EPA PROPOSAL TO SET RECOMMENDED MAXIMUM CONTAMINANT LEVELS FOR NINE VOLATILE SYNTHETIC ORGANIC CHEMICALS IN DRINKING WATER (49 FR 24330; June 12, 1984)

40 CFR Part 141

[OW-FRL-2514-3]

National Primary Drinking Water Regulations; Volatile Synthetic Organic Chemicals

AGENCY: Environmental Protection Agency (EPA).

ACTION: Proposed rulemaking.

SUMMARY: This proposed rule under the Safe Drinking Water Act (42 U.S.C. 300f *et seq.*) establishes Recommended Maximum Contaminant Levels (RMCLs) for the following volatile synthetic organic chemicals (VOCs) in drinking water: trichloroethylene, tetrachloroethylene, carbon tetrachloride, 1,1,1-trichloroethane, vinyl chloride, 1,2-dichloroethane, benzene, 1,1-dichloroethylene, and p-dichlorobenene. RMCLs (goals) for non-carcinogens are proposed based upon chronic toxicity data, and RMCLs (goals) for carcinogens are proposed at the zero level. VOCs that are not included in this proposal may be considered for subsequent rulemaking as appropriate.

RMCLs are *non-enforceable health goals* which are to be set at levels which would result in no known or anticipated adverse health effects with an adequate margin of safety. This proposal is the initial stage in rulemaking for the establishment of primary drinking water regulations for the VOCs. Following this proposal, Maximum Contaminant Levels (MCLs) and monitoring/reporting requirements will be proposed when the RMCLs are promulgated. MCLs are *enforceable standards* and are to be set as close to the RMCLs as is feasible and are based upon health, treatment technologies, cost and other factors.

Public comments are solicited on the approach to setting RMCLs as proposed in this notice as well as on the alternatives presented. Specifically, comments are requested on the following: Should the RMCLs for carcinogens be zero or a level of exposure considered to constitute a negligible incremental lifetime risk, say one in one million, based [illegible] carcinogens be established at the limits of analytical detection?

DATES: Written comments should be submitted by September 10, 1984. A public hearing will be held in Washington, D.C. on August 6 and 7, 1984, if needed beginning at 9:00 a.m.

ADDRESSES: Send written comments to Comment Clerk, Criteria and Standards Division, Office of Drinking Water (WH-550), Environmental Protection Agency, 401 M Street, S.W., Washington, D.C. 20460. A copy of the comments and supporting documents will be available for review during normal business hours at the EPA, Room 55EB, 401 M Street, S.W., Washington, D.C. 20460. The public hearing will be held in Room 3906, EPA, 401 M. St. S.W., Washington, D.C. It is requested that anyone planning to attend the public hearing (especially those who plan to make statements) register in advance by calling or writing Ms. Arnetta Davis at 202/382-7575, EPA, WH-550, 401 M St., S.W., Washington, D.C. 20460. Persons planning to make statements at the hearings are encouraged to submit written copies of their remarks at the time of the hearing.

References cited on section VII will be available for inspection at the Drinking Water Supply Branches of EPA's Regional Offices.

I. JFK Federal Bldg., Boston, MA 02203, Phone: (617) 223-6486, Jerome Healy
II. 26 Federal Plaza, Room 824, New York, NY 10278, Phone: (212) 264-1800, Walter Andrews
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Copies of the nine draft health criteria documents will be available for a fee from the National Technical Information Service, U.S. Department of Commerce, 5285 Port Royal Road, Springfield, Virginia 22161. The toll free number is 800/336-4700; local: 703/487-4650.

FOR FURTHER INFORMATION CONTACT: Contact Joseph A. Cotruvo, Ph. D., Director, Criteria and Standards Division, Office of Drinking Water (WH-550), Environmental Protection Agency, 401 M Street, S.W., Washington, D.C. 20460, telephone (202) 382-7575.

SUPPLEMENTARY INFORMATION:

I. Statutory Requirements

The Safe Drinking Water Act (42 USC 300f, *et seq.*) ("SDWA" or "the Act") requires the EPA to establish primary drinking water regulations which: (1) Apply to public water systems; (2) specify contaminants which in the judgment of the Administrator, may have any adverse effect on the health of persons; (3) specify for each contaminant either (a) maximum contaminant levels (MCLs) or (b) treatment techniques. See section 1401(1), 42 U.S.C. 300f. A treatment technique requirement would only be set if "it is not economically or technologically feasible" to ascertain the level of a contaminant in drinking

section 1412, 42 U.S.C. 300g-1. Interim regulations were to be established within 180 days of enactment of the SDWA. Revised regulations are to be developed in two steps: the Agency is to establish recommended maximum contaminant levels (RMCLs) and then establish maximum contaminant levels (MCLs) as close to the RMCLs as feasible. MCLs are to be proposed at the time of promulgation of the RMCLs.

RMCLs are non-enforceable health goals. RMCLs are to be set at a level which, in the Administrator's judgment, "no known or anticipated adverse effects on the health of persons occur and which allows an adequate margin of safety". Section 1412(b)(1)(B). The House Report on the 1974 legislation provides congressional guidance on developing RMCLs:

> • • • the recommended maximum [contaminant] level must be set to prevent the occurrence of any known or anticipated adverse effect. It must include an adequate margin of safety, unless there is no safe threshold for a contaminant. In such a case, the recommended maximum contaminant level should be set at zero level.

House Report No. 93-1185, July 10, 1974, at 20.

MCLs are the enforceable standards. MCLs must be set as close to RMCLs as is feasible. Feasible means "with the use of the best technology, treatment techniques and other means, which the Administrator finds are generally available (taking costs into consideration)." Section 1412(b)(3).

RMCLs in themselves have no impact on public water systems or the public. By promulgating RMCLs, no system is forced to reduce contaminants to this level or to take other action regarding contaminants. RMCLs serve as goals for the Agency in the course of setting MCLs and are therefore initial steps in the MCL rulemaking that will follow. In some cases, the MCLs will be set very close to the RMCLs; in other cases control processes or economic considerations may dictate an MCL that is not as close. Public water systems must comply with the MCL; non-compliance with an RMCL cannot be the basis of an enforcement action under section 1414 of the Safe Drinking Water Act.

In addition, the SDWA specifies that primary drinking water regulations contain criteria and procedures to assure a supply of water that complies with the MCLs (i.e., *monitoring and reporting requirements).* Section 1401(1)(D). Section 1445(a) authorizes EPA to require by regulation any public water supplier to keep records, make [illegible] compliance with the SDWA, in evaluating health risks of unregulated contaminants, or in advising the public of such health risks.

The SDWA also requires that the revised primary drinking water regulations be reviewed every three years and amended whenever changes in technology, treatment techniques or other factors permit greater health protection.

In addition to the regulatory mandates, the SDWA provides authorities for ensuring the safety of the nation's drinking water in a non-regulatory contest. Section 1442(a)(2)(B) authorizes EPA to provide technical assistance to States and publicly owned water systems in response to and alleviation of any emergency situation which the Administrator determines to be a substantial danger to public health. In the absence of appropriate State or local action, section 1431 authorizes EPA to take such actions as the administrator deems necessary to protect public health from a contaminant that may present an imminent and substantial endangerment to the health of persons.

II. Regulatory Framework

The issuance of Revised Primary Drinking Water Regulations is the third step in the evolution of the primary drinking water regulations mandated by the SDWA.

In the *first step*, the National Interim Primary Drinking Water Regulations (NIPDWR) were promulgated on December 24, 1975, with an effective date of June 24, 1977. Amendments were issued in 1976, and 1979 and 1980. See 40 CFR 141. Maximum contaminant levels (MCLs) and monitoring and reporting requirements were set for numerous microbiological, inorganic, organic, and radionuclide contaminants (40 CFR, Part 141, Subpart B). At the direction of the Congress, EPA based the NIPDWR in large part on the 1962 U.S. Public Health Service (PHS) Standards for drinking water which in turn were derived from previous standards dating as far back as 1915 for the microbiological standards and the 1940's for the MCLs for some of the inorganic chemcials.

As the *second step*, section 1412(e) of the SDWA directed EPA to arrange for the National Academy of Sciences (NAS) or an equivalent organization to conduct a study to assess the health effects of contaminants in drinking water and to provide proposals for RMCLs at levels at which there were "no known or anticipated effects on the health of persons • • •," and a list of contaminants whose levels in drinking health of persons. The NAS submitted its initial report, "Drinking Water and Health," to EPA in 1977 which was published in the Federal Register for public comment; four additional reports have been received. While Congress envisioned that the NAS would provide proposals for RMCLs in the report, the NAS stated essentially that it would do toxicological assessments of contaminants in drinking water but that developing proposals for RMCLs was not an NAS responsibility but an EPA regulatory function. In the words of the Academy, "determining safe levels to protect the health of persons' drinking water containing contaminants requires consideration of other factors in addition to the harmful properties of the contaminants" (John S. Coleman, Executive Officer, NAS, Feb. 20, 1975). The NAS reports have provided EPA with toxicological assessments of contaminants in drinking water and based upon this information and data from other scientific sources, EPA is developing the RMCLs.

As the *third step*, section 1412(b)(1)(B) provided that EPA must propose and promulgate National Revised Primary Drinking Water Regulations (NPDWR) that would include RMCLs, MCLs and monitoring and reporting requirements for those contaminants that may have an adverse effect on human health.

Regulatory Development Approach

Development of the NPDWR will be accomplished in four phases:

- Phase I Volatile Synthetic Organic Chemicals,
- Phase II Synthetic Organic Chemicals, Inorganic Chemicals and Microbiological Contaminants,
- Phase III Radionuclides,
- Phase IV Disinfectant By-Products including Trihalomethanes.

In general the approach for all four phases will be similar.

- Initially an ANPRM will be published followed by a comment period and a public meeting. Public technical workshops will also be held. The workshops provide an opportunity for EPA to present the issues that must be addressed in development of the regulations and to receive information on scientific and technical matters as well as receive comments on regulatory approaches.
- RMCLs will then be proposed followed by a public comment period and a public hearing(s).
- RMCLs will then be promulgated and proposals published for MCLs or treatment techniques, monitoring and reporting, and other requirements

will be identified that were used as the basis of determining the MCLs; in addition, generally available treatment technologies (GAT) will be identified for use in compliance with the MCLs and the issuance of variances.

- The MCLs or treatment techniques, monitoring and reporting, and other requirements including GAT will then be promulgated.

An ANPRM for Phase I (VOCs) was issued on March 4, 1982 (47 FR 9350, et seq.), and a public meeting was held in Washington, D.C., on April 28, 1982. In addition, four public technical workshops were conducted across the country (June–August 1982) on volatile synthetic organic chemicals (VOCs) in drinking water.

III. Background and Summary of Comments

The ANPRM identified the VOCs listed below as among those most commonly detected in drinking water based upon data available at that time.

trichloroethylene
tetrachloroethylene
carbon tetrachloride
1,1,1-trichloroethane
1,2-dichloroethane
vinyl chloride
dichloromethane
benzene
chlorobenzene
dichlorobenzene
trichlorobenzene
1,1-dichloroethylene
cis-1,2-dichloroethylene
trans-1,2-dichloroethylene

The purpose of the ANPRM was to solicit comments on the many scientific, technical, legal and economic questions associated with determining the proper approach under the Safe Drinking Water Act (SDWA) to limit human exposure to VOCs.

The ANPRM was published to initiate discussions that would assist the Agency in determining the proper approach under the SDWA for minimizing human exposure to VOCs. The public was invited to comment on the following broad issues:

- What is the significance of contamination of drinking water by VOCs?
- Should national standards be set for VOCs?
- If standards are appropriate, how should levels be established?

In addition to the above broad questions, comments were requested on specific technical and scientific questions. Also, available reference materials on occurrence, health effects, analytical methods, and treatment costs of VOCs in drinking water were

Summary of Public Comments

A total of 136 public written comments were received with the comment period ending on September 30, 1982.

The National Drinking Water Advisory Council (NDWAC) met in Washington, D.C., on September 23–24, 1982, to discuss the VOC ANPRM and its related issues. The NDWAC provided its recommendations to the Administrator in a letter dated January 5, 1983.

Public comments pertinent to this proposal are summarized in this section and in Appendix A. Comments pertinent to proposal of the MCLs and monitoring/reporting requirements will be summarized in that proposal. The public workshops conclusions and recommendations and the NDWAC recommendations are briefly summarized below. As representative of comments received by drinking water industry associations and public interest groups, comments submitted by the American Water Works Association and Natural Resources Defense Council (NRDC), respectively, are also summarized.

Summary of Comments From Public Workshops

Overall, it was concluded that contamination by VOCs is a national problem warranting action. There was sentiment in favor of establishing MCLs and some sort of monitoring program, provided the health effects data are valid and indicate the need to reduce human exposure.

The health effects work groups believed that there are sufficient data to cause concern. Three groups suggested that MCLs be set. However, every group qualified its recommendation by saying, variously, that the data are limited, more studies are needed, and that the difference between genotoxic and non-genotoxic carcinogens should be addressed by EPA.

Aeration and granular activated carbon were identified as generally available technologies, effective in reducing VOC levels to 10 μg/l (micrograms per liter or parts per billion (ppb)) or lower. Cost projections presented by EPA were considered to be reasonable but they should be updated.

The proposed analytical methods were found to be suitably accurate and the best available at this time.

Concerning monitoring, the consensus seemed to be that EPA should provide minimum requirements within which States could develop their own monitoring plans, if data show that VOC contamination can be adequately predicted. EPA would provide criteria predicting which systems were vulnerable to contamination by VOCs and thus be monitored.

American Water Works Association (AWWA)

The AWWA recommended that contaminants be controlled at their source through EPA's existing statutory authorities. They believed MCLs are not appropriate at this time, since "safe" levels of VOCs cannot be determined from existing health-effects data. However, when the health effects data have been evaluated by a recognized independent scientific organization (i.e., National Academy of Sciences (NAS)), the AWWA felt that MCLs should be established if a significant health risk exists.

In the interim, AWWA recommended that national monitoring for specific compound identification should be implemented for all water supplies, preferably using the purge and trap procedure (EPA Method 502.1 or equivalent), but requirements for systems serving less than 10,000 people would be at the discretion of the State. The initial monitoring frequency should be similar to the trihalomethane (THM) rule. In addition, guidance in the form of contamination levels, and action categories for five of the VOCs (i.e., vinyl chloride, trichloroethylene, tetrachloroethylene, carbon tetrachloride, 1,2–dichloroethane) should be established for all water supplies.

Natural Resources Defense Council (NRDC)

The NRDC recommended comprehensive national standards for volatile organic chemicals (VOCs) saying that the occurrence and health effects data show a significant national problem that warrants action under the SDWA. NRDC stated the EPA should establish RMCLs and MCLs for the 14 VOCs addressed in the ANPRM as well as an RMCL and MCL for total VOCs supported by mandatory national monitoring requirements. Other comments by NRDC included:

- Recommended Maximum Contaminant Levels (RMCLs) should be set at zero for carcinogens. RMCLs for non-carcinogens may be set at a no-observed-effect-level with an adequate margin of safety because RMCLs are health goals and are not intended to reflect feasibility of attainment.
- The multi-stage model as modified by the Carcinogen Assessment Group (CAG) should not be used in establishing RMCLs for carcinogens. Mathematical models at best provide crude estimates of the risks resulting

• Calculations of exposure levels corresponding to lifetime cancer risks of 10^{-6} should provide the upper limit for MCLs. That is, contaminant levels should be set at concentrations corresponding to lifetime cancer risks of no greater than 10^{-6}. MCLs for non-carcinogens should be set at correspondingly conservative levels.

NDWAC Recommendations

The National Drinking Water Advisory Council (NDWAC) provided the following recommendations and analyses.

1. The occurrence data derived primarily from the random surveys conducted by EPA and selected data produced by the States in conjunction with the health risk data, warrant establishing controls for 5 of the VOCs found in drinking water. These are: trichloroethylene, tetrachloroethylene, carbon tetrachloride, 1,2–dichloroethane and 1,1,1–trichloroethane.

2. Regulations under the Safe Drinking Water Act should be established for those 5 chemicals at this time. Additional data would be needed before a decision could be made on other volatile organics found in drinking water. Health advisory type guidance should be provided for these compounds in lieu of establishing MCLs.

3. Sufficient animal toxicology does exist at this time for establishing RMCLs for those 5 chemicals noted in 1. above. Quantitative risk calculations using a linearized multi-stage model should be used for establishing RMCLs for the carcinogens. A 1 in 100,000 target risk is recommended as the RMCL. For 1,1,1-trichloroethane, which the current data indicate is not carcinogenic, the RMCL should be calculated from the No Observed Effect Level (NOEL) for neurotoxicity with appropriate safety factors.

4. The analytical methodology for detecting and quantitating VOCs is well established (i.e., EPA Method 502.1 using the Purge and Trap technique and similar procedures). No information was provided to the Council on the availability of laboratory services; however, it is assumed that services would be available to meet ultimate demand. The Council believes that monitoring is technically and economically feasible.

5. Sufficient data exists at this time to determine that granular activated carbon and aeration are "generally available technologies" for central treatment application. Appropriately designed point of use devices, when shown to be effective for VOC control, can also be considered for some small water systems if they are cost/effective and properly managed.

IV. Volatile Synthetic Organic Chemicals in Drinking Water

Hundreds of chemicals have been detected at one time or another in drinking water in the U.S., but the vast majority have been detected infrequently and at very low concentrations. Selection of candidate chemicals for revised national primary drinking water regulations is made from an analysis of data on the occurrence frequency, concentrations detected, size of the exposed populations and the toxicology of the chemicals. This section briefly summarizes the available occurrence data, provides an overview of population exposure estimates, and discusses the health effects data for the VOCs. Additional information can be found in the references listed in section VII.

