NATIONAL INTERIM PRIMARY DRINKING WATER REGULATIONS

For Use With Homestudy Course 3014-G

ENVIRONMENTAL PROTECTION AGENCY
OFFICE OF WATER SUPPLY

CENTER FOR PROFESSIONAL DEVELOPMENT AND TRAINING
U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
PUBLIC HEALTH SERVICE
Centers for Disease Control

Preface

The National Interim Primary Drinking Water Regulations published herein were promulgated on December 24, 1975, in accordance with the provisions of the Safe Drinking Water Act (Public Law 93-523). Additional Interim Primary Regulations for radioactivity in drinking water were promulgated on July 9, 1976. These regulations become effective on June 24, 1977, and become in essence the standards by which all public drinking water supplies are judged.

These regulations will replace the Public Health Service Drinking Water Standards of 1962.

The background material on which the various Maximum Contaminant Levels were based, known as the Statement of Basis and Purpose, is included herein as appendices. The Statement of Basis and Purpose also includes background material on some contaminants which were omitted from the Regulations and thus provides an explanation for those omissions.

Certain contaminants which were listed in the Public Health Service Drinking Water Standards are not included in the National Interim Primary Drinking Water Regulations because the contaminants are not directly related to the safety of drinking water but rather are related to the esthetic quality. Such contaminants, and others, will be listed in Secondary Drinking Water Regulations, to be published separately.

The National Interim Primary Drinking Water Regulations, including any amendments or revisions which may be added later, should be useful in evaluating the quality and safety of all water supplies generally.

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AUTHORITY: Secs. 1412, 1414, 1445, and 1450 of the Public Health Service Act, 88 Stat. 1660 (42 U.S.C. 300g-1, 300g-3, 300j-4, and 300j-9).

Appendix A—Background Used in Developing the National Interim Primary Drinking Water Regulations

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Subpart A–General

Section 141.1 Applicability.

This part establishes primary drinking water regulations pursuant to section 1412 of the Public Health Service Act, as amended by the Safe Drinking Water Act (Pub. L. 93-523); and related regulations applicable to public water systems.

Section 141.2 Definitions.

As used in this part, the term:

(a) "Act" means the Public Health Service Act, as amended by the Safe Drinking Water Act, Pub. L. 93-523.

(b) "Contaminant" means any physical, chemical, biological, or radiological substance or matter in water.

(c) "Maximum contaminant level" means the maximum permissible level of a contaminant in water which is delivered to the free flowing outlet of the ultimate user of a public water system, except in the case of turbidity where the maximum permissible level is measured at the point of entry to the distribution system. Contaminants added to the water under circumstances controlled by the user, except those resulting from corrosion of piping and plumbing caused by water quality, are excluded from this definition.

(d) "Person" means an individual, corporation, company, association, partnership, State, municipality, or Federal agency.

(e) "Public water system" means a system for the provision to the public of piped water for human consumption, if such system has at least fifteen service connections or regularly serves an average of at least twenty-five individuals daily at least 60 days out of the year. Such term includes (1) any collection, treatment, storage, and distribution facilities under control of the operator of such system and used primarily in connection with such system, and (2) any collection or pretreatment storage facilities not under such control which are used primarily in connection with such system. A public water system is either a "community water system" or a "non-community water system."

(i) "Community water system" means a public water system which serves at least 15 service connections used by year-round residents or regularly serves at least 25 year-round residents.

(ii) "Non-community water system" means a public water system that is not a community water system.

(f) "Sanitary survey" means an onsite review of the water source, facilities, equipment, operation and maintenance of a public water system for the purpose of evaluating the adequacy of such source, facilities, equipment, operation and maintenance for producing and distributing safe drinking water.

(g) "Standard sample" means the aliquot of finished drinking water that is examined for the presence of coliform bacteria.

(h) "State" means the agency of the State government which has jurisdiction over public water systems. During any period when a State does not have primary enforcement responsibility pursuant to Section 1413 of the Act, the term "State" means the Regional Administrator, U.S. Environmental Protection Agency.

(i) "Supplier of water" means any person who owns or operates a public water system.

(j) "Dose equivalent" means the product of the absorbed dose from ionizing radiation and such factors as account for differences in biological effectiveness due to the type of radiation and its distribution in the body as specified by the International Commission on Radiological Units and Measurements (ICRU).

(k) "Rem" means the unit of dose equivalent from ionizing radiation to the total body or any internal organ or organ system. A "millirem (mrem)" is 1/1000 of a rem.

(l) "Picocurie (pCi)" means that quantity of radioactive material producing 2.22 nuclear transformations per minute.

(m) "Gross alpha particle activity" means the total radioactivity due to alpha particle emission as inferred from measurements on a dry sample.

(n) "Man-made beta particle and photon emitters" means all radionuclides emitting beta particles and/or photons listed in Maximum Permissible Body Burdens and Maximum Permissible Concentration of Radionuclides in Air or Water for Occupational Exposure, NBS Handbook 69, except the daughter products of thorium-232, uranium-235 and uranium-238.

(o) "Gross beta particle activity" means the total radioactivity due to beta particle emission as inferred from measurements on a dry sample.

Section 141.3 Coverage.

This part shall apply to each public water system, unless the pubilic water system meets all of the following conditions:

(a) Consists only of distribution and storage facilities (and does not have any collection and treatment facilities);

(b) Obtains all of its water from, but is not owned or operated by, a public water system to which such regulations apply:

(c) Does not sell water to any person; and

(d) Is not a carrier which conveys passengers in interstate commerce.

Section 141.4 Variances and exemptions.

Variances or exemptions from certain provisions of these regulations may be granted pursuant to Sections 1415 and 1416 of the Act by the entity with primary enforcement responsibility. Provisions under Part 142, *National Interim Primary Drinking Water Regulations Implementation*—subpart E (Variances) and subpart F (Exemptions)—apply where EPA has primary enforcement responsibility.

Section 141.5 Siting requirements.

Before a person may enter into a financial commitment for or initiate construction of a new public water system or increase the capacity of an existing public water system, he shall notify the State and, to the extent practicable, avoid locating part or all of the new or expanded facility at a site which:

(a) Is subject to a significant risk from earthquakes, floods, fires or other disasters which could cause a breakdown of the public water system or a portion thereof; or

(b) Except for intake structures, is within the floodplain of a 100-year flood or is lower than any recorded high tide where appropriate records exist.

The U.S. Environmental Protection Agency will not seek to override land use decisions affecting public water systems siting which are made at the State or local government levels.

Section 141.6 Effective date.

The regulations set forth in this part shall take effect 18 months after the date of promulgation.

Subpart B–Maximum Contaminant Levels

Section 141.11 Maximum contaminant levels for inorganic chemicals.

(a) The maximum contaminant level for nitrate is applicable to both community water systems and non-community water systems. The levels for the other inorganic chemicals apply only to community water systems. Compliance with maximum contaminant levels for inorganic chemicals is calculated pursuant to § 141.23.

(b) The following are the maximum contaminant levels for inorganic chemicals other than fluoride:

Contaminant	*Level, milligrams per liter*
Arsenic	0.05
Barium	1.
Cadmium	0.010
Chromium	0.05
Lead	0.05
Mercury	0.002
Nitrate (as N)	10.
Selenium	0.01
Silver	0.05

(c) When the annual average of the maximum daily air temperatures for the location in which the community water system is situated is the following, the maximum contaminant levels for fluoride are:

Temperature Degrees Fahrenheit	Degrees Celsius	Level, milligrams per liter
53.7 and below	12.0 and below	2.4
53.8 to 58.3	12.1 to 14.6	2.2
58.4 to 63.8	14.7 to 17.6	2.0
63.9 to 70.6	17.7 to 21.4	1.8
70.7 to 79.2	21.5 to 26.2	1.6
79.3 to 90.5	26.3 to 32.5	1.4

Section 141.12 Maximum contaminant levels for organic chemicals.

The following are the maximum contaminant levels for organic chemicals. They apply only to community water systems. Compliance with maximum contaminant levels for organic chemicals is calculated pursuant to § 141.24.

	Level, milligrams per liter
(a) Chlorinated hydrocarbons:	
Endrin (1, 2, 3, 4, 10, 10-hexachloro-6,7-epoxy-1, 4,	0.0002

4a, 5, 6, 7, 8, 8a-octahydro-1, 4-endo, endo-5, 8 - dimethano naphthalene).	
Lindane (1, 2, 3, 4, 5, 6-hexachlorocyclohexane, gamma isomer).	0.004
Methoxychlor (1, 1, 1-Trichloroethane). 2, 2 - bis [p-methoxyphenyl].	0.1
Toxaphene ($C_{10}H_{10}Cl_8$-Technical chlorinated camphene, 67-69 percent chlorine).	0.005
(b) Chlorophenoxys:	
2,4 - D, (2, 4-Dichlorophenoxyacetic acid).	0.1
2, 4, 5-TP Silvex (2, 4, 5-Trichlorophenoxypropionic acid).	0.01

Section 141.13 Maximum contaminant levels for turbidity.

The maximum contaminant levels for turbidity are applicable to both community water systems and non-community water systems using surface water sources in whole or in part. The maximum contaminant levels for turbidity in drinking water, measured at a representative entry point (s) to the distribution system, are:

(a) One turbidity unit (TU), as determined by a monthly average pursuant to § 141.22, except that five or fewer turbidity units may be allowed if the supplier of water can demonstrate to the State that the higher turbidity does not do any of the following:

(1) Interfere with disinfection;

(2) Prevent maintenance of an effective disinfectant agent throughout the distribution system; or

(3) Interfere with microbiological determinations.

(b) Five turbidity units based on an average for two consecutive days pursuant to § 141.22.

Section 141.14 Maximum microbiological contaminant levels.

The maximum contaminant levels for coliform bacteria, applicable to community water systems and non-community water systems, are as follows:

(a) When the membrane filter technique pursuant to § 141.21(a) is used, the number of coliform bacteria shall not exceed any of the following:

(1) One per 100 milliliters as the arithmetic mean of all samples examined per month pursuant to § 141.21 (b) or (c);

(2) Four per 100 milliliters in more than one sample when less than 20 are examined per month; or

(3) Four per 100 milliliters in more than five percent of the samples when 20 or more are examined per month.

(b) (1) When the fermentation tube method and 10 milliliter standard portions pursuant to § 141.21(a) are used, coliform bacteria shall not be present in any of the following:

(i) more than 10 percent of the portions in any month pursuant to § 141.21 (b) or (c);

(ii) three or more portions in more than one sample when less than 20

samples are examined per month; or

(iii) three or more portions in more than five percent of the samples when 20 or more samples are examined per month.

(2) When the fermentation tube method and 100 milliliter standard portions pursuant to § 141.21(a) are used, coliform bacteria shall not be present in any of the following:

(i) more than 60 percent of the portions in any month pursuant to § 141.21 (b) or (c);

(ii) five portions in more than one sample when less than five samples are examined per month; or

(iii) five portions in more than 20 percent of the samples when five or more samples are examined per month.

(c) For community or non-community systems that are required to sample at a rate of less than 4 per month, compliance with paragraphs (a), (b) (1), or (b) (2) of this section shall be based upon sampling during a 3 month period, except that, at the discretion of the State, compliance may be based upon sampling during a one-month period.

Section 141.15 Maximum contaminant levels for radium-226, radium-228, and gross alpha particle radioactivity in community water systems.

The following are the maximum contaminant levels for radium-226, radium-228, and gross alpha particle radioactivity:

(a) Combined radium-226 and radium-228—5 pCi/1.

(b) Gross alpha particle activity (including radium-226 but excluding radon and uranium)—15 pCi/1.

Section 141.16 Maximum contaminant levels for beta particle and photon radioactivity from man-made radionuclides in community water systems

(a) The average annual concentration of beta particle and photon radioactivity from man-made radionuclides in drinking water shall not produce an annual dose equivalent to the total body or any internal organ greater than 4 millirem/year.

(b) Except for the radionuclides listed in Table A, the concentration of man-made radionuclides causing 4 mrem total body or organ dose equivalents shall be calculated on the basis of a 2 liter per day drinking water intake using the 168 hour data listed in "*Maximum Permissible Body Burdens and Maximum Permissible Concentration of Radionuclides in Air or Water for Occupational Exposure,*" NBS Handbook 69 as amended August 1963, U.S. Department of Commerce. If two or more radionuclides are present, the sum of their annual dose equivalent to the total body or to any organ shall not exceed 4 millirem/year.

TABLE A.—*Average annual concentrations assumed to produce a total body or organ dose of 4 mrem/yr*

Radionuclide	Critical organ	pCi per liter
Tritium	Total body	20,000
Strontium-90	Bone marrow	8

Subpart C–Monitoring and Analytical Requirements

Section 141.21 Microbiological contaminant sampling and analytical requirements.

(a) Suppliers of water for community water systems and non-community water systems shall analyze for coliform bacteria for the purpose of determining compliance with § 141.14. Analyses shall be conducted in accordance with the analytical recommendations set forth in "Standard Methods for the Examination of Water and Wastewater," American Public Health Association, 13th Edition, pp. 662-688, except that a standard sample size shall be employed. The standard sample used in the membrane filter procedure shall be 100 milliliters. The standard sample used in the 5 tube most probable number (MPN) procedure (fermentation tube method) shall be 5 times the standard portion. The standard portion is either 10 milliliters or 100 milliliters as described in § 141.14 (b) and (c). The samples shall be taken at points which are representative of the conditions within the distribution system.

(b) The supplier of water for a community water system shall take coliform density samples at regular time intervals, and in number proportionate to the population served by the system. In no event shall the frequency be less than as set forth below:

Population served:	*Minimum number of samples per month*
25 to 1,000	1
1,001 to 2,500	2
2,501 to 3,300	3
3,301 to 4,100	4
4,101 to 4,900	5
4,901 to 5,800	6
5,801 to 6,700	7
6,701 to 7,600	8
7,601 to 8,500	9
8,501 to 9,400	10
9,401 to 10,300	11
10,301 to 11,100	12
11,101 to 12,000	13
12,001 to 12,900	14
12,901 to 13,700	15
13,701 to 14,600	16
14,601 to 15,500	17
15,501 to 16,300	18
16,301 to 17,200	19
17,201 to 18,100	20
18,101 to 18,900	21
18,901 to 19,800	22
19,801 to 20,700	23

Population served:	*Minimum number of samples per month*
90,001 to 96,000	95
96,001 to 111,000	100
111,001 to 130,000	110
130,001 to 160,000	120
160,001 to 190,000	130
190,001 to 220,000	140
220,001 to 250,000	150
250,001 to 290,000	160
290,000 to 320,000	170
320,001 to 360,000	180
360,001 to 410,000	190
410,001 to 450,000	200
450,001 to 500,000	210
500,001 to 550,000	220
550,001 to 600,000	230
600,001 to 660,000	240
660,001 to 720,000	250
720,001 to 780,000	260
780,001 to 840,000	270
840,001 to 910,000	280
910,001 to 970,000	290
970,001 to 1,050,000	300
1,050,001 to 1,140,000	310

20,701 to 21,500	24	1,140,001 to 1,230,000	320
21,501 to 22,300	25	1,230,001 to 1,320,000	330
22,301 to 23,200	26	1,320,001 to 1,420,000	340
23,201 to 24,000	27	1,420,001 to 1,520,000	350
24,001 to 24,900	28	1,520,001 to 1,630,000	360
24,901 to 25,000	29	1,630,001 to 1,730,000	370
25,001 to 28,000	30	1,730,001 to 1,850,000	380
28,001 to 33,000	35	1,850,001 to 1,970,000	390
33,001 to 37,000	40	1,970,001 to 2,060,000	400
37,001 to 41,000	45	2,060,001 to 2,270,000	410
41,001 to 46,000	50	2,270,001 to 2,510,000	420
46,001 to 50,000	55	2,510,001 to 2,750,000	430
50,001 to 54,000	60	2,750,001 to 3,020,000	440
54,001 to 59,000	65	3,020,001 to 3,320,000	450
59,001 to 64,000	70	3,320,001 to 3,620,000	460
64,001 to 70,000	75	3,620,001 to 3,960,000	470
70,001 to 76,000	80	3,960,001 to 4,310,000	480
76,001 to 83,000	85	4,310,001 to 4,690,000	490
83,001 to 90,000	90	4,690,001 or more	500

Based on a history of no coliform bacterial contamination and on a sanitary survey by the State showing the water system to be supplied solely by a protected ground water source and free of sanitary defects, a community water system serving 25 to 1,000 persons, with written permission from the State, may reduce this sampling frequency except that in no case shall it be reduced to less than one per quarter.

(c) The supplier of water for a non-community water system shall sample for coliform bacteria in each calendar quarter during which the system provides water to the public. Such sampling shall begin within two years after the effective date of this part. If the State, on the basis of a sanitary survey, determines that some other frequency is more appropriate, that frequency shall be the frequency required under these regulations. Such frequency shall be confirmed or changed on the basis of subsequent surveys.

(d) (1) When the coliform bacteria in a single sample exceed four per 100 milliliters (§ 141.14(a)), at least two consecutive daily check samples shall be collected and examined from the same sampling point. Additional check samples shall be collected daily, or at a frequency established by the State, until the results obtained from at least two consecutive check samples show less than one coliform bacterium per 100 milliliters.

(2) When coliform bacteria occur in three or more 10 ml portions of a single sample (§ 141.14(b) (1)), at least two consecutive daily check samples shall be collected and examined from the same sampling point. Additional check samples shall be collected daily, or at a frequency established by the State, until the results obtained from at least two consecutive check samples show no positive tubes.

(3) When coliform bacteria occur in all five of the 100 ml portions of a single sample (§ 141.14(b) (2)), at least two daily check samples shall be collected and examined from the same sampling point. Additional check

samples shall be collected daily, or at a frequency established by the State, until the results obtained from at least two consecutive check samples show no positive tubes.

(4) The location at which the check samples were taken pursuant to paragraphs (d) (1), (2), or (3) of this section shall not be eliminated from future sampling without approval of the State. The results from all coliform bacterial analyses performed pursuant to this subpart, except those obtained from check samples and special purpose samples, shall be used to determine compliance with the maximum contaminant level for coliform bacteria as established in § 141.14. Check samples shall not be included in calculating the total number of samples taken each month to determine compliance with § 141.21 (b) or (c).

(e) When the presence of coliform bacteria in water taken from a particular sampling point has been confirmed by any check samples examined as directed in paragraphs (d) (1), (2), or (3) of this section, the supplier of water shall report to the State within 48 hours.

(f) When a maximum contaminant level set forth in paragraphs (a), (b) or (c) of § 141.14 is exceeded, the supplier of water shall report to the State and notify the public as prescribed in § 141.31 and § 141.32.

(g) Special purpose samples, such as those taken to determine whether disinfection practices following pipe placement, replacement, or repair have been sufficient, shall not be used to determine compliance with § 141.14 or § 141.21 (b) or (c).

(h) A supplier of water of a community water system or a non-community water system may, with the approval of the State and based upon a sanitary survey, substitute the use of chlorine residual monitoring for not more than 75 percent of the samples required to be taken by paragraph (b) of this section, *Provided*, That the supplier of water takes chlorine residual samples at points which are representative of the conditions within the distribution system at the frequency of at least four for each substituted microbiological sample. There shall be at least daily determinations of chlorine residual. When the supplier of water exercises the option provided in this paragraph (h) of this section, he shall maintain no less than 0.2 mg/l free chlorine throughout the public water distribution system. When a particular sampling point has been shown to have a free chlorine residual less than 0.2 mg/l, the water at that location shall be retested as soon as practicable and in any event within one hour. If the original analysis is confirmed, this fact shall be reported to the State within 48 hours. Also, if the analysis is confirmed, a sample for coliform bacterial analysis must be collected from that sampling point as soon as practicable and preferably within one hour, and the results of such analysis reported to the State within 48 hours after the results are known to the supplier of water. Analyses for residual chlorine shall be made in accordance with "Standard Methods for the Examination of Water and Wastewater," 13th Ed., pp. 129-132. Compliance with the

maximum contaminant levels for coliform bacteria shall be determined on the monthly mean or quarterly mean basis specified in § 141.14, including those samples taken as a result of failure to maintain the required chlorine residual level. The State may withdraw its approval of the use of chlorine residual substitution at any time.

Section 141.22 Turbidity sampling and analytical requirements.

(a) Samples shall be taken by suppliers of water for both community water systems and non-community water systems at a representative entry point(s) to the water distribution system at least once per day, for the purpose of making turbidity measurements to determine compliance with § 141.13. The measurement shall be made by the Nephelometric Method in accordance with the recommendations set forth in "Standard Methods for the Examination of Water and Wastewater," American Public Health Association, 13th Edition, pp. 350-353, or "Methods for Chemical Analysis of Water and Wastes," pp. 295-298, Environmental Protection Agency, Office of Technology Transfer, Washington, D.C. 20460, 1974.

(b) If the result of a turbidity analysis indicates that the maximum allowable limit has been exceeded, the sampling and measurement shall be confirmed by resampling as soon as practicable and preferably within one hour. If the repeat sample confirms that the maximum allowable limit has been exceeded, the supplier of water shall report to the State within 48 hours. The repeat sample shall be the sample used for the purpose of calculating the monthly average. If the monthly average of the daily samples exceeds the maximum allowable limit, or if the average of two samples taken on consecutive days exceeds 5 TU, the supplier of water shall report to the State and notify the public as directed in § 141.31 and § 141.32.

(c) Sampling for non-community water systems shall begin within two years after the effective date of this part.

(d) The requirements of this § 141.22 shall apply only to public water systems which use water obtained in whole or in part from surface sources.

Section 141.23 Inorganic chemical sampling and analytical requirements.

(a) Analyses for the purpose of determining compliance with § 141.11 are required as follows:

(1) Analyses for all community water systems utilizing surface water sources shall be completed within one year following the effective date of this part. These analyses shall be repeated at yearly intervals.

(2) Analyses for all community water systems utilizing only ground water sources shall be completed within two years following the effective date of this part. These analyses shall be repeated at three-year intervals.

(3) For non-community water systems, whether supplied by surface or ground water sources, analyses for nitrate shall be completed within two

years following the effective date of this part. These analyses shall be repeated at intervals determined by the State.

(b) If the result of an analysis made pursuant to paragraph (a) indicates that the level of any contaminant listed in § 141.11 exceeds the maximum contaminant level, the supplier of water shall report to the State within 7 days and initiate three additional analyses at the same sampling point within one month.

(c) When the average of four analyses made pursuant to paragraph (b) of this section, rounded to the same number of significant figures as the maximum contaminant level for the substance in question, exceeds the maximum contaminant level, the supplier of water shall notify the State pursuant to § 141.31 and give notice to the public pursuant to § 141.32. Monitoring after public notification shall be at a frequency designated by the State and shall continue until the maximum contaminant level has not been exceeded in two successive samples or until a monitoring schedule as a condition to a variance, exemption or enforcement action shall become effective.

(d) The provisions of paragraphs (b) and (c) of this section notwithstanding, compliance with the maximum contaminant level for nitrate shall be determined on the basis of the mean of two analyses. When a level exceeding the maximum contaminant level for nitrate is found, a second analysis shall be initiated within 24 hours, and if the mean of the two analyses exceeds the maximum contaminant level, the supplier of water shall report his findings to the State pursuant to § 141.31 and shall notify the public pursuant to § 141.32.

(e) For the initial analyses required by paragraph (a) (1), (2) or (3) of this section, data for surface waters acquired within one year prior to the effective date and data for ground waters acquired within 3 years prior to the effective date of this part may be substituted at the discretion of the State.

(f) Analyses conducted to determine compliance with § 141.11 shall be made in accordance with the following methods:

(1) Arsenic—Atomic Absorption Method, "Methods for Chemical Analysis of Water and Wastes," pp. 95-96, Environmental Protection Agency, Office of Technology Transfer, Washington, D.C. 20460, 1974.

(2) Barium—Atomic Absorption Method, "Standard Methods for the Examination of Water and Wastewater," 13th Edition, pp. 210-215, or "Methods for Chemical Analysis of Water and Wastes," pp. 97-98, Environmental Protection Agency, Office of Technology Transfer, Washington, D.C. 20460, 1974.

(3) Cadmium—Atomic Absorption Method, "Standard Methods for the Examination of Water and Wastewater," 13th Edition, p.p. 210-215, or "Methods for Chemical Analysis of Water and Wastes," pp. 101-103, En-

vironmental Protection Agency, Office of Technology Transfer, Washington, D.C. 20460, 1974.

(4) Chromium—Atomic Absorption Method, "Standard Methods for the Examination of Water and Wastewater," 13th Edition, pp. 210-215, or "Methods for Chemical Analysis of Water and Wastes," pp. 105-106, Environmental Protection Agency, Office of Technology Transfer, Washington, D.C. 20460, 1974.

(5) Lead—Atomic Absorption Method, "Standard Methods for the Examination of Water and Wastewater," 13th Edition, pp. 210-215, or "Methods for Chemical Analysis of Water and Wastes," pp. 112-113, Environmental protection Agency, Office of Technology Transfer, Washington, D.C. 20460, 1974.

(6) Mercury—Flameless Atomic Absorption Method, "Methods for Chemical Analysis of Water and Wastes," pp. 118-126, Environmental Protection Agency, Office of Technology Transfer, Washington, D.C. 20460, 1974.

(7) Nitrate—Brucine Colorimetric Method, "Standard Methods for the Examination of Water and Wastewater," 13th Edition, pp. 461-464, or Cadmium Reduction Method, "Methods for Chemical Analysis of Water and Wastes," pp. 201-206, Environmental Protection Agency, Office of Technology Transfer, Washington, D.C. 20460, 1974.

(8) Selenium—Atomic Absorption Method, "Methods for Chemical Analysis of Water and Wastes," p. 145, Environmental Protection Agency, Office of Technology Transfer, Washington, D.C. 20460, 1974.

(9) Silver—Atomic Absorption Method, "Standard Methods for the Examination of Water and Wastewater", 13th Edition, pp. 210-215, or "Methods for Chemical Analysis of Water and Wastes", p. 146, Environmental Protection Agency, Office of Technology Transfer, Washington, D.C. 20460, 1974.

(10) Fluoride—Electrode Method, "Standard Methods for the Examination of Water and Wastewater", 13th Edition, pp. 172-174, or "Methods for Chemical Analysis of Water and Wastes," pp. 65-67, Environmental Protection Agency, Office of Technology Transfer, Washington, D.C. 20460, 1974, or Colorimetric Method with Preliminary Distillation, "Standard Methods for the Examination of Water and Wastewater," 13th Edition, pp. 171-172 and 174-176, or "Methods for Chemical Analysis of Water and Wastes," pp. 59-60, Environmental Protection Agency, Office of Technology Transfer, Washington, D.C. 20460, 1974.

Section 141.24 Organic chemical sampling and analytical requirements.

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

(1) For all community water systems utilizing surface water sources, an-

alyses shall be completed within one year following the effective date of this part. Samples analyzed shall be collected during the period of the year designated by the State as the period when contamination by pesticides is most likely to occur. These analyses shall be repeated at intervals specified by the State but in no event less frequently than at three year intervals.

(2) For community water systems utilizing only ground water sources, analyses shall be completed by those systems specified by the State.

(b) If the result of an analysis made pursuant to paragraph (a) of this section indicates that the level of any contaminant listed in § 141.12 exceeds the maximum contaminant level, the supplier of water shall report to the State within 7 days and initiate three additional analyses within one month.

(c) When the average of four analyses made pursuant to paragraph (b) of this section, rounded to the same number of significant figures as the maximum contaminant level for the substance in question, exceeds the maximum contaminant level, the supplier of water shall report to the State pursuant to § 141.31 and give notice to the public pursuant to § 141.32. Monitoring after public notification shall be at a frequency designated by the State and shall continue until the maximum contaminant level has not been exceeded in two successive samples or until a monitoring schedule as a condition to a variance, exemption or enforcement action shall become effective.

(d) For the initial analysis required by paragraph (a) (1) and (2) of this section, data for surface water acquired within one year prior to the effective date of this part and data for ground water acquired within three years prior to the effective date of this part may be substituted at the discretion of the State.

(e) Analyses made to determine compliance with § 141.12(a) shall be made in accordance with "Method for Organochlorine Pesticides in Industrial Effluents," MDQARL, Environmental Protection Agency, Cincinnati, Ohio, November 28, 1973.

(f) Analyses made to determine compliance with § 141.12(b) shall be conducted in accordance with "Methods for Chlorinated Phenoxy Acid Herbicides in Industrial Effluents," MDQARL, Environmental Protection Agency, Cincinnati, Ohio, November 28, 1973.

Section 141.25 Analytical Methods for Radioactivity.

(a) The methods specified in *Interim Radiochemical Methodology for Drinking Water*, Environmental Monitoring and Support Laboratory, EPA-600/4-75-008, USEPA, Cincinnati, Ohio 45268, or those listed below, are to be used to determine compliance with § § 141.15 and 141.16 (radioactivity) except in cases where alternative methods have been approved in accordance with § 141.27.

(1) Gross Alpha and Beta—Method 302 "Gross Alpha and Beta Radioactivity in Water" *Standard Methods for the Examination of Water and Wastewater*, 13th Edition, American Public Health Association, New York,

N.Y., 1971.

(2) Total Radium—Method 304 "Radium in Water by Precipitation" Ibid.

(3) Radium-226—Method 305 "Radium-226 by Radon in Water" Ibid.

(4) Strontium-89, 90—Method 303 "Total Strontium and Strontium-90 in Water" Ibid.

(5) Tritium—Method 306 "Tritium in Water" Ibid.

(6) Cesium-134—ASTM D-2459 "Gamma Spectrometry in Water," *1975 Annual Book of ASTM Standards, Water and Atmospheric Analysis*, Part 31, American Society for Testing and Materials, Philadelphia, PA. (1975).

(7) Uranium—ASTM D-2907 "Microquantities of Uranium in Water by Fluorometry," Ibid.

(b) When the identification and measurement of radionuclides other than those listed in paragraph (a) is required, the following references are to be used, except in cases where alternative methods have been approved in accordance with § 141.27.

(1) *Procedures for Radiochemical Analysis of Nuclear Reactor Aqueous Solutions*, H. L. Krieger and S. Gold, EPA-R4-73-014. USEPA, Cincinnati, Ohio, May 1973.

(2) *HASL Procedure Manual*, Edited by John H. Harley. HASL 300, ERDA Health and Safety Laboratory, New York, N.Y., 1973.

(c) For the purpose of monitoring radioactivity concentrations in drinking water, the required sensitivity of the radioanalysis is defined in terms of a detection limit. The detection limit shall be that concentration which can be counted with a precision of plus or minus 100 percent at the 95 percent confidence level (1.96σ where σ is the standard deviation of the net counting rate of the sample).

(1) To determine compliance with § 141.15(a) the detection limit shall not exceed 1 pCi/l. To determine compliance with § 141.15(b) the detection limits shall not exceed 3 pCi/l.

(2) To determine compliance with § 141.16 the detection limits shall not exceed the concentrations listed in Table B.

TABLE B.—DETECTION LIMITS FOR MAN-MADE BETA PARTICLE AND PHOTON EMITTERS

Radionuclide	*Detection limit*
Tritium	1,000 pCi/l.
Strontium-89	10 pCi/l.
Strontium-90	2 pCi/l.
Iodine-131	1 pCi/l.
Cesium-134	10 pCi/l.
Gross beta	4 pCi/l.
Other radionuclides	1/10 of the applicable limit.

(d) To judge compliance with the maximum contaminant levels listed in sections 141.15 and 141.16, averages of data shall be used and shall be

rounded to the same number of significant figures as the maximum contaminant level for the substance in question.

Section 141.26 Monitoring Frequency for Radioactivity in Community water systems.

(a) Monitoring requirements for gross alpha particle activity, radium-226 and radium-228.

(1) Initial sampling to determine compliance with § 141.15 shall begin within two years of the effective date of these regulations and the analysis shall be completed within three years of the effective date of these regulations. Compliance shall be based on the analysis of an annual composite of four consecutive quarterly samples or the average of the analyses of four samples obtained at quarterly intervals.

(i) A gross alpha particle activity measurement may be substituted for the required radium-226 and radium-228 analysis *Provided,* That the measured gross alpha particle activity does not exceed 5 pCi/l at a confidence level of 95 percent (1.65σ where σ is the standard deviation of the net counting rate of the sample). In localities where radium-228 may be present in drinking water, it is recommended that the State require radium-226 and/or radium-228 analyses when the gross alpha particle activity exceeds 2 pCi/l.

(ii) When the gross alpha particle activity exceeds 5 pCi/l, the same or an equivalent sample shall be analyzed for radium-226. If the concentration of radium-226 exceeds 3 pCi/l the same or an equivalent sample shall be analyzed for radium-228.

(2) For the initial analysis required by paragraph (a) (1), data acquired within one year prior to the effective date of this part may be substituted at the discretion of the State.

(3) Suppliers of water shall monitor at least once every four years following the procedure required by paragraph (a) (1). At the discretion of the State, when an annual record taken in conformance with paragraph (a) (1) has established that the average annual concentration is less than half the maximum contaminant levels established by § 141.15, analysis of a single sample may be substituted for the quarterly sampling procedure required by paragraph (a) (1).

(i) More frequent monitoring shall be conducted when ordered by the State in the vicinity of mining or other operations which may contribute alpha particle radioactivity to either surface or ground water sources of drinking water.

(ii) A supplier of water shall monitor in conformance with paragraph (a) (1) within one year of the introduction of a new water source for a community water system. More frequent monitoring shall be conducted when ordered by the State in the event of possible contamination or when changes in the distribution system or treatment processing occur which may increase the concentration of radioactivity in finished water.

(iii) A community water system using two or more sources having different concentrations of radioactivity shall monitor source water, in addition to water from a free-flowing tap, when ordered by the State.

(iv) Monitoring for compliance with § 141.15 after the initial period need not include radium-228 *except when* required by the State, *Provided*, That the average annual concentration of radium-228 has been assayed at least once using the quarterly sampling procedure required by paragraph (a) (1).

(v) Suppliers of water shall conduct annual monitoring of any community water system in which the radium-226 concentration exceeds 3 pCi/l, when ordered by the State.

(4) If the average annual maximum contaminant level for gross alpha particle activity or total radium as set forth in § 141.15 is exceeded, the supplier of a community water system shall give notice to the State pursuant to § 141.31 and notify the public as required by § 141.32. Monitoring at quarterly intervals shall be continued until the annual average concentration no longer exceeds the maximum contaminant level or until a monitoring schedule as a condition to a variance, exemption or enforcement action shall become effective.

(b) Monitoring requirements for man-made radioactivity in community water systems.

(1) Within two years of the effective date of this part, systems using surface water sources and serving more than 100,000 persons and such other community water systems as are designated by the State shall be monitored for compliance with § 141.16 by analysis of a composite of four consecutive quarterly samples or analysis of four quarterly samples. Compliance with § 141.16 may be assumed without further analysis if the average annual concentration of gross beta particle activity is less than 50 pCi/l and if the average annual concentrations of tritium and strontium-90 are less than those listed in Table A, *Provided*, That if both radionuclides are present the sum of their annual dose equivalents to bone marrow shall not exceed 4 millirem/year.

