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Survey of Trihalomethane Levels in Chlorinated Drinking Water of Michigan Treatment Plants

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SURVEY OF TRIHALOMETHANE LEVELS IN CHLORINATED DRINKING WATER OF MICHIGAN TREATMENT PLANTS

By

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ABSTRACT

Survey of Trihalomethane Levels in Chlorinated Drinking Water of Michigan Treatment Plants

By

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Raw and finished water of 40 Michigan treatment plants were analyzed for trihalomethane presence by the liquid-liquid extraction method.

Total trihalomethane (TTHM) levels in the finished water of the 40 treatment plants ranged from undetectable to 281.8 μg/L with a mean of 44.0 μg/L and a median of 20.5 μg/L. Range and median values are lower than those of the NOR and NOM surveys. Chloroform was usually present at the highest concentrations followed by bromodichloromethane, dibromochloromethane and bromoform.

Chlorine dose influences the amount of TTHM formed. After adjusting for this influence, treated drinking water supplies derived from surface water had significantly higher TTHM levels than either Great Lakes or groundwater. TTHM levels of Great Lakes and groundwater were significantly different only when chlorine dose effect was not accounted for, perhaps because the organic levels are low compared to surface water. However, when chlorinated, Great Lakes water tended to have higher TTHM levels than did groundwater.

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INTRODUCTION

Discovery and Prevalence of THM's in Drinking Water

In most instances, the chlorination of drinking water results in the presence of trihalomethanes (THM's) where none were previously found. Though other researchers (Kleopfer and Fairless, 1972; Novak et al, 1973; Tardiff and Deinzer, 1973) previously noted the presence of THM's in drinking water, the reaction of chlorine with naturally occurring organics to form THM's was first noted by Rook in 1974, and then, later the same year, reported also by Bellar, Lichtenberg and Kroner (1974).

Following this discovery, the Environmental Protection Agency (EPA) undertook a nationwide survey called the National Organics Reconnaissance Survey (NORS) to determine the extent of contamination of drinking water by halogenated organics including the THM's (Symons et al., 1975). Of the 79 cities surveyed which chlorinated drinking water, all had THM's in the finished water. When THM's were located in raw water, it was at very low levels.

Later an additional survey, the National Organics Monitoring Survey (NOMS), of 113 public water systems was undertaken by the EPA (U.S.EPA, 1978; U.S. EPA, 1979). The results of the Phase I portion of NOMS are similar to those of NORS. Median and range concentrations of both surveys are given in Table 1.

Table 1. Median and range concentrations of four trihalomethanes found in chlorinated drinking water by NORS and NOMS (phase I).

	NORS		NOMS	
	Concentration, µg/L			
Trihalomethane	Median	Range	Median	Range
$CHCl_3$	21	LD*- 311	27	LD - 271
$CHCl_2Br$	6	LD - 116	10	LD - 183
$CHClBr_2$	1	LD - 100	LD	LD - 190
$CHBr_3$	5	LD - 92	LD	LD - 39
Total THM	27	LD - 482	45	LD - 457

*LD, less than detection limit.

Human Health Effects

No chloroform was detected in the plasma of New Orleans residents (Dowty et al., 1975). However, in later studies, trihalomethanes have been detected in blood and adipose tissues of individuals who drink chlorinated water. In a 1976-1979 survey of Miami, Florida area residents, all participants were found to have chloroform levels of 10 to 60 µg/L in blood (Enos, 1979). In a later study of the same city trihalomethane levels of 2 to 400 µg/L were found in the blood and adipose tissue (Pfaffenberger, 1980).

Many epidemiological studies have been done which link drinking water to cancer mortality. Two basic approaches of analysis have been attempted. The first compares cancer mortality rates between people

who drink groundwater and those who drink surface water (Kuzma et al., 1977; Wilkins et al., 1979). By the second approach, cancer mortality rates are compared between individuals drinking chlorinated and nonchlorinated water, or cancer mortality rates are compared according to THM levels in drinking water (Alavanja et al., 1978; Cantor et al., 1977; Hogan et al., 1979; Wilkins et al., 1979).

From the accumulated studies, there appears to be a relationship between drinking water and mortality incidences from total cancers, and various urinary and gastrointestinal cancers. Many criticisms, however, of these preliminary epidemiological studies do exist (Wilkins et al., 1979; Maugh, 1981a) which do not allow a causative ruling of drinking water's effect on cancer mortality. Presently, more comprehensive epidemiological studies are being done to clarify this possibility (Maugh, 1981a).

Animal Metabolism and Toxicity

After exposure to chloroform, chloroform accumulates mostly in fat, then in the liver, and to a lesser extent in blood, brain, kidney, and muscle (Cohen and Hood, 1969). Metabolism appears to occur in both the liver and the kidney, though more is known of chloroform metabolsim in the liver. Metabolism occurs in the microsomes isolated from the liver by cytochrome P-450 (a co-enzyme) mediated mixed function oxidase (Pohl and Krishna, 1978, McMartin et al., 1981). Factors which induce cyctochrome P-450, such as phenobarbital (Gopinath and Ford, 1975; McMartin et al., 1981), polybrominated biphenyls (Kluwe et al., 1978a; Kluwe et al., 1978b) and fasting (Nakajima and Sato, 1979; McMartin et

al., 1981) increase the rate of metabolism. The final metabolites appear to be carbon dioxide and chloride (Rubenstein and Kanics, 1964; Van Dyke et al., 1964; Fry et al., 1972; Brown et al., 1974; Taylor et al., 1974).

Three pathways to these end products have been suggested. The first proposed intermediate to CO_2 formation is a free radical (Brown et al., 1974; U.S. EPA, 1980). Evidence for this pathway was obtained mostly from what is known of carbon tetrachloride metabolism (Smuckler, 1976; Reynolds, 1977; Poyer et al., 1978) and from the chemical properties of chloroform (Van Dyke, 1969).

The second proposed intermediate is methylene chloride. The pathway entails the reduction of chloroform to methylene chloride and its subsequent oxidation to formaldehyde, formic acid and finally carbon dioxide. None of the intermediates have been isolated in an in vitro or in vivo system (Rubenstein and Kanics, 1964).

In the final pathway, phosgene is the suggested intermediate to CO_2 formation. After administering choroform, phosgene was trapped using cysteine to form 2-oxothiazolidene-4-carboxylic acid (Mansuy et al., 1977; Pohl et al., 1977; Pohl and Krishna, 1978). Oxygen, microsomes, cytochrome P-450 and NADPH appear to be necessary for phosgene formation. Pohl and coworkers (1977) and Mansuy and coworkers (1977) proposed that chloroform is oxidized to trichloromethanol which spontaneously dehydrochlorinates to give phosgene.

The metabolite of chloroform may do one of two things. If glutathione, a tripeptide containing the sulfhydryl-bearing amino acid cysteine sensitive to electrophilic attack (Cagen and Klaassen, 1980), is present, the metabolite binds to it (Ekstrom and Hogberg, 1980).

Glutathione is necessary for metabolism of chloroform to carbon dioxide (Rubenstein and Kanics, 1964). When glutathione levels are depleted, the chloroform metabolite then binds covalently to microsomal protein and lipids (Ilett et al., 1973; Uehleke and Werner, 1975; Brown et al., 1974; Ekstrom and Hogberg, 1980).

Pieces of evidence suggest that phosgene is a highly reactive electrophile (Mansuy et al., 1977) capable of binding with glutathione, and it is the reactive metabolite responsible for hepatotoxicity, nephrotoxicity and CO_2 formation (Pohl et al., 1980). Removal of phosgene with the trapping agent cysteine results in both reduced CO_2 formation and reduced covalent binding of phosgene to microsomal protein (Pohl et al., 1980). Figure 1 gives the proposed pathway of chloroform metabolism.

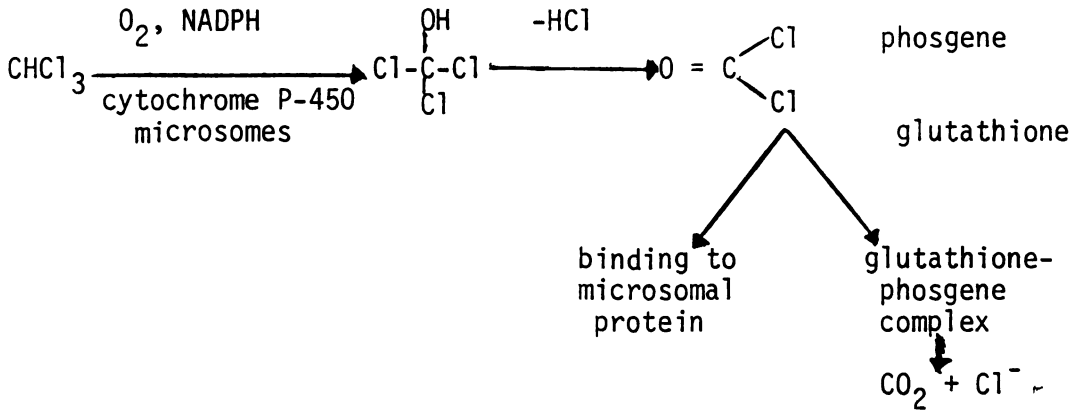


Figure 1. Proposed metabolism of chloroform having as an intermediate phosgene (modified from Mansuy et al. (1977) and Pohl et al. (1977)).

Chloroform-induced toxicity occurs mostly in the liver and to a lesser extent in the kidney (Ilett et al., 1973). Most of the current research is on hepatotoxicity. Two phases of metabolism led to hepatotoxicity and possibly nephrotoxicity (Ekstrom and Hogberg, 1980). During the first phase, chloroform metabolism occurs followed by glutathione depletion caused by binding of metabolite to glutathione and inhibition of glutathione synthesis, and finally binding of the metabolite to cellular protein. The second phase entails glutathione deficiency, lipid peroxidation and necrosis.

Phenobarbital, an inducer of cytochrome P-450, induces the proliferation of endoplasmic reticulum in the centrilobular areas of the liver (Burger and Herdson, 1966). Covalent binding of the metabolite occurs mostly in this area (Ilett et al., 1973). From autoradiographic analysis, covalent binding was found to be located mostly in the necrotic lesions (Ilett et al., 1973).

Another mode of hepatotoxicity appears to be related to the ability of the metabolite to inhibit the microsomal ATP-dependent calcium pump which could lead to intracellular calcium accumulation (Moore, 1980). Both altered calcium metabolism (Mikkelson, 1978; Cheung, 1980) and binding of metabolites to tissue macromolecules (Miller and Miller, 1966) have been implicated in carcinogenicity.

In order for necrosis and possibly carcinogenicity to occur, glutathione depletion must first occur (Brown et al., 1974; Ekstrom and Hogberg, 1980). This evidence suggests a threshold level is necessary before deleterious effects occur.

Animal Carcinogenicity

From available studies (Table 2), chloroform given by oral intubation in an oil-based vehicle caused liver, thyroid and kidney tumors in mice and rats of both sexes. The National Academy of Science (1977) reviewed the available animal toxicity studies and concluded that a human health risk exists from exposure to THM's in chlorinated water. Criticism of the use of the available animal carcinogenicity studies to determine human health risk is abundant (U.S.EPA, 1979). Risk estimation was based mostly on the National Cancer Institute (NCI) study (1976). The major criticisms stem from the use of an oil-based vehicle rather than water, chloroform was given by gavage rather than ad libitum, and very high doses and a small range of doses were used (Tardiff, 1977; Reitz et al., 1978; U.S.EPA, 1979; Budiansky, 1980). The studies do not duplicate actual THM exposure from drinking water consumption. Furthermore, Eschenbrenner (1944) noted that hepatomas did not occur unless hepatic necrosis first occurred. This study however, was relatively short-term; it was over by the fifth month.

EPA defended its approach to risk assessment by stating that from high dose to low dose and animal to man extrapolation of data is acceptable, and methods do not exist enabling a determination of threshold levels for adverse effects from long-term exposure (U.S.EPA, 1979). In dealing with human health effects, EPA takes a conservative approach favoring safety. To establish interim regulations, EPA needs only to suspect an adverse human health effect (U.S. EPA, 1979). Presently a carcinogenicity study on mice and rats' exposure to low-dose chloroform in drinking water is being performed by the EPA (Jorgenson and Rushbrook, 1980).

Table 2. Summarization of chloroform carcinogenicity studies in animals.

