the reactant sites are only dependent on temperature and pH. In this way, the variation due to water of differing quality could be represented by differing fictive concentrations of the reactants, and the chlorine decay data from different studies could be more directly compared. These are readily interpretable parameters and they described the underlying processes. With a single set of parameters, it appears to be sufficient to apply parallel two or four reactant models to fit chlorine decay in several very different types of waters. By using this approach, researchers have been able to achieve good fits for chlorine decay data from different fractions of natural organic matter for the same water (Jegatheesan, 2008) from surface water undergoing different types of treatment (McClellan, 2000; Jegatheesan, 2009; Fisher et al., 2004) and even from waters of different sources (McClellan, 2000; Fisher et al., 2012).

5.4 Experimental Procedures

Water samples were collected from the effluent of the water filtration plant for Northampton, MA. The plant includes an upflow roughing filter (adsorption clarifier; 7.6 gpm/ft^2 at average flow) and a downflow granular activated carbon filter (5 ft of Filtrasorb 820; 3.2 gpm/ft^2 at average flow). Alum (8-9 mg/L typical dose) is used as the primary coagulant along with small amounts of polymer. Sodium hypochlorite (1.5-1.7 mg/L typical dose) is added to the filtered water just before entering the clearwell. The finished water samples were transported to the University of Massachusetts laboratory in insulated coolers and stored at 4 ^{o}C until chlorination. The key organic characteristics of this water are listed in Table 5.1.

Water samples were first brought to 20 ^{o}C in an incubator for an hour before chlorination. A standardized stock solution of reagent-grade sodium hypochlorite was used for chlorination. Buffers (pH 6, 7, and 8) were prepared with monopotassium phosphate and varying amounts of sodium hydroxide such that the desired pH was achieved, and the final phosphate concentration was 10 mM. To prepare the final test solutions, the requisite volume of the stock buffer was transferred to 4L borosilicate bottles which were then filled with water samples. The pH of these samples was measured. Variable-volume, headspace-free "piston bottles" were prepared from borosilicate glass media bottles, Teflon lined caps and polyethylene centrifuge tubes (conical bottom). The preparation of the "piston bottles" was described in Liu and Reckhow (2013). The intent of this experimental design was to simulate chlorine decay for water traveling through a distribution system at ambient temperature and then undergoing in-home heating. Chlorine was added to the buffered water samples using

stock sodium hypochlorite solution in order to achieve a dose of 3 $mg \cdot L^{-1}$. The 4L borosilicate bottles containing chlorinated samples were incubated at 20 ^{o}C in the dark for different contact times representing a range of water ages (1h, 2 h, 6 h, 24 h, 48 h, 72 h and 96 h). At the end of each prescribed contact time, the chlorine residual was measured. After collection of these first samples, the remaining volume (except for those at 1hr, and the 2hr) was carefully partitioned into the piston bottles, headspace-free with the piston in place at the fully depressed position so that it could rise as the water sample expanded during incubation in the hot water bath. Each bottle was then tightly sealed and placed in the water bath pre-equilibrated to 55^{o} C. At each defined heating contact time (0.5hr, 3hr, 6hr and 24hr), the piston bottles were withdrawn from the 55 ^{o}C water bath, and placed in a chilled water bath (4 ^{o}C) for rapid cooling. The chlorine residuals were measured immediately.

The DPD ferrous titrimetric method, 4500-C1 F (APHA, 1998) was used to standardize the chlorine stock and analyze the chlorine residuals. The chlorine residual values used in this modeling effort are the average of two measurements if their difference is within 5% of the average. If the 5% agreement criterion is not met, a third measurement is made, and failing a third time, the data are discarded.

5.5 Methodology

5.5.1 Fitness function

The optimal parameters were obtained by minimizing an objective function (also called a fitness function of optimization) representing the sum of the squared differences between the model estimates and the actual experimental data, namely, sum of squared

errors (SSE). Under each pH condition, the chlorine decay in our system can be described as a combination of ambient temperature reactions (incubation with no heating; main process), and reactions at elevated temperature, initiated at 6, 24, 48, 72 or 96 h (incubation after subsequent heating; sub-process). Denote $n_{pH} = 3$ as the number of pH conditions, and $n_{sub} = 5$ as the number of sub-processes under each pH condition. Also denote $n_{mesMain} = 7$ (excluding the measurement at $T = 0$ where the decay starts) and $n_{mesSub} = 4$ as the number of measurements on each main process and the number of measurements on each sub process, respectively. Despite different pH values, $n_{mesMain}$ and n_{mesSub} remain the same. The SSE of the main processes can therefore be written as follows:

$$SSE_{main}(\boldsymbol{\theta}) = \sum_{i=1}^{n_{pH}} \sum_{j=1}^{n_{mesMain}} \left(\left[Cl_{F,P,main}(\boldsymbol{\theta}) \right]_{i,j} - \left[Cl_{F,M,main} \right]_{i,j} \right)^2 \quad (5.16)$$

where $\left[Cl_{F,P,main}(\boldsymbol{\theta}) \right]$ and $\left[Cl_{F,m,main} \right]$ are the model predicted concentration given parameter set $\boldsymbol{\theta}$ (vector) and actual laboratory measurements. Subscript "T" denotes time, "P" and "M" stand for "predicted" and "measured" respectively. Similarly, the SSE of the sub processes is

$$SSE_{sub}(\boldsymbol{\theta}) = \sum_{i=1}^{n_{pH}} \sum_{j=1}^{n_{sub}} \sum_{k=1}^{n_{mesSub}} \left(\left[Cl_{F,P,sub}(\boldsymbol{\theta}) \right]_{i,j,k} - \left[Cl_{F,M,sub} \right]_{i,j,k} \right)^2 \quad (5.17)$$

Thus, the fitness function, namely the SSE of the whole system, is written as the following:

$$F(\boldsymbol{\theta}) = SSE(\boldsymbol{\theta}) = SSE_{main}(\boldsymbol{\theta}) + SSE_{sub}(\boldsymbol{\theta}) \quad (5.18)$$

where again, θ is the vector of parameters.

