federal register

Thursday
November 29, 1979

Part III

Environmental Protection Agency

National Interim Primary Drinking Water Regulations; Control of Trihalomethanes in Drinking Water; Final Rule

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 141

[FRL 1312-2]

National Interim Primary Drinking Water Regulations; Control of Trihalomethanes in Drinking Water

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final Rule.

SUMMARY: This amendment to the National Interim Primary Drinking Water Regulations establishes a Maximum Contaminant Level (MCL) of 0.10 mg/l and associated monitoring and reporting requirements for total trihalomethanes (TTHMs), including chloroform, that are introduced into drinking water by the reaction of naturally occurring substances with chlorine in the course of water treatment. The proposed requirement to utilize granular activated carbon (GAC) or equivalent technology in those public water systems subject to significant contamination by synthetic organic chemicals has been separated from this promulgation and will be reproposed for additional public comment in the near future.

EFFECTIVE DATES: For community water systems serving 75,000 or more persons, monitoring must begin 1 year following promulgation and the effective date of the MCL is 2 years following promulgation. For community water systems serving 10,000 to 75,000 persons, monitoring must begin within 3 years from the date of promulgation and the effective date of the MCL is 4 years from the date of promulgation. Effective immediately, systems that plan to make significant modifications to their treatment processes for the purpose of complying with the TTHM MCL are required to seek and obtain State approval of their treatment modification plans.

FOR FURTHER INFORMATION CONTACT: Joseph A. Cotruvo, Director, Criteria and Standards Division, Office of Drinking Water (WH-550), Environmental Protection Agency, 401 M Street, S.W., Washington, D.C. 20460. (202-472-5016).

SUPPLEMENTARY INFORMATION:

History of Rulemaking

On July 14, 1976, EPA published an Advance Notice of Proposed Rulemaking (ANPRM), entitled "Control Options for Organic Chemicals in Drinking Water" (41 FR 28991 *et seq.*). The ANPRM summarized the many facets of the issue of organic chemicals in drinking water including the legislative background, health effects data, the state of available control technology and costs. Advantages and disadvantages of various regulatory and non-regulatory options were examined, and the ANPRM solicited comments and information regarding the problem and options presented. On February 9, 1978, the EPA published a proposed rule (43 FR 5756, *et seq.*) To amend the National Interim Primary Drinking Water Regulations to include an MCL and associated monitoring and reporting requirements for TTHMs. At the same time, a requirement for the use of GAC or equivalent technology was proposed for application to those drinking water sources subject to significant contamination by synthetic organic chemicals of industrial origin. Subsequently, on July 6, 1978, EPA published a Supplemental Notice of Proposed Rulemaking (43 FR 29135, *et seq.*) soliciting comment on EPA's reassessment of the economic impact analysis for the proposal, providing additional documentation in support of the proposal, clarifying certain aspects concerning the effects of organic chemicals in drinking water, and extending the public comment period from July 31, 1978, to September 1, 1978.

The two Federal Register Notice preambles and the supporting documentation cited therein provided a detailed discussion of EPA's rationale for proposing controls on organic chemicals in drinking water. The subjects covered included: assessments of the sources and occurrences of, and human exposure to, THMs and other organic chemicals in drinking water; discussion of the toxicology and epidemiology studies that relate to possible human health risks; rationale for the selection of the MCL for TTHMs and associated requirements; and a discussion of the control technology, economic impact and air pollution and energy impacts of the proposal. EPA's analyses of these subjects have been revised to incorporate information gained during the public comment period.

A total of 598 written comments were received in response to the proposed regulations of which 391 addressed the subject of THMs. In a number of cases the commenters confused the two different regulations being proposed for organic chemical control. For example, some commenters incorrectly assumed that GAC was proposed as the requirement for control of THMs and objected accordingly.

Public hearings were held between March and July, 1978, in Miami, Florida; New Orleans, Louisiana; Boston, Massachusetts; Los Angeles, California St. Louis, Missouri; Louisville, Kentucky; Washington, D.C. and Dallas, Texas. A total of 259 witnesses testified at the public hearings, and of these, 157 commented on the proposed regulations for THMs. Commenters included water utilities, state and local officials, public interest groups, federal health regulatory and research agencies, engineering consulting firms and individual citizens and scientists. In addition, there were 496 communications from members of Congress, and both the House and Senate Appropriations Committees, and the Council on Wage and Price Stability offered comments on the proposed regulations. The National Drinking Water Advisory Council was also consulted for their comments on the regulations. A number of the comments were duplicative, in that often the same persons or organizations submitted both written and oral comments and such comments often induced inquiries from members of Congress on the same subject. EPA has thoroughly considered all comments received in formulating the final regulations. A detailed breakdown of the comments and the Agency's responses to them are attached as Appendices.

Legal Authority

These final regulations are issued under the authority of the Safe Drinking Water Act, as amended (SDWA), 42 U.S.C. 300f *et seq.*, specifically, sections 1401, 1412, 1445 and 1450. They constitute amendments to the National Interim Primary Drinking Water Regulations (NIPDWR), 40 CFR Part 141, as authorized by Section 1412(a)(1).

As noted in the preamble to the proposed regulations (43 FR at 5759), EPA considered establishing these regulations as Revised Primary Drinking Water Regulations but concluded that they would be more appropriate as amendments to the NIPDWR. This means that the feasibility of control measures under the NIPDWR must be adjudged to have been available as of December, 1974, when the SDWA was enacted. As prescribed by Section 1412(a)(2), these Interim Regulations protect health to the extent feasible, using technology, treatment techniques, and other means which the Administrator determines are generally available (taking costs into consideration) on the date of enactment (of the SDWA).

Although Congress clearly contemplated the comprehensive control of organic chemical contaminants in the

Revised Regulations, the statute nowhere precludes EPA from establishing requirements as amendments to the Interim regulations even after the issuance of the report of the National Academy of Sciences under Section 1412(e). The statute does not require that all regulations subsequent to the NAS report be issued as Revised Regulations. All that is required is that the applicable statutory criteria be met. Given Congress' early concern with the presence of organic chemicals in drinking water, the availability of control measures to reduce the level of TTHMs to 0.10 mg/l since 1974, and EPA's finding that THMs "may have an adverse effect on the health of persons," amending the Interim Regulations to include these requirements as a first step toward controlling organic chemical contaminants in drinking water is clearly authorized at this time.

On February 10, 1978, one day after the publication of EPA's proposal in this rulemaking in the **Federal Register**, the United States Court of Appeals for the District of Columbia Circuit issued its opinion in *Environmental Defense Fund v. Costle*, No. 75-2224, 578 F.2d 337. In that case, EDF sought more comprehensive control by EPA of organic chemicals in the NIPDWR that were promulgated in December 1975. Following a review of the statutory provisions and the legislative history regarding the scope of the Interim Regulations, the Court found that EPA could exercise a degree of administrative discretion in deciding whether to control organic chemical contaminants under the NIPDWR. The Court also stated:

> As we have indicated above, we believe the legislature contemplated that the interim regulations would, where feasible, control every contaminant that may prove injurious to health. The failure of the challenged regulations to do so thus becomes suspect. In light of the clear language of the legislative history, the incomplete state of our knowledge regarding the health effects of certain contaminants and the imperfect nature of the available measurement and treatment techniques cannot serve as justification for delay in controlling contaminants that may be harmful. (578 F.2d at 345).

The Court deferred final resolution of the issue by remanding the record to EPA for a report regarding "significant changes that have occurred, since the promulgation of the interim regulations, in (EPA's) assessment of the problem of controlling organic contaminants in drinking water," and to advise the Court "as to whether it plans to propose *amended interim regulations* in light of newly acquired data" (emphasis added) (578 F.2d at 346). This evidenced the Court's recognition that amendments to the Interim Regulations were not restricted to mere modifications to existing requirements, as argued by one commenter. Following EPA's submission of its February 9, 1978, proposed regulations, the Court affirmed EPA's earlier rulemaking action without prejudice to the filing by EDF of a petition to review any action or inaction of the EPA concerning proposed regulations dealing with organic contaminants and without prejudice to the filing by EDF of a motion to recall the mandate should circumstances warrant such action. (Court's order, dated July 14, 1978). These final regulations directly address the Court's concerns as they were set forth in that opinion.

Summary of the Regulations

Section 141.12 of the Interim Regulations has been amended to add a new maximum contaminant level of 0.10 mg/l for TTHMs. TTHMs in § 141.2 are defined as the arithmetic sum of the concentrations of the THM compounds (trichloromethane (chloroform), dibromochloromethane, bromodichloromethane and tribromomethane (bromoform)) rounded to two significant figures. This MCL is applicable to all community water systems serving 10,000 or more persons that add a disinfectant to their treatment process. The effective dates of the MCL are specified at § 141.6 as two years from the date of promulgation for those systems serving a population of 75,000 persons or more and four years from the date of promulgation for those systems serving a population of 10,000 to 75,000. At this time, systems serving fewer than 10,000 persons are not covered by these regulations unless States exercise their discretion and expand their coverage to these smallest systems.

Under new Section 141.30, systems serving 75,000 or more persons are required to begin monitoring within one year from the date of promulgation of this regulation and systems serving from 10,000 to 75,000 persons are required to begin monitoring within three years from the date of promulgation. No monitoring is required for systems serving fewer than 10,000 persons under the federal regulations, but the States may extend coverage at their discretion.

The minimum total number of samples required to be taken by the system is required to be determined on a per plant basis, with the exception that wells drawing raw water from a single aquifer may, with State approval, be considered on treatment plant. Thus, if a system has only one treatment plant, the minimum number of samples is four samples per quarter; if it has two treatment plants, the minimum is eight samples per quarter; if it has three treatment plants, the minimum is twelve samples per quarter. All samples taken at the established frequency (e.g., quarterly, annually) must be collected on the same day.

Community water systems using surface sources and systems using ground water sources are, at a minimum, required to monitor for TTHMs at quarterly intervals, with a minimum of four samples each quarter for each treatment plant used by the system. Each quarter, the system's sampling scheme must insure that at least 25% of the samples are taken at locations within the distribution system reflecting maximum residence time of the water in the system, and that no more than 75% of the samples are taken at other representative locations within the distribution system. In selecting representative sampling locations for TTHM monitoring, the regulations provide that the system shall take into account the number of persons served, source of raw water and treatment methods used. To the extent possible, representative sampling for systems with more than one treatment plant should reflect the distributed water from each plant separately.

Systems are further required to average the results of all analyses performed per quarter and to report the results to the State, and to EPA if such monitoring requirements have not yet been adopted by the State with primary enforcement responsibility. All samples collected must be used in computing the average, unless the analytical results are invalidated for technical reasons by a responsible official. Compliance will then be determined based upon a running annual average of the quarterly samples.

The regulations also provide that this sampling frequency of four samples for TTHMs per quarter per year may be reduced by the State to a minimum of one sample for TTHMs per quarter per year (for each plant used by the system) if, after the system has monitored for at least one full year in accordance with the original schedule, it can demonstrate to the State that the water it serves is consistently below the TTHM MCL of 0.10 mg/l. This minimum single TTHM sample must be taken at a point in the distribution system that reflects maximum residence time to insure adequate protection. The system would be required to immediately revert back to the "four samples per quarter" sampling frequency if the single TTHM

sample exceeds the standard and such results have been confirmed by at least one check sample, or in the event of any significant change in its source of water or treatment program. The system must continue such program for at least one year before it could be eligible for reduced monitoring again. The regulations also authorize the States (and EPA, where the State does not have primary enforcement responsibility) to increase the monitoring frequencies at their discretion where such is deemed necessary and appropriate to insure consistent compliance with the MCL throughout the distribution system.

Special consideration is given in the regulations to community water systems which draw their water exclusively from groundwater sources by allowing them to have their monitoring requirements reduced by the State at the outset based upon a judgment by the State that such systems are not likely to be subject to TTHM contamination. The regulations require that such a system must demonstrate to the satisfaction of the State based on at least one sample for each treatment plant used by the system that it has a maximum total trihalomethane potential (MTP) of less than 0.10 mg/l. Thus, if the results from at least one MTP sample are less than 0.10 mg/l and after an examination of local conditions, the State may reduce the monitoring requirements of such a ground water system to not less than one sample for MTP per year. "Maximum total trihalomethane potential" is defined under new § 141.2(s). Any system using exclusively groundwater sources whose MTP is equal to or greater than 0.10 mg/l, which results have been confirmed by a check sample, must comply with the four TTHM samples per quarter per year requirement for at least one full year. Thereafter, the monitoring may be reduced by the State to one TTHM sample per quarter if the TTHM levels are consistently less than 0.10 mg/l, or to one MTP sample per year if the MTP is shown to be less than 0.10 mg/l.

Systems are required to report to the State (and EPA until the State adopts these regulations) the results of each quarterly sampling within 30 days of receipt of such results. Once the MCL takes effect, public notification as well as reporting to the State is required whenever the running average of quarterly samples during the previous 12 months indicates that the MCL of 0.10 mg/l has been exceeded.

To ensure the continued microbiological quality of the drinking water as TTHM levels are being reduced, water systems are required to seek and receive State approval of their plans to make significant modifications to their treatment processes. State approval shall be conditional upon inclusion of additional monitoring and other requirements prescribed by the State to assure microbiological quality in accordance with the guidance provided by EPA. Finally, analyses must be performed by approved laboratories and in accordance with EPA specified methods.

Trihalomethanes

As explained in the preamble to the proposed regulations, the THMs found in drinking water are members of the family of organohalogen compounds which are named as derivatives of methane, where three of the four hydrogen atoms have been replaced by three atoms of chlorine, bromine or iodine. Ten distinct compounds are possible by various combinations of three halogenated atoms, one hydrogen and carbon atom. Current analytical methodology applied to drinking has thus far detected chloroform (trichloromethane), bromodichloromethane, dibromochloromethane, bromoform (tribromomethane) and dichloroiodomethane and monitoring methods are currently available for the brominated and chlorinated THMs but not the iodinated THMs because of chemical instability.

The principal source of chloroform and other trihalomethanes in drinking water is the chemical interaction of the chlorine added for disinfection and other purposes with the commonly present natural humic and fulvic substances and other precursors. The actual levels of TTHMs in drinking water, however, will vary depending upon the season, chlorine contact time, water temperature, pH, type and chemical composition of raw water and treatment methodology. Since the natural organic precursors are more commonly found in surface waters, water taken from a surface source is more likely than ground water (with notable exceptions) to produce high THM levels.

Generally, the THM producing reaction is as follows:

Chlorine + (Bromide ion or iodide ion) + Precursors = Trihalomethanes and other Halogenated Compounds

Chloroform is the most common THM found in drinking water and it is also usually present in the highest concentration. In a number of cases, the concentrations of the brominated THMs were found to far exceed the chloroform concentrations. The mixed THMs appear to form by way of an initial oxidation of bromide ion in solution by added chlorine, followed by rapid bromination of the organic precursors. Bromine and chloroform may also be introduced as contaminants of chlorine.

Chloroform and other THMs were first reported in drinking water in late 1974. EPA initiated the National Organics Reconnaissance Survey (NORS) of 80 water utilities, which confirmed that THMs were being formed during chlorination in drinking water treatment process. Concentrations in finished water appeared to be roughly related to the amounts of natural chemicals present in the water.

In late 1975, EPA initiated the National Organics Monitoring Survey (NOMS) in 113 cities. The NOMS demonstrated that considerable amounts of THMs could form in the water after it has entered the distribution systems on the way to the consumer's tap. It also showed that THMs far exceeded the concentrations of other synthetic organic contaminants in finished drinking water, and that brominated THMs could also exceed the chloroform concentrations. Other studies have shown that the TTHMs are only a portion of the chlorinated chemicals generated in water after chlorination. Additional information is contained in EPA's "Statement of Basis and Purpose" accompanying this regulation.

Review of Major Issues

During this rule-making, EPA specifically solicited and received comments on the following major issues: The rationale for setting an MCL for TTHMs and the magnitude of the MCL; the feasibility of and timing for phased reduction of the MCL; the concept of phasing the application of the MCL based upon system size; an alternative of making the MCL applicable to all public water systems and to phase the implementation by a deferred monitoring schedule linked to population size; the method for determining compliance, including the number, frequency and location of sampling sites and the averaging of results; the availability of technology to achieve compliance, and the need for restrictions to assure that biologically safe water would be maintained in the course of achieving TTHM reduction; and the costs incurred by public water systems to achieve compliance with the MCL.

Magnitude and Rationale for the MCL

These final regulations adopt unchanged EPA's proposed MCL for

TTHMs of 0.10 mg/l. The majority of commenters responding to this issue felt that setting an MCL of 0.10 mg/l for THMs lacked supporting justification, both in terms of establishment of the need for a regulation to protect public health and also the numerical value that was proposed, while others supported the proposed MCL and some recommended that a lower MCL be selected. Many argued that an unenforceable goal instead of an MCL should be established, or that the MCL should be higher than 0.10 mg/l.

The Coalition for Safe Drinking Water (CSDW), a member organization of both municipal and investor-owned water utilities formed specifically to comment on EPA's proposed regulations, recommended that an MCL be established only for chloroform and that the MCL should be no lower than 0.3 mg/l. The CSDW presented a number of witnesses at the various public hearings and submitted voluminous written comments on the THM regulation. Among the arguments presented were: That chloroform is not known to be a human carcinogen; that other THMs are not known to be animal carcinogens; that the bioassy of chloroform conducted by the National Cancer Institute was flawed; that a threshold level could be established for carcinogenic risk; that the epidemiological studies purporting to indicate human risk were flawed or misinterpreted; that the cancer risks from chloroform could be considerably lower than those computed using the conservative linear or multi-stage models. One (Roe) stated that chloroform might be beneficial. EPA evaluated the CSDW's comments but found their arguments unpersuasive. A detailed analysis of the CSDW's comments is contained in EPA's response to comments, Appendix A. A summary of their specific comments is presented in Appendix B.

Comments from the National Cancer Institute (NCI), National Academy of Sciences (NAS), the National Drinking Water Advisory Council (NDWAC), the National Institute of Environmental Health Sciences (NIEHS) and federal regulatory agencies such as the Occupational Safety and Health Administration (OSHA), Food and Drug Administration (FDA) and the Consumer Product Safety Commission (CPSC), generally supported EPA's proposal. A summary of their specific comments is presented in Appendix B. They stated that sufficient scientific evidence had been accumulated to conclude that chloroform is an animal carcinogen as shown from a properly conducted bioassay and should be presumed to be a risk to humans and that, as such, prudent public health policy warrants reasonable measures to reduce human exposure. The NDWAC also specifically concurred with the 0.10 mg/l MCL proposal for TTHM. The Environmental Defense Fund (EDF) suggested that a lower MCL would be feasible.

EPA's decision to regulate THM levels in drinking water is based on a number of factors which were extensively discussed in the preambles to its proposal notices of February 9 and July 6, 1978. They include, in summary, the potential human health risks of chloroform and other THMs; the fact that drinking water is the major source of human exposure to THMs; the fact that THMs are the most ubiquitous synthetic organic chemicals found in drinking water in the U.S. and are generally found at the highest concentrations of any such chemicals; the fact that THMs are introduced in the course of water treatment as by-products of the chlorination process and thus are readily controllable; that low cost and feasible means have been generally available since 1974 to reduce their concentrations in drinking water; that monitoring is feasible; and that the THMs are also indicative of the presence of a host of other halogenated and oxidized, potentially harmful by-products of the chlorination process that are concurrently formed in even larger quantities but which cannot be readily characterized chemically.

In concluding that exposure to THMs in drinking water poses a human health risk, EPA followed the four principles on human risk assessment set forth in the 1977 report of the National Academy of Sciences, "Drinking Water and Health," which EPA feels are representative of the consensus of scientific opinion. As stated in the proposal, they are as follows:

1. Effects in animals, properly qualified, are applicable to man.
2. Methods do not now exist to establish a threshold for long-term effects of toxic agents.
3. Exposure of experimental animals to toxic agents in high doses is a necessary and valid method of discovering possible carcinogenic hazards in man.
4. Material should be assessed in terms of human risk, rather than as "safe" or "unsafe."

In the specific case of chloroform and other THMs, EPA has relied primarily on animal studies demonstrating the toxicology of chloroform. These are described in the NAS report, "Drinking Water and Health", and in the "Statement of Basis and Purpose" accompanying this regulation. The bioassay results from studies conducted by the NCI have demonstrated the carcinogenicity of chloroform in both rats and mice. Dr. Arthur Upton, Director of NCI, concluded in his comments that chloroform and other chemicals have been "proven as carcinogens in bioassays." Mechanisms for the metabolism and toxicity of chloroform are being investigated and include information demonstrating covalent binding of chloroform metabolites to DNA and the probable intermediate formation of phosgene as a metabolite.

EPA has also concluded that the available epidemiological evidence relative to THM concentrations or other drinking water quality factors and cancer morbidity/mortality has not been conclusive but is hypothesis generating and at least suggestive of a health risk. The NAS in its review of 13 preliminary epidemiological studies affirmed EPA's interpretation and concluded that the risks were probably small but that important confounding factors could not be distinguished in indirect ecological studies to allow a precise evaluation of the contributions from THMs. They pointed out the lack of sensitivity of epidemiological procedures due to lack of exposure data for individuals, population diversity and mobility, inability to control for all known contributing variables such as smoking, occupational exposures, diet, alcohol consumption, socio-economic and urbanization factors, and the usual 20–40 year latency period required for most cancers. The NAS also pointed out that sufficient evidence was available from animal toxicology studies to conclude that exposure to chloroform did pose a risk to human health. Additional studies are underway. Since epidemiology *per se* cannot "prove" causality, and because it may well be impossible to epidemiologically establish a strong causal association that THMs and related chemicals in drinking water contribute to higher cancer rates, EPA has extrapolated from the results of animal studies to assess the risk posed by THMs to humans.

EPA has also concluded that it would be inappropriate at this time to distinguish between an MCL for chloroform and other THMs. As a family of compounds, the THMs are similar in chemical composition and nature and are formed concurrently during the chlorination of drinking water. Brominated THM levels greater than 0.6 mg/l have been detected in some drinking waters. Their relative distribution in finished water is a

function of the organic and halide precursor concentrations which can be highly variable and unpredictable. The other THMs are under further study in the NCI bioassay program because of human exposure and structural similarity to chloroform. Mutagenicity studies in *Salmonella typhimurium* bacterial test systems have shown that brominated and iodinated THMs are more mutagenic than chloroform. The gas chromatographic analytical method concurrently analyses all four THMs, and treatment methods that would be employed would simultaneously reduce all of the THMs.

Excluding brominated THMs from these regulations would permit a substantial number of communities with low chloroform levels, but otherwise high THM and other by-product contamination, to avoid any improvement of treatment practice and, by implication, water quality.

Even though the toxicology of each of the other THMs has not at this time been as thoroughly studied by the scientific community as chloroform, the available toxicological information, their structural similarities to chloroform, and the fact that effective treatment is generally available to reduce public exposure to these potentially harmful contaminants as well as for chloroform, leads EPA to conclude that it would be inappropriate to exclude them from regulation.

Commenters had suggested that an MCL of 0.30 mg/l for chloroform could be computed as a "safe" level for human consumption by incorporating an uncertainty factor of 2,000 into Roe's "no observed effect dose." EPA has concluded that such an approach is totally inappropriate when dealing with human risk from chronic exposure to a potential carcinogen. That approach assumes the existence of a threshold level for carcinogens below which no risk would exist. It is thus inconsistent with the principles stated by the NAS in "Drinking Water and Health." In addition, 0.30 mg/l is well above the levels that are currently achievable in the large majority of public water systems by generally available methods that are technically and economically feasible. The comment was rejected. These comments and the Agency responses are detailed in Appendix A.

Because of the technical inability to determine a "safe" level for a carcinogen and the conclusion, therefore, that some risk must be assumed at any dose, regulatory agencies have attempted to minimize human exposure to carcinogens to the extent feasible. This approach was endorsed in the comments received from the National Cancer Institute, National Institute of Environmental Health Science, National Academy of Sciences, Consumer Product Safety Commission, National Institute of Environmental Health Sciences, Food and Drug Administration, Occupational Safety and Health Administration, as well as the National Drinking Water Advisory Council. See Appendix B.

EPA's selection of an interim MCL of 0.10 mg/l was based on a balancing of public health considerations and the feasibility of achieving such levels in public water systems in the United States. This balancing reflects the existing and generally available technology for water treatment which relies heavily on the proven use of chlorine to produce biologically safe water. It includes the existence of monitoring methods and trained personnel, economic considerations, and the limited amount of technical assistance available from EPA and the States, but primarily the risks that may be introduced in some cases from possibly inadvisable and improperly managed fundamental changes in disinfection practice.

Thus, the interim MCL should not be construed as an absolutely "safe" level, but rather a feasible level achievable with water treatment technology available since 1974. The preponderance of the current scientific thought on human exposure to substances that have been demonstrated to be carcinogens in animals in appropriate tests is that they be considered potential carcinogenic risks to humans. The presumptions are that human health risk is related to the extent of exposure and that no threshold level without risk can be experimentally demonstrated for a genetically diverse population. Translated into regulatory policy, exposure should be minimized so as to minimize unnecessary risks. Therefore, public water systems should strive to reduce TTHMs and related contaminant concentrations to levels as low as is economically and technologically feasible without compromising protection against the transmission of pathogenic microorganisms via drinking water.

The latest comprehensive information on concentrations of TTHMs in the U.S. drinking water was obtained from the National Organics Monitoring Survey (NOMS) of 113 communities sampled 3 times in 1975–77. This represented a wide range of water types including both surface and ground waters, and waters with minimal and substantial TTHM formation potentials. Mean levels of TTHM for Phase II and Phase III were 0.12 mg/l and 0.10 mg/l, respectively, in samples allowed to react to completion (terminal). Averages of both dechlorinated and terminal samples could be considered estimates of likely concentrations to be found at the tap of the average consumer. These were 0.09 mg/l and 0.08 mg/l, respectively, in Phase II and Phase III. However, maximum TTHM levels ranged as high as 0.70 mg/l and 0.78 mg/l in terminal samples. Therefore, an interim MCL of 0.10 mg/l will result in substantial reductions of TTHM concentrations in many water systems now exceeding the MCL.

Many commenters conceded that TTHMs were undesirable constituents of drinking waters, but preferred that a goal rather than an enforceable MCL should be established. In other words, it was suggested that compliance with a TTHM limit should be optional. However, neither the SDWA nor the facts at hand support such a course of action at this time. The SDWA provides for goals only in the case of the Administrator's list of recommended MCLs (Section 1412(b)(1)(B)), and, even then, the goal is to be selected as the value that would result in no known or anticipated adverse health effects and would allow an adequate margin of safety. Revised regulations must specify MCLs that come as close to the recommended levels as is feasible using the best technology, treatment techniques and other means which the Administrator finds are generally available (taking costs into consideration) (section 1412(b)(3)).

The SDWA clearly requires that EPA take regulatory action by establishing enforceable standards, not merely health goals. Since the issuance of EPA's ANPRM and proposal in this rulemaking, only a limited number of systems have voluntarily reduced the levels of TTHMs in their water supplies. Only in the presence of a mandatory requirement can EPA expect the full commitment in time and resources by community water systems and the oversight by State regulatory agencies necessary to achieve compliance nationally.

MCL Summary

Thus, based on the foregoing considerations set forth in the rulemaking record, the Administrator believes that an MCL for TTHMs of 0.10 mg/l in the Interim Regulations will protect human health to the extent feasible as prescribed by Section 1412(a)(2) of the SDWA. Since the optimum and only totally "safe" dose for any carcinogen would be zero, EPA strongly encourages all public water systems, not only those that exceed the

interim MCL, to implement measures to minimize the amounts of TTHMs and ılated by-products in finished water. THM levels in finished water are a nction of the raw water quality (precursor content) and the sequence of treatments applied. Based upon the performance of developing technologies, it appears that ulitmately many public water supplies with currently high TTHM levels may be able to achieve TTHM concentrations as low as 0.010 to 0.025 mg/l and EPA suggests those values as future goals. The MCL will be reconsidered in the Revised National Primary Drinking Water Regulations based upon an updated assessment of technological and economic feasibility, implementation experience and additional toxicological information.

Population Coverage and Phase-In of the MCL and Monitoring Requirements

The proposed regulations would have initially applied the MCL only to those community water systems serving 75,000 or more people, and would have only required that monitoring data be collected for one year in communities serving between 10,000 and 75,000 people. Systems smaller than 10,000 would not be initially covered. The oposed effective date of the MCL was months after promulgation.

EPA solicited comments on ternative approaches for coverage and implementation, for example by applying the MCL to all systems and phasing-in implementation through a deferred monitoring schedule (i.e., systems larger than 75,000 required to begin monitoring within one year of promulgation, 10,000–75,000 within three years of promulgation, and all other communities within five years).

The majority of commenters felt that the regulations should not be limited to the larger than 75,000 population community water systems, although some agreed that some phasing mechanism would be appropriate. The NDWAC suggested that utilities serving 10,000 to 75,000 should be included beginning three years after implementation of regulations in the larger than 75,000 group. The NDWAC also recommended in its initial comments that implementation in communities smaller than 10,000 should be at the option of the State.

EPA has concluded that the coverage of these regulations should be expanded to include community water systems rving 10,000 or more persons. Systems rving 75,000 or more people are quired to comply within two years of omulgation, and systems serving between 10,000 and 75,000 are required to comply within four years of promulgation.

This still means that systems serving fewer than 10,000 people are not required to comply with the TTHM MCL. However, EPA does not believe that this approach will result in those persons served by the smallest systems being afforded reduced health protection. This is because the great majority (about 80%) of these smallest systems are served by groundwater sources that are low in THM precursor content. The proportion of small community water systems that utilize chlorine is less than that of large systems and transport time within the distribution system, which increases the extent of TTHM formation, is generally shorter in small systems. Therefore, their drinking water is less likely to be subject to TTHM contamination.

Moreover, the smallest systems incur a greater risk of adversely affecting the microbiological quality of their drinking water when steps are taken to reduce TTHMs. The majority of waterborne disease outbreaks attributable to inadequate treatment practice still occur in the smallest systems. Such systems also have limited or no access to the resources and professional expertise needed for TTHM control. Thus, EPA believes that it would be premature to divert their already sparse resources away from improving their disinfection practices by requiring compliance with a TTHM MCL at this time.

It is imperative that any changes in current treatment practice must be carefully supervised and supported by technical assistance from the States or EPA. However, it is not administratively feasible for the States and EPA to adequately supervise the approximately 57,000 systems which each serves communities of fewer than 10,000 people.

The approximately 60,000 community water systems in the U.S. range in size from 25 persons to several million and serve a total of about 213 million people. The 390 systems exceeding 75,000 population serve about 101 million people, and the 2,300 systems between 10,000 and 75,000 serve an additional 66 million people. Thus, the final regulations cover approximately 80% of the U.S. population served by community water systems. Most of these larger systems have at least potential access to the technical personnel needed to safely and successfully carry out any fundamental changes in disinfection practice. The smallest systems serve only 20% of the population but comprise a sufficiently large number of systems to make careful supervision effectively impossible in the short-term. Nevertheless, EPA does not intend that these smallest systems be excluded from coverage of the TTHM regulations indefinitely.

EPA considered specifying monitoring requirements for these smallest systems and/or making the MCL applicable to such systems with an extended timeframe for compliance. However, considerable additional time would have been necessary to insure availability of laboratory capability to handle the increased number of TTHM analyses and adequate State and EPA technical assistance. Therefore, it did not seem prudent to specify requirements now for which compliance would be required so far in the future. The considerable experience that will be gained from the efforts of the larger systems to comply with the TTHM MCL will serve to make compliance by the smaller systems more feasible. For that reason, EPA expects that small systems will be subject to a TTHM MCL under the Revised Primary Drinking Water Regulations when they are established. In those States which choose to exercise their discretion to extend coverage to the small systems, EPA expects that additional phasing may be appropriate within this size category based on greatest likelihood of TTHM contamination, such as by first including those systems with surface water supplies.

Implementation Timing

The majority of commenters on the question of the timing of the effective date of the MCL felt that 18 months after promulgation was inadequate to allow for design and implementation of the most cost-effective treatment system for compliance. They stated that eighteen months would only be adequate if minor modifications were needed. EPA has reevaluated the treatment methods most likely to be used and has concluded that in most cases relatively minor technical modifications will be sufficient to substantially reduce TTHM levels below the MCL. Therefore, a delay in the effective date would not have been justified on this ground.

Other commenters pointed out that insufficient laboratories were available to analyze TTHM samples and that a quality assurance program would need to be developed; some suggested that monitoring should be delayed for those reasons. EPA agrees with those commenters concerned about the availability of sufficient numbers of laboratories capable of providing acceptable analytical data. At this time, only relatively few laboratories have demonstrated the capability of

consistently producing data with the required accuracy and precision.

EPA has, therefore, decided to extend the time frame for initiation of the monitoring requirement for systems serving 75,000 or more persons from the proposed three months after promulgation to one year after promulgation. This will allow additional time for State and private laboratories to develop their capabilities and to become certified to provide data in support of compliance determinations. Since the effective date for initiation of monitoring is one year after promulgation and one year of monitoring results is required to determine compliance, the effective date of the MCL for those systems is established as 2 years after promulgation. To accommodate the large incremental monitoring load, application of the monitoring requirements to the approximately 2,300 systems serving 10,000 to 75,000 persons is established at 3 years following promulgation and the effective date of the MCL in this population range is 4 years after promulgation. Despite these extended deadlines, EPA encourages water systems to initiate monitoring and corrective measures sooner than this schedule whenever it is feasible to do so, especially where high TTHM levels are suspected.

EPA will immediately initiate an interim certification program for State laboratories (and others if appropriate) that will be based on their ability to analyze Performance Evaluation samples which will be provided by EPA's Environmental Monitoring and Support Laboratory (EMSL). Two analytical methods (Purge and Trap and Liquid-Liquid Extraction) have been approved under § 141.30(e) of the regulations and the written procedures are available on request from EPA's EMSL, 26 W. St. Clair Street, Cincinnati, Ohio 45268.

To qualify for Interim Certification, laboratories will be required to demonstrate their ability to analyze the Performance Evaluation samples provided to them to within 20% of the "true value" for each of the THMs as well as for the total of the THMs in the samples, using at least one of the approved methods. As the certification program develops and more laboratories gain expertise, it is likely that the precision and accuracy requirements will become more stringent. A quality assurance program will be established to insure that continued certification is dependent upon the laboratories' continued ability to perform quality analyses.

State Primacy and Exemptions

The time frame of these amendments to the NIPDWR will significantly affect two other statutory provisions of the SDWA: continuation of State primary enforcement responsibility (or primacy) under Section 1413 and the issuance of exemptions from MCLs under Section 1416.

With respect to State primacy, the Agency will shortly be proposing amendments to its State implementation regulations, 40 CFR Part 142, which will provide primacy States adequate time to amend their regulations without jeopardizing primacy while more stringent federal regulations take effect. States are encouraged to begin the process of amending their regulations as quickly as possible. However, no action to withdraw primacy will be taken pending the establishment of new EPA regulations under Part 142.

Under Section 1416(b)(2)(B) of the SDWA, schedules attendant to exemptions from the NIPDWR must require compliance by no later than January 1, 1981 (or January 1, 1983, for systems that enter into enforceable agreements to become part of a regional water system). This will, in most cases, preclude the issuance of exemptions from the requirements promulgated today. Since the issuance of exemptions is discretionary with the State, or EPA where the State does not have primary enforcement responsibility, the unavailability of exemptions *per se* is not believed to be a fatal deficiency in the regulations. Nevertheless, EPA recognizes that some systems may not achieve compliance by the effective dates despite their best efforts. EPA is planning to seek from Congress an extension of the exemption deadlines as they may apply to these regulations when the Agency's implementation of the Act is the subject of oversight hearings. The States and EPA may also exercise their enforcement discretion in those cases where compliance with the MCL for TTHMs is not achieved before the applicable effective date despite the system's good faith efforts to comply.

Summary

Therefore, EPA has accepted the recommendation of the NDWAC and many other commenters to broaden the coverage of the THM regulations and to phase-in its implementation as follows:

Water systems serving more than 75,000 are required to be in compliance by two years from the date of promulgation of these regulations. Systems serving between 10,000 and 75,000 are required to be in compliance by four years from the date of promulgation.

Monitoring must be initiated no later than one year from the promulgation date by those water systems 75,000 or larger, and three years from promulgation by those systems in the 10,000 to 75,000 population range. However, EPA urges that compliance and monitoring be accelerated in those water systems where this is feasible and where assistance is available from the primacy authority, especially where high TTHM levels are suspected.

Compliance with the MCL and monitoring in communities smaller than 10,000 would only be required if the primacy State adopts regulations that are more expansive than these federal regulations. EPA will consider expanding the coverage of THM regulations to include smaller systems when it establishes Revised Primary Drinking Water Regulations.

Monitoring Requirements

The proposed monitoring requirements for systems exceeding 75,000 population included quarterly sampling consisting of at least five water samples collected on the same day. The sampling locations were to be representative of TTHM concentrations at the consumer's tap; no more than 20% to be collected at the entry point of the distribution system, no less than 20% at the extremes of the system and the remaining 60% representative of population density throughout the distribution system. Compliance would be determined by averaging the quarterly values from the preceding 12 months. Surveillance monitoring only for one year was proposed for systems between 10,000 and 75,000 population. This consisted of two samples per quarter to be collected at the entry to the distribution system. One sample would be dechlorinated and the other stored for seven days to permit completion of the chlorination reaction. These final regulations eliminate any distinction (except for timing) between the largest and medium size systems and modify the requirement somewhat.

The majority of the comments on this issue were in agreement with the concept of determining compliance by an annual average of quarterly samples. Others disagreed, arguing that averaging might mask fluctuations, and some felt that averaging results in the distribution system would result in higher exposures to those populations residing in the extremes of the system. A few felt that the extreme values rather than averages should be used to compute compliance. Some commenters suggested that systems using deep ground water should

be exempted because of probable low THM formation potential. Others disagreed with a continued monitoring requirement, even at a reduced frequency, after it had been established that TTHM concentrations were unlikely to approach or exceed the MCL. A number agreed with monitoring requirements but objected to public notification of results.

The intent of the monitoring requirements is to provide a reasonable representation of the normal concentrations of TTHMs and related chemicals at the tap of the typical consumer. Data has shown that there can be wide variation of TTHM concentrations particularly in surface waters and groundwaters with high precursor levels on a day to day basis and that levels at various points in a distribution system can differ markedly. The variations can be due to a number of factors that include seasonal or other changes in precursor concentrations in the raw water, the amount of precipitation and surface run-off, the treatment method, the presence of combined or free residual chlorine, chlorine contact time, pH, temperature and transit time during distribution.

EPA feels that it would be unreasonable at this time to demand the kind of pinpoint control that would be necessary to maintain TTHM levels below a particular figure at all times and at all locations in the distribution system of every water system. This Interim Regulation is intended to reduce the extremes of TTHM concentrations that have been found in some of the nation's public water systems, and thus, to reduce the variability that may occur within a given distribution system. TTHMs in drinking water do not present acute or short-term risks but rather chronic or lifetime risks that increase with long-term exposure. Therefore some variations are tolerable and probably do not contribute to a change in overall risk. Thus, EPA has concluded that an averaging approach is appropriate and the use of a 12 month running average for computing compliance is retained in the regulations.

The frequency of monitoring must be based upon its usefulness for determining the concentrations of TTHMs in finished water. It should also reflect the potential for variability of the contaminant concentration, and this is highly dependent upon site-specific factors such as distance from the treatment plant, source water quality and treatment methods used. These factors are particularly important in selecting sampling locations which will be truly representative of water served to consumers regardless of their location within the distribution system, especially when a system uses more than one treatment plant.

The consensus of the comments was that quarterly monitoring was adequate in most cases but many argued for more samples. Quarterly monitoring has been retained in the regulation because EPA considers this to be the minimum acceptable frequency in those places where the water has a potential for seasonal variability in TTHM levels. EPA strongly urges that States review each water system's monitoring program to insure that the monitoring is reflective of seasonal and other variation factors. More frequent monitoring should be required where this is necessary for adequate consistent year-round control of TTHM levels below the MCL. Such discretion to require more frequent monitoring is provided for in these regulations.

In further response to those comments encouraging more frequent monitoring to reflect variations of water quality in the distribution system, EPA agrees that some conditions lead to a greater potential for wide variations of TTHM levels. For example, if a community water system uses more than one treatment plant to provide water, different water sources may be used as well as different treatment processes, leading to the possibility of widely differing TTHM levels in parts of the distribution system. For this reason, the proposed sampling scheme has been changed to increase the weighting of distribution system samples. Samples taken at the entry point to the distribution system can no longer be included in the quarterly or annual averages. No less than 25% of the samples shall be collected at locations within the distribution system reflecting maximum residence time of the water in the system and no more than 75% from representative locations within the distribution system taking into account number of people served, source of water and treatment methods used. Thus, the required number of samples is reduced by 20% yet the results should be more representative of tap levels throughout the system, because the deleted entry point sample would not have reflected TTHM levels for a substantial portion of the population served. Of course, these compliance monitoring requirements do not preclude water systems from utilizing plant samplings for process control.

Moreover, a minimum of four compliance samples is required each quarter for each treatment plant used by the system, except that wells drawing raw water from a single aquifer may, with State approval, be considered one treatment plant for the purpose of determining the minimum number of samples required to be taken by the system. By determining the minimum number of samples per system based upon the number of separate treatment plants used by the system, sampling locations should be selected to reflect water quality in identifiable portions of the distribution systems associated with each plant to the extent possible. Larger systems are those most likely to have more than one treatment plant, and therefore more samples are both desirable in insuring consistent water quality throughout the distribution system and not likely to significantly increase the per capita cost of monitoring. However, it would not be reasonable to increase the number of samples to be taken proportionate to the number of wells drawn from a single aquifer even though each well might literally be considered a single treatment plant; water quality is likely to be consistent throughout the aquifer and many systems have a large number of wells. Therefore, with State approval, wells drawing raw water from a single aquifer may be deemed to be a single treatment plant for purposes of determining the minimum number of samples required to be taken by the system. The regulations do not provide for similar flexibility for systems drawing water from a single surface source due to the likelihood of much greater variability in raw water quality and treatment methods at different plants.

The sampling locations are important because TTHM levels will likely be higher in those parts of the distribution system where residence time of the water is longest, which is served by surface water sources, and where chlorination, as opposed to other disinfection practices, is used. Even though the samples will be averaged for determining compliance with the MCL, EPA expects that sampling will be conducted in such a way so as to insure that all parts of the distribution system are serving water to consumers in compliance with the MCL. Thus, where a system draws its raw water from multiple sources, or has more than one treatment plant utilizing different treatment methods, high THM levels in specific parts of the distribution system should be identified where possible, and such levels reduced to the extent feasible. EPA intends to address more comprehensively the problems of systems with multiple source waters and

multiple plants with differing treatment programs, when it proposes Revised Primary Drinking Water Regulations in the future.

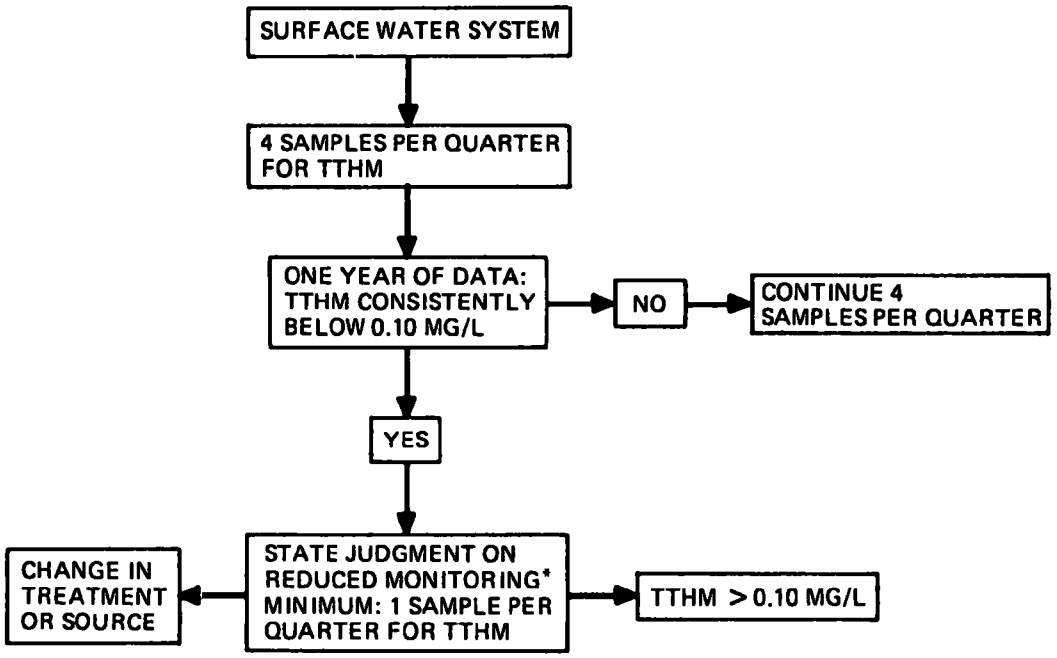
EPA also recognizes that there are a number of public water systems, such as those utilizing ground waters and some surface water supplies, where, because of the consistent quality of the source water and the treatment method employed, the probability that finished water would approach or exceed the MCL is remote. After a satisfactory record has been established, through one year of monitoring at a frequency of four TTHM samples per quarter, a water system may request that the State allow a reduction of the monitoring frequency. Upon the State's examination of at least one year of compliance data and a finding by the State that local conditions are such that TTHM concentrations are consistently below the maximum contaminant level, the system's monitoring frequency may be reduced to a minimum of one TTHM sample per quarter taken at a point in the distribution system that reflects the maximum residence time of the water served. Should the system experience a significant change in either its source of water or its treatment program, it must immediately reinstitute the four samples per quarter monitoring program initially required and continue on that program for at least another year before its sampling frequency could be reduced again so that the data baseline can be re-established. The original sampling requirements must also be reinstated immediately if the results from any analysis for TTHMs are found to exceed 0.10 mg/l and such results are confirmed by at least one check sample taken promptly after the results of the first analysis are received.

The State's decision to reduce a system's monitoring frequency must be made on a case-by-case basis taking into account such factors as the monitoring data, the quality and stability of the source of raw water, low total organic carbon (TOC) values, low maximum TTHM potential (MTP) during the time period when THM formation would most likely be at a maximum and the type of treatment employed. Except in certain ground water cases, monitoring cannot be reduced to less than one TTHM sample per quarter. This minimum monitoring is deemed necessary and is sufficient to demonstrate that conditions have not changed to the extent that the MCL might be exceeded. Intermittent use of another water source may also require additional monitoring at the discretion of the State. This flexibility is included in the regulations to allow States to modify the generally applicable monitoring requirements where appropriate only on a case-by-case basis to insure adequate public health protection. Figure 1 presents the basic steps to be followed by those systems (other than special ground water cases discussed below) that seek State approval to have their monitoring requirements reduced from four samples to one sample of TTHMs per quarter per year. "Maximum total trihalomethane potential (MTP)" is defined as the maximum concentration of TTHMs produced in a given water containing excess free chlorine after seven days at a temperature of 25° C. Determination of maximum TTHM potential should not be confused with measurement of terminal TTHM concentrations. The latter is measured under the ambient conditions of the distribution system with regard to temperature and storage time.

BILLING CODE 6560-01-M

FIGURE 1
CONSIDERATIONS FOR REDUCED MONITORING REQUIREMENTS SURFACE WATER SYSTEMS

THE MINIMUM MONITORING REQUIREMENT IS FOUR SAMPLES PER QUARTER PER PLANT. REDUCED MONITORING REQUIREMENTS MAY BE APPROPRIATE IN CERTAIN CASES; UPON WRITTEN REQUEST FROM THE PUBLIC WATER SYSTEM, STATES MAY REDUCE THE REQUIREMENTS THROUGH CONSIDERATION OF APPROPRIATE DATA AS FOLLOWS:



*FACTORS FOR CONSIDERATION:
- MONITORING DATA, MTP, TTHM, TOC
- QUALITY AND STABILITY OF SOURCE WATER
- TYPE OF TREATMENT

BILLING CODE 6560-01-C

Ground Water Sources

As several commenters suggested and EPA agrees, many, if not most, ground waters contain such small amounts of precursor organic compounds (as demonstrated by low total organic carbon levels and low measured maximum TTHM potential) and are so stable, as to virtually preclude the possibility of generating TTHM levels approaching or exceeding 0.10 mg/l even when free chlorine is employed as a disinfectant. For this reason, the regulations provide that the monitoring frequency applicable to systems using exclusively ground water sources may be reduced at the outset so that they may be relieved from the more rigorous monitoring program of four samples, or even one sample, per quarter per year which is applicable to systems using surface water sources in whole or in part.

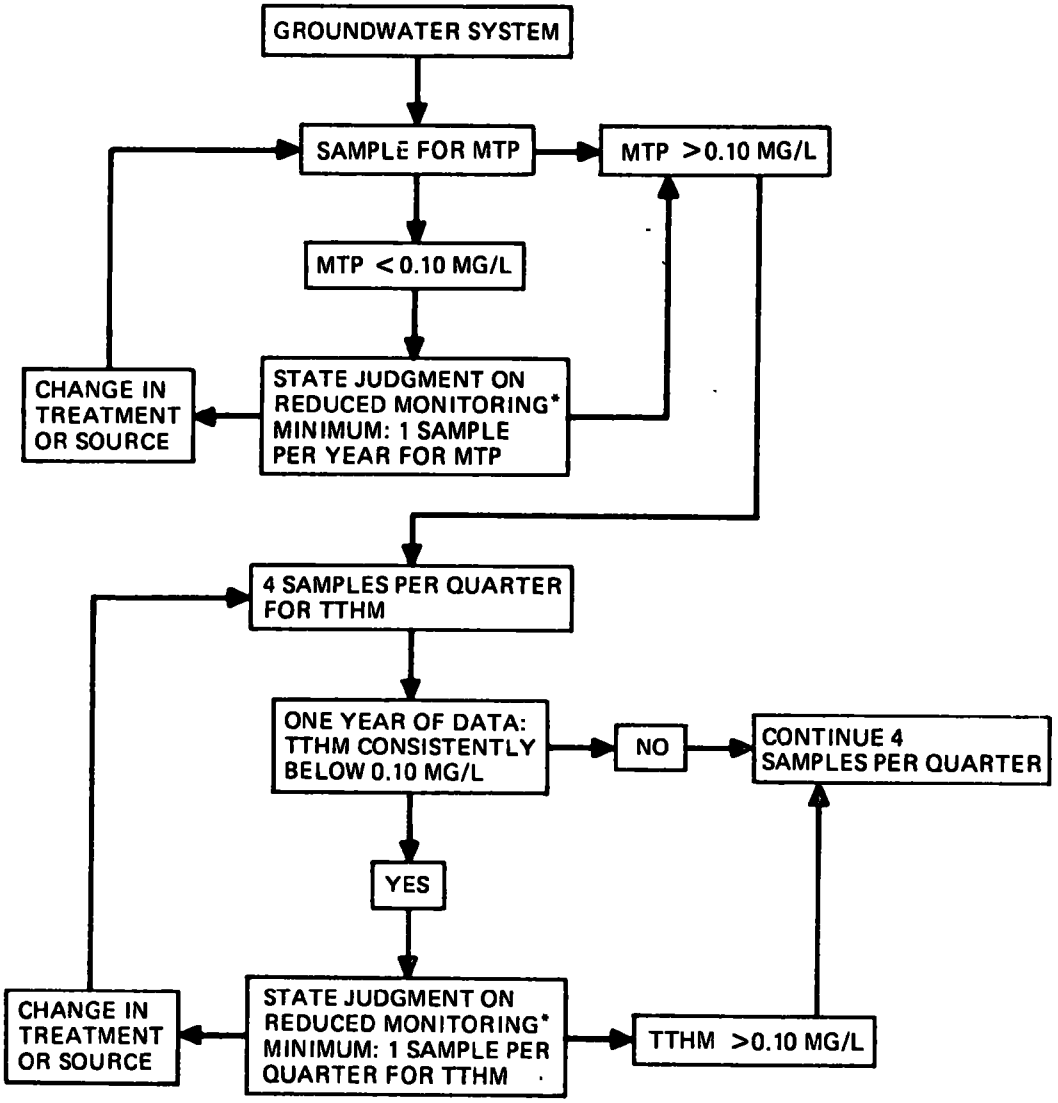
Thus, a system that draws its water exclusively from ground water sources may have its monitoring requirements reduced by the State if the results from a single sample taken at a point in the distribution system reflecting maximum residence time of the water in the system and analyzed for maximum TTHM potential (MTP) are less than 0.10 mg/l and the State determines in writing that, based on an examination of the local conditions, the system is not likely to approach or exceed the TTHM MCL. The State is expected to consider such factors as monitoring data, the quality and stability of the system's raw water source, low TOC values, low maximum TTHM potential during the time period when THM formation would most likely be at a maximum and the type of treatment employed. Such sampling frequency cannot be reduced to less than one sample for MTP per year. If such a system experiences a significant change in its source of water or treatment program, it must immediately take an additional sample for MTP analysis to determine whether it should be authorized to continue on the reduced monitoring program following the change. If the MTP is ever greater than 0.10 mg/l and such results are confirmed by a check sample taken promptly after the results of the original sample are received, the system must immediately begin taking and analyzing four samples per quarter per year for one full year. The year's results would then be averaged for determining whether the system was in compliance with the TTHM MCL. "Maximum total trihalomethane potential" is defined in the regulations at new § 141.2(s).

Figure 2 presents the basic steps to be followed by those systems using exclusively ground water sources that seek to have their monitoring frequency reduced at the outset to one sample analyzed for MTP per year, as opposed to the four samples for TTHMs per quarter per year otherwise applicable.

