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CONTROL OF HALOACETIC ACID FORMATION IN NORTH CAROLINA DRINKING WATER

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ABSTRACT

The objective of this research was to explore methodologies for limiting the formation of haloacetic acids (HAAs) in North Carolina drinking water so that water utilities within the State would be able to comply with the maximum contaminant levels (MCLs) for both trihalomethanes (THMs) and HAAs. Two techniques for evaluating HAA control were explored: pH adjustment and secondary chloramination.

The approach used in the pH adjustment portion of the study was to collect settled water samples from several water treatment plants in the State, chlorinate them at different pH values under controlled laboratory conditions, measure the extent of THM and HAA formation as a function of pH, and evaluate the impact of pH on THM and HAA speciation. The approach used in the chloramination portion of the research was either to sample treatment plants that already used chloramination as a secondary disinfectant or to simulate the impact of chloramination for treatment plants that used only free chlorine. In the latter case, chlorinated filtered water and/or finished water was adjusted to pH 8.0 and treated with ammonia at a concentration equal to one-fourth of the residual free chlorine concentration (on a weight basis). The treated samples were held for various times, after which the concentrations of THMs and HAAs were measured.

The results show that total THM (TTHM) formation increases and overall HAA formation decreases with increasing pH, consistent with the published literature. Trichloroacetic acid (TCAA) concentrations decreased appreciably with increasing pH, whereas dichloroacetic acid (DCAA) concentrations increased slightly. This suggests that dihaloacetic acids behave differently than trihaloacetic acids, and need to be treated differently with respect to disinfection by-product (DBP) control strategies. Chloramination effectively stopped DCAA, TCAA and overall HAA formation, as well as TTHM formation. This effect was more pronounced when ammonia was added to finished water compared to filtered water.

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I. Introduction

The application of chlorine as a disinfectant of drinking water since the early twentieth century has effectively controlled microbial diseases such as cholera, typhoid fever and diarrhea. However, in recent years, there has been much concern on the possible toxicological effects of disinfection by-products (DBPs) generated from chlorination practice.

The first and usually the dominant class of DBPs discovered in finished drinking water was the trihalomethanes (THMs). Later, researchers found a number of other DBPs that were formed as a result of chlorination practice. With the development of more sophisticated analytical techniques, several hundred halogenated and non-halogenated DBPs have been identified in treated drinking water. The haloacetic acids (HAAs) have been found to be the second most dominant class of halogenated DBPs in most drinking waters.

It has been well established that humic substances, the major component of total organic carbon (TOC) in drinking water, are the principal precursors of DBPs. Therefore, their character will determine the formation and distribution of DBPs in finished water. Other factors that also influence the formation of DBPs are type of disinfectant, chlorine dose and residual, temperature, pH, reaction time, and bromide ion concentration.

In subsequent toxicological studies conducted since their discovery in finished drinking water, some of these halogenated DBPs have been identified as possible human carcinogens and, accordingly, the US Environmental Protection Agency (USEPA) is mandated to establish maximum contaminant levels (MCLs) regulating their presence in finished drinking water. The current MCL for total trihalomethanes (TTHMs) is 100 μg/L (USEPA, 1979). Recently, the Disinfectant/Disinfection By-Product (D/DBP) Rule was published (USEPA, 1994). Stage I of this Rule (USEPA, 1998) sets a MCL for TTHMs at 80 μg/L and a MCL for five of the haloacetic acids (HAA5) of 60 μg/L. The five HAAs are monochloroacetic acid (MCAA), dichloroacetic acid (DCAA), trichloroacetic acid (TCAA), monobromoacetic acid (MBAA) and dibromoacetic acid (DBAA). Stage II of the D/DBP Rule is expected to be promulgated in May 2002.

In order to comply with increasingly stringent Federal rules, various strategies for controlling halogenated DBP formation have been explored. The most widely used strategies include precursor removal (e.g. enhanced coagulation, granular activated carbon adsorption and membrane filtration) and the use of alternative disinfectants (e.g. monochloramine, ozone, chlorine dioxide and UV irradiation).

A recent survey of selected utilities in North Carolina (Singer et al. 1995) indicated that HAA formation in North Carolina drinking water exceeds THM formation, and that utilities that are able to comply with the THM regulation will have a difficult time complying with the new MCL for HAAs. Therefore, the objective of this research was to explore methodologies for limiting the formation of HAAs in North Carolina drinking water utilities so that the utilities would be able to comply with the new MCLs for both THMs and HAAs.

Two of the less costly and easier to implement methodologies for controlling HAA formation involve pH adjustment and chloramination. These were the methodologies explored in this study. To evaluate the pH adjustment approach, settled water samples were collected from six treatment plants in North Carolina, then chlorinated in the laboratory under controlled conditions at several pH values (pH 6.0-8.5). The extent of HAA and THM formation was measured as a function of chlorination pH.

The approach for evaluating chloramination as an HAA control strategy was conducted in two stages. First, two treatment plants using chloramination (combined chlorine) as a secondary disinfectant were

sampled preceding the point of ammonia addition, immediately following the point of ammonia addition, and at several points of the distribution system to determine the effectiveness of chloramination in controlling HAA and THM formation. Second, chloramination was simulated in sample bottles by adding ammonia to chlorinated filtered and finished water from treatment plants currently using only free chlorine. THM and HAA levels were subsequently measured after 24 and 72 hours of storage under controlled conditions.

II. Background

2.1 FORMATION OF HALOGENATED DBPS IN DRINKING WATER

Research into DBP formation from the chlorination of drinking water was initiated more than twenty five years ago with the discovery of chloroform in municipal drinking waters (Rook 1974; Bellar et al. 1974). The widespread occurrence of haloforms in chlorinated waters was subsequently established by the National Organics Reconnaissance Survey (Symons et al. 1975).

While the focus of governmental regulatory attention was on the THMs (chloroform, bromodichloromethane, dibromochloromethane, and bromoform) as the most significant and prevalent class of halogenated DBPs, the list of DBPs has grown over the years. Besides the THMs, other halogenated DBPs that have been identified in finished drinking water are the HAAs (consisting of monochloroacetic acid, dichloroacetic acid, trichloroacetic acid, monobromoacetic acid, dibromoacetic acid, tribromoacetic acid, bromochloroacetic acid, bromodichloroacetic acid, dibromochloroacetic acid), haloacetonitriles (including dichloroacetonitrile, trichloroacetonitrile, dibromoacetonitrile, tribromoacetonitrile, bromochloroacetonitrile), cyanogen halides (including cyanogen chloride, cyanogen bromide), halopicrins (chloropicrin, bromopicrin), haloketones, haloaldehydes, halophenols, and halogenated furanones such as MX (3-chloro-4-(dichloromethyl)-5-hydroxy-2(5H)-furanone).

To date, hundreds of halogenated DBPs have been identified (Stevens et al. 1989), but they only account for about fifty percent of the total organic halide (TOX) concentration in finished drinking water (Christman et al. 1979; Reckhow and Singer 1984; Singer and Chang 1989). The other fifty percent of the TOX is attributed to halogenated DBPs that have not yet been identified. Di- and trichloroacetic acids have been identified as major DBPs occurring in chlorinated water (Christman et al. 1979; Quimby et al. 1980; Miller and Uden 1983; Reckhow and Singer 1984; Krasner et al. 1989). The brominated and mixed halogenated HAA species were also detected when raw water with higher bromide concentration was chlorinated (Pourmoghaddas et al. 1993; Cowman and Singer 1996).

In a survey of the occurrence of DBPs in US drinking water (Krasner et al. 1989), thirty-five treatment plants were selected to provide a comprehensive assessment of the impact of water quality parameters and treatment processes on DBP formation. The findings showed that on a weight basis, THMs were the largest group of halogenated DBPs while HAAs were the second largest fraction. The median level of THMs was almost twice that of HAAs. In a later study, Singer et al. (1995) indicated that HAA levels were as much as 50 percent greater than THM levels in finished drinking water in North Carolina.

2.2 MECHANISMS OF THM AND HAA FORMATION

Many researchers have shown that THM concentrations in finished drinking water are correlated with the total organic carbon (TOC) concentrations in raw water. The major component of TOC in most waters is natural organic matter (NOM), e.g. humic substances. It has been postulated that aquatic humic materials are a primary reaction precursor for DBP formation (Rook 1977; Babcock and Singer 1979; Christman et al. 1979; Stevens et al. 1989).

To establish a reaction pathway for the formation of chloroform and the chlorinated acids, knowledge of the structure of humic material is necessary. Humic materials are geopolymers formed from lignin, carbohydrates, proteins, and fatty acids by microbiological degradation and enzymatic or autooxidative coupling reactions (Gjessing 1976). Although the chemical structures present in aquatic humic substances are not known with any certainty, several structures have been proposed (Christman and Ghassemi 1966; Schnitzer and Khan 1972).

Because the structure of aquatic humic substances is so complex, many researchers have focused on the reaction of chlorine with simple model compounds. Many types of organic species have been found as reactive substrates for $CHCl_3$ production, such as aliphatic carboxylic acids (Rockwell and Larson 1978), hydroxybenzoic acids (Rockwell and Larson 1979), phenols and pyrrole nitrogen derivatives (Morris and Baum 1978).

Rook first documented that 1,3-dihyroxybenzenes produced high yields of chloroform under alkaline conditions (Rook 1976; Rook 1977). By conducting similar experiments at lower pH, Boyce and Hornig (1980) confirmed these observations. Christman et al. (1979) systematically studied the chlorination reactions of derivatives of NOM, notably vanillic acid, syringic acid, and 3,5-dihydroxybenzoic acid. These compounds were selected as representative structures for the phenolic polymer core of humic substances based on products detected from the alkaline copper sulfate degradation of soil and aquatic organic matter. Of the substrates chosen for study, structures related to 3,5-dihydroxybenzoic acid, i.e., resorcinol and orcinol, were the most efficient precursors of $CHCl_3$ (Norwood et al. 1980). A more detailed mechanistic study was conducted by Boyce and Hornig (1983) through a systematic analysis of the halogenation reactions of a series of dihydroxyaromatic model compounds. Their data indicated that the conversion of 1,3-dihydroxyaromatic precursors to THMs occurs in two stages. Extensive incorporation of halogen by electrophilic substitution and addition processes is followed by a complex series of hydrolysis and decarboxylation steps leading to THM formation via carbon-carbon bond cleavage at the C2-site of the aromatic ring.

It has long been accepted that a major mechanism associated with the formation of chloroform and the other THMs is the so-called haloform reaction. The haloform reaction, which occurs with methylketones or compounds oxidizable to that structure, is believed to proceed through three consecutive steps: enolization, halogenation and hydrolysis. Morris and Baum (1978) indicated that the increase of THM formation occurred without significant changes in chlorine consumption, which strongly suggests the presence of chlorinated intermediates that require alkaline conditions for hydrolysis.

There have not been many mechanistic studies on HAA formation. Reckhow and Singer (1985) developed a generalized conceptual model involving a polyfunctional unsaturated organic molecule with both aliphatic and aromatic components in order to elucidate the formation of $CHCl_3$, TCAA and DCAA. In this model, $CHCl_3$, TCAA and DCAA production was attributed to highly activated compounds containing β-diketone moieties or structures that can be oxidized. When the activated carbon on a β-diketone type moiety becomes fully-substituted with chlorine, hydrolysis analogous to that of dichloroacetylacetone would occur. DCAA is formed if the remaining R group is -OH or -OR. Otherwise this structure would be further chlorinated to a base-hydrolyzable trichloromethyl species such as trichloropyruvate. If electron pair donation occurs for the rest of the molecule, TCAA might be the major product. On the other hand, chloroform will be produced through hydrolysis without such an oxidative cleavage.

2.3 FACTORS AFFECTING THM AND HAA FORMATION

There are various factors that will influence halogenated DBP formation. These factors include pH, contact time, temperature and season, nature and concentration of NOM, chlorine dose and residual, and bromide concentration. Most of the studies conducted to date are limited to the influence of these factors on THM formation, rather than on the formation of other DBPs.

In the presence of free chlorine, THM formation increases with increasing pH, while overall HAA formation decreases with increasing pH. TOX formation decreases with increasing pH, which allows THMs to constitute a higher percentage of DBP formation at alkaline pH values (Miller and Uden 1983;

Reckhow and Singer 1985; Stevens et al. 1989; Reckhow and Singer 1990). A more detailed discussion of pH effects is given in Chapter 4, along with the experimental findings of this research.

Numerous reports have shown that TOX, THM, and HAA (total HAA as well as individual HAA species) concentrations increase with increasing contact time as long as free chlorine remains in solution (Fleischacker and Randtke 198; Reckhow and Singer 1985; Stevens et al. 1989). However, the kinetics of their formation are difficult to characterize. Generally, total THMs and HAAs, as well as their major individual species (chloroform, TCAA and DCAA), form rapidly at first followed by a slow continuing rate of formation (Miller and Uden 1983; Carlson and Hardy 1998). It was proposed by Carlson and Hardy that some components of NOM react rapidly with chlorine while others react more slowly.

Large seasonal fluctuations have been observed in THM formation. Seasonal variations may be caused by different temperatures, the nature of NOM, or bromide concentrations, all of which may change with season. For example, during the summer when temperatures are high, reaction kinetics for DBP formation are fast. One study (Kavanaugh et al. 1980) showed that the rate of THM formation doubled with every 10 degree Celsius increase in temperature. The source and composition of NOM may change with season depending upon the nature of the vegetation in the watershed. Low rainfall causes increased salt water intrusion into natural fresh waters along the coast, thereby producing increases in the formation of mixed and brominated DBP species.

NOM is comprised of humic and non-humic fractions. The humic fraction is more hydrophobic in nature and includes humic and fulvic acids. The non-humic fraction is more hydrophilic in nature and is comprised of carbohydrates, hydrophilic acids, proteins and amino acids. Historically, the humic fractions have been considered to be more important with respect to their contribution to DBP formation. More specifically, Reckhow et al. (1990) showed that the activated aromatic structures of aquatic humic material were strongly correlated to chlorine consumption and correspondingly DBP production. Quantitative parameters such as TOC concentration and ultraviolet absorbance at 254 nm (UV-254) have been demonstrated to be effective surrogates for DBP precursors. Good correlations have also been developed between THM and TOX concentrations and the values of TOC and UV-254 (Singer et al. 1981; Edzwald et al. 1985; Singer and Chang 1989). On the other hand, Owen et al. (1995) indicated that a significant amount of DBPs can be formed from the non-humic fraction of the TOC as well.

Increased chlorine dosage will result in increased DBP formation under typical water treatment conditions. In fact, there is almost a linear relationship between chlorine dosage and THM formation (Carlson and Hardy 1998). When chlorine dosage increases to a point where a constant chlorine residual is obtained, only moderate increases in DBP formation are observed (Trussell and Umphres 1978; Fleischacker and Randtke 1983; Engerholm and Amy 1983). Chlorine dosage and residual can also influence the species distribution of DBPs. At higher dosages and residuals, HAAs will form to a greater extent than THMs (Singer 1994).

Bromide ion can directly affect the rate of THM and HAA formation, the final concentration of THMs and HAAs, and their species distribution. For example, a clear correlation was found between brominated THM levels and saltwater intrusion into aquifers (Lange and Kawczynski 1978). During chlorination, if bromide ion is present, it is oxidized by chlorine to hypobromous acid that can then form brominated THMs. It is believed that when reacting with NOM, chlorine acts as a more efficient oxidizing agent while bromine is a more active halogen-substituting agent (Rook 1978; Luong et al. 1982; Symons et al. 1993). The overall rate of THM formation has been found to increase as the initial bromide concentration increases (Minear et al. 1980). An increase in total THM concentration and a simultaneous shift to the more brominated species have also been observed following chlorination of waters with increasing bromide concentration (Lange and Kawczynski 1978; Oliver 1980). Like THMs, mixed bromochloro and

brominated HAA species have also been found to exist at high bromide concentrations (Pourmoghaddas et al. 1993; Cowman and Singer 1996).

2.4 HEALTH EFFECTS OF THMS AND HAAS

All four THM species have been shown to induce tumors in one or more species or strains of experimental animals (Bull and Kopfler 1990). In various studies, chloroform has been shown to induce liver tumors in mice, kidney tumors in rats and thyroid tumors in female rats (Jorgenson et al. 1985; NCI 1976; Roe et al. 1979). Bromodichloromethane can greatly increase the incidence of renal tumors in both mice and rats, liver tumors in female mice, and intestinal tumors in both male and female rats (NTP 1986). Dibromochloromethane has been found to induce liver tumors only in male B6C3F1 mice (Dunnick et al. 1985). Bromoform was shown to induce a low incidence of rare intestinal tumors in both male and female F344 rats (NTP 1989). A recent assessment of the carcinogenic potential of chlorinated water also shows that the THMs were carcinogenic in the liver, kidney, and/or intestine of rodents (Dunnick, 1993).

Since chlorinated water has a variable composition and contains many different kinds of halogenated by-products, assessment of the risk from a specific chemical is difficult to achieve using epidemiological studies. For example, an analysis of ten epidemiological studies suggested an association between the consumption of chlorinated drinking water and increased risk of urinary, bladder and rectal cancers in humans (Morris et al. 1992). Because THMs are measured routinely in most drinking waters and are highly correlated with other chlorinated by-products, their concentration is often used as an indicator of the level of all chlorination by-products in drinking water. A population-based case-control study was conducted in Ontario, Canada using THM concentration as an estimate of the total chlorinated disinfection by-product level. The results indicated that the risk of bladder cancer increased with both duration and concentration of exposure to chlorinated by-products (King and Marrett 1996). Another study in southwest England also showed that THM concentration was one of the water quality indicators that was significantly associated with acute leukemia and myeloproliferative disorders (Foster et al. 1997).

The correlation between reproductive outcomes known to be sensitive to environmental toxicants, and long-term, low dose exposure to DBPs has received increased attention in recent years. Kramer et al. (1992) conducted a study in Iowa to evaluate the relationship between pre-term delivery, low birth weight, small for gestational age (SGA) and chlorination by-products. Chloroform concentrations above 10 μg/L were shown to be associated with a small increased risk for low birth weight and a somewhat greater risk for SGA. Another relevant study in northern New Jersey also found the risk of adverse reproductive outcomes (birth weight, pre-term delivery, SGA births and fetal deaths) was generally elevated slightly in relation to both surface (versus ground) water use and elevated THM levels (Bove et al. 1992). Similar results were obtained in a later research effort in central North Carolina (Savitz et al. 1995). These authors indicated that their results, along with the literature as a whole, can not distinguish between the absence of association and the presence of a modest association between chlorination by-products and adverse pregnancy outcome; more refined evaluations are needed.

Much less health information is available for the HAAs. DCAA and TCAA were among the first HAA species investigated because their levels were comparable to the concentration of chloroform in finished drinking water. Based on animal studies, DCAA is believed to be a more potent carcinogen than any of the THMs (Bull et al. 1990). Both DCAA and TCAA have been shown to induce hepatic tumors in mice (Bull et al. 1990; DeAngelo et al. 1991). Adverse developmental effects in rats have been observed for DCAA and TCAA (Smith et al. 1989; Smith et al. 1992). For DCAA, testicular damage (Cicmanec et al., 1991 Bhat et al. 1991) and adverse effects on sperm motility and morphology (Toth et al. 1992) have also been reported.

Both DCAA and MCAA can cause neurological damage in both man and animals (Katz et al. 1981; Bererdi et al. 1987; Stacpoole 1989). MCAA was observed by Bererdi et al. to produce permanent brain damage in animals exposed to a single oral dose (320-380 mg/Kg body weight).

When bromide is present in source waters, mixed halogen and/or brominated acetic acids can also form as a result of chlorination. Although brominated acetic acids are ubiquitous in chlorinated drinking water containing bromide, little or no health information is available for these compounds. Among the limited studies, one investigation showed that DBAA is an active spermatotoxicant. MBAA, even though it may have greater acute toxicity, has little or no antispermatogenic activity (Linder et al. 1994). The effects of HAA in whole embryo cultures have also been studied. All HAAs can directly alter development and produce dysmorphogenesis in a wide range of concentrations. The reported order of increasing potency for these chemicals was DCAA < MCAA < TBAA < TCAA < DBAA < MCAA < MBAA (Rogers et al. 1997).

2.5 REGULATION OF DBPS IN DRINKING WATER

On November 29, 1979, a maximum contaminant level (MCL) of 100 μg/L was established for total THMs (TTHMs) by the US Environmental Protection Agency (USEPA) (USEPA 1979). The MCL was based on the annual average of four quarterly sets of measurements. Compared to maximum contaminant level goals (MCLGs) which are nonenforceable, health-based levels, the MCLs are enforceable standards. They are set as close to the MCLGs as possible, but are feasible to achieve using the best available technology taking cost into consideration.

In 1981, the USEPA published a guidance document for controlling THMs in finished drinking water (Pontius 1993). Various technologies were suggested for THM control, such as precursor removal, improvement of source water quality, and use of alternative disinfectants. This guidance document also stated that other by-products may be produced by the alternative disinfectants.

On June 19, 1986, the Safe Drinking Water Act (SDWA) amendments were enacted (Pontius 1993). In addition to regulating 83 contaminants by June, 1986, these amendments also required the USEPA to subsequently regulate 25 additional contaminants every three years. On January 22, 1988, the USEPA published the first group of 25 contaminants under this regulation. These included disinfectants, THMs, and other DBPs.

From winter 1992 to spring 1994, a regulatory negotiation was conducted by the USEPA to develop revised regulations for disinfectants and DBPs (Pontius 1997). This negotiation resulted in the development of a three-rule package: the Disinfectants/Disinfection By-Products (D/DBP) Rule, the Enhanced Surface Water Treatment Rule (ESWTR) to ensure microbial quality, and an Information Collection Rule (ICR) to produce data to support the other two rules.

On July 29,1994, the USEPA published its proposed D/DBP rule (USEPA 1994). Because of a lack of knowledge regarding DBP occurrence and control and the delicate balance between the risk of microbial disease outbreaks and risks associated with disinfectants and their by-products, the D/DBP Rule was developed in two stages. Stage I proposed an MCL for TTHMs at 80 μg/L and an MCL for five of the HAAs (HAA5) of 60 μg/L. The five HAAs are MCAA, DCAA, TCAA, MBAA and DBAA. These five HAAs were selected based on existing occurrence data. Compliance with the MCLs is still based on running annual averages of measurements that are taken quarterly. Enhanced coagulation or granular activated carbon (GAC) adsorption with a 10-min empty bed contact time were proposed as the best available technologies (BATs) for compliance with the MCLs for Stage I of the D/DBP Rule. The

proposed D/DBP Rule also proposed Stage II MCLs for TTHMs and HAA5 of 40 and 30 μg/L, respectively. These levels were to be considered by stakeholders for future discussion. Additional DBPs were also to be considered in Stage II. Stage II was to be revisited after collection of additional information under the ICR and after more definitive health effects data becomes available (Pontius 1997).

2.6 METHODS FOR CONTROLLING DBP FORMATION

The methods used for controlling halogenated DBP formation include source control of precursors, precursor removal, use of alternative disinfectants and removal of DBPs after they are formed (Singer et al. 1995).

The aim of source control is to limit the presence of NOM and bromide in the water source. Algal growth has been shown to be a contributor to DBP formation in drinking water (Wachter and Andelman 1984; Karimi and Singer 1991). Therefore controlling algal growth, e.g. by controlling nutrient enrichment or nutrient cycling in source waters, can reduce DBP formation. Successful approaches include development of storm-water detention basins to trap nutrients, controlling land use activities in watersheds used as drinking water sources, installation of hypolimnetic aeration systems, and harvesting aquatic plants.

Another source control strategy that has been demonstrated to reduce DBP formation in finished water is aquifer storage and recovery (ASR). In this approach, water is abstracted and treated during seasons when raw water quality is best, then stored in aquifers, and distributed to consumers at a later date when raw water quality is not as good.

The best available technology for DBP control focuses on removal of the organic precursors, NOM. The latter is often characterized in terms of its TOC or dissolved organic carbon (DOC) concentration. Enhanced coagulation is considered to be the major strategy for TOC removal in Stage I of the D/DBP rule (Pontius 1993). However, GAC adsorption or membrane filtration may be required to meet the potential lower DBP limits for Stage II of the D/DBP rule.

For utilities that already use conventional coagulation, their BAT is enhanced coagulation. Aluminum and ferric salts are common coagulants in treatment practice and have been shown to remove NOM effectively (Reckhow and Singer 1984; Hubel and Edzwald 1987; Hundt and O'Melia 1988). The optimal pH range for enhanced coagulation with alum is 5.5 to 6.0. Factors that influence the capability of coagulants for removing TOC involve the pH of coagulation, raw water alkalinity, TOC concentrations and the hydrophobic/hydrophilic distribution of the TOC.

GAC is currently considered the most feasible adsorption option to remove NOM compared to powdered activated carbon (PAC) and other adsorptive materials. For best performance, an additional post-filtration absorber is required rather than using GAC in a filter/absorber mode. More than 15 minutes of empty-bed contact time (EBCT) is desirable and a three to six-month regeneration frequency is needed.

The greatest removal of NOM (up to 90%) can be accomplished using membrane filtration. Nanofilters, with a molecular weight cutoff of 200-500 daltons, are generally used to achieve high (>75%) TOC removal (Amy et al. 1990; Laine et al. 1993).

Many oxidants and disinfectants have been investigated as alternatives to chlorine for reducing DBP formation. These include combined chlorine (monochloramine), ozone, chlorine dioxide, permanganate, advanced oxidation processes (AOPs) and UV irradiation.

For treatment plants using chloramines as a secondary disinfectant, THMs, HAAs and other halogenated DBPs are still observed in the finished water, but at a lower level compared to what would be produced

using free chlorine. This is primarily due to the fact that, in typical practice, ammonia is added after the addition of chlorine. Moreover, several researchers have indicated that DCAA may be formed at significant levels and that cyanogen chloride may be formed at greater concentrations than from free chlorination (Smith et al. 1993; Cowman and Singer 1994). Since monochloramine requires much higher CT values (concentration of disinfectant times contact time) for disinfection than free chlorine, many utilities comply with disinfection and DBP control requirements by using free chlorine as the primary disinfectant and monochloramine as the secondary disinfectant for residual maintenance in the distribution system. Chloramination may cause nitrification problems; therefore a weight ratio of 1:4 or 1:4.5 for N:Cl2 is suggested (Wolfe et al. 1990; Lieu et al. 1993).

For chlorine dioxide, studies have been focused on its inorganic by-products, chlorite (ClO_2^-), and chlorate (ClO_3^-). It has been reported that 50-70% of the chlorine dioxide consumed during water treatment is reduced to chlorite (Werdehoff and Singer 1987). When reacting with NOM, chlorine dioxide most likely will produce oxidation by-products similar to those produced by ozonation (see below). No significant concentrations of halogenated DBPs have been observed (Rav-Acha et al. 1984; Richardson et al. 1994). MCLs of 1.0 mg/L and 0.8 mg/L have been proposed for chlorite and chlorine dioxide, respectively (USEPA 1994).

Recently, ozone has become the most attractive alternative disinfectant to chlorine because of its high microbial inactivation ability and the fact that it does not produce chlorinated DBPs. However, ozonation cannot maintain a persistent residual and may also lead to the formation of various oxidation by-products when ozone reacts with NOM (Yamada and Somiya 1989; Reckhow et al. 1992; Weinberg et al. 1993). These by-products include aldehydes, aldo- and ketoacids, carboxylic acids and peroxides. Brominated DBPs such as bromate, bromoform and brominated acetic acids, are correspondingly formed when ozone is applied to bromide-containing waters. Bromate is being regulated at an MCL of 10 μg/L; its formation can be controlled to some degree by ozonation at lower pHs (Siddiqui and Amy 1993).

Oxidation by-products of ozonation often increase the content of assimilable organic carbon (AOC) and biodegradable organic carbon (BDOC) in water. This may lead to bacterial growth problems in treatment plant basins, filters, and in the distribution system. An effective control strategy for these biodegradable oxidation by-products is to use biologically active filtration to remove them prior to distribution (Weinberg et al. 1993; Krasner et al. 1993).

Permanganate (MnO_4^-), an effective alternative oxidant for taste and odor control and iron and manganese oxidation, cannot be employed for disinfection purposes because it is a poor disinfectant. The formation of insoluble manganese dioxide [$MnO_2(s)$] can also cause operational problems if not properly controlled.

Advanced oxidation processes (AOPs) are strategies using combined oxidants, such as hydrogen peroxide and ozone, ultraviolet irradiation and ozone, or ultraviolet irradiation and peroxide. AOPs have been found to be useful in controlling taste, odor and synthetic organic contaminants such as trichloroethylene and atrazine. These processes, however, are not effective disinfection processes. Bromate and other oxidation by-products can also be formed by AOPs.

Ultraviolet (UV) irradiation can only achieve effective disinfection in low turbidity waters, and in waters with low concentrations of UV-absorbing materials. Because it does not leave a residual, UV light can only be used as a primary disinfectant. Together with the application of monochloramine as a secondary disinfectant, UV light has become an attractive option for disinfection of filtered surface water and groundwater.

Since air stripping is only effective for eliminating volatile DBPs, for example THMs, it is not considered to be a viable strategy for controlling non-volatile DBPs, such as the HAAs.

2.7 CHEMISTRY OF CHLORINATION AND CHLORAMINATION

This section describes the chemistry of chlorine and its reactions in aqueous solutions. One of the most important reactions following the addition of chlorine to an aqueous solution is the formation of hypochlorous acid. This species of chlorine is the most germicidal of all chlorine compounds with the possible exception of chlorine dioxide.

The source of free chlorine can be chlorine gas, a hypochlorite solution such as NaOCl, or $Ca(OCl)_2$ (a solid).

$Cl_2 + H_2O \rightarrow HOCl + H^+ + Cl^-$ (2-1)

$Ca(OCl)_2 + 2H_2O \rightarrow Ca^{2+} + 2HOCl + 2OH^-$ (2-2)

$NaOCl(aq) \rightarrow Na^+ + OCl^-$ (2-3)

Hypochlorous acid is a weak acid, which means that it tends to undergo partial dissociation as follows:

$HOCl = H^+ + OCl^-$ (2-4)

At 20°C, the pKa of HOCl is 7.58. HOCl dominates at pH < pKa, while OCl^- dominates at pH > pKa. At pH 7.58, the two species exist at almost equal concentrations. Morris (1966) developed a formula for the acid dissociation constant pKa as a function of temperature:

$$pKa = \frac{3000}{T} - 10.0686 + 0.0253T \quad (2\text{-}5)$$

where T = absolute temperature (°C).

The distribution of various chlorine species (e.g. OCl^- ion and undissociated HOCl) at various pH values can be calculated as follows:

$$\frac{[HOCl]}{[HOCl]+[OCl^-]} = \frac{[H^+]}{[H^+]+Ka} \quad (2\text{-}6)$$

An important aspect of water chlorination is the reaction between chlorine and various forms of nitrogen in the water. These compounds of nitrogen can be generally grouped as inorganic nitrogen and organic nitrogen species. Ammonia, nitrite and nitrate are primary inorganic nitrogen species, while amino acids and proteins are part of the organic nitrogen fraction.

The reaction of chlorine with nitrogenous compounds containing the nitrogen atom with one or more hydrogen atoms attached will form a compound classified as a chloramine. There are two distinct classes of chloramines, organic and inorganic. The inorganic chloramines are formed by the reaction of chlorine in aqueous solution with ammonia that is naturally present in the raw water or is added to intentionally produce chloramines.

In dilute aqueous solutions, the reaction between ammonia nitrogen and chlorine forms three types of inorganic chloramines in the following reactions:

$HOCl + NH_3 \rightarrow NH_2Cl$ (monochloramine) $+ H_2O$ (2-7)

$NH_2Cl + HOCl \rightarrow NHCl_2$ (dichloramine) $+ H_2O$ (2-8)

$$NHCl_2 + HOCl \rightarrow NCl_3 \text{ (trichloramine)} + H_2O \quad (2\text{-}9)$$

These parallel and sequential reactions are dependent upon pH, temperature, contact time, chlorine to ammonia ratio, and most of all, upon the initial concentrations of chorine and ammonia nitrogen.

The first of the three reactions will convert all of the chlorine to monochloramine when the ratio of chlorine to ammonia is equimolar (5:1 by weight) or less. The rate of this reaction is extremely pH-sensitive. According to reaction rates established by Morris et al. (1965), the fastest conversion of HOCl to NH_2Cl occurs at pH 8.3. The reaction rate decreases as temperature drops. The pH dependence of this reaction can be explained by the HOCl-OCl^- equilibrium (pKa = 7.58) and the NH_3-NH_4^+ equilibrium (pKa = 9.3).

In typical water treatment practice, if the chemistry of Eqn.(2-7) is practiced, the process is known as chloramination. The purpose of chloramination is to stop DBP formation and to maintain a stable disinfectant residual in the distribution system. Equations (2-8) and (2-9) are related to the "breakpoint" phenomenon. In the presence of ammonia, as the dose of chlorine is increased, the total chlorine residual increases to a certain point and then drops to a very low value, approaching zero. Subsequently, at higher chlorine doses, the residual increases linearly with chlorine dose.

In the chlorination of drinking water in the presence of organic N, both free chlorine and monochloramine react to form organochloramines. Free chlorine reacts almost instantaneously, whereas monochloramine takes several minutes. The longer the contact time, the greater will be the shift from monochloramine to organochloramine (White 1992).

It is common knowledge that chloramines are poor disinfectants compared to free chlorine. Kabler (1953), for example, showed that for the same conditions of contact time, temperature, and a pH range of about 6-8, it will take at least 25 times more monochloramine than free chlorine to produce the same germicidal efficiency. This has been explained by the slow production of HOCl by hydrolysis of monochloramine. Most organic chloramines are considered to be non-germicidal and non-toxic to aquatic life (Morris 1975).

2.8 IMPACT OF CHLORAMINATION ON DBP FORMATION

Using chloramines or combined chlorine instead of free chlorine as a secondary disinfectant can reduce THM levels significantly. Chloramines are produced by simultaneous addition of chlorine and ammonia or by adding ammonia at a later stage to convert the free chlorine residual in the water to combined chlorine. The ratio of ammonia to chlorine most often used for chloramination is 1 to 4 on a mg/mg basis (White 1992). This approach is especially attractive when chloramination is adopted as a secondary disinfectant for protection of the distribution system after primary disinfection within the treatment plant has been accomplished with free chlorine.

Once free chlorine is converted to combined chlorine, THM production essentially stops because the free chlorine residual no longer exists. The amount of THM reduction achieved depends on the amount of free chlorine contact time prior to the conversion of free chlorine to monochloramine. Some researchers reported that they reduced THM levels as much as 80 percent through the use of chloramines (Mitcham et al. 1983).

There have also been a number of studies that observed continuing formation of THMs after the addition of ammonia. For example, significant concentrations of THMs were formed in the pre-breakpoint region, and the closer the chlorine to ammonia ratio was to the breakpoint, the greater was the THM production (Speed et al. 1987; Rebhun et al. 1990). Other factors that may contribute to continuing THM formation

include hydrolysis of chlorinated intermediates at the time of ammonia addition and poor efficiency of the chloramination reaction due, for example, to poor mixing, improper pH, and low temperature.

Jensen et al. (1985) examined the reactions between monochloramine and naturally occurring organics by studying the characterization of nonpurgeable organic halide (NPOX) produced by chloramination. Compared to free chlorine, monochloramine was found to be relatively unreactive toward fulvic acid based on NPOX production, chlorine/chloramine demand values, and product identification. No products that were ether-extractable from an acidified solution were identified by gas chromatography-flame ionization techniques. More hydrophilic and higher molecular weight compounds were produced from monochloramine than from free chlorine.

Amy et al. (1990) also compared purgeable organic halide (POX) and NPOX formation as well as THM formation at two treatment utilities that used free chlorine compared to levels produced by monochloramine. Although the use of chloramination led to a reduction in the formation of THMs and POX, significant amounts of NPOX were still formed.

Cowman and Singer (1994) showed that chloramination is an effective method to reduce the formation of HAAs, a major component of NPOX. The formation of trihalogenated HAA species stopped, while DCAA formation continued, but to a much lower degree than with free chlorine. Total HAA formation was decreased by more than ninety percent in chloraminated waters.

One recent research investigation studied the effect of chloramination on both THM and HAA formation in two treatment plants (Norton and LeChevallier 1997). Chloramination resulted in lower overall DBP formation in both plants, yet one plant experienced higher TTHM reduction while the other plant experienced higher overall HAA reduction.

III. EXPERIMENTAL METHODS

3.1 EXPERIMENTAL APPROACH AND DESIGN

Two techniques for evaluating HAA control were explored: pH adjustment and chloramination. The approach used in evaluating pH adjustment for HAA control was to collect settled water samples from six treatment plants in North Carolina, chlorinate them in the laboratory under several pH conditions, and measure the extent of HAA and THM formation as a function of pH. The results from the laboratory pH testing were compared to the actual levels found in the distribution system to determine what would be expected if pH had been adjusted in the plant prior to the application of free chlorine.

The approach for evaluating chloramination as an HAA control strategy was conducted in two stages. In the first stage, two treatment plants using chloramination (combined chlorine) were sampled to determine the impact of chloramination on HAA and THM formation. Samples were collected immediately preceding the point of ammonia addition, immediately following the point of ammonia addition, and at several locations in the distribution system. In the second stage, chloramination was simulated for four water treatment plants currently using only free chlorine. This was accomplished by adding ammonia to samples of chlorinated filtered and finished water after first adjusting the pH to 8.0.

3.2 PREPARATION OF GLASSWARE

Glassware for general laboratory use was soaked in Alconox detergent overnight, rinsed with tap water, soaked in 10% nitric acid overnight, soaked in distilled, de-ionized water, then oven-dried at 110°C overnight. After drying, the glassware was either glass-stoppered or covered with aluminum foil. This procedure was repeated every 2 to 3 months. In the time period between these rigorous cleanings, glassware that had been used was rinsed copiously with de-ionized and distilled water and oven-dried.

All 40-mL vials used for sample collection, storage and extraction were soaked in detergent overnight, rinsed with tap water, soaked in a 10% nitric acid solution overnight, soaked in distilled, de-ionized water, and oven-dried at 110°C overnight. All plastics (caps, septa, etc.) were soaked in detergent overnight, soaked in distilled, de-ionized water, then oven-dried at 80°C.

All glassware used in the chlorination and chloramination study was made chlorine demand-free by soaking in a chlorine solution (approximately 25 mg/L as Cl_2) for at least 3 hours, then rinsing it well with de-ionized chlorine demand-free water (Dracor Inc., Durham, NC). Dracor water was used as de-ionized and organic-free water (DOFW) for rinsing glassware and preparing chemical solutions and aqueous standards. The Dracor water system is composed of a 1 μm pre-filter, a 1/2 cubic foot activated carbon tank that is used to remove chlorine and organic material, and two mixed bed de-ionizers. A macro-reticular resin removes any remaining organics.

Diazomethane generators for HAA analysis were cleaned according to the following procedure. After each experiment, the inner tube was immediately placed into a 5.0 N sodium hydroxide (NaOH) bath. Then silicic acid was immediately added to the outside tube to quench any residual diazomethane solution, and the tube was placed in the 5.0 N NaOH bath. Subsequently, both the inner and outside tubes were rinsed twice with tap water and were soaked in a 10% nitric acid bath overnight. The apparatus was then rinsed with DOFW three times and baked at 110°C for at least 2 hours in an oven.

Volumetric flasks used for the preparation of calibration standards were immediately rinsed and soaked with DOFW overnight after use. They were then rinsed three times with DOFW and methanol. The flasks

were then inverted to drain on a rack. They were allowed to air-dry in the fume hood for at least 3 hours prior to use.

3.3 SAMPLE COLLECTION AND STORAGE

In this research, six treatment plants in North Carolina were studied for the impact of pH adjustment and chloramination on HAA and THM formation. These plants were OWASA (Chapel Hill), Cary, High Point, Durham, Greenville and Fayetteville. All of the utilities used surface waters as their source of supply. Cary and High Point used chlorine as their primary disinfectant and monochloramine as their secondary disinfectant. The rest of the plants used chlorine as both their primary and secondary disinfectants. Each plant was sampled twice during the project, in different seasons, approximately 3-5 months apart. For each plant, water samples were collected at the treatment plant and at two locations in the distribution system.

The generic sampling flowchart (Figure 3.1) shows a schematic of the water treatment processes at the plants, the points of disinfectant and ammonia addition and the locations where samples were taken. As can be seen from the figure, the main unit treatment processes consisted of coagulation, sedimentation, filtration and storage. The treated water was then pumped into the distribution system.

Points of disinfectant addition are shown in the upper part of the flowchart. Generally, for utilities using free chlorine as both primary and secondary disinfectants, chlorine was usually added after sedimentation, at the filter influent (Cl_2 ①). For some plants, chlorine was also applied after filtration (Cl_2 ②) and/or after storage (Cl_2 ③). For utilities using free chlorine as the primary disinfectant and combined chlorine as the secondary disinfectant, chlorine was added after sedimentation (Cl_2 ①) and after filtration (Cl_2 ②). Ammonia was applied either prior to or after storage, as shown in the figure. Chlorine and ammonia dosages were taken from plant records.

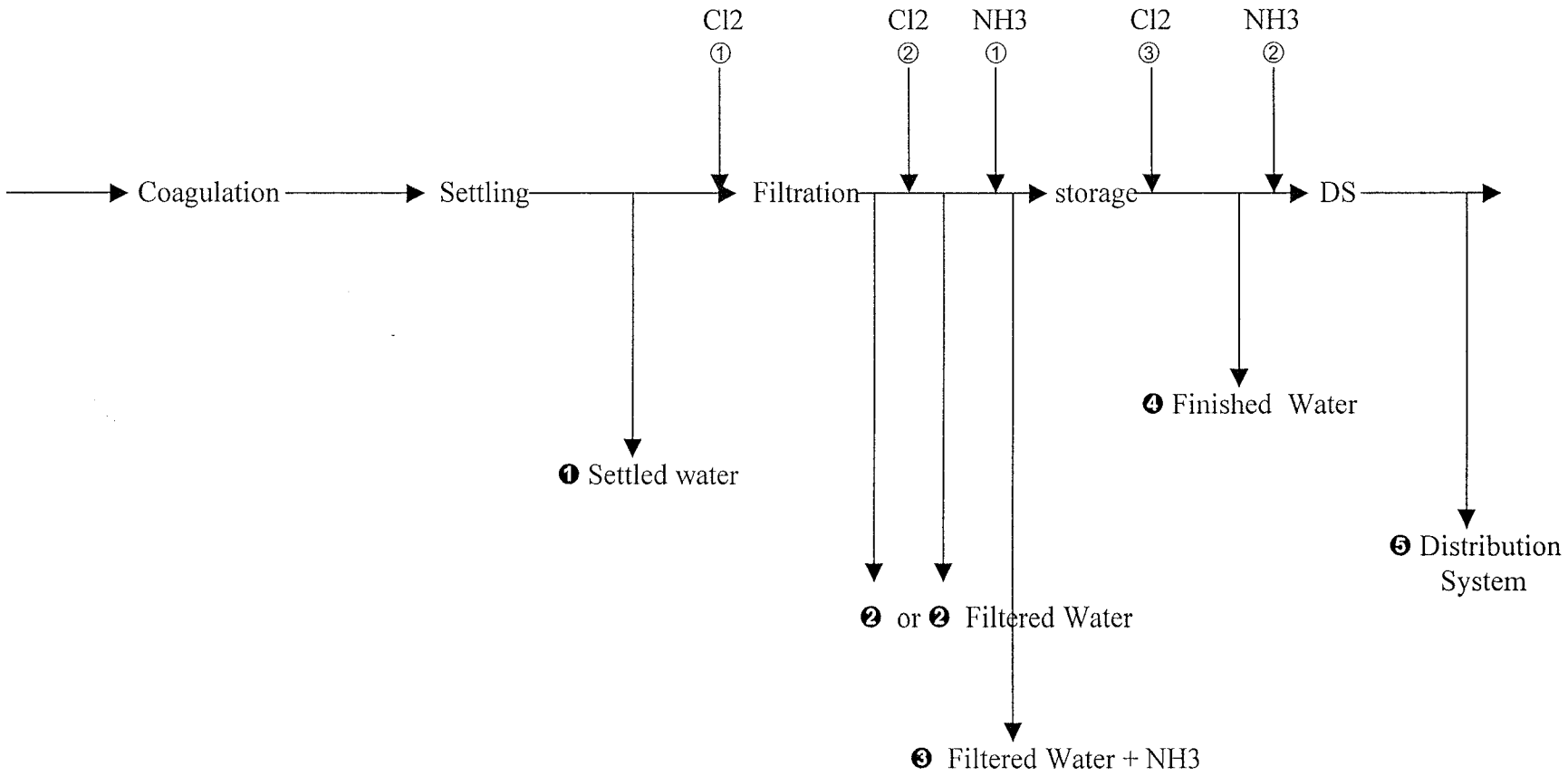
Locations of sample collection are illustrated in the lower part of Figure 3.1. At point ❶, settled water from each plant was collected for the pH adjustment experiments. These locations were prior to the application of any chlorine. The TOC concentration of these samples was measured, and the settled water pH and temperature were taken from plant records. For plants using free chlorine as both primary and secondary disinfectants, samples of filtered water ❷ and finished water ❹ were collected as indicated. For plants using combined chlorine, samples of filtered water were collected at ❷and after ammonia addition (NH_3 ①) at ❸) if ammonia was applied at NH_3 ①. Finished water samples, after storage, were also taken immediately before ammonia addition (NH_3 ②) at ❹. Detailed sampling flowcharts for each plant are given in Appendix A. Chlorine residual was measured at the time of sample collection. THM samples were quenched with sodium sulfite (ACS Grade, Fisher Scientific, Pittsburgh, PA) and HAA samples were quenched with ammonium sulfate (ACS Grade, Fisher Scientific, Pittsburgh, PA), on-site, and stored for subsequent HAA and THM analysis. Others were adjusted with phosphate buffer to pH 8.0, after which ammonia was added at a 1:4 ratio by weight of ammonia nitrogen to residual chlorine. In the chloraminated samples, HAA and THM formation, along with chlorine residuals, were measured after 24 and 72 hours of storage at 20°C.

Samples collected from the distribution system were quenched on-site and taken back to the laboratory for subsequent HAA and THM analysis. Chlorine residuals were measured at the time of sample collection.

Chlorinated and chloraminated samples were collected headspace-free, in duplicate, in 40-mL glass vials with polypropylene screw caps and Teflon-lined septa. Each vial contained the following quenching agents: 150 mg of ammonium sulfate for the samples to be analyzed for HAAs and 200 mg of sodium sulfite for the samples to be analyzed for THMs. For samples from the distribution system, the sampling

taps were allowed to flush for approximately 4-5 minutes to allow any stagnant lines to be flushed. In the treatment plants, samples were taken from running laboratory sample taps. Bottles were not rinsed before filling and were not allowed to overflow. Samples were sealed headspace-free and stored at 4°C for no more than 7 days prior to THM and HAA analysis.

Figure 3.1 Generic sampling flowchart



3.4 CHLORINATION PROCEDURES

For the chlorination studies, about 5 liters of settled water was collected from each water treatment plant on two occasions and approximately 3-5 months apart. Water samples were immediately stored in the refrigerator at 4°C after each sampling trip. The samples were brought to room temperature before each experiment. Samples were also taken for measurement of TOC. Temperature and pH values for the settled water were taken from plant records.

Chlorine working solutions were prepared by diluting 4-6% sodium hypochlorite solution (Fisher Scientific, Fair Lawn, NJ) into DOFW at a final concentration of 100 mg/L chlorine as Cl_2 for the chlorine demand study and 400 mg/L chlorine as Cl_2 for the chlorination study. Concentrations of the working solutions were determined by the iodometric method (Standard Method #4500-Cl B; APHA 1995). These solutions were prepared fresh on the day of use and were stored at room temperature in a glass-stoppered amber bottle during use.

A 0.1 M phosphate buffer (Na_2HPO_4, Mallinckrodt Specialty Chemicals Co., Paris, KY) at pH 8.0 was prepared by dissolving appropriate weights of Na_2HPO_4 into DOFW and adjusting to pH 8.0 by adding concentrated HCl (Fisher Scientific, Fair Lawn, NJ) dropwise to the solution.

A chlorine demand study was performed before each chlorination study. Prior to treatment, waters were divided into a series of five 100-mL samples. Samples were then buffered at pH 8.0 with 5 mL 0.1 M Na_2HPO_4 which yielded a 0.05 M phosphate buffer, and aliquots of the chlorine working solution were added to each sample to produce a dosage of 3-7 mg/L chlorine as Cl_2. Samples were then stored in the dark at 20°C for 24 hours. After 24 hours, the chlorine residual was measured for each sample using the DPD Ferrous Titrimetric Method (Standard Method #4500-Cl F; APHA 1995). The chlorine demand for the settled water was calculated by deducting the measured chlorine residual from the chlorine dosage. The value of the chlorine demand was used to determine the dosage of chlorine required for the chlorination study.

All pH adjustment and chlorination experiments were performed in 585 mL chlorine demand-free clear glass bottles. Samples were brought to room temperature prior to treatment. These samples were then buffered at pH 8.0 with 0.05 M Na_2HPO_4. Samples were adjusted to pH 6.0, 6.5, 7.0, 7.5 with HCl, and adjusted to pH 8.5 with NaOH. All chlorinated samples received a chlorine dose that would ensure a residual of 1-2 mg/L chlorine as Cl_2 after 24 hours of storage at 20°C.

The chlorinated samples were stored in an incubator at 20°C, in the dark, for 24 hours. After 24 hours, the chlorine residual was measured for each sample using the DPD Ferrous Titrimetric Method (Standard Method #4500-Cl F; APHA 1995). Then residual chlorine was quenched with sodium sulfite (for THMs) and ammonium sulfate (for HAAs). Samples were then taken for the measurement of THMs and HAAs.

3.5 CHLORAMINATION PROCEDURES

Two treatment plants (Cary and High Point) already using chloramination were sampled twice during the project to determine the impact of chloramination on HAA and THM formation. Samples were collected immediately preceding the point of ammonia addition, after the point of ammonia addition and at several locations in the distribution system representative of the bulk of the service population. For each sample, the free and total chlorine residuals were measured using a Hach chlorine test kit (Hach Company, Loveland, CO). The chlorine residual was then quenched with sodium sulfite (for THMs) and ammonium sulfate (for HAAs) and samples were taken for subsequent THM and HAA analysis. Chlorine and ammonia dosages, pH and temperature were taken from plant records.

Four treatment facilities (OWASA, Greenville, Durham and Fayetteville) not currently using combined chlorine were evaluated to simulate the impact of chloramination at these plants. These plants were the same ones used in the pH adjustment investigation. The chloramination simulations were performed twice, approximately 3-5 months apart. Samples of chlorinated water immediately downstream of the filters and at the point of entry to the distribution system (finished water) were collected and quenched with sodium sulfite and ammonium sulfate for THM and HAA analysis, respectfully. For purposes of simulating chloramination, ammonia was applied to a parallel set of samples and the treated samples were held for 24 and 72 hours at 20°C. Ammonia was added at a 1:4 ratio by weight of ammonia nitrogen to residual chlorine. The waters were first buffered at pH 8.0 with 0.05 M phosphate buffer to maximize the kinetics of monochloramine formation. The ammonia dosing solution was prepared by dissolving an appropriate amount of ammonium chloride (NH_4Cl, Aldrich Chemical Co., Milwaukee, WI) in DOFW to yield a concentration of 1 mg NH_3 as N/mL. After these holding times, residual chlorine was measured, after which the samples were quenched with sodium sulfite and ammonium sulfate and taken for the measurement of THMs and HAAs.