Occurrence of VOCs in Drinking Water

One or more VOCs have been detected in numerous public water systems across the country. Typically, contamination is at low levels (i.e., less than 1 part per billion, μg/l) but some systems have found higher levels. The VOCs are man-made chemicals, their presence may indicate that a pollution incident has occurred, and some of them are among the most frequently detected contaminants around hazardous waste sites. Several of these chemicals are suspected carcinogens, with differing degrees of evidence, while certain of these are mutagens and/or teratogens in some test systems.

In 1982, EPA conducted a national sampling (Ground Water Supply Survey (GWSS)) of almost 1000 drinking water systems using ground water; 500 were selected at random and 500 were selected by the States as having high potential for VOC contamination (non-random). Table 1 presents results of the random portion of the GWSS. Approximately 21 percent of the systems in the random set had one or more of the VOCs at detectable levels (mostly in the sub μg/l range). The data showed a distinct difference in the frequency of occurrence of VOCs between larger and smaller systems; approximately 28 percent of samples in systems serving over 10,000 detected one or more VOCs in the drinking water whereas 17 percent of samples in systems serving less than 10,000 detected VOCs. Six tenths percent of all public water systems serving less than 10,000 were sampled in the survey whereas 15 percent of systems greater than 10,000 were sampled.

Six national surveys have been conducted by EPA since 1975. These include:

• National Organics Reconnaissance Survey (NORS)
• National Organics Monitoring Survey (NOMS)
• National Screening Program for Organics in Drinking Water (NSP)
• Community Water Supply Survey (CWSS)
• Rural Water Survey (RWS)
• Ground Water Supply Survey (GWSS)

Based upon the above six surveys, projections of national occurrence and human exposure potential for the VOCs are summarized in Table 2 for levels associated with various risk rates. These surveys were conducted for various purposes over an eight year period which saw a rapidly developing state-of-the-art in water analytical methods. Different analytical procedures were used and, consequently, some surveys were able to detect and measure particular VOCs at lower concentrations than other surveys were able to do. The most significant portion of the data base on VOCs, however, is derived from the Ground Water Supply Survey and the Community Water Supply Survey.

In combining the survey data, the national projections of the frequency of occurrence of VOCs at various concentrations can be provided only for those concentrations at or above the level at which all of the surveys were capable of detecting and measuring them. This level, referred to as the lowest common quantifiable concentration, is generally the highest detection limit or minimum quantifiable concentration from among the surveys that are combined. Table 2 shows the estimated frequency of occurrence of the VOCs at or above the lowest common quantifiable concentration. Individual surveys using detection limits or minimum quantifiable concentrations less than the lowest common quantifiable concentration may report a higher frequency of occurrence of some VOCs. For example, according to Table 2, 3.6% of the nation's ground water supplies are projected to have trichloroethlyene at or above the lowest common quantifiable concentration of 0.5 μg/l, whereas the GWSS (random sample), using a minimum quantifiable nominal concentration of 0.2 μg/l, reported trichloroethylene to be present in 6.4% of the supplies sampled (Table 1). (Note: The GWSS random sample was found to have 4.1% at or above 0.5 μg/l.)

TABLE 1.—Summary of GWSS Occurrence Data

[Random sample: n = 466]

Parameter	Quantification limit µg/l	Positives		Median of + µg/l	Max µg/l
		No.	Percent		
Tetrachloroethylene	0.2	34	7.3	0.5	23
Trichloroethylene	.2	30	6.4	1	78
1,1,1-Trichloroethane	.2	27	5.8	.8	16
1,1-Dichloroethane	.2	18	3.9	.5	3.2
1,2-Dichloroethylenes (cis and/or trans)	.2	16	3.4	1.1	2
Carbon tetrachloride	.2	15	3.2	.4	16
1,1-Dichloroethylene	.2	9	1.9	.3	6.3
m-Xylene	.2	8	1.7	.3	1.5
o-+p-Xylene	.2	8	1.7	.3	.9
Toluene	.5	6	1.3	.8	2.9
1,2-Dichloropropane	.2	6	1.3	.9	21
p-Dichlorobenzene	.5	5	1.1	.7	1.3
Bromobenzene	.5	4	.9	1.8	5.8
Ethylbenzene	.5	3	.6	.8	1.1
Benzene	.5	3	.6	3	15
1, 2-Dichloroethane	.5	3	.6	.6	1
Vinyl chloride	1	1	.2	1.1	1.1
1, 2-Dibromo-3-chloropropane	5	1	.2	5.5	5.5
1, 1, 2-Trichloroethane	.2	0			
1, 1, 1, 2-Tetrachloroethane	.2	0			
1, 1, 2, 2-Tetrachloroethane	.5	0			
Chlorobenzene	.5	0			
n-Propylbenzene	.5	0			
o-Chlorotoluene	.5	0			
p-Chlorotoluene	.5	0			
m-Dichlorobenzene	.6	0			
o-Dichlorobenzene	.5	0			
Styrene	.5	0			
Isopropylbenzene	.5	0			

TABLE 2.—APPROXIMATE PERCENT OF GROUND WATER SYSTEMS AND SIZE OF POPULATION PROJECTED TO EXCEED THE NOMINAL INDICATED RISK LEVEL

Substance	Risk level	Drinking water		Nearest drinking water concentration for which data are available µg/l	Percent of systems	Population exposed (thousands)
		Concentration	Risk level (µg/l)			
Trichloroethylene	10^{-6}	2.8	[1] 1.8	0.5	3.4	6,620
	10^{-5}	28	18	20	.4	510
	10^{-4}	280	180	100	.1	40
Tetrachloroethylene	10^{-6}	1		.5	3.8	4,270
	10^{-5}	10		10	.5	440
	10^{-4}	100		80	0	0
Carbontetrachloride	10^{-6}	.4	[1] 0.27	.5	1.8	1,100
	10^{-5}	4	2.7	5	.3	160
	10^{-4}	40	27	40	0	0
Benzene	10^{-6}	.67		.5	1.5	1,000
	10^{-5}	6.7		5	.4	210
	10^{-4}	67		70	0	0
1,1-Dichloroethylene	10^{-6}	.23	[1] 0.24	.2	2.6	1,810
	10^{-5}	2.3	2.4	.5	.1	70
	10^{-4}	23	24	20	0	0
1,2-Dichloroethane	10^{-6}	.95	[1] 0.5	.5	.3	1,230
	10^{-5}	9.5	5	5	0	0
	10^{-4}	95	50	20	0	0
Vinyl chloride	10^{-6}	1	[1] 0.015			
	10^{-5}	10	0.15			
	10^{-4}	100	1.5	1	.06	160
1,1,1-Trichloroethane [2]	10^{-6}	21.7		10	.3	270
	10^{-5}	217		100	.01	180
	10^{-4}	2,170				
p-Dichlorobenzene		[3] 75				

[1] Recent draft calculations by EPA's Carcinogen Assessment Group.
[2] Preliminary data; non carcinogenic Adjustable Acceptable Daily Intake (AADI) is 1,000 µg/l; proposed RMCL is 200 µg/l. Not considered in this proposal as a carcinogen. Developing data may change this classification. See text.
[3] Random sample found 5 occurrences all below this level.

Estimating the occurrence of VOCs as a class in public water supplies is difficult because not all of the six surveys looked for all the listed VOCs and because the detection limits or minimum quantifiable concentrations for specific VOCs varied from one survey to another. However, some insight to the overall occurrence of VOCs can be gained from analyses of the data from the GWSS and CWSS. As shown in Table 3, in the GWSS, 99 of 466 (21.2%) randomly selected ground water supplies had at least one of the 2 VOCs identified in that survey. In the CWSS, 50 of the 330 (15.2%) ground water supplies had at least one of 10 VOCs identified in that survey; 14 of 10 (13.2%) surface water supplies were found to have one or more of the VOCs present.

Occurrence of VOCs at levels above µg/l appears to be more likely in groun water rather than surface water; however the detection frequencies may be similar. Virtually all persistent occurrences of VOCs above 50 µg/l are expected to be in ground water. However, the frequency of specific VOCs occurring above that higher leve is expected to be much less than 1%.

Table 3 also provides data on multip occurrences of VOCs; 44 of 466 (9.4%) randomly selected sites in the GWSS ha measurable levels of two or more VOCs, while 19 of 330 (5.8%) of the ground water supplies in the CWSS ha two or more present.

TABLE 3.—Summary of Single and Multiple Occurrence of VOCs as a Class

No. of contaminants	GWSS [1]	CWSS [2]	
	Random [3]	Ground water [4]	Surface water [5]
0	367(78.8%)	280(84.9%)	92(86.8%)
>1	99(21.2%)	50(15.2%)	14(13.2%)
>2	44 (9.4%)	19 (5.8%)	5 (4.7%)
>3	26 (5.6%)	6 (1.8%)	1 (0.9%)
>4	14 (3.0%)	4 (1.2%)	0
>5	6 (1.7%)	2 (0.6%)	0
>6	4 (0.9%)	0	0
>7	2 (0.4%)	0	0
>8	0	0	0

[1] Based on analyses for 29 VOCs.
[2] 466 supplies studied.
[3] Based on analyses for 10 VOCs.
[4] 330 supplies studied.
[5] 106 supplies studied.

Table 4 shows the frequency of occurrence of supplies with total concentrations of the 29 VOCs examined in the GWSS (random sample) above the indicated levels.

In addition to the EPA national survey data, numerous incidents of contamination have been reported by States across the country, and contamination in some public water wells has been in the range of 100 µg/l to 1,000 µg/l and higher. Usually when concentrations in that range have been detected, corrective measures have been rapidly taken; this could explain the relatively small number detected in the random surveys.

Several States, including California, Michigan, New York, and Connecticut, have monitored comprehensively for VOCs while others have generally responded to incidents of contamination. Table 5 summarizes State data that were available to EPA. The estimates of population exposed to VOCs in Table 2 are based only on the data from the EPA surveys; the State data and miscellaneous information were not included because those data were only from a few States and therefore not geographically representative. Furthermore, since much of the State data were obtained in response to incidents of recognized contamination problems, these data may not be representative of typical conditions existing nationally. However, while these data were not used for computing the national projections, they (including the GWSS non-random data) do provide a valuable and necessary perspective for evaluating those projections.

TABLE 4.—CUMULATIVE OCCURRENCE OF SUPPLIES IN THE GWSS RANDOM SAMPLE WITH TOTAL CONCENTRATION OF 29 VOC'S ABOVE THE INDICATED LEVELS

Total number of supplies sampled	>Minimum quantifiable concentrations	>5 µg/l	>10 µg/l	>50 µg/l	>100 µg/l
466	99 (21.2 percent)	20 (4.3 percent)	12 (2.8 percent)	2 (0.4 percent)	0.

TABLE 5.—SUMMARY OF STATE OCCURRENCE DATA [1]

Parameter	Number of States	Number of samples	Number of positives	Max (ppb)
Tetrachloroethylene	17	3,636	628	1,000
Trichloroethylene	19	4,228	624	510,000
1,1,1,-Trichloroethane	16	3,3330	715	2,250
1,2-Dichloroethylenes (cis and/or trans)	13	1,249	197	860
Carbon tetrachloride	15	2,645	368	1,300
Benzene	2	646	4	17
1,2-Dichloroethane	15	2,628	177	2,100
Vinyl chloride	9	1,793	126	380

[1] The State data are not a comprehensive data base. The data represent a collection of available data from various State agencies, are normally in response to contamination incidents, and are not considered to be statistically representative of national occurrence. In addition, not all the data are from public water systems since private and industrial wells are included in some cases.

Occurrence and Exposure Assessment

As part of the basis for determining how to reduce human exposure to VOCs and determine the appropriate regulatory actions, the occurrence data on VOCs are used in two principal areas. As input to the health risk assessment of the VOCs, an estimate is

in the United States exposed to various levels of the VOCs in drinking water from public water supplies. Information on Dietary intake and respiratory intake from ambient air is provided and is used to estimate the relative contributions of the three sources, particularly of drinking water, to the total dose received by individuals. While it is recognized that some individuals may be exposed to the VOCs from other sources, such as occupational settings or the use of particular consumer products, these analyses are limited to drinking water, food and air because these are the major exposure routes common to all individuals.

In addition to serving as an input to the health assessment, the exposure assessment supports EPA efforts to estimate the economic impact of the regulatory alternatives being considered. To aid in that effort, projections are provided to estimate the number of public water supplies of various water source and system size categories likely to have VOCs present, and the distribution of the VOCs levels in those water supplies.

There are approximately 60,000 public water supplies in the United States. These systems fall into two major categories according to water source (i.e., surface water and ground water) and for purposes of estimating the potential regulatory impact are divided into eleven size categories according to the number of individuals served.

Probability distributions for computing the expected number of systems with concentrations in specified intervals were examined and tested by statistical significance procedures. Ideally, separate probability distributions should be developed for each water soure and system size category: however, the available data were too limited for this. Therefore, it was necessary to consolidate some of the size categories to have sufficient data for developing the probability distribution. Specifically, for ground water it was necessary to collapse the data into two size categories: less than 10,000 people served and 10,000 or more people served. For surface water, there were insufficient data for statistical analysis even when all size categories were combined. The delta distribution was found to be reasonable for the available data and was used for determining the probability of contamination at various levels within the two ground water size categories. For completing the national estimates for ground water, it was assumed that the probability distribution function

category was directly applicable to each of the systems in a particular source/size category. Concentrations of VOCs within a given interval were calculated as the product of the probability associated with the interval and the total number of systems in that source/size category.

As noted previously, Table 2 summarizes the estimated population exposures at various levels of contamination. Details of the data base used in these projections for each of the VOC's can be found in the occurrence documents referenced in section VII.

Human Health Considerations

The underlying principles used to assess the potential health risks of exposure to chemicals are discussed in this section. Brief summaries of the toxicology of each selected VOC are also provided. A more detailed evaluation of the health effects of the chemicals is given in the individual health criteria documents referenced in section VII.

Development of RMCLs for Non-carcinogens

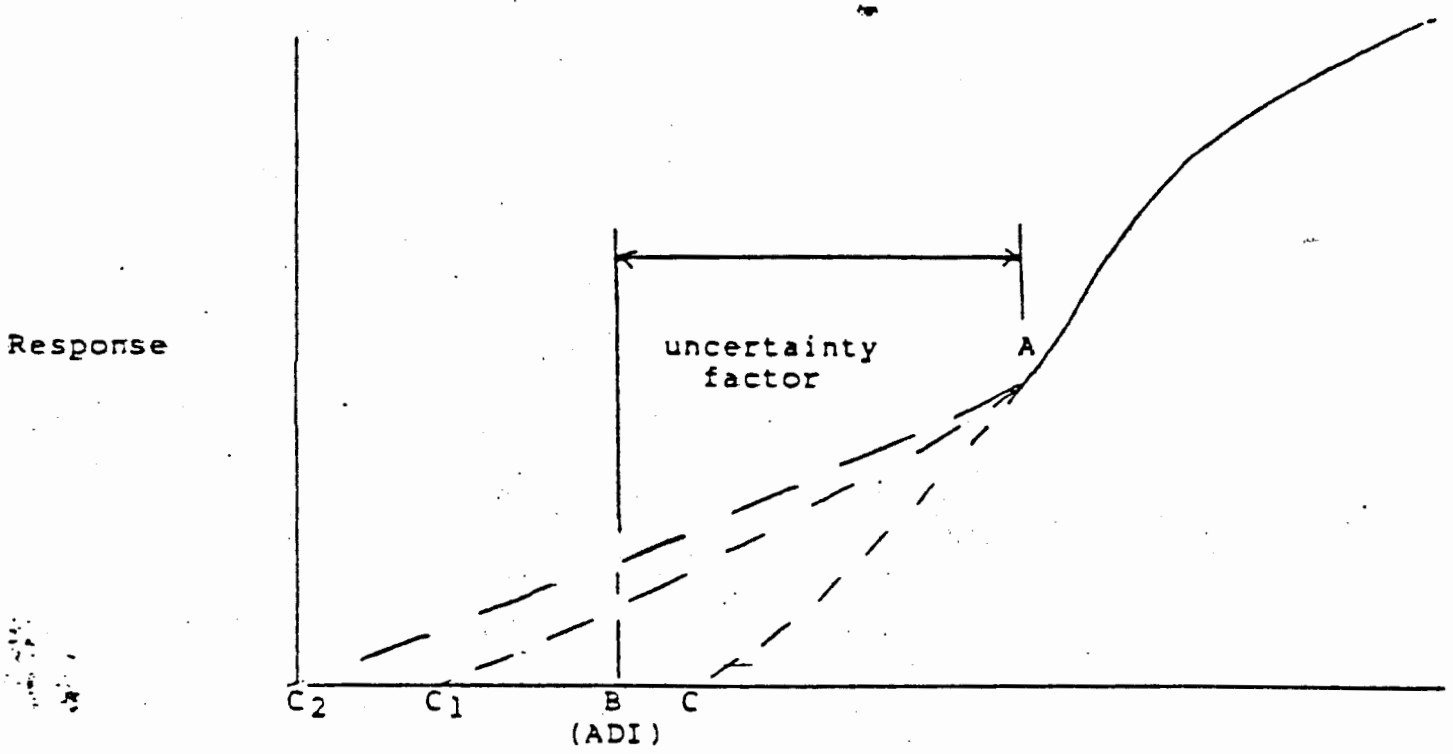
When appropriate data are available from human epidemiology or animal studies, determination of the "no known or anticipated adverse effect levels" for RMCL purposes for toxic agents not considered to have carcinogenic potential is a relatively well-accepted procedure. "No effect" levels for chronic or lifetime periods of exposure including a margin of safety are referred to commonly as ADIs or Acceptable Daily Intakes. These ADI's are considered to be exposure levels which would be without significant risk to humans when received daily over a lifetime. For non-carcinogenic end-points of toxicity, it is assumed that an organism can tolerate and detoxify some amount of a toxic agent without ill effect up to a certain does or threshold. As the threshold is exceeded, the extent of the response will be a function of the dose applied and the length of time exposed.