BILLING CODE 6560-01-M

FIGURE 2
CONSIDERATIONS FOR REDUCED MONITORING REQUIREMENTS GROUNDWATER SYSTEMS

THE MINIMUM MONITORING REQUIREMENT IS FOUR SAMPLES PER QUARTER PER PLANT; SYSTEMS USING MULTIPLE WELLS DRAWING RAW WATER FROM A SINGLE AQUIFER MAY WITH STATE APPROVAL BE CONSIDERED AS ONE TREATMENT PLANT. REDUCED MONITORING REQUIREMENTS MAY BE APPROPRIATE IN CERTAIN CASES; UPON WRITTEN REQUEST FROM THE PUBLIC WATER SYSTEM, STATES MAY REDUCE THE REQUIREMENTS THROUGH CONSIDERATION OF APPROPRIATE DATA AS FOLLOWS:



*FACTORS FOR CONSIDERATION:
- MONITORING DATA, MTP, TTHM, TOC
- QUALITY AND STABILITY OF SOURCE WATER
- TYPE OF TREATMENT

BILLING CODE 6560-01-C

Technical Feasibility of TTHM Reduction

In establishing an MCL for TTHMs, EPA is not required to specify any particular method to achieve that standard. However, in establishing Interim Regulations, EPA must find that technology was generally available in 1974 to achieve the MCL. Thus, the preamble to the proposal did discuss a number of approaches that could be utilized to achieve the MCL depending on the individual circumstances. The "Interim Treatment Guide for the Control of Chloroform and Other Trihalomethanes" was also published and made available to commenters to provide information on successful techniques that should be considered. It is incorporated by reference as part of the Statement of Basis and Purpose for these regulations.

Three general alternatives have been presented:

(1) Use of a disinfectant (oxidant) that does not generate (or produces less) THMs in water;

(2) Treatment to reduce precursor concentrations prior to chlorination; and

(3) Treatment to remove THMs after formation. Many possible choices exist within each category. For example, alternate disinfectants or oxidants that might be considered include ozone, chlorine dioxide, and chloramines (combined chlorine). Precursor reduction processes include off-line raw water storage, aeration, improved coagulation, ion exchange resins, granular activated carbon (GAC), powdered activated carbon (PAC), and ozone enhanced biological activated carbon (BAC). TTHM reduction has also been achieved by merely moving the chlorine addition point to later stages in the conventional treatment process, and by substituting prechlorination with some other preoxidation process. TTHM removal processes include GAC, aeration or macroreticular resins. A combination of these methods may be necessary to comply with the TTHM MCL.

Few comments discussed the feasibility of the available treatments, and three suggested that additional research should be performed on the subject. EPA has concluded that many methods have been shown to be effective for meeting the 0.10 mg/l MCL for TTHMs and it remains only for the individual water systems to select the one or more procedures that are optimal for their particular water characteristics.

Which treatment method (or combination of treatment methods) is ultimately selected by a water supplier to achieve compliance with the MCL must be based upon a case-by-case assessment of the system's entire treatment process, and an evaluation of the precursor content of its raw water source and TTHM formation potential as well as the need to assure optimal biological quality of drinking water derived from contaminated sources.

In determining what technologies were "generally available" in 1974 for achieving the standard, EPA has taken cost into consideration. The legislative history of the SDWA clearly requires that the reasonableness of costs must be based on "what may reasonably be afforded by large metropolitan or regional public water systems" (House Report No. 93-1185, p. 18). Moreover, the Administrator must assume that most intake waters are sufficiently uncontaminated so that the MCLs can be met with the application of those technologies found to be "generally available" at reasonable cost in 1974 (House Report No. 93-1185, p. 13).

EPA has estimated the costs of various treatment methods available in 1974 to achieve compliance with the TTHM MCL of 0.10 mg/l. They appear in the report prepared for EPA by Culp/Wesner/Culp entitled, "Estimating Costs for Water Treatment As a Function of Size and Treatment Efficiency" and EPA's "Interim Treatment Guide for the Control of Chloroform and other Trihalomethanes." The cost assumptions in those documents in large part serve as the basis for EPA's Economic Impact Analysis for these regulations. These documents are incorporated by reference as part of the Agency's Statement of Basis and Purpose for these regulations.

Based on these documents, EPA has concluded that the use of any of the alternative disinfectants discussed above has been clearly available at reasonable costs since 1974 to any large public water system to achieve the MCL of 0.10 mg/l. Alternatives, such as changing the point of disinfection, off-line raw water storage and improved coagulation are also relatively inexpensive and are also found to be "generally available" at reasonable costs.

With respect to the use of adsorbants, the reasonableness of costs will be dependent upon the particular operational parameters that are employed. For purposes of establishing these regulations, EPA assessed the costs that would be incurred by systems utilizing GAC as a replacement for their existing filter media, with a regeneration frequency of one year. Although most systems are expected to select the less expensive treatment methods where they are effective in achieving compliance with the MCL, the use of GAC under these operating conditions has also been found to be "generally available" at reasonable cost since 1974 for achieving the standard. Systems with very high raw water TOC may need to use GAC with more stringent operating parameters or additional treatment methods to achieve the MCL. For this reason, EPA has also assessed the cost of using biological activated carbon (ozone plus GAC) with a regeneration frequency for the carbon of two years; this cost has also been found to be reasonable.

Disinfectant Restrictions and the Standard Plate Count

Restrictions were proposed on the excessive use of chlorine dioxide because of possible by-product chlorite toxicity, and also on misuse of chloramines because of their low-potency as disinfectants compared to free chlorine. The proposal also admonished those considering modifications to their treatment process to reduce TTHMs that any such modification must not in any way affect the microbiological quality of drinking water so as to increase the possibility of transmission of infectious disease. Also, EPA espoused the fundamental principle that water treatment should aim at producing water of high quality and low chemical content prior to application of the oxidant, so as to maintain pathogen control while minimizing oxidant use and by-product demand.

Because of possible adverse effects on finished water quality from ill-advised treatment modification, the following three conditions were specifically proposed to apply in cases where changes to current treatment practice would be utilized to reduce TTHMs:

1. The total quantity of chlorine dioxide added during the treatment process should not exceed 1 milligram per liter of water.

2. Chloramines should not be utilized as the primary disinfectant. Chloramines may be added for the purpose of maintenance of an active chlorine residual in the distribution system only to water that already meets primary drinking water regulations.

3. Monitoring for general bacteria populations (Standard Plate Count) should be performed as determined by the State but at least daily for at least one month prior to and six months subsequent to the modifications.

These restrictions have been deleted from the final regulations to provide the States with greater discretion to prescribe requirements as necessary on a case-by-case basis. This should not be construed to reflect EPA's lack of concern regarding microbiological

quality. As described below, EPA is requiring that water systems obtain State approval of any proposed significant modifications to their treatment process. Once a system's plan is approved, the system must follow the plan. Moreover, these regulations prescribe those minimum conditions which must be satisfied by the plan before State approval can be granted. EPA will also publish guidance for the States that will serve as a useful reference in approval of system plans.

This approach is believed to be more reasonable than the inclusion of specific nationally applicable restrictions which may or may not be applicable in every case. Because systems will begin making modifications to their disinfection processes immediately upon promulgation of this regulation (and in fact, some systems have already begun to make such changes), EPA has determined that good cause exists to make the requirements of § 141.30(f) (approval of system treatment modification plans) effective immediately upon promulgation. This is necessary to ensure that all system treatment modifications are made subject to close State and EPA supervision at the earliest possible time.

Chlorine Dioxide

Oxidation/reduction reactions of chlorine dioxide in water produce chlorite and some chlorate and ultimately chloride ions. Preliminary studies with cats and rats had indicated that excessive exposures (above 10 mg/l) to chlorite had resulted in deleterious effects on red blood cells in some animals. A limit on applied chlorine dioxide of 1 mg/l was proposed to provide a margin of safety from the possible effects of ingested chlorine dioxide and chlorite and chlorate, and assumed that a portion of the chlorine dioxide would be spontaneously reduced to chloride which is not toxic. In a more recent study in a human population using drinking water treated seasonally with chlorine dioxide, statistically significant blood effects were not found at concentrations of approximately 5 mg/l of oxidant in water; however, this was a short duration test that terminated earlier than expected. One individual shown to be deficient in glucose 6-phosphate dehydrogenase, a genetic defect that is present in a small percent of the U.S. population that would possibly be sensitive to oxidants, showed an effect, but, it was within the range of effects of some of the normal population.

Only ten comments were received on the proposed chlorine dioxide restriction and nine were opposed claiming insufficient evidence of adverse health risk. Several suggested acceptable levels as high as 2 or 3 mg/l, but did not submit supporting data.

EPA has concluded that while there is evidence that exposure to chlorine dioxide by-products can result in detectable if not clinically significant blood effects, restrictions should be more appropriately placed on the residual oxidants (ClO_2, ClO_2^- and ClO_3^-) in the water rather than on the amount of ClO_2 added. The extent of the oxidation/reduction of the added ClO_2 and the formation of the intermediate chlorite and chlorate would be a function of the reducing agents present in the water, and the chlorine dioxide that would be completely reduced to chloride is of no toxicological significance.

In the 1979 update of "Drinking Water and Health", the NAS reviewed the data as of 1978 and estimated acceptable exposure values of 0.38 mg/l and 0.21 mg/l for chlorine dioxide and chlorite respectively. These were computed from data in rats and cats and incorporated an uncertainty factor of 100. The NAS also noted that the computed value for chlorine dioxide was consistent with EPA's proposal limiting the amount added to 1 mg/l assuming 50% conversion to chloride. Very recent incomplete data obtained from controlled studies with normal male volunteers detected slight but not clinically significant effects at higher than normal doses. These experiments are continuing and will produce more definitive results within the next year.

Therefore, although the restriction on chlorine dioxide addition has been deleted from the regulation, EPA feels that whenever chlorine dioxide is used residual oxidants should be monitored and kept below 0.5 mg/l. EPA will consider establishing an MCL for chlorine dioxide, chlorite and chlorate or the aggregate as total oxidant for inclusion in the Revised Regulations after further studies have been fully evaluated.

Chloramines

Chloramine (combined chlorine) has been shown to be a simple and readily available means of reducing the formation of THMs in many water supplies in those cases where raw water quality and treatment methods permit.

The proposal to restrict the use of chloramines in THM control in inappropriate circumstances was based upon the well known fact that chloramines, in themselves, are very weak disinfectants for bacteria, virus and protozoa compared to free chlorine as HOCl, ozone and chlorine dioxide. Thus, the use of chloramines as a primary disinfectant, (i.e., to kill or inactivate pathogens in raw water), may increase the risk of pathogens reaching the consumer. The proposed restriction would not have affected the use of chloramines for disinfection maintenance in distribution systems.

Opponents of the restriction argued that chloramines had been effectively used in many systems. Other commenters agreed with the proposal that chloramines should be restricted from use as a primary disinfectant. Those opposed to the restriction did not distinguish between the common use of chloramines to maintain an active combined chlorine residual (as a secondary disinfectant by EPA's definition) and total reliance on chloramines (as a primary disinfectant). None of the commenters contradicted the experimental fact that chloramines are much less efficient bacteriocides and virocides than chlorine (HOCl), ozone, and chlorine dioxide. The NDWAC felt that the proposed limitation was unduly restrictive.

Providing the necessary barrier against waterborne disease transmissions is the function of the total process of providing water to the consumer. This process begins with selection of the best available source, and its protection from contamination and is followed by the treatment train, that may consist of off-line storage, coagulation, sedimentation and filtration and/or lime treatment and pH adjustment, along with several increments of oxidant (disinfectant). It concludes with protecting the finished water in transit by maintenance of the integrity of the distribution system. EPA recognizes the use history as well as the risks inherent in misuse of chloramines and has concluded that the decision is best made on a case-by-case basis by the State or primacy authority in its review and approval of a water system's plan under § 141.30(f) to provide the necessary supervision. This subject is also included in EPA's guidance to the States for approval of system treatment modification plans.

Standard Plate Count

The presence of coliform bacteria is considered to be the most reliable indicator of possible fecal contamination and associated enteric microorganism. Current National Interim Primary Drinking Water Regulations (40 CFR 141.21, 40 FR 59556) require monitoring for coliforms on a frequency based upon population served in the community water system and include an MCL of 1 coliform per liter as determined by the membrane filter

technique. Nevertheless, certain bacteria, viruses and cysts are more resistant to disinfectants and are capable of surviving in water longer than the coliform indicator organisms.

Because of the possibility that, in the course of applying treatment modifications to reduce TTHMs, some water systems might be tempted to utilize less efficient disinfectants such as chloramines or shorter contact times with free chlorine, the proposal contained a requirement to utilize the Standard Plate County (SPC) analysis during transition periods when current treatment practice was being modified. This was intended to be applied as a more sensitive indicator of general biological quality to signal the possibility of a deterioration of treatment effectiveness and therefore increased potential of undetected pathogens.

Of the comments on this issue, more than half opposed or questioned the significance of the SPC as an indicator of water quality. However, somewhat less than half of the commenters agreed with the proposal that SPC should be required during treatment modification. A few suggested that SPC should be required only for those water sources receiving discharges of municipal waste. Others felt that SPC should be used at the discretion of the State. The NEWAC recommended that the SPC should not be a regulatory requirement but rather a matter of State discretion.

In "Drinking Water and Health," the Safe Drinking Water Committee of the NAS underscored the usefulness of SPC applied in conjuction with total coliform tests to measure the sanitary quality of drinking water. The Committee recommended use of SPC to:

1. Provide a method for monitoring for changes in the microbiological quality of finished water;

2. Determine whether the normal flora of a water supply may be interfering with coliform detection; and

3. Monitor the effectiveness of a disinfectant or treatment practice within the plant and distribution system and provide an indication of filter-effluent quality deterioration and the occurrence of the breakthrough of microorganisms.

EPA remains convinced that the SPC is an appropriate adjunct to coliform monitoring and a sensitive indicator of process performance and distribution system integrity, and that it should be employed particularly during periods when treatment modifications are being introduced. Many public water systems have extensively used the test as a routine quality monitor. Its application is particularly essential in drinking water drawn from raw water sources contaminated by sewage effluent. SPC has been deleted as a requirement from these regulations, but should be a condition for State approval of system plans where disinfection process modifications are contemplated. SPCs are therefore included in the guidance to States for approval of system treatment modification plans.

Microbiological Considerations—State Approval of System Treatment Modification Plans to Reduce TTHMs

Historically, the States have had the responsibility of ensuring that drinking water in public water systems has received adequate treatment before it is distributed. When systems alter traditional treatment practices to reduce TTHMs, States must continue to exercise control to assure that water is provided to the consumer by public water systems that is microbiologically and chemically safe and of optimal quality. Where States lack primacy enforcement responsibility, that responsibility falls to the EPA Regional Office.

The goal of disinfection has been and still is to produce water that is biologically safe to drink; this goal is attained by killing pathogens in the water. However, potentially harmful chemicals are now known to be produced during disinfection. Quality control thus necessitates careful consideration of all appropriate factors for each public water system modifying disinfection processes to control production of those chemicals, and States should exercise their full authority to see that the public is protected.

The National Academy of Sciences' reports, "Drinking Water and Health" and "The Disinfection of Drinking Water" and the Office of Drinking Water (EPA) Report, EPA-570/9-78-002, "Evaluation of the Microbiology Standards for Drinking Water" address the principles of drinking water disinfection and their effect on microbial problems. These documents, along with the guidance accompanying this regulation, should be consulted early in the development of the public water supply's program to reduce TTHM formation.

The basic principle in achieving compliance with the TTHM MCL is that as TTHM control practices are conceived and put into practice, the water supplied to the consumer must be of optimal quality. Systems must be carefully supervised to ensure that water quality is not allowed to deteriorate as a result of changes in treatment practice, thereby creating risks to the public health from particular chemicals or infectious agents. The integrity of the bacteriological quality of the drinking water must not be compromised.

EPA is therefore requiring that public water systems contemplating significant changes in treatment practice to control TTHMs submit an action plan to the State for approval and after approval has been received, to follow the conditions set forth in the approved plan, that will be based upon the guidance provided by EPA.

The following summarizes the major principles set forth in the EPA guidance to the States:

1. Prior to any significant modification, the entire system should be evaluated to detect the presence of sanitary defects and to determine the risks from breakthrough of microbiological contaminants in the source water, through treatment and in the distribution system. Virus studies are essential where source waters are heavily contaminated with sewage effluents.

2. A comprehensive evaluation of existing treatment practices and available options should be conducted to determine the most effective treatment modifications that would result in optimum finished water biological quality and TTHM control. Any system deficiencies that are found during the examination should be promptly corrected.

3. A baseline water quality survey of source water, water undergoing treatment prior to disinfection and water within the distribution system particularly in the extremes of the system and in deadends should be conducted prior to the initiation of the TTHM control practices at a sufficient frequency and time span to establish an understanding of the water quality. Measured parameters should include coliform and fecal coliform bacteria, fecal streptococci, standard plate count incubated at 35° C and 20° C, phosphate, ammonia nitrogen, TOC and others directed by the State based on the particular characteristics of local water quality. In systems using poor quality source water, for example, a weekly or more frequent sampling frequency may be necessary.

4. Following modification, the water quality survey (in item 3 above) should be continued for one year to determine the performance of the treatment system for all seasons. The parameters in the baseline study should continue to be examined using samples from the same locations.

5. Treatment practices for THM control should also provide effective post disinfection to control microbial

populations, and an active disinfectant residual should be maintained in all parts of the distribution system.

6. If the present point of chlorination is altered, the supply should maintain proper pH control and allow sufficient contact time for optimal disinfection.

7. Monitoring for chlorate, chlorite and chlorine dioxide should be performed when chlorine dioxide is used as a disinfectant. Residual concentrations of total residual oxidants (except for HOCl derivatives) in the water should not exceed 0.5 mg/l in the interim until further EPA studies are completed.

8. Chloramines are less efficient as disinfectants particularly for virus and protozoans as compared to chlorine, chlorine dioxide and ozone. If chloramines are used with contaminated source water, the total treatment process should be capable of compensating for any potential reduction in disinfection efficiency.

9. Ozone is not an appropriate disinfectant for high TOC containing waters unless the potential for post treatment biological growth can be controlled such as by the use of processes that control biodegradable chemicals in the source water and the finished water.

10. Systems presently utilizing pre-chlorination for disinfection purposes must be certain that alternative pretreatment practices are sufficient to protect the public if changes are introduced.

11. Any oxidant (disinfectant) used to treat drinking water will interact with chemicals already in the water to form undesirable by-products in the finished water. Therefore the basic principle should be to maximize precursor removal prior to the addition of the oxidant so as to minimize a disinfectant demand and by-product formation. Otherwise, an excessive disinfectant demand could reduce the efficiency of any disinfectant practice and add, in the process, substantial amounts of undesirable and perhaps toxic compounds.

12. Varied and extensive modification of existing treatment processes often result in changes in the chemical and microbial quality of treated water. Increased monitoring of coliform bacteria and the use of other indicators of the sanitary quality of water (e.g., SPC) are advisable.

Individual system plans for TTHM control should include the design of the vulnerability and baseline data surveys and the additional surveillance monitoring to assure maintenance of biological quality with the altered treatment system and must be approved by the State prior to their implementation. The plan should also include information on current treatment practices and their performance and other information as directed by the State. EPA believes that if States and public water systems follow the guidance and technical assistance is provided as needed, TTHM control will be safely achieved.

Economic Impact Assessment

The economic impact of these regulations was projected based on the three principal control options available to the approximately 2,700 community water systems serving more than 10,000 people required to comply with the regulatory requirements—modifying chlorination or associated treatment procedures, changing disinfectants, using an adsorbent, or some combination of the above. The calculation of total national cost projections for the TTHM regulation required an estimate of the number of systems choosing each control option and the incremental costs associated with each option considered. An incremental expense will accrue to all systems covered, whether or not treatment is required, to cover monitoring expenses. These expenses for all systems covered are included in the following estimates of total costs for the TTHM regulation.

This analysis employed a probabilistic and structured approach for determining the choice of control options that each public water system would make since no empirical method exists for predetermining that choice. A logical sequence of decision points was designed to distribute the systems anticipated to be covered by the regulation according to the most likely path they would follow. The decision made at each point is consistent with the following criteria:

1. The treatments currently used: If a system does not add chlorine it will not be affected by a THM regulation, and therefore will require no new treatment.

2. Water source used: If a system uses surface water (except the Great Lakes and some high quality mountain water) as its primary source, it is more likely to exceed a given level of THM contamination. Hence the number of water systems using water from ground or surface sources affects the number of systems which will exceed the MCL and will therefore require treatment.

3. Degree to which water quality exceeds MCL: If the presence of TTHMs is only slightly in excess of the initial MCL, then minimal modifications to current treatment procedures may be adequate for compliance. As the level of contamination increases, a system must consider more significant (and costly) treatment techniques.

4. Economic considerations: The presumption was that systems would adopt the least costly treatment strategy that satisfies the regulations.

5. Treatment effectiveness: Many systems with TTHM concentrations only slightly above the MCL can comply by modifying treatment procedures. Others may need to change disinfectants. Finally, precursor concentrations resulting in very high THM formation potentials can probably be best controlled by the use of adsorbents. This is because of the likelihood that high disinfectant demand waters cannot be disinfected adequately without generating considerable amounts of by-products of unknown hazard or without exceeding the MCL. Consequently, some of those systems with very high levels of TTHMs are projected to use adsorbents.

Based on all information available to EPA of the 390 public water systems that serve more than 75,000 people, 61 purchase the majority of their water from other systems that are presumed to provide treatment. Thus, a total of 329 systems would be initially affected although 7 of these were excluded because they do not presently add a disinfectant. Of the remaining 322, some 95 systems were estimated to have TTHM levels above 0.10 mg/l and hence would require changes in their treatment processes.

Since the final regulation phases in coverage to include systems serving between 10,000 and 75,000 people, the economic analysis has also included the costs these systems will bear in achieving compliance. Of the 2,295 public water systems that serve between 10,000 and 75,000 people, 355 are known to purchase the majority of their water from other systems that are presumed to provide treatment. Thus a total of 1,940 systems between 10,000 and 75,000 population would be initially affected, although 281 of these are excluded because they do not presently add a disinfectant. Of the remaining 1,659, some 420 systems were estimated to have TTHM levels above 0.10 mg/l and hence would require changes in their treatment processes to comply by the applicable effective date in the regulation.

The following projections were made based upon information presented during the comment period primarily from the water utilities and consultants. Of the systems estimated to be in the range of 1 to 1.5 times the MCL, 60 percent were expected to modify their chlorination procedures and 40 percent were expected to change disinfectants. Of the systems with TTHM levels in the

range of 1.5 to 2.5 times the MCL, 25 percent were expected to change their chlorination procedures with 75 percent changing disinfectants. Finally, of the systems exceeding 2.5 times the MCL, 80 percent were anticipated to change disinfectants and the remaining 20 percent would likely use an adsorbent. On the basis of the above assumptions, national cost estimates for compliance with these final regulations are as follows:

Summary of Estimated Total Costs for an MCL Regulation With the Trihalomethane Concentration of 0.10 mg/l

(In millions of 1980 dollars)

	Categories according to population served by average system		
	10,000–75,000	Over 75,000	Total
Capital Expenditures	$40	$45	$85
Operation and Maintenance	5	5	10
Revenue Requirements	9	10	19
Annual per Carta Costs of Treatment [1] (dollars)	0 60	0 90	0 70
Increase in Annual Residential Bill [1] (dollars)	1 20	1 80	1 40

[1] Includes only systems projected to incur treatment costs associated with the THM regulations

Per capita costs will vary depending upon the type of treatment selected, the system size, and many other factors. Given an MCL of 0.10 mg/l, the range of annual residential bill increases for a typical family of 3 would be from $0.32 to $1.89 for systems using an alternative disinfectant and $4.44 to $11.18 for systems using an adsorbent in combination with ozonation assuming a 720 day regeneration cycle.

The costs presented in this final analysis are considerably lower than EPA's previous national cost estimates for the TTHM regulations as set forth in the February 9, 1978, notice and later revised in the July 6, 1978, supplemental notice, even though they are now stated in 1980 dollars while the August 1977 report accompanying the proposed regulations used 1976 dollars. The differences causing this reduction result from numerous changes in the underlying data, based on information received during the comment period, including: (a) Revised estimates of the number of systems using disinfectants; (b) revised estimates of the level of TTHMs in a given ground or surface system; (c) changes in the probabilities assigned to branches of the decision tree used to select among control options with more systems using chloramines and many fewer using GAC; (d) revisions of unit cost data to reflect inflation to 1980 dollars and increases in assumed levels of professional fees (resulting in an approximate 28 percent increase in costs); (e) changes in the GAC costs to reflect longer projected regeneration cycles (from 60 days to 360 days for GAC alone and 720 days for GAC and ozone), more off-site regeneration at regional facilities and use of GAC in existing filter beds. Detailed analysis of the costs of various options and the underlying data are contained in the "Economic Impact Analysis of a Promulgated Trihalomethane Regulation for Drinking Water," available on request, and incorporated by reference as part of the Statement of Basis and Purpose for this regulation.

Although the typical economic impacts appear to be reasonable, it is possible that some utilities will have unique problems which lead to financial hardships. This would take the form of an inability to raise capital needs for improvements in treatment necessary to comply with the TTHM regulation. Should a situation arise, opportunities exist which can ease these financing difficulties. The Office of Drinking Water provides technical assistance in this area, and interested parties should contact: Victor J. Kimm, Deputy Assistant Administrator for Drinking Water (WH–550), Environmental Protection Agency, 401 M Street, SW., Washington, D.C. 20460 for additional information.

Energy Impact Assessments

The TTHM regulation will have a negligible impact on annual domestic energy consumption. The total energy requirements associated with the regulation are 508×10^9 BTU's, or 0.0007 percent of 1977 U.S. energy consumption. The *annual* energy requirements of the various treatment alternatives selected by utilities to meet the MCL for TTHMs are as follows: Electric power, 39.9 million kilowatt-hours; diesel fuel, 64,000 gallons; and natural gas, 76.4 million cubic feet. In 1980 dollars these total annual energy requirements are estimated to cost $2.3 million per year. The annual electric power demand of 39.9 kwhr is approximately 0.002 percent of 1977 total domestic electric power sales. The annual diesel fuel demand represents only 0.00002 percent of the 1977 total domestic demand for refined oil products. At 76.4 million cubic feet, the annual natural gas demand represents less than 0.004 percent of the 1977 domestic natural gas demand.

Approximately 87 percent of the electric power demand is due to ozone disinfection processes. GAC treatment and ozonation together represent 96 percent of the total electric power demand.

The diesel fuel and natural gas requirements are created by the GAC regeneration process. For those water utilities without on-site GAC regeneration, transport of GAC to remote processing sites will require diesel fuel. The regeneration process itself requires either oil or natural gas as an energy source. In preparing these energy demand estimates, EPA assumed that only natural gas would be used in GAC regeneration furnaces. The energy impacts of this regulation are reduced from those associated with the proposal because fewer systems are expected to resort to the more energy intensive treatment methods to achieve compliance with the MCL.

Evaluation Plan

As noted previously, these regulations are considered to be an initial step in controlling disinfection by-products, with TTHMs being a surrogate. As the regulations are implemented, an extensive data collection effort will begin through the self-monitoring programs at the applicable public water systems. These data will include levels of TTHMs associated with disinfection of various types of raw water sources and the specific technologies utilized for control of TTHMs.

Compliance with the regulations will be determined by State program staffs and the compliance data will be included in the Model State Information System and Federal Data Reporting Systems (computer systems). This will allow easy access to evaluation of national compliance with the regulations.

The compliance data will be evaluated along with results of ongoing research and development efforts which are examining the toxicology of disinfection by-products and available treatment alternatives for control. The evaluation will be used to determine the appropriateness of the level of the MCL and will be the basis of further regulatory actions controlling disinfection by-products. These evaluations will be conducted no later than three years after the promulgation of the regulations. The Director, Criteria and Standards Division, Office of Drinking water, should be contacted if further information is desired.

Under Executive Order 12044, EPA is required to judge whether a regulation is "significant" and therefore subject to the procedural requirements of the Order or whether it may follow other specialized development procedures. EPA labels these other regulations "specialized." I have reviewed this regulation and determined that it is a specialized

regulation not subject to the procedural requirements of Executive Order 12044.

Dated: November 5, 1979.

Douglas M. Costle,

Administrator.

Accordingly, Part 141, Title 40 of the Code of Federal Regulations is hereby amended as follows:

1. By amending § 141.2 to include the following new paragraphs (p) through (t):

§ 141.2 Definitions.

* * * * *

(p) "Halogen" means one of the chemical elements chlorine, bromine or iodine.

(q) "Trihalomethane" (THM) means one of the family of organic compounds, named as derivatives of methane, wherein three of the four hydrogen atoms in methane are each substituted by a halogen atom in the molecular structure.

(r) "Total trihalomethanes" (TTHM) means the sum of the concentration in milligrams per liter of the trihalomethane compounds (trichloromethane [chloroform], dibromochloromethane, bromodichloromethane and tribromomethane [bromoform]), rounded to two significant figures.

(s) "Maximum Total Trihalomethane Potential (MTP)" means the maximum concentration of total trihalomethanes produced in a given water containing a disinfectant residual after 7 days at a temperature of 25° C or above.

(t) "Disinfectant" means any oxidant, including but not limited to chlorine, chlorine dioxide, chloramines, and ozone added to water in any part of the treatment or distribution process, that is intended to kill or inactivate pathogenic microorganisms.

2. By revising § 141.6 to read as follows:

§ 141.6 Effective dates.

(a) Except as provided in paragraph (b) of this section, the regulations set forth in this part shall take effect on June 24, 1977.

(b) The regulations for total trihalomethanes set forth in § 141.12(c) shall take effect 2 years after the date of promulgation of these regulations for community water systems serving 75,000 or more individuals, and 4 years after the date of promulgation for communities serving 10,000 to 74,999 individuals.

3. By revising the introductory paragraph and adding a new paragraph (c) in § 141.12 to read as follows:

§ 141.12 Maximum contaminant levels for organic chemicals.

The following are the maximum contaminant levels for organic chemicals. The maximum contaminant levels for organic chemicals in paragraphs (a) and (b) of this section apply to all community water systems. Compliance with the maximum contaminant levels in paragraphs (a) and (b) is calculated pursuant to § 141.24. The maximum comtaminant level for total trihalomethanes in paragraph (c) of this section applies only to community water systems which serve a population of 10,000 or more individuals and which add a disinfectant (oxidant) to the water in any part of the drinking water treatment process. Compliance with the maximum contaminant level for total trihalomethanes is calculated pursuant to § 141.30.

* * * * *

(c) Total trihalomethanes (the sum of the concentrations of bromodichloromethane, dibromochloromethane, tribromomethane (bromoform) and trichloromethane (chloroform)) 0.10 mg/l.

4. By revising the title, the introductory text of paragraph (a) and paragraph (b) of § 141.24 to read as follows:

§ 141.24 Organic chemicals other than total trihalomethanes, sampling and analytical requirements.

(a) An analysis of substances for the purpose of determining compliance with § 141.12(a) and § 141.12(b) shall be made as follows:

(b) If the result of an analysis made pursuant to paragraph (a) of this section indicates that the level of any contaminant listed in § 141.24 (a) and (b) exceeds the maximum contaminant level, the supplier of water shall report to the State within 7 days and initiate three additional analyses within one month.

5. By adding a new § 141.30 tc read as follows:

§ 141.30 Total trihalomethanes sampling, analytical and other requirements.

(a) Community water system which serve a population of 10,000 or more individuals and which add a disinfectant (oxidant) to the water in any part of the drinking water treatment process shall analyze for total trihalomethanes in accordance with this section. For systems serving 75,000 or more individuals, sampling and analyses shall begin not later than 1 year after the date of promulgation of this regulation. For systems serving 10,000 to 74,999 individuals, sampling and analyses shall begin not later than 3 years after the date of promulgation of this regulation. For the purpose of this section, the minimum number of samples required to be taken by the system shall be based on the number of treatment plants used by the system, except that multiple wells drawing raw water from a single aquifer may, with the State approval, be considered one treatment plant for determining the minimum number of samples. All samples taken within an established frequency shall be collected within a 24-hour period.

(b)(1) For all community water systems utilizing surface water sources in whole or in part, and for all community water systems utilizing only ground water sources that have not been determined by the State to qualify for the monitoring requirements of paragraph (c) of this section, analyses for total trihalomethanes shall be performed at quarterly intervals on at least four water samples for each treatment plant used by the system. At least 25 percent of the samples shall be taken at locations within the distribution system reflecting the maximum residence time of the water in the system. The remaining 75 percent shall be taken at representative locations in the distribution system, taking into account number of persons served, different sources of water and different treatment methods employed. The results of all analyses per quarter shall be arithmetically averaged and reported to the State within 30 days of the system's receipt of such results. Results shall also be reported to EPA until such monitoring requirements have been adopted by the State. All samples collected shall be used in the computation of the average, unless the analytical results are invalidated for technical reasons. Sampling and analyses shall be conducted in accordance with the methods listed in paragraph (e) of this section.

(2) Upon the written request of a community water system, the monitoring frequency required by paragraph (b)(1) of this section may be reduced by the State to a minimum of one sample analyzed for TTHMs per quarter taken at a point in the distribution system reflecting the maximum residence time of the water in the system, upon a written determination by the State that the data from at least 1 year of monitoring in accordance with paragraph (b)(1) of this section and local conditions demonstrate that total trihalomethane concentrations will be consistently below the maximum contaminant level.

(3) If at any time during which the reduced monitoring frequency prescribed under this paragraph applies, the results from any analysis exceed 0.10 mg/l of TTHMs and such results are confirmed by at least one check sample taken promptly after such results are received, or if the system makes any significant change to its source of water or treatment program, the system shall immediately begin monitoring in accordance with the requirements of paragraph (b)(1) of this section, which monitoring shall continue for at least 1 year before the frequency may be reduced again. At the option of the State, a system's monitoring frequency may and should be increased above the minimum in those cases where it is necessary to detect variations of TTHM levels within the distribution system.

(c)(1) Upon written request to the State, a community water system utilizing only ground water sources may seek to have the monitoring frequency required by subparagraph (1) of paragraph (b) of this section reduced to a minimum of one sample for maximum TTHM potential per year for each treatment plant used by the system taken at a point in the distribution system reflecting maximum residence time of the water in the system. The system shall submit to the State the results of at least one sample analyzed for maximum TTHM potential for each treatment plant used by the system taken at a point in the distribution system reflecting the maximum residence time of the water in the system. The system's monitoring frequency may only be reduced upon a written determination by the State that, based upon the data submitted by the system, the system has a maximum TTHM potential of less than 0.10 mg/l and that, based upon an assessment of the local conditions of the system, the system is not likely to approach or exceed the maximum contaminant level for total TTHMs. The results of all analyses shall be reported to the State within 30 days of the system's receipt of such results. Results shall also be reported to EPA until such monitoring requirements have been adopted by the State. All samples collected shall be used for determining whether the system must comply with the monitoring requirements of paragraph (b) of this section, unless the analytical results are invalidated for technical reasons. Sampling and analyses shall be conducted in accordance with the methods listed in paragraph (e) of this section.

(2) If at any time during which the reduced monitoring frequency prescribed under paragraph (c)(1) of this section applies, the results from any analysis taken by the system for maximum TTHM potential are equal to or greater than 0.10 mg/l, and such results are confirmed by at least one check sample taken promptly after such results are received, the system shall immediately begin monitoring in accordance with the requirements of paragraph (b) of this section and such monitoring shall continue for at least one year before the frequency may be reduced again. In the event of any significant change to the system's raw water or treatment program, the system shall immediately analyze an additional sample for maximum TTHM potential taken at a point in the distribution system reflecting maximum residence time of the water in the system for the purpose of determining whether the system must comply with the monitoring requirements of paragraph (b) of this section. At the option of the State, monitoring frequencies may and should be increased above the minimum in those cases where this is necessary to detect variation of TTHM levels within the distribution system.

(d) Compliance with § 141.12(c) shall be determined based on a running annual average of quarterly samples collected by the system as prescribed in subparagraphs (1) or (2) of paragraph (b) of this section. If the average of samples covering any 12 month period exceeds the Maximum Contaminant Level, the supplier of water shall report to the State pursuant to § 141.31 and notify the public pursuant to § 141.32. Monitoring after public notification shall be at a frequency designated by the State and shall continue until a monitoring schedule as a condition to a variance, exemption or enforcement action shall become effective.

(e) Sampling and analyses made pursuant to this section shall be conducted by one of the following EPA approved methods:

(1) "The Analysis of Trihalomethanes in Finished Waters by the Purge and Trap Method," Method 501.1, EMSL, EPA Cincinnati, Ohio.

(2) "The Analysis of Trihalomethanes in Drinking Water by Liquid/Liquid Extraction,"Method 501.2, EMSL, EPA Cincinnati, Ohio.

Samples for TTHM shall be dechlorinated upon collection to prevent further production of Trihalomethanes, according to the procedures described in the above two methods. Samples for maximum TTHM potential should not be dechlorinated, and should be held for seven days at 25° C prior to analysis, according to the procedures described in the above two methods.

(f) Before a community water system makes any significant modifications to its existing treatment process for the purposes of achieving compliance with § 141.12(c), such system must submit and obtain State approval of a detailed plan setting forth its proposed modification and those safeguards that it will implement to ensure that the bacteriological quality of the drinking water served by such system will not be adversely affected by such modification. Each system shall comply with the provisions set forth in the State-approved plan. At a minimum, A State approved plan shall require the system modifying its disinfection practice to:

(1) Evaluate the water system for sanitary defects and evaluate the source water for biological quality;

(2) Evaluate its existing treatment practices and consider improvements that will minimize disinfectant demand and optimize finished water quality throughout the distribution system;

(3) Provide baseline water quality survey data of the distribution system. Such data should include the results from monitoring for coliform and fecal coliform bacteria, fecal streptococci, standard plate counts at 35° C and 20° C, phosphate, ammonia nitrogen and total organic carbon. Virus studies should be required where source waters are -- heavily contaminated with sewage effluent;

(4) Conduct additional monitoring to assure continued maintenance of optimal biological quality in finished water, for example, when chloramines are introduced as disinfectants or when pre-chlorination is being discontinued. Additional monitoring should also be required by the State for chlorate, chlorite and chlorine dioxide when chlorine dioxide is used as a disinfectant. Standard plate count analyses should also be required by the State as appropriate before and after any modifications;

(5) Demonstrate an active disinfectant residual throughout the distribution system at all times during and after the modification.

This paragraph (f) shall become effective on the date of its promulgation.

Appendix A—Summary of Public Comments and EPA Responses on Proposed Amendments to the National Interim Primary Drinking Water Regulations for Control of Trihalomethanes in Drinking Water

The following is a summary and discussion of the principal public comments to EPA's proposed regulations for the control of

trihalomethanes (THMs) in drinking water and EPA's responses to them. Many comments have already been addressed in the preamble which should be referred to for additional explanation of the agency's responses. In its February 9, 1978, notice of proposed rulemaking, EPA specifically solicited comments on the following six questions:

1. The reasonableness of the concept of phasing the application of the regulation by making the MCL mandatory initially only for large water systems and for the time being requiring monitoring only in others, and no requirements in the smallest systems. Should the regulations differentiate in their application between ground and surface water supplies? Are monitoring frequencies sufficient to identify locations with high TTHM levels?

An alternative approach on which public comments are solicited would be to make the MCL applicable to all public water systems and affect phasing of implementation by establishing a deferred monitoring schedule. Systems serving more than 75,000 people would be required to begin monitoring within one year of promulgation, systems serving between 10,000 and 75,000 would be required to begin monitoring within three years and all other communities within five years.

2. The magnitude of the MCL at 0.10 mg/l. Does the current information warrant more restrictive regulations at this time, for example, 0.050 mg/l or less? How rapidly can the MCL be reduced to lower feasible levels?

3. The feasibility and timing of the treatment modifications that will be necessary to achieve compliance. Will 18 months provide adequate time for most impacted systems to take steps to come into compliance?

4. The economic impact on large, medium, and small water systems either for the proposed regulation or for more restrictive regulations. Are EPA's estimates of the cost of compliance reasonable?

5. The concept of averaging the concentrations of the TTHMs for compliance—both the annual averaging of quarterly samples, and the averaging of representative samples within the distribution system.

6. The use of the Standard Plate Count as a more sensitive indicator of microbiological quality while treatment modifications are being introduced and the limitations on chlorine dioxide and chloramines.

In addition, the proposed regulations generated comments on other issues, including such issues as whether the States with primary enforcement responsibility had been provided sufficient time to make State regulations consistent with the federal regulations by the effective date. The majority of commenters did not address all of the issues that were posed by EPA; many commented on just a few issues or only on a single issue.

In all, EPA received 598 written comments and 259 oral statements were presented in the eight public hearings. The total of 857 comments came from various interested parties, including 390 from water utilities, 32 from private industries, 28 from consulting engineers, 95 from special interest groups, 80 from private individuals, 33 from educational institutions, 13 from Federal government agencies, 98 from local governments, 75 from local and State health and environmental departments, and 13 from other groups including some members of Congress. An additional 496 communications from members of Congress were received and responded to directly. Many of the comments were duplicative; some commenters presented both written and oral comments, or the comments were repeated in substance by many commenters, including members of Congress. In a number of cases, commenters simply endorsed the official position taken by a particular organization. For example, 124 water utilities and local governments responded by endorsing the position of the American Water Works Association (AWWA) which recommended an alternative program for the control of organic chemical contamination in drinking water. Comprehensive comments were also received from the Coalition for Safe Drinking Water (CSDW), a member organization of both municipal and investor-owned water utilities formed specifically to comment on EPA's proposed regulations, Calgon Corporation, a large manufacturer of carbon, and the National Drinking Water Advisory Council. These and other major comments are summarized in Appendix B. The following discussion summarizes comments received on the proposed regulations and the Agency's responses to those comments.

1. A majority of public comments disagreed with EPA's proposal to limit the applicability of the TTHM MCL to systems serving greater than 75,000 people. Most commenters preferred to have all water systems included under the regulation if control of chloroform was indeed deemed necessary (many of them did not feel any regulation was necessary). Phasing-in the applicability of the regulation to smaller systems in time was also opposed by some commenters, but a large number thought such a phasing approach to be logical.

The population cut-off of 75,000 received a total of 158 comments. Among the commenters, 132 felt that the regulations should be applied to all systems regardless of size; 22 commenters thought the population cut-off and phasing approach were reasonable. The main reason given by those who opposed the population cut-off was that they felt such an approach was contradictory to the intent of the SDWA which was to protect all persons served by community water systems. Therefore, these commenters said that if there was a health concern, all systems should be required to comply with the TTHM MCL, not just those who are served by a large water system. The commenters who thought that the population cut-off and phasing approach were reasonable cited as their reasons economic and technical feasibility, realizing that the larger water utilities would be better financed and staffed.

In response to the comments, EPA has accepted the recommendation of the National Drinking Water Advisory Council and many other commenters to broaden the coverage of the TTHM regulations to include those systems serving as few as 10,000 people and to phase-in the effective dates of the MCL by system size as follows:

- Water systems serving 75,000 or more people are required to be in compliance with the TTHM MCL within two years from the date of promulgation of the regulations.
- Systems serving between 10,000 and 75,000 people are required to be in compliance by four years from the date of promulgation.

This still means systems serving fewer than 10,000 persons are not covered by these regulations. EPA does not believe that this approach violates the intent of the SDWA to protect all persons served by community water systems. The great majority of smallest systems are served by ground water sources that are low in THM precursor content. Therefore, their drinking water is less likely to be subject to significant THM contamination. EPA is also concerned that measures taken by the smallest systems to reduce THM levels are more likely to result in drinking water of poor microbiological quality since they generally lack the expertise and access to technical assistance necessary for careful supervision of alterations in disinfection practice. Commenters are referred to the preamble to these regulations for a more complete discussion of EPA's rationale for excluding these smallest systems from

the coverage of these amendments to the Interim Regulations.

As discussed in the preamble, EPA's decision to phase-in the effective date of the MCL by system size has been based in part on the present limited laboratory capability available for TTHM analyses and the need for careful supervision of any alterations to the disinfection process. The systems in the 10,000 to 75,000 population range will be able to draw upon the experience gained by the first group of largest systems who must achieve compliance in the shortest feasible time-frame. By that time, laboratory resources and technical assistance from the States and EPA will be available to handle the increased number of systems. It was believed to be unreasonable to make the regulations effective for all systems at once for these reasons.

2. Thirty-seven comments were received on whether the regulations should differentiate between surface and ground water sources. Twenty-five opposed the idea of differentiation and said that the regulations should be based on water quality rather than water sources. Nine believed differentiation between sources was a good approach because in general ground water contains relatively less precursor material than surface water and therefore has less chance to produce TTHMs during chlorination practice. Three thought that the States should make the decision whether to distinguish between surface and ground water.

In response to these comments, the TTHM MCL applies equally to ground and surface water supplies within the population range covered. Water quality serves as the basic distinguishing factor to the extent that only those systems that exceed the MCL will be required to take steps to reduce TTHM levels in the finished drinking water. However, the monitoring requirements have been modified from the proposal to accommodate the valid concerns of some commenters that systems with relatively stable groundwater sources should not be required to incur the expense of regular monitoring where it is demonstrated that TTHM levels are not likely to approach or exceed the MCL. As discussed more fully in the preamble, the States have been accorded some flexibility to modify the monitoring requirements on a case-by-case basis under such circumstances.

3. Four comments were received on the monitoring and compliance timeframes established in the proposal. One of these commenters asked what would happen at the end of one year of monitoring for systems serving 10,000 to 75,000 people. He questioned why no action would be required if the TTHM levels exceeded the MCL. One commenter suggested that monitoring requirements be extended to systems which serve less than 10,000 population and report the results to customers as well as authorities. One commenter suggested that water systems serving more than 75,000 should start monitoring within 6 months, systems serving 10,000–75,000 should start monitoring within 1 year while the rest of the communities should begin monitoring within 3 years. One commenter felt that more discretion should be left to the States to determine which systems should be brought into compliance first.

EPA has responded to the comment concerning compliance by those systems serving between 10,000 and 75,000 persons by applying the TTHM MCL to those systems within 4 years of the promulgation of these regulations. Thus, systems in that size category that exceed the MCL would be required to take measures to reduce TTHM levels in their drinking water.

The monitoring requirements have not been extended to systems serving fewer than 10,000 people in the final regulations. Monitoring and public notification of the results were not believed to be warranted unless and until those smallest systems were also going to be required to reduce TTHM levels when the monitoring results showed that the MCL was exceeded. EPA was also concerned about the availability of laboratories for conducting TTHM analyses for the approximately 57,000 systems that fall within this size category. EPA's rationale for excluding these systems from the coverage of the MCL has already been addressed in response to other comments and in the preamble to these regulations.

The alternative monitoring timeframe suggested by one commenter was presumably intended to lengthen the timeframe that EPA had originally proposed as well as to require monitoring by the smallest size systems within a definite timeframe. In these final regulations, EPA has expanded the timeframe it originally proposed by requiring the largest systems to begin monitoring within one year from the promulgation of these regulations and the next size category within 3 years. EPA found that requiring the largest systems to begin monitoring within three to six months would not have provided adequate time for sufficient numbers of laboratories to become properly certified to perform quality TTHM analyses. An additional two years was believed to be necessary to insure the existence of quality laboratory capability to accommodate the approximately 2,300 more systems in the next size category. EPA's reasons for not requiring monitoring by the smallest size systems have already been discussed.

With respect to the comment suggesting that the States should have more discretion to determine which systems should be brought into compliance first, this regulation does not impair the State's prerogative to give highest enforcement priority to those systems with, for example, the highest TTHM levels. However, applying a uniform effective date for the MCL to the largest size systems first insures a fair application of the regulation among systems and achieves public health protection for the most people in the shortest timeframe. While it is the State's responsibility to enforce compliance with the MCL, it is each system's responsibility to achieve compliance by the applicable date.

4. Other monitoring-related issues submitted by commenters included: Seven commenters said that the proposed timing for monitoring was inadequate; several commenters said that it was premature at this time to require the water utilities to monitor for TTHMs while other commenters urged EPA to establish a deferred monitoring schedule; and two commenters felt that the monitoring requirement and the setting of a MCL should be a two-step action including initial monitoring followed by setting the MCL. One commenter believed that it was necessary to establish an occurrence data base prior to setting a MCL and recommended that monitoring must span at least a 2 to 3 year period in order to determine the varying concentrations of these contaminants.

As noted previously, the effective date of the monitoring requirements has been extended to one year and three years for the two size categories, respectively. This extension will allow adequate time for development of laboratory capabilities. In regard to the two step approach suggested by two commenters and the establishment of an occurrence data base prior to setting an MCL, the EPA agrees with the commenter's concept and has included both steps in the regulations: monitoring followed by compliance with the MCL. A sufficient data base has been established for setting the MCL and monitoring for one year prior to the effective date of the MCL will provide more precise information on variations in TTHM levels. Of course, systems may, at their

option, begin monitoring prior to the effective date.

5. With regard to EPA's proposed monitoring frequencies for TTHMs of five analyses per quarter, 37 comments were received. Eleven comments said that the proposed monitoring frequencies were reasonable. Twenty-two felt that quarterly sampling was insufficient, and some suggested more frequent sampling, such as one sample every month. Two commenters thought the proposed frequencies were too frequent and suggested that monitoring be conducted twice a year. Two commenters suggested that the frequency should be proportionate to the population served and at regular intervals.

EPA has retained the quarterly sampling requirements of the proposal as the minimum acceptable frequency for determining the effect of differing treatment practices and seasonal variations in raw water quality on TTHM concentrations in the finished drinking water. Four instead of five samples per quarter are required based on the number of treatment plants used by the system. Thus, more samples must be taken by those larger systems most likely to utilize more than one plant. This also allows for more representative sampling since TTHM levels may vary depending upon the system's raw water source or treatment program at a particular plant. Systems may seek State approval to have multiple wells drawing raw water from a single aquifer considered as a single treatment plant for the purpose of determining the minimum number of samples.

In response to those comments seeking more frequent sampling, generally, the final regulations provide that the States may require more frequent sampling where it is necessary to insure adequate and consistent control of TTHM levels below the MCL in the water served to all consumers of the system. EPA also recognizes that, in some situations, quarterly sampling should not reasonably be required because the maximum TTHM potential in some ground waters is consistently well below the TTHM MCL. Thus, the final regulations also allow the States to exercise their discretion to reduce the monitoring frequency in those situations. The requirements of these regulations have thus been fashioned to establish a minimum regular monitoring frequency while providing for case-by-case flexibility, recognizing that the optimal monitoring frequency for TTHM control will depend largely on site-specific circumstances.

6. Many comments were received charging that EPA's action of setting a TTHM MCL of 0.10 mg/l was arbitrary, premature and lacking in supporting data. 243 comments suggested that EPA adopt 0.10 mg/l TTHM as a goal rather than a regulation while additional data were being collected and more research on the health effects of the TTHMs was being conducted.

EPA believes that a TTHM MCL of 0.10 mg/l is adequately supported by the evidence in the rulemaking record demonstrating that THMs "may cause any adverse effect on the health of persons" (Section 1401) and that such a standard "shall protect health to the extent feasible, using technology, treatment techniques, and other means, which the Administrator determines are generally available (taking costs into consideration) on the date of enactment" of the SDWA, as required by Section 1412. Although new information will always be forthcoming on any regulatory subject, EPA must make the critical decision of when a sufficient basis is established to support regulatory action in order to comply with the protective intent of the SDWA. Citing the House Report accompanying the Act, the United States Court of Appeals for the District of Columbia Circuit has noted that "controls were not to be delayed pending the development of more refined data on health effects and more efficient detection and treatment technology" (*EDF v. Costle*, 578 F.2d 337, 344 (D.C. Cir. 1978). As discussed in the preamble to these regulations, EPA's mandate to protect the public health to the extent feasible does not contemplate the mere establishment of "goals" which utilities may choose to ignore when the evidence demonstrates that protective action is warranted.

7. Ten comments suggested that if a MCL were to be set for TTHMs, the MCL should be 0.30 mg/l. Other comments suggested higher TTHM MCLs than EPA's 0.10 mg/l ranging from 0.25 mg/l to 15 mg/l. Although most of these suggested MCLs were offered without supporting data, two commenters submitted suggested MCLs based upon their own studies or formulas. One commenter suggested a MCL of 0.3 mg/l for chloroform based upon his studies on dogs, rats and mice in the laboratory while another commenter calculated an MCL for chloroform in drinking water of 0.429 mg/l. Thirty-four comments supported the proposed MCL of 0.10 mg/l for TTHM while 11 comments said that a MCL of 0.10 mg/l should be lower but did not provide supporting data.

In establishing a TTHM MCL of 0.10 mg/l as an Interim Regulation, EPA has struck a reasonable balance between requiring the reduction of TTHM levels in drinking water to protect the public health and what public water systems could reasonably have been expected to achieve in 1974, taking into account technological and economic feasibility. EPA has also been mindful of the fact that corrective measures taken to comply with a TTHM MCL have the potential for adversely impacting the microbiological quality of a system's drinking water. Although technologies are available to reduce TTHM levels below 0.10 mg/l, EPA believes that a more stringent standard at this time would unnecessarily jeopardize the overriding need for quality disinfection. Moreover, EPA expects that many systems striving to comply with the standard of 0.10 mg/l will, in fact, achieve lower TTHM levels as well as a reduction in other potentially harmful disinfection by-products. Thus, EPA's approach to the regulation of THMs, as discussed more fully in the preamble to the regulations, has been both deliberate and cautious.

EPA does not believe that a less stringent MCL is warranted. Based upon EPA's occurrence data, if a less stringent standard were established, very few systems would be required to reduce the TTHM levels in their drinking water, resulting in no improvement of water quality served to their consumers. While this would relieve many systems from any costs, it would clearly not further the protective intent of the SDWA. EPA has determined that treatment methods have been generally available since 1974 at reasonable cost to reduce TTHM levels to 0.10 mg/l, and therefore, a higher standard would not be justified.