3.6 ANALYTICAL METHODS

3.6.1 Analysis of Trihalomethanes

Samples were analyzed for THMs by a modification of Standard Method 6232B (APHA 1995). In summary, a 20 mL sample aliquot was extracted with 4 mL of pentane. Two microliters of the extract was then injected into a gas chromatograph (GC) equipped with a fused silica capillary column and a linearized electron capture detector for separation and analysis. Procedural standard calibration was used in quantitative analysis.

Equipment and supplies used in this methodology included: 2-mL autosampler vials and their crimp caps (Laboratories Supplies Distributor, Mt. Laurel, NJ); micropipetors (VWR Scientific, McGaw Park, IL); 100-mL glass-stoppered volumetric flasks; disposable Pasteur pipettes for extract transfer; 5-mL amber glass vials equipped with screw caps and Teflon-lined septa for the storage of standards; vortexer; 500-mL amber glass bottles equipped with dispensers; and a muffle furnace and an analytical balance with an accuracy of 0.0001 g.

A HP-5890 Series II GC (Hewlett Packard, Palo Alto, CA) with an electron capture detector, a split/splitless injector and a model 8035 autosampler was used for the analysis. The analytical column was a fused silica DB-1 (J&W Scientific, Inc., Folsom, Calif.) with a 0.25 μm film thickness, internal diameter of 0.25 mm and 30 m in length. The carrier and make-up gases were high purity (99.999%) He and N_2, respectively.

A high purity analytical standard THM mixture in methanol (containing $CHCl_3$, $CHCl_2Br$, $CHClBr_2$ and $CHBr_3$) was obtained from Supelco (Bellefonte, PA) at a concentration of 2 g/L. The standard was kept for no more than six months. THM-grade pentane (VWR Scientific, McGaw Park, IL) was used as the extraction solvent. ACS reagent-grade sodium sulfate (Na_2SO_4) was pretreated before the experiment. It was placed in a muffle furnace and baked at 400°C overnight. It was then allowed to dry in the furnace for an hour, after which it was stored in a capped glass bottle before use.

The reference internal standard 1,2-dibromopropane was 98% pure (Aldrich Chemical Co., Milwaukee, WI). The internal standard stock solution was prepared by weighing approximately 10 mg of 1,2-dibromopropane into a 10-mL volumetric flask and bringing it to volume with methanol (Ultra-resin Grade, Doe & Ingalls, Durham, NC). This yielded approximately a 1 g/L stock solution which was used

for six months. The extraction solution with approximately 30 μg/L of the internal standard was prepared by diluting an appropriate amount of the stock solution into 500 mL of THM-grade pentane (VWR Scientific Products, McGaw Park, IL).

Aqueous calibration standards were prepared in DOFW by injecting a measured amount of the multicomponent primary or secondary standard mixture directly into water. Primary and secondary standard mixtures were made by diluting the 2 g/L standard mixture to 5 mg/L and 50 mg/L. Nine different concentration levels (0, 0.1, 0.5, 1, 5, 10, 25, 50, 100 μg/L) were prepared in 100-mL volumetric flasks.

Samples and standards were removed from storage and allowed to reach room temperature. Twenty mL aliquots of sample and aqueous calibration standards were delivered to 40-mL vials with screw caps and Teflon-faced septa by a 25-mL graduated cylinder. Exactly 4.0 mL of pentane was added with a dispenser. Six grams of oven-dried Na_2SO_4 was then added to each sample vial. The vials were recapped and the samples were mixed by vortexing for 1 minute using a Genie-2 vortex mixer (VWR Scientific Products, McGaw Park, IL).

Sample vials were allowed to sit until the pentane layer was separated. Using a disposable Pasteur pipette, a portion of the solvent phase was transferred from the 40-mL vial to an autosampler vial. Care was exercised to be sure that no water was carried over into the autosampler vial. If a dual phase appeared in the autosampler vial, it was removed and discarded using a Pasteur pipette.

The column oven temperature was programmed as follows:

[1] Hold at 35°C for 10 minutes;
[2] Increase temperature to 50°C at a rate of 5°C/minute and hold at 50°C for 1 minute;
[3] Increase temperature to 250°C at a rate of 10°C/minute and hold at 250°C for 5 minutes.

Other system parameters were:

Injector temperature: 180°C
Detector temperature: 280°C
Carrier Gas Flow rate: 1 mL/minute ultra high purity helium
Injection volume: 2 μL

Sample components were identified by comparison of retention times to retention time data from the calibration standard analysis. If the retention time of an unknown compound corresponded within limits to the retention time of a standard compound, then identification was considered positive. Sample quantitation is discussed in the section on data analysis.

3.6.2 Analysis of Haloacetic Acids

Samples were analyzed for HAAs by a modification of Standard Method 6251B (APHA 1995). In general, a 20-mL volume of sample was extracted with 4 mL of Doe & Ingalls (Durham, NC) Ultra-resin grade methyl-tert-butyl-ether (MTBE) at an acidic pH below 0.5 in order to extract the non-dissociated acid and with a salting agent Na_2SO_4 to increase the extraction efficiency. The HAAs that had been partitioned into the organic phase were then converted to their methyl esters by the addition of diazomethane in order to produce chromatographable methyl ester derivatives. Analysis was performed by a GC equipped with a fused silica capillary column and an electron capture detector for separation and analysis. Aqueous calibration standards were extracted, esterified, and analyzed in the same manner as the samples in order to compensate for extraction efficiency.

Equipment and supplies used in this methodology were the same as those used in the THM analysis, with the following exceptions: 2-mL volumetric flask with screw caps and Teflon-lined septa; a 2.5 mL syringe; ACS plus-grade sulfuric acid (Fisher Scientific, Pittsburgh, PA); MNNG diazald methane generators; diazald; carbitol; 50% KOH; silicic acid (Aldrich Chemical Co., Milwaukee, WI).

A HP-5890 Series II GC (Hewlett Packard, Palo Alto, CA) with electron capture detector, split/splitless injector and a model 8035 autosampler was used for the analysis. The analytical column was a fused silica DB-1701 (J&W Scientific, Inc., Folsom, Calif.) with a 0.25 μm film thickness, internal diameter of 0.25 mm and 30 m in length. The carrier and make-up gases were high purity (99.999%) He and N_2, respectively.

A high purity analytical standard HAA mixture in MTBE (containing MCAA, MBAA, BCAA, DBAA, DCAA, TCAA) was obtained from Supelco (Bellefonte, PA) at a concentration of 2 g/L. The other three HAAs (BDCAA, CDBAA and TBAA) were purchased individually from Supelco at a concentration of 1 g/L. A mix of HAA6 and HAA9 esters were also obtained from the same source. Standards were kept for no more than six months. Ultra-resin grade MTBE (Doe & Ingalls, Durham, NC) was used as the extraction solvent. ACS reagent-grade sodium sulfate (Na_2SO_4) was pretreated in the same manner as in the THM analysis.

The same internal standard stock solution of 1,2-dibromopropane was used as in the THM analysis. An extraction solution with approximately 30 μg/L internal standard was prepared by diluting appropriate amounts of the stock solution into 500 mL of MTBE.

The surrogate used in this method was 2,3-dibromopropionic acid. A surrogate stock solution was obtained from Supelco (Bellefonte, PA) at a concentration of 1 g/L in MTBE. A surrogate spiking solution was made by delivering 50 μL of the surrogate stock solution into a 5-mL volumetric flask. The spiking solution was stored at -11°C for no more than three months.

Aqueous calibration standards were prepared in Dracor water by injecting a measured amount of the multicomponent primary or secondary standard mixture directly into water. Primary and secondary standard mixtures were made by diluting the 2 g/L and 1 g/L standard mixtures to 5 mg/L and 50 mg/L. Seven different concentration levels (0, 1, 5, 10, 20, 50, 70 μg/L) were prepared from the HAA standard mixtures in 100-mL volumetric flasks. Since bromide ion concentration in North Carolina raw waters is quite low, brominated HAAs were expected to be present only in low concentrations. For the three individual brominated HAAs, six different concentration levels (0, 1, 2, 5, 10, 20 μg/L) were prepared.

Samples and standards were removed from storage and allowed to reach room temperature. The whole procedure was carried out in a fume hood. Twenty mL aliquots of sample and aqueous calibration standards were delivered to 40-mL vials with screw caps and Teflon-lined septa by a 25-mL graduated cylinder. Twenty μl of the surrogate spiking solution at a concentration of 0.01 g/L was added to each vial. The vial was gently inverted once to mix the contents.

During the extraction process, 2 mL of concentrated H_2SO_4 was added to each vial to lower the pH to less than 0.5. Exactly 4.0 mL of MTBE was then added with a dispenser. Six grams of oven-dried Na_2SO_4 was added to each sample vial. The vials were recapped and the samples were mixed by vortexing for 1 minute using a Genie-2 vortex mixer (VWR Scientific Products, McGaw Park, IL).

The derivatization and quenching process was performed as follows. Using a disposable Pasteur pipette, 2 mL of the MTBE layer was transferred from the 40-mL vial to a 2-mL volumetric flask. Care was taken to ensure that no water was carried over into the autosampler vial. If a dual phase appeared in the

autosampler vial, it was removed and discarded using a Pasteur pipette. The volumetric flask was placed in the freezer for 7 minutes. Cooling the extracts was unnecessary if the diazomethane was cold. 175 µl of a cold diazomethane/MTBE solution was then added to each volumetric flask. The mixture was gently inverted once. The flasks were held for 15 minutes in a 4°C explosion-proof refrigerator. After that, the extracts were placed in the hood for 15 minutes until they reached room temperature. 0.1 g silicic acid was added to quench excess diazomethane. The samples were then transferred to autosampler vials with disposal Pasteur pipettes. Unused diazomethane reagent was also quenched with silicic acid.

Diazomethane was prepared according to the following procedures. The MNNG diazomethane-generation apparatus was set up on ice, and a 5 N NaOH bath was prepared to destroy unreacted diazald. Three mL of MTBE was added to the outer tube of the generator; 1 mL MTBE was added to the inner tube through its screw-cap opening, along with 1 mL of carbitol. Then 0.3-0.4 g of diazald was added to the inner tube and the two parts were assembled together.

The generator was clamped, and the screw-cap top with the Teflon-lined septa was closed and allowed to cool on ice for 15 minutes. Afterwards, 1.5 mL of 37% KOH was added drop-wise to the inner tube with a gas-tight syringe. Diazomethane was allowed to form for 30-45 minutes on ice. Every 10 minutes, the apparatus was gently shaken by hand. After this time period, the septum was punctured with a syringe to release pressure. The diazomethane was transferred to a 40-mL vial and stored in an explosion-proof freezer for no longer than 1 day.

The column oven was temperature-programmed as follows:

[1] Hold at 35°C for 10 minutes;
[2] Increase temperature to 75°C at a rate of 5°C/minute and hold at 75°C for 15 minutes;
[3] Increase temperature to 100°C at a rate of 5°C/minute and hold at 100°C for 5 minutes;
[4] Increase temperature to 135°C at a rate of 5°C/minute and hold at 135°C for 10 minutes.

Other system conditions were:

Injector temperature: 200°C
Detector temperature: 260°C
Carrier Gas Flow rate: 1 mL/minute ultra high purity helium
Injection volume: 2 µL

Sample components were identified by comparison of retention times to retention time data for the nine HAA ester standards. If the retention time of an unknown compound corresponded within limits to the retention time of a standard compound, then identification was considered positive. The quantitation of HAA concentrations is discussed in the next section.

The HAA6 species were measured in all samples collected; the other three HAA species (BDCAA, DBCAA, TBAA) were measured in only some of the samples.

3.7 QUALITY CONTROL AND DATA ANALYSIS

Quality Control:

Before processing any samples, reagent blanks (both solvent and DOFW) were run in order to demonstrate that all glassware and reagent interference was under control. Duplicate analyses were made for both calibration standards and samples. The relative standard deviation for the internal standard and component peak area was always less than 10%.

In the analysis of HAAs, a surrogate analyte was added to all calibration standards and samples. The surrogate concentration was chosen to give a peak response that was somewhere between that of the most predominant peak in the sample and that producing the lowest response which was quantifiable. The surrogate response was used to monitor the experimental procedure from extraction precision to quantitative analysis. The relative standard deviation for the surrogate peak area was always lower than 10%.

Calibration and Standardization:

Seven or more calibration standards were used in these analyses. One was at a concentration of the analytes that was near to but greater than the method detection limit. The others were evenly distributed throughout the concentration range expected in the unknowns. These standards were subject to change due to the different expected concentrations of the samples.

Calibration curves were made by plotting relative peak area versus concentration. Relative peak area was calculated as follows:

$$\text{Relative Peak Area} = \text{Component Peak Area}/\text{Area of Internal Standard} \quad (3.1)$$

One value that was used to monitor the linearity and credibility of the calibration curve was R^2. When this value was larger than 0.99, the calibration curve was accepted. Otherwise the deviant points were discarded and the data were re-analyzed.

Data Analysis and Calculation:

Peak identification for the HAA analysis was conducted by direct injection of the methyl ester standard mix with the internal standard to determine relative retention times (RRTs). Sample components were identified by comparison of the retention time to the retention time of the standard esters.

A standard of moderate concentration (i.e. 40 μg/L) was then run. Each component was identified by comparing the relative location of each chromatogram to the standard chromatogram. After identification of each component, the RRT of each component was calculated as follows:

$$\text{RRT} = \text{Retention Time of Component}/\text{Retention Time (RT) of Internal Standard (IS)} \quad (3.2)$$

For each experiment, the solvent with the internal standard (IS) was run first. Then the retention time of each component for every experiment was confirmed as:

$$\text{Retention Time of Component} = \text{RRT of Component} \times \text{RT of IS in the experimental run} \quad (3.3)$$

Peak areas from the chromatograms were first incorporated into an EXCEL spreadsheet (Microsoft Corporation) for analysis. Then the concentrations of the unknowns in the samples were calculated from the calibration curve for each component. Results were accepted if the relative standard deviation of the relative peak area did not exceed 10%. The average of replicates was reported as the final concentration for a particular sample.

IV. RESULTS AND DISCUSSION

4.1 PLANT SCALE RESULTS

Among the six treatment plants that were studied, four plants used free chlorine as both their primary and secondary disinfectants. Two used chlorine as the primary disinfectant and monochloramine as the secondary disinfectant. Results are presented and discussed for only one of the plants using free chlorine; the results for the others are similar and are given in Appendix B. Results for both plants that used combined chlorine are presented and discussed in this section. Each of the six plants was visited on two occasions, approximately 3-5 months apart.

4.1.1 Results for Systems Using Free Chlorine

Table 4.1 presents the chlorine dosage, sample locations and respective chlorine residuals for each sampling point in Greenville. Referring to the generic flowchart (Figure. 3.1), Table 4.1 indicates the positions where samples were collected, relevant chlorine dosages and points of application, and settled water quality information. Parallel tables and figures are given in Appendix B for the other three systems using only free chlorine. Greenville applied chlorine to the settled water immediately ahead of the filters and again before clearwell storage. Samples were collected two times from this utility, in August and December. Finished water was collected within the plant, after filtration and clearwell storage, and immediately ahead of the high-service pumps. Samples were also collected at two representative locations in the distribution system, MacThrift and Darryls. These are two of the regular compliance monitoring stations used by Greenville.

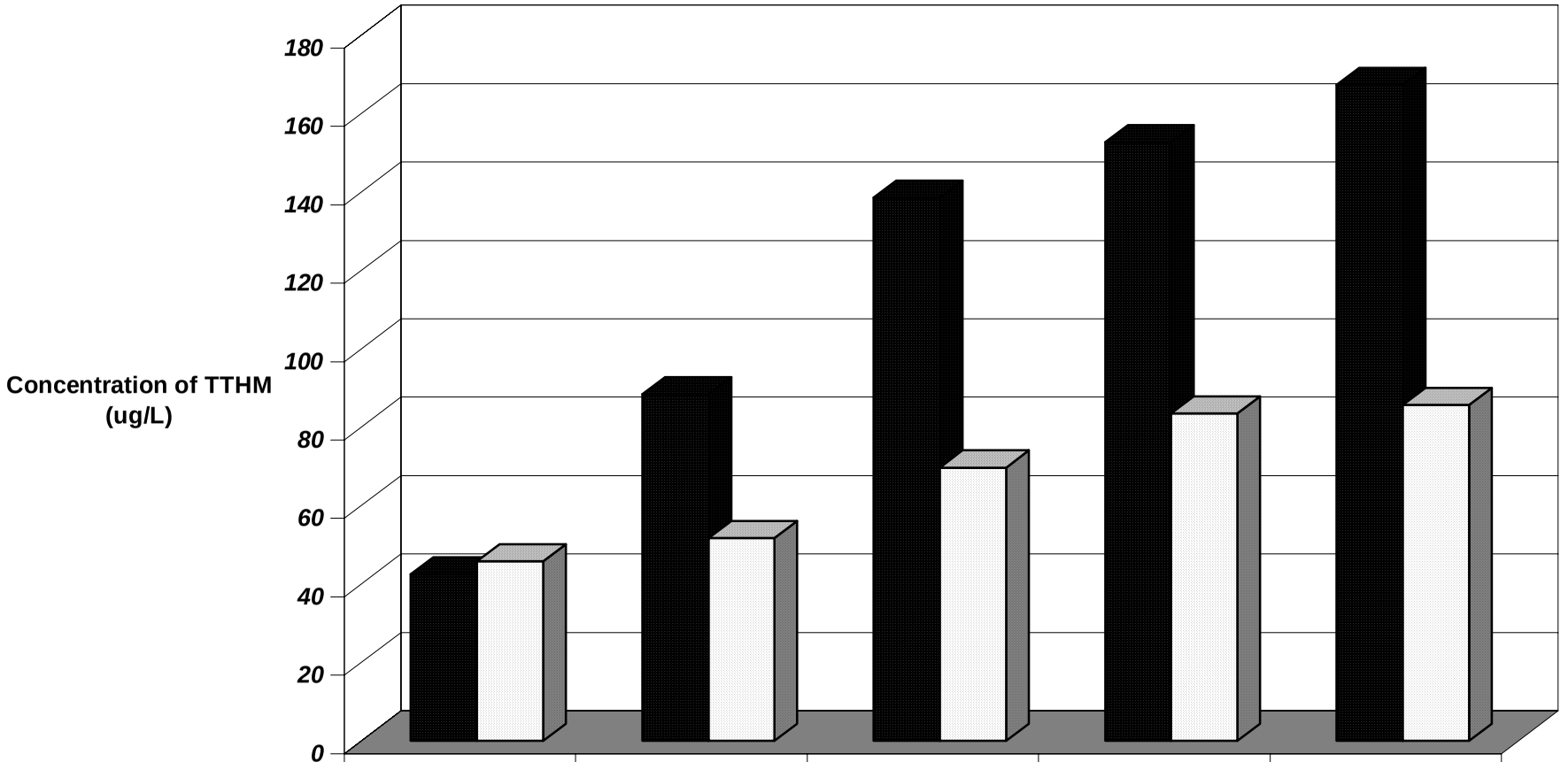
Table 4.1 Chlorine residual in plant and distribution system (Greenville, NC)

	Sample Location				
	Filtered	*Clearwell*	*Finished*	*MacThrift*	*Darryls*
Cl_2 residual mg/L (8/6/97)	0.9	3.17	2.19	1.9	1.4
Cl_2 residual mg/L (12/1/97)	0.7	2.85	1.75	1.45	1.25

* On 8/6/97, settled water Cl_2 dose was 1.8 mg/L, filtered water Cl_2 dose was 2.6 mg/L. Temperature was 27°C, TOC of the settled water was 4.04 mg/L.
On 12/1/97, settled water Cl_2 dose was 2.0 mg/L, filtered water Cl_2 dose was 1.7 mg/L. Temperature was 11°C, TOC of the settled water was 2.9 mg/L.

The patterns of formation of TTHM, HAA6, DCAA and TCAA in the plant and distribution system are shown in Figures 4.1-4.4. TTHM concentrations and HAA6 concentrations increase with increasing contact time through the treatment plant and out into the distribution system. Concentrations of these DBPs were higher in August than in December, which may due to the higher temperature, higher TOC concentration, and/or the greater dose of chlorine applied in August than in December. The two principal HAA species, DCAA and TCAA, increase correspondingly. HAA6 and TTHM concentrations were similar.

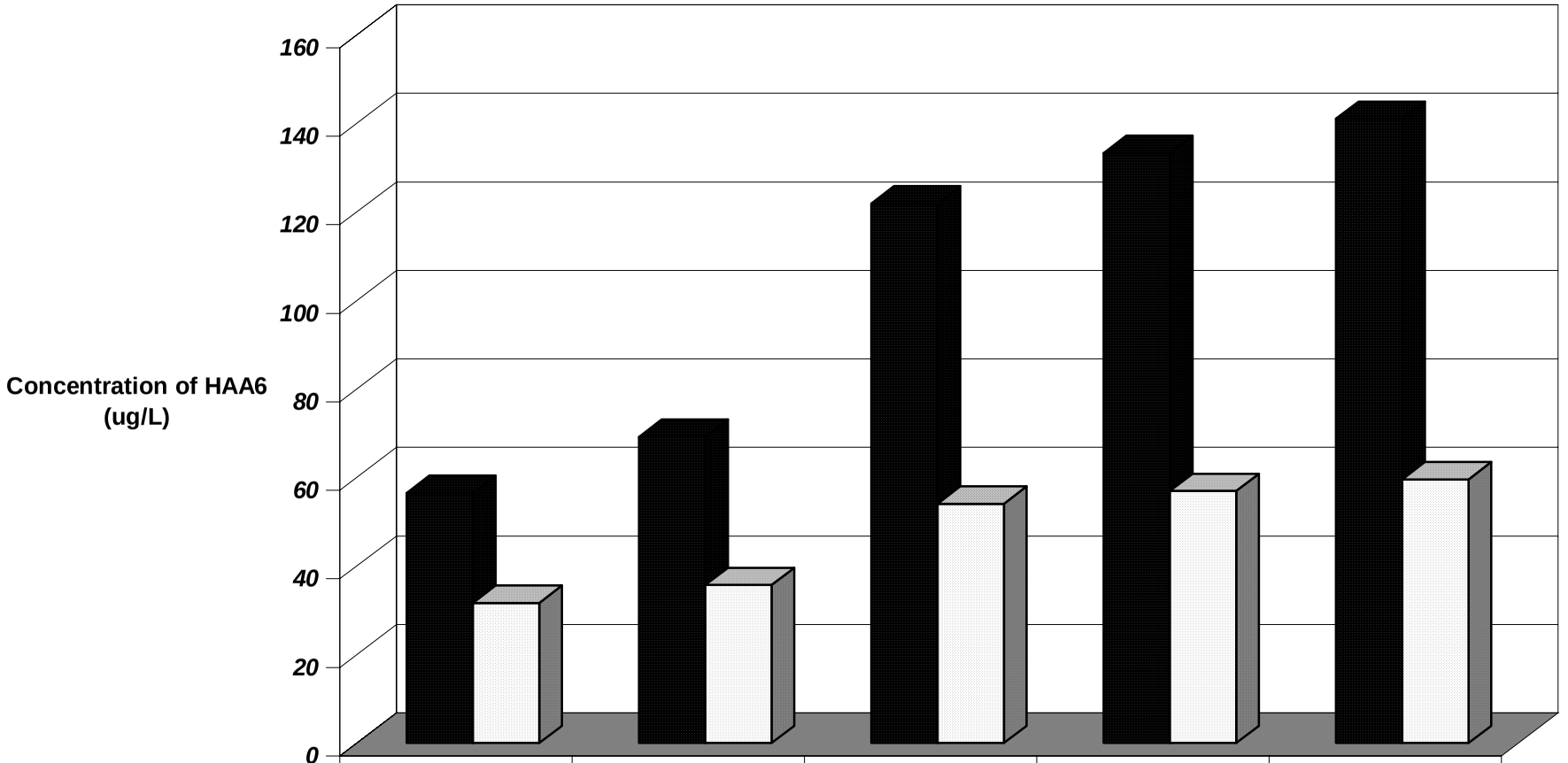
Figure 4.1 TTHM Concentrations in plant and distribution system (Greenville, NC)



	Filtered	Clearwell	Finished	MacThrift	Darryls
■ TTHM ug/L (8/6/97)	42.5	88.4	138.6	152.7	167.3
□ TTHM ug/L (11/10/97)	45.8	51.7	69.7	83.5	85.7

Sampling points

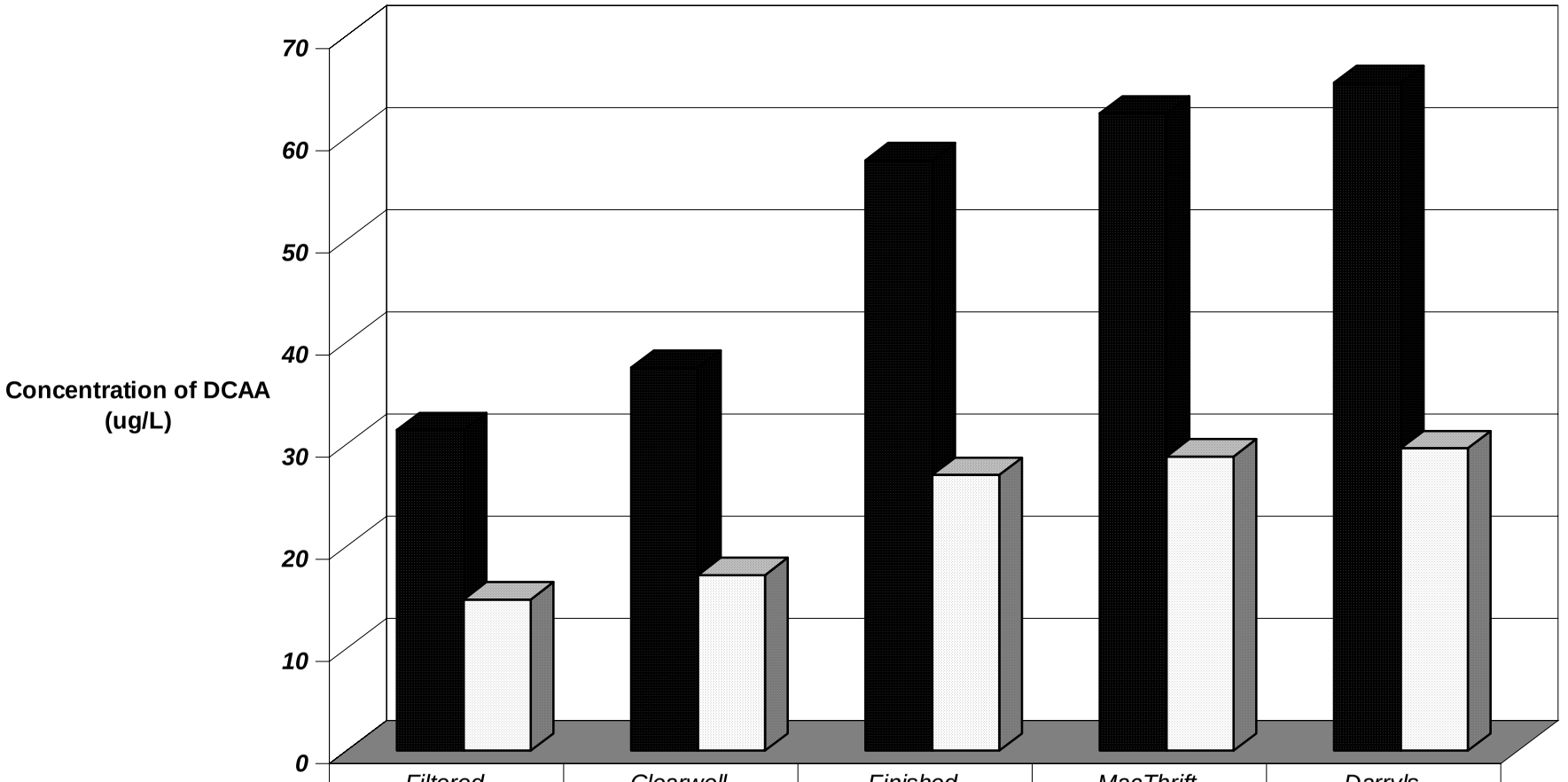
Figure 4.2 HAA6 Concentrations in plant and distribution system (Greenville, NC)



	Filtered	Clearwell	Finished	MacThrift	Darryls
HAA6 ug/L (8/6/97)	56.5	69.2	121.9	133.3	141
HAA6 ug/L (11/10/97)	31.7	35.7	54.1	57	59.6

Sampling points

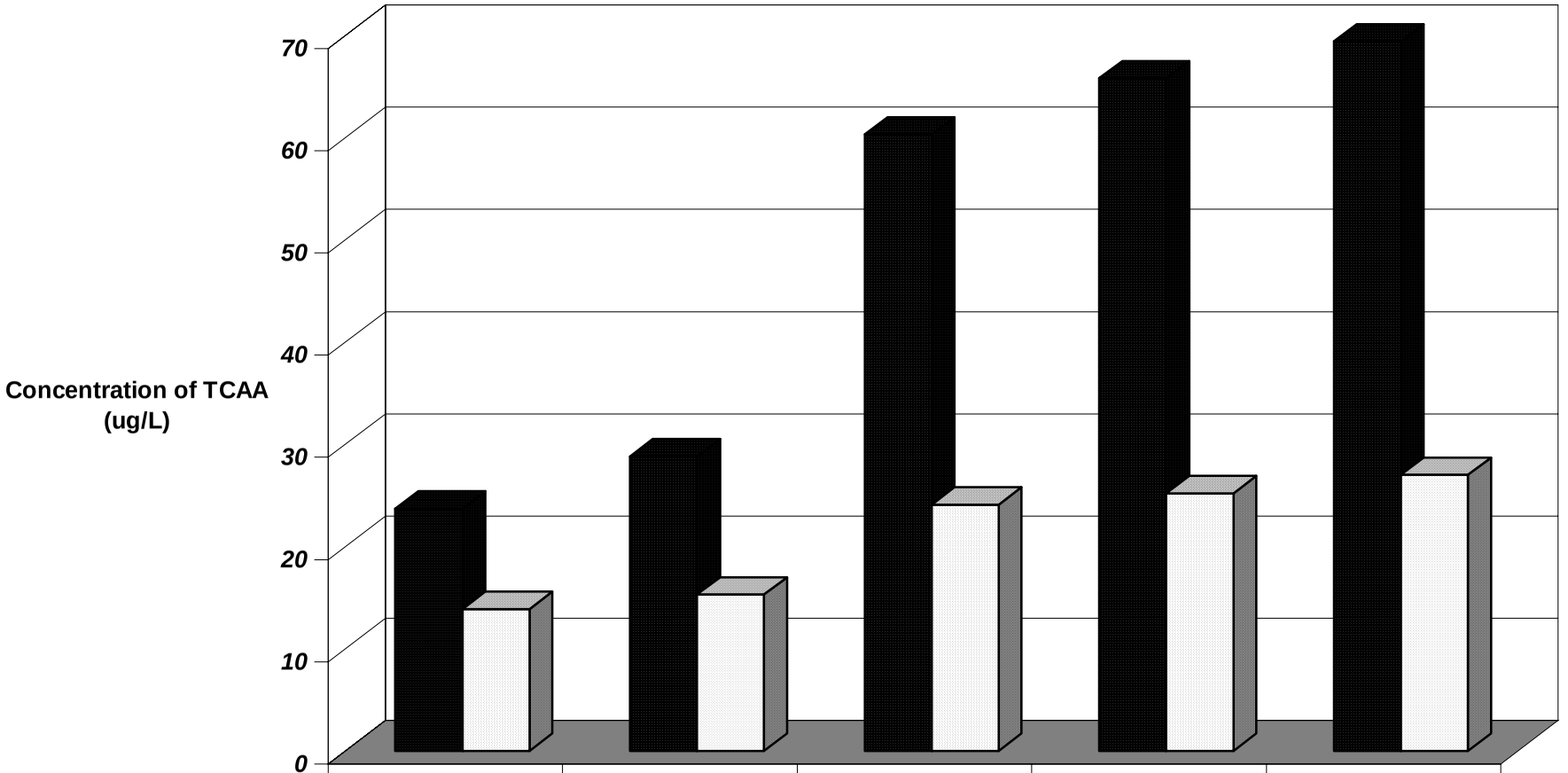
Figure 4.3 DCAA Concentrations in plant and distribution system (Greenville, NC)



	Filtered	Clearwell	Finished	MacThrift	Darryls
DCAA ug/L (8/6/97)	31.4	37.5	57.8	62.4	65.4
DCAA ug/L (11/10/97)	14.8	17.2	27	28.8	29.6

Sampling points

Figure 4.4 TCAA Concentrations in plant and distribution system (Greenville, NC)



	Filtered	Clearwell	Finished	MacThrift	Darryls
■ TCAA ug/L (8/6/97)	23.7	28.8	60.3	65.8	69.4
□ TCAA ug/L (11/10/97)	13.9	15.3	24.1	25.2	27

Sampling points

For all samples analyzed, $CHCl_3$ was the dominant THM species, and DCAA and TCAA were the principal HAA6 species. An attempt was made to measure HAA9, but analytical difficulties were encountered due to contamination of the standards and instability of the bromine-containing trihaloacetic acids.

4.1.2 Results for Systems Using Combined Chlorine

Cary adds free chlorine as a primary disinfectant before and after filtration. Ammonia is applied to convert free chlorine to monochloramine just before the water enters the distribution system (at NH_3 ②, Figure 3.1). Table 4.2 shows the sample locations, chlorine dosages and respective chlorine residuals for each sampling point. The addition of ammonia stabilizes the chlorine residual as NH_2Cl at about 3 mg/L in the distribution system. The free chlorine residual decreases to very low levels after the addition of ammonia.

Figures 4.5-4.8 show that THMs and HAAs continue to form in the plant in the presence of free chlorine, but addition of ammonia to the finished water essentially stops subsequent TTHM and HAA6 formation. Concentrations of TTHM and HAA6 at Preston Crossroads and Town Hall in the distribution system are essentially the same as those in the finished water leaving the plant.

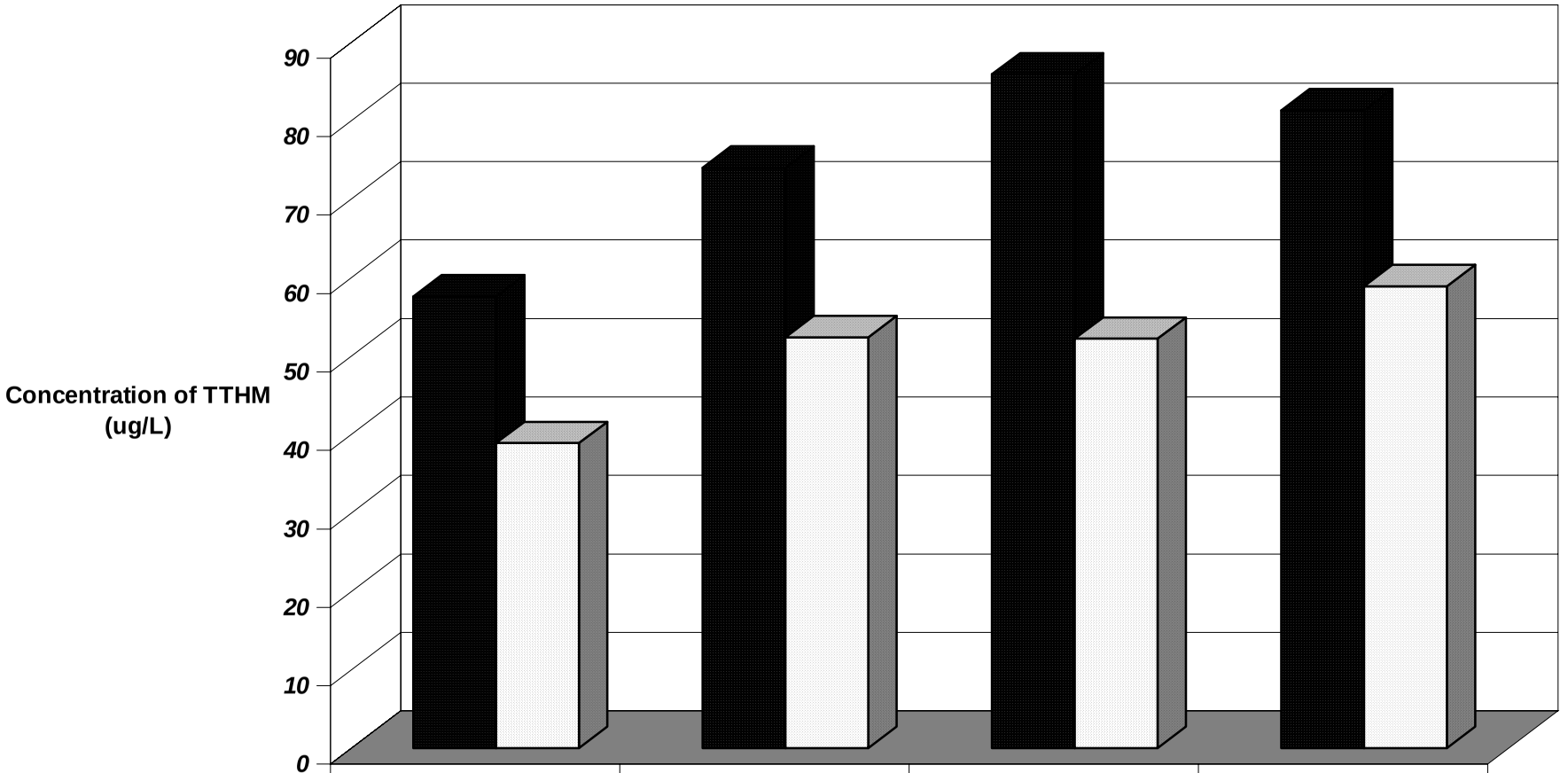
Monochloramine formation also stops the formation of both DCAA and TCAA. The concentration of DCAA in the finished water and distribution system is essentially the same as that of TCAA. The value of TCAA for Town Hall in May is questionable due to possible analytical error. This also lowered the apparent HAA6 concentration in May at Town Hall. The levels of TTHM and HAA6 were similar for both trips.

Table 4.2 Chlorine residual in plant and distribution system (Cary, NC)

	Sample Location			
	Filtered	*Finished*	*Preston Crossroads*	*Town Hall*
Free Cl_2 residual mg/L (5/29/97)	3.6	3.5	0.2	0.1
Total Cl_2 residual mg/L (5/29/97)	N/M	N/M	3.0	2.8
Free Cl_2 residual mg/L (10/14/97)	4.7	3.7	0.3	0.2
Total Cl_2 residual mg/L (10/14/97)	N/M*	N/M	3.3	3.1

* N/M = not measured
* On 5/29/97, settled water Cl_2 dose was 3.5 mg/L, filtered water Cl_2 dose was 3.05 mg/L, finished water NH_3 dose was 0.85 mg/L. Temperature was 22°C, TOC of the settled water was 3.5 mg/L.
On 10/14/97, settled water Cl_2 dose was 2.8 mg/L, filtered water Cl_2 dose was 4.7 mg/L, finished water NH_3 dose was 1.04 mg/L. Temperature was 24°C, TOC of the settled water was 2.44 mg/L.

Figure 4.5 TTHM Concentrations in plant and distribution system (Cary, NC)



	Filtered	Finished	Preston Crossroads	Town Hall
■ TTHM ug/L (5/29/97)	57.6	74	85.9	81.3
□ TTHM ug/L (10/14/97)	38.9	52.4	52.2	58.9

Figure 4.6 HAA6 Concentrations in plant and distribution system (Cary, NC)

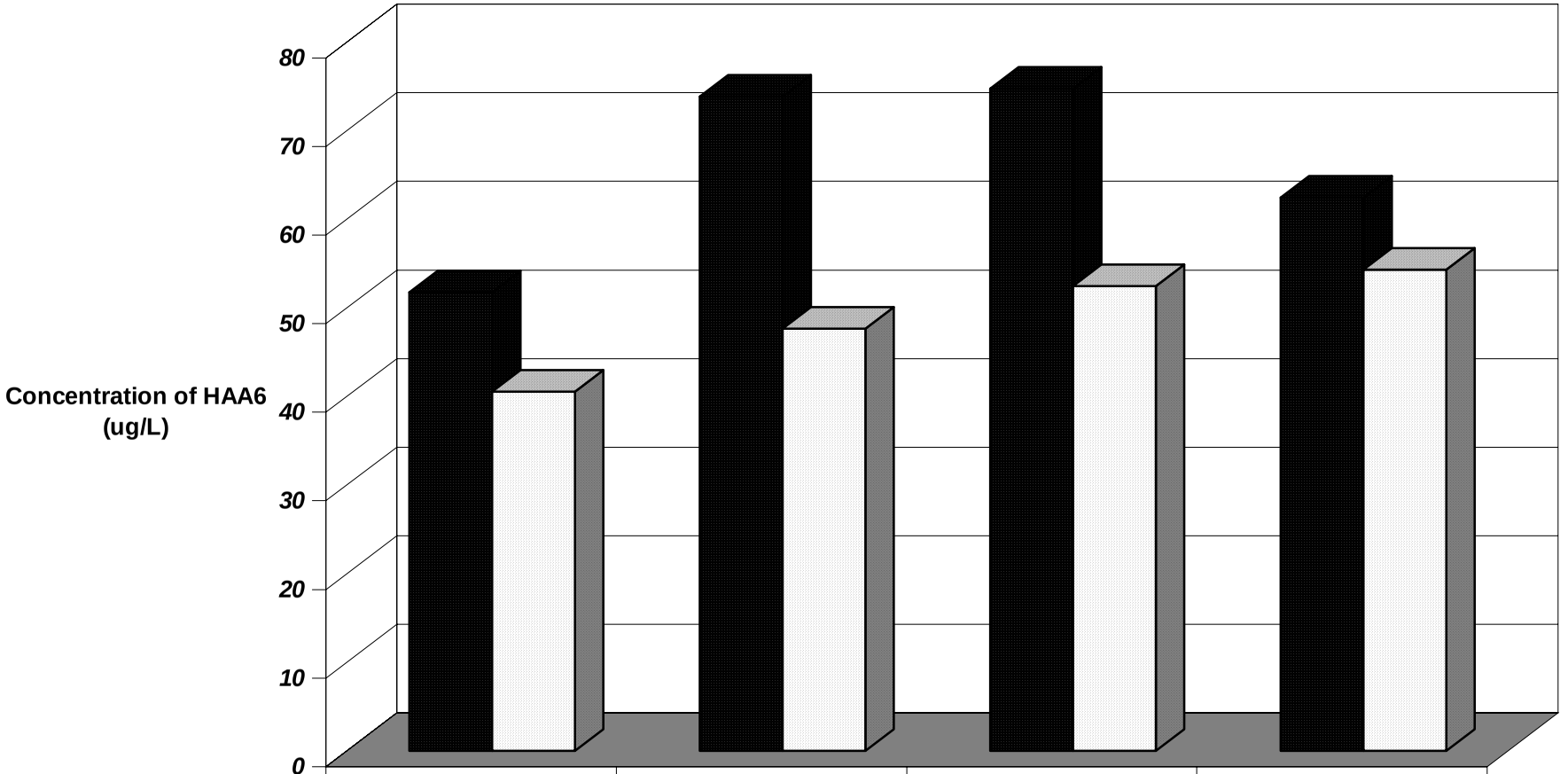


Figure 4.7 DCAA Concentrations in plant and distribution system (Cary, NC)

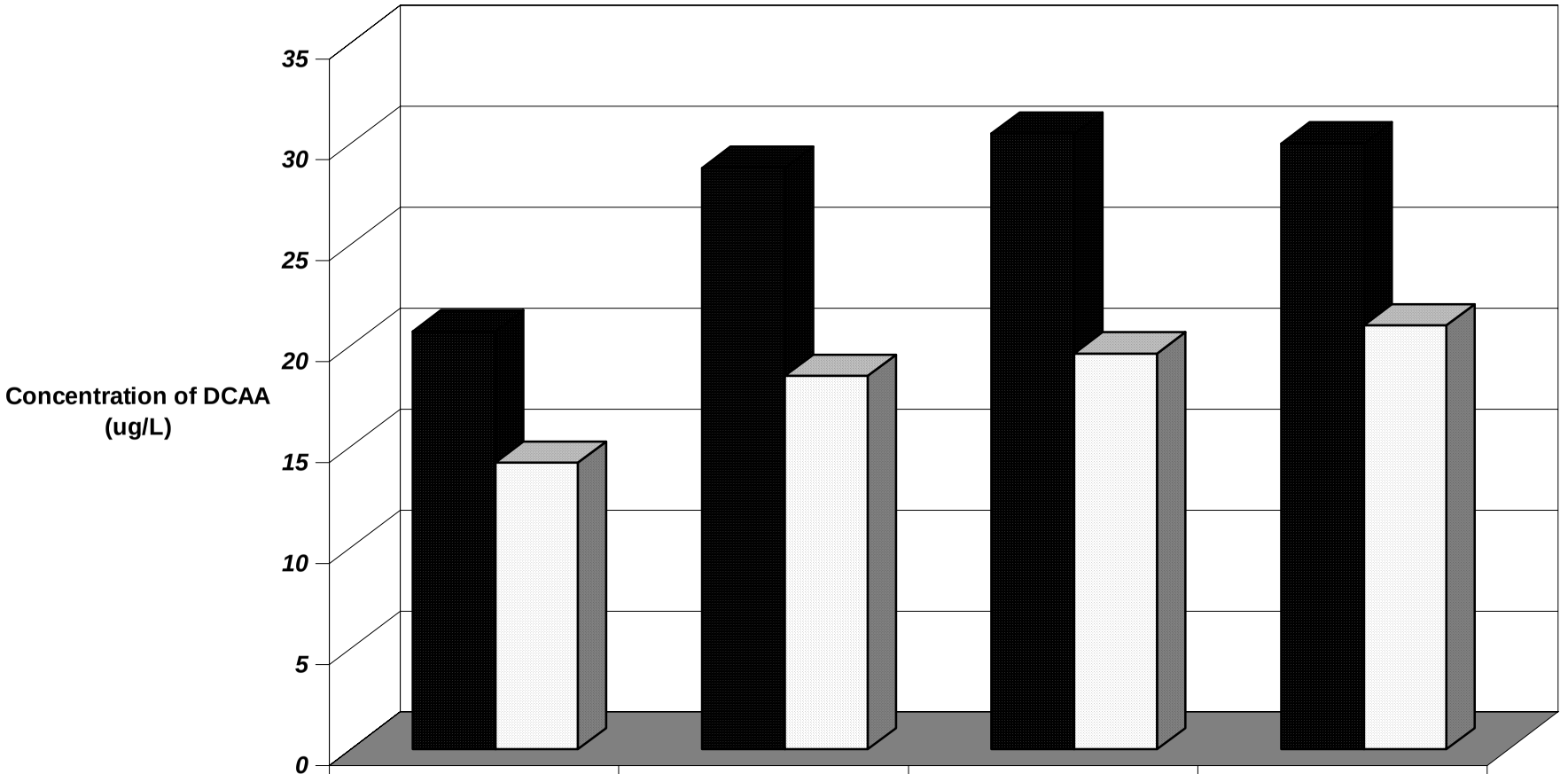
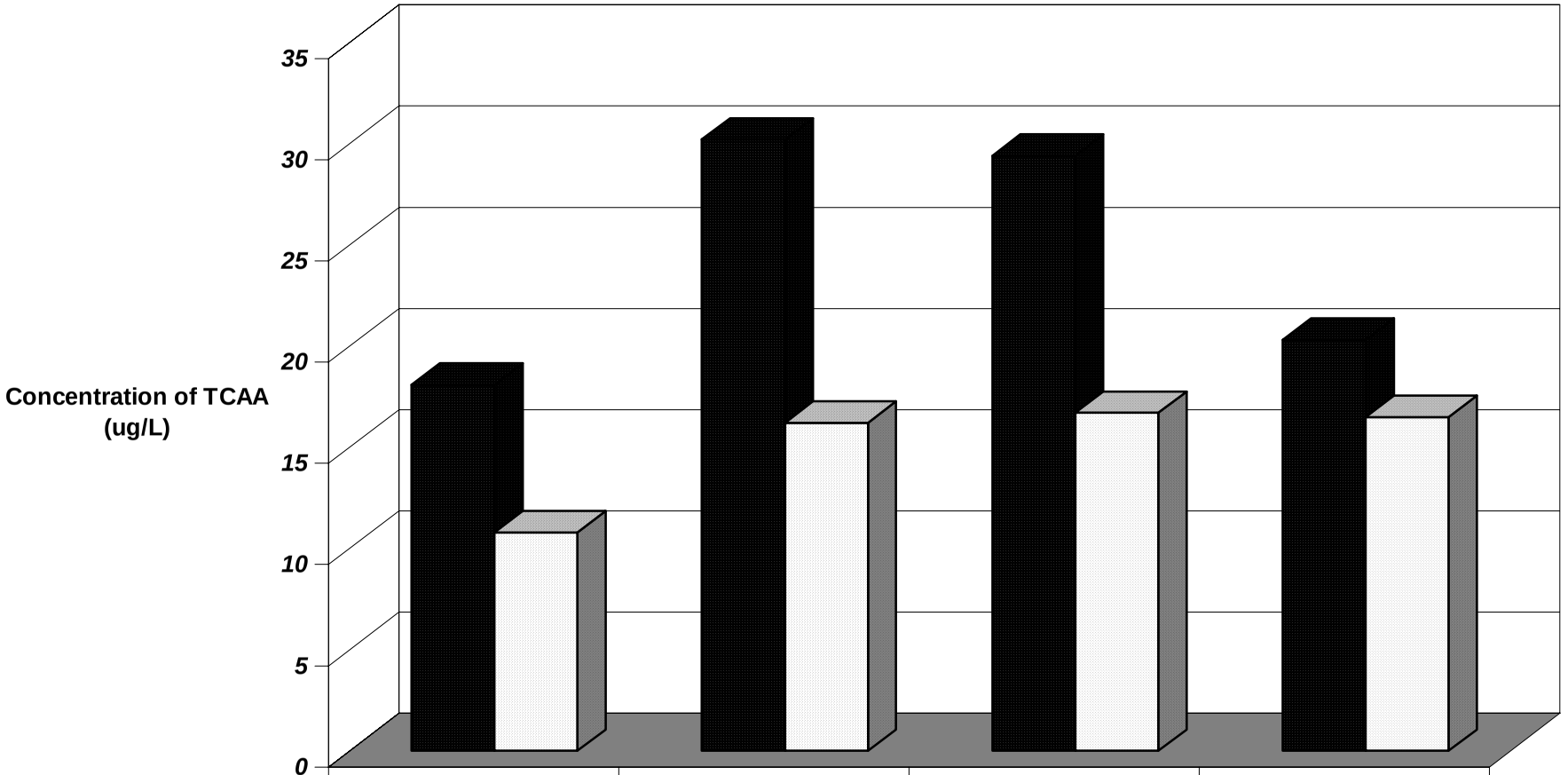


Figure 4.8 TCAA Concentrations in plant and distribution system (Cary, NC)



Like Cary, High Point applies free chlorine as a primary disinfectant before and after filtration, but in High Point, ammonia is added to the filtered water after chlorination and before storage and subsequent pumping into the distribution system (see Figure 3.1). Table 4.3 gives chlorine residuals for each sampling location in High Point, along with the chlorine and ammonia dosages. Measurements in the distribution system could only be quantified up to 3.5 mg/L combined chlorine. Any value larger than this is reported as >3.5 mg/L.

Table 4.3 Chlorine residual in plant and distribution system (High Point, NC)

	Sample Location			
	Filtered	*Finished*	*Eastchester*	*Westchester*
Free Cl_2 residual mg/L (6/13/97)	2.2	N/M	0.5	0.9
Total Cl_2 residual mg/L (6/13/97)	N/M	4.6	>3.5	>3.5
Free Cl_2 residual mg/L (10/29/97)	1.6	1.2	0.5	0.2
Total Cl_2 residual mg/L (10/29/97)	N/M	5.0	>3.5	>3.5

* N/M = not measured
* On 6/13/97, settled water Cl_2 dose was 1.6 mg/L, filtered water Cl_2 dose was 5.2 mg/L, filtered water NH_3 dose was 1.7 mg/L. Temperature was 21.2°C, TOC of the settled water was 2.52 mg/L. On 10/29/97, settled water Cl_2 dose was 1.8 mg/L, filtered water Cl_2 dose was 5.5 mg/L, filtered water NH_3 dose was 1.6 mg/L. Temperature was 17°C, TOC of the settled water was 1.9 mg/L.