(i) If the gross beta particle activity exceeds 50 pCi/l, an analysis of the sample must be performed to identify the major radioactive constituents present and the appropriate organ and total body doses shall be calculated to determine compliance with § 141.16.

(ii) Suppliers of water shall conduct additional monitoring, as ordered by the State, to determine the concentration of man-made radioactivity in principal watersheds designated by the State.

(iii) At the discretion of the State, supplies of water utilizing only ground waters may be required to monitor for man-made radioactivity.

(2) For the initial analysis required by paragraph (b) (1) data acquired within one year prior to the effective date of this part may be substituted at the discretion of the State.

(3) After the initial analysis required by paragraph (b) (1) suppliers of water shall monitor at least every four years following the procedure given in paragraph (b) (1).

(4) Within two years of the effective date of these regulations the supplier of any community water system designated by the State as utilizing waters contaminated by effluents from nuclear facilities shall initiate quarterly monitoring for gross beta particle and iodine-131 radioactivity and annual monitoring for strontium-90 and tritium.

(i) Quarterly monitoring for gross beta particle activity shall be based on the analysis of monthly samples or the analysis of a composite of three monthly samples. The former is recommended. If the gross beta particle activity in a sample exceeds 15 pCi/l, the same or an equivalent sample shall be analyzed for strontium-89 and cesium-134. If the gross beta particle activity exceeds 50 pCi/l, an analysis of the sample must be performed to identify the major radioactive constituents present and the appropriate organ and total body doses shall be calculated to determine compliance with § 141.16.

(ii) For iodine-131, a composite of five consecutive daily samples shall be analyzed once each quarter. As ordered by the State, more frequent monitoring shall be conducted when iodine-131 is identified in the finished water.

(iii) Annual monitoring for strontium-90 and tritium shall be conducted by means of the analysis of a composite of four consecutive quarterly samples or analysis of four quarterly samples. The latter procedure is recommended.

(iv) The State may allow the substitution of environmental surveillance data taken in conjunction with a nuclear facility for direct monitoring of man-made radioactivity by the supplier of water where the State determines such data is applicable to a particular community water system.

(5) If the average annual maximum contaminant level for man-made radioactivity set forth in § 141.16 is exceeded, the operator of a community water system shall give notice to the State pursuant to § 141.31 and to the public as required by § 141.32. Monitoring at monthly intervals shall be continued until the concentration no longer exceeds the maximum contaminant level or until a monitoring schedule as a condition to a variance, exemption or enforcement action shall become effective.

Section 141.27 Alternative analytical techniques.

With the written permission of the State, concurred in by the Administrator of the U.S. Environmental Protection Agency, an alternative analytical technique may be employed. An alternative technique shall be acceptable only if it is substantially equivalent to the prescribed test in both precision and accuracy as it relates to the determination of compliance with any maximum contaminant level. The use of the alternative analytical technique shall not decrease the frequency of monitoring required by this part.

Section 141.28 Approved laboratories.

For the purpose of determining compliance with § 141.21 through § 141.27, samples may be considered only if they have been analyzed by a laboratory approved by the State except that measurements for turbidity and free chlorine residual may be performed by any person acceptable to the State.

Section 141.29 Monitoring of consecutive public water systems.

When a public water system supplies water to one or more other public water systems, the State may modify the monitoring requirements imposed by this part to the extent that the interconnection of the systems justifies treating them as a single system for monitoring purposes. Any modified monitoring shall be conducted pursuant to a schedule specified by the State and concurred in by the Administrator of the U.S. Environmental Protection Agency.

Subpart D–Reporting, Public Notification, and Record Keeping

Section 141.31 Reporting requirements.

(a) Except where a shorter reporting period is specified in this part, the supplier of water shall report to the State within 40 days following a test, measurement or analysis required to be made by this part, the results of that test, measurement or analysis.

(b) The supplier of water shall report to the State within 48 hours the failure to comply with any primary drinking water regulation (including failure to comply with monitoring requirements) set forth in this part.

(c) The supplier of water is not required to report analytical results to the State in cases where a State laboratory performs the analysis and reports the results to the State office which would normally receive such notification from the supplier.

Section 141.32 Public notification.

(a) If a community water system fails to comply with an applicable maximum contaminant level established in Subpart B, fails to comply with an applicable testing procedure established in Subpart C of this part, is granted a variance or an exemption from an applicable maximum contaminant level, fails to comply with the requirements of any schedule prescribed pursuant to a variance or exemption, or fails to perform any monitoring required pursuant to Section 1445 (a) of the Act, the supplier of water shall notify persons served by the system of the failure or grant by inclusion of a notice in the first set of water bills of the system issued after the failure or grant and in any event by written notice within three months. Such notice shall be repeated at least once every three months so long as the system's failure continues or the variance or exemption remains in effect. If the system issues water bills less frequently than quarterly, or does not issue water bills, the notice shall be made by or supplemented by another form of direct mail.

(b) If a community water system has failed to comply with an applicable maximum contaminant level, the supplier of water shall notify the public of such failure, in addition to the notification required by paragraph (a) of this section, as follows:

(1) By publication on not less than three consecutive days in a newspaper or newspapers of general circulation in the area served by the system. Such notice shall be completed within fourteen days after the supplier of water learns of the failure.

(2) By furnishing a copy of the notice to the radio and television stations serving the area served by the system. Such notice shall be furnished within seven days after the supplier of water learns of the failure.

(c) If the area served by a community water system is not served by a daily newspaper of general circulation, notification by newspaper required

by paragraph (b) of this section shall instead be given by publication on three consecutive weeks in a weekly newspaper of general circulation serving the area. If no weekly or daily newspaper of general circulation serves the area, notice shall be given by posting the notice in post offices within the area served by the system.

(d) If a non-community water system fails to comply with an applicable maximum contaminant level established in Subpart B of this part, fails to comply with an applicable testing procedure established in Subpart C of this part, is granted a variance or an exemption from an applicable maximum contaminant level, fails to comply with the requirement of any schedule prescribed pursuant to a variance or exemption or fails to perform any monitoring required pursuant to Section 1445(a) of the Act, the supplier of water shall give notice of such failure or grant to the persons served by the system. The form and manner of such notice shall be prescribed by the State, and shall insure that the public using the system is adequately informed of the failure or grant.

(e) Notices given pursuant to this section shall be written in a manner reasonably designed to inform fully the users of the system. The notice shall be conspicuous and shall not use unduly technical language, unduly small print or other methods which would frustrate the purpose of the notice. The notice shall disclose all material facts regarding the subject including the nature of the problem and, when appropriate, a clear statement that a primary drinking water regulation has been violated and any preventive measures that should be taken by the public. Where appropriate, or where designated by the State, bilingual notice shall be given. Notices may include a balanced explanation of the significance or seriousness to the public health of the subject of the notice, a fair explanation of steps taken by the system to correct any problem and the results of any additional sampling.

(f) Notice to the public required by this section may be given by the State on behalf of the supplier of water.

(g) In any instance in which notification by mail is required by paragraph (a) of this section but notification by newspaper or to radio or television stations is not required by paragraph (b) of this section, the State may order the supplier of water to provide notification by newspaper and to radio and television stations when circumstances make more immediate or broader notice appropriate to protect the public health.

Section 141.33 Record Maintenance.

Any owner or operator of a public water system subject to the provisions of this part shall retain on its premises or at a conventient location near its premises the following records:

(a) Records of bacteriological analyses made pursuant to this part shall be kept for not less than 5 years. Records of chemical analyses made pursuant to this part shall be kept for not less than 10 years. Actual laboratory

reports may be kept, or data may be transferred to tabular summaries, provided that the following information is included:

(1) The date, place and time of sampling, and the name of the person who collected the sample;

(2) Identification of the sample as to whether it was a routine distribution system sample, check sample, raw or process water sample or other special purpose sample;

(3) Date of analysis;

(4) Laboratory and person responsible for performing analysis;

(5) The analytical technique/method used; and

(6) The results of the analysis.

(b) Records of action taken by the system to correct violations of primary drinking water regulations shall be kept for a period not less than 3 years after the last action taken with respect to the particular violation involved.

(c) Copies of any written reports, summaries or communications relating to sanitary surveys of the system conducted by the system itself, by a private consultant, or by any local, State or Federal agency, shall be kept for a period not less than 10 years after completion of the sanitary survey involved.

(d) Records concerning a variance or exemption granted to the system shall be kept for a period ending not less than 5 years following the expiration of such variance or exemption.

Appendix A
Background Used In Developing The National Interim Primary Drinking Water Regulations

The National Interim Primary Drinking Water Regulations have been predicated on the best and latest information available at the time of their promulgation. The concepts and rationale included in this Appendix were derstanding, judgment, and discretion.
used in arriving at specific limits and should enable those whose responsibility it is to interpret, apply, or enforce the Regulations to do so with un-

A. SOURCE AND FACILITIES
B. MICROBIOLOGICAL QUALITY
C. CHEMICAL QUALITY

A — SOURCE AND FACILITIES

Mounting pollution problems indicate the need for increased attention to the quality of source waters. Abatement and control of pollution of sources will significantly aid in producing drinking water that will be in full compliance with the provisions of these Standards and will be esthetically acceptable to the consumer, but they will never eliminate the need for well designed water treatment facilities operated by competent personnel.

Production of water that poses no threat to the consumer's health depends on continuous protection. Because of human frailties associated with protection, priority should be given to selection of the purest source. Polluted sources should not be used unless other sources are economically unavailable, and then only when personnel, equipment, and operating procedures can be depended on to purify and otherwise continuously protect the drinkink water supply.

Although ground waters obtained from aquifers beneath impervious strata, and not connected with fragmented or cavernous rock, have been considered sufficiently protected from bacterial contamination to preclude need for disinfection, this is frequently not true as ground waters are becoming polluted with increasing frequency, and the resulting hazards require special surveillance. An illustration of such pollution is the presence of pollutants originating either from sewage or industrial effluents.

Surface waters are subjected to increasing pollution and should never be used without being effectively disinfected. Because of the increasing hazards of pollution, the use of surface waters without coagulation and filtration must be accompanied by adequate past records and intensive surveillance of the quality of the raw water and the disinfected supply in order to assure constant protection. This surveillance should include a sanitary survey of the source and water handling, as well as biological examination of the supply.

The degree of treatment should be determined by the health hazards involved and the quality of the raw water. When in use, the source should be under continuous surveillance to assure adequacy of treatment in meeting the hazards of changing pollution conditions. Continuous, effective disinfection shall be considered the minimum treatment for any water supply except for ground waters in which total coliforms can be shown to be continually absent from the raw water. During times of unavoidable and excessive pollution of a source already in use, it may become necessary to provide extraordinary treatment (e.g., exceptionally strong disinfection[1], improved coagulation, and/or special operation). If the pollution cannot be removed satisfactorily by treatment, use of the source should be discontinued until the pollution has been reduced or eliminated.

The adequacy of protection by treatment should be judged, in part, on a record of the quality of water produced by the treatment plant and the relation of this quality to the requirements of these Regulations. Evaluation of adequacy of protection by treatment should also include frequent inspection of treatment works and their operation. Conscientious operation by well-trained, skillful, and competent operators is an essential part of protection by treatment. Operator competency is encouraged by a formal program leading to operator certification or licensing.

Delivery of a safe water supply depends on adequate protection by natural means or by treatment, and protection of the water in the distribution system. Minimum protection should include programs that result in the *provision* of sufficient and safe materials and equipment to treat and distribute the water; *disinfection* of water mains, storage facilities, and other equipment after each installation, repair, or other modification that may have subjected them to possible contamination; *prevention* of health hazards, such as cross-connections or loss of pressure because of overdraft in excess of the system's capacity; and *routine analysis* of water samples and frequent survey of the water system to evaluate the adequacy of protection. The fact that the minimum number of samples are taken and analyzed and found to comply with specific quality requirements of these Standards, *is not sufficient evidence that protection has been adequate.* The protection procedures and physical facilities must be reviewed along with the results of water quality analyses to evaluate the adequacy of the supply's protection. Knowledge of physical defects or of the existence of other health hazards in the water supply system is evidence of a deficiency in protection of the water supply. Even though water quality analyses have indicated that the quality requirements have been met, the deficiencies must be corrected before the supply can be considered safe.

[1] See reference to relationship of chlorine residual and contact time required to kill viruses in section on Microbiological Quality.

B — MICROBIOLOGICAL QUALITY

Coliform Group

Coliform bacteria traditionally have been the bacteriological tool used to measure the occurrence and intensity of fecal contamination in stream-pollution investigations for nearly 70 years. During this time, a mass of data has accumulated to permit a full evalution of the sensitivity and specificity of this bacterial pollution indicator.

As defined in *Standard Methods for the Examination of Water and Wastewater* (1), "the coliform group includes all of the aerobic and facultative anaerobic, Gram-negative, non-spore-forming rod-shaped bacteria which ferment lactose with gas formation within 48 hours at 35° C." From this definition, it becomes immediately apparent that this bacterial grouping is somewhat artificial in that it embodies a heterogeneous collection of bacterial species having only a few broad characteristics in common. Yet, for practical applications to stream pollution studies, this grouping of selected bacterial species, which we shall term the "total coliform group," has proved to be a workable arrangement.

The total coliform group merits consideration as an indicator of pollution because these bacteria are always present in the normal intestinal tract of humans and other warm-blooded animals and are eliminated in large numbers in fecal wastes. Thus, the absence of total coliform bacteria is evidence of a bacteriologically safe water.

Some strains included in the total coliform group have a wide distribution in the environment but are not common in fecal material. *Enterobacter aerogenes* and *Enterobacter cloacae* are frequently found on various types of vegetation (2-5) and in materials used in joints and valves (6-7).

The intermediate-aerogenes-cloacae (I.A.C.) subgroups may be found in fecal discharges, but usually in smaller numbers than *Escherichia coli* that is characteristically the predominant coliform in warm-blooded animal intestines (8-10). *Enterobacter aerogense* and intermediate types of organisms are commonly present in soil (11-14) and in waters polluted some time in the past. Another subgroup comprises plant pathogens (15) and other organisms of indefinite taxonomy whose sanitary significance is uncertain. All of these coliform subgroups may be found in sewage and in the polluted water environment.

Survival Times

Organisms of the I.A.C. group tend to survive longer in water than do fecal coliform organisms (16-18). The I.A.C. group also tends to be somewhat more resistant to chlorination than *E. coli* or the commonly occurring bacterial intestinal pathogens (19-22). Because of these and other reasons, the relative survival times of the coliform subgroups may be useful in distinguishing between recent and less recent pollution. In waters recently contaminated with sewage, it is expected that fecal coliform organisms will be present in numbers greater than those of the I.A.C. subgroup; but in

waters that have been contaminated for a considerable length of time or have been insufficiently chlorinated, organisms of the I.A.C. subgroup may be more numerous than fecal coliform organisms (23).

Differentiation of Organisms

Because various numbers of the coliform group normally grow in diverse natural habitats, attempts have been made to differentiate the population in polluted waters, with specific interest directed toward those coliforms that are derived from warm-blooded animal contamination. In his pioneering research, McConkey (23, 24) defined the aerogenes group in terms of certain fermentation characteristics, ability to produce indole, and reaction in the Voges-Proskauer test. Other developments refined techniques that progressed to differentiate the coliform group on the basis of indole production, methyl red, and Voges-Proskauer reactions, and citrate utilization (IMViC tests) into the *E. coli*, *Enterobacter aerogenes*, intermediate, and irregular subgroups (24-28).

In another approach to coliform differentiation, Hajna and Perry (29) and Vaughn, Levine, and Smith (30) further developed the Eijkman test (31) to distinguish organisms of fecal origin from those of nonfecal origin by elevating the incubation temperature for lactose fermentation. Geldreich, and associates, (31, 32) further refined the procedure and developed additional data to indicate the specific correlation of this elevated temperature procedure to the occurrence of fecal contamination.

Fecal Coliform Measurements

The fecal coliform bacteria, a subgroup of the total coliform population, does have a direct correlation with fecal contamination from warm-blooded animals. The principal biochemical characteristic used to identify fecal coliform is the ability to ferment lactose with gas production at 44.5° C. Research data have shown that 96.4 percent of the coliforms in human feces were positive by this test (10). Examination of the excrement from other warm-blooded animals, including livestock, poultry, cats, dogs, and rodents (33-34), indicate the fecal coliforms contribute 93.0 to 98.7 percent of the total coliform population. The predominant fecal coliform type most frequently found in the intestinal flora is *E. coli*. Occasionally, other coliform IMViC types may predominate for periods of several months before a shift occurs in type distribution. For this reason, it is more significant to be able to measure all coliforms common to the intestinal tract. In man, particularly, there is a significantly greater positive correlation with the broader fecal coliform concept (96.4 percent) than with identification of *E. coli* by the traditional IMViC biochemical reactions (87.2 percent).

Application to Treated Water

The presence of any type of coliform organism in treated water suggests either inadequate treatment or contamination after post-chlorination (23). It is true there are some differences between various coliform strains with

regard to natural survival and their chlorination resistance, but these are minor biological variations that are more clearly demonstrated in the laboratory than in the water treatment system. The presence of any coliform bacteria, fecal or nonfecal, in treated water should not be tolerated.

Insofar as bacterial pathogens are concerned, the coliform group is considered a reliable indicator of the adequacy of treatment. As an indicator of pollution in drinking water supply systems, and indirectly as an indicator of protection provided, the coliform group is preferred to fecal coliform organisms. Whether these considerations can be extended to include rickettsial and viral organisms has not been definitely determined.

Sample Size

The minimum official sample volume cited in the earlier editions of the *Drinking Water Standards* and *Standard Methods for the Examination of Water and Wastewater* was either stated or implied to be 50 ml because of the requirement to inoculate a series of 5 lactose broth fermentation tubes, each with a 10 ml or 100 ml portion of the sample. Few laboratories ever routinely employed the larger portions in the multiple tube procedure because of the attendant problems of preparing, handling and incubating the larger sized sample bottles that are required. Thus, when the multiple tube procedure was used, it became a practice to examine only 50 ml. With the development of the membrane filter procedure for routine potable water testing, the examination of larger sample volumes became practical, limited only by the turbidity of water and excessive bacterial populations.

Since many water supplies are sampled infrequently during the month, it is statistically more meaningful to examine a large sample for greater test precision with reduced risk of failing to detect some low level occurrence of coliforms. Increasing the sample portion examined will tighten the base line sensitivity and is particularly important for measuring the coliform reduction capacity of disinfection that approaches the magnitude essential for control of waterborne virus. Mack et al (35) reported poliovirus type II could be isolated from a restaurant well water supply using a flocculant in the 2.5 gallon samples prior to centrifugation to concentrate the low density virus particles. Bacteriological examinations of 50 ml portions of the unconcentrated water samples were negative for coliforms. However, coliforms were found in the concentrated sediment pellets. Future studies on coliform to virus occurrence in potable water may require further tightening of the coliform standard, possibly to a one-liter base (36).

The recommendations to increase the sample size to 100 ml for bacteriological examinations of water is supported in the 13th Edition of *Standard Methods* where the larger volume is stated as preferred. A study of State Health Laboratory procedures indicates that 39 or 78 percent of these laboratory systems are currently using 4 oz sample bottles to collect 100 ml of sample, and 25 of these State Health Laboratory networks are examining all public water samples by the membrane filter procedure. These figures

suggest that the stronger position now being proposed on a minimum sample size of 100 ml for statistically improved coliform monitoring is not unrealistic in terms of current practice.

Application to Source Waters and Untreated Potable Supplies

In the monitoring of source water quality, fecal coliform measurements are preferred, being specific for fecal contamination and not subject to wide-range density fluctuation of doubtful sanitary significance.

Although the total coliform group is the prime measurement of potable water quality, the use of a fecal coliform measurement in untreated potable supplies will yield valuable supplemental information. Any untreated potable supply that contains one or more fecal coliforms per 100 ml should receive immediate disinfection.

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Substitution of Residual Chlorine Measurement for Total Coliform Measurement

The best method of assuring the microbiological safety of drinking water is to maintain good clarity, provide adequate disinfection, including maintenance of a disinfectant residual, and to make frequent measurements of the total coliform density in the distributed water. In the 1962 U.S. Public

Health Service Drinking Water Standards, the major emphasis was on the *measurement of total coliform densities* and a sampling frequency graph relating number of samples per month to population served was included. The sampling frequency ranged from two per month for populations of 2,000 and less to over 500 per month, for a population of 8 million.

The effectiveness of this approach for assuring microbiological safety was evaluated during the 1969 Community Water Supply Survey. The results of this evaluation by McCabe, et al., (1) are paraphrased below.

Microbiological Quality

To determine the status of the bacteriological surveillance program in each of the 969 water supply systems investigated, records in the State and county health departments were examined for the number of bacteriological samples taken and their results during the previous 12 months of record. Based on this information, only 10 percent had bacteriological surveillance programs that met the "criteria," while 90 percent either did not collect sufficient samples, or collected samples that showed poor bacterial quality, or both. The table below summarizes the results.

	Bacteriological Surveillance			
Population	500 or Less	501 100,000	Greater than 100,000	All Populations
Number of Systems	446	501	22	969
	Percent of Systems			
Met Criteria	4	15	36	10
Did not meet Criteria	95	85	64	90

Sampling Frequency

Insufficient samples were taken in more than one of the previous 12 months of record from 827 systems (85 percent of the survey total). Even considering a sampling rate reduced by 50 percent of that called for in the criteria, 670 systems (69 percent of the survey total) still would not have collected sufficient samples.

Recommendation

The water utility should be responsible for water quality control, but the bacteriological surveillance collection requirements are not being met in most small water systems even though only two samples per month are required. A more practical technique must be developed if the public's health is to be protected. If all systems were chlorinated, a residual chlorine determination might be a more practical way of characterizing safety.

The validity of the recommendation that the measurement of chlorine residual might be a substitute for some total coliform measurements has been investigated by Buelow and Walton (2) Because the recommended rate of sample collection could not be or was not being used, alternative

methods of indicating safety were considered. One suggestion was to substitute the measurement of chlorine residual for some of the bacteriological samples. This method has the advantage of being easy to perform, and thus providing an immediate indication of safety. Further, data from London, U.K.; Cincinnati, Ohio; and the 1969 Community Water Supply Survey (CWSS) has shown that present sampling locations do not protect all consumers and that chlorine residual can be used to replace some coliform determinations.

Sampling Location

During 1965-66, the London Metropolitan Water Board using its Standards, made bacteriological examinations of 11,371 samples of water entering the distribution system, 947 samples taken from distribution reservoirs, 2,720 samples taken following pipeline breaks anl 689 samples from miscellaneous locations (complaints, hospitals, etc.). Most of the unsatisfactory results were associated with reservoir problems. Main breaks and miscellaneous samples were responsible for most of the remaining unsatisfactory samples.

Chlorine Residual

In Cincinnati during the 1969-70 period of free chlorine residual, approximately 24 samples were collected from each of 143 sampling stations. None of the samples from 116 of these stations showed presence of coliform, and 23 of the remaining sampling stations showed coliform bacteria in only one out of the approximately 24 samples examined. At the other four stations where 2 or more coliform-positive tests were obtained from the 24 samples, three had no chlorine residual at the time the coliform-positive samples were collected. The question is raised, therefore, as to the need for examining samples routinely collected from a large number of stations scattered throughout the system without regard to the water's residual chlorine content. Maintaing a free chlorine residual of 0.2 mg/l in the Cincinnati, Ohio, distribution system reduced the percentage of coliform positives to about 1 percent. The table below from the CWSS data, shows that the presence of a trace or more of chlorine residual drastically reduced or eliminated total coliforms from distribution system samples.

PERCENT OF VARIOUS TYPES OF WATER SUPPLY SYSTEMS FOUND TO HAVE AVERAGE TOTAL COLIFORMS GREATER THAN 1/100ML

Type of System	Non-Chlorinated	Chlorinated No Residual	With Any Detectable Residual
Spring	39	17	0
Combined Spring and Well	41	28	0
Well	8	5	0
Surface	64	7	2
Combined Surface and Well	100	16	3

These findings indicate that *a major portion of a distribution system, exclusive of deadends, reservoirs, etc., could be monitored for bacteriological safety by the use of chlorine residual.* (Emphasis added.) Therefore, when chlorine substitution is used, determination of total coliform densitities should be continued in problem areas, and some samples, as a check, should be collected in the main part of the distribution system.

These two studies led to the inclusion in the Regulations of Par. 141.21(h) on the substitution of chlorine residual tests for a portion of the required total coliform determinations. Par. 141.21(h) states that any substitution must be approved by the State on the basis of a sanitary survey. The following four items should be specified by the State:

1. The number and location of samples for which chlorine residuals are to be substituted.
2. The form and concentration of chlorine residual to be maintained;
3. The frequency of chlorine residual determinations; and
4. The analytical method to be used.

While each approval must be made individually, taking into account individual circumstances, the following may offer some guidance. The first requirement is the establishment of the relationship between chlorine residual and the absence of total coliforms in any given water. This may not be too difficult in larger supplies where both of these measurements are routinely made, but it might be quite difficult for the smaller purveyors (where the most help is needed) who have not been making either measurement.

The number and location of samples for which chlorine residuals are to be substituted

Total coliform measurements should continue to be made of the finished water as it enters the distribution system and at known trouble spots such as reservoirs and dead ends. Substition can be considered in the free-flowing portion of the distribution system.

The chlorine residual to be maintained

In general, a low turbidity water with a free chlorine residual of about 0.2 mg/l at a pH of less than 8.5 will be free from total coliforms although these conditions may vary from water to water. However, a higher free chlorine residual or the use of some other disinfectant is required prior to the water entering the distribution system, where disinfection is practiced, if initial disinfection is to be adequate.

The frequency of chlorine residual determinations

Because the chlorine residual test is so easy to perform, it is reasonable to expect the substition of several chlorine residual determinations for each total coliform test deleted. In this way wider coverage of the distribution system can be achieved, thereby increasing the protection to the consumer. Since, for maximum protection, chlorination must be continuous, it is also reasonable to expect that a minimum of one daily determination of chlorine

residual be performed whenever the chlorine residual option has been chosen. By limiting the extent of substitution to 75% of the required bacteriological samples, a sufficient number of bacteriological samples will still be taken to enable the assessment of the adequacy of disinfection and to assure the continuity of water quality records.

The analytical method to be used

An analytical method free of interferences to eliminate false residuals must be recommended. For this reason the DPD method is specified.

Finally, when the chlorine residual option is in use and a free chlorine residual concentration less than that agreed to is measured at a sampling point, then a sample for total coliform analysis must be taken immediately from that point.

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General Bacterial Population

The microbial flora in potable water supplies is highly variable in numbers and kinds of organisms. Those bacterial groups most frequently encountered in potable waters of poor quality include: *Pseudomonas, Flavobacterium, Achromobacter, Proteus, Klebsiella, Bacillus, Serratia, Corynebacterium, Spirillum, Clostridium, Arthrobacter, Gallionella,* and *Leptothrix* (1-5). Substantial populations of some of these organisms occurring in potable water supplies may bring a new area of health risk to hospitals, clinics, nurseries, and rest homes (6-11). Although *Pseudomonas* organisms are generally considered to be non-pathogenic, they can become a serious "secondary pathogenic invader" in post-operation infections, burn cases, and intestinal-urinary tract infections of very young infants and the elderly population of a community. These organisms can persist and grow in water containing a minimal nutrient source of nitrogen and carbon. If *Pseudomonas* becomes established in localized sections of the distribution lines, it may persist for long periods and shed irregularly into the consumer's potable water supply (12). A continual maintenance of 0.3 to 0.6 mg/l free chlorine residual will suppress the development of an extensive microbial flora in all sections of the distribution network.

Flavobacterium strains can be prevalent in drinking water and on water taps and drinking-fountain bubbler-heads. A recent study of stored emergency water supplies indicated that 23 percent of the samples contained *Flavobacterium* organisms with densities ranging from 10 to 26,000 per 1 ml. *Flavobacterium* must be controlled in the hospital environment be-

cause it can become a primary pathogen in persons who have undergone surgery (13).

Klebsiella pneumoniae is another secondary invader that produces human infection of the respiratory system, genito-urinary system, nose and throat, and occasionally this organism has been reported as the cause of meningitis and septicemia (14). *Klebsiella pneumoniae*, like *Enterobacter aerogenes*, (15) can multiply in very minimal nutrients that may be found in slime accumulations in distribution pipes, water taps, air chambers, and aerators.

Coliform Suppression

The inhibitory influence of various organisms in the bacterial flora of water may be important factor that could negate detection of the coliform group (16-17). Strains of *Pseudomonas*, *Sarcina*, *Micrococcus*, *Flavobacterium*, *Proteus*, *Bacillus*, *Actinomycetes*, and yeast have been shown to suppress the detection of the coliform indicator group (18-21). These organisms can coexist in water, but when introduced into lactose broth they multiply at a rapid rate, intensifying the factor of coliform inhibition (22). Suspensions of various antagnoistic organisms in a density range of 10,000 to 20,000 per 1 ml, added to lactose tubes simultaneously with a suspension of 10 *E. coli* per 1 ml, resulted in reduction in coliform detection (19). This loss of test sensitivity ranged from 28 to 97 percent, depending on the combination of the mixed strains.

Data from the National Community Water Supply Survey (23) on bacteriological quality of distribution water from the 969 public water supplies were analyzed (Table 1) for bacterial plate count relationship to detection of total coliforms and fecal coliforms. It is interesting to note that there was a significant increase in total and fecal coliform detection when the bacterial counts increased up to 500 per 1 ml. However, further increase in the detection of either coliform parameter did not occur when the bacterial count per 1 ml was beyond 500 organisms. There was, in fact, progressively decreased detection of both coliform parameters as the bacterial count continued to rise. This could indicate an aftergrowth of bacteria in distribution system water or a breakpoint where coliform detection was desensitized by the occurrence of a large general bacterial population that included organisms known to suppress coliform recovery.

Control of the General Bacterial Population

Density limits for the general bacterial population must be related, in part, to a need to control undesirable water quality deterioration and practical attainment for water throughout the distribution system. This necessity for monitoring the general bacterial population is most essential in those supplies that do not maintain any chlorine residual in the distribution lines and in special applications involving desalinization. This bacteriological measurement would serve as a quality control on water treatment processes and sanitation of distribution line sections and storage tanks that could

be shedding various quantities of organisms into the system, thereby degrading the water quality.

TABLE 1.—Bacterial Plate Count vs. Coliform Detection in Distribution Water Networks for 969 Public Water Supplies

General Bacterial Population*		Total Coliform		Fecal Coliform	
Density Range per 1 ml	Number of Samples	Occurrences	Percent	Occurrences	Percent
1 - 10	1013	47	4.6	22	2.2
11 - 30	371	28	7.5	12	3.2
31 - 100	396	72	18.2	28	7.1
101 - 300	272	48	17.6	20	7.4
301 - 500	120	30	25.0	11	9.2
501 - 1,000	110	21	19.1	9	8.2
1,000	164	31	18.9	5	3.0
TOTAL	2446	277	—	107	—

*Standard Plate Count (48 hrs. incubation, 35°C)

Practical attainment of a low general bacterial population can best be judged by a study of data from the National Community Water Supply Survey. Data presented in Table 2 demonstrate the effectiveness of chlorine residual in controlling the general bacterial population in a variety of community water supply distribution systems. Although the number of samples on each distribution system in this special study was small, it does reflect bacterial quality conditions in numerous large and small water systems examined in each of the eight metroplitan areas and the entire State of Vermont.

These data indicate that the general bacterial population in distribution lines can be controlled to a value below 500 organisms per 1 ml by maintaining a residual chlorine level in the system. Increasing the chlorine residual above 0.3 mg/l to levels of 0.6 and 1.0 mg/l did not further reduce the bacterial population by any appreciable amount. Restricting such bacterial densities to a limit of 500 organisms per ml is, therefore, not only attainable in the distribution system, but is also desirable to prevent loss in coliform test sensitivity definitely observed at approximate densities of 1000 organisms per ml, thereby producing a safety factor of at least two.

TABLE 2.—The Effect of Varying Levels of Residual Chlorine on the Total Plant Count in Potable Water Distribution Systems*

Standard Plate Count**	Residual Chlorine (mg/l)							
	0.0	0.01	0.1	0.2	0.3	0.4	0.5	0.6
<1	8.1***	14.6	19.7	12.8	16.4	17.9	4.5	17.9
1 - 10	20.4	29.2	38.2	48.9	45.5	51.3	59.1	42.9
11 - 100	37.3	33.7	28.9	26.6	23.6	23.1	31.8	28.6
101 - 500	18.6	11.2	7.9	9.6	12.7	5.1	4.5	10.7
501 - 1000	5.6	6.7	1.3	2.1	1.8	0	0	0
>1000	10.0	4.5	3.9	0	0	2.6	0	0
Number of Samples	520	89	76	94	55	39	22	28

*Data from a survey of community water supply systems in 9 metropolitan areas (23)
**Standard Plate Count (48 hrs. incubation, 35°C)
***All values are percent of samples that had the indicated standard plate count.

Any application of a limit for the general bacterial population in potable water will require a definition of medium, incubation temperature, and incubation time so as to standardize the population to be measured. The 13th edition of Standard Methods for the Examination of Water and Wastewater does specify these requirements for a Standard Plate Count (SPC) to be used in collection of water quality control data. Because many organisms present in potable waters are attenuated, initial growth in plate count agar frequently is slow; thus, incubation time should be extended to 48 hours at 35 C. This time extension will permit a more meaningful standard count of the viable bacterial population. Samples must be collected in bottles previously sterilized within 30 days and adequately protected from dust accumulation. Examination for a Standard Plate Count should be initiated within 8 hours of collection. This time may be extended to periods up to 30 hours only if these samples are transported in iced containers.

With maintenance of a chlorine residual and turbidity of less than one Turbidity Unit, the need for a bacteriological measurement of the distribution system may become less critical. For this reason, it is recommended that such water supplies be monitored routinely for baseline data on the general bacterial population and correlated with chlorine residual and turbidity measurements in the distribution lines. It is also recommended that water plant personnel be alert to unusual circumstances that may make it desirable to monitor the general bacterial population more often in a check of water plant treatment efficiencies.

For these reasons, the general bacterial population should be limited to 500 organisms per 1 ml in distribution water. In theory, the limitation of the general bacterial population to some practical low level would also indirectly and proportionally limit any antagonistic organisms that could suppress coliform detection and reduce the exposure and dosage level for health effect organisms that might be present.

While no maximum contaminant level for general bacterial populations is included in the Interim Primary Drinking Water Regulations, it is recommended that the limit mentioned above be used as an operational guide in assessing the quality of drinking water delivered.

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Enteric Viruses in Water

Viruses of fecal and/or urinary origin from any species of animal may pollute water. Especially numerous, and of particular importance to health, are those viruses of human enteric origin. They include polioviruses, coxsackieviruses, echoviruses, adenoviruses, reoviruses, and the infectius hepatitis virus(es). Each group or subgroup consists of a number of different

serological types so that to date more then 100 different human enteric viruses are recognized.