As to those commenters who suggested that an MCL of 0.3 mg/l for chloroform could be computed as a "safe" level for human consumption by incorporating an uncertainty factor of 2,000 into Roe's "no observed effect dose." EPA has concluded that such an approach is inappropriate when dealing with human risk from chronic exposure to a potential carcinogen. That approach assumes the existence of a threshold level below which no risk would exist. It is thus inconsistent with the principles stated by the NAS in its report, "Drinking Water and Health". In addition, 0.3 mg/l is well above the levels that are currently achievable in the large majority of public water systems by generally available methods that are technically and economically feasible. Roe's study has been specifically addressed elsewhere in this Appendix.

8. Sixteen comments responded specifically to the question of whether the current information warrants more restrictive regulations at this time and how rapidly the MCL could be reduced to lower feasible levels. Except for one commenter who said that a TTHM MCL of 0.05 mg/l would be technically feasible today at reasonable cost, the other 15 commenters all said that a more restrictive regulation was unnecessary due to questions regarding the health basis of 0.10 mg/l. Further, they expressed serious doubts that a much lower MCL could be met without extensive modification in treatment processes. Several comments disapproved of the agency's intention to make the MCL more stringent in the future, noting that it might be difficult for water utilities to cope with a moving target since the economics of system improvements frequently depend upon the level of control sought. State activities would be seriously disrupted because utilities would have to re-modify their treatment processes whenever new standards were set (modifications would require State approval), and the States would have to change their regulations to retain primary enforcement responsibility.

EPA has already explained its rationale for not imposing a more restrictive standard for TTHMs at this time in its response to other comments and in the preamble to these regulations. EPA's health basis for these regulations is also discussed elsewhere in the preamble and in this Appendix. EPA agrees that reducing TTHM levels to 0.05 mg/l would necessarly result in increased costs greater than those estimated to achieve EPA's MCL of 0.10 mg/l; it is, however, EPA's concern for the potential adverse impact on disinfection practices and microbiological quality rather than the increased cost that has let EPA to conclude that a more stringent standard is not justified at this time.

When EPA establishes Revised Primary Drinking Water Regulations, the Act clearly authorizes and indeed requires, more stringent and more comprehensive regulations of those contaminants which may have an adverse effect on human health, including TTHMs. Congress contemplated that, as new technologies were developed to reduce the level of contaminants in drinking water, EPA's regulations would be reevaluated accordingly. Since new information regarding health effects and treatment technology will continue to be generated, it would be unrealistic to expect that EPA's requirements would remain static. However, EPA recognizes the increased burden placed on water utilities and the States when more stringent regulations are promulgated; when this occurs, adequate opportunity for public comment and time for compliance with any more stringent regulation will be provided.

9. On the question of feasibility of compliance with EPA's proposed TTHM MCL, three commenters said that more research is needed to study the feasibilities of different treatment processes for the removal of TTHMs. One expressed the need for EPA's assistance in evaluating the appropriate treatment for his system. One suggested that ozone in combination with a chlorine residual, when the two are properly used together as part of a total treatment scheme, often results in a significant reduction in the ultimate TTHM levels. One said that granular activated carbon (GAC) is good for TTHM removal as well as taste and odor.control. One stated that the type of treatment modification used for compliance with the MCL should be determined by the water utility.

EPA believes that despite the ongoing research being conducted on control of THMs in drinking water, sufficient evidence exists to demonstrate that technology and treatment methods were generally available in 1974 at reasonable cost for water systems to achieve TTHM levels of 0.10 mg/l. Such methods include both relatively inexpensive alterations of a system's disinfecton practices, which will be sufficient in most cases to reduce TTHM levels to below the standard, as well as more complex treatment modifications, such as those suggested by two commenters. EPA's findings regarding the feasibility of TTHM control are fully set forth in the report "Interim Treatment Guide for the Control of Chloroform and Other Trihalomethanes," which has been incorporated by reference as part of the Agency's Statement of Basis and Purpose for these regulations.

A 1978 report prepared by J. S. Zagorski, G. D. Allgeier and R. L. Mullins, Jr., "Removal of Chloroform from Drinking Water," studying the reduction of chloroform formation upon subsequent chlorination, reported that various common treatment processes including sedimentation; sedimentation followed by chemical coagulation and precipitative softening; sedimentation, chemical coagulation, precipitative softening and rapid sand filtration; and sedimentation followed by chemical coagulation, precipitative softening, rapid sand filtration and GAC adsorption resulted in substantial reductions of the chloroform formation potential. They also reported that both alum and polymers at moderately large dosages were capable of reducing the potential of Ohio River water to form chloroform and other THMs. Both ozonation and powdered carbon at high doses also reduced THM formation potential. In the plant-scale studies, the same investigators also reported that moving the point of chlorination from the head of pre-sedimentation reservoirs to the head of the coagulation process significantly reduced the concentration of $CHCl_3$ in finished water, and that ammoniation at the head of precipitative softening ceased the THM formation reaction and markedly reduced the level of THMs in softened water. Aeration also was able to reduce chloroform in finished water.

As explained in EPA's response to other comments and in the preamble, in light of currently available information, EPA need not wait for the results of additional research before establishing regulations to control TTHMs. Rather, any new information will be considered by EPA when it develops Revised Primary Drinking Water Regulations.

EPA agrees with the comment that the type of treatment modification used to comply with the TTHM MCL must be determined by the water utility that has the ultimate responsibility to select a method for achieving compliance. Many commenters appeared to erroneously confuse the TTHM regulation with EPA's proposal of a specific treatment technique for control of pollution-related synthetic organic chemicals in drinking water. Nevertheless, technical assistance will be provided by EPA and the States on a case-by-case basis. Systems that modify their treatment processes to comply with the TTHM MCL are also required to obtain State approval of their plans prior to implementation to insure proper supervision of alterations in disinfection practice.

Significant reductions in THMs can normally be achieved by making relatively minor modifications to existing water treatment systems, such as maximizing the efficiency of precursor removal during coagulation/filtration or changing the point of chlorination. Where minor modifications to existing treatment methods prove insufficient to bring the system into compliance with the MCL, the system may need to use an adsorbent technology, such as GAC, to reduce precursors and thereby achieve compliance with the MCL. Thus, each system will probably be using a

combination of the available treatment options that will be most effective for its situation. Because of these treatment alternatives, total reliance upon an adsorbent for reduction of the THMs to below the MCL will not likely occur. The EPA has estimated that of the approximately 2,700 systems serving more than 10,000 people required to comply with the MCL, approximately 25 systems may ultimately need to install adsorbent technology to control THMs.

10. One commenter stated that GAC has never been tested or proven in full-scale operation in the United States and therefore constitutes a nationwide experiment in water treatment.

The availability and efficacy of GAC technology has been clearly demonstrated by the large extent of use by numerous facilities in the United States as well as overseas. GAC technology has been used for many years in the water treatment industry, and today over 60 drinking water plants presently use GAC in their treatment facilities. Extensive use of GAC is practiced in the food and beverage industry for removal of organic contaminants from process waters and in the treatment of industrial and municipal waste waters prior to discharge to receiving waters. GAC for removal of organic chemical contaminants has been in use by numerous European municipal drinking water plants since the 1960's and as industrial activity continues to increase, more facilities using GAC are being installed.

Most drinking water plants in the U.S. have been using GAC as a replacement for the media in their existing filters for the stated purpose of removal of taste and odors. However, with the development of more sophisticated analytical procedures which are capable of detecting and measuring levels of organic chemicals (including THMs and THM precursors) in drinking water, EPA now knows that such chemicals are actually being removed by GAC and that their presence, previously undetectable by analytical measurement, was being manifested through taste and odor problems.

Commenters nevertheless question the availability of means for the regeneration of GAC and use of GAC in post contactors for removal of organic compounds. Regeneration of GAC has been demonstrated in numerous locations including a full scale operation at a drinking water facility in the late 1960's in the U.S. Some European drinking water plants have also been regenerating GAC for several years. The frequency at which drinking water plants in the U.S. replace the GAC ranges from less than six months to two to three years. The GAC is usually removed from the facility and replaced by virgin carbon.

In addition to its use by numerous and varied types of drinking water systems in the U.S. and overseas, GAC has been widely and successfully used for the treatment of municipal waste waters for removal of organic chemical pollutants. For example, since the mid-1960's, the municipality of Lake Tahoe has used GAC in contactors with on-site regeneration. Thus, regeneration technology has been applied both on site and at central furnace facilities. Frequency of regeneration will necessarily be dependent upon TTHM reduction needed on a case-by-case basis. Numerous drinking water treatment plants are presently operating modules of full scale GAC systems or pilot plants to more fully correlate GAC performance with various regeneration frequencies.

11. One commenter stated that the GAC treatment process may result in serious problems and these may outweigh the alleged environmental benefits associated with GAC treatment. These problems include potential air pollution from regeneration and the waste water associated with air pollution scrubbers as well as waste water from backwash and drainage from carbon slurries.

GAC is normally regenerated at furnace temperatures of 750° C to 900° C and at these temperatures, data do not show that most pollutants are oxidized to other than harmless compounds. EPA has considered potential waste disposal problems including air and water pollution relating to GAC reactivation and has found that techniques are available to control wastes from these facilities.

In regard to discharge of backwash water or drainage from carbon slurries (if at the water treatment plant), no additional water is expected to be necessary. In fact, less water is normally used in backwashing with GAC than with conventional media in the filter. Any drainage from carbon slurries at the off-site GAC regeneration facility is not large in volume and normally is discharged to municipal treatment plants.

12. Several commenters were concerned that the use of GAC may constitute a larger health hazard than means for improvement of water quality. The alleged health hazards associated with GAC included desorption, chromatographic effect (competitive displacement), resorption (leaching) of heavy metals and polycyclic aromatic hydrocarbons contained in the virgin or regenerated carbon, release of carbon fines, promotion (catalytic reactions) on the carbon itself of hazardous compounds due to chemical reactions between chlorine and organic compounds, bacterial growth on the carbon and air pollution from regeneration facilities. Commenters also noted that indirect hazards were associated with GAC usage through the manufacture of GAC and the production of energy necessary to operate GAC facilities. They said these industries, such as the coal industry, pose a high risk of morbidity and mortality to the workers. Because of these concerns, they urged that additional research and testing should be conducted prior to implementation of GAC in this country's major waterworks. It was suggested that toxicological evaluations be conducted using concentrated effluents from GAC to assess these potential hazards.

EPA has evaluated the potential hazards associated with the use of GAC. The items listed can be shown to occur under specific laboratory conditions directed at obtaining a specific reaction, such as the promotion reaction or the chromatographic effect, but no significant hazard is expected under actual use conditions so long as proper operating procedures are followed. For example, use of GAC for THM control will not result in desorption of TTHM to levels above the MCL since the GAC would be regenerated at the point where THM levels in the effluent approached those in the influent. Also, bacterial growth on GAC is common, is frequently encouraged by adding oxygen to the influent waters, and assists in reduction of precursor compounds. Control of bacteria in the finished drinking water is effectively accomplished by disinfection and the alleged slugs of bacteria breaking through the GAC do not occur with proper operation; in any event, proper disinfection with a residual throughout the distribution system would eliminate this potential hazard.

In addition, present data have not shown a health hazard associated with the use of GAC in its many applications in drinking water treatment. Nevertheless, EPA is continuing to conduct research on these questions. For example, short term bioassay studies are being conducted with animals using concentrated raw and finished waters to assess the toxicological significance of various disinfectants, such as chlorine and ozone, and the use of various treatment technologies, including GAC. However, the methodologies used in these studies are only now being developed and must be verified by more established methods.

13. Twenty-seven comments were received discussing the proposed effective date of the TTHM regulations. In general, the commenters thought that the compliance dates for either the monitoring requirement or the MCL were unreasonable. A number of these commenters had apparently confused the effective date for the TTHM regulations with that for the treatment technique requirement and commented accordingly.

Specifically, 11 commenters said the allowed time for compliance with the proposed regulations was unreasonable without specifically referring to whether the comment was addressed to the monitoring schedule or the MCL. Nine commenters, however, submitted specific time-tables that they felt would be required for compliance with the proposed TTHM regulations ranging from monitoring beginning 3 months after promulgation of the regulations to as long as 8 years for the completion of plant modifications.

One commenter submitted his suggestion of a specific time-table including the following: (1) Request for variance or exemption should be submitted no later than the effective date, (2) design specifications should be submitted to States for approval no later than 18 months after the effective date, (3) by no later than 24 months after the effective date, final design plans and specifications should be submitted to States for approval, (4) construction should be completed and operation should begin no later than 4 years after effective date, and (5) operational data should then be submitted to States for evaluation. One commenter suggested postponement of the regulations and instead conducting a two-year comprehensive monitoring program. One commenter felt that the proposed time-table of the TTHM regulation was adequate.

Thirty-four commenters said that EPA's proposed effective date, allowing 18 months for compliance, was unreasonable and that it was technically impossible for systems to design the most cost-effective treatment system within that timeframe. These comments suggested allowing additional time for compliance, ranging from 3 to 7 years. Four thought the allowed time of 18 months was adequate. Three said the regulation should be more flexible with regard to the time for compliance and the type of treatment modification used and suggested that the States make these decisions. One commenter said that the allowed 18 months was adequate if only minor modifications were needed but that additional time would be required if major changes to the treatment plant were needed. Another commenter said that whether the allowed timing was adequate would depend upon whether the particular water system would need to use GAC to remove TTHMs. One stated that the primacy States should have a minimum of two years to revise their regulations to be consistent with the regulations finally adopted by EPA before they became effective requirements for the water supplies. One commenter said that although the proposed timing was feasible, in most cases, the final regulations should provide for a delay in the effective date for systems that could show the need for additional time. One commenter said that the proposed compliance schedule was appropriate if the MCL were established at 0.30 mg/l.

EPA has responded to the comment seeking more time to achieve compliance by extending the effective date of the TTHM MCL for systems serving more than 75,000 people to two years after the promulgation of these regulations. Systems serving between 10,000 and 75,000 people have been given four years to achieve compliance with the MCL. Both dates take into account the need for one year of monitoring data to be established and the need for adequate time to develop quality laboratory capability for TTHM analyses. The two-year effective date of the MCL for the first size category also serves to provide primacy States with sufficient time to amend their regulations before the MCL takes effect. In the meantime, EPA will not allow State primacy to be needlessly jeopardized. The Agency will be proposing regulations shortly as amendments to 40 CFR Part 142 which will allow for a reasonable amount of time for States to conform their regulations to the federal requirements.

The extended timeframes suggested by some commenters do not appear to be warranted for applicability to all systems. It appears that these commenters may have been erroneously assuming that GAC was being required for control of TTHMs in all cases. On the contrary, EPA believes that most systems will be able to achieve compliance with the TTHM MCL of 0.10 mg/l with relatively minor changes to their existing treatment processes. Therefore, the timeframe provided in the final regulations should provide ample time for compliance measures to be implemented. However, EPA recognizes that additional time may be needed by those few systems that will need to institute more complex treatment modifications to comply with the TTHM MCL. In such cases, Section 1416 normally provides for the issuance of exemptions. Due to the belated issuance of these amendments to the Interim Regulations, an extension of the compliance deadlines presently established in Section 1416 will be needed to authorize exemptions from the TTHM MCL. EPA will seek a legislative extension of the exemption deadline. So long as good faith efforts are being taken by systems to comply with the TTHM MCL, EPA and the States may exercise their enforcement discretion to insure compliance as expeditiously as practicable.

14. Seventy comments addressed the specific cost estimates for installation of the technologies as well as the projected national cost impacts of the regulations. The majority said that EPA's estimates were not reasonable and that the actual costs would be considerably higher. A few comments felt that the costs were reasonable or "in the ball park."

Of these comments, 32 stated that the costs for installation of the technologies were low while five thought that the estimates were reasonable. Some of these felt that the EPA estimates in most cases did not conform to local economic conditions. Other commenters said the EPA's costs were underestimated and submitted cost estimates for their particular utilities in support of their argument. They indicated that compliance with the MCL would require far larger investments by the utility than those estimated by EPA. In addition, one commenter provided data showing that the cost impacts would be higher because his public water system used 225 gallons per capita per day (gpcd) as compared to the 179 gallons per capita per day used by EPA in the estimates. The commenter also used maximum daily and hourly flows of 240 percent and 390 percent of average daily flows, respectively, and 65 percent of the total year's flow occurred during the four summer months.

EPA's analysis of the cost and economic impact of the final regulation is discussed in the preamble and described in detail in the "Economic Impact Analysis for a Promulgated Regulation for Trihalomethanes in Drinking Water". The costs of treatment are based upon average national costs and were determined from an analysis of the costs of materials and labor rates in various parts of the United States. The costs of treatment represent those of an average size utility in each of several size categories, and serve as the basis for assessment of the national cost impacts. It is expected that some utilities would experience costs that are

higher than the average system in its size category, while others would be lower. In order to reflect site-specific factors for a utility, contingency factors are incorporated into the treatment cost estimates.

The base flows used in the cost analysis are values representing the average flow conditions for a certain size range of systems. The values are based upon a recent survey of 1,000 water systems in the United States during which it was determined that larger systems have higher water usage per capita than do smaller systems. This is a result of commercial and industrial customers. Thus, a different flow base was used for each size category ranging from 155 to 210 gpcd for systems serving one million persons or more. Capital costs were based upon capacity flows and O&M costs were based upon average daily flows. The exception was that capital costs of GAC were based upon the average day in the peak month which was less than the capacity flow. Commenters are referred to EPA's document "Economic Analysis" for further details.

15. One comment noted that it was difficult to determine whether EPA's estimates of the cost for compliance were reasonable. He felt that debt service, the additional water treatment plant personnel laboratory assistance and control, and more sophisticated monitoring equipment, were not adequately considered. One commenter stated that it would cost $20,000 to $30,000 per year to conduct monitoring for his utility. Four said that the compliance cost for TTHM analyses estimated by EPA at $25 per sample was low and that the current rate for commercial TTHM analyses was approximately $100 per sample exclusive of sampling and delivery costs. Two other commenters suggested that prices of $75 and $120 per sample, respectively, were appropriate. Three commenters agreed with the EPA's estimation of monitoring costs.

EPA's analysis of the costs of treatment specifically considered each of the items of concern to the commenter. Debt service is included in the annual costs (revenue requirements) and includes interest rates on capital of 8% and 10% for public and privately-owned utilities, respectively. The rate for privately-owned utilities was revised from the 9% rate used in the cost estimates supporting the proposed regulations to take into account the current and projected cost of capital. Additional plant personnel were included in the O&M costs and thereby in the annual costs.

In regard to monitoring costs, the total required monitoring costs were estimated to be $800 per year per system based upon four samples per quarter. As noted in the preamble, monitoring costs for some systems will be higher than $800 per year because these systems have more than one plant, thereby necessitating (in some cases) additional sampling. This cost estimate included costs of analysis at $50 per sample. The cost of sampling and mailing samples to an outside laboratory was not considered to be significant. No additional sophisticated monitoring equipment was included in the estimate; however, it was anticipated that many systems would purchase analytical equipment to perform their own analyses. While commercial rates for TTHM analyses varied from $25 per sample to more than $100 per sample, $50 was used as a reasonable estimate and this was increased from the value of $25 per sample used in the proposed regulations. However, it is expected that the cost per sample will likely be lower, since increased availability of analytical services, competition between laboratories and the increased number of samples for analyses will provide opportunities for cost-savings.

In addition to the costs associated with the required monitoring, additional costs will be incurred by some systems in the monitoring conducted to assure that the bacteriological quality of the drinking water will be maintained during and after treatment modifications for the purpose of reducing THM levels. Costs of this monitoring will vary between systems but will not likely exceed approximately $5,000 at systems with the most extensive monitoring program. This estimate was based upon use of outside contract laboratories, and it is expected that most water systems will conduct some of the analyses in their own laboratories, thereby reducing the costs. Nevertheless, this cost is considered reasonable for those systems which will need the most extensive monitoring (e.g., for systems serving 10,000 people, this cost would be $0.50 per person), and is a one-time expense (as opposed to continued requirements for quarterly TTHM monitoring).

16. One commenter said that the use of a forty-year amortization period to determine the yearly cost for capital improvements was unreasonable in that the life of the water treatment facility would be considerably less than 40 years.

Forty years was used as representative of the average expected life of equipment in public water systems. While some equipment may require replacement sooner than 40 years, other equipment has a life greater than 40 years. While privately-owned utilities often depreciate equipment at a 20-year rate, this is primarily for tax advantages and does not represent the true life of the equipment. Publicly owned utilities most often use rates of approximately 40 years since no tax advantages are available. Since over 80% of water systems are covered by this regulation and are publicly-owned, it is reasonable to use the 40-year amortization period as the basis of annual costs.

17. One commenter said that EPA's use of $5.58 per hour for labor in its EPA cost estimates was too low, stating $7.00 per hour for labor cost would be more appropriate. The cost estimates have been revised and now include labor costs at $11.75 per hour including fringe benefits. In addition, it should be noted that contrary to the commenter's statement, the proposed regulations were based upon an average labor cost of $7.50 per hour.

18. A number of commenters argued that the costs were underestimated because of specific factors in the analysis. For example, one commenter stated, based upon the use of GAC, that the difference between his potential national cost estimates and EPA's estimates could be explained primarily by four factors. It was not clear to what extent these comments differentiated between costs for GAC for TTHM control and costs for GAC to control other synthetic organic chemicals in the separate treatment technique requirement. The four specific areas of difference noted by this commenter and EPA's responses are as follows:

(a) EPA determined its estimated capital costs for a system based upon the capacity of the entire system; whereas, the commenter estimated the system capital costs as equal to the sum of the capital costs for each treatment plant based on the capacity of each plant.

The EPA recognizes that several large public water systems use more than one treatment plant and thereby might be required to install necessary treatment at each plant if they utilize the same or similar source waters. Due to the limitations of available data, the cost estimates were based upon installation of treatment for the total flow capacity of each water system, rather than separate flows from each plant. EPA does not believe that per plant costs would significantly affect the national cost estimates. Treatment costs depend upon flow capacity whether apportioned per plant or taking the system as a whole. In some cases, costs could be

reduced if only the flow from a single plant required treatment to reduce TTHMs. These effects have been taken into account by including contingencies in the cost estimates. Moreover, since it is generally the larger systems that have multiple plants, additional costs of treatment will be borne by a greater number of customers, reducing the per capita impact.

(b) EPA's estimates were based upon the system capacity on the average day of the peak month; whereas, the commenter's estimates were based upon the actual capacity of each treatment plant.

As presented in the cost analysis and discussed above, costs were determined based upon system capacity except for the use of GAC which was based upon the average day in the peak month. This was determined to be an appropriate cost base rather than total plant capacity because compliance with the MCL will be based upon a running annual average of average quarterly monitoring results and not a peak value.

(c) EPA assumed that some of the affected systems would design facilities for a 9-minute empty bed contact time (EBCT); whereas, the commenter assumed that all GAC facilities would be designed for an 18-minute EBCT.

It is anticipated that for most systems, 9 minutes EBCT will be adequate to achieve the MCL. It is possible that certain systems may require additional contact time but use of an average condition is entirely appropriate in the development of a national cost estimate. Use of 18 minutes EBCT as the base of the national cost estimate would have inflated the costs unrealistically.

(d) The commenter's estimates for specific systems, based on the costing out of the individual components, were 30–80% higher than EPA's proposed estimates.

As stated previously, EPA's cost estimates have been substantially revised to take into account many of the commenter's concerns. The cost estimates have been based upon the most accurate and recent sources of information and cost data available and that have been reviewed within the industry. Differences between the commenter's costs and EPA's proposed cost estimates were primarily due to differences in the base year for the estimates (EPA was 1976 dollars and the commenter was 1978) and differences in EBCT (9 minutes vs. 18 minutes). In any event, the commenter's detailed estimates have been evaluated and the EPA estimates have been revised appropriately.

The commenter's O&M cost estimates were higher than EPA's primarily because they were based on expenses at multiple treatment plants. Certain specific costs, such as the price of GAC and fuel costs, also account for portions of the differences and have been revised in the final cost analysis. EPA's GAC costs were based upon current and projected costs and ranged from $0.65 to $0.84 per pound of GAC depending upon the size of the public water system. Fuel costs were also projected and included estimates for 1980 of $0.84 per gallon for diesel fuel, $0.0038 per cubic feet for natural gas, and $0.038 per kilowatt-hour for electricity. The commenter's revenue requirement estimates were higher than EPA's primarily because of the higher estimates of capital and O&M costs.

19. Two comments stated that the costs were understated because the increased demand for materials required to comply with the regulations would cause costs to rise beyond normal inflation rates. This concern has been evaluated and, as shown in the economic analysis, no single chemical or component of any of the available treatment technologies is expected to experience a sufficiently large demand so as to affect its price. For example, the initial demand for GAC (to meet these regulations) is estimated to be four million pounds whereas the industry has excess GAC capacity of more than 100 million pounds per year.

20. One commenter stated that the EPA estimates did not include costs for land that would be necessary for installation of the GAC facilities. As shown in the economic analysis, costs of land acquisition were included in the capital cost estimates.

21. One commenter indicated that the EPA's estimates were based upon 1976 costs. He felt that approximately 20 percent increase was needed just due to elapsed time to date (1978) and that at the time of construction of needed facilities, another 50 percent inflationary increase would be applicable.

EPA's costs have been revised to reflect anticipated use of 1980 dollars to meet the regulations. The estimates were increased to 1980 dollars through the use of the available cost indices which included separate indices for labor, steel, excavation, concrete, manufactured equipment, pipes and valves, electrical and instrumentation, housing, and producer prices. These indices took into account anticipated inflation to 1980 and the precise index values are presented in the economic analysis and supporting documents. Overall, unit costs have been increased by approximately 36% as a result of this change from 1976 to 1980 dollars.

22. A number of commenters stated that the use of GAC will have substantial financial impact upon water supplies and that actual costs are very difficult to predict and are understated. For example, the average capital cost for a system serving over one million people was alleged to exceed $106 million with annual costs of more than $23 million. These commenters estimated that rate increases for residential customers would be in the range of 40–70% and that these rates could double where there were site-specific problems, such as land acquisition. The commenters claimed that these costs may result in insurmountable problems at some utilities in obtaining financing for GAC treatment facilities. They charged that EPA's assessment of the feasibility of financing the GAC treatment facilities was totally out of step with the realities of both the financing markets and operating needs of the public utilities.

Costs for GAC treatment are highly dependent on the substances being removed and the target level in finished water. The use of GAC to control THM precursors would not require the most stringent design and operating characteristics in most cases. Thus, the cost for this application would likely be very much less than the cost for using GAC to control synthetic organic chemicals. As noted in the preamble, EPA's cost estimates for using GAC for TTHM control were revised from those costs supporting the proposed regulations. For purposes of the economic analysis supporting this final THM regulation, EPA estimated the costs for a system using GAC by replacing its existing filter media with GAC and regenerating its carbon no more frequently than once every 12 months. Only systems with severely contaminated raw water sources will require the extent of GAC usage that the estimates accompanying the original proposal were based upon (post-filtration contractors with two month regeneration cycles). The data indicate that in most cases the raw waters were relatively uncontaminated and this was used in determining feasibility of treatment and reasonableness of costs for purposes of establishing the MCL. Thus, the revised costs are significantly lower than those in the economic analysis of the proposed regulations. Of course, the economic impact analysis is based upon a specific model system and costs will vary depending upon specific details at each site. To a reasonable extent, site-specific factors were included in the revised analysis and EPA's supporting economic document should be consulted for details. The document also examined the feasibility

of financing and found that financing is available.

23. Nine commenters said that the cost estimation should be more realistically based upon results from controlled experiments such as field studies. As the commenters suggested, one of the primary factors considered by EPA in developing the cost estimates has been the engineering application of the available treatment technologies. EPA has revised its cost estimates to reflect the engineering costs developed by Culp, Wesner, and Culp, consulting engineers with extensive experience in water treatment technology.

24. One commenter stated that the costs for GAC did not include the investment necessary for disposal of the concentrated organics removed from the off-gases by either landfill or underground injection. The cost estimates for use of GAC are based upon off-site regeneration, and all aspects of regeneration of GAC, including disposal of scrubber waters and other waste products were taken into account.

25. Two commenters stated that the cost estimates were low because EPA did not include the costs of installation of conventional treatment (coagulation, sedimentation, filtration) followed by THM control. The commenters indicated that some water supplies use sources from such places as the Adirondacks which do not necessitate conventional filtration but have TTHM levels at 150 to 250 mg/l. One of the commenters stated that for his system, which serves 140,000 people, to meet the MCL filtration would have to be installed at a capital cost of $12 million, an annual cost of $1.3 million, and a rate increase of 60 percent.

Most public water systems use conventional treatment technology and thus EPA's cost estimates included only those treatment technologies that are additions or adjustments to such conventional treatment. It would not be appropriate to include the costs of conventional technology in these regulations since, in most cases, compliance with other requirements of the NIPDWR (e.g., turbidity) necessitate use of conventional treatment. Therefore, the cost of conventional treatment should not be directly attributable to this regulation. Nevertheless, many of these systems are expected to be able to comply with the regulations through adjustment of chlorination procedures or use of an alternate disinfectant.

26. One commenter stated that the economic impact assessment did not take into account the costs of treating waste water from GAC operations, such as backwash waters, wet scrubbers and drainage from carbon slurries. It was estimated that 50,000 gallons of waste water will be generated for every one million gallons of drinking water treated and half of that amount would need to be discharged. This commenter concluded that this would result in increased flows and an approximate 4% increase in operation and maintenance costs at municipal waste water treatment facilities.

EPA's estimates did take into account disposal of any additional waste waters from the use of GAC. For example, the cost estimates were based upon regeneration of carbon at an off-site, privately owned, regeneration facility. The costs of regenerated carbon utilized in the estimates were based upon actual manufacturer's estimates and operating rates. Overall rates included costs of GAC regeneration and all ancillary activities such as air pollution control and disposal of waste waters.

27. Several commenters stated that the estimates were low because EPA did not include the administrative, environmental, overhead, and political costs of implementing the regulations. Two of these commenters felt that additional dollars would be required for such items as cost of processing variances, public hearings, research costs into health and treatment aspects of the regulations, monitoring compliance, laboratory instrumentation and facilities, and laboratory certification programs.

The Agency agrees that each of the above items has some degree of costs associated with it and has taken appropriate costs into account in the revised cost estimates. Systems would not be expected to conduct research into the health aspects of the regulations, and only research into treatment aspects to the extent necessary to determine which treatment would be most effective in meeting the MCL. Costs attributable to administrative or legal (or political) factors, processing, variances, and public hearings are difficult to precisely estimate. They have been included in appropriate parts of the estimate. Thus, administrative and legal costs have been included in the engineering costs at a rate of 12% of the total treatment cost. Some of the overhead costs have been included in the O&M costs which include labor rates with fringe benefits. Further, costs associated with monitoring have been included in the monitoring costs; environmental costs have been considered in GAC regeneration costs which would take into account such items as air pollution control equipment and disposal of by-products; finally, any other costs not included in those components of the total cost have been included in the contingency added to the costs.

28. One commenter said that the costs associated with the treatment cost analysis were inflated. He stated that the cost analysis was based upon NOMS data which averaged values of THM concentrations measured in over 100 finished water supplies across the United States. The commenter believed that the cost analysis should have been done in two phases: one for summer conditions and one for winter, using quenched values for all 117 cities, and measured at the point in the distribution system most distant from the source to accurately measure the THM concentration reaching the consumer.

EPA's national cost estimate has been based upon NOMS which is the most recent available data base with regard to the levels of THMs in finished drinking water supplies. Certainly a more refined and extensive survey would provide a higher degree of confidence for its estimates; however, for the purposes of assessing the national cost impact of these regulations, the NOMS data base was felt to be a reasonable representation of THM occurrence.

29. One commenter estimated that the cost to the consumers in his system could increase 50 to 75 cents per 1,000 gallons and the needed treatment modifications would also result in reducing his filter capacity up to 70 percent. Other estimated rate increases reported by several commenters reached as high as 120 percent, while it was stated by one commenter that a 5.4% increase would be necessary for his utility.

EPA's projected national capital expenditures total $85 million in 1980 dollars resulting in a overall rate increase of 2% which is a considerable reduction from EPA's original estimates. EPA's original estimates were $154 million (1976 dollars), equivalent to $210 million in 1980 dollars, and included only those impacted communities larger than 75,000 population. EPA's revised cost estimates now include those communities between 10,000 and 75,000 population, and assume that a total of 515 water systems would be required to institute some type of change in current processes. Fewer systems are expected to use the more expensive treatment technologies. Available technologies range from no-cost or very low cost changes such as improving coagulation or moving the point of chlorination (172 systems estimated), to low to moderate cost changes, such as modification of

the disinfectant (319 systems estimated), to high cost changes, such as use of an adsorbent like GAC (24 systems estimated).

EPA restructured its decision tree based upon several factors regarding the treatment technology alternatives that are available to meet the MCL and the number of systems by size that would be likely to modify or install treatment because they exceeded the MCL. It is not anticipated that the existing filter capacity, as suggested by the commenter, would be reduced by application of these technologies. These projections have been derived based to a large degree upon information received during the comment period. For example, considerably wider use of chloramines and less usage of GAC is expected to be selected to reduce THMs. Primarily, for those reasons, the cost estimates have significantly changed, and the typical costs per family (i.e. residential bill increase) are expected in the range of $1.40 per year. In those few cases (24) where GAC is necessary, costs per family have been estimated to be up to $11.20 per year, less than $1.00 per month. After review of existing rates, rates for other utilities, and the specific costs involved, EPA does not believe that such increases will have an unreasonable impact on a family budget.

30. Twenty commenters thought that the monitoring costs were excessive for the water utilities to pay and they felt that the federal government or EPA should conduct or fund the monitoring program. One questioned whether Federal funds would be available to assist in the additional financial burden of the regulations. However, another stated that no federal grants should be issued to public water systems because of their prior record of providing services and supporting themselves from their own resources.

Monitoring costs required by these regulations amount to approximately $800 per system per year. These costs are not considered to be excessive; for example, minimum cost per capita for monitoring for systems serving 10,000 people will be $0.08 per year and for systems serving one million people, $0.0008 per year. As noted above, the costs associated with this regulation generally are not significant and federal financial assistance should not be needed in the size range covered by this regulation. If it is needed, federal financial assistance programs are available for public water system improvements. It is also probable that in many cases the States may provide analytical services for their communities.

31. One commenter was concerned that compliance with the regulations by systems that will require major modifications would be difficult because of the economic and social burden; the commenters also questioned how the regulations relate to the President's urban policy. Several commenters were concerned that the burdens of increased water rates would be difficult for those least able to afford it; that is, low income and high unemployment groups, minorities, and retirees. One felt that the required rate increases for both normal system maintenance and to meet the regulations might not be supported by the customers, concluding that this could eventually result in deterioration of the water supply facilities because the cost of meeting the regulations would take needed capital away from maintenance type programs. One felt that the cost of the regulations would take money away from the needy and could result in poorer and less nutritious diets.

Because of the relatively low costs associated with these regulations, the impact on consumers' other needs are not considered to be significant. EPA believes that providing healthful drinking water must be a high national priority and that these regulations do not conflict with the President's urban policy.

32. A commenter said that it was not clear that GAC would effectively reduce TTHM concentrations more than movement of the chlorination point or changing disinfectants; the choice of installing GAC filtration by water treatment plant managers might produce only slight reduction in TTHM concentrations at a very high cost and therefore might not be a feasible alternative.

EPA estimates that GAC will only be used by about 25 systems to comply with the MCL because less expensive technology alternatives are available, such as changing the point of chlorination or using an alternate disinfectant. For these 25 systems, it is expected that a comprehensive evaluation of the existing treatment will be made to determine the most cost-effective technique for compliance with the MCL. These systems will most likely use a combination of the alternative treatments, such as changing the point of chlorination or maximizing coagulation/filtration efficiencies. Use of GAC for TTHM control has been found to be effective for not only reducing precursor compounds which contribute to TTHM formation, but also to some degree for removing THMs once they are formed.

33. One commenter felt that increases in State program grants would be necessary for States to implement these regulations.

These requirements are not expected to be an undue burden upon State programs. Implementation of these regulations will require State review and approval of proposed plans for treatment modifications for approximately 515 systems. Because of the relatively small number of systems within each State, the phasing-in of the two population segments, and the fact that, for the most part, minor modifications will be necessary, this is expected to be accomplished with minimal disruption to existing State programs. Further, many States already review system plans for any modifications to existing treatment. Compliance monitoring will also be required but this will only be a minor addition to the system already in use by State programs for checking compliance with the NIPDWR in effect.

34. One commenter stated that EPA underestimated the costs of implementing the regulation by underestimating the number of impacted systems. This commenter disagreed with EPA's use of a specific model for the water supply industry, assumptions regarding the number of systems that purchase water and use alternate disinfectants, and assumptions and predictions based upon NOMS for determining the level of THMs and if systems would be impacted. Instead, they said EPA should have conducted sampling at all systems and based its estimates upon those results. They further commented that EPA's estimate of 390 systems serving greater than 75,000 persons was not derived from EPA's Inventory of Systems but was based upon a policy testing model which left out numerous systems including all Federal Systems (e.g. District of Columbia) and the States of Hawaii and Alaska. They criticized EPA for not confirming the hypothetical results of the model with empirical data. Finally, they said EPA's assumptions regarding the number of systems using specific treatment systems such as GAC or no-cost modifications were arbitrary.

EPA has based its assumptions regarding the number of public water systems upon the actual inventory of water supply systems in the U.S. as ascertained in the Federal Reporting Data System (FRDS), and thus the number of systems is as accurate as possible. Certainly, surveys at every plant in the U.S. as suggested by the commenter would provide actual results rather than an estimate of TTHM levels, but NOMS is considered to be a valid representation of national exposure

levels. NOMS is the most recent and extensive data base and is adequate for estimating national cost impacts. In regard to disinfectant use, EPA based its estimates upon an EPA national survey in 1976 of drinking water plant operations. The determination of the number of systems that are expected to use specific types of treatment was discussed in the preamble and are reasonable estimates based upon the TTHM levels and available technologies. Finally, the commenter was unfamiliar with the policy testing model which the Agency uses to support economic and financial analysis. A description of this model is presented in Appendix A of the economic analysis document. It is used only to generate the aggregated costs and financial impacts, based upon inputs from treatment cost data, water supply inventory data, and water supply operating characteristics data.

35. One commenter stated that the EPA should provide a cost estimate of the stated goal of lowering the MCL at a later time to 50 ppb or 10 ppb.

Prior to lowering the MCL to any level, a full economic impact analysis would have to be conducted and available for public comment as part of an entire rulemaking proceeding. The 0.010 to 0.025 mg/l was merely stated as an indication of future technological performance potential.

36. One commenter was concerned that EPA had underestimated the financial implications of the TTHM regulations on water utilities, for example, by assuming that the rate increase required to finance the necessary revenue requirements would be easily obtained. This commenter noted that projections of future capital requirements in addition to the cost of the GAC process for various water systems had not been factored into the analysis. Another commenter stated that in order to install GAC, water utilities would need to raise capital through large rate increases. They noted that there were substantial regulatory barriers which could preclude water utilities from obtaining the necessary rate increases. Even if utilities were able to raise the capital funds, the quality of their credit and the attractiveness of their common stock would be severely reduced; this would reduce their ability to obtain external financing for normal water supply activities.

EPA believes that the estimated costs will not result in an undue burden upon water utilities and therefore, revenue requirements will be reasonably obtained in most cases. Further, EPA did not factor in capital requirements for such items as system maintenance or expansion into the analysis since these are not directly related to the regulations. Since implementation of these regulations will improve drinking water quality, utilities should be in a favorable position to obtain rate increases. Further, it is not expected that bond rating of the utilities will be significantly affected or that regulatory barriers will seriously prevent systems to obtain financing for complying with these regulations.

37. Two commenters stated that EPA was required to prepare an Environmental Impact Statement (EIS) in conjunction with these regulations. They noted that EPA had not addressed the significant primary and secondary environmental problems associated with the use of GAC treatment facilities and that EPA's assessment had not evaluated the full environmental impact potential of the regulations so as to be functionally equivalent to an EIS.

EPA is not required to prepare a formal EIS for these regulations. Section 102(2)(C) of the National Environmental Policy Act (NEPA) requires the preparation of an EIS for "major Federal actions significantly affecting the quality of the human environment." However, the courts have exempted EPA rulemaking from this requirement where the Agency's action in carrying out its statutory obligations is designed to protect the environment and amounts to the "functional equivalent" of the requirements of NEPA. Although the courts have not specifically addressed the applicability of NEPA under the SDWA, the "functional equivalent" standard is equally appropriate and clearly satisfied here. This rulemaking has involved extensive efforts by EPA, including public participation, for evaluating the primary environmental impacts related to the control of TTHMs in drinking water. The potential negative impacts included air and water pollution impacts of GAC and its attendant regeneration furnaces, waste disposal issues related to such furnaces, adverse effects on the microbiological quality of drinking water, as well as risks associated with the use of GAC. Many other environmental impacts will be positive since human exposure to harmful chemicals will clearly be reduced. Moreover, the legislative history of the SDWA indicates that proposed provisions that would have required literal compliance with NEPA for actions taken under the SDWA were rejected by Congress. The secondary impacts were found to be too remote for consideration in EPA's analysis but are also believed to be negligible.

38. Two commenters stated that EPA was required to prepare an Inflationary Impact Statement (IIS) in conjunction with these regulations.

EPA does not believe that it was required to prepare an IIS for these regulations. Under Executive Orders Nos. 11821 and 12044, only major regulatory actions which may have a significant impact on inflation require the preparation of such statements. A major or significant regulation is one which has associated annual costs of greater than $100 million, causes an increase in price of greater than five percent, or is so designated by the Agency's Administrator. For the TTHM regulation, annual costs are estimated at $19 million, and average increases in the price of water are less than one percent. The Administrator has not designated this regulation as significant. Nevertheless, EPA has conducted a full economic and financial impact analysis of these regulations which is reported in the economic analysis document.

39. Comments were received concerning the air pollution and energy impacts associated with the use of regeneration furnaces for GAC. These commenters were concerned that the regulations would promote substantial new consumption of energy through use of GAC as well as in secondary energy consumption such as in the production of the energy that will be used in GAC regeneration, or the energy usage associated with the manufacture and transportation of GAC. One commenter stated that the Agency did not address the cost and environmental impact of such furnaces. One commenter was concerned about the availability and costs of energy for on-site regeneration of GAC as well as increased energy consumption.

EPA issued a supplemental notice of proposed rulemaking on July 6, 1979 (43 FR 29135 at 29147) which addressed precisely these concerns. EPA has concluded that the air pollution and energy impact of these regulations will be negligible. Air pollution associated with GAC furnaces will be minimized by the use of scrubbers whose cost have been included in EPA's estimated cost of compliance for those systems that will be required to use GAC for meeting the TTHM MCL. Since fewer systems are expected to have to install GAC than EPA originally proposed, these impacts have further been reduced. Secondary energy impacts, such as transportation costs, are too tangential to be estimated with any degree of accuracy, but are also considered to be insignificant. Energy consumption will increase consumption by an estimated 508×10^9

BTU's per year or 0.0007 percent of present U.S. energy consumption. These figures do include a number of secondary energy impacts. Commenters are referred to the preamble and EPA's economic impact analysis accompanying these regulations for further details on these issues.

40. One commenter noted that EPA was required to analyze the costs of its actions in terms of the benefits hoped to be obtained and had failed to do so.

EPA has conducted a thorough analysis of the costs of this regulation and has examined in a qualitative source the perceived benefits from reducing levels of human exposure to THMs. It has been determined that the costs of this regulation are reasonable and therefore risks associated with exposure to THMs should be reduced accordingly. However, EPA is not required under the SDWA to perform a quantitative cost/benefit analysis nor to base regulatory decisions solely on the basis of such an analysis. Rather EPA is directed to establish an MCL which requires contaminants which may have any adverse effect on human health, including carcinogens, to be reduced to the extent feasible and that is the basis of EPA's establishment of the TTHM MCL at 0.10 mg/l TTHMs. Further reduction was not considered to be feasible at this time because of the potential trade-off of compromising the bacteriological quality of the drinking water due to less effective disinfection practices. Commenters are also referred to the discussion that follows below.

41. Information on the relative benefits related to the costs of the TTHM regulation was provided in an NAS Report, "Non-Fluorinated Halomethanes in the Environment" (1978). Dr. Andelman, using GAC and aeration as the tool to demonstrate a methodology of evaluating cost and benefits, concluded that in the absence of any other perspective, it was not cost-beneficial to use GAC or aeration simply to reduce chloroform concentrations in drinking water.

The report stated:

> From the viewpoint of economics, the central policy issue in controlling human exposure to any toxic substance is whether the benefits of reducing deaths, suffering, illness, and other losses outweigh the costs of controls. This involves identification of population exposure levels and a determination of when the costs of additional controls exceed the benefits of a further reduction in exposures.

The report applied four concepts and principles including: (1) The discounted value of an individual's production, (2) extrapolations from risk premiums, (3) costs of illness and human suffering, and (4) the Pareto Improvement principle, and applied the empirical estimates of values of reducing the probability of death to develop his benefit-cost evaluation. The report concluded that:

> Depending on the methodology that is used to compute costs, from these examples, the most reasonableness estimates of the per capita value associated with reducing the probability of death by 100 percent range from $100,000 to $1,000,000.

EPA has reviewed this NAS report and believes that the cost side of a benefit-cost equation that is used in the control of toxic substances should have been calculated for each specific control technique because the costs per person benefited may vary greatly among the available control options. The NAS report selected only aeration and GAC adsorption process for the control of THM concentration in drinking water and failed to consider other less expensive treatment methods which will, in fact, be used by most systems to comply with the TTHM MCL.

The report assumed that the most significant effect of human exposure to chloroform in drinking water was cancer and that all of these cancers result in death; effects other than cancer mortality were presumed to be negligible. Therefore, the report said the benefits of reducing human exposure to chloroform in drinking water could be estimated by multiplying data on lifetime risk of cancer by the economic value of reducing the risk of death from cancer in a population. The benefits also could be calculated by multiplying the daily per capita uptakes of chloroform by the risk of a cancer death over an average lifetime from a given daily dose of the carcinogen by the economic value of reducing the risk of a cancer death.

Based upon the above principles and other assumptions, the report found that:

> Very high concentrations of chloroform in drinking water are associated with enough risk of cancer to justify the costs, on economic grounds alone, of treatment processes for removal of this compound. The potential magnitude of the problem is even greater if allowance is made for the upper limit of risk. Furthermore, justification for treatment rises with the value imputed to avoiding a death. However, the current cost of treatment to remove chloroform from drinking water is sufficiently high that the economic justification for removing chloroform from drinking water in the United States, assuming the most probable risk, exists only in those cases where maximum initial concentrations of chloroform are found in drinking water, there is maximum fluid intake, and the risk of death is valued at $1,000,000 or more. Using a more typical and more statistically justifiable value of reducing the risk of death, i e , $300,000, the high cost of removing chloroform alone cannot be justified on economic grounds for the most probable risk conditions, even when there are maximum concentrations and intake.

EPA believes the analysis in this NAS report has everal serious shortcomings which obviates its conclusions. As is stated in the report itself, the analysis was designed primarily "to demonstrate a methodology," rather than to draw strong conclusions about the particular example used. In EPA's view, the following assumptions made in the analysis bias it against regulation of THM: (1) The risk extrapolation used for chloroform is lower by a factor of 8.5 from that derived by the NAS in *Drinking Water and Health* (the existence of so large a discrepancy in an estimate by the same organization using the same model illustrates the difficulties in making a fine-grained comparison of risks and costs), (2) no account is taken of the benefits of GAC other than removal of THM, such as removal of other disinfection by-products, synthetic organic chemicals present in the raw water, and substances with objectionable taste and odor, and (3) it does not take into account much cheaper technologies for THM control. In spite of these biasing assumptions, the analysis still concludes that, for an assumed value per cancer case avoided of $500,000, a community would be justified in installing GAC for TTHM control if its TTHM level exceeded 164 ug/l, a conclusion which is not at all inconsistent with an MCL of 100 ug/l.

EPA agrees that the costs and benefits of alternative regulations should be examined in deciding whether and how stringently to regulate, where the statutory framework does not prohibit such examination. While no such prohibitions are contained in the SDWA, EPA believes that the uncertainties in quantifying the health benefits of regulatory actions, particularly given the great scientific uncertainties about the effects of low levels of carcinogens, make formal cost-benefit analysis of limited usefulness in regulatory decision making.

The quantification of risk is sorely limited by the lack of demonstrable accuracy and precision of any statistical model, the inability to identify more than a portion of the substances that would be generated by chlorination in water, the inability to predict the toxic potency of those chemicals individually let alone as a variable complex mixture, the inability to quantify the contributions of these chemicals to and their interactions with the mass of toxic chemicals that are part of human body burdens, and the inability to identify

particularly susceptible high risk segments of the population.

The costs that were used in the NAS nalysis dwelled on GAC and aeration which are among the most expensive options and which only a small number of water systems would need to use. Prevention or reduction of THM formation potential prior to introduction of chlorine is much less costly than removal after formation. The NAS estimate of benefits associated with the THM regulation considered removal only of chloroform and none of the other by-products, and also did not consider any other water quality improvements. The study's cost of not controlling THMs in public water systems did not include the considerable offset of increased cost to consumers and society by increased reliance on bottled water or home devices that ostensibly reduce organic chemicals at the tap. Morbidity costs, lost wages and health treatment costs were also not considered. Thus, risks and benefits can easily be underestimated, and costs overestimated. Considering costs, risks and benefits is of course an essential part of any regulatory process, but the judgment of an acceptable societal cost for a human life is a matter of policy hat requires many more complex and ubtle factors that are not within the urrent state-of-the-art for these types of uantitative analyses. Additional discussion of cost-benefit analyses is provided below in the reponse to the comments submitted by the Council on Wage and Price Stability.

42. The Council on Wage and Price Stability (CWPS) said that the EPA studies contain:

(a) No analysis of the benefits of alternative performance standards or of alternative population-size cut-offs, and

(b) No analysis of either the costs or the benefits of alternative design standards.

Consequently, CWPS believed that the EPA analyses shed no light on the reasonableness (i.e., the cost-effectiveness) of these decisions. They said that EPA provided no information about the consistency of these regulatory decisions with each other or with other EPA regulations. CWPC believed that because the resources available for health-related programs are limited, it is important that those resources be allocated in a way that maximizes the benefits (in terms of lives saved or cases of illness or injury avoided). This in turn would require that he incremental cost per cancer case voided be at least approximately uated for different regulations or ifferent adopted standards. CWPS felt that it was incumbent upon EPA to support its proposed regulations with careful risk-assessment and cost-benefit analyses, employing the best estimates available regarding uncertain variables, parameters, and relationships. CWPS made some preliminary calculations and suggested that more lives could be saved with no increase in costs by tightening up on the performance standard for THM (i.e., lowering the allowable concentration below 100 ug/l and concomitantly relaxing the population cut-off (higher than 75,000)). CWPS said that:

(a) The incremental cost of lowering the population cut-off from 100,000 to 75,000 (given a 100 ug/l standard) is $12.2 million per additional cancer case avoided.

(b) The incremental cost of strengthening the performance standard from 100 ug/l to 50 ug/l (given a population cut-off of 75,000) is $6.3 million per additional cancer case avoided.

(c) Thus, the cost of avoiding cancer cases by applying the MCL to communities with populations of 75,000 and above, which EPA had done, is double the cost of avoiding cancer cases by strengthening the standards to 50 ug/l, which EPA did not propose.

(d) CWPS also said, "These calculations do not necessarily mean that the performance standard should be tightened to 50 ug/l, but they do suggest that the (two) proposed regulations are internally inconsistent."

The CWPS comments raise two separate types of issues with respect to the THM regulation. The first concerns the use of cost-benefit analysis to determine whether a regulation is justified and what its overall level of stringency should be. The second concerns whether, *given* that a regulation limiting THM levels is to be implemented, the proposal would be the most cost-effective way of using a given level of social resources to reduce the population's exposure to THMs.

On the first issue, CWPS did not draw any conclusions as to whether the regulation was justified, but recommended that cost-benefit analyses be an integral part of the Agency's decision process.

EPA has reviewed the subject of using cost-benefit analysis in regulatory decision-making under the SDWA and reached the following conclusions. First, benefit-cost analysis is most useful to decision-makers when benefits can be specified with the same degree of certainty as the costs. However, when dealing with long-term health risks, such as cancer-causing contaminants like THMs, while it is possible to establish the existence of a risk, it is beyond the state-of-the-art of current scientific knowledge to establish the exact degree of risk. Crude indications of risk can be made, and these can be used to develop a range of health benefits associated with a regulation, however, the range is so broad that its use in benefit cost analysis overwhelms these elegant and sensitive analytical procedures. In addition, there is little agreement on the dollar value which should be ascribed to the avoidance of a case of cancer. Past estimates have ranged from $10,000 to $158 million. Therefore, due to these two fatal deficiencies, it is not possible to place excessive significance on cost-benefit analysis for the long-term health risks related to this regulation.

Despite these inherent difficulties, EPA conducted an analysis of regulation alternatives. Constraints to decisionmaking involving technical and administrative issues tended to limit the range of alternatives. Within this framework, however, it was possible to establish that for the regulation the marginal cost of a case of cancer avoided is approximately $200,000 (counting only the benefits of THM reduction). This is similar to that suggested in the NAS report cited by Dr. Andelman. Further discussion is included in the Statement of Basis and Purpose.

On the second issue, EPA agrees that any regulation should make the most efficient possible use of the social resources devoted to compliance, to the extent that it is possible to predict. CWPS presented an analysis which purported to show that, for the same total cost, a greater reduction in THM exposure might be obtained by reducing the MCL and increasing the population cut-off figure. However, the assumption had been made that systems exceeding the MCL would reduce their THM levels precisely to the MCL; in fact, many of the control technologies would actually reduce THM levels to much lower levels in practice. When account is taken of this fact, the analysis shows that EPA's proposed regulation is more cost-effective than the CWPS' suggested alternative. After staff-level discussion, CWPS recognized this and other technical deficiencies in its analysis in a letter to EPA dated January 31, 1979.