Study	Animal	Dosing Protocol	Significant Results
Roe, 1969-1976*	Male ICI-Swiss mice	60 mg/kg; in arachis oil; for 80 wks; by gavage; observed 24 wks. longer	hepatic tumors renal tumors
Roe, 1969-1976*	Sprague-Dawley rats (males and females)	60 mg/kg; in toothpaste; for 95 weeks; by gavage	hepatic tumors renal tumors
NCI, 1976	Osborne-Mendel rats (males and females)	male - 90 and 180 mg/kg; female - 100 and 200 mg/kg; in corn oil; 5 times per week for 78 weeks; observed 33 wks. longer; by gavage	liver tumors thyroid tumors male only - kidney tumors
NCI, 1976	$B6C3F_1$ mice (males and females)	male - 138 and 277 mg/kg; females - 238 and 477 mg/kg; in corn oil, by gavage; 5 times per week for 78 wks; observed 14 weeks longer	hepatic tumors lymphomas
Weisburger, 1977	Osborne-Mendel rats (males and females)	up to 180 mg/kg; 5 times per wk for 78 wks; in corn oil; by gavage; observed 12 weeks longer	males - kidney tumors females - thyroid tumors

Table 2 (Cont.)

Study	Animal	Dosing Protocol	Significant Results
Weisburger, 1977	$B6C3F_1$ mice (males and females)	males - up to 300 mg/kg; females - up to 500 mg/kg; 5 times per week for 78 weeks; in corn oil; by gavage; observed 12 weeks longer	hepatocellular carcinoma
Eschenbrenner, 1944	Strain A mice (males and females)	37.5 to 600 mg/kg; once every four days for 16 weeks; observed 4 weeks longer; in olive oil; by gavage	hepatomas

*Information from Reuber, 1979.

Trihalomethane Formation

Chlorine is dispersed in water as Cl_2, H_2OCl^+, Cl^+, Cl_3^-, HOCl, OCl^- and Cl^- (Morris, 1976). The relative amount of each species depends upon pH. At conditions usual for drinking water chlorination, the species of any consequence are H_2OCl^+, Cl_2, HOCl and OCl^-. Based upon relative concentrations and specific reactivity of each species to nitrogenous compounds at pH 7 and 15°C, net relative reactivity is much higher for HOCl followed by Cl_2, H_2OCl^+ and finally OCl^-. Hypochlorous acid can dissociate to hypochlorite ion and hydrogen ion. The dissociation constant ranges from 1.6 to 3.2 x 10^{-8} for the temperature range 0 to 25°C (Morris, 1966). Hypochlorous acid concentration decreases with increasing pH with a corresponding increase in hypochlorite ion concentration. Hypochlorite ion is the dominant species at pH values greater than 7.8 to 7.5. By pH 9, hypochlorite ion accounts for about 96% of aqueous chlorine (Morris, 1976). Based upon reaction studies with nitrogenous compounds, HOCl is 10,000 times more reactive than OCl^-.

HOCl is an electrophilic agent (Morris, 1976). Reaction may occur at either the oxygen or chlorine atom; however, because the latter is more electropositive the reactions probably occur more often at the chorine site.

Chlorine, when first added to water, reacts rapidly with the reducing agents such as ferrous ion, sulfide, nitrite, and easily oxidized organic compounds. Ammonia and chlorinated ammonia are the next compounds to react with HOCl, followed by the organic and inorganic compounds which are more difficult to oxidize (Johnson, 1976). Reaction of the more resistant organics with chlorine is slow

and depends upon chlorine concentration and pH.

The classical haloform reaction, a base catalyzed reaction of aqueous hypohalites or hypohalous acid with simple methyl ketones to yield trihalomethanes, has been known since 1822 (Fuson and Bull, 1934).

The reaction, as given in Figure 2, requires the enolization of the methyl ketone by proton dissociation from the alpha-carbon resulting in an enolate carbanion. The enolate carbanion then undergoes electrophilic attack by HOCl or OCl^- (Morris, 1976). After three enolizations and electrophilic attack sequences, hydrolysis occurs resulting in an organic carboxylic acid and a chloroform.

Bellar and coworkers (1974) proposed that ethanol is oxygenated to acetaldehyde which subsequently reacts with free chlorine to form chloral hydrate which then decomposes to form chloroform. The basis of this proposal is the observed presence of ethanol in tap water. Alcohols and amines which are oxidized to ketones are capable of reacting in the haloform reaction (Fuson and Bull, 1934).

The rate determining step is the enolization of the ketone. The stabilization of the enol is pH dependent, increasing with increasing pH. At pH 7 simple methyl ketones - such as acetone, acetaldehyde and acetophenone - do not react sufficiently to produce significant amounts of trihalomethanes (Stevens et al., 1976). Acetone concentration in raw water is not high enough to account for the observed THM levels. At pH values above 8 or 9, acetone and other simple methyl ketones may significantly contribute to THM formation (Morris, 1976; Stevens et al., 1976). These levels are above the usual water chlorination pH values of 6 to 8.

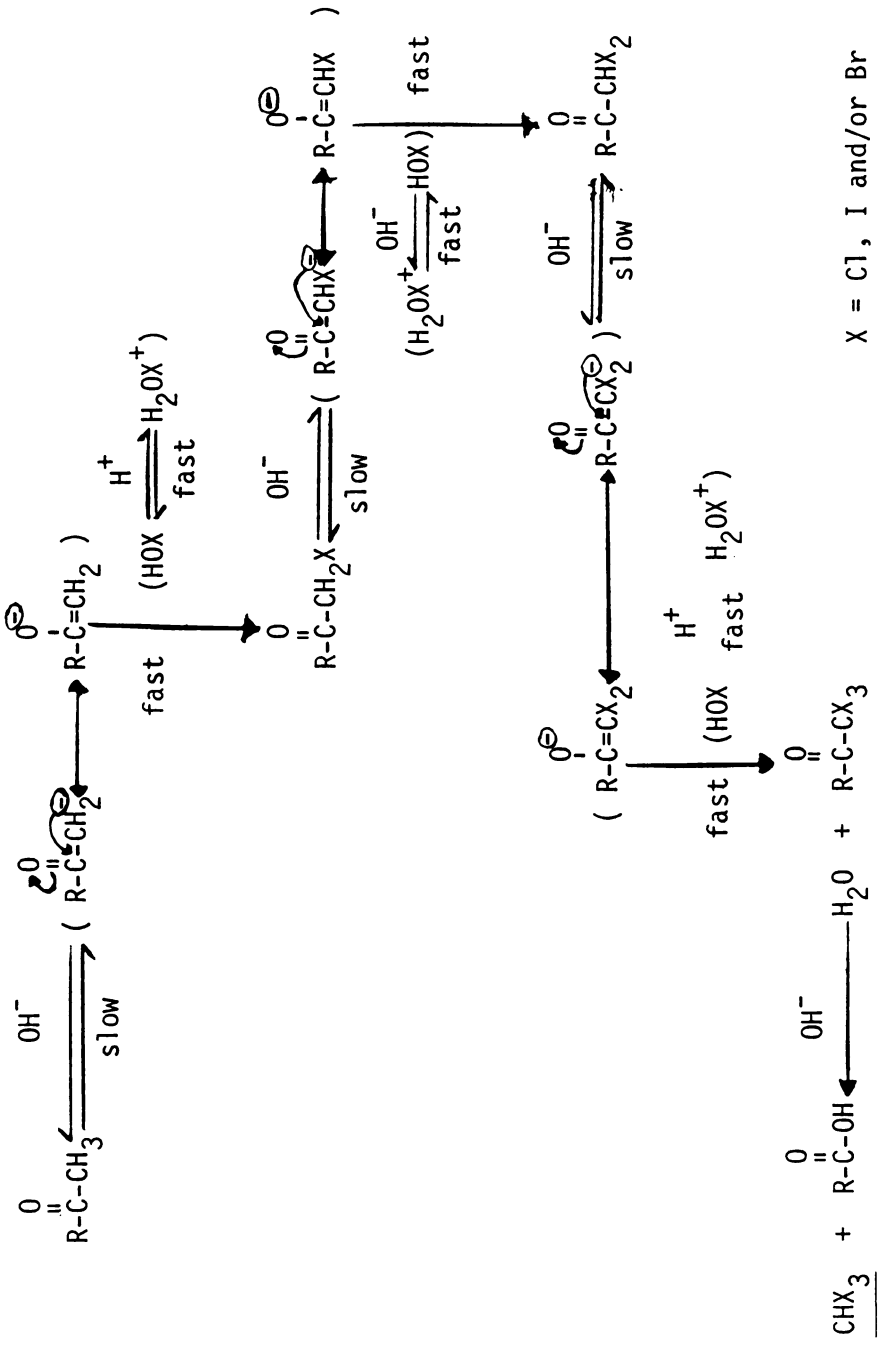


Figure 2. The classical haloform reaction (from Morris, 1976; Morris and Baum, 1978).

The presence of brominated haloforms in chlorinated water has been attributed to two reasons. The first, as suggested by Bellar and coworkers (1974), is the presence of bromine impurities in chlorine. Rook (1974) noted that 100 mg chlorine gas was contaminated with less than 0.04 mg (0.04%) bromine, and concluded that bromine contribution by chlorine was negligible.

The second reason stems from the oxidizing potential of HOCl. The potential is strong enough to oxidize aqueous Br^- and I^-, but not F^-, to their respective hypohalous acids (Bunn et al., 1975). HOBr and HOI then participate in the haloform reaction forming the respective brominated and iodinated haloforms. After spiking raw water with 5 mg/L bromide and 5 mg/L iodide followed by chlorination with calcium hypochlorite (1.2 mg/ml as available chorine), the following haloforms were detected: chloroform, bromodichloromethane, dibromochloromethane, bromoform, dichloroiodomethane, chlorodiiodomethane, iodoform, dibromoiodomethane, bromodiiodomethane, and bromochloroiodomethane. Hypobromous acid reacts faster with ammonia than does hypochlorous acid, and may also react faster with haloform precursors (Morris, 1976). This would account for the greater proportion of brominated haloforms present than could be accounted for by the concentration of natural bromide in raw water (Rook, 1974).

In 1950, Booth and Saunders noted that the following groups of compounds participated in the haloform (iodoform) reaction.

1. $CH_3\text{-}\overset{O}{\overset{\|}{C}}\text{-R}$, when R=H, hydrocarbon radical, COOH and esters, $(CH_2)_nCOOH$ and esters. R cannot be OH, substituted OH, NH_2 or certain substituted amino groups.

2. $CH_3\text{-}\overset{OH}{\overset{|}{C}}\text{H-R}$, when easily oxidized to the above compound.

3. Oximes, when easily oxidized to a methyl ketone.

4. Compounds containing $-COCH_2CO-$, or $-CH(OH)CH_2CH(OH)-$ if not connected to heavily substituted carbon atoms.

5. Quinone or hydroxyquinone with at least one unsubstituted position adjacent to the carbonyl or hydroxyl group.

6. Meta-dihydric phenols, such as resorcinol and phloroglucinol. Tautomerism to the keto-form may account for the reaction.

The possibility that humic substances could be precursors of trihalomethanes during water chlorination was first proposed by Rook (1974). Humic substances are naturally occurring organics responsible for the yellow to brown color in some natural waters (Packham, 1964), and are resistant to microbial degradation (Kuznetsov et al., 1970). Humic substances arise from one or a combination of three sources - water soluble extractions of living woody substances, degradation products of decaying wood, and soil organic matter (Christman and Ghassemi, 1966). Aqueous humic substances can be divided into three fractions - fulvic acid, an HCl-soluble fraction; humic acid, an HCl-insoluble and ethanol-insoluble fraction; and hymatomelanic acid, an HCl-insoluble and ethanol-soluble fraction (Black and Christman, 1963). Weber and Wilson (1975) suggest that the non-acidic ester group of humic acid breaks down by hydrolysis into an aromatic acid and an alcohol containing a carboxyl group, thus forming fulvic acids. This suggestion could explain the higher molecular weight of humic acid, greater number of alcohol hydroxyl groups in fulvic acids, and the similar phenol hydroxyl values of both acids.

Both humic and fulvic acids react with free chlorine to produce haloforms. Commercially available humic acid, when suspended in water

at pH 7 or dissolved at higher pH then readjusted to pH 7, had THM formation rate curves similar to those observed during chlorination of natural waters (Stevens et al., 1976). At both pH 6.7 and pH 9.2, the haloform reaction was complete after 100 hours. Of the available carbon in humic acid, 0.7% and 1.4% react forming haloforms at pH 6.7 and pH 9.2, respectively (Stevens et al., 1976). The increase in THM yield with increasing pH, may be explained by the presence of reactive sites on humic acids, normally unreactive at neutral pH, becoming reactive at higher pH values.