Most infections with enteric viruses are mild, and many infections are subclinical, i.e., the infected individual is not sick. It is, however, generally agreed that all human viruses are pathogens, and as clinical experience accumulates, it is evident that the enteric viruses have at least two distinct effects on man: (a) the acute effect, e.g., poliomyelitis, meningitis, infectious hepatitis, etc. (b) the delayed effect, e.g., spontaneous abortion, congenital heart anomalies, insulin-dependent diabetes, malignancies, etc. All available evidence to date indicates that the acute clinical effects of enteric virus infection are many times more common than the delayed clinical effects which appear to be extremely rare and, in many cases, speculative.

Mosley (1) reviewed the literature in 1968 and cited 50 waterborne outbreaks of infectious hepatitis and 8 waterborne outbreaks of poliomyelitis. Nine of these infectious hepatitis outbreaks occurred in the United States, and 3 of these were reportedly from chlorinated municipal supplies. One is not certain, however, whether these 3 water supplies were really adequately treated. Only one of the 8 polimyelitis epidemics occurred in the United States, and this was the result of cross-connection contamination. Since Mosley's publication there have been three other reports of waterborne infectious hepatitis outbreaks in this country, all reportedly due to either sewage pollution of well water or cross-connection contamination. An estimated 20,000 - 40,000 cases of infectious hepatitis were reported in Delhi, India, in 1955-56 (2) atrributable to a municipal water supply source heavily overloaded with raw sewage. This outbreak, however, was not accompanied by noticeable increases of typhoid fever or other enterobacterial diseases, suggesting that, in practice, the virus(es) of infectious hepatitis may be more resistant to chlorine or chloramines than are vegetative bacteria. Weibel and co-workers (3) listed 142 outbreaks of gastroenteritis during the period of 1946 to 1970 in which epidemiologic evidence suggested a waterborne nature. More than 18,000 persons were affected in these outbreaks. Mosley (1) suspected that a significant portion of these cases must have been caused by viruses.

It is well recognized that many raw water sources in this country are polluted with enteric viruses. Thus, water supplies from such sources depend entirely upon the treatment processes used to eliminate these pollutants. Even though the processes may be perfectly effective, an occasional breakdown in the plant or any marginal practice of treatment could still allow the pollutants to reach the finished water supplies. It should be noted that Coin and his associates (4) have reported the recovery of viruses from raw and finished waters in Paris, France. Coin estimated that the Paris water probably contained one tissue culture unit of virus per 250 liters. Very recently, Mack et al (5) reported that poliovirus was recovered in water

from a deep well in Michigan. Although the well had a history of positive coliforms, coliforms and virus were not recovered from an unconcentrated water sample; only after a 2.5 gallon sample of water was subjected to high speed centrifugation were both virus and coliforms recovered. This study would seem to indicate that the present method of using the coliform test is not adequate to provide assurance of the non-presence of viruses. In summary, in the United States, most waterborne virus disease outbreaks have resulted from contamination of poorly treated drinking water by sewage either directly or through cross-connections. Overt outbreaks of virus disease from *properly* treated municipal water supplies are not known to have occurred. Proper treatment of surface water usually means clarification followed by effective disinfection.

Chang (6), however, has theorized that some water supplies that practice only marginal treatment may contain low levels of human viruses, and that this small amount of virus might initiate infection or disease in susceptible individuals. He believes that such individuals might thus serve as "index cases" and further spread the virus by person-to-person contact. Whether this hypothesis is true, can be proved only by an intensive survey for viruses in numerous drinking water supplies in this country, and such a survey has never been conducted. If viruses were detected in a survey of drinking water supplies, it would be necessary to conduct in-depth epidemiological studies to determine if actual infection or disease was being caused by these agents. Additionally, it would be necessary to determine what modifications would be required in the water treatment processes to eliminate these viruses.

The relative number of viruses and coliform organisms in domestic sewage is important in assessing the significance of the coliform test and the "virus safety" of water. Calculations by Clarke et al (7) have indicated the following virus-coliform ratios in feces, sewage, and polluted waters.

CALCULATED VIRUS-COLIFORM RATIOS

	Virus	Coliform	Ratio
Feces	200/gm	13×10^8/gm	1:65,000
Sewage	500/100 ml	46×10^6/100ml	1:92,000
Polluted Surface Water	1/100 ml	5×10^4/100 ml	1:50,000

It is apparent that coliform organisms far outnumber human enteric viruses in feces, sewage, and polluted surface water. It should be emphasized that these calculated ratios are only approximations and that they would be subject to wide variations and radical changes, particularly during a virus disease epidemic. Additionally, both bacteria and virus populations in sewage and polluted waters are subject to reductions, at different rates, from die-off, adsorption, sedimentation, dilution, and various other undetermined causes; thus, the coliform-virus ratio changes, depending upon conditions resulting from the combined effect of all factors preesnt. Thus, one must take

into consideration the most unfavorable conditions although they may be encountered very infrequently. Such conditions may impose considerable demands on the indicator system and treatment processes.

The efficacy of various water treatment processes in removing or inactivating viruses has recently been reviewed by Chang (6) and also in a Committee Report, "Engineering Evaluation of Virus Hazards in Water" (8). These reports indicate that natural "die-off" cannot be relied upon for the elimination of viruses in water. Laboratory pilot plant studies indicate that combination of coagulation and sand filtration is capable of reducing virus populations up to 99.7 percent if such treatments are properly carried out (9). It should be noted, however, that a floc breakthrough, sufficient to cause a turbidity of as little as 0.5 Turbidity Units, was usually accompanied by a virus breakthrough in a pilot plant unit seeded with high doses of virus (9). Disinfection, however, is the only reliable process by which water can be made free of virus. In the past, there have been numerous studies conducted on the chlorination of viruses. Recent work by Liu, et al (10), has confirmed early observations and has reemphasized two possible weaknesses in these early reports: (a) the number of virus types studied was very small, thus generalization on such results is not without pitfalls, (b) the early chlorination studies were usually conducted with reasonably pure virus suspensions derived from tissue cultures or animal tissue and may not represent the physical state of the virus as it exists under natural conditions (clumped, embedded in protective material, etc.) which would make the virus much more resistant to disinfectants. Thus, it is imperative that good clarification processes be used on turbid waters to reduce their turbidity levels that will ensure effective disinfection. Additionally, Liu's data show the wide variation in resistance to chlorine exhibited by viruses, e.g., four minutes were required to inactivate 99.99 percent of a reovirus population as contrasted to 60 minutes to achieve the same percent inactivation of coxsackievirus.

Virology techniques have not yet been perfected to a point where they can be used to routinely monitor water for viruses. Considerable progress on method development, however, has been made in the past decade. The methods potentially useful include: two-phase polymer separation (11), membrane filtration (12), adsorption on and elution from chemicals (13, 14, 15), and the gauze pad technique (16) to name a few. From the concerted efforts of virus-water laboratories throughout the world, it is hoped that a simple and effective method will become available for viral examination of water. In the interim, control laboratories having access to facilities for virus isolation and identification should be encouraged to use available procedures for evaluating the occurrence of human enteric viruses in treated waters.

As noted above, no simple and effective method for the viral examination of water is available at this time. When such a method is developed, and

when there are sufficient data to provide the necessary basis, a maximum contaminant level for virus will be proposed

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Turbidity

Drinking water should be low in turbidity prior to disinfection and at the consumer's tap for the following reasons:

(1) Several studies have demonstrated that the presence of particulate matter in water interferes with effective disinfection. Neefe, Baty, Reinhold, and Stokes (1) added from 40 to 50 ppm of feces containing the causative agent of infectious hepatitis to distilled water. They then treated this water

by varying techniques and fed the resultant liquid to human volunteers. One portion of the water that was disinfected to a total chlorine residual after 30 minutes of 1.1 mg/l caused hepatitis in 2 of the 5 volunteers. A similar experiment in which the water was first coagulated and then filtered, prior to disinfection to the same concentration of total residual, produced no hepatitis in 5 volunteers. This was repeated with 7 additional volunteers, and again no infectious hepatitis occurred.

Chang, Woodward and Kabler (2) showed that nematode worms can ingest enteric bacterial pathogens as well as virus, and that the nematode-borne organisms are completely protected against chlorinations even when more than 90 percent of the carrier worms are immobilized.

Walton (3) analyzed data from three waterworks treating surface waters by chlorination only. Coliform bacteria were detected in the chlorinated water at only one waterworks, the one that treated a Great Lakes water that usually did not have turbidities greater than 10 turbidity units (TU), but occasionally contained turbidities as great as 100 TU.

Sanderson and Kelly (4) studied an impounded water supply receiving no treatment other than chlorination. The concentration of free chlorine residual in samples from household taps after a minimum of 30 minutes contact time varied from 0.1 to 0.5 mg/l and the total chlorine residual was between 0.7 and 1 mg/l. These samples consistently yielded confirmed coliform organisms. Turbidities in these samples varied from 4 to 84 TU, and microscopic examination showed iron rust and plankton to be present. They concluded " . . . coliform bacteria were imbedded in particles of turbidity and were probably never in contact with the active agent. Viruses, being smaller than bacteria, are much more likely to escape the action of chlorine in a natural water. Thus, it would be essential to treat water by coagulation and filtration to nearly zero turbidity if chlorination is to be effective as a viricidal process."

Hudson (5) reanalyzed the data of Walton, above, relating them to the hepatitis incidence for some of the cities that Walton studied plus a few others. A summary of his analysis is shown in Table I. Woodward does, however, in a companion discussion warn against over interpreting such limited data and urges more field and laboratory research to clearly demonstrate the facts.

TABLE I.—Filtered-Water Quality and Hepatitis Incidence, 1953

City	Average Turbidity TU	Final Chlorine Residual mg/l	Hepatitis cases/100.00 people
G	0.15	0.1	3.0
C	0.10	0.3	4.7
H	0.25	0.3	4.9
B	0.2	—	8.6
M	0.3	0.4	31.0
A	1.0	0.7	130.0

Tracey, Camarena, and Wing (6) noted that during 1963, in San Francisco, California, 33 percent of all the coliform samples showed 5 positive tubes, in spite of the presence of chlorine residual. During the period of greatest coliform persistence, the turbidity of this unfiltered supply was between 5 and 10 TU.

Finally, Robeck, Clarke, and Dostal (7) showed by laboratory demonstration that virus penetration through a granular filter was accompanied by a breakthrough of floc, as measured by an increase in effluent turbidity above 0.5 turbidity unit in a pilot unit seeded with an extremely high dose of virus.

These 7 studies show the importance of having a low turbidity water prior to disinfection and entrance into the distribution system.

(2) The 1969 Community Water Supply Survey (8) revealed that unpleasant tastes and odors were among the most common customer complaints. While organics and inorganics in finished water do cause tastes and odors, these problems are often aggravated by the reaction of chlorine with foreign substances. Maintenance of a low turbidity will permit distribution with less likelihood of increasing taste and odor problems.

(3) Regrowth of microorganisms in a distribution system is often stimulated if organic matter (food) is present. An example of this possibility occurred in a Pittsburgh hospital (9). One source of this food is biological forms such as algae which may contribute to gross turbidity. Therefore, the maintenance of low turibidity water will reduce the level of this microbial food and maintain a cleanliness that will help prevent regrowth of bacteria and the growth of other microorganisms.

(4) The purpose of maintaining a chlorine residual in a distribution system is to have a biocidal material present throughout the system so that the consumer will be protected if the integrity of the system is violated. Because the suspended material that causes turbidity may exert a chlorine demand, the maintenance of a low turbidity water throughout the distribution system wil facilitate the provision of proper chlorine residual.

For these reasons, the limit for turbidity is one (1) Turbidity Unit (TU) as the water enters the distribution system. A properly operated water treatment plant employing coagulants and granular filtration should have no difficulty in consistently producing a finished water conforming to this limit.

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C—CHEMICAL QUALITY

The following pages present detailed data and the reasoning used in reaching the various limits.

In general, limits are based on the fact that the substances enumerated represent hazards to the health of man. In arriving at specific limits, the total environmental exposure of man to a stated specific toxicant has been considered. An attempt has been made to set lifetime limits at the lowest practical level in order to minimize the amount of a toxicant contributed by water, particularly when other sources such as milk, food, or air are known to represent the major exposure to man.

The Regulations are regarded as a standard of quality that is generally attainable by good water quality control practices. Poor practice is an inherent health hazard. The policy has been to set limits that are not so low as to be impracticable nor so high as to encourage pollution of water.

No attempt has been made to prescribe specific limits for every toxic or undesirable contaminant that might enter a public water supply. While the need for continued attention to chemical contaminants of water is recognized, the Regulations are limited to need and available scientific data or implications on which judgments can be made. Standards for innumerable substances which are rarely found in water would require an impossible burden of analytical examination.

The following table indicates the percent of samples analyzed in the Community Water Supply Study which exceeded 75% of the 1962 PHS Drinking Water Standards limits. This table shows the relationship of the existing quality of water analyzed during the study to the drinking water standards in effect at that time.

PERCENT OF SAMPLES IN THE COMMUNITY WATER SUPPLY STUDY WITH VALUE EXCEEDING 75% OF EACH LIMIT IN THE 1962 DRINKING WATER STANDARDS

Constituent	DWS Limit	DWS Limit X 0.75	Percent of Samples Exceeding
Arsenic	0.05 mg/l	0.0375 mg/l	1.24%
Barium	1 mg/l	0.75 mg/l	0.08%
Cadmium	0.010 mg/l	0.0075 mg/l	1.45%
Chloride	250 mg/l	187.5 mg/l	1.56%
Chromium	0.05 mg/l	0.0375 mg/l	1.43%
Color	15 C.U.	11.25 C.U.	3.54%
Copper	1 mg/l	0.75 mg/l	2.47%
Cyanide	0.2 mg/l	0.15 mg/l	0.00%
Foaming Agents	0.5 mg/l	0.375 mg/l	0.08%
Iron	0.3 mg/l	0.225 mg/l	15.81%
Lead	0.05 mg/l	0.0375 mg/l	3.32%
Manganese	0.05 mg/l	0.0375 mg/l	11.91%
Nitrate	45 mg/l	33.75 mg/l	3.46%
Selenium	0.01 mg/l	0.0075 mg/l	8.35%
Silver	0.05 mg/l	0.0375 mg/l	0.00%
Sulfate	250 mg/l	187.5 mg/l	3.37%
Zinc	5 mg/l	3.75 mg/l	0.35%

DAILY FLUID INTAKE

For the purpose of these Regulations, a daily intake of water or water based fluids of two liters was assumed. This figure was taken as being representative of the fluid consumption of a normal adult male, and was obtained by consulting standard textbooks on physiology and numerous journal articles concerning water consumption.

It was realized that tremendous variation in individual consumption would exist, but since women and children drink less than the average man, it was decided that a large percent of the population would consume less than two liters a day.

There have been numerous reports of individuals or groups of persons who consume abnormally large quantities of water or waterbased fluids. For example, the consumption of six liters of beer in a day (1, 2) is not unknown. However, it should be noted that anyone who consumes this quantity of beer would be getting more than 240 ml (½ pint) of pure alcohol which is close to the maximum tolerable dose for a day.

The Boy's Life Magazine (1971) (3) survey indicated that 8% of 10-17 year-old boys drink more than 8 soft drinks per day. This survey can be viewed from another angle and a statement made that 92% of such boys drink *less* than 8 soft drinks per day. It would probably be valid to state that the average consumption is far les than 8.

Guyton (1951) (4) properly indicates that diseased persons having diabetes insipidus consume great quantities of water a day but even raising the "daily fluid intake" to 6 liters a day would not protect these individuals who excrete up to 15 or more liters of urine per day. It might also be pointed out that diabetes insipidus is a relatively rare disease and that these patients could not be considered average consumers.

Welch, et al (5) show that at temperatures up to 75°F 2 liters or less of fluid are drunk per day by adult males.

Molnar, et al (6) found that average fluid intake in the desert was 5.90 liters per day with a standard deviation of ±2.03 whereas average fluid intake in the tropics was 3.26 liters with a standard deviation of ±1.09. These men were performing their normal duties including truck driving, guard duty, hiking, etc. Five percent of the men in the tropics drank as little as 1 liter a day.

Wyndham and Strydom (7) indicated that marathon runners lost between 1,500 and 4,200 ml of sweat in 20 miles of running at about 60°F. To replace their fluid that day would require from 2.5 to 5 liters of water.

In "Clinical Nutrition" (8) the normal water loss per day shown for a normal adult ranges from 1,500 ml - 2,100 ml. The breakdown for a 2,600 ml water intake is shown as 1,500 from fluids, 800 ml from food and 300 ml from metabolism.

In "Physiology of Man in the Desert" (9) the average intake of fluid for 91 men in the desert was 5.03 liters with a standard deviation of ±1.67.

This indicates that some men only drank three liters a day in a desert environment where temperatures went as high as 105°F.

In Best and Taylor's book, "The Physiological Basis of Medical Practice," (1945) (10) an average adult is shown to require 2,500 ml of water from all sources under ordinary circumstances. The sources of this water are shown as:

Solid and semisolid food	1200 cc
Oxidation of food	300 cc
Drinks (water, milk, coffee, beer, etc.)	1000 cc

This reference points out that cooked lean meat contains from 65 to 70 percent water.

It should be noted that certain references refer to water loss per day instead of drinking water intake. Water loss per day is approximately 1½ liters higher than the drinking water intake figure would be.

"Human Designs" (11) by Beck (1971) indicates that between 2200 ml and 2800 ml are required for an average adult with an average 2500 ml daily fluid intake. This author, however, reverses the food and drink quantities shown above. Both of these references indicate that 1 cc of water is required per calorie of food intake.

Two articles relating to the fluid intake of children might be cited here. One, by Galagan, et al (12), used children from under one year of age to age ten and showed that total fluid intake per pound of body weights was highest among infants and decreased with age. The water intake listed average 0.40 ounces (12 ml) per day per pound of body weight. They also found that water intake increased directly with increases in temperature.

The second article by Bonham, et al (13) concerns six-year old children and lists 0.70 ounces (21 ml) per day per pound. This is total fluid and includes milk. If a child of this age weighed 50 lbs., he would drink about one liter per day.

The "Bioastronautics Data Book" (14) lists an average of 2400 ml total water intake but indicates the breakdown as 1,500 ml from drinking water, 600 ml from food and 30 ml from oxidation of food.

More recently, the Task Group on Reference Man (1974) (15) estimated the water-based fluid intake of an adult man to be 1650 ml/day, with corresponding values for an adult woman of 1200 ml/day and for a child of 950 ml/day.

Considering all the information we have available, two liters per day drinking water consumption for the average man should be a reasonable estimate. It is twice the amount listed by some authors and 30% higher than other authors list as an average figure and is therefore defensible as a reference standard.

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ARSENIC

The high toxicity of arsenic and its widespread occurrence in the environment necessitate the setting of a limit on the concentration of arsenic in drinking water.

The presence of arsenic in nature is due mainly to natural desposits of the metalloid and to its extensive use as a pesticidal agent. Arsenic concentrations in soils range from less than one part per million (mg/l) to several hundred mg/l in those areas where arsenical sprays have been used for years. Despite relatively high concentrations of arsenic in soils, plants rarely take up enough of the element to constitute a risk to human health (1, 2). Despite the diminishing use of arsenicals as pesticides, presently several arsenites are used as herbicides and some arsenates as insecticides. In 1964, farmers in the U.S. used a combined total of approximately 15 million pounds of arsenicals (3).

The chemical forms of arsenic consist of trivalent and pentavalent inorganic compounds and trivalent and pentavalent organic agents. It is not known which forms of arsenic occur in the drinking water. Although combinations of all forms are possible, it can be reasonably assumed that the pentavalent inorganic form is the most prevalent. Conditions that favor chemical and biological oxidation promote the shift to the pentavalent specie; and conversely, those that favor reduction will shift the equilibrium to the trivalent state.

The population is exposed to arsenic in a number of ways. Arsenic is still used, albeit infrequently, to treat leukemia, certain types of anemia, and certain skin diseases (4). In the diet, vegetables and grain contain an average of 0.44 ppm and meats an average of 0.5 ppm of arsenic (5). Organic arsenicals are deliberately introduced into the diet of poultry and pigs as growth stimulators and pesticides. The Food and Drug Administration has set tolerance limits for residues of arsenicals on fruits and vegetables (3.5 mg as As_2O_3 per kg) and in meat (0.5 to 2.0 mg as As per kg) (6). Shellfish are the dietary components that usually contain the highest concentrations of arsenic, up to 170 mg/kg (2, 7, 8).

For the entire U.S., the arsenic concentrations in air range from a trace to 0.75 ug/m^3 (9). Airborne arsenic is usually the result of operating cotton gins, manufacturing arsenicals, and burning coal.

Arsenic content of drinking water ranges from a trace in most U.S. supplies to approximately 0.1 mg/l (10). No adverse health effects have been reported from the ingestion of water containing 0.1 mg/l of arsenic.

The toxicity of arsenic is well known, and the ingestion of as little as 100 mg can result in severe poisoning. In general, inorganic arsenicals are more toxic to man and experimental animals than the organic analogs; and arsenic in the pentavalent state is less toxic than that in the trivalent form.

Inorganic arsenic is absorbed readily from the gastro-intestinal tract, the lungs, and to a lesser extént from the skin, and becomes distributed through-

out the body tissues and fluids (4). Inorganic arsenicals appear to be slowly oxidized *in vivo* from the trivalent to the pentavalent state; however, there is no evidence that the reduction of pentavalent arsenic occurs within the body (5, 11-13). Inorganic arsenicals are potent inhibitors of the intracellular sulfhydryl (-SH) enzymes involved in cellular oxidations (14). Arsenic is excreted via urine, feces, sweat, and the epithelium of the skin (15-20). A single dose is usually excreted largely in the urine during the first 24 to 48 hours after administration; but elimination of the remainder of the dose continues for 7 to 10 days thereafter. During chronic exposure arsenic accumulates mainly in bone, muscle, and skin, and to a smaller degree in liver and kidneys. After cessation of continuous exposure, arsenic excretion may last up to 70 days (14).

A number of chronic oral toxicity studies with inorganic arsenite and arensate (21-25) demonstrated the minimum-effect and no-effect levels in dogs, rats, and mice. Three generations of breeding mice were exposed to 5 ppm of arsenite in the diet with no observable effects on reproduction. At high doses (i.e., 200 mg/l or greater) arsenic is a physiological antagonist of thyroid hormones in the rat (26). Arsenic is also an antagonist of selenium and has been reported to counteract the toxicity of seleniferous foods when added to argicultural animals' feed water (27, 28). Rats fed shrimp meat containing a high concentration of arsenic retain very little of the element as compared to rats fed the same concentrations of either arsenic trioxide or calcium arsenate (29), suggesting that the arsenic in shellfish tissues may be less toxic to mammals than that ingested in other forms.

In man, subacute and chronic arsenic poisoning may be insidious and pernicious. In mild chronic poisoning, the only symptoms present are fatigue and loss of energy. The following symptoms may be observed in more severe intoxication: gastrointestinal catarrh, kidney degeneration, tendency to edema, polyneuritis, liver cirrhosis, bone marrow injury, and exfoliate dermatitis (30, 31). In 1962, thirty-two school-age children developed a dermatosis associated with cutaneous exposure to arsenic trioxide (32, 33). It has been claimed that individuals become tolerant to arsenic. However, this apparent effect is probably due to the ingestion of the relatively insoluble, coarse powder, since no true tolerance has ever been demonstrated (14).

Since the early nineteenth century, arsenic was believed to be a carcinogen; however, evidence from animal experiments and human experience has accumulated to strongly suggest that arsenicals do not produce cancer. One exception is a report from Taiwan showing a dose-response curve relating skin cancer incidence to the arsenic content of drinking water (44). Some reports incriminated arsenic as a carcinogen (34, 35), but it was later learned that agents other than the metalloid were responsible for such cancers (36). Sommers and McManus (37) reported several cases of cancer in individuals who had at some time in their lives been exposed to thera-

peutic doses of arsenic trioxide (usually in Fowler's Solution). Patients displayed characteristic arsenic keratosis, but there was no direct evidence that arsenic was the etiologic agent in the production of the carcinoma.

Properly controlled studies (38, 39) have demonstrated that industrial workers do not have an increased prevalence of cancer despite continued exposure to high concentrations of arsenic trioxide. In the study by Pinto and Bennett (39), the exposure was estimated by comparing the arsenic excreted in urine of control and exposed populations. In the experimental group, some workers who had been exposed to arsenic trioxide for up to 40 years, excreted 0.82 mg of arsenic per liter, or more than six times the concentration of the control population. In addition, attempts to demonstrate through animal studies that arsenic is tumorigenic have met with failure (23, 35, 40-42). The possible co-carcinogenic role of arsenic trioxide in the production of methycholanthrene-induced skin tumors has been investigated and found to have no significant effect (43).

However, some recent evidence supports the view that arsenic is carinogenic. Industrial workers in a plant manufacturing arsenic powder were exposed to arsenic dust and showed a higher incidence of skin and lung cancer than other occupational groups (44, 45, 46). Ulceration of the nasal septum appears to be a common finding among workers exposed to inorganic arsenic. The incidence of skin cancer has also been reported to be unusually high in areas of England where arsenic was present in drinking water at a level of 12 mg/l (47). More recently Lee and Fraumeni found that the mortality rate of white male smelter workers exposed to both arsenic trioxide and sulfur dioxide exceeded the expected mortality rate by a statistically significant margin and found that lung cancer deaths among these workers increased with increasing lengths of exposure to arsenic trioxide. They concluded that their findings were "consistent with the hypothesis that exposure to high levels of arsenic trioxide, perhaps in interaction with sulphur dioxide or unidentified chemicals in the work environment, is responsible for the three-fold excess of respiratory cancer deaths among smelter workers" (48).

Similarly, Ott, et al., found, in a study for the Dow Chemical Company, that exposed employees in a dry arsenical manufacturing plant experienced a three-fold increase in lung cancer over the rate for non-exposed employees (49).

Baetjer, et al., in a study for the Allied Chemical Company, found that 19 of the 27 deaths occuring in this population between 1960 and 1972 were due to cancer as compared to an expected number, based on figures adjusted for age, race, and sex, of 7.3 cancer-related deaths (50).

Additional medical problems relating to arsenic content of drinking water have been reported from several other countries. Several epidemiological studies in Taiwan (51-55) have reported the correlation between increased incidence of hyperkeratosis and skin cancer with the consumption of water

with arsenic content higher than 0.3 mg/l. A similar problem has been reported in Argentina (56-58). Dermatological manifestations of arsenicism were noted in children of Antofagasto, Chile, who used a water supply with 0.8 mg/l. A new water supply was provided, and preliminary data show that arsenic levels of hair have decreased, and further study will be made of the health of persons born since the change in supply (59). Arsenicism affecting two members of a family where the arsenic content of the family's well varied between 0.5 and 2.75 mg/l over a period of several months, was reported in Nevada (60). A study in California found that a greater proportion of the population had elevated concentrations of arsenic in the hair when the drinking water had more than 0.12 mg/l than when it was below this concentration, but illness was not noted (61). In none of the cited incidents of apparent correlation of arsenic in drinking water with increased incidence of hyperkeratosis and skin cancer has there been any confirmed evidence that arsenic was the etiologic agent in the production of carcinomas.

Arsenic is a geochemical pollutant, and when it occurs in an area it can be expected to be in the air, food, and water, but in other cases it is due to industrial pollution. In some epidemiological studies it is difficult to determine which exposure is the greater problem. A recent study (62) of metallic air pollutants showed that arsenic levels of hair were related to exposure from this source, but other exposures were not quantitated. The Taiwan studies were able to compare quite similar populations that differed only in the water intake. Deep wells contained arsenic, but persons using shallow wells were not exposed.

The change in water supply in Chile provided a unique experience to demonstrate the effect of arsenic in drinking water in spite of other arsenic exposures.

It is estimated that the total intake of arsenic from food is an average of 900 ug/day (5). At a concentration of 0.05 mg per liter and an average intake of 2 liters of water per day, the intake from water would not exceed 100 ug per day, or approximately 10 percent of the total ingested arsenic.

In light of our present knowledge concerning the potential health hazard from the ingestion of arsenic, the concentration of arsenic in the drinking water shall not exceed 0.05 mg/l.

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BARIUM

Barium is recognized as a general muscle stimulant, including especially the heart muscle (1). The fatal dose for man is considered to be from 0.8-0.9 g as the chloride (550-600 mg Ba). Most fatalities have occurred from mistaken use of barium salts incorporated in rat poison. Barium is capable of causing nerve block (2) and in small or moderate doses produced transient increase in blood pressure by vasoconstriction (3). Aspirated barium sulfate has been reported to result in granuloma of the lung (4) and other sites in man (5). Thus, evidence exists for high acute toxicity of ingested soluble barium salts, and for chronic irreversible changes in tissues resulting from the actual despostion of insoluble forms of barium in sufficient amounts at a localized site. On the other hand, the recent literature reports no accumulation of barium in bone, muscle, or kidney from experimentally administered barium salts in animals (6). Most of the administered dose appeared in the liver with far lesser amounts in the lungs and spleen. This substantiates the prior finding of no measurable amounts of barium in bones or soft tissues of man (7). Later, more accurate analysis of human bone (British) showed 7 ug Ba/g ashed sample (8), but no increase in bone barium occurred from birth to death. Small amounts of barium have been shown to go to the skeleton of animals when tracer amounts of barium-140 were used (9), but no determinations of barium have been made in animals to which barium had been repeatedly administered for long periods.

No study appears to have been made of the amounts of barium that may be tolerated in drinking water or of effects from prolonged feeding of barium salts from which an acceptable water guideline may be set. A rational basis for a water guideline may be derived from the threshold limit of 0.5 mg Ba/m^3 air set by the American Conference of Governmental Industrial Hygienists (10) by procedures that have been discussed (11). By assuming that 75% of the barium inhaled is absorbed into the blood stream and that 90% is a reasonable factor for absorption via the gastrointestinal tract, a value of 2 mg/l can be derived as an approximate limiting concentration for a healthy adult population. The introduction of a safety factor to account for heterogeneous populations results in the derivation of 1mg/l as a limit that should constitute a "no effect" level in water. Because of the seriousness of the toxic effects of barium on the heart, blood vessels, and nerves, drinking water shall not contain barium in a concentration exceeding 1mg/l.

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CADMIUM

As far as is known, cadmium is biologically a nonessential, non-beneficial element of high toxic potential. Evidence for the serious toxic potential of cadmium is provided by: (a) poisoning from cadmium-contaminated food (1) and beverages (2); (b) epidemiologic evidence that cadmium may be associated with renal arterial hypertension under certain conditions (3); (c) epidemiologic association of cadmium with "Itai-itai" disease in Japan (4); and (d) long-term oral toxicity studies in animals.

The possibility of cadmium being a water contaminant has been reported in 1954 (5); seepage of cadmium into ground water from electroplating plants has resulted in cadmium concentrations ranging from 0.01 to 3.2 mg/l. Other sources of cadmium contamination in water arise from zinc-galvanized iron in which cadmium is a contaminant. The average concentration of cadmium in drinking water from community supplies is 1.3 ug per liter in the United States. Slight amounts are common, with 63 percent of samples taken at household taps showing 1 ug per liter or more. Only 0.3 percent of tap samples would be expected to exceed the limits of 10 ug per liter (6).

Several instances have been reported of poisoning from eating substances contaminated with cadmium. A group of school children were made ill by eating popsicles containing 13 to 15 mg/l cadmium (1). This is commonly considered the emetic threshold concentration for cadmium. It has been stated (7) that the concentration and not the absolute amount determines the acute cadmium toxicity; equivalent concentrations of cadmium in water are likewise considered more toxic than equivalent concentrations in food probably because of the antagonistic effect of components in the food.

Chronic oral toxicity studies in rats, in which cadmium chloride was added to various diets at levels of 15, 45, 75, and 135 ppm cadmium. showed marked anemia, retarded growth, and in many instances death at the

135 ppm level. At lower cadmium levels, anemia developed later; only one cadmium-fed animal had marked anemia at the 15 ppm level. Bleaching of the incisor teeth occurred in rats at all levels, except in some animals at 15 ppm. A low protein diet increased cadmium toxicity. A maximal "no effect" level was thus not established in the above studies (8). A dietary relation to cadmium toxicity has been reported by others (9).

Fifty mg/l of cadmium administered as cadmium chloride in food and drinking water to rats resulted in a reduction of blood hemoglobin and lessened dental pigmentation. Cadmium did not decrease experimental caries (10).

In a study specifically designed to determine the effects of drinking water contaminated with cadmium, five groups of rats were exposed to drinking water containing levels from 0.1 to 10 mg/l. Although no effects of cadmium toxicity were noted, the content of cadmium in the kidney and liver increased in direct proportion to the dose at all levels including 0.1 mg/l. At the end of one year, tissue concentrations approximately doubled those at six months. Toxic effects were evident in a three-month study at 50 mg/l (11). Later work has confirmed the virtual absence of turnover of absorbed cadmium (12). More recently, the accumulation of cadmium in renal and hepatic tissue with age has been documented in man (13).

Recent epidemiological evidence strongly suggests that cadmium ingestion is associated with a disease syndrome referred to as "Itai-itai" in Japan (4). The disease syndrome is characterized by decalcification of bones, proteinuria, glycosuria and increased serum alkaline phosphatase, and other more subjective symptoms. Similar clinical manifestations have been noted in cadmium workers (14). Yamagatta and Shigematsu (15) have estimated the current daily intake of cadmium in an endemic "Itai-itai" area as 600 ug. The authors from a geological and topographical survey as well as knowledge of local customs, concluded that the daily cadmium intake on the endemic area was probably higher in the past. They concluded that 600 ug per day would not cause "Itai-itai" disease. The average ingestion of cadmium is 59 ug/day in non-polluted areas of Japan.

The association of cardiovascular disease, particularly hypertension, with ingestion of cadmium remains unsettled. Conflicting evidence has been found both in man (3, 16) and in anmials (17, 18). It is notable that hypertension has not been associated with "Itai-itai" disease (19).

The main sources of cadmium exposure in the United States to the general population appear to be the diet and cigarette smoking. R.E. Duggan and P.E. Corneliussen (20) of the FDA in a market basket survey of five geographic regions in the U.S. found the "daily intake" of cadmium to be 50 ug in 1969 and 30 ug in 1970. Each market basket represented a 2-week diet constructed for a 16-19 year-old male. Murthy and associates found the cadmium intake of children to be 92 ug per day from a study of institutional diets (21). Other estimates are also generally higher than FDA's — ranging

from 67 to 200 ug/day. A review of these data suggest 75 ug as a reasonable estimate of average daily dietary intake (22, 23, 24, 25).

Cigarette smoking has also been shown to be important. Twenty cigarettes per day will probably cause the inhalation of 2-4 ug of cadmium (26). However, the absorption rate associated with cigarette smoke inhalation is much larger than that associated with food ingestion. Lewis (27) has shown in autopsy studies that men who smoke one or more packages of cigarettes per day have a mean cadmium concentration in the renal cortex (wet weight) double the level in a control group of non-smokers. Hammer (24) in similar studies also found renal wet weight concentrations for those smoking 1½ or more packages of cigarettes per day to be more than twice as high as for non-smokers. In terms of effective body burden, then, cigarette smoking may double the level derived from food intake alone.