43. Sixty-nine comments were received on the proposed concept of averaging concentrations of TTHMs for compliance. A majority of the commenters approved of both the annual averaging of TTHM values from quarterly samples, and the averaging of TTHM values of representative samples within the distribution system. However, fourteen commenters thought that

averaging the quarterly results would mask fluctuations in TTHM levels as affected by seasonal and other site-specific factors. One said that quarterly averaging would be justified if EPA were concerned about the chronic but not acute effects of THMs. One said that flexibility should be retained in the regulation for later reconsideration of this averaging concept. Two commenters said that compliance should be determined by averaging all of the results of samples taken in the preceding 12 months. One suggested that a geometric mean should be used in compiling and averaging the sampling results. One felt that there was not enough information to determine whether the concept of averaging was reasonable.

On the question of averaging results of samples in the distribution system, several commenters felt that averaging values could mask high TTHM concentrations and fail to protect those individuals receiving maximum doses. Because flow patterns in the distribution system are likely to be relatively constant, these commenters believed that some residents could be unduly exposed to consistently high levels of TTHMs over a long period of time. One commenter opposed averaging the high values of TTHM analyses from samples taken at the extremes of a distribution system, with the lower results from other areas of the distribution system because it would result in uneven population exposure. Three others suggested that all samples should be taken at the extremes of the distribution system instead of averaging all sample results. One suggested that all samples should be incubated to obtain terminal TTHM and hence uniform results. One commenter said that all samples should be taken from the same point every time to avoid misrepresentation. One commenter thought that selection of sampling locations should be based upon results of a sanitary survey for each system.

EPA's proposal to determine compliance with the TTHM MCL based upon an annual average of the sampling results per quarter has been retained in the final regulations. EPA recognizes that TTHM levels may fluctuate depending upon seasonal and other site-specific factors. However, the MCL for TTHMs has been established primarily to protect the public from the adverse effects attributable to chronic exposure to these contaminants, rather than from any acute effects. EPA nevertheless retains the flexibility to amend these regulations should new information indicate that annual averaging of quarterly results is not adequately protective. On the other hand, EPA believes that it would not be reasonable to determine compliance by an annual average of all samples taken since this could clearly allow systems to mask fluctuations in TTHM levels over the year. In regards to use of a geometric mean as the basis of the MCL, the arithmetic mean is considered to be more appropriate because it is a more accurate representation of typical human exposure.

With regard to those commenters who expressed concern about EPA's proposed sampling program, it is noted that it would have required systems to average a minimum of five samples per quarter, no more than 20% of the samples to be taken at the entry point to the distribution system, no less than 20% at the extremes of the distribution system and the remaining 60% at representative points in the system relative to population density. In response, these final regulations have reduced to four the minimum number of samples to be taken per quarter, but no longer allow any samples to be taken at the entry point to the distribution system, where TTHM levels would have likely been lowest, and where few consumers would have actually been exposed to such levels. EPA believes that this sampling program will better reflect the average TTHM levels in the drinking water served to most consumers.

However, EPA rejected the suggestions to require all samples to be taken either at the extremes of the distribution system, or at the same point in the distribution system each time. Such sampling schemes would not fairly represent the water system as a whole. However, EPA is concerned that very high levels of TTHMs at the extreme ends of a distribution system be reduced. EPA believes that by requiring extreme sampling results to form a larger percentage of the quarterly average (25% as opposed to the proposed 20%), any great differences in TTHM concentrations in such locations may be detected and corrected.

In response to the remaining comments on EPA's proposed sampling program, EPA has not required all samples to be incubated to obtain terminal results because this would probably overestimate actual concentrations at the taps of most consumers. EPA agrees with the comment that sampling locations must be selected by the system on a case-by-case basis, preferably after a sanitary survey, depending upon the particular configuration of its distribution system. Systems are encouraged to work with the States and EPA in the selection of truly representative sampling points. EPA has required that the number of samples taken be commensurate with the number of treatment plants used by each system to allow sampling to detect differences in TTHM levels within each system attributable to different source waters and different treatment methods. Once problems are detected, systems should reduce extreme differences of TTHM levels within their distribution system.

44. Twenty-three commenters supported EPA's proposal to require use of the Standard Plate Count (SPC) as a more sensitive indicator (than the coliform test) of microbiological quality during treatment modifications. Thirty-seven commenters felt that the SPC was of questionable value or unreliable, and that the SPC requirement would impose an unnecessary administrative burden on water utilities. Five commenters suggested that the SPC should only be required for those systems whose water sources receive municipal point source discharges, and should not be required for all treatment modifications. Four commenters also felt that the SPC should only be used to confirm a questionable microbiological count and that the decision to use the SPC should be left to the discretion of the State regulatory agency.

In response to these comments, EPA has decided to delete from these regulations the SPC as a mandatory requirement for all systems that make treatment modifications to comply with the TTHM MCL. However, EPA still believes that compliance with the TTHM MCL should not be achieved at the expense of the microbiological integrity of the water and that the SPC can be a reliable and useful tool as an overall indicator of water quality. Therefore, in order to insure that disinfection is not compromised, while affording maximum flexibility to the States to address case-by-case situations, these final regulations have included a requirement whereby systems must seek and obtain State approval of any planned significant modifications to their treatment process made to comply with the TTHM MCL that could affect biological quality. The States (or EPA in non-primacy States) must therefore exercise careful supervision over system treatment changes by prescribing specific measures (which would include the SPC in appropriate cases) to insure the continued microbiological quality of the drinking water. The usefulness of the SPC and other biological tests are

discussed in greater detail in the preamble to these regulations and will be discussed in EPA's guidance to the States concerning approval of system treatment modification plans.

45. Ten comments were received on EPA's proposed restriction on the use of chlorine dioxide as an alternative disinfectant to free chlorine. Nine opposed the restriction of using chlorine dioxide at a maximum dose of 1 mg/l but provided no supporting data. One felt that EPA should encourage the testing and use of alternative disinfectants while others felt that the limit of 1 mg/l for chlorine dioxide was arbitrarily set and that up to 2 to 3 mg/l chlorine dioxide should be allowed. One commenter reported that chlorine dioxide was effective in reducing the TTHM concentration in his system from 284 mg/l to 16 mg/l.

In response to these comments, EPA has deleted from the final regulations its proposed restriction on the amount of added chlorine dioxide. EPA is nevertheless concerned about the uncertain state of knowledge concerning the potential for adverse effects associated with chlorite, chlorate and chlorite ion, which are produced from oxidation/reduction reactions of chlorine dioxide in water. EPA will be considering proposing limitations on the residual oxidants (ClO_2, ClO^-_2, and ClO^-_3) in the finished drinking water rather than on the amount of chlorine dioxide added. In the meantime, additional research on the health effects of alternative disinfectants will continue. MCLs may be developed for inclusion in the Revised Regulations after further studies have been fully evaluated.

By requiring all systems significantly modifying their treatment process to comply with the TTHM MCL to obtain State approval of their modification plan, EPA expects that where restrictions on chlorine dioxide are necessary, the States will impose such restrictions as appropriate in accordance with EPA guidance including monitoring for residual oxidants and maintaining their concentration at a low level. Where chlorine dioxide is completely reduced to chloride, no restrictions would be necessary since by-products are believed to be of no toxicological significance. Case-by-case judgments can also be made to impose restrictions when the presence of reducing agents in the raw water of a particular system would result in excess formation of chlorite and chlorate. Additional discussion on the use of chlorine dioxide as an alternative disinfectant is contained in the preamble to these regulations and will be contained in additional EPA guidance to the States for approval of system treatment modification plans.

46. Fifty-five commenters opposed EPA's proposed limitation on the use of chloramines as a primary disinfectant. They argued that chloramines would solve some of the problems of using chlorine for drinking water treatment because chloramines do not react with precursors to produce TTHMs, and chloramines have been in use in many water systems for many years without any problems. Eleven commenters agreed that chloramines should be restricted from use as a primary disinfectant. One of these commenters reported that preliminary data had indicated that chloramines may not be effective in neutralizing viruses and amoebic or Giardia cysts. One commenter suggested that chloramines may be used after the primary disinfection step for the purpose of maintaining an active disinfectant residual. The NDWAC felt that the proposed limitation was unduly restrictive.

EPA found that most of the commenters opposed to the imposition of restrictions on the use of chloramines failed to recognize that EPA's proposed restriction was limited to prohibiting its use as a primary disinfectant. EPA does not disagree with those commenters who endorsed the use of chloramines as an effective secondary disinfectant (to maintain an active combined chlorine residual). Nevertheless, in response to these comments, EPA has decided to delete the chloramine restriction from the final regulations, allowing appropriate restrictions to be imposed in necessary situations by the States in approving system treatment modification plans. Use of chloramines instead of free chlorine has been shown to be a simple and readily available means for reducing the formation of TTHMs in many examples. However, they are also known to be weak disinfectants for certain bacteria, viruses and protozoa, compared to free chlorine as HOCl, ozone and chlorine dioxide. Therefore, where such contamination is suspected, appropriate restrictions should be imposed. Additional information on the use of chloramines as an alternative disinfectant is contained in the preamble to these regulations and in additional EPA guidance to the States on approval of system treatment modification plans.

47. Eleven comments were received opposing the concept of setting an MCL to control TTHMs in drinking water. Two commenters said that EPA lacked legal authority to regulate the TTHMs under the Amendments to the National Interim Primary Drinking Water Regulations (NIPDWR). One commenter noted that the feasibility of control measures under the NIPDWR must be adjudged to have been available as of December 1974, when the SDWA was enacted. Three commenters said the NAS report, "Drinking Water and Health" fell far short of providing the needed scientific definition and did not recommend EPA to set MCLs for TTHMs. One commenter said that the EPA should not yield to the pressure from some public interest groups to set a MCL for TTHMs before the health risks have been established. Four commenters believed that the main reason EPA proposed an MCL for TTHMs was because EPA was anti-chlorination and was trying to abolish chlorination practice in water treatment. One of the four suggested that instead of an MCL, EPA should tighten chlorine specifications so that no contamination of the water will result during chlorination practice. Three others recommended that the regulations provide guidance on the proper use of chlorine as a disinfectant, either free or combined, for case-by-case applications.

EPA's response to those comments addressing the Agency's authority to establish these regulations as Interim Primary Drinking Water Regulations is contained in the preamble, and commenters are referred thereto. EPA agrees that the feasibility of control measures under the NIPDWR must be based on technology generally available as of 1974 and has found that these regulations satisfy the statutory test.

With respect to those commenters that cited the NAS Report "Drinking Water and Health" to support their position that regulation of TTHMs is premature, EPA disagrees with their interpretation that the NAS only recommended further research. In fact, the NAS concluded that: "strict criteria be applied when limits for chloroform in drinking water are established to protect the public health." Moreover, Dr. Riley Housewright of the NAS Safe Drinking Water Committee, stated that: "chloroform and other THMs present a health hazard and that steps should be taken to prevent their formation or to remove them from drinking water." As noted in the preamble and EPA's responses to other comments in this Appendix, EPA believes that sufficient information is known about the potential for adverse health effects from the presence of THMs in drinking water

to warrant regulation at this time. Although further research will continue to be forthcoming, EPA need not wait for definitive proof of harm before it takes regulatory action under the SDWA.

With respect to those commenters who charged that EPA's establishment of a TTHM MCL evidenced EPA's intent to abolish chlorination as a drinking water treatment practice, EPA disavows such an intent. However, EPA does believe that improper or careless use of chlorine, as well as any other disinfectant, can result in the unnecessary formation of potentially harmful by-product chemicals in the finished drinking water. EPA acknowledges that chlorine is currently the most widely used, highly effective drinking water disinfectant and expects that use to continue. However, control of TTHMs should lead to a more judicious use of chlorine and will serve to minimize human health risks from exposure to other disinfection by-products. EPA also agrees that better quality control in the manufacture of chlorine for drinking water treatment is necessary to avoid harmful contaminants contained therein and will address such concerns in conjunction with its overall review of water treatment additives. EPA's guidance to the States for approval of system treatment modification plans will contain additional information on proper chlorine use.

48. A total of 306 comments were received expressing a concern for the basis of health effects data that support the proposed TTHM regulations. The majority of the commenters felt that the proposed MCL was not based upon incontrovertible health effects information and urged that additional health effects research and epidemiological studies should be conducted. Only a few commenters said the supporting health effects data for the proposed THM regulations were adequate and that the regulatory action was justified now.

Specifically, 292 comments said that the available health effects data, both epidemiological studies and laboratory animal tests, were not conclusive and were disputed by many scientists. These commenters, therefore, believed that the setting of an MCL for TTHMs was not warranted at this time. They suggested that more research should be conducted specifically on the toxicological assessment procedures and the health effects of long term exposure to low dosage of THMs.

EPA has reviewed these comments in light of all available health effects information and has concluded that long term low level exposure to TTHMs may be harmful to human health. EPA's conclusions are supported by comments and statements of policy by representatives of the National Cancer Institute, National Academy of Sciences, National Drinking Water Advisory Council, National Institute of Environmental Health Sciences, Food and Drug Administration, Occupational Safety and Health Administration, and the Consumer Product Safety Commission. These commenters emphatically stated that EPA should not wait for additional evidence to proceed with regulatory action to control chloroform and trihalomethanes in drinking water which was warranted now. These comments are summarized in Appendix B.

The following discussion summarizes the specific concerns expressed by commenters regarding the health basis of the regulations and presents the Agency's responses.

49. Comments were received that argued that chloroform poses no potential cancer risk and there are no available data that support the premise of a causal relationship between the concentrations of THMs normally found in drinking water and cancer in humans. They noted that the epidemiological studies that have been conducted concerning drinking water and a possible connection with cancer risk in humans were inconclusive.

EPA reviewed the available 18 epidemiological studies concerning the relationship between cancer morbidity/mortality and constituent concentration in drinking water supplies. In summary, many but not all of the preliminary studies have found positive correlations between some drinking water quality factors and some cancer mortality and morbidity statistics such that the general hypothesis is supported. Further evaluations are necessary due to the confounding factors inherent in epidemiological studies of this nature. Therefore, EPA has relied primarily on the results of animals studies in concluding that TTHMs in drinking water pose a risk to humans. Thus, EPA does not disagree with the comment that data do not exist to demonstrate a causal relationship between the concentrations of synthetic organic chemicals including THMs in drinking water and cancer in humans. However, the positive correlation of cancer morbidity/mortality and contaminants in drinking water are suggestive and are not inconsistent with the carcinogenic potential of chloroform as demonstrated by well conducted animal experiments at high doses.

50. Some commenters opposed EPA's reliance on animal studies for its finding that TTHMs in drinking water pose a health risk on the grounds that extrapolation of results in animal cancer studies to humans is fraught with problems and uncertainties.

EPA recognizes the problems of extrapolating animal data to man. The state-of-the-art in toxicology as illustrated by the NAS in the report "Drinking Water and Health" is that the effects in animals, properly qualified, are applicable to man. Chloroform has been shown to be carcinogenic in experimental animals; its metabolic pattern in animals is similar to that in humans; EPA therefore believes that the carcinogenic effect of chloroform as observed in animals do indicate risks from human exposure to TTHMs in drinking water.

51. Some commenters argued that the study (by National Cancer Institute (NCI)) cited by EPA to support the carcinogenicity of chloroform was "a preliminary screening test and not a definitive study." They said that the study was not intended to be used to extrapolate health effects of chloroform to drinking water levels and that the NCI study was inadequately controlled and did not follow proper scientific protocols. Since a new EPA/NCI study is underway it was recommended that the implementation of any regulations be delayed until this study was completed. They claimed that the NCI study was not intended to be used to extrapolate the adverse health effects of the tested animals to the potential human health risk posed by the low levels of chemicals that are found in drinking water, since many researchers believe that the high morbidity rates in the animal experiments suggested acute toxicity rather than chronic toxicity.

Based on the NAS review and the NCI report, EPA has concluded that the NCI chloroform-carcinogen bioassay with all its short-comings is a valid test. It has been accepted by the other federal agencies for regulatory purposes. The morbidity noted took months or years to develop and would not be an acute effect by definition which would occur in 3–7 days. In addition, the studies performed as early as 1945 by Eschenbrenner and Miller pointed out the carcinogenic potential of chloroform and the metabolic similarity of chloroform in humans and animals. The NCI study on the carcinogenic potential of chloroform has been used by the NAS as well as by the EPA's Cancer Assessment Group (CAG) for risk estimation. Additional refining studies are continuing, but sufficient evidence

exists to indicate potential human risk
ıd, therefore, to reduce human
cposure.

52. Several commenters stated that Dr. Roe's studies with chloroform on dogs, rats and four strains of mice at low dose levels did not produce tumors in animals. Dr. Roe recommended a level of 300 ppb THM in drinking water based upon his results. It was claimed that Roe's studies showed a no observed effect at 595,000 (drinking water equivalent) ppb of chloroform in drinking water. Therefore, he argued that 300 ppb would provide margin of safety of 2,000. It was argued that EPA had used 500 as a margin of safety in other regulations. Based upon his chosen statistical extrapolation model, he found that a THM MCL of no lower than 0.30 mg/l (300 ppb) would provide a more than adequate margin of safety. however, it was also stated that this level is still too low to be justified on a cost-benefit basis if GAC were required.

EPA has concluded that Dr. Roe's studies with chloroform on dogs, rats and four strains of mice at low dose levels further strengthens the hypothesis of chloroform carcinogenicity. In one study, the mice fed 17 mg/kg/day chloroform showed no incidence of
ınal carcinoma, but an excess of
ımors of the renal cortex were
ɔserved in the male ICI—Swiss mice, at a dose level of 60 mg/kg/day. The negative results observed in the dog experiment may be attributed to the fact that either the animals were not exposed for a suitable length of time (i.e., duration of life span) or that an insufficient number of animals were tested. The negative results of the rat study may be attributed to the lack of strain sensitivity.

Using a no-observed-effect-level for chloroform of 17 mg/kg/day, Dr. Roe recommended 300 ppb chloroform in drinking water as an acceptable level. According to his calculation this would provide a margin of safety of 2000 for a standard person drinking two liters of water per day. The NAS Safe Drinking Water Committee and many other scientists now believe that the methods at present do not exist to establish a threshold for long-term effects of carcinogens; thus, the safety factor of 2000 referred to in Roe's recommendation of 300 ppb THM does not apply to carcinogens since no exposure can be considered to be absolutely "safe". EPA is directed by the
DWA to reduce human exposure to
armful contaminants in drinking water
) the extent feasible. EPA's THM MCL
ıf 0.10 mg/l can be feasibly achieved. The comment regarding the costs vs. the benefits of the use of GAC is discussed elsewhere in this Appendix.

54. Some commenters said that EPA's proposed MCL of 100 ppb was needlessly low and will require costly additions or changes to water treatment facilities without achieving any corresponding benefit in water quality.

EPA has found that exposure to TTHMs should be minimized. The level of the MCL at 0.10 mg/l TTHMs was determined to be a feasible level for achievement under the interim regulations. Systems are encouraged to reduce the level of TTHMs below the MCL if technically feasible. EPA expects that compliance with the MCL will benefit drinking water consumers in reduced exposure to THMs as well as reduced exposure to other disinfection by-products which may have adverse health effects. For some systems the aesthetic quality of the water will also improve because taste and odor producing compounds will be reduced along with reductions in TTHM levels. As discussed in the preamble and in EPA's Economic Analysis accompanying this final regulation, costs are not considered to be significant in that most required changes will be relatively minor.

54. It was stated by several commenters that there are a lack of health effects data on THMs other than chloroform and therefore, if an MCL is set, it should only apply to chloroform.

EPA has found that the THMs other than chloroform (bromoform, dibromochloromethane, dichlorobromomethane) are structurally similar to chloroform, and possibly undergo similar metabolic pathways and exert similar bioeffects. Like chloroform, bromoform exposure leads to fatty degeneration and centrilobular necrosis of the liver. Bromoform, dibromochloromethane and dichlorobromomethane have been reported to be mutagenic in Ame's bacterial test system. This test provides information indicative of the potential of genetic damage in biological systems. Thus, because of the chemical similarities in chemical structure and biological activity, EPA's concern regarding potential toxic effects of these chemicals and setting the MCL for TTHMs is reasonable.

55. Several commenters stated that there was no hard evidence that low level exposure to TTHMs produces cancer.

Based on current scientific knowledge, EPA must extrapolate from the results of animal tests using higher dosages to determine potential human health risks from exposure to low levels of particular contaminants. With chemicals such as chloroform that have been shown to be carcinogenic in animals, no level of exposure can be presumed safe. Therefore, EPA has concluded that TTHMs in drinking water must be reduced to the extent feasible as required by the SDWA.

Nevertheless, recent studies using low levels of 2-amino N-acetyl-fluorene (2-AAF) in mice suggest that low level exposure of animals to this compound produces liver tumors when applied. These adequately controlled studies (23,000 animals) showed a no threshold effect (liver cancer) was observed for AAF at the 1% level. In order to be able to measure below the 1% effect somewhere in the order of 100,000 animals would be required.

56. Some commenters claimed that other animal experiments have suggested the existence of definite threshold limits for toxic and carcinogenic effects.

EPA's position is that available data suggest a non-threshold response for carcinogenesis. As an example, the recent Acetyl Amino Fluorene experiments were consistent with a no threshold mechanism for liver tumor induction. This position is supported by the comments of Drs. Upton, Kennedy, Bingham and King from the National Cancer Institute (NCI), Food and Drug Administration (FDA), Occupational Safety and Health Administration (OSHA) and Consumer Products Safety Commission (CPSC), respectively, as noted in the preamble and presented in Appendix B. EPA's position is discussed in both the preamble and the Statement of Basis and Purpose. Also, the National Academy of Sciences addressed this issue in "Drinking Water and Health" (NAS, 1977) as follows:

> Carcinogenic effects may well not have threshold dose-effect relationships. If an effect can be caused by a single hit, a single molecule, or a single unit of exposure, then the effect in question cannot have a threshold in the dose-response relationship, no matter how unlikely it is that the single hit or event will produce the effect. Mutations in prokaryotic and eukaryotic cells can be caused by a single cluster of ion pairs produced by a beam of ionizing radiation. We would expect that mutations can be caused by a single molecule or perhaps group of molecules in proximity of DNA. The necessary conclusion from this result is that the dose-response relationship for radiation and chemical mutagenesis cannot have a threshold and must be linear, at least at low doses.
>
> We therefore conclude that, if there is evidence that a particular carcinogen acts by directly causing a mutation in the DNA, it is likely that the dose response curve for carcinogenicity will not show a threshold and will be linear with dose at low doses (pp. 37–38).

Methods Do Not Now Exist to Establish a Threshold for Long-Term Effects of Toxic Agents

With respect to carcinogenesis, it seems plausible at first thought, and it has often been argued, that a threshold must exist below which even the most toxic substance would be harmless. Unfortunately, a threshold cannot be established experimentally that is applicable to a total population. A time-honored practice of classical toxicology is the establishment of maximum tolerated (no-effect) doses in humans based on finding a no-observed-adverse-effect dose in chronic experiments in animals, and to divide this dose by a "safety factor" of, say, 100, to designate a "safe" dose in humans. There is no scientific basis for such estimations of safe doses in connection with carcinogenesis. For example, even if no tumors are obtained in an assay of 100 animals, this means only that at a 95% confidence level, the true incidence of cancer in this group of animals is less than 3%. Even if we were to carry out the formidable task of using 1,000 animals for the assay and no tumors appeared we could only be 95% sure that the true incidence were less than 0.3%. Obviously, 0.3% is a very high risk for a large human population.

In fact, there are no valid reasons to assume that false-negative results of carcinogenicity tests are much less frequent than false-positive ones. To dismiss all compounds that did not induce tumors in one or two mouse and rat experiments as non-carcinogenic is wrong. Labeling as "carcinogens" all substances that gave rise to increased incidence of tumors is justified only if there is conclusive evidence of a causal relationship. The "relative risk" of compounds that are not found to induce tumors in animal experiments must also be considered. But this requires evaluation of data other than those collected in chronic toxicity studies on rodents.

Experimental procedures of bioassay in which even relatively large numbers of animals are used are likely to detect only strong carcinogens. Even when negative results are obtained in such bioassays, it is not certain that the agent tested is unequivocally safe for man. Therefore, we must accept and use possibly fallible measures of estimating hazard to man.

57. As noted by a number of commenters, the assumption of parallel response between test animals and humans does not hold for many species.

EPA believes that animal experiments that demonstrate a carcinogenic response are indicative of a potential carcinogenic response in the human population. This is supported by Drs. Upton, Kennedy, Bingham, and King from the NCI, FDA, OSHA, CPSC, and NIEHS, respectively, whose testimony is presented in the preamble and Appendix B.

58. Some commenters stated that EPA's extrapolation procedure erroneously utilized two "very consecutive" techniques to determine the MCL for THMs. They said that either technique could probably be justified, but not both.

The level of the MCL is based upon feasibility of available treatment technology and maintenance of biological safety and not on an extrapolation technique from experimental data. The need to limit human exposure is demonstrated by the potential adverse health effects from long term exposure to chloroform from animal studies.

59. Comments were received that alleged that EPA estimates of environmental exposures to chloroform appear to be erroneous and suggested that EPA make every effort to obtain correct values for contributions from air, food and water. Also, they suggested the possibility that *in vivo* formation of chloroform and other THMs in the human body might occur. The commenters felt that the available data suggest that more cost-effective avenues, such as control of chloroform in the work place, may be available for reducing THMs in the environment than by implementing the proposed TTHM MCL.

EPA's estimates of environmental exposure to chloroform were based upon the most recent available data and are considered to be adequate representations of exposure levels. The speculation of *in vivo* formation of chloroform and other THMs in the human body contradicts what is known concerning the fate of chloroform in a mammalian system although this may be occurring from ingestion of chlorine in water. In mammalian systems, chloroform is metabolized to carbon dioxide and other metabolites. The rate of metabolism will be dependent upon the species. Therefore, there is little chance of chloroform being biochemically produced endogenously in the human body.

With regard to the suggestion that there may be more cost-effective means for controlling chloroform in other aspects of the environment, EPA has found that drinking water is a significant contributor to overall human exposure to THMs. Moreover, control of THMs in drinking water is not a significant burden upon water utilities, and will result in reduced human exposure to other potentially harmful disinfection by-products as well. Thus, EPA believes that these regulations are necessary for reducing human exposure to chloroform from a significant source. OSHA and FDA have likewise taken action to reduce human exposure to chloroform under their respective statutory authorities.

60. Some commenters noted that the concentrations of THMs found in public water systems present no mutagenic, teratogenic, acute, subchronic, or chronic toxicological health risk to the public.

Based on the evidence in EPA's rulemaking record, EPA has concluded that THMs pose a carcinogenic risk at the levels found in drinking water. No safe level can be deemed to exist for human exposure to carcinogens and therefore, levels of these contaminants should be reduced to the extent feasible.

61. Some commenters alleged that EPA misconstrued the four general "principles" for risk assessment stated by the NAS in its report "Drinking Water and Health." They argued that EPA did not properly use these principles and ignored the available data. Specifically, with regard to the first NAS principle, EPA was faulted for not taking into account a number of variables in extrapolation of the animal data to humans, including differences between species response to carcinogens, weight, intake of food and water, and routes of exposure. With regard to the second NAS principle, they argued that EPA ignored animal experiment data that showed a threshold level for no-effect responses with respect to a number of suspected carcinogens, as well as experiments involving animals and humans suggesting a no-effect level for chloroform. In support of their claim that threshold levels can be established for carcinogens, they cited the existence of *in vivo* biological processes and human exposure to natural carcinogens without adverse health effects. With regard to the third NAS principle, they claimed EPA did not consider the significance of the detoxification and repair mechanisms operative in animals and humans in its health assessment of THMs. Finally, with regard to the fourth NAS principle, they claimed EPA ignored the guidelines for assessing risk for chloroform as set forth in EPA's "Interim Guideline for Carcinogen Risk Assessment." The comments also faulted EPA for using only the linear model for extrapolating the NCI animal data to humans, while ignoring the data presented by Roe, Eschenbrenner, and Miller, as well as the estimates of risk by Tardiff using the "margin of safety," "probit-log" and "two step" extrapolation models.

The EPA has carefully evaluated all available data and believes it has properly followed the four NAS principles. Each of the commenters' concerns have been thoroughly considered in determining the health

basis of the regulation. EPA has used the present state-of-the-art in toxicology in using the NCI bioassay study on the carcinogenicity of chloroform for assessing cancer risk to humans. The studies by Roe, Eschenbrenner, and Miller were not suited for risk extrapolation because either the dosages were not high enough to observe the response or the experiments were not performed for long enough time periods to observe tumorigenic response.

The question of threshold and/or no threshold for carcinogens is discussed elsewhere in this Appendix, in the preamble and in EPA's Statement of Basis and Purpose accompanying these regulations. The linear non-threshold model is a conservative risk model and consistent with the method used by the NAS. The basis of the regulation is that a human health risk exists even though precise quantification of the risk cannot be made using current toxicological procedures. Therefore, EPA's regulatory approach is to minimize human exposure to these potential carcinogens to as low a level as is feasible.

62. Some commenters said that EPA ignored the relationship between dose and time-to-tumor observation in assessing the health risk of a carcinogenic material.

EPA does recognize the potential relationship between dose and time-to-tumor, but this has not been taken into consideration in the calculation of risk because scientific methods and data are not currently available to adequately perform such a computation.

63. Dr. Timothy DeRouen, representing the Coalition for Safe Drinking Water, critiqued the epidemiological studies cited by EPA in the proposed regulations. He discussed the studies for a possible relationship between chlorinated drinking water and cancer mortality. His principal points and EPA's responses are as follows:

(1) Dr. DeRouen commented that although some consistencies exist to support the premise of a relationship between organic chemicals in drinking water and cancer risk, comparable inconsistencies exist that were not pointed out by EPA.

EPA has concluded that in epidemiological studies, inconsistencies are always present, due to one or more confounding factors. Because of this and as noted in the preamble and EPA's Statement of Basis and Purpose, EPA did not rely upon the epidemiology studies as a basis for the regulations. Rather, they have been found to support the hypothesis, as Dr. DeRouen noted, that some relationship may exist between cancer risk and chloroform in drinking water. EPA's conclusions based on animal studies are justified.

(2) Dr. DeRouen said that correlational studies are the crudest kind of epidemiology investigation and their results should be used to suggest more definite studies. However, they are not considered accurate enough for decision-making.

EPA believes that since several of the individual correlational studies when evaluated collectively suggest that chloroform in water poses a risk, the hypothesis is strengthened. Drs. Upton and Schneiderman of the NCI supported this conclusion and suggested that reducing TTHM concentrations by 100 micrograms per liter could lead to a decrease in cancer rates of up to 7.5% in men and 10% in women for bladder cancer and between 7.5% and 8.5% in large intestinal cancer for women and men, respectively, assuming the validity of one of the studies.

(3) Dr. DeRouen also commented that the epidemiological studies did not adequately adjust the data for confounding variables such as urbanization and industrialization. He noted that in a recent study where additional variables were considered, the statistical significance "dissipated" relative to GI and urinary tract cancers.

As noted previously, taking into account the multitude of interplaying factors in epidemiology studies is a complex problem. EPA has carefully evaluated the available study results, and taken collectively, they generally support the hypothesis of the risk of chloroform in drinking water. The commenter's concerns that the impact of several variables "dissipated" when re-examined may be valid but these issues do not vitiate the basis of the regulations. EPA's finding that chloroform may pose a carcinogenic risk to humans is based primarily upon animal toxicity studies.

(4) Dr. DeRouen noted that the epidemiological studies would have more credence if the health effects were uniformly distributed over all race-sex groups, but that this was usually not the case in the drinking water/organics studies.

EPA believes that it is not necessary to have a uniformly distributed effect over all race-sex groups, although when this is the case conclusions can be more strongly supported. Rarely in even well-controlled experimental studies are the effects uniformly distributed among sex groups even in in-bred strains of test animals.

(5) Dr. DeRouen stated that unexpected and unlikely statistically significant correlations were reported for some cancer sites, and significant relationships were not seen in humans for liver or kidney cancers, which were the effects seen in the animal tests.

EPA believes that site-specific cancers are not necessarily observed across species. This was supported by Drs. Upton and Kennedy of NCI and FDA, respectively.

(6) Dr. DeRouen commented that in many studies, the presence of statistically significant results would change depending upon the statistical or analytical model selected. In general, therefore, the statistical methods are usually specified in the protocol before performing the study.

EPA agrees with this comment and it is supported by Dr. Hoel from NIEHS. The epidemiological studies cited were correlational, preliminary and hypothesis generating, rather than case-control or prospective in nature. It is therefore expected that further studies could be designed based on those already conducted which could be more definitive. EPA has pointed out many of these same problems in its evaluation of the epidemiological studies in the preamble accompanying the February 9, 1978, proposal, and EPA's Statement of Basis and Purpose as did the NAS, Safe Drinking Water Committee, in its review of the studies. The primary basis for the regulations is the animal toxicology studies including the NCI bioassay results demonstrating that chloroform was an animal carcinogen under conditions of the test. EPA has concluded that the epidemiological studies conducted so far are sufficient hypothesis-generating studies, and taken as a whole are supportive of the animal data in pointing out the possible human risk. The pros and cons of the studies are discussed in more detail in the Agency's Statement of Basis and Purpose for these regulations.

64. Dr. F. J. C. Roe, representing the Coalition for Safe Drinking Water, submitted written and oral comments. He also submitted copies of his recent studies on chloroform carcinogenicity. His major points and EPA's responses are as follows:

(1) Dr. Roe stated that regulatory contexts usually do not distinguish between highly dangerous cancer-causing agents and those such as chloroform for which the evidence is equivocal.

EPA has concluded that the SDWA directs EPA to protect the public health from any contaminant which "may have any adverse effect" on human health. Nevertheless, EPA evaluated the risk of exposure to chloroform to the general population based on its toxic effects, cancer potential and exposure potential. Chloroform has been found to be an

animal carcinogen with well known acute and chronic effects. Its presence in treated finished drinking water potentially exposes over 100 million people over their lifetime. EPA believes this to pose a substantial risk.

(2) He stated that the NCI bioassay was faulty because it erroneously used corn oil as the vehicle for administering chloroform to the test animals, not enough control animals were used, and concommitant exposure to other carcinogens occurred. He urged that prior to setting an MCL, the study should be repeated in a wider dose range and under better controlled conditions.

Although additional studies taking into account the above objections may lead to slightly different responses one way or the other, EPA believes that the findings of carcinogenicity would remain unchanged in light of previously reported studies on other carcinogens and the statistically significant results obtained in the NCI chloroform bioassay. EPA is sponsoring a study that takes into account Dr. Roe's suggestions. However, it would not be prudent to delay setting an MCL for TTHMs pending refinement of the data, given the existence of credible data to date demonstrating an adverse health risk.

(3) Dr. Roe stated that the Theiss (pulmonary adenomas) study produced erroneous statistical results.

As stated in the Statement of Basis and Purpose, EPA did not rely on the Theiss study to reach its conclusions. The study was only included as background information to the published positive results.

(4) Dr. Roe said that the four principles of the NAS (1977) and the non-threshold risk concept for carcinogenesis are not scientifically sound.

As discussed previously, EPA relied upon the judgment of the National Cancer Institute who commissioned and evaluated the bioassay of chloroform in rats and mice and concluded that significant rates of chloroform-related tumors were detected in both rats and mice under conditions of the test. The National Academy of Sciences in "Drinking Water and Health" (1977) concluded that chloroform had been shown by those and other studies to be an animal carcinogen and, as such, should be considered a risk to humans. Other studies sponsored by EPA are underway further refining our knowledge of the toxicology and carcinogenicity of chloroform, which may provide more information on dose-response relationships.

Federal health regulatory agencies have carefully considered various approaches for dealing with potential human carcinogens and the possible presence or lack of thresholds for carcinogens. These agencies have concluded as a matter of policy that in the absence of evidence to the contrary it must be assumed that substances that have been shown to be animal carcinogens in properly conducted tests, must be assumed to be potential human carcinogens, and that threshold exposure levels below which there would be no risk have not been demonstrated experimentally.

Drs. Upton, Kennedy, Bingham, King and Bates/Hoel of NCI, FDA, OSHA, CPSC, and NIEHS, respectively, supported EPA and these principles enunciated by the NAS.

(5) Dr. Roe also submitted results of three additional mouse studies that were conducted on chloroform along with his written comments.

In the first of these studies, the mice of an outbred Swiss albino strain (ICI) were given daily (six days per week) oral doses of 17 mg/kg or 60 mg/kg chloroform in tooth paste base for 77–80 weeks. The animals were observed for an additional 16 weeks. Twenty-two percent of the high dose males developed adenomas or hydronephromas of the kidney. In the second study male mice of the same strain responded similarly, with 18% of the high dose having histologically the same tumors.

In the third mouse study, the response of the male mice of four strains were compared. In each of the four strains, 52 male mice were given 60 mg of chloroform per kilogram (six days per week) using the same experimental design as previously outlined. As in the previous experiments, mice of the ICI Swiss strain developed more kidney tumors than did the vehicle control mice. No excess tumors were found in the remaining three strains.

Dr. Cipriano Cueto (representing the National Cancer Institute) stated to the National Drinking Water Advisory Council (1978) that Dr. Roe's results were entirely consistent with the NCI studies. Dr. Cueto also said that the results of other studies relied upon by Roe using rat and Beagle dog study were also not surprising based on the doses administered and the previous NCI results.

(6) Dr. Roe calculated that a 70 kilogram man consuming one liter of water containing 100 ppb of chloroform would have a 7,000 fold safety factor. Dr. Roe assumed that the mouse was the most sensitive animal model and that 10 mg/kg was the "no effect level" for kidney tumor enhancement.

As discussed previously, the EPA has found that thresholds for carcinogens have not been sufficiently demonstrated and that this type of calculation therefore contradicts that policy and does not take into account many of the principles enunciated by the NAS. Thus, EPA has rejected Dr. Roe's approach as unacceptable for regulating carcinogens in drinking water.

(7) Dr. Roe also stated that it was "reasonable to assume that none (of the THMs) is more active than chloroform itself," and, therefore, a level of 300 ppb for chloroform alone would be as protective as a similar limit for all THMs as proposed by EPA. However, Dr. Roe did not present any scientific facts or principles to support his statement that other THMs are less potent than chloroform.

As discussed earlier, EPA has found that *in vitro* mutagenicity data indicate that the other THMs are more active mutagens than chloroform. EPA's regulation of total THMs has also been based upon the similar chemical structures and expected biological activity, of all THMs, the availability of analytical methods that analyze for total THMs, and the fact that all THMs are produced as a result of disinfection practice.

(8) Dr. Roe stated that animal detoxification mechanisms were overwhelmed by the administration of very high doses of chloroform in the animal studies. He based his comment on the following observations:

(1) Females of the species did not appear at risk.

(2) Ames type assays were negative.

(3) Tumor formation was dependent upon an indirect mechanism which involved both sex hormone status and a deviation from normal metabolic breakdown pathways.

In EPA's opinion, there are many experimental conditions under which one sex or the other is more sensitive to the compound under test and therefore this difference in the results is not surprising. The *in vitro* assays of the Ames type have been shown to be insensitive to certain chemical classes; simple chlorinated hydrocarbons appear to be one of these chemical classes. Dr. Roe presented direct evidence to support his third hypothesis; however, other studies have shown a relationship between chloroform toxicity and testosterone levels in animals.

(9) Dr. Roe asserted that consistent increased survival of three different species exposed to chloroform suggested a beneficial effect.

EPA has carefully reviewed the available data and EPA does not believe

the evidence is sufficient to support this contention.

64. Dr. Arthur Furst, representing the Coalition for Safe Drinking Water, submitted comments, many of which are similar to those detailed previously. His comments and EPA's responses are set forth below:

(1) Dr. Furst commented that the NCI chloroform bioassay was not definitive, that results from animal studies using high dosages (100,000 ppb) cannot be extrapolated to predict human health effects at low dosages (100 ppb), and that human risks cannot be extrapolated from animal data. These comments have been responded to elsewhere in this Appendix.

(2) He also faulted EPA's risk assessment for not following the sigmoid curve which he claimed should represent the dose-response that one would expect from biologically active compounds. EPA has found that the dose-response curve for carcinogens would not be expected to be represented by a sigmoid curve. Rather a linear non-threshold curve is believed to be appropriate in assessing a health risk from carcinogens. Carcinogenic, reversible, or non-reversible progressive chronic response are not "all-or-none" responses, nor do they lend themselves to easily definable criteria for categorizing the biological response. Therefore, carcinogenic responses do not satisfy the conditions upon which use of the sigmoid curve is based.

(3) Dr. Furst also claimed that there is a threshold for carcinogens, and that the histological type of tumors produced in the experimental animals was not related to the human tumor response.

As discussed previously, EPA's policy with respect to risk assessment for potential carcinogens is to include the conservative linear-dose response curve and not a carcinogenic response threshold level so as not to underestimate potential risks. With regard to the type of tumors in animals versus human tumor responses, EPA has concluded that the animal toxicity studies can be related to man irrespective to differences in tumor sites. This is supported by Drs. Upton, Kennedy, Bingham, King, Bates and Hoel of NCI, FDA, OSHA, CPSC, and NIEHS, respectively.

(4) Dr. Furst claimed that release of benzo(a)pyrene could be a factor to be considered when GAC treatment is used. He questioned the use of GAC, claiming that the treatment of water by GAC may be replacing THMs with more potent carcinogens such as benzo(a)pyrene.

EPA has evaluated the available studies involving extraction of GAC with distilled water and the total level of PAHs in the effluent were found to be insignificant.

(5) Dr. Furst suggested that a time to tumor experimental design be undertaken using multiple dose levels. EPA is currently proceeding with additional tests. However, regulatory action need not await the outcome to such studies.

(6) Dr. Furst stated that carcinogens in the environment can interact, thus modifying each others' responses. He stated that there is no association between organic chemicals in New Orleans drinking water and cancer rates.

EPA agrees that synergistic interactions between toxic chemicals can occur which is all the more reason to consider approaches that will reduce human exposures where feasible. The association between New Orleans drinking water and increased cancer rates has been suggested by epidemiology studies but is far from conclusive. EPA's discussion of the epidemiological studies is set forth elsewhere in this Appendix, in the preamble, and in EPA's Statement of Basis and Purpose.

(7) Dr. Furst objected to the conditions under which the NCI bioassay was carried out. He felt that a single massive dose by oral gavage does not compare with a minute fraction of the dose ingested throughout the day. The doses used in this bioassay overwhelmed the ability of the liver to detoxify the THMs. EPA has concluded that high dose animal studies are necessary and valid methods of determining risks from human exposure at lower doses.

These questions are more fully addressed elsewhere in this Appendix and in the Statement of Basis and Purpose.

65. Comments submitted by Dr. Frank L. Lyman on behalf of the Coalition for Safe Drinking Water and EPA's responses are as follows:

(1) Dr. Lyman commented that the 100 ppb level for TTHMs is unnecessarily restrictive.

As discussed thoroughly in the Statement of Basis and Purpose, EPA believes that human exposure to carcinogenic chemicals should be minimized to the extent feasible. The level of 0.10 mg/l TTHM in this interim regulation is based upon technological and economical feasibility in that the level is achievable and is consistent with the SDWA mandate to reduce exposure to contaminants in drinking water to the extent feasible, taking into consideration the potential health risks.

(2) Dr. Lyman stated that the possible benefits of GAC are unknown and GAC itself may have harmful effects on water quality.

The questions of benefits and release of harmful chemicals have been addressed previously in this Appendix. Data to date do not support the speculation that there are adverse effects from GAC use.

(3) Dr. Lyman noted that chloroform has been found in tomatoes, grapes and milk and is also produced in food processing. He urged that the total body burden must be considered in regulating chloroform.

As discussed previously in this Appendix and in the Statement of Basis and Purpose, EPA has examined several exposure routes of chloroform and feels that regulations controlling chloroform in drinking water are necessary since water can be the most significant source of exposure under typical conditions.

(4) Dr. Lyman commented that, in spite of wide-spread chronic industrial exposure to chloroform, there is no evidence of human carcinogenesis.

The unavailability of occupational risk data showing a precise relationship between exposure to chloroform in the work place and human carcinogenesis does not mean that chloroform poses no risk to humans. Systematic and scientifically sound studies have not yet been conducted to evaluate the possibility. However, in view of the positive carcinogenic response in the animal studies, EPA feels that regulations are appropriate at this time. This will result in reduced human exposure to many disinfection by-products, not only chloroform and THMs.

(5) Dr. Lyman stated that animal studies are useful in comparing effects on laboratory animals to human toxicity. EPA concurs with the use of animals in evaluating toxic effects of chemicals. EPA believes that carcinogenicity is one of several end points of toxicity and the statement by Dr. Lyman presented below also applies to the carcinogenic effect: "The toxicologist uses lower animals to predict the effects of chemicals on humans. Generally, the toxicity of a compound in lower animals is similar to that in humans on a dose per unit of body weight, particularly if the metabolic pathways and detoxification mechanisms are similar." Thus, EPA believes that cancers produced by chemicals in animals are evidence of human risk. Drs. Upton, Kennedy, Bingham, King, Bates and Hoel of NCI, FDA, OSHA, CPSC, and NIEHS, respectively, support this belief as presented in Appendix B.

(6) Dr. Lyman criticized EPA's use of the results of animal studies exposing them to high dosages to extrapolate

human health risks associated with exposure to low dosages on the grounds that high dose exposures were more likely than low doses to cause tissue damage which he claimed was a prerequisite to cancer introduction by chloroform. In support of his argument, he noted that high doses of liver and kidney toxins cause cancer to develop in those organs. He concluded that because lower dosages were less likely to damage tissue, they were also less likely to result in the development of tumors.

EPA does not agree with Dr. Lyman's hypothesis that tissue damage is necessary for cancer induction. The scientific community has not yet reached a consensus on this point. There are chemcials that cause the kind of tissue damage Dr. Lyman describes that do not go on to cause cancer (i.e., 1,1,1-trichloroethane). Thereby, tissue damage does not invariably lead to a carcinogenic response. Therefore, it is prudent and consistent with current scientific thought to assume that low level exposure to carcinogens, which may or may not cause direct tissue damage poses a human health risk. FDA, CPSC, NIEHS, NCI, and EPA agree that site-specific cancers are not necessarily found across species.

(7) Dr. Lyman also said that thresholds for carcinogens exist.

EPA believes that thresholds for carcinogens have not been experimentally demonstrated to date. This is thoroughly discussed in the preamble and in response to previous comments.

(8) Dr. Lyman commented that in order to produce tumors in people it would require drinking 15,000–30,000 gallons of water daily with a concentration of 311 ppb to produce tumors in humans.

EPA has evaluated this estimate and has concluded that the direct comparison of dosages from animals to humans in this way neither scientifically valid nor relevant.

(9) Dr. Lyman noted that one must differentiate between a real and potential risk.

EPA believes that sufficient information has been presented to demonstrate a risk from THM exposure that reduction of that risk is feasible and regulation is warranted and required by the SDWA.

66. Comments submitted on behalf of the Coalition for Safe Drinking Water by Farrel R. Robinson and EPA's responses are as follows:

(1) Dr. Robinson said that surveys of drinking water in various cities did demonstrate the presence of THMs but there were no realistic historical data with which these levels could be compared; the available epidemiological data are unreliable.

EPA is relying primarily on the animal toxicity data as the basis of the regulation. The correlational epidemiology is not inconsistent with this data, and assuming that similar raw water quality and chlorine dosage have been used over previous years which is a reasonable assumption in most cases, THM levels would not be significantly different.

(2) Dr. Robinson commented that there are significant problems in interpreting animal data and extrapolating their results to humans.

This has been responded to in detail above and in the preamble and Statement of Basis and Purpose.

(3) Dr. Robinson said NCI bioassays are only applicable to that strain of animals under the conditions of testing.

EPA believes that properly conducted studies in test animals do provide evidence of potential human risks from those chemicals. This is thoroughly discussed elsewhere in this Appendix, the preamble and the Statement of Basis and Purpose.

(4) Dr. Robinson commented that there is a threshold for carcinogens. He claimed that threshold cancer response extrapolations are contrary to scientific fact.

EPA believes that thresholds for carcinogens have not been demonstrated at this time. This is discussed in detail in the preamble, this Appendix and in the Statement of Basis and Purpose.

(5) Principles enunciated by the NAS are not principles but opinions.

EPA has relied on the NAS as representing the consensus of scientific opinion on these subjects.

67. Comments submitted by Dr. Alexander Grendon on behalf of the Coalition of Safe Drinking Water were as follow, that:

(1) EPA has not balanced costs against benefits for GAC. He stated that the costs were enormous while the theoretical benefits are minor.

(2) That there is a threshold for carcinogenesis.

(3) That cancer death rates have been declining for 25 years.

(4) That a person would have to live 74 years before a tumor would develop due to chloroform exposure.

(5) A person would have to live 35 lifetimes before dying from chloroform induced cancer.

Most of these comments has been addressed previously in this appendix and in the preamble. In regards to the time-to-tumor question, EPA feels that the state-of-the-art of toxicology does not provide for estimates such as those Dr. Grendon submitted. Rates of some types of cancer have declined but other types have risen in the past 25 years.

68. Comments submitted by Dr. Richard Reitz, representing Dow Chemical Company, and EPA's responses are as follows:

(1) Dr. Reitz commented that the use of GAC for organic chemical removal may release chemicals into treated waters that are carcinogenic. EPA has responded to this comment elsewhere in this Appendix.

(2) Dr. Reitz criticized EPA's use of the most conservative model for assessing human risk which he said greatly overestimated the risk of trace levels of organic chemicals in drinking water. He said that NCI should develop two separate risk extrapolation models, one for direct-acting carcinogens and another for metabolically model activated carcinogens. He commented that the extrapolation developed by Dr. David Rall and used by EPA's Cancer Assessment Group (CAG) was not appropriate for THMs since THMs are not direct-acting carcinogens but are carcinogens generally "involved in the variable drug metabolizing system," for which that model was not designed.

In support of his argument that EPA used an inappropriate risk model, he cited inconsistencies between the mouse and rat data in the NCI study. He noted that although based on the model one would have expected rats to be more sensitive to chloroform than mice, even though metabolism was required to activate chloroform, the opposite results were obtained. He therefore concluded that EPA's model overestimated the risk to rats by eleven-fold and overestimated the risk to humans by an even greater margin. Using pharmakokinetic data, Dr. Reitz predicted that the "chloroform risk" was one order of magnitude lower than that estimated by EPA.

EPA recognizes that other risk estimation models exist. Depending upon various assumptions, the computed levels can be significantly different among models. EPA has relied on the scientific expertise in the area of risk assessment of the NAS and EPA's CAG for its risk models which are considered to be state-of-the-art. While these models may be more conservative than Dr. Reitz's model, EPA believes that this was a reasonable and responsible choice in view of the SDWA's mandate to protect the public health.

EPA further found that the NAS–CAG models were appropriate for use for chloroform based on the best scientific evidence available. The fact that the results from the rat studies showed them to be four times less sensitive to

chloroform than mice does not mean hat the data cannot be used for human isk extrapolation. Species variability in ancer inductions mechanisms could be n explanation for this apparent nconsistency.

(3) Dr. Reitz stated that the doses of chloroform used in the NCI study produced gross liver damage long before the production of tumors. Thus, he said it was impossible to determine whether the carcinogenicity of chloroform was due to a genotoxic reaction or simply a secondary reaction to the extensive liver and kidney necrosis (i.e., epigenic).

As discussed previously, EPA feels high dosage tests are necessary and valid. EPA believes that large doses over long periods of time are required to produce effects in relatively small populations of animals and to increase the experimental sensitivity. The NCI, FDA, CPSC and NIEHS have concurred with this conclusion.

Moreover, one cannot conclude that the use of high dosages in animal experiments means that the resulting carcinogenicity is attributable solely to a toxic assault on the organ. Rather, toxic assaults leading to organ damage do not always evoke a carcinogenic response. Therefore, the particular hemical, in this case chloroform, must lso be implicated as a factor when a arcinogenic response is found.

(4) Dr. Reitz commented that since chloroform belongs to the class of chemicals which require metabolic activation for toxicity, one would expect the incidence of oncogenicity to be greater in those species with greater capacities to metabolize the chemical. Dr. Reitz assumed that the metabolic capability of rats was greater than mice and that of humans was greater than rats. He also postulated that glutathione availability was the limiting factor in the rate of macromolecular binding (a factor hypothesized as being a critical step in carcinogenicity).

Since more glutathione was expected to be available after lower dose exposures, Dr. Reitz argued that the chemical's carcinogenic potential at low dosages would be lower than if exposure had occurred at higher dosages. Based on these assumptions, he concluded that the human risk for chloroform was 71 times less than that estimated by CAG. Dr. Reitz said his calculations would result in an MCL between 0.01 mg/l and 0.1 mg/l for incremental risk of 10^{-6} and 10^{-5}, respectively.

EPA does not agree to with Dr. Reitz's ssumptions. His hypothesis concerning lutathione availability as a limiting actor in cancer induction has been shown not to be valid in tests using other similarly metabolized carcinogens at low exposure levels. Despite the differences between Dr. Reitz's and EPA's risk estimates, no specific risk value served as the basis for EPA's TTHM MCL, which was based upon technical feasibility factors.

(5) Dr. Reitz cited a study whereby chronic industrial exposure (50–125 ppm) of British Confectionary workers to chloroform for up to 10 years twenty years ago did not produce convincing epidemiology to link chloroform with increased cancer risk. EPA recognizes the difficulties involved with conducting epidemiology studies and this subject has been addressed previously.

(6) Dr. Reitz recommended the following changes be incorporated into the proposed THM regulation:

(a) That the MCL should be increased to 1.0–10 mg/l based on health effects data and risk models.