Yields of haloforms from chlorination of fulvic acids vary from 0.3% to 0.9% of available carbon depending upon chlorination conditions (Rook, 1976). After chlorine (870 mg/L) was in contact with fulvic acid (250 mg/L) as total organic carbon (TOC) for four hours at 10°C, a large increase in THM concentration was noted at pH values between 8 and 10. The reaction was less affected by a pH change in the range from 6 to 8 (Rook, 1976).

Oliver and Lawrence (1979) and later Oliver and Visser (1980) reported a small difference for THM formation between humic acids and fulvic acids. The reactions were carried out at pH 11 and 20°C for 72 hr. The chlorine concentration was 15 mg/L and humic material concentration was 1 mg/L as TOC. Under different reaction conditions (pH 6.5, 20°C, 100 hr, chorine dose of 10 mg/L, and humic material concentration of 2 mg/L as TOC) haloform yields from fulvic acids was less than half that of humic acids (Babcock and Singer, 1979).

Ultimately fulvic acids may be the major source of THM precursors. From evaluation of ten 20 or 40 liter samples of raw water, source or sources unknown, fulvic acid was determined to represent about 87% of

the humic substances. Hymatomelanic acid and humic acid account for only 12% and 1%, respectively, of the humic substances (Black and Christman, 1963). These findings agree with Packham (1964) who found fulvic acids to be the major organic constituent of colored waters.

The molecular structures of humic substances are unknown; however, various models have been proposed. The first model, as described by Kuznetsov (1970), is for humic acid. The humic acid molecule may contain an aromatic nucleus with cyclical and chain-form organic nitrogen and carbohydrates covalently bound to it.

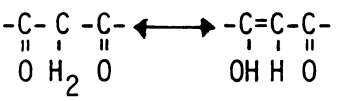
The second model is for fulvic acid, and consists predominantly of an aliphatic or alicyclic backbone (Anderson and Russell, 1976). A polymaleic acid model was proposed with unsaturation and vicinal carboxyl groups present. The polymer also contained phenolic derivatives, polysaccharides, ammonium ion and amino acid residues, but these groups were determined to be of less importance.

Khan and Schnitzer (1972) determined, contrary to the findings of Anderson and Russell (1976) that the major oxidation products of humic and fulvic acids were predominantly aromatic, that is, aromatic carboxylic acids and phenolic acids. Aliphatic carboxylic acids were also found but at a much lower concentration. Methylation of humic substances resulted in degradation products that could be isolated. Since methylation is known to reduce hydrogen-bonding, Khan and Schnitzer (1971) proposed that humic substances consist of "phenolic and benzene carboxylic acids joined by hydrogen-bonds, on which alkanes, fatty acids, and dialkyl phthalates are adsorbed."

Various degradation products of fulvic acid and other humic substances have been isolated (Christman and Ghassemi, 1966).

Chlorination studies have been performed on many of these degradation products. The results of some of these studies are given below.

Diketoalicyclic rings give a positive haloform reaction when the keto groups are beta to each other (Rook, 1976). Dimedone, 1,3-indandione and 1,3-cyclohexanedione are examples of such compounds. Like methylketone THM yield increases with increasing pH values because alkaline conditions favor the enolization reaction



thus THM formation.

Resorcinol (meta-dihydroxybenzene) also reacts in the haloform reaction (Rook, 1977). Because hydroxyl groups are ortho and para activators, they doubly activate the carbon atom to favor formation of a carbanion and electrophilic halogenation. Rook (1977) proposed a reaction mechanism (Figure 3) based upon the observation by Moye (in Rook, 1977) of pentachlororesorcinol as a product of the chlorination of resorcinol with nonaqueous chlorine.

Resorcinol, as found in natural waters, may be substituted by R_1, R_2, and R_3, which could be the fulvic acid matrix once or twice, H, OH, OCH_3 or COOH. Chlorination of the activated carbon (I) is rapid. The intermediate carbanion (II) can be rapidly protonated or halogenated to compounds III and IV, respectively. Hydrolytic or oxidative cleavage of compounds III and IV is responsible for most of the known degradation products of fulvic acids. Cleavage occurs along dotted lines a, b and c. The resulting products, depending upon the groups present at the R positions, include chloroform, methylene

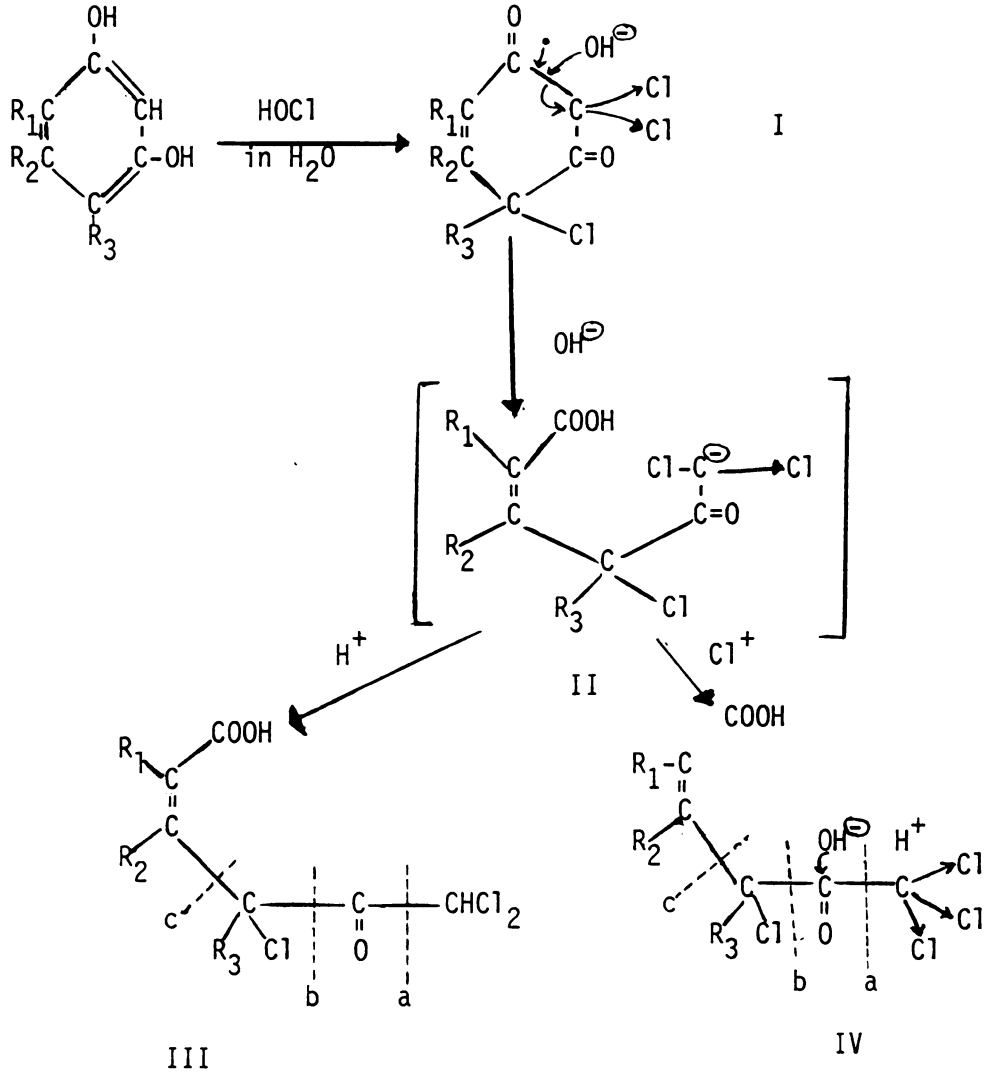


Figure 3. Proposed degradation pathway of fulvic acids and resorcinol. Cl^+ represents in a simplified way any electrophilic halogenating species of the series XOH_2^+, H_2, HOX, X_2O where X = I, Br or Cl (from Rook, 1977).

chloride, tetrachloroacetone, pentachloroacetone, hexachloroacetone, chloral, dichloromaleic acid and trichloroethylene.

The presence of a stable chlorinated cyclopentene, such as 3,5,5-trichlorocyclopent-3-ene-1,2-dione, upon chlorination of resorcinol, was detected by Christman and coworkers (1978). The quantity of this compound increased as resorcinol decreased, and along with chloroform appeared to be the major product of chlorination. At molar chlorine to carbon ratios below the optimal ratio for chloroform production (1.14), cyclopentene exists. As the reaction endpoint of 1.14 Cl_2/C is approached, the cyclopentene begins to disappear and is gone when the endpoint exists. The formation of stable cyclopentene does not agree with the postulated reaction mechanism of Rook. The product does, however, confirm Rook's suggestion that the carbon between the two hydroxyl groups becomes the chloroform carbon atom.

Alkaline conditions also increase the reaction rate by favoring phenoxide ion formation (Rook, 1977) which is a stronger activator than is an aromatic hydroxyl group. The presence of the phenoxide ion may be why THM formation rate increases with increasing pH values.

Substitution of the meta-dihydroxybenzene compound with various groups can alter chloroform yield (Norwood et al., 1980) by altering the activation of the carbon ortho to the two hydroxyl groups. At pH 7 and 25°C, chloroform yield from resorcinol, or a molar basis, was 88%. The addition of a methyl group at the position meta to the two hydroxyl groups did not significantly affect chloroform yield (85%). The replacement of the methyl group with a carboxyl group, however, reduced chloroform yield to 45%. Meta-dimethoxybenzene, a diester, produces much less chloroform since keto-enol stabilization reaction does not

occur as readily. The chloroform yield for 3,5-dimethoxybenzoic acid was 0.9%. Unsaturated alkyl side chains tend to increase chloroform yield. The presence of a hydroxyl group on the carbon inbetween the two hydroxyl or methoxy groups greatly decreases chloroform yield by interfering with keto-enol stabilization. For example, ortho- and para-dihydroxybenzenes yield much less chloroform than meta-dihydroxybenzene, 0.5%, 1.5%, and 85%, respectively at pH 7, because double activation of any carbon does not occur for the ortho- and para-compounds (Rook, 1977). Chloroform yield for both these compounds increases somewhat at higher pH values, but not to the 100% yield observed for meta-dihydroxybenzene.

Quinones and compounds oxidized to quinones, such as tannic acid, could undergo degradation by a pathway similar to that of resorcinol as proposed by Rook (in Youssefi and Zenchelsky, 1978).

Citric acid is another possible precursor which has been found in natural water (Bjork, 1975) and in waste water (Afghan et al., 1974). Unlike most proposed precursors, chloroform yield from citric acid reaches a maximum at pH 7 rather than at more alkaline conditions (Larson and Rockwell, 1978). The proposed reaction mechanism is given in Figure 4. According to this scheme, the rate limiting step would be the oxidative decarboxylation of citric acid. If this reaction mechanism requires the stabilization of the citrate trianion, the pH of maximum reaction would be halfway between the pH of maximum trianion concentration (pK_a 6.40) and hypochlorous acid concentration (pK_a 7.49), or pH 6.94, a nearly neutral pH (Larson and Rockwell, 1978).

The chlorination of amino acids leads to the formation of dichloroacetonitrile (Maugh, 1981b) which decomposes to form

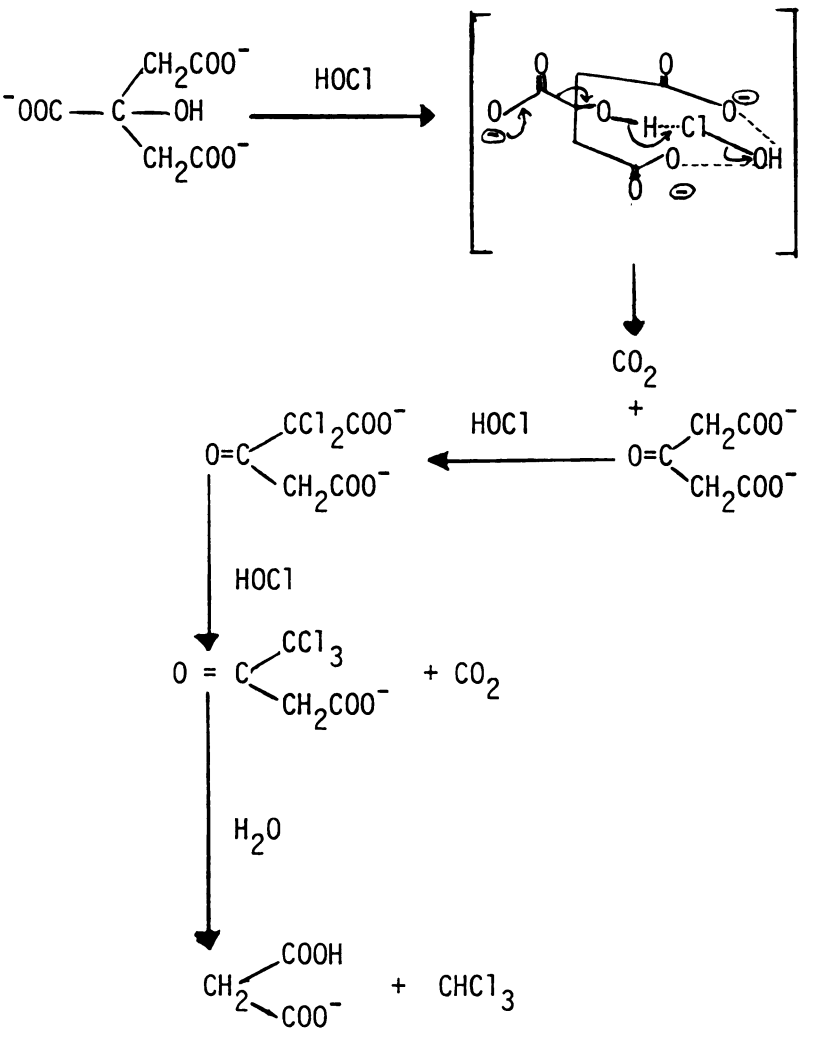


Figure 4. Proposed mechanism of $CHCl_3$ production from citric acid (from Larson and Rockwell, 1978).

chloroform. Amino acids occur in natural waters usually associated with the humic material at concentrations equivalent to about 1% of the carbon fraction (Lytle and Perdue, 1981).