The intent of a toxicological analysis performed as part of the regulatory development process is to identify the highest no-observed-adverse-effect-level (NOAEL) based upon assessment of human or animal data (usually from animal experiments). To determine the ADI or "no effect" level, the NOAEL is divided by appropriate "uncertainty" or "safety" factors. This process makes accommodations for the extrapolation of animal data to the human, for the existence of weak or insufficient data and for individual differences in human sensitivity toxic agents, among other factors. General guidelines were provided by the NAS Safe Drinking Water Committee (*Drinking Water and Health*, Vol. I) which state that an uncertainty factor of 10 is used if there exist valid experimental results via ingestion in humans; an uncertainty factor of 100 is used if there exist valid experimental results on long-term feeding studies on experimental animals; and an uncertainty factor of 1000 is used if there exist inadequate animal data. Additional factors and variations also may be used if the circumstances dictate it.

Figure 1 illustrates a process by which an ADI for humans is compiled. Figure shows the lower end of a typical sigmoid-shaped dose-response curve as might be generated experimentally for a non-carcinogenic end-point of toxicity believed to have a threshold. The solid line represents the curve as experimentally-determined. Point A represents the highest NOAEL determined during the experiment. Point C represents the theoretical threshold dose at or above which an adverse effect might occur in the most sensitive case.

Figure 1

Non-Carcinogenic Effect



A: NOAEL (experimentally derived)

B: ADI or "no effect" level

C: Presumed threshold for adverse effect

C_1: Another possible presumed threshold for adverse effect

C_2: Non-threshold end point of toxicity

To derive the human "no effect" level or ADI based upon the experimentally-derived data displayed in Figure 1, the appropriate margin of safety (i.e., uncertainty factor) is applied to establish an acceptable level of exposure, depicted as Point B. The objective of applying the uncertainty factor is to make Point B fall below Point C. Thus, Point B would represent the ADI or "no effect" level with a margin of safety. It is possible that the actual dose response curve would result in Point C_1 not detected in the experiment, in which case the calculated ADI (i.e., Point B) might not be below the actual threshold for an adverse effect.

There is suggestive scientific evidence available to postulate that thresholds do exist for non-carcinogenic end-points of toxicity. In the absence of irrefutable evidence, however, it remains theoretically possible that one or more non-carcinogenic end-points may not have a demonstrable threshold. The dose-response curve for this case is depicted as the dashed line from Point A to the origin or C_2. C_2 represents the threshold dose and the "no effect" level in this case would thus be zero.

Table 5 summarizes the suggested Adjusted Acceptable Daily Intakes (AADIs) for the VOCs based upon chronic toxicity data without consideration of the potential carcinogenic risk. These values were not used for developing proposed RMCLs

for chemicals considered to be potential carcinogens, but are provided to add some perspective on the chemical's total toxicity including potential non-carcinogenic end-points.

In addition, these values may have some practical application as guidance on the levels at which no adverse health effects would be expected to occur based upon non-carcinogenic data. This would be especially useful for substances considered to be "weak" carcinogens. Comment is requested on these values.

The AADI's were calculated by:

- Determining the highest No-Observed-Adverse Effect Level (NOAEL), or the lowest observed adverse effect level (LOAEL) in mg/kg body weight/day,
- Dividing by appropriate safety or uncertainty factor(s) (U.F.),
- Multiplying by the weight of an adult (70 kg), and
- Dividing by the amount of water consumed by an adult per day (2 liters/day). (This allocates the ADI totally to drinking water which would have to be modified to consider other routes of exposure when the RMCL or MCL is computed.) The formula for this calculation is as follows:

$$\frac{\text{(NOAEL in mg/kg/day) (70 kg)}}{\text{U.F.(s)} \times \text{2 liters/day}} = \text{AADI mg/l}$$

TABLE 6.—SUGGESTED ADJUSTED ACCEPTABLE DAILY INTAKE: VOC'S

(Does not consider carcinogenicity and excludes contributions from air and food)

Compound	AADI
Tetrachloroethylene	0.085 mg/L
Trichloroethylene	0.26 mg/L
Carbon tetrachloride	0.025 mg/L
1,1,1-Trichloroethane	1.0 mg/L
1,2-Dichloroethane	0.26 mg/L
Vinyl chloride	0.06 mg/L
Benzene	0.025 mg/L
1,1-Dichloroethylene	0.35 mg/L
p-Dichlorobenzene	3.75 mg/L

The calculated AADIs above assume that the total exposure was from drinking water. Since normally exposure also comes from air and food, in addition to drinking water, and since drinking water is frequently a minor contributor to the total exposure, the RMCL or MCL should be modified to take into account the relative source contributions. The World Health Organization, in "Guidelines for Drinking Water Quality" (1983), assigned as little as 1 percent of the ADI to drinking water where the chemical was known to bioaccumulate to a high degree, while greater proportions were assigned where the chemical was known to bioaccumulate to a lesser degree. In "Drinking Water and Health" (1977), the National Academy of Sciences provided projections of 1 percent and 20 percent as illustrations of drinking water contributions. In the National Interim Primary Drinking Water Regulations for six organic chemicals, drinking water was assumed to contribute 20 percent of the total daily intake.

Because of the wide range of environmental exposure distributions that would occur across urban and rural populations as well as because of age and occupationally-related differences, assumption of a 20 percent contribution from drinking water would be reasonably conservative and protective. Thus, in this case, if an AADI value for a non-carcinogen were to be the basis for an RMCL, it would be reduced by 80 percent to account for up to 20 percent contribution from drinking water to the total daily burden.

Development of RMCLs for Carcinogens. Evaluations of the toxicology of substances which may possess carcinogenic potential is a two-phase process. In the first phase, the toxicological data base for non-carcinogenic end-points of toxicity was evaluated in the same manner as described above for "non-carcinogens" (Table 6). In the second phase, assessment was made of the evidence of the carcinogenic potential (e.g., long-term bioassays in rodents and human epidemiology) as well as information which provides indirect evidence (e.g., mutagenicity and other short-term test results). This process is complex since the production of cancer probably is a multi-stage event, determined by a multiplicity of mechanisms, the nature of which remain, for the most part, hypothesized rather than identified.

To date, scientists have been unable to demonstrate experimentally a threshold of effect for "carcinogens," acording to the 1977 report of the NAS Safe Drinking Water Committee. This leads to the assumption that since no threshold dose can be demonstrated for carcinogens, any exposure might represent some finite level of risk. Depending upon the potency of the specific carcinogen and the level, such a risk could be vanishingly small at very low doses.

Human epidemiology data are extremely limited in their ability to identify carcinogenic risks. Thus, animal experiments are conducted from which potential human risk is extrapolated. In the first volume of *Drinking Water and Health* (1977), the NAS Safe Drinking Water Committee provided principles to serve as guidance to EPA when assessing the irreversible effects of long-term exposure to non-threshold substances at low doses:

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: Material should be assessed in terms of human risk, rather than "safe" or "unsafe".

Tumors appear spontaneously in experimental animals, at different rates and different sites depending upon the species and strain. It is unlikely that an increased tumor incidence could be detected following exposure of experimental animals to most carcinogens at dose levels occurring in the ambient environment. Very large numbers of animals would be required to distinguish between treated and control groups. It is possible, as was shown in the 24,000 animal "mega-mouse" study on 2-acetylaminofluorene at the National Center for Toxicology Research (NCTR), that a definitive answer would not necessarily be forthcoming at the low dose levels. Mathematical extrapolation still would be required to project human risk. Relying on this type of study for individual assessments is impractical because of its great expense and lingering scientific uncertainty.

In order to produce quantitative estimates, the assumption has been made that estimated excess cancer risk in humans at low dose levels can be extrapolated using various technique from results observed in animals at high dose levels. Conventionally, designed carcinogenicity bioassay studies are conducted using both sexes of two species of test animals (usually rat and mouse) with each group of 50 animals exposed at the maximum tolerated dose or one-half the maximum tolerated dose. In addition to the possible existence of thresholds, other sources of uncertainty in high to low dose extrapolation include: (1) heterogeneity of sensitivity in the exposed populations, (2) the pharmacokinetic behavior of the test agent in animals vs. the human and (3) mechanisms of action (i.e., whether the agent initiates the process or acts at a later stage). Classification of carcinogens into genotoxic vs. non-genotoxic carcinogens based on possible mechanisms has also been consid-

but a scientific consensus has not been achieved. Fundamental changes in normal cells are the most probable basis for the conversion of normal cells to cancer cells; however, the nature of these changes and how they are brought about is still a scientific uncertainty. Many scientists believe that the most likely mechanism involves direct alteration of DNA by carcinogens. Many carcinogens are capable of altering DNA; chemically-induced alteration of DNA in germinal cells can also cause heritable changes, or mutations; thus, when a chemical shows a positive response in short-term mutagenicity tests, there is concern that it could also be a carcinogen. Scientists also generally believe that cancer results from a multi-stage process. However, these processes are not well understood and available evidence is insufficient to differentiate between carcinogens on the basis of mechanism (IARC, 1983). Therefore in this proposal EPA did not make a differentiation based upon potential mechanisms.

Thus, quantitative risk extrapolation procedures can provide only a rough projection of carcinogenic hazard because of the many unknown factors which enter into these estimates. Models using different assumptions may produce estimates ranging over several orders of magnitude. Since there is currently no way to demonstrate the accuracy of any model at low doses, this process is a subject of debate in the scientific community. However, in spite of these difficulties, quantitative risk estimation does provide the decision-maker one means of setting priorities among pollutants and some gauge of the potential seriousness of environmental hazards (see NCI Subcommittee report referenced in section VII).

EPA's Carcinogen Assessment Group employs a multi-stage model among various others to extrapolate potential excess cancer risk expected at doses of the chemical found in the environment from results in high dose animal studies (U.S. EPA, 1980). Equivalent human doses are established either on a body weight basis (mg/kg) such that the ratio of human to animal body weights is raised to the ⅓ power:

$$\left[\frac{\text{human body weight}}{\text{animal body weight}}\right]^{1/3}$$

or on a body surface area comparison.

The multi-stage model is used for several reasons: (1) it is more systematic than the one-hit model, (2) it invokes fewer arbitrary assumptions, (3) the assumption of low dose linearity is not essential in the use of the model and (4) it incorporates data from all of the dosage groups which are consistent with the multi-stage model. At the same time, it is conceptually consistent with the linear, non-threshold concept. With this model, CAG estimated the upper bound excess cancer risk rate at a specific exposure level for a 70 kg adult who consumes 2 liters of drinking water per day, every day over a 70 year lifespan.

These calculated risk rates have associated uncertainties. This uncertainty has many sources, including such uncertainties as the shape of the dose-response relationship at low doses, differences in responses between humans and laboratory animals, and the effects of artificial dosing regimens. A relatively minor source of uncertainty is statistical fluctuation that results from the finite sample size necessarily used in any experiemental study. This is the only uncertainty that can be readily quantified; it is expressed in EPA's methodology by giving the upper-95% confidence limit of the observed response. Other confidence limits could also be calculated. (In more technically precise terms, the confidence limit is calculated on the coefficient of the linear term in the multi-stage model, assuming that all the statistical uncertainty is loaded on that term.)

Excess cancer risk rates also can be projected using variations within a specific model or other models, such as the one-hit model, the Weibull model, and logit and probit models. There exists no solid basis in the current understanding of the biological mechanisms involved in cancer to say that one model provides a better estimate of the true risk. The estimates of risk at low doses for these models can differ by several orders of magnitude. However, the linear non-threshold model usually has the best, even if limited, scientific biological basis of any of the currently available models for giving an upper limit estimate. The multi-stage model is presumed to usually give a conservative risk estimate (i.e., less likely to underestimate the actual risk) and thus would usually be consistent with a protective regulatory philosophy. A similar model was used by the NAS Safe Drinking Water Committee in the calculations provided to EPA in "Drinking Water and Health". The NDWAC recommended that the multi-stage model be used in the estimation of cancer risk associated with the VOCs. Various calculations using multi-stage models are presented in Table 7.

Shown along with the risk estimates in Table 7 is a qualification of the degree of evidence of carcinogenicity exhibited by the chemicals. The International Agency for Research on Cancer (IARC) provides guidance for categorizing chemicals having sufficient or limited evidence of carcinogenicity. In the IARC Monographs Supplement =1 the definition for sufficient evidence for carcinogenicity indicates that there need be an increased incidence of malignant tumors: (a) In mulitple species or strains, or (b) in multiple experiments, or (c) to an unusual degree with regard to incidence, site or type of tumor, or age at onset. Sufficient evidence of *human* carcinogenicity indicates a causal association between exposure and human cancer. *Limited evidence* of carcinogenicity means that the data suggest a carcinogenic effect but are limited because: (a) The studies involve a single species, strain, or experiment; of (b) the experiments have an inadequate period of follow-up, poor survival, too few animals, or inadequate reporting; or (c) the neoplasms produced often occur spontaneously or are difficult to classify as malignant by histological criteria alone. Limited evidence of *human* carcinogenicity indicate a possible carcinogenic effect in humans, although the data are not sufficient to demonstrate a causal association. In general, although a single study may be indicative of a cause-effect relationship, confidence in inferring a causal association is increased when several independent studies are concordant in showing the association, when the association is strong, when therer is a dose-response relationship, or when a reduction in exposure is followed by a reduction in the incidence of cancer.

The National Academy of Sciences in their report, *Drinking Water and Health*, Vol. I, (1977) classified chemical carcinogens into four categories: human carcinogens, suspected human carcinogens, animal carcinogens and suspected animal carcinogens.

Figure 2 presents a typical dose-response curve for animal experiments dealing with carcinogens. Usually only two data points are available either from an NTP bioassay or other chronic study. Points A_1 amd A_2 represent the tumor incidence observed in the animal experiment at the high and low dose levels, respectively. Point B represents the mathematically extrapolated tumor incidence esimated to occur at an exposure level below those experimentally applied. This exposure level would correspond to a level likely to exist in the ambient environment (usually far below the experimental

dose). Identification of this point (B) and others along the extrapolated lower end of the curve then allows for the projection of an associated excess human carner risk.

TABLE 7.—CANCER RISK ESTIMATES FOR VOCs; PROJECTED UPPER LIMIT LIFETIME CANCER RISKS

Compound	Projected upper limit excess lifetime cancer risk	Concentration in drinking water (µg/l) CAG	CAG *	NAS	Quality of evidence **
Trichloroethylene	10^{-5}	28	18	45	Limited (animal).
	10^{-6}	2.8	1.8	4.5	
Tetrachloroethylene	10^{-5}	10		35	Limited (animal).
	10^{-6}	1		3.5	
Carbon tetrachloride	10^{-5}	4	2.7	45	Sufficient (animal).
	10^{-6}	0.4	0.27	4.5	
1,2-Dichloroethane	10^{-5}	9.5	5.0	7.0	Sufficient (animal).
	10^{-6}	0.95	0.5	0.7	
Vinyl chloride ††	10^{-5}	20	0.15	10	Sufficient (human).
	10^{-6}	2	0.015	1	
1,1-Dichloroethylene	10^{-5}	2.3	2.4		Limited *** (animal).
	10^{-6}	0.23	0.24		
Benzene ††	10^{-5}	6.7			Sufficient (human).
	10^{-6}	0.67			
1,1,1-Trichloroethane (1)	10^{-5}	217		168	Limited **** (animal).
	10^{-6}	21.7		16.8	
p-Dichlorobenzene					Inadequate.

† 95% confidence limit.

†† Vinyl chloride and benzene classified as human and suspected human carcinogens, respectively, by NSA. Both have been classified as human carcinogens by IARC (1982).

CAG = EPA Carcinogen Assessment Group; NAS = National Academy of Sciences Safe Drinking Water Committee; IARC = International Agency for Research on Cancer.

* Recent draft updated calculations by CAG.

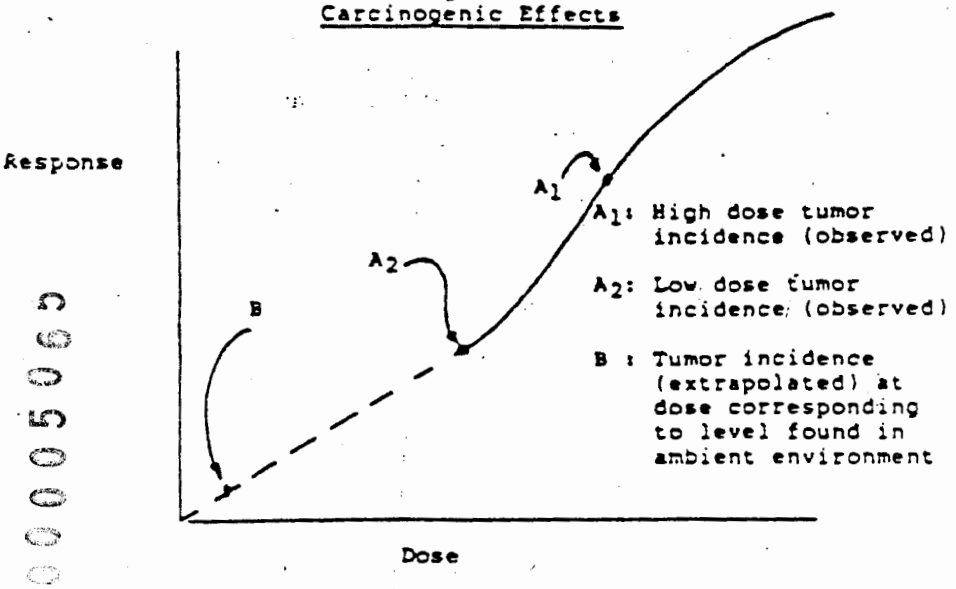
** Based upon IARC unless otherwise noted. Indicates strength of evidence as an animal carcinogen.