Figures 4.9-4.12 demonstrate that, in High Point too, the addition of ammonia basically stops THM and HAA formation. During June, when the water was warm and kinetics of chloramine formation were rapid, ammonia addition stopped THM and HAA formation immediately after filtration. However, presumably due to slower reaction kinetics in October or due to the different nature of the DBP precursors, some THM and HAA formation continued through the clearwell, but essentially stopped thereafter. DCAA and TCAA display the same pattern. For High Point, HAA6 concentrations were much lower than TTHM concentrations compared to Cary and Greenville, where HAA6 and TTHM concentrations were similar. DCAA formed to a greater degree than TCAA; however, the formation of both stopped with ammonia addition.

These findings with respect to DBP formation in the distribution systems for the two chloraminated waters are very different from those in Greenville and the other three locations where free chlorine is the secondary disinfectant. In the distribution system, THM and HAA formation essentially stops in the chloraminated waters, while in the chlorinated waters, THM and HAA formation continue.

Figure 4.9 TTHM Concentrations in plant and distribution system (High Point, NC)

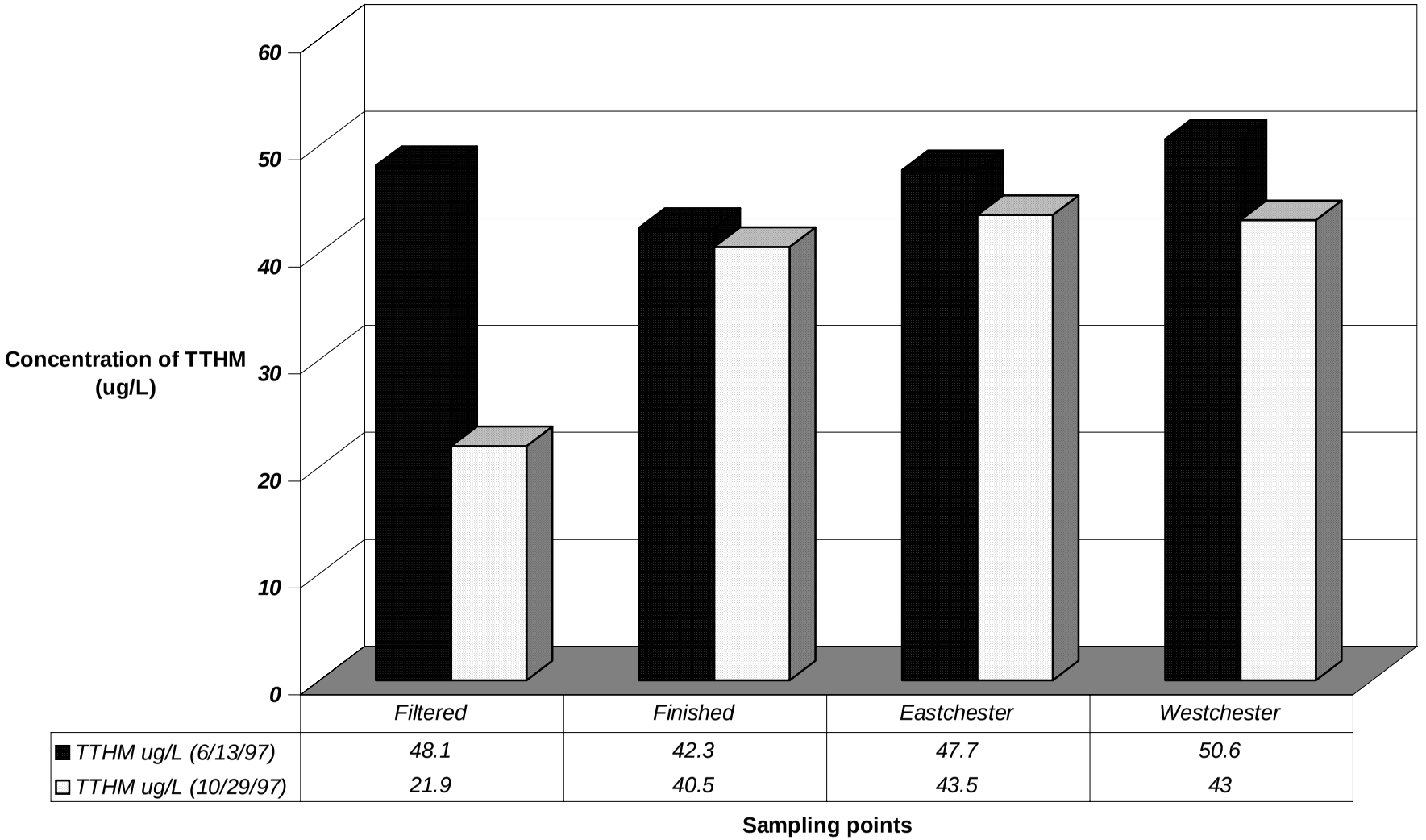
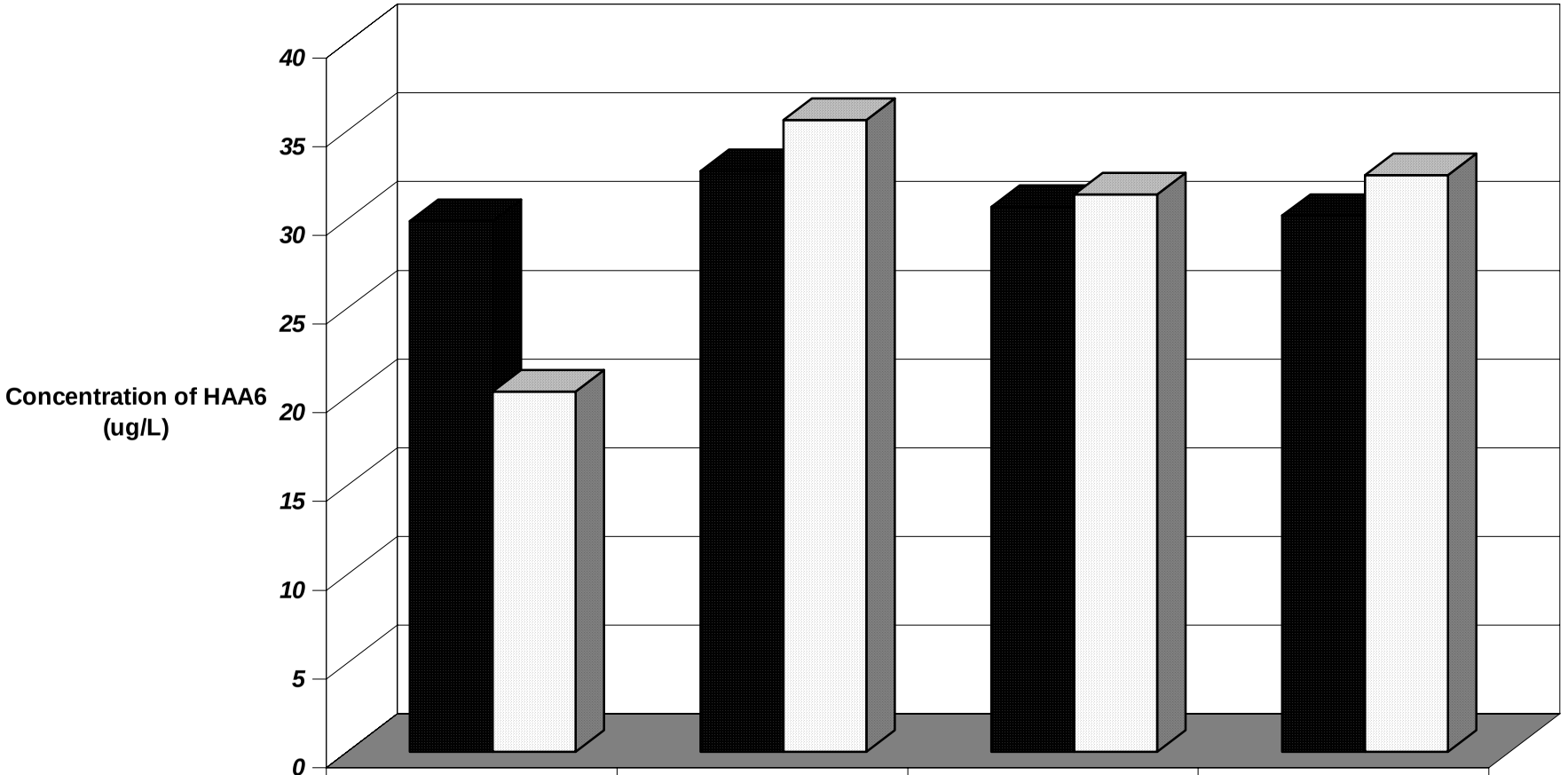


Figure 4.10 HAA6 Concentrations in plant and distribution system (High Point, NC)



	Filtered	Finished	Eastchester	Westchester
■ HAA6 ug/L (6/13/97)	29.9	32.7	30.7	30.2
□ HAA6 ug/L (10/29/97)	20.3	35.6	31.4	32.5

Figure 4.11 DCAA Concentrations in plant and distribution system (High Point, NC)

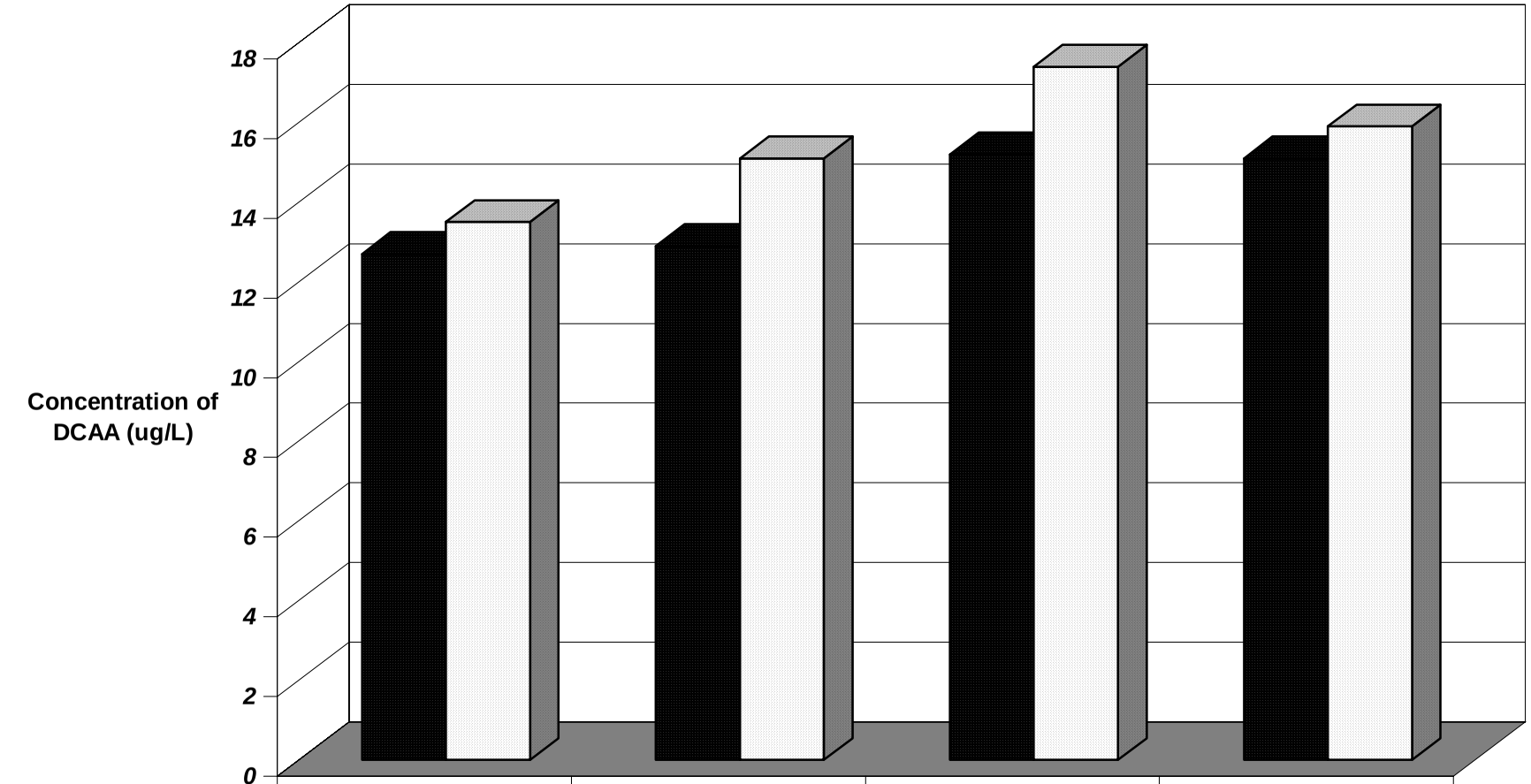
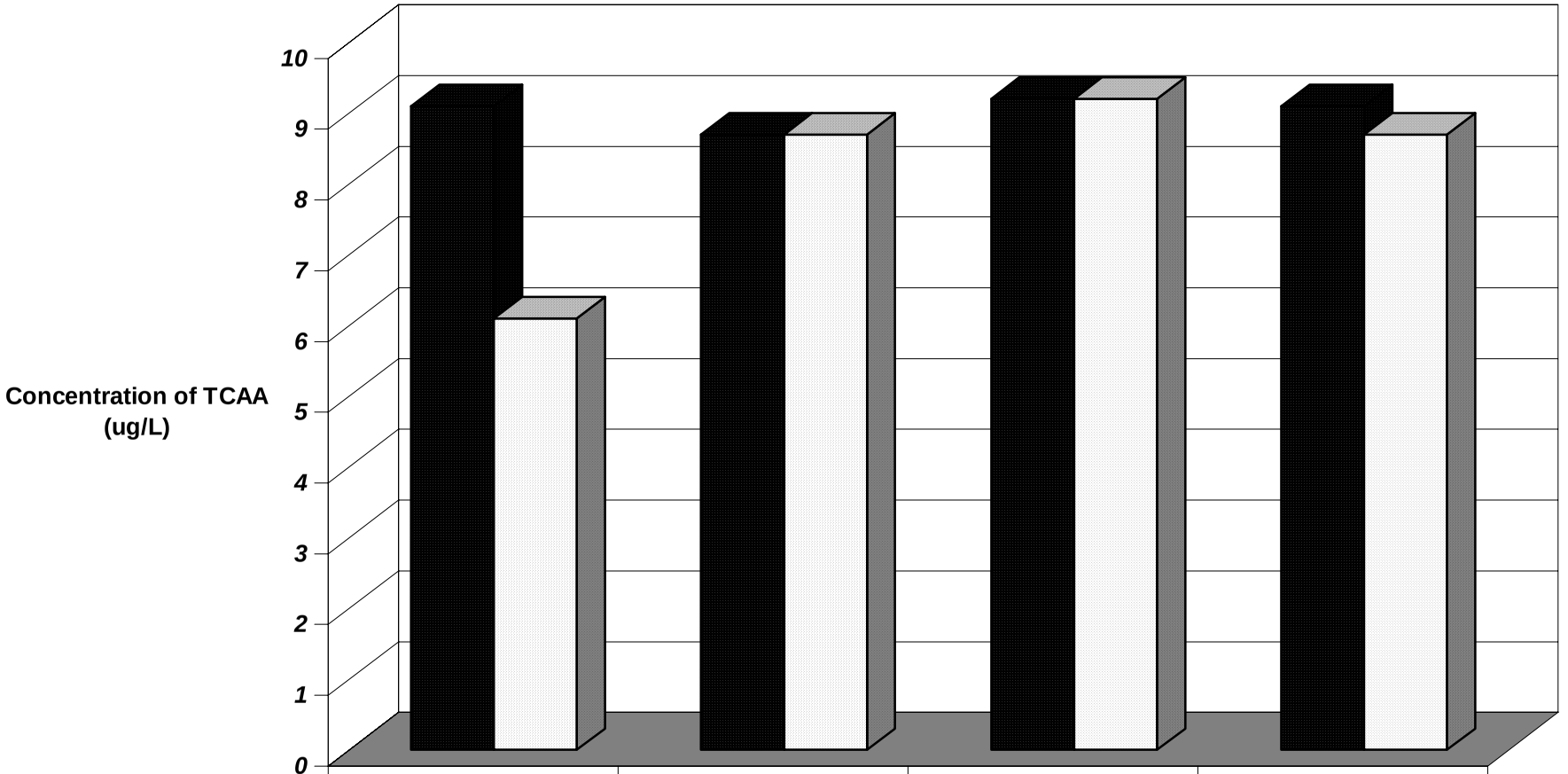


Figure 4.12 TCAA Concentrations in plant and distribution system (High Point, NC)



4.2 CONTROLLED CHLORAMINATION EXPERIMENTS

In this section, findings from the controlled chloramination experiments on filtered and finished water at a number of the facilities using only free chlorine are discussed. In these tests, filtered and finished chlorinated water was collected at the sampling taps in the treatment plant laboratories, adjusted to pH 8.0, and treated with ammonia at an N:Cl_2 ratio of 1:4 by weight based on the measured free chlorine residual for that sample. TTHM and HAA6 concentrations were measured prior to the addition of ammonia, and 24 and 72 hours after ammonia addition.

The values for the chlorine residuals and ammonia dosages for chloramination of the filtered water are summarized in Table 4.4. Figures 4.13-4.20 show the corresponding controlled chloramination results for filtered water. In most cases, the controlled laboratory results verify the observations from the plants using chloramination, i.e. that ammonia addition essentially stops THM and HAA formation. Ammonia addition to filtered water was not performed at Fayetteville because the chlorine residual of the filtered water was below 0.5 mg/L.

Table 4.4 Summary of chlorine residuals and ammonia dosages for filtered water in chloramination experiments

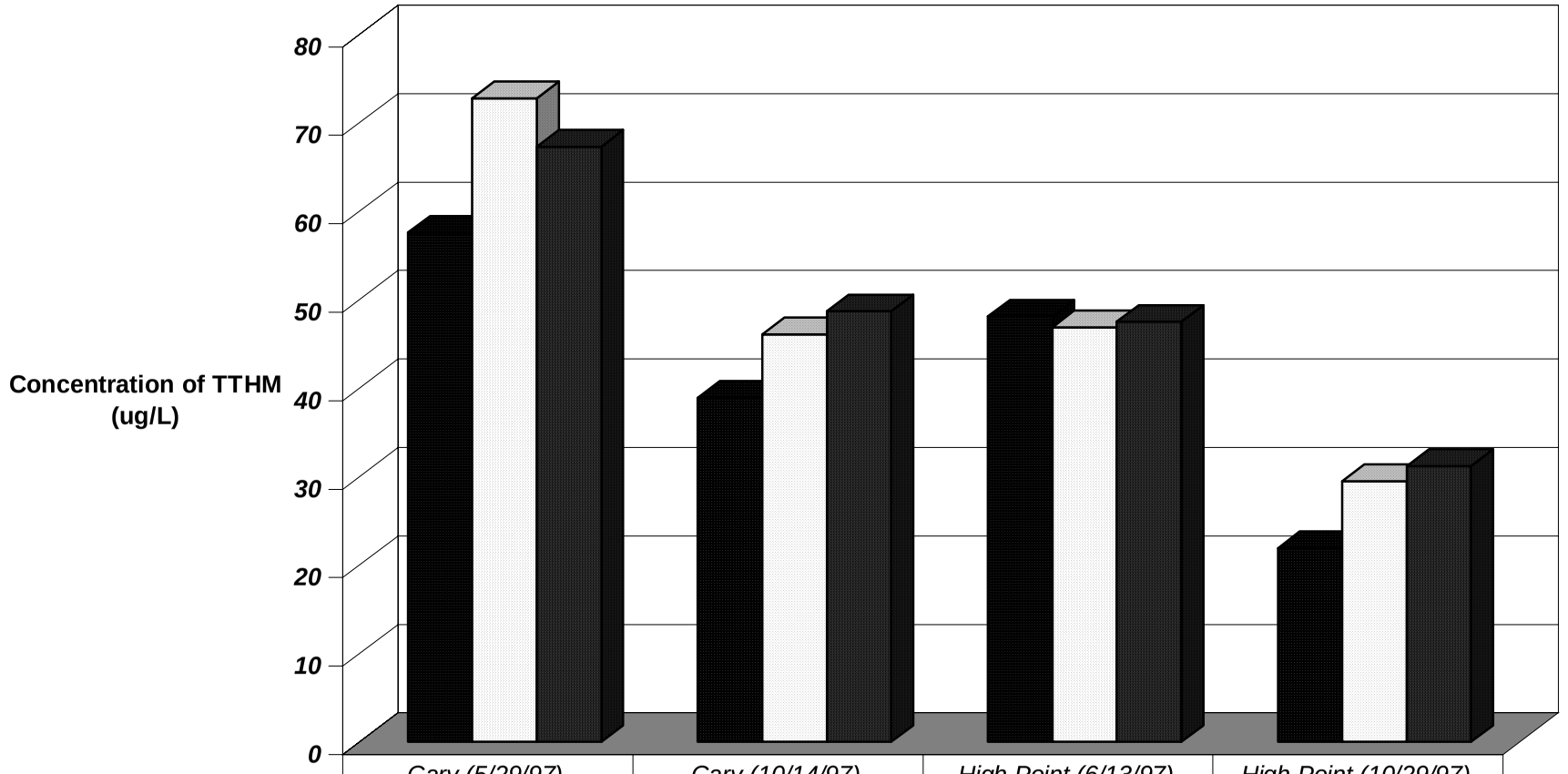
	Cl_2 *residual (mg/L)*	*Ammonia dosage (mg/L as N)*
Cary (5/29/97)	3.6	0.9
Cary (10/14/97)	4.7	1.2
High Point (6/13/97)	2.2	0.55
High Point (10/29/97)	1.6	0.4
OWASA (10/3/97)	1.96	0.5
Durham (9/4/97)	2.4	0.6
Durham (11/10/97)	2.6	0.65
Greenville (8/6/97)	0.9	0.2
Greenville (12/1/97)	0.7	0.2

* Ammonia added as NH_4Cl, after water was buffered to pH 8.0

For TTHMs, sometimes the addition of ammonia stops the DBP formation immediately (see High Point, June), while in other cases, there is still some continuing reaction even up to 72 hours after ammonia addition (e.g. Cary, October; High Point, October; Durham, September and November). These findings suggest that there is still some free Cl_2 due to the slow conversion of free Cl_2 to NH_2Cl, or that the presence of reactive NOM continues to produce DBPs even with NH_2Cl, or that the conversion of reactive intermediates produced by chlorine will slowly hydrolyze and revert to THMs. This phenomenon is less noticeable for HAA6, where the addition of NH_3 has a much more immediate effect in stopping subsequent HAA formation. In most cases, the formation of DCAA and TCAA ceases immediately after ammonia addition. This was more evident for TCAA than for DCAA.

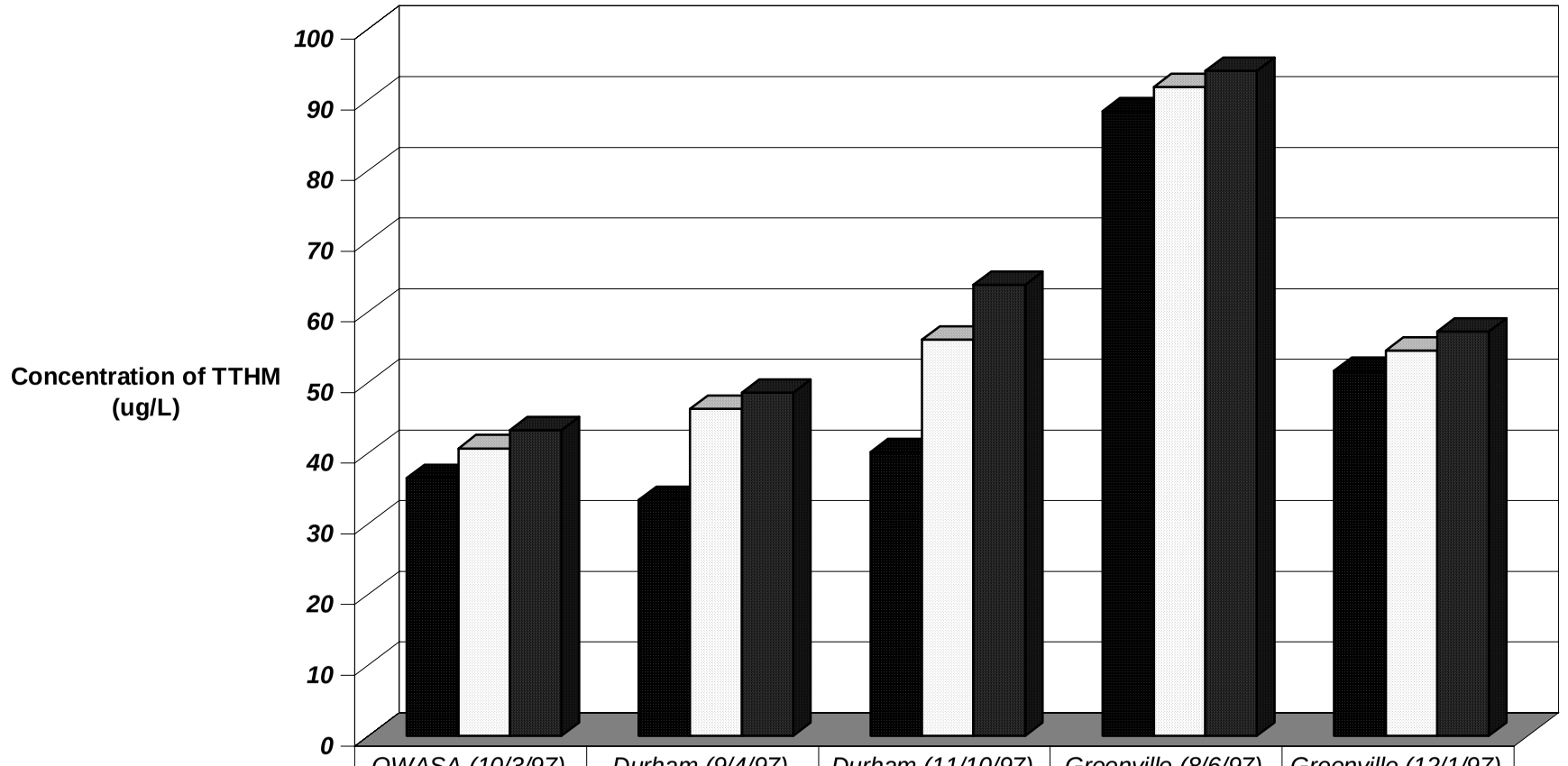
Table 4.5 summarizes the values for chlorine residuals and ammonia dosages for chloramination experiments on the finished waters. The corresponding DBP results for the controlled chloramination experiments for finished water are illustrated in Figures 4.21-4.28. High Point is not included here because the finished water in the plant was already chloraminated. These findings again confirm that ammonia addition generally stops THM and HAA formation.

Figure 4.13 TTHM concentrations with addition of ammonia to filtered water- I



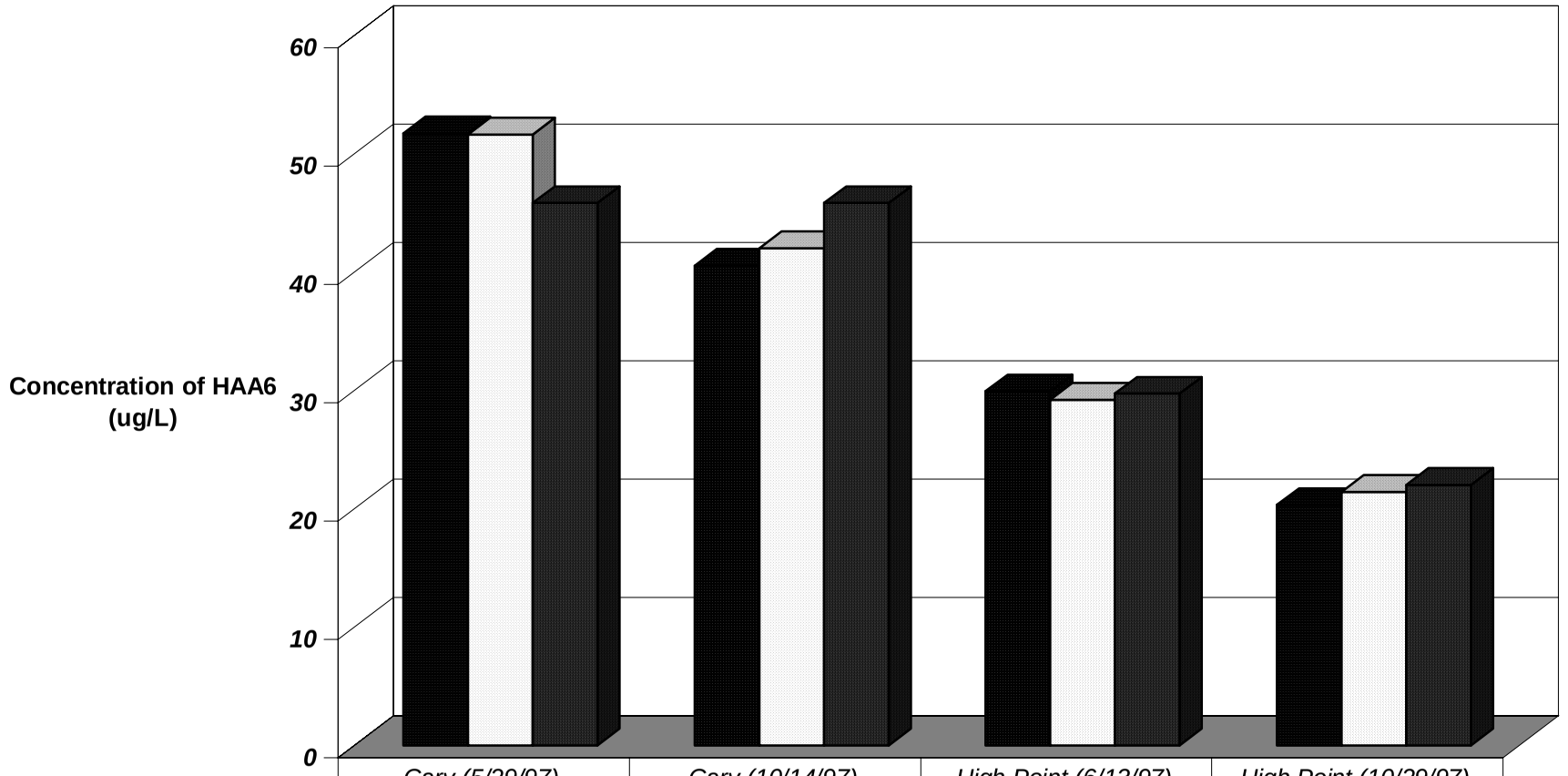
	Cary (5/29/97)	Cary (10/14/97)	High Point (6/13/97)	High Point (10/29/97)
TTHM ug/L @ t<0, before NH3	57.6	38.9	48.1	21.9
TTHM ug/L @ t=24hr after NH3	72.8	46.1	46.9	29.5
TTHM ug/L @ t=72hr after NH3	67.3	48.7	47.5	31.2

Figure 4.14 TTHM concentrations with addition of ammonia to filtered water-II



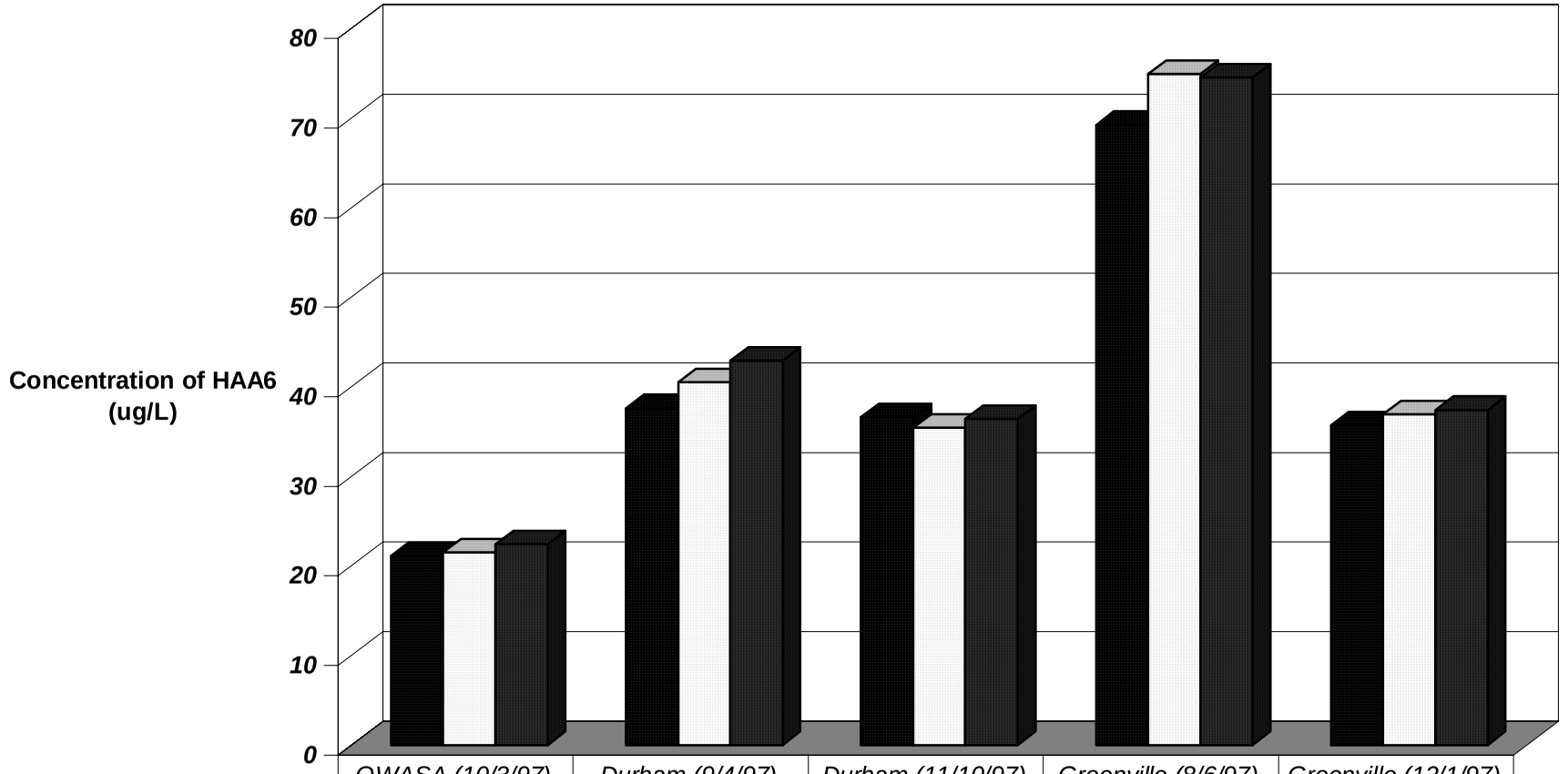
	OWASA (10/3/97)	Durham (9/4/97)	Durham (11/10/97)	Greenville (8/6/97)	Greenville (12/1/97)
TTHM ug/L @ t<0, before NH3	36.5	33.4	40.2	88.4	51.7
TTHM ug/L @ t=24hr after NH3	40.7	46.3	56.1	91.8	54.5
TTHM ug/L @ t=72hr after NH3	43.3	48.6	63.8	94.1	57.2

Figure 4.15 HAA6 concentrations with addition of ammonia to filtered water-I



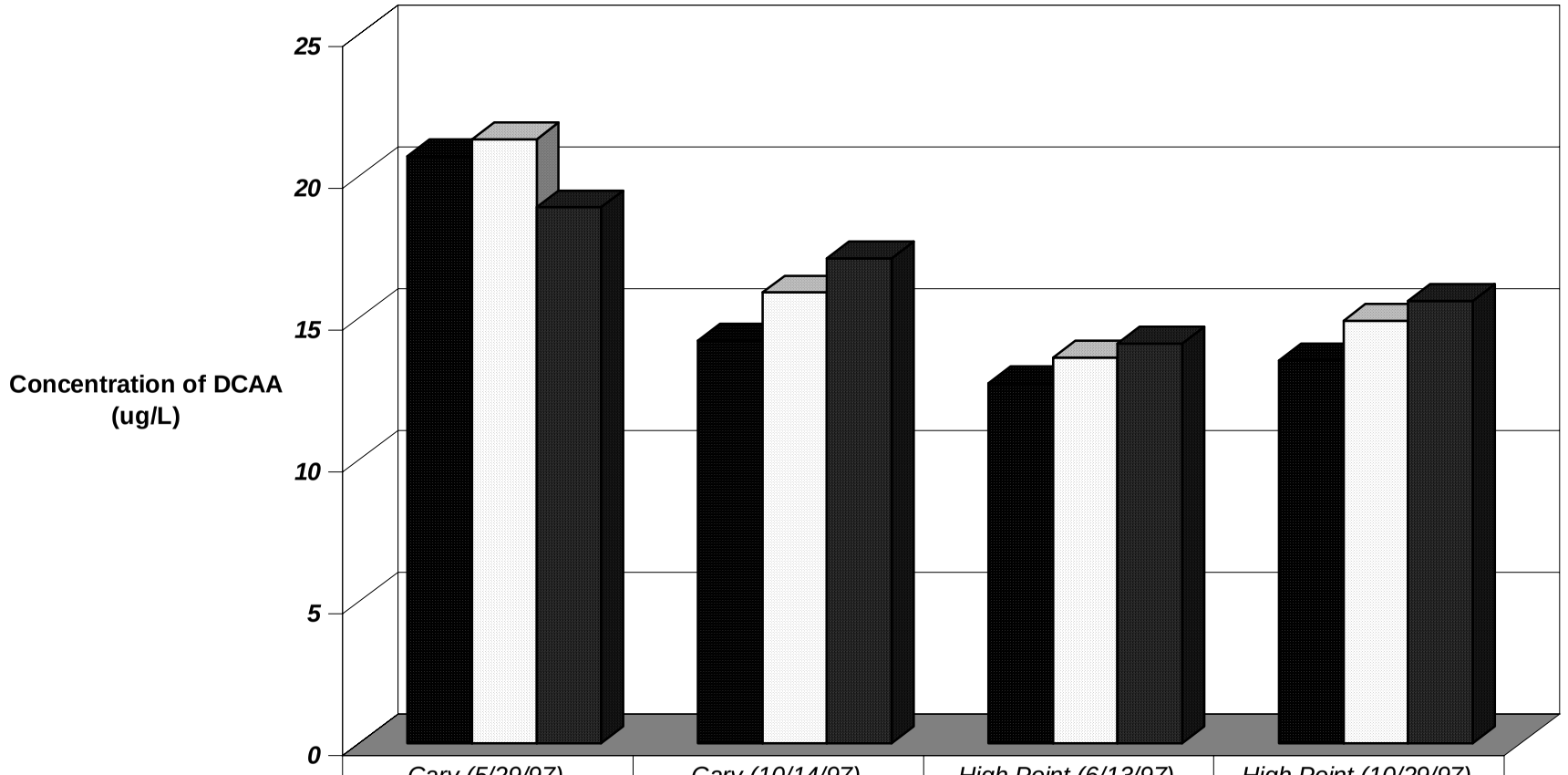
	Cary (5/29/97)	Cary (10/14/97)	High Point (6/13/97)	High Point (10/29/97)
HAA6 ug/L @ t<0, before NH3	51.7	40.5	29.9	20.3
HAA6 ug/L @ t=24hr after NH3	51.6	42	29.2	21.4
HAA6 ug/L @ t=72hr after NH3	45.8	45.8	29.7	22

Figure 4.16 HAA6 concentrations with addition of ammonia to filtered water-II



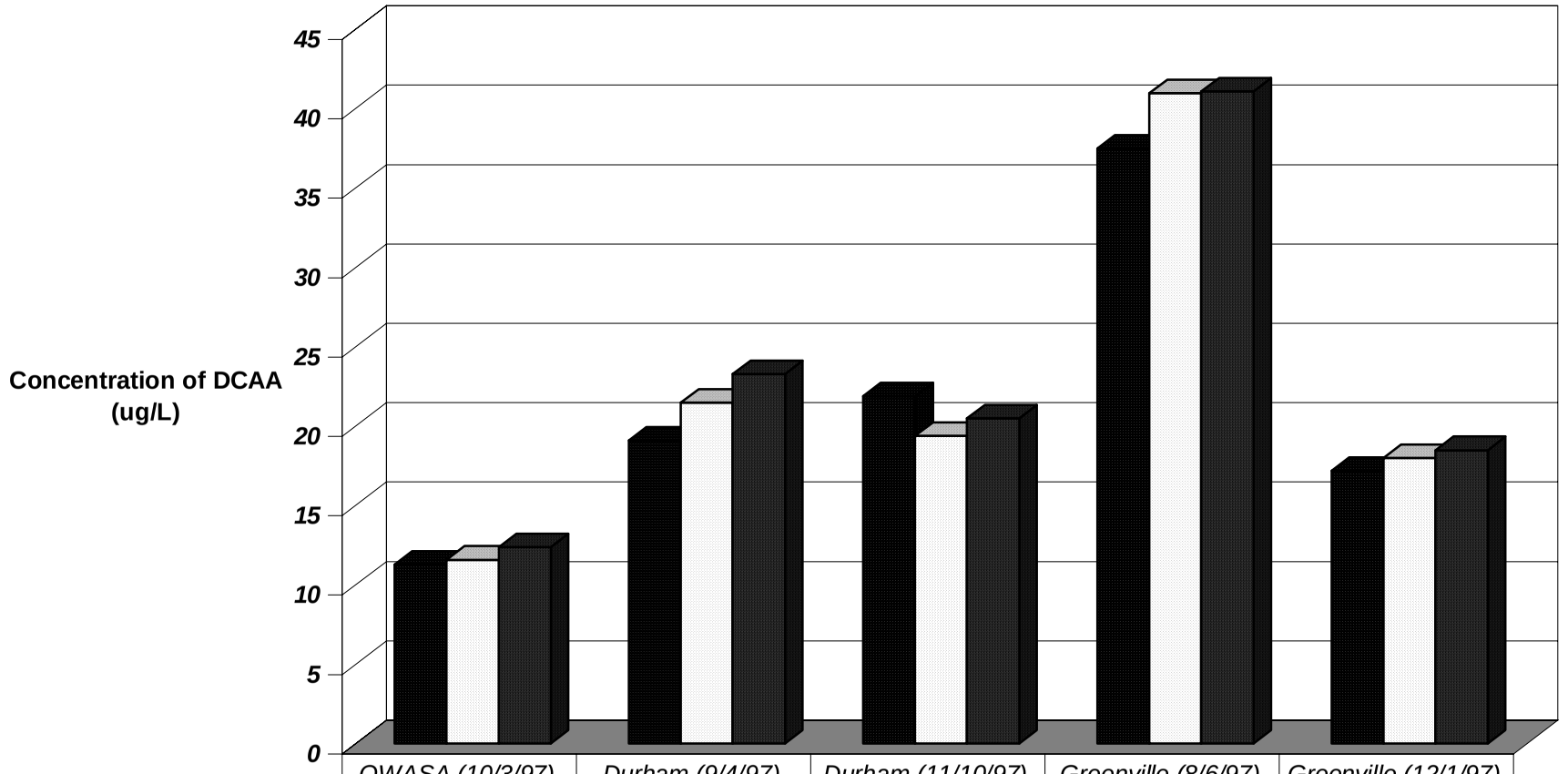
	OWASA (10/3/97)	Durham (9/4/97)	Durham (11/10/97)	Greenville (8/6/97)	Greenville (12/1/97)
HAA6 ug/L @ t<0, before NH3	21.1	37.6	36.6	69.2	35.7
HAA6 ug/L @ t=24hr after NH3	21.5	40.5	35.4	74.9	36.9
HAA6 ug/L @ t=72hr after NH3	22.4	42.9	36.4	74.5	37.4

Figure 4.17 DCAA concentrations with addition of ammonia to filtered water-I



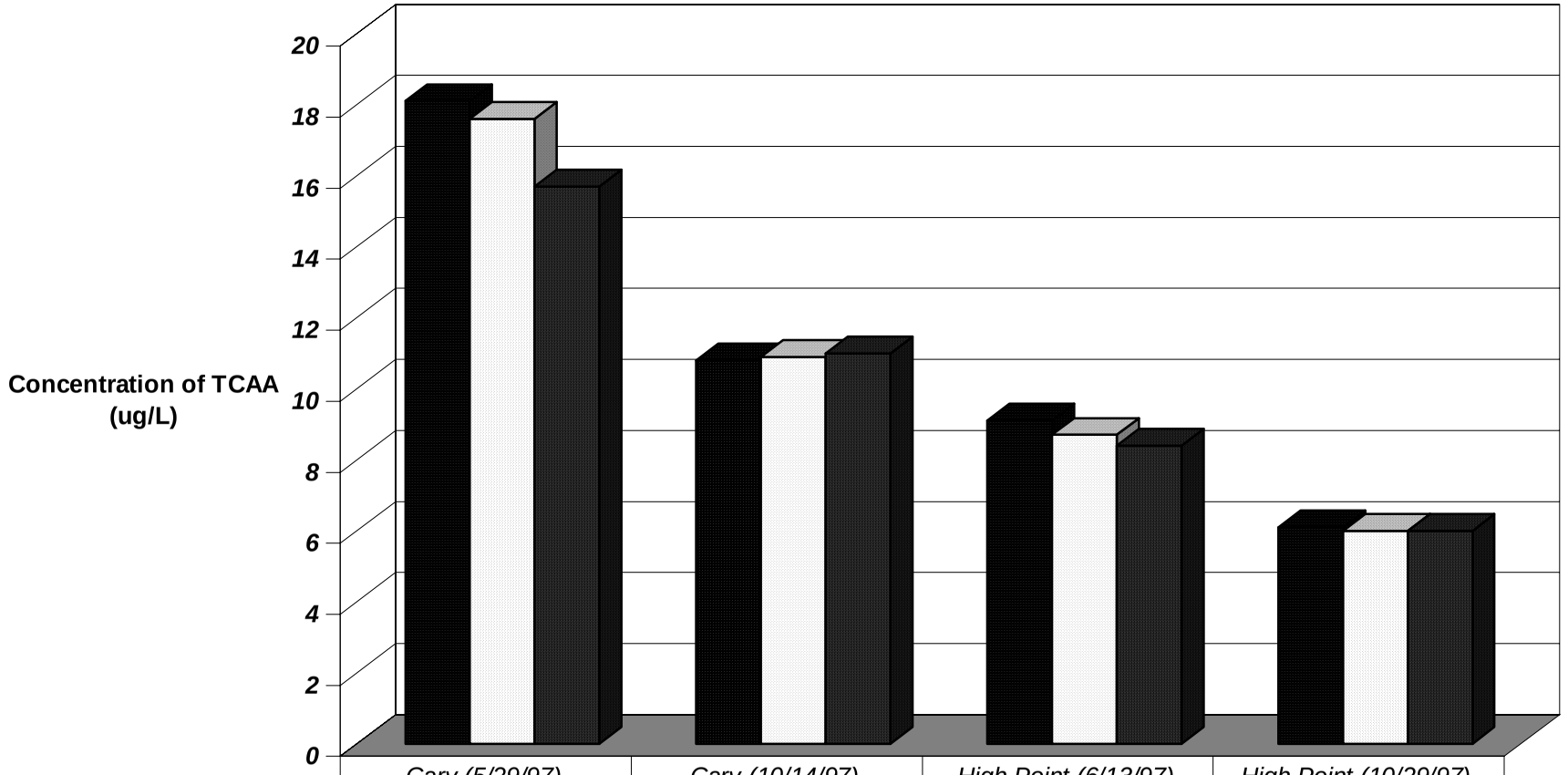
	Cary (5/29/97)	Cary (10/14/97)	High Point (6/13/97)	High Point (10/29/97)
■ DCAA ug/L @ t<0, before NH3	20.7	14.2	12.7	13.5
□ DCAA ug/L @ t=24hr after NH3	21.3	15.9	13.6	14.9
■ DCAA ug/L @ t=72hr after NH3	18.9	17.1	14.1	15.6

Figure 4.18 DCAA concentrations with addition of ammonia to filtered water-II



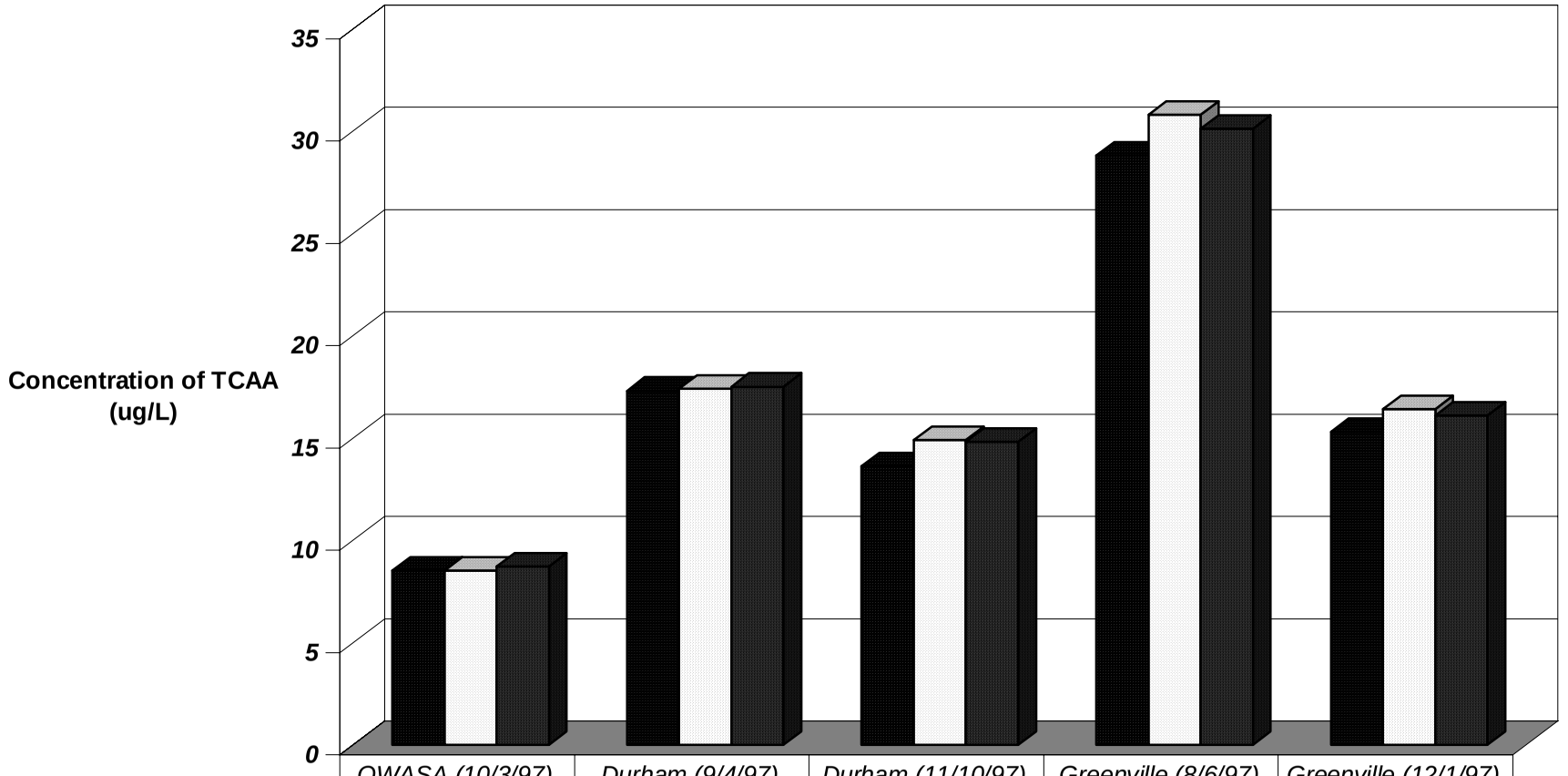
	OWASA (10/3/97)	Durham (9/4/97)	Durham (11/10/97)	Greenville (8/6/97)	Greenville (12/1/97)
DCAA ug/L @ t<0, before NH3	11.3	19.1	21.9	37.5	17.2
DCAA ug/L @ t=24hr after NH3	11.6	21.5	19.4	41	18
DCAA ug/L @ t=72hr after NH3	12.4	23.3	20.5	41.1	18.5

Figure 4.19 TCAA concentrations with addition of ammonia to filtered water-I



	Cary (5/29/97)	Cary (10/14/97)	High Point (6/13/97)	High Point (10/29/97)
TCAA ug/L @ t<0, before NH3	18.1	10.8	9.1	6.1
TCAA ug/L @ t=24hr after NH3	17.6	10.9	8.7	6
TCAA ug/L @ t=72hr after NH3	15.7	11	8.4	6

Figure 4.20 TCAA concentrations with addition of ammonia to filtered water-II



	OWASA (10/3/97)	Durham (9/4/97)	Durham (11/10/97)	Greenville (8/6/97)	Greenville (12/1/97)
TCAA ug/L @ t<0, before NH3	8.5	17.3	13.6	28.8	15.3
TCAA ug/L @ t=24hr after NH3	8.5	17.4	14.9	30.8	16.4
TCAA ug/L @ t=72hr after NH3	8.7	17.5	14.8	30.1	16.1

For the finished waters, the impact of ammonia addition appears to be more immediate than for ammonia addition to the filtered waters. In most cases, TTHM concentrations 24 hours after ammonia addition are the same as they were before ammonia addition (see OWASA; Greenville; Fayetteville, December; Cary; Durham, September). The results suggest that the more reactive NOM precursors had already reacted by the time ammonia was added. Again, DCAA and TCAA formation was also observed to cease immediately after the addition of ammonia in most cases; the effect of ammonia addition was more pronounced for TCAA than for DCAA.