5.5.2 Optimization algorithm

To find the optimal parameter set that best fits the data, we minimize the SSE, as defined in equation 5.18. It is obvious that the SSE is not convex with respect to the parameters we need to solve for, hence multiple local minima may exist. Therefore, conventional nonlinear regression algorithms such as the Levenberg-Marquardt or Nelder-Mead method are not suitable for such a problem, because results from these methods are very sensitive to the initial trial parameters. For example, in one of their testing scenarios (Jonkergouw et al., 2008), the solution of parameters from mechanistic chlorine decay model did not converge to a global optimum by a stochastic optimization algorithm followed by a derivative-based Levenberg-Marquardt method, largely because their stochastic optimization algorithm had difficulty ending up in the vicinity of the global optimum, which is necessary for the follow-up Levenberg-Marquardt to converge.

In this work, differential evolution, a global stochastic optimization algorithm implemented in Matlab, is utilized to search for the set of parameters that properly minimize the SSE without being trapped into some local minimum (see a pseudo code included in Appendix A). Differential evolution (DE) is a type of population-based evolutionary algorithm of function minimizer (Storn and Price, 1997). DE has been demonstrated to be generally more effective at finding the global minimum compared to other stochastic optimization algorithms such as Particle Swarm Optimization, Genetic Algorithm, and Simulated Annealing (Vesterstrøm and Thomsen, 2004). DE stochastically alters the population (generation) of agents (parameter sets) using the difference between randomly picked agents from the current generation. In searching for a lower value of the fitness function, this method updates its current population in a

greedy way in the sense that only the offspring agents that generate better or equal fitness are kept. Briefly, after the first generation of agents is initialized (usually in a random manner), the evolution of the agent population proceeds with a sequence of mutation, crossover (recombination) and selection. Once a candidate agent with better fitness is identified, it will be kept to replace the old (target) agent from which it originates. Although there are several variants of DE, population update procedure toward best fitness value is essentially the same. After experimenting with different DE variants, we adopt the variant developed by Brest et al. (2006) to serve our optimization purpose. This particular type of DE, called jDE, has been demonstrated to be simple yet effective in finding the parameter set that yields the globally best fitness value without sacrificing the capability of exploring the entire parameter space.

Another challenge in our optimization with DE is to properly apply constraints on the parameters. In this work, a set of constraints is necessary mainly for two reasons. First, constraints are needed to break the degeneracy of multiple latent processes, because they are otherwise indistinguishable. Second, a set of proper, physically motivated constraints will shrink the feasible domain and can often help facilitate the optimization. So far there has not been any generally-accepted way to handle constraints in DE, or in any kind of stochastic optimization algorithms, but several alternatives have been proposed (Michalewicz and Schoenauer, 1996). Here, we adopt a constraint handling technique suggested by Huang and Qin (2006), such that this optimization problem can be written as a nonlinear programming problem with constraints stated as the following:

$$\min \ \mathrm{F}(\boldsymbol{\theta}), \boldsymbol{\theta} = (\theta_1, \theta_2, \dots, \theta_{\mathrm{n}}), \boldsymbol{\theta} \in \boldsymbol{S} \tag{5.19}$$

$$s.\mathrm{t.} \quad \mathrm{g_i}(\boldsymbol{\theta}) \leq 0, i = 1, \dots, m \tag{5.20}$$

where $\boldsymbol{\theta}$ is the vector of parameters, $\boldsymbol{S}$ is the parameter space, and m stands for the number of inequality constraints. Given the candidate parameter set $\boldsymbol{\theta}$, for each constraint i, we define the constraint violation,$\mathrm{G_i}(\boldsymbol{\theta})$, as

$$\mathrm{G_i}(\boldsymbol{\theta}) = \max\{\mathrm{g_i}(\boldsymbol{\theta}), 0\}, \mathrm{i} = 1, \ldots, \tag{5.21}$$

We further define the total constraint violation, $\mathrm{v}(\boldsymbol{\theta})$, as

$$\mathrm{v}(\boldsymbol{\theta}) = \frac{\sum_{\mathrm{i}=1}^{\mathrm{m}} \mathrm{w_i G_i}(\boldsymbol{\theta})}{\sum_{\mathrm{i}=1}^{\mathrm{m}} \mathrm{w_i}} \tag{5.22}$$

where w_i is the weight of each constraint violation, which is defined as the reciprocal of the maximum violation obtained so far for constraint i, i.e., $\mathrm{w_i} = 1/\mathrm{G_{i,max}}(\boldsymbol{\theta})$. The purpose of using a weight factor is to scale each constraint violation properly so that each violation can be (roughly) equally reflected in the total constraint violation, $\mathrm{v}(\boldsymbol{\theta})$. By definition, it is obvious that $\mathrm{v}(\boldsymbol{\theta}) \geq 0$. A parameter set with zero violation is called a feasible parameter set, otherwise it is infeasible.