*** Assessment made by EPA Carcinogen Assessment Group (CAG), and IARC.

**** Limited evidence as determined by the NAS Safe Drinking Water Committee (1983) and CAG (1983) from preliminary data.

(1) Not considered in this proposal as a carcinogen. Risk estimates are provided for perspective. Developing data may change this classification. See text.

Figure 2
Carcinogenic Effects



Toxicology of VOCs

The following are short discussions of the toxicity of VOCs for which RMCLs are proposed. Detailed assessments are found in the draft health criteria documents that have been prepared for each VOC and are provided for public comment; see section VII, References.

Trichloroethylene. Trichloroethylene has been shown to exhibit non-carcinogenic bioeffects at high (non-environmental) doses in humans and several other animal species, including dogs, rabbits, guinea pigs, rats and mice. The major effects demonstrated are liver and kidney damage, central nervous system effects and depression in myocardial contractility.

In the calculation of a suggested adjusted ADI for trichloroethylene, liver toxicity was used as the most sensitive end-point with respect to adverse health effects, not including the potential carcinogenic risk that may result from exposure to the chemical. A study in which rats were exposed to trichloroethylene through inhalation with resulting elevation of liver weights was used to calculate a suggested Adjusted ADI of 0.257 mg/l. This value was calculated based upon a minimal-effect-level of 300 mg/m³ (55 ppm), since rats exposed to this dose level (5 days a week for 14 weeks) showed elevation of liver weights. An uncertainty factor of 1000 was applied due to the fact that an animal study, where the no-observed-adverse-effect-level was not identified, was used and because the study was only of 14 weeks duration. One hundred percent exposure from drinking water and a 70 kg adult consuming 2 liters of water per day were assumed in the calculations.

The NAS has not calculated a chronic, non-carcinogenic Suggested No-Adverse-Response Level (equivalent to an Adjusted ADI) for trichloroethylene, because every long-term study, with the exception of the National Cancer Institute (NCI) carcinogenesis investigation, involves trichloroethylene administration by inhalation. The NCI bioassay did not determine a "no-effect level" and thus it was not considered appropriate for use in the deviation of a chronic, noncarcinogenic value.

Bacterial mutagenicity studies have shown trichloroethylene to be mutagenic in several systems, including metabolically activated *Salmonella typhimurium* and *E. coli* K12 strain; however, a later study reported trichloroethylene to be non-mutagenic in the Ames test system.

Commercial grade trichloroethylene was tested by the National Cancer Institute (NCI) (1976) and was reported to induce hepatocellular carcinomas in male and female mice by oral gavage. A repeat bioassay by the National Toxicology Program (1983) using purified trichloroethylene in corn oil found it to cause hepatocellular carcinomas in both sexes of mice, at a dose of 1,000 mg/kg per day, five days per week for 2 years, administered by gavage. Trichloroethylene was not carcinogenic in female rats under the test conditions and the results in male rats were determined to be insufficient to make a

adequate evaluation of the carcinogenicity. The doses administered to the rat were 1,000 and 500 mg/kg/day.

The International Agency for Research on Cancer (IARC) has concluded that trichloroethylene has limited evidence of carcinogenicity, based upon experimental animal studies and inadequate evidence from available human data. This means that the data suggest a carcinogenic effect in one species, but lack of confirmation in others. The World Health Organization (1981) has recommended a tentative guideline value of 30 μg/l for trichloroethylene in drinking water.

EPA's Carcinogen Assessment Group has used the linearized non-threshold multi-stage model to calculate projected excess cancer risk estimates extrapolated from high dose animal studies. For trichloroethylene, these estimates were based upon the NCI bioassay data. Calculated risks correspondingly to various doses are listed in Table 7.

Tetrachloroethylene. The principal non-carcinogenic effects of tetrachloroethylene in humans and other animals from both acute and longer-term exposures at relatively high (non-environmental) doses include central nervous system depression and fatty infiltration of the liver and kidney with concomitant changes in serum enzyme activity levels indicative of tissue damage.

A suggested adjusted ADI for tetrachloroethylene, considering adverse health effects other than the potential carcinogenic risk, was calculated based upon a series of studies in which rats were exposed by inhalation to tetrachloroethylene with effects observed on the central nervous system, immune system and certain blood components. The value of 0.085 mg/l was derived from these studies, based upon a no-observed-adverse-effect level of 10 mg/m³ (1.5 ppm) and an uncertainty factor of 100. This uncertainty factor was considered appropriate for use with a no-observed-adverse effect level from an animal study with no comparable human data. Daily exposure of a 70 kg adult drinking 2 liters of water per day was assumed in the calculations.

Tetrachloroethylene in corn oil was tested for carcinogenic potential in mice and rats by gavage in the NCI Bioassay Program (1977). In these bioassays, it was shown that tetrachloroethylene increased the incidence of hepatocellular carcinomas in both sexes of mice, but not in rats. A dose rate of 531 mg/kg per day, 5 days/week in male mice and 386 mg/kg in female mice resulted in a tumor incidence rate of 65 percent and 40 percent, respectively. Because of an excessive dose related mortality in the gavage experiment and low dose level in the inhalation study, no conclusion can be made about the carcinogenicity of tetrachloroethylene in rats. Data from the recent gavage study has been withdrawn for the time being pending the results of an indepth audit by the NTP due to unresolved problems with the study as conducted.

The majority of mutagenicity studies on tetrachloroethylene were negative. Two positive studies have been reported; however, the purity of the tetrachloroethylene was questioned in these cases.

The IARC has concluded that tetrachloroethylene has limited evidence of carcinogenicity in animals and inadequate evidence from available human data. This means that the data suggest a carcinogenic effect in one species, but lack confirmation in others. The World Health Organization has recommended a tentative guideline value of 10 μg/l for tetrachloroethylene in drinking water.

EPA's Carcinogen Assessment Group has used the linearized multi-state model to calculate projected excess cancer risk estimates extrapolated from high-does animal studies. For tetrachloroethylene, these estimates were based upon the 1977 NCI bioassay in mice. Calculated risks corresponding to various doses are listed in Table 7.

1,1,1-Trichloroethane. The principal toxic effects of 1,1,1-trichloroethane from which (non-environmental) dose exposure in animals and humans are depression of the central nervous system, increase in liver weight and cardiovascular changes.

Liver toxicity was used as the most sensitive end-point with respect to adverse health effects, not including the potential carcinogenic risk, in the calculation of an adjusted ADI for 1,1,1-trichloroethane. An inhalation study which examined exposure of mice to 1,1,1-trichloroethane was used to calculate a suggested Adjusted ADI of 1.0 mg/l. This study demonstrated changes in the livers of the mice at various dose levels.

Two animal bioassays by the National Cancer Institute (NCI) have been completed in rats and mice (1977; 1983). In the earlier bioassay, rats and mice were treated with 1,1,1-trichloroethane in corn oil by gavage. Because only 3 percent of the animals survived to the end of the experiment, due in part to chronic murine pneumonia which was determined to be the most probable cause of the high incidence of natural deaths among the animals, it was concluded that carcinogenicity could not be determined from this study. A repeat carcinogenesis bioassay of 1,1,1-trichloroethane was conducted in which doses of 3,000 or 1,500 mg/kg were administered by gavage to both sexes of mice, and rates were given does of 750 or 375 mg/kg. In the preliminary report of this study, 1,1,1-trichloroethane was carcinogenic in both male and female mice showing an increased incidence of hepatocellular carcinomas but not in rats; however, these initial results have been questioned.

1,1,1-Trichloroethane has been tested for mutagenicity in several test systems. Both negative and positive results were reported in mutagenicity tests in various *Salmonella typhimurium* strains, and 1,1,1-trichloroethane was not shown to be mutagenic in studies using yeast as an indicator organism.

EPA's Carcinogen Assessment group has used the linearized non-threshold multi-stage model to calculate preliminary excess cancer risk estimates extrapolated from the preliminary reported incidence of hepatocellular carcinomas in female mice in the study cited above. Calculated risks corresponding to various doses are listed in Table 7.

Similar calculations were made by the NAS (*Drinking Water and Health*, Vol. V) except that the average of the results in both male and female mice were used as the basis.

The latest bioassay data, on 1,1,1-trichloroethane is currently undergoing audit by the NTP and a final report has not been issued. Therefore this proposal will use the noncancer inhalation data as the basis for the proposed RMCL. This notice will be amended if the final NTP report determines that 1,1,1-trichloroethane was carcinogenic under the conditions of the tests.

Carbon Tetrachloride. Carbon tetrachloride (CCl_4) has been shown to exhibit non-carcinogenic effects in humans and animals following acute and chronic exposures. The principal effects seen at high doses are liver changes such as fatty liver with centrilobular necrosis developing if exposure is continued.

A chronic AADI for CCl_4 of 0.025 mg/1 was calculated from a recent report of a study (Bruckner, et al., 1983) which has not yet been published or peer reviewed at this juncture.

Rats weighing 200–500 g were randomly divided into groups of 15 to 16 animals each. The animals were given by gavage 0, 1, 10, 33 mg CCl_4/kg bw (in corn oil). The animals were dosed on a daily basis, 5 times weekly, for a total period of 12 weeks. Blood samples were obtained from alternate animals at the

following intervals: 2, 4, 6, 8, 10 and 12 weeks post-treatment. The serum was analyzed for BUN, GPT, SDH and OCT. At 1 mg/kg, there were no significant biochemical/histopathological changes. SDH, the most sensitive index of hepatotoxicity, was elevated ($p < 0.05$) in rats receiving 10 mg/kg for 12 weeks. Also, these rats exhibited mild centrilobular vacuolization. At 33 mg/kg, levels of GPT, SDH, and OCT were increased ($p < 0.01$) and marked hepatic lesions were apparent. There was no evidence that CCl_4 was nephrotoxic.

Comments on the experimental protocols and interpretations of the data are requested.

Carbon tetrachloride has been shown to be carcinogenic in rats, mice and hamsters through oral administration. In the NCI (1976) bioassay for trichloroethylene, carbon tetrachloride was used as the positive control. Carbon tetrachloride was administered in corn oil by gavage to rats at two dose levels: 47 and 94 mg/kg for males and 80 and 159 mg/kg for females. In mice, the chemical was administered at 1,250 and 2,550 mg/kg. Carbon tetrachloride was determined to increase carcinomas of the liver in both rats and mice in this bioassay.

Carbon tetrachloride has not been shown to be mutagenic in any of the reported Salomonella (Ames) assays. However, mutagenic activity associated with carbon tetrachloride has been observed in a test system using the yeast *Sacchcromyces cerevisiae*.

The IARC has concluded that sufficient evidence of carcingenicity in animals exists for carbon tetrachloride. The NCI has also identified carbon tetrachloride as an animal carcinogen and has used it as a positive control in several bioassays. The World Health Organization (1981) has recommended a tentative guideline value of 3 μg/l for carbon tetrachloride in drinking water.

EPA's Carcinogen Assessment Group has used the linearized non-threshold multi-stage model to calculate projected excess cancer risk estimates extrapolated from high dose animal studies. For carbon tetrachloride, the latest draft estimates were based upon the geometric mean of the four cancer studies. Calculated risks corresponding to various doses are listed in Table 7.

1,2-Dichloroethane. The toxic effects of 1,2-dichloroethane in humans and other animals from both acute and longer-term exposures at relatively high levels include central nervous system depression, liver and kidney damage, gastrointestinal distress, adrenal and pulmonary effects and circulatory disturbances.

A series of inhalation studies in which a variety of animal species were exposed for up to 8 months to 1,2-dichloroethane were used to calculate a suggested Adjusted ADI for 1,2-dichloroethane. The most sensitive end-points, not including the potential carcinogenic risk, identified in these studies were pulmonary congestion, diffused myocarditis, and fatty degeneration of the liver, kidney, adrenal and heart. A value of 0.260 mg/l was calculated, based upon a no-observed-adverse-effect-level of 405 mg/m^3 (100 ppm). A variety of animal species exposed to this dose level for 6 to 7 hours/day, 5 days/week yielded no adverse effects as measured by general appearance, behavior, mortality rates, growth rates, organ function and blood chemistry. An undertainty factor of 1000 was used to account for an animal study with no equivalent human data, and for the use of a study of less than lifetime exposure. One hundred percent exposure from drinking water and a 70 kg adult consuming 2 liters of water per day were assumed in the calculations.

1,2-Dichloroethane has been shown to significantly increase tumor incidences at several sites in both rats and mice when administered by gavage, but not following inhalation exposure. In the NCI bioassay, doses of 47 or 95 mg/kg in corn oil administered by gavage to rats and 97 or 195 mg/kg given to male mice and 149 or 299 mg/kg given to female mice were shown to increase the incidence of several types of tumors. 1,2-Dichloroethane has also been shown to be mutagenic in a number of biological systems, including *Drosophila melanogaster, Salmonella typhimurium* and *E. coli*.

The IARC has concluded that sufficient evidence of carcinogenicity in animals exists for 1,2-dichloroethane. The World Health Organization (1981) has recommended a tentative guideline value of 10 ug/l for 1,2-dichloroethane in drinking water.

EPA's Carcinogen Assessment Group has used the linearized non-threshold multi-stage model to calculate projected excess cancer risk estimates extrapolated from high-dose animal studies. For 1,2-dichloroethane, these estimates were based upon the NCI bioassay data. Calculated risks corresponding to various doses are listed in Table 7.

Vinyl chloride. Acute and chroic toxicity studies with vinyl chloride have shown the major non-carcinogenic effects resulting from high dose exposures to be congestion and edema of the lungs and hyperemia of the kidney and liver. Other non-carcinogenic effects have been noted, including disturbances of the central nervous system, pulmonary insufficiency, cardiovascular manifestations, gastrointestinal symptoms and acroosteolysis.

A suggested Adjusted ADI for vinyl chloride of 0.06 mg/l considering adverse health effects not including carcinogenic risk, was calculated based upon an oral toxicity study in rats in which a variety of carcinogenic and non-carcinogenic effects were observed at all dose levels. A minimal-effect-level of 1.7 mg/kg was used in the calculations, as histopathological changes in the liver including clear-cell foci, extensive necrosis, cysts and liver-cell polymorphism were observed at this dose level. An uncertainty factor of 1000 was applied to account for an animal study where the no-observed-adverse-effect level was not identified. One hundred percent exposure from drinking water and a 70 kg adult consuming 2 liters of water per day were assumed in the calculations.

Vinyl chloride has been shown to have carcinogenic effects in animals and humans. Animal studies have demonstrated the production of liver angiosarcomas, mammary carcinomas, pulmonary angiosarcomas and other tumor types in rats following oral exposure and carcinogenic effects in mice, rats and hamsters by inhalation exposure have been reported. In humans, studies have linked vinyl chloride with angiosarcoma of the liver and other forms of neoplasm. The IARC has concluded that sufficient evidence of carcinogenicity exists for vinyl chloride from animal studies and human studies, and that vinyl chloride should be considered a human carcinogen with target organs of the liver, brain, lungs and haemo-lymphopoietic system.

Vinyl chloride was shown to be mutagenic in the test system using metabolically activated *Salmonella typhimurium*, *E. coli* K12 strain, in germ cells of *Drosophila* and Chinese hamster V79 cells.

EPA's carcinogen assessment Group has used the linearized non-threshold multi-stage model to calculate projected excess cancer estimates extrapolated from high dose animal studies. For vinyl chloride, these estimates were based upon an inhalation study in rats in which vinyl chloride concentrations ranging from 50 to 10,000 ppm resulted a total tumor incidence rate of 17 percent to 62 percent, respectively. The NAS has also used the multi-stage model to calculate excess cancer risk values. They based their estimates up the same study as did CAG (Maltoni, *et al.*, 1975), except ingestion data instead of inhalation data were used. The NA

risk estimation used ingestion exposure and thus may be more appropriate for estimating risks from drinking water exposure. Calculated risks corresponding to various doses are listed in Table 7. In addition, data from a recent draft CAG calculation using an ingestion study in rats (EPA, 1984) are also included for comment.

Benzene. The toxic effects of benzene in humans and other animals include central nervous system effects, hematological effects as well as immunological effects. The toxicity of benzene to the hematopoietic system of humans experiencing chronic exposure to benzene is well documented. Repeated exposure effects include myelocytic anemia, thrombocytopenia and leukemia. In laboratory animals, leukopenia is the most commonly observed effect of chronic benezene exposure.

A suggested Adjusted ADI for benzene, considering adverse health effects not including carcinogenic risk, was calculated based upon data from a gavage study in rats in which leucopenia was observed at specific dose levels. A value of 0.025 mg/l was calculated using a no-observed-adverse-effect level of 1 mg/kg and an uncertainty factor of 1000. This uncertainty factor was used to account for an animal study with no equivalent human data, and for the use of a study of less than lifetime exposure. One hundred percent exposure from drinking water and a 70 kg adult consuming 2 liters of water per day were assumed in the calculations.