Exactly what exposure to cadmium will cause proteinuria, the earliest manifestation of chronic cadmium poisoning, is unknown. From animal experiments and very limited human observation in cases of industrial exposure, it is believed that a cadmium level of 200 ppm wet weight in the renal cortex will be associated with proteinuria. (However, it should be noted that in one case a level of 446 ppm was found by Axelsson and Piscator without proteinuria) (29). It has been estimated that with 5% gastrointenstinal absorption, rapiod excretion of 10% of the absorbed dose, and 0.05% daily excretion of the total body burden, it would take 50 years with a daily ingestion of 352 ug of Cd to attain the critical level of 200 ppm wet weight in the renal cortex. The percentage absorption in man is unknown. If the gastrointestinal absorption of cadmium in man really is about 3%, it would probably take about 500-600 ug ingested per day to cause proteinuria.

Concentration of cadmium shall be limited to 0.010 mg/l in drinking water. At this level it would contribute 20 ug per day to the diet of a person ingesting 2 liters of water per day. Added to an assumed diet of 75 ug/day, this would provide about a four-fold safety factor. This does not, however, take cigarette smoking into account.

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CHROMIUM

Chromium, particularly in the hexavalent state, is toxic to man, produces lung tumors when inhaled, and readily induces skin sensitizations. Chromium occurs in some foods, in air including cigarette smoke, and in some water supplies (see Table I). It is usually in an oxidized state in chlorinated or aerated waters, but measurements for total chromium are easily made by atomic absorption, so the somewhat conversative total value is used for this guideline.

TABLE I

U.S. urban air concentrations range, 1965 (1)	0-0.028 ug/m^3
Chromium content in cigarette tobacco (2)	1.4 ug/cigarette
Chromium in foods cooked in stainless-steel ware (3)	0-0.35 mg/100 g
Chromium concentration range in water supplies 1969 (4)	0-0.08 mg/l

Comparatively little data are available on the incidence and frequency of distribution of chromium in foods. Although most information has limited applicability, one study (5) determined the occurrence of chromium and other elements in institutional diets. In that investigation, the concentrations of chromium in foods ranged from 0.175 to 0.470 mg/kg.

Chromium has not been proved to be an essential or a beneficial element in the body. However, some studies suggest that chromium may indeed by essential in minute quantities (5, 6, 7). At present, the levels of chromium that can be tolerated by man for a lifetime without adverse effects on health are still undetermined. A family of four individuals is known to have drunk water for periods of 3 years at a level as high as 0.45 milligrams chromium per liter without known effects on their health, as determined by a single medical examination (8).

A study by MacKenzie *et al* (8) was designed to determine the toxicity to rats of chromate (Cr^{+6}) and chromic (Cr^{+3}) ion at various levels in the drinking water. This study showed no evidence of toxic responses after one year at levels from 0.45 to 25 mg/l by the tests employed, viz., body weight, food consumption, blood changes and mortality. Significant accumulation of chromium in the tissues occurred abruptly at concentrations above 5 mg/l; however, no study has been made of the effects of chromium on a cancer-susceptible strain of animal. Recent studies demonstrated that 0.1 mg of potassium dichromate per kg enhances the secretory and motor activity of the intestines of the dog (10).

From these and other studies of toxicity (11-15), it would appear that a concentration of 0.05 mg/l of chromium incorporates a reasonable factor of safety to avoid any hazard to human health.

In addition, the possibility of dermal effects from bathing in water containing 0.05 mg/l would likewise appear remote, although chromium is recognized as a potent sensitizer of the skin (3). Therefore, drinking water shall not contain more than 0.05 mg/l of chromium.

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CYANIDE

Cyanide in reasonable doses (10 mg or less) is readily converted to thiocyanate in the human body and is thus much less toxic for man than fish. Usually, lethal toxic effects occur only when the detoxifying mechanism is overwhelmed. The oral toxicity of cyanide for man is shown in the following table.

ORAL TOXICITY OF CYANIDE FOR MAN

Dosage	Response	Literature Citations
2.9-4. 7 mg/l	Noninjurious	(1)
10 mg, single dose	Noninjurious	(2)
19 mg/l in water	Calculated from threshold limit for air to be safe	(3)
50-60 mg, single dose	Fatal	(4)

Proper chlorination to a free chlorine residual under neutral or alkaline conditions will reduce cyanide to very low levels. The acute oral toxicity of cyanogen chloride, the chlorination product of hydrogen cyanide, is approximately one-twentieth that of hydrogen cyanide (5). It should be noted that at a pH of 8.5 cyanide is readily converted to cyanate which is much less toxic.

Because of the above considerations, and because cyanide occurs, however rarely, in drinking water primarily as the result of spills or other accidents, there appears to be no justification for establishing a maximum contaminant level for cyanide.

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FLUORIDE

The Food and Nutrition Board of the National Research Council has stated that fluoride is a normal constituent of all diets and is an essential nutrient (1). In addition, fluoride in drinking water will prevent dental caries. When the concentration is optimum, no ill effects will result, and the caries rate will be 60-65 percent below the rates in communities with little or no fluoride (2, 3).

Excessive fluoride in drinking water supplies produces objectionable dental fluorosis which increases with increasing fluoride concentration above the recommended upper control limits. In the United States, this is the only harmful effect observed to result from fluoride found in drinking water (4, 5, 6, 7, 8, 9, 10, 11). Other expected effects from excessively high intake levels are: (a) bone changes when water containing 8-20 mg fluoride per liter (8-20 mg/l) is consumed over a long period of time (7); (b) crippling fluorosis when 20 or more mg of fluoride from all sources is consumed per day for 20 or more years (12); (c) death when 2,250-4,500 mg of fluoride (5,000-10,000 mg sodium fluoride) is consumed in a single dose (7).

The optimum fluoride level (see Table 1) for a given community depends on climatic conditions because the amount of water (and consequently the amount of fluoride) ingested by children is primarily influenced by air temperature. This relationship was first studied and reported by Galagan and Associates in the 1950's (13, 14, 15, 16), but has been further investigated and supported by Richards, et al (17) in 1967. The control limits for fluoride supplementation, as shown in Table 1, are simply the optimum concentrations for a given temperature zone, as determined by the Public Health Service, DHEW, from the data cited, plus or minus 0.1 mg/liter.

Many communities with water supplies containing less fluoride than the concentration shown as the lower limit for the appropriate air temperature range have provided fluoride supplementation (18, 19, 20, 21). Other communities with excessively high natural fluoride levels have effectively reduced fluorosis by partial defluoridation and by change to a water source with more acceptable fluoride concentration (22, 23, 24).

Richards, et al (17) reported the degree of fluorosis among children where the community water supply fluoride content was somewhat above the optimum value. From such evidence, it is apparent that an approval limit (see Table 1) slightly higher than the optimum range can be tolerated without any mottling of teeth, so where fluorides are native to the water supply, this concentration is acceptable. Higher levels should be reduced by treatment or blending with other sources lower in fluoride content. In such a case, the optimum value should be sought and maintained.

TABLE 1

Annual Average of Maximum Daily Air Temperatures F	Recommended Control Limits Fluoride Concentrations in mg/l			Approval Limit mg/l
	Lower	Optimum	Upper	
50.0 - 53.7	1.1	1.2	1.3	2.4
53.8 - 58.3	1.0	1.1	1.2	2.2
58.4 - 63.8	0.9	1.0	1.1	2.0
63.9 - 70.6	0.8	0.9	1.0	1.8
70.7 - 79.2	0.7	0.8	0.9	1.6
79.3 - 90.5	0.6	0.7	0.8	1.4

It should be noted that, when supplemental fluoridation is practiced, it is particularly advantageous to maintain a fluoride concentration at or near the optimum. The reduction in dental caries experienced at optimal fluoride concentrations will be diminished by as much as 50% when the fluoride concentration is 0.2 mg/l below the optimum. (25, 26).

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LEAD

Lead is well known for its toxicity in both acute and chronic exposures, Kehoe (1) has pointed out that in technologically developed countries, the widespread use of lead multiplies the risk of exposure of the population to excessive lead levels. For this reason, the necessity of constant surveillance of the lead exposure of the general population via food, air, and water is imperative.

The clinical picture of lead intoxication has been well documented (2). Unfortunately, the general picture of the symptoms is not unique (i.e., gastrointestinal disturbances, loss of appetite, fatigue, anemia, motor nerve paralysis, and encephalopathy) to lead intoxication and often this has resulted in misdiagnosis (3, 4). Several laboratory tests that are sensitive to increased lead blood levels have been developed for diagnostic purposes, but their relationship to the effects of lead intoxication are incompletely understood. The most sensitive of these is the inhibition of red cell-aminolevulinic acid dehydrase (ALAD) which correlates well with blood lead levels from 5-95 ug/100 g blood (5, 6). Because this is not the rate-limiting step in porphyrin biosynthesis, accumulation of aminolevulinic acid (ALA) does not occur until high blood lead levels are reached. Other such tests, which correlate with blood lead to a lesser degree and at higher levels, are the measurment of urinary coproporphyrins, the number of coarsely stippled red-blood cells and the basophilic quotient (6). These changes, in themselves, have little known significance in terms of the danger to the health of the normal individual, for although red cell life-time can be shown to decrease (7), high lead concentrations are required for the development of the anemia typical of lead intoxication (8). Urinary ALA, however, has been shown to be closely related to elevated lead levels in soft tissues (9, 10) and is considered to be indicative of a probable health risk (11).

Young children present a special case in lead intoxication, both in terms of the tolerated intake and the severity of the symptoms (8). Lead encephalopathy is most common in children up to three years of age (12). The most prevalent source of lead in these cases of childhood poisoning has been lead-containing paint still found in many older homes (1, 12). Prognosis of children with lead encephalopathy is poor, with or without treatment. Up to 94% of the survivors have been found to have psychological abnormalities (13). It is still unknown whether smaller intakes of lead without encephalopathy or subclinical lead poisoning causes mental retardation or psychological abnormalities. Several studies in man and animals suggest this (14, 15, 16, 17), but a well-controlled prospective study in man has yet to be done. ALAD in baby rats' brains is suppressed by excess lead (18); however, the significance of this finding to humans is unknown. Some groups of individuals who experienced lead intoxication at an early age and survived have demonstrated a high incidence of chronic nephritis in later life (19). Recent work has demonstrated a high incidence of aminoaciduria and

other biochemical changes of kidney disease in children in Boston with excessive lead exposure (17). A recent study found anemia in children with blood levels from 37-60 ug/100 ml to be common (20). There is evidence that lead in high doses in animals affects the immunological system (21, 22, 23, 24); this, however, has not yet been demonstrated in man.

The average daily intake of lead via the diet was 0.3 mg in 1940 (25) and rarely exceeded 0.6 mg. Data obtained subsequent to 1940 indicate that the intake of lead appears to have decreased slightly since that time (1, 26). Inhaled lead contributes about 40% to total body burden of lead (1, 27) in the average population. Cigarette smoking in some studies in the past has also been associated with slightly elevated blood lead levels (3).

Accumulation of lead with age in non-occupationally exposed individuals has been demonstrated (26, 28, 29). The bulk of this lead distributes to bone, while soft tissues levels vary only slightly from normal even with high body burdens (30). Blood levels vary only slightly from normal even with high body burdens (30). Blood levels of lead in persons without unusual exposure to lead range up to 40 ug/100 g and average about 26 ug/100 g (1). The U.S. Public Health Service (31) considers 40 ug/100 g lead or over in whole blood in older children and adults on two separate occasions as evidence suggestive of undue absorption, either past or present. Levels of 50-79 ug/100 g require immediate evaluation as a potential poisoning case. Eighty ug/100 g or greater is considered to be unequivocal lead poisoning. The 40 ug/100 g lead level in blood probably has a biological effect as the National Academy of Science Lead Panel (11) concluded:

> ". . . the exponential increase in ALA excretion associated with blood lead content above approximately 40 ug/100 g of blood signifies inhibition of ALAD that is significant physiologically in vivo."

In addition animal experiments show beginning renal injury at about the same exposure level causing urinary ALA increase (32).

Blood lead is increased in urban vs. suburban (28, 33, 34), near to vs. distant from large motorways (35, 36) and in occupational exposure to areas of high traffic density (37, 38, 39). Lead in soil has epidemiologically been implicated in increased blood lead in children (40).

The World Health Organization Committee (41), assuming 10% of lead from food and water is absorbed, established *in adults* a "*Provisional tolerable weekly intake*" of 3 mg of lead per person (the maximum lead exposure the average person can tolerate without increased body burden). (Kehoe considers 600 ug per day the limit). Assuming 10% absorption from the gastrointestinal tract, approximately 40 ug of lead per day would be absorbed, by the WHO standard. With the average diet containing 100-300 ug lead per day, and the average urban air containing 1 to 3 ug/m^3 of air, the average urban man would absorb 16 to 48 ug of lead per day. (The contribution from 1 ug/m^3 lead in air at 20 m respiratory volume with 30% absorption is 6 ug). Just from food and air alone, some urban dwellers

would have excessive exposure by the WHO standard. Urban children are further exposed by dust with levels of over 1000 ug/g (40, 42, 43) and because airborne lead particles vary in density inversely from the distance from the ground (44, 45). Rural children have significantly less exposure than do urban children to these sources. Additionally, children have increased risk, because they have food and air intakes proportionally greater than their size and they might absorb a larger percentage from their gut, possibly 50% of ingested lead (46). Lead might also have a greater effect on their developing neurological, hematological, and immunological systems (18, 20-24, 47, 48). Likewise, fetuses of mothers unduly exposed may be at risk (49, 51), and McIntire concluded that there is a definite fetal risk maximal in the first trimester from intrauterine exposure to increased lead in maternal blood (52).

The lead concentrations in finished water ranged from 0 to 0.64 mg/liter in the Community Water Supply Study conducted in 1969 (53). Of the 969 water supplies surveyed, 1.4% exceeded 0.05 mg/liter of lead in drinking water. Five of the water supplies in this sample had sufficient lead to equal or exceed the estimated maximum safe level of lead intake (600 ug/day) without considering the additional contribution to the total intake by other routes of exposure. Under certain conditions, (acidic soft water, in particular), water can possess sufficient plumbosolvency to result in appreciable concentrations of lead in water standing in lead pipes overnight (54).

As a result of the narrow range between the lead exposure of the average American in every day life and exposure which is considered excessive (especially in children) it is imperative that lead in water be maintained within rather strict limits. Since a survey (55) of lead in surface water of the U.S.A. and Puerto Rico found only 3 of 726 surface waters to exceed 0.05 mg/l; the standard of 0.05 mg/l should be obtainable. For a child one to three years old drinking one liter of water a day (probably the most a child would drink), the contribution would be 0.05 mg/l x 1.0 liter equals 0.050 mg. The diet is estimated by scaling down the average adult's diet to be 150-200 ug (56). Assuming the fraction of lead absorbed is the same for lead in food and water, water would contribute 25 to 33% of the lead normally ingested. For an adult drinking 2 liters per day, the contribution would be 0.1 mg/0.3 mg, or 33% of food. At lower concentrations, for example, 0.015 mg/l, the average concentration in drinking water, the contribution of water in an adult or child would be less than 10% of that of food.

It should be reemphasized that the major risk of lead in water is to small children (50). The potentially signifcant sources of lead exposure to children which have been documented include paint, dust (40, 42, 43), canned milk (58, 59), tooth paste (60, 61), toys, newsprint ink (62, 63), and air. Although paint is most strongly implicated eqidemiologically, there is growing evidence that others, such as dust, are important (40). There is a serious problem with excess lead in children; it is well documented. It can lead to

lead poisoning. Lead poisoning does cause death and morbidity in children. A survey of 21 screening programs (64) testing 344, 657 children between 1969 and 1971 found 26.1% or over 80,000 children with blood leads of over 40 ug/l (which is considered evidence of excessive exposure.) Several recent studies suggest that the frequency of intellectual and psychological impairment is increased among children overexposed to lead who were not thought to have had overt clinical lead poisoning (14, 15, 16, 17). With the widespread prevalence of undue exposure to lead in children, its serious potential sequelae, and studies suggesting increased lead absorption in children (chronic brain or kidney damage, as well as acute brain damage); it would seem wise at this time to continue to limit the lead in water to as low a level as practicable. Data from the Community Water Supply Study and other sources indicate that a lead concentration of 0.05 mg/l or less can be attained in most drinking water supplies. Experience indicates that less than four precent of the water samples analyzed exceed the 0.05 mg/l limit and the large majority of these are due to stability (corrosion) problems not due to naturally occurring lead content in the raw waters.

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MERCURY

Environmental exposure of the population to mercury and its compounds poses an unwarranted threat to man's health. Since conditions indicate an increasing possibility that mercurials may be present in drinking water, there is a need for a guideline that will protect the health of the water consumer.

Mercury is distributed throughout the environment. And as a result of industrial and agricultural applications, large increases in concentrations above natural levels in water, soils, and air may occur in localized areas around chlor-alkali manufacturing plants and industrial processes involving the use of mercurial catalysts, and from the use of slimicides primarily in the paper-pulp industry and mercurial seed treatment.

Mercury is used in the metallic form, as inorganic mercurous (monovalent) and mercuric (divalent) salts, and in combination with organic molecules (viz. alkyl, alkoxyalkyl, and aryl).

The presence of mercury in fresh and sea water was demonstrated more than 50 years ago (1-4). In early studies in Germany, Stock (5, 6) found mercury in tap water, springs, rain water, and beer. In all water, the concentration of mercury was consistently less than one ug/l; however, beer occasionally contained up to 15 ug/l. A recent survey (7) demonstrated that most U.S. streams and rivers contain 0.1 ug of dissolved mercury or less per liter.

Presently the concentration of mercury in air is ill-defined for lack of analytical data. In one study (8) the concentration of mercury contained in particulates in the atmosphere of 2 U.S. cities was measured and ranged from 0.03 to 0.21 ug/m^3. One review (9) cited values up to 41 ug/m^3 of particulate mercury in one U.S. metropolitan area.

Outside of occupational exposure, food, particularly fish, is the greatest contributor to body burden of mercury. In 1967 a limited study of mercury residues in foods was conducted, involving 6 classes of foods. The results indicated levels of mercury in the order of 2 to 50 ug/kg. The Atomic Energy Commission sampled various foods for mercury in its tri-city study and reported levels between 10 and 70 ug of of mercury per kg of meats, fruits, and vegetables. In 1970, it was discovered that several types of fresh and salt water fish contained mercury (mostly in the alkyl form) in excess of the FDA guideline of 0.5 ppm (500 ug/kg). Mercury in bottom sediments had been converted by micro-organisms to the alkyl form, entered the food chains, and had accumulated in the higher members of the chains. Game birds were also discovered to have high levels of mercury in their tissues, persumably from the ingestion of mercury-treated seeds or of smaller animals that had ingested such seeds. The Food and Drug Administration has established a guideline of 0.5 ppm for the maximum allowable concentra-

tion of mercury in fish for human consumption; but for all other foodstuffs, no tolerances have been established.

Mercury poisoning may be acute or chronic. Generally mercurous salts are less soluble than mercuris salts and are consequently less toxic acutely. Acute intoxication is usually the result of suicidal or accidental exposure. For man the fatal dose of mercuric salts ranges from 20 mg to 3 g. The acute syndrome consists of an initial phase referable to local effects (viz. pharyngitis, gastroenteritis, vomiting, and bloody diarrhea) followed later by symptoms of systemic poisoning (viz. anuria with uremia, stomatitis, ulcerative-hemorrhagic colitis, nephritis, hepatitis, and circulatory collapse) (10).

Acute intoxication from the inhalation of mercury vapor or dusts leads to the typical symptoms of mercury poisoning coincident with lesions of the mucous membranes of the respiratory tract which may ultimately develop into bronchitis and bronchopneumonia. Inhalation of mercury in concentrations of 1,200 to 8,500 ug/m^3 results in acute intoxication (10). In severe cases, signs of delayed neurotoxic effects, such as muscular tremors and psychic disturbances, are observed. The Threshold Limit Value for all forms of mercury except alkyl is 0.05 mg/m^3 in the U.S. (11).

Chronic mercury poisoning results from exposure to small amounts of mercury over extended periods of time. Chronic poisoning from inorganic mercurials has been most often associated with industrial exposure, whereas that from the organic derivatives has been the result of accidents or environmental contamination.

Workers continually exposed to inorganic mercury are particularly susceptible to chronic mercurialism. Usually the absorption of a single large dose by such individuals is sufficient to precipitate the chronic disease that is characterized mainly by central nervous systems toxicity (10, 12, 13). Initially, non-specific effects, such as headaches, giddiness, and reduction in the power of perception, are observed. Fine tremors gradually develop primarily in the hands and are intensified when a particular movement is begun. In prolonged and severe intoxication, fine tremor is interspersed with coarse, almost choreatic, movements. Excessive salivation, aften accompanied by a metallic taste and stomatitis, is common. As the illness progresses, nervous restlessness (erethismus mercurialis) appears and is characterized by psychic and emotional distress and in some cases hysteria. Although the kidney is less frequently affected in this type of poisoning, chronic nephrosis is occasionally observed.

Several of the compounds used in agriculture and industry (such as alkoxyalkyls and aryls) can be grouped, on the basis of their effects on man, with inorganic mercury to which the former compounds are usually metabolized.

Alkyl compounds are the derivatives of mercury most toxic to man, producing illness from the ingestion of only a few milligrams (21, 24).

Chronic alkyl mercury poisoning, also known as Minamata Disease, is an insidious form of mercurialism whose onset may appear after only a few weeks of exposure or may not appear until after a few years of exposure. Poisoning by those agents is characterized mainly by major neurological symptoms and leads to permanent damage or death. The clinical features in children and adults include numbness and tingling of the extremities, incoordination, loss of vision and hearing, and intellectual deterioration. Autopsy of the clinical cases reveals severe brain damage throughout the cortex and cerebellum. There is evidence to suggest that compensatory mechanisms of the nervous system can delay recognition of the disease even when partial brain damage exists.

Several episodes of alkyl mercury poisoning have been recorded. As early as 1865, two chemists became ill and died as a result of inhaling vapors of ethyl mercury (14). One of the largest outbreaks occurred in a village near Minamata Bay, Japan, from 1953 through 1960. At least 121 children and adults were affected (of whom 46 died) by eating fish containing high concentrations of methyl mercury (15). Of the population affected, 23 infants and children developed a cerebral palsy-like disease which was referred to as Congenital (or Fetal) Minamata Disease. Similarly, in 1964 and 1965, the disease was reported among 47 persons, 6 of whom died, in Niigata, Japan. Hunter *et al* (16) reported 4 cases of industrial intoxication from handling of these agents. In Guatemala, Iraq, Pakistan, and the United States, the human consumption of grain treated with alkyl mercurials for seed purposes has led to the poisoning of more than 450 persons, some of whom died (17-20).

The congenital (fetal) disease observed in Minamata and Niigata emphasize the devastating and insidious nature of these agents. Of particular significance are the facts that (1) the affected children had not eaten contaminated fish and shellfish, and (2) the mothers apparently were not affected although they had consumed some contaminated food. Exposure of the fetus to mercury via the placenta and/or the mother's milk is believed to be the etiologic basis for this disease, thus indicating the greater susceptibility of infants to alkyl mercury.

Absorption is a factor important in determining the toxicity of alkyl mercurials. Berglund and Berlin (21) estimated that methyl mercury is absorbed at more than a 90% rate via gastro-intestinal tract as compared with 2% mercuric ion (22). In addition, methyl mercury crosses the placenta into the fetus and achieves a 30% higher concentration in fetal erythrocytes than in maternal red blood cells (23). However, the fetal plasma concentration of mercury is lower than that of the mother. The rate of uptake of methal mercury into the fetal brain is as yet unknown. Alkyl mercury can cross the blood-brain barrier more easily than other mercurials, so that brain levels of mercury are much higher after a dose of alkyl mercury than after a corresponding dose of any other mercurial.

Excretion is of equal importance in determining the health hazard. Unlike inorganic mercury, alkyl mercury is excreted mainly in the feces. After exposure to methyl mercury, approximately 4% of the dose is excreted within the first few days, and about 1% per day thereafter (24). The biological half-life of methyl mercury in man is approximately 70 days.

Safe levels of ingested mercury can be estimated from data presented in "Methyl Mercury in Fish" (24). From epidemiological evidence, the lowest whole-brood concentration of methyl mercury associated with toxic symptoms is 0.2 ug/g. This blood concentration can be compared to 60 ug Hg/g hair. These values, in turn, correspond to prolonged, continuous exposure at approximately 0.3 mg Hg/70 kg/day. By using a safety factor of 10, the maximum dietary intake should be 0.03 mg Hg/person/day (30 ug/70 kg/day). Although the safety factor is computed for adults, limiting ingestion by children to 30 ug Hg/day is believed to afford some, albeit smaller, degree of safety. If exposure to mercury were from fish alone, the limit would allow for a maximum daily consumption of 60 grams (420 g/week) of fish containing 0.5 mg Hg/kg. In a given situation, if the total daily intake from all sources, air, water, and food, is approaching 30 ug/person/day, the concentration of mercury and/or the consumption of certain foods will have to be reduced if a safety factor of 10 is to be maintained. Fortunately, since only a small fraction of the mercury in drinking water is in the alkyl form, the risk to health from waterborne mercury is not nearly so great as is the risk from mercury in fish. Also fortunately, mercury in drinking water seldom exceeds 0.002 mg/l. Drinking water containing mercury at the approval limit of 0.002 mg/l will contribute a total of 4 ug Hg to the daily intake, and will contribute less than 4 ng methyl mercury to the total intake. (Assuming that less than 0.1% of the mercury in water is in the methyl mercury form.) Since the Regulations approval limit is seldom exceeded in drinking water, the margin of safety gained from the restricted intake of mercury in drinking water can be applied to the total intake with minimal economic impact.

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NITRATE

Serious and occasionally fatal poisonings in infants have occurred following ingestion of well waters shown to contain nitrate (NO_3) at concentrations greater than 10 mg/l nitrate nitrogen. This has occurred with sufficient frequency and widespread geographic distribution to compel recognition of the hazard by assigning a limit to the concentration of nitrate in drinking water at 10 mg/l as nitrogen. This is about 45 mg/l of the nitrate ion.

Nitrate in drinking water was first associated in 1945 with a temporary blood disorder in infants called methemoglobinemia (1). Since then, approximately 2000 cases of this disease have been reported from North America and Europe, and about 7 to 8 percent of the infants died (2, 3, 4). Evidence in support of the limit for nitrate is given in detail by Walton (2) in a survey of the reported cases of nitrate poisoning of infants before 1951. The survey shows that no cases of poisoning were reported when the water contained less than 10 mg/l nitrate nitrogen. More recent surveys (3, 4) involving 467 and 249 cases tend to confirm these findings. Frequently, however, water was sampled and analyzed retrospectively and therefore the concentration of nitrate which caused illness was not really known. Many infants have drunk water when the nitrate nitrogen was greater than 10 mg/l without developing the disease. Many public water supplies in the United States have levels of nitrate that routinely exceed the standard, but only one case associated with a public water supply has been reported (5).

A basic knowledge of the development of the disease is essential to understanding the rationale behind protective measures. The development of methemoglobinemia, largely confined to infants less than three months old, is dependent upon the bacterial conversion of the relatively innocuous nitrate ion to nitrite. Nitrite then converts hemoglobin, the blood pigment that carries oxygen from the lungs to the tissues, to methemoglobin. Because the altered pigment can no longer transport oxygen, the physiologic effect of methemoglobinemia is that of oxygen deprivation, or suffocation.

The ingestion of nitrite directly would have a more immediate and direct effect on the infant because the bacterial conversion step in the stomach would be eliminated. Fortunately, nitrite rarely occurs in water in significant amounts, but waters with nitrite nitrogen concentrations over 1 mg/l should not be used for infant feeding. Waters with a significant nitrite concentration would usually be heavily polluted and would be unsatisfactory on a bacteriological basis as well.

There are several physiological and biochemical features of early infancy that explain the susceptibility of the infant less than three months of age to this disorder. First, the infant's total fluid intake per body weight is approximately three times that of an adult (6). In addition, the infant's incompletely developed capability to secrete gastric acid allows the gastric

pH to become high enough (pH of 5-7) to permit nitrate-reducing bacteria to reside high in the gastrointestinal tract. In this location, the bacteria are able to reduce the nitrate before it is absorbed into the circulation (7). To further predispose the infant, the predominant form of hemoglobin at birth, hemoglobin F (fetal hemoglobin), is more susceptible to methemoglobin formation than the adult form of hemoglobin (hemoglobin A) (8). Finally, there is decreased aotivity in the enzyme predominantly responsible for the normal methemoglobin reduction (NADH-dependent methemoglobin reductase) (9).

Winton reports on a study (10) where methemoglobin levels in blood were measured on infants to determine subclinical effects. He indicates that at intakes over 10 mg of nitrate ion per kilogram of body weight (2.2 mg/kg measured as nitrate nitrogen) the methemoglobin concentration is slightly elevated over normal. The methemoglobin levels returned to normal when the babies were changed to bottled water free of nitrate nitrogen. When a baby is fed a dehydrated formula that is made with water that the mother boils, (increasing the concentration), the intake of 2.2 mg NO_3-N/kilogram can be reached if the water contains 10 mg/l nitrogen. To determine if a slight elevation of an infant's methemoglobin concentration has an adverse health effect will require a large and elaborate study.

In some circumstances, which are not understood, the standard does not have a safety factor. Cases of illness might occur, but for the usual situation the limit of 10 mg/l NO_3-N will protect the majority of infants. Older children and adults do not seem to be affected, but the Russian literature reports (11) elevated methemoglobin in school children where water concentrations of NO_3-N were high, 182 mg/l.

Treatment methods to reduce the nitrate content of drinking water are being developed and should be applied when they are ready if another source of water cannot be used. If a water supply cannot maintain the NO_3-N concentration below the limit, diligent efforts must be made to assure that the water is not used for infant feeding. Consumption of water with a high concentration of NO_3-N for as short a period as a day may result in the occurrence of methemoglobinemia.

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ORGANIC CHEMICALS

The Environmental Protection Agency's problem of how to deal with the organics in drinking water is very complex. Several facts are undisputed:

1. Organics (synthetic and natural), some of which are produced during the disinfection of water with chlorine, are present in all drinking waters to some extent;

2. The organic compounds in raw source waters are from municipal and industrial point source discharges and from urban and rural non-point sources; the major portion of organics in most waters is of natural origin;

3. Most of the specific organic compounds in drinking water have not been identified and analysis for many of them is very difficult;

4. Most of the identified organics in drinking water have not been bioassayed;

5. Some of the organics that have been identified in drinking water in small quantities are toxicants, carcinogens, mutagens, and teratogens as indicated by animal bioassay tests conducted at high dosages;

6. The effect on humans of long-term ingestion of very low levels (ng/l to mg/l) of organic chemicals in drinking water is not known, and the portion of human exposure from drinking water versus the total exposure from all sources (food, air) is seldom known although the drinking water portion is usually considered to be small.

7. Some preliminary epidemiological studies have suggested a correlation between cancer mortality and the concentration of certain organics in drinking water but the conclusions are not firm;

8. With the passage of the Safe Drinking Water Act, Pub. L. 93—523, Congress intended that at least some organic contaminants in drinking water would be regulated;

9. Treatment processes are available for limiting the concentrations of most known organic contaminants of concern;

10. Treatment for the control of organic compounds, other than those that add taste and odor, is largely not practiced by water utilities in the United States, although some organics are undoubtedly removed by conventional treatment, which is commonly practiced;

11. Treatment for the control of organics would be an added expense and an added operational burden for the water works industry.

Given these facts, a course of action is not clear. EPA is deeeply concerned about the health of consumers of drinking water, but it does not wish to regulate frivously without more knowledge of costs and benefits.

Only within the last few years have instrumentation and techniques sophisticated enough to measure very small quantities of contaminants been applied to drinking water. With the aid of modern analytic techniques, such as gas chromatography and mass spectrometry, many types of organic chemicals have been detected in drinking water in various locations for the first

time. The subsequent discoveries of chemical contaminants, including known or suspected carcinogens, which may pose a threat to human health, contributed to the passage of the Safe Drinking Water Act (SDWA) in December 1974.

Certain industrial, agricultural, and environmental practices have allowed potentially harmful chemicals to enter the nation's drinking water. New compounds such as various pesticides and other organic chemicals have been introduced into the environment before full knowledge of their ultimate health effects were known. In order to cope with these realities yet protect human health to the maximum extent feasible, certain provisions were added to the Public Health Service Act by Pub. L. 93—523 to allow for greater and more comprehensive protection of public health from drinking water contaminants.

Under the SDWA, EPA is required to prescribe national drinking water regulations for contaminants that may adversely affect public health. Pursuant to section 1412(a) (1), EPA promulgated Interim Primary Drinking Water Regulations (40 CFR, Subpart D, FR Vol. 40, No. 248, pp. 59566 to 59587, Wednesday, December 24, 1975) which become effective in June 1977. These are based on a review and updating of the 1962 Public Health Service Standards and include Maximum Contaminant Levels (MCL's) for microbiological and chemical contaminants (primarily selected inorganic ions and organic pesticides) and turbidity (cloudiness in water). In addition, monitoring frequencies and public notification requirements for violations were established. National coverage was thereby expanded to approximately 40,000 community water systems and 200,000 other public water systems. Maximum Contaminant Levels for natural and man-made radioactivity were proposed in August 1975, promulgated in July 1976, and will also become effective in June 1977.

Revised Primary Drinking Water Regulations are scheduled for proposal in March 1977, followed by promulgation 6 months later; becoming effective 18 months thereafter (March 1979). These will either specify MCL's or require the use of specific treatment techniques, which in the Administrator's judgment will prevent known or anticipated adverse effects on health to the extent feasible. "Feasible" is defined in the SDWA as "use of the best technology, treatment techniques and other means which the Administrator finds are generally available (taking costs into consideration)."

Congress anticipated that organic chemicals would be dealt with primarily in the Revised Primary Drinking Water Regulations because of the paucity of data on the health effects of various organic chemicals, uncertainties over appropriate treatment techniques, and the need for additional information on the incidence of specific organic chemical in drinking water supplies. Because the Interim Primary Drinking Water Regulations did not contain Maximum Contaminant Levels for organic chemicals other than certain pesticides, EPA concurrently published Special Monitoring Regulations

(40 CFR, Subpart E, Vol. 40, No. 248, pp. 59587-59588, Wednesday, December 24, 1975) pursuant to Sections 1445 and 1450 of the Act, that provide for a national evaluation of the presence in drinking water of approximately 20 specific organic chemicals and simultaneously attempt to correlate their presence with several general organic measurement parameters.