The MCL was based on a positive qualitative findings of carcinogenicity from animal bioassays and not on any quantitative risk extrapolation. The MCL for chloroform is that level which can be achieved given technological and economic feasibility factors.

(b) That definitive interspecies metabolism studies be carried out to allow a rationale species/species extrapolation. EPA agrees that this would provide additional information and has additional studies underway. However, regulatory action need not await the outcome of such studies.

(c) That a complete evaluation of the chloroform carcinogenicity potential below 200 mg/l be conducted. More research can always be conducted. EPA has an ongoing carcinogenicity study to evaluate chloroform at low levels of exposure. Again, regulatory action need not be delayed.

69. Dr. Joseph Schlosser, of Tulane Medical School, stated that:

(1) Bronchiogenic cancer should not be related to the Mississippi River and drinking water.

(2) The petrochemical industry could be the cause of increased cancer in Southern Louisiana.

(3) There is no consistent thinking about what the reason is for the high incidence of cancer in the New Orleans area. EPA's conclusions regarding the human epidemiology data, including that involving New Orleans, has been discussed elsewhere in this Appendix, in the preamble, and in the Statement of Basis and Purpose.

70. Three commenters said that separate MCLs should be set for each THM, such as chloroform, instead of for total THMs. One of these said that MCLs should only be established for those specific contaminants proven to be human or animal carcinogens. It was argued that, while all THMs were included in the proposed standards, only chloroform has been shown to produce a dose-response relationship for epithelia tumors of the kidney and renal pelvis in the rat and for hepatocellular carcinomas in mice. The other commenters felt that if standards were set for the THMs, concentrations of all THMs should be converted to the same base such as milliequivalents because grouping THMs on a weight basis and expressing the total THMs as mg/l was scientifically incorrect.

EPA's rationale for establishing a MCL for total THMs, instead of for only chloroform or for each THM separately, is set forth in greater detail in the preamble to these regulations and commenters are referred thereto. Although less is known about the health effects of the other THMs than about chloroform, EPA believes that carcinogenicity need not be proven before regulatory action may proceed. Based upon the similarity in chemical structure of all the THMs and the best available information on the health effects of the other THMs, EPA believes that they, as well as chloroform, pose adverse health risks which should be minimized to the extent feasible. It is also reasonable to regulate total THMs as a group because the gas chromatographic analytical method concurrently analyzes all four THMs; also treatment methods that would be employed to reduce chloroform would simultaneously reduce all of the THMs, since they are all formed through the use of chlorine in the disinfection process.

On the question of the use of milliequivalents instead of milligrams, EPA does not believe that such an approach would necessarily be meaningful since insufficient information is available to judge the relative potency of the four THMs to warrant that approach. Moreover, milligrams per liter have been used as the standard measurement for other drinking water MCLs in the NIPDWR and this term has become familiar to the water utilities that must comply with such standards.

71. In addition to those comments previously discussed, 136 comments were received discussing other issues related to sampling and monitoring for TTHMs. Of these, 43 commenters said they supported the sampling and monitoring requirements in the proposed regulations and found them to be adequate and reasonable. Many of these commenters, however, felt that EPA or the States should conduct or pay for the analyses. Seven commenters opposed

the monitoring program because of the added cost burden on utilities and noted the lack of laboratory facilities and skilled personnel. Fifty-one comments favored the monitoring requirements but opposed any requirement to notify the public of such results on the grounds that the public notification requirement would create unnecessary, expensive paper work as well as a "bad-feeling" among the public. One commenter felt that the reporting of THM monitoring data to EPA by utilities should apply only to States that are qualified for primacy.

EPA has already responded in this Appendix to those comments addressing the cost of monitoring. Under the SDWA, the cost of compliance with these regulations must be borne by the water utilities and EPA has taken this factor into consideration in determining minimum monitoring frequencies and has found that such costs are reasonable. With respect to public notification of the results of TTHM monitoring, Section 1414(c) of the SDWA requires that systems notify the public of any failure to comply with an applicable MCL as well as any failure to perform required monitoring. EPA does not believe that the costs of such public notification are unreasonable and any public notice may include appropriate explanation so that the public is adequately informed, but not misled.

The results of all monitoring are required to be reported to the States so that compliance with the regulations can be properly enforced and technical assistance can be provided to correct problems at the earliest possible time. Systems are also required to report results to EPA until such requirements are adopted by the States with primacy.

72. Twenty-four additional commenters raised questions regarding laboratory capabilities, quality assurance of results, and sampling and analytical procedures. They commented about the lack of qualified and experienced laboratories in the U.S. to perform TTHM analyses and about the fact that analytical procedures were not very well defined. They urged that the laboratory certification process be expedited and the analytical procedures be defined as soon as possible.

On the issue of the availability of laboratory facilities and analytical procedures, EPA has responded to those commenters concerned about the availability of sufficient numbers of laboratories capable of providing acceptable analytical data by extending the time frame for initiation of monitoring by systems serving more than 75,000 people from the proposed three months after promulgation to one year after promulgation. The 10,000 to 75,000 size category of systems are given 3 years from promulgation to begin monitoring. This will allow additional time for State and private laboratories to develop their capabilities and to become certified by EPA to provide data in support of compliance determinations. A quality assurance and certification program is also being developed by EPA, to determine the capable laboratories and to insure the reliability of data.

73. One commenter noted that EPA had failed to quantify the contribution of industrial and municipal discharges to the total concentrations of THMs and their precursors. EPA was urged to control THMs and precursor materials at their source; much of the THM in drinking water could be eliminated by not permitting any industrial or municipal discharges of THMs or THM precursors.

While THMs do occur in some drinking water sources as a result of municipal and industrial discharges, EPA has found that such levels are generally significantly lower than the levels associated with chlorination by-products in the finished drinking water. Most THMs in drinking water are the result of the reaction between chlorine and natural precursor compounds in the treatment process. Therefore, in most cases, control of THMs or precursor compounds municipal or industrial discharges would not likely have any significant effect upon THM levels in the drinking water.

74. One commenter noted that because of the inaccuracy and imprecision inherent in the analytical procedure for measurement of THMs, the MCL should include an allowance for the variations in analytical results.

Although EPA has established a single numerical value for the TTHM MCL, the variabilities associated with the analytical procedures have been taken into account in determining what laboratories will be deemed qualified for performing TTHM analyses. EPA has determined that 20% of 0.10 mg/l TTHM will be an allowable variation in the analytical results for purposes of laboratory approval and certification. Recent data show variations in properly run procedures of 10% to 20% and it is expected that as more experience is gained, the allowable variation will be reduced. Thus, while it is necessary to establish a single MCL value, quality control of laboratories is believed to be the most appropriate way of taking into account analytical variability.

Appendix B—Summary of Major Comments (for responses, see Appendix A)

I. Coalition for Safe Drinking Water

A. Introduction

The Coalition for Safe Drinking Water is a group of approximately 90 water systems—both investor and municipally owned—formed to present information and comments concerning EPA's proposed regulations.

The Coalition's doubts and disagreements about the substance of the proposed regulation centered upon EPA's conclusions that:

(1) The trace amount of THMs normally found in drinking water may pose a health risk, and,

(2) The GAC treatment technique is, at this time, required to reduce the levels of THMs in drinking water.

The Coalition also doubted EPA's authority to propose these new requirements as "amendments" to the interim primary drinking water regulations.

B. Legal Issues

1. EPA lacks the authority to promulgate the regulations as amendments to the National Interim Primary Drinking Water Regulations. The regulations are entirely new regulations and not modifications and to propose these regulations requires recommendations from NAS. The NAS has not made this recommendation. Further, the GAC technology was not available in December 1974 and all exemptions for water systems to avoid hardship will end on January 1, 1981.

C. Health Issues

1. Chloroform poses no potential cancer risk and there are no available data that support the premise of a causal relationship between the concentrations of THMs normally found in drinking water and cancer in humans.

2. The epidemiological studies that have been conducted concerning drinking water and a possible connection with cancer in humans are inconclusive.

3. EPA has relied upon animal studies for the hypothesis that trace organics pose a health concern. However, extrapolation of results in animal cancer studies to humans is fraught with its own set of problems and uncertainties.

4. The proposed regulations are based upon fear of the unknown using equivocal animal data and extrapolation models and methods which are unreliable.

5. The study cited by EPA to support the carcinogenicity of chloroform was "a preliminary screening test (by the

National Cancer Institute (NCI)) and not a definitive study." The study was not intended to be used to extrapolate health effects of chloroform to drinking water levels. The NCI study was inadequately controlled and did not follow proper scientific protocols. A new EPA/NCI study is underway and corrects deficiencies of the previous study and it is recommended that the implementation of any regulations be delayed until the studies are complete.

6. Dr. Roe's studies showed a no observed effect at 595,000 (drinking water equivalent) ppb chloroform in drinking water. Dr. Roe recommended a level of 300 ppb THM in drinking water based upon his studies of chloroform. Dr. Francis J. Roe's study with chloroform on dogs, rats and four strains of mice at low dose levels does not produce tumors in animals. Three hundred ppb would provide a margin of safety of 2,000. However, EPA uses 500 as a margin of safety.

7. EPA's proposed MCL of 100 ppb is needlessly low and will require costly additions or changes to water treatment facilities without any corresponding benefit being obtained.

8. There are no health effects data which support carcinogenicity of the other THMs.

9. Based upon most appropriate statistical extrapolation model, the level of the THM MCL should be no lower than 0.30 mg l since this provides a more than adequate margin of safety. However, this level is still too low to be justified on a cost-benefit basis if GAC is required.

10. There is no hard evidence that low level exposure to any of the chemicals produces cancer.

11. EPA estimates of environmental exposures to chloroform appear to be erroneous and it is suggested that EPA make every effort to obtain correct values for contributions from air, food and water. Also, there is the possibility that *in vivo* formation of chloroform and other THMs in the human body may occur. At this point, the available data suggest that more cost-effective avenues, such as control of chloroform in the work place, may be available for reducing THMs in the environment than by implementing the proposed THM MCL.

12. The concentrations of THMs detected in water systems present no mutagenic, teratogenic, or acute, subchronic, and chronic toxicological health risk to the public.

13. EPA has misconstrued the four very general "principles" stated by NAS. EPA has not properly used these principles and has ignored the available data. With regard to the first principle, EPA has not taken into account a number of variables in extrapolation of the animal data to humans; some of these variables include differences in such items as species response to carcinogens, weight between animals and man, intake of food and water, and routes of exposure. With regard to the second principle, EPA has ignored existing scientific data that show a threshold for no-effect responses with respect to a number of suspected carcinogens; there are a number of suspected carcinogens for which animal experiments have established a threshold level of effects; experiments involving humans suggest a no-effect level exists for chloroform; *in vivo* biological processes militate in favor of a no-effect level; and human exposure to natural carcinogens without adverse health effects support thresholds. With regard to the third principle, EPA has not considered the significance of the detoxification and repair mechanisms operative in animals and humans in its health assessment of THMs. With regard to the fourth principle, EPA has ignored the guidelines for assessing risk for chloroform as set forth in EPA's "Interim Guidelines for Carcinogen Risk Assessment." EPA used only the linear model for extrapolating the NCI data to humans, ignored the data of Roe, Eschenbrenner, and Miller, and ignored the estimates of risk by Tardiff using the "margin of safety," "probit-log" and "two step" extrapolation models.

14. EPA has ignored the relationship between dose and time-to-tumor observation in assessing the health risk of a carcinogenic material.

D. Treatment Technology and Economic/Energy Assessments

1. GAC has never been tested or proven on a full-scale operation in the United States and therefore constitutes a nationwide experiment for water treatment.

2. The use of GAC will have substantial financial impact upon water supplies and actual costs are very difficult to predict and are understated. For example, the average capital cost for a system serving over one million people will exceed $106 million with annual costs of more than $23 million. Rate increases for residential customers could be in the range of 40–70% and these rates could double where there are specific problems, such as land acquisition. These costs may result in insurmountable problems for some utilities in obtaining financing for GAC treatment facilities. EPA's assessment of the feasibility of financing the GAC treatment facilities is totally out of step with the realities of both the financing markets and operating needs of the public utilities.

3. The regulations will promote substantial new consumption of energy in operation of the treatment technologies as well as in secondary energy consumptions such as energy usage for GAC regeneration or energy associated with the manufacture and transportation of GAC.

4. The economic impact assessment did not take into account the costs of treating wastewater from GAC operations, such as backwash waters, wet scrubbers and drainage from carbon slurries. It is estimated that 50,000 gallons of waste water will be generated for every one million gallons of drinking water treated and half of that amount will need to be discharged. This will result in increased flows and higher O&M costs at municipal waste water treatment facilities on the order of four percent.

5. The costs were underestimated because of specific factors in the analysis. Based upon the use of GAC, the difference between their potential national cost estimates and EPA's estimates could be explained primarily by four factors (It was not clear to what extent these comments differentiated between costs for GAC for TTHM control and costs for GAC to control other synthetic organic chemicals in the separate treatment technique requirement):

(a) EPA determined its estimated capital costs for a system based upon the capacity of the entire system; whereas, the coalition estimated the system capital costs as equal to the sum of the capital costs for each treatment plant based on the capacity of each plant.

(b) EPA's estimates were based upon the system capacity on the average day of the peak month; whereas, the coalition's estimates were based upon the actual capacity of each treatment plant.

(c) EPA assumed that some of the affected systems would design facilities for a 9-minute empty bed contact time (EBCT); whereas, the coalition assumed that all GAC facilities would be designed for an 18-minute EBCT.

(d) The coalition's estimates for specific systems, based on the costing out of the individual components, were 30–80% higher than EPA's proposed estimates.

6. EPA has underestimated the costs of implementing the regulation by underestimating the number of impacted systems. This is the result of basing the analyses upon a model for the water supply industry and using a number of unfounded assumptions regarding the

number of systems that purchase water and use alternate disinfectants. Also, assumptions and predictions based upon NOMS were used to determine the level of THMs and the extent to which systems would be impacted further. Instead, EPA should have conducted sampling at all systems and based its estimates upon those results. The estimate of the 390 systems serving greater than 75,000 persons was not derived from EPA's Inventory of Systems but was based upon the TBS Policy Testing Model which left out numerous systems including all Federal Systems (e.g. District of Columbia) and the States of Hawaii and Alaska. Also, the hypothetical results of the TBS Model were never checked on to compare with reality. Finally, the number of systems using specific treatment systems such as GAC or no cost modifications were arbitrary assumptions.

7. EPA should provide a cost estimate of the stated goal of lowering the MCL at a later time to 50 ppb or 10 ppb.

8. The financial implications on water utilities have been underestimated by EPA. The financial analysis assumed that the rate increase required to finance the necessary revenue requirements would be obtained easily. Also, projections of future capital requirements in addition to the cost of the GAC process for various water systems were not factored into the analysis.

9. In order to install GAC, water utilities will need to raise capital through large rate increases. There are substantial regulatory barriers which could preclude water utilities from obtaining the necessary rate increases. Even if utilities are able to raise the capital funds, the quality of their credit and the attractiveness of their common stock will be severely reduced; this will reduce their ability to obtain external financing for normal water supply activities.

10. The GAC treatment process may result in serious problems and these may outweigh the alleged environmental benefit associated with GAC treatment. These problems include potential air pollution from regeneration and the waste water associated with GAC from contactor disinfection, backwashing, GAC quenching and transport, drainage from carbon slurries, and the regeneration furnace scrubbers. The total volume of waste water resulting from GAC facilities will be approximately 43,000 gallons per million gallons of water treated. Some of the waste water can be recycled but some will require pretreatment prior to disposal.

11. The use of GAC may constitute a larger health hazard than that of the alleged improvement of water quality. The potential health hazards associated with GAC include desorption, chromatographic effect (competitive displacement), resorption (leaching) of heavy metals and polycyclic aromatic hydrocarbons contained in the virgin or regenerated carbon, release of carbon fines, promotion (catalytic reactions) on the carbon itself of hazardous compounds due to chemical reactions between chlorine and organic compounds, bacterial growth on the carbon and air pollution from regeneration facilities. Indirect hazards associated with the GAC usage derive from the manufacture of GAC and the production of energy necessary to operate GAC facilities. These industries, such as the coal industry, pose a high risk of morbidity and mortality to the workers. Because of these concerns, additional research and testing should be conducted prior to implementation of GAC in this country's major waterworks. It is suggested that toxicological evaluations be conducted using concentrated effluents from GAC to assess these potential hazards.

12. EPA is required to analyze the costs of its actions in terms of the benefits hoped to be obtained but EPA has not done that.

E. Other Comments

1. EPA has failed to quantify the contribution of industrial and municipal discharges to the total concentrations of THMs and their precursors. EPA should control the THMs and precursor materials at their source, and much of the THM in drinking water could be eliminated by not permitting any industrial or municipal discharges of THMs or THM precursors.

2. Because of the inaccuracy and imprecision inherent in the analytical procedure for measurement of THMs, the MCL should include an allowance for the variations in analytical results.

3. If there is a necessity for a MCL for THMs, the MCL should apply to all water systems.

4. The EPA has not addressed the significant primary and secondary environmental problems associated with the use of GAC treatment facilities. Such concerns would normally be considered in an Environmental Impact Statement (EIS) prepared in accordance with the National Environmental Policy Act. However, EPA has stated that the supporting documentation for the regulations is the functional equivalent of an EIS. The EPA documents are not the functional equivalent of an EIS as they have not remotely analyzed the full potential environmental impact.

II. American Water Works Association

The AWWA's recommendations were:

1. Expanded and accelerated health-effects research on THM and synthetic organics as recommended by the NAS.

2. Establishment of 100 ppb level of TTHMs as a goal for all public water supply systems.

3. Elimination of EPA's proposed requirement of GAC as a treatment technique. In its place, EPA sponsorship of at least four plant-size research projects to gather financial and operating, as well as scientific data.

4. Adoption of EPA's proposed monitoring program for TTHM, except that public notification should not be required.

5. Establishment of an EPA financed and operated monitoring program for synthetic organic chemicals.

III. Environmental Defense Fund

The scientific evidence supporting the regulations is massive and convincing. A number of epidemiology studies have been conducted and provide strong support for the regulations in that taken as a whole they show a consistent pattern of association between drinking water and cancer mortality rates at certain sites.

Using the NAS model and Dr. Roe's data, the estimated risk of ingesting 200 ppb of chloroform over a lifetime in a community the size of one million would be predicted to result in 20 excess cancer deaths.

In a case study in New York State, it was found that for urban area populations drinking chlorinated water had a relative risk of 2.7 compared to populations in urban areas that do not drink chlorinated water. This would result in 250 excess cancer deaths per year in a population of one million.

The benefits of the regulation far outweigh the costs.

Because chloramines are quite ineffective in killing viruses and because viruses are not monitored for in drinking water supplies, any encouragement of chloramine usage should proceed with great caution.

The overwhelming consensus of the scientific community is that testing animals with high dosages is perfectly adequate for relating to humans.

Any delay in promulgating the regulations would be unconscionable, in view of the health effects data, and improper, in view of the requirements of the SDWA.

It is abundantly clear that the public wants safer drinking water since large

numbers are turning to alternative sources of water (bottled water) or to home water treatment devices. Unfortunately, all of the available evidence indicates that these alternatives are not adequate substitutes for municipally treated drinking water.

A regulation applicable to only half the population is not good enough and is inconsistent with the congressional intent that maximum feasible protection of public health be provided. The coverage should be expanded.

The level of the MCL should be the level achievable by the application of the most effective THM reducing technique applied to a relatively clean water source, such as an average water supply. A level of 50 ppb was suggested as a possible alternative to the proposed MCL.

IV. Supporting Comments on Health Basis of Regulation

A. Dr. Samuel Epstein, from the University of Illinois, endorsed the following principles:

1. There is no safe level of exposure to a carcinogen.
2. Animal carcinogens should be considered as human carcinogens.
3. Chemicals found to be carcinogenic at high doses in animals are carcinogenic at much lower doses in humans.
4. Chloroform is not the only chemical of concern in contaminated drinking water.
5. If the effects of cigarette smoking are eliminated, cancer rates are not in decline for many sites.
6. There have been 13 epidemiological studies which in context demonstrate an association between chlorinated drinking water and gastrointestinal urinary tract cancer.
7. GAC is a proven water treatment technology.

Dr. Epstein summarized the scientific basis for the regulations as follows:

1. Less than 10% of the 700 chemicals identified have been tested "for their toxicologic and carcinogenic effects."
2. NCI lists 23 of these as carcinogens, 30 as mutagens and 11 as promoting agents.
3. Fish and shellfish which live in polluted water have a high incidence of tumors.
4. Organic extracts of drinking water have been shown to be carcinogenic and mutagenic in animal tests.
5. Organic chemicals in drinking water have shown reproductive effects in one preliminary laboratory test.
6. Epidemiologic studies suggest association between drinking water contaminants and cancer.

B. Susan B. King, chairperson of the U.S. Consumer Product Safety Commission (CPSC) testified that CPSC concurred with the four principles for safety and risk assessment set forth by the NAS in its report, "Drinking Water and Health" and that CPSC also utilized them in their regulations of carcinogens. CPSC also concurred in EPA's conclusion that humans are also susceptible to effects observed in animals, as properly qualified. Ms. King noted that thresholds have not been demonstrated at which a "no effect" level for a carcinogen could be presumed and that varying individual susceptibilities must be considered in a heterogenous human population. She endorsed testing of chemicals at high levels in animals for assessing possible human risks. CPSC uses factors such as potency, extent and nature of human exposure and human uptake factors in evaluating risks from carcinogens. CPSC's interim policy for regulating carcinogens consists of prohibiting use if a reasonable substitute exists and prohibiting use in the absence of a reasonable substitute unless this would result in both unacceptable social and economic costs. CPSC's approach is comparable to EPA's in that the extent of the exposure and risk are considered as well as the availability and costs of alternatives.

C. Dr. Donald Kennedy, Commissioner of the Food and Drug Administration (FDA), stated that FDA was in full accord with the objective of protecting public health from organic chemicals in drinking water, and endorsed EPA's efforts to reduce exposure to THMs. FDA's recent actions to remove chloroform from drug and cosmetic products were consistent with this position.

The FDA agreed that feeding high doses of a carcinogen to test animals provides the most practical way to predict whether a chemical may cause cancer in humans. Dr. Kennedy noted that "the NCI study was a good one that provided a clear demonstration that chloroform is carcinogenic in experimental animals." FDA concurred with EPA's assessment that, since one cannot conclude with certainty that cholorform is or is not a human carcinogen, prudent public health policy demands that we assume the potential for carcinogenesis in humans unless there is strong evidence to the contrary.

Dr. Kennedy submitted as part of his written comments a paper entitled "What Animal Research Says About Cancer." In summary, it noted that testing with large doses of a chemical is the usual, and in most instances, the *only* way to determine whether it causes cancer. Epidemiology is fraught with unreasonable confounding factors from retrospective designs, and therefore, the threshold hypothesis has been rejected on the grounds that no threshold has yet been demonstrated for a carcinogen. However, animal testing can be used to confirm a cause-and-effect relationship between dosage and the incidence of cancer—a relationship general enough to be applied confidently to most hazardous chemicals used over long periods. Moreover, the similarities between cancer in animals and human beings, such as the fact that cancer cells are capable of metastasizing—breaking away from the original cancer and seeding themselves elsewhere—as well as the growing evidence that cancer-causing chemicals interfere with the biochemistry of genetic material, are powerful arguments for the appropriateness of using animals as models for people.

Finally, he found persuasive the comparison between the substances known to cause cancer in human beings and their effect on laboratory animals; or 18 such substances, all but two were also found to be carcinogenic in animals.

D. Dr. Eula Bingham, Assistant Secretary of the Department of Labor and head of the Occupational Safety and Health Administration (OSHA), concurred with Dr. Donald Kennedy's testimony. Dr. Bingham stated that trace contaminants may increase the risk of human cancer and produce other chronic effects. Large numbers of people are placed at risk to chemicals if they are present in drinking water.

Dr. Bingham supported limiting exposure to carcinogens to the lowest feasible level. She stated that animal evidence provides the best qualitative test for assessing potential human carcinogenic risk and that there is presently no means for determining a safe exposure level to a carcinogen. Due to the long latency period for chemical carcinogenesis, it would be imprudent to await the results of human epidemiological studies.

Thus, OSHA's generic proposal to regulate carcinogens relies on animal extrapolation for the detection of carcinogenic activity of chemicals. Because of the statistical insensitivity of laboratory bioassays conducted with limited numbers of animals, she stated that positive test results with experimental animals should generally supersede negative results and that it is appropriate to test chemicals at high exposure levels.

E. Dr. Arthur Upton, Director of the National Cancer Institute submitted

comments. Those points not previously included are stated below.

There are currently 32 carcinogens or suspected carcinogens, 30 mutagens or suspected mutagens, and 11 promoters in drinking water identified from a 1976 list of organic compounds.

Two sets of studies have been carried out to explore the relationship in humans between THMs in drinking water and possible increases in cancer. The first set used presumed measures of THM contamination (i.e., surface waters likely to be chlorinated) vs. ground water (likely to not be chlorinated). The second set used actual measures of THM levels. Nine of ten indirect studies showed a number of statistically significant associations between water quality and cancer.

From the three quantitative studies one could tentatively conclude that cancer of the urinary bladder, and perhaps large intestine are correlated, with THMs in water. He noted that a decrease of 100 micrograms per liter of chloroform in water could lead to a decrease in cancer rates of up to 7.5% in men and 10% in women for bladder cancer and between 7.5 and 8.5% in large intestinal cancer for women and men, respectively. Although these studies did not purport to "prove" a cause-effect association between THMs and cancer, Dr. Upton testified that the weight of evidence showed a "high index of suspicion" of such a relationship.

The additive or more than additive effects from multiple exposure to an array of organic carcinogens in drinking water are of such significance as to warrant an appraisal of the opportunity for modification of the total carcinogenic burden which may be traceable or produced by water processing to reduce the levels of total exposure.

The fact that source carcinogens from drinking water may persist in body tissues makes quantification of these effects difficult.

In the absence of conclusive and quantitative empirical evidence, Dr. Upton supported EPA's reliance on the NAS principles set forth in "Drinking Water and Health." He stated that every dose of a demonstrated carcinogen should be regarded as carrying some potential or presumptive risk. Animal studies must be used to evaluate human carcinogenic risk and to predict the safety of environmental chemicals if human victims are to be spared. He endorsed EPA's proposed TTHM MCL of 100 ppb as a "comprehensive public health measure" in the direction of cancer prevention. Measures taken to control large classes of contaminants were deemed useful for reducing levels of material whose carcinogenic or mutagenic potential was still unknown.

F. Dr. Upton was accompanied by Dr. Marvin Schneiderman and Dr. Umberto Saffiotti, from NCI, who explained the difficulties in predicting with any degree of accuracy, human risk posed by carcinogens due to low levels of exposure, variability in such levels, measurement problems, long latency periods and other confounding factors. They also endorsed EPA's approach to regulating THMs. Those points stated by Dr. Marvin Schneiderman of the NCI not covered previously are outlined below.

The experimental conditions to detect cancer in 1 in 100 or 1% of the time requires 20,000 animals. Experiments performed with 100 animals per dose group can detect approximately a 3% incidence. Three percent is an enormously high incidence. After all, breast cancer, the most common human cancer has a lifetime probability of 7.5% and lung cancer is 6%. Therefore, three percent is in line with the most common of cancers that cause the greatest concern.

G. Dr. Riley Housewright, National Academy of Sciences, provided a review of the NAS report, "Drinking Water and Health" and stated the following:

> Drinking Water regulations have not always been based entirely on health considerations even though protection of consumer health is the unqualified logical goal. For various reasons, drinking water standards have historically been set on the basis of: 1. contaminant background levels, 2. analytical detection limits, 3. technological feasibility of treatment processes, 4. aesthetic considerations, 5. health effects, and combinations of the above. In our report we have attempted to summarize the current knowledge of the health effects of contaminants in drinking water with the purpose of providing the scientific information required for establishing regulations based on health effects.

The NAS report did provide a relatively long list of recommendations for research but these recommendations were not be in lieu of establishing a standard for chloroform. He stated, "there appears to be no question but that, first of all, chloroform is found in drinking water, and it is a carcinogen."

Dr. Housewright also stated that the hazards of ingesting chemical pollutants in drinking water can be assessed in two general ways: epidemiology studies and laboratory studies of toxicity. The insidious effects of chronic exposure to low doses of toxic agents are difficult to recognize, because there are few, if any, early warning signs, and, when signs are ultimately observed, they often imply irreversible effects. In evaluating the potential effects on health of organic compounds found in drinking water, the NAS principal concern was to assess their carcinogenicity. The risk associated with the ingestion of compounds that were identified as carcinogenic were calculated by extrapolation from animal data. Chloroform was one of the compounds that produced cancer in both rats and mice. The NAS Safe Drinking Water Committee believed that: "these tests were valid and there is a hazard to man associated with the ingestion of chloroform," and that "chloroform and other THMs present a health hazard and that steps should be taken to prevent their formation or to remove them from drinking water." He stated that "Our committee believed these tests were valid and that there is a hazard to man associated with the ingestion of chloroform."

In addition, Dr. Housewright stated the following:

Some early epidemiological studies suggested an association between THMs and cancer. Our review of ten epidemiological studies concluded that the association was small and that there was a large margin of error. In most of the studies evaluated, the THM exposure and duration levels were inferred and confounding factors known to affect cancer incidence, such as cigarette-smoking, occupation, use of alcohol and drugs, socio-economic status and many others, were inadequately controlled. The failure of these studies to clearly establish a positive or negative cause and effect relationship between THMs and cancer resides to some extent in the complexities inherent in doing such studies.

We believe that THMs in drinking water present a human health hazard. The principal basis for this is that exposure to them results in cancer in two species of experimental animals. This conclusion is neither confirmed nor denied by the results of epidemiological studies now available; confirmation would require more sensitive epidemiological studies than have been conducted thus far. The examination of currently available epidemiological evidence gives no reason to change the conclusion of the study *Drinking Water and Health* which recommends that "strict criteria be applied when limits for chloroform in drinking water are established to protect the public health."

H. Dr. Richard Bates, National Institute of Environmental Health Sciences, and Dr. David Hoel, National Institute of Environmental Health Services and National Academy of Sciences, stated that determination of a quantitative standard for a contaminant in drinking water must be based upon a

judgment of the risk that is socially acceptable and upon a scientific estimation of the actual risk posed by the contaminant. Scientific estimation of risk from carcinogenic chemicals is not yet an exact science and until that time, regulatory agencies will have to act according to the most likely interpretation of scientific information while resolving uncertainties in a way that assures protection of the public health.

The four principles from NAS are consistent with what is now known about chemical carcinogenesis. The first principle is now widely accepted. Because epidemiology studies have problems of sensitivity and specificity and harmful effects can only be noted after the damage is done, experimental studies must be relied upon to judge the potential carcinogenicity of a chemical to humans. This practice is supported by the observation that most known human carcinogens are also carcinogenic in experimental animals, that generally the same kinds of metabolic enzymes that activate and detoxify chemical carcinogens are present in both human tissues and experimental animals, and that the general process of cancer development is similar in humans and experimental in animals.

With regard to the second and third principles, which discuss the inability to establish thresholds for carcinogens and the validity of using high doses, the fundamental reason for testing at high dose levels is to enhance the sensitivity of the experimental bioassay to detect a chemical carcinogen. A study of 100 animals can only detect the induction of cancer in no less than one percent of the animals. In order to detect lower levels of risk, it would be necessary to test much larger numbers of animals or to use mathematical procedures to estimate the level of risk from lower levels of exposure. The former approach is normally economically infeasible. The latter approach is based upon debatable scientific assumptions including that there is no threshold below which exposure to a carcinogen entails no risk. At the present time, it cannot be determined unequivocally whether or not thresholds exist or to determine which individuals in the population may or may not be able to tolerate additional exposure to carcinogenic chemicals.

The methods described in "Drinking Water and Health" are the best available to provide guidance on low level risks. In view of the many uncertainties, the safest action is always to reduce exposure to a chemical carcinogen to the lowest feasible level.

With regard to the numerical values that are produced by the models in terms of human risk per unit of exposure, Dr. Hoel stated that because of the inability to estimate the possible biological errors, biological differences between species and within species, and the experiences with the empirical data, the use of model predictions in ascribing some certain number of deaths in a population is not necessarily appropriate. He stated that the models could be used to rank carcinogens relative to their potency.

V. Calgon Corporation

As an example of comments providing information and data concerning the technical basis of the regulations, comments submitted by Calgon Corporation are summarized below.

1. GAC has been widely used for over 18 years in potable water applications to control taste, odor, and color in the U.S. and presently over 60 plants in the U.S. use GAC. In these applications, GAC has worked effectively with minimal problems without hazard or injury.

2. GAC is used to remove organic chemical contaminants from potable water in 21 cities in Europe and have been operating for up to 10 years. Most of these plants have on-site reactivation and have been operating without any adverse effects or undue difficulties.

3. GAC does not get into the water system from the filter beds. The bulk of the carbon lost is lost during the periodic backwashing of the carbon beds.

4. GAC does not add heavy metals or polynuclear aromatics (PAH) to the finished water. A composite sample of four activated carbons contained 7.36% ash of which 0.08% was soluble in water. Analysis for inorganic compounds showed very low levels but most significant is that the soluble portion of the ash is dissolved and discarded during the backwashing operation. During reactivation, the ash compounds are liberated and driven off in the furnace or the quench tank which contains boiling hot water, extracting any water soluble ash that is present.

Activated carbon is made by a multi-step process which is not conducive to the formation or retention of PAHs. The raw material, coal, is subject to an oxidation step, followed by a devolatilization step, followed by a long term high temperature (up to 2,000° F) activation step during which time the carbon granules are constantly turned in a reducing atmosphere. This process will drive off any materials with boiling points characteristic of PAHs. Experiments by a U.S. FDA laboratory have not been able to extract any PAHs from activated carbon. Activated carbon is such a strong adsorbent that even a small amount of polynuclear aromatics that might exist would be strongly adsorbed by the carbon.

5. GAC adsorbs organics and allow bacteria naturally present in the water to grow within the carbon bed. However, these bacteria are removed during backwashing and any bacteria in the effluent are easily controlled by disinfection following GAC. Bacterial growth has not been a problem at the more than 60 plants in the U.S. or in the systems in Europe.

6. In addition to its effectiveness in removing taste, odor and color from potable waters, GAC provides other advantages to the water treatment plant, such as savings in the amount of backwash water that is needed. Twenty to 40% savings over conventional media has been experienced by plants using GAC. Also, the demand for chlorine was reduced in these plants by 13% to 14% because organic contaminants had been reduced. Finally, use of GAC has extended service life between backwashes because of a reduction in head loss.

7. Energy requirements, based upon actual experiences, with reactivation of GAC used to treat industrial waste waters, are approximately 8,000 BTUs per pound of reactivated carbon. It is reasonable to expect that reactivation of GAC used to treat drinking water would require less energy. While the reactivation process is relatively energy intensive, the consumption of additional energy for reactivation of GAC from drinking water facilities will be insignificant in view of national consumption of energy.

8. Experience with furnaces reactivating GAC from industrial waste water facilities has shown that proper application of air pollution control technologies can be operated to comply with applicable air pollution requirements.

9. Compliance with the MCL is feasible and use of GAC for this purpose would most likely be for precursor removal.

10. The allotted time for compliance with the MCL is adequate. Systems that elect to use GAC to reduce THMs could be modified very quickly. For most applications, replacement of the existing filter media with GAC will be adequate. For greater bed depths, the necessary contact time can probably be achieved with relatively simple modifications of the existing filter systems. A few systems may require greater bed depth and thus additional time will probably be needed for those systems to make the modifications.

11. The utilities' cost estimates for GAC are overstated in that the capital

required for reactivation is based upon a redundant furnace. Based upon actual experience, it has not been necessary to have such substantial stand-by reactivation capacity. A more reasonable approach would be to utilize two furnaces of equal size with a total capacity equal to the peak flow rates and provide for stocking of buffer carbon to meet needs during periods of maintenance. Also, the use of an outside reactivation service could be used during long down times of the furnace. Detailed cost estimates were provided for GAC for two system sizes.

12. In order for the demand for GAC to be spread over a reasonable time frame, it is recommended that the regulations be phased in three segments separated by three months each.

VI. National Drinking Water Advisory Council

It is the opinion of the National Drinking Water Advisory Council (NDWAC) that EPA is justified in establishing an MCL of 100 ppb for THMs in finished drinking water on the basis of health hazard and feasibility. However, the MCL should not be restricted to utilities serving greater than 75,000 persons. The Council recommends that an MCL of 100 ppb THM also apply to utilities serving between 10,000 and 75,000 persons beginning three years after implementation of the regulation covering those utilities serving greater than 75,000 persons.

The Council also recommends that the implementation of the MCL of 100 ppb THM for utilities serving less than 10,000 persons be at the option of the agency having primacy in each state. The agency having primacy will be more familiar with the water supplies in that state and be better able to evaluate the potential for THM formation as a result of chlorine disinfection. This would serve to avoid unnecessary financial burdens on these utilities. The decision for compliance by those utilities should be made within five years.

The Council believes that the THM requirements should initially apply to all water sources (surface and ground). Where no THM problem is determined, the state should have the responsibility to determine the need for future monitoring requirements in order to assure that THMs do not pose a problem in the future.

It is imperative that the EPA publicly clarify its position relative to lowering the MCL for THM below 100 ppb. If the Agency believes the current health effects data supports an MCL lower than 100 ppb a detailed justification should be provided.

It is recommended that the EPA reconsider its restriction on the use of chloramines. Chloramines have been effectively used for disinfection in certain water systems for many years. Consequently, the Council believes that EPA's proposed regulation is unduly restrictive.

As previously expressed, the NDWAC is of the opinion that the standard plate count, although useful to the utility operator, should not be established as a regulatory requirement.

The Council concurs with the averaging method described in the proposed regulation for determining the level of THM in drinking water supplies.

Appendix C—Analysis of Trihalomethanes

Part I: The Analysis of Trihalomethanes in Drinking Water by the Purge and Trap Method

1. *Scope*

1.1 This method (1) is applicable in the determination of four trihalomethanes, i.e. chloroform, dichlorobromomethane, dibromochloromethane, and bromoform in finished drinking water, raw source water, or drinking water in any stage of treatment. The concentration of these four compounds is totaled to determine total trihalomethanes (TTHM).

1.2 For compounds other than the above-mentioned trihalomethanes, or for other sample sources, the analyst must demonstrate the usefulness of the method by collecting precision and accuracy data on actual samples as described (2).

1.3 Although the actual detection limits are highly dependent upon the gas chromatographic column and detector employed, the method can be used over a concentration range of approximately 0.5 to 1500 micrograms per liter.

1.4 Well in excess of 100 different water supplies have been analyzed using this method. Supplementary analyses using gas chromatography mass spectrometry (GC/MS) have shown that there is no evidence of interference in the determination of trihalomethanes (3). For this reason, it is not necessary to analyze the raw source water as is required with the Liquid/Liquid Extraction Method (4).

2. *Summary*

2.2 Trihalomethanes are extracted by an inert gas which is bubbled through the aqueous sample. The trihalomethanes, along with other organic constituents which exhibit low water solubility and a vapor pressure significantly greater than water, are efficiently transferred from the aqueous phase to the gaseous phase. These compounds are swept from the purging device and are trapped in a short column containing a suitable sorbent. After a predetermined period of time, the trapped components are thermally desorbed and backflushed onto the head of a gas chromatographic column and separated under programmed conditions. Measurement is accomplished with a halogen specific detector such as electrolytic conductivity or microcoulometric titration.

2.3 Confirmatory analyses are performed using dissimilar columns, or by mass spectrometry (5).

2.4 Aqueous standards and unknowns are extracted and analyzed under identical conditions in order to compensate for extraction losses.

2.5 The total analysis time, assuming the absence of other organohalides, is approximately 35 minutes per sample.

3. *Interferences*

3.1 Impurities contained in the purge gas and organic compounds outgasing from the plumbing ahead of the trap usually account for the majority of contamination problems. The presence of such inteferences are easily monitored as a part of the quality control program. Sample blanks are normally run between each set of samples. When a positive trihalomethane response is noted in the sample blank, the analyst should analyze a method blank. Method blanks are run by charging the purging device with organic-free water and analyzing in the normal manner.

If any trihalomethane is noted in the method blank in excess of 0.4 μg/l, the analyst should change the purge gas source and regenerate the molecular sieve purge gas filter. *Subtracting the blank values is not recommended.* The use of non-TFE plastic tubing, non-TFE thread sealants, or flow controllers with rubber components should be avoided since such materials generally out-gas organic compounds which will be concentrated in the trap during the purge operation. Such out-gasing problems are common whenever new equipment is put into service; as time progresses, minor out-gasing problems generally cure themselves.

3.2 Several instances of accidental sample contamination have been noted and attributed to diffusion of volatile organics through the septum seal and into the sample during shipment and storage. The sample blank is used as a monitor for this problem.

3.3 For compounds that are not efficiently purged, such as bromoform, small variations in sample volume, purge time, purge flow rate, or purge temperature can affect the analytical

result. Therefore, samples and standards must be analyzed under identical conditions.

3.4 Cross-contamination can occur whenever high-level and low-level samples are sequentially analyzed. To reduce this likelihood, the purging device and sample syringe should be rinsed twice between samples with organic-free water. Whenever an unusually concentrated sample is encountered, it is highly recommended that it be followed by a sample blank analysis to ensure that sample cross contamination does not occur. For samples containing large amounts of water soluble materials, it may be necessary to wash out the purging device with a soap solution, rinse with distilled water, and then dry in a 105°C oven between analyses.

3.5 Qualitative misidentifications are a problem in using gas chromatographic analysis. Whenever samples whose qualitative nature is unknown are analyzed, the following precautionary measures should be incorporated into the analysis.

3.5.1 Perform duplicate analyses using the two recommended columns (4.2.1 and 4.2.2) which provide different retention order and retention times for the trihalomethanes and other organohalides.

3.5.2 Whenever possible, use GC/MS techniques which provide unequivocal qualitative identifications (5).

4. *Apparatus*

4.1 The purge and trap equipment consists of three separate pieces of apparatus: the purging device, trap, and desorber. Construction details for a purging device and an easily automated trap-desorber hybrid which has proven to be exceptionally efficient and reproducible are shown in Figures 1 through 4 and described in 4.1.1. through 4.1.3. An earlier acceptable version of the above-mentioned equipment is described in (1).

4.1.1 Purging Device—Construction details are given in Figure 1 for an all-glass 5 ml purging device. The glass frit installed at the base of the sample chamber allows finely divided gas bubbles to pass through the sample while the sample is restrained above the frit. Gaseous volumes above the sample are kept to a minimum to eliminate dead volume effects, yet allowing sufficient space for most foams to disperse. The inlet and exit ports are constructed from heavy-walled ¼-inch glass tubing so that leak-free removable connections can be made using "finger-tight" compression fittings containing Teflon ferrules. The removable foam trap is used to control samples that foam.

4.1.2 Trapping Device—The trap (Figure 2) is a short gas chromatographic column which at <35° C retards the flow of the compounds of interest while venting the purge gas and, depending on which sorbent is used, much of the water vapor. The trap should be constructed with a low thermal mass so that it can be heated to 180° C in less than 1 minute for efficient desorption, then rapidly cooled to room temperature for recycling. Variations in the trap ID, wall thickness, sorbents, sorbent packing order, and sorbent mass could adversely affect the trapping and desorption efficiencies for compounds discussed in this text. For this reason, it is important to faithfully reproduce the trap configurations recommended in Figure 2. Traps containing Tenax only, or combinations of Tenax and other sorbents are acceptable for this analysis.

4.1.3 Desorber assembly—Details for the desorber are shown in Figures 3, and 4. With the 6-port valve in the Purge Sorb position (Figure 3), the effluent from the purging device passes through the trap where the flow rate of the organics is retarded. The GC carrier gas also passes through the 6-port valve and is returned to the GC. With the 6-port valve in the Purge-Sorb position, the operation of the GC is in no way impaired; therefore, routine liquid injection analyses can be performed using the gas chromatograph. After the sample has been purged, the 6-port valve is turned to the desorb position (Figure 4). In this configuration the trap is coupled in series with the gas chromatographic column allowing the carrier gas to backflush the trapped materials into the analytical column. Just as the valve is actuated, the power is turned on to the resistance wire wrapped around the trap. The power is supplied by an electronic temperature controller. Using this device, the trap is rapidly heated to 180° C and then maintained at 180° C with minimal temperature overshoot. The trapped compounds are released as a "plug" to the gas chromatograph. Normally, packed columns with theoretical efficiencies near 500 plates/foot under programmed temperature conditions can accept such desorb injections without altering peak geometry. Substituting a non-controlled power supply, such as a manually-operated variable transformer, will provide nonreproductible retention times and poor quantitative data unless Injection Procedure (8.9.2) is used.

4.1.4 Several Purge and Trap Devices are now commercially available. It is recommended that the following be taken into consideration if a unit is to be purchased:

a. Be sure that the unit is completely compatible with the gas chromatograph to be used for the analysis.

b. Use a 5-ml purging device similar to that shown in Figure 1.

c. Be sure the Tenax portion of the trap meets or exceeds the dimensions shown in Figure 2.

d. With the exception of sample introduction, select a unit that has as many of the purge trap functions automated as possible.

4.2 Gas chromatograph—The chromatograph must be temperature programmable and equipped with a halide specific detector.

4.2.1 Column I is an unusually efficient column which provides outstanding separations for a wide variety of organic compounds. Because of its ability to resolve trihalomethanes from other organochlorine compounds, column I should be used as the primary analytical column (see Table 1 for retention data using this column).

4.2.1.1 Column I parameters: Dimensions—8 feet long x 0.1 inch ID stainless steel or glass tubing. Packing—1% SP-1000 on Carbopack-B (60/80) mesh. Carrier Gas—helium at 40 ml/minute. Temperature program sequence: 45° C isothermal for 3 minutes, program at 8° C/minute to 220° C then hold for 15 minutes or until all compounds have eluted.

Note.—It has been found that during handling, packing, and programming, active sites are exposed on the Carbopack-B packing. This results in tailing peak geometry and poor resolution of many constituents. To correct this, pack the first 5 cm of the column with 3% SP-1000 on Chromosorb-W 60/80 followed by the Carbopack-B packing. Condition the precolumn and the Carbopack columns with carrier gas flow at 220° C overnight. Pneumatic shocks and rough treatment of packed columns will cause excessive fracturing of the Carbopack. If pressure in excess of 60 psi is required to obtain 40 ml/minute carrier flow, then the column should be repacked.

4.2.1.2 Acceptable column equivalent to Column I: Dimensions—8 feet long x 0.1 inch ID stainless steel or glass tubing. Packing—0.2% Carbowax 1500 on Carbopack-C (80/100) mesh. Carrier Gas—helium at 40 ml/minute. Temperature program sequence—60° C isothermal for 3 minutes, program at 8° C /minute to 160° C, then hold for 2 minutes or until all compounds have eluted.

Note.—It has been found that during handling, packing, and programming, active sites are exposed on the Carbopack-C packing. This results in poor resolution of constituents and poor peak geometry. To correct this, place a 1 ft. 0.125 in. OD x 0.1 in.

ID stainless steel column packed with 3% Carbowax 1500 on Chromosorb–W 60/80 mesh in series before the Carbopack–C column. Condition the precolumn and the Carbopack columns with carrier gas flow at 190° C overnight. The two columns may be retained in series for routine analyses. Trihalomethane retention times are listed in Table 1.

4.2.2 Column II provides unique organohalide-trihalomethane separations when compared to those obtained from Column I (see Figures 5 and 6). However, since the resolution between various compounds is generally not as good as those with Column I, it is recommended that Column II be used as a qualitative confirmatory column for unknown samples when GC/MS confirmation is not possible.

4.2.2.1 Column II parameters: Dimensions—6 feet long x 0.1 inch ID stainless steel or glass. Packing—n-octane on Porisil–C (100/120 mesh). Carrier Gas—helium at 40 cc/minute. Temperature program sequence—50° C isothermal for 3 minutes, program at 6°/minute to 170° C, then hold for 4 minutes or until all compounds have eluted. Trihalomethane retention times are listed in Table 1.

5.8 Organic-free water is defined as water free of interference when employed in the purge and trap analysis.

5.8.1 Organic-free water is generated by passing tap water through a carbon filter bed containing about 1 lb. of activated carbon. Change the activated carbon bed whenever the concentration of any trihalomethane exceeds 0.4 μg/l.

5.8.2 A Millipore Super-Q Water System or its equivalent may be used to generate organic-free water.

5.8.3 Organic-free water may also be prepared by boiling water for 15 minutes. Subsequently, while maintaining the temperature at 90° C, bubble a contaminant-free inert gas through the water for one hour. While still hot, transfer the water to a narrow-mouth screw-cap bottle with a Teflon seal.

5.8.4 Test organic free water each day it is used by analyzing according to Section 8.

5.9 Standards.*

5.9.1 Bromoform—96%—available from Aldrich Chemical Company.

5.9.2 Bromodichloromethane 97%—available from Aldrich Chemical Company.

5.9.3 Chlorodibromomethane—available from Columbia Chemical Inc., Columbia, S.C.

5.9.4 Chloroform—99%—available from Aldrich Chemical Company.

* As a precautionary measure, all standards must be checked for purity by boiling point determinations or GC/MS assays (5).

5.10 Standard Stock Solutions

5.10.1 Place about 9.8 ml of methyl alcohol into a ground glass stoppered 10 ml volumetric flask.

5.10.2 Allow the flask to stand unstoppered about 10 minutes or until all alcohol wetted surfaces have dried.

5.10.3 Weigh the flask to the nearest 0.1 mg.

5.10.4 Using a 100 μl syringe, immediately add 2 drops of the reference standard to the flask, then reweigh. *Be sure that the 2 drops fall directly into the alcohol without contacting the neck of the flask.*

5.10.5 Dilute to volume, stopper, then mix by inverting the flask several times.

5.10.6 Transfer the solution to a dated and labeled 15 ml screw cap bottle with a Teflon cap liner.

Note.—Because of the toxicity of trihalomethanes, it is necessary to prepare primary dilutions in a hood. It is further recommended that a NIOSH/MESA approved toxic gas respirator be used when the analyst handles high concentrations of such materials.

5.10.7 Calculate the concentration in micrograms per microliter from the net gain in weight.

5.10.8 Store the solution at 4° C.

Note.—All standard solutions prepared in methyl alcohol are stable up to 4 weeks when stored under these conditions. They should be discarded after that time has elapsed.

5.11 Aqueous Calibration Standard Precautions.

5.11.1 In order to prepare accurate aqueous standard solutions, the following precautions must be observed.

a. Do not inject more than 20 μl of alcoholic standards into 100 ml of organic-free water.

b. Use of 25 μl Hamilton 702N microsyringe or equivalent. (Variations in needle geometry will adversely affect the ability to deliver reproducible volumes of methanolic standards into water.)

c. Rapidly inject the alcoholic standard into the expanded area of the filled volumetric flask. Remove the needle as fast as possible after injection

d. Mix aqueous standards by inverting the flask three times only.

e. Discard the contents contained in the neck of the flask. Fill the sample syringe from the standard solution contained in the expanded area of the flask as directed in Section 8.5.

f. Never use pipets to dilute or transfer samples or aqueous standards.

g. Aqueous standards when stored with a headspace are not stable and should be discarded after one hour.

h. Aqueous standards can be stored according to Sections 6.4 and 8.6.

5.11.2 Prepare, from the standard stock solutions, secondary dilution mixtures in methyl alcohol so that a 20 μl injection into 100 ml or organic-free water will generate a calibration standard which produces a response close (±10%) to that of the sample (See 9.1).

5.11.3 Purge and analyze the aqueous calibration standards in the same manner as the samples.

5.11.4 Other calibration procedures (3) which require the delivery of less than 20 μl of a methanolic standard into a 5.0 ml volume of water already contained in the sample syringe are acceptable only if the methanolic standard is delivered by the solvent flush technique (6).

5.12 Quality Check Standard (2.0 μg/l)

5.12.1 From the standard stock solutions, prepare a secondary dilution in methyl alcohol containing 10 ng/μl of each trihalomethane (See Section 5.10.8 Note).

5.12.2 Daily, inject 20.0 μl of this mixture into 100.0 ml of organic-free water ana analyze according to Section 8.

6. *Sample Collection and Handling*

6.1. The sample containers should have a total volume of at least 25 ml.

6.1.1 Narrow mouth screw cap bottles with the TFE fluorocarbon face silicone sepata cap liners are strongly recommended.

6.2 Sample Bottle Preparation

6.2.1 Wash all sample bottles and TFE seals in detergent. Rinse with tap water and finally with distilled water.

6.2.2 Allow the bottles and seals to air dry at room temperature, then place in a 105° C oven for one hour, then allow to cool in a area known to be free of organics.

Note.—Do not heat the TFE seals for extended period of time (>1 hour) because the silicone layer slowly degrades at 105° C.

6.2.3 When cool, seal the bottles using the TFE seals that will be used for sealing the samples.

6.3 Sample Stabilization—A chemical reducing agent (Section 5.6) is added to the sample in order to arrest the formation of trihalo-methanes after sample collection (3, 7). *Do not add the reducing agent to samples when data on maximum trihalomethane formation is desired.* If chemical stabilization is employed, the reagent is also added to the blanks. The chemical agent (2.5 to 3 mg/40 ml) is added to the empty sample bottles just prior to shipping to the sampling site.

6.4 Sample Collection

6.4.1 Collect all samples in duplicate.

6.4.2 Fill the sample bottles in such a manner that no air bubbles pass through the sample as the bottle is filled.

6.4.3 Seal the bottles so that no air bubbles are entrapped in it.

6.4.4 Maintain the hermetic seal on the sample bottle until analysis.

6.4.5 Sampling from a water tap.

6.4.5.1 Turn on water and allow the system to flush until the temperature of the water has stabilized. Adjust the flow to about 500 ml/minute and collect duplicate samples from the flowing stream.