Therefore, the constraints augment the selection phase of DE with the following criteria. For a trial parameter set (agent), it will be kept to replace the target parameter set if it satisfies any of the 3 conditions: (1) it is feasible and the target parameter set is infeasible; (2) both are feasible but the trial parameter set yields a smaller fitness function value (equation 5.18), and (3) neither is feasible but the new parameter set has smaller constraint violation.

In order to distinguish the different latent decays and let DE search in the meaningful parameter domain, the constraints on the parameters are defined and listed below,

$k_{S_i,HOCl,20} > k_{S_{i+1},HOCl,20}$

$k_{S_i,OCl,20} > k_{S_{i+1},OCl,20}$

$E_{\alpha_{i,HOCl}i,j} < E_{\alpha_{i,HOCl}(i+1),j}$

$0.1 < k_{s_1} < 50$

$k_{s_2} > 0.001$

In addition to these constraints, there were no constraints placed on the parameters except for non-negativity. In addition, since we have a large parameter space without much knowledge about the value of optimal parameter set, DE is set to explore the parameter space logarithmically. We independently run DE optimization assuming there are 1, 2 or 3 latent decaying processes as previously mentioned. We terminate the optimization after at least 100,000 evaluations of fitness function, and the fitness value has not improved by more than 0.1% in the past 25 generations. In fact, this stopping criterion is very empirical and our experiments show, in this work, that such stopping criteria should be sufficient to prevent the search from missing further significant improvement.

5.6 Results and Discussion

The models fit the data with different pH values and heating conditions simultaneously by minimizing the fitness function (equation 5.18). The best-fit parameters from DE are summarized and shown in Table 5.2.

Figures 5.1, 5.2 and 5.3 show the optimized process along with the actual observations. It was found that the model assuming only one reactive constituent does a poor job of fitting the data, which was in agreement with findings of other researchers (i.e. Broccelli et al., 2003; Kastl et al., 1999).However, excellent agreement is found between laboratory-measured chlorine residual data and the best-fit model predictions assuming either two reactive constituents or three reactive constituents. In particular, the E_a/R values for the slowest reactive sites are in the range of 7700-9300 K, which means the decay rate doubles with temperature increments of 7.9 ^oC and 6.5 ^oC. The optimized E_a/R values are of similar magnitude to the single E_a/R values derived for Australian waters by Fisher et al. (2012). The results show that the concentration of the slowest reactive sites is the highest, and the chlorine decay in the heating process is largely caused by these sites.

The DE with constraints implemented in this work always converges to a global minimum given adequate evaluations of the fitness function, as shown in Figure 5.4. It can be seen that, in the case of 2 latent reactive constituents, independent DE optimizations always converge to the same global minima after approximately 2500 generations. In the case of 3 latent reactive constituents, the same behavior is observed as well. This demonstrates that the DE algorithm employed in this work is fairly robust.

5.6.1 Choosing the preferred model

In what follows, we discuss the plausibility of the models assuming 1, 2 or 3 reactive constituents. We denote a model with i constituents as $\mathcal{M}_i$. As shown above, models assuming 2 or 3 reactive constituents ($\mathcal{M}_2$ and $\mathcal{M}_3$) both show good agreement with laboratory measurements, while the one assuming 1 reactive constituent, $\mathcal{M}_1$, fails

to fit the data. Among the three models, it is expected that model $\mathcal{M}_3$ should produce the best fit of the data (with the smallest SSE), because the other two are nested to it. However, it also runs a greater risk of over-fitting. For this study, the Bayesian factor is chosen as a preferable model plausibility measure, since R^2 is believed to be an inadequate measure for goodness-of-fit of a nonlinear model and the Bayesian factor performs significantly better (Spiess and Neumeyer, 2010). From a Bayesian point of view, the relative plausibility of two different models can be parameterized by the Bayesian factor, which, in short, stands for the ratio of Bayesian evidence of two different models. As suggested by Raftery (1995), the logarithm of a Bayesian Factor between $\mathcal{M}_\mathrm{i}$ and $\mathcal{M}_\mathrm{j}$, $\mathrm{B_{ij}}$, can be approximated by:

$$\log(\mathrm{B_{ij}}) = \frac{1}{2}\left(BIC_{\mathcal{M}_\mathrm{j}} - BIC_{\mathcal{M}_\mathrm{i}}\right) \tag{5.23}$$

where BIC stands for the Bayesian Information Criterion. Empirically, if the logarithm of a Bayes factor is greater than 3, then it can be considered as strong evidence that model $\mathcal{M}_\mathrm{i}$ is more plausible than model $\mathcal{M}_\mathrm{j}$ (see, e.g., Raftery 1995, Weinberg 2013). Given a model with parameter set θ, denote $\log(\mathrm{L}|\theta)$ as the log-likelihood of the data, BIC can be expressed as:

$$\mathrm{BIC} = -2\log(\mathrm{L}|\theta) + \mathrm{k}\cdot\log(\mathrm{n}) \tag{5.24}$$

where k, n stands for the number of parameters and the sample size, respectively. According to BIC, an overly simplistic model will likely be penalized by its smaller likelihood; on the other hand a model with more parameters will be penalized for its complexity with a term proportional to the number of parameters, $\mathrm{k}\cdot\log(\mathrm{n})$, despite a

higher likelihood. This is particularly true when the models are nested. Thus, of several different models, the one with smallest BIC is statistically closer to representing the real situation. Assuming the residual follows a Gaussian distribution with a constant standard deviation, the BIC can be estimated by

$$\mathrm{BIC} \approx \mathrm{n} \cdot \log\left(\frac{\mathrm{SSE}}{\mathrm{n}}\right) + \mathrm{k} \cdot \log(\mathrm{n}) + \mathrm{n} \cdot \log(2\pi) + \mathrm{n} \tag{5.25}$$