In accordance with these Special Monitoring Regulations, EPA is currently conducting an extensive year long National Organics Monitoring Survey (NOMS) of drinking water supplies in 113 cities nationwide, which will reflect long-term and seasonal variations and represent various types of drinking water sources and treatment processes. Laboratory analyses will be used to evaluate the extent and nature of organic chemical contamination of drinking water, and to evaluate the validity of the several organic parameters as surrogates for measurement of potentially harmful organic chemicals.

The National Academy of Sciences is currently conducting a major study for EPA of the health effects related to contaminant levels in drinking water of many potential toxicants including organic chemicals, as mandated by the Safe Drinking Water Act. In this study the NAS will collect and evaluate currently available published and unpublished information relating to the toxicology of those substances in animals and humans and where possible, where they believe sufficient data exists, make recommendations of "safe" levels for humans. Among the factors the Academy will consider in this study are: the margin of safety required to protect particularly susceptible segments of the population; the contributions of various routes of exposure including water, air, food, and occupations; synergism among contaminants; and the relative risk of different levels of exposure. The Academy will also evaluate and report those contaminants that may pose a threat to human health, but whose current level in drinking water cannot be determined. For those contaminants, the Academy will recommend studies and test protocols for future research. The project, initiated in June 1975, is scheduled for completion by December 16, 1976.

Based on the NAS report, EPA will publish:

(1) Recommended maximum contaminant levels (health goals) for substances which may have adverse effects on humans. These recommended levels will be set so that no known or anticipated adverse effects would occur, allowing an adequate margin of safety. A list of contaminants which may have adverse effect on health, but which cannot be accurately measured in water, will also be published.

(2) Revised primary National Drinking Water Regulations. These will specify MCL's or require the use of treatment techniques. MCL's will be as close to the recommended levels for each contaminant as is feasible. Required treatment techniques for those substances which cannot be adequately measured will reduce their concentrations to a level as close to the recommended level as is feasible.

The Organics Problem

Thus far, more than 300 specific organic chemicals have been identified in various drinking water supplies in the United States. These compounds result from such sources as industrial and municipal discharges, urban and rural runoff, natural decomposition of vegetative and animal matter, as well as water and sewage chlorination practices. Although compositions and concentrations vary from locality to locality and from time to time, the occurrence of organic compounds in tap water is universally acknowledged. The human health effects of exposure to these compounds via drinking water are as yet unclear. However, some of them have been shown to be carcinogenic in animal tests and a few are known to be human carcinogens.

The majority of organic chemicals identified in drinking water have not been examined for potential health effects. Even in the case of those with recognized effects from studies at higher doses, the actual risk posed by ingesting very low concentrations over an extended period of time is not currently known. Some statistical correlations between water containing certain organics and cancer incidences have been suggested in some very preliminary studies. However, such correlations would not establish causality even if they were statistically valid. Health effects research and epidemiological studies involving organic chemical contamination of drinking water are underway in an attempt to assess the effect on human health of exposure to these substances from drinking water as well as the contribution of drinking water to total human exposure.

Chloroform, one of the trihalomethanes, serves as one example of the organics problem with which EPA is dealing. Advanced analytical techniques have facilitated the detection of chloroform in small amounts of drinking water. The National Organics Reconnaissance Survey (NORS) in 1975 confirmed the widespread presence of several previously determined organics in drinking water and, further, served to attribute the presence of chloroform and related trihalomethanes to the chlorination disinfection process itself. These results were subsequently supported by a further survey of 83 utilities within EPA's Region V.

The range of the levels of chloroform found in those chlorinated water surveys was from less than 1 microgram per liter to 366 micrograms per liter; 20 micrograms per liter median. Chloroform seldom was detected in the raw waters of those systems. The principal source of chloroform and other trihalomethanes in drinking water is the chemical interaction of the chlorine added for disinfection with the commonly present natural humic substances found in raw water. The extent of trihalomethane formation however, will vary depending upon season, contact time, water temperature, pH, type and chemical composition of raw water, and treatment methodology.

To help assess the health risk, EPA in 1975 sought the advice of its Science Advisory Board regarding potential carcinogenic or other adverse

health effects resulting from exposure to organic compounds in drinking water. Principal attention was directed to chloroform, carbon tetrachloride, chloroethers, and benzene.

The Board prefaced its Report with the caveat that the chemicals thus far identified in drinking water account for only a small fraction of the total organic content. Thus, the possibility exists that additional substances of equal or greater toxicological significance may be present. The Board also expressed concern that future studies should take into account possible synergistic effects of common combinations of contaminants. It recommended that a complete analysis of the problem consider data from all routes of exposure, such as dietary and occupation exposure, to these substances in addition to drinking water. Some of these additional sources of exposure may pose a much greater potential intake and risk than the consumption of drinking water.

The Report indicated that, in general, for all the compounds reviewed, the carcinogenicity data and experimental designs were either inappropriate or below the standard of current toxicological practice and protocols for carcinogenicity testing. Additional well-designed experimental studies to determine the carcinogenicity of lifetime exposures by ingestion were sorely needed.

According to the Report, carbon tetrachloride, a demonstrated carcinogen in laboratory studies, occurs in drinking water generally at much lower levels and is much less widespread than chloroform and related trihalomethanes. Benzene has not been clearly established to be carcinogenic in experimental animals, although epidemiological and clinical studies, largely of occupational exposures, suggest that possibility. Certain haloethers, chloro-olefins, and polynuclear aromatic hydrocarbons have been demonstrated to be carcinogenic in laboratory animals and have been identified in some drinking waters.

The Report concluded that some human health risk probably does exist from exposure through drinking water, although this risk is currently unquantifiable. The Report recommended that EPA seek ways to reduce exposure to these compounds without increasing the risk of infectious disease transmission.

In an early attempt to explore whether or not there is a relationship between water consumption and cancer, data obtained from the National Organics Reconnaissance Survey were compared with cancer mortality occurring in populations served by these water utilities. One preliminary study utilizing data from 50 of the 80 water utilities samples indicated a statistically significant correlation between the cancer mortality for all anatomical sites and both sexes combined in the years 1969-71, with the chloroform concentration in the sample collected in spring 1975. Such a correlation was not noted with total mortality or with the sum of the concentrations of the four trihalomethanes in the drinking waters.

In contrast to the above result, a similar epidemiological analysis of 43 cities from the Region V survey of 83 cities did not show any statistically significant correlation between chloroform or trihalomethanes and cancer mortality. Neither of these analyses attempted to correct for other variables that are known to be related to cancer mortality, and which might have had a fortuitous correlation with chloroform concentrations in water. Thus they must be considered preliminary. These preliminary results do, however, underline the need for more definitive analyses, which are now being attempted.

The recently released National Cancer Institute (NCI) Report on the bioassay of chloroform in rats and mice showed that chloroform caused cancers under the laboratory test conditions. EPA is very concerned with these findings and has asked NAS to study the NCI findings and other data on the carcinogenicity of chloroform as a part of its report to EPA under the Safe Drinking Water Act.

Taking note of the NCI Report, the Food and Drug Administration has banned the use of chloroform in human drugs, cosmetics and food packaging. On April 6, Dr. Alexander M. Schmidt, Commissioner, stated:

> The experiments on animals by no means prove that chloroform causes cancer in humans. The amount fed to the test animals exceeds, by far, the amount to which any person could be exposed with present products, but the benefits of cholorform are minimal and do not warrant any risk, however small.

Based on the information available at the time the Administrator of EPA stated that the prudent course of action was to take steps to reduce exposure to chloroform from drinking water wherever feasible by means that would not increase the risk of microbiological contamination. On March 29, 1976, EPA announced the institution of an experimental pilot cooperative chloroform reduction effort in which EPA would work through the states with a number of water utilities experiencing high chloroform levels. The program consists of carefully controlled modifications of existing water treatment processes in 10 to 20 water utilities. To provide the supporting information, a document titled "Interim Treatment Guide for the Control of Chloroform and Other Trihalomethanes" has been prepared by and is available from EPA's Water Supply Research Division in Cincinnati, Ohio. If successful the effort could be expanded to include many more systems. This technical assistance program will reduce human exposure to chloroform and other chlorination by-products in the short run, while providing information to support possible national regulations for organics is being developed.

Ongoing Research

In addition to the major NAS and NOMS studies in progress, research efforts designed to identify sources, distributions, treatment techniques and health effects of a variety of organic chemicals are being undertaken to find answers to the following questions:

1. What are the effects of commonly occurring organic compounds on human health?

2. What analytical procedures should be used to monitor finished drinking water to assure that any Primary Drinking Water Regulations dealing with organics are met?

3. Because some of these organic compounds are formed during water treatment, what changes in treatment practices are required to minimize the formation of these compounds in treated water?

4. What treatment technology must be applied to reduce contaminant levels to concentrations that may be specified in the Primary Drinking Water Regulations? What is the cost of this technology?

This research will involve health effects and epidemiological studies, investigations of analytical methodology, as well as pilot plant and field studies of organic removal unit processes.

The NAS and NOMS studies of drinking water contaminants with other additional research efforts will provide an overview of the drinking water problem essential in determining future national strategies. The results of all these efforts in conjunction with public comment and advice should contribute to the determination of whether an adequate basis exists, and if so, provide that basis for establishing maximum contaminant levels for specific organic contaminants that are found to be widespread, and/or for a general organics parameter(s), and/or treatment requirements that may be incorporated into the Primary Drinking Water Regulations. This information will enable the Administrator to determine appropriate health goals for these contaminants and then after considering technological and economic feasibility, to establish levels for National Primary Drinking Water Regulations. However, although treatment technology development is processing rapidly, significant new health effects information will probably not be available before regulatory decisions must be made because of the time required for completion of animal feeding studies (usually 3 years).

Future Action

Although health effects research is underway, definitive relationships between human health effects of low level exposure to specific chemicals from drinking water will be very difficult to establish, and such research requires considerable time lags between its inception and conclusion. EPA feels that the prudent action at this time is to consider the practically and feasibility of the available control technologies which may be applied to reduce exposure to many chemicals of unknown hazard and thus reduce the risks, whatever they may be, because of the following factors:

1. A large number of different chemicals have been found in drinking water albeit in low levels: several are considered carcinogens, others may have chronic toxic effects and more are likely to be found;

2. The large exposed population and the variable physiological susceptibilities of the individuals;

3. Statistical estimates of possible health effects, which although not definitive, suggest that some level of risk may exist;

4. The complexities of possible health effects from interactions of the many substances to which humans are exposed from a multitude of sources, including drinking water.

According to the SDWA, Primary Drinking Water Regulations shall protect health to the extent feasible, using technology treatment techniques and other means which EPA determines are generally available (taking cost into consideration). In light of those considerations and the difficulty in obtaining the essential health data and quantifying risks in limited time, technologically and economically feasible solutions must be considered which will reduce risks of exposure where necessary.

Possible Regulatory Options

Generally, organic chemicals in drinking water could be divided by sources and type under the following headings:

1. Chemicals derived from natural sources (e.g. humus);

2. Contaminants introduced as a result of treatment technology (e.g. trihalomethanes);

3. Synthetic chemicals from point sources (e.g. industrial chemicals);

4. Chemicals from non-point sources (e.g. pesticides or aromatics).

Several categories of contaminants must be considered and therefore several regulatory strategies may be necessary to address the problems fully. Additonal complications are raised by local factors including raw water quality, size of the water system, financial and personnel limitations, as well as the cost and availability of substances essential for treatment operation including granular absorbants, such as activiated carbon, reactivation facilities or disinfection chemicals.

The impact of any regulations for organics will be especially great on the small public water systems; those serving between 25 and 1000 or 10,000 persons. The installation, operation, and maintenance of some fairly sophisticated control processes and the monotoring requirements may result-in very substantial per capita costs for small systems. EPA pointed out this problem in the Interim Primary Drinking Water Regulations and is seeking means of alleviating it. Fortunately many of those small systems utilize ground water sources and some others may be able to switch to purer underground sources which would not require extensive treatment. Since many ground waters are already low in organics, they also would produce very little chloroform (trihalomethanes) and minimal, if any, treatment for organics control would be necessary in many cases. Thus, the following regulatory options would likely impact primarily surface water supplies and shallow ground water sources.

There are two basic regulatory philosophies possible within the SDWA: (1) Set Maximum Contaminant Levels for chemicals, or (2) Establish treatment technique requirements for substances which cannot be monitored

feasibly. Within each category several actions are possible. These include, for the MCL approach: (1) Establishing MCL's for each hazardous compound, or (2) developing general indicators of organic contamination and setting MCL's for these or (3) a combination of (1) and (2). Options within the designated treatment technology category include: (1) Modification of treatment and disinfection processes to eliminate specific contaminants such as chloroform (this could include substitution of other disinfection techniques for chlorine) or (2) requiring the use of a treatment technique such as granular activated carbon (GAC) to remove almost all organic compounds. These MCL and treatment options are not mutually exclusive, thus more than one might be appropriate. Some of them relate specifically to chloroform control and others are inclusive.

Establishment of MCL's for specific compounds or for a general organics contamination indicator would designate the maximum amount of the substance which is permitted to be in drinking water. The standards would be applicable in every public water system and periodic monitoring would be required to assure compliance. If an MCL were exceeded, the water utility would be required to notify the State and the water consumers and take corrective action.

The MCL approach would result in consistent health protection of drinking water throughout the nation. It offers flexibility by allowing each water system to use any acceptable means to achieve the standard. These could include: use of alternative water sources, blending, or treatment methods which could be optimized to be most cost effective in each specific case. In general, monitoring costs would be dependent upon the number and types of analyses required; and the problem is that many different substances might have to be regulated. Monitoring costs tend to be sensitive economic issues, particularly for small systems, where per capita expenditures may be substantial.

Owing to the number of MCL's which might be necessary to regulate organics in drinking water, and the feasibility of monitoring for such contaminants, it could also be appropriate to establish a treatment technique requirement for organics in drinking water. Under this approach (which could be phased-in according to system size), all public water systems would essentially be required to apply the best treatment available for total organics or, for example, chloroform removal.

A system may obtain a variance (deferral) of an MCL if the system cannot comply with an MCL, despite the application of the best technology available, because of the poor quality of the raw water which is reasonably available to the system. As with an exemption, the system must demonstrate that the variance will not result in an unreasonable risk to public health. The system must also comply with the MCL as expeditiously as practicable, in accordance with a compliance schedule to be established after a public hearing. It should be noted, however, that a variance from an MCL should not

be granted unless the system has already installed the best technology available so as to at least minimize the contamination in the drinking water.

A system would not be required to comply with a prescribed treatment technique if it can obtain a variance from the requirement under section 1415(a) (1) (B). A variance may be obtained if the system can demonstrate to the satisfaction of a State (or EPA if a State does not have primary enforcement responsibility) that such treatment technique is not necessary to protect the health of persons because of the nature of the raw water source of such system. In other words, if EPA prescribed GAC as a treatment technique for total organic contaminants, a system would not have to install GAC if it could demonstrate to the satisfaction of the State that its finished and/or raw water supply did not contain "harmful" quantities of total organics. This determination would presumably be based on federal and State guidelines taking into account local raw water conditions.

Section 1416 of the Act provides for temporary exemptions from MCL's or treatment techniques. Exemptions enable a public water system to remain out of compliance with an MCL or treatment technique for a limited period (up until 1981 under the interim regulations for most systems), subject to a compliance schedule. In order to obtain an exemption, a public water system must demonstrate to a State with primary enforcement responsibility (or otherwise to EPA) that (1) it was in operation in June of 1977; (2) there are compelling reasons (e.g. economic or technical) for such an exemption; and (3) the grant of such an exemption will not result in an unreasonable risk to public health. Within one year of the grant of an exemption, a State (or EPA) must hold a public hearing and establish a compliance schedule to enable the system to meet the applicable requirements.

In short, a public water system may defer the impact of an MCL or treatment technique upon a showing that such a deferral is necessary. However, the duration of such a deferral is limited by Statute and the compliance schedule established by the State (or EPA).

FWPCA

In addition to the SDWA, various elements of the Federal Water Pollution Control Act (Pub. L. 92—500) impact on the quality of drinking water sources; including control of effluents from point sources under sections 304, 307, 311 and others, non-point source controls, areawide waste treatment management under section 208 and possible reporting requirements under section 308. Use of Pub. L. 92—500 would prevent contamination of certain water sources by some organic chemicals, and any reduction in organic load in raw water would help a water utility maintain good finished water quality. Regulation sunder sections 304, 307, and 311 control the quality of receiving waters or limit effluent discharges. Under section 308, monitoring and reporting by dischargers can be required so that sources of pollutants can be identified. Non-point sources of contamination are even

more difficult to control and in situations where several sources of the same contaminant existed, enforcement becomes more difficult.

Non-regulatory Options

A. Other short run actions, though not necessarily general regulatory options, need to be considered. Under the Safe Drinking Water Act, EPA has the authority to take action to deal with an imminent and substantial endangerment to human health involving a public water system. Unlike maximum contaminant levels or required treatment techniques, which cannot take effect until June 24, 1977, the imminent hazard authority can be used immediately. However, as a practical matter this authority could only be used in a limited number of cases and does not appear to be appropriate for dealing on a national basis with widespread problems.

B. An interim alternative specifically for chloroform reduction would recommend the measurement of chloroform in finished water and offer technical assistance to interested states and water utilities wanting to alter their treatment procedure in order to lower chloroform concentrations, and thus the risk from chloroform exposure. Some initial monitoring would be necesary to determine which water utilities may need to alter their treatment procedures. Since this would not be regulatory or mandatory, not all water utilities which might need to take action will do so. Therefore, the health risk reduction to the population would not be as great as it would be by regulation. This was the interim approach outlined in the Administrator's statement of March 29, described earlier in this notice.

C. Another interim alternative, short of establishment of MCL's or treatment requirements, would be the issuance of regulations requiring monitoring for many organic chemicals. This would produce a large data base from which to develop future regulations, increase awareness of the presence of these contaminants, and point out the existence of potentially hazardous substances where they were previously not suspected. This could result in voluntary corrective actions, including the identification of sources so that some of these would be controlled.

D. Lastly, a choice may be to recommend that no change in current water treatment practices be made for the time being. Taking no regulatory action at this time would avoid impacting water utilities with treatment requirements that may be changed in the near future. Also, not taking regulatory action until additional data becomes available may be reasonable. However, the negative aspect of this action would be that no change in water works practice means no change in the current organic levels in finished water, and no reduction of potential health risks. However, it should be noted that the Agency has been challenged in the U.S. Court of Appeals for the District of Columbia, in part because more extensive organic standards were not contained in the Interim Primary Regulations of December 1975.

Advantages and Disadvantages of Various Options

I. *Maximum Contaminant Level Options.* A. *Establish MCL's for specific organic chemicals.* Based on nationwide distribution and health effects data, MCL's might be established for many specific organic substances. Because of limited health data now available, a major factor in many cases would be feasibility, based on economics and practicality of attaining lower risk exposure levels. Thus far, MCL's for 6 pesticides have been established in the Interim Primary Regulations. Others are currently being considered for regulation and more information is being gathered in the current NOMS program. The acceptable MCL's would undoubtedly be quite low (mg/l or ug/l level and below) thus both sophisticated monitoring (gas chromatography/mass spectrometry, (GC/MS)), and treatment methods would be necessary. Since a large number of chemicals would be candidates for MCL's, monitoring would probably be frequent and costly. A large number of systems would probably require some kind of treatment; variances and exemptions are possible under the Act but would only temporarily delay action. The most likely means of achieving the standards would involve use of less polluted source water or adsorbants.

Example, MCL for Chloroform (Trihalomethanes). Setting an MCL provides a legal requirement for a standard to be met on a nationwide basis by all public water systems and would require periodic monitoring along with public notification if an MCL is being exceeded. The means of achieving the MCL would be the prerogative of the individual water system. These could include: treatment process modifications; switching to a raw water source which contains less of the precursor compounds (e.g. groundwater); using a disinfectant other than chlorine (e.g. ozone, chlorine dioxide, chloramines); use of adsorbants to remove either precursor substances (more likely) or to remove chloroform (less likely). In some cases the action might be needed only intermittently (e.g. seasonally). Local conditions, including economics and available personnel, could determine which approach is the most practical.

If it were determined that the MCL approach is appropriate, a set of three possibilities related to chloroform are presented below as examples. A similar approach could be applied to some other compounds. The several control levels could also be applied consecutively in a phased approach starting with the less stringent levels and reducing them over some period of time as widespread compliance became more possible.

a. Establish Interim Levels to Cover Worst Case Situations: e.g. Chloroform, 100 ug/l: A small percentage of water utilities, mostly on surface sources, would be affected by a regulation at these levels. Such an MCL could be imposed under Amended Interim Primary Regulations, then reduced to the maximum extent feasible under the Revised Primary Drinking Water Regulations. This might be cost-effective since in many cases, only modest or seasonal modifications would be required to meet the standards.

This could include adjustment of chlorination practices, use of alternative disinfectants, or blending. Use of adsorbants for treatment would increase, but longer than optimal periods between reactivation (regeneration) could be employed because chloroform levels below 100 ug/l might be maintainable for several months in many localities. Considerable reduction of exposure would result at least seasonally for a fairly large population group.

b. Establish Levels at the Wintertime Median Found in the EPA National Survey: e.g. Chloroform, 20 ug: A very large number of water systems could be affected and considerable treatment would be necessary in many cases, at least seasonally. Granular activated carbon (GAC) or other adsorbants or alternative disinfectants would be necessary in many cases. A much broader population segment would be consuming water of considerably improved quality. Some phasing would undoubtedly be necessary, resulting in issuance of many variances or exemptions until widespread compliance could be achieved. Considerable increases in demand for adsorbants, ozone, chlorine dioxide, ammonia feeders, reactivation facilities and engineering service would result.

c. Establish Very Low Limit Levels: e.g. Chloroform, 5 ug/l or less: Virtually every surface water and many ground water systems would be affected and adsorbants or alternate disinfectants would be needed for treatment. Extensive phasing would be necessary, therefore, variances and exemptions would be extensively used. Demand for new equipment and chemicals and engineering services would be intense for several years. If GAC were used, reactivation would probably be required in many systems on monthly or shorter schedule and consumers would be receiving water of very high quality with respect to many chemicals as well as chloroform.

B. *Establish MCL's for general organic contaminant indicators.* Because of the probably multitude of organic contaminants in drinking water, the difficulties in toxicologically distinguishing between many of them at the low levels generally found in drinking water, and the impracticability and costs of monitoring and enforcing standards for tens or hundreds of individual contaminants, MCL's for groups of compounds or general organic indicators should be considered. This is analogous to the use of coliform bacteria as the indicator of microbiological contamination in water. These general parameters might consist of standards for groups such as polynuclear aromatics, or nitrosamines, or element analyses such as Non-Purgeable Total Organic Carbon (NPTOC), Total Organic Carbon (TOC), Total Organic Halogen (TOH), or Total Organic Nitrogen (TON).

Since a general indicator cannot distinguish individual compounds, some relationship should exist between the indicator's value and the levels of toxic compounds in the water, although the general organics indicator might not be as sensitive as the most sophisticated single compound analyses. The indicator could also be used as a trigger to indicate the need for more detailed analyses.

NPTOC is probably not sensitive to low level pollution from synthetic chemicals (pesticides and other non-humus type compounds). Total Organic Halogen (TOH) is used somewhat in Europe and it may be an acceptable indicator of the many halogenated industrial and pesticide compounds and halogenated trihalomethanes precursors. Total Organic Nitrogen (TON) may be an acceptable indicator of nitrogenous compounds, some of which may be precursors to nitrosamine formation. Ultraviolet absorption and fluorescence have also been suggested as possibilities. The Organics-Carbon Adsorbable (O-CA) test was suggested in the proposed Interim Primary Drinking Water Regulations but was rejected in the promulgated regulations. A common problem with these general indicators is the cost and availability of apparatus which is sensitive in low analytic ranges (sub mg/l). NPTOC analysis is the most highly developed and TOC and TAH development work is in progress. The NOMS is expected to provide data on several of these general indicators. Neither NPTOC, O-CA, UV or fluorescence correlated well with chloroform concentrations in the NORS.

Monitoring could probably be less frequent than for individual compounds and cost per analysis would be relatively small (less than $10 if performed externally), however instrument costs could be substantial ($6,000 to $10,000 each). Since the resulting numerical value is non-specific, additional analytical data might be necessary if the indicator value is exceeded.

Because of the insensitivity and non-specific nature of general organic indicators, selection of MCL's based on a direct health relationship is difficult, except by utilizing the principle (similar to the coliform indicator for microbiological contamination) that the lower the level of total organics, the smaller the possibility of adverse effects.

By analogy to the chloroform MCL discussion, MCL's for a general organic indicator, for example NPTOC, could be selected from several possible levels (e.g. 5 mg/l or 1 mg/l or 0.1 mg/l), and a phased reduction could be applied.

That approach has at least two problems: (1) NPTOC does not measure volatile compounds such as chloroform and (2) most utilities could be affected ultimately, and many variance and exemption requests would have to be processed. The second problem would be considerably alleviated if a reasonable phase-in schedule were employed. The ultimate benefit would be that drinking water of high quality, considering both health risk and esthetics (taste and appearance), would result.

C. *Combination of MCL's for specific compounds and general organic indicators.* Many water supplies that are known to be free of industrial or human waste discharge contamination contain a high concentration of a general organics indicator (e.g. NPTOC) because of the presence of large amounts of natural substances such as humus. Conversely, some waters contaminated with potentially hazardous chemicals at the microgram per liter level might have a low NPTOC at the milligram per liter level.

At least an initial possible solution might be to categorize water systems based on contamination type (e.g. natural or synthetic) and to establish MCL's combining both general indicators and appropriate specific chemicals. In this way, those water supplies contaminated with substances of greatest concern for which MCL's would be written would be required to take action first. Some element of phasing would be introduced, such that the high NPTOC (or other general indicator) and low synthetic organic contamined water sources would be affected later if adjustments in the standards were deemed appropriate. However, this approach assumes that the naturally occurring substances in water are normally less hazardous than the others, which may be true in general, but much more analytical and health effects work must be performed to determine if that indeed is the case. In addition, the definition and application of the distinction between natural and synthetic chemicals would be difficult in practice.

II. *Designated Treatment Technology to Control Either Specific Contaminants (e.g. Trihalomethanes) or Total Organics.*

Monitoring for a number of organic MCL's might be infeasible, and moreover, the MCL approach might not encompass all possible components of the problems. The SDWA allows EPA to establish treatment techniques requirements if it is not feasible to monitor for a given contaminant. Thus, a treatment technique requirement would prescribe one or more available technologies that public water systems must apply instead of meeting particular MCL's.

For example, methods are available to analyze for trihalomethanes in water, however, other products of chlorination may be much more difficult to quantify. The formation of chloroform can be avoided or reduced in water by certain chlorination process modifications, use of absorbants such as GAC prior to chlorination or by using an alternate disinfectant such as ozone or chlorine dioxide instead of chlorine. Unless an absorbant was being used, the concentrations of other organic contaminants would not be materially affected, except for the by-product of reaction with the disinfectants.

A treatment regulation for control of total organics would probably require the use of an adsorbant. The operation of the process would probably be monitored by the breakthrough of some general organics parameter (e.g. NPTOC), or of some indicator chemical (e.g. chloroform). Such a technology requirement could be applied to public water systems in a phased manner based on treatment plant size. A schedule could be selected such that utilities of greater than 100 MGD could be affected initially in amended Interim Primary Regulations and smaller systems could be included later on a prescribed schedule (e.g. 100 MGD by June 1977, 50 MGD by June 1978, 10 MGD by June 1979 etc.).

Treatment would not necessarily have to be in place on the effective date of the regulation. States with primary enforcement responsibility or EPA

could grant the variances if the water system could demonstrate that its water supply did not contain harmful quantities of organics. Conditions for granting variances probably would involve a survey of discharges into the source water and detailed organic analyses, and so enable a State to make an essentially "case by case" determination. Thus, in an area frequently sprayed by a particular pesticide or subjected to particular discharges these individual problems could be considered.

Exemptions for a limited period could also be granted from treatment techniques upon a showing of necessity (e.g. to install equipment or to raise the necessary capital), and that the exemption would not result in an undue risk to health.

Processing variance or exemption requests is an administrative burden under either an MCL or treatment technique approach. Somewhat fewer than 700 water utilities have an average flow of 10 MGD or greater. Of these, nearly 300 use ground water as the source. By phasing in a treatment technique requirement for plants over 10 MGD before 1980, the States or EPA would be able to carefully process applications for variances and exemptions and benefit the largest population segments initially, and it would become more feasible for public water systems to construct or develop the necessary technologies. Subsequently, smaller plants could also be required to adopt a treatment technique for organics.

During the phase-in period, the smaller systems which were not yet affected by the treatment requirement could be required to meet one or more MCL limits (eg. chloroform). Thus some level of protection would be available immediately in all cases.

Several treatment technique possibilities involving both specific contaminants and total organics are described below.

A. *Modify the chlorination process.* Chlorination is currently the principal method of disinfection of water supplies and it is the major line of defense against waterborne disease caused by bacterial and viral contamination. EPA has been actively examining alterations in the chlorination process to find ways of reducing the amount of trihalomethanes that are produced. Although of questionable biocidal value, addition of ammonia following chlorination is also a way that eliminates the chlorine that would be available for further reaction with organic compounds.

It appears that changes in the point of application of chlorine can significantly reduce the quantity of chlorine applied and the amounts of trihalomethanes and other chlorinated organics generated in some systems using filtration of source waters which contain the natural organics precursors. For example, the common practice of prechlorination of raw surface water to insure adequate disinfection is likely to produce greater quantities of trihalomethanes, compared to chlorination after the water has been coagulated and settled, resulting in some chloroform precursor removal. For this reason, EPA has been critically reviewing chlorination practices to see if

simple modifications (such as ceasing raw water chlorination in favor of chlorination just prior to filtration) can be made that would minimize the formation of chloroform yet still provide microbiologically safe drinking water. Initial results have been promising.

The discontinuation of raw water chlorination would be easily accomplished by most water utilities, and it could be done at low cost since additional equipment would usually not be needed and chlorine use would decline. Modifying the chlorination process could avoid causing a sudden demand for water treatment chemicals and not further tax limited energy resources. Discontinuation of raw water chlorination would not completely eliminate chloroform from the finished water, so some continued risk from chloroform exposure would exist.

Ceasing the disinfection of the raw water could possibly result in poorer microbiological quality in the finished water, so increased microbiological monitoring might be necessary. EPA is currently examining the practicality of this approach in a limited number of water systems (vide supra Non Regulatory Option B). Results will be released as they become available.

B. *Use of alternate disinfectants.* The principal source of chloroform in drinking water is the chemical interaction of chlorine added for disinfection with common humic substances formed from the nautral decomposition of vegetation. One possible way of avoiding the formation of trihalomethanes would be to substitute other disinfectants such as chlorine dioxide or ozone for chlorine, where possible. Any action to control chloroform and other organics in drinking water must not increase risk of waterborne disease by reducing the level of protection or by introducing other unknown risks (eg. from chemical by-products of other disinfection processes).

a. *Chlorine dioxide.* Because of its oxidizing properties, chlorine dioxide has been used to some extent for taste and odor control, but because of its cost it is not widely used in water treatment practice for disinfection. Some studies have shown that disinfection by this method is satisfactory and that a residual can be maintained to insure against bacteriological contamination in the distribution system. The problem with using chlorine dioxide now, is that our present knowledge is lacking regarding the products of its interaction with organic chemicals in water and on the possible toxicity of the inorganic ions that it generates.

Installation of a unit for chlorine dioxide generation would not be particularly costly or complicated relative to chlorination. Disinfection cost should average between 1 and 2 cents per 1000 gallons in larger systems, and about 3 cents per 100 gallons for a 1 MGD plant, when chlorine dioxide is generated from sodium chlorite.

b. *Ozonation.* Ozone is a strong disinfectant but has the disadvantage of not producing a disinfectant residual to carry throughout the water system. Thus chlorine, chlorine dioxide or chloramines might have to be used following ozone. Ozonation of drinking water is practiced in several hundred

systems throughout the world. However, little is known about the by-products of ozonation of chemicals in water.

In general, both substitute treatment methods have the advantage of reducing the health risk of exposure of chlorinated compounds. Most of the disadvantages associated wtih implementing this option are due to a lack of complete information as to the doses required for disinfection, the reactions involved, possible toxic organic by-products of these reactions and the operational experience needed. In addition there is the question of availability of equipment, chemicals, electric power and operators; particularly with ozone.

Replacement of a chlorination unit with ozone would require installation of ozonators, but the average cost for larger systems >10 MGD is not expected to exceed 1 cent per 1000 gallons. For a 1 MGD plant, ozone would cost about 4 to 5 cents per 1000 gallons compared to 3 cents per 1000 gallons for chlorination. If post-chlorination were necessary after ozonation the cost would be additive.

C. *Granular activated carbon to remove organic chemicals.* The best method yet developed for removing environmental organic contaminants such as pesticides and aromatics from water is the use of adsorbants such as granular activated carbon. GAC is also capable of removing trihalomethanes and their precursors. Installation and proper operation of GAC would affect the concentration of a large number of chemicals in water.

The simplest, although perhaps not ultimately the most efficient, approach applying GAC in systems already practicing filtration would be replacement of the present media with GAC in the existing filter to a depth of at least 30 inches. Preliminary estimates indicate that approximtaely 10 tons of GAC per MGD capacity would be needed. Chemical breakthrough rates and consequent carbon reactivation frequencies have not yet been established in large scale operations. Monitoring for chloroform breakthrough is a possible process control indicator, because chloroform is commonly present in chlorinated water and rather weakly bound to GAC. Monitoring frequencies would have to be at least weekly, particularly in the later stages of use nearing the reactivation time. If organic removal is to be maximized, the adsorbant might require renewal when the NPTOC or TOC concenrtation in the effluent exceeded 0.1 or 0.2 mg/l.

Deeper and/or countercurrent beds of GAC may be more cost effective in the long run but time for redesign and construction would delay implementation. Since reactivation of GAC is essential to its operation, another limiting factor in the short run is the almost complete lack of vailable reactivation facilities in appropriate locations.

More sophisticated operation and monitoring of GAC filtration would require personnel and apparatus not commonly available at this time. Studies are not sufficiently advanced currently to determine the exact length of operation before the activated carbon needs to be reactivated for a wide

variety of chemicals. GAC can, however, lose its effectiveness for general organic carbon removal after a few weeks as evidenced by an increase of NPTOC values in the effluent with time, although some substances such as polynuclear aromatics are effectively adsorbed. Current experience indicates that the effective life of GAC for the removal of trihalomethane precursors is somewhat limited; a one month regeneration frequency has been generally assumed.

Costs will vary widely depending on factors such as labor costs, carbon costs, reactivation frequency, carbon loss due to attrition, and system size, average production to design size ratio, and power costs. Therefore, it is difficult to predict costs for specific systems. For a 1 MGD plant with 1 month reactivation frequency the cost might be more than 10 cents per 1000 gallons, but for large systems (over 100 MGD) between 5 and 7 cents per 1000 gallons. This assumes that sand in existing filters is to be replaced with GAC.