Table 4.5 Summary of chlorine residuals and ammonia dosages for finished water in chloramination experiments

	Cl_2 residual (mg/L)	*Ammonia dosage (mg/L as N)*
Cary (5/29/97)	3.5	0.9
Cary (10/14/97)	3.7	0.9
Durham (9/4/97)	1.8	0.45
Durham (11/10/97)	2.2	0.55
OWASA (10/3/97)	1.8	0.45
Greenville (8/6/97)	2.19	0.55
Greenville (12/1/97)	1.75	0.45
Fayetteville (9/18/97)	1.8	0.45
Fayetteville (12/10/97)	1.23	0.3

* Ammonia added as NH_4Cl, after water was buffered to pH 8.0

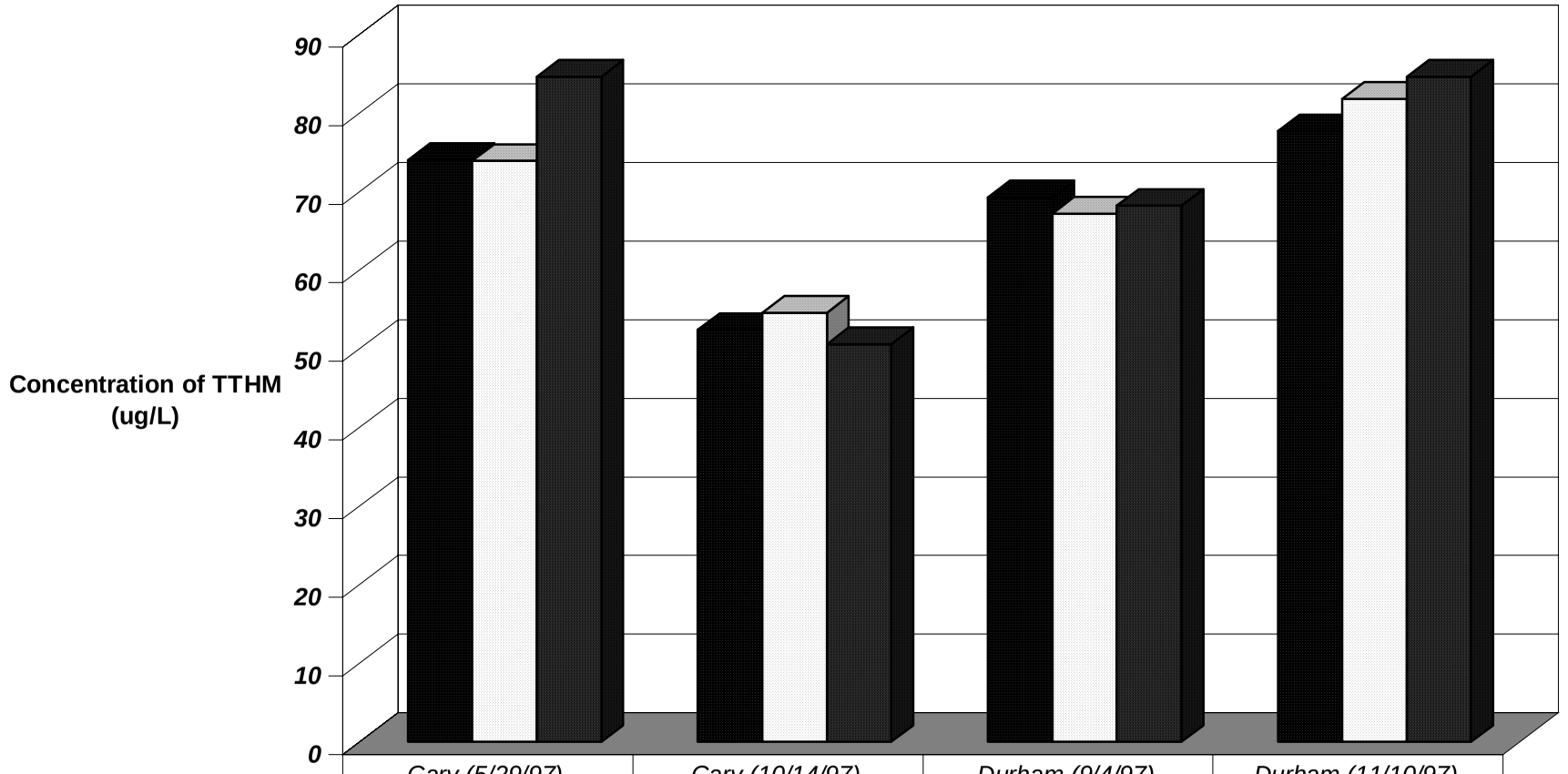
4.3 DISCUSSION OF CHLORAMINATION FINDINGS

Many earlier works have shown that the addition of ammonia dramatically reduces THM production (e.g. Stevens et al. 1976; Brodtmann and Russo 1979; Norman et al. 1980; Mitcham et al. 1983). Similar results are also evident in this research. However, some researchers have found that pre-formed NH_2Cl will still produce some THMs and HAAs and have proposed several mechanisms. For example, it was suggested that monochloramine still served as a Cl^+ donor even though the concentration of chloroform formed using monochloramine as the chlorination agent was two orders of magnitude less than that using free available chlorine as the chlorination agent (Topudurti and Haas 1985).

Johnson and Jensen (1986) indicated that the major mechanisms in the reaction between disinfectants and naturally occurring organic materials in water are oxidation and substitution. Chlorine can oxidize organics by accepting electrons from the organic substrate or can substitute into the organic matrix. Even though monochloramine is a weaker substituting agent than free chlorine, it still can form significant concentrations of substitution products, e.g. halogenated DBPs (TOX), although THM and HAA formation tends to be relatively small.

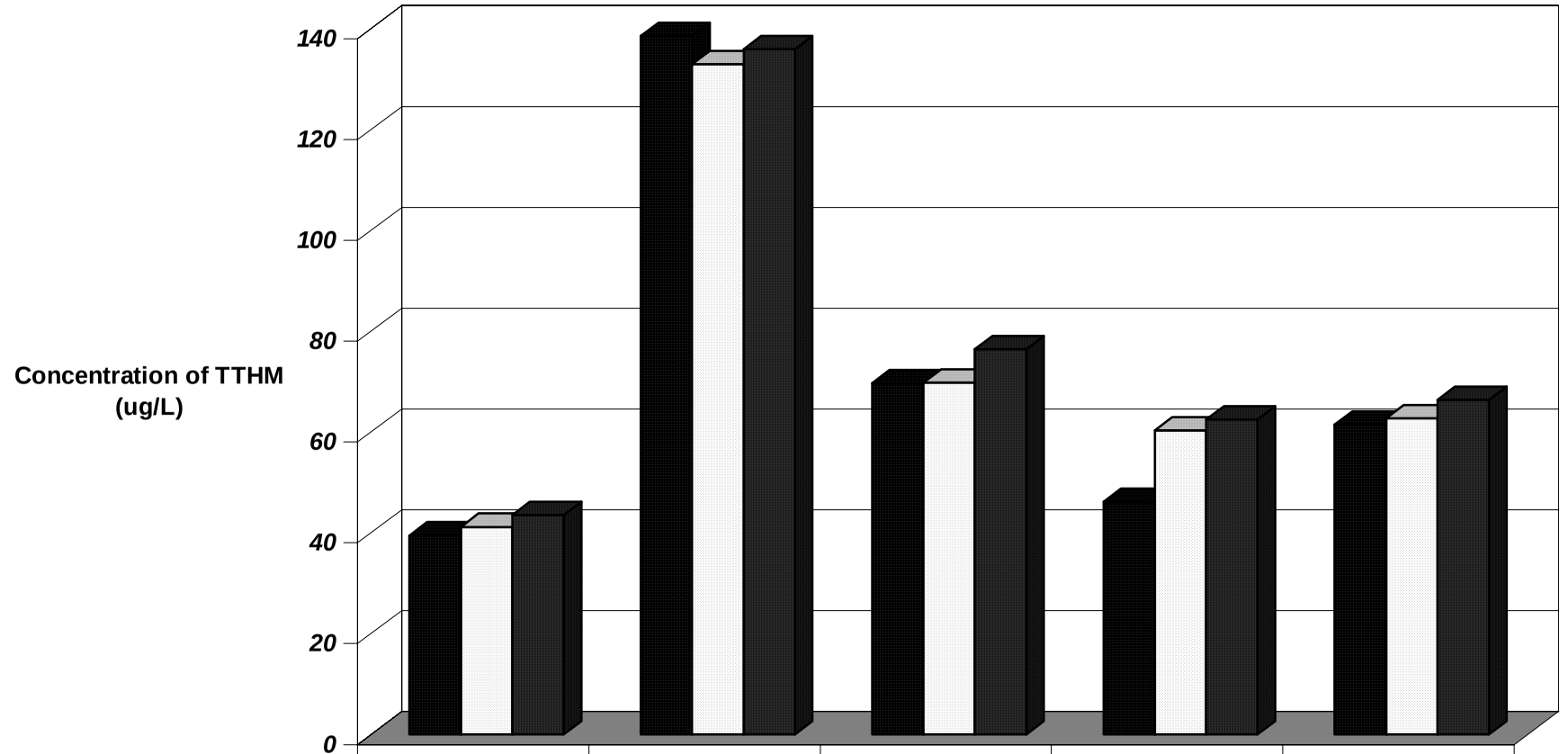
These reports suggest that, although using monochloramine as the chlorination agent will result in much less by-product formation than from using free chlorine as the chlorination agent, monochloramine can still add to organics by substitution reactions. This is consistent with the results observed in this study that show a small but continuing increase in TTHM concentrations after addition of ammonia. TTHMs increased from 0 to 23.6 μg/L (Durham, November) 72 hours after the addition of ammonia. The average

Figure 4.21 TTHM concentrations with addition of ammonia to finished water-I



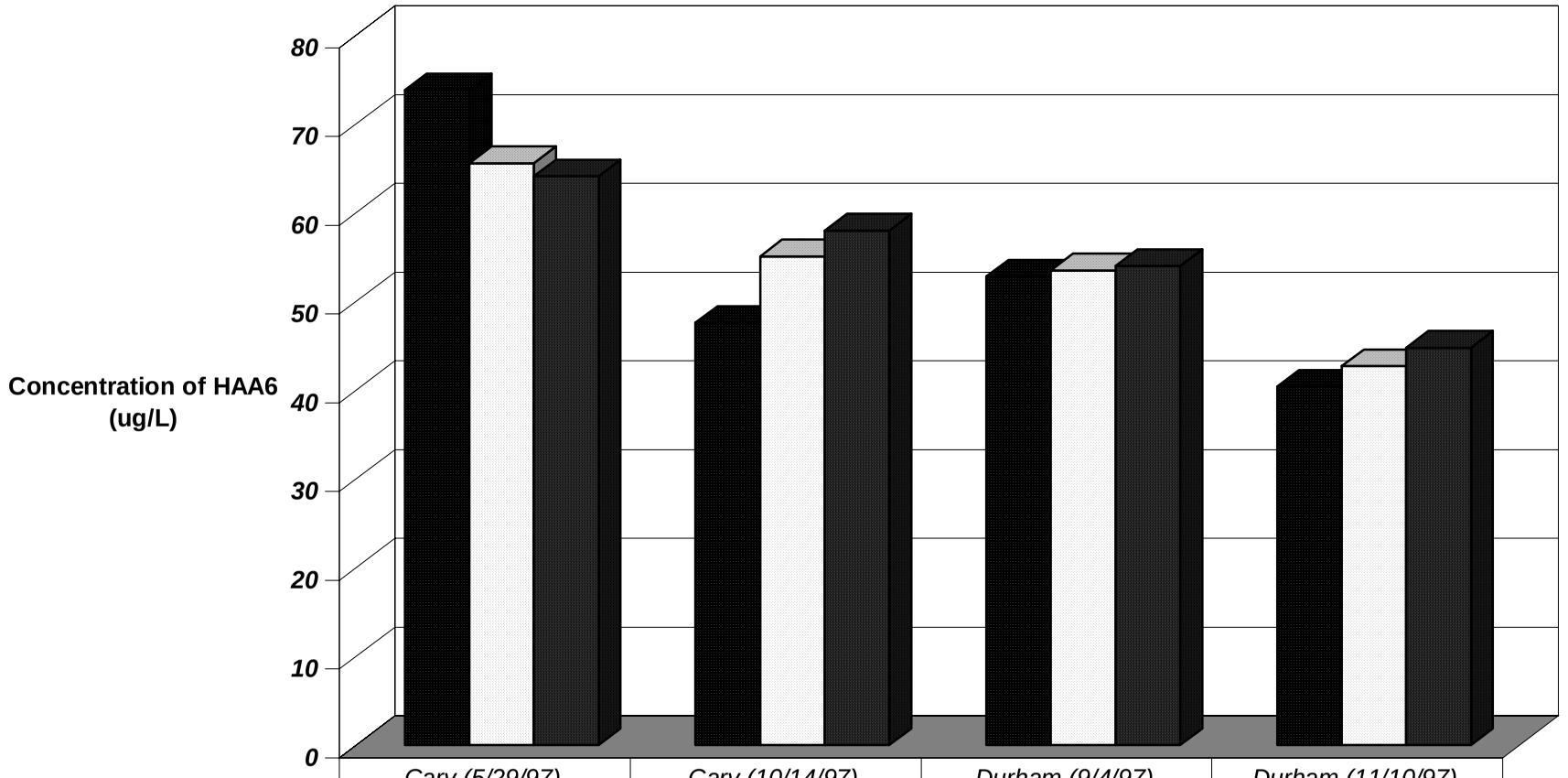
	Cary (5/29/97)	Cary (10/14/97)	Durham (9/4/97)	Durham (11/10/97)
TTHM ug/L @ t<0, before NH3	74	52.4	69.3	77.7
TTHM ug/L @ t=24hr after NH3	73.9	54.6	67.2	81.8
TTHM ug/L @ t=72hr after NH3	84.6	50.6	68.2	84.6

Figure 4.22 TTHM concentrations with addition of ammonia to finished water-II



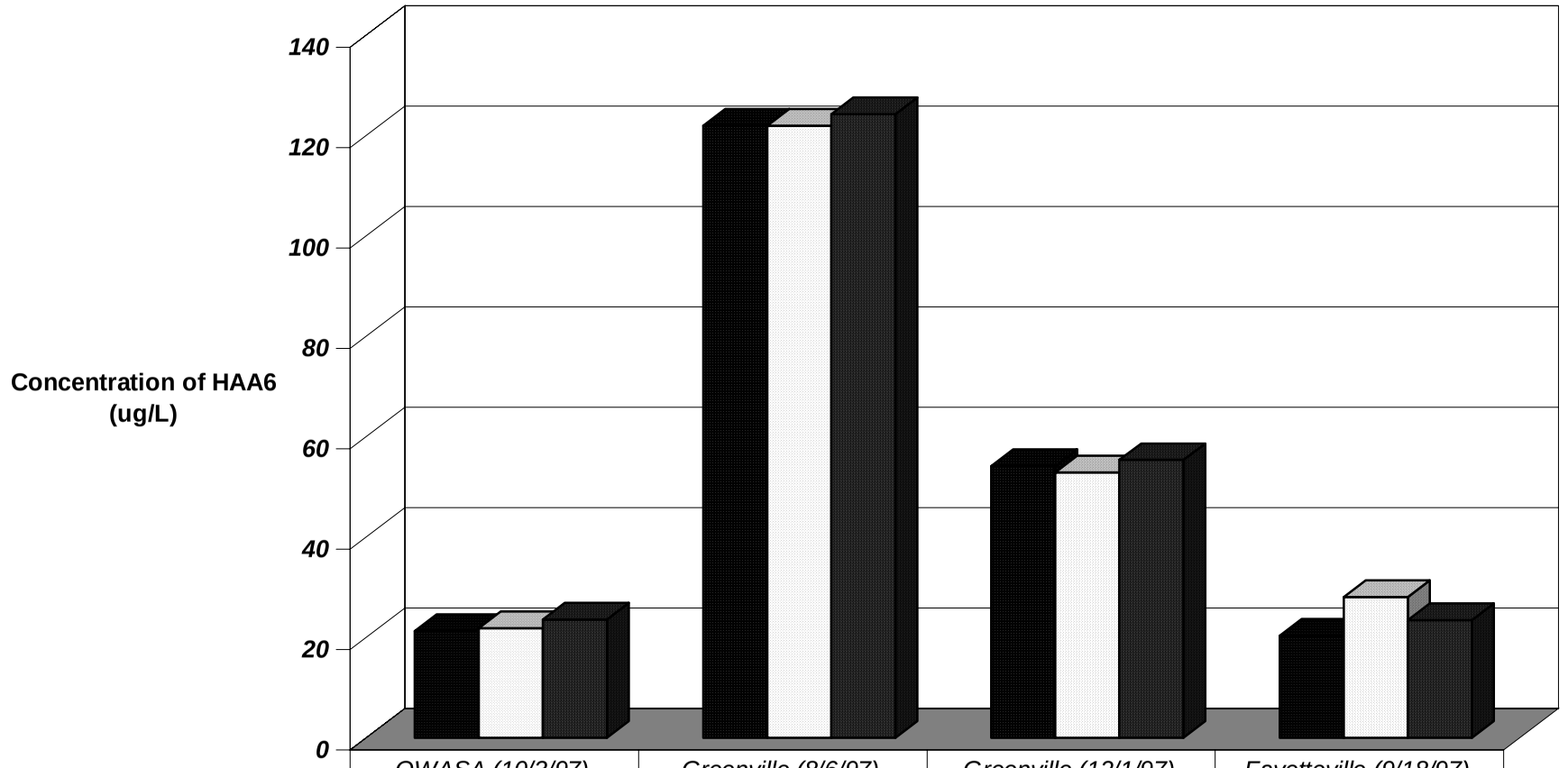
	OWASA (10/3/97)	Greenville (8/6/97)	Greenville (12/1/97)	Fayetteville (9/18/97)	Fayetteville (12/10/97)
■ TTHM ug/L @ t<0, before NH3	39.5	138.6	69.7	46.2	61.5
□ TTHM ug/L @ t=24hr after NH3	41.2	133	69.8	60.3	62.7
■ TTHM ug/L @ t=72hr after NH3	43.5	136	76.5	62.5	66.4

Figure 4.23 HAA6 concentrations with addition of ammonia to finished water-I



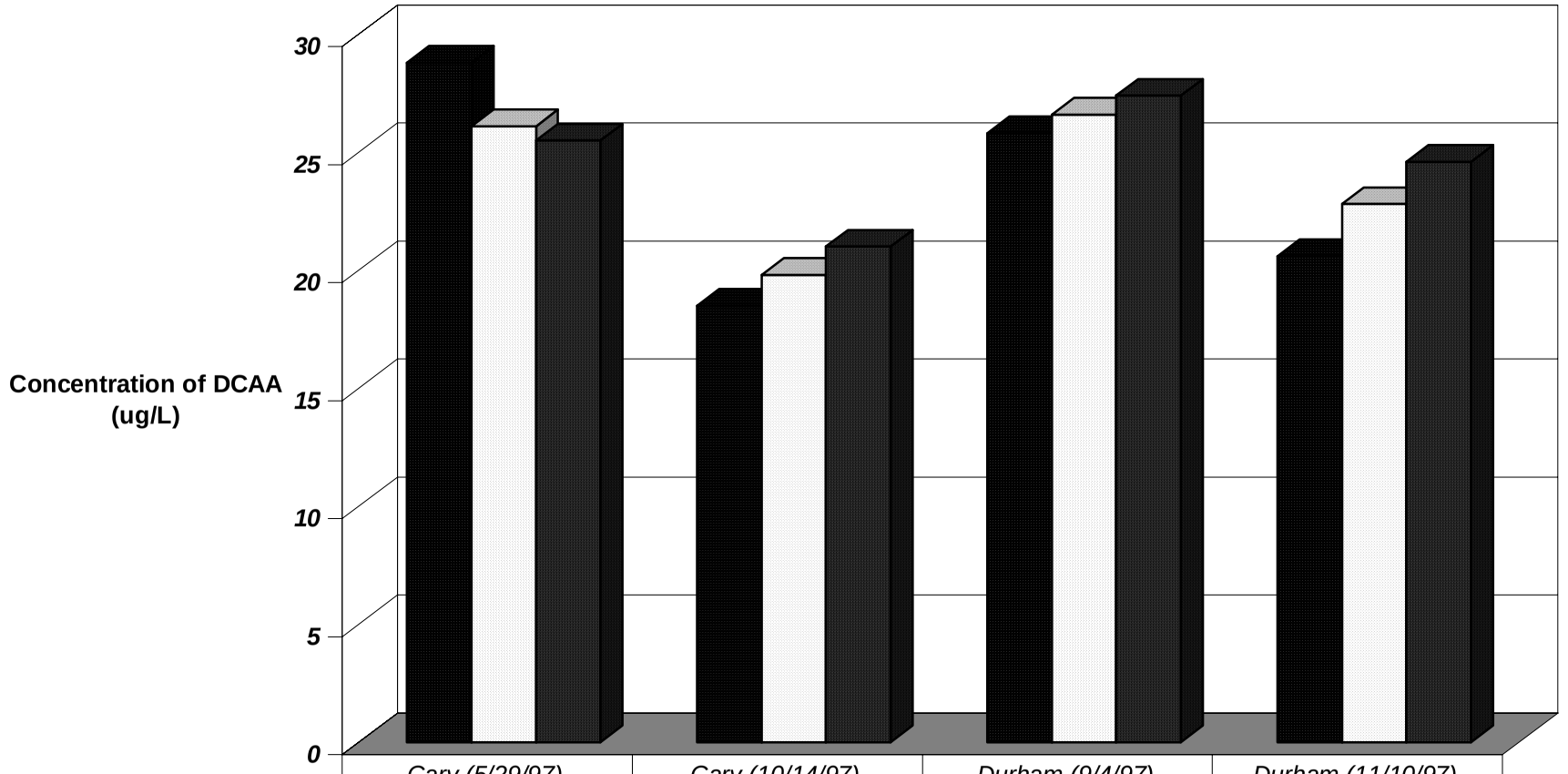
	Cary (5/29/97)	Cary (10/14/97)	Durham (9/4/97)	Durham (11/10/97)
HAA6 ug/L @ t<0, before NH3	73.8	47.6	52.8	40.4
HAA6 ug/L @ t=24hr, after NH3	65.6	55.1	53.5	42.7
HAA6 ug/L @ t=72hr, after NH3	64.1	58	54	44.8

Figure 4.24 HAA6 concentrations with addition of ammonia to finished water-II



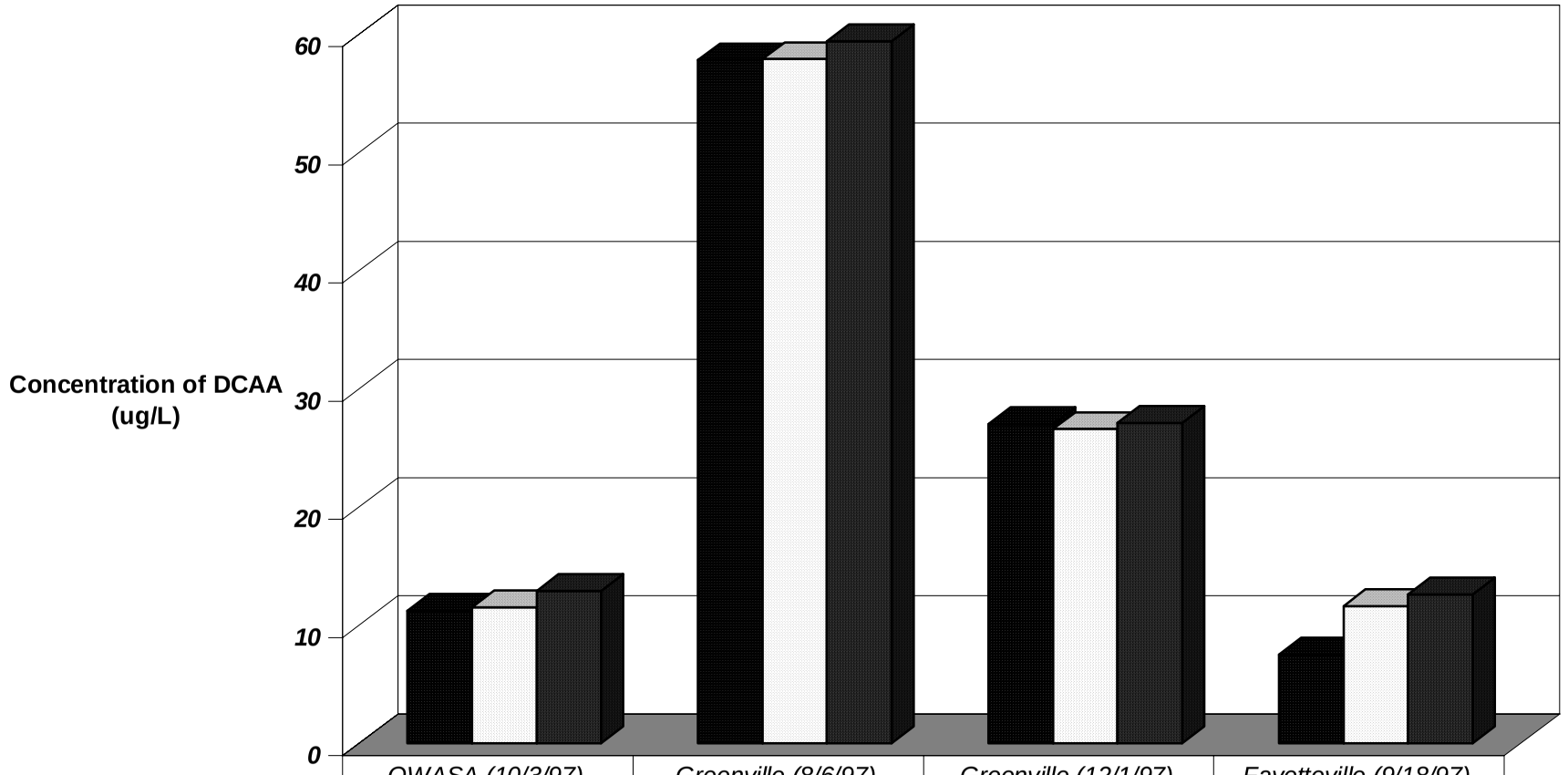
	OWASA (10/3/97)	Greenville (8/6/97)	Greenville (12/1/97)	Fayetteville (9/18/97)
■ HAA6 ug/L @ t<0, before NH3	21.2	121.9	54.1	20.3
□ HAA6 ug/L @ t=24hr after NH3	21.8	121.9	52.8	28
■ HAA6 ug/L @ t=72hr after NH3	23.5	124.2	55.3	23.4

Figure 4.25 DCAA concentrations with addition of ammonia to finished water-I



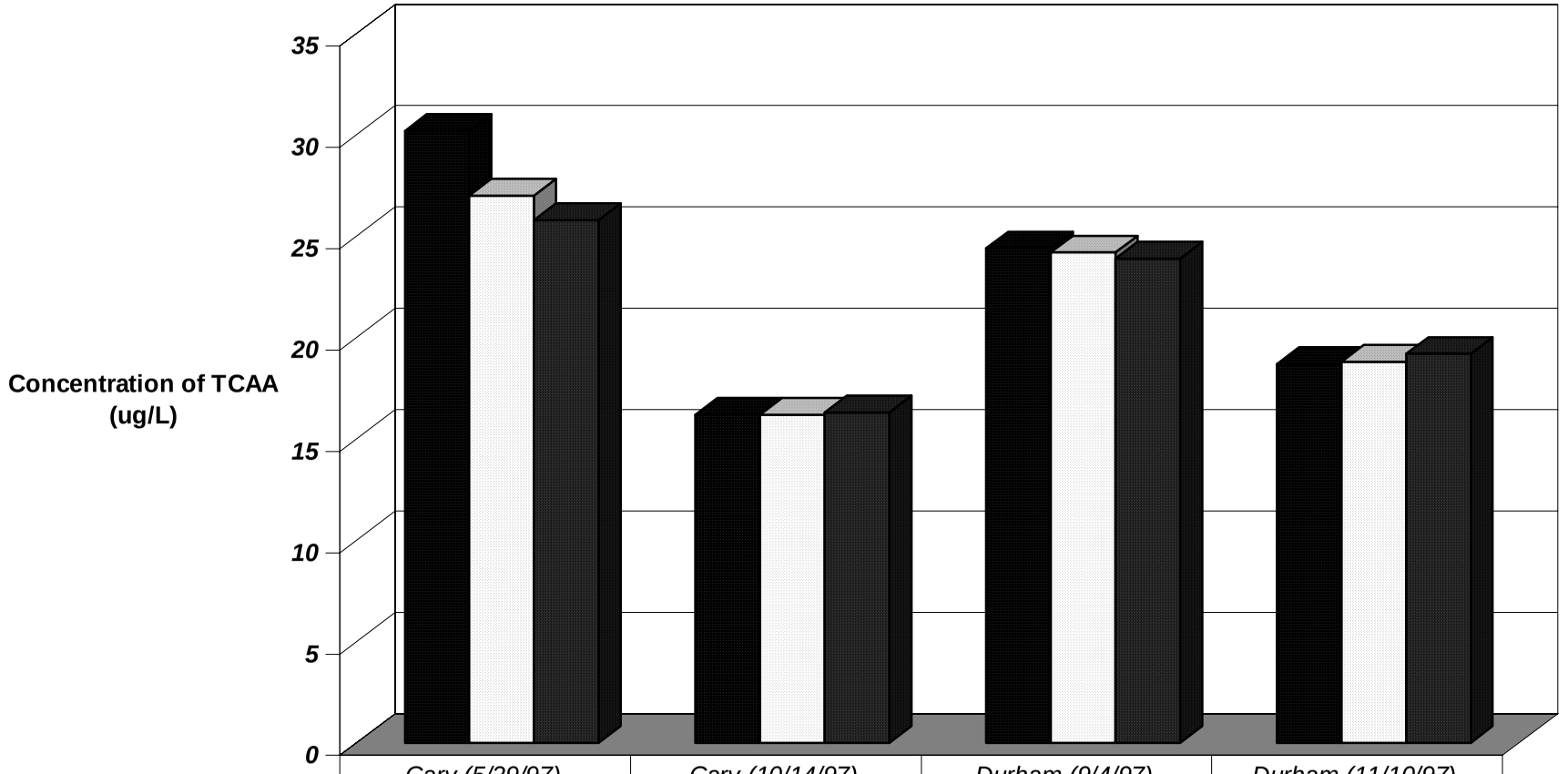
	Cary (5/29/97)	Cary (10/14/97)	Durham (9/4/97)	Durham (11/10/97)
DCAA ug/L @ t<0, before NH3	28.8	18.5	25.8	20.6
DCAA ug/L @ t=24hr after NH3	26.1	19.8	26.6	22.8
DCAA ug/L @ t=72hr after NH3	25.5	21	27.4	24.6

Figure 4.26 DCAA concentrations with addition of ammonia to finished water-II



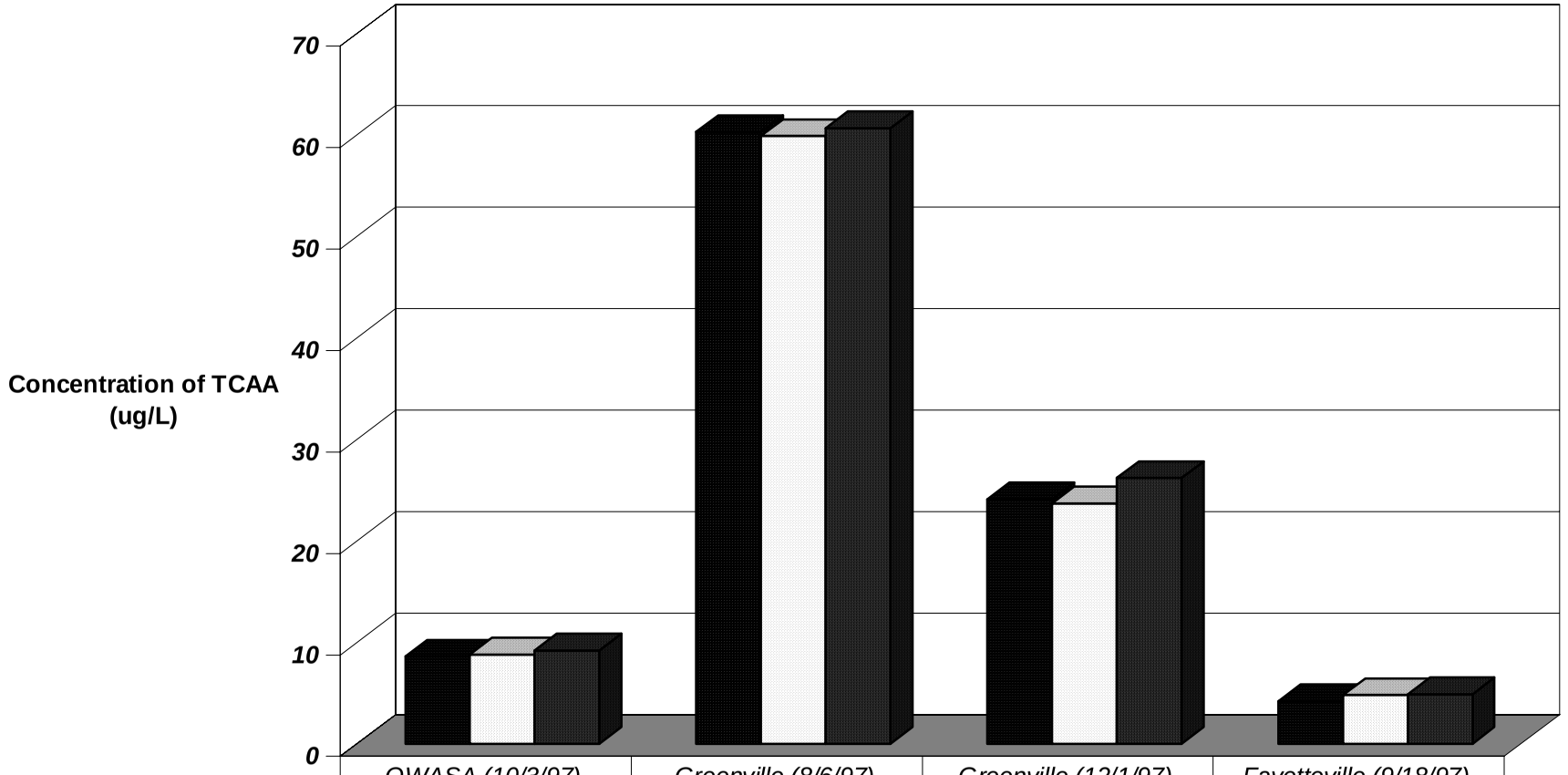
	OWASA (10/3/97)	Greenville (8/6/97)	Greenville (12/1/97)	Fayetteville (9/18/97)
■ DCAA ug/L @ t<0, before NH3	11.2	57.8	27	7.5
□ DCAA ug/L @ t=24hr after NH3	11.5	57.9	26.6	11.6
■ DCAA ug/L @ t=72hr after NH3	12.9	59.4	27.1	12.6

Figure 4.27 TCAA concentrations with addition of ammonia to finished water-I



	Cary (5/29/97)	Cary (10/14/97)	Durham (9/4/97)	Durham (11/10/97)
TCAA ug/L @ t<0, before NH3	30.2	16.2	24.4	18.7
TCAA ug/L @ t=24hr after NH3	27	16.2	24.2	18.8
TCAA ug/L @ t=72hr after NH3	25.8	16.3	23.9	19.2

Figure 4.28 TCAA concentrations with addition of ammonia to finished water-II



	OWASA (10/3/97)	Greenville (8/6/97)	Greenville (12/1/97)	Fayetteville (9/18/97)
TCAA ug/L @ t<0, before NH3	8.6	60.3	24.1	4.2
TCAA ug/L @ t=24hr after NH3	8.8	59.9	23.7	4.8
TCAA ug/L @ t=72hr after NH3	9.2	60.7	26.2	4.9

increase was 11 μg/L for TTHMs. Similarly, a range from 0 to 5.9 μg/L (Cary, October) and an average increase of 3 μg/L was observed for HAA6 concentrations 72 hours after addition of ammonia.

Cowman and Singer (1996) showed that DCAA was the principal HAA species formed from chloramination, while TCAA formation was greatly suppressed by chloramination. In this research, the average increases in TCAA concentrations 24 and 72 hours after ammonia addition were observed to be 0.3 and 0.5 μg/L, respectively. DCAA formation 24 and 72 hours after ammonia addition increased by an average of 1.2 and 2.1 μg/L, respectively. The results show that more DCAA was produced from NH_2Cl than TCAA, as suggested by Cowman and Singer (1996).

Overall, the formation of TTHM and HAA6 seems to be effectively stopped by the addition of ammonia.

4.4 EFFECT OF pH ON DBP FORMATION

The impact of pH on DBP formation was investigated by chlorinating settled water from each treatment plant at various pH values under controlled laboratory conditions. pH has been shown to be a dominant factor influencing both the production of total DBPs and specific DBP components.

Figure 4.29 shows that TTHM formation increases with increasing pH for Greenville settled water. A higher increase was observed in August than in December. In contrast to this, the overall concentration of HAA6 decreases with increasing pH as shown in Figure 4.30. The decrease in HAA6 with increasing pH reflects the effects of both TCAA and DCAA, the primary HAA species, and their combined behavior with respect to pH. TCAA formation decreases markedly with increasing pH, as shown in Figure 4.31, but DCAA formation either increases slightly with increasing pH or is not influenced by pH, as shown in Figure 4.32.

In this work, the pH adjustment experiments were performed twice for each treatment plant. Figures 4.33-4.36 summarize TTHM, HAA6, TCAA and DCAA formation, respectively, for all six plants during the first set of tests. Likewise, Figures 4.37-4.40 present parallel results for the second round of testing.

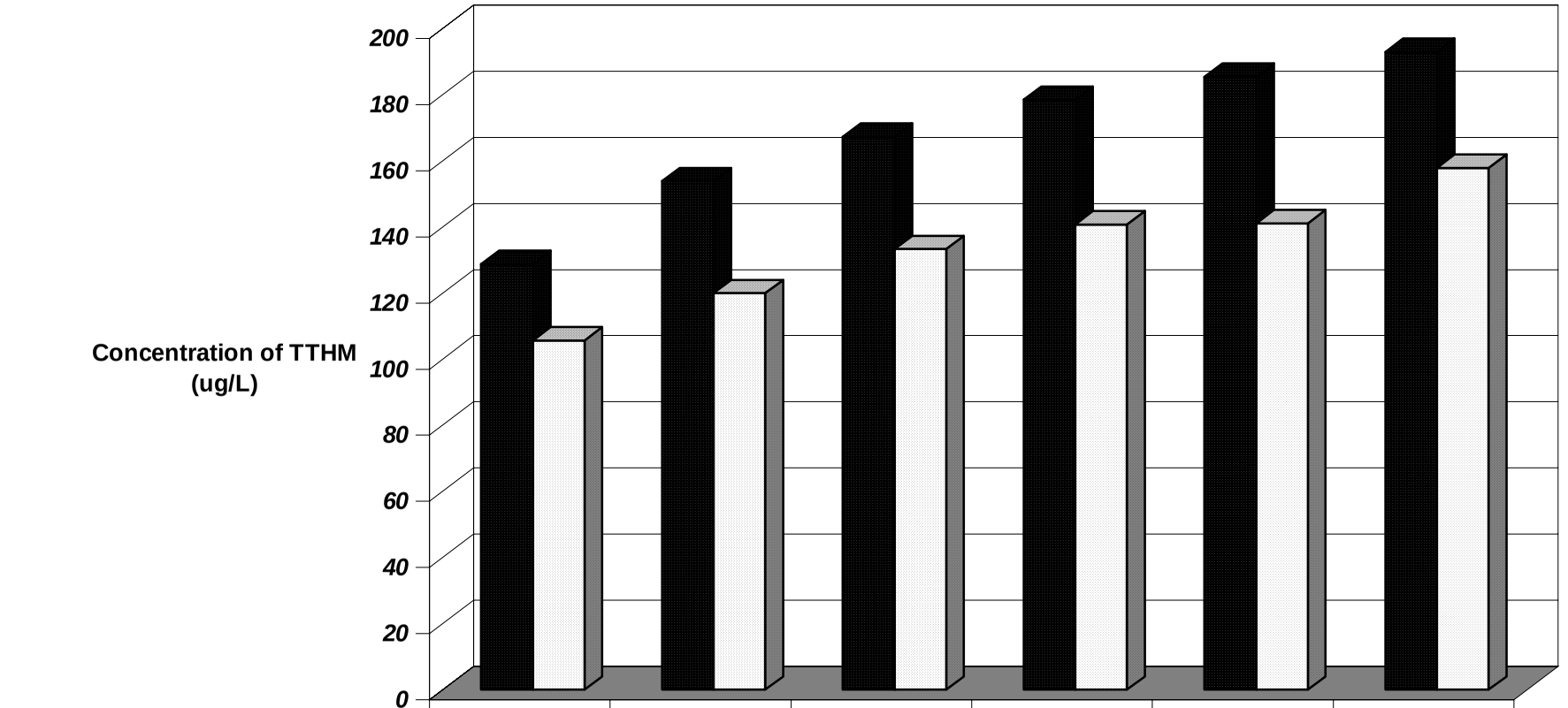
For all six plants and both rounds of testing, there is a consistent trend for each DBP species measured. As can be seen in Figures 4.33 and 4.37, TTHM concentrations consistently increase with increasing pH. TCAA consistently decreases with increasing pH as can be seen in Figures 4.35 and 4.39. Figures 4.34 and 4.38 show that, due to the behavior of TCAA, overall HAA6 formation decreases as pH increases. DCAA formation exhibited a minor increase with increasing pH in all cases (see Figures 4.36 and 4.40).

4.5 DISCUSSION OF pH RESULTS

The impact of pH on the formation of DBPs is quite complex as is seen in the formation of THMs and HAAs in Figures 4.33-4.40. For the pH range studied in this research, the predominant chlorine species in these waters should be either hypochlorous acid at pH values less than 7.58 or hypochlorite ion at pH values greater than 7.58. Hypochlorous acid is known to be a more powerful oxidizing agent than hypochlorite ion (White, 1992). Therefore, pH's at which hypochlorous acid is dominant should also be the pH's at which the more highly oxidized chlorinated compounds are abundant. Yet other factors that may influence DBP formation and species distribution should also be taken into consideration, such as the chemical nature of the precursors and the different pathways that form each DBP species.