For a model that poorly fits the data (in our case,$\mathcal{M}_1$), the Gaussian assumption for residual distribution becomes less relevant because the likelihood is nevertheless low. As for models that fit the data better ($\mathcal{M}_2$ and $\mathcal{M}_3$), such an assumption may be important. The D'Agostino-Pearson's K2 normality test on the residuals from $\mathcal{M}_2$ and $\mathcal{M}_3$ yields a p-value of 0.014 and 0.018, respectively. This means we cannot reject the null hypothesis (that the residuals do follow a Gaussian distribution) at a 1% level. Thus, BIC approximation, equation 5.25, is considered applicable. For the three models in this study, the relevant BIC information is compiled in Table 5.3.

As shown in Table 5.3, model $\mathcal{M}_2$ has the smallest BIC value. Based on equation 5.23, the logarithm of its Bayes factor with respect to $\mathcal{M}_1$ and $\mathcal{M}_3$, are $\log(\mathrm{B}_{21}) = 22.35$ and $\log(\mathrm{B}_{23}) = 7.05$, respectively. Both are well above 3. Therefore, we conclude that model $\mathcal{M}_2$ is strongly preferred by the BIC model selection rule. In other words, model $\mathcal{M}_2$ is a significantly better model for the underlying decay processes.

5.7 Conclusions

This paper proposes a new approach to modeling chlorine decay in bulk water. The derivative-based mechanistic model incorporates the effect of pH and temperature

into the chlorine decay model structure. The adoption of fictive concentrations of reactive constituents other than TOC allows the parameters to be interpretable and leads to general application of this model to different waters under varied initial conditions and scenarios. The model combines the use of a stochastic algorithm, differential evolution, to find the optimal parameters. The Bayesian factor was calculated to facilitate the selection of the most appropriate number of reactive sites. The simultaneous calibration of the model parameters was performed for laboratory controlled simulated chlorine decay tests under ambient incubation and heating conditions. The results indicate that the two-site reactive model with a single set of ten parameters is superior to the one- and three-site models for predicting chlorine residuals under these temperature varying conditions.

5.8 Appendix

Pseudo-code for parameter estimation using constrained differential evolution

Pseudo-code, an informal high-level description of the operating principle and implementation of the constrained differential evolution algorithm is listed below:

Randomly generate initial population in the parameter space with size NP = 10D

Do

For each parameter set $\boldsymbol{\theta}_{\mathbf{i,G}}$ in generation G

Randomly pick 3 parameter sets $\boldsymbol{\theta}_{\mathbf{r1,G}}, \boldsymbol{\theta}_{\mathbf{r2,G}}, \boldsymbol{\theta}_{\mathbf{r3,G}}$, where r1 ≠ r2 ≠ r3 ≠ i

Randomly pick an integer i_{rand} from [1,NP]

Choose $f_{i,G+1} = \begin{cases} 0.1 + rand_1 \cdot 0.9, & \text{if } rand_2 < \tau \\ f_{i,G}, & \text{otherwise} \end{cases}$

Choose $CR_{i,G+1} = \begin{cases} rand_3 & \text{if } rand_4 < \tau \\ CR_{i,G}, & \text{otherwise} \end{cases}$

For target parameter set $\mathbf{u}'_{\mathbf{i,G+1}}$, calculate each element $u'_{j,i,G+1}$

$$u'_{j,i,G+1} = \begin{cases} \theta_{j,r1,G} + f_{i,G+1} \cdot (\theta_{j,r2,G} - \theta_{j,r3,G}), & \text{if } rand_5 < CR_{i,G+1} \text{ or } j = i_{rand} \\ \theta_{j,i,G}, & \text{otherwise} \end{cases}$$

End For

Update

$$\boldsymbol{\theta_{i,G+1}} = \begin{cases} \boldsymbol{u'_{i,G+1}}, \text{ if } \boldsymbol{v}(\boldsymbol{u'_{i,G+1}}) = \mathbf{0} \text{ and } \boldsymbol{v}(\boldsymbol{\theta_{i,G}}) > \mathbf{0} \\ \boldsymbol{u'_{i,G+1}}, \text{ if } v\left(\boldsymbol{u'_{i,G+1}}\right) = v(\boldsymbol{\theta_{i,G}}) = 0 \text{ and } F\left(\boldsymbol{u'_{i,G+1}}\right) < F(\boldsymbol{\theta_{i,G}}) \\ \boldsymbol{u'_{i,G+1}}, \text{ if } v(\boldsymbol{\theta_{j,G}}) > v\left(\boldsymbol{u'_{i,G+1}}\right) > 0 \\ \boldsymbol{\theta_{i,G}}, \text{otherwise} \end{cases}$$

End For

Until the termination condition is met

Notation:

NP: size of population.

D: dimension of parameter space.

f: random control parameter in jDE.