Organic compounds containing the nitrogen-bearing pyrrolic ring, such as chlorophyll, produce chloroform under increased alkaline conditions (Morris and Baum, 1978). Like those in phenol, the hydrogens alpha to the nitrogen are activated and become the site of electrophilic halogenation. Significant haloform formation occurs only at elevated pH.

Algae upon chlorination yield $CHCl_3$, though not to the extent of algal by-products (Hoehn et al., 1980). Algal extracellular products, such as D-mannitol (Crane et al., 1980), yield THM's upon chlorination (Hoehn et al., 1980). From studies with extracellular products, it was found that as much chloroform per unit carbon was formed as from humic and fulvic acids, and the THM yields are greater under more alkaline conditions than at neutral pH (Hoehn et al., 1980). Due to the ease of bacterial degradation, algal by-products may not exist very long (Hoehn et al., 1980), thus their contribution to THM formation may be small.

Therefore, a large variety of compounds normally found in natural waters can react with chlorine to form haloforms. Not all the aforementioned compounds need to be associated with the humic fraction because fulvic acid and related compounds appear to be the major THM precursors. The contribution of each compound depends upon its relative concentration and reactivity as well as the chlorination conditions such as chlorine concentration and pH. These same conditions also determine which other halogenated compounds are produced (Morris and Baum, 1978; Norwood et al., 1980).

THM Level Regulated

Based upon the Safe Drinking Water Act (Public Law 93-523), an amendment to the Public Health Service Act (U.S. Congress, 1974), the EPA has the authority to establish the National Primary Drinking Water Regulation for those chemicals the EPA administrator determines may be harmful to human health. Causative proof of adverse effect is not necessary to establish a regulation. Since many organic contaminants are present at only trace concentrations, this factor is important. A significant health risk may exist since exposure is chronic. Adverse human health effects are determined from epidemiological and toxicological evidence (U.S. EPA, 1978). The Safe Drinking Water Act requires the EPA to set regulations which protect human health to the maximum level using economically feasible treatment methods available in 1974.

On November 29, 1979, EPA amended the National Interim Primary Drinking Water Regulations by establishing a Maximum Contaminant Level (MCL) of 100 μg/L for total trihalomethanes (U.S. EPA, 1979). This regulation affects community water supply systems servicing 10,000 or more people.

Within one year of the date of promulgation, water suppliers servicing 75,000 or more people are required to monitor THM levels for one year. The following year each system must comply with the set MCL. Three years after promulgation water suppliers servicing 10,000 to 75,000 people must monitor THM levels for one year. The following year the established MCL must be complied with.

The Study

Due to the health concerns and EPA's regulations, the amount of trihalomethanes present in Michigan's public water supply is of interest. A survey was conducted of 40 water treatment plants on a one time basis. The results are compared to those of the National Organics Reconnaissance and National Organics Monitoring Surveys, and are examined for a possible geographic distribution pattern. Since treatment conditions affect THM yield, results were analyzed to determine if THM yield is related to water source and chlorine dose.

METHODS AND MATERIALS

Treatment Plant Selection

An index summary and addresses of treatment plants were supplied by the Michigan Department of Public Health (1979, 1980). From this supplied information 173 treatment plants were selected as possible participants because water chlorination was practiced, and when supplied by other treatment plants, the intake water was not already chlorinated. Each of the 173 treatment plants were sent a letter (Figure A-1) requesting their cooperation in the survey and a preliminary questionnaire (Figure A-2) requesting information about their chlorination procedure. The 40 treatment plants that agreed to participate were sent a sampling kit, sampling instructions (Figure A-3) and a second questionnaire (Figure A-4).

THM Sampling and Extraction

Water sampling and organohalide extraction are given in Figure A-3. Organohalide extraction was a modified liquid-liquid extraction method of Mieure (1977) and Richard and Junk (1977) developed by Kaczmar (1979). Fisher's certified methylcyclohexane lot number 792990 was used to extract the organohalides from the aqueous solutions.

Analytical Procedure

Instrument: Varian 1800 Gas Chromatograph

Detector: Tritium foil electron capture

Standing current: 1.184×10^{-8} amps

Column: 2 m x 3 mm i.d. nickel column packed with 10% squalane on chromosorb W-AW

Column temperature: 80°C, isothermal

Injection: On-column (injection precision curve for chloroform-Figure A-5)

Injection port temperature: 200°C

Detector temperature: 165°C

Carrier gas: nitrogen, head pressure of 32 psi

Sample size: 2 µl

Mixed standards were obtained from Environmental Monitoring and Support Laboratory, U.S. Environmental Protection Agency and were called Water Supply Quality Control Check Samples for Volatile Organics. Stock solutions containing chloroform (1369.2 µg/L), 1,2-dichloroethane (544.2 µg/L), 1,1,1-trichloroethane (223.8 µg/L), trichloroethylene (379.4 µg/L), carbon tetrachloride (251.4 µg/L), tetrachloroethylene (175.2 µg/L), bromodichloromethane (238 µg/L), dibromochloromethane (343 µg/L) and bromoform (284.8 µg/L) were made up monthly in methylcyclohexane. Working standards were made by serially diluting stock solution with methylcyclohexane. Eluted peaks were identified by comparing elution time with that of individual organohalides obtained from RCK-001 Organohalide Kit Lot Number I-139 (Analabs, Inc., New Haven, CT). Elution order is given in Figure A-6.

Statistical Analyses

Analysis of covariance and Student's t test were used to determine significance of chlorine dose on total trihalomethane levels. One-way analysis of covariance and Bonferroni's t-test were used to determine the effect of water source on total trihalomethane levels after chlorine dose effect was accounted for. When chlorine dose effect was not accounted for, analysis of variance and Bonferroni's t-test were used to determine the effect of water source on total trihalomethane level (Gill, 1978a; Gill, 1978b; Gill, 1981).

An attempt was made to develop a model for predicting TTHM levels in drinking water using a computer program for multiple regression (SAS Institute Inc., 1979). Four approaches were used to analyze the data: stepwise forward, stepwise backward, stepwise minimum, and stepwise maximum.

RESULTS

General Results of Survey

Information from questionnaires - including sampling date, water source, chlorine dose, residual chlorine concentration, water temperature, pH, turbidity, color, and treatment procedure - along with trihalomethane survey results are given in Table B-1 (parts I and II). Total trihalomethane levels, in the finished water of 40 Michigan drinking water plants, ranged from undetectable to 281.8 µg/L with a mean of 44.0 µg/L and a median of 20.5 µg/L.

The mean, median, and range concentrations of the four individual trihalomethanes detected in finished water are given in Table 3. Both range, median, and mean values of individual trihalomethanes decreased with decreasing chlorine atom presence in each trihalomethane; chloroform was present at the highest concentrations followed by bromodichloromethane, dibromochloromethane, and finally bromoform.

Table 3. Mean, median and range of trihalomethane levels found in finished water of forty Michigan drinking water plants.

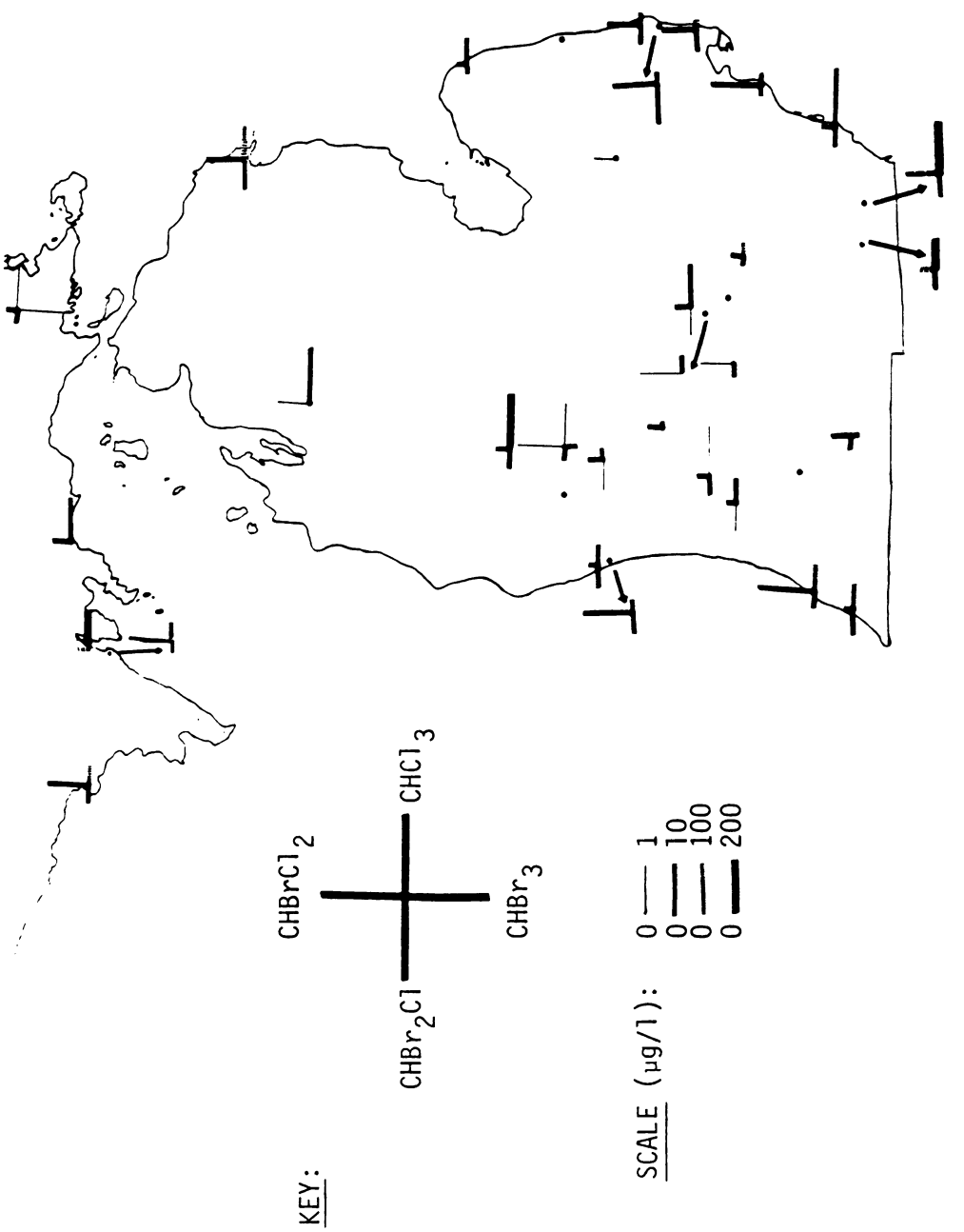
Contaminant	Concentration (µg/L) Mean	Median	Range
$CHCl_3$	33.4	16.7	UD*-201.4
$CHCl_2Br$	6.4	2.7	UD - 54.2
$CHClBr_2$	4.1	2.2	UD - 39.6
$CHBr_3$	0.1	UD***	UD - 1.6
TTHM**	44.0	20.5	UD -281.8

*UD, undetectable
**TTHM, total trihalomethane
***Only three treatment plants had bromoform detected in finished waters.