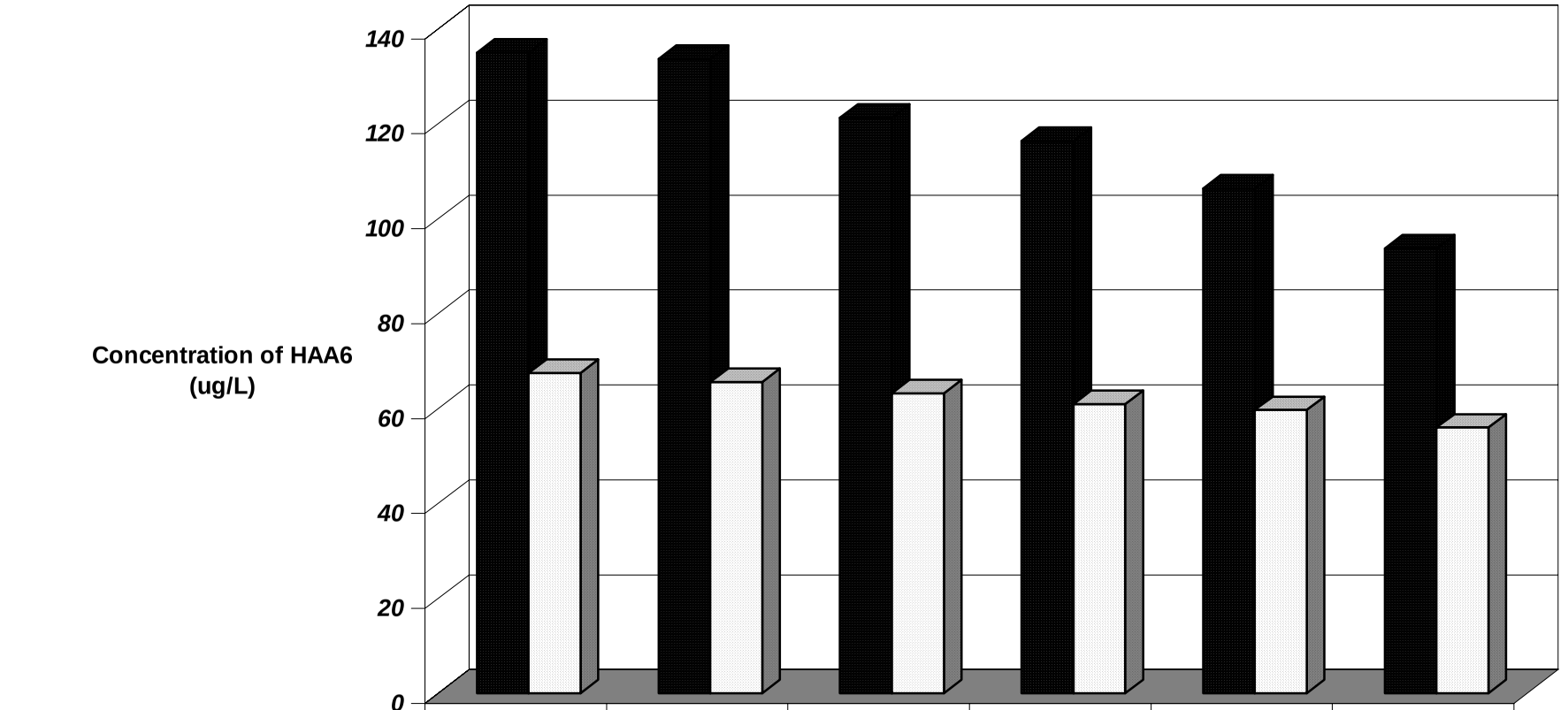
Numerous researchers have observed a reduction in THM formation by reducing the pH (Stevens et al. 1978; Trussell and Umphres 1978; Engerholm and Amy 1983; Fleischacker and Randtke 1983). Other researchers studied the influence of pH on HAA formation and speciation (Miller and Uden 1983;

Figure 4.29 TTHM Concentrations at various pH values (Greenville, NC)



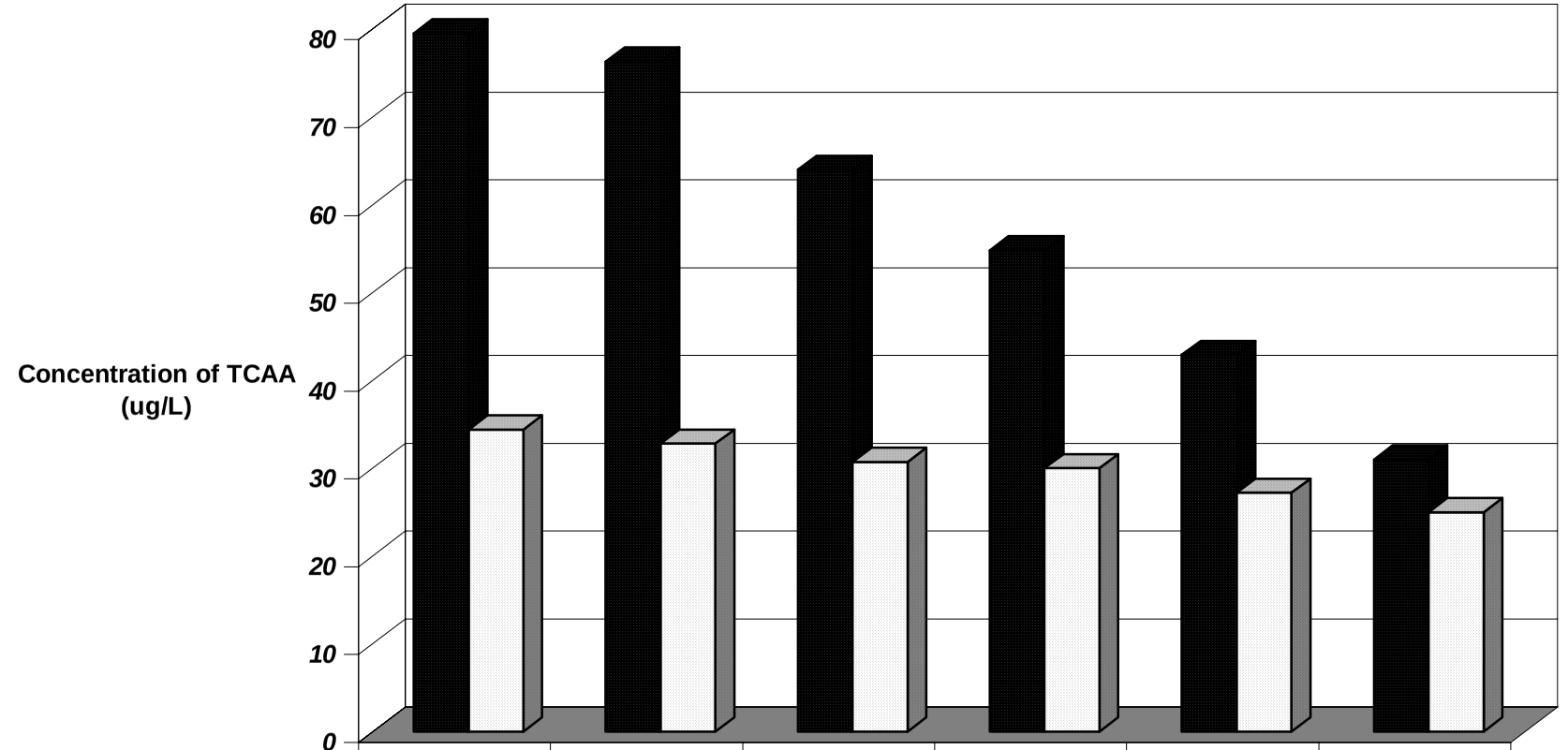
	pH 6.0	pH 6.5	pH 7.0	pH 7.5	pH 8.0	pH 8.5
■ TTHM ug/L (8/6/97); TOC 4.04 mg/L	128.7	153.8	167.2	178.4	185.4	192.9
□ TTHM ug/L (12/1/97); TOC 2.9 mg/L	105.7	119.9	133.3	140.7	141.1	157.8

Figure 4.30 HAA6 Concentrations at various pH values (Greenville, NC)



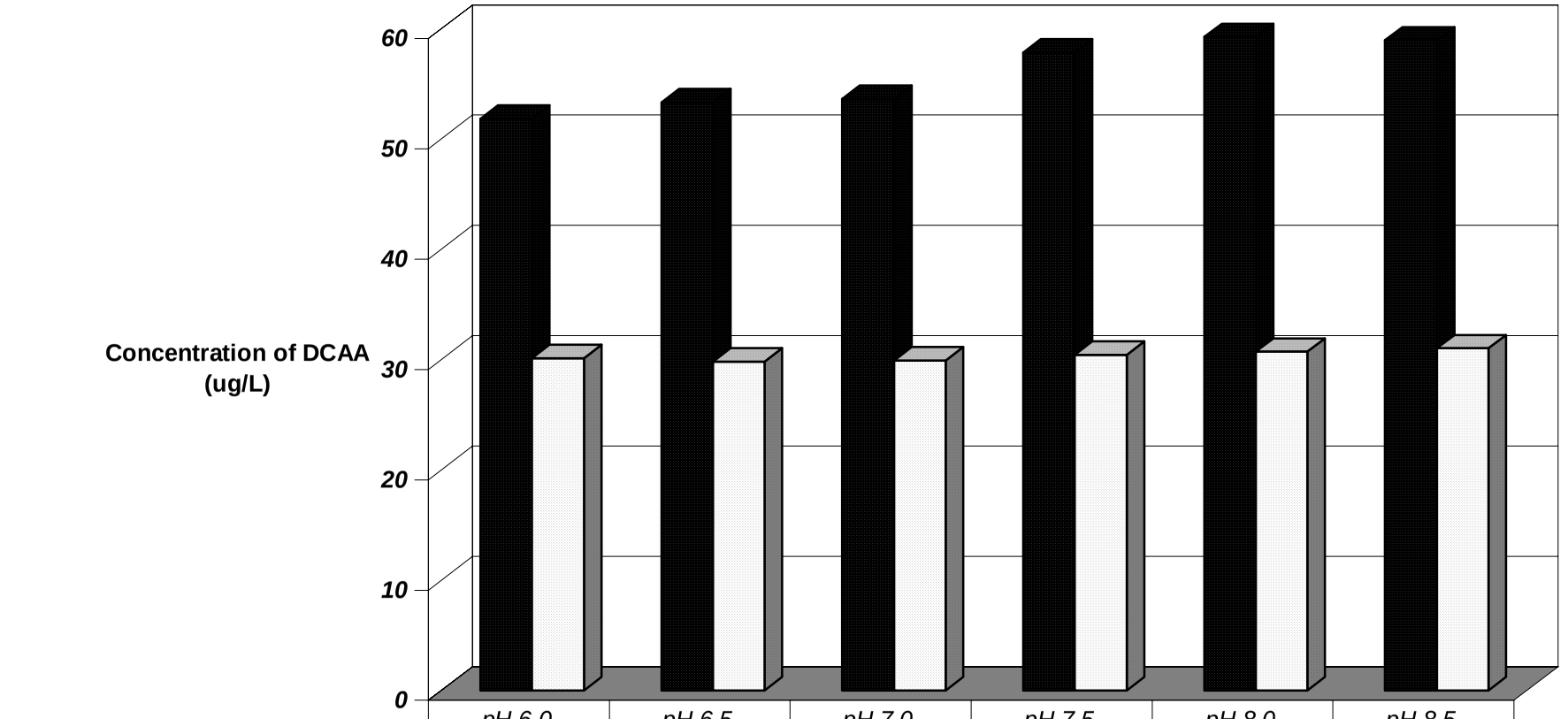
	pH 6.0	pH 6.5	pH 7.0	pH 7.5	pH 8.0	pH 8.5
■ HAA6 ug/L (8/6/97); TOC 4.04 mg/L	135	133.7	121.3	116.4	106.4	93.7
□ HAA6 ug/L (12/1/97); TOC 2.9 mg/L	67.5	65.6	63.2	60.9	59.7	56

Figure 4.31 TCAA Concentrations at various pH values (Greenville, NC)



	pH 6.0	pH 6.5	pH 7.0	pH 7.5	pH 8.0	pH 8.5
■ TCAA ug/L (8/6/97); TOC 4.04 mg/L	79.5	76.3	64	54.8	42.9	31
□ TCAA ug/L (12/1/97); TOC 2.9 mg/L	34.4	32.8	30.7	30	27.2	25

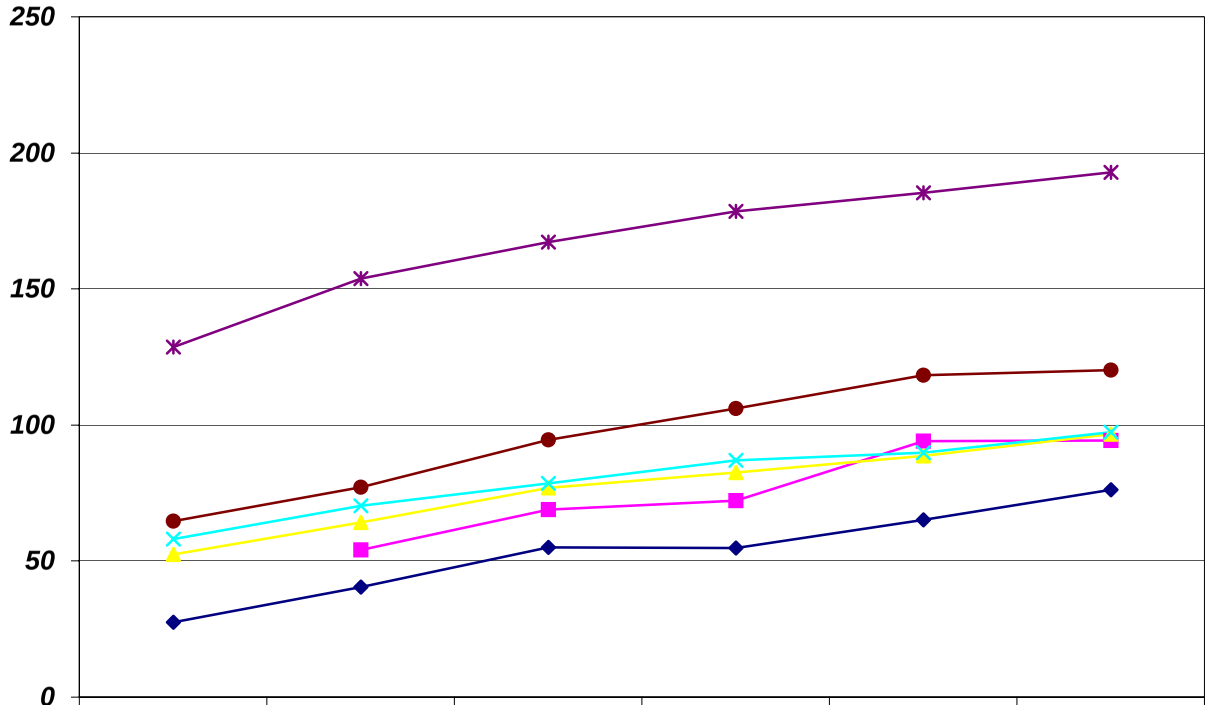
Figure 4.32 DCAA Concentrations at various pH values (Greenville, NC)



	pH 6.0	pH 6.5	pH 7.0	pH 7.5	pH 8.0	pH 8.5
■ DCAA ug/L (8/6/97); TOC 4.04 mg/L	51.8	53.3	53.6	57.8	59.2	58.9
□ DCAA ug/L (12/1/97); TOC 2.9 mg/L	30.1	29.8	29.9	30.4	30.7	31

pH

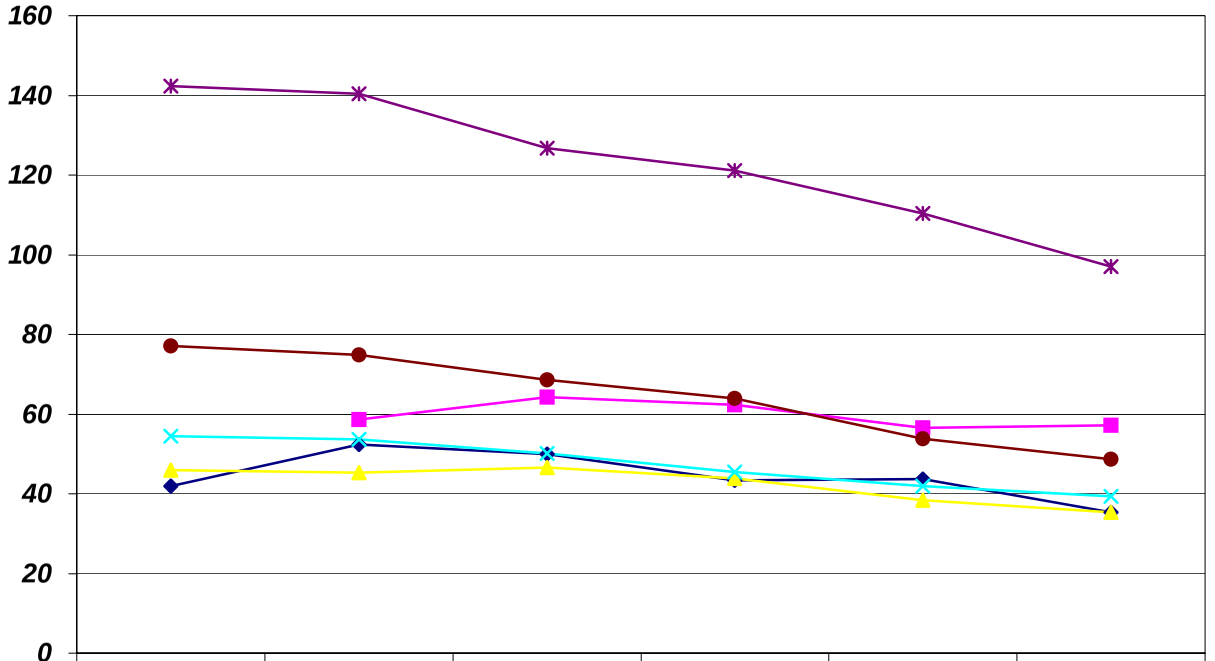
Figure 4.33 TTHM concentrations for chlorination of settled water at various pH values during first round of testing



	pH 6.0	pH 6.5	pH 7.0	pH 7.5	pH 8.0	pH 8.5
OWASA (5/6/97)	27.5	40.5	55.1	54.7	65.1	76.3
Cary (5/29/97)		54	68.9	72.3	94.1	94.2
High Point (6/13/97)	52.5	64.2	76.8	82.6	88.7	96.6
Durham (9/4/97)	58.2	70.3	78.5	87.1	89.8	97.4
Greenville (8/6/97)	128.7	153.8	167.2	178.4	185.4	192.9
Fayetteville (9/18/97)	64.7	77.1	94.6	106	118.2	120.1

pH

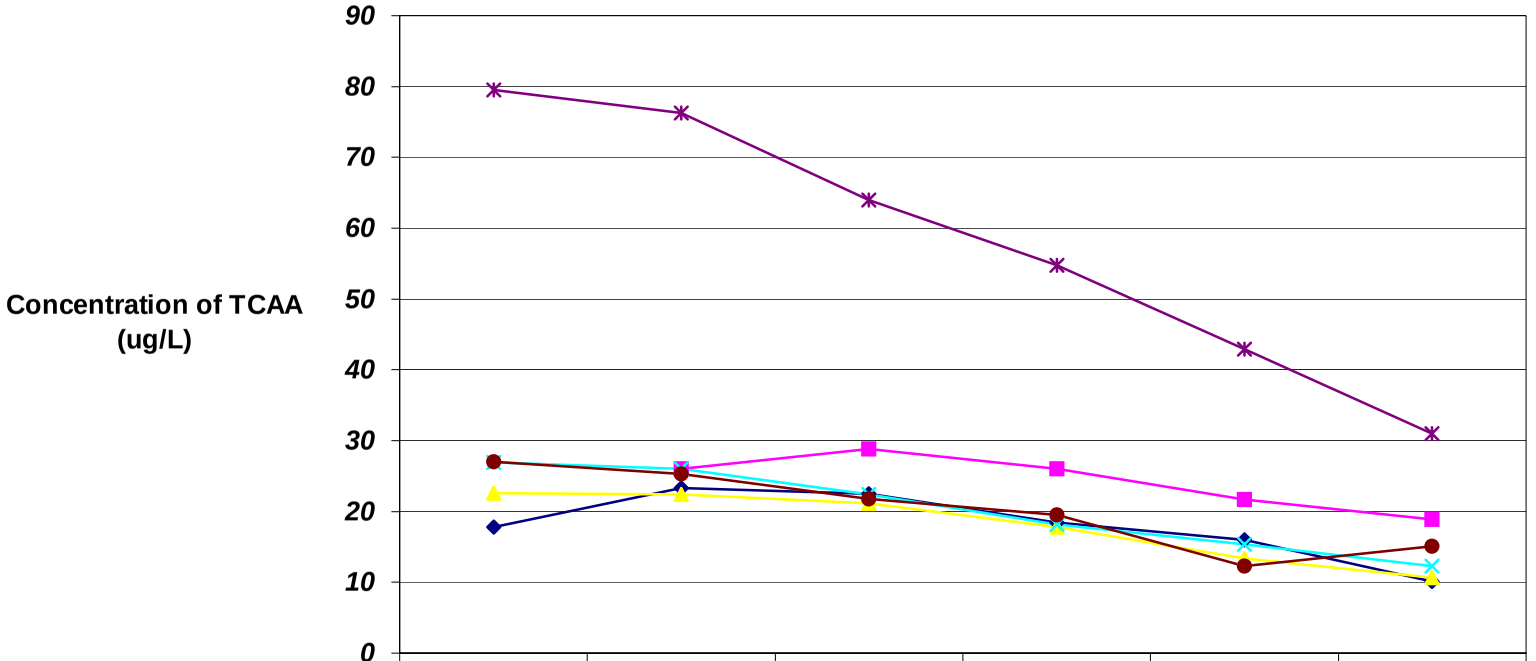
Figure 4.34 HAA6 concentrations for chlorination of settled water at various pH values during first round of testing



	pH 6.0	pH 6.5	pH 7.0	pH 7.5	pH 8.0	pH 8.5
OWASA (5/6/97)	42	52.3	50	43.4	43.7	35.4
Cary (5/29/97)		58.6	64.3	62.3	56.6	57.2
High Point (6/13/97)	45.9	45.3	46.6	43.8	38.4	35.4
Durham (9/4/97)	54.4	53.7	50.2	45.4	42	39.3
Greenville (8/6/97)	142.3	140.4	126.7	121.2	110.3	97
Fayetteville (9/18/97)	77.1	74.9	68.6	64	53.8	48.6

pH

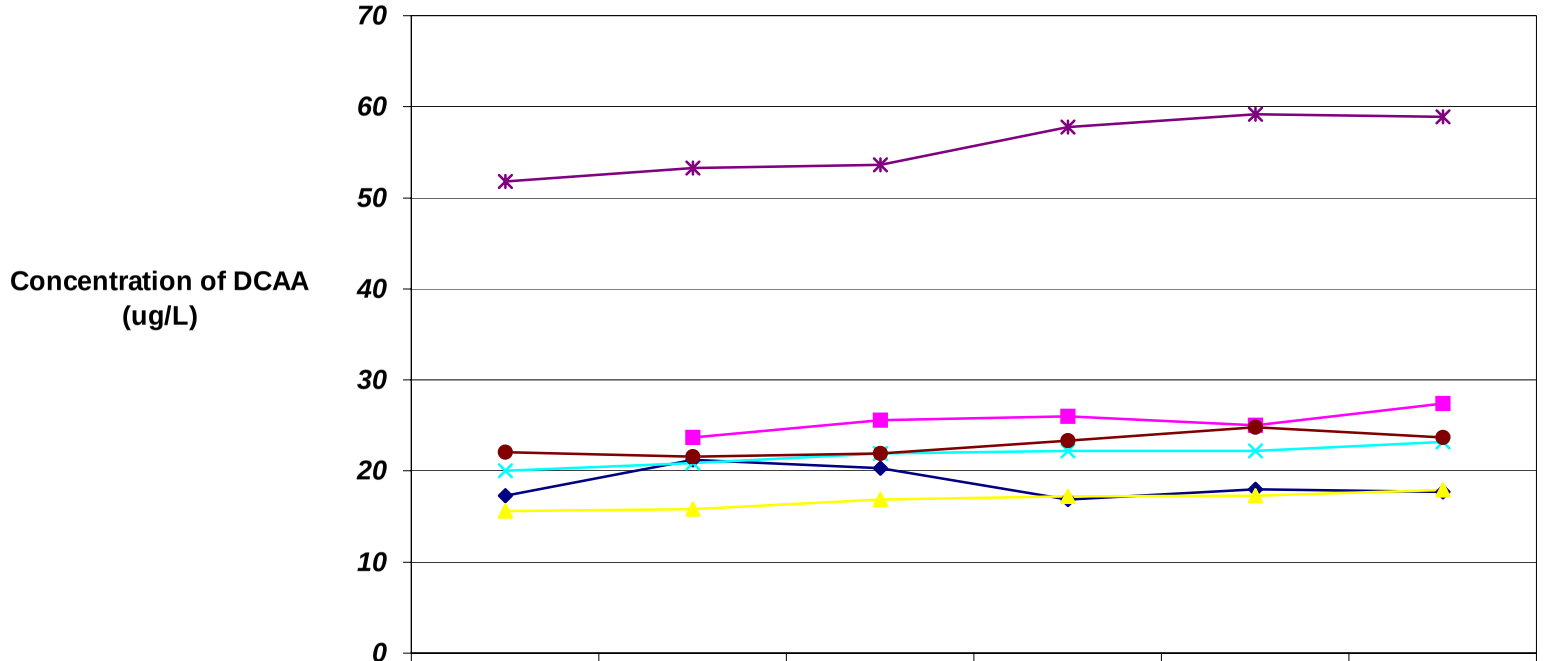
Figure 4.35 TCAA concentrations for chlorination of settled water at various pH values during first round of testing



	pH 6.0	pH 6.5	pH 7.0	pH 7.5	pH 8.0	pH 8.5
OWASA (5/6/97)	17.8	23.3	22.5	18.4	16	10.1
Cary (5/29/97)		26	28.8	26	21.7	18.9
High Point (6/13/97)	22.6	22.4	21.1	17.8	13.4	10.7
Durham (9/4/97)	26.9	26	22.4	18.2	15.4	12.3
Greenville (8/6/97)	79.5	76.3	64	54.8	42.9	31
Fayetteville (9/18/97)	27	25.3	21.8	19.5	12.3	15.1

pH

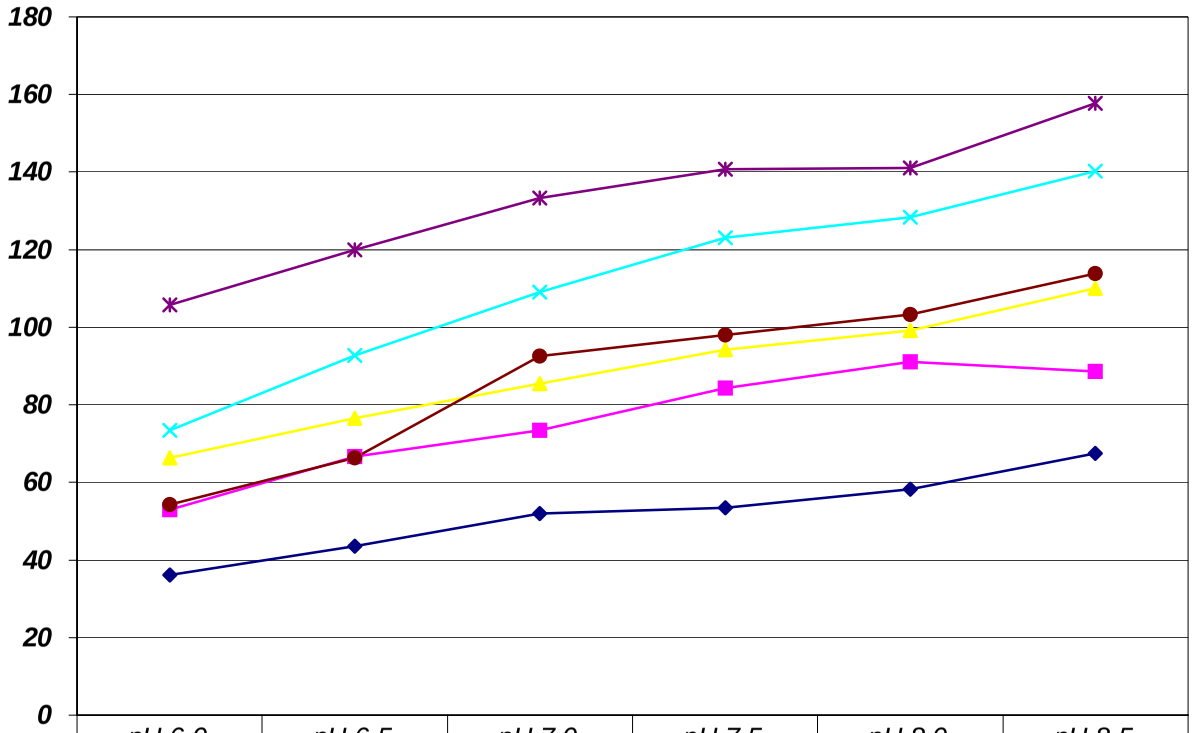
Figure 4.36 DCAA concentrations for chlorination of settled water at various pH values during first round of testing



	pH 6.0	pH 6.5	pH 7.0	pH 7.5	pH 8.0	pH 8.5
OWASA (5/6/97)	17.3	21.2	20.3	16.9	18	17.7
Cary (5/29/97)		23.7	25.6	26	25	27.4
High Point (6/13/97)	15.6	15.8	16.9	17.2	17.3	17.9
Durham (9/4/97)	20	20.9	21.9	22.2	22.2	23.2
Greenville (8/6/97)	51.8	53.3	53.6	57.8	59.2	58.9
Fayetteville (9/18/97)	22.1	21.6	21.9	23.3	24.8	23.7

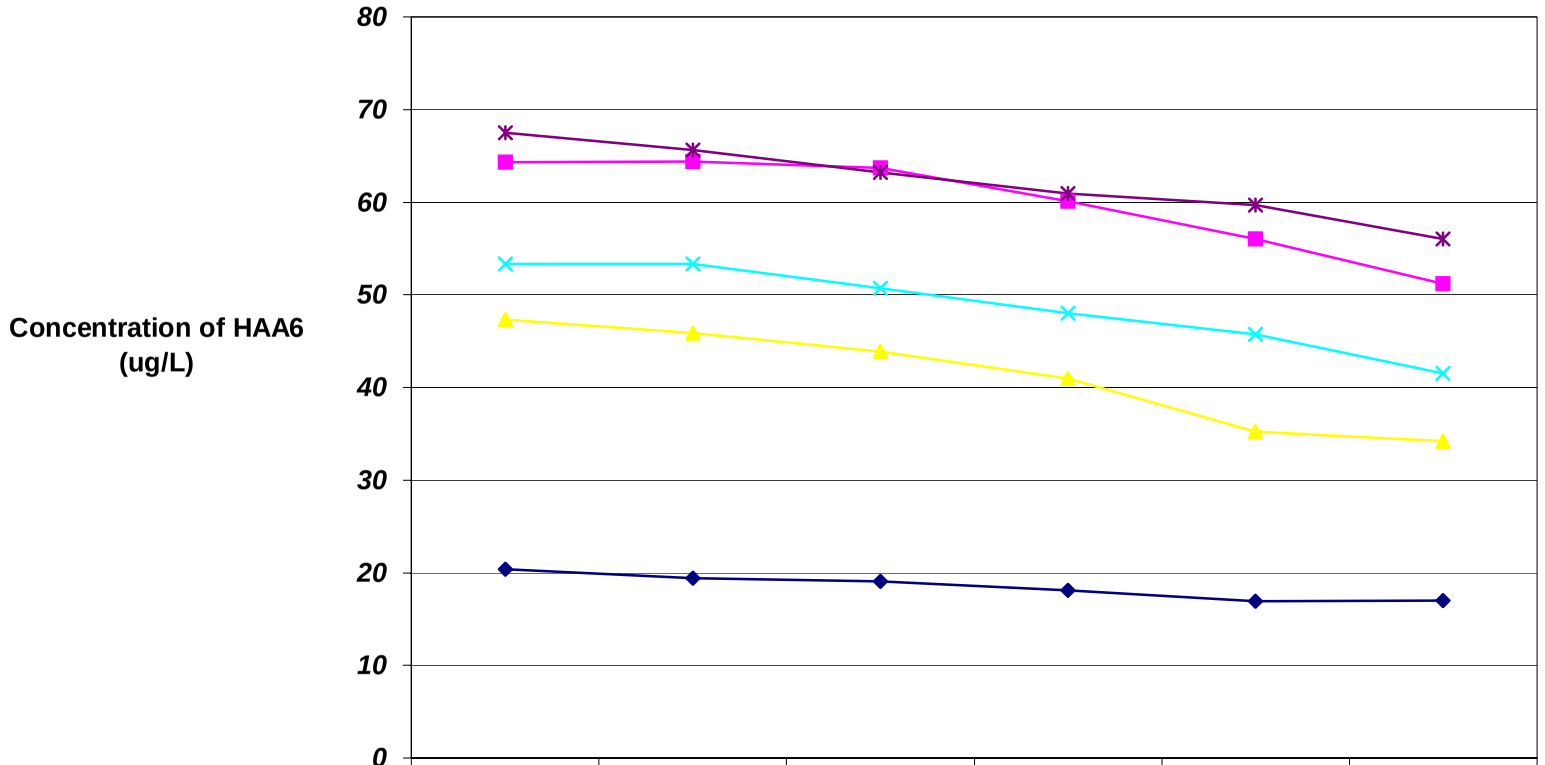
pH

Figure 4.37 TTHM concentrations for chlorination of settled water at various pH values during second round of testing



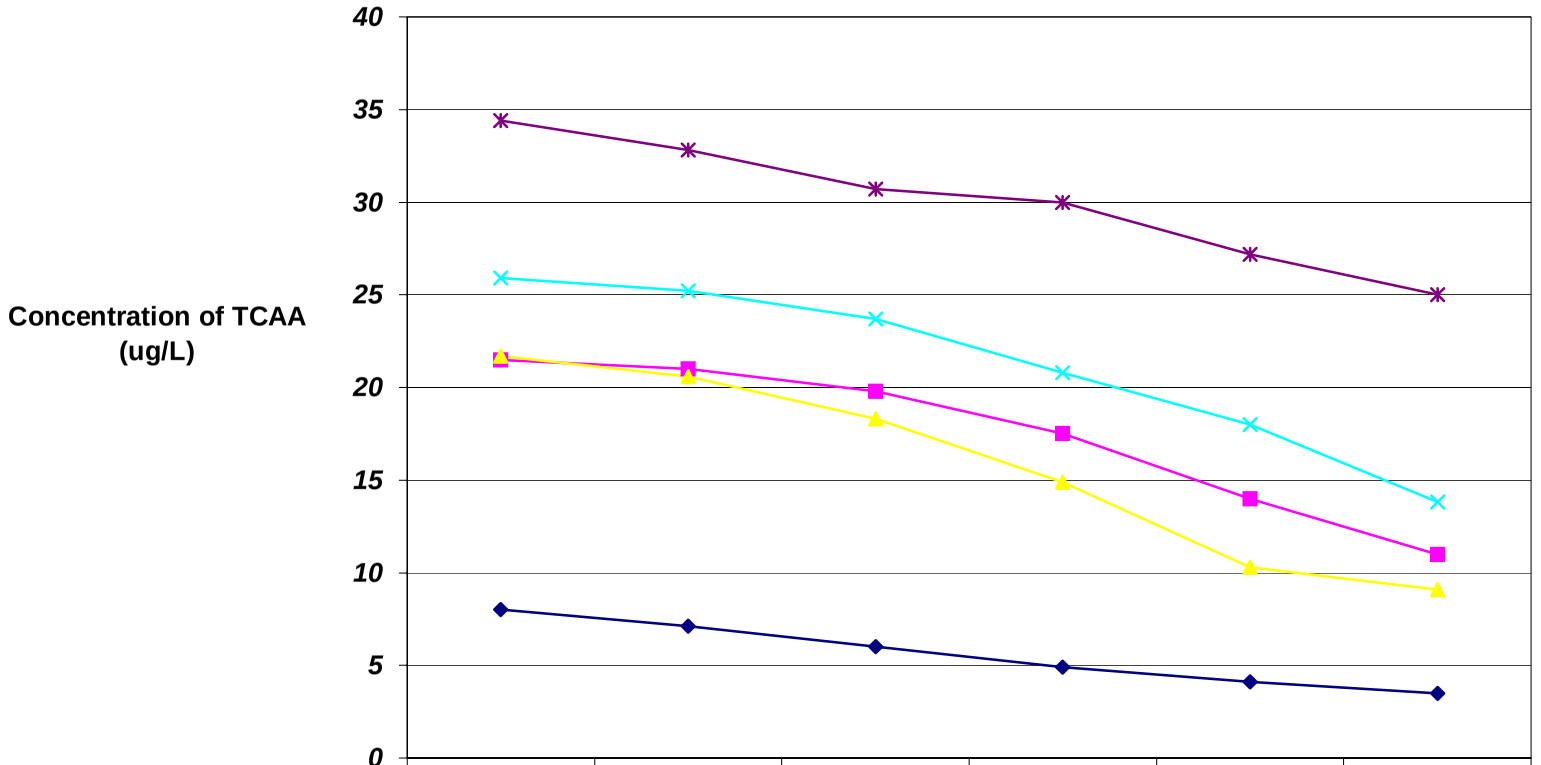
	pH 6.0	pH 6.5	pH 7.0	pH 7.5	pH 8.0	pH 8.5
OWASA (10/3/97)	36.1	43.6	51.9	53.5	58.3	67.4
Cary (10/14/97)	53	66.7	73.5	84.3	91	88.6
High Point (10/29/97)	66.4	76.5	85.4	94.2	99.2	110.1
Durham (11/10/97)	73.5	92.7	109	123.1	128.3	140.2
Greenville (12/1/97)	105.7	119.9	133.3	140.7	141.1	157.8
Fayetteville (12/10/97)	54.2	66.3	92.5	98	103.3	113.9

Figure 4.38 HAA6 concentrations for chlorination of settled water at various pH values during second round of testing



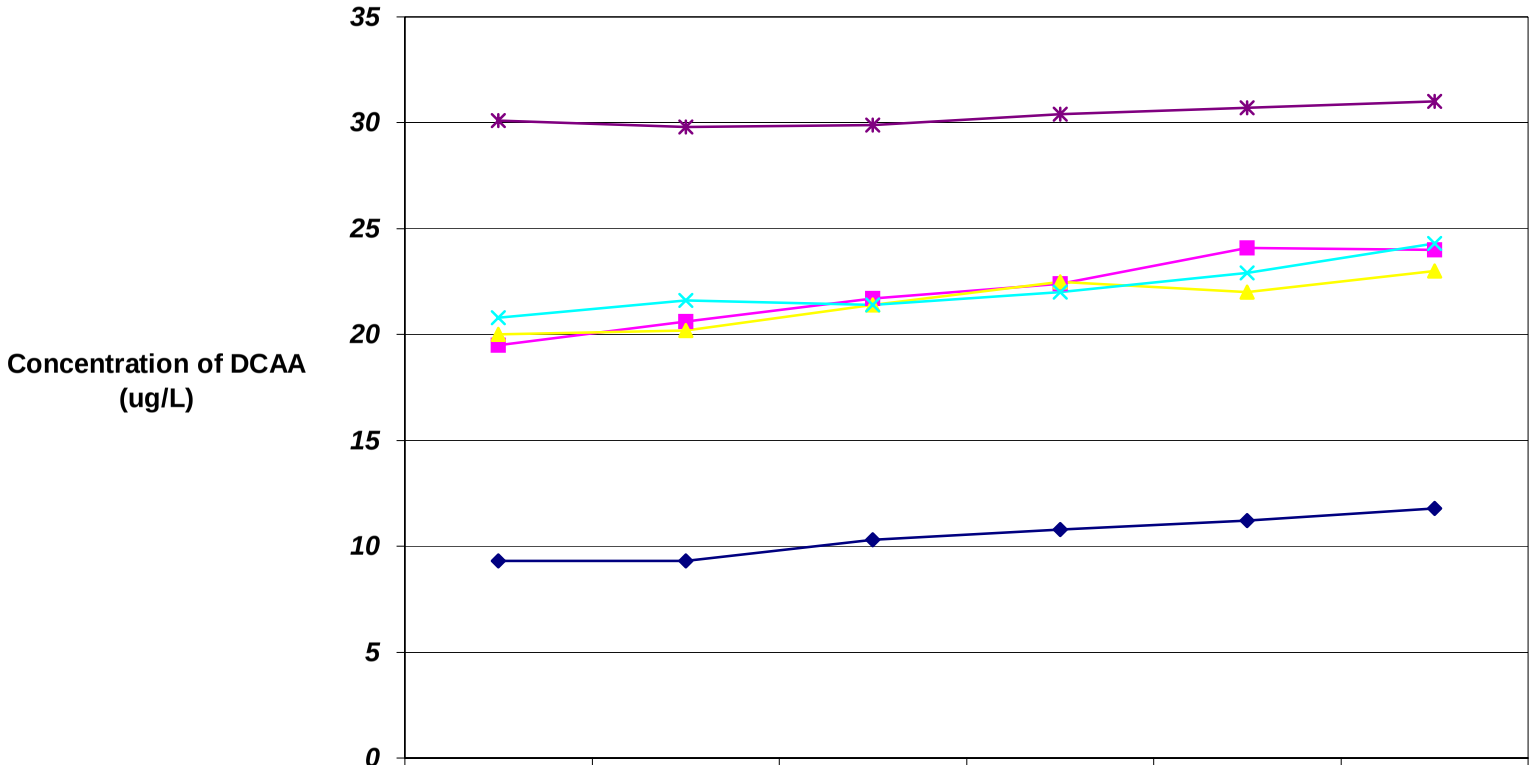
	pH 6.0	pH 6.5	pH 7.0	pH 7.5	pH 8.0	pH 8.5
OWASA (10/3/97)	20.4	19.4	19.1	18.1	16.9	17
Cary (10/14/97)	64.3	64.4	63.7	60.1	56	51.2
High Point (10/29/97)	47.3	45.9	43.9	41	35.2	34.2
Durham (11/10/97)	53.3	53.3	50.7	48	45.7	41.5
Greenville (12/1/97)	67.5	65.6	63.2	60.9	59.7	56

Figure 4.39 TCAA concentrations for chlorination of settled water at various pH values during second round of testing



	pH 6.0	pH 6.5	pH 7.0	pH 7.5	pH 8.0	pH 8.5
Cary (10/14/97)	21.5	21	19.8	17.5	14	11
High Point (10/29/97)	21.7	20.6	18.3	14.9	10.3	9.1
Durham (11/10/97)	25.9	25.2	23.7	20.8	18	13.8
Greenville (12/1/97)	34.4	32.8	30.7	30	27.2	25

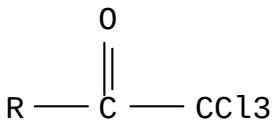
Figure 4.40 DCAA concentrations for chlorination of settled water at various pH values during second round of testing



	pH 6.0	pH 6.5	pH 7.0	pH 7.5	pH 8.0	pH 8.5
Cary (10/14/97)	19.5	20.6	21.7	22.4	24.1	24
High Point (10/29/97)	20	20.2	21.4	22.5	22	23
Durham (11/10/97)	20.8	21.6	21.4	22	22.9	24.3
Greenville (12/1/97)	30.1	29.8	29.9	30.4	30.7	31

Reckhow and Singer 1984; Stevens et al. 1989; Pourmoghaddas et al. 1993). They found that TCAA had lower formation at higher pH's, and DCAA had similar formation at both low and high pH's.

Reckhow and co-workers (Reckhow and Singer 1985; Reckhow et al. 1986; Reckhow et al. 1990) have suggested that both $CHCl_3$ and TCAA were generated from the same group of trichloromethyl keto precursors, as shown below:



(4.1)

These species are not very reactive to chlorine addition at neutral pH, but at elevated pH, they are much more reactive and become a major source of potential THM formation.

Based on a haloform-type mechanism, increasing pH will help THM formation by increasing the stability of carbonanions of the structure indicated above. At the same time, higher pH values will shift the chlorination mechanism from oxidation to substitution as the presence of the stronger oxidizing agent HOCl decreases. Additionally, alkaline conditions result in a significant base-catalyzed hydrolysis reaction that forms THMs.

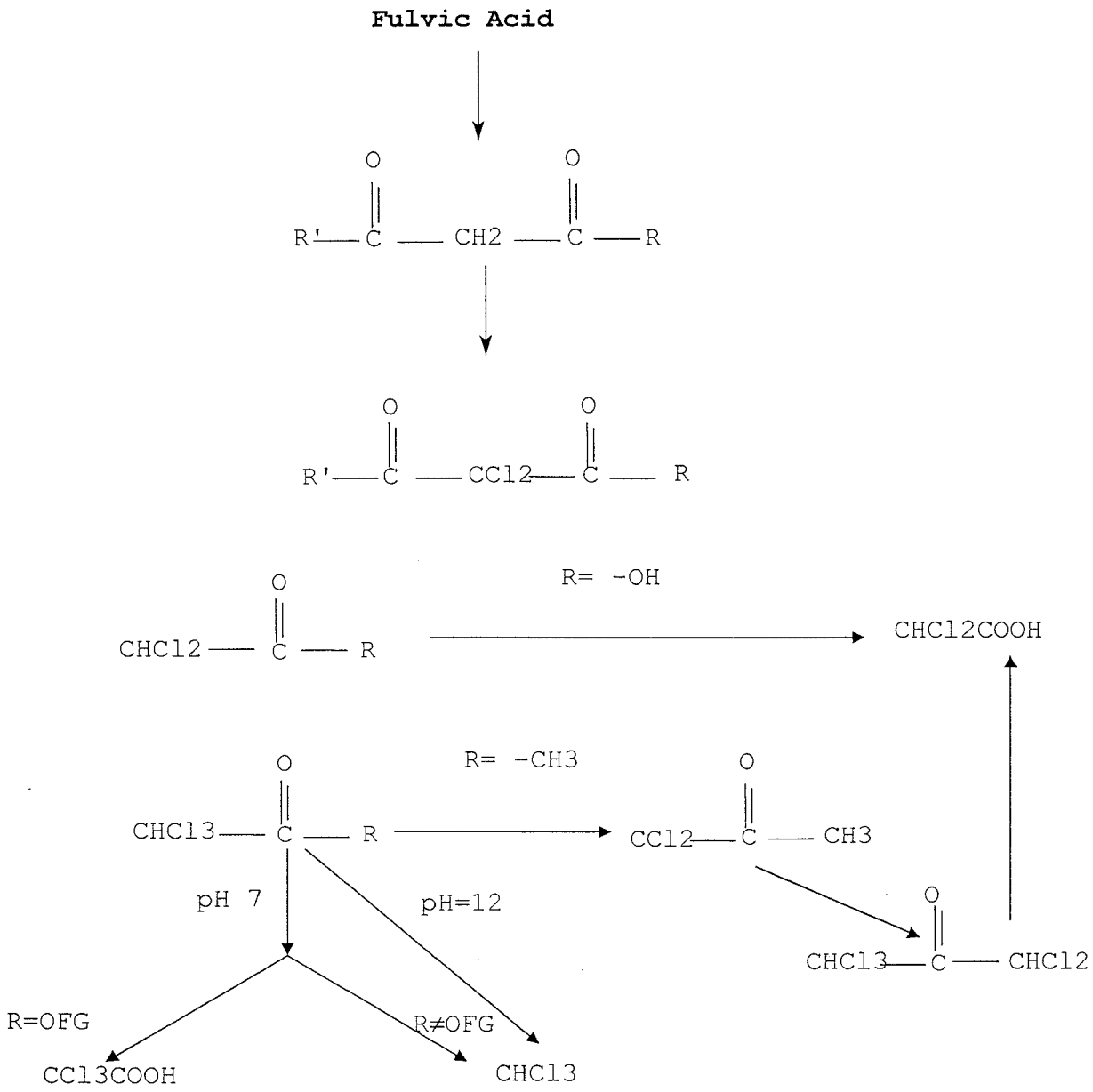
TCAA formation tends to depend to a much larger degree on the rigorous oxidation of this trichloromethyl species while haloform reactions are base-catalyzed. With a combination of the pH effect on the distribution of chlorine species and on the hydrolysis reaction that forms $CHCl_3$, it is easy to predict that the domination of HOCl at low pH's will favor TCAA formation whereas hydrolysis reactions favoring $CHCl_3$ formation would prevail at high pH's.

However, DCAA exhibits more complex formation behavior with respect to pH. It was proposed by Reckhow and Singer (1985) that the oxidation reactions of humic materials will lead to significant formation of transient β-diketone-type moieties. The activated carbon atom is then fully substituted with chlorine. Afterwards, the structure would undergo hydrolysis and produce DCAA if the remaining R group is -OH. Otherwise, the reaction will lead to the base-hydrolyzable trichloromethyl species. Figure 4.41 illustrates another pathway for DCAA formation. Both mechanisms do not appear to be affected by pH.

Miller and Uden (1983) reported that DCAA formation is slightly favored at lower pH's. Reckhow and Singer (1985) claimed that Miller and Uden;s work results actually showed that DCAA formation had a local maximum at pH 7 with a decrease at both high and low pH; their work showed similar trends (Reckhow and Singer 1985). However, in a later paper, Reckhow et al. (1990) concluded that DCAA formation showed no significant change with increasing pH. Stevens et al. (1989) indicated similar formation of DCAA at pH 5 and 9.4, and perhaps slightly higher formation at pH 7.

In general, the observed results of this research are consistent with the previous discussion in that THM and $CHCl_3$ formation increase with increasing pH, HAA6 and TCAA concentrations decrease with increasing pH, and DCAA formation tends to be insensitive to variations in pH.

Figure 4.41 Simplified conceptual model for the formation of major organic halide products from fulvic acid (OFG = oxidizable functional group). Adapted from Reckhow and Singer (1985).



4.6 ADDITIONAL DATA ANALYSIS

Regression analyses were conducted for a number of parameters for all twelve sets of field data and chlorination experiments to evaluate correlations between TTHM and HAA6 concentrations, chlorine consumption and TTHM or HAA6 formation, and TOC concentration and TTHM or HAA6 formation.

4.6.1 Correlation Between TTHM and HAA6 Concentrations

Figures 4.42 and 4.43 show correlations between TTHM (primarily chloroform, and HAA6 (primarily DCAA and TCAA) in these low bromide-containing waters) for the settled water chlorinated at pH 6 and 8, respectively. The correlation coefficients (R^2) are 0.65 and 0.69, respectively. Both TTHM and HAA6 concentrations are reported as the sum of the species concentrations in units of micrograms per liter. Figure 4.44 illustrates the linear regression for filtered water, finished water and waters collected from the distribution system of all twelve facilities during this study. There is also a reasonably good correlation ($R^2 = 0.78$) between TTHM and HAA6 concentrations for waters collected from the treatment systems themselves.

Table 4.6 summarizes the statistics of the relationships between TTHM and HAA6 concentrations for settled water chlorinated at each of the various pH's examined and for samples collected in the treatment plants and distribution systems. The table also summarizes these findings in micromolar units for each class of species. For the settled water chlorinated at various pH values, the slope of the HAA6/TTHM ratio decrease with increasing pH, reflecting the fact that HAA6 concentrations decrease with increasing pH while TTHM formation increases with increasing pH. Note that the slope for the plant samples is consistent with the fact that the utilities chlorinate alum-coagulated settled water at pH values of 6.0-6.5, where HAA concentrations relative to THM concentrations tend to be greater.

Singer et al. (1995) researched the extent of DBP formation in North Carolina drinking waters with a desire to compare their findings to the DBP occurrence survey conducted by the Metropolitan Water District of Southern California (MWDSC) (Krasner et al. 1989). The former found that the mean and median DCAA and TCAA concentrations were almost as high as the corresponding chloroform concentrations. Overall HAA concentrations were about twice as high as TTHM concentrations (see Table 4.7).