CR: random control parameter in jDE.

τ: fixed control parameter in jDE, usually takes the value of 0.1.

i_{rand}: random integer from [1, NP].

$rand_{\text{k}}$: k[th] random real number from (0,1) in the current iteration, where $k = 1,2,3,4,5$.

Table 5.1 Organic Characteristic of the Northampton Water

Parameter	TOC($mg \cdot L^{-1}$)	DOC($mg \cdot L^{-1}$)	UV254(cm^{-1})	SUVA($L \cdot mg^{-1} m^{-1}$)[α]
Value	0.96	0.78	0.006	0.8

[α] SUVA (specific ultraviolet absorbance) was calculated from ultraviolet absorbance at 254 nm (UVB254) divided by the dissolved organic carbon (DOC).

Table 5.2 Parameters Determined from the Fit of Experimental Data

		1 reactive constituent	2 reactive constituents	3 reactive constituents
Parameter Name	Unit	Optimized feasible parameters		
$k_{S_1,HOCl,20}$	h^{-1} mg/L^{-1}	4.26E-3	0.194	0.215
$k_{S_1,OCl,20}$	h^{-1} mg/L^{-1}	3.86E-3	0.070	0.074
$E_{\alpha_{1,HOCl}}/R$	K	7430	3042	6403
$E_{\alpha_{1,OCl}}/R$	K	8286	4.54E-6	8.67E-8
$[S_1]$	mg Cl-equiv/L	2.650	0.657	0.597
$k_{S_2,HOCl,20}$	h^{-1} mg/L^{-1}	_	1.34E-3	4.09E-3
$k_{S_2,OCl,20}$	h^{-1} mg/L^{-1}	_	1.09E-3	4.88E-4
$E_{\alpha_{2,HOCl}}/R$	K	_	7379	8485
$E_{\alpha_{2,OCl}}/R$	K	_	9054	7489
$[S_2]$	mg Cl-equiv/L	_	3.468	1.141
$k_{S_3,HOCl,20}$	$h^{-1}mg/L^{-1}$	_	_	5.43E-5
$k_{S_3,OCl,20}$	$h^{-1}mg/L^{-1}$	_	_	2.31E-4
$E_{\alpha_{3,HOCl}}/R$	K	_	_	8507
$E_{\alpha_{3,OCl}}/R$	K	_	_	7742
$[S_3]$	Mg Cl-equiv/L	_	_	14.88

Table 5.3 Summary of regression and Bayesian Information Criteria

	SSE	n	k	BIC
$\mathcal{M}_1$	4.011	81	5	8.40
$\mathcal{M}_2$	1.760	81	10	-36.3
$\mathcal{M}_3$	1.598	81	15	-22.2