Post treatment for larger systems is slightly less expensive, but installation time would result in a substantial lag time before widespread compliance. Cost-wise the most serious problems appear to be with plants under 1 MGD. The problem could be much more serious if conventional filtration units would have to be constructed. Costs for smaller systems could be mitigated somewhat if joint regeneration facilities could be used.

Some constraints may exist to prevent immediate and widespread installation of GAC treatment. Despite excess GAC production capacity available at present, industry might not be able to supply enough GAC needed for potable water treatment in the short run, if a rapid increase in demand occurred. Questions of whether or not enough regeneration furnaces can be produced quickly is a serious concern.

Some of the principal problems facing EPA in the control of the quality of the Nation's drinking water are pressing the limits of current research capabilities in health, science and technology. Regulatory decisionmaking is further compounded by the dearth of definitive information, the lack of agreement within the scientific community on many questions, and minimal data on costs and impacts of changes in current technology at the national, State, and local levels.

PESTICIDES

A. *Chlorinated Hydrocarbon Insecticides*

The chlorinated hydrocarbons are one of the most important groups of synthetic organic insecticides because of their wide use, great stability in the environment, and toxicity to mammals and insects. When absorbed into the body, some of the chlorinated hydrocarbons are not metabolized rapidly but are stored in the fat.

As a general group of insecticides, the chlorinated hydrocarbons can be absorbed into the body through the lungs, the gastro-intestinal tract, or the skin. The symptoms of poisoning, regardless of the compound involved or the route of entry, are similar but may vary in severity. Mild cases of poisoning are characterized by headache, dizziness, gastro-intestinal disturbances, numbness and weakneess of the extremities, apprehension, and hyperirritability. In severe cases, there are muscular fasciculations spreading from the head to the extremities, followed eventually by spasms involving whole muscle groups, leading finally to convulsions and death from cardiac or respiratory arrest. The severity of symptoms is related to the concentration of the insecticides in the nervous system, primarily the brain (1).

Criteria Based on Chronic Toxicity

Except as noted below, the approval limits (AL's) for chlorinated hydrocarbons in drinking water have been calculated primarily on the basis of the extrapolated human intake that would be equivalent to that causing minimal toxic effects in mammals (rats and dogs). Table I lists the levels of several chlorinated hydrocarbons fed chronically to dogs and rats (2, 3, 4) that produced minimal toxicity or no effects.

For comparison, the dietary levels are converted to mg/kg body weight/day. Endrin and lindane had lower minimal effect/no-effect levels in dogs than in rats; whereas, for toxaphene and methoxychlor the converse was observed.

Human studies have also been conducted for methoxychlor, although they were of short duration (8 weeks). The highest level tested for methoxychlor was 2 mg/kg/day (5). No illness was reported in these subjects.

Such data from human and animal investigations may be used to derive exposure standards, as for drinking water, by adjusting for factors that influence toxicity such as inter- and intra- species variability, length of exposure, and extensiveness of the studies. To determine a "safe" exposure level for man, conventionally a factor of 1/10 is applied to the data derived from human exposure studies conducted longer than 2 months at which no effects have been observed; whereas, a factor of 1/100 is applied to data derived from human exposure studies conducted for 2 months or less as is the case for the human methoxychlor data cited. A 1/100 factor is applied to animal data when adequate human data are available for corroboration and a factor of 1/500 is generally used on animal data when no adequate

and comparable human data are available. The minimal effect levels of endrin, lindane, and toxaphene are adjusted by 1/500 since no adequate data are available for comparison. These derived values are considered the maximum safe exposure levels from all sources. Since these values are expressed as mg/kg/day, they are then readjusted for body weight to determine the total quantity to which persons may be safely exposed.

Analysis of the maximum safe levels (mg/man/day) reveals that these levels are not exactly the same when one species is compared with another. The choice of a level on which to base an AL for water requires the selection of the lowest value from animal experimentation, provided that the human data are within the same order of mangnitude. Thus the human data should substantiate the fact that man is no more sensitive to a particular agent than is the rat or the dog.

To set a standard for a particular medium necessitates that account be taken for exposure from other media. In case of the chlorinated hydrocarbons, exposure is expected to occur mostly through the diet. Occasionally, aerial sprays of these agents will result in their inhalation. Dietary intake of pesticide chemicals has been determined by the investigations of the Food and Drug Administration from "market basket" samples of food and water. Duggan and Corneliussen (6) report on this activity from 1964-1970. The average dietary intakes (mg/man/day) are listed in Table I. Comparing the intake from the diet with what are considered acceptable safe levels of these pesticides, it is apparent that only traces of methoxychlor and toxaphene are present in the diet. Less than 10% of the maximum safe level of endrin or lindane are ingested with the diet.

The AL's for chlorinated hydrocarbon insecticides reflect only a portion of man's total exposure to the compounds. In general, 20% of the total acceptable intake is taken to be a reasonable apportionment to water. However, the AL for toxaphene was lowered because of organoleptic effects (7, 8) at concentrations above 0.005 mg/l.

The approval limits for the chlorinated hydrocarbon insecticides are listed in Table I. These limits are meant to serve only in the event that these chemicals are inadvertently present in the water. Deliberate addition of these compounds is neither implied nor sanctioned.

TABLE I. Derivation of Approval Limits (AL's) for Chlorinated Hydrocarbon Insecticides

		Lowest Long-Term Levels With Minimal or no Effects		Calculated Maximum Safe Levels			Intake from Diet		Water	
Compound	Species	ppm in diet	mg/kg body weight/day[a]	Safety Factor (X)	mg/kg/day	mg/man/day[b]	mg/man/day (6)	% of Safe Level	% of Safe Level	Recommended MAL (mg/l)[c]
Endrin	Rat	5.0(3)	0.83	1/500	0.00166	0.1162				
	Dog	1.0(3)	0.02	1/500	0.00004	0.0026[d]	0.00035	4.1	20	0.0002
	Man	N.A.	N.A.	—	—	—				
Lindane	Rat	50.0(2)	8.3	1/500	0.0166	1.162				
	Dog	15.0(2)	0.3	1/500	0.0006	0.042[d]	0.0035	8.3	20	0.004
	Man	N.A.	N.A.	—	—	—				
Methoxychlor	Rat	100.0(2)	17.0	1/100	0.17	11.9				
	Dog	4000.0(2)	80.0	1/100	0.3	21.0	T	T	20	0.1
	Man	—	2.0(5)	1/100	0.02	1.4[d]				
Toxaphene	Rat	10.0(2)	1.7	1/500	0.0034	0.238[d]	T	T	20	(0.025)[f]
	Dog	400.02(2)	8.0	1/500	0.016	1.12				0.005[e]
	Man	N.A.	N.A.	—	—	—				

[a]Assume weight of rat =0.3 kg and of dog =10 kg; assume average daily food consumption of rat =0.05 kg and of dog =0.2 kg.

[b]Assume average weight of human adult =70 kg.

[c]Assume average daily intake of water for man =2 liters.

[d]Chosen as basis on which to derive MAL.

[e]Adjusted for organoleptic effects.

[f]Calculated MAL in parentheses.

NA—no data available.

T--infrequent occurrence in trace quantities.

Criteria Based on Potential Carcinogenicity

To establish AL's for Compounds such as DDT, aldrin, and dieldrin, a different method for deriving AL's must be used, since DDT, aldrin, and dieldrin might represent a potential carcinogenic hazard to humans, based on experiments with rats and mice. (9, 10, 11, 12). Aldrin is readily converted to dieldrin by animals, soil microorganisms, and insects, and thus the potential carcinogenicity of aldrin will be considered to be equivalent to that of dieldrin (13).

It is recognized that scientists have yet to determine if there is any level of exposure to chemical carcinogens that is completely free of risk of cancer. For the purpose of setting standards we will assume that the risk of inducing cancer decreases with decreasing dose. Thus, the limits for these possible carcinogens could be derived by estimating the health risk associated with various concentrations and comparing these concentrations with ambient levels to assess the attainability of the proposed limits with presently known means of technology.

Risk estimates at very low levels of exposure are subject to great uncertainties. Extrapolation techniques such as the "one-hit" model and the Mantel-Bryan use of the probit model (14) are being intensively reviewed by several agencies of the federal government.

Aldrin-Dieldrin

Experiments carried out on mice (strain CF1) fed dieldrin in their daily diet, at levels varying from 0.1 to 20 ppm during their normal life span, resulted in significant increases in the incidence of liver tumors (11). The results of this studp appear to be, at present, the most appropriate for calculating the risk associated with a range of concentrations of dieldrin in drinking water.

DDT:

Although earlier studies of the carcinogenic effect of DDT have yielded generally negative results, three recent studies in experimental animals conflict with these previous findings. Using tumor-susceptible hybrid strains of mice, Innes at al (15) produced significantly increased incidences of tumors with the administration of large doses of DDT (46.4 mg/kg/day). In a separate study in mice extending over five generations, a dietary level of 3 ppm of DDT produced a greater incidence of leukemia and malignancies beginning with the F2 and F3 generations (16).

More recent information (12) on the effect of DDT on long-term exposure in mice indicated a higher incidence of liver tumors in the treated population. CF-1 minimal inbred mice were given technical DDT mixed into the diet at the dose levels at 2, 10, 50 and 250 parts per million (ppm) for the entire life span for two consecutive generations. Exposure to all four levels of DDT resulted in a significant increase of liver tumors in

males, this being most evident at the highest level used. In females, the incidence of liver tumors was slightly increased following exposure to 250 ppm. In DDT-treated animals the liver tumors were observed at an earlier age than in untreated controls. The age at death with liver tumors and the incidence of liver tumors appear to be directly related to the dose of DDT to which the mice were exposed. Four liver tumors, all occurring in DDT-treated mice, gave metastases. Histologically, liver tumors were either well-differentiated nodular growths, pressing but not infiltrating the surrounding parenchyma, or nodular growths in which the architecture of the liver was obliterated showing glandular or trabecular patterns. The results of this study appear to be, at present, the most appropriate to use as a basis for extrapolating the risk associated with a range of concentrations of DDT in drinking water.

Chlordane and Heptachlor

Because recent evidence also implicates chlordane and heptachlor as potential carcinogens, establishment of limits for these pesticides must be based on considerations similar to those for aldrin, dieldrin and DDT.

* * *

A national survey for aldrin, dieldrin and DDT in drinking water was carried out during 1975. A total of 715 samples of raw and finished drinkink water were analyzed for the presence of aldrin, dieldrin, DDT, and DDT metabolites. Dieldrin was found in 94 samples at concentration levels of 4 ppt (minimum level of detection) to 10 ppt; 13 with levels of 11-20 ppt; 4 with levels of 21-29 ppt; and 6 with levels from 56-110 ppt. These 6 samples represented 3 raw and 3 finished waters from one location. Of these 6 samples, 3 also contained aldrin with concentrations between 15-18 ppt. DDT at levels between 10-28 ppt was found in 6 other dieldrin-containing samples. DDT only at 15 and 32 ppt was found in 2 samples. Based on the initial data, 30 "high potential" samples were selected and analyzed for chlordane, heptachlor and heptachlor epoxide. None of them were found above the detection limits of 5 ppt, 10 ppt and 5 ppt respectively. Note that these ambient levels where measurable are approximately one ten thousandths of the amounts that were employed in the animal tests described above.

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B. *Chlorophenoxy Herbicides*

Aquatic weeds have become substantial problems in the U.S. in recent years, and chemical control of this vegetation has won wide acceptance. Since waters to which applications of herbicides are made are sometimes employed as raw water sources of drinking water, there is the possibility that herbicides may enter potable source water. Consequently, a standard is needed for the more extensively used herbicides so as to protect the health of the water consumer.

Two widely used herbicides are 2, 4-D (2, 4-dichlorophenoxyacetic acid) and 2, 4, 5-TP (silvex) [2-(2, 4, 5-trichlorophenoxy) propionic acid]. [A closely related compound, 2, 4, 5-T (2, 4, 5-trichlorophenoxyacetic acid) had been extensively used at one time, but has been banned for major aquatic uses.] Each of these compounds is formulated in a variety of salts and esters that may have a marked difference in herbicidal properties, but all of which are hydrolyzed rapidly to the corresponding acid in the body.

The acute toxicity following oral administration to a number of experimental animals is moderate. Studies (1-4) of the acute oral toxicity of the chlorinated phenoxyalkyl acids indicate that there is approximately a threefold variation between the species of animals studied. It appears that acute oral toxicity of the three compounds is of about the same magnitude within each species (e.g., in the rat, an oral LD of about 500 mg/kg for each agent).

The subacute oral toxicity of chlorophenoxy herbicides has been investigated in a number of species of experimental animals (1-6). The dog was the most sensitive species studied and often displayed mild injury in response to doses of 10 mg/kg/day for 90 days, and serious effects from a dose of 20 mg/kg/day for 90 days. Lehman (6) reported that the no-effect level of 2, 4-D is 50* mg/kg/day in the rat, and 8.0 mg/kg/day in the dog.

Although 2, 4, 5-T has been banned for all aquatic uses there is considerable interest as to why this action was taken, so for informational purposes, a discussion of the toxicity of this herbicide is included. In a study of various pesticides and related compounds for teratogenic effects, Courtney, et al. (7) noted terata and embryotoxicity from 2, 4, 5-T. These effects were evidenced by statistically increased proportions of litters affected and of abnormal fetuses within the litters (notably, cleft palate and cystic kidneys). Effects were noted in both mice and rats, although the rat appeared to be more sensitive to this effect. A dosage of 21.5 mg/kg produced no harmful effects in mice, while a level of 4.6 mg/kg caused minimal, but statistically significant, effects in the rat. More recent work (8) has indicated that a contaminant (2, 3, 7, 8-tetrachlorodibenzo-p-dioxin) which was present at approximately 30 ppm in the 2, 4, 5-T formulation originally tested was highly toxic to experimental animals and produced fetal and maternal toxicity at levels as low as 0.005 mg/kg. However, purified 2, 4, 5-T has also produced teratogenic effects in both hamsters and rats at relatively high dosage rates (9). Current production samples of 2, 4, 5-T that contain less than 1 ppm of dioxin did not produce embryotoxicity or terata in rats at levels as high as 24 mg/kg/day (10).

The subacute and chronic toxicity of 2, 4, 5-TP has been studied in experimental animals (11). The results of 90-day feeding studies indicate that the no-effect levels of the sodium and potassium salts of 2, 4, 5-TP are 2 mg/kg/day in rats and 13 mg/kg/day in dogs. In 2-year feeding studies with these same salts, the no-effect levels were 2.6 mg/kg/day in rats and 0.9 mg/kg/day in dogs.

Some data are available on the toxicity of 2, 4-D to man. A daily dosage of 500 mg (about 7 mg/kg) produced no apparent ill effects in a volunteer

*In the March 14, 1975, issue of this document, this figure was erroneously written as 0.5.

over a 21-day period (12). When 2, 4-D was investigated as a possible treatment for disseminated coccidioidomycosis, the patient had no side effects from 18 intravenous doses during 33 days; each of the last 12 doses in the series was 800 mg (about 15 mg/kg) or more, the last being 2000 mg (about 37 mg/kg) (13). A nineteenth and final dose of 3600 mg (67 mg/kg) produced mild symptoms.

The acute oral dose of 2, 4-D required to produce symptoms in man is probably 3000 to 4000 mg (or about 45 to 60 mg/kg). A comparison of other toxicity values for 2, 4, 5-TP indicates that the toxicity of these two agents is of the same order of magnitude. Thus, in the absence of any specific toxicologic data for 2, 4, 5-TP in man, it might be estimated that the acute oral dose of 2, 4, 5-TP required to produce symptoms in man would also be about 3000 to 4000 mg.

In addition to these specific data, the favorable record of use experience of 2, 4-D is also pertinent. Sixty-three million pounds of 2, 4-D were produced in 1965 while there were no confirmed cases of occupational poisoning and few instances of any illness due to ingestions (14, 15). One case of 2,4-D poisoning in man has been reported by Berwick (16).
Berwick (16).

Table 1 displays the derivation of the approval limits for the two chlorophenoxy herbicides most widely used. The long-term no-effect levels (mg/kg/day) are listed for the rat and the dog. These values are adjusted by 1/500 for 2, 4-D and 2, 4, 5-TP. The safe levels are then readjusted to reflect total allowable intake per person. Since litle 2, 4-D or 2, 4, 5-TP are expected to occur in foods, 20% of the safe exposure level can be reasonably allocated to water without jeopardizing the health of the consumer.

The approval limits for these herbicides are meant to serve in the event that these chemicals inadvertently occur in the water. Deliberate addition of these compounds to drinking water sources is neither implied nor sanctioned.

TABLE I. *Derivation of Approval Limits (AL) for Chlorophenoxy Herbicides*

		Lowest Long-Term Levels with Minimal or No Effects	Calculated Maximum Safe Levels From all Sources of Exposure			Water	
Compound	Species	mg/kg/day[a]	Safety Factor (X)	mg/kg/day	mg/man/day[b]	% of Safe Level	AL (mg/l)[c]
2, 4-D	Rat	50 (6)	1/500	0.1	7.0		
	Dog	8.0 (6)	1/500	0.016	1.12[d]	20	0.1
2, 4, 5-TP	Rat	2.6 (12)	1/500	0.005	0.35		
	Dog	0.9 (12)	1/500	0.002	0.14[d]	20	0.01

[a]Assume weight of rat =0.3 kg and of dog =10 kg; assume average daily food consumption of rat =0.05 kg and of dog =0.2 kg.

[b]Assume average weight of human adult =70 kg.

[c]Assume average daily intake of water for man =2 liters.

[d]Chosen as basis on which to derive AL.

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SELENIUM

The 1962 Drinking Water Standards Committee lowered the limit for selenium in drinking water primarily out of concern over the possible carcinogenic properties of the element. Data supporting the carcinogenicity of selenium has not been forthcoming, and more recent findings concerning the nutritional requirement for selenium has required a comprehensive review of the data available concerning the toxicity of selenium and its compounds.

The controversy over the present limits of selenium acceptable in the environment is largely the result of the demonstration by Schwartz and Foltz (2) that the element was an integral part of "factor 3," recognized for some time as essential in animal nutrition. While definite evidence is still lacking for a nutritional requirement for selenium in man, certain cases of protein-resistant kwashiorkor have been shown to be responsive to administration of the element (3).

Consideration of a maximal concentration of selenium allowable in drinking water is further complicated by the many secondary factors known to affect both the efficacy of selenium in alleviating deficiency syndromes and the intakes associated with toxicity. The chemical form of selenium (4), the protein content of the diet (5), the source of dietary protein (6), the presence of other trace elements (1, 7, 8), and the vitamin E intake (9, 10, 11) all affect the beneficial and/or adverse effects of selenium in experimental animals. The fact that these interactions are not simple is illustrated by the comments of Frost (1) on the well-known antagonism of arsenic in selenium toxicity (1, 7, 8, 12). He has found that arsenic in drinking water accentuates the toxicity of selenium in drinking water in contrast to the protective effect of arsenic seen when selenium was administered via the diet. Consequently, when considering "safe" levels of selenium in drinking water, consideration must also be given to the variability in these other factors which are certain to occur in any given population.

The current limit of 0.01 mg/liter of selenium in drinking water is based on the total selenium content. No systematic investigation of the forms of selenium in drinking water sources with excessive concentrations has ever been carried out. Since elemental selenium must be oxidized to selenite or selenate before it has appreciable solubility in water (13), one would predict that these would be the principal inorganic forms that occur in water. Organic forms of selenium occur in seleniferous soils and have sufficient mobility in an aqueous environment to be preferentially absorbed over selenate in certain plants (14). However, the extent to which these compounds might occur in source waters is essentially unknown.

There is considerable difficulty involved in determining what the required level and toxic levels of selenium intake in humans might be. The basic problem is that dietary selenium includes an unknown variety of selenium compounds in varying mixtures. Toxicologic examination of plant sources of

selenium has revealed that selenium present in seleniferous grains is more toxic than inorganic selenium added to the diet (16). Although there is a fairly extensive literature on industrial exposures to selenium (see Cerwenka and Cooper, 1961 (17), and Cooper, 1967 (18) for reviews of this subject), the results do not apply well to environmental exposures since the only studies that made an attempt to document systemic absorption involved elemental selenium (19). Elemental selenium is virtually non-toxic to plants and animals that have been shown to be very sensitive to the water soluble forms of selenium.

Only one documented case of human selenium toxicity for a water source uncomplicated with selenium in the diet has been reported (21). Members of an Indian family developed loss of hair, weakened nails, and listlessness after only 3 months' exposure to well-water containing 9 mg/l. The children in the family showed increased mental alertness after use of water from the seleniferous well was discontinued, as evidenced by better work in school (22).

Smith and co-workers (23, 24) reported the results of their studies dealing with human exposure to high environmental selenium concentrations in the 1930's. They reported a high incidence of gastrointestional problems, bad teeth, and an icteriod skin color in seleniferous areas. The individuals exhibiting these symptoms had urinary selenium levels of 0.2-1.98 ug/liter as compared to the 0.0-0.15 ug/liter that Glover (19) indicates to be the normal range. The gastrointestinal disturbances and the icteriod discoloration of the skin apparently have their counterparts in the anorexia (23) and bilirubinemia (7), respectively, in rats fed selenium. The effect of selenium on teeth has had some marginal documentation in rats (26); and has been supported by Hadjimarkos (27) and refuted by Cadell and Cousins (28) in epidemiologic studies.

From urinary concentrations of selenium, Smith and Westfall (24) estimated that the individuals displaying these symptoms were ingesting 0.01 to 0.10 mg/kg/day, and possibly as much as 0.20 mg/kg/day. For the 70 kg man, this would amount to a daily intake of 700 to 7000 ug/day. Smith (24, 29) also presented the range of selenium concentrations found in various food classes in the areas in which the field studies had been conducted. With the use of the table provided in *Dietary Levels of Households in the U.S., Spring 1965* (U.S.D.A. Agri. Res. Service), calculations from these data result in a range of intake of 600-6300 ug/day, very close to the estimates made from urinary concentrations of selenium. These intakes of selenium correspond in the main with the levels producing adverse effects in other mammalian species. Tinsley et al. (25) found that an intake of 0.125 mg/kg/day adversely affected early growth in rats. 1.1 mg/kg, administered twice weekly (ca. 0.3 mg/kg/day), has been found to adversely affect growth and to increase mortality in Hereford steers (30). Mortality in ewes was increased at 0.825 mg/kg/day. The steers were administered

sodium selenite; the ewes sodium selenate. Although these levels are slightly higher than those reported for the human exposures, it must be remembered that the parameters measured would not be acceptable either in terms of severity or incidence in the human population.

Few studies have been performed to specifically examine the toxicity of selenium administered in drinking water. Pletnikova (31) found the rabbit to be very sensitive to selenium as selenite. Ten ug/l in drinking water resulted in a 40% reduction in the elimination of bromosulphalein by the liver. Since no apparent consideration was given to the selenium content of the diet of these animals, the meaning of this result in terms of liver function is obscure. If the sole intake of selenium were from the water in these studies, the controls had to be deficient and the experimental group marginal, at best, in terms of the dietary requirement for selenium. The duration of the study was 7½ months. Schroeder (32) has indicated that intake of selenite from drinking water is more toxic than when mixed with food. However, this suggestion was not based on a direct experimental comparison. Rosenfeld and Beath (33) studied the effects of sodium selenate in drinking water on reproduction in rats. Selenium concentrations of 2.5 mg/l reduced the number of young reared by the second generation of mothers, and 7.5 mg/l prevented reproduction in females.

Early work (34), using both naturally occurring, and a selenide salt, indicated the formation of adenomas and low-grade non-metastasizing hepatic cell carcinomas in 11 of 53 rats surviving 18 months of diets containing selenium. Harr et al. (24), in a much more extensive study using selenite and selenate salts, found no evidence of neoplasms that could be attributed to the addition of these selenium compounds to the diet at 0.5 - 16 ppm. Volganev and Tschenkes (35) negated their earlier results, which had indicated that 4.3 mg/l selenium as selenite in the diet gave rise to tumors, but had not used proper controls. It should be noted that these studies are not a direct negation of the earlier studies implicating selenium as a carcinogen, since entirely different compounds of selenium were used in the early work. Consequently, the possibility that other compounds of selenium, besides selenite, possess carcinogenic properties cannot be strictly ruled out. The carcinogenic properties of selenium are further complicated by recent reports of the effectiveness of selenium, 1 mg/l (as selenite), in reducing papillomas induced by various chemicals in mice (36).

Any consideration of a maximum allowable concentration of selenium must include the evidence that the element is an essential dietary requirement. A range of 0.04 to 0.10 mg/l in the diet is considered adequate to protect animals from the various manifestations of selenium deficiency (10, 37, 38). Using the recent data on Morris and Levander (39), an estimate of the present average daily intake of selenium by the American population may be calculated. This figure approximates 200 ug/day and some variation around this figure would be anticipated primarily as the result

of individual preferences, particularly in meats. Since no deficiency diseases of selenium have been reported to date in the U.S., it may be assumed that 200 ug/day of selenium is nutritionally adequate.

Signs of selenium toxicity have been seen at an estimated level of selenium intake of 0.7-7 mg/day according to the data of Smith et al. (23, 24). At the present limit on selenium content of drinking water, water would increase the basal 200 ug/day intake of selenium by only 10%, if one assumes a 2-liter ingestion of water per day. This results in a minimum safety factor of 3, considering the lower end of the range of selenium intakes that have been associated with minor toxic effects in man. In view of the relative scarcity of data directly applicable to the apparent small margin of safety brought about by selenium contained in the diet, selenium concentrations above 0.01 mg/liter shall not be permitted in the drinking water.

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SILVER

The need to set a water standard for silver (Ag) arises from its intentional addition to waters as a disinfectant. The chief effect of silver in the body is cosmetic. It consists of a permanent blue-grey discoloration of the skin, eyes, and mucous membranes which is unsightly and disturbing to the observer as well as to the victim. The amount of colloidal silver required to produce this condition (argyria, argyrosis), and to serve as a basis of determining the water standard, in not known, however, but the amount of silver from injected Ag-arsphenamine, which produces argyria is precisely known. This value is any amount greater than 1 gram of silver, 8g Ag-arsphenamine, in an adult (1, 2).

From a review (2) of more than 200 cases of argyria, the following additional facts were derived. Most common salts of silver produce argyria when ingested or injected in sufficient doses. There is a long-delayed appearance of discoloration. No case has been uncovered that has resulted from an idiosyncrasy to silver. There was, however, considerable variability in predisposition to argyria; the cause of this is unknown, but individuals concurrently receiving bismuth medication developed argyria more readily. Although there is no evidence that gradual deposition of silver in the body produces any significant alteration in physiologic function, authorities are of the opinion that occasional mild systemic effects from silver may have been overshadowed by the striking external changes. In this connection, there is a report (3) of implanted silver amalgams resulting in localized argyria restricted to the elastic fibers and capillaries. The histopathologic reaction resembled a blue nevus simulating a neoplasm with filamentous structures and globular masses. Silver affinity for elastic fibers had been noted a half-century earlier (5).

A study (5) of the metabolism of silver from intragastric intake in the rat, using radio-silver in carrier-free tracer amounts, showed absorption to be less than 0.1-0.2 percent of the silver administered; but this evidence is inconclusive because of the rapid elimination of silver when given in carrier-free amounts. Further study indicated, however, that silver is primarily excreted by the liver. This would be particularly true if the silver were in colloidal form. Silver in the body is transported chiefly by the blood stream in which the plasma proteins and the red cells carry practically all of it in extremely labile combinations. The half-time of small amounts of silver in the blood stream of the rat was about 1 hour. A later report (6), using the spectrographic method on normal human blood, showed silver unmistakably in the red blood cell and questionably in the red cell ghosts and in the plasma. Once silver is fixed in the tissues, however, negligible excretion occurs in the urine (7).

A study (8) of the toxicologic effects of silver added to drinking water of rats at concentrations up to 1,000 ug/l (nature of the silver salt unstated)

showed pathologic changes in kidneys, liver, and spleen at 400, 700, and 1,000 ug/l, respectively.

A study (9) of the resorption of silver through human skin using radiosilver Ag^{111} has shown none passing the dermal barrier from either solution (2 percent $AgNO_3$) or ointment, within limits of experimental error (±2 percent). This would indicate no significant addition of silver to the body from bathing waters treated with silver.

Uncertainty currently surrounds any evaluation of the amount of silver introduced into the body when silver-treated water is used for culinary purposes. It is reasonable to assume that vegetables belonging to the family Brassicoceae, such as cabbage, turnips, cauliflower, and onions, would combine with residual silver in the cooking water. The silver content of several liters of water could thus be ingested.

Because of the evidence (7) that silver, once absorbed, is held indefinitely in tissues, particularly the skin, without evident loss through usual channels of elimination or reduction by transmigration to other body sites, and because of the probable high absorbability of silver bound to sulfur components of food cooked in silver-containing waters [the intake for which absorption was reported in 1940 to amount to 60-80 ug per day (10)], the concentration of silver in drinking water shall not exceed 0.05 mg/l.

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SODIUM

Man's intake of sodium is mostly influenced by the use of salt. Intake of sodium chloride for American males is estimated to be 10 grams per day, with a range of 4 to 24 grams (1). This would be a sodium intake of 1600 to 9600 mg per day. Intake of these amounts is considered by most to have no adverse effect on normal individuals. Even Dahl, who has been one of the strong advocates of the need for restricting salt intake, has felt that an intake of 2000 mg of sodium could be allowed for an adult without a family history of hyertension. Intake of sodium from hospital "house" diets has been measured recently (2). The sodium content of a pool of 21 consecutive meals that were seasoned by the chef or the dietitian from twenty selected general hospitals was determined each quarter. The average sodium intake per capita per day was 3625 ± 971 (SD) milligrams. The intake could be greatly changed between individuals who never add salt to the food at the table and the individuals who always add salt even before tasting.

The taste threshold of sodium in water depends on several factors (3). The predominant anion has an effect; the thresholds for sodium were 500 mg/l from sodium chloride, 700 mg/l from sodium nitrate, and 1000 mg/l from sodium sulfate. A heavy salt user had a threshold of taste that was 50 percent higher, and the taste was less detectable in cold water.

Six of 14 infants exposed to a sodium concentration of 21, 140 mg/l died when salt was mistakenly used for sugar in their formula (4). Sea water would have about 10, 000 mg/l of sodium.

Severe exacerbation of chronic congestive heart failure due to sodium in water has been documented (3). One patient required hospitalization when he changed his source of domestic water to one that had 4200 mg/l sodium. Another patient was readmitted at two-to-three-week intervals when using a source of drinking water of 3500 mg/l sodium.

Sodium-restricted diets are used to control several disease conditions of man. The rationale, complications, and practical aspects of their use were reviewed by a committee on food and nutrition of the National Research Council (5). Sodium-restrictive diets are essential in treating congestive cardiac failure, hypertension, renal disease, cirrhosis of the liver, toxemias of pregnancy, and Meniere's disease.

Hormone therapy with ACTH and cortisone is used for several diseases. Sodium retention is one of the frequent metabolic consequences following administration of these therapeutic agents, and sodium-restricted diets are required, especially for long periods of treatment. More recent medical text books continue to point out the usefulness of sodium-restricted diets for these several diseases where fluid retention is a problem (6).

When disease causes fluid retention in the body, with subsequent edema and ascites, there is a diminished urinary excretion of sodium and of water. If the sodium intake is restricted in these circumstances, further fluid retention will usually not occur, and the excess water ingested will be excreted

in the urine because the mechanisms that maintain the concentration of sodium in the extracellular fluid do not permit the retention of water without sodium.

Almost all foods contain some sodium, and it is difficult to provide a nutritionally adequate diet without an intake of about 440 mg of sodium per day from food; this intake would be from the naturally occurring sodium in food with no salt added. The additional 60 mg that would increase the intake to the widely used restricted diet of 500 mg per day must account for all non-nutrition intake that occurs from drugs, water and incidental intakes. A concentration of sodium in drinking water up to 20 mg per liter is considered compatible with this diet. When the sodium content exceeds 20 mg/l, the physician must take this into account to modify the diet or prescribe that distilled water be used. Water utilities that distribute water that exceeds 20 mg/l must inform physicians of the sodium content of the water so that the health of consumers can be protected. About 40 percent of the water supplies are known to exceed 20 mg/l and would be required to keep physicians informed of the sodium concentration (7). Most of the State health departments have made provision for determining the sodium content of drinking water on a routine basis and are now informing physicians in their jurisdiction (8). If change of source or a treatment change such as softening occurs that will significantly increase the sodium concentration, the utility must be sure that all physicians that care for consumers are aware of the impending change. Diets prescribing intakes of less than 500 mg per day must use special foods such as milk with the sodium reduced, or fruits that are naturally low in sodium.

It is not known how many persons are on sodium-restricted diets and to what extent the sodium intake is restricted. To reduce edema or swelling, the physician may prescribe a diuretic drug, a sodium-restricted diet, or a combination of the two. Therapy, of course, depends on the patient's condition, but there are also regional differences that probably result from physician training. The American Heart Association (AHA) (9) feels that diuretics may allow for less need of very restricted diets and that diurectics are necessary for quick results in acute conditions. For long-term, use, a sodium-restricted diet is simpler, safer, and more economical for the patient. It is preferable, especially when a moderate or mild sodium-restricted diet will effectively control the patient's hypertension and water retention. Literature is provided to physicians by the AHA to distribute to their patients explaining the sodium-restricted diets. These cover the "strict" restriction - 500 mg. sodium, "moderate" restriction - 1000 mg sodium, and the "mild" restricted diet-2400 to 4500 mg sodium. From 1958 through June 1971, there were 2,365,000 pieces of this literature distributed: 37% - 500 mg; 34% - 1000 mg; and 29% - "mild" (10). There are many ways a physician can counsel his patients other than using this literature, so the total distribution does not reflect the extent of the problem, but the pro-

portion of booklets distributed may provide an estimate of the portion of diets that are prescribed. The "mild" restricted diet could require just cutting down on the use of salt, and literature for the patient would not be as necessary.

The AHA estimates that hypertension affects more the 2 million Americans, and in more than half of these cases put enough strain on the heart to be responsible for the development of hypertensive heart disease (11). Congestive heart failure is a sequelae of several forms of disease that damage the heart and would affect some unknown portion of the 27 million persons with cardiovascular disease. Thus, from 21 to 27 million Americans would be concerned with sodium intake.

Toxemias of pregnancy are common complications of gestation and occur in 6 to 7 percent of all pregnancies in the last trimester (12). Thus, about 230,000 women would be very concerned with sodium intake each year. Other diseases are treated with restricted sodium intake, but no estimate can be made on the number of people involved.

Questions about salt usage were asked on the ninth biennial examination of the National Heart Institute's Framingham, Massachusetts Study (13). The study population was free of coronary heart disease when the study began in 1949 and now are over 45 years of age. There were 3,333 respondents. Forty-five percent of the males and 30 percent of the females reported that they add salt routinely to their food before tasting. But at the other extreme, 9 percent of the men and 14 percent of the women avoid salt intake. More of the people 60 and over avoid salt intake than the 45 to 59 population. It is not determined if the salt restriction was medically prescribed nor how extensively the sodium intake was restricted.