Table 4.6 Summary of Linear Regression Relationships Between TTHM and HAA6 Concentrations

	n	*HAA6/TTHM* (*μg/μg*)	R^2	*HAA6/TTHM* (*μM/μM*)	R^2
pH 6.0	10	0.88	0.65	0.70	0.70
pH 6.5	11	0.69	0.66	0.57	0.64
pH 7.0	11	0.58	0.62	0.48	0.60
pH 7.5	11	0.52	0.61	0.44	0.60
pH 8.0	11	0.52	0.69	0.45	0.67
pH 8.5	11	0.41	0.60	0.35	0.59
Plant Samples@	43	0.76	0.78	0.68	0.79

@ Plant samples including filtered and finished water from the treatment plants and water samples collected from the distribution system

Figure 4.42 Correlation between TTHM and HAA6 concentrations for settled water chlorinated at pH 6

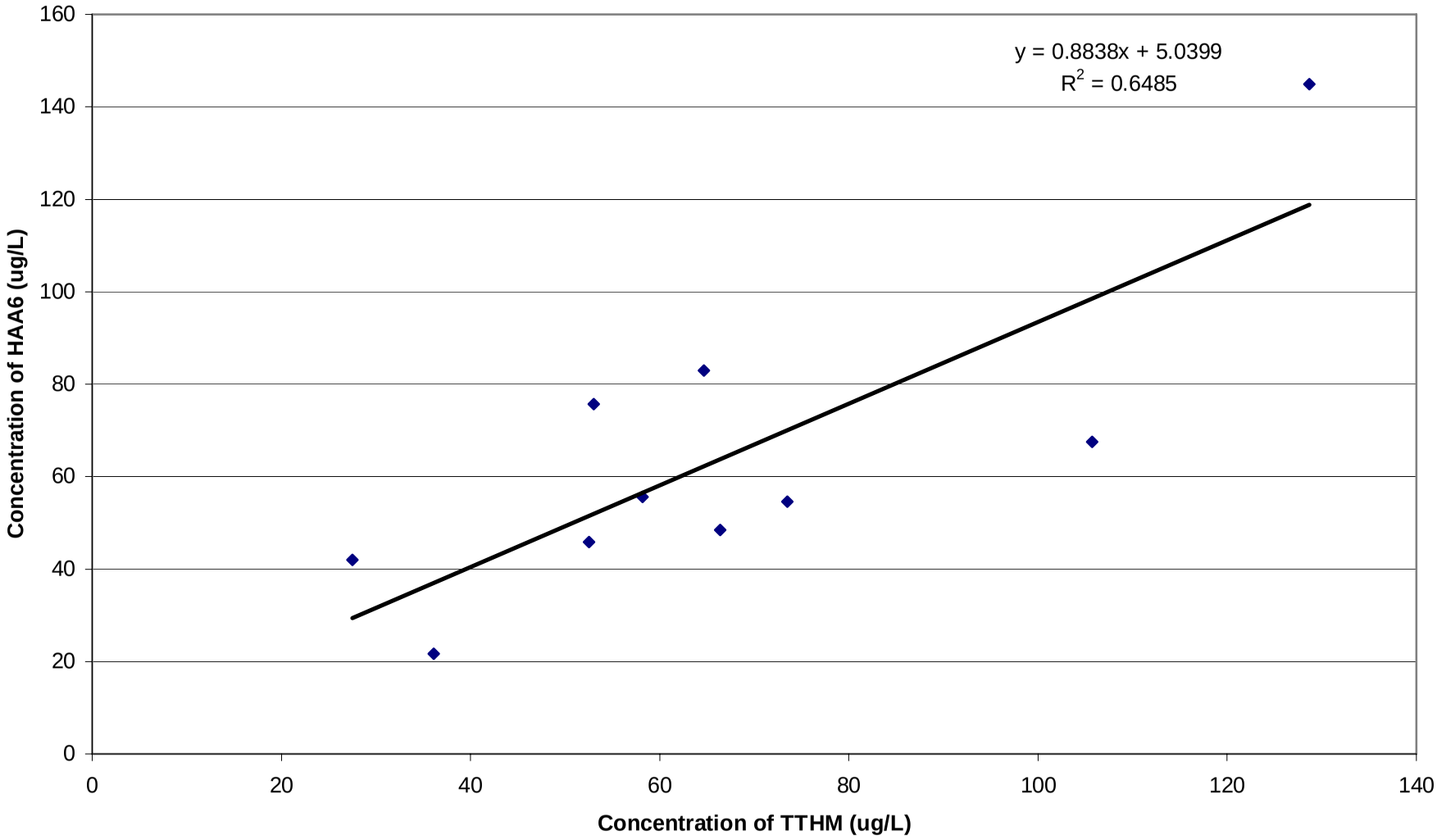


Figure 4.43 Correlation between TTHM and HAA6 concentrations for settled water chlorinated at pH 8

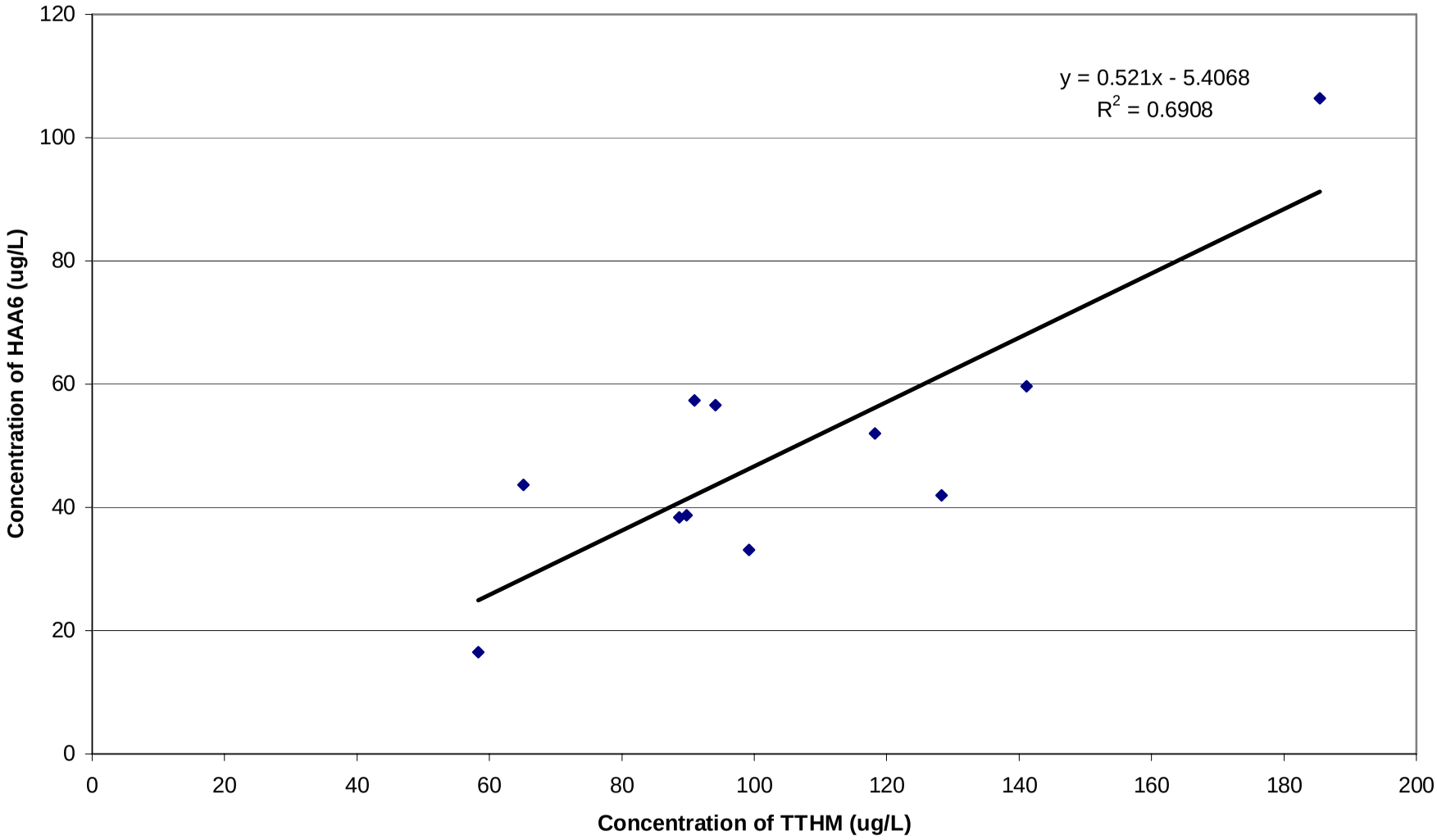


Figure 4.44 Correlation between TTHM and HAA6 concentrations for waters sampled in treatment plant and distribution system

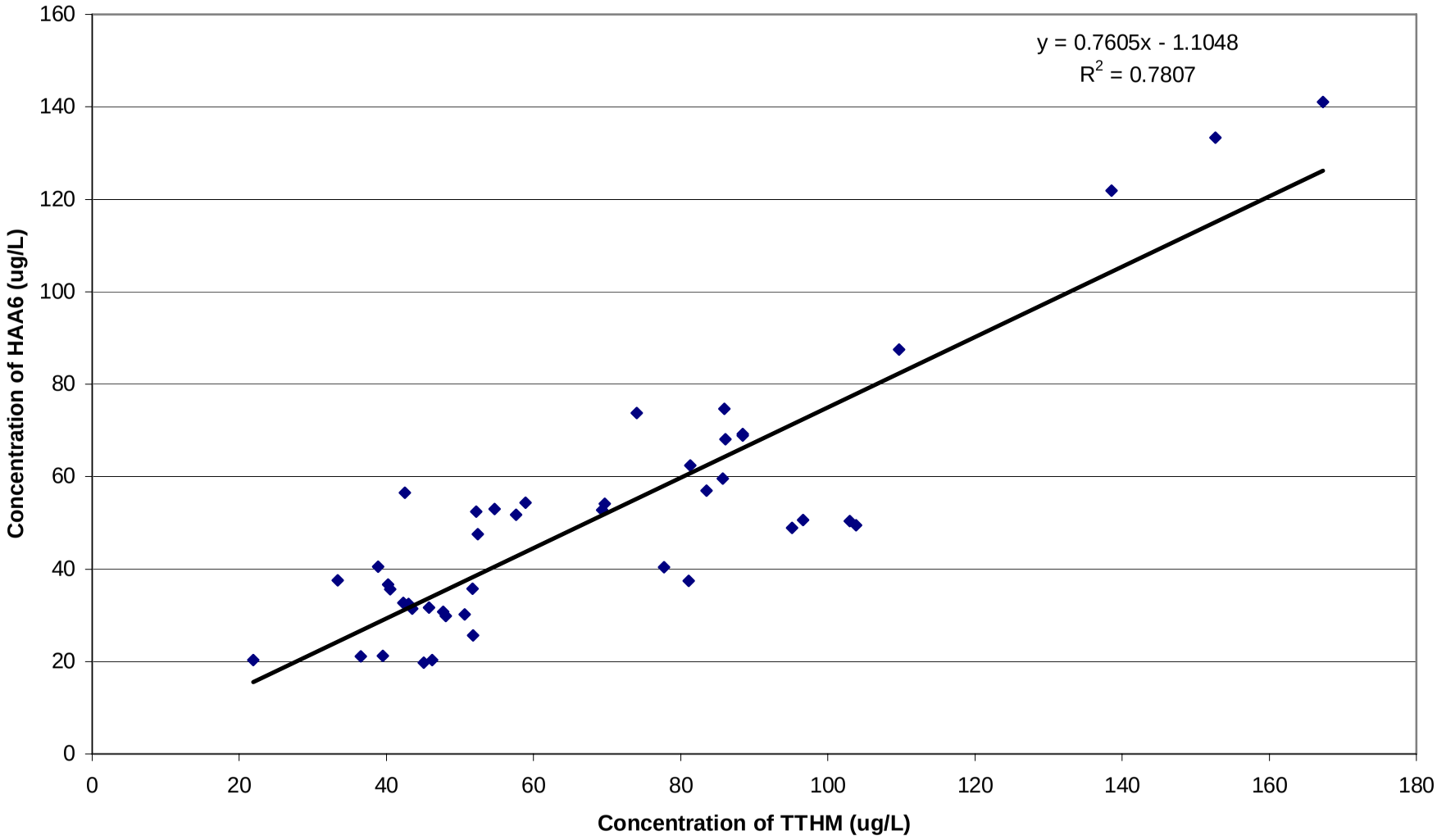


Table 4.7 Summary of DBP Concentrations in Finished Water and Distribution System
(adapted from Table 2, Singer et al. 1995)

	Finished water (n=24)	*Distribution system (n=42)*
median TTHM (μg/L)	35	46
median HAA4@ (μg/L)	64	81
HAA4/TTHM (μg/μg)	1.83	

@ Monochloroacetic acid, dichloroacetic acid, trichloroacetic acid, bromoacetic acid

These results were in sharp contrast to those reported by Krasner and co-workers (Krasner et al. 1989). In their MWDSC 35-city survey, it was observed that the median HAA5 concentration was only about half of the median TTHM concentration (see Table 4.8). Elevated pH values of chlorination and higher bromide concentrations that masked true overall HAA formation were cited as contributing to the relatively low HAA values in Krasner et al's work (Singer et al. 1995).

Table 4.8 Quarterly Median Values of DBPs in Drinking Water
(adapted from Table 4, Krasner et al. 1989)

	Spring 1988	*Summer 1988*	*Fall 1988*	*Winter 1989*
TTHM (μg/L)	34	44	40	30
HAA5@ (μg/L)	18	20	21	13
HAA5/TTHM	0.53	0.46	0.53	0.43

@ Monochloroacetic acid, dichloroacetic acid, trichloroacetic acid, bromoacetic acid, dibromoacetic acid

For the settled waters chlorinated at different pH values in this research, it was found that HAA6 concentrations were 88% and 52% of the TTHM concentration at pH 6 and pH 8, respectively. For the plant and distribution system samples, a value of 0.76 was observed for the HAA6/TTHM ratio(μg/μg). These numbers are intermediate between Singer et al.'s (1995) and Krasner et al.'s (1989) results.

Table 4.9 shows data for the average ratio of HAA6/TTHM concentrations for each system. Cary had the highest HAA6/TTHM ratio (0.90) while Fayetteville had the lowest ratio (0.44). The lowest value is comparable to Krasner et al.'s (1989) results; the highest value is only half of the HAA4/TTHM value reported by Singer et al. (1995).

Table 4.9 Average Ratios of HAA6/TTHM Concentrations for Each System

	HAA6/TTHM (μg/μg)	*HAA6/TTHM (μM/μM)*
OWASA	0.70	0.62
Cary	0.90	0.84
High Point	0.74	0.67
Durham	0.72	0.66
Greenville	0.81	0.72
Fayetteville	0.44	0.45

4.6.2 Correlation Between Chlorine Consumption and TTHM and HAA6 Formation

In a survey of THM formation in North Carolina drinking water (Singer et al. 1981), a significant correlation ($R^2 = 0.81$) was observed between the 7-day chlorine consumption of raw water and the corresponding terminal THM concentration. The terminal THM concentration was the concentration of THMs produced after seven days of storage under controlled temperature and pH conditions and with sufficient chlorine dosage to maintain a free chlorine residual after seven days. Accordingly, this terminal THM concentration can be considered as a surrogate of THM precursors in the water.

In another survey of DBPs in chlorinated North Carolina drinking waters (Singer et al. 1995), regression analysis was conducted between TTHM and HAA4 concentrations and chlorine consumption. The correlation coefficients (R^2) were 0.805 and 0.691, respectively. It was proposed that chlorine consumption is a fairly good parameter to predict DBP formation.

Figures 4.45 and 4.46 demonstrate the correlation between Cl_2 consumption and TTHM concentrations at pH 6 and pH 8, respectively, for the data collected in this research. Figures 4.47 and 4.48 illustrate the correlation between Cl_2 consumption and HAA6 concentrations at pH 6 and 8, respectively. The correlation coefficients range from 0.61 to 0.77, similar to those reported previously. However, for waters sampled from the treatment plants and distribution systems, no correlation was found between Cl_2 consumption and TTHM or between Cl_2 consumption and HAA6 (Figures 4.49 and 4.50).

Table 4.10 summarizes the regression analyses for Cl_2 consumption and TTHM concentration. A moderate association is shown, with correlation coefficients in the range 0.54 to 0.62 when concentrations are calculated on a weight basis. The R^2 values are slightly lower when concentrations are calculated on a molar basis. There is no clear trend in the slope of the association with changing pH values.

Table 4.10 Summary of Correlation Analyses Between Cl_2 Consumption and TTHM Concentration

	n	*TTHM/Cl_2 (μg/mg)*	*R^2*	*TTHM/Cl_2 (μM/mM)*	*R^2*
pH 6.0	11	36.1	0.61	29.1	0.53
pH 6.5	12	42.7	0.60	34.6	0.53
pH 7.0	12	44.1	0.56	35.7	0.48
pH 7.5	12	40.8	0.54	33.0	0.47
pH 8.0	12	38.9	0.62	31.8	0.54
pH 8.5	12	46.1	0.54	37.8	0.45
Plant Samples	34		0.05		0.04

Figure 4.45 Correlation between Cl2 demand and TTHM concentrations for settled water chlorinated at pH 6

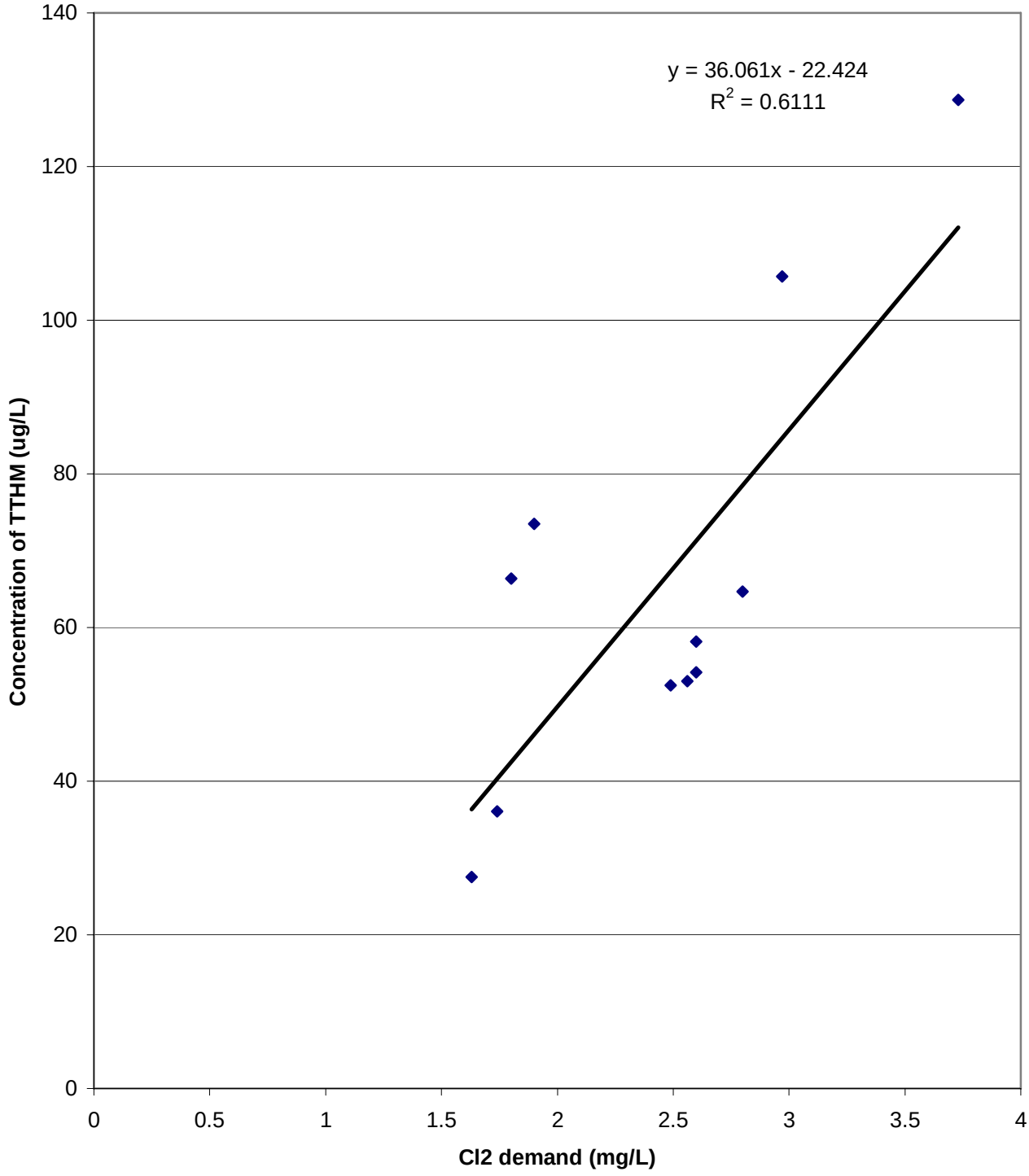


Figure 4.46 Correlation between Cl2 demand and TTHM concentrations for settled water chlorinated at pH 8

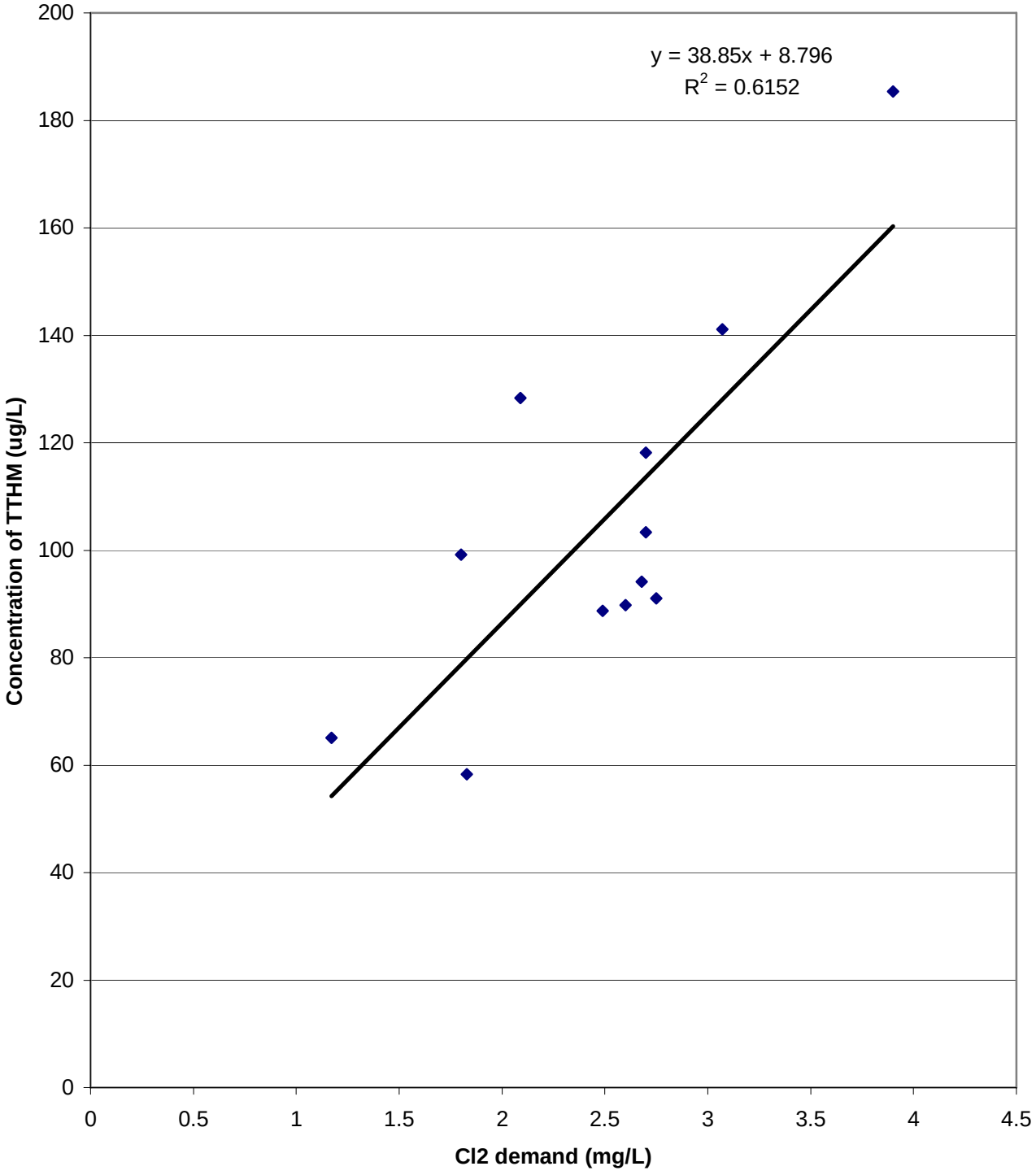


Figure 4.47 Correlation between Cl2 demand and HAA6 concentrations for settled water chlorinated at pH 6

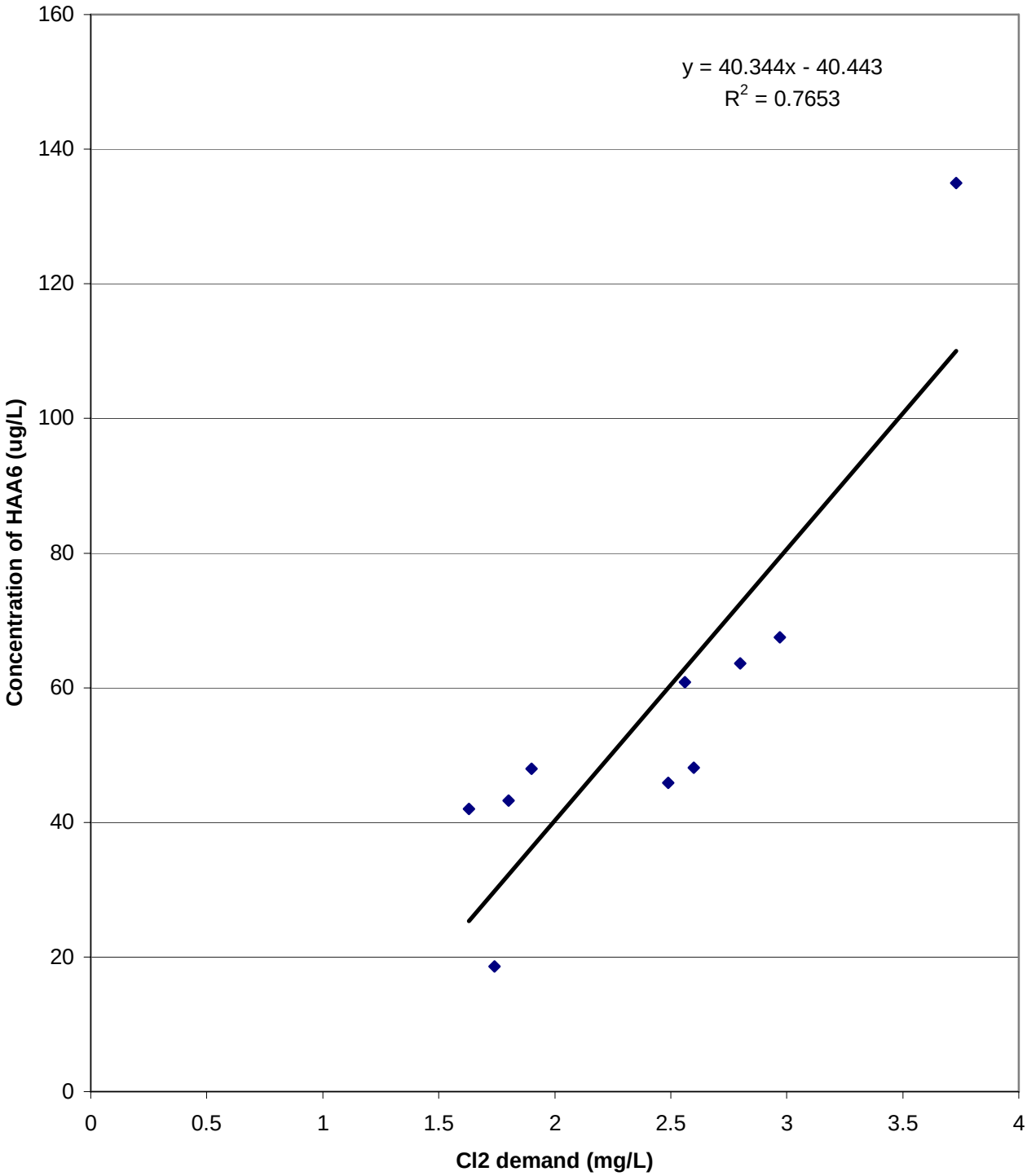


Figure 4.48 Correlation between Cl2 demand and HAA6 concentrations for settled water chlorinated at pH 8

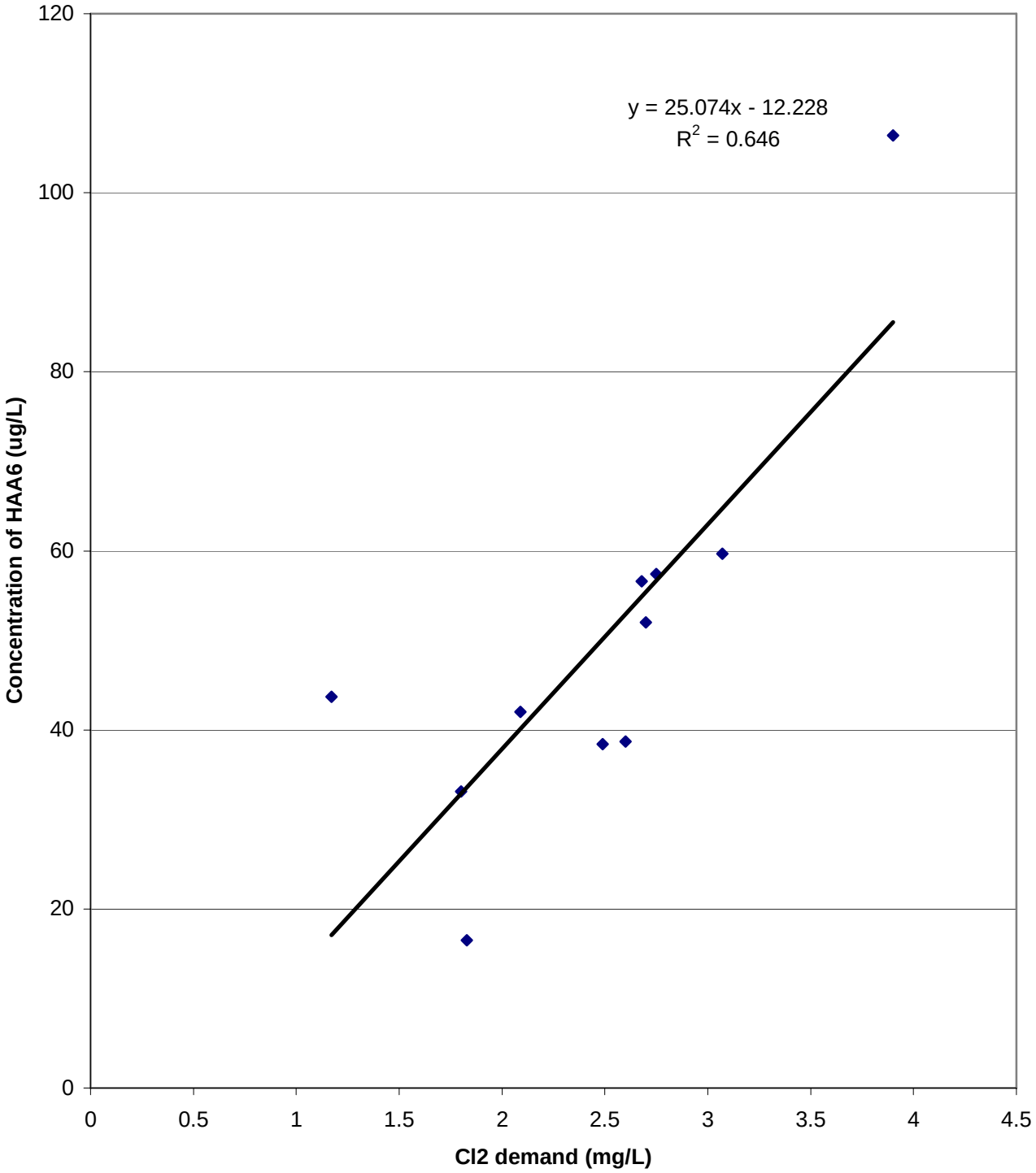


Figure 4.49 Correlation between Cl2 demand and TTHM concentration for waters sampled in treatment plant and distribution system

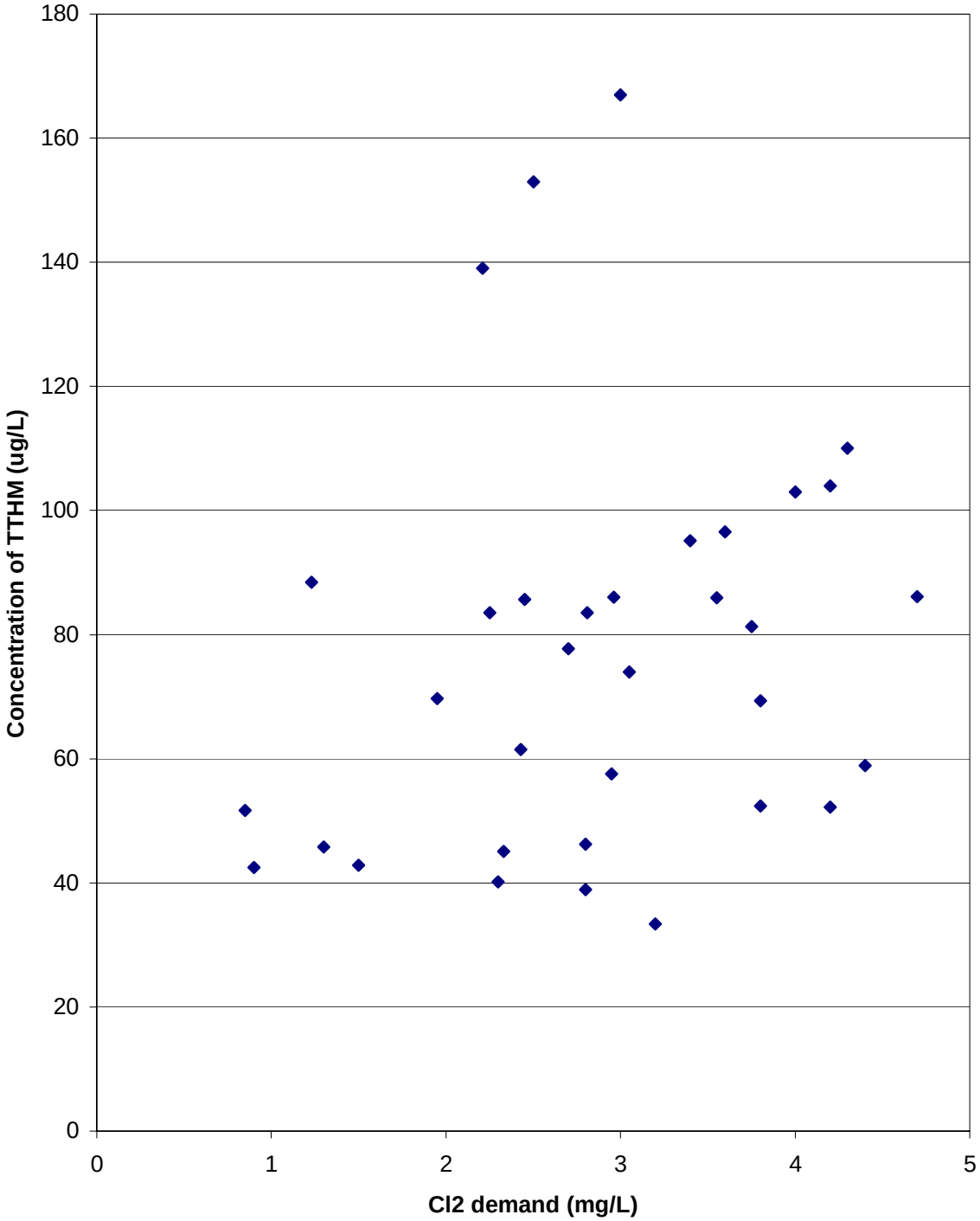


Figure 4.50 Correlation between Cl2 demand and HAA6 concentration for waters sampled in treatment plant and distribution system

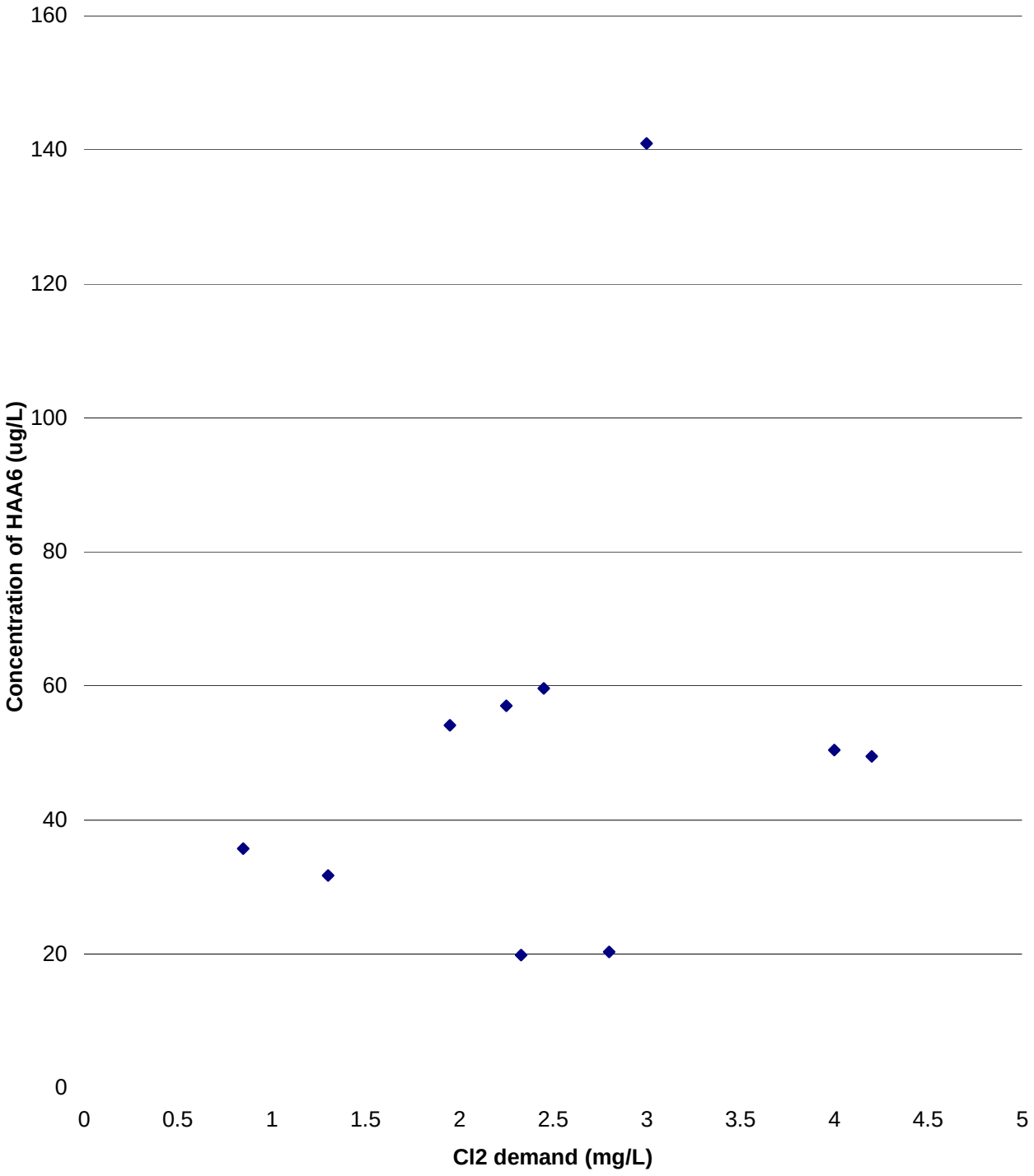


Table 4.11 summarizes the corresponding analysis conducted for Cl_2 consumption and HAA6 concentration. Compared to the TTHM case, the correlation coefficients are somewhat higher, showing a better association between Cl_2 consumption and HAA6 concentration. The slope of the correlation appears to decrease with increasing pH when concentrations are calculated on either a weight or a molar basis. Accordingly, there is a smaller percentage of consumed chlorine incorporated into HAA formation at higher pH.

These observations are in agreement with the results discussed earlier. Increasing pH will encourage chlorine substitution into THMs, therefore increasing THM formation. On the other hand, increasing pH will diminish chlorine substitution into HAAs, thus decreasing HAA formation. The theoretical basis for this phenomenon has been discussed in a previous section (Section 4.3)

Table 4.11 Summary of Correlation Analyses Between Cl_2 Consumption and HAA6 Concentration

	n	*HAA6/Cl_2 (μg/mg)*	*R^2*	*HAA6/Cl_2 (μM/mM)*	*R^2*
pH 6.0	10	40.3	0.77	24.1	0.72
pH 6.5	11	41.9	0.81	25.1	0.77
pH 7.0	11	40.8	0.88	24.1	0.83
pH 7.5	11	31.7	0.75	18.7	0.72
pH 8.0	11	25.1	0.65	14.7	0.63
pH 8.5	11	31.1	0.87	18.0	0.85
Plant Samples	30		0.006		0.005

4.6.3 Correlation Between TOC Concentration and TTHM/HAA6 Formation

It has been widely demonstrated that there is a linear relationship between THM formation and TOC concentration in samples chlorinated under controlled experimental conditions (Rook 1976; Babcock and Singer 1979; Fleischacker and Randtke 1983). THM concentrations in finished water and TOC concentrations in raw water have also been found to be well correlated (Symons et al. 1975). Accordingly, TOC has been suggested as a useful surrogate for THM precursor concentrations in finished water (Singer et al., 1981). Similar trends that increasing TOC concentrations will result in increasing by-product formation were observed for DCAA, TCAA and HAA6 (HAA5 plus BCAA) in a series of TOC-spiked samples prepared from agricultural drainage water (Krasner et al. 1996).

Tables 4.12 and 4.13 summarize the correlation analysis for TOC concentrations in settled water and TTHM or HAA6 formation from controlled laboratory chlorinations at different pH values. The data indicate that TOC does not provide for a very good overall correlation for THMs (R^2 = 0.25~0.39), but tends to be a better predictor of HAA6 (R^2 = 0.64~0.72). The reason for this may be due to the different nature of the NOM in the different waters, and other different water quality parameters, such as alkalinity, etc.

Table 4.12 Summary of Correlation Analyses Between TOC Concentration in Settled Water and TTHM Concentration

	n	*TTHM/C (μg/mg)*	R^2
pH 6.0	11	31.9	0.39
pH 6.5	12	27.2	0.25
pH 7.0	12	30.1	0.29
pH 7.5	12	30.4	0.25
pH 8.0	12	35.2	0.37
pH 8.5	12	33.0	0.30

Table 4.13 Summary of Correlation Analyses Between TOC Concentration in Settled Water and HAA6 Formation

	n	*TTHM/C (μg/mg)*	R^2
pH 6.0	10	43.2	0.72
pH 6.5	11	36.8	0.64
pH 7.0	11	34.0	0.69
pH 7.5	11	33.1	0.69
pH 8.0	11	30.4	
pH 8.5	11	26.3	0.67

4.7 IMPLICATIONS OF RESULTS FOR TTHM AND HAA6 CONTROL

In North Carolina, most utilities apply chlorine to settled water in which the pH has been depressed to 6.0-6.5 as a result of coagulation with alum. Increasing the pH of settled water prior to chlorination would be expected to lower HAA levels. However, the strategy of pH adjustment can only be utilized if both THMs and HAAs are controlled and disinfection effectiveness is not compromised. Adopting chloramination as a secondary disinfectant has been demonstrated in this research to be a successful method of controlling THM and HAA formation.

Table 4.14 summarizes the average concentration of TTHM and HAA6 in finished water and waters sampled from the distribution systems of the six treatment plants studied. The values reported for finished water are the average TTHM and HAA6 concentrations in the finished water during the two sampling trips. The values reported for the distribution systems are the average TTHM and HAA6 concentrations in waters sampled at the two distribution system compliance monitoring stations during the two sampling trips. The concentrations of TTHM and HAA6 for OWASA finished water in May are not available; therefore the values shown are the concentrations of THM and HAA6 in October only. Similarly, the values reported for HAA6 concentrations in the finished water and waters sampled in the distribution system at Fayetteville are actually the concentrations of HAA6 in September.

Table 4.14 Summary of the Average Concentrations of TTHM and HAA6 in Finished Water and Waters Sampled in the Distribution System

	TTHM Finished Water Average (μg/L)*	*TTHM Distribution System Average@ (μg/L)*	*HAA6 Finished Water Average (μg/L)*	*HAA6 Distribution System Average (μg/L)*
Cary	63.2	69.6	60.7	61.0
High Point	41.4	46.2	34.2	31.2
OWASA	39.5	69.0	21.2	46.2
Durham	73.5	97.0	46.6	63.8
Greenville	104.4	122.3	88.0	98.0
Fayetteville	53.9	94.1	20.3	50.0

* The average of the concentrations in finished water for each plant.
@ The average of the concentrations in waters sampled in the distribution system for each plant.

For TTHM concentrations in the finished water, one (Greenville, with 104 μg/L TTHMs) out of six plants exceeded the proposed 80 μg/L MCL for TTHM in the Stage I D/DBP rule. For TTHM concentrations in the distribution system, three (Durham with 97 μg/L, Greenville with 122 μg/L, and Fayetteville with 94 μg/L) out of six plants exceeded the proposed 80 μg/L MCL for TTHM.

For HAA6 concentrations in the finished water, one (Greenville, with 88 μg/L HAA6) out of six plants exceeded the proposed 60 μg/L MCL for HAA5 in the Stage I D/DBP rule. For HAA6 concentrations in the distribution system, three (Cary with 61 μg/L, Durham with 64 μg/L, and Greenville with 98 μg/L) out of six plants exceeded the proposed 60 μg/L MCL for HAA5.

For these utilities, the concentrations of TTHMs exceeded the proposed MCL to a much larger degree than the concentrations of HAA6. For example, Cary and Durham only had marginal excesses relative to the proposed MCL for HAA5. Therefore, it appears that these plants will have more problems in complying with the MCL for TTHMs than with the MCL for HAA5.

Among the treatment plants using free chlorine as both primary and secondary disinfectants, Greenville had the most serious problem in complying with the MCLs for both TTHMs and HAA5. Relatively high TOC concentrations seems to be the major cause of the elevated DBP formation. More rigorous precursor removal techniques, such as GAC adsorption or membrane filtration, or use of an alternative disinfectant to free chlorine, are recommended for this utility.

The concentrations of both TTHMs and HAA6 in Durham finished water were well below the proposed MCLs. However, the concentrations of TTHMs and HAA6 in the distribution system exceeded the proposed MCLs for both groups of DBPs because of their continued formation with increasing contact time. Accordingly, chloramination may be a good strategy for THM and HAA control. Ammonia can be added just before the water enters the distribution system. For Fayetteville, chloramination is also a control strategy that should be considered.

With the use of chloramines as a secondary disinfectant, both Cary and High Point were able to depress THM and HAA formation. The difference between Cary and High Point was that Cary applied ammonia just before water was pumped into the distribution system, while High Point applied ammonia after filtration, before clearwell storage. Accordingly, Cary was only able to marginally comply with the

proposed MCL for HAA5 in the distribution system while High Point was easily able to comply with the Stage I MCLs for both TTHM and HAA6. If Cary can apply ammonia earlier in the treatment plant without compromising disinfection, TTHM and HAA6 can be depressed even further.

It should be emphasized that Table 4.14 and the discussion that derives from it are based only on two sets of samples collected 3 to 5 months apart, and do not represent a full year's worth of monitoring results.

V. **CONCLUSIONS**

IMPACT OF CHLORAMINATION ON DBP FORMATION

For plants using free chlorine as a primary disinfectant and monochloramine as a secondary disinfectant, THMs and HAAs form in the plant while the water is in contact with free chlorine, but the addition of ammonia essentially stops subsequent THM and HAA formation. Both TCAA and DCAA formation stops after ammonia addition, in a manner similar to that observed for TTHMs.

However, in some cases involving ammonia addition to chlorinated filtered water, there is still a small continuing production of THMs and HAAs even after ammonia addition. This suggests that the chloramination reaction is not instantaneous and that there is still some slow formation of THMs and HAAs due to the presence of chlorine residual reacting with the highly reactive residual NOM species. For finished water, THM and HAA formation essentially stops after ammonia addition. This suggests that, in this case, the more reactive NOM precursors have already reacted by the time ammonia is added, and that the remaining NOM is relatively un-reactive to any residual free or combined chlorine.

IMPACTS OF PH ADJUSTMENT ON DBP FORMATION

TTHM concentrations consistently increase with increasing pH in the range 6-8.5. HAA6 formation, however, decreases as pH increases. TCAA formation decreases with increasing pH, but DCAA formation is relatively insensitive to changes in pH.

IMPLICATIONS FOR DRINKING WATER UTILITIES IN NORTH CAROLINA

In North Carolina, most utilities apply chlorine to settled water that has a pH of 6.0-6.5 as result of coagulation with alum. Hence, increasing the pH of the settled water prior to chlorine addition would be expected to lower HAA levels. In this research, however, THMs were found to increase with pH to a larger extent than HAAs decreased. Therefore, pH adjustment is not a recommended method for controlling both THMs and HAAs at the treatment plants investigated.

Chloramination can effectively control both THM and HAA formation as observed in the full-scale plants using monochloramine as a secondary disinfectant and in the laboratory simulations of chloramination. Application of ammonia immediately after the necessary contact time for primary disinfection with free chlorine should allow most of the plants to meet the proposed Stage I MCLs for TTHMs and HAA5.

For those plants with relatively high TOC concentrations (e.g. Greenville), chloramination will not be an effective strategy to enable them to comply with the proposed MCLs. Therefore, more rigorous precursor removal techniques, such as GAC adsorption or membrane filtration, or use of an alternative disinfectant to free chlorine, is recommended.

VI. REFERENCES

Amy, G.L., B.C. Alleman and C.B. Cluff. 1990. Removal of Dissolved Organic Matter by Nanofiltration. Jour. AWWA 116:1:200.

Amy, G.L., J.H. Greenfield and W.J. Cooper. 1990. Organic Halide Formation During Water Treatment Under Free Chlorine Versus Chloramination Conditions. Water Chlorination: Environmental Impact and Health Effects, Vol.6, Ann Arbor Science Publishers Inc., Ann Arbor, MI. 605.

Arber, R., M.A. Speed and F. Scully. 1985. Significant Findings Related to Formation of Chlorinated Organics in the Presence of Chloramines. Water Chlorination: Environmental Impact and Health Effects, Vol.5, Ann Arbor Science Publishers Inc., Ann Arbor, MI. 951.

Bellar, T.A., J.J. Lichtenberg and R.C. Kroner. 1974. The Occurance of Organohalides in Chlorinated Drinking Waters. Jour. AWWA, 66:12:703

Bererdi, M., R. Snyder, R.S. Waritz and K.R. Cooper. 1987. Monochloroacetic Acid Toxicity in the Mouse Associated With Blood-Brain Barrier Damage. Fundam. Appl. Toxicol. 9:469.

Bhat, H.K., M.F. Kanz, G.A. Campbell and G.A.S. Ansari. 1991. Ninety Day Toxicity Study of Chloroacetic Acids in Rats. Fundam. Appl. Toxicol. 17:240.

Bove, F. J. Fulcomer M. C. Klotz J. B. Esmart J. Dufficy E. M. and Zagraniski R. T. 1992. Report on Phase IV-A:Public Drinking Water Contamination and Birthweight, Fetal Deaths, and Birth Defects. A Cross-Sectional Study. Trenton, NJ: New Jersey Department of Health

Boyce, S.D. and J.F. Hornig 1979. Formation of Chloroform Form the Chlorination of Diketones and Polyhydroxybenzenes in Dilute Aqueous Solution. Water Chlorination: Environmental Impact and Health Effects, Vol.3, Ann Arbor Science Publishers Inc., Ann Arbor, MI. 131.

Boyce, S.D., S.V. Barefoot, D.R. Britton and J.F. Hornig 1983. Formation of Trihalomethanes From the Halogenation of 1,3-Dihydroxybezenes in Dilute Aqueous Solution: Synthesis of 2-13C-Resorcinol and Its Reaction With Chlorine and Bromine. Water Chlorination: Environmental Impact and Health Effects, Vol.4, Ann Arbor Science Publishers Inc., Ann Arbor, MI. 253.

Brodtmann, N.V. and P.J. Russo. 1979. The Use of Chloramines for Reduction of THMs and Disinfection of Drinking Water. Jour. AWWA 71:2:179.

Bull, R.J, I.M. Sanchez, M.A. Nelson, J.L. Larson, and A.J. Lansing. 1990. Liver Tumor Induction in B6C3F1 Mice by Dichloroacetate and Trichloroacetate. Toxicology 16:337.

Bull, R.J. and F. Kopfler. 1990. Review of Available Health Effects Data on Disinfection By-products. AWWA Research Foundation. Denver, Colorado

Carlson, M. and D. Hardy. 1998. Controlling DBPs With Monochloramine. Jour. AWWA 90:2:95.

Christman R.F. and M. Ghssemi. 1966. Chemical Nature of Organic Color in Water. Jour. AWWA, 58:6:723

Christman R.F., J.D. Johnson, F.K. Pfaender, D.L. Norwood, M.R. Webb, J.R. Hass and M.J. Bobenrieth.

1979. Chemical Identification of Aquatic Humic Chlorination Products. Water Chlorination: Environmental Impact and Health Effects

Cicmanec, J.L., L.W. Condie, G.R. Olson and Shin-Ru Wang. 1991. 90-Day Toxicity Study of Dichloroacetate in Dogs. Fundam. Appl. Toxicol. 17:376.

Cowman, G.A. and P.C. Singer. 1996. Effect of Bromide Ion on Haloacetic Acid Speciation Resulting Form Chlorination and Chloramination of Aquatic Humic Substances. Envir. Sci. and Tech. 30:1:16-24.

De Laat, J., N. Merlet and M. Dore. 1982. Chlorination of Organic Compounds: Chlorine Demand and Reactivity in Relationship to the Trihalomethane Formation. Water Research 16:1437.

DeAngelo, A.B., F.B. Daniel, J.A. Stober and G.R. Olson. 1991. The Carcinogenicity of Dichloroacetic Acid in the Male B6C3F1 Mouse. Fundam. Appl. Toxicol. 16:337.

Dunnick, J.K. and R.L. Melnick. 1993. Assessment of the Carcinogenic Potential of Chlorinated Water: Experimental Studies of Chlorine, Chloramine, and Trihalomethanes. Journal of the National Cancer Institute 85:10:817.

Dunnick, J.K., J.K. Haseman, H.S. Lilja and S. Wyand. 1985. Toxicity and Carcinogenicity of Chlorodibromomethane in Fischer 344/N Rats and B6C3F1 Mice. Fundam. Appl. Toxicol. 5:1128.

Edzwald, J.K., W.C. Becker and K.L. Wattier. 1985. Surrogate Parameters for Monitoring Organic Matter and Trihalomethane Precursors in Water Treatment. Jour. AWWA 77:4:122.

Fleischacker, S.J. and S.J. Randtke. 1983. Formation of Organic Chlorine in Public Water Supplies. Jour. AWWA 75:3:132.

Foster A.M., A.G. Prentice, J.A. Copplestone, R.A. Cartwright and C. Ricketts. 1997. The Distribution of Leukemia in Association With Domestic Water Quality in South West England. European Journal of Cancer Prevention 6:11.

Glaze, W.H. and R. Rawley. 1979. A Preliminary Survey of Trihalomethane Levels in Selected Texas Water Supplies. Jour. of AWWA 71:9:509.

Gurol, M.D., A. Wowk, S. Myers and I.H. Suffet. 1985. Kinetics and Mechanism of Haloform Formation From Trichloroacetone. Water Chlorination: Environmental Impact and Health Effects, Vol.4, Ann Arbor Science Publishers Inc., Ann Arbor, MI. 269-84.

Herren-Freund, S.L, M.A. Pereira, M.D. Khoury, and G. Olson. 1987. The Carcinogenicity of Trichloroethylene and Its Metabolites, Trichloroacetic Acid and Dichloroacetic Acid. Toxicol. Appl. Pharmacol. 63:341.

Hubel, R.E. and J.K. Edzwald. Removing Trihalomethane Precursors by Coagulation. Jour. AWWA 79:7:98.

Hundt, T.R. and C.R. O'Melia. 1988. Aluminum-Fulvic Interactions: Mechanisms and Applications. Jour. AWWA 80:4:176.

Jensen, J.N. et al. 1985. Characterization of the Reaction Between Monochloramine and Isolated Aquatic Fulvic Acid. Water Chlorination: Environmental Impact and Health Effects, Vol.5, Ann Arbor Science Publishers Inc., Ann Arbor, MI. 939.

Johnson, J.D. and J.N. Jensen. 1986. THM and TOX Formation: Routes, Rates, and Precursors. Jour. AWWA 78:4:156.

Kabler, P.W. 1953. Relative Resistance of Coliform Organisms and Enteric Pathogens in the Disinfection of Water with Chlorine. Jour. AWWA 43:553

Katz, R.C., N. Tai, R.M. Diener, R.F. McConnell and D.E. Semonick. 1981. Dichloroacetate, Sodium: 3-Month Oral Toxicity Studies in Rats and Dogs. Toxicol. Appl. Pharmacol. 57:273.

Kavanaugh, M.C., A.R. Trussell, J. Cromer and R.R. Trussell. 1980. An Empirical Kinetic Model of Trihalomethane Formation: Applications to Meet the Proposed THM Standard. Jour. AWWA 72:10:578.

King W.D. and L.D. Marrett. 1996. Case-Control Study of Bladder Cancer and Chlorination By-Products in Treated Water (Ontario, Canada). Cancer Causes and Control 7: 596.