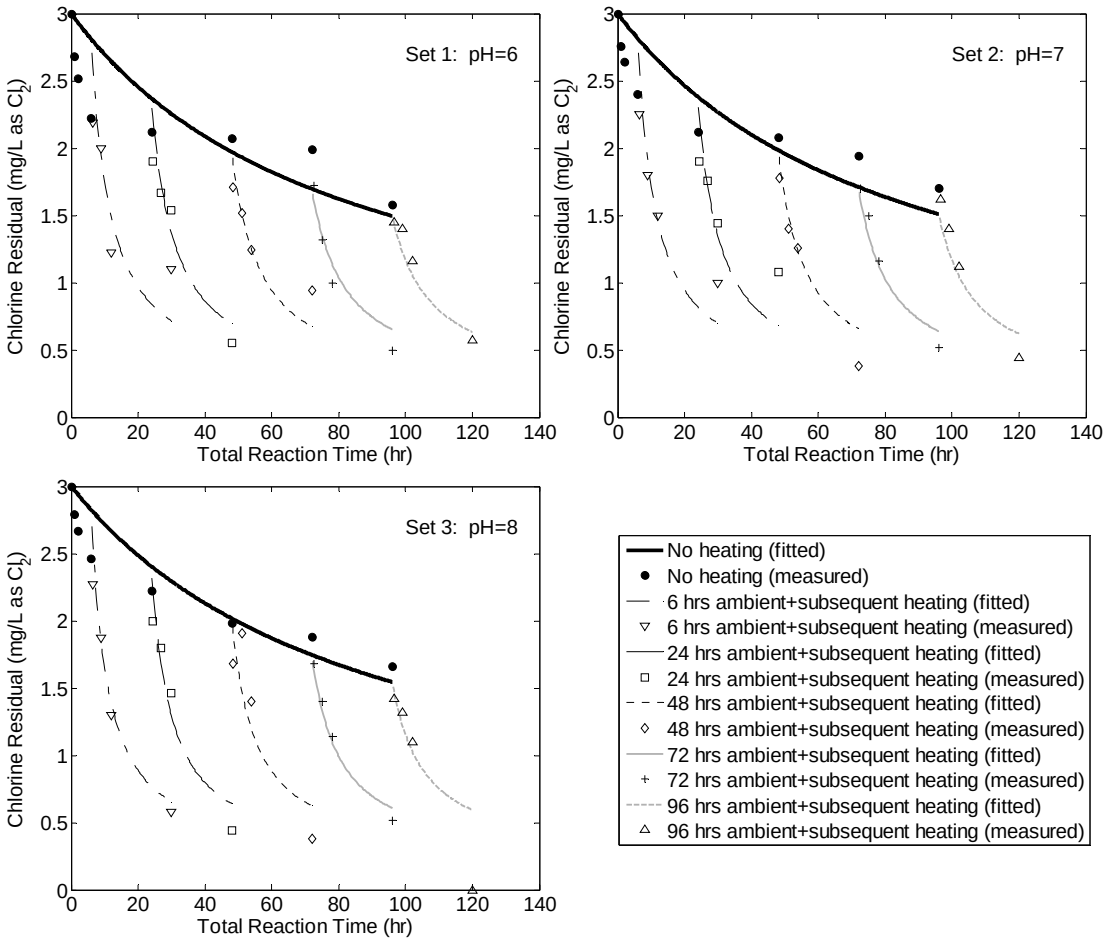

Figure 5.1 Chlorine Decay in Northampton Water for the three pHs under ambient incubation and lab heating conditions (Only one reactive constituent is assumed. It can be seen that the best-fit model does not agree with the measurements, especially at the early phase of the process)

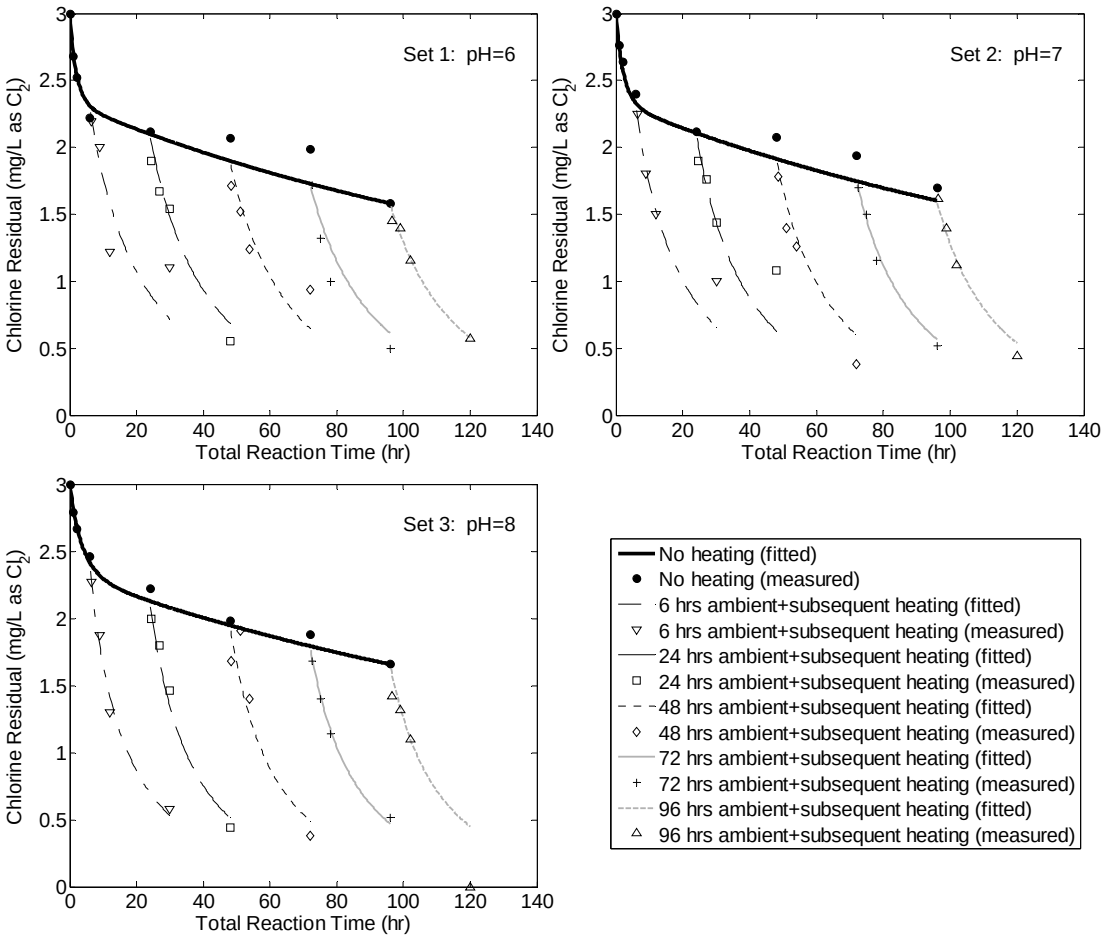

Figure 5.2 Chlorine Decay in Northampton Water for the three pHs under ambient incubation and lab heating conditions (Two reactive constituents are assumed)