Benzene has been shown to be carcinogenic in Sprague-Dawley rats, causing tumors at dose levels of 50 mg/kg and 250 mg/kg. An increase in zymbal gland carcinomas, luekemias and mammary carcinomas in rats has also been observed. Toxic effects on bone marrow cells of rats and other laboratory animals from benzene exposure include changes in chromosome number and chromosome breakage. These types of effects have also been observed in humans.

EPA's Carcinogen Assessment group has used the linearized non-threshold multi-stage model to calculate projected excess cancer estimates extrapolated from high-dose animal and human studies. For benzene, these estimates were based upon an epidemiologic study of workers exposed to benzene vapors on their jobs. Calculated risks corresponding to various doses are listed in Table 7.

1,1-Dichloroethylene. 1,1-Dichloroethylene has been shown to cause liver and kidney injury in animals from high dose exposures. Liver damage in rats, mice and guinea pigs has been documented, along with renal toxicity, CNS depression and sensitization of the heart.

An Adjusted ADI of 350 µg/l for 1,1-dichloroethylene considering adverse health effects not including the potential carcinogenic risk was calculated based upon toxic liver effects using a NOAEL of 10 mg/kg and 100 percent exposure from drinking water.

The NAS (1983) has calculated a chronic, suggested-no-adverse-response level (equivalent to an adjusted ADI) of 0.1 mg/l based upon non-carcinogenic effects only for 1,1-dichloroethylene, from data in the National Toxicology Program bioassay (1982) in rats and mice. A no-observed-adverse-effect level of 2 mg/kg was used and an uncertainty factor of 100, and complete absorption from the GI tract. Twenty percent exposure from drinking water and a 70 kg adult consuming 2 liters of water per day were assumed in the calculations, along with conversions from a 5 d/week dosing regime to a 7 d/week exposure.

1,1-Dichloroethylene was found to be mutagenic with microsomal activation in *Salmonella typhimurium* and *E. coli* test systems. However, mutagenicity was not observed with V79 Chinese hamster cells or in dominant lethal studies in mice and rats.

1,1-Dichloroethylene was shown to produce kidney adenocarcinomas in mice and rats in one study (Maltoni, 1977). However, most of the other studies have failed to demonstrate significant carcinogenic activity of the chemical. A study by the National Toxicology Program (1982) examined 1,1-dichloroethylene exposures of 1 mg/kg or 5 mg/kg 5 times per week in rats and 2 mg/kg or 10 mg/kg 5 times per week in mice. In this bioassay, there was no evidence that 1,1-dichloroethylene was carcinogenic for either the rats or the mice. However, there was some question as to whether the maximum tolerated dose had been used in this study. The NAS (1983) has concluded that information on 1,1-dichloroethylene is not sufficient to reach a definite conclusion on the carcinogenicity of the compound.

EPA's Carcinogen Assessment Group found 1,1-dichloroethylene to have limited evidence of carcinogenicity in animals. They have used the linearized, non-threshold, multi-stage model to calculate projected excess cancer estimates extrapolated from high-dose animal studies. For 1,1-dichloroethylene, these estimates were based on results of inhalation studies in mice and rats. Calculated risks corresponding to various doses are listed in Table 7. EPA's SAB has recently questioned validity of this study result. This tentative classification of 1,1-DCE as a carcinogen will be reexamined during the comment period. Comment is solicited in this regard.

p-Dichlorobenzene. Non-carcinogenic adverse effects observed in animal studies include liver and kidney damage, porphyria, pulmonary edema and congestion and splenic weight changes. In humans, exposure to fairly high concentrations of the dichlorobenzenes has been reported to result in anorexia, nausea, yellow atrophy of the liver and blood dyscrasias.

A suggested Adjusted ADI of 3.75 mg/l for p-dichlorobenzene considering adverse health effects other than carcinogenic potential was calculated. This value was based upon the rat subchronic gavage study which served as the dose range-finding study for the NTP bioassay. The ADI was based upon a NOAEL of 150 mg/kg/day. Uncertainty factors of 100 and 10 were used to account interspecies extrapolation and use of data from an exposure duration significantly less than lifetime.

p-Dichlorobenzene has been shown to induce abnormal mitotic division in higher plants. The compound was not seen to be mutagenic when tested in the *Salmonella typhimurium* or *E. coli* WP2 systems, and no evidence of mutagenicity in animals has been reported to date.

In June 1980, a carcinogenesis bioassay of p-dichlorobenzene in mice and rats was undertaken by the National Toxicology Program. Doses of 200 mg/kg or 600 mg/kg were administered by gavage to both sexes of mice and to female rats. Male rats were given 150 or 300 mg/kg. The results of this study have not yet been released.

V. RMCL Development Rationale

The ANPRM requested public comment on the appropriate approach to deal with VOCs in drinking water, specifically requesting consideration of the following:

- What approach should be followed under the SDWA to reduce human exposure to VOCs?
- For which VOCs should regulations be set?
- What approach should be followed in setting RMCLs for suspected carcinogens?

Each of these issues is discussed below in regard to the rationale used by the Agency in development of this proposal and the Agency's consideration of the public comments, the

requirements of the SDWA, and the available scientific information.

VOCs: Regulatory Approach

Alternative approaches. The major alternatives considered for limiting human exposure to VOCs in drinking water as discussed in 47 FR 9350 are provided below.

(1) *No federal regulations. Provision of health advisories for State action as appropriate.* Health advisories and advice on treatment and analytical methods are currently being provided to States and public water systems for use in dealing with incidents of VOC contamination.

Each State would design its own control strategies to address incidents of contamination on a case-by-case basis or state-wide. Health advisories were developed to deal primarily with isolated incidents of short-term contamination in lieu of standards and not as a substitute for MCLs. Experience has shown that, as would be expected, States have interpreted and applied the health advisories in different ways. Some States have applied the health advisories as if they were standards or considered adopting them as State standards.

(2) *Set federal monitoring regulations and provide health advisories for State action as appropriate.* This option would set monitoring requirements for VOCs under section 1445 and provide health advisories for State action as needed. This alternative would result in all public water systems determining if they have VOCs in their drinking water and could be proposed and promulgated in a shorter period of time than alternative 3. Different States would probably adopt different control options and action levels.

(3) *Set Primary Drinking Water Regulations for certain of the VOCs.* This option would set RMCLs, MCLs, monitoring and reporting requirements for a number of VOCs and would result in consistent, nation-wide controls on VOCs.

Proposed regulatory approach. The SDWA authorizes EPA to establish RMCLs for "each contaminant which, in [the Administrator's] judgment * * * may have any adverse effect on the health of persons" section 1412(b)(1)(B). A primary drinking water regulation is to be established for each contaminant for which an RMCL is established. Section 1412(b)(2). In implementing this broad statutory mandate, EPA is considering the following factors to select contaminants appropriate for regulations. These include:

- Whether the frequency of occurrence and the concentrations detected in drinking water and the extent of the population exposed warrant establishment of national primary drinking water regulations.
- Whether the available toxicology data are sufficient to warrant a determination that adverse effects may be known or anticipated at levels found in drinking water.

Notwithstanding these factors, EPA feels that primary drinking water regulations may be appropriate in some instances for substances which to date have not been found at high concentrations or frequencies in drinking water, but where in the Administrator's judgement it would be appropriate to anticipate possible future potential for drinking water contamination from spills or improper disposal.

Other factors that must be considered as part of the decision on the type of regulation (MCL or treatment requirement) include:

- Whether monitoring is technically and economically feasible.
- Whether treatment technologies are available to reduce the contaminants to appropriate levels.

In addition, some guidance was provided in the legislative history to the SDWA Senate Report on possible candidates for Revised Regulations. Contaminants listed in the following sources were expected to be considered for regulation.

- World Health Organization: "Maximum Permissible Concentrations of Harmful Substances in the Water of Water Courses used for Hygienic and Domestic Purposes (1970)."
- World Health Organization: "European Standards for Drinking Water," 2nd edition, Revised, Geneva (1970).
- National Institute of Occupational Safety and Health annual list of toxic substances.
- Toxic Substances listed under section 307 of the Federal Water Pollution Control Act.

Information provided by the NAS in the Drinking Water and Health series is an additional source.

While numerous contaminants are listed in these sources, this proposal in Phase I of EPA's National Primary Drinking Water Regulations addresses a limited number of contaminants in the VOC category found in drinking water. Because of EPA's desire to avoid delay in developing regulations for certain VOCs that have been detected in ground waters and the need to prioritize the expenditure of limited resources, only nine VOCs are addressed in this initial proposal. Other VOCs for which sufficient occurrence and health effects information become available will be addressed in Phase II and later iterations of the National Primary Drinking Water Regulations along with other contaminants. Specific VOCs considered in this proposal are those that have appeared to be the highest priority for regulation based upon occurrence, health risk considerations and available data.

Several VOCs have been found across the country in numerous drinking water supplies. In the GWSS, 21 percent of systems had at least one VOC detected. EPA has concluded that sufficient health effects data are available to cause concern about potential human exposure to certain VOCs via drinking water. Various of the VOCs are suspected or proven mammalian carcinogens, some are known human carcinogens, some are active in certain mutagenic test systems and exposure to certain of the VOCs at high doses has shown other non-carcinogenic toxic effects. EPA recognizes that interpretation of health risk data raises numerous scientific issues. However, drawing upon the conclusions/recommendations of the NAS, IARC and the NDWAC, EPA believes that the data adequately demonstrate concern such that RMCLs and primary drinking water regulations are warranted. Thus, EPA has determined that human exposure to certain VOCs via drinking water may have an "adverse effect upon the health of persons" thereby warranting regulatory action.

Selection of VOCs for Regulation

This section provides a discussion of the factors used to select the specific contaminants for which RMCLs are proposed at this time. VOCs that were not included in this proposal will be reconsidered in Phase II of the Revised Regulations as additional data become available.

Factors considered. A number of factors were considered in determining which VOCs should be regulated; however, there is no established formula or set criteria for these determinations. The SDWA states that regulations should be set for contaminants that the Administrator determines "may have any adverse effect upon the health of persons" but little additional guidance was provided. Obviously, it is impossible to consider for regulation every chemical that may appear in drinking water and that theoretically may adversely affect health in some remote circumstances. What is needed is some prioritization of contaminants in drinking water so that a reasonable

number of contaminants of sufficient concern can be addressed in regulations.

To best employ its resources, EPA must select contaminants for regulation based upon considerations that will advance the goals of the Act to assure the safety of drinking water. EPA believes that the most relevant criteria are the: (1) Analytical ability to detect a contaminant in drinking water, (2) the frequency and level of occurrence and population exposed, and (3) potential health aspects of the contaminants. In addition EPA considers regulation when there are sufficient incidents or contamination potential such that national guidance in the form of a Primary Drinking Water Regulation is desirable to assist States and public water systems which must determine appropriate responses.

Analytical methods. Analytical methods must be available such that the presence of the chemicals in water can be validly determined. This factor is an important part in determining whether the substance can be regulated and whether an MCL or a treatment technique regulation should be promulgated.

National or limited significance. Consideration of occurrence data encompasses both the frequency of occurrence, the level of occurrence and ther extent of the population exposed. The occurrence data allow EPA to determine whether contamination of drinking water represents isolated or localized incidents of contamination more appropriately dealt with by States, or whether contamination has occurred or has the potential for occurring in numerous locations across the country involving a sufficient number of water supplies and population exposed to warrant action under the Safe Drinking Water Act. In the ANPRM for Phase II of the NPDWR, 48 FR 45502, *et seq.*, EPA described a categorization system for differentiation between widespread and limited contamination potential.

Health effects. Consideration of the potential health effects of a chemical encompasses the: (1) Suitability of the available data for assessing the toxicology of the chemical, and (2) the possibility of human health concern from exposure from drinking water. When it is possible scientifically, section 1412(e)(3) of the SDWA also requires consideration of the impact of the following:

(A) The existence of groups or individuals in the population which are more susceptible to adverse effects than the normal healthy adult.

(B) The exposure to contaminants in other media than drinking water (including exposures in food, in the ambient air and in occupational settings) and the resulting body burden of contaminants.

(C) Synergistic effects resulting from exposure to or interaction by two or more contaminants.

(D) The contaminant exposure and body burden levels which alter physiological function or structure in a manner reasonably suspected of increasing the risk of illness.

These factors were addressed in assessing the potential health effects of each of the VOCs and are discussed in each of the health effects criteria documents as referenced in section VII. However, applicable data are seldom available for any of these factors except B (to a limited extent) which is addressed in both the occurrence and health effects documents.

Other considerations. Additional factors considered in determining which VOCs should be regulated and how are discussed below.

• One approach that might be considered would be to set RMCLs by category, i.e., the same RMCL for each VOC or subcategories of VOCs. In effect this is being proposed for the category determined to be non-threshold toxicants. However, a categorical RMCL for non-carcinogenic VOCs is not scientifically supportable due to differing relative toxicities of individual substances (different thresholds) and different toxic endpoints.

• *Strength of evidence.* Pertaining to either the extent of contamination or to the potential health risks of exposure, the amount of available data of sufficient quality on a certain chemical was considered. For example:

—A chemical proven to be a human carcinogen, even though occurring relatively infrequently in drinking water supplies might be appropriate for regulation, e.g., vinyl chloride and benzene.

—A chemical occurring at a higher frequency in drinking water supplies but for which the strength of evidence on potential health risks was weaker could be appropriate for regulation, e.g., trichloroethylene, tetrachloroethylene, carbon tetrachloride, 1,1-dichloroethylene, 1,1,1-trichloroethane, 1,2-dichloroethane.

• *Notional guidance to address incidents of contamination.* Regulations provide a benchmark for potential action by State and local officials in evaluating incidents of contamination. In certain cases, this factor may be a major consideration in determining if regulations are appropriate. For example, regulations would be appropriate for a chemical that occurs but at levels normally below those associated with potential health risks, e.g., p-dichlorobenzene and 1,1,1-trichloroethane. The MCL would provide guidance that no action was necessary for these systems with less than that level; without regulations, these types of situations have met widely varying responses by States and public water systems. Regulations can provide a basis for rational and uniform responses to incidents of contamination.

• *Potential impact.* The potential impact of setting regulations can be considered in a general manner; however, this factor is primarily considered during establishment of MCLs. This evaluation considers potential burdens including such factors as the affordability of treatment systems, the technical feasibility of meeting MCLs, and other possible impacts such as monitoring and reporting.

The results of setting regulations for VOCs will vary widely from no impact to installation of treatment systems for reduction of VOCs. Recognizing that the great majority of public water systems do not have VOCs in the drinking water, the only burden on these systems would be monitoring and reporting. These burdens could be minimized through flexible monitoring requirements (see 48 FR 45502) that would provide states with authority to determine appropriate requirements beyond the national minimum. In addition, the VOCs are somewhat unique in the sense that several of them can be analyzed for in a single analytical procedure.

• *Other factors.* Surrogate parameters or aggregate parameters may be needed to take into account other potential effects not considered in setting RMCLs and MCLs for individual chemicals, such as possible additive or synergistic risks of simultaneous exposure to more than one VOC.

Proposed VOCs. The ANPRM listed fourteen VOCs being considered for regulations. Detailed occurrence and health effects information were provided for six of the fourteen VOCs.

Since the ANPRM was published, EPA completed the Ground Water Supply Survey (GWSS) in which twenty-nine VOCs were looked for in each sample using the "purge and trap" analytical procedure employing gas chromatography (Method 502.1 and Method 503.1, U.S. EPA, Environmental Monitoring and Support Laboratory). As shown in Table 1, not all of the ANPRM list of 14 VOCs were detected in the GWSS.

Based upon the above considerations, public comments and recommendations of the NDWAC and other information, EPA has concluded that these chemicals

"may have an adverse effect upon the health of persons" and that RMCLs and primary drinking water regulations under Section 1412 should be proposed at this time. They are:

trichloroethylene
tetracholoroethylene
1,1,1-trichloroethane
carbon tetrachloride
1,2-dichloroethane
benzene
vinyl chloride
p-dichlorobenzene
1,1-dichloroethylene

As presented previously, the NDWAC recommended developing regulations for the first five of the above nine VOCs. Their rationale was based upon an evaluation of the available occurrence and health effects data for each of the VOCs. The NDWAC evaluated the information in September 1982. Since that time additional data have become available and the Agency has concluded that four additional VOCs warrant regulation.

The background occurrence and health effects data used as the basis for determining which VOCs warranted regulations is summarized below.

Trichloroethylene. Occurrence: GWSS (Random): 6.4%; max: 78 μg/l; medium: 1.0 μg/l. GWSS (Non-random): 12.7%; max: 130 μg/l; median: 1.4 μg/l. State Data: 624 positives/4228 sampled, max: 510.000 μg/l.

Health Effects: Non-carcinogenic effects (at high doses): liver and kidney damage, central nervous system effects, depression in myocardial contractility. Carcinogenic effects: mutagenic in some test systems; carcinogenic in NCI test: mice. Limited evidence.

Tetrachloroethylene. Occurrence: GWSS (Random): 7.3%; max: 23 μg/l; median: 0.5 μg/l. GWSS (Non-random): 9.4%; max: 69 μg/l; median: 0.7 μg/l. State Data: 628 positive/3636 sampled, max: 1.000 μg/l.

Health Effects: Non-carcinogenic effects (at high doses); central nervous system depression, fatty infiltration of liver and kidney, tissue damage. Carcinogenic effects: carcinogenic in NCI test: mice; limited evidence.

1,1,1-Trichloroethane. Occurrence: GWSS (Random): 5.8%; max: 18 μg/l; median: 0.8 μg/l. GWSS (Non-random): 10.6%; max: 21 μg/l; median: 1.0 μg/l. State Data: 715 positive/3330 sampled, max: 2.250 μg/l.