Kramer, M.D., C.F. Lynch, P. Isacson and J.W. Hanson. 1992. The Association of Waterborne Chloroform With Intrauterine Growth Retardation. Epidemiology 3:5:407.

Krasner, S.W., M.J. McGuire, J.G. Jacangelo, N.L. Patania, K.M. Reagan, E.M. Aieta. 1989. The Occurrence of Disinfection By-products in US Drinking Water. Jour. AWWA 81:8:41.

Krasner, S.W., M.J. Sclimenti, R. Chinn, Z.K. Chowdhury and D.M. Owen. 1996. The Impact of TOC and Bromide on Chlorination By-Product Formation. Disinfection By-Product in Water Treatment by CRC Press, Inc. 59.

Lange, A.L. and E. Kawczynski. 1978. Controlling Organics: The Contra Costa County Water District Experience. Jour. AWWA 70:12:653.

Lieu, N.I., R.L. Worlfe and E.G. Means. 1993. Optimizing Chloramine Disinfection for the Control of Nitrification. Jour. AWWA 85:2:84.

Linder, R.E., G.R. Klinefelter, L.F. Strader, J.D. Suarez and C.J. Dyer. 1994. Acute Spermatogenic Effects of Bromoacetic Acids. Fundam. Appl. Toxicol. 22:422.

Luong, T.V., C.J. Peters and R. Perry. 1982. Influence of Bromide and Ammonia Upon the Formation of Trihalomethanes Under Water-Treatment Conditions. Envir. Sci. and Technol. 16:473.

McGuire M.J. 1989. Preparing for the Disinfection By-products Rule: A Water Industry Status Report. Jour. AWWA 81:8:35.

Miller, J.W. and P.C. Uden. 1983. Characterization of Nonvolatile Aqueous Chlorination Products of Humic Substances. Envir. Sci. And Technol. 17:150-7.

Mitcham, R.P., M.W. Shelley and C.M. Wheadon. 1983. Free Chlorine Versus Ammonia-Chlorine: Disinfection, Trihalomethane Formation, and Zooplankton Removal. Jour.AWWA 75:4:196.

Morris, C.J. and B. Baum. 1977. Precursors and Mechanisms of Haloform Formation in the Chlorination of Water Supplies. Water Chlorination: Environmental Impact and Health Effects, Vol.2, Ann Arbor Science Publishers Inc., Ann Arbor, MI. 29-47.

Morris, J.C. 1966. The Acid Ionization Constant of HOCl From 5 to 35°C. J. Phys. Chem. 70:3798.

Morris, J.C. 1975. Aspects of the Quantitative Assessment of Germicidal Efficiency. Disinfection: Water and Waste Water. Ann Arbor Science Publishers Inc., Ann Arbor, MI. 21-33.

Morris, J.C. The Chemistry of Aqueous Chlorine in Relation to Water Chlorination. 1977. Water Chlorination: Environmental Impact and Health Effects, Vol.2, Ann Arbor Science Publishers Inc., Ann Arbor, MI. 1.

Morris, R.D., A.M. Audet, I.F. Angelillo et al. 1992. Chlorination, Chlorination by-Products, and Cancer: A Meta-Analysis. American Journal of Public Health 82: 955.

NCI. 1976. Report on The Carcinogenesis Bioassay of Chloroform. National Cancer Institute, Bethesda, MD, NTIS PB-264-0188 (Government Report).

Ngan, F. and M. Toofan. 1991. Modification of Preparation of Diazomethane for Methyl Esterification of Environmental Samples Analysis by Gas Chromatography. Journ. Of Chromatographic Science

Norman, T.S., L.L. Harms and R.W. Looyenga. 1980. The Use of Chloramines to Prevent Trihalomethane Formation in Huron, S.D., Drinking Water. Jour. AWWA 72:3:176.

Norton, C.D. and M.W. LeChevallier. 1997. Chloramination: Its Effect on Distribution System Water Quality. Jour. AWWA 89:7:66.

Norwood, D.L., J.D. Johnson, R.F. Christman and D.S. Millington. Chlorination Products From Aquatic Humic Material At Neutral pH. Water Chlorination: Environmental Impact and Health Effects, Vol.4, Ann Arbor Science Publishers Inc., Ann Arbor, MI. 191-200.

Norwood, N.L., J.D. Johnson and R.F. Christman. 1980. Reaction of Chlorine With Selected Aromatic Models of Aquatic Humic Material. Envir. Sci. and Technol. 14:2:187.

NTP. 1986. Toxicology and Carcinogenesis Studies of Bromodichloromethane in F344/N Rats and B6C3F1 Mice. National Toxicology Program, US Dept. of Health and Human Svs. TRS No. 321, NIH Publ. No. 88-2537, CAS. NO. 75-27-4.

NTP. 1989. Toxicology and Carcinogenesis Studies of Trihalomethane (Bromoform) in F344/N Rats and B6C3F1 Mice. National Toxicology Program, US Dept. of Health and Human Svs. TRS No. 350, NIH Publ. No. 89-2805, CAS. NO. 75-25-2 (Government Report).

Oliver, B.G. 1978. Chlorinated Non-Volatile Organics Produced by the Reaction of Chlorine With Humic Materials. Can. Res. 11:6:21.

Oliver, B.G. 1980. Effect of Temperature, pH and Bromide Concentration on the Trihalomethane Reaction of Chlorine With Aquatic Humic Material. Water Chlorination: Environmental Impact and Health Effects, Vol.3, Ann Arbor Science Publishers Inc., Ann Arbor, MI. 141-9.

Owen, D.M., G.L. Amy, Z.K. Chowdhury, R. Paode, G. McCoy and K. Viscosil. 1995. NOM Characterization and Treatability. Jour. AWWA

Pontius, F.W. D-DBP Rule to Set Tight Standards. 1993. Jour. AWWA 85:11:22

Pontius, F.W. Reg-Neg Process Draws to a Close. 1993. Jour. AWWA 85:9:18.

Pontius, F.W. Strengthening the Surface Water Treatment Rule. 1993. Jour. AWWA 85:12:20.

Pontius, F.W. Inside the Information Collection Rule. 1996. Jour. AWWA 88:8:16.

Pontius, F.W. An Update of the Federal Regs. 1997. Jour. AWWA 89:3:36.

Pontius, F.W. Future Directions in Water Quality Regulations. 1997. Jour. AWWA 89:3:40.

Pourmoghaddas, H., A.A. Stevens, R.N. Kinman, R.C. Dressman, L.A. Moore and J.C. Ireland. 1993. Effect of Bromide Ion on Formation of HAAs During Chlorination. Jour. AWWA 85:1:82-87.

Rav-Acha, C., A. Serri, E. Choshen and B. Limoni. 1984. Disinfection of Drinking Water Rich in Bromide With Chlorine and Chlorine Dioxide, While Minimizing the Formation of Undesirable By-Products. Water Science and Technology 17:611.

Rebhun, M.L., J. Heller-Grosman, D. Manka, Kimel and B. Limoni. 1990. Trihalomethane Formation and Distribution in Bromide-Rich and Ammonia-Containing Lake Water. Water Chlorination: Environmental Impact and Health Effects, Vol.6, Ann Arbor Science Publishers Inc., Ann Arbor, MI. 665.

Reckhow, D.A. and P.C. Singer. 1985. Mechanisms of Organic Halide Formation During Fulvic Acid Chlorination and Implications With Respect to Preozonation. Water Chlorination: Environmental Impact and Health Effects, Vol.5, Ann Arbor Science Publishers Inc., Ann Arbor, MI. 1229.

Reckhow, D.A. and P.C. Singer. 1984. The Removal of Organic Halide Precursors by Preozonation and Alum Coagulation. Jour. AWWA 76:4:151.

Reckhow, D.A. and P.C. Singer. 1990. Chlorination of Humic Materials: Byproduct Formation and Chemical Interpretations. Envir. Sci. and Technol. 24:11:1655-64.

Reckhow, D.A., J.T. Tobiason and M.S. Switzenbaum et al. 1992. Control of Disinfection By-Products in Drinking Water. Proc., Annu. Conf., American Water Works Association, Denver, Colorado.

Richardson, S.D., A.D. Thruston and T.W. Collette et al. 1994. Multispectral Identification of Chlorine Dioxide Disinfection By-Products in Drinking Water. Envir. Sci. and Technol. 28:4:592.

Roe, F.J.C., A.K. Palmer, A.N. Worden and N.J. Van Abbe. 1979. Safety Evaluation of Toothpaste Containing Chloroform: I. Long-Term Studies in Mice. Jour. Environ. Path. Toxicol. 2:799.

Rogers, E.H., J.E. Schmid, A.M. Richard and E.S. Hunter. 1995. Effects of Haloacetic Acid Drinking Water Contaminants in Whole Embryo Culture. Teratology 51:3:195.

Rook, J.J. 1977. Chlorination Reactions of Fulvic Acids in Natural Waters. Envir. Sci. and Technol.

Rook, J.J. 1974. Formation of Haloforms During Chlorination of Natural Waters. Water Treat. Exam. 23:2:234-43.

Rook, J.J. 1976. Haloforms in Drinking Water. Jour. AWWA 68:3:168-72.

Rook J.J. 1979. Possible Pathways for the Formation of Chlorinated Degradation Products During Chlorination of Humic Acid and Resorcinol. Water Chlorination: Environmental Impact and Health Effects, Vol.3, Ann Arbor Science Publishers Inc., Ann Arbor, MI. 85-98.

Ruff, F. and I.G. Csizmadia. 1994. Organic Reactions: Equiblibria, Kinetics and Mechanism. Elsevier.

Savitz, D.A., K.W. Andrews and L.M. Pastore. 1995. Drinking Water and Pregnancy Outcome in Central North Carolina: Source, Amount, and Trihalomethane Levels. Environmental Health Perspectives 103:6:592.

Schnitzer, M., and S. U. Khan. 1972. Chemical Structure of Humic Substances. Humic Substances in the Environment. Marcel Dekker, New York. 50.

Semmens, M.J., G.E. Norgaard, G. Hohenstein and A.B. Staples. 1986a. Influence of pH on the Removal of Organics by Granular Activated Carbon. Jour. AWWA 78:5:89.

Semmens, M.J., A.B. Staples, G. Hohenstein and G.E. Norgaard. 1986b. Influence of Coagulation on Removal of Organics by Granular Activated Carbon. Jour. AWWA 78:8:80.

Siddiqui, M.S. and G.L. Amy. 1993. Factors Affecting DBP Formation During Ozone-Bromide Reactions. Jour. AWWA 85:1:63.

Simmon V.F. and Tardiff R.G. 1978. Water Chlorination: Environmental Impact and Health Effects; Volume 2, Ann Arbor Science, MI. 417-31.

Singer, P.C. 1988. An Evaluation of Alternative Oxidant and Disinfectant Treatment Strategies for Controlling Trihalomethane Formation in Drinking Water. Cincinnati, Ohio, Water Engineering Research Laboratory, Office of Research and Development, USEPA.

Singer P.C., and S.D. Chang. 1989. Correlations Between Trihalomethanes and Total Organic Halides Formed During Drinking Water Treatment. Jour. AWWA 81:8:61.

Singer, P.C., J.J. Barry, G.M. Palen and A.E. Scrivner. 1981. Trihalomethane Formation in North Carolina Drinking Waters. Jour. AWWA 73:8:392.

Singer, P.C., A. Obolensky and A. Greiner. 1995. DBPs in Chlorinated North Carolina Drinking Waters. Jour. of AWWA 87:10:83-91.

Smith, M.E., G.A. Cowman and P.C. Singer. 1993. The Impact of Ozonation and Coagulation on DBP Formation in Raw Waters. Proc., Annu. Conf., American Water Works Association, Denver, Colorado.

Speed M.A. et al. 1987. Treatment Alternatives for Controlling Chlorinated Organic Contaminants in Drinking Water. [EPA/60012-87/011]. U.S. Environmental Protection Agency, Washington, DC.

Stacpoole, P.W. 1989. The Pharmacology of Dichloroacetate. Metabolism 11:1124.

Standard Methods for the Examination of Water and Waste Water. 1995. 19th Edition. APHA, AWWA and WPCF. Washington, DC

Stevens A.A., L.A. Moore and R.J. Miltner. 1989. Formation and Control of Non-Trihalomethane Disinfection By-Products. Jour. AWWA 81:8:54.

Stevens, A.A. et al. 1989. Chlorinated Humic Acid Mixtures Establish Criteria for Detection of Disinfection By-products in Drinking Water. Aquatic Humic Substances. Suffet I.H. and MacCarthy P. editors. adv. in Chem. Ser. 219. ACS, Washington, D.C.

Symons, J.M., T.A. Bellar, J.K. Carswell, J. DeMarco, K.L. Kropp, G.G. Robeck, D.R. Seeger, C.J. Slocum, B.L. Smith and A.A. Stevens. 1975. National Organics Reconnaissance Survey for Halogenated Organics. Journ. AWWA. 67:11:634

Symons, J.M., A.A. Stevens, R.M. Clark, E.E. Geldreich, O.T.Jr. Love and J. DeMarco. 1993. Measurement of THM and Precursor Concentration Revisited: the Effect of Bromide Ion. Jour. AWWA 85:1:51.

Topudurti, K.V. and C.N. Haas. 1989. Chloroform Formation by the Transfer of Active Chlorine Form Monochloramine to Phloroacetophenone. Water Chlorination: Environmental Impact and Health Effects, Vol.6, Ann Arbor Science Publishers Inc., Ann Arbor, MI. 649.

Toth, G.P., K.C. Kelty, E.L. George, E.J. Read and M.K. Smith. 1992. Adverse Male Reproductive Effects Following Subchronic Exposure of Rats to Sodium Dichloroacetate. Fundam. Appl. Toxicol. 19:57.

US Environmental Protection Agency. 1979. National Interim Primary Drinking Water Regulations; Control of Trihalomethanes in Drinking Water. Federal Register, 44(231; Nov 29), 68624

US Environmental Protection Agency. 1994. National Primary Drinking Water Regulations; Disinfectants/Disinfection by-products; Proposed Rule. Federal Register, 59(145, Jul 29), 38667

Wachter, J.K. and J.B. Andelman. 1984. Organohalide Formation on Chlorination of Algal Extracellular Products. Envir. Sci. and Technol. 18:111:811.

Weinberg, H.S., W.H. Glaze, S.K. Krasner and M.J. Sclimenti. 1993. Formation and Removal of Aldehydes in Plants That Use Ozone. Jour. AWWA 85:5:72.

Werdehoff, K. S. and Singer P. C. 1987. Chlorine Dioxide Effects on THMFP, TOXFP, and the Formation of Inorganic By-Products. Jour. AWWA 79:9:107.

White, G.C. 1992. Handbook of Chlorination and Alternative Disinfectants. Van Nostrand Reinhold, New York.

Wolfe, R.L., N.I. Lieu, G. Izaguirre and E.G. Means. 1990. Ammonia-Oxidizing Bacteria in a Chloraminated Distribution System: Seasonal Occurrence, Distribution, and Disinfection Resistance. Appl. Envir. Microbiol. 56:2:451.

LIST OF ABBREVIATIONS

AOPs	Advanced Oxidation Processes
BAT	Best Available Technology
BCAA	Bromochloroacetic Acid
BDCAA	Bromodichloroacetic Acid
BDOC	Biodegradable Organic Carbon
CT	Concentration × Time
D	Disinfectant
DBAA	Dibromoacetic Acid
DCAA	Dichloroacetic Acid
DBCAA	Dibromochloroacetic Acid
DBPs	Disinfection By-products
DOC	Dissolved Organic Carbon
DPD	N,N-diethyl-1,4-phenylenediamine
EBCT	Empty Bed Contact Time
ECD	Electron Capture Detector
ESWTR	Enhanced Surface Water Treatment Rule
GAC	Granular Activated Carbon
GC	Gas Chromatography
HAAs	Haloacetic Acids
ICR	Information Collection Rule
IS	Internal Standard
MBAA	Bromoacetic Acid
MCAA	Chloroacetic Acid

MCL	Maximum Contaminant Level
MCLG	Maximum Contaminant Level Goal
MtBE	Methyl Tertiary-butyl Ether
NOM	Natural Organic Matter
NPTOX	Non-purgeable Total Organic Halide
OWASA	Orange Water and Sewer Authority
PAC	Powdered Activated Carbon
POX	Purgeable Organic Halide
Rt	Retention Time
RRI	Relative Retention Index
RRt	Relative Retention Time
SDWA	Safe Drinking Water Act
TBAA	Tribromoacetic Acid
TCAA	Trichloroacetic Acid
THAA	Total Haloacetic Acid
THMs	Trihalomethanes
TTHM	Total Trihalomethanes
TOC	Total Organic Carbon
TOX	Total Organic Halogen
USEPA	U.S. Environmental Protection Agency

APPENDICES

APPENDIX A: Sampling Flowcharts

Figure A.1 Sampling flowchart for OWASA, NC (May 6, 1997)

OWASA, 5/06/97, 4:00 pm:

Temperature: 19.1°C

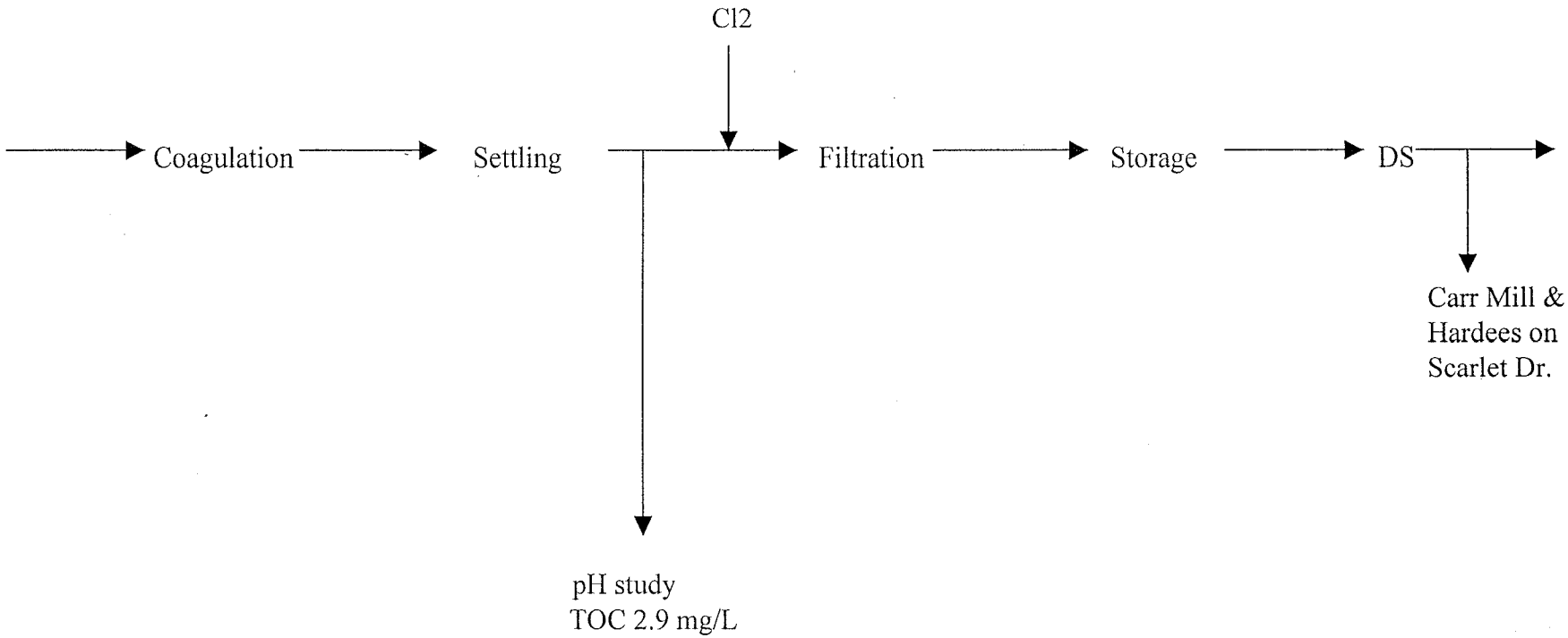


Figure A. 2 Sampling flowchart for OWASA, NC (October 3, 1997)

OWASA, 10/03/97, 11:30 am:

Temperature: 21.4°C

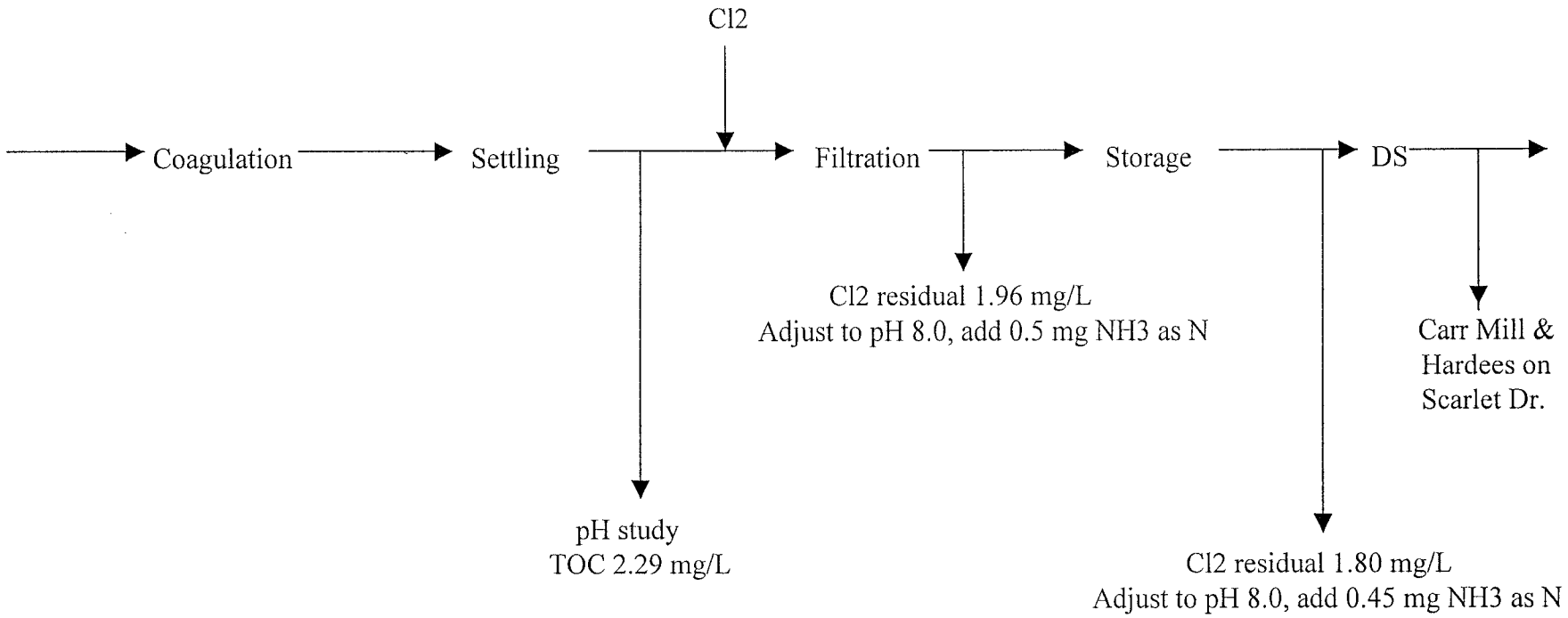


Figure A. 3 Sampling flowchart for Cary, NC (May 29, 1997)

Cary, 5/29/97, 11:00 am:
Temperature: 22°C

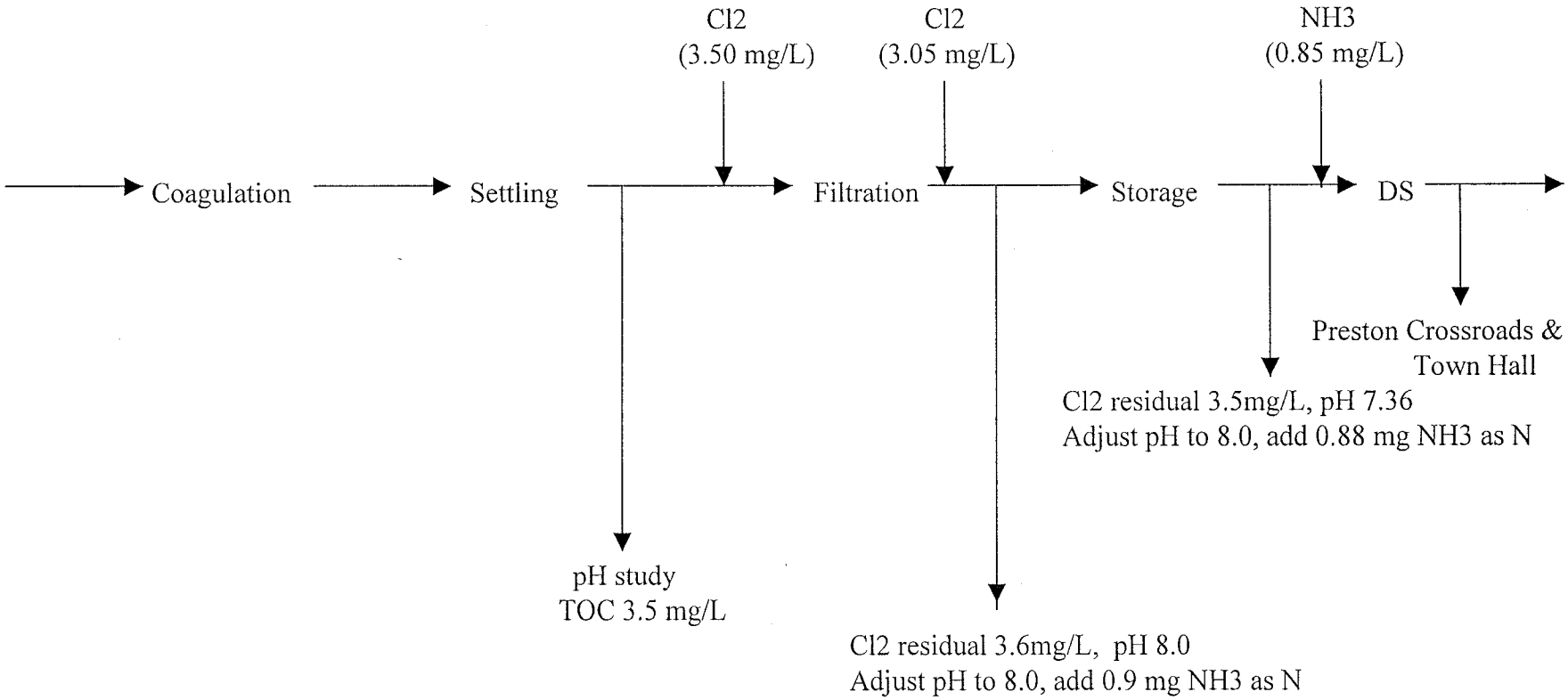


Figure A. 4 Sampling flowchart for Cary, NC (October 14, 1997)

Cary, 10/14/97, 10:30 am:
Temperature: 24°C

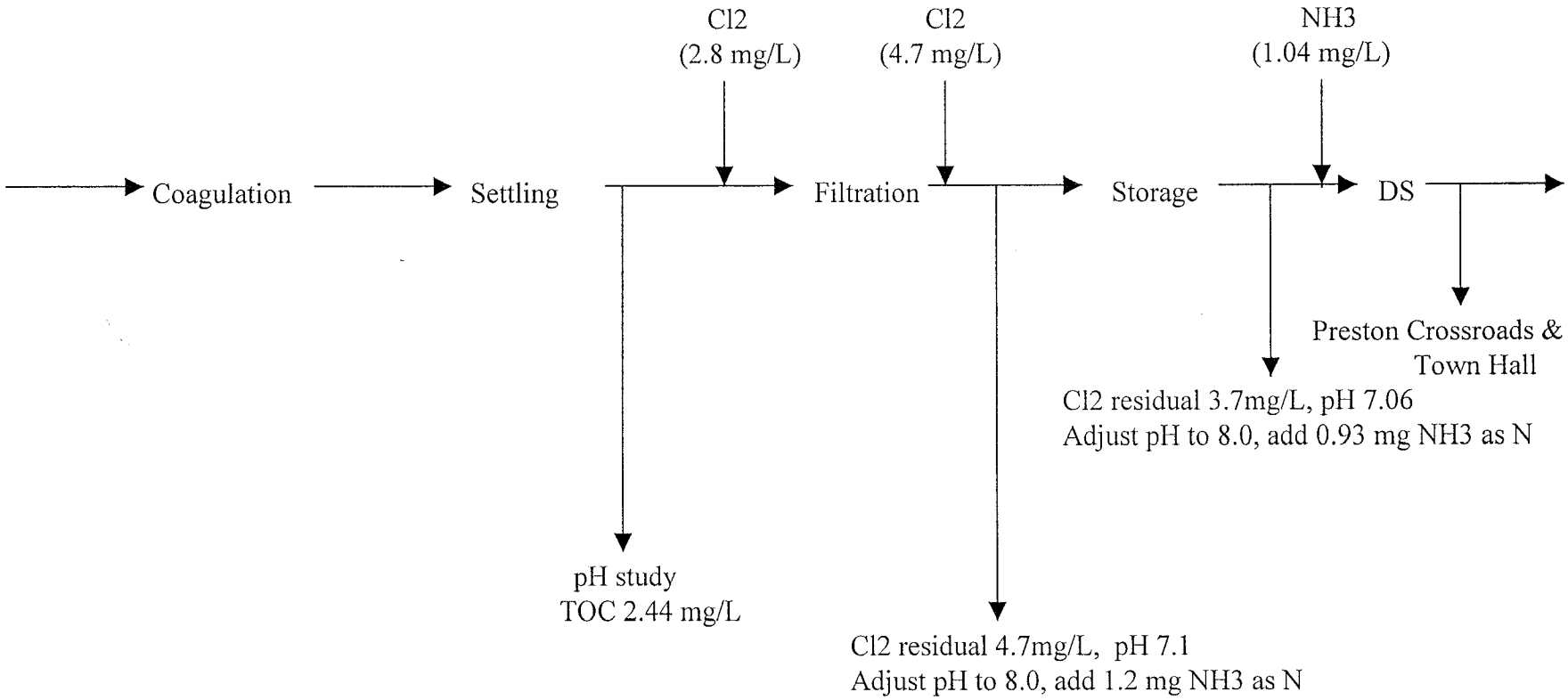


Figure A. 5 Sampling flowchart for High Point, NC (June 13, 1997)

High Point, 6/13/97, 11:55 am:
Temperature: 21.2°C

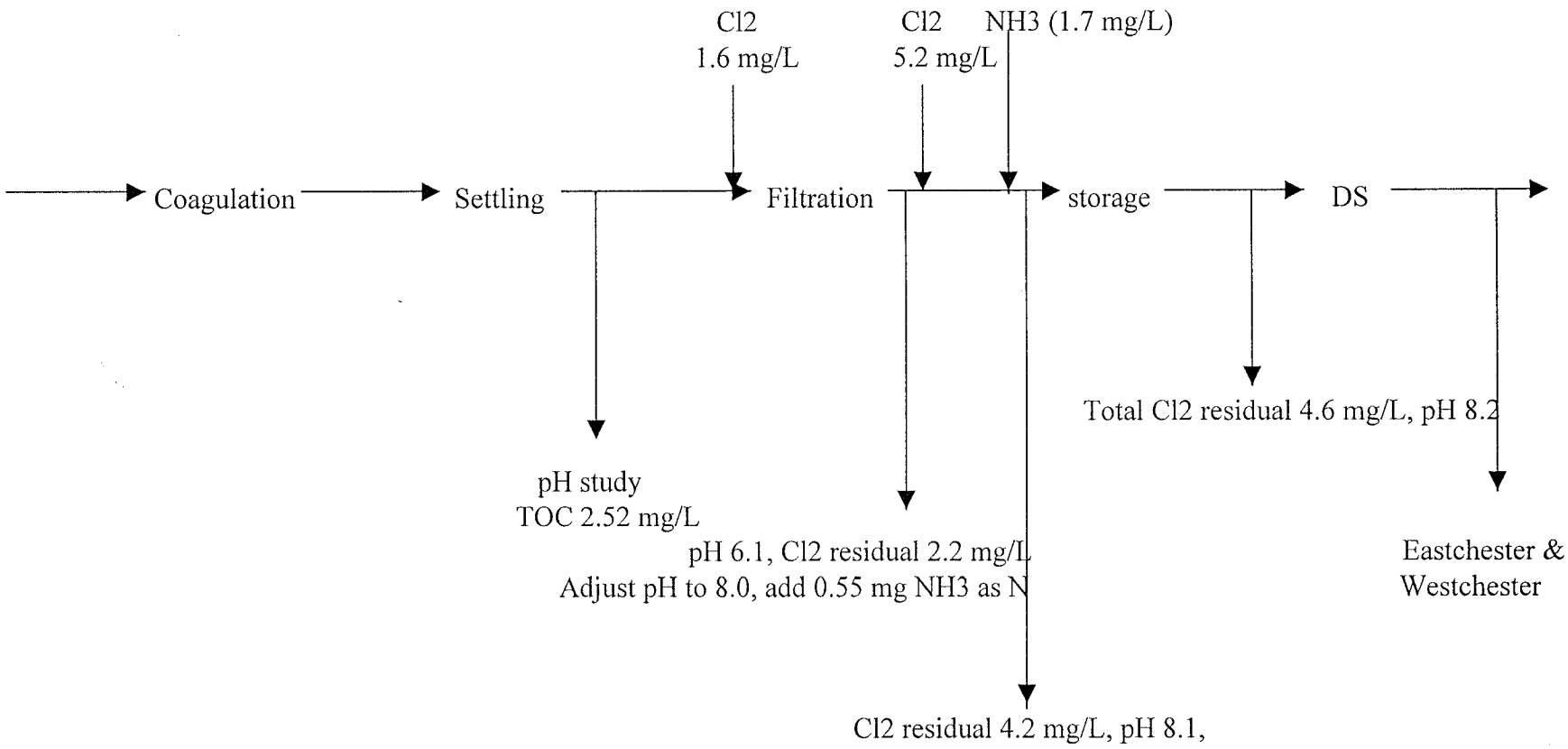


Figure A. 6 Sampling flowchart for High Point, NC (October 29, 1997)

High Point, 10/29/97, 11:00 am:
Temperature: 17.0°C

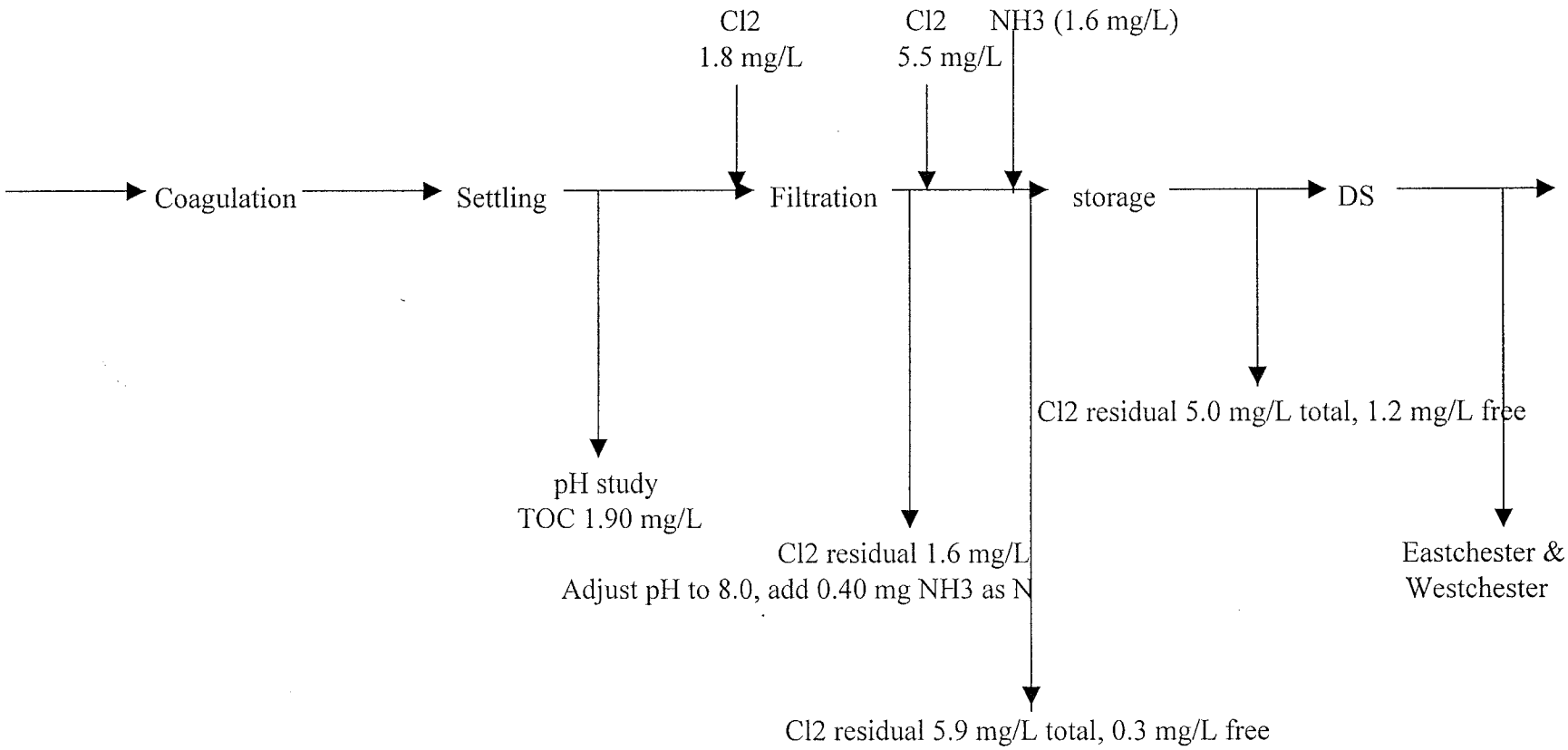


Figure A. 7 Sampling flowchart for Durham, NC (September 4, 1997)

Durham, 9/04/97, 10:00 am:
Temperature: 26.5°C

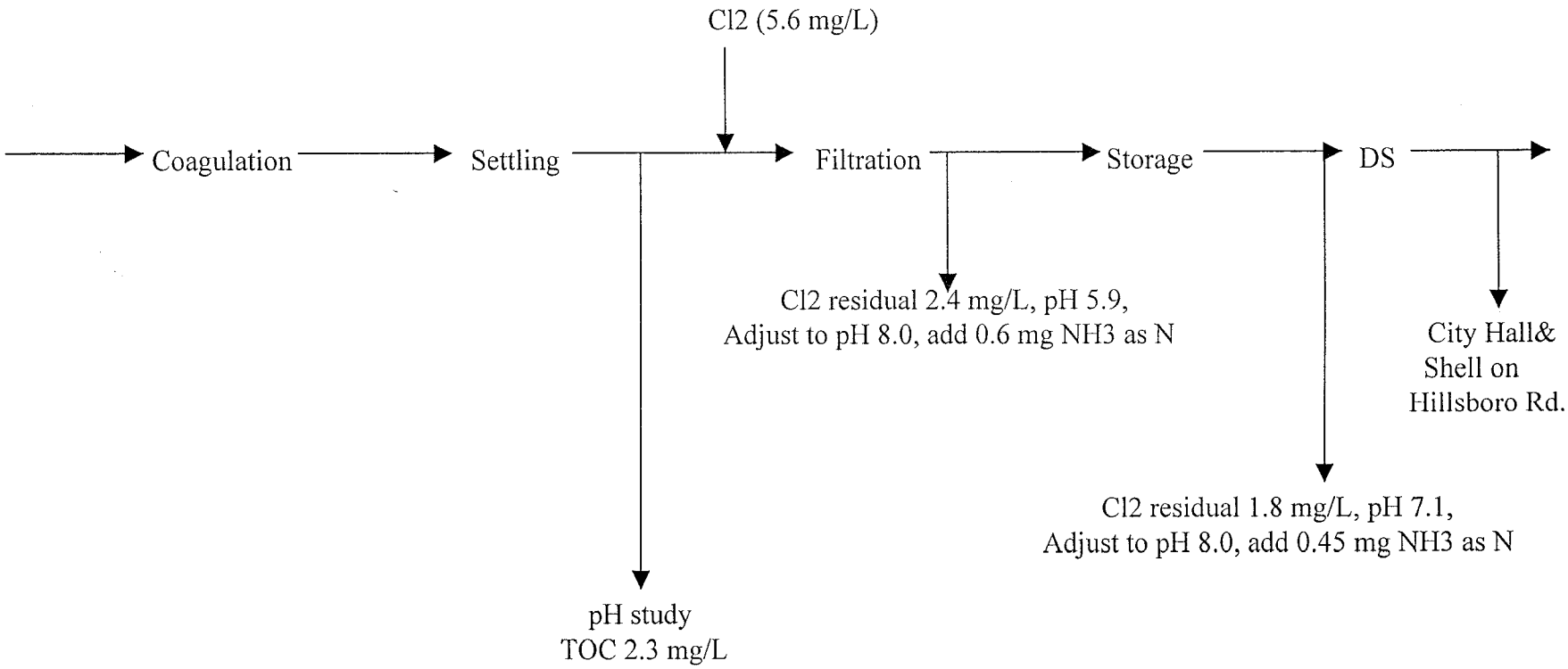


Figure A. 8 Sampling flowchart for Durham, NC (November 10, 1997)

Durham, 11/10/97, 11:05 am:
Temperature: 15°C

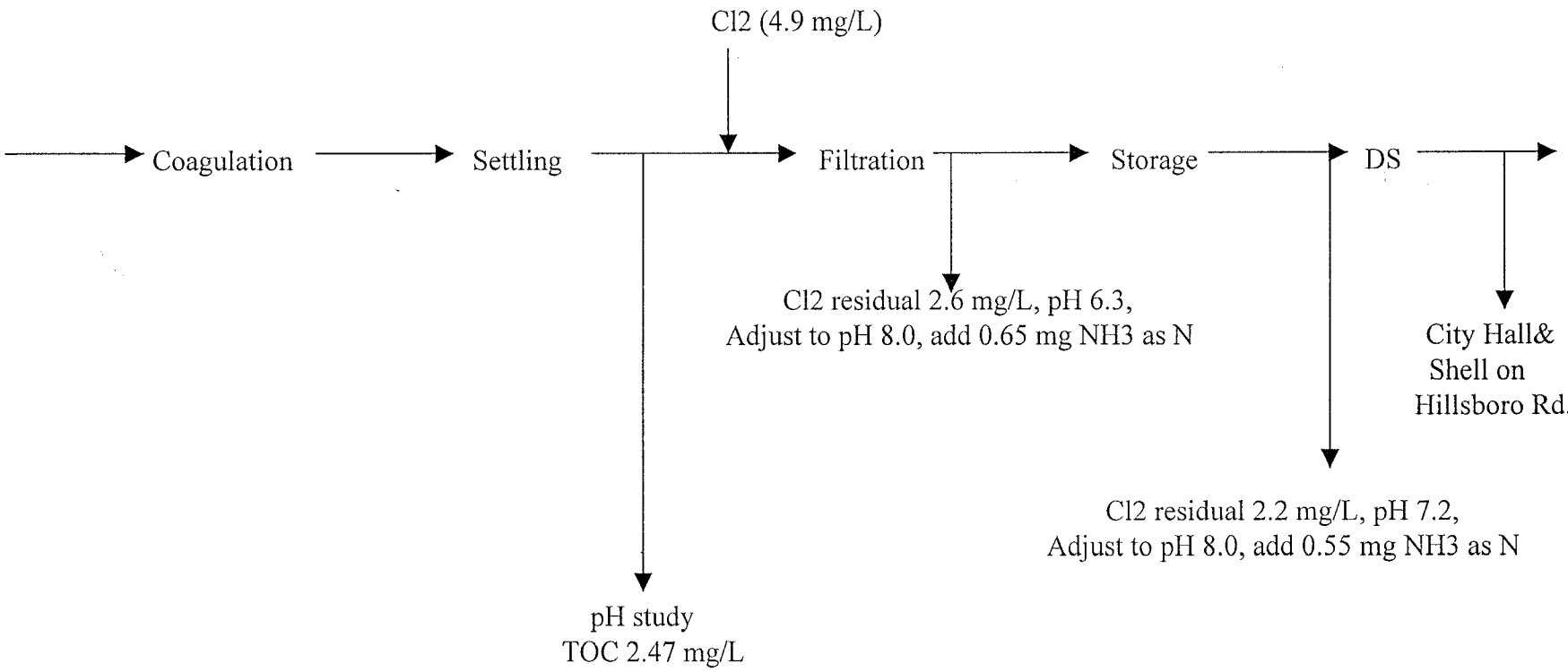


Figure A. 9 Sampling flowchart for Greenville, NC (August 6, 1997)

Greenville, 8/06/97, 11:00 am:
Temperature:27°C

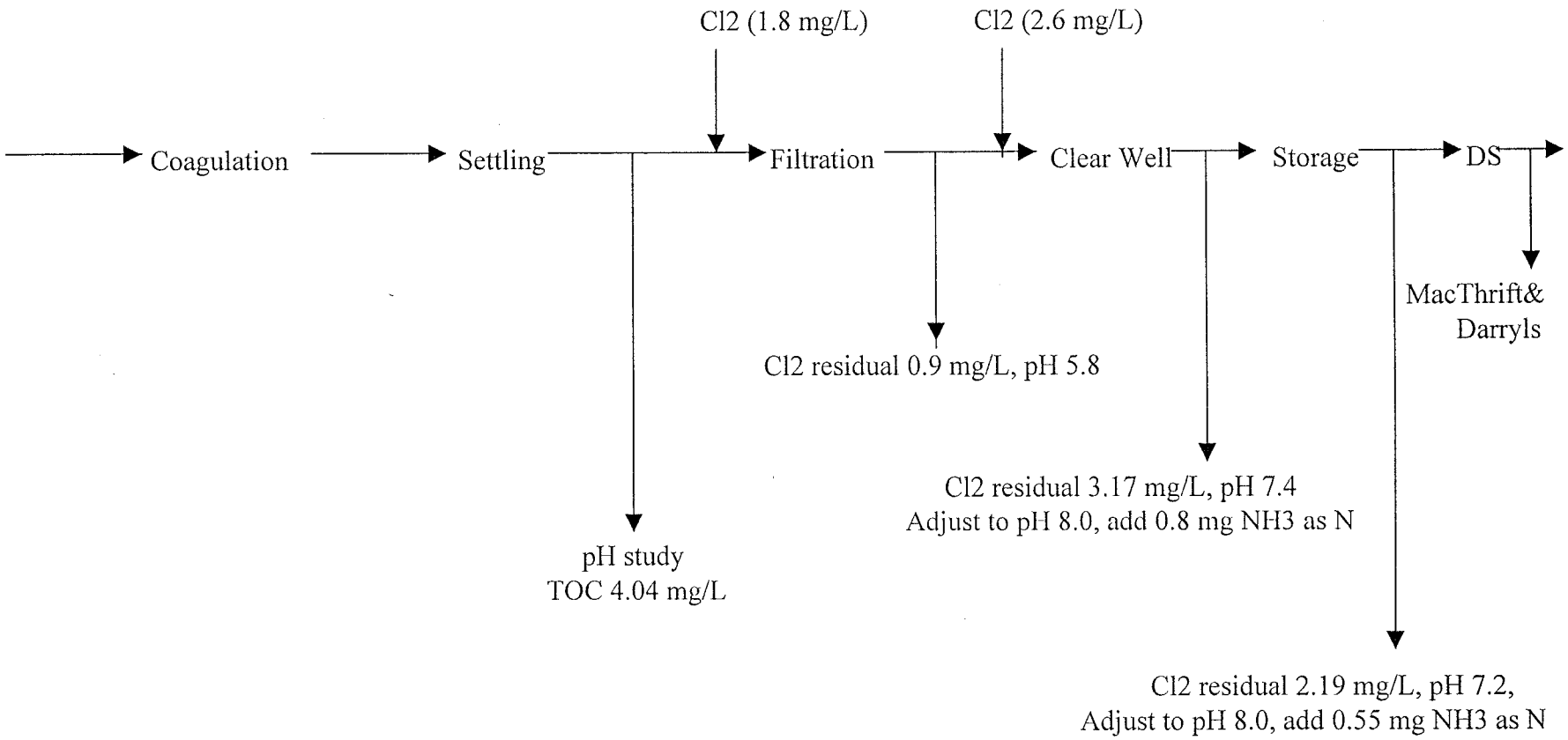


Figure A. 10 Sampling flowchart for Greenville, NC (December 1, 1997)

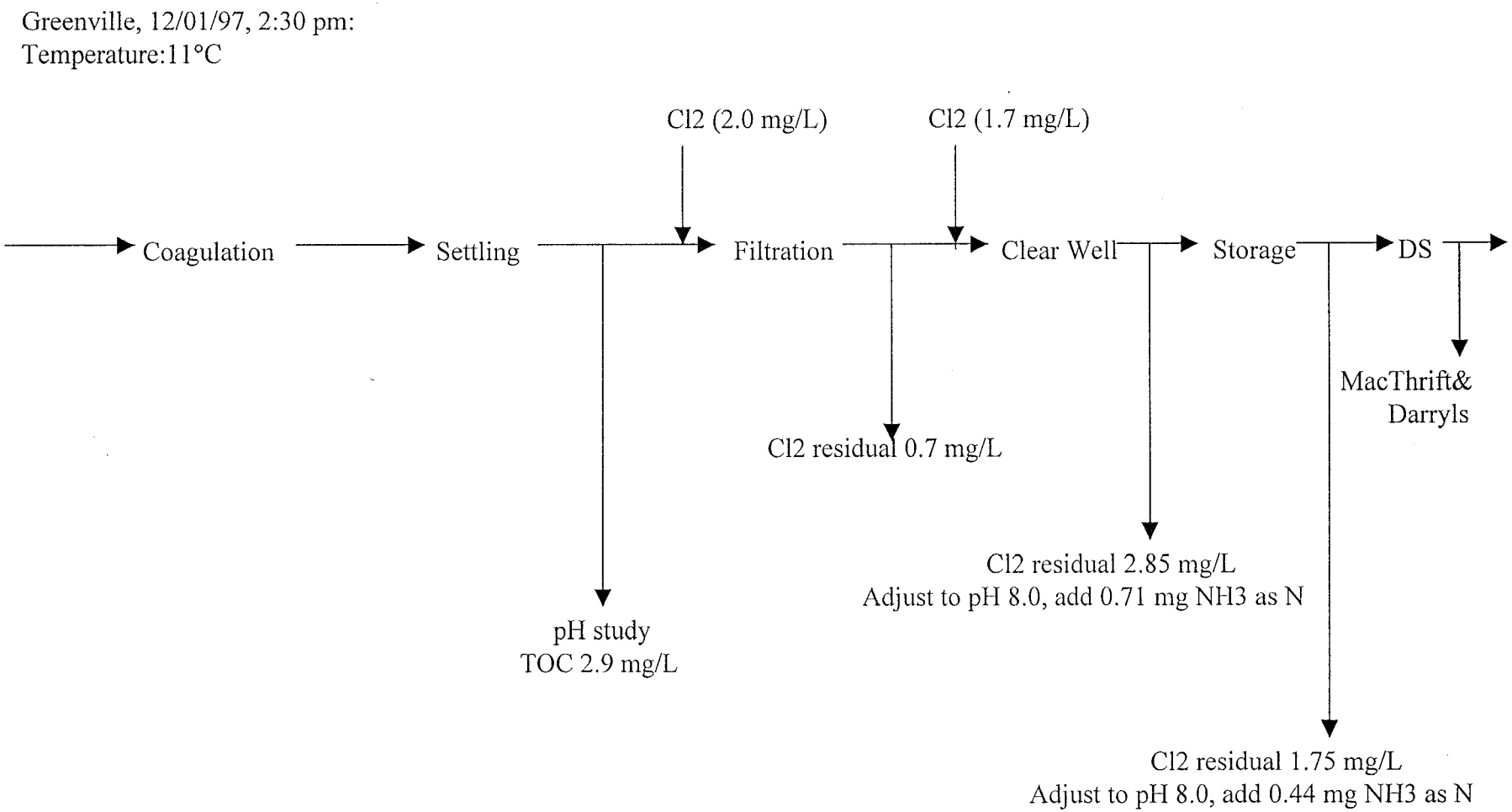


Figure A. 11 Sampling flowchart for Fayetteville, NC (September 18, 1997)