It can be seen that a significant proportion of the population needs to and is trying to curtail its sodium intake. The sodium content of drinking water should not be significantly increased for frivolous reasons. This is particularly true of locations where many of the people using the water would be susceptible to adverse health effects, such as hospitals, nursing homes, and retirement communities. The use of sodium hypochlorite for disinfection, or sodium fluoride for control of tooth decay, would increase the sodium content of drinking water but to an insignificant amount. The use of sodium compounds for corrosion control might cause a significant increase, and softening by either the base exchange or lime-soda ash process would significantly increase the sodium contentof drinking water. For each milligram per liter of hardness removed as calcium carbonate by the exchange process, the sodium content would be increased about one-half mg per liter. The increase in excess lime softening would depend on the amount of soda ash added. A study in North Carolina found that the sodium content of 30 private well-water supplies increased from 110 mg/l to 269 mg/l sodium on the average after softening (14). The sodium content of the softened water was much higher shortly after the softener had been regen-

erated than later in the cycle. A case has been reported where a replacement element type softener was not flushed, and the drinking water had a sodium content of 3,700 mg/l when the unit was put back in service.

As a further deterrent to softening of water, it should be noted that there is considerable evidence of an inverse relationship between water hardness and certain cardiovascular diseases. Research in the area is being accelerated to determine cause and effect relationships. Until the full significance of water hardness is known, and because of the increase in sodium content of softened waters, utilities should carefully consider the consequences of installing softening treatment.

All consumers could use the water for drinking if the sodium content was kept below 20 mg per liter, but about 40 percent of the U.S. water supplies have a natural or added sodium content above this concentration (7). Many industrial wastes and runoff from deiced highways may increase the sodium pollution of surface water (15). The problem is most acute when ground water is polluted with sodium (16, 17) because it remains for a long time. Removal of sodium from water requires processes being developed by the Office of Saline Water (18) and are economically feasible only in certain situations.

The person who is required to maintain a restricted sodium intake below 500 mg per day can use a water supply that contains 20 mg or less sodium per liter. If the water supply contains more sodium, low sodium bottled water or specially treated water will have to be used. In the moderately restricted diet that allows for a consumption of 1000 mg sodium per day the food intake is essentially the same, but the diet is liberalized to allow the use of 1/4 teaspoon of salt, some regular bakery bread, and/or some salted butter. If persons on the moderately restricted diet found it necessary to use a water with a significant sodium content they could still maintain their limited sodium intake with a water containing 270 mg/liter. This would require allocating all the liberalized intake to water (the original 20 mg/l and 250 mg/l more with two liter domestic use, drinking or cooking, per day). High sodium in water causes some transfer of sodium to foods cooked in such water (5).

It is essential that the sodium content of public water supplies be known and this information be disseminated to physicians who have patients in the service area. Thus, diets for those who must restrict their sodium intake can be designed to allow for the sodium intake from the public water supply or the persons can be advised to use other sources of drinking water. Special efforts of public notification must be made for supplies that have very high sodium content so that persons on the more restricted sodium intakes will not be overly stressed if they occassionally use these water supplies.

The 1963 Sodium Survey (7) had the following percent distribution of sodium concentration from 2100 public water supplies:

Range of Sodium Ion Concentration	*Percent of Total Samples*
mg/l	%
0 - 19.9	58.2
20 - 49.9	19.0
50 - 99.9	9.3
100 - 249.9	8.7
250 - 399.9	3.6
400 - 499.9	0.5
500 - 999.9	0.7
Over 1000	0.1

While the question of a maximum contaminant level for sodium is still under consideration by the National Academy of Sciences and others, no specific level will be proposed for the Interim Primary Drinking Water Regulations. The Environmental Protection Agency believes that the available data do not support any particular level for sodium in drinking water, and that the regulation of sodium by a maximum contaminant level is a relatively inflexible, very expensive means of dealing with a problem which varies greatly from person to person.

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SULFATE

The presence of sulfate ion in drinking water can result in a cathartic effect. Both sodium sulfate and magnesium sulfate are well-known laxatives. The laxative dose for both Glauber salt ($Na_2SO_4 \cdot 10H_2O$) and Epsom salt ($MgSO_4 \cdot 7H_2O$) is about two grams. Two liters of water with about 300 mg/l of sulfate derived from Glauber salt, or 390 mg/l of sulfate from Epsom salt, would provide this dose. Calcium sulfate is much less active in this respect.

This laxative effect is commonly noted by newcomers and casual users of waters high in sulfates. One evidently becomes acclimated to use of these waters in a relatively short time.

The North Dakota State Department of Health has collected information on the laxative effects of water as related to mineral quality. This has been obtained by having individuals submitting water samples for mineral analyses complete a questionnaire that asks about the taste and odor of the water, its laxative effect (particularly on those not accustomed to using it), its effect on coffee, and its effect on potatoes cooked in it.

Peterson (1) and Moore (2) have analyzed part of the data collected, particularly with regard to the laxative effect of the water.

Peterson found that, in general, the waters containing more than 750 mg/l of sulfate showed a laxative effect and those with less than 600 mg/l generally did not. If the water was high in magnesium, the effect was shown at lower sulfate concenrations than if other cations were dominant. Moore showed that laxative effects were experienced by the most sensitive persons, not accustomed to the water, when magnesium was about 200 mg/l and by the average person when magnesium was 500-1,000 mg/l. Moore analyzed the data as shown in Table 1. When sulfates plus magnesium exceed 1,000 mg/l, a majority of those who gave a definite reply indicated a laxative effect.

Table 2 presents some data collected by Lockhart, Tucker and Merritt (3) and Whipple (4) on the influence of sulfate on the taste of water and coffee. Because of the milder taste of sulfate over chloride (5) (6) a taste standard for sulfate would probably be in the 300-400 mg/l range. The Peterson data (1) and Table 1 (2), however, indicate that from 600 to 1000 mg/l of sulfate has a laxative effect on a majority of users.

While a limit for sulfate may be included in Secondary Drinking Water Regulations, on the basis of the effect of sulfate on water taste, no maximum contaminant level is being proposed at this time. As noted above, a relatively high concentration of sulfate in drinking water has little or no known effect on regular users of the water, but transients using high sulfate water sometimes experience a laxative effect. Whether this effect will occur, and its severity, varies greatly with such factors as the level of sulfate in the water being consumed and the level of sulfate to which the transient is accustomed. Because of this great variability, the available data do not support

TABLE 1

Determination	Range mg/l	Number of Wells in Range	Laxative Yes	Laxative No	Effects Present Not Stated	Percent of Yes Answers *
Magnesium plus sulfate	0-200	51	9	34	8	21
	200-500	45	7	27	11	21
	500-1,000	56	11	38	17	28
	1,000-1,500	36	18	10	8	64
	1,500-2,000	14	6	4	4	60
	2,000-3,000	21	13	3	5	81
	Over 3,000	14	5	1	8	83
Sulfate	0-200	56	10	36	10	22
	200-500	47	9	28	10	24
	500-1,000	56	13	26	17	33
	1,000-1,500	34	16	10	8	62
	1,500-2,000	16	9	4	3	69
	2,000-3,000	20	9	3	8	75
	Over 3,000	8	3	0	5	100

*This percentage is based only on the total of yes and no answers. It is probable that a large proportion of the wells for which no statements were made were not regularly used as water supplies.

TABLE 2. Data on the Influence of Sulfate Salts on the Taste of Water and Coffee

	Threshold Concentration — mg/l			
	Median		Range	
Salt	Salt	Anion	Salt	Anion
Na_2SO_4	350	327	250-550	169-372 (4)
$CaSO_4$	525	370	250-900	177-635 (4)
$MgSO_4$	525	419	400-600	320-479 (4)
	Average			
$MgSO_4$	500	400 (3)		

the establishment of any given maximum contaminant level. The Environmental Protection Agency recommends that the States institute monitoring programs for sulfates, and that the transients be notified if the sulfate content of the water is high. Such notification should include an assessment of the possible physiological effects of consumption of the water.

In the meantime, research is being undertaken to determine if the health effects of sulfate in drinking water warrant further consideration. If data are generated to support a maximum contaminant level, this level will be proposed for inclusion in Revised Interim Primary Water Regulations.

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Appendix B–Radionuclides

Introduction

The Safe Drinking Water Act directs the Administrator to set interim primary standards for drinking water that "shall protect health to the extent feasible, using technology, treatment techniques and other means, which the Administrator determines are generally available (taking costs into consideration)." The cost considerations referred to are limited to treatment techniques and other means which are under the control of the water supplier. The Agency believes that the establishment of maximum contaminant levels for radioactivity (1) will protect health to the extent feasible and aid achievement of the national goal of safe drinking water.

General Considerations

In determining maximum contaminant levels for radioactivity in drinking water the Agency has given consideration to several important factors including the diversity of sources causing radioactivity to be present in drinking water. Radioactivity in public water systems may be broadly categorized as naturally occurring or man-made. Radium-226 is the most important of the naturally occurring radionuclides likely to occur in public water systems. Although radium may occassionally be found in surface water due to man's activities, it is usually found in ground water where it is the result of geological conditions, not subject to prior control. In contrast to radium, man-made radioactivity is ubiquitous in surface water because of fallout radioactivity from nuclear weapons testing. In some localities this radioactivity is increased by small releases from nuclear facilities (such as nuclear power plants), hospitals, and scientific and industrial users of radioactive materials. The Agency recognizes that, for both man-made and naturally occurring radioactivity, a wide range of both controllable and uncontrollable sources can influence the concentration of radioactivity in water served by public systems.

Variability in the quality of source waters is not unique for radioactive contaminants; other contaminents in drinking water also differ widely in their occurrence. Limits to protect public health can not be based on some proven harmless intake of radioactive material. Rather, maximum contaminant levels for radioactivity are based on the assumption that there is no harmless level of dose from ionizing radiation and that any detrimental effects on health produced by the radiation will be proportional to the dose equivalent delivered by the radioactivity in drinking water.

The Agency recognizes that for the low doses and dose rates expected from intakes of drinking water, the risk to an individual is small and that the potential health effects associated with the risk are no different in the types of diseases manifested spontaneously, representing in fact only small potential increases in the normal incidences in these diseases. The Agency also recognizes that the number of health effects caused by ionizing radia-

tion at very low doses and dose rates is presently unknown and unlikely to be quantified more precisely in the immediate future. Therefore, the Environmental Protection Agency has adopted a prudent policy which assumes that any dose of ionizing radiation may produce potential harmful effects to human health and that the extent of such harm can be estimated from effects that have been observed at higher doses and dose rates than are likely to be encountered from environmental sources of radiation. Acceptance of this policy by the Agency cannot be based solely on the scientific evidence but must include an operational judgment, for practical reasons, in applying present knowledge to the establishment of standards. A more detailed statement of this policy on the relationship between radiation dose and effects is reprinted in Appendix I.

Depending on the circumstances of the exposure, risks from ionizing radiation may or may not be accompanied by an offsetting benefit. In the case of radium contaminated ground water there is no benefit, per se, from the geological processes causing the radiocontamination. On the other hand, man-made radioactivity in public water supply systems may be deliberate due to man's use of nuclear energy to produce electric power, or to his use of radionuclides in the diagnosis and treatment of diseases or research and industrial applications. Balancing the risks and benefits from these activities and specifying appropriate controls for the resultant liquid effluent waste streams is required by other Federal statutes. The Administrator is limited under the Safe Drinking Water Act to regulating the water supplier. However, the Interim Regulations for radioactivity take full account of the fact that control regulations established under authority of the Atomic Energy Act as amended (PL 83-703) and Environmental Protection Standards promulgated under this Act by EPA (Radiation Protection for Nuclear Power Reactors, 42FR2859, January 13, 1977 as well as Federal Radiation Council Guides are intended to limit liquid radioactive discharges into surface waters to the extent practicable.

In addition to man-made radioactivity in drinking water due to effluents from nuclear facilities, surface waters may contain radioactive materials from aerial effluent releases and from nuclear weapons testing. The residual radioactivity in surface waters from fallout due to atmospheric nuclear weapons testing is mainly strontium-90 and tritium, the former being the more important in health considerations. Current data on the impact of fallout strontium-90 on public water supplies is incomplete. However, the available data indicate strontium-90 concentrations are about 1 pCi per liter, corresponding to a dose equivalent to bone marrow of less than 0.5 mrem annually.* Tritium concentrations in surface water rarely exceed 1000 pCi per liter, corresponding to a dose equivalent of less than 0.2 millirem per year.

*Definitions of units and terms are given in the regulations; dosimetry calculations in Appendix IV.

Unplanned releases of radioactive materials are another source of possible contamination. It is not anticipated that the proposed maximum contaminant levels for radioactivity would apply to transient situations such as might follow a major contaminating event. In accident situations it is necessary to balance, on a case-by-case basis, the potential risk from radiation exposure against the practicality and consequences of any remedial measures taken to ameliorate that risk. In such situations Federal guidance as promulgated in the Federal Register Notices of August 22, 1964 and May 22, 1965 will apply and the emergency plans of the States, as provided for in Section 1413(A) (5) of the Safe Drinking Water Act should reflect this Federal Guidance.

Radium in drinking water is primarily a problem of the smaller public water systems. About 40 percent of the U.S. population is served by 243 regional systems supplying large metropolitan areas. Yet, most of the nation's 40,000 community water systems serve less than 500 persons. In general, the large regional systems utilize surface water which on the whole contains very low concentrations of radium. Small supplies commonly use ground water, water which in some cases may contain radium. Therefore, the impact of maximum contaminent levels for radium is more likely to fall on some small supply systems which generally have limited resources. Although one of the intentions of the Safe Drinking Water Act is to encourage the regionalization of these small systems, the availability of local resources for the control and monitoring of radioactivity has been of concern to the Agency. This concern is balanced by the belief that the identification of an atypical radium concentration and the introduction of its control is a direct benefit to the user population. This benefit is a reduction in any health risks due to radium in drinking water.

Health Risks From Radionuclides in Drinking Water

Risk estimates from total body and to a lesser extent, partial body exposure have been made using data published in the NAS-BEIR Report (National Academy of Sciences Report of the Advisory Committee on the Biological Effects of Ionizing Radiation) (2). Such estimates are based on the likely conservative, but nevertheless prudent assumption that the radiation effects are linearly proportional to the dose* and that the number of cancers per rem that have been observed at high doses and dose rates is a practical predictor of the effects per rem at the low doses and dose rates encountered from environmental sources of radiation. The degree of conservatism in such an approach has not been documented but it is likely to be less for ingested alpha particle emitting radionuclides than for those man-made sources of radioactivity which decay by beta and gamma ray emission.

*For the purpose of this statement "dose" means "dose equivalent" as defined in the regulations.

The NAS-BEIR risk estimates are for the U.S. population in the year 1967. For an exposed group having the same age distribution, the individual risk of a fatal cancer from a lifetime total body dose rate of 4 mrem per year ranges from about 0.4 to 2 x 10^{-6} per year depending on whether an absolute or relative risk model is used. *The NAS-BEIR Committee does not choose between these two models but their "most likely estimates" correspond to an average of the absolute and relative risk estimate i.e., about twice the absolute risk. For fatal cancer, an individual risk of 0.8 x 10^{-6} per year from a 4 mrem annual total body dose is believed to be a reasonable estimate of the annual risk from the lifetime ingestion of drinking water at the maximum contaminant level for man-made beta and photon emitting radioactivity. The risk from the ingestion of water containing lesser amounts of radioactivity would be proportionately smaller.

The estimated total health risk from radiation exceeds that due to fatal cancers alone. The NAS-BEIR committee projected that the incidence of non-fatal cancers would be about the same as fatal cancers. The incidence of genetic effects is more difficult to estimate; but the increase, expressed over several generations, would be comparable to the increased incidence of fatal cancer (2).

The estimated risks of a fatal cancer due to a lifetime exposure of ionizing radiation can be compared to the risk without additional radiation by normalizing the NAS-BEIR data for the 1967 population in terms of a single individual's exposure history. Based on U.S. Vital Statistics, (3) the probability that an individual will die of cancer is about 0.19. This probability may be increased by 0.1% from a lifetime dose equivalent rate of 15 mrem per year. Maximum contaminant levels for man-made beta and photon emitters limit the dose equivalent from the drinking water pathway to 4 mrem per year, corresponding to a lifetime risk increase of 0.025% to exposed groups.

For partial body irradiation, which is not uncommon for ingested radionuclides since the radioactivity may be largely concentrated in a particular organ or group of organs, the estimated risk is somewhat less than for total body exposure where all organs are irradiated. For example, the estimated thyroid cancer incidence rate from the thyroid gland receiving 10 mrem per year continuously ranges from about 0.5 to 1.3 per year per million exposed persons (averaged over all age groups). Fatality due to thyroid cancers varies with age, from nearly zero for children and young adults to about 20 percent of the incidence for persons well past middle age. Although it is noted that estimated fatalities from thyroid exposure are at least five times less than that from whole body exposure, other factors bearing on the health impact are significant.

*Absolute risk estimates are based on the reported number of cancer deaths per rad; relative risk estimates, on the percentage increase in cancer mortality per rad.

The incidence in thyroid tissue of non-cancerous neoplasms, (benign nodules), following radiation exposures is much higher than the incidence of thyroid cancers, particularly in the young (2). Since the most likely treatment for such nodules is severe, thyroidectomy, the medical consequences are underestimated by a consideration of cancers only. In addition, there is clinical evidence that the young appear to be particularly susceptible to radiation induced cancer of the thyroid, perhaps by as much as a factor of 10 (2, 3). While it is appropriate to calculate risks due to the dose permitted by an ambient standard on the basis of the average risk throughout life and not just childhood alone, as in the Interim Regulations, the Agency recognizes a need for some conservatism where the major impact of the allowed radiation may fall on a particular subgroup.

Radium locates primarily in bone where 80 to 85 percent of the retained radium is deposited. However, other organs are also irradiated to a lesser extent and the total health risk from radium ingestion has been estimated by summing the dose and resultant risk from all organs, Appendix II. Risk estimates derived from the BEIR Report (2) indicate that continuous consumption of drinking water containing radium-226 or radium-228 at the maximum contaminant level of 5 pCi/l may cause between 0.7 and 3 cancers per year per million exposed persons. Almost all of these cancers would probably be fatal. Although the maximum contaminant level for radium is much nearer Federal Radiation Council guides than the limit for man-made radioactivity, see below, the estimated risks from maximum contaminant levels for radium and for man-made radioactivity are nearly the same. It should be noted that these risk estimates apply only to the relatively small proportion of the population exposed to radioactivity at the maximum contaminant level.

While it is incorrect to speak of safety factors in radiation standards, since only in the complete absence of radiation can any effects be avoided completely, some perspective may be gained by comparing the dose due to drinking water at maximum contaminant levels to dose levels established for population groups by the Federal Radiation Council (4). The radiation protection guide for all sources of total body exposure except radiation received for medical purposes and that due to natural background is 170 millirem per year. At this dose rate effects are not expected to be necessarily non-existent but rather non-detectable, except perhaps by rigorous statistical analysis involving a large exposed population. The annual dose allowed by the proposed maximum contaminant levels for man-made radionuclides is over forty times smaller (4 millirem vis-a-vis 170 millirem) for a single exposure pathway, drinking water. Similarly, in the case of radium-226, Federal Guides for total ingestion recommend that the daily intake not exceed 20 pCi,* twice that allowed by the maximum contaminant level, 5 pCi/l and an intake of 2 liters per day.

*Upper limit of Range II (5).

In addition to the maximum contaminant level for radium-226 and radium-228 of 5 pCi/l, the Interim Regulations specify a maximum condium-226.* A limit is placed on gross alpha particle activity rather than dium-226.** A limit is placed on gross alpha particle activity rather than each alpha particle emitting radionuclide individually since it is impractical at the present time to require identification of all alpha particle emiting radionuclides because of analytical costs.

The maximum contaminant level for gross alpha particle activity is based on a consideration of the radiotoxicity of other alpha particle emitting contaminants relative to radium. The 15 pCi per liter gross alpha particle limit (which includes radium-226) is based on the conservative assumption that if the radium concentration is 5 pCi/l and the balance of the alpha particle activity is due to the next most radiotoxic alpha particle emitting chain, starting with lead-210, the total dose to bone would be equivalent to less than 6 pCi/l of radium-226 (6).

As stated in Section 141.15(b) in the Interim Regulations, the maximum contaminant level for gross alpha particle activity does not include any uranium or radon that may be present in the sample. The Agency may consider proposing maximum contaminant levels for these radionuclides at a later date after determining the national need for such regulations, the cost of water treatment to remove these radionuclides and their dosimetry and potential for causing adverse health effects. It should be noted that the maximum contaminant level for gross alpha particle activity includes man-made as well as naturally occurring radioactive materials, Section 141.2(m).

The Control of Radium in Public Water Systems

In contrast to man-made radioactivity, for which the environmental impact is controlled by a number of regulatory agencies, the abatement of radium radioactivity in drinking water has received little attention. Therefore, radium contamination of drinking water is often of more concern from a regulatory standpoint than that due to man-made radioactivity. Radium-226 is distributed widely in the U.S., and is found frequently in ground water, particularly in the midwestern and Rocky Mountain States. (In a comparatively few cases radium-228, a beta emitter having a chain of daughter radionuclides which decay by alpha particle emission, like radium-226, is also present.) Unlike the situation for ground water, radium is infrequently found in any appreciable quantity in U.S. surface waters. In most of the public supply systems utilizing surface water the radium content is extremely low, less than 0.1 pCi per liter. In contrast to surface waters the concentration of radium in ground waters used by public supply systems can be appreciable, concentrations as large as 50 pCi per liter have been

*Radium-228 is a beta particle emitter.

reported and perhaps as many as 500 community water systems supply water that exceeds 5 pCi per liter.

Several remedial measures are applicable to radium control. In some instances it should be possible to utilize surface or other ground water sources containing less radium. Dilution with less radioactive waters is an acceptable abatement technique for complying with the interim regulations. Depending on the quality of the source water, such common water treatment practice as coagulation may remove about 25% of the radium (7). However, in some cases more rigorous treatments will be required to meet the maximum contaminant level for radium-226 and radium-228. Radium removal by means of conventional technology is feasible. A number of public water systems currently remove radium as part of their water softening treatment processing. The most efficient and in many cases the most economical treatment method for radium removal is based on the use of zeolite as an ion exchange medium. In this process calcium and radium are exchanged for sodium. The Agency is aware that if the mineral content of the source water is high, the exchange of calcium with sodium could result in a marked increase in the sodium content of the drinking water. However, ingestion of sodium via drinking water in such cases would still be lower than the normal dietary intake level. Even so, persons on low sodium diets should be informed of any significant changes in sodium concentration.

National Cost For Radium Removal

In order to estimate the total national cost to remove radium from all public water systems it is necessary to know both the local concentration of radium and the population served by each system. Such complete information is not available since the majority of U.S. systems have not been analyzed for radium. However, many systems have been radioassayed, particularly in the Midwest where radium contamination is encountered most often. The estimated costs of radium removal, given below, are based on a sample of public water systems identified by Straub in his search of the relevant literature on radium contamination (8). Straub listed 306 community water systems serving radium-226 at a concentration of 0.5 pCi/l or more. While his list is probably representative of the population size of systems serving water at various radium concentrations, it is not of course complete and contains some bias since radium assay has been extensive only in areas known to have a potential for higher radium levels. A second source of bias is that larger water systems are more likely to be selected for study by public health authorities than small community systems serving only a few persons. At best the sample of 306 systems represents a minimum estimate of the total number of impacted systems. However, in view of the extent of national monitoring that has occurred in recent years, it is doubtful that the sample is low by an order of magnitude. For the purpose of this

analysis, EPA has estimated Straub's sample represents about 30% of the systems in the U.S. having radium concentrations greater than 0.5 pCi/l. This may underestimate the number of supplies but probably overestimates the population impacted because of the likely bias in the sample, as outlined above. Since costs for radium removal are directly related to population, the estimate of national costs developed below may be somewhat high.

The cost of achieving various control levels and the estimated health benefits are shown in Table 1. It is seen that the total national cost for radium removal increases rapidly with decreasing concentrations of radium not only because of the increased marginal cost for treatment at low concentrations (Appendix III) but also because both the number of supply systems impacted and the average population served becomes larger. The Administrator believes that because of the limited data on the cost of radium removal and the extent of radium contamination in community water supplies currently available it would be unwise to prescribe radium removal at concentrations lower than 5 pCi per liter. It should be noted, however, that under the Safe Drinking Water Act of 1974 (PL 93-523), States may set more stringent standards if they so desire.

TABLE 1. Annual National Cost and Health Savings for Achieving Radium Control Limits

Control Limit	Estimated Number of Systems	Average Size of Systems	Average Cost Per Systems	National Cost to Achieve Limit	Estimated Total Number of Lives Saved per yr.
pCi/l	#	Population	Thousands dollars/yr.	Millions dollars/yr.	#
9	240	4,200	6.0	1.4	0.6
8	300	5,400	8.0	2.4	1.1
7	370	5,000	9.2	3.4	1.6
6	450	7,450	12.4	5.6	2.5
*5	500	8,800	17.5	8.8	3.7
4	670	9,500	21.3	14.	5.5
3	800	12,000	30.4	24.	8.2
2	860	12,100	41.6	36.	11
1	980	18,400	70.2	70.	15
0.5	1100	20,800	90.2	100.	20

Includes systems currently exceeding 10 pCi/l.
*Interim maximum contaminant level for radium.

At the maximum contaminant level selected it is estimated that as many as 500 community water systems may need to remove radium or utilize additional source waters containing a lower radium concentration. If ion exchange were the method selected to lower radium concentrations the average cost per supply would be $18,000 per year or about two dollars per person served. The estimated cost effectiveness of radium removal to avoid a potential fatal cancer is not high, mainly because only about one-half percent of the treated water is consumed as drinking water. In some cases it may be

possible to minimize costs by not treating water used only for commercial purposes.

The methodology used to estimate the marginal cost of ion exchange to remove radium and the cost-effectiveness of radium removal to prevent health effects is outlined in Appendix III. It must be understood that other abatement measures such as dilution will have lower costs than those predicted in Appendix III and that the effects of radium removal in terms of reducing the predicted excess cancer incidence is uncertain by at least a factor of four. Therefore, the estimated cost effectiveness of radium removal should not be given undue weight in evaluating the proposed maximum contaminant levels. However, the cost estimates are not affected by the uncertainty in health effect models and have been used by EPA to project the national cost of various control limits considered by the Agency in its selection of a maximum contaminant level for radium.

Impact of Maximum Contaminant Levels for Man-made Radionuclides

Though man-made radioactivity in public water systems is sometimes a matter of concern it is important to recognize that unlike the case for radium, current ambient concentrations are less than the proposed limits because of regulatory concern for these radionuclides. Drinking water is not a major pathway for exposure from nuclear power plants. The Agency has reviewed all the Envionmental Impact Statements for power reactors currently available. Based on the design of these reactors the estimated total body doses due to drinking water served by public water systems from these facilities range from 0.00001 to 0.3 millirem per year with 90% of the expected doses less than 0.04 millirem per year. The average total body dose is 0.3 millirem per year. Thyroid doses are somewhat larger, ranging from 0.0003 to 0.8 millirem per year, with an average annual dose of 0.08 millirem per year.

Data on ambient levels in public water systems indicate that almost all of the radioactivity in the aquatic environment is due to residual radioactivity from nuclear weapons testing. The historical trend of radioactivity in the Great Lakes and in other waterways shows this source of radioactivity is diminishing (9).

The maximum contaminant level for man-made radionuclides is expressed in terms of the annual dose rate (millirem per year) from continuous ingestion. Specifying maximum contaminant levels in terms of radioactivity concentration (pCi per liter) was considered but rejected in view of the short length of time such limits would be appropriate, since presently available dose conversion factors for ingested radioactivity are obsolescent and the ICRP is developing new dose models. When appropriate models for doses due to environmental contamination become available, the Agency will revise the Interim Regulations to permit the use of newer data. The concentrations yielding 4 millirem annually, given in Appendix IV, are based on

NBS Handbook 69 as required by the Interim Regulations, 41 FR 133, p. 28402, July 9, 1976.

Monitoring for Radioactivity in Community Water Systems

The Agency has developed monitoring requirements for radioactivity with two ends in view. Information must be available to the supplier so he can control the quality of the water he serves. However, the cost of the monitoring should not result in an undue economic burden in terms of other financial requirements for safe operation of the system. To an extent these are conflicting requirements since more information can always be purchased for more money. The Agency has tried to limit the monitoring to that which is essential for determining compliance with maximum contaminant limits under most conditions. As State capability for effective monitoring is augmented, States are encouraged to introduce more rigorous monitoring of particular supplies because of local knowledge of their potential for radiocontamination. In addition Federal monitoring requirements for radioactivity are limited to community water systems as defined in Section141.2 of the Interim Regulations. Since the proposed limits are based on lifetime exposure, any radiation risk to transient populations is minimal.

In general, the Interim Regulations call for quarterly sampling. In the case of naturally occurring radioactivity it is often thought that a single sample can be used to determine the average annual concentrations. This is not the case for some ground water sources where the annual discharge cycle of the aquifers has a pronounced effect on radium concentration. In such cases, a single yearly grab sample could show a low concentration, resulting in the acceptance of water containing more than a maximum contaminant level. Conversely, an abnormally high level could lead to the institution of expensive control measures where the annual average concentration is really acceptable. Although sampling at monthly intervals might be advisable in certain locations and situations (and should be required by the State where necessary) the Agency believes quarterly sampling will be sufficient to determine the average annual concentration in most cases. Where the average annual concentration has been shown to be less than one-half the relevant maximum contaminant level, a yearly sampling procedure is permitted by the regulations.

In order to reduce monitoring costs, the Interim Regulations allow composited samples to be radioassayed, usually at yearly intervals. In such cases care must be taken to prevent the loss of activity by means of absorption on container walls. Acidification with 1 milliliter of 16N HNO_3 per liter of sample is a method suggested in "Interim Radiochemical Methodology for Drinking Water" (10). In the case of iodine-131, hydrochloric rather than nitric acid should be used for acidification and sodium bisulfite should be added to the sample. In some cases State laboratoroies may prefer to count quarterly samples rather than keep track of quarterly aliquots. If so, the estimated costs given below will be exceeded. The increased cost is not large,

however, and quarterly measurements are recommended, particularly for the monitoring of gross beta activity.

It should be noted that from the definition of "maximum contaminant level" in the Interim Regulations, section 141.2(c), samples should be collected from free flowing outlets, not at the source of supply water. Since in some cases, several sources may contribute water to the system, samples should be taken at representative points within the system so as to truly reflect the maximum concentration of radioactivity received by users. In cases where more than one source is utilized, suppliers shall monitor source water, in addition to water from a free flowing tap, when ordered by the State.

Although monitoring a typical community water system is relatively inexpensive, less than five dollars per year, the total national cost of monitoring for radium-226, radium-228, and gross alpha particle activity is not trivial because of the large number of supplies involved, 40,000. In order to minimize cost, the Agency is proposing that a water supplier initially obtain a relatively low cost analysis of gross alpha particle activity. In most cases this test will indicate that no significant activity is present and additonal tests will not be required. However, when the gross alpha measurement indicates the alpha particle activity may exceed 5 pCi per liter, a further test for radium-226 is required.

Although not in the same decay chain, radium-228 sometimes accompanies radium-226. Only rarely, however, does the radium-228 concentration exceed that of radium-226. Therefore, a radium-228 analysis, which is relatively expensive, is only required when the radium-226 concentration exceeds 3 pCi per liter. In localities where radium-228 may be present in drinking water, it is recommended that the State require radium-226 and/or radium-228 analyses when the gross alpha particle activity exceeds 2 pCi/l.

The Interim Regulations require sampling and measurement at quarterly intervals where the limits are exceeded so that the water supplier can follow the variation of radium concentration through the yearly cycle and thereby institute remedial measures, such as additional dilution or treatment, during periods when concentrations are unusually high. Monitoring at quarterly intervals shall be continued until the annual average concentration no longer exceeds the maximum contaminant level or until a monitoring schedule as a condition to a variance, exemption or enforcement action shall become effective.

Monitoring Costs for Radium and Alpha Particle Activity

Estimated monitoring costs are based on the assumption that 40,000 community water systems will initially monitor for gross alpha particle activity as required by the regulations. If a composite of quarterly collected samples is assayed to minimize analytical expenses the cost for initial survey will be $400,000, Table 2, which lists estimated monitoring costs. The Agency recognizes that the Interim Regulations impose a national program to determine once and for all which community water systems require fur-

ther testing for radium contamination. In order to ameliorate the financial impact of this requirement, the Interim Regulations allow samples to be collected over a three year interval and the substitution of measurements made one year previous to the effective date of the regulations. The Agency considered the possibility of using geological information in selecting which systems should be tested for radium contamination. The poor predictive value shown in the past by such information, however, indicates such a procedure could fail to identify systems which exceed the maximum contaminant levels.

ESTIMATED NATIONAL COSTS FOR MONITORING RADIOACTIVITY IN ALL COMMUNITY WATER SYSTEMS*

	Initial Survey Dollars	Annual Cost (succeding year) Dollars per Year
Public water systems serving more than 100,000 persons	15,000	4,000
Community systems potentially impacted by nuclear facilites	20,000	20,000
Gross alpha particle activity in all community water systems	400,000	100,000
Radium-226 and radium-228	133,000	60,000
Estimated totals	568,000	184,000

*Based on an estimated 40,000 community water systems including an estimated 60 systems impacted by nuclear facilities. The estimates of initial cost are high since States are permitted to substitute equivalent data.

Cost estimates for radium-226 and radium-228 analyses are based on the assumption that, nationally, ten percent of the approximately 35,000 systems using ground water will exceed the screening level for gross alpha activity and therefore require further testing. The Agency recognizes that in some States a much higher percentage of the systems will require radium analyses and that these costs will be distributed very unevenly. Of the 3500 systems analyzing for radium it is assumed that about 700 will also be required to assay for radium-228, Table 1.

After the initial survey, a subsequent gross alpha particle anlysis is required every four years both for those systems utilizing surface water and for those using ground water. Nationwide total annual cost in succeeding years is estimated as $184,000, based on estimated assay costs of $10 for gross alpha activity, $30 for radium-226, by the precipitation method and an additional $15 if a subsequent radium-228 analysis is required.

The annual cost for radium assay in succeeding years is difficult to estimate because it is highly dependent on the findings of the initial survey. For the present the Agency has assumed that 500 systems will continue radium-226 monitoring on a quarterly basis. This is the number of systems thought to exceed the maximum contaminant limit, Table 1. The frequency at which

these 500 systems are monitored will be reduced as they come into compliance with maximum contaminant levels.

The cost estimates shown in Table 2 do not make allowance for the cost saving that will be realized by those States which use data already collected.

Monitoring Costs for Man-made Radioactivity

National monitoring costs for man-made radioactivity are smaller than for natural radioactivity but costs for analysis of individual samples are somewhat greater, Table 3.