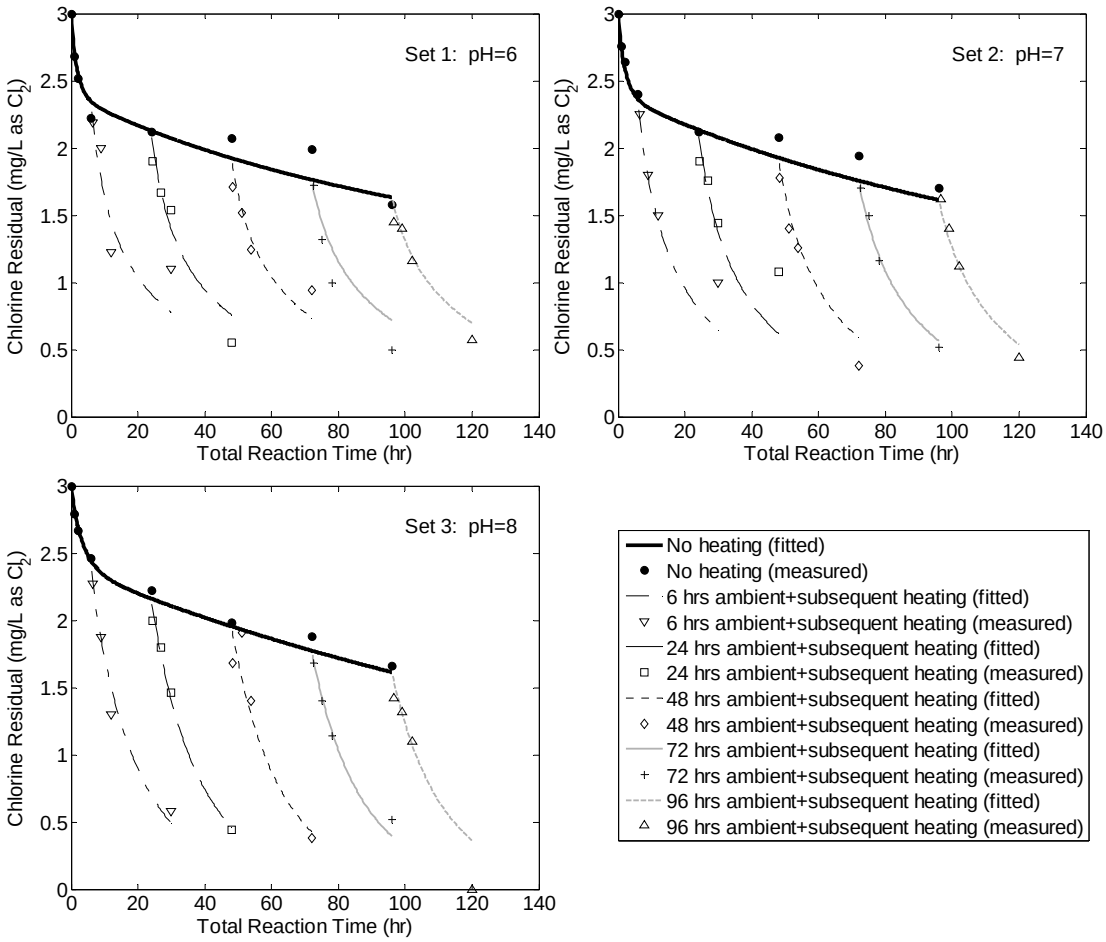

Figure 5.3 Chlorine Decay in Northampton Water for the three pHs under ambient incubation and lab heating conditions (Two reactive constituents are assumed)

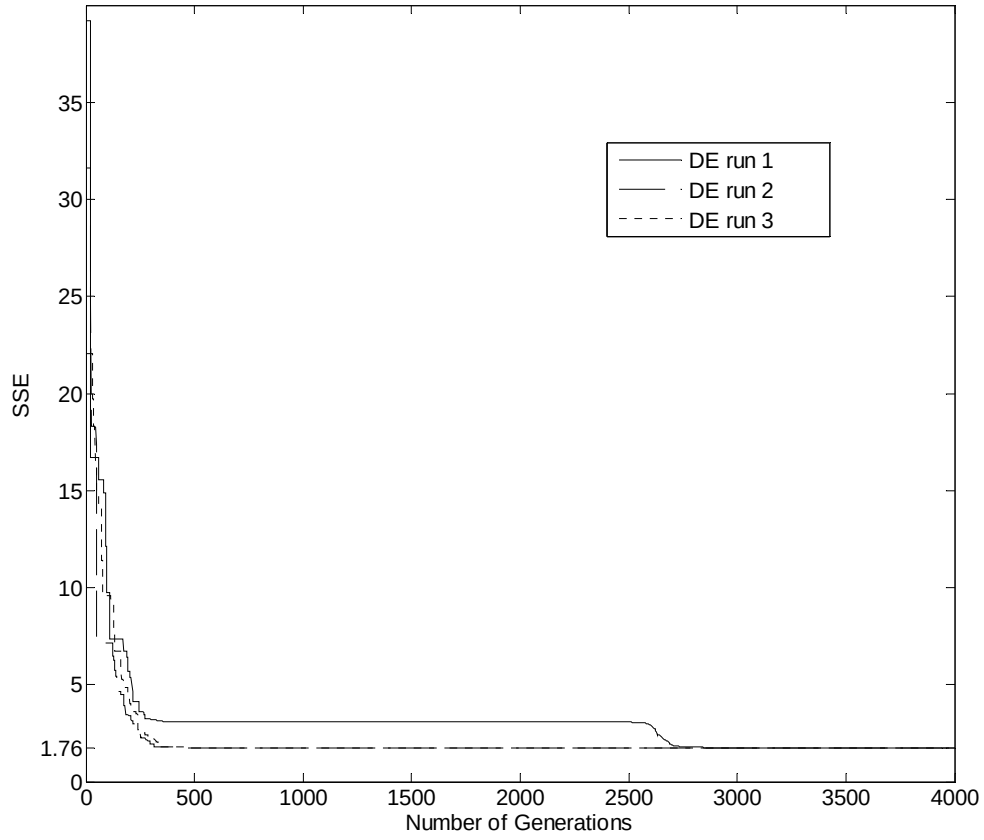

Figure 5.4 Three independent DE optimizations with random initial population, for the process with 2 assumed latent constituents