Health Effects: Non-carcinogenic effects (at high doses): central nervous system depression, increase in liver weight, cardiovascular changes. Carcinogenic effects: carcinogenic in preliminary report from NTP test: mice; limited evidence. This report is currently being evaluated.

Carbon tetrachloride. Occurrence (may be a contaminant in chlorine) GWSS (Random): 3.2% max: 16 μg/l; median: 0.4 μg/l. GWSS (Non-random): 3.1%; max: 15 μg/l; median: 0.5 μg/l. State Data: 368 positive/2646 sampled, max: 1.200 μg/l.

Health Effects: Non-carcinogenic effects: liver effects such as fatty liver with centrilobular necrosis. Carcinogenic effects: mutagenic in some test systems; carcinogenic in NCI test: mice, rats, hamsters; sufficient evidence.

1,2-Dichloroethane. Occurrence: GWSS (Random); 0.6%, max: 1.0 μg/l; median: 0.5 μg/l. GWSS (Non-random): 1.5%; max: 10 μg/l; median: 2.5 μg/l. State Data: 177 positive/1793 sampled, max: 2.100 μg/l.

Health Effects: Non-carcinogenic (at high doses): central nervous system depression, liver and kidney change, gastro-intestinal distress, adrenal and pulmonary effects, circulatory disturbances. Carcinogenic effects: mutagenic in most test systems; carcinogenic in NCI test: mice, rats: sufficient evidence.

Vinyl chloride. Occurrence: GWSS (Random): 0.2%; max: 1.1 μg/l; median: 1.1 μg/l. GWSS (Non-random): 1.3% max: 8 μg/l; median: 2.7 μg/l. State Data: 126 positive/1793 sampled, max: 380 μg/l.

Health Effects: Non-carcinogenic (at high doses): congestion and edema of the lungs, hyperemia of the kidneys and liver. Carcinogenic effects: mutagenic: carcinogenic in animal studies: mice, rats, hamsters; sufficient evidence for human carcinogenicity.

Benzene. Occurrence: GWSS (Random): 0.6% max: 15 μg/l; median: 3 μg/l. GWSS (non-random): 1.7%; max: 12 μg/l; median: 1.6 μg/l. State Data: 4 positive/645 sampled, max: 17 μg/l.

Health Effects: non-carcinogenic: central nervous system effects, hematological and immunological effects. Carcinogenic effects: sufficient evidence for human carcinogenicity.

1,1-Dichloroethylene. Occurrence: GWSS (Random): 1.9%, max: 6.3 μg/l; median: 03. μg/l. GWSS (non-random): 3.1%, max: 3.0 μg/l; median: 0.4 μg/l. State Data: NA.

Health Effects: non-carcinogenic effects (at high doses): liver and kidney damage, renal toxicity, CNS depression and sensitization of the heart. Carcinogenic effects: mutagenic, carcinogenic in one animal study: mice and rats: limited evidence.

p-Dichlorobenzene. Occurrence: GWSS (Random): 1.1%, max: 1.3 μg/l, median: 0.7 μg/l. GWSS (Non-random): 0.8%, max: 0.9 μg/l. median: 0.7 μg/l. State Data: N/A.

Health Effects: non-carcionogenic (at high doses): kidney and liver damage. pulmonary edema and congestion, spieneic weight changes. Carcinogenic effects: NTP test underway.

Other VOCs. Several additional VOCs listed in the ANPRM (47 FR 9350) have been found in some drinking water samples but the available data has been judged to be insufficient to propose RMCLs at this time.

- Cis-1,2-dichloroethylene and trans-1,2-dichloroethylene

These two VOCs have not been tested for carcinogenicity by the NTP and adequate studies on non-carcinogenic toxicity have not been conducted.

- Chlorobenzene

While some occurrence has been reported by a number of States, the GWSS did not detect any chlorobenzene in the random sample; however, it was found twice in the non-random sample. The toxicology evaluation has not been completed.

- Trichlorobenzene(s)

States have detected trichlorobenzene in a number of water samples; however the number of drinking water versus non-drinking water incidences could not be determined from the data. In addition, analytical difficulties in analyzing samples in the GWSS precluded obtaining representative occurrence data.

- Dichloromethane

Because of problems of laboratory contamination and quality assurance, the available occurrence data for dichloromethane was not considered reliable. In addition, the NTP initial report on carcinogenicity has been withdrawn and the NTP is currently conducting an in-depth audit of the data.

These VOCs and several others will be considered in the Phase II portion of the Primary Drinking Water Regulations when sufficient occurrence and toxicology data become available. Among the other compounds being evaluated are such VOCs as ethylene dibromide, 1,1-dichloroethane, xylenes, toluene, bromobenzene, dibromochloropropane, 1,2-dichloropropane, and ethylbenzene (see ANPRM, October 5, 1983, 48 FR 45502). Other chemicals in the random GWSS for which no occurrence information was obtained but which will receive some consideration in Phase II or other iterations include: 1,1,2-trichloroethane, 1,1,2,2-tetrachloroethane, 1,1,1,2-tetrachloroethane, n-propylbenzene, o-chlorotoluene, p-chlorotoluene, m-dichlorobenzene, o-dichlorobenzene, styrene, isopropylbenzene.

Total VOCs

In addition to regulations for individual VOCs, the inclusion of RMCLs and MCLs for total VOCs (TVOC) is being considered. TVOC is not formally proposed in this regulation. Public comments are being solicited on whether it would be proper to include TVOC in drinking water regulations or in supporting guidance.

TVOC would represent summation of the levels of the individual VOCs for which RMCLs and MCLs have been set. The objective of a TVOC standard is to provide some additional protection from simultaneous exposure to multiple VOCs. As indicated in Table 4, drinking water often contains several VOCs. Generally, toxicology has not yet been able to provide a scientifically based conclusion on possible effects of simultaneous exposure to more than one chemical. Chemicals are normally tested separately and the possible synergistic, antagonistic, or additive health effects are not known. However, the NAS suggestion in this area was that in the absence of any other procedures, exposure to multiple carcinogens could be assessed by adding the risk rates. Comment is requested on the technical validity of this approach.

The potential problem that EPA feels must be addressed is a situation where a public water system finds several VOCs in its drinking water at levels slightly below the MCLs. For example, assume that MCLs are set for trichloroethylene, tetrachloroethylene, and carbon tetrachloride; a public water system with the following levels would technically be in compliance with the MCLs:

Compound	Measured level	Hypothetical MCL
Trichloroethylene	9 µg/l	10 µg/l
Tetrachloroethylene	14 µg/l	15 µg/l
Carbon tetrachloride	4 µg/l	5 µg/l

While technically in compliance with the standards, this condition probably represents an increased risk over any single chemical but the question that cannot be scientifically answered is whether this would be significant. EPA feels that multiple exposures could be more significant than indicated from just consideration of individual substances and requests public comments considering the myriad of possibilities in assessing multiple exposures, the costs and feasibility to reduce all the VOCs by application of one treatment technology, and the unknown aggregate health risk and the SDWA intent to err on the side of safety. If an RMCL and MCL for total VOCs (TVOCs) were appropriate, should EPA adopt the NAS suggestion that risks be considered additive be an appropriate approach?

RMCLs: Regulatory Approach

EPA is to set RMCLs at levels which, "no known or anticipated adverse effects on the health of persons occur and which allow an adequate margin of safety". Section 1412(b)(1)(B). Recommended MCLs are health goals and not enforceable standards. The proposed RMCLs for non-carcinogens can be determined using the scientific procedures set forth previously by calculating an AADI. However, determination of the "no effect" levels for carcinogens is a much more complex decision on what constitutes the safe level for non-threshold toxicants. Guidance on levels for the RMCLs was provided in House Report 93–1185 which stated that "It [The RMCL] must include an adequate margin of safety, unless there is no safe threshold for a contaminant. In such a case, the recommended maximum contaminant level should be set at zero level." EPA has considered the following approaches for setting RMCLs for carcinogens:

1. Set the RMCLs at zero.
2. Set the RMCLs at the analytical detection limit.
3. Set the RMCLs at a non-zero level based upon a calculated negligible contribution to lifetime risk.

Although one of these is proposed at this time, EPA requests comments on all three approaches. EPA's analysis of these approaches and the issues they raised are provided below.

Alternative 1: Set RMCLs at zero. One approach would be to establish RMCLs at zero for substances considered to be non-threshold toxicants. The existence of a threshold for the action of genotoxic carcinogens cannot be demonstrated by current science; thus, it could be conservatively assumed that no threshold exists, absent evidence to the contrary. Since distinctions between mechanisms of action of most carcinogens also cannot be conclusively made at this time, virtually all substances determined to be "carcinogens" would be assumed to be "non-threshold". Variation of this approach would be to limit the selection of RMCLs at zero only for those substances known to function by genotoxic processes, or perhaps only those determined to be human carcinogens, or only those for which "sufficient" rather than "limited" evidence of mammalian carcinogenicity exists.

Setting RMCLs for carcinogens at zero would follow the guidance provided in House Report 93–1185 and would express a general philosophy that *as a goal* carcinogens should not be present in drinking water. The Agency believes that the RMCLs (as a goal) should express the ideal concept that drinking water should be free from avoidable contamination and risk and that quality degradation should not be permitted.

If RMCLs are set at zero, some explanation may be needed to differentiate an RMCL from an MCL that would not be zero, since MCLs consider factors such as potential health risk, costs of treatment and feasibility of meeting the MCL. If these factors changed substantially, MCLs would need to be reexamined.

Alternative 2: Set RMCLs at the analytical detection limit. Due to limitations in analytical techniques, it will always be impossible to say with certainty that the substance is not present. In theory, RMCLS at zero will always be unachievable (or at least not demonstrable). While zero could be the theoretical goal for carcinogens in drinking water, in practice, a goal of achieving the analytical detection limits for specific carcinogens would have to be followed.

One possible approach would be for EPA to specify RMCLs for carcinogens based upon defined state-of-the-art analytical detection limits. The verifiable detection limits (i.e., the RMCLs) would probably fall in the vicinity of 1 µg/l depending upon the specific VOC. EPA believes this approach is justifiable in that zero is analytically undefinable and the detection limit may be the functional equivalent of zero. Of course, analytical detection limits are also moving targets as the state-of-the-art of analytical chemistry progresses, but at least they do provide a measurable target.

Alternative 3: Set RMCLs at a non-zero level based upon a calculated negligible contribution to lifetime risk. Alternative 3 would establish a non-zero level as the RMCL. A level could be selected that would present a negligible risk. In practical terms, such a low nominal risk would effectively preclude any discernable adverse effect on the health of the population and, because of the conservative nature of the risk calculation process, may not result in any actual adverse effects on an individual. EPA would have to conclude that this very low risk would result in "no known or anticipated adverse effect on the health of persons and which allows an adequate a margin of safety". This approach would provide some

quantitative guidance to public water systems of the ultimate goal which they might wish to use in the operation of water treatment facilities and in the design of future planned facilities. However, it should be recognized that just as with analytical detection limits (Option 2) a calculated risk target would also be moving target, because: (1) calculation methods change, and (2) the subjective determination of what is a negligible risk might change.

One possible variation of Option 3 would be to set RMCLs as a range of finite risk levels. This alternative would recognize the lack of accuracy and precision of risk calculations and the inherent difficulties in selecting one finite level as the *only* appropriate health goal in view of the numerous scientific uncertainties of risk estimates. However, this approach has a number of disadvantages including: lack of national uniformity and lack of specific guidance from EPA.

If a non-zero level is determined as appropriate for the RMCLs, two questions must be considered.

(1) What level should be used as representing the "no effect" level?

(2) How can an "adequate margin of safety" be incorporated into the finite risk level?

The NAS principles (*Drinking Water and Health*, Vol. 1) state that human exposure to carcinogens should be addressed in terms of risk rather than safe or non-safe. Because zero is not definable in an analytical sense, rather than speaking in terms of zero concentrations for carcinogens RMCLs for carcinogens could be set at levels at which the risks are so small that they are considered virtually nonexistent.

Determination of RMCLs for carcinogens at a finite level would be based on available science and the only quantitative tools available are cancer risk models. These are based upon animal studies and none of the models is experimentally verifiable as there is no scientifically valid method for determining the actual risks at low environmental exposure levels. Scientific issues surround their use in such areas as the data used, extrapolation techniques, and various factors in the analysis. Risk models are recognized as imperfect but they are the best tool available for estimating toxic potency or risk at low exposure levels. The commonly used risk models are generally conservative in their estimation of human risk of exposure to a contaminant. Selection of a target risk based upon a conservative risk model, such as the linearized multi-stage model, is arguably in accord with the SDWA, which requires the RMCL to be set at a no effect level "with an adequate margin of safety." The Agency believes that there is no exact or precise way to determine this level. The decision is judgmental—not strictly based upon science but upon a social judgment on what constitutes a negligible risk.

Federal regulations for environmental contaminants have generally fallen in the 10^{-4} to 10^{-6} lifetime risk range, as calculated from a linear multi-stage model. Most of those decisions incorporated consideration of costs and feasibility.

The negligible risk concept considered here is based strictly on individual risk rates and exposure. It does not include other economic or technical considerations that are part of setting the enforcement standards (i.e., the MCLs). The level for the MCLs (not RMCLs) would thus be considered to be the upper limits of risk that are considered to be acceptable based upon our current evaluation of the feasibility and costs of controls.

Under this approach to setting drinking water RMCLs, EPA has considered two risk levels as possibly representing an upper limit for a risk: one in 100,000 (10^{-5}) probability per 70 years of exposure and one in 1,000,000 (10^{-6}) probability. An incremental lifetime risk level of 10^{-6} would probably be more representative than 10^{-5} as the "no effect" level for these chemicals in drinking water with a margin of safety as envisioned by Congress. The NDWAC stated that 10^{-5} would be an appropriate target. However, a level of 10^{-6} is the level of concern that commonly has been discussed as the lower limit of concern over the potential health risks of exposure, especially for the generally involuntary risk from exposure to a drinking water contaminant.

In addition, if RMCLs were to be set at a non-zero level, use of the linearized multi-stage model would often appear to be more appropriate than others to meet the Congressional intent. The conservative nature of the model could actually mean that the real risk of exposure was probably lower (e.g., 10^{-7} or 10^{-9}) if any risk actually exists (assuming a non-threshold mechanism were operative) because the model was structured to be conservative and because of the nature of many of the assumptions in the model.

As an example of what 10^{-6} would mean in terms of the U.S. population, a total of 20 cases of cancer would result if 10 percent of the population were exposed at a dose level equivalent to a 10^{-6} risk for 70 years. Stated another way, that would be one-third of a cancer case per year as an upper limit in the U.S. population compared to the appropriately 500,000 annual cancer deaths that occur. The actual number of cases attributable to that particular substance would probably be less and perhaps none at all would occur unless some additive or synergistic interaction with other substances resulted in enhanced toxicity.

Proposed RMCLs: Conclusions. This proposal selects RMCLs for potential carcinogens at zero; the alternatives were carefully considered in view of the intent of the SDWA and public comments. It should be recognized that regardless of which of the three alternatives is ultimately selected for the RMCL, it is unlikely that the MCL for a particular substance would be affected, since normally all of the approaches would yield targets that are likely to be below levels that are "technically and economically feasible" using available technologies. MCLs will be set as close to the RMCLs as feasible. Preliminary analyses indicate that the MCLs may fall roughly in the range of 5 to 50 μg/l for most of the VOCs being considered in this proposal.

Proposed RMCLs for the following substances considered carcinogenic are "zero": tetrachloroethylene, trichloroethylene, carbon tetrachloride, 1,2-dichoroethane, vinyl chloride, benzene, 1,1-dichloroethylene.

The proposed RMCL for 1,1,1-trichloroethane is 0.2 mg/l, derived from the calculated AADI of 1.0 mg/l assuming 20 percent contribution from drinking water to total exposure. If the preliminary NTP report on the carcinogenicity of this compound is affirmed, the RMCL would be zero. EPA would provide formal notice if and when this occurs.

The proposed RMCL for p-dichlorobenzene (1,4-dichlorobenzene) is 0.75 mg/l, derived from the calculated AADI of 3.75 mg/l assuming 20 percent contribution from drinking water to total exposure.

Three of these substances (trichloroethylene, tetrachloroethylene and 1,1-dichloroethylene) have only "limited" animal evidence of carcinogenicity, as this term is used in the IARC criteria. Factors which contribute to this classification include lack of replication in multiple experiments or multiple species, as well as defects in particular studies. In addition, indicators of certain types of tumors, such as in the mouse liver, are considered by some scientists to have less weight than others in predicting carcinogenicity in humans. Data of this type, obtained by corn oil gavage, introduces another variable that

complicates interpretation. While evidence for these three substances is of a weaker nature than for others that EPA is proposing to regulate as carcinogens, it is nevertheless evidence that must be weighed by the Administrator.

The strictly scientific evaluation of such evidence (known as "risk assessment") can only describe its strength and weaknesses. EPA's risk assessment is summarized above and described in detail in the documents referenced in Section VII. Health Assessment documents for these three substances were reviewed by EPA's Science Advisory Board in April and May of 1984. Those reviews will be considered in this rulemaking action under the SDWA and become part of the record.

Decisions about what actions to take on the basis of the evidence (known as "risk management"), including decisions about how strong the scientific evidence should be to justify regulating a substance, require policy judgments which must be made by the Administrator, after public comment, in the light of the Agency's statutory mandates.