Geographic Distribution

The trihalomethane levels in the finished waters of the surveyed Michigan water treatment plants are given by location of treatment plants on an outline map of Michigan (Figure 5). Treatment plants located along the shores of the Great Lakes and connecting waters tended to have higher trihalomethane levels than those located inland. Four treatment plants located inland from the Great Lakes had trihalomethane levels in finished water comparable to those of treatment plants located along the Great Lakes. These four treatment plants received river or inland lake water.

Figure 5. Geographic distribution of levels of four trihalomethanes found in finished water of 40 Michigan water treatment plants.


KEY:
$CHBrCl_2$
$CHCl_3$
$CHBr_2Cl$
$CHBr_3$
SCALE (µg/l):
0 — 1
0 — 10
0 — 100
0 — 200

Effect of Water Source and Chlorine Dose on TTHM Levels

The results of statistical analyses are given in Appendix C. Total trihalomethane level in finished water is significantly affected by the quantity of chlorine applied to raw water (Figure C-2). The higher the chlorine concentration, the more the TTHM level tends to increase.

Chlorine dose effects were accounted for before determining the effect of water source on TTHM levels (Figure C-3). Of 35 treatment plants surveyed, five received water from a river or inland lake source (surface water), 17 received water from the Great Lakes and connecting waters, and the final 13 plants received groundwater (Table C-1). Five treatment plants were not used for statistical analyses since chlorine dose information was not available for these plants. Finished water obtained from surface water had TTHM levels significantly different from those of Great Lakes ($P < 0.01$) and groundwater ($P < 0.01$) (Figure C-4). Treatment plants using surface water tended to have the highest TTHM levels.

TTHM levels were not significantly different when the water source was from the Great Lakes and/or connecting waters or from groundwater (Figure C-4). When the quantity of chlorine applied to raw water was assumed to have no effect on TTHM levels, mean TTHM levels in finished water obtained from Great Lakes or connecting waters was significantly different from that when groundwater was the source ($P < 0.05$) (Figure C-5). When the Great Lakes was the water source, TTHM levels were higher than levels from groundwater.

Predicting Trihalomethane Levels

A model for predicting trihalomethane levels in drinking water was developed using multiple regression. The independent variables used were those most often supplied by the treatment plant operators: chlorine dose, hydrogen ion concentration, turbidity, and temperature. Only 26 plants supplied data for all four variables.

The models developed are given in Table D-1. Only two variables, chlorine dose and hydrogen ion concentration, helped explain the variability in TTHM levels observed in the data. When chlorine dose is the only independent variable used, the model is

$$[\text{TTHM}] = -5.74 + 23.08\ (\text{chlorine dose}).$$

The model has a Fisher's exact test value of 96.12 which is highly significant at $p < 0.0001$. This model explains 80% of the variability found in the TTHM levels.

Alone, hydrogen ion concentration does not significantly explain the variability in TTHM levels. When added to the model with chorine dose --

$$[\text{TTHM}] = -13.5 + 22.24\ (\text{chlorine dose}) + (2.38 \times 10^{8})\ \text{HCON}$$

-- the model explains an additional 3% of the variability in TTHM levels for a total of 83%. This model is significant with a Fisher's exact test value of 57.06 and $p < 0.0001$. The addition of either of the two remaining variables, turbidity and temperature, added less than 1% to the percent variability explained.

DISCUSSION

The median and range TTHM values for the Michigan survey (Table 3) are lower than both the NORS and NOMS values (Table 1). Many possible reasons exist for this difference; two, however, are particularly relevant. First, water sampling for the Michigan survey took place from November through March when less trihalomethanes are formed due to a combination of lower water temperature and less organics available for reaction (Stevens et al., 1976; Young and Singer, 1979; Otson et al., 1981). Sampling for the NOR survey took place from late January to late April (Symons et al., 1975). The time period is about the same, but the NORS sampling often occurred after the spring thaw. Second, the NOR survey analyzed proportionally more surface waters and less ground- and Great Lakes waters than this survey. Surface waters, as will be discussed later, contain more organics available for THM formation than do ground- or Great Lake waters.

In addition to the trihalomethanes, two alkylhalides, carbon tetrachloride and tetrachloroethylene, were detected in chlorinated water (Table B-1, part II). While both compounds were infrequently detected in raw and finished waters, in some finished waters where total trihalomethane levels exceeded 60 μg/L, the concentrations of these compounds were often greater than in raw water. In the NOR survey carbon tetrachloride and 1,2-dichloroethane, but not tetrachloroethylene, were detected in a small proportion of chlorinated

waters (Symons et al., 1975); however, due to 1,2-dichloroethane and 1,1,1-trichloroethane eluting together, 1,2-dichloroethane could not be quantified in this survey.

No one factor determines the amount of trihalomethane ultimately formed during chlorination; however, chlorine concentration and concentration of organics seem particularly important. Surface water, generally, has a higher total organic carbon content than does groundwater (Symons et al., 1975). Oliver and Lawrence found levels of THM precursors to be "relatively low" in Great Lakes water (in Anon., 1977). Differences in total organic carbon content between the three sources of water could account for the observed differences in the amount of trihalomethanes formed. According to the results, however, the difference between THM formation in Great Lakes water and groundwater may be due more to chlorine dose than to organic load, because TTHM levels significantly differed only when mean TTHM values were not adjusted for chlorine dose effects (Figures C-3, C-4, and C-5).

The observation that chlorine dose affects THM yield agrees with the findings of other researchers (Zogorski et al., 1978; Moore et al., 1979; Vajdic, 1080; Otson et al, 1981). Otson and coworkers (1981) noted that chlorine dosages and chlorine demands were the dominant factors influencing THM yield when river water was used as the source. A variable amount of the chlorine dose is used to satisfy the demand. The remaining free residual chlorine is available to form trihalomethanes. Therefore, assuming total organic carbon is not limiting, free residual chlorine should influence THM formation more than chlorine dose would. Residual chlorine values are given in

Table B-1, part I. Unfortunately water suppliers often did not specify whether the values were free, combined, or total residual chlorine. No statistical analysis could be made.

A general description of water treatment procedure for each treatment plant is given in Table B-1, part II. Treatment procedure can influence THM formation; however, no conclusions could be drawn from the supplied information. Treatment conditions which affect THM yield are the presence of lime water softening by raising pH, recarbonation of softened water which lowers pH, placement and type of filtration, oxidation, and disinfection, and finally chlorine contact time (Zogorski et al., 1978; Kavanaugh, 1978; U.S. EPA, 1979; McGuire and Suffet, 1979; Brodtmann and Russo, 1979; Norman et al., 1979).

Of the models developed to predict TTHM levels, the two variable model containing chlorine dose and hydrogen ion concentration was the best. It explained 83% of the variability in TTHM levels generated in this survey. However, as a predictive tool, the model is inadequate. To be of value the model should explain nearly 100% of the variability (Geisy, 1982).

The four variables used, chlorine dose, hydrogen ion concentration, turbidity, and temperature, were the variables most often supplied by treatment plant operators. A model using variables available to plant operators would be useful. A model has been developed using those variables known to influence THM formation (Moore et al., 1979).

From this model alone, chlorine dose and hydrogen ion concentration cannot be said to directly influence THM formation because interrelationships with unmeasured variables are unknown

(Geisy, 1982). However, the results do support the previous statistical test showing the importance of chlorine dose in THM formation. Of the unmeasured variables, total organic carbon would probably be the most important. Unfortunately no treatment plant operator supplied data for this variable.

Five of the 40 plants had TTHM levels above the federally set MCL of 100 µg/L (Table B-1, part II). Analyses were done at one point in time, during winter when trihalomethane concentration is expected to be at its lowest. At other times, more favorable to THM formation, such as in spring, summer or fall, temperature and organic load increase and chlorine dose is normally increased, more plants producing potable water with TTHM values about 100 µg/L would be expected.

One hundred and seven Michigan plants, including 13 of the 40 plants surveyed (Michigan DPH, 1979) will be affected by the limit set by the EPA. The population served and TTHM levels measured in the finished water of the 13 affected plants are given in Table 4. Two of the thirteen plants had TTHM levels above 100 µg/L at the time of sampling.

This survey does not statistically represent trihalomethane levels present in finished waters of Michigan water treatment plants. To be a statistically representative survey, a portion of those treatment plants which did not agree to participate would need to have trihalomethane analysis of finished water (Gill, 1981). Of the 951

Table 4. The thirteen surveyed treatment plants which are affected by the EPA regulation, and their population size served and TTHM levels found in finished water.

WSSN	Township Served	Population*	TTHM, µg/L
0710	Big Rapids	12,000	227
0040	Adrian	20,382	151
0160	Alpena	14,000	68
0600	Benton Harbor	16,481	57
2890	Grosse Pointe Farms	11,700	49
4570	Muskegon	44,631	47
2170	Escanaba	15,100	46
4580	Muskegon Heights	17,304	35
5480	Port Huron	37,500	25
5950	Sault Sainte Marie	15,000	15
4340	Michigan State University	50,000	11
3760	Lansing	131,546	4
5520	Portage	25,000	ND**

*Population size obtained from Michigan DPH, 1979
**ND, none detected

Michigan water system suppliers, 488 chlorinate their water (Michigan DPH, 1979). The 40 treatment plants which participated in the survey account for 8.2% of the Michigan treatment plants which practice chlorination. However, this study does indicate the THM levels of those plants surveyed and could be used as a beginning point for an expanded survey. In addition, trends were noted regarding the influence of water source and chlorine dose on TTHM levels.

SUMMARY

1. Total trihalomethane levels, in the finished water of 40 Michigan drinking water plants, ranged from undetectable to 281.8 µg/L with a mean of 44.0 µg/L and a median of 20.5 µg/L. Range and median values are lower than those of the NOR and NOM surveys. Chloroform was usually present at the highest concentrations followed by bromodichloromethane, dibromochloromethane and bromoform.
2. At the time of sampling, five of the 40 plants had TTHM levels above the EPA MCL of 100 µg/L, of which two would be affected by the EPA regulation.
3. Chlorine dose influences the amount of TTHM formed. After adjusting for this influence, treated drinking water supplies derived from surface water had significantly higher TTHM levels than either Great Lakes or groundwater. TTHM levels of Great Lakes and groundwater were significantly different only when chlorine dose effect was not accounted for, perhaps because the organic levels are low compared to surface water. However, when chlorinated, Great Lakes water tended to have higher TTHM levels than did groundwater.

APPENDICES

APPENDIX A

Methods and Materials

Figure A-1. Cover letter for preliminary questionnaire.

MICHIGAN STATE UNIVERSITY

INSTITUTE OF WATER RESEARCH
NATURAL RESOURCES BUILDING

EAST LANSING · MICHIGAN · 48824

April 29, 1980

Dear Sir:

To determine the effect of water chlorination on the haloform content of finished drinking water, I would like to enlist your cooperation to answer the enclosed questionnaire and send water samples to our laboratory for analysis. In return for your cooperation, I will be happy to send you your individual results and a compilation of the total data. Obviously, the more participation we have, the more comprehensive the study will be.

The first step is to answer the enclosed questionnaire which has a pre-addressed, postage-paid return envelope. If you signify that you are also willing to send the water samples, you will then be sent the implements and simple instructions. It will require very little time to fill the six sample vials that will be enclosed with a syringe, needle, and pre-addressed, postage-paid return container.

The sampling procedure entails placing 5 ml of water in each vial and shaking it for one minute. Three vials are for raw water and three for finished drinking water.

Your assistance in this survey is much appreciated. It will give you more information about the quality of your own drinking water and also will enable us to learn more about the overall quality of drinking water in the state of Michigan.

If you have further questions, you can contact me or Eileen A. Nickerson, a graduate student who is working on this project, at either of the following telephone numbers: (517) 353-3742 or (517) 485-8188.

Thank you for your time and cooperation.

Sincerely yours,

Frank M. D'Itri
Professor of
Water Chemistry

tjw

Enclosure

Figure A-1

QUESTIONNAIRE

WATER SOURCE

1. Source of raw water?

   Ground _____ Stream or River _____

   Great Lake _____ Lake or Pond _____ Other _____

   Name of Source: _____

   Location: _____

2. Is this source shared with any other water treatment plants?

   Yes _____ No _____

   Plant names(s): _____

3. What type of intake valve is used? (e.g., buried intake valve): _____

4. If known, what is the organic content of the raw water?

   Dissolved organics _____

   Particulate organics _____

   Total organics _____

5. Is there any industry, agriculture, sewage outfall, chemical dump near the water source?

   Yes _____ No _____

   Name(s): _____

PREVIOUS STUDIES

6. Has any organohalide, in particular halomethane, study ever been performed at the treatment plant before?

   Yes _____ No _____

   Briefly, what were the results, or where can the results be obtained? _____

7. Has the organic content of the water ever been measured before?

   Yes _____ No _____

   Briefly, what were the results, or where can they be obtained? _____

Figure A-2. Preliminary questionnaire for determining which treatment plants may be of value for THM analysis and individual treatment conditions.