6.4.6 Sampling from an open body of water.

6.4.6.1 Fill a 1-quart wide-mouth bottle with sample from a representative area. Carefully fill duplicate sample bottles from the 1-quart bottle as noted in 6.4.2.

6.4.7 If a chemical reducing agent has been added to the sample bottles, fill with sample just to overflowing, seal the bottle, and shake vigorously for 1 minute.

6.4.8 Sealing practice for septum seal screw cap bottles.

6.4.8.1 Open the bottle and fill to overflowing, place on a level surface, position the TFE side of the septum seal upon the convex sample meniscus and seal the bottle by screwing the cap on tightly.

6.4.8.2 Invert the sample and lightly tap the cap on a solid surface. The absence of entrapped air indicates a successful seal. If bubbles are present, open the bottle, add a few additional drops of sample and reseal the bottle as above.

6.4.9 Blanks.

6.4.9.1 Prepare blanks in duplicate at the laboratory by filling and sealing sample bottles with organic-free water just prior to shipping the sample bottles to the sampling site.

6.4.9.2 If the sample is to be stabilized, add an identical amount of stabilization reagent to the blanks.

6.4.9.3 Ship the blanks to and from the sampling site along with the sample bottles.

6.4.9.4 Store the blanks and the samples collected at a given site (sample set) together. A sample set is defined as all the samples collected at a given site (i.e., at a water treatment plant, the duplicate raw source waters, the duplicate finished waters and the duplicate blank samples comprise the sample set).

6.5 When samples have been collected according to Section 6, no measurable loss of trihalomethanes has been detected over extended periods of storage time (3). It is recommended that all samples be analyzed within 14 days of collection.

7. *Conditioning Traps*

7.1 Condition newly packed traps overnight at 180° C with an inert gas flow of at least 20 ml/min.

7.1.1 Vent the trap effluent to the room, not to the analytical column.

7.2 Prior to daily use, condition traps 10 minutes while backflushing at 180° C. It may be beneficial to routinely condition traps overnight while backflushing at 180° C.

7.2.1 The trap may be vented to the analytical column; however, after conditioning, the column must be programmed prior to use.

8. *Extraction and Analysis*

8.1 Adjust the purge gas (nitrogen or helium) flow rate to 40 ml/min.

8.2 Attach the trap inlet to the purging device. Turn the valve to the purge-sorb position (Figure 3).

8.3 Open the syringe valve located on the purging device sample introduction needle.

8.4 Remove the plungers from two 5 ml syringes and attach a closed syringe valve to each.

8.5 Open the sample bottle and carefully pour the sample into one of the syringe barrels until it overflows. Replace the syringe plunger and compress the sample. Open the syringe valve and vent any residual air while adjusting the sample volume to 5.0 ml. Close the valve.

8.6 Fill the second syringe in an identical manner from the same sample bottle. This second syringe is reserved for a duplicate analysis, if necessary (See Sections 9.3 and 9.4).

8.7 Attach the syringe-valve assembly to the syringe valve on the purging device.

8.8 Open the syringe valve and inject the sample into the purging chamber. Close both valves. Purge the sample for 11.0±.05 minutes.

8.9 After the 11-minute purge time, attach the trap to the chromatograph (turn the valve to the desorb position) and introduce the trapped materials to the GC column by rapidly heating the trap to 180°C while backflushing the trap with an inert gas between 20 and 60 ml/min for 4 minutes.

8.9.1 If the trap can be rapidly heated to 180°C and maintained at this temperature, the GC analysis can begin as the sample is desorbed, i.e., the column is at the initial 45°C operating temperature. The equipment described in Figure 4 will perform accordingly.

8.9.2 With other types of equipment (see Section 4.1.4 and Reference 1) where the trap is not rapidly heated or is not heated in a reproducible manner, it may be necessary to transfer the contents of the trap into the analytical column at <30°C where it is once again trapped. Once the transfer is complete (4 minutes), the column is rapidly heated to the initial operating temperature for analysis.

8.9.3 If injection procedure 8.9.1 is used and the early eluting peaks in the resulting chromatogram have poor geometry or variable retention times, then Section 8.9.2 should be used.

8.10 After the extracted sample is introduced into the gas chromatograph, empty the gas purging device using the sample introduction syringe, followed by two 5-ml flushes of organic-free water. When the purging device is emptied, leave the syringe valve open allowing the purge gas to vent through the sample introduction needle.

8.11 Analyze each sample and sample blank from the sample set in an identical manner (see Section 6.4.9.4) on the same day.

8.12 Prepare calibration standards from the standard stock solutions (Section 5.10) in organic-free water that are close to the unknown in trihalomethane composition and concentration (Section 9.1). The concentrations should be such that only 20 μl or less of the secondary dilution need be added to 100 ml of organic-free water to produce a standard at the same level as the unknown.

8.13 As an alternative to Section 8.12, prepare a calibration curve for each trihalomethane containing at least 3 points, two of which must bracket the unknown.

9. *Analytical Quality Control*

9.1 Analyze the 2 μg/l check sample daily before any samples are analyzed. Instrument status checks and lower limit of detection estimations based upon response factor calculations at five times the noise level are obtained from these data. In addition, response factor data obtained from the 2 μg/l check standard can be used to estimate the concentration of the unknowns. From this information, the appropriate standard dilutions can be determined.

9.2 Analyze the sample blank to monitor for potential interferences as described in Sections 3.1, 3.2, and 3.4.

9.3 Spiked Samples

9.3.1 For laboratories analyzing more than 10 samples a day, each 10th sample should be a laboratory generated spike which closely duplicates the average finished drinking water in trihalomethane composition and concentration. Prepare the spiked sample in organic-free water as described in Section 5.11.

9.3.2 For laboratories analyzing less than 10 samples daily, each time the analysis is performed, analyze at least 1 laboratory generated spike sample which closely duplicates the average finished drinking water in

trihalomethane composition and concentration. Prepare the spiked sample in organic-free water as described in Section 5.11.

9.4 Randomly select and analyze 10% of all samples in duplicate.

9.4.1 Analyze all samples in duplicate which appear to deviate more than 30% from any established norm.

9.5 Maintain an up-to-date log on the accuracy and precision data collected in Sections 9.3 and 9.4. If results are significantly different than those cited in Section 11.1, the analyst should check out the entire analyses scheme to determine why the laboratory's precision and accuracy limits are greater.

9.6 Quarterly, spike an EMSL-Cincinnati trihalomethane quality control sample into organic-free water and analyze.

9.6.1 The results of the EMSL trihalomethane quality control sample should agree within 20% of the true value for each trihalomethane. If they do not then the analyst must check each step in the standard generation procedure to solve the problem (Section 5.9, 5.10, and 5.11).

9.7 Maintain a record of the retention times for each trihalomethane using data gathered from spiked samples and standards.

9.7.1 Daily calculate the average retention time for each trihalomethane and the variance encountered for the analyses.

9.7.2 If individual trihalomethane retention time varies by more than 10% over an eight hour period or does not fall with 10% of an established norm, the system is "out of control." The source of retention data variation must be corrected before acceptable data can be generated.

10. *Calculations*

10.1 Locate each trihalomethane in the sample chromatogram by comparing the retention time of the suspect peak to the data gathered in 9.7.1. The retention time of the suspect peak must fall within the limits established in 9.7.1 for single column identification.

10.2 Calculate the concentration of the samples by comparing the peak height or peak areas of the samples to the standard peak height (8.12). Round off the data to the nearest μg/l or two significant figures.

$$\mu g/l \left(\frac{\text{peak height sample}}{\text{peak height standard}}\right) \times (\text{conc std } \mu g/l)$$

10.3 Report the results obtained from the lower limit of detection estimates along with the data for the samples.

10.4 Calculate the total trihalomethane concentration (TTHM) by summing the 4 individual trihalomethane concentrations in μg/l. TTHM (μg/l) = (Conc. $CHCl_3$) + (Conc. $CHBrCl_2$) + (Conc. $CHBr_2Cl$) + (Conc. $CHBr_3$).

10.5 Calculate the limit of detection (LOD) for each trihalomethane not detected using the following criteria:

$$\text{LOD } (\mu g/l) = \left(\frac{A \times ATT}{B \times ATT}\right) (2\ \mu g/l)$$

where B = peak height (mm) of 2 μg/l quality check standard

A = 5 times the noise level in (mm) at the exact retention time of the trihalomethane or the baseline displacement in (mm) from the theoretical zero at the exact retention time of the trihalomethane.

ATT = Attenuation factor

11. *Accuracy and Precision*

11.1 One liter of organic-free water was spiked with the trihalomethanes and used to fill septum seal vials which were stored under ambient conditions. The spiked samples were randomly analyzed over a 2-week period of time. The single laboratory data listed in Table II reflect the errors due to the analytical procedure and storage.

References

1. Bellar, T. A., J. J. Lichtenberg. Determining Volatile Organics at the Microgram per Litre Levels by Gas Chromatography, Journal AWWA, *66*, 739 (December 1974).

2. "Handbook for Analytical Quality Control in Water and Wastewater Laboratories," Analytical Quality Control Laboratory, National Environmental Research Center, Cincinnati, Ohio, June 1972.

3. Brass, H. J., et al., "National Organic Monitoring Survey Sampling and Purgeable Organic Compounds, Drinking Water Quality Through Source Protection," R. B. Pojasek, Editor, Ann Arbor Science, p. 398, 1977

4. "The Analysis of Trihalomethanes in Finished Water by the Liquid/Liquid Extraction Method, Method 501.2" Environmental Monitoring and Support Laboratory, Environmental Research Center, Cincinnati, Ohio, 45268, May 15, 1979.

5. Budde, W. L. and J. W. Eichelberger, "Organics Analysis Using Gas Chromatography-Mass Spectrometry," Ann Arbor Science, Ann Arbor, Michigan, 1979

6. White, L. D. et al., "Convenient Optimized Method for the Analysis of Selected Solvent Vapors in the Industrial Atmosphere," AIHA Journal, Vol. 31, p. 225, 1970

7. Kopfler, F. C., et al. "GC/MS Determination of Volatiles for the National Organics Reconnaissance Survey (NORS) or Drinking Water, Identification and Analysis of Organic Pollutants in Water," L. H. Keith, Editor, Ann Arbor Science, p. 87, 1976

Table I—*Retention Data for Trihalomethanes*

Trihalomethane	Retention time minutes		
	Column I 1% sp1000 Carbopack B	Acceptable Alternative to column I 0.4% Carbowax Carbopack	Column II n-octane Porasil C
Chloroform	10.7	8.2	12.2
Bromodichloromethane	13.7	10.8	14.7
Chlorodibromomethane (Dibromochloromethane)	16.5	13.2	16.6
Bromoform	19.2	15.7	19.2

Table II—*Single Laboratory Accuracy and Precision for Trihalomethanes*

Spike μg/l	Number samples	Mean μg/l	Precision standard deviation	Accuracy percent recovery
		Chloroform		
1.2	12	1.2	0.14	100
12.0	8	11	0.16	92
119.0	11	105	7.9	88
		Bromodichloromethane		
1.6	12	1.5	0.05	94
16.0	8	15	0.39	94
160.0	11	145	10.2	91
		Chlorodibromomethane		
2.0	12	1.9	0.09	95
20.0	8	19	0.70	95
196.0	11	185	10.6	94
		Bromoform		
2.3	12	2.3	0.16	100
23.0	8	23	1.38	100
231.0	11	223	16.3	97

BILLING CODE 6560-01-M

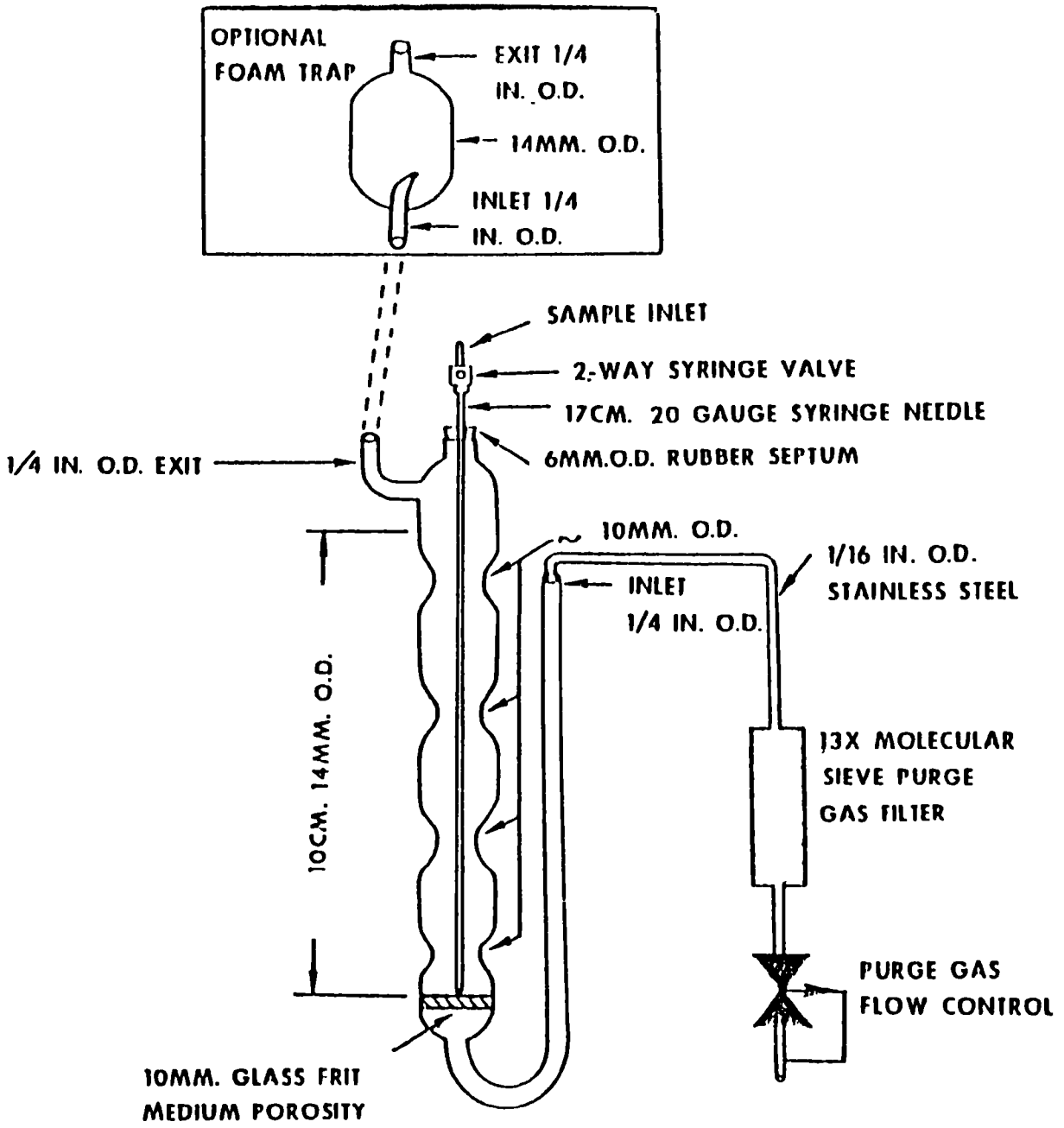


FIGURE 1. PURGING DEVICE

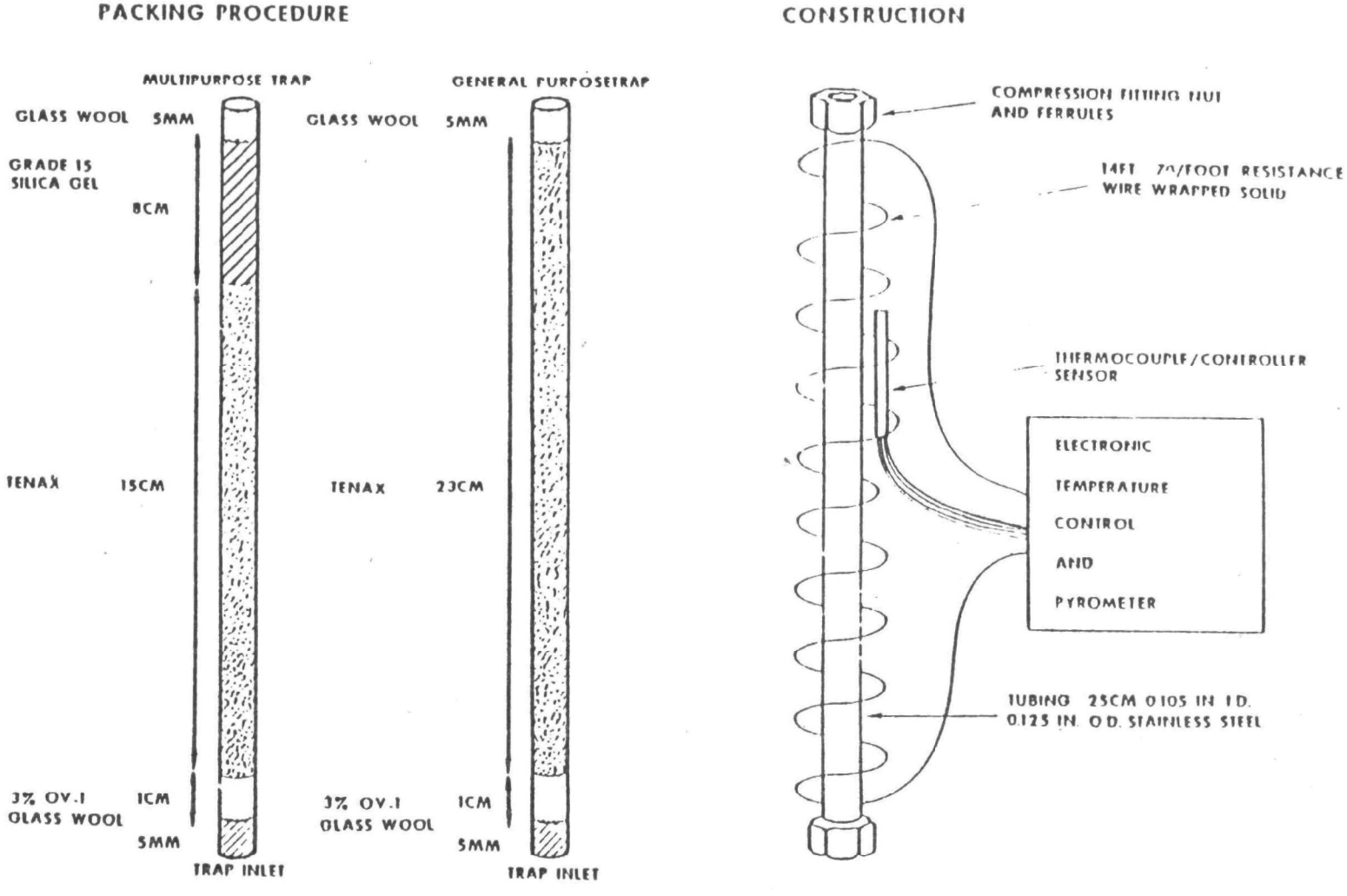


FIGURE 2 TRAP

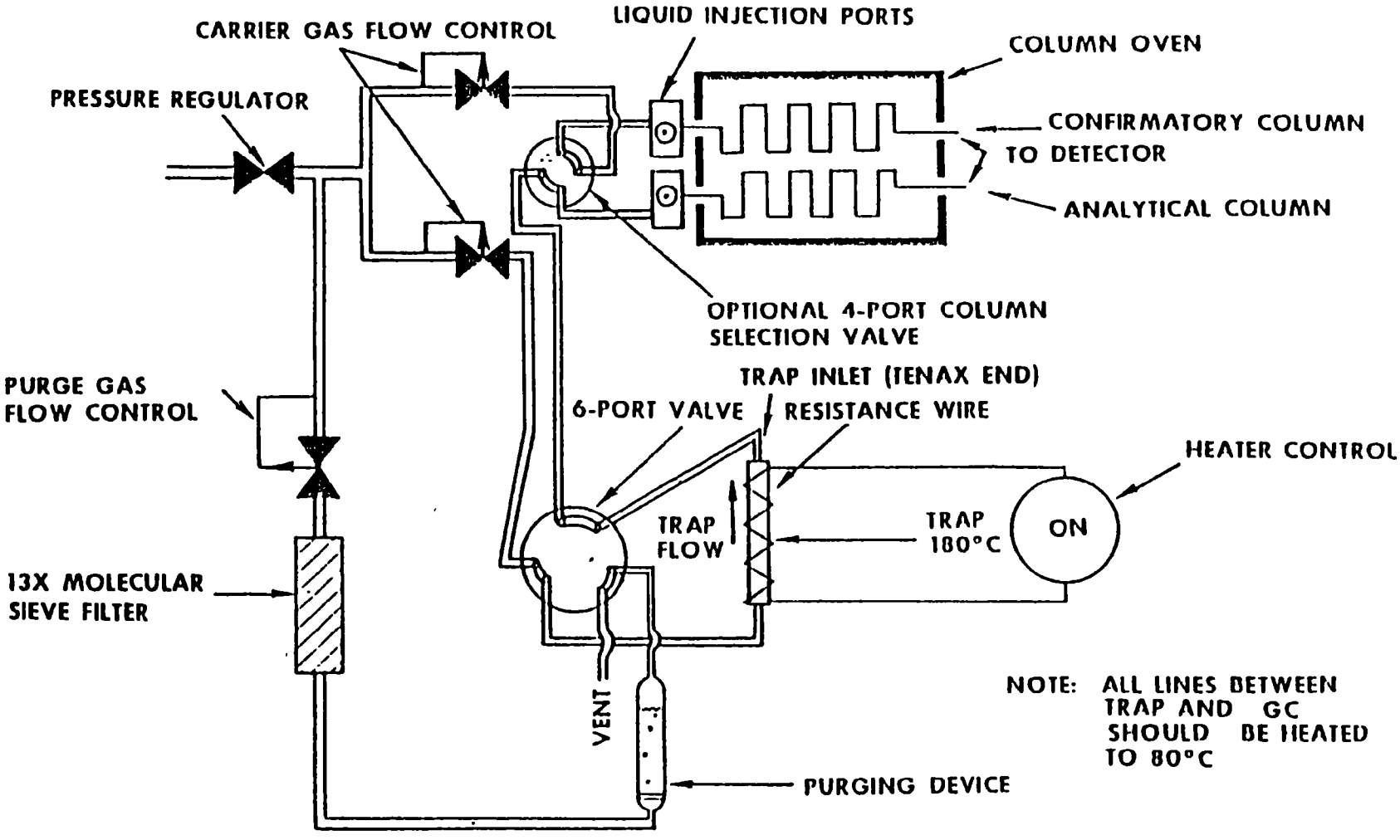


FIGURE 4 PURGE-TRAP SYSTEM (DESORB MODE)

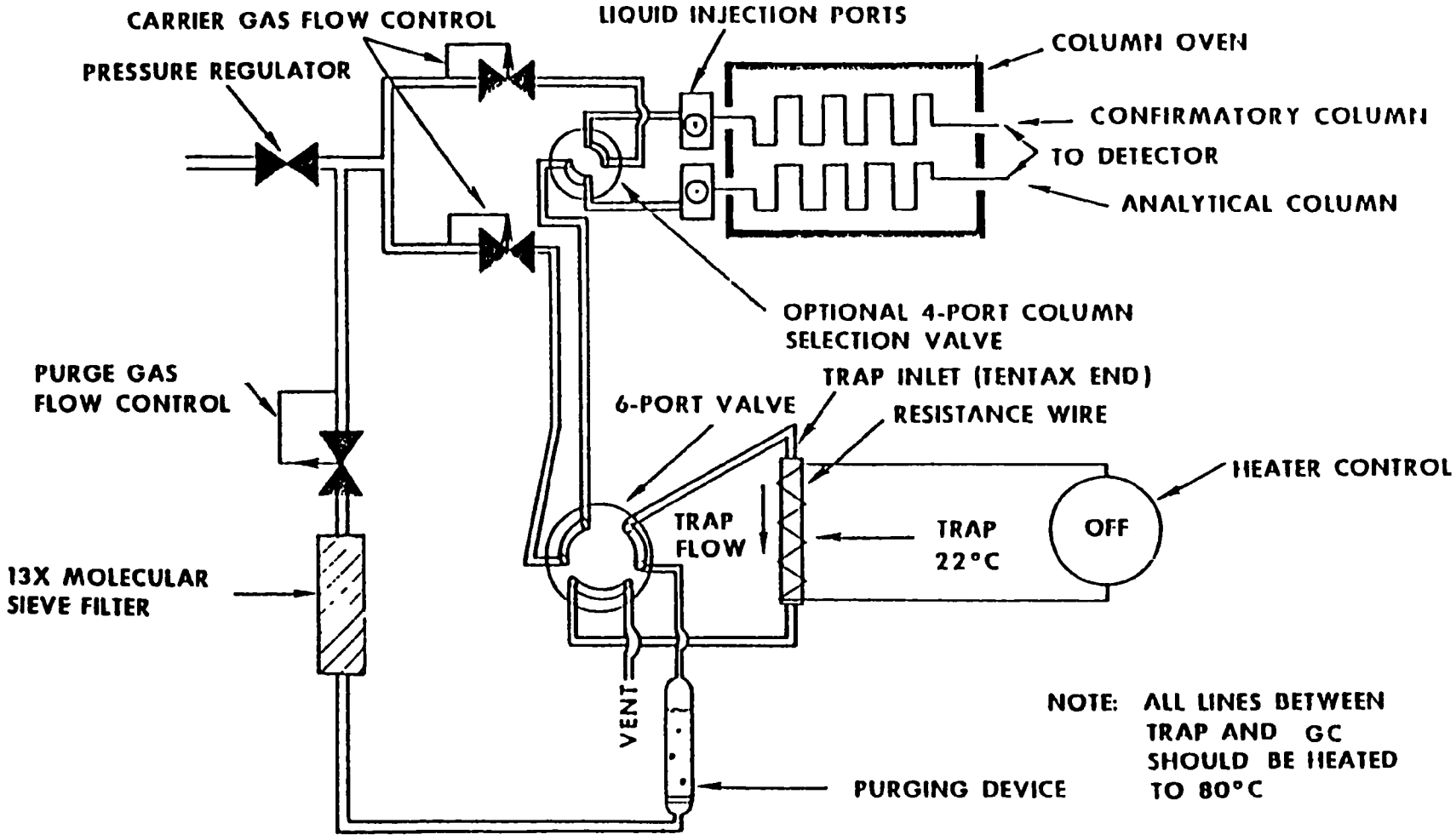


FIGURE 3 PURGE-TRAP SYSTEM (PURGE-SORB MODE)

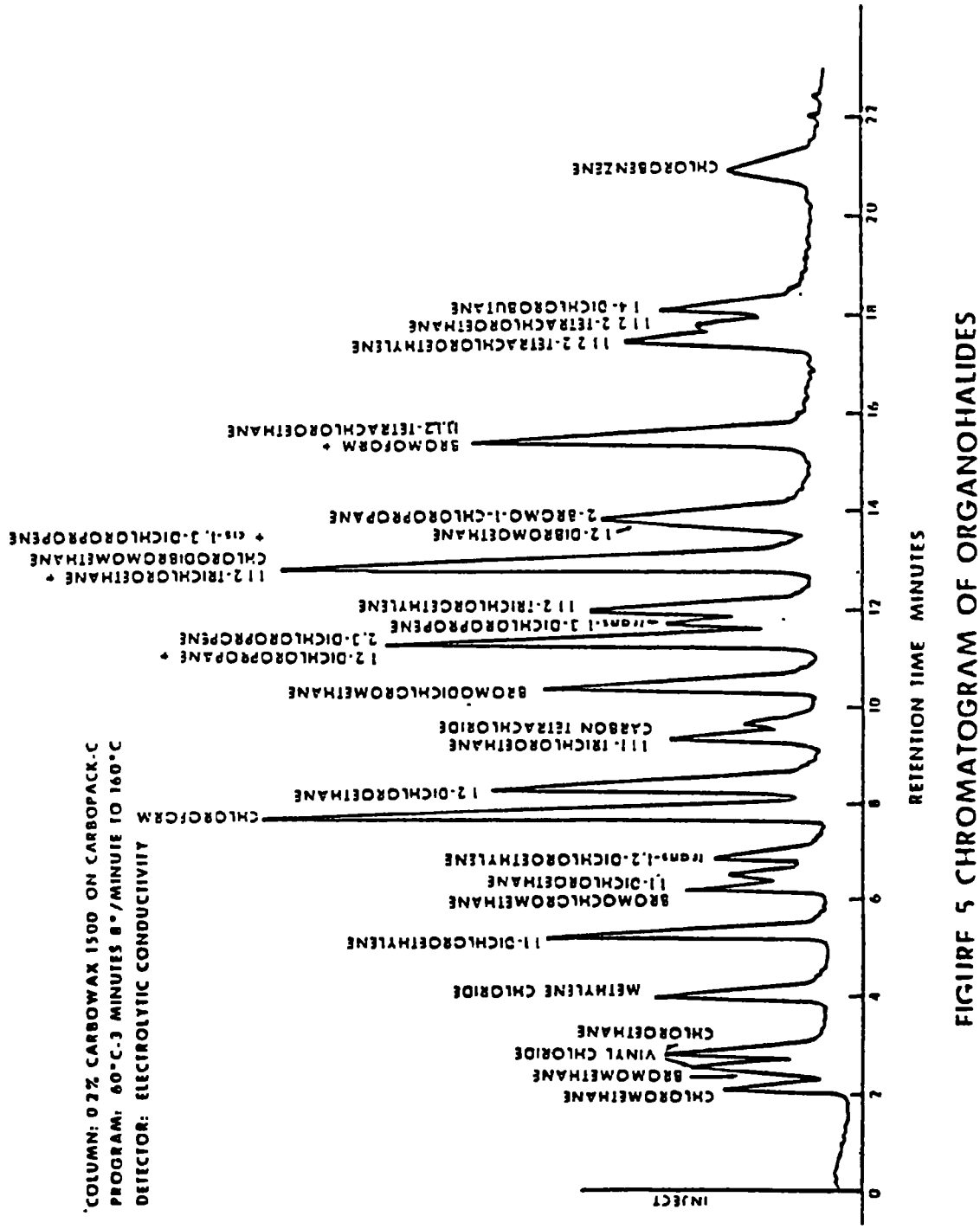


FIGURE 5 CHROMATOGRAM OF ORGANOHALIDES

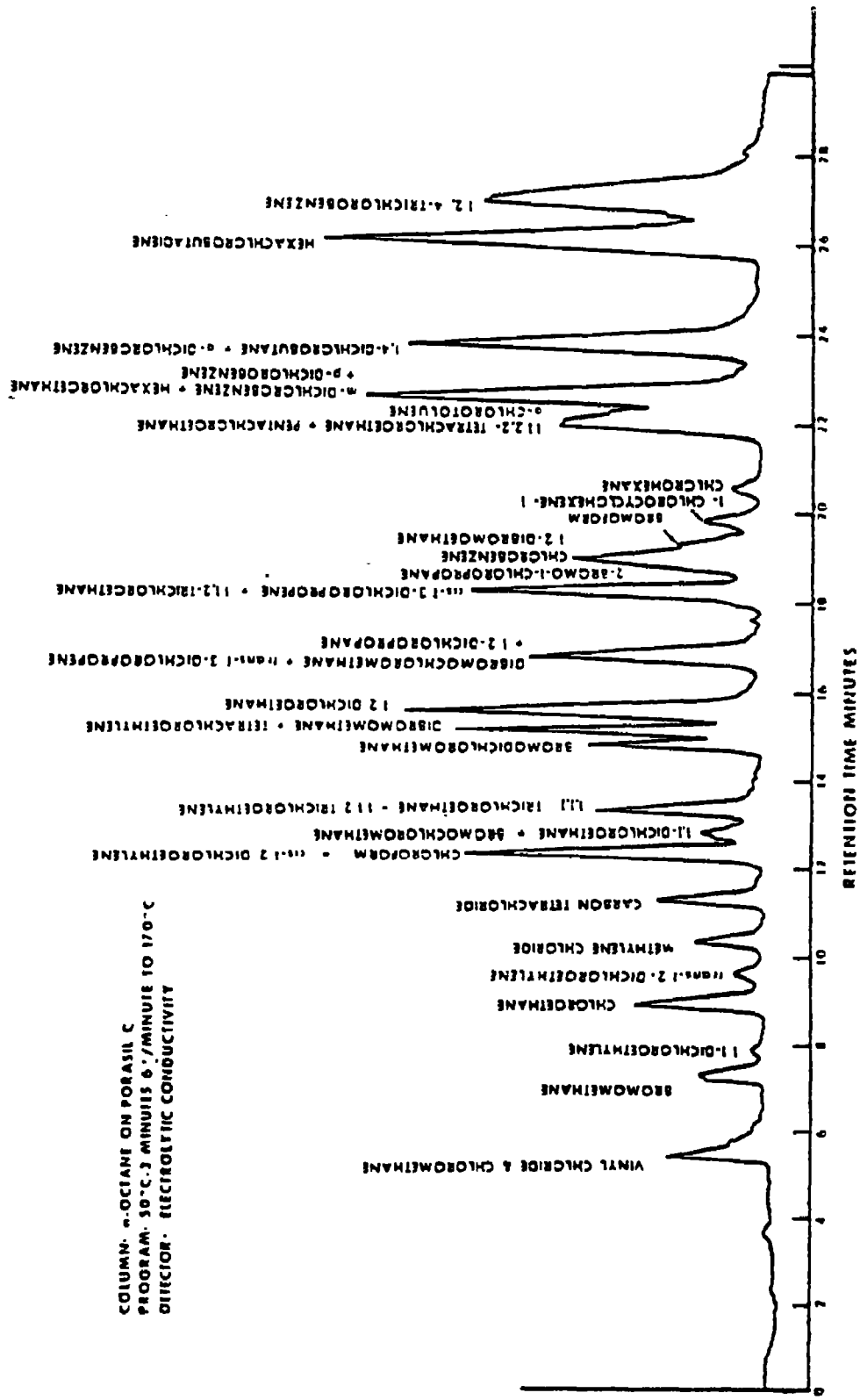


FIGURE 6 CHROMATOGRAM OF ORGANOHALIDES

BILLING CODE 6560-01-C

Part II: Analysis of Trihalomethanes in Drinking Water by Liquid/Liquid Extraction

1. *Scope.*

1.1 This method (1,2) is applicable only to the determination of four trihalomethanes, i.e., chloroform, bromodichloromethane, chlorodibromomethane, and bromoform in finished drinking water, drinking water during intermediate stages of treatment, and the raw source water.

1.2 For compounds other than the above-mentioned trihalomethanes, or for other sample sources, the analyst must demonstrate the usefulness of the method by collecting precision and accuracy data on actual samples as described in (3) and provide qualitative confirmation of results by Gas Chromatography/Mass Spectrometry (GC/MS) (4).

1.3 Qualitative analyses using GC/MS or the purge and trap method (5) must be performed to characterize each raw source water if peaks appear as interferences in the raw source analysis.

1.4 The method has been shown to be useful for the trihalomethanes over a concentration range from approximately 0.5 to 200 μg/l. Actual detection limits are highly dependent upon the characteristics of the gas chromatographic system used.

2. *Summary*

2.1 Ten milliliters of sample are extracted one time with 2 ml of solvent. Three μl of the extract are then injected into a gas chromatograph equipped with a linearized electron capture detector for separation and analysis.

2.2 The extraction and analysis time is 10 to 50 minutes per sample depending upon the analytical conditions chosen. (See Table 1 and Figures 1, 2, and 3.)

2.3 Confirmatory evidence is obtained using dissimilar columns and temperature programming. When component concentrations are sufficiently high (>50 μg/l), halogen specific detectors may be employed for improved specificity.

2.4 Unequivocal confirmatory analyses at high levels (>50 μg/l) can be performed using GC/MS in place of the electron capture detector. At levels below 50 μg/l, unequivocal confirmation can only be performed by the purge and trap technique using GC/MS (4, 5).

2.5 Standards dosed into organic free water and the samples are extracted and analyzed in an identical manner in order to compensate for possible extraction losses.

2.6 The concentration of each trihalomethane is summed and reported as total trihalomethanes in μg/l.

3. *Interferences*

3.1 Impurities contained in the extracting solvent usually account for the majority of the analytical problems. Solvent blanks should be analyzed before a new bottle of solvent is used to extract samples. Indirect daily checks on the extracting solvent are obtained by monitoring the sample blanks (6.4.10). Whenever an interference is noted in the sample blank, the analyst should reanalyze the extracting solvent. The extraction solvent should be discarded whenever a high level (>10 μg/l) of interfering compounds are traced to it. Low level interferences generally can be removed by distillation or column chromatography (6); however, it is generally more economical to obtain a new source of solvent or select one of the approved alternative solvents listed in Section 5.1. Interference free solvent is defined as a solvent containing less than 0.4 μg/l individual trihalomethane interference. Protect interference-free solvents by storing in a non-laboratory area known to be free of organochlorine solvents. *Subtracting blank values is not recommended.*

3.2 Several instances of accidental sample contamination have been attributed to diffusion of volatile organics through the septum seal on the sample bottle during shipment and storage. The sample blank (6.4.10) is used to monitor for this problem.

3.3 This liquid/liquid extraction technique efficiently extracts a wide boiling range of non-polar organic compounds and, in addition, extracts the polar organic components of the sample with varying efficiencies. In order to perform the trihalomethane analysis as rapidly as possible with sensitivities in the low μg/l range, it is necessary to use the semi-specific electron capture detector and chromatographic columns which have relatively poor resolving power. Because of these concessions, the probability of experiencing chromatographic interferences is high. Trihalomethanes are primarily products of the chlorination process and generally do not appear in the raw source water. The absence of peaks in the raw source water analysis with retention times similar to the trihalomethanes is generally adequate evidence of an interference-free finished drinking water analysis. Because of these possible interferences, in addition to each finished drinking water analysis, a representative raw source water (6.4.5) must be analyzed. When potential interferences are noted in the raw source water analysis, the alternate chromatographic columns must be used to reanalyze the sample set. If interferences are still noted, qualitative identifications should be performed according to Sections 2.3 and 2.4. If the peaks are confirmed to be other than trihalomethanes and add significantly to the total trihalomethane value in the finished drinking water analysis, then the sample set must be analyzed by the purge and trap method (5).

4. *Apparatus*

4.1 Extraction vessel—A 15 ml total volume glass vessel with a Teflon lined screw-cap is required to efficiently extract the samples.

4.1.1 For samples that do not form emulsions 10 ml screw-cap flasks with a Teflon faced septum (total volume is ml) are recommended. Flasks and caps—Pierce—#13310 or equivalent. Septa—Teflon silicone—Pierce #12718 or equivalent.

4.1.2 For samples that form emulsions (turbid source water) 15 ml screw cap centrifuge tubes with a Teflon cap liner are recommended. Centrifuge tube—Corning 8062-15 or equivalent.

4.2 Sampling containers—40 ml screw cap sealed with Teflon faced silicone septa. Vials and caps—Pierce #13075 or equivalent. Septa—Pierce #12722 or equivalent.

4.3 Micro syringes—10, 100 μl.

4.4 Micro syringe—25 μl with a 2-inch by 0.006-inch needle—Hamilton 702N or equivalent.

4.5 Syringes—10 ml glass hypodermic with luerlok tip (2 each).

4.6 Syringe valve—2-way with luer ends (2 each)—Hamilton #86570—1FM1 or equivalent.

4.7 Pipette—2.0 ml transfer.

4.8 Glass stoppered volumetric flasks—10 and 100 ml.

4.9 Gas chromatograph with linearized electron capture detector. (Recommended option—temperature programmable. See Section 4.12.)

4.10 Column A—4 mm ID x 2m long glass packed with 3% SP-1000 on Supelcoport (100/120 mesh) operated at 50°C with 60 ml/min flow. (See Figure 1 for a sample chromatogram and Table 1 for retention data.)

4.11 Column B—2 mm ID x 2m long glass packed with 10% squalane on Chromosorb WAW (80/100 mesh) operated at 67°C with 25 ml/min flow. This column is recommended as the primary analytical column. Trichloroethylene, a common raw source water contaminate, coelutes with bromodichloromethane. (See Figure 2 for a sample chromatogram and Table 1 for retention data.)

4.12 Column C—2 mm ID x 3m lor glass packed with 6% OV-11/4% SP-2100 on Supelcoport (100/120 mesh) temperature program 45°C for 12

minutes, then program at 1°/minute to °C with a 25 ml/min flow. (See Figure for a sample chromatogram and Table or retention data.)

4.13 Standard storage containers—15 ml amber screw-cap septum bottles with Teflon faced silicone septa. Bottles and caps—Pierce #19830 or equivalent. Septa—Pierce #12716 or equivalent.

5. Reagents

5.1 Extraction solvent—(See 3.1). Recommended—Pentane[a]. Alternative—hexane, methylcyclohexane or 2,2,4-trimethylpentane.

5.2 Methyl alcohol—ACS Reagent Grade.

5.3 Free and combined chlorine reducing agents—Sodium thiosulfate ACS Reagent Grade—sodium sulfite ACS Reagent Grade.

5.4 Activated carbon—Filtrasorb—200, available from Calgon Corporation, Pittsburgh, PA, or equivalent.

5.5 Standards.[b]

5.5.1 Bromoform 96%—available from Aldrich Chemical Company.

5.5.2 Bromodichloromethane 97%—available from Aldrich Chemical Company.

5.5.3 Chlorodibromomethane—available from Columbia Chemical, [illegible]orporated, Columbia, S.C.

5.5.4 Chloroform 99%—available [illegible]m Aldrich Chemical Company.

5.6 Organic-free water—Organic-free water is defined as water free of interference when employed in the procedure described herein.

5.6.1 Organic-free water is generated by passing tap water through a carbon filter bed containing carbon. Change the activated carbon whenever the concentration of any trihalomethane exceeds 0.4 μg/l.

5.6.2 A Millipore Super-Q Water System or its equivalent may be used to generate organic-free deionized water.

5.6.3 Organic-free water may also be prepared by boiling water for 15 minutes. Subsequently, while maintaining the temperature at 90° C, bubble a contaminant free inert gas through the water at 100 ml/minute for one hour. While still hot, transfer the water to a narrow mouth screw cap bottle with a Teflon seal.

5.6.4 Test organic free water each day it is used by analyzing it according to Section 7.

5.7 Standard stock solutions.

5.7.1 Fill a 10.0 ml ground glass stoppered volumetric flask with approximately 9.8 ml of methyl alcohol.

5.7.2 Allow the flask to stand unstoppered about 10 minutes or until all alcohol wetted surfaces dry.

5.7.3 Weigh the unstoppered flask to the nearest 0.1 mg.

5.7.4 Using a 100 μl syringe, immediately add 2 to 3 drops of the reference standard to the flask, then reweigh. *Be sure that the reference standard falls directly into the alcohol without contacting the neck of the flask.*

5.7.5 Dilute to volume, stopper, then mix by inverting the flask several times.

5.7.6 Transfer the standard solution to a dated and labeled 15 ml screw-cap bottle with a Teflon cap liner.

Note.—Because of the toxicity of trihalomethanes, it is necessary to prepare primary dilutions in a hood It is further recommended that a NIOSH/MESA-approved toxic gas respirator be used when the analyst handles high concentrations of such materials.

5.7.7 Calculate the concentration in micrograms per microliter from the net gain in weight.

5.7.8 Store the solution at 4° C.

Note.—All standard solutions prepared in methyl alcohol are stable up to 4 weeks when stored under these conditions. They should be discarded after that time has elapsed.

5.8 Aqueous calibration standard precautions.

5.8.1 In order to prepare accurate aqueous standard solutions, the following precautions must be observed:

a. Do not inject more than 20 μl of alcoholic standards into 100 ml of organic-free water.

b. Use a 25 μl Hamilton 702N microsyringe or equivalent. (Variations in needle geometry will adversely affect the ability to deliver reproducible volumes of methanolic standards into water.)

c. Rapidly inject the aloholic standard into the expanded area of the filled volumetric flask. Remove the needle as fast as possible after injection.

d. Mix aqueous standards by inverting the flask three times only.

e. Discard the contents contained in the neck of the flask. Fill the sample syringe from the standard solution contained in the expanded area of the flask as directed in Section 7.

f. Never use pipets to dilute or transfer samples and aqueous standards.

g. Aqueous standards, when stored with a headspace, are not stable and should be discarded after one hour. Aqueous standards can be stored according to Sections 6.4.9 and 7.2.

5.9 Calibration standards.

5.9.1 Prepare, from the standard stock solutions, a multicomponent secondary dilution mixture in methyl alcohol so that a 20 μl injection into 100 ml of organic-free water will generate a calibration standard which produces a response close (± 25%) to that of the unknown. (See 8.1.)

5.9.2 Alternative calibration procedure.

5.9.2.1 Construct a calibration curve for each trihalomethane containing a minimum of 3 different concentrations. Two of the concentrations must bracket each unknown.

5.9.3 Extract and analyze the aqueous calibration standards in the same manner as the unknowns.

5.9.4 Other calibration procedures (7) which require the delivery of less than 20 μl of methanolic standards to 10.0 ml volumes of water contained in the sample syringe are acceptable only if the methanolic standard is delivered by the solvent flush technique (8).

5.10 Quality Check Standard Mixture.

5.10.1 Prepare, from the standard stock solutions, a secondary dilution mixture in methyl alcohol that contains 10.0 ng/μl of each compound. (See 5.7.6 and 5.7.8.)

5.10.2 Daily, prepare and analyze a 2.0 μg/l aqueous dilution from this mixture by dosing 20.0 μl into 100 ml of organic-free water (See Section 8.1).

6. Sample Collection and Handling.

6.1 The sample containers should have a total volume of at least 25 ml.

6.1.1 Narrow-mouth screw-cap bottles with the TFE fluorocarbon faced silicone septa cap liners are strongly recommended.

6.2 Glassware Preparation.

6.2.1 Wash all sample bottles, TFE seals, and extraction flasks in detergent. Rinse with tap water and finally with distilled water.

6.2.2 Allow the bottles and seals to air dry, then place in an 105° C oven for 1 hour, then allow to cool in an area known to be free of organics.

Note.—Do not heat the TFE seals for extended periods of time (>1 hour) because the silicone layer slowly degrades at 105° C

6.2.3 When cool, seal the bottles using the TFE seals that will be used for sealing the samples.

6.3 Sample stabilization—A chemical reducing agent (Section 5.3) is added to all samples in order to arrest the formation of additional

---

[a] Pentane has been selected as the best solvent for this analysis because it elutes, on all of the columns, well before any of the trihalomethanes. High altitudes or laboratory temperatures in excess of 75°F may make the use of this solvent impractical For these reasons, alternative solvents are acceptable, however, the analyst may experience baseline variances in the elution areas of the trihalomethanes due to coelution of these solvents The degree of difficulty appears to be dependent upon the design and condition of the [illegible]tron capture detector Such problems should be [illegible]nificant when concentrations of the coeluting [illegible]lomethane are in excess of 5 μg/L

[b] As a precautionary measure, all standards must [illegible] checked for purity by boiling point determinations or GC/MS assays

trihalomethanes after sample collection (7,9) and to eliminate the possibility of free chlorine reacting with impurities in the extraction solvent to form interfering organohalides. *DO NOT ADD THE REDUCING AGENT TO SAMPLES AT COLLECTION TIME WHEN DATA FOR MAXIMUM TRIHALOMETHANE FORMATION IS DESIRED.* If chemical stabilization is employed, then the reagent is also added to the blanks. The chemical agent (2.5 to 3 mg/40 ml) is added in crystalline form to the empty sample bottle just prior to shipping to the sampling site. If chemical stabilization is not employed at sampling time then the reducing agent is added just before extraction.

6.4 Sample Collection.

6.4.1 Collect all samples in duplicate.

6.4.2 Fill the sample bottles in such a manner that no air bubbles pass through the sample as the bottle is filled.

6.4.3 Seal the bottle so that no air bubbles are entrapped in it.

6.4.4 Maintain the hermetic seal on the sample bottle until analysis.

6.4.5 The raw source water sample history should resemble the finished drinking water. The average retention time of the finished drinking water within the water plant should be taken into account when sampling the raw source water.

6.4.6 Sampling from a water tap.

6.4.6.1 Turn on the water and allow the system to flush until the temperature of the water has stabilized. Adjust the flow to about 500 ml/minute and collect duplicate samples from the flowing stream.

6.4.7 Sampling from an open body of water.

6.4.7.1 Fill a 1-quart wide-mouth bottle with sample from a representative area. Carefully fill duplicate sample bottles from the 1-quart bottle as in 6.4.

6.4.8 If a chemical reducing agent has been added to the sample bottles, fill with sample just to overflowing, seal the bottle, and shake vigorously for 1 minute.

6.4.9 Sealing practice for septum seal screw cap bottles.

6.4.9.1 Open the bottle and fill to overflowing. Place on a level surface. Position the TFE side of the septum seal upon the convex sample meniscus and seal the bottle by screwing the cap on tightly.

6.4.9.2 Invert the sample and lightly tap the cap on a solid surface. The absence of entrapped air indicates a successful seal. If bubbles are present, open the bottle, add a few additional drops of sample, then reseal bottle as above.

6.4.10 Sample blanks.

6.4.10.1 Prepare blanks in duplicate at the laboratory by filling and sealing sample bottles with organic-free water just prior to shipping the sample bottles to the sampling site.

6.4.10.2 If the sample is to be stabilized, add an identical amount of reducing agent to the blanks.

6.4.10.3 Ship the blanks to and from the sampling site along with the sample bottles.

6.4.10.4 Store the blanks and the samples, collected at a given site (sample set), together in a protected area known to be free from contamination. A sample set is defined as all the samples collected at a given site (i.e., at a water treatment plant, duplicate raw source water, duplicate finished water and the duplicate sample blanks comprise the sample set).

6.5 When samples are collected and stored under these conditions, no measurable loss of trihalomethanes has been detected over extended periods of time (7). It is recommended that the samples be analyzed within 14 days of collection.

7. *Extraction and Analysis.*

7.1 Remove the plungers from two 10-ml syringes and attach a closed syringe valve to each.

7.2 Open the sample bottle [c] (or standard) and carefully pour the sample into one of the syringe barrels until it overflows. Replace the plunger and compress the sample. Open the syringe valve and vent any residue air while adjusting the sample volume to 10.0 ml. Close the valve.

7.3 Fill the second syringe in an identical manner from the same sample bottle. This syringe is reserved for a replicate analysis (see 8.3 and 8.4).

7.4 Pipette 2.0 ml of extraction solvent into a clean extraction flask.

7.5 Carefully inject the contents of the syringe into the extraction flask.

7.6 Seal with a Teflon faced septum.

7.7 Shake vigorously for 1 minute.

7.8 Let stand until the phases separate ($\int$60 seconds).

7.8.1 If the phases do not separate on standing then centrifugation can be used to facilitate separation.

7.9 Analyze the sample by injecting 3.0 µl (solvent flush technique, (8)) of the upper (organic) phase into the gas chromatograph.

8. *Analytical Quality Control.*

8.1 A 2 µg/l quality check standard (See 5.10) should be extracted and analyzed each day before any samples are analyzed. Instrument status checks and lower limit of detection estimations based upon response factor calculations at 5 times the noise level are obtained from these data. In addition, the data obtained from the quality check standard can be used to estimate the concentration of the unknowns. From this information the appropriate standards can be determined.

8.2 Analyze the sample blank and the raw source water to monitor for potential interferences as described in Sections 3.1, 3.2, and 3.3.

8.3 Spiked samples.

8.3.1 For those laboratories analyzing more than 10 samples a day, each 10th sample analyzed should be a laboratory-generated spike which closely duplicates the average finished drinking water in trihalomethane composition and concentration. Prepare the spiked sample in organic-free water as described in section 5.9.

8.3.2 In those laboratories analyzing less than 10 samples daily, each time the analysis is performed, analyze at least one laboratory generated spike sample which closely duplicates the average finished drinking water in trihalomethane composition and concentration. Prepare the spiked sample in organic-free water as described in section 5.9.

8.3.3 Maintain an up-to-date log on the accuracy and precision data collected in Sections 8.3 and 8.4. If results are significantly different than those cited in Section 10.1, the analyst should check out the entire analysis scheme to determine why the laboratory's precision and accuracy limits are greater.

8.4 Randomly select and analyze 10% of all samples in duplicate.

8.5 Analyze all samples in duplicate which appear to deviate more than 30% from any established norm.

8.6 Quarterly, spike an EMSL-Cincinnati trihalomethane quality control sample into organic-free water and analyze.

8.6.1 The results of the EMSL trihalomethane quality control sample should agree within 20% of the true value for each trihalomethane. If they do not, the analyst must check each step in the standard generation procedure to solve the problem.

8.7 It is important that the analyst be aware of the linear response characteristics of the electron capture system that is utilized. Calibration curves should be generated and rechecked quarterly for each trihalomethane over the concentration range encountered in the samples in order to confirm the linear response range of the system. Quantitative data cannot be calculated from non-linear

[c] If for any reason the chemical reducing agent has not been added to the sample, then it must be added just prior to analyses at the rate of 2.5 to 3 mg/40 ml or by adding 1 mg directly to the sample in the extraction flask.

responses. Whenever non-linear responses are noted, the analyst must dilute the sample for reanalysis.

8.8 Maintain a record of the retention times for each trihalomethane using data gathered from spiked samples and standards.

8.8.1 Daily calculate the average retention time for each trihalomethane and the variance encountered for the analyses.

8.8.2 If individual trihalomethane retention time varies by more than 10% over an eight hour period or does not fall within 10% of an established norm, the system is "out of control." The source of retention data variation must be corrected before acceptable data can be generated.

9. *Calculations*

9.1 Locate each trihalomethane in the sample chromatogram by comparing the retention time of the suspect peak to the data gathered in 8.8.1. The retention time of the suspect peak must fall within the limits established in 8.8.1 for a single column identification.

9.2 Calculate the concentration of each trihalomethane by comparing the peak heights or peak areas of the samples to those of the standards. Round off the data to the nearest μg/l or two significant Figures.

Concentration, μg/l = sample peak height/standard peak height × standard concentration, μg/l.