EPA strongly believes that its risk assessments should be consistent among Agency programs. On the other hand, risk management decisions can and should vary in the light of differing circumstances or statutory mandates. It is therefore possible that some of these substances might be regulated differently in other Agency programs. For example, EPA plans to decide whether to list several of these substances as hazardous air pollutants under section 112 of the Clean Air Act. The same scientific evidence will be considered along with other factors relevant to that decision. This may or may not lead to a conclusion to list and to regulate them as carcinogens.

Public comments are requested on setting RMCLs for carcinogens at zero, the analytical detection limit, and at some finite value based upon risk estimation. Comment is also requested on appropriate analytical detection limits, and on the method for calculating the finite risk value and for determining the risk target. Comments are also requested on the RMCLs for non-carcinogenic substances and the assumption of an exposure factor of 20 percent from drinking water, absent quantitative multi-media exposure data.

Comment is also directed to technical determinations, AADI calculations, the draft revised CAG risk calculations, and the inclusion of substances with "limited evidence" in the carcinogen category. If, on the basis of the record, it is determined that one or more of these substances should not be treated as carcinogens, then the AADI calculations modified by an allocation of 20 percent to drinking water would be the basis for the promulgated RMCL.

VI. Other Considerations for Public Comment

The next regulatory steps will be promulgation of the RMCLs and proposal of MCLs and monitoring and reporting requirements. Supporting documentation for the MCL proposal will include: (1) Exposure and risk assessments, (2) an assessment of generally available technology, (3) an assessment of available analytical methods and costs of monitoring, and (4) an economic and financial impact analysis. Available information to support several of the assessments is referenced in the next section. The public is requested to review those references and provide comments and other supporting information and data. The public is also requested to comment on the issues and information discussed below on available treatment techniques and costs and current estimates of the potential impact of VOC regulations.

Treatment of Control of VOCs

Economics, treatment technologies and feasibility are not factors involved in the determination of RMCLs; however brief discussions are provided here. These factors are key elements in the determination of the MCL which will be proposed when the RMCLs are promulgated.

Methods for removal of these volatile organic chemicals include aeration and granular activated carbon (GAC). The available data do not show powdered activated carbon treatment or conventional drinking water treatment (i.e., coagulation, sedimentation, and filtration) to be sufficiently effective for long term application. Macroreticular resins may eventually prove to have value for removing VOCs; questions still exist concerning their use. Data describing actual exhaustive capacity of the resins are not available to define the regeneration frequencies to be expected with the resins. Thus, costs have not yet been estimated for application of resin technology. At this time, substantial operational experience and/or experimental data are available only for aeration and GAC.

Costs of treatment. Preliminary designs and cost estimates have been developed for a hypothetical ground water contamination situation involving trichloroethylene (TCE). Table 8 provides relevant estimated cost information for treatment of TCE at the 90 percent and 99 percent removal levels, respectively, for aeration and GAC technologies.

TABLE 8.—PRELIMINARY COSTS FOR CONTROLLING TCE IN DRINKING WATER

[1983 dollars]

Type of treatment	Estimated costs—System size—Population served		
	100 to 500 (0.05 mgd)	1,000 to 2,500 (0.43 mgd)	10,000 to 25,000 (4.0 mgd)
Packed tower aeration:			
For 90 percent removal, e.g., source 500 µg/l MCL 50 µg/l:			
Capital cost	$16,000	$90,000	$269,000
Cost per thousand gallons (cents)	128	34	8
For 99 percent removal, e.g., source 500 µg/l MCL 5 µg/l:			
Capital cost	$26,900	$129,000	$510,000
Cost per thousand gallons (cents)	181	41	11
Granular activated carbon:			
For 90 percent removal, e.g., source 500 µg/l MCL 50 µg/l:			
Capital cost	$28,400	$84,000	$486,500
Cost per thousand gallons (cents)	143	36	19
For 99 percent removal, e.g., source 500 µg/l MCL 5 µg/l:			
Capital cost	$28,400	84,00	486,500
Cost per thousand gallons (cents)	149	39	22

Basis for designs:
Notes:
For packed tower aeration—fiberglass reinforced plastic shell with plastic packing material and separate housing; Kavanaugh & Trussell design procedure; contingency factors of 25 percent for engineering, 25 percent for overhead and profit, 25 percent for shipping and installation; electricity costs 8 cents per kWh; interest rate of 12 percent; amortization period of 20 years.
For granular activated carbon—empty-bed-contact-time of 10 minutes; pressure contractors based upon quoted prices of various manufacturers; initial charge carbon costs 65 cents per lb.; contingency factors of 25 percent of engineering, 25 percent for overhead and profit, 25 percent of shipping and installation; electricity costs 8 cents per kWh; interest rate of 12 percent; amortization period of 20 years.

Potential Impact of Regulations

The nominal limits of detection attained by the laboratories performing analyses in the GWSS were usually in the 0.2 to 0.5 µg/l range depending upon the specific chemical, although it appears that precision and accuracy requirements for regulatory compliance determination might require that regulations (MCLs) be set at least one

order of magnitude higher. The feasible application of aeration and granular carbon might also lead to MCLs in a similar range, i.e., on the order of 5–50 µg/l. At this level, very preliminary projections are that about 1,000 systems would probably need to reduce VOC levels either through treatment technologies or other options such as blending or shutting down wells; most of these would be communities utilizing ground water.

Therefore, based upon current exposure estimates, risks of most VOCs would not appear likely to represent a high impact, nor would regulations result in a significant number of cancer cases avoided based upon total cancer rates and projected risks using the linear multi-stage model. Although VOC contamination is widespread across the country, it is usually at low levels, and the overall population at risk is quite low. Moreover, most VOCs do not appear to be highly potent carcinogens. However, in those communities where exposed levels are relatively high, resulting in correspondingly higher risks per individual, control is obviously essential. On the other hand, where that is not the case, the non-quantifiable benefits would probably be of most importance in determining the proper approach. These would include such items as providing federal standards to be used as a benchmark in responding to incidences of contamination, and use in ground water protection and clean-up programs.

Economic impact analysis. The proposal of an RMCL is different than proposal of an MCL in that an RMCL is, by law, to be based only on health and safety considerations, while an MCL is to take costs into consideration. Therefore, this RMCL proposal notice does not include an analysis of the economic impacts of various possible RMCLs. However, we intend to fully analyze the probable impacts of the various MCL alternatives, and will report on them at the time an MCL is proposed.

Because the economic impact analysis is an import part of the rulemaking decision process, and because some reviewers of this notice may be concerned that insufficient attention is being paid to economic considerations, below is a brief indication of how EPA will conduct the economic analysis of alternative MCLs, and what is considered from the results of the analysis.

Executive Order 12291 and the Regulatory Flexibility Act specify how and when to analyze the probable impacts of a Federal action. In essence, information on the impacts to industry, consumers and the nation is assembled. Where possible, this information is put in the form of an analysis of the net benefits of the various alternatives. This "regulatory impact" information then becomes a part of the official record in support of whatever action EPA finally takes, and is used by decision-makers when an alternative MCL is selected for proposal, and when final MCL is promulgated.

The types of impacts which will be examined for each of the various regulatory and non-regulatory alternatives are of three basic types. The impacts of the alternatives on the water supply industry wil be examined. This will be done by reviewing three elements, the capital cost of technology, the operating and maintenance cost and the feasibility of financing new treatments. The first two elements are derived by the engineering analysis of treatment technologies, and the cost of treatments. The third element, the ability to finance new treatments is derived from an analysis of the water supply industry. A financial model of the industry has been developed by EPA, and this model indicates how likely it is for water systems to be able to finance new treatments, based on the historical financial performance of water systems.

The second type of impact is the impact on the consumer. Information on the cost of water to consumers is assembled, based on the cost data prepared during the engineering analysis of treatment technologies. These costs are compared with the historical cost of water.

The third type of impact is the impact on the nation as a whole. The purpose of this analysis is to allow balancing of the cost of a federal action, in this case MCLs, with the benefit to be derived from the action. In some cases, it is not possible to describe the value of the benefits in the same terms as the costs, i.e., dollars. The benefits which will accrue to the nation are derived from an analysis of the contamination occurrence, the reduction in human exposure likely to result from an alternative, and the health effect averted by the reduction.

The cost of the various alternative MCLs is more than merely the cost to industry. It also includes the cost to government of implementing the regulation. These national costs are summarized and presented with the national benefits, and this too becomes a part of the record supporting the proposed and final MCL.

Because these various analyses are based on estimates, an additional analysis is conducted which indicates the sensitivity of analytical results to the assumptions maded during the analysis. This sensitivity analysis completes the general regulatory and non-regulatory analysis required under E.O. 12291. A summary of these analyses will be presented in the preamble of the MCL proposal notice, and full documentation of the underlying analyses will be entered into the formal record of the rulemaking procedure.

VII. References

The following supporting documentation for this proposal is available on request from the address listed at the beginning of this notice.

Bellar, T.A., Lichtenberg, J.J. "The Determination of Halogenated Chemical Indicators of Industrial Contamination in Water by the Purge and Trap Method: Method 502.2." U.S. EPA. EMSL #600/4–81–059.

Bellar, T.A., Lichtenberg, J.J. "The Analysis of Aromatic Chemicals in Water by the Purge and Trap Method: Method 503.1," U.S. EPA, EMSL. EPA 600/4–81–057.

Love, O. Thomas, Jr. and Richard G. Eilers, "Treatment for the Control Trichloroethylene and Related Industrial Solvents in Drinking Water," U.S. EPA, Office of Research and Development, February 1981.

Environmental Science and Engineering, "Treatment for Control of VOCs in Drinking Water," August 1983.

EPA, Criteria and Standards Division, Draft Criteria Document for Trichloroethylene, EPA, Office of Drinking Water, February 1984.

EPA, Criteria and Standards Division, Draft Criteria Document for Tetrachloroethylene, EPA, Office of Drinking Water, February 1984.

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EPA, Criteria and Standards Division, Occurrence of Volatile Organic Chemicals in Drinking Water, 1,1,1-Trichloroethane, June 1982.
EPA, Criteria and Standards Division, Occurrence of Volatile Organic Chemicals in Drinking Water, 1,2-Dichloroethane, November 1983.
EPA, Criteria and Standards Division, Occurrence of Volatile Organic Chemicals in Drinking Water, Carbon Tetrachloride, November 1983.
EPA, EMSL, "Total Organic Halide, Method 450.1-Interim," EPA 600/4-81-056.
EPA, Office of Health and Environmental Assessment, Draft Health Assessment Document for Vinylidene Chloride, Office of Research and Development, October 1983.
EPA, Office of Health and Environmental Assessment, Draft Health Assessment Document for Carbon Tetrachloride, Office of Research and Development, August 1983.
EPA, Office of Health and Environmental Assessment, Draft Health Assessment Document for 1,1,1-Trichloroethane, Office of Research and Development, November 1983.
EPA, Office of Health and Environmental Assessment, Draft Health Assessment for Tetrachloroethylene, Office of Research and Development, December 1983.
EPA, Office of Health and Environmental Assessment, The Carcinogen Assessment Group's Evaluation of the Carcinogenicity of Benzene (DRAFT), Office of Research and Development, March 1983.
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EPA, "Review of a Carcinogenicity Study on Vinyl Chloride," Memo from Robert E. McGaughy, Office of Research and Development to Joseph A. Cotruvo, Office of Drinking Water, Jan. 6, 1984.
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VIII. Request for Comments

EPA requests public analyses, comments and information on all aspects of this proposal. The questions for which comment is being specifically solicited are listed below. Comment will be of great assistance to EPA in formulating a protective and practical approach to reducing human exposure to VOCs in drinking water.

• How strong should the scientific evidence be to justify regulating a substance, particularly for carcinogenicity?

—When positive evidence exists but is sparse or inconclusive, how should it affect decision-making? Should there be a well-defined and uniform minimum level of evidence of carcinogenicity in animals or humans? If so, what evidence would comprise this minimal level?

—When substantial doubt exists as to whether a substance causes a serious health or environmental risk, how should EPA balance its mandate to err on the side of protection against the competing risk of imposing costly regulations on substances which may later be shown to be benign?

• How should evidence of mouse liver tumors be weighed? If evidence is limited to mouse liver tumors, is that sufficient evidence to warrant regulating that substance as a carcinogen? Conversely, what would be the scientific basis for giving mouse liver tumors less weight in the evaluation of the potential for human carcinogenicity?

• What level should be set for RMCLs that would represent a level such that "no known or anticipated adverse effect would result with an adequate margin of safety"?

—For non-carcinogens, is the approach used for computing the AADIs scientifically acceptable? Is providing for an assumed contribution of 20 percent from drinking water appropriate when more precise data is not available.

—Should RMCLs for carcinogens be set at zero? If RMCLs are set at zero, what guidance, if any, should be provided on the actually attainable target levels in drinking water?

—Should RMCLs for carcinogens be set at the analytical detection limit? What would this be for each VOC considered in this proposal?

—Should setting RMCLs for carcinogens be established at a non-zero level based upon a negligible risk determination? What non-zero level and upon what basis? Which model and which assumptions? Does an incremental lifetime risk level of 10^{-6} represent a virtually non existent or negligible risk? Should higher or lower risk rates be considered? Would another level be more representative yet meet the needs for practical implementation of the SDWA? Would use of the linearized multi-stage model in the non-zero RMCL calculations meet the Congressional intent to incorporate a margin of safety into the RMCLs?

—Should a range of finite risk levels for each RMCL be selected such as 10^{-5} to 10^{-6} instead of zero or a single value?

• How should the degree of evidence of potential carcinogenicity be factored into the RMCL determinations? If there is sufficient experimental evidence of human carcinogenicity, should the RMCL be either zero or the one in one million risk equivalent, or some other calculated value? Should the RMCL be set at a higher concentration and higher nominal risk (to indirectly reflect less concern) as the strength of evidence of carcinogenicity is reduced? For example, if there is only sufficient evidence of animal carcinogenicity, should the RMCL be in the 10^{-5} up to the 10^{-6} range, whereas if there is only limited evidence of animal carcinogenicity, should the RMCL be in the 10^{-4} to 10^{-5} risk range? If less than "limited evidence" is available, should the RMCL be determined based upon an ADI calculation?

As another example, could RMCLs for substances such as TCE and PCE with limited, insufficient, or less convincing evidence of carcinogenicity be produced on the basis of chronic toxicity, but with an additional margin of safety or based upon the minimum measured cancer producing dose level such as was suggested by Weil (Toxicology and Applied Pharmacology *21* 454–163 (1972))? This would differentiate those from substances such as benzene or vinyl chloride which have the most complete evidence and therefore warrant the most conservative regulatory treatment.

• Should an RMCL and an MCL be set for total VOCs to address multiple exposure to VOCs? On what basis?

A public hearing will be held in Washington, D.C., for the interested public to comment and provide information and data on the regulatory approach.

EPA recognizes that many significant questions surround the issue of the control of volatile synthetic organic chemicals in drinking water. The Agency has attempted in this proposal to portray current scientific uncertainties in a measured and objective manner. In this way, any data gaps or errors in logic which may exist can be identified and corrected. For that reason, careful review of and thoughtful comment on the information in this proposal is encouraged.

Under the Regulatory Flexibility Act, 5 U.S.C. 601 et seq., I certify that this action will not have a significant impact on a substantial number of small entities. This proposed action will have no economic impact in and of itself because these are non-enforceable health goals.

Under Executive Order 12291, EPA must judge whether a regulation is "major" and therefore subject to the requirements of a Regulatory Impact Analysis. This proposed action does not constitute a "major" regulatory because it will not have a major financial or adverse impact on the community and it is a non-enforceable action. This regulation was submitted to the Office of Management and Budget for review as required by Executive Order 12291.

List of Subjects in 40 CFR Part 141

Chemicals, Water supply.

(42 U.S.C. 300/SDWA 1412

Dated: June 1, 1984.

William D. Ruckelshaus,

Administrator.

For the reasons set out in the preamble, Part 141 of Chapter I of Title 40 of the Code of Federal Regulations is proposed to be amended as follows:

PART 141—NATIONAL PRIMARY DRINKING WATER REGULATIONS

1. The title of Part 141 is revised to read as set forth above.

2. In § 141.2, paragraph (u) is added to read as follows:

§ 141.2 [Amended]

* * * * *

(u) "Recommended maximum contaminant levels" means the maximum level of a contaminant in drinking water at which no known or anticipated adverse effect on the health of persons would occur and which includes an adequate margin of safety.

3. A new Subpart F, consisting of §§ 141.50 and 141.51, is added as follows:

Subpart F—Recommended Maximum Contaminant Levels

§ 141.50 Recommended maximum contaminant levels for organic chemicals.

The following are Recommended Maximum Contaminant Levels for organic chemicals. They are non-enforceable goals for public water systems.

(a) Recommended Maximum Contaminant Levels are zero for the following substances: trichloroethylene, tetrachloroethylene, carbon tetrachloride, 1,2-dichloroethane, vinyl chloride, 1,1-dichloroethylene, and benzene.

(b) Recommended Maximum Contaminant Levels for the following substances are as indicated:

	Milligrams per liter
1,1,1-trichloroethane	0.2
p-dichlorobenzene (1,4-dichlorobenzene)	0.75

10 mg/l = 1 ppb

§ 141.51 [Reserved]

Appendix A—Summary of Public Comments Pertinent to the Proposed Recommended Maximum Contaminant Levels (RMCLs) for Volatile Synthetic Organic Chemicals (VOCs) in Drinking Water

The following is a summary and discussion of the principal public comments to EPA's proposed rule for the establishment of RMCLs for certain VOCs in drinking water. EPA specifically solicited comments on the following three issues in its March 4, 1982, Advance Notice of Proposed Rulemaking:

1. What is the significance of contamination of drinking water by VOCs?

2. What approach should EPA take to deal with VOCs in drinking water?

3. What level should be set for RMCLs such that, "no known or anticipated adverse effect" will result? How should the health basis be determined for any MCLs?