Questionnaire
Page 2

8. Would you be willing to send water samples to our laboratory for haloform analysis?

   Yes _____ No _____

TREATMENT PROCESS

9. On a separate sheet of paper, question 6 is presented in the form of a chart. The following may aid you in completing the chart.

   A. Column 1 - Sequence the treatment steps listed in Column 2 from the time the raw water enters the plant until it is ready for distribution. Please mark the first treatment step one (1), the next step two (2), and so on.

   B. Fill in the other columns with the appropriate information or NA if it is not applicable.

   C. If the answer to a question is not known, please write "Unknown" in the proper block.

10. What is the chlorine breaking point? _____

11. How much residual chlorine and organics remain in the finished water?

    Residual chlorine: _____

    Total organics: _____

12. How much water is treated per day or per year? _____

Figure A-2. (Cont.)

Question 6.

Sequence Number	Treatment Steps	Method	Water Temperature and pH	Treatment Time and Any Way Contact is Increased	Chemical: Type and Dosage	Organic Concentration (If Known)	Chemicals Which Conflict With Disinfection Chemicals and Are Present In The Water (e.g., Ammonia)	Comments (Use Opposite Side for Additional Comments)
	Pretreatment							
	Coagulation							
	Sedimentation							
	Filtration							
	Oxidation/ Disinfection							
	Metal Removal							
	Softening							
	Other							
	Other							

Figure A-2. (Cont.)

Figure A-3. Sampling and extraction instructions sent to water treatment facilities.

SAMPLING AND EXTRACTION INSTRUCTIONS

List of Materials:

6 or 9 vials (pre-labeled and containing the extrant)
1 syringe
1 needle
scotch tape
1 plastic bag
1 envelope
mailing label
mailing tape
return postage

Directions: *(Read carefully before you begin.)*

1. To minimize contamination, raw water should be sampled before finished water. Select the three vials labeled Raw 1, Raw 2, and Raw 3, and remove the protective tape from around the cap. Attach the needle to the syringe, and then rinse the unit by drawing raw water fully into and then expelling from the syringe three times.

2. Draw 5 ml of raw water into the syringe and place the water in vial Raw 1. Let the water run down the side of the vial to minimize the volatization of the trihalomethanes (see Figure 1). Cap the vial and shake it for 60 seconds.

3. Repeat step two for the vials labeled Raw 2 and Raw 3.

4. In some instances, additional water sampling is necessary before sampling of chlorinated water. If you have vials labled Pre-1, Pre-2, and Pre-3, you have been chosen to participate in this step. The water to be sampled: _____

_____

Rinse the syringe as before, except rinse it 5 times using this water. Remove the protective tape from the vials labeled Pre-1, Pre-2, and Pre-3. Using this source of water, repeat step 2 for each of the three vials.

5. Proceed to finished water sampling. Select the three vials labeled Final 1, Final 2, and Final 3, and remove the protective tape. Rinse the syringe and needle 5 times. Using finished water, repeat step 2 for each of the three vials.

6. In some instances, 3 control vials containing the extrant have also been sent. These vials are labeled MC-1, MC-2, and MC-3. If you do not have these vials, proceed with step 6. Vial MC-1 should not be uncapped, it is a contamination control. At the time of sampling for raw water, vial MC-2 should have its protective tape removed and then uncapped. Recap the vial and shake it for 60 seconds. Vial MC-3 should be treated as was vial MC-2, except during sampling of finished water.

Figure A-3

7. When the sampling is finished, make certain the caps are tight and wrap scotch tape around the cap end of the vial such that the cap and some of the vial are covered.

8. Replace the vials into the plastic bag and the completed questionnaire into the envelope. Place both the envelope and the bag into the box. Reseal the box with the supplied mailing tape. Cover the old mailing address with the new label and place the supplied postage on the box.

9. Discard the syringe and needle.

10. Thank you.

Figure A-4. Questionnaire used at time of sampling.

QUESTIONNAIRE

1. Name and address of treatment plant: _____

_____

_____

2. Time of sampling: Raw:_____ AM PM _____/_____/_____

Finished:_____ AM PM

3. Retention time between chlorination and sampling of finished water:

_____

4. pH:
   Raw Water: _____

   At Time(s) of Chlorination: _____

   Final Water: _____

5. Temperature:

   Raw Water: _____

   At Chlorination: _____

   Final Water: _____

6. An indication of organic lode:

   Method: _____

   Measure: Raw Water: _____

   At Chlorination: _____

   Finished Water: _____

   (Examples of methods: turbidity, colorimetry, organic carbon, Biological Oxygen Demand, Chemical Oxygen Demand.)

7. Chlorination:

   Chlorine Dose: _____

   Amount of Residual Chlorine: _____

8. Is any method used to reduce volatile organics following chlorination (e.g., aeration, activated carbon)? YES_____ NO_____

   If Yes, method: _____

_____

Figure A-4

Figure A-5. Precision curve of chloroform from injection of a 2 μl mixed standard into gas chromatograph (mean ± standard deviation of 3 injections).

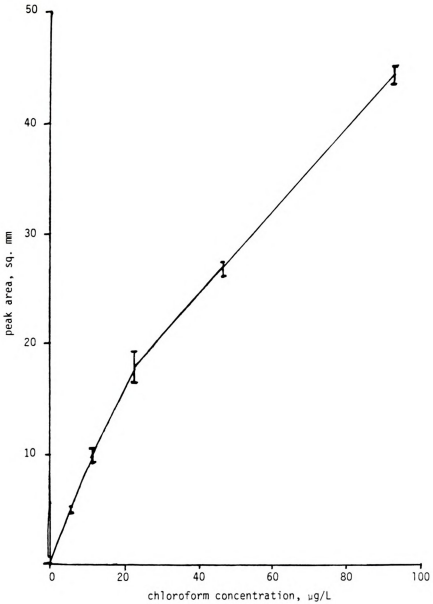

50
40
30
20
10
peak area, sq. mm
0
20
40
60
80
100
chloroform concentration, µg/L

Figure A-5

Figure A-6. Elution order of organohalide mixed standard.

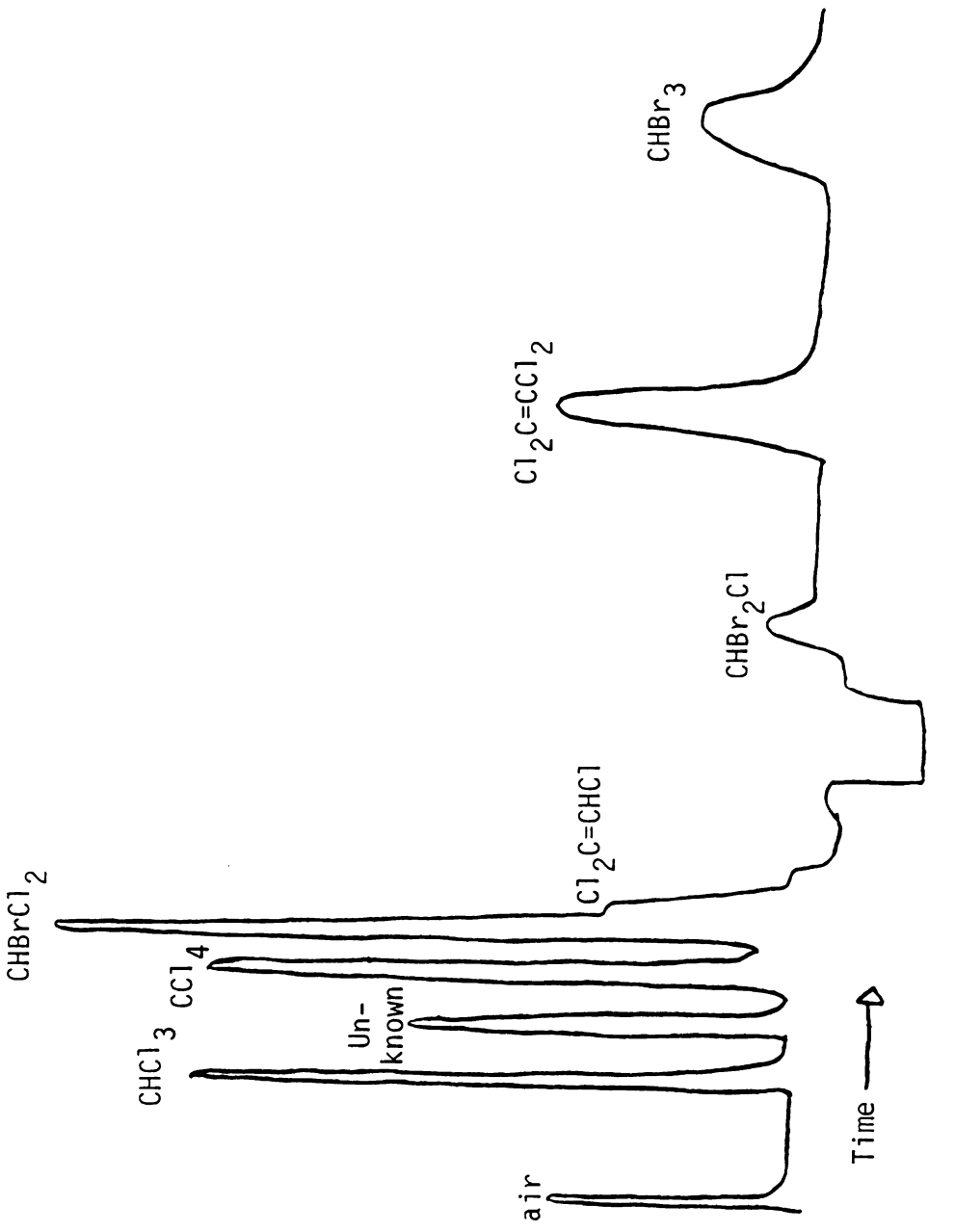

CHBrCl2
CHCl3
CCl4
Un-
known
air
Cl2C=CHCl
CHBr2Cl
Cl2C=CCl2
CHBr3
Time

Figure A-6

APPENDIX B

Survey of Michigan Water Treatment Plants

Table B-1. Survey of Michigan water treatment plants: total trihalomethane content of chlorinated drinking water. (Part I)

WSSN[a]	Treatment Plant	Sampling Date	Source	Chlorine Dose,[b] mg/L	Residual Chlorine,[c] mg/L	Sample[d]	Temperature °C	pH	T[e]	C[f]
1770	Deerfield	11/14/80	River Raisin	14.0	2.0	Raw	5.6	7.7	2.5	--
						Final	7.2	7.1	0.6	--
0710	Big Rapids	2/26/81	Muskegon River	5.8	0.7	Raw	1.0	7.9	12.0	--
						Final[k]	2.0	6.8	0.2	--
0040	Adrian	11/13/80	Lake Adrian on Wolf Creek	3.66	2.1	Raw[t]	10.5	8.1	4.1	--
						Pre[m]	10.5	10.31	1.2	--
						Final[k]	11.5	9.5	0.32	--
2150	Enrico-Fermi Detroit Edison	11/18/80	Lake Erie	6.8	F 2.3 T 2.5	Raw	7.0	7.8	8.7	--
						Pre[k,m]	7.0	7.8	8.7	--
						Final[k]	9.8	9.3	0.11	--
2640	Gladstone	2/27/81	Lake Michigan (Little Bay de Noc)	3.0 pr. 1.4 po.	0.9	Raw	3.0	7.5	3.7[p]	14
						Pre[k,m]	3.0	7.4	3.7[p]	14
						Final[k]	3.0	7.3	0.13[p]	1
4040	Manistique	2/26/81	Indian River	2.8 pr. 0.9 po.	0.8 to 1.2	Raw	3.0	7.3	--	--
						Pre[n]	5.0	7.3	--	--
						Final	4.0	7.1	--	--
0160	Alpena	11/13/80	Lake Huron (Thunder Bay)	3.25	1.6	Raw[k]	3.9	8.1	1.4	2
						Final[k]	5.0	7.6	0.14	0
5470	Port Hope	11/16/80	Lake Huron	3.4	1.5	Raw[k]	3.0	7.6	8.5	--
						Final	5.0	7.1	0.25	--

Table B-1. (Cont.)