TABLE 3.—*Estimated Assay Costs for Man-made Radionuclides*

	$ Costs per sample
Gross beta activity	10
Tritium	20
Strontium-90	30
Iodine-131	60
Strontium-89	30
Cesium-134	30

Except for community water systems directly impacted by nuclear facilities, only an estimated 243 systems serving more than 100,000 persons and utilizing surface water are required to monitor for man-made radioactivity. Since monitoring for gross beta particle, tritium and strontium-90 activity is required, the initial survey cost will be $15,000 and the annual cost for resurvey every four years is $4,000.

The Administrator is allowing wide discretion to the States in determining where quarterly monitoring in the vicinity of nuclear facilities will be required. Community water systems near nuclear facilities other than power reactors and support facilities for the Uranium Fuel Cycle may be monitored for man-made radionuclides at the option of the State. In some local situations a State may want to consider monitoring for contamination from waste storage areas, and large experimental facilities and medical centers. Monitoring is not expected at all community water systems within an impacted water shed but only in those systems most likely to be contaminated.

At present about 40 nuclear power reactors have a potential for introducing man-made radioactivity into community water systems. The estimated annual national cost for monitoring potentially impacted community water systems is $20,000 based on the assumption that 60 community water systems may require assay. This cost will increase, of course, as the number of nuclear facilities increases. The annual cost to an impacted system is estimated as $330 per year.

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APPENDIX I

EPA Policy Statement on Relationship Between Radiation Dose and Effect, March 3, 1975

The actions taken by the Environmental Protection Agency to protect public health and the environment require that the impacts of contaminants in the environment or released into the environment be prudently examined. When these contaminants are radioactive materials and ionizing radiation, the most important impacts are those ultimately affecting human health. Therefore, the Agency believes that the public interest is best served by the Agency providing its best scientific estimates of such impacts in terms of potential ill health.

To provide such estimates, it is necessary that judgements be made which relate the presence of ionizing radiation or radioactive materials in the environment, i.e., potential exposure, to the intake of radioactive materials in the body, to the absorption of energy from the ionizing radiation of different qualities, and finally to the potential effects on human health. In many situations the levels of ionizing radiation or radioactive materials in the environment may be measured directly, but the determination of resultant radiation doses to humans and their susceptible tissues is generally derived from pathway and metabolic models and calculations of energy absorbed. It is also necessary to formulate the relationships between radiation dose and effects; relationships derived primarly from human epidemiological studies but also reflective of extensive research utilizing animals and other biological systems.

Although much is known about radiation dose-effect relationships at high levels of dose, a great deal of uncertainty exists when high level dose-effect relationships are extrapolated to lower levels of dose, particularly when given at low dose rates. These uncertainties in the relationships between dose received and effect produced are recognized to relate, among many factors, to differences in quality and type of radiation, total dose, dose distribution, dose rate, and radiosensitivity, including repair mechanisms, sex, variations in age, organ, and state of health. These factors involve complex mechanims of interaction among biological, chemical, and physical systems, the study of which is part of the continuing endeavor to acquire new scientific knowledge.

Because of these many uncertainties, it is necessary to rely upon the considered judgments of experts on the biological effects of ionizing radiation. These findings are well-documented in publications by the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), the National Academy of Sciences (NAS), the International Commission on Radiological Protection (ICRP), and the National Council on Radiation Protection and Measurements (NCRP), and have been used by the Agency in formulating a policy on relationship between radiation dose and effect.

It is the present policy of the Environmental Protection Agency to assume a linear, nonthreshold relationship between the magnitude of the radiation dose received at environmental levels of exposure and ill health produced as a means to estimate the potential health impact of actions it takes in developing radiation protection as expressed in criteria, guides or standards. This policy is adopted in conformity with the generally accepted assumption that there is some potential ill health attributable to any exposure to ionizing radiation and that the magnitude of this potential ill health is directly proportional to the magniture of the dose received.

In adopting this general policy, the Agency recognizes the inherent uncertainties that exist in estimating health impact at the low levels of exposure and exposure rates expected to be present in the environment due to human activities, and that at these levels the actual health impact will not be distinguishable from natural occurrences of ill health, either statistically or in the forms of ill health present. Also, at these very low levels, meaningful epidemiological studies to prove or disprove this relationship are difficult, if not practically impossible, to conduct. However, whenever new information is forthcoming, this policy will be reviewed and updated as necessary.

It is to be emphasized that this policy has been established for the purpose of estimating the potential human health impact of Agency actions regarding radiation protection, and that such estimates do not necessarily constitute identifiable health consequences. Further, the Agency implementation of this policy to estimate potential human health effects presupposes the premise that, for the same dose, potential radiation effects in other constituents of the biosphere will be no greater. It is generally accepted that such constituents are no more radiosensitive than humans. The Agency believes the policy to be a prudent one.

In estimating potential health effects it is important to recognize that the exposures to be usually experienced by the public will be annual doses that are small fractions of natural background radiation to at most a few times this level. Within the U. S. the natural background radiation dose equivalent varies geographically between 40 to 300 mrem per year. Over such a relatively small range of dose, any deviations from dose-effect linearity would not be expected to significantly affect actions taken by the Agency, unless a dose-effect threshold exists.

While the utilization of a linear, nonthreshold relationship is useful as a generally applicable policy for assessment of radiation effects, it is also EPA's policy in specific situations to utilize the best available detailed scientific knowledge in estimating health impact when such information is available for specific types of radiation, conditions of exposure, and recipients of the exposure. In such situations, estimates may or may not be based on the assumptions of linearity and a nonthreshold dose. In any case, the

assumptions will be stated explicitly in any EPA radiation protection actions.

The linear hypothesis by itself precludes the development of acceptable levels of risk based solely on health considerations. Therefore, in establishing radiation protection positions, the Agency will weigh not only the health impact, but also social, economic and other considerations associated with the activities addressed.

APPENDIX II

Risk to Health from Internal Emitters

A. *The Dose and Health Risk from Radium Ingestion*

The Federal Radiation Council has also recommended radium-226 ingestion limits for the general population and stated that such limits should be based on environmental studies *not* the models used to establish occupational dose limits (1). The FRC ingestion limit is based on the assumption that the skeletal radium-226 burden does not exceed 50 times the daily radium intake. This assumed relationship between ingestion and body burden agrees quite well with the measurements of skeletal body burdens and radium ingestion data reported by the U. N. Scientific Committee on the Effects of Atomic Radiation (2). By comparing Tables 9 and 10 in reference (2) it is seen that the skeletal burden is about forty times the estimated daily radium-226 intake.

The FRC limit on radium ingestion is 20 pCi per day.* After continuous ingestion at this limit the skeletal body burden is 1000 pCi. Ingestion of 2 liters of drinking water per day containing radium-226 at a maximum contaminant level of 5 pCi per liter would result in a skeletal burden of 500 pCi.

In order to estimate potential health effects from radium ingestion, it is necessary to express the dose equivalent from this body burden in terms of the ICRP dose model which was used in the dose estimates made in the NAS BEIR Report (3). The ICRP model predicts an average dose to bone of about 30 rem per year from a body burden of 100,000 pCi(2). A body burden of 500 pCi would therefore cause an average dose of 150 mrem per year.

The NAS BEIR Report (Table 3-2) gives the rate of *absolute risk* from bone cancer as four percent of all non leukemia type cancers (3). For a lifetime risk plateau and continuous lifetime exposure (Table 3-1 in reference 3) the number of bone cancers per year is 3 per 10^6 man-rem per year, estimated on the basis of *absolute risk*.

Relative risk, the number of cancers expected on the basis of their percent increase in an irradiated population, is also estimated in the BEIR for total body exposure, Table 3-1. The NAS-BEIR committee risk report does not give a breakdown by cancer site of the relative risk per rem. Assuming that bone cancers are four percent of the relative risk from total body exposure, excluding leukemia as before, the *relative risk* of bone cancer is about 17 per year per 10^6 man-rem per year.

Bone cancer is not the only risk from radium ingestion. About 15 percent of the radium is deposited in soft tissue where bone marrow is the primary tissue at risk. Doses to soft tissue relative to those in bone from ingested radium have been calculated in reference 2, Table 9. The risk to these tissues from radium ingestion has been calculated by weighing the risk estimates

*Range II, averaged over a suitable sample (1).

for leukemia (and other cancers) given in the NAS-BEIR Report, by the appropriate organ dose. The total *absolute risk* due to bone and soft tissue cancers is 60 percent larger than that from bone cancer alone; the *relative risk*, 16 percent greater. Therefore, the *annual rate* of total cancers from ingesting radium ranges from 4.8 (3 x 1.6) to 20 (17 x 1.16) per million man-rem/year depending on whether an *absolute* or *relative* risk model is used.

Combining these estimates of the annual risk of total cancer with the ICRP dose to bone, 0.15 rem per year, from the ingestion of 10 pCi of radium-226 per day yields the range of estimated health effects from radium ingestion, 0.7 to 3 cancers per year, per million exposed persons. Almost all of any induced cancers would be fatal. Bone cancer fatality is estimated at nearly 90 percent, that for leukemia is much higher.

Given the assumption that radiation damage occurs at incremental doses greater than those due to external background radiation, the total health impact from a public water supply system can be estimated on the basis of the total dose received by the population it serves. This aggregate dose can be calculated by multiplying the number of persons served by the average dose received by a reference man consuming two liters of drinking water per day. Based on the geometric mean of the individual risk discussed above, a radium concentration of 5 pCi per liter in a water system serving 1,000,000 persons could result in an estimated health impact of 1.5 fatalities per year or about 3×10^{-7} per person per year for each pCi per liter of radium-226 or radium-228 in the drinking water. As is shown in Appendix III, this number can be used to estimate the marginal cost effectiveness of radium control in public water systems to prevent cancer. However, it must be kept in mind that the risk estimates are uncertain by a factor of four or more.

B. *The Relative Health Risk of Radium-228 as Compared to Radium-226*

Unfortunately, guidance on the body burden from chronic radium-228 ingestion was not provided by the Federal Radiation Council in their discussion of radium-226 limits. Because Handbook 69, which is based on 1959 ICRP dose models (4), gives a maximum permissible concentration in water for radium-228 that is three times greater than for radium-226, many persons have concluded that these two isotopes are not equally toxic. However, more recent data (particularly that in the 1972 UNSCEAR report (2) and the 1972 ICRP Report (4) on alkaline earth metabolism) indicates that radium-228 is at least as toxic as radium-226.

There are two major difficulties with the old ICRP model. It assumes for radium-226 an effective half-life in bone of 1.6×10^4 days (44 years) and because of the shorter physical half-life of radium-228 an effective half-life of 2.1×10^3 days (5.8 years) for radium-228. Therefore, using the old ICRP model, on the basis of effective half-life the body burden due radium-226 would be 7.6 times greater than that calculated for radium-228 for equal daily intakes of each.

The recent report from the ICRP Committee II task group on alkaline earth metabolism shows that the old ICRP bone model overestimated the effective half-life of radium-226 and that 17.1 years, not 44, is currently the best estimate of the half-time for radium retention (5).* On this basis the effective half-life of radium-228 (physical half-life 5.75 years) (5) is 4.3 years, assuming the half-time of radium-226 retention is a reasonable estimate of the biological half-life of radium. In light of this new information, the body burden from chronic radium-226 ingestion is about four times greater than that from radium-228, not 7.6 times greater as predicted by the old ICRP model.

The old ICRP model also underestimates the effective energy delivered to bone from a given body burden. The old ICRP model assumes that 50 percent of the radon-220 (physical half-life 55 secs) produced in the radium-228 decay chain escapes from bone as compared to an assumed 70 percent escape of the radon-222 (physical half-life 3.8 days) produced in the radium-226 decay chain. Speculation on this point is unnecessary. The MIT Radioactivity Center has measured the escape of this short half-life radon-220 from bone and found it to be about one to two percent (6).

Since almost all of the radon-220 decay products are retained in bone, the effective energy per disintegration of radium-228 in bone is about 330 MEV, not 190 MEV as given by the old ICRP #2 model. The effective energy for radium-226 in the old ICRP model is 110 MEV, a factor of three less than that for radium-228.

The average dose to bone due to continuous radium ingestion (based on an expontential retention function) is proportional to the effective half-life and effective energy;

for radium-226 this product is 17.1 years x 110 MEV = 1880.

for radium-228 this product is 4.3 years x 330 MEV = 1420.

which indicates that even on the basis of a single exponential retention model, as used in reference (4) these two radionuclides give approximately the same dose per unit activity ingested.

Actually, a simple exponential retention model is not a very good approximation of radium retention in man and the more sophisticated model based on studies in humans that were not available in 1959 (5) is currently being considered by ICRP Committee II.

This new ICRP model on alkaline earth metabolism, indicates that for equal intakes the 50 year dose to bone surfaces from radium-228 is significantly greater than that from radium-226. Experimental data given in the 1972 UNSCEAR report supports this viewpoint (2). In the United States the average daily ingestion of radium-226 and radium-228 is about equal, Table 10 in reference 2. Table 9 in reference 2 shows that the dose to bone

*n.b. that since the old ICRP model was used to calculate both radium doses and health effects this change does not change the risk estimates given in II-A.

surfaces, calculated on the basis of measured skeletal body burdens of radium-226 and radium-228, is greater for radium-228 than for radium-226.

Since radium carcinogenicity is associated with the dose to bone surfaces (7), it is likely that radium-228 is more of a health risk than radium-226. Experimental findings in dogs bear this out. The measured relative biological effectiveness of radium-228 is over twice as great as radium-226 when death by osteosarcomas is used as an end point (8). Though the carcinogencity of radium-228 relative to radium-226 may not be as great in man as in dogs, it is prudent to assume chronically ingested radium-228 is at least as dangerous as radium-226.

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APPENDIX III

The Cost and Cost-Effectiveness of Radium Removal

The United States Environmental Protection Agency planning guide for water use provides estimates of the amount of water used per day by various population groups (1). Per capita water consumption increases with community size because of industrial and commercial usage. In this cost analysis a water use of 100 gallons per person day is assumed. This may be somewhat high since mainly small community systems, serving less than 10,000 persons, would be impacted by the proposed regulations.

"Selecting a Softening Process," by Frank O. Wood, has served as the Agency's primary reference for assessing the cost of zeolite treatment to remove radium(2). Wood surveyed a representative sample of community water systems to determine their construction and operating costs for water softening in order to compare the economics of lime-soda ash softening with treatment by ion exchange. Zeolite ion exchange was the lower cost operation for public water systems serving fewer than about 50,000 persons and therefore is applicable to all systems which may require radium abatement.

Wood's report shows that while the cost per 1000 gallons increases slightly with system capacity, 8¢ per 1000 gallons is a conservative average value for systems supplying less than 1 million gallons per day. Because plants examined by Wood had been built over a period of several years, he normalized costs in terms of the 1967 wholesale price index to place them on an equal chronology basis. For this analysis Wood's estimates have been updated to 1975 by means of the "Sewage Treatment Plant Construction Cost Index," prepared by the United States Environmental Protection Agency Office of Water Programs Operations. From 1967 to January 1975 the index increased by about 90%. Therefore, for the cost analysis for radium removal the Agency has assumed a treatment cost of 15¢ per 1000 gallons. It should be noted that these costs include amortization of capital costs over a 20 year period as well as chemical costs for regeneration of the zeolite system. Labor costs for equipment operation are not included since these costs were too small to be included in Wood's analysis; the equipment is essentially automatic.*

Usually only a fraction of the supply water need be treated since the mixing of treated and untreated water is an acceptable abatement procedure. The fraction of water treated, F, to achieve a given radium concentration is calculated as follows:

$$F = \frac{1 - C_a/C_u}{e}$$

where C_u is the radium concentration in untreated water, C_a is the average radium concentration in treated and untreated waters and e is the efficiency of radium removal.

*Recently completed studies indicate that addition of labor costs would increase the treatment cost by about 2¢ per 1000 gallons (3).

The efficiency at which radium is removed from water by a zeolite ion exchange column is very high, approaching 99% for a newly charged column and falling to around 90% just before breakthrough in a spent column. The results listed below are based on an estimated overall removal efficiency of 97 percent.

The volume of water that must be treated per person year to reduce the radium concentration from (n) pCi/l to (n-1) pCi/l is shown in Table III-1 along with the annual marginal cost per pCi/l removed to treat this volume of water. Costs are based on 15¢ per 1000 gallons, as outlined above. For concentrations greater than 5 pCi/l the annual per capita cost ranges from about 60 cents to 90 cents per pCi/l removed depending on the initial concentration.

Each decrement of the average annual concentration of radium by 1 pCi/l, corresponds to an estimated health savings of approximately 3×10^{-7} excess cancers averted per year, Appendix II-A. Dividing this number by the annual expenditure required to obtain a given concentration yields the estimated marginal costs per cancer averted shown in Table III-1. The marginal cost increases slowly as the radium concentration is decreased until at about 2-3 pCi per liter the cost per estimated excess cancer averted increases more rapidly due to the larger fraction of the water needing treatment to achieve smaller concentrations.

TABLE III-1—*The Marginal Cost-Effectiveness of Radium Removal**

Initial Radium Concentration	Volume of Water Treated Per Person Year	Annual Cost Per Person to Remove One pCi per liter	Marginal Cost to Prevent One Cancer
(pCi/l)	(1000 gallons)	(dollars)	(millions of dollars)
10	3.8	0.57	1.88
9	4.2	0.63	2.09
8	4.7	0.71	2.35
7	5.2	0.78	2.61
6	6.3	0.94	3.14
5	7.5	1.13	3.77
4	9.4	1.41	4.71
3	12.6	1.88	6.28
2	18.9	2.82	9.41
1	36.5	5.48	18.83

*by zeolite ion exchange

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APPENDIX IV

Dosimetric Calculations for Man-Made Radioactivity

A. *Calculations Based on NBS Handbook 69*

The dose rate from radioactivity in drinking water is calculated on the basis of a 2 liter daily* intake. Except for tritium and strontium-90, see below, the concentrations of man-made radionuclides causing 4 millirem per year have been calculated using the data in NBS Handbook 69 (1) and are tabulated in Table IV-2A and IV-2B. The dose models used in preparing Handbook 69 are outlined in reference 2. Maximum Contaminant Levels are defined in terms of the annual dose equivalent to the total body or any internal organ. Handbook 69 lists the critical organ for each radionuclide. Often the total body is listed as the critical organ. The 168 hour maximum permissible concentrations for ingestion in Handbook 69 are not calculated on the basis of the same annual dose to each critical organ as in the Interim Regulations, rather different organ doses are permitted by occupational radiation protection limits (ORL), Table IV-1.

TABLE IV-1. Occupational Radiation Limits

	(ORL)
Critical Organ	ORL (rems)
Total body	5
Gonads	5
Thyroid	30
Bone	29.1 (a)
Other Organs	15

(a) Based on the alpha energy deposited in bone by 0.1 μCi of radium-226.

The maximum permissible concentrations for a 168 hour week, MPC, in Handbook 69, assume ingestion at 2.2 liters per day and are in units of μCi per cc. The various numerical factors can be combined to find C_4, the concentration causing 4 mrem per year from 2 liters daily ingestion of drinking water as follows:

$$C_4 = 4.4 \times 10^6 \times \frac{\text{MPC}}{\text{ORL}} \ldots\ldots \text{pCi per liter}$$

Critical organs are identified by boldface type in Handbook 69 so that an appropriate ORL can be selected from Table IV-1.

To illustrate, a sample calculation, taken from page 24 of Handbook 69 is given:

*The recent ICRP publication #23, "Report of the Task Group on Reference Man," (3) gives the total daily water intake as 3 liters, 1.95 liters by fluid intake, the balance by food and food oxidation. Almost all of the fluid intake is from tap water and water based drinks (Page 360).

Radionuclide

Beryllium-7 MPC (168 hours) = 0.02 uCi/cm^3

Listed critical organ GI(LLI) gastrointestional tract (lower large intestine)

$$C_4 = 4.4 \times 10^6 \times \frac{0.02}{15} \frac{\text{pCi}}{\text{l}} = 5867 \text{ pCi/l}$$

$$= 6000 \text{ pCi/l}$$

Rounding is appropriate since the values in Handbook 69 are given to one significant figure.

Calculation of the dose resulting from the ingestion of drinking water containing a known mixture of radionuclides is straightforward. Let A, B, . . . be the concentrations, in pCi per liter, of isotopes a, b, . . . in the water and let $C_4^A(X)$ be the average annual concentrations of isotope A yielding 4 millirem per year to organ X, $C_4^B(X)$ the same quantity for B, etc. The total annual dose to organ X in one year is, then

$$\left[\frac{A}{C_4^A(X)} + \frac{B}{C_4^B(X)} + \ldots \right] \times 4 \text{ millirem}$$

Therefore, the 4 millirem limit is not exceeded if

$$\left[\frac{A}{C_4^A(X)} + \frac{B}{C_4^B(X)} + \ldots \right] \leq 1.0$$

It should be noted that although limits for the various radionuclides may be based on different critical organs, the resultant dose is additive with respect to a specific organ when the total body is the designated critical organ for one of the radionuclides. For example, consider drinking water which has on an annual basis a strontium-90 concentration of 4 pCi/l and a tritium concentration of 15,000 pCi/l. The annual dose to bone marrow from the strontium-90 is 2 mrem. The total body dose from the tritium is 3 mrem annually. Even though the annual concentration of each contaminant alone is permissible, the total dose to bone marrow is 5 mrem annually and therefore the MCL is exceeded. Tabular values for C_4 for photon and beta emitters are listed in Table IV-2A and IV-2B below.

B. *The Dose from Tritium and Strontium-90 in Drinking Water*

For the majority of radionuclides, the models given in Handbook 69 to estimate doses to occupationally exposed workers are also appropriate for environmental contaminants. They are not, however, appropriate for all man-made radionuclides, particularly tritium and strontium-90. Concentrations yielding 4 millirem annually for these radionuclides are given in Table A of the Interim Regulations and listed in Table IV-2A.

Some radionuclides are isotopes of elements which are incorporated into organic molecules within the body so that the single exponential excretion models assumed in the development of Handbook 69 underestimate the

dose. An example is tritium where two or three exponentials may be needed to describe the dose-time relationship of ingested tritium (4). Some investigators have estimated that following chronic ingestion organically bound tritium may increase the dose by a factor of 1.4 to 1.5 over that predicted by Handbook 69 (5). Such estimates are too high because organically bound tritium irradiates the total body mass, and not just the mass of body water, as assumed in the model used in Handbook 69 (2).

Consideration of the daily intake of hydrogen and water shows that the tritium concentration (specific activity) in any organ is no greater than 120% of the tritium concentration in body water. The concentration of tritium in body water following chronic ingestion is T/3 where T is daily intake of tritium in pCi and the total water intake, including that in food, is 3 liters per day (3). Water content by weight of any organ does not exceed 80 percent (4). Therefore, equilibrium concentration of tritium in any organ due to its water content, can not exceed 0.8 T/3 = .267 T pCi/kg.

Because of organically bound hydrogen an organ's hydrogen (and tritium) content is greater than that due to water alone. The daily hydrogen intake is .35. kg per day (3) and, since no organ contains more than 11 percent hydrogen by weight (4), the maximum tritium concentration in any organ following chronic ingestion is .11 T/.35 = .314 T pCi/kg. The specific activity of tritium in any organ due to bound and unbound hydrogen exceeds that due to its water content alone by the ratio .314/.267 = 1.18. Therefore, the dose to any organ due to organically bound tritium exceeds the dose to body water, given in Handbook 69, by no more than about twenty percent.

The Agency is aware that the ICRP is developing new tritium dose models more suitable for environmental sources of tritium exposure than the model used in Handbook 69. Until these models are published and recommended by the Agency, the maximum contaminant level for tritium is calculated on the basis of 80 percent of the value calculated using NBS Handbook 69.* For tritium in drinking water:

$$C_4 = 0.8 \times 4.4 \times 10^6 \times \frac{0.03}{5} = 21{,}120 \text{ pCi/l}$$

$$= 20{,}000 \text{ pCi/l}$$

The maximum contaminant level for strontium-90 in the Interim Regulations is based on the dose model used by the Federal Radiation Council (FRC) to predict the dose to bone marrow (6). According to the FRC model a continuous daily intake of 200 pCi per day of strontium-90 will result in a body burden of 50 pCi per gram of calcium in bone. The annual

*n.b. In accordance with current guidance to Federal agencies, a quality factor of 1.7, as in Handbook 69, is used in this calculation.

dose rate to bone marrow from this body burden would be 50 mrem per year (7). Therefore, continuous ingestion of 16 pCi per day would result in 4 mrem per year, the limit for man-made radionuclides in drinking water. For two liters ingestion of water per day:

$$C_4 = \frac{16 \text{ pCi}}{2} = 8 \text{ pCi/l}$$

C. *Concentrations yielding an Annual Dose of 4 Millirem*

Tables IV-2A and IV-2B give C_4 the annual average concentrations for man-made radionuclides which are assumed to yield an annual dose of 4 millirem to the indicated organ. Table IV-2A comprises those nuclides having half-lives greater than one day. Table IV-2B contains shorter half-life radionuclides not expected to appear in drinking water supplies. Ingestion at a rate of 2.0 liters per day is assumed. The values shown were calculated from the Maximum Permissible Concentrations listed in Handbook 69 (1) as outlined above.

TABLE IV-2A. *Annual Average Concentrations Yielding 4 Millirem per Year for a Two Liter Daily Intake*

(Half-life greater than 24 hours)

Radionuclide	Critical Organ	C_4 (pCi/l)
Tritium	Total Body	20,000
$^{4}Be^{7}$	GI (LLI)	6,000
$^{6}C^{14}$	Fat	2,000
$^{11}Na^{22}$	Total Body	400
$^{15}P^{32}$	Bone	30
$^{16}S^{35}$	Testis	500
$^{17}Cl^{36}$	Total Body	700
$^{20}Ca^{45}$	Bone	10
$^{20}Ca^{47}$	Bone	80
$^{21}Sc^{46}$	GI (LLI)	1,000
$^{21}Sc^{47}$	GI (LLI)	300
$^{21}Sc^{48}$	GI (LLI)	80
$^{23}V^{48}$	GI (LLI)	90
$^{24}Cr^{51}$	GI (LLI)	6,000
$^{25}Mn^{52}$	GI (LLI)	90
$^{25}Mn^{54}$	GI (LLI)	300
$^{26}Fe^{55}$	Spleen	2,000
$^{26}Fe^{59}$	GI (LLI)	200
$^{27}Co^{57}$	GI (LLI)	1,000
$^{27}Co^{58}$	GI (LLI)	9,000
$^{27}Co^{60}$	GI (LLI)	100
$^{28}Ni^{59}$	Bone	300
$^{28}Ni^{63}$	Bone	50
$^{30}Zn^{65}$	Liver	300
$^{32}Ge^{71}$	GI (LLI)	6,000
$^{33}As^{73}$	GI (LLI)	1,000
$^{33}As^{74}$	GI (LLI)	100
$^{33}As^{76}$	GI (LLI)	60
$^{33}As^{77}$	GI (LLI)	200

$^{34}Se^{75}$	Kidney	900
$^{35}Br^{82}$	GI (LLI)	100
$^{37}Rb^{86}$	Total Body	600
$^{37}Rb^{87}$	Pancreas	300
$^{38}Sr^{85}$	GI (SI)	21,000
$^{38}Sr^{89}$	Bone	20
$^{38}Sr^{89}$	Bone Marrow (FRC)	80
$^{38}Sr^{90}$	Bone Marrow (FRC)	8
$^{39}Y^{90}$	GI (LLI)	60
$^{39}Y^{91}$	GI (LLI)	90
$^{40}Zr^{93}$	GI (LLI)	2,000
$^{40}Zr^{95}$	GI (LLI)	200
$^{41}Nb^{93m}$	GI (LLI)	1,000
$^{41}Nb^{95}$	GI(LLI)	300
$^{42}Mo^{99}$	Kidney	600
$^{43}Tc^{96}$	GI(LLI)	300
$^{43}Tc^{97m}$	GI(LLI)	1,000
$^{43}Tc^{97}$	GI(LLI)	6,000
$^{43}Tc^{99}$	GI(LLI)	900
$^{44}Ru^{97}$	GI(LLI)	1,000
$^{44}Ru^{103}$	GI(LLI)	200
$^{44}Ru^{106}$	GI(LLI)	30
$^{45}Rh^{105}$	GI(LLI)	300
$^{46}Pd^{103}$	GI(LLI)	900
$^{46}Pd^{109}$	GI(LLI)	300
$^{47}Ag^{105}$	GI(LLI)	300
$^{47}Ag^{110m}$	GI(LLI)	90
$^{47}Ag^{111}$	GI(LLI)	100
$^{48}Cd^{109}$	GI(LLI)	600
$^{48}Cd^{115m}$	GI(LLI)	90
$^{48}Cd^{115}$	GI(LLI)	90
$^{49}In^{115}$	GI(LLI)	300
$^{50}Sn^{113}$	GI(LLI)	300
$^{50}Sn^{125}$	GI(LLI)	60
$^{51}Sb^{122}$	GI(LLI)	90
$^{51}Sb^{124}$	GI(LLI)	60
$^{51}Sb^{125}$	GI(LLI)	300
$^{52}Te^{125m}$	Kidney	600
$^{52}Te^{127m}$	Kidney	200
$^{52}Te^{127}$	GI(LLI)	900
$^{52}Te^{129m}$	GI(LLI)	90
$^{52}Te^{129}$	GI(S)	2,000
$^{52}Te^{131m}$	GI(LLI)	200
$^{52}Te^{132}$	GI(LLI)	90
$^{53}I^{126}$	Thyroid	3
$^{53}I^{129}$	Thyroid	1
$^{53}I^{131}$	Thyroid	3
$^{55}Cs^{131}$	Total Body	20,000
$^{55}Cs^{134}$	GI(S)	20,000
$^{55}Cs^{135}$	Total Body	900
$^{55}Cs^{136}$	Total Body	800
$^{55}Cs^{137}$	Total Body	200
$^{56}Ba^{131}$	GI(LLI)	600

$^{56}Ba^{140}$	GI(LLI)	90
$^{57}La^{140}$	GI(LLI)	60
$^{58}Ce^{141}$	GI(LLI)	300
$^{58}Ce^{143}$	GI(LLI)	100
$^{59}Pr^{143}$	GI(LLI)	100
$^{61}Pm^{149}$	GI(LLI)	100
$^{62}Sm^{151}$	GI(LLI)	1,000
$^{62}Sm^{153}$	GI(LLI)	200
$^{63}Eu^{152}$	GI(LLI)	60
$^{63}Eu^{154}$	GI(LLI)	200
$^{63}Eu^{155}$	GI(LLI)	600
$^{64}Gd^{153}$	GI(LLI)	600
$^{65}Tb^{160}$	GI(LLI)	100
$^{66}Dy^{166}$	GI(LLI)	100
$^{67}Ho^{166}$	GI(LLI)	90
$^{68}Er^{169}$	GI(LLI)	300
$^{69}Tm^{170}$	GI(LLI)	100
$^{69}Tm^{171}$	GI(LLI)	1,000
$^{70}Yb^{175}$	GI(LLI)	300
$^{71}Lu^{177}$	GI(LLI)	300
$^{72}Hf^{181}$	GI(LLI)	200
$^{73}Ta^{182}$	GI(LLI)	100
$^{74}W^{181}$	GI(LLI)	1,000
$^{74}W^{185}$	GI(LLI)	300
$^{75}Re^{183}$	GI(LLI)	2,000
$^{75}Re^{186}$	GI(LLI)	300
$^{75}Re^{187}$	GI(LLI)	9,000
$^{76}Os^{185}$	GI(LLI)	200
$^{76}Os^{191}$	GI(LLI)	600
$^{76}Os^{193}$	GI(LLI)	200
$^{77}Ir^{190}$	GI(LLI)	600
$^{77}Ir^{192}$	GI(LLI)	100
$^{78}Pt^{191}$	GI(LLI)	300
$^{78}Pt^{193m}$	GI(LLI)	3,000
$^{78}Pt^{193}$	Kidney	3,000
$^{78}Pt^{197}$	GI(LLI)	300
$^{79}Au^{196}$	GI(LLI)	600
$^{79}Au^{198}$	GI(LLI)	100
$^{81}Tl^{204}$	GI(LLI)	300
$^{82}Pb^{203}$	GI(LLI)	1,000
$^{83}Bi^{206}$	GI(LLI)	100
$^{83}Bi^{207}$	GI(LLI)	200
$^{91}Pa^{233}$	GI(LLI)	300

TABLE IV - 2B

Annual Average Concentrations Yielding 4 Millirem per Year for a Two Liter Daily Intake (Half-life less than 24 hours)

Radionuclide	*Critical Organ*	C_i *(pCi/l)*
$^{9}F^{18}$	GI(SI)	2,000
$^{14}Si^{31}$	GI(S)	3,000
$^{17}Cl^{38}$	GI(S)	1,000

$^{19}K^{42}$	GI(S)	900
$^{25}Mn^{56}$	GI(LLI)	300
$^{27}Co^{58m}$	GI(LLI)	300
$^{28}Ni^{65}$	GI(LLI)	300
$^{29}Cu^{64}$	GI(LLI)	900
$^{30}Zn^{69m}$	GI(LLI)	200
$^{30}Zn^{69}$	GI(S)	6,000
$^{31}Ga^{72}$	GI(LLI)	100
$^{38}Sr^{85m}$	Total Body	900
$^{38}Sr^{91}$	GI(LLI)	200
$^{38}Sr^{92}$	GI(ULI)	200
$^{39}Y^{91m}$	GI(SI)	9,000
$^{39}Y^{92}$	GI(ULI)	200
$^{39}Y^{93}$	GI(LLI)	90
$^{40}Zr^{97}$	GI(LLI)	60
$^{41}Nb^{97}$	GI(ULI)	3,000
$^{43}Tc^{96m}$	GI(LLI)	30,000
$^{43}Tc^{99m}$	GI (ULI)	20,000
$^{44}Rh^{105}$	GI(ULI)	300
$^{45}Rh^{103m}$	GI(S)	30,000
$^{49}In^{113m}$	GI(ULI)	3,000
$^{49}In^{114m}$	GI(LLI)	60
$^{49}In^{115m}$	GI(ULI)	1,000
$^{53}I^{132}$	Thyroid	90
$^{53}I^{133}$	Thyroid	10
$^{53}I^{134}$	Thyroid	100
$^{53}I^{135}$	Thyroid	30
$^{55}Cs^{134m}$	Total Body	80
$^{59}Pr^{142}$	GI(LLI)	90
$^{60}Nd^{149}$	GI(LLI)	900
$^{63}Eu^{152}$	GI(LLI)	200
$^{64}Gd^{159}$	GI(LLI)	200
$^{66}Dy^{165}$	GI(LLI)	1,000
$^{68}Er^{171}$	GI(ULI)	300
$^{74}W^{187}$	GI(LLI)	200
$^{75}Re^{188}$	GI(LLI)	200
$^{76}Os^{191m}$	GI(LLI)	9,000
$^{77}Ir^{194}$	GI(LLI)	90
$^{78}Pt^{197m}$	GI(ULI)	3,000
$^{81}Tl^{202}$	GI(LLI)	300

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