EPA received 136 written comments during the 210-day public comment period and five oral statements were presented at the public meeting held in Washington, D.C., on April 28, 1982. The comments included 26 public interest groups, 14 water utilities, 10 chemical manufacturing companies, 11 state governments and state organizations, 12 local governments, 40 private citizens and 18 from other groups including some members of Congress.

The following discussion summarizes comments received on the ANPRM for VOCs.

1. What is the significance of contamination of drinking water by VOCs?

A total of 66 commenters addressed this issue. A majority of comments (41) felt that VOC contamination in drinking water is a significant national problem because of the frequency of occurrence and potential health risk warranting action to limit exposure to VOCs. Their reasoning is based on the following: Local problems of severe VOC contamination, the number of VOCs in drinking water is continually increasing and VOCs have been demonstrated to cause serious carcinogenic and non-carcinogenic toxic effects. Some of the toxic effects are as follows: Some VOCs are known animal carcinogens and vinyl chloride is both an animal and human carcinogen, causes hepatomas in animals and in some cases in humans, is toxic to the kidneys, has serious effects on the reproductive system, and depresses the central nervous system.

One commenter stated that the results of a monitoring study conducted by New Jersey showed 17 percent of the 1,200 wells tested contained VOCs at concentrations above 10 ppb. They felt that, "the toxic properties of these chemicals, including the potential increased risks of cancer and birth defects, warrant federal action".

Twenty-five commenters felt that neither the occurrence data, the health effects data, nor the combined data, demonstrate on a national basis the significance of VOC contamination in drinking water; therefore action to limit human exposure to VOCs is not warranted. Reasons cited were: VOC contamination in drinking water is a localized problem, not a widespread national problem; more information is needed on occurrence and health effects, especially in order to assess the significance of VOC contamination; state data represented emergency spill situations which are not considered to be statistically representative of national occurrence; when present, VOCs usually occur at low part per billion concentrations, whereby significant health risks would not be expected; and the results of the Ground Water Supply Survey (GWSS) should be considered questionable because the detection limits that were used (i.e., 0.2 µg/l) are extremely sensitive and can rarely be reached. One comment stated that "positive occurrence data does not present a case for regulation".

In general, these commenters felt that the major shortcomings of the available health effects data is that it is not scientifically established at this time and subject to debate among the scientific community. One commenter specifically stated that "until the extent of the threat of human health of VOCs, if any, can be established, federal regulations governing VOCs are not justifiable on mere occurrence data alone".

2. What approach should EPA take to deal with VOCs in drinking water?

A total of 118 comments were received that addressed this issue. Comments favored one of the three approaches provided in the ANPRM which are:

- Non-federal regulatory approach
- Establish montioring requirements and provide Health Advisories (formerly termed SNARLs) for State response as appropriate
- Establish national regulations for monitoring and MCLs

The non-federal regulatory approach was favored by 22 commenters. Several commenters stated that recent surveys by EPA and their own sampling did not indicate a major VOC contamination problem within certain States, thus, MCLs and monitoring requirements are not warranted.

A number of commenters felt that the health effects data were insufficient to show a health risk that would warrant regulation. One commenter stated that "EPA has not presented any evidence that there is any risk to the population requiring Federal regulation. Furthermore, where the contamination is the result of improper disposal of solvents, guidance is needed by those implementing the RCRA/Superfund cleanup as to an adequate effort". One commenter summed up this sentiment as "there is a need for a rational[e] and consistent approach to the problem of low levels of carcinogens in drinking water, but the science is not sufficiently developed to guide regulatory and utility actions with any degree of certainty".

Some commenters are opposed to national regulations for VOCs because, "shrinking federal and state resources are creating problems for the already existing drinking water programs". They felt that "EPA should focus on source-protection and rapid reaction to ground water contamination than attempt to cover all possibilities by regulation".

The majority of these commenters favored the continued use of Health Advisories to handle contaminant situations. However, two comments were received that specifically requested that "once a health advisory is released, it should be published in the Federal Register, detailing EPA's derivation of the health advisory". "Based on the scientific input and occurrence information received, EPA must issue an updated health advisory and can then decide if there is a need to establish MCLs." One commenter stated that "Health Advisories should be the first step in determining whether or not it is necessary to establish an MCL for a particular contaminant". In addition, one commenter favored the use of Health Advisories as opposed to national monitoring requirements, in that the latter would only gather more occurrence data.

In general, these commenters favored EPA continuing to provide research data and technical advise (i.e., Health Advisories) when dealing with contaminant situations. In addition, "routine, repetitive monitoring requirements must not be put into regulations because monitoring programs must be flexible and can best be developed by States and water utilities".

Option 2, whereby EPA should establish monitoring requirements and provide Health Advisories for State response as appropriate was favored by 13 commenters. The basis for the comments which recommended this approach was two-fold: (1) Localized VOC contaminant situations, especially in ground water necessitates monitoring requirements, and (2) the health effects data is unclear and insufficient to establish MCLs since "safe" levels of VOCs cannot be determined at this time. Health Advisories should be used in dealing with contaminant situations.

Generally, these commenters felt that gound water contamination is a problem in some places, which must be addressed; however, VOC contamination is not widespread enough to require highly formalized and restrictive requirements. Furthermore, the available data are insufficient to determine the scope of the problem and only monitoring should be done to determine where problems exist. Therefore, instead of setting MCLs, guidance should be provided. These commenters generally supported giving States considerable authority for implementation of the monitoring requirements and for determining appropriate action when contamination is found.

A few of the comments received, which favored the monitoring requirements option, proposed an action-oriented approach in the form of contamination levels and action categories. They felt that guidance for five of the VOCs should be established as follows:

Compound	Concentration levels categories (ug/1)		
	I	II	III
Vinyl chloride	> 100	10–100	< 10
Trichloroethylene	> 500	50–500	< 50
Tetrachloroethylene	> 500	50–500	< 50
Carbon tetrachloride	> 500	50–500	< 50
1, 2-Dichloroethane	> 250	25–250	< 25

Note:
Category I—high concentration and consequently greater risks. Immediate action warranted to reduce contaminant level.
Category II—intermediate concentration with lower risk. Prompt action warranted to step up surveillance and consider control strategies. Action should reflect whether the concentration is at higher or lower part of the range.
Category III—very low concentration. Little risk associated with these concentrations. Only routine monitoring is needed.

The third option which would require establishing national regulations for monitoring and MCLs, was favored by 82 commenters. Numerous commenters stated that MCLs and monitoring requirements should be set for the VOCs. A number of these commenters qualified their statements by saying MCLs should be set if it is shown that the occurrence of VOCs is widespread and the health effects data show that VOCs are a health risk. However, most commenters felt that sufficient data were available showing VOCs to be a widespread problem, that data did show a potential health hazard, and that MCLs were needed.

The following related statements were made:

- Problems with organic chemicals have been shown in several States and without enforceable standards; the problems will continue to spread; the justification for cleaning contaminated aquifers will be challenged on a case-by-case basis.
- Consistent, nation-wide standards are needed for VOCs (numerous commenters explicitly stated that "uniform, mandatory and enforceable standards" are needed) to provide adequate public health protection in each State. Latitude for stricter rules by the States was suggested by one commenter.
- While Health Advisories were noted to have been very useful in addressing incidences of VOC contamination, several public water systems commented that the States have adopted widely varying approaches to dealing with VOCs in drinking water. Some States have adopted the Health Advisories as enforceable standards and consequently public water systems have been forced to make permanent and costly decisions on the basis of health guidance.
- Alternative involving determination of the acceptable levels of contaminants by individual States, based on EPA advisory options, will not be effective. Their reasoning is that EPA advisory

opinions on health effects will be misinterpreted and misused as demonstrated by many States that have used the Health Advisories as rigid criteria by which suitability of a water supply is measured. The fact that Health Advisories are developed without consideration for possible carcinogenic properties of a compound and with disregard for economic and technological feasibility of achieving them is forgotten. We are better off with MCLs established in the process involving public participation and *intended* to be the rigid standards by which quality of drinking water is measured.

• If left to the States, drinking water guidelines will differ from each other. "This will lead to confusion and poor public image for State agencies which recommend guidelines less stringent than others. In addition, leaving the regulatory activity to States will require more human resources in the area of toxicological evaluation and standard setting. Many States do not have these resources. Thirdly, enforcement will be very difficult, if not impossible, if neighboring States have different drinking water standards. Contamination has no boundaries."

One commenter summarized their argument for MCLs in the following manner: "Contamination of water supplies in the U.S. with VOCs is indicative of a national trend. Setting legally enforceable national standards will be important in reversing that trend. It will establish a ceiling on how much contamination of drinking water is acceptable and will trigger remedial action in situations where the ceiling is almost reached or exceeded."

A number of comments were received that addressed monitoring requirements and treatment costs; however, these comments will be addressed when the proposed rule for the establishment for MCLs is published.

3. How should the potential health risks of exposure to VOCs be assessed? What level should be set for RMCLs such that "no known or anticipated adverse effects" will result?

In assessing the potential health risks of exposure to VOCs, numerous questions arise such as:

(1) Whether or not a compound can be classified as "genotoxic" or "non-genotoxic"?

(2) Should different risk models or approaches be used for carcinogens that are not genotoxic?

(3) Would the risk of exposure to two or more VOCs be considered additive, antagonistic, synergistic?

(4) Which subgroup of the general population should be addressed?

(5) How should exposure to VOCs from other routes (i.e., air or food) be addressed?

Consideration of the potential health effects of a chemical encompasses the evaluation of available data and the potential for human health effects from exposure via drinking water. A number of comments addressed the aforementioned issues.

First, the issue as to what criteria could be used to classify a compound as "genotoxic" or "non-genotoxic" was addressed by seven comments. Four of the commenters suggested that the criteria to classify a compound as "genotoxic" include:

1. a reliable, positive demonstration of genotoxicity in appropriate prokaryotic and eukaryotic systems *in vitro;*

2. positive results in studies on binding to DNA; and

3. evidence of biochemical or biologic consequences of DNA damage.

One commenter submitted data and information on recent developments in the understanding of the various mechanisms by which a carcinogenic response can be produced in laboratory animals. These comments can be summarized as follows:

> Based on the extent of a chemical's interaction with DNA, it appears that chemicals that have a greater propenity to directly interact with DNA are appropriately classified as genotoxic. Those that do not have this propensity to interact directly with DNA, but lead to tumors via recurrent tissue injury or other secondary events are classified as non-genotoxic or epigenetic carcinogens. The carcinogenic risk to man posed by such epigenetic carcinogens appears to be substantially less than that posed by purely genetic carcinogens. Whereas, there has been relatively less disagreement over appropriate measures for the control of those materials categorized as human carcinogens, there has been considerable disagreement among scientists regarding appropriate measures for the control of the numbers materials categorized as animal carcinogens on the basis of tests in rats, mice or hamsters.

The above commenters stated that different risk models should be used to account for the differentiation of carcinogens recognizing different mechanisms. All of these comments reject the CAG risk model because it is too conservative and that both the upper and lower bound risks must be taken into account. In other words, EPA's cancer risk estimation process overstates the potential risk posed by these chemicals in a manner which may mislead the public. Furthermore, they believe that EPA has accepted animal data at face value without any critical review. They recommended that the health criteria documents be subject to independent peer review before further government and industry resources are spent on discussing approaches to regulate chemicals which may be non-hazardous or pose an insignificant risk.

Three comments were received that recommended EPA should continue to use the CAG model for both genotoxic and nongenotoxic carcinogens. Even though knowledge of the carcinogenic mechanism should be a major factor in selecting the most appropriate risk model, this information is generally not available for environmental carcinogens. One commenter stated that a distinction between caracinogenic mechanisms is arbitrary because there is a lack of experimental data establishing a threshold for non-mutagenic carcinogens or showing that the dose-response curve is different in the lower range from that for substances that cause gene mutations. In addition, thresholds observed in experiments with an inbred animal populations cannot be estrapolated with any degree of certainty to a diverse human population; therefore, no distinction between carcinogenic mechanisms should be made at this time.

One commenter added that the multi-stage model as modified by the Carcinogen Assessment Group (CAG) should be used in establishing MCLs for carcinogens regardless of mechanisms of action. Mathematical models at best provide crude estimates of the risks resulting from exposure to a carcinogen.

The third issue as to how the risk of exposure to two or more VOC's should be considered was addressed by five comments. Four commenters felt that two or more chemicals found to be toxic to the same organ system should be considered to be additive in their cumulative effect on the body. Added margins of safety should then be included in the health basis of each MCL. The magnitude of the safety factor should reflect where possible, current understanding of synergistic interaction between chemicals and should be considered at least additive in proportion to the absolute and relative levels of exposure. In addition, no evidence has been put forth that suggests that these interactions could never be a problem. One recommended approach was to set an RMCL and MCL for "total VOCs".

One commenter believed that the risk of exposure to two or more VOCs is *not* additive. The reasoning was based on two studies in which two chlorinated solvents were administered simultaneously for 3 to 6 months, in which no synergism was indicated and, in fact, the effects were less than

additive. Thus, the commenter felt that an increase in the margin of safety is not required.

The issue of which subgroup of the general population should be protected received six comments. Three commenters felt that the 10 kg child should be used because adequate protection should be provided to all segments of the general population. Two commenters felt that the 70 kg adult should be the basis for potential MCLs because life-time exposure should be used in the calculation. A 10 kg child is not exposed over a 70-year lifetime. One commenter recommended that if MCLs are warranted, the level should be set to protect all signficant populations groups (i.e., children, pregnant women, aged adults, etc.). Also, short term exposure risk calculations should be based on a 10 kg child, long term exposures based on a 70 kg adult, and the worst case would be controlling.

Lastly, the issue as to how exposure to VOCs from other routes should be addressed in the development of RMCLs received eight comments. Five commenters felt that relative source contribution should *not* be a major factor in determining the acceptable risk. One commenter suggested that EPA state the likely other sources of VOC exposure and average levels. Another commenter put it this way, "The contribution of drinking water to the total exposure to a contaminant should be considered in light of the risk to public health and not in terms of its relative significance to other sources of exposure".

Three comments recommended that the total allowable body burden from all media (air, food and water) should be taken into account, based on health effects data.

Twenty-six comments were received on what level should be set for RMCLs such that "no known or anticipated adverse effects" will result. Twenty-two commenters recommended that the RMCLs for carcinogens be set at zero. Their reasoning was based on the premise that an RMCL is a health goal, which is not intended to reflect cost and feasibility of treatment, and that scientific evidence to date cannot be used to establish a no adverse health effect threshold for carcinogens.

Four commenters recommended that the RMCLs for carcinogens be set at a finite risk level and not zero. Their reasoning was that every water supply contains at least some of the chemicals listed in the ANPRM. A finite risk level is the only realistic basis. Furthermore, it is impossible to establish with any degree of certainty that the concentration of a contaminant in water is zero, due to limited analytical capability. One commenter stated that, "The question of the level of the RMCL for carcinogens is the most fundamental in the ANPRM. RMCLs are confusing and an RMCL set at zero is not useful because it could not be measured." Instead a regulatory target level (RTL), set as a negligible risk level should be established. The level should be 10^{-5}, based upon the National Academy of Sciences projections, not CAG's."

Another commenter felt that "RMCLs for compounds shown to increase tumors in test animals through non-genetic mechanisms, should be set at a finite number based on the toxicity of the contaminant (i.e., incorporating the threshold concept)".

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Journal

CONGRESS

Senate Bills and Resolutions Introduced

June 6, 1984.

(INTERIOR) to amend the Wild and Scenic Rivers Act to permit control of the lamprey eel and to designate a portion of the Au Sable River in Michigan as a component of the National Wild and Scenic Rivers System. S 2732 (Levin and Riegle) Energy.

June 7, 1984.

(HAZARDOUS WASTE) to amend the Solid Waste Disposal Act to clarify liability for criminal acts involving hazardous wastes. S 2741 (Lautenberg and Bradley) Environment.

Senate Committee Action

June 7, 1984.

Appropriations, approved HR 5713, authorizing fiscal 1985 funds for the Environmental Protection Agency.

House Bills and Resolutions Introduced

June 6, 1984.

(ENVIRONMENT) to amend the Clean Air Act to control acid deposition, and for other purposes. HR 5794 (Eckart and Others) Energy.

EXECUTIVE BRANCH

President of the United States

June 12, 1984.

President Reagan signed into law S 518, the Environmental Programs Assistance Act of 1984, directing EPA to develop programs to assist state and local governments in using the talents of older Americans to aid in pollution abatement and control.

Departments and Agencies

Council on Environmental Quality June 11 announced three open meetings to discuss draft contractor report on a national research center on water resources and national clearinghouse for water resources information. The meetings are scheduled for: Aug. 22 at 10 a.m. in Marlboro Rooms A & B, New Orleans Hilton Hotel, No. 2 Poydras St., New Orleans; Aug. 24 at 10 a.m. in the Golden/Baldwin Rooms, Rodeway Inn, Airport, Denver; and Aug. 27 at 10 a.m. in the Amphitheater, Federal Home Loan Bank Board, 1700 G St., N.W., Washington, D.C. To make presentations, contact Harvey Doerksen, Project Director, Environmental Monitoring and Data, CEQ, 722 Jackson Pl., N.W., Washington, D.C. 20006; telephone (202) 395-5754.

Environmental Protection Agency June 8 approved Delaware's application for final authorization of its hazardous waste management program under the Resource Conservation and Recovery Act (49 FR 23837).