WSSN[a]	Treatment Plant	Sampling Date	Source	Chlorine Dose,[b] mg/L	Residual Chlorine,[c] mg/L	Sample[d]	Temperature °C	pH	T[e]	C[f]
0850	Bridgman	3/29/81	Lake Michigan	2.8	1.2	Raw	6.0	7.5	0.20	--
						Final	7.0	7.4	0.19	--
0600	Benton Harbor		Lake Michigan	2.8	0.9	Raw	3.3	7.6	5.0	--
						Final	3.3	7.4	0.35	--
2890	Grosse Point Farms	11/14/80	Lake St. Clair	1.85	0.76	Raw[k]	8.0	7.8	6.0	--
						Final[k]	8.0	7.3	0.09	--
4570	Muskegon	3/19/81	Lake Michigan	1.90	1.12	Raw	1.7	8.3	1.4	5
						Final	2.2	7.5	0.03	1
2170	Escanaba	3/3/81	Lake Michigan (Little Bay de Noc)	2.2	1.0	Raw	2.0	8.2	6.1	2
						Pre[t,m]				
						Final[k]	2.0	7.9	0.08	0
3400	Iron Mountain	3/12/81	Lake Antoine	3.09	F 0.63	Raw	4.4	8.3	0.5	7
						Final[k]	5.6	8.4	0.4	1
4160	Marysville	11/25/80	St. Clair River (From L. Huron)	1.54	1.06	Raw[k]	5.0	8.1	3.0[p]	--
						Final[k]	7.0	7.6	0.13[p]	--
4580	Muskegon Heights	3/18/81	Lake Michigan	2.3	1.1	Raw	3.0	8.2	1.8	3
						Final	3.0	7.4	0.03	0
4090	Marine City	11/18/80	St. Clair River	1.4	0.85	Raw	8.0	8.1	2.4	--
						Final	9.0	7.4	0.10	--
5370	Plainfield	3/17/81	Ground	0.8	0.6	Raw	11.5	6.5	0.18	--
						Final	11.5	9.2	0.19	--

Table B-1. (Cont.)

WSSN[a]	Treatment Plant	Sampling Date	Source	Chlorine Dose,[b] mg/L	Residual Chlorine,[c] mg/L	Sample[d]	Temperature °C	pH	T[e]	C[f]
5480	Port Huron	11/12/80	St. Clair River	2.0	1.0	Raw	6.7	8.0	6.1	--
						Final	7.8	7.5	0.05	--
4560	Munising	2/25/81	Lake Superior	1.82	1.2	Raw	0.0	7.1	0.35	--
						Final	0.0	7.0	0.29	--
7080	White Pine Copper Co.	2/26/81	Lake Superior	1.65	0.9	Raw[k]	0.0	8.7	3.4	15
						Final[l]	2.0	8.5	0.4	0
3670	L'Anse	2/26/81	Lake Superior (Keeweena Bay)	1.06	0.8	Raw	1.0	7.6	1.25	--
						Final	1.0	7.5	0.91	--
1260	Cedar Springs	3/18/81	Ground	1.1		Raw	10.0	7.6	--	--
						Final	10.0	7.4	--	--
5950	Saulte Ste. Marie	2/27/81	St. Marie's River (Upper)	0.8	0.7	Raw	0.75	6.8	0.47	--
						Final	0.75	6.8	0.51	--
4340	Michigan St. University	11/11/80	Ground	1.4	0.5	Raw[k]	12.2	7.3	0.4[p]	--
						Final	13.3	7.5	0.5[p]	--
3560	Kalkaska	3/18/81	Ground		F 0.3	Raw	--	--	--	--
					T 1.1	Final	--	--	--	--
6940	Wayland	3/18/81	Ground	1.0	0.4	Raw	10.0	--	--	--
						Final	10.0	--	--	--
0120	Allegan		Ground	1.40	F 0.55	Raw	12.5	7.4	--	--
					T 1.15	Final	14.2	9.0	--	--

Table B-1. (Cont.)

WSSN[a]	Treatment Plant	Sampling Date	Source	Chlorine Dose,[b] mg/L	Residual Chlorine,[c] mg/L	Sample[d]	Temperature °C	pH	T[e]	C[f]
				a b	a b					
1540	Colon[j]	3/31/81	Ground	0.3/0.2	0.8/0.5	Raw	11.1	9.0	--	--
						Final a	11.1	8.2	--	--
						Final b	11.1	8.2	--	--
3240	Howard City	2/26/81	Ground	1.1	0.3	Raw[k]	10.6	7.0	--	--
						Final	4.4	7.0	--	--
3630	Kincheloe Air Force Base	2/26/81	Ground	0.46	0.13	Raw	11.0	7.2	--	--
						Final	13.0	7.3	--	--
3760	Lansing	12/9/80	Ground	1.9	1.1	Wise	11.1	7.0	5.0	--
						Final	11.7	9.7	0.0	--
		12/9/80	Ground	2.35	0.92	Dye	11.7	7.1	10.0	--
						Final[k]	12.8	9.3	0.13	--
3950	Lowell	3/20/81	Ground	0.5	F 0.6	Raw	11.0	7.6	0.3	--
						Final	11.0	9.0	0.6	--
1340	Charlotte	3/26/81	Ground	0.92	F 0.24	Raw	11.0	7.8	--	--
					T 0.36	Final	11.0	7.8	--	--
0700	Powell	3/2/81	Ground			Raw	8.3	--	--	--
						Final	8.9	--	--	--
1570	Columbiaville	12/2/80	Ground	$\bar{X}$ = 1.97 for Nov.	F 0.3	Raw	8.3	--	--	--
					T 0.7	Final	8.9	--	--	--

Table B-1. (Cont.)

WSSN[a]	Treatment Plant	Sampling Date	Source	Chlorine Dose,[b] mg/L	Residual Chlorine,[c] mg/L	Sample[d]	Temperature °C	pH	T[e]	C[f]
4170	Mason	11/24/80	Ground	~1.0		Park[t]	12.8	7.4	--	--
						Curtis[t]	12.8	7.4	--	--
						Howell[t]	12.2	7.4	--	--
						Final	17.2	7.4	--	--
4710	Newaygo	3/19/81	Ground	15 lbs./ 24 hrs.	F 0.4	Raw	10.0	7.6	--	--
					T 1.8	Final	6.7	7.5	--	--
5500	Port Sanilac	11/22/80	Ground		F 0.5	Raw	10.6	7.8	0.0	--
					T 0.6	Final	11.1	7.9	0.0	--
5520	Portage	3/30/81	Ground		Westfield[t]					
					Final[t]					
					Winterforest[t]					
					Final[t]					
					Milham[t]					
					Final[t]					

Table B-1. Survey of Michigan water treatment plants: Total trihalomethane content of chlorinated drinking water.[r] (Part II)

WSSN[a]	Sample[d]	Organohalide Concentration, µg/L							Treatment Procedure[s]
		CCl_4	$Cl_2C = CCl_2$	$CHCl_3$	$CHCl_2Br$	$CHClBr_2$	$CHBr_3$	TTHM[h]	
1770	Raw	0.16	ND[q]	ND	ND	ND	ND	ND	1-2, 3, 4
	Final	0.24	ND	188	54.2	39.6	ND	281.8	
0710	Raw	0.11	ND	ND	ND	ND	ND	ND	1-2-3-9,
	Final[k]	0.21	ND	201.4	22.4	3.1	ND	226.9	4
0040	Raw[g]	0.13	ND	ND	ND	ND	ND	ND	2, 13, 3,
	Pre[m]	0.12	ND	1.3	0.2	ND	ND	1.5	1, 4, 5
	Final[k]	0.19	0.36	131.0	16.2	4.2	ND	151.4	
2150	Raw	0.14	ND	ND	ND	ND	ND	ND	6, 1, 11, 12,
	Pre[k,m]	0.14	<0.10	73.0	14.8	12.4	ND	100.2	8, 4, 5
	Final[k]	0.14	0.11	98.0	17.2	27.0	ND	142.2	
2640	Raw	<0.06	ND	ND	ND	ND	ND	ND	1, 2, 3-9,
	Pre[k,m]	<0.07	ND	122.0	10.8	0.1	ND	132.9	4-7, 5
	Final[k]	0.06	ND	124.0	11.9	0.1	ND	136.0	
4040	Raw	0.08	ND	ND	ND	ND	ND	ND	1-9-7-2,
	Pre[n]	0.50	0.40	36.4	1.4	ND	ND	37.8	3, 4, 5
	Final	0.72	2.00	78.5	3.0	ND	ND	81.5	
0160	Raw[k]	0.08	ND	ND	ND	ND	ND	ND	1, 2, 3, 4,
	Final[k]	0.12	ND	55.0	7.6	5.4	ND	68.0	7, 8
5470	Raw[k]	0.08	<0.10	ND	ND	ND	ND	ND	1, 2, 3, 4
	Final	0.08	1.44	42.0	12.0	11.0	ND	65.0	
0850	Raw	ND	ND	ND	ND	ND	ND	ND	1, 3, 4, 7
	Final	ND	ND	42.0	11.2	4.4	ND	57.6	

Table B-1. (Cont.)

WSSN[a]	Sample[d]	Organohalide Concentration, µg/L							Treatment Procedure[s]
		CCl_4	$Cl_2C = CCl_2$	$CHCl_3$	$CHCl_2Br$	$CHClBr_2$	$CHBr_3$	TTHM[h]	
0600	Raw	ND	ND	ND	ND	ND	ND	ND	1-2-7-11
	Final	ND	ND	43.9	10.0	2.8	ND	56.7	3, 4
2890	Raw[k]	0.14	ND	ND	ND	ND	ND	ND	1, 2, 3, 4
	Final[k]	0.14	ND	22.0	9.2	17.4	ND	48.6	
4570	Raw	ND	ND	ND	ND	ND	ND	ND	2, 3, 4, 5
	Final	ND	ND	32.4	10.8	4.0	ND	47.2	
2170	Raw	0.08	ND	ND	ND	ND	ND	ND	1, 2, 3, 7,
	Pre[g,m]	0.08	ND	31.6	7.6	2.0	ND	41.2	9, 4, 5
	Final[k]	0.08	ND	36.0	8.0	2.4	ND	46.4	
3400	Raw	0.08	ND	ND	ND	ND	ND	ND	1, 13, 2,
	Final[k]	0.30	ND	34.0	7.2	3.0	ND	44.2	14, 4, 8
4160	Raw[k]	0.14	ND	ND	ND	ND	ND	ND	1, 2, 3, 4
	Final[k]	0.14	ND	21.0	8.0	8.0	ND	37.0	
4580	Raw	0.06	ND	ND	ND	ND	ND	ND	1, 2, 3, 4
	Final	0.06	ND	21.8	8.4	4.4	34.6		
4090	Raw	0.15	0.19	ND	ND	ND	ND	ND	1-2, 3, 4
	Final	0.20	ND	16.4	6.2	3.7	ND	26.3	
5370	Raw	0.06	ND	ND	ND	ND	ND	ND	1, 2-11,
	FInal	<0.06	ND	21.6	1.9	1.4	ND	24.9	4-7-8
5480	Raw	0.10	0.14	ND	ND	ND	ND	ND	1, 2, 3, 4,
	Final	0.15	Trace	15.6	5.7	3.3	ND	24.6	5

Table B-1. (Cont.)

WSSN[a]	Sample[d]	Organohalide Concentration, µg/L							Treatment Procedure[s]
		CCl_4	$Cl_2C = CCl_2$	$CHCl_3$	$CHCl_2Br$	$CHClBr_2$	$CHBr_3$	TTHM[h]	
4560	Raw	0.25	ND	20.8	3.2	0.4	ND	24.4	1, 7
	Final	0.20	ND	17.8	2.7	0.5	ND	21.0	
7080	Raw[k]	0.05	ND	ND	ND	ND	ND	ND	1, 2-9, 3,
	Final[l]	0.05	ND	17.0	2.6	0.3	ND	19.9	4, 7-9
3670	Raw	ND	ND	ND	ND	ND	ND	ND	1
	Final	ND	ND	17.4	2.0	ND	ND	29.4	
1260	Raw	ND	ND	ND	ND	ND	ND	ND	1
	Final	ND	ND	16.0	2.4	0.5	ND	18.9	
5950	Raw[k]	0.12	0.36	ND	ND	ND	ND	ND	1
	Final	0.16	0.24	12.8	2.0	0.5	ND	15.3	
4340	Raw[k]	0.14	ND	2.9	0.2	ND	ND	3.1	1-3-7-8-9,
	Final	0.14	ND	8.1	2.3	0.5	ND	10.9	10
3560	Raw	0.08	0.22	ND	ND	ND	ND	ND	1
	Final	0.16	0.22	10.0	0.6	ND	ND	10.6	
6940	Raw	ND	ND	1.6	2.3	3.2	ND	7.1	1-7-8, 10
	Final	ND	ND	1.0	2.0	3.1	ND	6.1	
0120	Raw	<0.06	ND	ND	ND	ND	ND	ND	13, 2, 3,
	Final	<0.06	ND	4.3	1.5	0.5	ND	6.3	5, 7, 4
1540	Raw	ND	ND	ND	ND	ND	ND	ND	15, 1
	Final[a]	ND	ND	ND	3.2	0.8	ND	4.0	
	Final[b]	ND	ND	ND	3.2	1.8	1.3	6.3	

Table B-1. (Cont.)