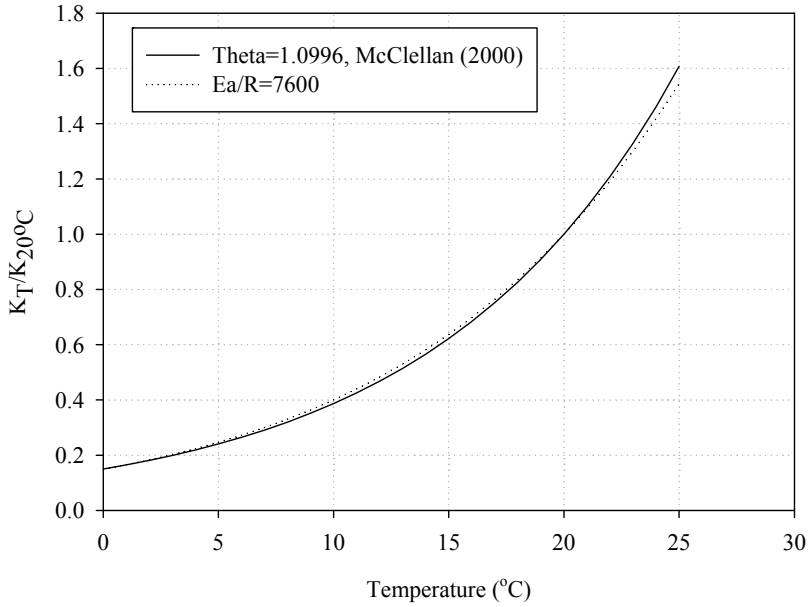

Figure 5.5 k_T/k_{20^oC} as a function of temperature

CHAPTER 6

SUMMARY

Accurate assessment of DBPs exposure is a challenging but crucial step for epidemiologic studies on DBPs. Despite limitations, this dissertation shed light on the possible misinterpretation of DBP exposure and tried to fill the gap left by the epidemiologic studies with DBP profiles in the hot tap water. Additionally, this dissertation pointed out that possible attention should be paid to cold tap water quality parameters, heating time and water age when dermal and inhalation exposure are considered for the individual's total exposure assessment to DBPs. Although direct analysis of the dermal and inhalation exposure assessment was not conducted in this dissertation, the four chapters in this dissertation demonstrate that the DBP profile and chlorine concentration in the hot tap water are quite different from those for cold tap water.

The summary of my major findings are:

1. When there are chlorine residuals in the cold tap water that feeds the water tank, chlorine is consumed quickly in hot water tank and pipes, and the elevation of THMs, DCAA and BCAA is expected. The extent of this elevation is proportional to the heating time or stagnation period of water in the hot water tank. However, the TCAA was only elevated in the hot tap water when there is high chlorine dose in the cold tap water. Disinfection byproducts, such as DCP, CP, and DCAN reveal a significant degradation in additional to the initial

increase. TCP and BDCAA have a faster degradation rate and therefore, their concentrations decrease with time under thermal conditions.

2. Although the lowest water age site has relatively lower concentration of THMs and DCAA in cold tap water, the heating of the cold tap water at the site lead to no less formation of THMs, DCAA compared to sites with highest water age.
3. The strategies that utilities use to control DBPs in cold tap water (i.e. Lower pH) may not be helpful for controlling DBP formation in hot tap water. The initial distribution system temperature also has an impact on the concentration of DBPs in hot tap water.
4. The concentrations of THM, DCAA, DCAN, TCP and CP in hot tap water out of tankless heaters are statistically different from those in the hot tap water out of tank heaters. The long term heating changed the concentrations of DBPs significantly.
5. Chlorine residual in the tap water could be modeled using differential evolution to accommodate pH and temperature effects in the two-site chlorine decay model.

Several recommendations could be given to epidemiological researchers, utilities and portable water consumers. The recommendations would be:

1. For epidemiological researchers, the stagnation and heating of the tap water should be considered instead of only relating the biomarkers and DBPs in the cold tap water (i.e. Zhang et al., 2009). When selecting the sites for epidemiological experiments on DBPs, the sites with similar water age should be picked.

2. For consumers, it is recommended to increase ventilation of air of the enclosed shower room when taking a shower and sustain use the cold and hot tap water to eliminate the water stagnation.
3. It may also be helpful for consumers to use GAC filter for chlorine residual removal to reduce the DBPs in hot water out of the water tank.
4. For water utilities, it is recommended to use low TOC/TN (total nitrogen) raw water and only provide adequate chlorine dose at the point of entry, In the future water utilities may need to monitor the terminal hot tap water for distribution system sampling point with shortest water age for THMs and HAAs.

6.1 Future Research

Researchers found that brominated DBPs have significant higher biological toxicity compared with their chlorinated analogs (Plewa et al., 2008) and temperature has a significant impact on the speciation of DBP species in the hot tap water (Dion-Fortier et al., 2009). Therefore, more research needs to be conducted to investigate the speciation distribution of the DBPs in the presence of high bromide water under the heating conditions. More detailed research should be conducted to investigate the formation, conversion and degradation of TOX and UTOX under the heating conditions. More efforts should be made to build a mechanistic model for DBPs in hot tap water based on the physical and chemical parameters and DBP concentrations of cold tap water. This approach will be useful to relate the DBPs in the distribution system to individual's actual exposure to DBPs.

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