9.3 Calculate the total trihalomethane concentration (TTHM) by summing the 4 individual trihalomethane concentrations in μg/l: TTHM (μg/l) = (conc. $CHCl_3$) + (conc. $CHBrCl_2$) + (conc. $CHBr_2Cl$) + (conc. $CHBr_3$)

9.4 Calculate the limit of detection (LOD) for each trihalomethane not detected using the following criteria:

$$\text{LOD } (\mu g/l) = \left(\frac{(AXATT)}{(BXATT)}\right) \times (2\ \mu g/l)$$

Where

B = peak height (mm) of 2 μg/l quality check standard

A = 5 times the noise level in mm at the exact retention time of the trihalomethane or the base line displacement in mm from theoretical zero at the exact retention time for the trihalomethane.

ATT = attenuation factor.

9.5 Report the results obtained from the lower limit of detection estimates along with the data for the samples.

10. *Precision and Accuracy*

10.1 Single lab precision and accuracy. The data in Table II were generated by spiking organic-free water with trihalomethanes as described in 5.9. The mixtures were analyzed by the analyst as true unknowns.

Table I.—*Retention Times for Trihalomethanes*

Trihalomethane	Retention time minutes		
	Column A	Column B	Column C
Chloroform	1.0	1.3	4.9
Bromodichloromethane	1.5	[a]2.5	11.0
Chlorodibromomethane (Dibromochloromethane)	2.6	5.6	23.1
bromoform	5.5	10.9	39.4

[a] On this column, trichloroethylene a common raw source water contaminate, coelutes with bromodichloromethane

Table II.—*Single Laboratory Accuracy and Precision*

Compound	Dose level μg/l	Number of samples	Mean μg/l	Precision relative standard deviation, percent	Accuracy percent recovery
$CHCl_3$	9.1	5	10	11	110
$CHCl_3$	69	3	73	5.3	106
$CHBrCl_2$	1.2	5	1.3	9.8	108
$CHBrCl_2$	12	2	15	1.4	125
$CHBr_2Cl$	2.7	5	2.0	17	74
$CHBr_2Cl$	17	3	16	9.9	94
$CHBr_3$	2.9	5	2.2	10	76
$CHBr_3$	14	3	16	12	114

References

1. Mieure, J. P., "A Rapid and Sensitive Method for Determining Volatile Organohalides in Water." *Journal AWWA, 69*, 60, 1977.

2 Reding, R., et al. "THM's in Drinking Water Analysis by LLE and Comparison to Purge and Trap". Organics Analysis in Water and Wastewater, STP 686 ASTM, 1979.

3 "Handbook for Analytical Quality Control in Water and Waste water Laboratories," Analytical Quality Control Laboratory, National Environmental Research Center, Cincinnati, Ohio, June 1972.

4. Budde, W. L., J. W. Eichelberger, "Organic Analysis Using Gas Chromatography-Mass Spectrometry," Ann Arbor Science, Ann Arbor, Michigan, 1979.

5. "The Analysis of Trihalomethanes in Finished Water by the Purge and Trap Method," Environmental Monitoring and Support Laboratory, Environmental Research Center, Cincinnati, Ohio, 45268, May 15, 1979.

6 Richard J. J.; G. A. Junk, "Liquid Extraction for Rapid Determination of Halomethanes in Water, *Journal AWWA, 69* 62, January 1977.

7 Brass, H. J., et al., "National Organic Monitoring Survey: Sampling and Purgeable Organic Compounds, Drinking Water Quality Through Source Protection," R. B. Pojasek, Editor, Ann Arbor Science, p. 398, 1977.

8. White, L. D., et al. "Convenient Optimized Method for the Analysis of Selected Solvent Vapors in Industrial Atmosphere," AIHA Journal, Vol 31, p. 225, 1970.

9 Kopfler, F. C., et al "GC/MS Determination of Volatiles for the National Organics Reconnaissance Survey (NORS) or Drinking Water, Identification and Analysis of Organic Pollutants in Water," L. H Keith, Editor, Ann Arbor Science, p. 87, 1976.

BILLING CODE 6560-01-M

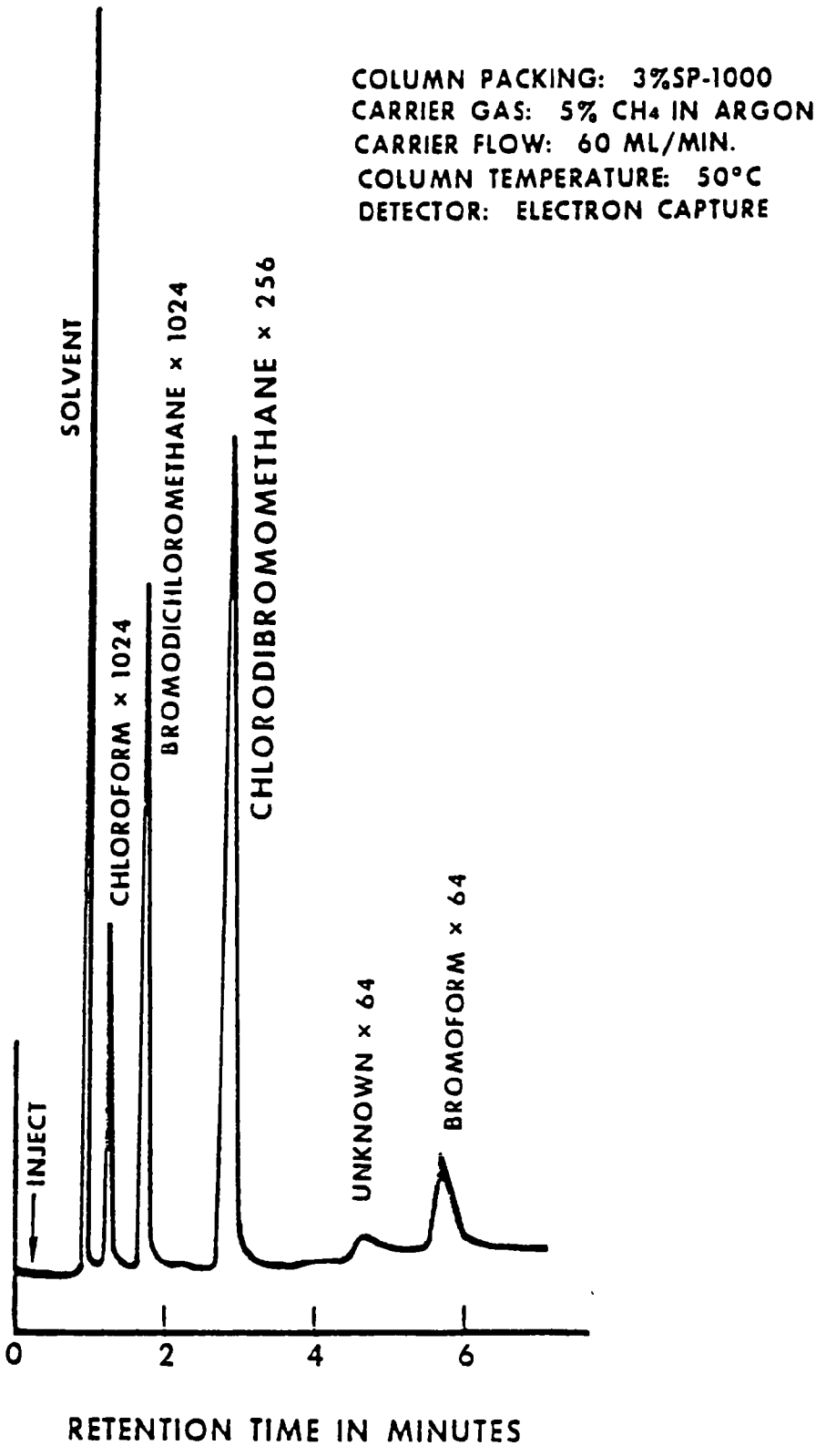


FIGURE 1. FINISHED WATER EXTRACT

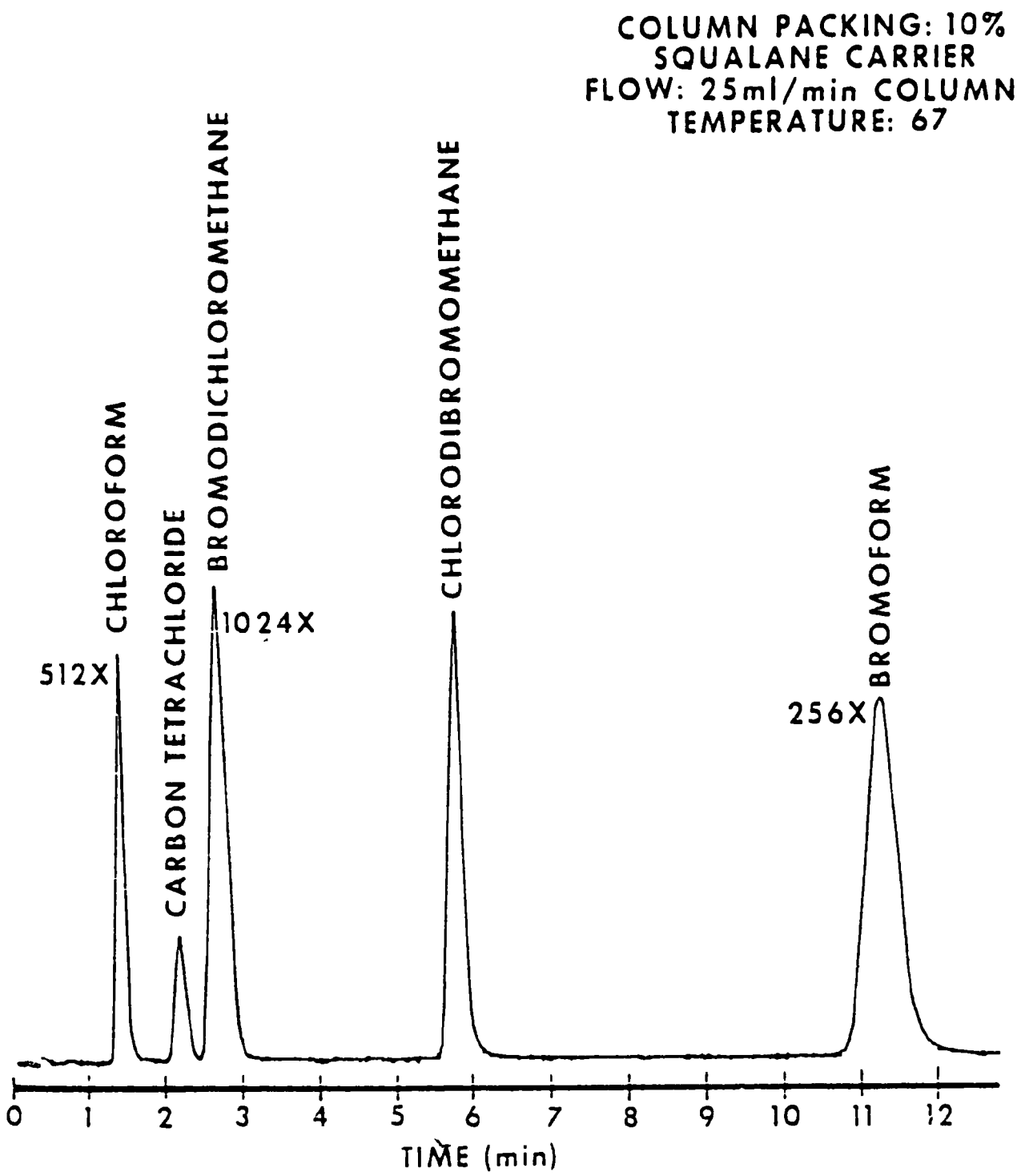


FIGURE 2. EXTRACT OF STANDARD

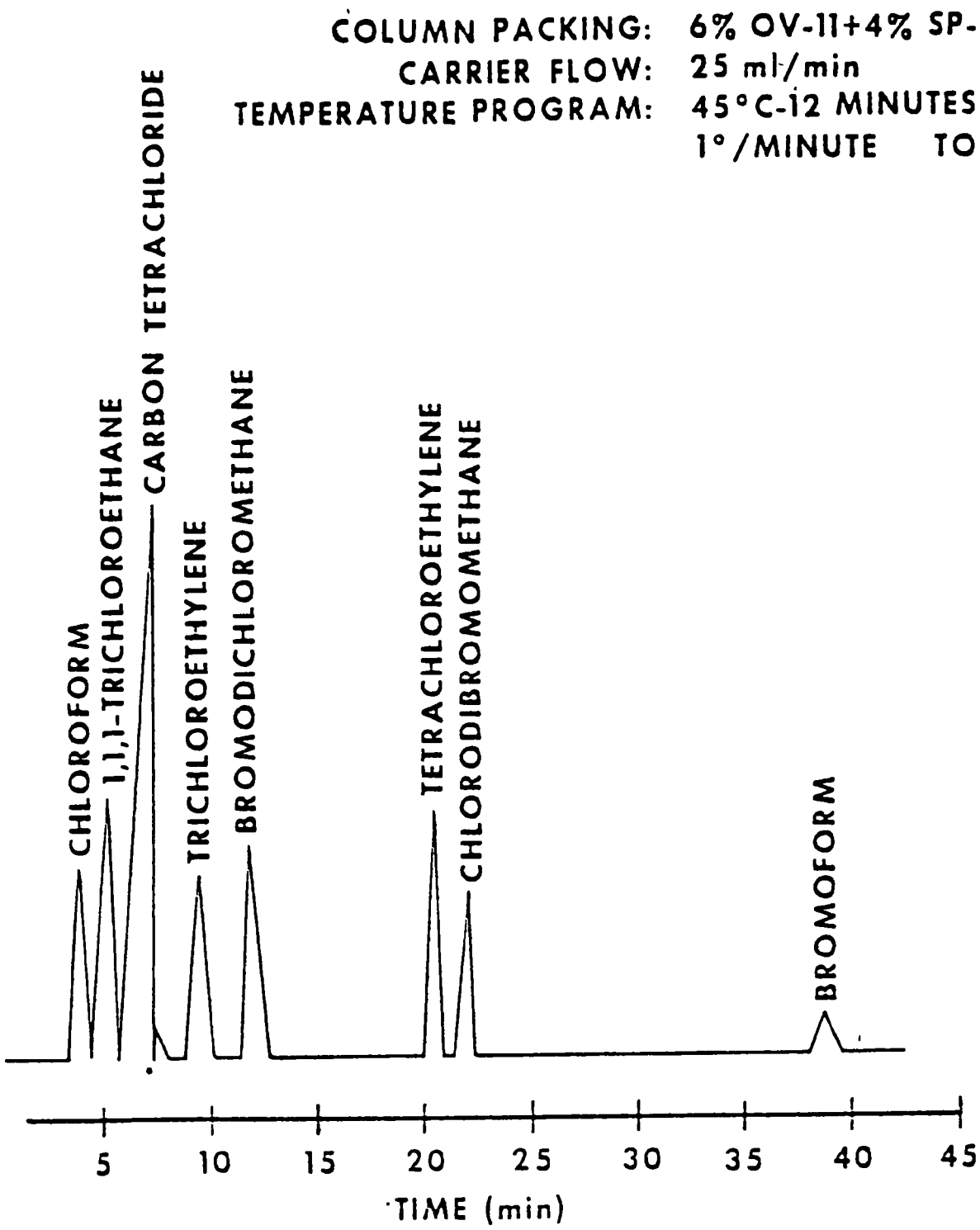


FIGURE 3. EXTRACT OF STANDARD

BILLING CODE 6560-01-C

Part III—Determination of Maximum Total Trihalomethane Potential (MTP)

The water sample used for this determination is taken from a point in the distribution system that reflects maximum residence time. Procedures for sample collection and handling are given in EMSL Methods 501.1 and 501.2. No reducing agent is added to "quench" the chemical reaction producing THMs at the time of sample collection. The intent is to permit the level of THM precursors to be depleted and the concentration of the THMs to be maximized for the supply being tested.

Four experimental parameters affecting maximum THM production are pH, temperature, reaction time and the presence of a disinfectant residual. These parameters are dealt with as follows:

Measure the disinfectant residual at the selected sampling point. Proceed only if a measurable disinfectant residual is present. Collect triplicate 40 ml water samples at the pH prevailing at the time of sampling, and prepare a method blank according to the EMSL methods. Seal and store these samples together for 7 days at 25°C or above. After this time period, open one of the sample containers and check for disinfectant residual. Absence of a disinfectant residual invalidates the sample for further analyses. Once a disinfectant residual has been demonstrated, open another of the sealed samples and determine total THM concentration using either of the EMSL analytical methods.

Attachment 7.—Statement of Basis and Purpose for an Amendment to the National Interim Primary Drinking Water Regulations on Trihalomethanes, August 1979

Office of Drinking Water Criteria and Standards Division, Environmental Protection Agency, Washington, D.C. 20460.

Table of Contents

I. Summary

The trihalomethanes (THMs) are a family of organic compounds, named as derivatives of methane, where three of the four hydrogen atoms are substituted by a halogen atom. Although halogens can include fluorine, chlorine, bromine and iodine, only chlorine and bromine substituents are now considered for the purpose of this regulation. THMs in drinking water are produced by the action of the chlorine added for disinfection or oxidation, with the naturally occurring organic precursors (e.g., humic or fulvic acids) commonly found in source waters.

THMs are commonly found in drinking water supplies throughout the United States. Chloroform has been found at concentrations ranging from 0.001–0.540 mg/l and (TTHM) potential concentrations as high as 0.784 mg/l have been detected. The concentrations of TTHM increase when raw water supplies are treated with chlorine for disinfection and other purposes. TTHM concentrations are indicative of the presence of other halogenated and oxidized organic chemicals that are produced in water during chlorination.

People are also exposed to chloroform in the air they breathe and the food they eat. Analyses of the relative contribution of chloroform in drinking water, air and food exposures assumed various levels of exposure based on monitoring studies. Drinking water may contribute from zero to more than 90% of the total body burden.

Chloroform has been shown to be rapidly absorbed on oral and intraperitoneal administration and subsequently metabolized to carbon dioxide, chloride ion, phosgene, and other unidentified metabolites. The metabolic profile of chloroform in animal species such as mice, rats, and monkeys is indicated in Table 4 and is qualitatively similar to that in man.

Mammalian responses to chloroform exposure include: central nervous system depression, hepatotoxicity, nephrotoxicity, teratogenicity, and carcinogenicity. These responses are discernible in mammals after oral and inhalation exposures to high levels of chloroform ranging from 30–350 mg/kg; the intensity of response is dependent upon the dose. Although less toxicological information is available for the brominated THMs, mutagenicity and carcinogenicity have been detected in some test systems. Physiological chemical activity should be greater for the brominated THMs than for chloroform.

Although short-term toxic responses to THMs in drinking water are not documented, the potential effects of chronic exposures to THMs should be a matter of concern. Prolonged administration of chloroform at relatively high dose levels (100–138 mg/kg) to rats and mice, manifested oncogenic effects. Oncogenic effects were not observed at the lowest dose level (17 mg/kg) in three experiments. Since methods do not now exist to establish a threshold no effect level of exposure to carcinogens, the preceding data do not imply that a "safe" level of exposure can be established for humans.

Human epidemiological evidence is inconclusive, although positive correlations with some sites have been found in several studies. There have been 18 retrospective studies shown in Table 7 that have investigated some aspect of a relationship between cancer mortality or morbidity and drinking water variables. Due to various limitations in the epidemiological methods, in the water quality data, and problems with the individual studies, the present evidence cannot lead to a firm conclusion that there is an association between contaminants in drinking water and cancer mortality/morbidity. Causal relationships cannot be proven on the basis of results from epidemiological studies. The evidence from these studies thus far is incomplete and the trends and patterns of association have not been fully developed. When viewed collectively, however, the epidemiological studies provide sufficient evidence for maintaining the hypotheses that there may be a potential health risk, and that the positive correlations may be reflecting a causal association between constituents of drinking water and cancer mortality.

Preliminary risk assessments made by the Science Advisory Board (SAB), the National Academy of Sciences (NAS), Tardiff, and EPA's Carcinogen Assessment Group (CAG) using different models have estimated the incremental risks associated with the exposure from chloroform in drinking water. The exposure to THMs from air and food have not been included in these computations. The risk estimates associated with the MCL at the 0.10 mg/l level are essentially the same from the NAS and CAG computations (3.4×10^{-4} and 4×10^{-4}) assuming two liters of water at 0.10 mg/l chloroform consumed daily for 70 years.

On the basis of the available toxicological data summarized in the following report, chloroform has been shown to be a carcinogen in rodents

(mice and rats) at high dose levels. Since its metabolic pattern in animals is qualitatively similar to that in man, it should be suspected of being a human carcinogen. Epidemiological studies also suggest a human risk. Therefore, because a potential human health risk does exist, levels of chloroform in drinking water should be reduced as much as is technologically and economically feasible using methods that will not compromise protection from waterborne infectious disease transmission.

Although documentation of their toxicity is not so well established, other THMs should be suspected of posing similar risks. Because the treatment process that can reduce drinking water levels of chloroform have about the same effectiveness in reducing levels of the other THMs, the proposed regulation is addressed to these substances, as well.

II. Introduction

The extent and significance of organic chemical contamination of drinking water or drinking water sources first came to public attention in 1972, when a report, "Industrial Pollution of the Lower Mississippi River in Louisiana" was published (EPA, 1972). While this report did not include quantification of the pollutants found, and was directed toward locating industrial discharges responsible for the pollution, the report did include analyses of finished (treated) drinking water and provided evidence of the presence of THMs. Subsequently, a more thorough examination of finished drinking water in the New Orleans area was carried out, using the most sophisticated analytical methods available (EPA, 1974). This latter study confirmed the presence of THMs and many other organic chemicals in finished drinking water, and furthermore it demonstrated that one of them, chloroform, was present in high relative concentrations.

The findings in New Orleans promoted other studies, primarily for the purpose of determining how widespread and serious the organic chemical contamination of drinking water was. Impetus was added by the passage of the Safe Drinking Water Act (Pub. L. 93-523), which directed the EPA to conduct a comprehensive study of public water supplies and drinking water sources to determine the nature, extent, sources, and means of control of contamination by substances suspected of being carcinogenic. The National Organics Reconnaissance Survey of Halogenated Organics (NORS) (Symons, et al, 1975), or "80 City Study", was aimed primarily at determining the extent of the presence of four THMs, *chloroform, bromodichloromethane, dibromochloromethane* and *bromoform*, along with carbon tetrachloride and 1,2-dichloroethane, and at determining what effect raw water source and water treatment practices had on the formation of these compounds (Table 1). The presence of THMs in finished drinking water was confirmed, and some trend relating non-volatile total organic carbon (NVTOC) of the raw water and the total trihalomethane (TTHM) was postulated. Chloroform occurred invariably in water which had been chlorinated, while it was absent or present at much lower concentrations in the raw water. Water samples were collected at the treatment plant in winter and iced for shipment but not dechlorinated. Thus, those values might approximate minima for human exposure in the areas selected. Of the various THMs, chloroform was found at the highest concentrations (averaging approximately 75 percent of the TTHM), with progressively less bromodichloromethane, dibromochloromethane and bromoform being detected. In some cases chloroform was found at concentrations greater than 0.300 mg/l; (the highest value found was 0.540 mg/l). Carbon tetrachloride and 1,2-dichloroethane were found at very low concentrations The concentration of these two components did not increase after chlorination; therefore, it can be assumed that these compounds are not related to the chlorination process.

BILLING CODE 6560-01-M

TABLE I - Analytical results of chloroform, bromoform, bromodichloromethane, and dibromochloromethane and total trihalomethanes in water supplies from NORS and NOMS

(Concentrations in milligrams per liter)

	NORS	NOMS			
		Phase I	Phase II	Phase III	
Chloroform				Dechlorinated	Terminal
Median	0.021	0.027	0.059	0.022	0.044
Mean	-	0.043	0.083	0.035	0.069
Range	NF-0.311	NF-0.271	NF-0.47	NF-0.20	NF-0.540
Bromoform					
Median	0.005	LD	LD	LD	LD
Mean	-	0.003	0.004	0.002	0.004
Range	NF-0.092	NF-0.039	NF-0.280	NF-0.137	NF-0.190
Dibromochloromethane					
Median	0.001	LD	0.004	0.002	0.003
Mean	-	0.008	0.012	0.006	0.011
Range	NF-0.100	NF-0.19	NF-0.290	NF-0.114	NF-0.250
Bromodichloromethane					
Median	0.006	0.010	0.014	0.006	0.011
Mean	-	0.018	0.018	0.009	0.017
Range	NF-0.116	NF-0.183	NF-0.180	NF-0.072	NF-0.125
Total Trihalomethanes					
Median	0.027	0.045	0.087	0.037	0.074
Mean	0.067	0.068	0.117	0.053	0.100
Range	NF-0.482	NF-0.457	NF-0.784	NF-0.295	NF-0.695

NF = not found
LD = less than detection limit

BILLING CODE 6560-01-C

A Joint Federal/State Survey of Organics and Inorganics in 83 Selected Drinking Water Supplies, carried out by EPA's Region V (Chicago) provided additional evidence of the ubiquitous nature of chloroform and other THMs in chlorinated drinking water (EPA, 1975). Two conclusions reached in that study were that raw water relatively free of organic matter results in finished water that is relatively free of chloroform and related halogenated compounds, and that there is a correlation in some instances between the concentrations of chloroform, bromodichloromethane, dibromochloromethane and bromoform in finished water and the amount of organic matter found in raw water.

The National Organics Monitoring Survey (NOMS), directed by § 141.40 of the National Interim Primary Drinking Water Regulations (40 FR 59574, December 24, 1975), was aimed not only at determining the presence of THMs in additional water supplies, but also at determining the seasonal variations in concentration of these substances.

The NOMS sampling included 113 public water systems designated by the Administrator, and also included analyses for approximately 20 specific synthetic organic chemicals deemed to be candidates of particular concern as well as analyses of several surrogate group chemical parameters which are indicators of the total amount of organic contamination. Three phases of this study were completed and the mean, minimum, and maximum values of chloroform and THMs in drinking water are reported in Table 1. Phase I analyses in the NOMS were conducted similarly to the NORS. Phase II analyses were performed after the THM-producing reactions were allowed to run to completion. Phase III analyses were conducted on both declorinated samples and on samples that were allowed to run to completion (terminal). Again chloroform was found at the highest concentrations in most cases, however, in a few cases bromoform was found to be the highest concentration of the THMs (0.280mg/l). The mean concentrations of chloroform were 0.043 mg/l, 0.083 mg/l, 0.035 mg/l, and 0.069 mg/l for Phase I, II, III (dechlorinated) and III (terminal), respectively; the mean concentrations for TTHMs were 0.068 mg/l, 0.117mg/l, 0.053 mg/l and 0.100 mg/l for Phase I, II, III (dechlorinated) and III (terminal), respectively.

III. The Role of Chlorine and Other Disinfectants

All available evidence indicates that chlorination of drinking water containing naturally occurring organic chemicals is the major factor in the formation of halogenated organic chemicals, particularly the THMs in finished drinking water. Chlorinated organic compounds, however, can also be introduced into drinking water from industrial outfalls, urban and rural runoff, rainfall, through polluted air, or from the chlorination in sewage and industrial wastewater.

Several studies in addition to those mentioned above, have demonstrated increased THM concentrations in drinking water. Work by J. J. Rook (1974) in the Netherlands, and the studies by Bellar, Lichtenberg and Kroner (1974), showed that chloroform and other halogenated methanes are formed during the water chlorination process. It should be noted that these findings came as a result of the development and application of more sensitive and refined analytical techniques. Recent work by Rook (1974, 1977) has provided some insight into the organic precursors which might be responsible for the formation of the THMs. Studies by Sontheimer and Kuhn (1977) indicate that the THMs may represent only a portion of the total halogenated products of chlorination of water. Bunn et al. (1975), have demonstrated that hypochlorite in the presence of bromide and iodide ions but not fluoride will react with natural organic matter to produce all ten possible trihalogenated methanes.

It can be concluded from the above studies and others that the THMs occur in chlorinated drinking waters, and that the concentrations of the various THMs are dependent on the type and quality of organic precursor substances, the amount of chlorine used, and the presence of other halogen ions as well as contact time, temperature and pH.

A number of methods are available for reducing levels of THMs in drinking water. These options include modifications of current treatment practices, such as moving the point of chlorination, the use of alternative disinfectants such as chlorine dioxide, chloramines, or ozone, and various methods that will reduce organic precursor concentrations such as use of adsorbents like granular activated carbon (GAC).

Two chemicals often mentioned as alternative disinfectants, chlorine dioxide and ozone, are both well known as effective disinfectants and chemical oxidents, and some history of their practical use in water treatment has been accumulated particularly in Europe, but also in the United States.

Chlorine dioxide is usually prepared at the water plant by the reaction of chlorine (either as gas or as sodium hypochlorite) with sodium chlorite. Unless an excess of chlorine is used, there will be unreacted sodium chlorite left over from the reaction. When chlorine dioxide reacts with organic matter in the water, one of the reaction products is the chlorite ion. Thus, whenever chlorine dioxide is used to treat water, the presence of chlorite ion in the treated water can be expected.

EPA is studying the health effects of chlorine dioxide in water, utilizing several animal species as well as human volunteers. Studies of the toxicology of chlorine dioxide and chlorite ion in drinking water reveal considerable variations. These compounds have been reported to affect the hematopoietic systems such as oxidative changes in hemoglobins and hemolysis of red blood cells. Other bioeffects observed include gastrointestinal disturbances. The preliminary results indicate species variability in biological manifestations. Cats and African green monkeys appear to lie at the extreme ends of the spectrum from among the species studied; cats are very sensitive to hematopoietic effects whereas monkeys were apparently insensitive even at levels as high as 400 mg/l (Bull, 1979). An upper limit for chlorine dioxide by-product exposure is being considered primarily because of the lack of data concerning the safety of this material, and particularly its decomposition products, at higher concentrations (Musil et al., 1963 and Fridyland and Kagan, 1971. Studies with cats have shown that chlorite, which is oxidant that can cause anemias, has a deleterious effect on red blood cell survival rate at chlorine dioxide concentrations above 10 mg/l. Preliminary studies in a small human population did not demonstrate substantial blood chemistry changes, except possibly in one person known to be deficient in glucose-6-phosphotase dehydrogenase. Lack of sufficient health effects data on human toxicity for ClO_2 and its by-products prevents establishment of an MCL at this time, however, work in progress is expected to provide much additional information within the coming year. In the meantime, EPA recommends that monitoring be conducted when chlorine dioxide is used, and that residual oxidant should not exceed 0.5 mg/l as ClO_2.

A preliminary study concerning ozonation of 29 organic compounds potentially present in water supply sources indicated the formation of a number of products (Cotruvo, Simmon, Spanggord, 1976, 1977). These reaction mixtures were assayed for mutagenic activity employing 1) five strains of *Salmonella typhimurium* (Ames

Salmonella/microsome assay); and 2) mitotic recombination in the yeast *Saccharomyces cerevisiae D3.* After very extensive ozonation in water some of the organic compounds exhibited mutagenic activity in these systems. Similar more recent studies under extreme conditions with chlorine dioxide by-products did not exhibit mutagenic activity (SRI Report).

Combining ammonia with chlorine to form chloramines has been called the chloramine process, chloramination, and combined residual chlorination. The products of this process are monochloramine, dichloramine or trichloramines (nitrogen trichloride) depending on the pH and the chlorine to ammonia ratio. The production of the latter species may contribute to taste and odor problems in the finished water; however, chloramination does not reduce the formation of THMs.

Based on the results of numerous investigations, the comparative disinfectant efficiency of chloramines ranks last when compared to ozone, chlorine dioxide, hypochlorous acid (HOCl), and hypochlorite ion (OCl^-) (NAS, 1977, 1979). Early studies by Butterfield and Watles (1944, 1946, 1948) demonstrated that chloramines required approximately a 100-fold increase in contact time to inactivate coliform bacteria and enteric pathogens as compared to free available chlorine at pH 9 5. This work was later confirmed by Kabler (1953) and by Clarke et al., (1962).

Results with cysts of *Entamoeba histolytica* and viruses also confirm the decreased effectiveness of chloramines as disinfectants. Studies by Fair, et al., (1947) showed that additional dichloramine is about 60 percent and monochloroamine about 22 percent as effective as hypochlorous acid at pH 4.5 against cysts of *E. histolytica.* Kelly and Sanderson (1960) found that chloramines in the concentration of 1 mg/l at 25° C required 3 hours at pH 6, or 6 to 8 hours at pH 10 to achieve 99.7 percent inactivation of polio virus. With 0.5 mg/l free chlorine at pH 7.8, by comparison, inactivation of 99.99 percent of polio virus can be achieved in approximately 15 minutes (Liu and McGrowan, 1973). Chloramine treatment finds its widest application in maintenance of chlorine residuals in the distributing systems. The human health effects of consuming water treated with chloramine have not been studied in detail.

Although all of these disinfectants can reduce THM formation, questions have been raised on both their toxicity and the toxicity of their by-products. Studies are underway to clarify these matters, and could result in the designation of maximum permissible levels for certain disinfectants applied to drinking water.

The use of adsorbents for THM removal has also introduced some unknown factors. Assuming that the adsorption process is effective for its intended purpose, there is the possibility that a breakthrough of some of the adsorbed chemicals may occur, that these substances will be adsorbed and subsequently slough off to produce intermittent contamination, or that bacteria and/or toxins will be added to the water from growth on the adsorbent. All of these potential effects are controllable in practice, and EPA encourages the use of GAC to purify contaminated waters and to control THM precursors.

Thus, THM concentrations should be reduced, but without compromising public health from either increased risk of infectious disease transmission or from the chemicals that are used. Outbreaks of infectious waterborne disease have been noted when chlorination systems have been improperly operated. The alternative control methods outlined previously are effective, and are also being studied for their possible side effects. As soon as data become available, EPA will make specific recommendations regarding their use. At the present time, the best approach to reduce THMs in finished water is to reduce precursors prior to chlorination, such as with GAC. This approach has the benefit of reducing the concentration of many other organic chemicals in the water as well as to the precursors to THM and other chlorinated organics. Thus, once the organic chemical concentrations in the water have been reduced, the chemical demand for applied disinfectant will be reduced. Thus, human exposure to all disinfectant chemicals and their degradation products and by-products will be minimized. This is the intent of the regulation controlling THMs.

IV. Sources of Trihalomethane Exposure

McConnell et al. (1975), have reported that chloroform occurs in many common foods and that while some halogenated compounds in food may result from manufacturing, canning and pest control practices, chloroform may be introduced as the result of geochemical processes. Chlorinated compounds are the halogenated species most prevalent in food, but at least one food, Limu Kohu, a seaweed or algae eaten in Hawaii, contains an essential oil which is composed largely of bromoform (Burreson, et al 1975).

Chloroform was widely used as an anesthetic in the past, and, until recently, was a common ingredient in dentifrices and cough preparations. The Food and Drug Administration has taken action to halt the use of chloroform in drug products, cosmetic products, and food-contact articles (41 FR 145026, April 9, 1976). EPA has issued a notice of "rebuttable presumption against registration" of chloroform-containing pesticides (41 FR 14588, April 6, 1976). Thus, in addition to drinking water, exposure to some or all of the THMs is complicated by other environmental sources, however, exposure from some of these sources is being reduced.

The relative human chloroform exposures can be estimated for three major sources of human exposure: atmosphere, drinking water, and the food supply. The uptake calculations are based on the fluid intake, respiratory volume, and food consumption data for "reference man" as compiled by the International Commission on Radiological Protection. The combined uptake for adults from all three sources was derived by multiplying estimated exposure levels by the estimated annual intakes and combining the results [ODW protocol].

Human uptake of chloroform from air, food and drinking water is given in Table 2. Chloroform and TTHM uptake from drinking water was estimated by multiplying the chloroform and THM concentrations from NOMS data (Table 1) by the average consumption of 2 liters of water per day for the 70 kg adult male, by 365. One hundred per cent absorption of the amount of chloroform in drinking water is assumed for these calculations. The total chloroform uptake from water was estimated as a mean value of 64 mg per year. The maximum uptake value may be 394 mg per year.

To determine uptake of chloroform from foods, the concentration of chloroform in each food item in North American diets was multiplied by the average annual consumption of that food item by adults in the United States (NAS, 1977), and the results were combined again; one hundred per cent absorption of ingested chloroform was assumed. A calculated maximum value of about 16 mg of chloroform uptake per year from total food an a mean value of 9 mg based on ODW assumptions was obtained.

Table 2—*Human Uptake of Chloroform and Trihalomethanes from Drinking Water, Food, and Air*

Chemical	Exposure levels mg/year Mean (range)		
	Drinking water	Food	Air[1]
Chloroform	64 (0 73-343)	9 (2-15 97)	20 (0 41-204)
Trihalomethanes	85 (0 73-572)		

[1]Calculated from data supplied by Strategies and Air Standards Division, Office of Air Quality Planning and Standards Environmental Protection Agency Research Triangle Park The air samples were collected both from the rural and industrial areas during the years 1974-76 The mean value was derived from the concentrations obtained from urban industrialized areas, the minimum value from the rural area and the maximum value from an urban industrialized area

The calculation for the uptake of chloroform by humans from ambient air was based upon the assumptions that 63 percent of inhaled choloroform is absorbed, (NAS, 1977); the volume of air inhaled by an average adult is 8.1×10^6 liters per year; and 0.02 and 10 ppb (by volume) are the respective minimum and maximum chloroform concentrations in urban air. The minimum and maximum values for the annual uptake of chloroform by an adult were estimated at 0 41 and 204 mg, respectively. Assuming minimum exposures from all sources, the atmosphere contributes 12 percent of the total chloroform, the drinking water contributes 23 percent, and food is most significant (65%). Assuming maximum exposures from all sources, drinking water is the major contributor at 61 percent, with air at 36 percent. Thus, the relative contribution of drinking water to the total body burden of chloroform may range from a moderate to a maximum contributor as the annual exposure from water ranges from nil to 394 mg/year, and from 204 to 0.73 mg/year in ambient air (Table 3).

Table 3.—*Uptake of Chloroform for the Adult Human from Air, Water, and Food*

Source	Adult mg/yr	Percent uptake
Maximum Conditions		
Atmosphere	204	36
Water	343	61
Food supply	16	3
Total	563	100
Minimum Conditions		
Atmosphere	0 41	13
Water	0 73	23
Food supply	2 00	64
Total	3 14	100

	Max Water Min-Air	
Atmosphere	0 41	1
Water	343 00	97
Food supply	9 00	2
Total	352 41	100

B. Metabolism

Several reports (Brown, et al., 1974; Labigne & Marchand, 1974, Fry et al., 1972; Paul and Rubenstein, 1963; Taylor et al., 1974) have indicated that chloroform is rapidly absorbed on oral and intraperitoneal administration and subsequently metabolized to carbon dioxide and unidentified metabolites in urine. Species variation in the metabolism of chloroform has been summarized in Table 4. It is noteworthy that the mouse, a species which shows greater sensitivity to the oncogenic effect of chloroform (Eschenbrenner & Miller, 1945; Brown et al. 1974) metabolized chloroform extensively to carbon dioxide (80%) and unidentified metabolites (3%) from an oral dose of 60 mg/kg. Rats also metabolize chloroform to carbon dioxide but to a lesser extent (66%). In another report, Paul and Rubinstein (1963) recovered 4 percent carbon dioxide after administering 1484 mg/kg chloroform intradoudenally to rats. The discrepancy in these two results may be dose related.

Dose related differences in the metabolism of compounds are known and have recently been reported for the carcinogen vinyl chloride. Squirrel monkeys, when given 60 mg/kg of chloroform orally, excreted 97 percent of the dose, with 17 percent as carbon dioxide and 78 percent as chloroform. Fry, et al. (1972), recovered unmetabolized chloroform ranging from 17.8–66.6 percent of a 500 mg dose of chloroform given to human volunteers during an 8 hour time period (equivalent to about 7 mg/kg). Since the metabolism of chemicals is also dependent on age and sex, the widespread variation in the quantitative disposition of chloroform in human subjects may be due to the experimental protocols wherein subjects ranging from 18–50 years of age were used Individual variability in the non-homogenous human population is a major factor.

Metabolic similarities between carbon tetrachloride and chloroform include the appearance of halide ions in urine and carbon dioxide in breath. A related chemical, carbon tetrachloride, is a common contaminant of the chlorine used in water disinfection. Carbon tetrachloride also is metabolized to chloroform in trace amounts, which may in turn, be biotransformed to carbon dioxide. Both chloroform and carbon tetrachloride are proven animal carcinogens (see below). However, this is mentioned because of possible metabolic production of proximal carcinogens. Toxicity of carbon tetrachloride, however, has been attributed to a free radical (CCl_3) which is postulated as a metabolic intermediate. Chloroform appears to be metabolized to form phosgene (Krishna, 1979).

Table 4.—*Disposition of Chloroform—Species Variation*

Animal species	Sex	Strain	Dose mg/kg	Metabolism (percent) $CHCl_3$	CO_2	Urine feces	Total excretion	References
Mouse	M	CBA CF/ LP C57	60 po	6	80	3	[1]93	Brown *et al* (1974).
Rat	M	Sprague Dawley	60 po	20	66	7	93	Brown *et al* (1974)
Rat			1,484 id	70				Paul & Rubstein (1963)
Rat	M	Sprague Dawley	4,710 ip		0 39			
Monkey	M	Squirrel	60 po	78	17	2	97	Brown *et al* (1974)

[1]Includes radioactivity in carcas
Po Orally
id intradeudenally
ip - intraperitoneal

Many carcinogens have been reported to form complexes with proteins, DNA and RNA (Miller & Miller, 1966). In the case of chloroform, Ilett et al., (1973) reported covalent bonding of chloroform metabolite(s) to tissue macromolecules in mice. The covalent bonding increased or decreased when the animals were pretreated with phenobarbital or piperonyl butoxide, agents which stimulate or inhibit the metabolism of foreign compounds by mixed function

oxidase enzymes. This is suggestive of the involvement of chloroform metabolism in these processes. These results may be interpreted to mean that the potency of an ingested chemical will be dependent upon its rate of metabolism to the active form.

Information regarding the metabolism of bromoform and other haloforms is not available. However, the structural similarities of these haloforms with chloroform indicate that they should also be absorbed by the oral and inhalation routes of exposure and then metabolized into carbon dioxide and halide ions. Related halogenated hydrocarbons of the dihalomethane series (e.g., dichloromethane, dibromomethane and bromochloromethane) have been reported (Kubic et al. 1974) to be metabolized to carbon monoxide; the rate of metabolism of dibromomethane was higher than that of the dichloromethane.

VI. Acute and Chronic Health Effects in Animals

Mammalian responses to chloroform include effects on: the central nervous system, hepatotoxicity, nephrotoxicity, teratogenicity, and carcinogenicity. Reported oral LD_{50} values are as follows: for rats, 300 mg/kg (DHEW, 1978); and for mice, 705 mg/kg (Plaa, et al., 1958).

Jones, et al. (1958), reported the effect of various oral doses of chloroform on mice 72 hours after exposure:

35 mg/kg—threshold hepatotoxic effect—minimal midzonal fatty changes
70 mg/kg—minimal hepatic central fatty infiltration
140 mg/kg—massive hepatic fatty infiltration
350 mg/kg—hepatic centrilobular necrosis
1,100 mg/kg—minimum lethal dose

Acute effects of exposure to chloroform and bromoform vary among species. Reported lethal doses for chloroform and bromoform are:

Species	Subcutaneous lethal dose	Values in mg/kg
Mouse	LD_{50}	704 (Chloroform) 1820 (Bromoform)
Rabbit	LD_{50}	800 (Chloroform) 410 (Bromoform)

Data on the acute toxicity of dibromochloromethane and dichlorobromomethane are not available.

A. Hepatotoxicity

Plaa, et al. (1968) established a dose-response relationship in mice, measuring parameters indicative of hepatotoxicity. Median effective dose (ED_{50}) values of 1.4 mM/kg (166 mg/kg) were found in mice exposed to chloroform by subcutaneous injection. The inhalation exposure of chloroform by mice for 4 hours at concentrations ranging from 100–800 ppm resulted in fatty infiltration of the liver at all dose levels. These changes were observed at necropsy 1–3 days after exposure.

Like chloroform, bromoform exposure leads to fatty degeneration and centrilobular necrosis of the liver (von Oettingen, 1950).

Dibromochloromethane and dichlorobromomethane may bring about similar responses, although no experiments have been reported.

B. Nephrotoxicity

Nephrotoxic effects of chloroform were studied by Plaa and Larson (1965). The ED_{50} for orally administered chloroform in mice was 178 mg/kg as measured by phenolsulfo-phthalein excretion. Increases in urinary protein and glucose excretion, indices of kidney damage, indicated an ED_{50} of 104 mg/kg chloroform. Data concerning the nephrotoxic effect of other THMs are not available.

C. Central Nervous System Effects

Chloroform was used extensively as an anesthetic because of its effect on the central nervous system. Lehmann and Hasegawa (1910) reported dizziness and light intoxication during 20-minute exposures to chloroform concentrations of 4300–5100 ppm. Repeated exposures up to six days to concentrations as low as 920 ppm for 7 minutes resulted in symptoms of central nervous system depression (Lehman & Schmidt-Kehn, 1936). Additional important information has been submitted to EPA and is discussed below.

Effects of acute and subchronic chloroform exposure on cholinergic parameters in mouse brain were studied by Vocci, et al., (1977). Male Swiss Webster ICR mice were gavaged with single doses of chloroform (30 and 300 mg/kg) and sacrificed 15 minutes after administration of chloroform. In another experiment, the mice were gavaged with 14 or 90 daily doses of chloroform (3 or 30 mg/kg) and sacrificed 18 hours after the last administration. Neither of the above dosage regimens had any effect on *in vitro* [^{3}H] choline uptake in synaptosomes. In another study (ibid) of biosynthesis of acetylcholine in mouse brain, chloroform (30 mg/kg) significantly decreased the [^{3}H] acetylcholine synthesis (57% of control). Administration of chloroform (3 mg/kg) for 14 days produced a reduction in [^{3}H] acetylcholine (57% of control) (Vocci, Personal Communication, April 1979).

Chloroform, dichlorobromomethane, chlorodibromomethane and bromoform, at concentrations of 8×10^{-4} M did not alter the update of norepinephrine or dopamine into brain synaptosomes *in vitro* (Vocci, Personal Communication, April 1979).

D. Teratogenicity

Teratogenic responses to oral dosing of animals with chloroform were investigated. Rats and rabbits were administered chloroform at 126 and 50 mg/kg respectively. No significant fetal deformities were observed (Thompson et al. 1973). Inhalation of chloroform by Sprague Dawley rats at 30, 100 and 300 ppm for 7 hours a day, on days 6 through 15 of gestation revealed significant fetal abnormalities including: acaudia, imperforate anus, subcutaneous edema, missing ribs and delayed skull ossification (Schwetz et al. 1974).

In an attempt to explain reproductive failure in laboratory animals, i.e., mice and rabbits, McKinney et al. (1976) conducted a study using CD–1 mice wherein groups of mice were given tap water and purified tap water (passed through a Corning 3508 ORC and a Corning 3508 B demineralizer), respectively. Analysis indicated reduced amounts of chlorinated compounds in the purified water. The study could not relate chloroform and other chlorinated organics in tap water to reproductive failures in laboratory animals, since the concentrations of chlorinated organics in water were lowest in those months that reproductive failure was highest, although there did appear to be small, non-significant differences in this parameter between the highly purified and tap water. In a reevaluation involving the effect of Durham tap water and purified tap water as in the above study, Chernoff (1977) did not find striking differences in the reproductive success of CD–1 mice. No teratogenic studies on haloforms other than chloroform were available.

E. Mutagenicity

The THMs (chloroform, bromodichloromethane, dibromochloromethane, dibromochloromethane and bromoform) were assayed *in vitro* for mutagenic activity using strains of *Salmonella typhimurium* (TA 100 & TA 1535). The assays were conducted in desiccators to allow each compound to volatilize so that only the vapor phase came in contact with bacteria on the petri dishes. The activation system was tested and found not to be required for the bromohalomethanes since they wer positive in the absence of activation. The results obtained were as follows: (a)

Chloroform was not mutagenic in TA 00 with or without activation, nor in TA 535 without activation; (b) romodichloromethane was mutagenic n TA 100 without activation, with a doubling dose of approximately 25 microliters; (c) dibromochloromethane was mutagenic in TA 100 without metabolic activation, with a doubling dose of approximately 3.5 microliters; (d) bromoform was mutagenic in TA 100 without metabolic activation, with a doubling dose of approximately 25 microliters, and was also mutagenic in TA 1535 with metabolic activation, with a doubling dose of approximately 100 microliters (Tardiff, 1976). All three compounds demonstrating mutagenic activity did so in a dose-response mode. For certain classes of compounds, except for many chlorinated hydrocarbons (Ames, 1973) the Ames test which utilizes *Salmonella typhimurium* bacteria correlates highly (90 percent) with the *in vivo* carcinogenicity bioassay.

F. Carcinogenicity

Prolonged administration of chloroform at relatively high dose levels to animals, specifically mice and rats, manifested oncogenic effects. The nvestigation conducted by Eschenbrenner and Miller (1945) roduced hepatomas in female mice (strain A) given repeated dosages ranging from 0 145 to 2.32 mg of chloroform for a period of only four months. Minimum doses of 593 mg/kg chloroform per day (total of 30 doses) produced tumors in all of the surviving animals.

In a recent bioassay (NCI, 1976) linking chloroform with oncogenicity, rats and mice of both sexes were fed doses of chloroform ranging from 90 to 200 (rats), and 138–477 (mice) mg/kg. In this study, the lowest dose for observed carcinogenic effect (kidney epithelial tumors) in male rats was 100 mg/kg and for mice 138 mg/kg administered to the animals for a total period of 78 weeks. A related halogenated hydrocarbon, carbon tetrachloride, was carcinogenic in Osborne Mendel rats and in B6C3F1 mice at dosages ranging from 57 to 160 mg/kg and 1250 to 2500 mg/kg, respectively. The incidence of hepatocellular tumors formed in these animals at both dose levels almost approached one hundred percent (Table 5). The percent survival in mice treated with chloroform and carbon tetrachloride is depicted in Table 6. Almost all the animals on treatment with carbon tetrachloride died between 91–92 weeks whereas with chloroform treatment at both dose levels, 73 and 46 percent of the animals survived. Miklashevskii et al. (1966) fed chloroform to rats at 0.4 mg/kg apparently for 5 months and detected no histopathological abnormalities after this treatment. A recent study on the carcinogenic effect of chloroform at dose levels of 17 mg/kg/day and 60 mg/kg/day was conducted by Roe (1976), utilizing the rat (Sprague-Dawley), the beagle dog and four strains of mice (ICC Swiss, C57B1, CVA and CF/1). Comparison with the NCI study (1976) indicates that the number of animals and the duration of the experiment were essentially similar; the major differences were the dosages, which were lower than in the NCI study, and the vehicle, which was toothpaste. The only finding of neoplasia was an excess of tumors of the renal cortex in the male ICI-Swiss mice at a dose level of 60 mg/kg/day. However, animals fed 17 mg/kg/day of chloroform showed no incidence of renal carcinoma.

Table 5.—*Comparison of Hepatocellular Carcinoma Incidence in Chloroform and Carbon Tetrachloride-Treated Mice*

Animal group	Chloroform	Carbon tetrachloride
Males		
Controls	5/77	5/77
Low Dose	18/50	49/49
High Dose	44/45	47/48
Females		
Controls	1/80	1/80
Low Dose	36/45	40/40
High Dose	39/41	43/45

Table 6.—*Comparison of Survival of Chloroform and Carbon Tetrachloride-Treated Mice*

Animal group	Chloroform			Carbon tetrachloride		
	Initial No	78 weeks	90 weeks	Initial No	78 weeks	91–92 weeks
Males						
Controls	77	53	38	77	53	38
Low Dose	50	43	37	50	11	0
High Dose	50	41	35	50	2	0
Females						
Controls	80	71	65	80	71	65
Low Dose	50	43	36	50	10	0
High Dose	50	36	11	50	4	1

Some renal tumors were also seen in control animals in a later study. The negative results observed in the dog experiment may be explained on the basis that either the animals were not exposed for a suitable length of time (i e. duration of life span) or that an insufficient number of animals were tested, or that this species may not have been responsive to the oncogenic effect of chloroform. The negative results of the rat study may be explained on the basis of lack of strain sensitivity. Based on the extrapolation from the NCI study, the dose was too low to produce an effect in so few animals (Cueto, NCI, 1979).

Much less information is available on the carcinogenicity of bromohalomethanes. Preliminary results from the strain A mouse pulmonary tumor induction technique (Theiss et al., 1977) indicated that bromoform produced a positive pulmonary adenoma response while chloroform did not. Other studies (Poirier, et al., 1975) indicated that in several instances brominated compounds exhibited more carcinogenic activity than their chlorinated analogs in the pulmonary adenoma bioassay.

VII. Human Health Effects

A. NAS Principles of Toxicological Evaluation

The recent NAS (1977) report entitled "Drinking Water and Health" identified several principles for assessing the irreversible human effects of long and continued low dose exposure to carcinogenic substances.

Principle 1: Effects in animals, properly qualified, are applicable to man.

Principle 2 Methods do not now exist to establish a threshold for long-term effects of toxic agents.

Principle 3: The exposure of experimental animals to toxic agents in high doses is a necessary and valid method of discovering possible carcinogenic hazards in man.

Principle 4. Materials should be assessed in terms of human risk, rather than as "safe" or "unsafe".

On the basis of studies in animals and human toxicological data the NAS (1977) has recommended that strict criteria should be applied for establishing exposure limits to chloroform.

The National Institute for Occupational Safety and Health has recommended that the occupational exposure to chloroform should not

exceed 2 ppm determined as time-weighted average exposure for up to a 10 hour work day.