WSSN[a]	Sample[d]	Organohalide Concentration, µg/L							Treatment Procedure[s]
		CCl_4	$Cl_2C = CCl_2$	$CHCl_3$	$CHCl_2Br$	$CHClBr_2$	$CHBr_3$	TTHM[h]	
3240	Raw[k]	0.15	ND	ND	ND	ND	ND	ND	1
	Final	0.10	ND	0.8	0.9	2.7	1.6	6.0	
3630	Raw	0.10	ND	ND	ND	ND	ND	ND	1-7
	Final	0.10	ND	0.8	1.1	2.2	0.9	5.0	
3760	Wise	0.10	ND	ND	ND	ND	ND	ND	1-2-13-16,
	Final	0.10	ND	3.4	0.7	ND	ND	4.1	3, 4
	Dye	0.10	ND	ND	ND	ND	ND	ND	
	Final[k]	0.14	ND	2.9	0.9	ND	ND	3.8	
3950	Raw	ND	ND	ND	ND	ND	ND	ND	2-13, 4, 5
	Final	ND	ND	ND	1.0	2.4	ND	3.4	
1340	Raw	ND	ND	ND	ND	ND	ND	ND	1, 7
	Final	ND	ND	ND	0.6	2.2	ND	2.8	
0700	Raw	0.20	ND	ND	ND	ND	ND	ND	1
	Final	0.20	ND	2.1	ND	ND	ND	2.1	
1570	Raw	0.14	ND	ND	ND	ND	ND	ND	16, 1
	Final	0.10	ND	ND	0.4	ND	ND	0.4	
4170	Park[g]	0.08	0.4	ND	ND	ND	ND	ND	1. 7
	Curtis[g]	0.12	0.2	ND	ND	ND	ND	ND	
	Howell[g]	0.08	ND	ND	ND	ND	ND	ND	
	Final	0.12	ND	ND	ND	ND	ND	ND	
4710	Raw	ND	ND	ND	ND	ND	ND	ND	8, 4, 5
	Final	ND	ND	ND	ND	ND	ND	ND	

Table B-1. (Cont.)

WSSN[a]	Sample[d]	Organohalide Concentration, µg/L							Treatment Procedure[s]
		CCl_4	$Cl_2C = CCl_2$	$CHCl_3$	$CHCl_2Br$	$CHClBr_2$	$CHBr_3$	TTHM[h]	
5500	Raw	0.08	ND	ND	ND	ND	ND	ND	1. 16
	Final	0.08	ND	ND	ND	ND	ND	ND	
5520	Westfield[g]	ND	ND	ND	ND	ND	ND	ND	8, 7, 5
	Final[g]	ND	0.4	ND	ND	ND	ND	ND	
	Winterforest[g]	ND	ND	ND	ND	ND	ND	ND	
	Final[g]	ND	ND	ND	ND	ND	ND	ND	
	Milham[g]	ND	ND	ND	ND	ND	ND	ND	
	Final[g]	ND	ND	ND	ND	ND	ND	ND	

[a]WSSN, water supply system number.

[b]Chlorine dose; po - postchlorination dose, pr - prechlorination dose.

[c]Residual chlorine concentration: F - free residual chlorine; T - total residual chlorine. Where there is no initial, the type of residual chlorine was not specified.

[d]Where a name is given instead of "Raw," the name is that of the well where the raw water sample was obtained.

[e]T, turbidity. Units are NTU's (nephelometric turbidity units).

[f]C, color. Units are platinum-cobalt color units.

[g]The results of one water sample instead of the average of three.

[h]TTHM, total trihalomethane.

[j]"b" was further down the distribution line than was "a", hence "b" represents a longer chlorine contact time (the difference in contact time in unknown).

Table B-1. (Cont.)

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[k]The average of two water samples instead of three.

[m]Pre-sample was taken after filtration, but before post-chlorination.

[n]Pre-sample was taken after filtration, but before chlorination. No pre-chlorination was done.

[p]JTU, Jackson turbidity units.

[q]ND, none detected.

[r]Results are the average of organohalide content of three water samples, unless otherwise specified.

[s]Treatment procedure. Treatment steps are given in the order of occurrence. Dashes indicate treatments that occur in the same step. Commas separate sequential steps.

1. pre-chlorination
2. coaqulation
3. sedimentation
4. filtration
5. post-chlorination
6. oxidation
7. fluoridation
8. corrosion control (hexametaphosphate)
9. pH adjustment (soda ash)
10. order not specified
11. pH adjustment (lime)
12. clarification - settling
13. softening
14. recarbonation (liquid carbon dioxide)
15. aquadine
16. metal removal

APPENDIX C

Statistical Analyses.

Effect of Water Source and Chlorine Dose on TTHM Levels

Table C-1. Data used for statistical analyses.

Surface Water Data

WSSN Number*	TTHM** Level (µg/L)	Chlorine Dose (mg/L)
1770	281.8	14.0
0710	226.9	5.8
0040	151.4	3.66
4040	81.5	3.7
3400	44.2	3.9

$\bar{Y}_1 \pm$ S.E.M. = 157.16 ± 17.294 µg/L $\bar{X}_1$ = 6.05 mg/L r_1 = 5

Great Lakes and Connecting Waters Data

WSSN Number*	TTHM** Level (µg/L)	Chlorine Dose (mg/L)
2150	142.2	6.8
2640	136.0	4.4
0160	68.0	3.25
5470	65.0	3.4
0850	57.6	2.8
0600	56.7	2.8
2890	48.6	1.85
4570	47.2	1.90
2170	46.4	2.2
4160	37.0	1.54
4580	34.6	2.3
4090	26.3	1.4
5480	24.6	2.0
4560	21.0	1.82
7080	19.9	1.65
3670	19.4	1.06
5950	15.3	0.8

$\bar{Y}_2 \pm$ S.E.M. = 50.90 ± 9.379 µg/L $\bar{X}_2$ = 2.47 mg/L r_2 = 17

Table C-1. (Cont.)

WSSN Number*	Groundwater Data TTHM** Level (µg/L)	Chlorine Dose (mg/L)
5370	24.9	0.8
1260	18.9	1.1
4340	10.9	1.4
6940	6.1	1.0
0120	6.3	1.4
3240	6.0	1.1
3630	5.0	0.46
3760	4.1	1.9
3760	3.8	2.35
3950	3.4	0.5
1340	2.8	0.92
1570	0.4	1.97
4170	0.0	1.0
$\bar{Y}_3 \pm$ S.E.M. = 7.12 ± 10.725 µg/L	$\bar{X}_3$ = 1.22 mg/L	r_3 = 13

*WSSN, water supply system number.

**TTHM, total trihalomethane

Source of Variation	d.f.	SS_y	SP_{xy}	SS_x
Water Source (unadjusted)	t-1 = 2	$SS_{T(y)}$	SP_T	$SS_{T(x)}$
Plants/Source (unadjusted)	n-t = 32	$SS_{E(y)}$	SP_E	$SS_{E(x)}$
Total	n-1 = 34	SS_y	SP_{xy}	SS_x

$$SS_y = \sum_{i=1}^{t} \sum_{j=1}^{r} Y_{ij}^2 - (Y^2../n) = 142931.91$$

$$SS_{T(y)} = \sum_{i=1}^{t} (Y_i^2./r) - (Y^2../n) = 81329.66$$

$$SS_{E(y)} = SS_y - SS_{T(y)} = 61602.25$$

$$SS_x = \sum_{i=1}^{t} \sum_{j=1}^{r} X_{ij}^2 - (X^2../n) = 204.42156$$

$$SS_{T(x)} = \sum_{i=1}^{t} (X_i^2./r) - (X^2../n) = 84.21496$$

$$SS_{E(x)} = SS_x - SS_{T(x)} = 120.2066$$

$$SP_{xy} = \sum_{i=1}^{n} x_i y_i - \sum_{i=1}^{n} x_i \sum_{i=1}^{n} y_i/n = 3899.159$$

$$SP_T = \sum_{i=1}^{t} (x_i.y_i./r_i) - (x..y..)/n = 2613.457$$

$$SP_E = SP_{xy} - SP_T = 1285.702$$

$$SS_{R(E)} = SP_E^2/SS_{E(x)} = 13751.571$$

$$SS_E = SS_{E(Y)} - SS_{R(E)} = 47850.68$$

$$MS_E = SS_E/(n-t) = 1495.3338$$

Figure C-1. One-way analysis of covariance (unequal replication).

$$H : \beta = 0$$

$$t = b/\sqrt{MS_E/SS_{E(x)}} = 3.0326$$

$$\text{Student's } t : \pm t_{0.025,n-1} = 2.032$$

Since 3.0326 is greater than 2.032, the quantity of chlorine applied to raw water has a significant effect on TTHM levels at the 95% confidence limit.

Figure C-2. Statistical test to determine if quantity of chlorine applied to raw water has an influence on TTHM levels in finished drinking water.

$$\bar{Y}_{AK} = \bar{Y}_K - b(\bar{X}_K - \bar{X}), \text{ where } b = SP_E/SS_{E(x)}$$

$$b = 10.696$$

Surface water	$\bar{Y}_{A1} = 122.4$ µg/L
Great Lakes and Connecting Waters	$\bar{Y}_{A2} = 54.43$ µg/L
Groundwater	$\bar{Y}_{A3} = 24.02$ µg/L

Figure C-3. Mean total trihalomethane values adjusted for chlorine concentration effects.

Bonferroni t-test $t_\beta = (\bar{Y}_{A1} - \bar{Y}_{A2})/\sqrt{mS_E[(1/r_1)+(1/r_2)]}$

Degrees of freedom = n - t - 1 = 31

1. Surface water versus Great Lakes and connecting waters

   $t_\beta = 3.4555$

   Since 3.4555 is greater than 3.181 (t_β, 0.005, m=3), the TTHM levels are significantly different (P < 0.01).

2. Surface water versus groundwater

   $t_\beta = 4.834$

   Since 4.834 is greatr than 3.181 (t_β, 0.005, m=3), the TTHM levels are significantly different (P < 0.01).

3. Great Lakes and connecting waters versus groundwater

   $t_\beta = 2.135$

   Since 2.135 is smaller than 2.532 (t_β, 0.025, m=3), the TTHM levels are not significantly different (P > 0.05).

Figure C-4. Statistical difference between mean TTHM levels in the finished waters when Great Lakes, groundwater and surface water were used as water sources after chlorine concentration effect is corrected for.

Analysis of Variance

$$SS_E = 61602.25$$

$$MS_E = SS_E/n-t = 1925.0703$$

Degrees of freedom = n-t = 32

Bonferroni t-test

$$t_\beta = (\bar{Y}_2 - \bar{Y}_3)/\sqrt{mS_E[(1/r_2) + (1/r_3)]} = 2.709$$

Since 2.709 is greater than 2.526 (t_β, 0.025, m = 3), TTHM levels would be significantly different at 95% confidence limits.

Figure C-5. Statistical difference between mean TTHM levels in the finished waters when Great Lakes and groundwater were used as water sources assuming no chlorine concentration effect.

APPENDIX D

TTHM Predictive Models

Table D-1. Models developed using multiple regression to predict TTHM levels following water chlorination.

β_0	β_1	β_2	β_3	β_4	F*	Prob > F	% TTHM Variability explained
One variable models -							
- 5.7	23				96.12	0.0001	80.02
43		4.41×10^8			3.08	0.0921	1.14
70				-1.32	0.15	0.7033	0.62
57			19		0.09	0.7726	0.35
Two variable models -							
-13.5	22	2.38×10^8			57.06	0.0001	83.23
3.0	23			-1.41	48.15	0.0001	80.72
- 3.0	23		-12		46.47	0.0001	80.16
Three variable models							
- 9.2	22	2.50×10^8	-20		37.44	0.0001	83.62
-15.4	22	2.49×10^8		0.25	36.43	0.0001	83.24
Four variable model -							
-10.5	22	2.56×10^8	-20	0.16	26.82	0.0001	83.63

*Fisher's exact test

Note: To obtain the model, the intercept (β_0) and slope values (β_1, β_2, β_3, and β_4) are substituted into the formula - [TTHM,ppb] = β_0 + β_1(chlorine dose, mg/L) + β_2(hydrogen ion concentration, m) + β_3(turbidity, NTU) + β_4(temperature, °C).

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