Fayetteville, 9/18/97, 2:00 pm:
Temperature: 27°C

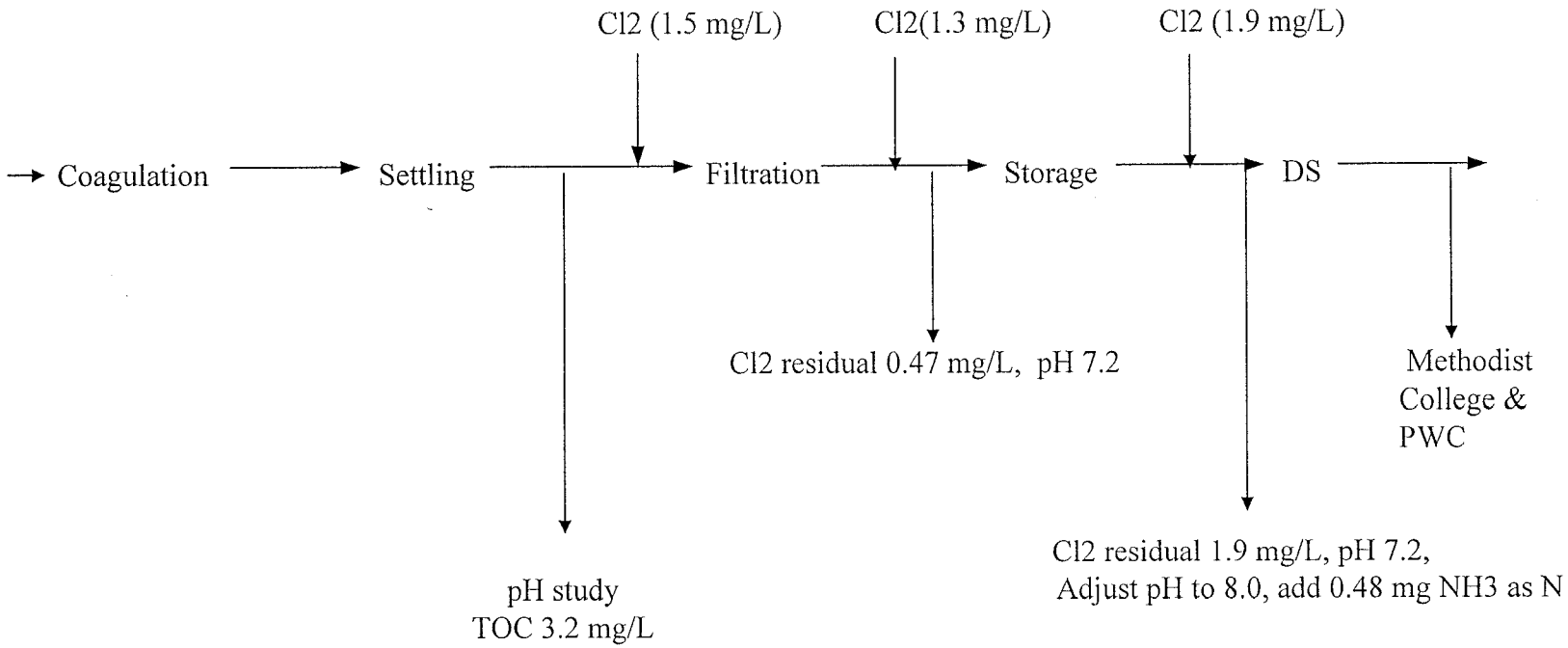
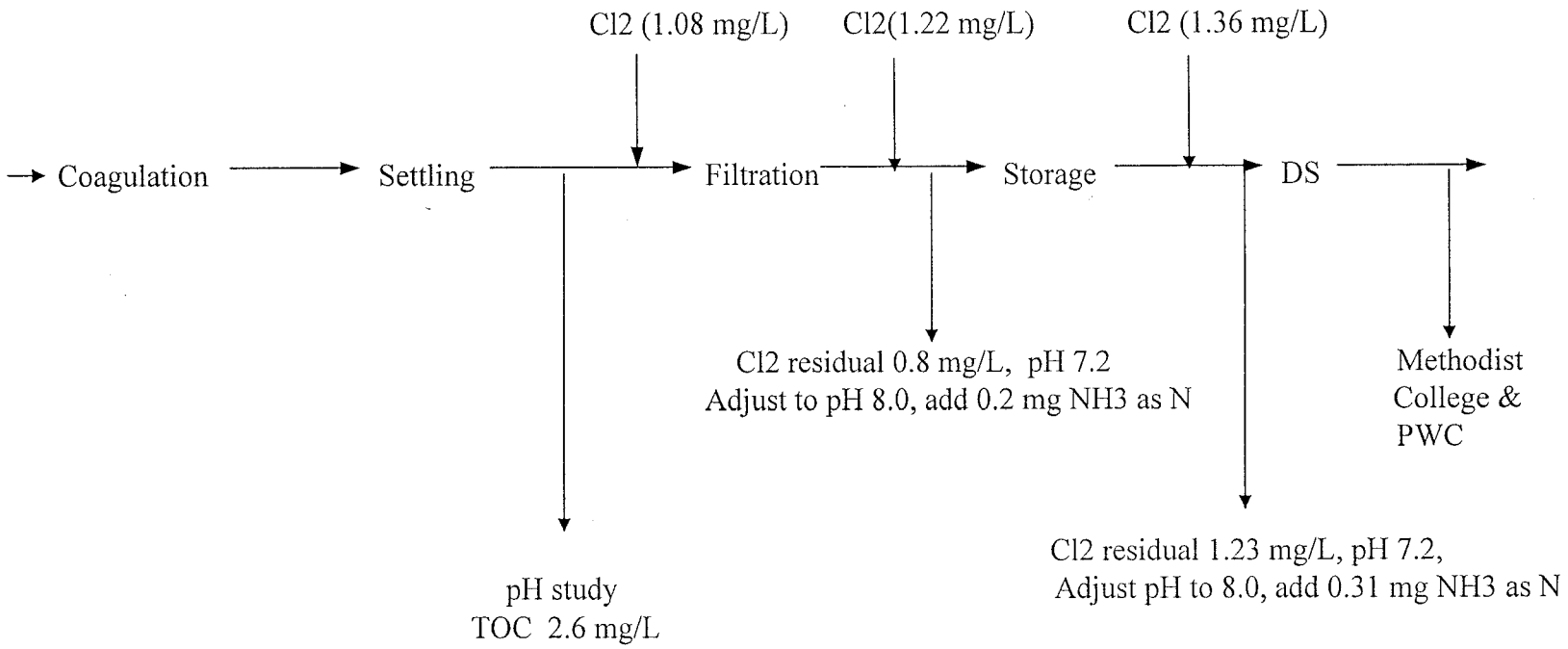


Figure A. 12 Sampling flowchart for Fayetteville, NC (December 10, 1997)

Fayetteville, 12/10/97, 2:00 pm:
Temperature: 6°C



APPENDIX B: DBP concentration in plant and distribution system (OWASA, Durham, Fayetteville)

Table B.1 Chlorine residual in plant and distribution system (OWASA, NC)

	Sample Location			
	Filtered	*Finished*	*Carr Mill*	*Hardees*
Cl_2 residual mg/L (5/6/97)	N/A	N/A	1.2	0.9
Cl_2 residual mg/L (10/3/97)	1.96	1.8	1.2	0.7

* N/A = Not Available
* On 5/6/97, temperature was 19.1°C, TOC of the settled water was 2.9 mg/L.
  On 10/3/97, temperature was 21.4°C, TOC of the settled water was 2.29 mg/L.

Table B.2 Chlorine residual in plant and distribution system (Durham, NC)

	Sample Location			
	Filtered	*Finished*	*Shell*	*City Hall*
Cl_2 residual mg/L (9/4/97)	2.4	1.8	1.3	0.9
Cl_2 residual mg/L (11/10/97)	2.6	2.2	1.5	1.3

* On 9/4/97, $Cl_2$① was 5.6 mg/L. Temperature was 27°C, TOC of the settled water was 2.3 mg/L.
  On 11/10/97, $Cl_2$① was 4.9 mg/L. Temperature was 11°C, TOC of the settled water was 2.47 mg/L.

Table B.3 Chlorine residual in plant and distribution system (Fayetteville, NC)

	Sample Location			
	Filtered	*Finished*		*Methodist College*
Cl_2 residual mg/L (9/18/97)	0.47	1.9	0.7	0.5
Cl_2 residual mg/L (12/10/97)	0.8	1.23	0.85	0.7

* On 9/18/97, $Cl_2$① was 1.5 mg/L, $Cl_2$② was 1.3 mg/L, $Cl_2$③ was 1.9 mg/L. Temperature was 27°C, TOC of the settled water was 3.2 mg/L.
On 12/10/97, $Cl_2$① was 1.08 mg/L, $Cl_2$② was 1.22 mg/L, $Cl_2$③ was 1.36 mg/L. Temperature was 6°C, TOC of the settled water was 2.6 mg/L.

Figure B.1 TTHM Concentrations in plant and distribution system (OWASA, Chapel Hill, NC)

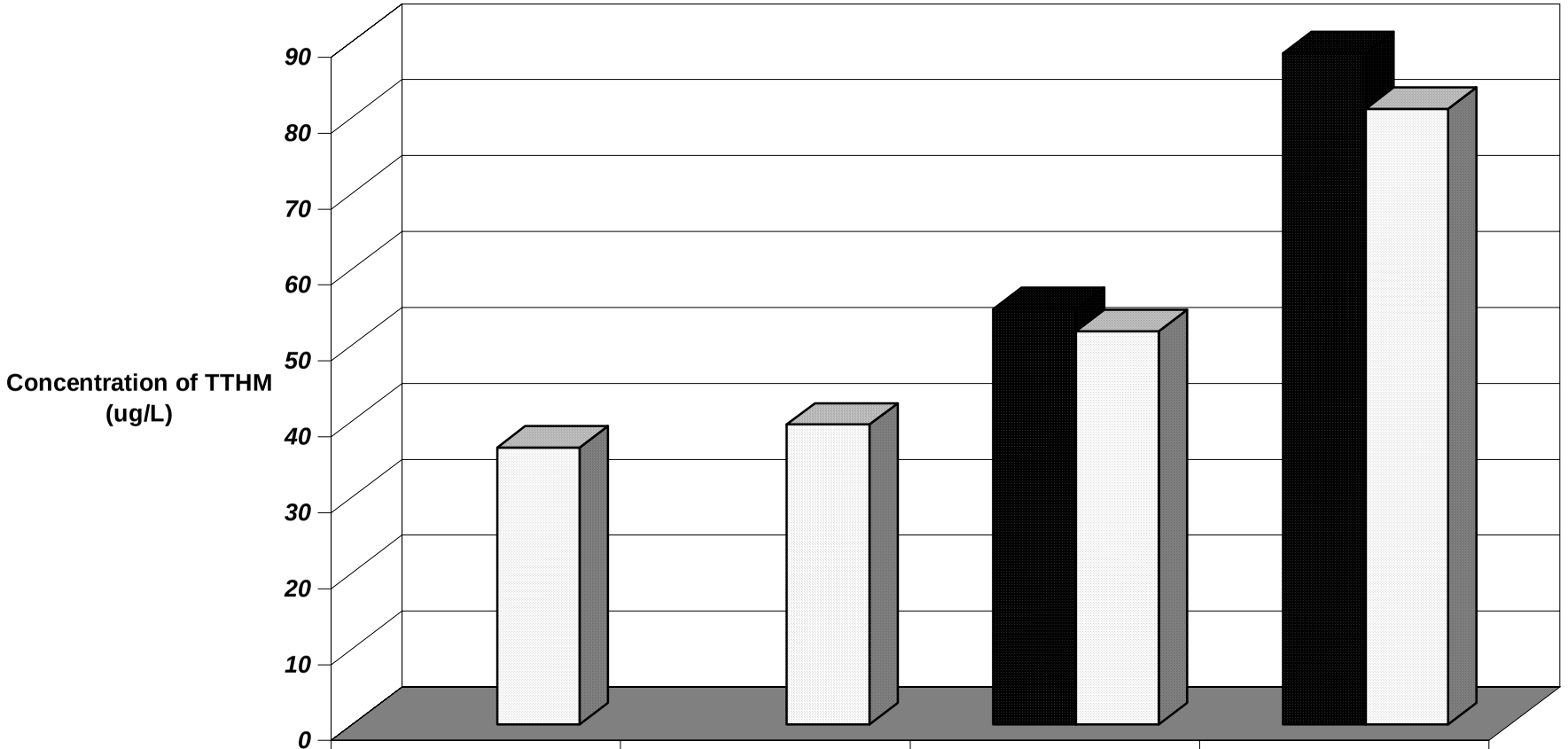
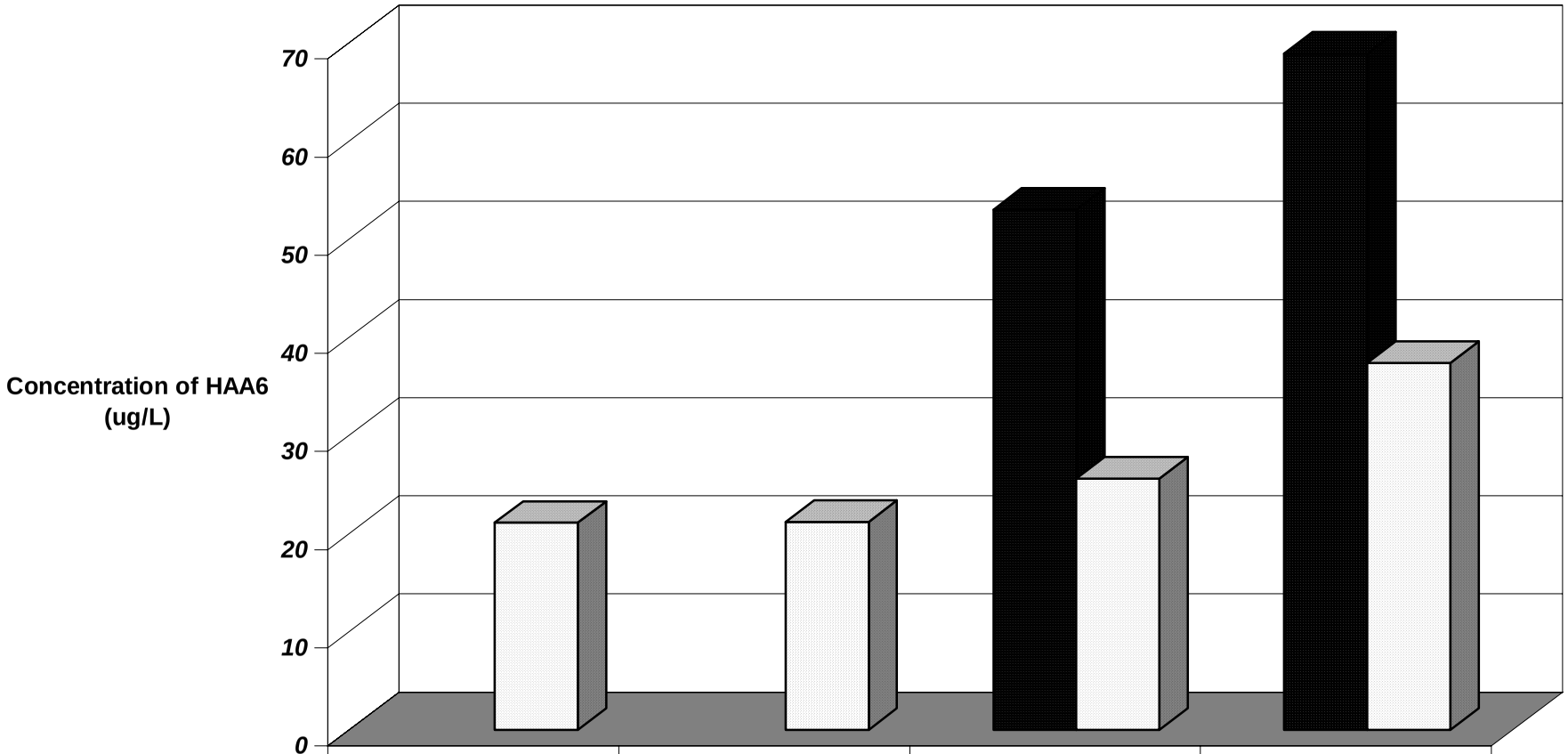


Figure B.2 HAA6 Concentrations in plant and distribution system (OWASA, Chapel Hill, NC)



	Filtered	Finished	Carr Mill	Hardees
■ HAA6 ug/L (5/6/97)			53	68.9
□ HAA6 ug/L (10/3/97)	21.1	21.2	25.6	37.4

Sampling points

Figure B.3 DCAA Concentration in plant and distribution systems (OWASA, Chapel Hill, NC)

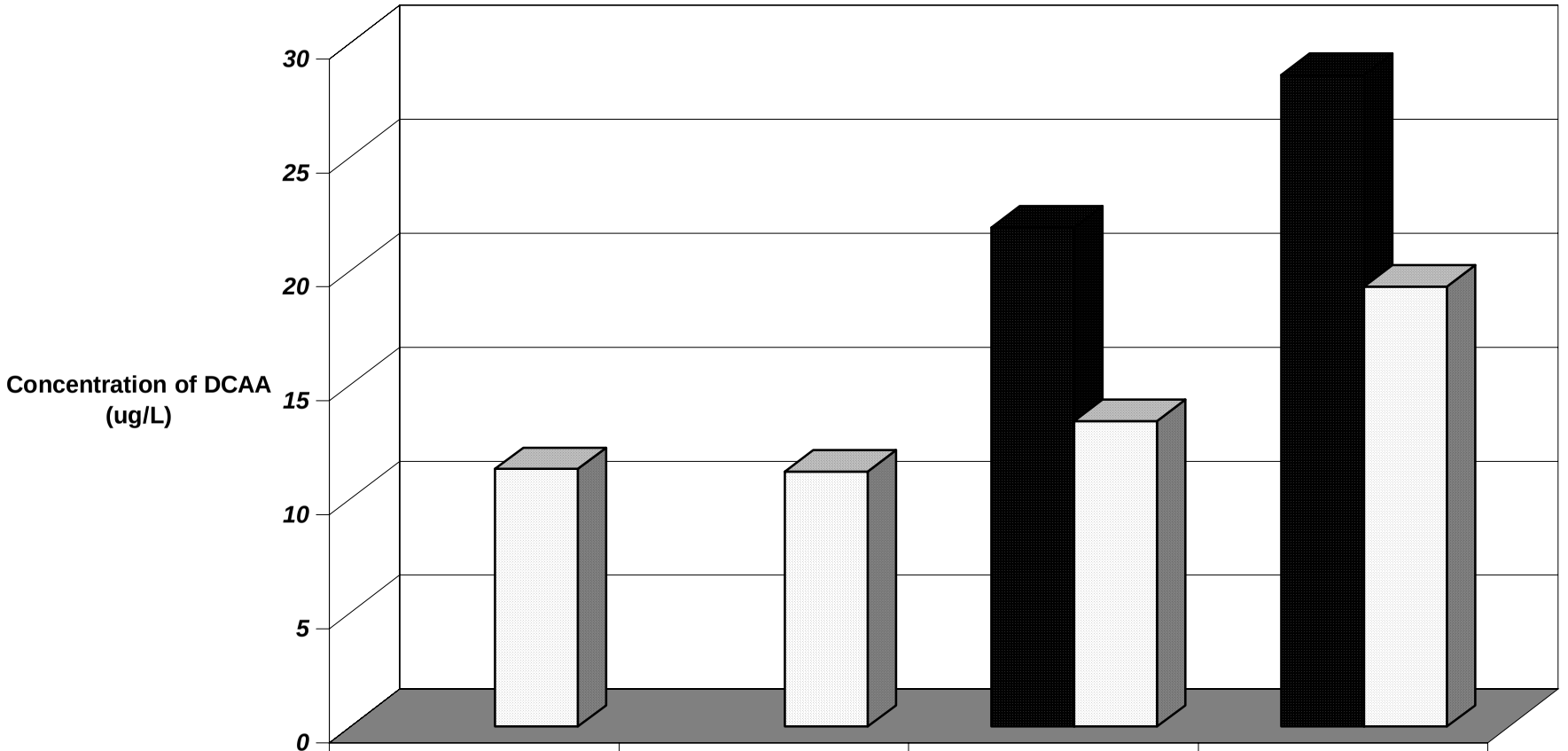
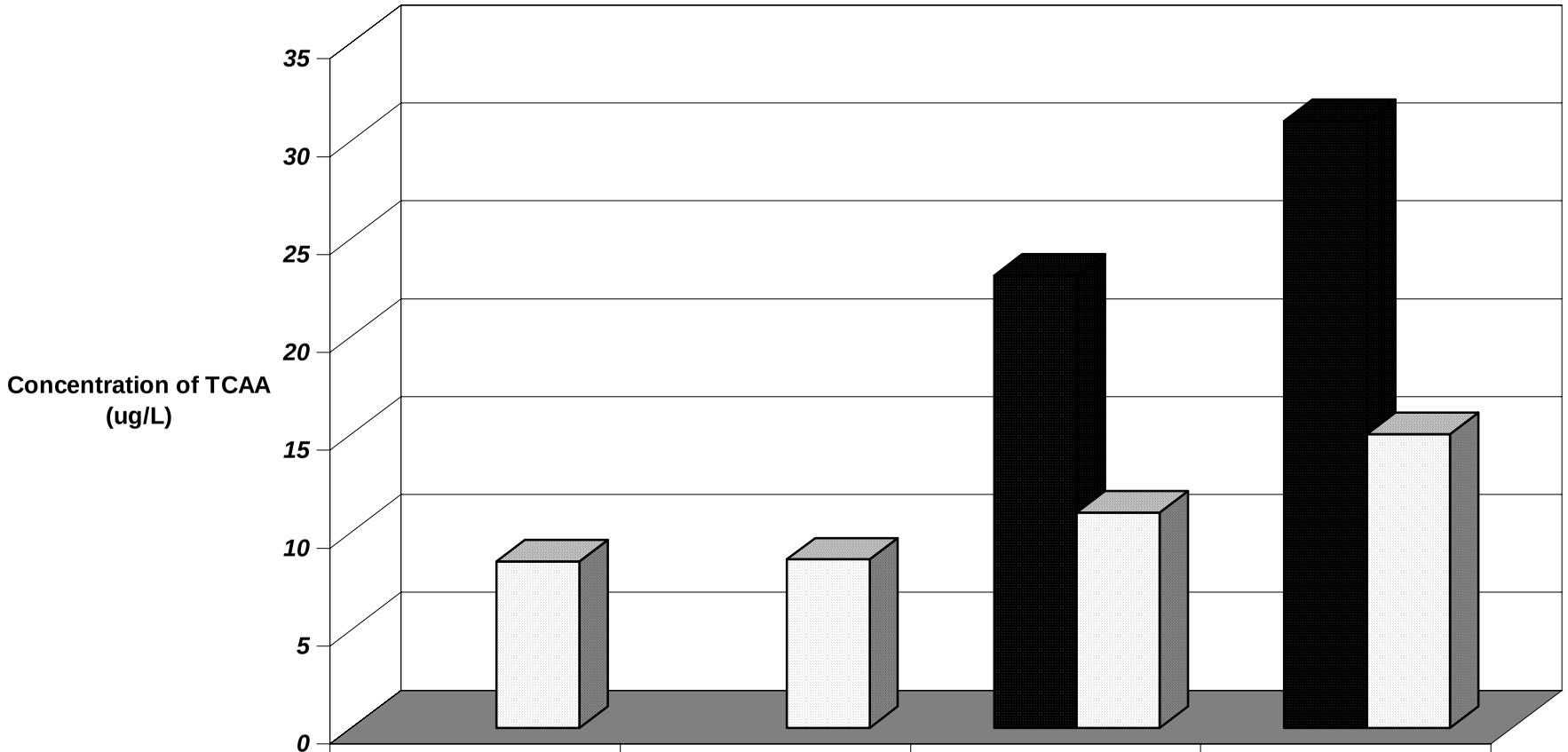


Figure B.4 TCAA Concentrations in plant and distribution system (OWASA, Chapel Hill, NC)



	Filtered	Finished	Carr Mill	Hardees
■ TCAA ug/L (5/6/97)			23.1	31
□ TCAA ug/L (10/3/97)	8.5	8.6	11	15

Sampling points

Figure B.5 TTHM Concentrations in plant and distribution system (Durham, NC)

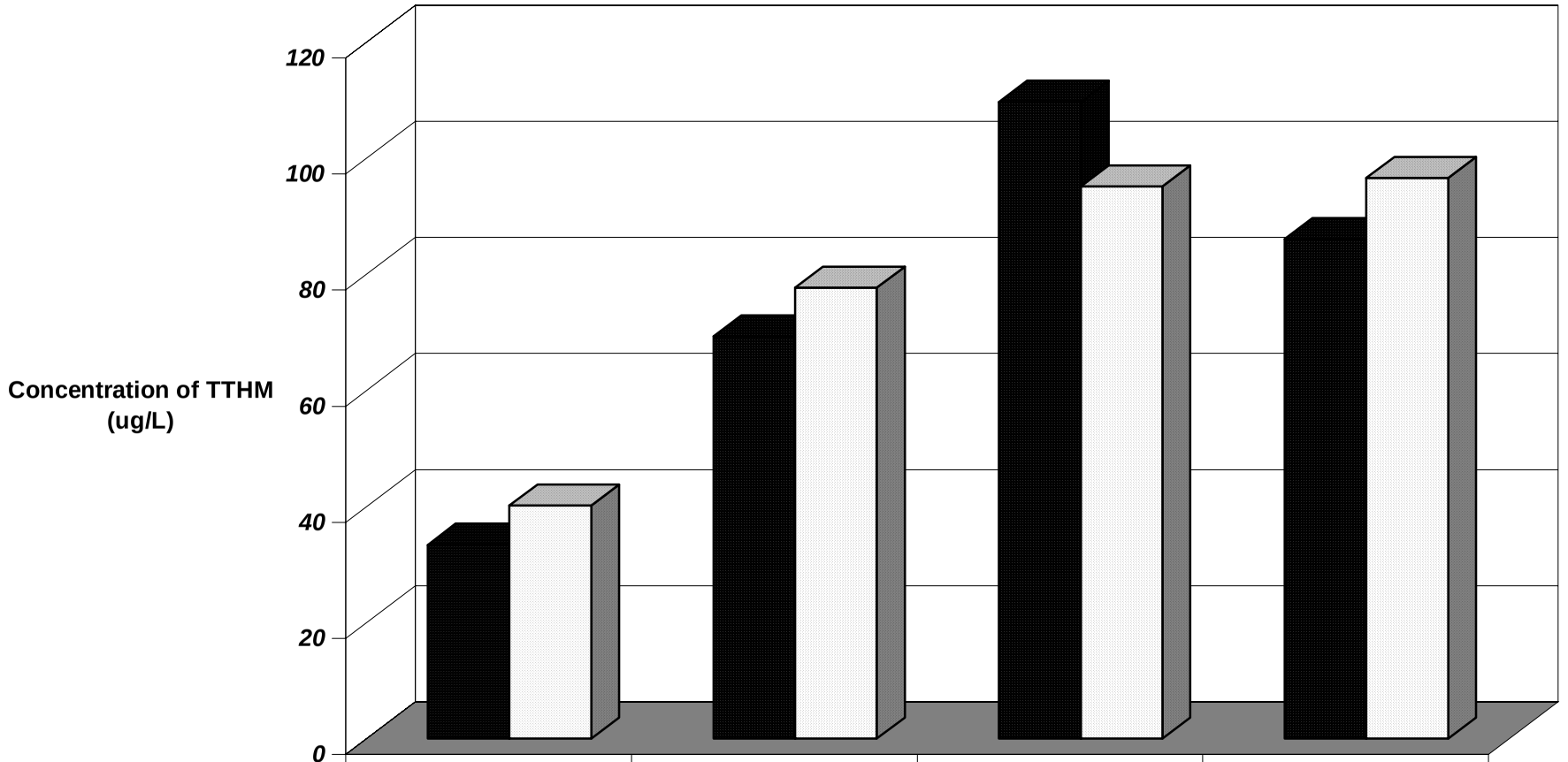


Figure B.6 HAA6 Concentrations in plant and distribution system (Durham, NC)

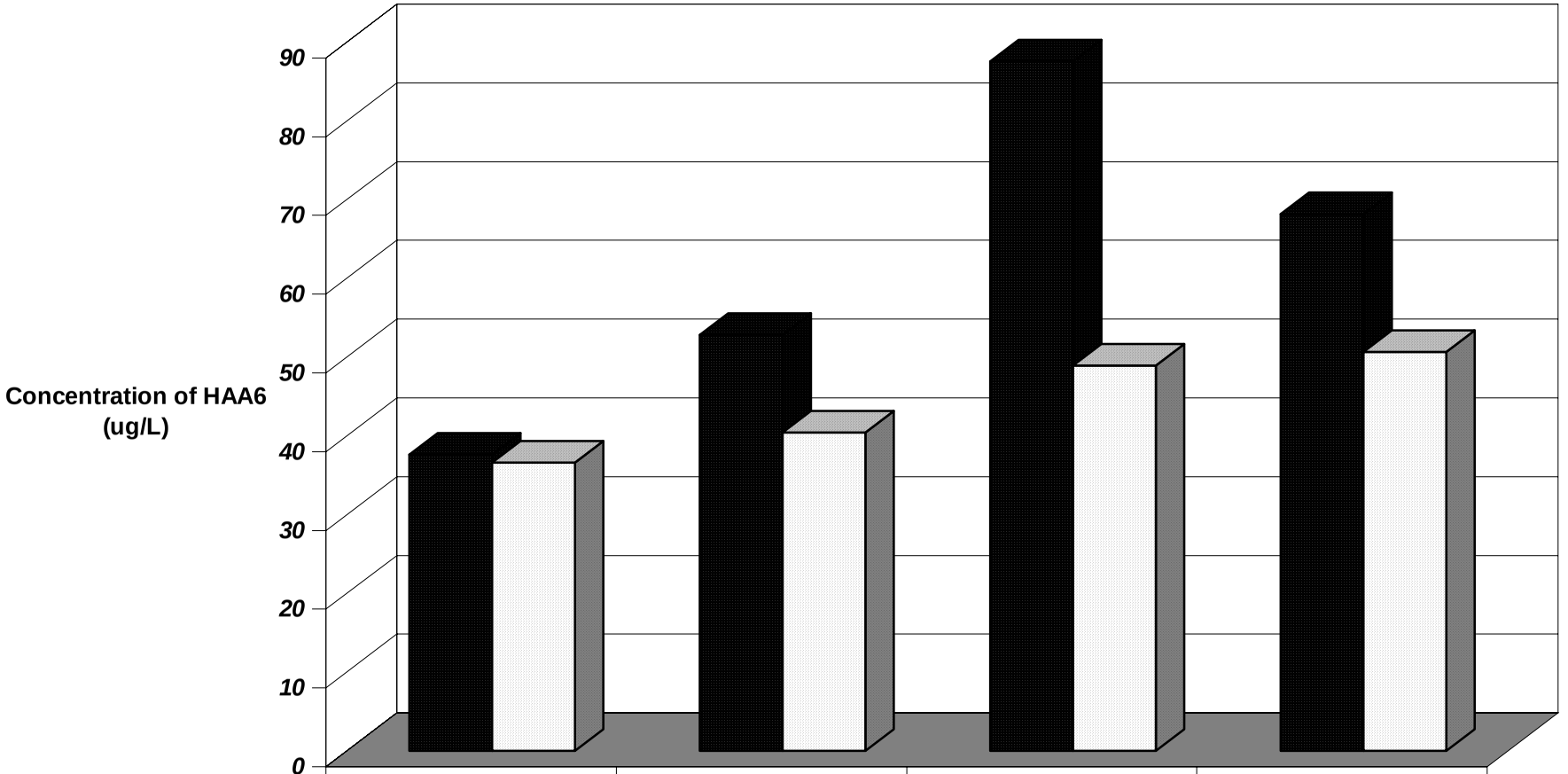


Figure B.7 DCAA Concentrations in plant and distribution system (Durham, NC)

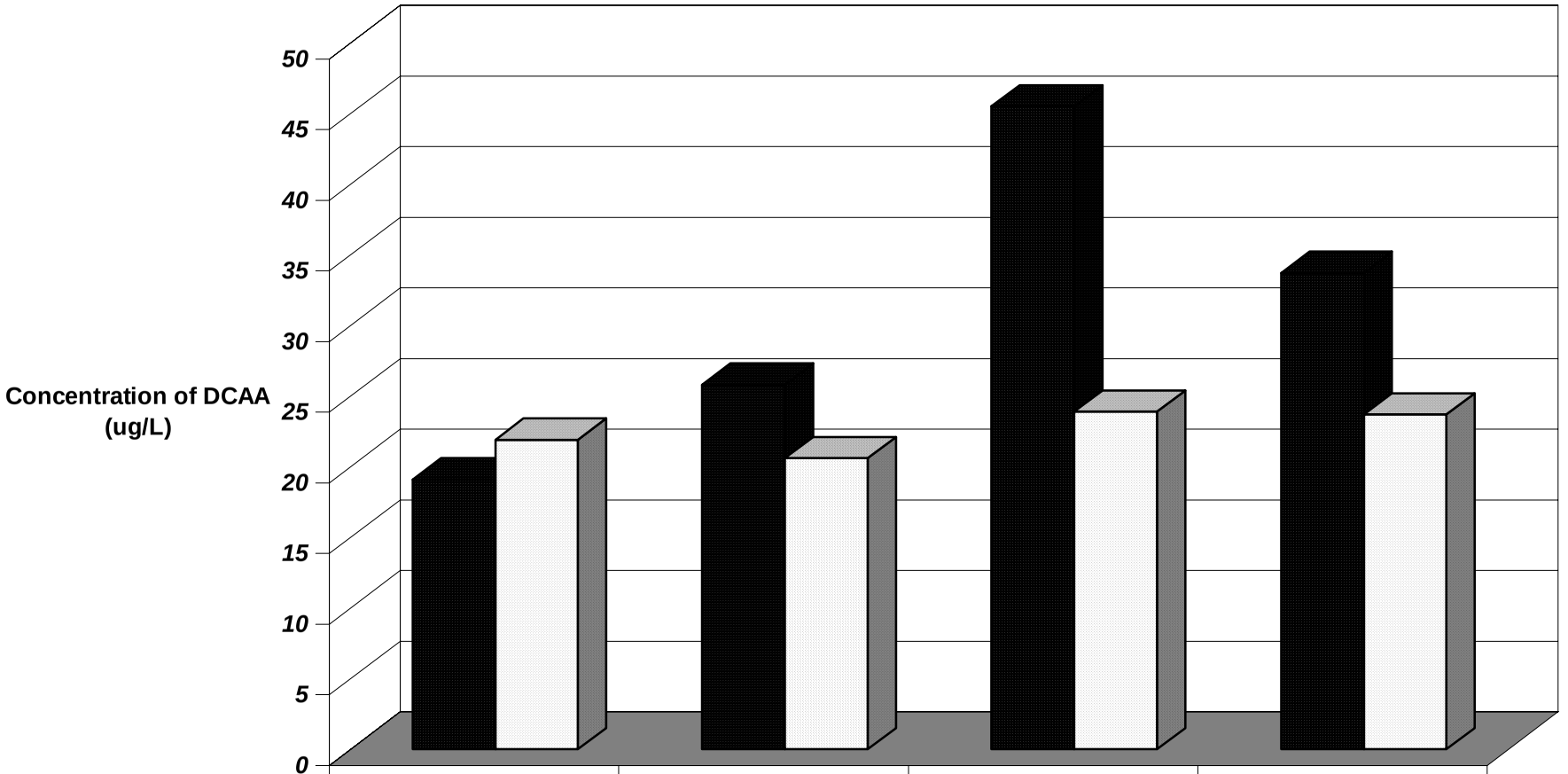


Figure B.8 TCAA Concentrations in plant and distribution system (Durham, NC)

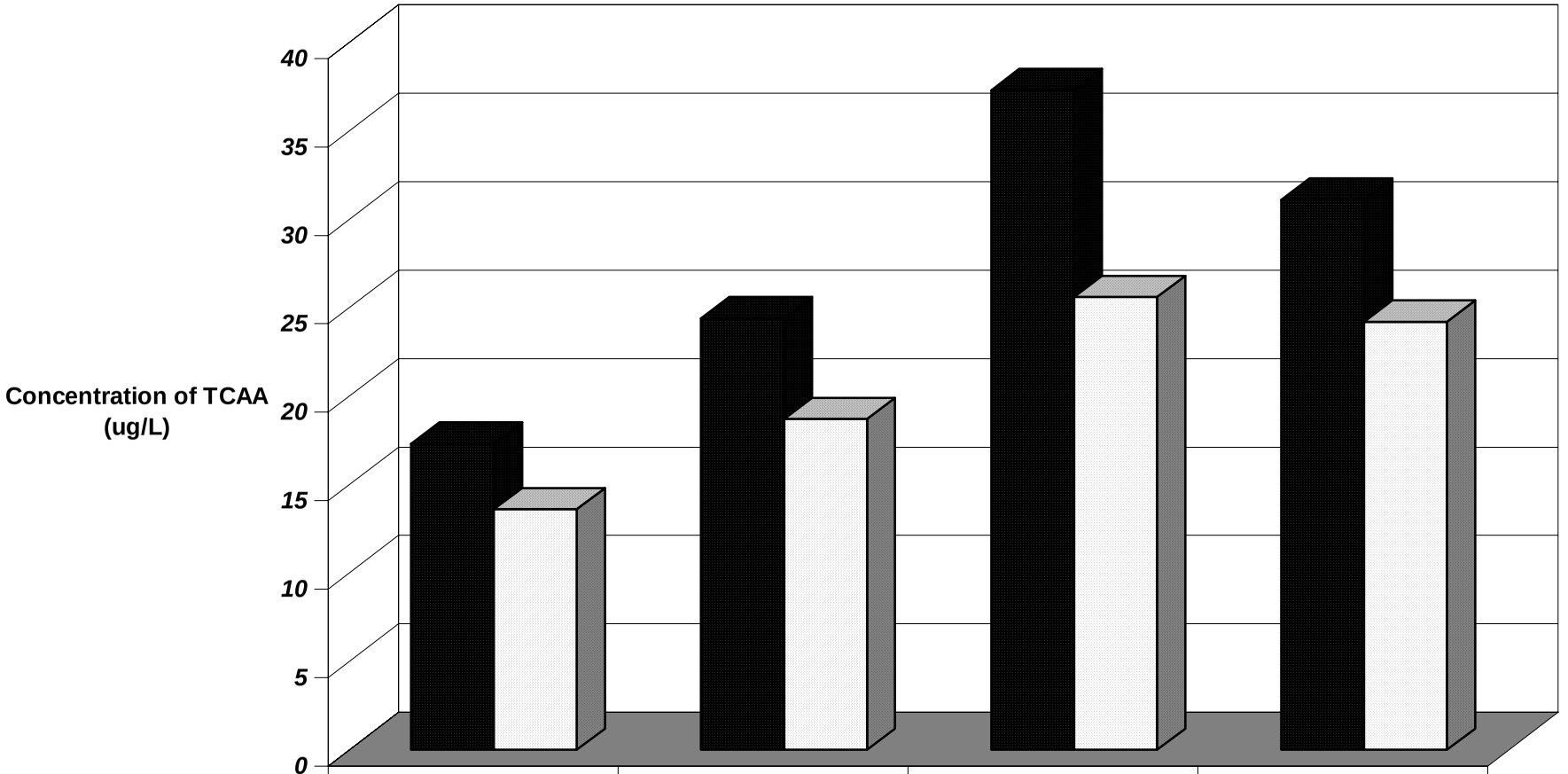


Figure B.9 TTHM Concentrations in plant and distribution system (Fayetteville, NC)

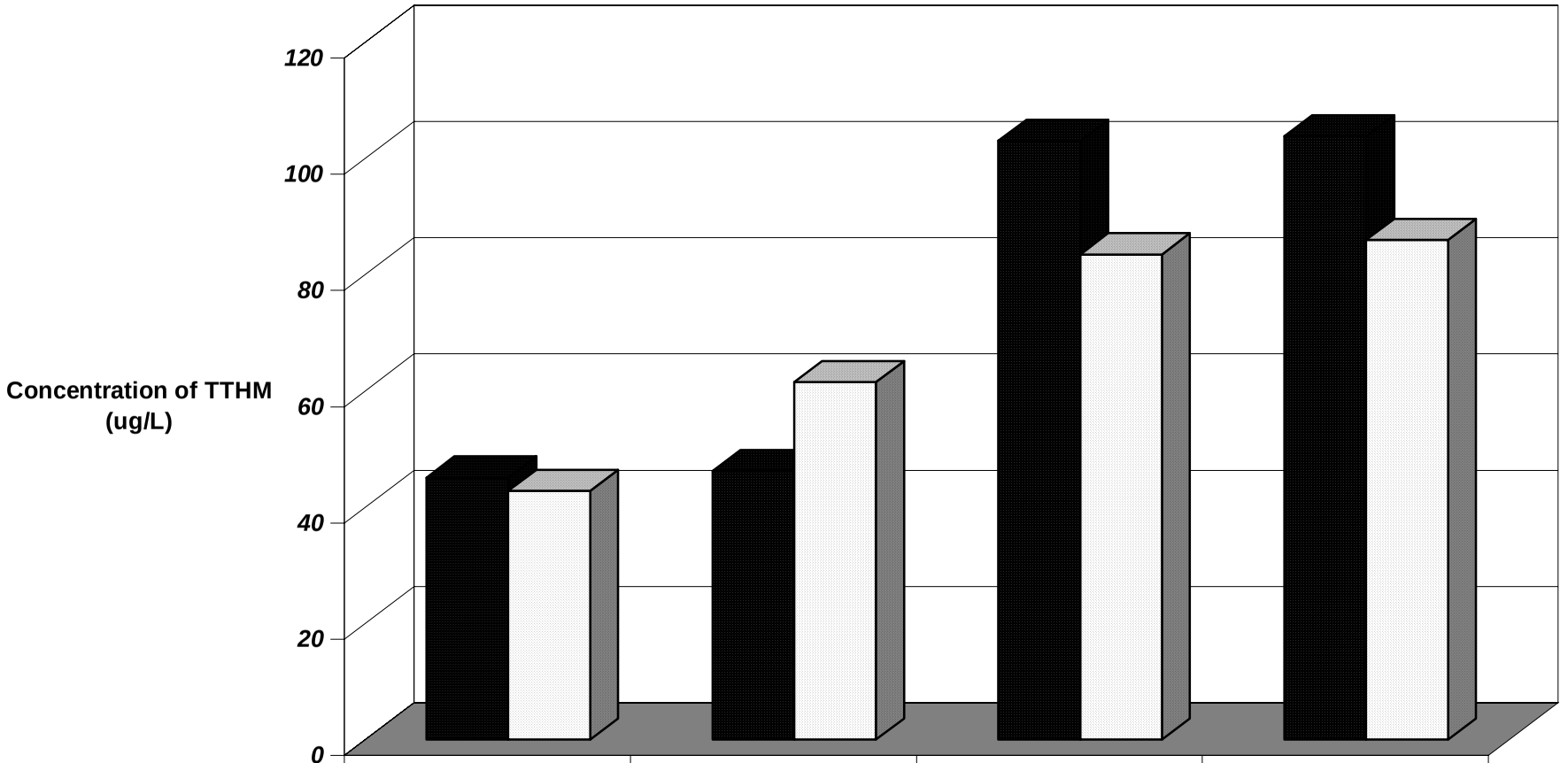


Figure B.10 HAA6 Concentrations in plant and distribution system (Fayetteville, NC)

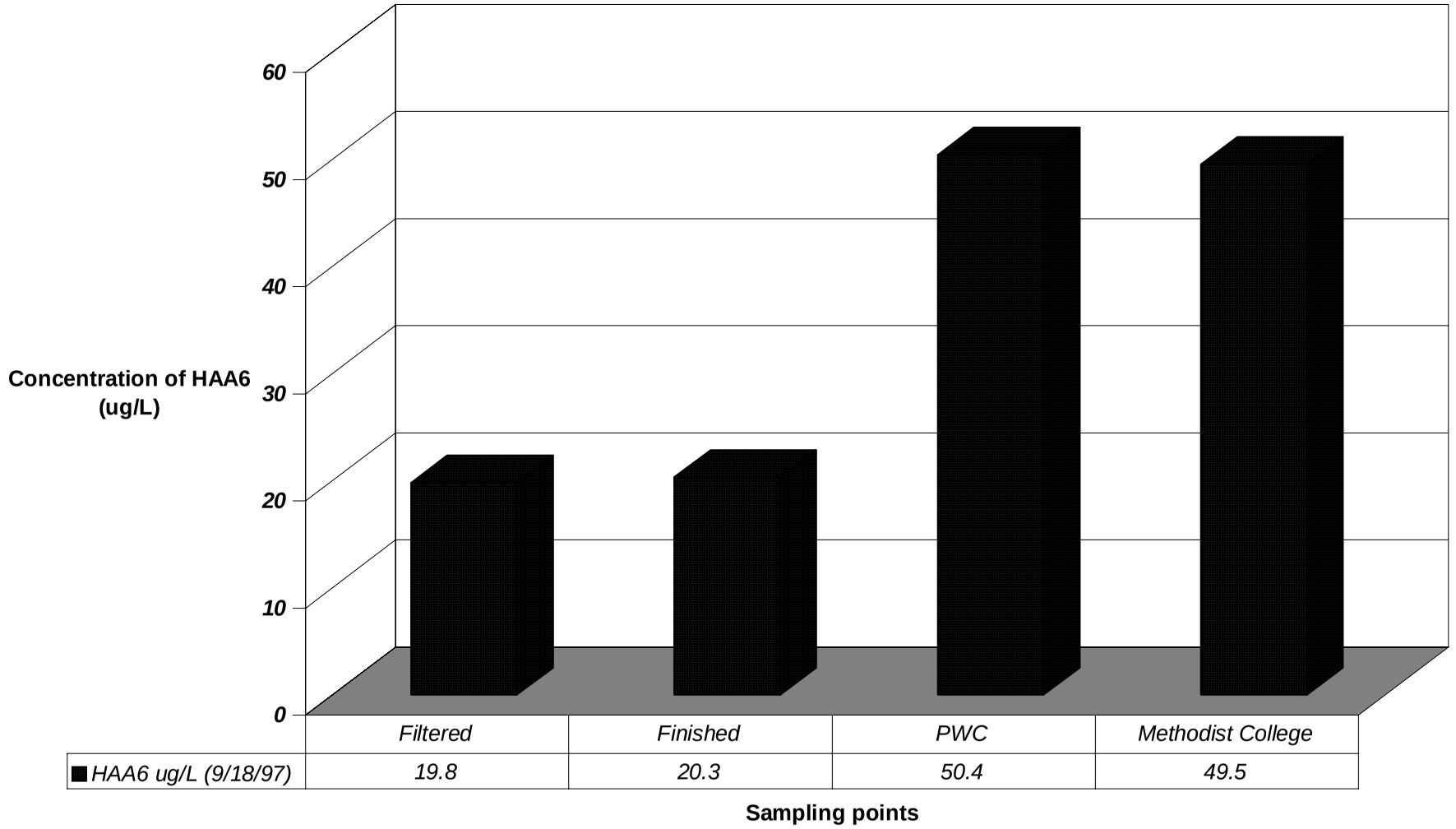


Figure B.11 DCAA Concentrations in plant and distribution system (Fayetteville, NC)

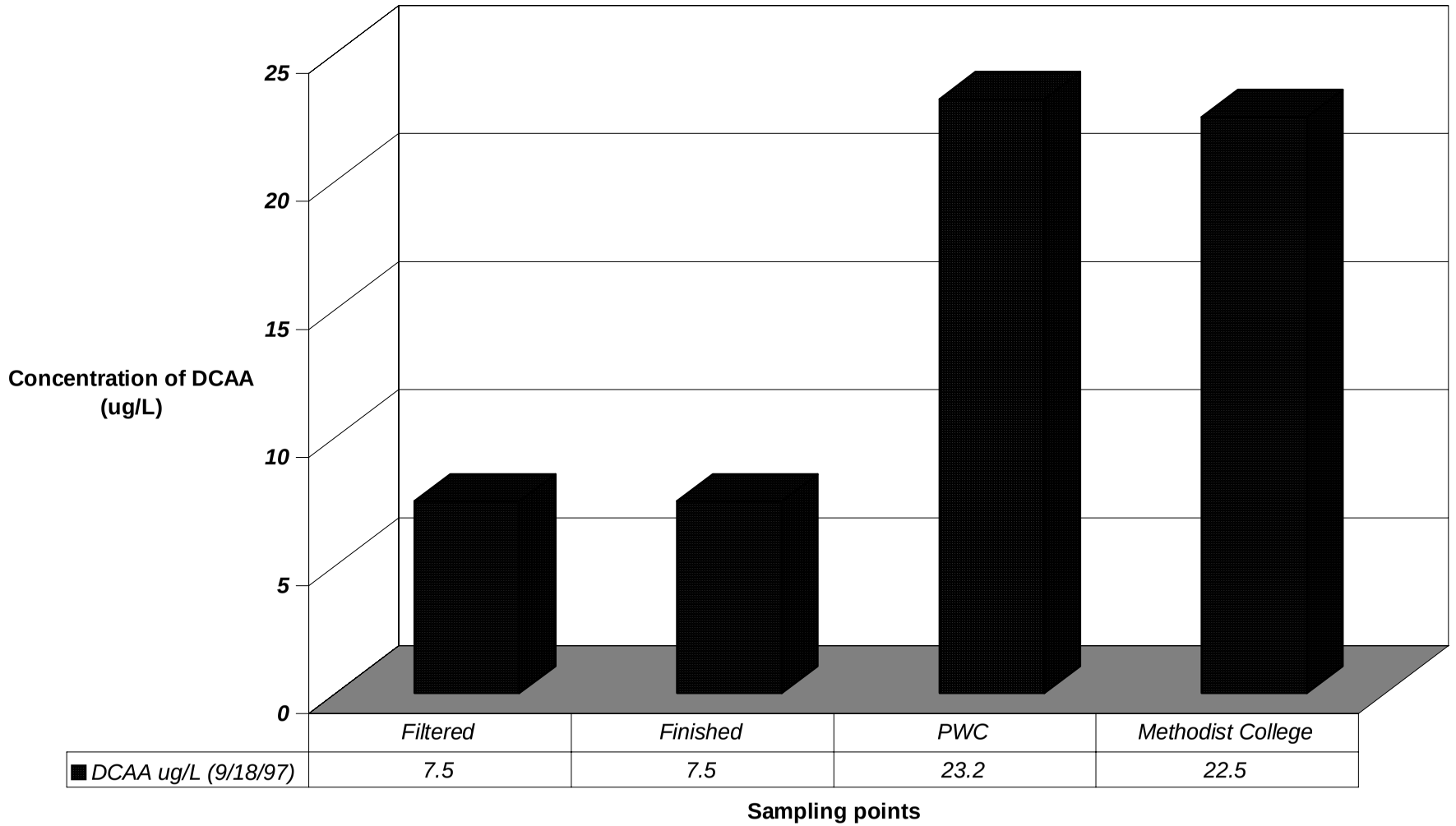


Figure B.12 TCAA Concentrations in plant and distribution system (Fayetteville, NC)