The human health effects as observed in accidental, habitual, and occupational exposures appear to indicate that the effects produced by exposure to chloroform are similar to those found in experimental animals. These include effects on the central nervous system, liver, and kidney.

The symptoms observed (Storms, 1973) in a 14 year old patient following an accidental exposure to an unknown amount of chloroform included cyanosis, difficulty in breathing and unconsciousness. Liver function tests measured by serum enzyme levels four days after ingestion indicated high levels of SGOT, SGPT, and LDH. The authors also noted damage to the cerebellum characterized by an instability of gait and a slight tremor on finger-to-nose testing. The symptoms disappeared in two weeks.

Several cases of habitual chloroform use have also been recorded by Heilbrunn et al. (1945). A case study of interest was a 33 year old male who had habitually inhaled chloroform for 12 years. The subject showed psychiatric and neurological symptoms including restlessness, hallucinations, convulsions, dysarthria, ataxia, and tremors of the tongue and fingers.

Lunt (1953) reported that delayed chloroform poisoning in obstetric patients, anaesthetized with chloroform is characterized by renal dysfunction as indicated by: Albumin, red blood cells, and pus in the urine. Chloroform exposure of humans by inhalation was studied by Lehman and Schmidt-Kehl (1936). Ten different concentrations of chloroform were used and the chloroform concentrations were determined by the alkaline hydrolysis method. Exposure at concentrations of 7 ppm for 7 minutes and at all higher levels up to 3000 ppm caused symptoms of central nervous system depression.

Desalva et al. (1975) studied the effects of chloroform in humans; the subjects were given dentifrice containing 3.4% chloroform and mouthwash with 0.43% chloroform for 1 to 5 years. No hepatotoxic effects were observed at estimated daily ingestion of 0.3 to 0.96 mg/kg chloroform. Reversible hepatotoxic effects were manifested at 23 to 27 mg/kg/day chloroform ingested for 10 years in a study conducted by Wallace (1959).

B. Epidemiologic Studies

By August 1979, 18 epidemiological studies, and additional unpublished reports discussed possible relationships between cancer mortality and morbidity and drinking water supplies. The results of the studies are shown in Table 7 in the approximate chronological order of completion. The table shows the statistically significant results of analysis by anatomical site. The statistically significant positive results are denoted by "M" for males and "F" to females and the statistically significant negative results are denoted by "—" before the "M" or "F".

BILLING CODE 6560-01-M

Table 7. Statis Significant Results of Epidemiological Cancer Studies on Drinking Water and Cancer Sites

No.	Author	Yr.	Oral	Esophagus	Stomach	Intestine (not rectum)	Rectum	Combined G.I.	Liver and Gallbladder	Pancreas	Respiratory (not lung)	Lung, trachae, bronchi	Breast	Cervix	Uterus, ovary	Prostate	Bladder	Kidney	Combined Urinary	Lymphatic	Leukemia	Non-Hodgkins Lymphoma	Hodgkins	Thyroid	Brain	Larnyx	All Cancer	Study Type
1	Page, H.E.	74 76						M F											M								M	E
2	Tarone	75										M															M	E
3	Vasilenko	75		—	F	(studied females only)						F															F	E
4	DeRouen	75			F	M F	M		-M						F		M		·	-M							F	E
5	Harris/ Reiches	76		M	M F			M		M							M		M								M F	E
6	Buncher	75			M F				F	F			F				M F											E
7	Kuzma	77			M F					F			F				M										M F	E
8	McCabe	75	(total cancer and total mortality only)																								M F	E
9	Cantor	78			-F					-M		F				M	M F								M			E
10	Hogan	79	-M	-M	-M -F	M F	F		-M								M F							-M -F				E
11	Salg	77		M	-F	M	M F	M				M	F				M			F			M F			M F	M	E
12	Kruse	77	(studied liver and kidney sites only)																									C-C
13	May	77	(Eight sites studied -- no statistically positive associations/geographic and data analysis problems)																									E
14	Alavanja	77 78		M	M F	M	M	M F				M					M		M F								M F	C-C
15	Wilkins	78							F								M		(liver, kidney and bladder only)									C-C
16	Rafferty, N.	79																										E
17	Brenniman	78	(Completed but results not useable) (Females Only)			F		F	F										F									C=C
18	Tuthill[1] Tuthill[2]	78 79		M	M,F							-F																E E

E = Epidemiological C = Case-Control R = Retrospective

BILLING CODE 6560-01-C

Five of the studies were published through August 1979. All of the studies were retrospective in design; sixteen were correlation studies, and four used a case-control approach. Four studies utilized cancer morbidity or incidence rather than mortality as a measure of disease frequency. The studies vary in sample size, cancer sites considered, factors selected as possible explanatory variables, parameters selected as indicators of water quality, and in the statistical techniques used for analysis, so caution must be used in comparing the results of one study with the results of another study.

There are several problems which make the results difficult to interpret: (1) There is limited water quality data on organics and other contaminants in the finished drinking water, and the data which exist cover less than five years; and (2) the water quality data are often from geographic areas other than those (usually counties) reporting cancer mortality data.

The water quality data are recent, and it is not known to what extent they reflect past exposure to THMs. This is important, since the latent period for most types of cancer is measured in decades. Comparison of the various study results is difficult also because of the different approaches used.

In general, retrospective epidemiological studies are a useful methodological tool in hypothesis generation. The results from these studies, when viewed collectively, can provide some insight into the postulation of causal relationships which then need to be tested further, using epidemiological designs such as case-control or cohort studies, for documentation.

When the evidence from all studies is weighed, an emphasis can be placed not only on the statistical significance of single correlation coefficients but on their consistency and patterns. When more than one independent study shows positive associations for *site-specific* cancers, then the association may not be due to chance alone. When the association is verified by consistent results across all four sex-race groups (white male, non-white male, white female, non-white female), the association is more likely to be used due to the variable considered and the evidence should be viewed more seriously. The studies done so far suggest the appropriateness of concern.

There is much evidence (both epidemiological and experimental) that most human cancers result from a combination of causes (Weisburger, 1977). Etiologic factors (e.g. smoking as a cause of lung cancer, soot as a cause of scrotal cancer in chimney sweeps) that result in increased relative risk greater than 5, were among the first to be discovered. The etiologic factors associated with cancers of gastrointestinal and urinary tract are more difficult to isolate from epidemiological studies because of the lower incidence and mortality rates, the interaction of environmental causes, and site-specific differences. The increased relative risk of populations exposed to most factors suspected of being associated with gastrointestinal and urinary cancers are less than three. Effects as small as, or smaller than these, are difficult to detect or quantify.

A number of the epidemiologic studies relating "water quality" to cancer did not define the water quality parameter by chemical constituents but instead compared cancers in persons who used water from different sources. Among the first of these was an investigation by Page, Talbot, and Harris (1974). The study considered Louisiana parish (county) cancer mortality rates for 1950–69, for total cancers and various selected cancer sites, and related these to the percentage of the parish populations drinking water from the Mississippi River, which is known to be contaminated by many organic chemicals (Laseter, 1972). The variables controlled were the rural-urban character of the parish, median income, population density, and proportion of population employed in the petroleum, chemical, and mining industries. An unweighted regression analysis showed a positive correlation between drinking water and total cancer (excluding cancer of the lung, urinary tract, GI tract, and liver), and then separately for cancer of the gastrointestinal organs and lung cancer. These investigations suggested an association between cancer mortality rates and use of drinking water from the Mississippi.

Meinhardt, et al. (1975), commenting on the Page-Harris report, looked at the cancer mortality gradient by apparent "dose" of river water and concluded that there was a random distribution of high and low cancer mortality rates among the river water consumers along the lengths of the Missouri and Mississippi River systems.

Subsequent reports by Page and Harris (1975, 1976) on the "Relation Between Cancer Mortality and Drinking Water in Louisiana" utilized explanatory variables and cancer sites similar to those in the first study; relationships for all four sex-race groups were considered. Positive regression coefficients for the water variable that were found statistically significant were:

Total cancer sites: WM, NWM, NWF.
All other than lung: WM.
Urinary Tract. WM, NWF.
Gastrointestinal. WM, NWM, WF, NWF.

Tarone and Gart (1975) reviewed the Page-Harris work and included an additional variable, elevation above sea level. By using a weighted regression analysis for four race-sex groups, statistically significant, positive correlations were found between the water variable and total cancer and lung cancer mortality for white males (WM), non-white males (NWM), and non-white females (NWF). The correlations were not statistically significant for white females (WF) for the same sites. Thus, there was a lack of consistency across the four sex-race groups for the aforementioned cancer sites.

Vasilenko and Magno (1975) conducted an ecological study in New Jersey and determined the relation between water source and age-adjusted cancer mortality from lung, stomach and urinary tract cancer of white females. Water quality was estimated from the ratio of the number of households served by public systems and private water companies to the number served by individual wells. Positive associations were found for lung and stomach cancer.

DeRouen and Diem (1975) also reviewed the relationship of cancer mortality in Louisiana and the Mississippi River as the drinking water source looking at ethnic variables as a possible confounding factor. By dividing Louisiana into a northern and southern section, they were able to mimic an ethnic division of the population. Many of the variables (urban-rural characteristics, median income, employment characteristics, and elevation above sea level) included in the previous studies were omitted. The water variable was handled differently by the investigators. Population groups were dichotomized into those who obtained none of the water from the Mississippi River, and those who obtained some or all from the river. The results show a positive relationship between cancer mortality and drinking water, for gastrointestinal cancer. The cancer mortality rates for southern parishes of Louisiana whose source of drinking water is the Mississippi River are higher than in the southern parishes whose source of drinking water is not the Mississippi River for the following:

Stomach: NWF.
Rectum: WM.
Large Intestine: WF, NWF.
Cervix: NWF.
Lung: NWF.
Total Cancer: NWF.

The cancer mortality rates tend to be higher for the southern parishes with ver water use than northern for river ater parishes for cancer of the urinary act, gastrointestinal tract, and the lung.

In another set of analyses and comments, DeRouen and Diem (1975) discuss the problems associated with interpretation of regression coefficients as they relate to the Page and Harris Report, particularly the problem of making interferences from correlational studies. They concluded that inconsistencies such as the failure to see the same relationships for all sex-race groups reduces the credibility of the hypothesis of a causal relationship between water source and cancer risk.

An analysis was done by McCabe (1975) of EPA using the 50 (of a total of 80) NORS cities with a 1950 population greater than 25,000 and 70 percent or more of the city's population receiving water comparable to that sampled by EPA. McCabe showed a statistically significant correlation between the chloroform concentrations in the drinking water and the cancer mortality rate by city for all cancers combined.

In a second analysis by McCabe using water quality data from Region V, correlations between chloroform and TTHMs and total cancer mortality were ot positive. When the same rrelations were done using Region V us NORS data for chloroform and total trihalogenated methane concentration levels, a positive statistically significant result was obtained.

Several epidemiological studies have been conducted in the Ohio River area. Buncher (1975) conducted a study of 88 counties (in Ohio, bordering the Ohio River) of which 14 used the Ohio River as a drinking water source. Buncher reports no significant relationship with drinking water from the Ohio River and the higher cancer mortality rates. There was a weak positive correlation between the chloroform concentration in 23 cities and the cancer mortality rate for all cancer sites in white males. Similar results were found in 77 cities (59 with surface water supplies) between chloroform concentrations and pancreatic cancer mortality in white females. For cities that accounted for more than 70 percent of the county population, there was a significant correlation between chloroform concentration and bladder cancer mortality rates for both white males and white females.

As a follow up on the Buncher study, a study by Kuzma, et al. (1977), nsidered the 88 Ohio counties, assified as either ground water or rface water counties based on the ource of the drinking water used by a majority of the county residents. A two-stage analysis was performed and no statistically significant results were shown between the drinking water from the Ohio River and cancer mortality rates. However, rates for stomach, bladder, and total cancers were higher for white males in counties served by surface water supplies (probably chlorinated) than in counties served by ground water supplies (probably not chlorinated).

Reiches, et al. (1976), re-examined the Ohio data using a different methodology. Correlations between the surface drinking water variable and cancer mortality rates for stomach cancer and total cancers for both white males and females were statistically significant. The correlations between the drinking water variable and cancer mortality rates of the pancreas, bladder, esophagus, gastrointestinal tract, and urinary organs were significant for white males only.

Although several studies defined the water quality parameter by chlorination or levels of chloroform, only one study has considered the relationships of cancer with all THMs, both collectively and separately. Cantor et al. (1978) studied the correlation of cancer mortality at sixteen anatomical sites with the presence of concentration levels for each THM and TTHM in drinking water for whites. Counties were grouped according to the percentage of the county population served by the sampled water supply. In both sexes, there was a positive dose-response gradient of increasing correlation between trihalomethane concentration and bladder cancer. The correlation was stronger for bromoform than with chloroform. There was a negative correlation in white females of stomach cancer with total THM levels. Kidney cancer in white males showed a positive correlation with chloroform levels. Lung cancer in white females showed a positive correlation with THM levels. Among white males non-Hodgkins' lymphoma showed a positive correlation with bromoform. A positive dose-response was observed between brain cancer mortality (in both sexes) with increasing use of water containing chloroform, but the associations were not strong.

Alavanja, et al. (1976) conducted a retrospective, case-control study of female cancer mortality and its relationship to drinking water chlorination in seven selected New York counties. A statistically significant association was found between a region being served from a chlorinated drinking water supply and combined gastrointestinal and urinary tract cancer mortality rates in that region. There was also a higher mortality for the summed gastrointestinal and urinary cancer in urban areas served by chlorinated surface or ground drinking water supplies than in urban areas served by nonchlorinated supplies, however, the results should be viewed cautiously due to the small numbers in the sample.

Alavanja (1977) expanded this study and included gastrointestinal and urinary cancer deaths. Results showed that males living in the chlorinated water areas of three counties and females living in the chlorinated water areas of two counties were at greater risk of gastrointestinal and urinary tract cancer mortality than individuals living in the non-chlorinated areas. Alavanja (1978) did a second study (shown on Table 7), which expanded the first to nineteen counties in New York and several specific cancer sites. Statistically significant positive associations were found for males and lung cancer and for females and pancreatic cancer. Statistically significant positive associations were found for both males and females and cancer of the large intestine, combined gastrointestinal, and all cancers.

Kruse (1977) conducted a retrospective, case control study of white males and females in Washington County, Maryland. The relationship between mortality and morbidity from liver (including biliary passages) and kidney cancer in areas supplied by chlorinated public water supplies was analyzed. While there was a higher incidence of liver cancer among the exposed group; i.e., the group which consumed chlorinated drinking water, the correlations were not statistically significant. It should be noted that the sample size was small and that fewer than 50 cases each of liver cancer and kidney cancer were counted.

Salg (1977) also conducted a retrospective study of various cancer mortality rates and drinking water from a variety of sources and receiving different types of treatment in 346 counties in seven states in the Ohio River Valley Basin. She compared mortality rates for white and non-white males and females using weighted regression analyses, surface water usage showed weak but statistically significant associations between chlorinated water supplies (regardless of source) and the following cancers: For white males—esophagus, respiratory organs, large intestine, rectum, bladder, other urinary organs and lymphosarcoma and reticulosarcoma; for white females—breast and rectum,

and for non-white females—esophagus and larynx. Rectal cancer showed positive correlations across all race-sex groups. It should be noted that the test of significance utilized for this study was $p < 0.10$, which is less stringent than that used in other studies.

Mah, et al. (1977), conducted a retrospective study of the white population in the Los Angeles County area of the relationship between cancer mortality and morbidity and the chlorinated drinking water supply. They did not reveal any trends and showed no significant relationships for either cancer mortality or morbidity. The authors pointed out several methodological problems, including the diluting effect of migration into the area covered by this study.

Hogan, et al. (1979) also utilized the NORS and Region V data sets and applied various statistical procedures to the data in order to determine the effects of using different statistical models. Their results were similar to previous studies showing a positive correlation between rectal-intestinal and bladder cancer mortality rates and chloroform levels in drinking water when weighted regression analysis were applied. However, as the authors pointed out, "the marked extent to which these results were dependent on (1) the weighting scheme adopted in the analysis, (2) the presumed appropriateness of the data, and (3) the characteristics of the statistical model, was also clearly illustrated."

Wilkins (1978) conducted a case-control study in Washington County, Maryland and investigated the association between liver, kidney and bladder cancer and chlorinated water source. A positive correlation was found for female liver cancer and male bladder cancer and the chlorinated drinking water source. Due to small numbers of cases the outcome of this study should be viewed with suspicion.

Rafferty (1979) studied associations between drinking water quality in North Carolina communities and cancer mortality rates. The drinking water supplies were characterized by domestic and/or industrial contribution. No significant positive association were found.

Tuthill and Moore (1978) investigated the association between cancer mortality rates and parameters of water quality for Massachusetts community public water supplies. The average annual chlorine dose was one of the independent water characteristics. Simple correlations showed that the average chlorine dose level in the water was negatively associated with female buccal cancer, and positively associated with female esophageal and male respiratory cancers. Occupation, population mobility, and other demographic variables were controlled.

In summary, many but not all of the studies have found positive correlations between some characteristics of drinking water and various cancer mortality/morbidity rates. However, these correlations are dependent upon the selection and appropriateness of the data, the weighting scheme and extrapolation in the analysis, and the characteristics of the statistical model. Because of these dependencies the quantitative, causal interpretation of results generated from an indirect or ecological study should be viewed as tenuous for the primary purpose of generating hypotheses and even questionable in most cases.

It is important in the evaluation process to consider the results from other epidemiological studies as they develop hypothesis of potential causal associations between cancer mortality and other agents. For example, the confounding factors of diet, occupation, and smoking all have been suggested as potential causative agents of bladder cancer, Cole (1972). Therefore, any epidemiological study that investigates the possible association between bladder cancer and drinking water should be designed to avoid the problems that result in confounding of the data. None of the studies completed thus far have obtained data on or controlled for diet; several studies have attempted to control for occupational exposure (Page and Harris, 1974 and 1975; Cantor, et al., 1978; Tuthill and Moore, 1978); only the studies by Kruse (1977) and Wilkins (1978) obtained smoking data. Only a few studies considered four sex-race groups (the number of non-whites is too small in some of the geographic areas) and of those studies only a few showed consistent patterns of association of *specific cancer sites*, e.g., Salg (1977)-rectum. Several studies which considered only white populations found positive correlation coefficients for both sexes: Kuzma (1977)—stomach; De Rouen (1975)— intestine, stomach and bladder; Buncher (1975)—bladder; Reiches (1976)—stomach; Cantor (1978)—bladder; Hogan (1979)—intestine and bladder; and Alavanja (1978)—intestine. Only a few studies defined the water quality variable by the chloroform concentrations (McCabe, 1975; Buncher, 1975; Cantor et al., 1977; Hogan et al., 1977; Alvanja, 1978), and by the THM concentrations (Cantor et al. 1977).

Of particular interest are possible correlations of liver and kidney cancer rates with drinking water, since the animal exposure data indicate that hepatocellular carcinomas and hepatic modular hyperplasias have been observed in B6C3F1 strains of mice after life time exposure to chloroform. Several of the preliminary studies grouped the cancer sites for the anatomical systems, e.g., gastrointestinal and urinary organs, in order to increase the sample size. One of the studies (Cantor, 1978) which considered site-specific cancer mortality showed a positive association between drinking water and cancer of the kidneys in white males. The absence of any positive association between drinking water and liver cancer mortality may be due in part to small sample sizes, very low incidence of the disease, or because the exposure levels of contaminants in trace amounts over a lifetime may be below a no-effect level (Weisburger, 1977). The incremental increase may be too small to measure for statistical significance. On the other hand, many scientists believe that the specific site in which cancer appears in animal tests need not necessarily be the same site in which the cancer is likely to appear in humans.

Thus, the evidence is incomplete and the trends and patterns of association have not been fully developed. As stated previously, a causal relationship cannot be established by correlation studies. When viewed collectively, the epidemiological studies completed thus far provide evidence for maintaining a hypothesis that there may be a health risk and that the positive correlations may be due to an association between some constituents of drinking water and cancer mortality. The animal test data alone provide a firm basis for policy decisionmaking. Additional epidemiological studies may provide evidence regarding the strength of the associations and the possibility of a causal relationship between drinking water and cancer mortality, and thus provide a stronger basis for further regulatory action.

The NAS Epidemiology Subcommittee of the Safe Drinking Water Committee reviewed the first thirteen of the aforementioned eighteen studies. In the report, "Epidemiological Studies of Cancer Frequency and Certain Organic Constituents of Drinking Water—A Review of Recent Literature Published and Unpublished," September 1978, the Committee reached the following conclusions, which are consistent with EPA. Among the group of studies that characterized water quality by actual measurements, the results suggest:

That higher concentrations of THMs in drinking water may be associated with an

increased frequency of cancer of the bladder. The results do not establish causality, and the quantitative estimates of increased or decreased risk are extremely crude. The positive association found for bladder cancer was small and had a large margin of error; not only statistical, but much more importantly, because of the very nature of the studies.

Further research is being conducted with more definitive analytical studies. A large case-control bladder cancer study with 3,000 cases and 6,000 controls is being conducted by the National Cancer Institute (NCI). Three other case-control colon cancer studies are being conducted in Louisiana, Pennsylvania, and Utah. The results of these studies may provide more solid evidence to answer the question of possible associations between water quality and increased incidence of bladder and colon cancer.

VIII. Mechanism of Toxicity

Biologic responses upon exposure of mammals to chloroform include effects on the central nervous system resulting in narcosis, hepatotoxicity, nephrotoxicity, teratogenicity and carcinogenicity. Elucidation of the mechanism of toxicity of chloroform and related compounds has been attempted by several researchers.

Scholler (1968) and McLean (1970) observed that phenobarbital pretreatment of rats caused an increase in liver necrosis after administration of chloroform. Later, Brown, et al. (1974) reported that exposure of rats to an atmosphere containing chloroform (0.5%) for 2 hour markedly decreases glutathione (GSH) concentration in the liver when the animals have been pretreated with phenobarbital. In an attempt to further elucidate the role of GSH in chloroform-induced hepatotoxicity, Docks and Krishna (1976) injected chloroform into rats pretreated with microsomal enzyme inducers–phenobarbital, 3-methlcholanthrene, acetone and isopropanal. A dose of chloroform as little as 0.2 mg/kg decreased liver GSH levels and caused centrilobular necrosis within 24 hours in phenobarbital pretreated rats. At a dose of 0.05 ml/kg, chloroform did not decrease liver GSH or cause liver necrosis. When the rats were not pretreated with phenobarbital, a chloroform dose of 0.2 ml/kg caused neither GSH depletion nor necrosis. In this connection, it is interesting to note that cysteine, which is a precursor of GSH and a common amino acid in one's diet, protected the liver from the hepatotoxicity produced by chloroform. The animals were also protected from the hepatotoxic effect by pretreatment with cystamine, not a precursor of GSH, thus suggestive of a mechanism other than of GSH depletion in the hepatotoxicity of $CHCl_3$.

Earlier reports by Ilett, et al. (1973) suggested the possibility of another mechanism involving the formation of an active metabolite of chloroform responsible for the chloroform-induced hepatotoxicity. This study correlated the renal and hepatic necrosis with covalent binding of chloroform metabolites to tissue macromolecule. Bioactivation of xenobiotics including chloroform, involves mixed function enzymes; the NADPH cytochrome reductase-cytochrome P–450 coupled systems. Sipes, et al. (1972) studied the bioactivation of carbon tetrachloride, chloroform and bromotrichloromethane utilizing ^{14}C-labled compounds and rat liver microsomes. The covalent binding of radiolabel to microsomal protein was used as a measure of conversion of the compounds to reactive intermediates. The authors concluded that cytochrome P–450 is the site of bioactivation of these three compounds rather than NADPH cytochrome C reductase. CCl_4 bioactivation proceeds by cytochrome P–450 dependent reductive pathways, while $CHCl_3$ activation, proceeds by cytochrome P–450 dependent oxidative pathways.

The isolation and identification of an active metabolite of chloroform supposedly responsible for toxicity was attempted by Pohl and his co-workers (1977). 2-oxithiazolidine-4-carboxylic-acid, an *in vitro* metabolite of chloroform, and presumably formed by the reaction of cysteine and phosgene ($COCl_2$), was isolated and characterized. When the incubation was conducted in an atmosphere of $[^{18}O]\ O_2$, the trapped $COCl_2$ contained $[^{18}O]$. These findings suggest that C–H bond of $CHCl_3$ is oxidized by a cytochrome P–450 mono-oxygenase to produce trichloromethanol which spontaneously dehydrochlorinates to phosgene. The electrophilic phosgene could react with water to form carbon dioxide, a known metabolite of $CHCl_3$ *in vitro* and *in vivo* or with microsomes to yield a covalently bound product. The *in vitro* oxidation of chloroform and its relationship to chloroform toxicity has been further substantiated by the studies wherein deuterated chloroform was used. Pohl and Krishna (1978) reported that $CDCl_3$ was metabolized slower than chloroform suggesting that the cleavage of C–H bond of chloroform is the rate determining step in the enzymatic process. The observation that $CDCl_3$ is less hepatotoxic than $CHCl_3$ indicates that the cleavage of the C–H bond is also the critical step in the process leading to $CHCl_3$ induced hepatotoxicity. The finding that $CDCl_3$ depletes less glutathione in the liver of rats than $CHCl_3$ suggests the active metabolite phosgene is responsible for the depletion of glutathione.

In the experiments involving the isolation and characterization of metabolites of chloroform, the evidence for the metabolism of chloroform to phosgene *in vitro*, by the oxidative pathway was present. Recent research has indicated the possibility of formation of phosgene *in vivo*. Pohl, et al. (1979), isolated and characterized 2-oxo-thiazolidine-4-carboxylic acid from the liver of rats pretreated with cysteine carboxylic acid after a dose of chloroform and/or deuterated chloroform. In these experiments, deuterated chloroform yielded less amount of metabolite, confirming once again the specificity of the cytochrome P–450 dependent enzymes in the mediation of oxidative dehalogenation of chloroform and its toxicity.

IX. Risk Assessment

The establishment of chloroform as an animal carcinogen, plus the epidemiological data and mutagenesis data on THMs, show that a potential human risk exists from the consumption of THMs, but these data do not quantify the risk. Methods have been developed to estimate the level of risk, based on an assumption that there is no threshold level for the action of a carcinogen. The state-of-the-art at the present time is such that no experimental tools can accurately define the absolute numbers of excess cancer deaths attributable to chloroform in drinking water. Due to the biological variability and a number of assumptions required, each of the risk-estimating procedures leads to a different value. There is wide variation among these estimates and their interpretation.

The EPA Science Advisory Board (SAB) (1975), using the highest levels of chloroform then reported in drinking water by the NORS data (0.300 mg/l) and assuming a maximum daily intake of 4 liters of water for a 70 kg man, attempted to estimate the risk. The estimates were based on the Eschenbrenner and Miller (1945) animal data, which themselves are subject to great variability since the experiments used only 5 animals per sex per dose. Using a linear extrapolation of the animal data over more than 2 orders of magnitude dose from mice to humans at the 0.300 mg/l concentration level, the lifetime incidence for liver tumors in man were estimated to range from 0 to .001 (95% confidence limits) or 0 to 100 γ.

10^{-5} in a lifetime. This rate may be compared with the lifetime incidence of 260×10^{-5} for malignancy of liver derived from the data of the Third National Cancer Survey (1976). This estimate would range from zero to approximately 40% of the observed incidence of liver cancer in the United States that may be attributable to exposure to chloroform in drinking water at the 0.300 mg/l level. It should be noted that this value is at the upper limit of the confidence interval and the linear non-threshold dose-effect model allows an estimate of maximal risk where a risk has actually been observed. Most other models would yield lower estimates. The SAB, however, also stated that a more reasonable assumption would yield lower estimates of the risk.

Tardiff (1976) using four different models, calculated the maximum risks from chloroform ingestion via tap water Using a margin of safety of 5000 applied to the minimum effect animal dose, i.e., the Weil conjecture, the "safe" level was calculated to be 0.2 mg/kg/day. Using the logprobit model and the slope recommended by Mantel and Bryan, the conclusion reached was that at a maximum daily dose of 0.01 mg/kg the risk would be between 0.016 and 0.683 cancers per million exposed population per year. Using the identical data, but with the experimental slope of the dose response curve as found in the mice as opposed to the slope of the one in the previous calculation, the conclusion reached was that a maximum daily dose of 0.01 mg/kg would produce less than one tumor per billion population per lifetime. Using the linear, or one hit model, usually considered to be the most conservative, a risk estimate of between 0.42 and 0.84 cancers per million population per year was calculated to result from a dosage level of 0.01 mg/kg/day. The two step model produced an estimated maximum risk of between 0.267 and 0.283 cancers per million population per year at a dose level of 0.01 mg/kg/day.

In the National Academy of Sciences (1977) report on "Drinking Water and Health," lifetime risks were estimated from the more recent, and much more extensive NCI animal data using a multi-stage model.

For a concentration of chloroform at 1 ug/liter the estimated incremental lifetime cancer risk would fall at approximately 1.7×10^{-6} per microgram per liter at the upper 95% confidence limit, assuming 70 year daily consumption of water at that level. Assuming lifetime exposure at the standard of 0.10 mg/l level in drinking water the incremental risk would be 3.4×10^{-4} assuming two liters of water at 0.10 mg/l consumed daily for 70 years.

In evaluating the risk estimates, it is important to compare the calculated maximum risk with the current cancer mortality data. Both liver and kidney cancer are rare diseases in the U.S. (< 5 per 100,000 population per year). The standardized mortality rates in the U.S. for white males and females combined are 52.5 per million per year for liver cancer and 29.2 per million per year for kidney carcinoma.

Based on the various risk estimates, Tardiff (1976) calculated that the percent of the annual cancer mortality attributable to chloroform in drinking water could be 1.60% and 1.44% for liver and kidney cancer respectively assuming the maximum exposure levels. Applying these percentages to the actual cancer mortality rates, the number of cancer deaths per year would be 168 from liver carcinoma or 84 from kidney carcinoma; an estimated maximum of 252 cancer deaths per year attributable to chloroform in drinking water.

Reitz, Gehring, and Park (1978) discussed EPA's procedures in estimating risk. They stated that EPA "seriously overestimates the actual potential of chloroform * * * (for) two major reasons." These are: (1) The mechanism through which chloroform exerts its toxicity, and (2) reliance on the NCI bioassay protocols which call for high doses of chloroform, and by not conducting studies at lower doses which usually induce relatively less carcinogenicity, there is a likelihood of ignoring a possible detoxification mechanism which protects test animals until they are overwhelmed by very large doses. They also suggest that an experiment to evaluate the carcinogenicity of chloroform at lower doses must be performed before high/low dose extrapolations can be performed. Definitive data do not exist to prove or to disprove the above claims.

The authors indicated that EPA's proposed standard for THMs of 0.10 mg/l in drinking water supplies was based on the carcinogenic risk estimates. It should be pointed out the EPA's proposed standard for THM was based upon that feasibility of achieving the TTHM concentration in drinking water, as well as the potential adverse health effects.

EPA's Office of Water Planning and Standards and Office of Research and Development with EPA's Carcinogen Assessment Group, developed a risk estimate in the draft document, "Chloroform—The Consent Decree Ambient Water Quality Criteria Document" (1979). The method used assumed consumption of 2 liters/per day of drinking water and 18.7 gm/per day of fish and shellfish. The lifetime risk estimates for excess cancers range from 10^{-5}, 10^{-6}, and 10^{-7} with corresponding consumption of 2.1 ug/l, 0.21 ug/l and 0.021 ug/l, respectively. The difference in these risk estimations may be explained by the assumption of daily fish consumption as well as other exposure sources. Without the fish consumption, the equivalent concentrations are 4.8 ug/l and 0.48 ug/l for estimated cancer risk of 1×10^{-5} and 1×10^{-6}, respectively. When this estimate is computed for the concentration of 0.10 mg/l for levels in drinking water, the incremental risk would be 4.0×10^{-4} assuming two liters of water at 0.10 mg/l was consumed daily for 70 years.

At an assumed lifetime exposure of 2 liters of water per day at 0.10 mg/l chloroform the risk reduction to the impacted population was estimated as a range of approximately 200–500 total cases. It should be noted however, that these average exposure levels in the impacted population may result in overestimates of the risk in light of the facts that: (1) The computations are based upon lifetime exposures. In actuality the proposed interim standard will likely be reduced in the future as technologically feasible, and, therefore, the lifetime exposure values will be less. (2) The interim standard encourages maximum reduction obtainable using current technology. A much lower average exposure is likely in the future because technology will most likely improve and result in greater exposure reductions. On the other hand, these may be underestimated because they are based upon toxicity exposure data from chloroform, which is only a portion of the TTHMs, which are only a portion of the by-products of the chlorination process; therefore, the magnitude of the contribution to the risk of the other THMs, which in some cases contribute significantly to TTHMs, is unknown. The exposure to THMs from air and food have not been included in these computations.

X. Selected Maximum Contaminant Levels (MCLs)

Since a risk to the public exists from exposure to TTHMs and other chlorination by-products in drinking water, the potential for that risk should be reduced as much as is technologically and economically feasible without increasing the risk of microbiological contamination. This can be accomplished by several means, and the Safe Drinking Water Act (Pub. L. 93-523) provides two major regulatory

avenues—(1) the establishment of an MCL, or (2) the institution of a treatment equirement.

EPA has determined that the establishment of an MCL in the Interim Primary Drinking Water Regulations, along with monitoring requirements, is the most effective and immediate approach to reducing the levels of THMs in drinking water. The Administrator has determined that monitoring is both technically and economically feasible (refer to "Economic Impact Analysis of a Trihalomethane Regulation for Drinking Water," EPA, 1977). Measures taken to reduce the THM concentrations will concurrently provide the additional benefit of reducing human exposure to the other undefined by-products of chlorination and possibly other synthetic organic contaminants.

Since it is known that chlorination of water is primarily responsible for the relatively high levels of THMs in drinking water, modifications in the chlorination process, the substitution of other disinfectants, and the use of adsorbents and other technologies to remove precursor chemicals are possible approaches to control. The optimal approach would be to reduce organic precursor concentrations prior to addition of a disinfectant in order to reduce disinfectant demand and minimize all by-products.

Use of a chlorine residual in a less active form such as combined chlorine or chloramine will significantly reduce THM formation; however, chloramines are much less potent disinfectants than free chlorine, and therefore, this approach must only be used after careful consideration, and assurance of maintenance of excellent biological quality. The two chemicals most often mentioned as substitute disinfectants, ozone and chlorine dioxide, are both well known as effective disinfectants and chemical oxidants. The issues of the biological effects and toxicity of these disinfectants and their by-products are being clarified by studies underway. In the meantime, EPA recommended that the residual total oxidant levels after application of chlorine dioxide should be limited to 0.5 milligram per liter.

The National Organics Monitoring Survey found that the mean total trihalomethane (TTHM) concentrations in the drinking water systems evaluated were approximately 0.068, 0.117, 0 053 and 0.100 mg/l for Phase I, II, III (dechlorinated) and III (terminal) respectively, with the highest levels of 0.784 mg/l in Phase II (refer to Table 1).

It is reasonable to assume that the various calculated risk estimates for chloroform indicate a potential risk to public health. It is possible that a percentage of the total number of liver and/or kidney cancers are attributable to exposure of chloroform in drinking water, although it is most likely that drinking water exposure would interact with a number of other variables such as smoking and diet as effect modifiers in a multifactorial manner. It is also likely that the other by-products of chlorination also present a potential risk.

Thus, based upon a number of risk extrapolations assuming various levels of exposure to chloroform in drinking water, it has been estimated that such exposures may cause an annual excess of cancers in the U.S. population (ranging from 0 to several hundred). At higher levels of exposure of chloroform (>0.300 mg/l) the cancer risk estimates are even higher.

The reduction of TTHMs to an MCL level of 0.10 mg/l would reduce the unnecessary and excessive exposure to these potential human carcinogens, mutagens, and chronic toxicants, and other effects. At the same time, measures taken to reduce THM levels (such as the use of adsorbents) will concurrently result in reduction of human exposure to other contaminants in drinking water.

Since it is economically and technologically feasible to reduce the THM levels in drinking water, and since benefits are achieved by reducing the health risks of exposure, EPA has decided to establish the MCL at 0.10 mg/l as the initial feasible step in a phased, regulatory approach. As more data become available from implementation experience, and toxicology and epidemiology, standards are expected to become more restrictive. In the meantime, EPA and the States should continue to take steps as necessary on a case-by-case basis to provide adequate protection for the delivery of safe drinking water to the public, by minimizing the amounts of toxic chemicals in the water.

XI. References

Alavanja, M., et al., 1978. "An Epidemiological Study of Cancer Mortality and Trihalomethanes in Drinking Water in 19 New York State Counties" U S EPA, Office of Research and Development, Health Effects Research Laboratory, Cincinnati, Ohio, Unpublished.

Alavanja, M., Goldstein, I., and Susser, M., 1977. "Case Control Study of Gastrointestinal Cancer Mortality in Seven Selected New York Counties in Relation to Drinking Water Chlorination" U.S. EPA, Office of Research and Development, Health Effects Research Laboratory, Cincinnati, Ohio, Unpublished.

Alavanja, M., Goldstein, I., and Susser, M., 1976 "Report of Control Study of Cancer Deaths in Four Selected New York Counties in Relation of Drinking Water Chlorination." U.S. EPA, Office of Research and Development, Health Effects Research Laboratory, Cincinnati, Ohio, Unpublished Draft.

American Conference on Industrial Government Hygienists, 1975. "Threshold Limit Values for Chemical Substance in Workroom Air" Cincinnati, Ohio.

Ames, B. N., W. E. Durston, E. Yamaski, and F D. Lee, 1973. "Carcinogens are Mutagens: A Simple Test System Combining Liver Homogenates for Activation and Bacteria for Detection." *Proc. Nat. Academy of Science*, USA, *70:* 2281–2286.

Bellar, R. A., Lichienberg, J J, and Kroner, R. C, 1974. "The Occurrence of Organohalides in Finished Drinking Waters." *Journal of American Water Works Association, 66:*

Brenniman, G, et al., 1979. "Relationship Between Cancer Mortality and Chlorinated Drinking Water" University of Illinois, Unpublished Draft.

Brown, D. M., Langley, P. F., Smith, D., and Taylor, D. C., 1974. "Metabolism of Chloroform I. The Metabolism of [C] Chloroform by Different Species." *Xenobiotica 4.* 155–163.

Buncher, C. R, 1975. "Cincinnati Drinking Water—An Epidemiologic Study of Cancer Rates." University of Cincinnati Medical Center, Cincinnati, Ohio.

Bunn, W. W., Haas, B B, Deane, E. R., and Kleopfer, R D., 1975. "Formation of Trihalomethanes by Chlorination of Surface Water" *Environmental Letters 10* 205–213

Burreson, C. J., Moore, R. E., and Roller, P. P., 1975. "Volatile Halogen Compounds in the Algae Aspargopis Taxiformis." *J. Agric. Food Chem. 24:* 856–861

Butterfield, C. T. and Wattie, S, 1944. "Influence of pH and Temperature on the Survival of Coliforms and Enteric Pathogens When Exposed to Free Chlorine." *Public Health Rep. 58:* 1837–1866.

Butterfield, C. T and Wattie, S., 1946. "Influence of pH and Temperature on the Survival of Coliforms and Enteric Pathogens When Exposed to Chloramine." *Public Health Rep. 61:* 157–192.

Canter, K, Hoover, R., Mason, T, and McCabe, L., 1978 "Association of Cancer Mortality with Halomethanes." *J. of National Cancer Inst 61:* 979–985.

Chernoff, 1977. "Personal Communication."

Clarke, N. A. and Kabler, P K., 1954. "Inactivation of Purified Coxsackie Virus in Water by Chlorine" *Am J. Hyg. 59* 119–127

Cotruvo, J A, Simmon, V F. and Spanggord, R J, 1977. "Investigation of Mutagenic Effects of Products of Ozonation Reactions in Water." *Annals of the New York Academy of Sciences*, in press, and "Personal Communications."

Cole, P C., Hoover, R, and Friedell, G. H., 1972. "Occupation and Cancer of the Lower Urinary Tract." *Cancer, 29:* 1250–60

DeRouen, T. A., and Diem, J. E, 1975. "Ethnic, Geographical Difference in Cancer Mortality in Louisiana." Tulane University, School of Public Health and Tropical Medicine, Unpublished.

DeRouen, T. A and Diem, J. E., 1975. "The New Orleans Drinking Water Controversy A Statistical Perspective." *American Journal of Public Health, 65.* (No 10) 1060

DeSalva, S., Volpe, A., Leigh, G., and Regan, T., 1975. "Long-Term Safety Studies of a Chloroform-Containing Dentrifice and Mouth Rinse in Man." *Food Cosmet. Toxicol. 13*: 529–532.

Dewey, W., Personal Communication, Medical College of Virginia.

Docks, E.L. and Krishna, G., 1976. "The Role of Glutathione in Chloroform Induced Hepatotoxicity." *Experimental and Molecular Pathology 24*: 13–22.

EPA, 1979. "Chloroform—The Consent Decree Ambient Water Quality Criteria Document." EPA, Office of Water Planning and Standards. Unpublished Draft.

EPA, April 1976. "Industrial Pollution of the Lower Mississippi River in Louisiana." Region VI, Dallas, Texas.

EPA, 1974. "Analytical Report, New Orleans Water Supply Study." Region VI, Dallas, Texas.

EPA, 1975. "A Report—Assessment of Health Risk from Organics in Drinking Water by an Ad Hoc Study Group to the Hazardous Materials Advisory Committee." Science Advisory Board. Unpublished.

EPA, June 1975. "Region V Joint Federal/State Survey of Organics and Inorganics in Selected Drinking Water Supplies." Region V, Chicago, Illinois

EPA, 1976. "Risk Evaluation of Chloroform." Office of Pesticides Programs, Criteria and Evaluation Division. Unpublished.

EPA, 1977. "Chloroform Risk Assessment in Drinking Water." Cancer Assessment Group. Unpublished.

EPA, August 1977. "Economic Impact Analysis of a Trihalomethane Regulation for Drinking Water," prepared by Temple, Barker, and Sloan, Inc. for EPA, Office of Drinking Water.

EPA, June 1976. "Interim Treatment Guide for the Chloroform and other Trihalomethanes." EPA, Water Supply Research Division, Municipal Environmental Research Laboratory, Cincinnati, Ohio.

Eschenbrenner, A.B. and Miller, E., 1945. "Introduction of Hepatomas in Mice by Repeated Oral Administration of Chloroform with Observation on Sex Differences." *J. Nat'l. Cancer Inst. 5* 251–255.

Fry, F.J., Taylor, T., and Hathaway, E.D., 1972. "Pulmonary Elimination of Chloroform and Its Metabolites in Man." *Arch. Int. Pharmacodyn 196* 98–111.

Hefferman, P., 1977, Personal Communication.

Heilbrunn, G., Liebert, E., and Szanto, P.B., 1945. "Chronic Chloroform Poisoning—Clinical and Pathological Report of a Case." *Arch. Neurol. Psych 53*: 68–72.

Hogan, M.D., Chi, P., Hoel, D G., and Mitchell, T.J., 1979 "Association Between Chloroform Levels in Finished Drinking Water Supplies and Various Site-Specific Cancer Mortality Rates." *J. of Env. Path. & Tox. 2*: 873–887.

Ilett, K.F., Reid, W.P., Sipes, I.G., and Krishna, G., 1973. "Chloroform Toxicity in Mice: Correlation of Renal and Hepatic Necrosis with Covalent Binding of Metabilities to Tissue Macromolecules." *Exptl. Molec. Pathol. 19*: 215–229.

Jones, W.M., Marguis, G., and Stephen, C.R., 1958. "Hepatoxicity of Inhalation Anesthetic Drugs." *Anesthesiology 19*: 715–23.

Kelly, S.M. and Sanderson, W.W., 1960. "The Effect of Chlorine in Water on Enteric Viruses. II. The Effect of Combined Chlorine on Poliomyelitis and Coxsackie Viruses." *Am. J. Public Health, 50*: 14–20.

Kubic, V.L., Anders, M.W., Engel, R.R., Barlow, C H., and Caughey, W.S., 1974. "Metabolism of Dihalomethanes to Carbon Monoxide In Vivo Studies." *Drug Metabolism and Disposition 2*: 53–57.

Kruse, C.W., 1977. "Chlorination of Public Water Supplies and Cancer—Washington County, Maryland Experience." A Final Report, John Hopkins University, School of Hygiene and Public Health to the Office of Research and Development, Health Effects Research Laboratory, Cincinnati, Ohio, Unpublished Draft.

Kuzma, R J., Kuzma, C J. and Buncher, C.R., 1977. "Ohio Drinking Water Source and Cancer Rates." *American Journal of Public Health 67*. 725–729.

Lehman, K B. and Hasegawa, 1910. "Studies of the Absorption of Chlorinated Hydrocarbons in Animals and Humans." *Archives of Hygiene 72*: 327.

Mah, R. A., Spivey, G. H., and Sloss, E., 1977 "Cancer and Chlorinated Drinking Water." A Final Report, University of California, Los Angeles, School of Public Health to the Office of Research and Development, Health Effects Research Laboratory, Cincinnati, Ohio, Unpublished.

McCabe, L. J., 1975. "Association Between Halogenated Methanes in Drinking Water and Mortality." EPA, Health Effects Research Laboratory, Cincinnati, Ohio, Unpublished.

McLean, A E. M., 1970. "The Effect of Protein Deficncy and Microsomal Enzyme Induction by DDT and Phenobarbitone on the Acute Toxicity of Chloroform and a Pryolizione Alkaloid, Retrosine." British Journal of Experimental Pathology 51: 317–21.

Meinhardt, T. J., Mareinfield, C. J., Miller, R. S., and Wright, H T., 1975. "River Water and Cancer Mortality." Environmental Health Surveillance Center, University of Missouri, Unpublished.

Michael, G E., et al., 1979. "Chlorine Dioxide Water Disinfection: A Prospective Epidemiologic Study" U.S. EPA, Office of Research and Development, HERL, Cincinnati, Ohio.

NAS, 1978. "Epidemiological Studies of Cancer Frequency and Certain Organic Constituents of Drinking Water—A Review of Recent Literature Published and Unpublished." Washington, D.C.

NAS, 1977. "Drinking Water and Health." Washington, D.C.

NAS, 1977. "Non-Fluorinated Halomethanes in the Environment." Washington, D C.

NAS, 1979. "The Disinfection of Drinking Water." Washington, D.C. Unpublished Draft.

NAS, 1979. "The Chemistry of Disinfectants in Water: Reactions and Products." Washington, D.C. Unpublished Draft.

National Cancer Institute, 1976. "Report on Carcinogenesis Bioassay of Chloroform."

National Institute of Occupational Safety and Health, 1974. "Criteria for a Recommended Standard, Occupational Exposure to Chloroform."

Page, T. and Harris, R. H., 1975. "Relation Between Cancer Mortality and Drinking Water in Louisiana" Unpublished.

Page, T., Harris, R. H., and Estein, S. S., 1976. "Drinking Water and Cancer Mortality in Louisiana." *Science 193*: 55–57.

Page, T., Talbot, E., and Harris, R. H., 1974 "The Implications of Cancer Causing Substances in Mississippi River Water." Environmental Defense Fund, Washington, D C.

Paul, B B. and Rubinstein, D., 1963. "Metabolism of Carbon Tetrachloride and Chloroform by the Tat." *J. Pharm Exp. Therap. 141*: 141–148.

Plaa, G. L., Evancs, E. A., and Hine, G. H., 1963. "Relative Hepatotoxicity of Seven Halogenated Hydrocarbons." *J. Pharmacol. Exp. Therap. 123*: 224–229.

Plaa, G. L. and Larson, R. E., 1965. "Relative Nephrotoxic Properties of Chlorinated Methane, Ethane and Ethylene Derivatives in Mice." *Toxicol. Appl. Pharmacol. 7*. 37–44.

Pohl, L. R., et al., 1977. "Phosgene: A Metabolite of Chloroform." *Biochemical and Biophysical Research Communications*, Bethesda, Maryland.

Pohl, L. R. and Krishna, G., 1978. "Deuterium Isotope Effect in Bioactivation and Hepatotoxicity of Chloroform." *Life Sciences*, Bethesda, Maryland.

Pohl, et al., 1979. "Deuterium Isotope Effect in Vivo Bioactivation of Chloroform and Phosgene."

Poirier, L. A., Stoner, G. D., and Shimkin, M. B., 1975. "Bioassay of Alkyl Halides and Nucleotides Base Analogs by Pulmonary Tumor Response in Strain A Mice." *Cancer Research 35*: 1411–1415.

Rafferty, P., 1979. "Drinking Water Quality and Cancer Mortality Patterns." University of North Carolina, Chapel Hill, North Carolina, Unpublished Masters Thesis.

Reiches, N A., Page T., Talbot, P., and Harris, R H., 1976. "Carcinogenic Hazards of Organic Chemicals in Drinking Water." Unpublished.

Reitz, R H., Gehring, P.J., and Park, C.N., 1978. "Carcinogenic Risk Estimation for Chloroform—An Alternative to EPA's Procedures." *J. Food & Cosmet. Toxicol. 16*: 511–514.

Roe, F.J C., 1976. "Preliminary Report of Long-Term Tests of Chloroform in Rats, Mice and Dogs." Hazelton Laboratories, Vienna, Virginia.

Rook, J.J., 1976. "Formation of Haloforms During Chlorination of Natural Waters." *Water Treatment & Examination 23*: 234–243.

Rook, J.J., 1977. "Chlorination Reactions of Fulvic Acids in Natural Waters." *Env. Sci. Technol. 11*. 478–482.

Salg, J., 1977. "Cancer Mortality Rates and Drinking Water in 346 Counties of the Ohio River Valley Basin." Final Report, University of North Carolina, Department of Epidemiology to the Office of Research and Development, Health Effects Research Laboratory, Cincinnati, Ohio, Unpublished.

Scholler, K.L., 1968. "Electron-Microscopic and Autoradiographic Studies on the Effect of Halomethane and Chloroform on Liver Cells." *Acta Anaesthesioly of Scandinavia 32*: 1–62.

Schwetz, B.A., Leong, B K.J., and Gehring, P.J., 1974. "Embryo and Fetotoxicity of

Inhaled Chloroform in Rats." *Toxicol. Appl. armacol. 28:* 442–451.

Sipes, G.I., Krishna, G., and Gillette, J R., 2. "Bioactivation of Carbon Tetrachloride, loroform, and Bromotrichloromethane: Role of Cytochrome P–450." *Life Sciences,* Bethesda, Maryland.

Southeimer, H., and Kuhn, W., 1977. The Engler-Bunte Institute, University of Karlsruhe, Karlsruhe, Germany. Personal Communication.

SRI International, March 1979. "The Effects of Ozonation Reactions in Water." Volume 1.

SRI International, March 1979. "The Effects of Reactions of Chlorine Dioxide in Water." Volume 2.

Storms, W.W., 1973. "Chloroform Parties." *JAMA, 225:* 160.

Symons, J.M., Bellar, T.A., Carswell, J.K., DeMarco, J , Kropp, K L., Robeck, G.G., Seeger, D.R., Slocum, C.J., Smith, B.L., and Stevens, A.A., 1975. National Organics Reconnaissance Survey for Halogenated Organics. *J. Am. Water Works Assn. 67:* 634–646.

Tardiff, R., 1976 Personal Communication.

Tardiff, R.G , 1976. "Health Effects of Organics. Risk & Hazard Assessment of Ingested Chloroform." The 96th Annual Conference of the American Water Works Association New Orleans, Louisiana.

Tarone, R.E. and Gart, J.J., 1975. "The Implications of Cancer-Causing Substances in Mississippi Rain Water." An unpublished review of the study by R H Harris.

Taylor, D.C , Brown, D M , Keeble, R. and Langley, P.F., 1974. "Metabolism of loroform II. A Sex Difference in the tabolism of [C] Chloroform in Mice." *iobiotica 4:* 165–174.

Theiss, J.C., Stoner, C.D , Shimkin, M B., and Weisburger, E K., 1977. "Test of Carcinogenicity of Organic Contaminants of United States Drinking Waters by Pulmonary Tumor Response in Strain A Mice." *Cancer Research 37.* 2717–2720.

Tuthill, R W. and G Moore, 1978. "Chlorination of Public Drinking Water Supplies and Subsequent Cancer Mortality: An Ecological Time-Lag Study." Division of Public Health, School of Health Sciences, University of Massachusetts, Unpublished.

United States, Department of Health, Education and Welfare, National Institute of Occupational Safety and Health, 1973. Toxic Substance List.

Vasilenko, P. and Magno, L., 1975. "Factors Relating to the Incidence of Cancer Mortality in New Jersey " Princeton University, Princeton, New Jersey, Unpublished

Vocci, F J., Martin, B R , Petty, S., and Dewey, W L., 1977. "Effect of Acute and Subchronic Chloroform Exposure on Chlonergic Parameters in Mouse Brain." *Journal of Toxicology and Applied Pharmacology.*

Wallace, C J , 1959 "Hepatitis and Nephrosis Due to Cough Syrup Containing Chloroform." *Calif Med. 73* 442.

Watanabe, P G , McGowan, G R., Madrid, E O., and Gehring, P J , 1976 "Fate of [C] Vinyl Chloride Following Inhalation posure in Rats." *Toxicol. Appl. Pharmacol.* 49–59.

Weisburger, J.H., 1977. "Social and Ethical plications of Claims for Cancer Hazards." *Medical and Pediatric Oncology 3:* 137–140.

Wilkins III, J.R., and C.W. Kruse, 1978. "Chlorination of Public Water Supplies and Cancer." School of Public Health, Johns Hopkins University, Baltimore, Maryland, Unpublished Doctoral Dissertation.

Von Oettingen, W.F., 1955. "The Halogenated Hydrocarbons: Toxicity and Potential Dangers." Public Health Service No. 414. Washington, D.C., U.S. Government Printing Office

[FR Doc. 79-36442 Filed 11-28-79; 8 45 am]

BILLING CODE 6560-01-M