

University of Nebraska - Lincoln

DigitalCommons@University of Nebraska - Lincoln

Public Health Resources

Public Health Resources

1962

Public Health Service Drinking Water Standards

Follow this and additional works at: <https://digitalcommons.unl.edu/publichealthresources>

This Article is brought to you for free and open access by the Public Health Resources at DigitalCommons@University of Nebraska - Lincoln. It has been accepted for inclusion in Public Health Resources by an authorized administrator of DigitalCommons@University of Nebraska - Lincoln.



CERTIFICATE

By Authority Of
THE UNITED STATES OF AMERICA
Legally Binding Document

By the Authority Vested By Part 5 of the United States Code § 552(a) and Part 1 of the Code of Regulations § 51 the attached document has been duly INCORPORATED BY REFERENCE and shall be considered legally binding upon all citizens and residents of the United States of America. *HEED THIS NOTICE:* Criminal penalties may apply for noncompliance.



Document Name: USPHS 956: Drinking Water Standards

CFR Section(s): 46 CFR 160.026-4(a)

Standards Body: U.S. Public Health Service



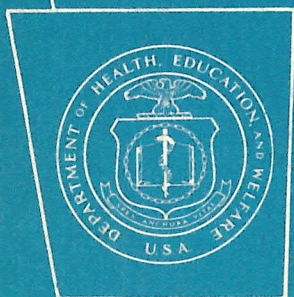
Official Incorporator:

THE EXECUTIVE DIRECTOR
OFFICE OF THE FEDERAL REGISTER
WASHINGTON, D.C.

Public Health Service

DRINKING WATER
STANDARDS

1962



**U.S. DEPARTMENT OF
HEALTH, EDUCATION, AND WELFARE**
Public Health Service

Public Health Service
Drinking Water Standards
Revised 1962



**U.S. DEPARTMENT OF HEALTH, EDUCATION,
AND WELFARE**

U.S. PUBLIC HEALTH SERVICE

Washington 25, D.C.

Public Health Service Publication No. 956

First Printed August 1962

Reprinted March 1963

Reprinted June 1963

Reprinted Sept. 1964

**UNITED STATES
GOVERNMENT PRINTING OFFICE
WASHINGTON : 1962**

For sale by the Superintendent of Documents, U.S. Government Printing Office
Washington, D.C., 20402 - Price 30 cents (paper cover)

cop 3

LIBRARY
UNIVERSITY OF TEXAS
AUSTIN, TEXAS

THE PUBLIC HEALTH SERVICE DRINKING WATER STANDARDS—1962

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE,
PUBLIC HEALTH SERVICE,
Washington 25, D.C., May 6, 1962.

The Standards published herein have been promulgated as Public Health Regulations in the Federal Register. As such they became effective April 5, 1962, as the Standards to which drinking water and water supply systems used by carriers and others subject to Federal quarantine regulations must conform.

The Division of Environmental Engineering and Food Protection is responsible for the application of these Standards to all carrier water supplies.

These Standards supersede the Public Health Service Drinking Water Standards—1946, as amended in 1956. The new Standards were developed with the assistance of an Advisory Committee appointed by the Public Health Service to revise the Standards of 1946. The Committee in its deliberations took cognizance of man's changing environment and its effect on water supplies. Accordingly, new sections, such as one on radioactivity, have been added and substantive changes have been made elsewhere.

The new Standards are in a form believed useful in evaluating the quality and safety of water supplies generally and they are hereby recommended for such use.

LUTHER L. TERRY,
Surgeon General, Public Health Service.

ENDORSEMENT BY THE AMERICAN WATER WORKS ASSOCIATION

Acting on behalf of the Officers and Directors, the AWWA Executive Committee adopted a resolution endorsing the 1962 revision of the USPHS Drinking Water Standards as "minimum" standards for all public water supplies.

The resolution, which will be included with the published standards, read:

WHEREAS, the 1962 Drinking Water Standards of the U.S. Public Health Service, as prepared by the Advisory Committee on Revision of U.S. Public Health Service 1946 Drinking Water Standards and promulgated for use in the administration of interstate quarantine regulations, are intended to apply only to water used on common carriers engaged in interstate commerce;

WHEREAS, the 1962 Drinking Water Standards are to serve as minimum requirements to protect the health and promote the well-being of individuals and of communities;

WHEREAS, it is the desire of the American Water Works Association to support all efforts to promote health through safe water supplies and to recognize reasonable standards of quality for water furnished by public water supply systems; and,

WHEREAS, it is the hope of the American Water Works Association that its acceptance of the 1962 Drinking Water Standards will establish these standards as minimum criteria of quality for all public water supplies in the United States; now, therefore, be it

Resolved by the Officers and Directors of the American Water Works Association, that the 1962 Drinking Water Standards of the U.S. Public Health Service be accepted as minimum standards for all public water supplies.

ADVISORY COMMITTEE REPORT

Domestic water supplies should protect the health and promote the well-being of individuals and the community. In this report on the revision of the 1946 edition of the Public Health Service Drinking Water Standards, the objective of the Committee is to recommend minimum requirements for reaching this goal.

The Public Health Service Drinking Water Standards were first adopted in 1914 to protect the health of the traveling public. The general and widespread use of these Standards since that time has led to a series of revisions which have been applicable to water supplies generally. The development of atomic energy and other technological advances requires that these Standards again be revised. To carry out this revision, the Chief Sanitary Engineer of the Public Health Service appointed the Advisory Committee. A Technical Subcommittee of Public Health Service Officers and a Toxicological Task Force were established to collect information and prepare suggestions for the consideration of the Advisory Committee.

In preparing this report on the revision of the Standards, the Committee established the following guidelines:

1. The proposed standards should be discussed widely and due cognizance should be given to International and other standards of water quality before a final report is submitted.

2. A new section on radioactivity should be added.

3. Greater attention should be given to the chemical substances being encountered increasingly in both variety and quantity in water sources.

4. In establishing limits for toxic substances, intake from food and air should be considered.

5. The rationale employed in determining the various limits should be included in an appendix.

6. The proposed format, with the exceptions noted above, should not differ greatly from the present Standards.

7. The Standards should be generally acceptable and should be applicable to all public water supplies in the United States, as well as those supplies used by carriers subject to the Public Health Service regulations.

8. The following two types of limits used in previous editions should be continued:

- (a) Limits which, if exceeded, shall be grounds for rejection of the supply. Substances in this category may have adverse effects on health when present in concentrations above the limit.

- (b) Limits which should not be exceeded whenever more suitable supplies are, or can be made, available at reasonable cost. Substances in this category, when present in concentrations above the limit, are either objectionable to an appreciable number of people or exceed the levels required by good water quality control practices.

9. These limits should apply to the water at the free-flowing outlet of the ultimate consumer.

This revision of the Drinking Water Standards includes, for the first time, limiting concentrations of radioactivity in water. The effects on large popula-

defined. The limits presented herein are an effort to derive conservative values from the best information now available and may be adjusted upward or downward as new and better data become available.

The Committee has taken cognizance of the growing problem of potentially harmful chemicals in sources of drinking water. Limits for several new chemicals have been added, including a gross limit for the concentration of some types of synthetic chemicals. It was not feasible, however, to include limits for all the many chemicals that have varying degrees of toxic potential. Consideration was given to the more common chlorinated hydrocarbon and organophosphate insecticides but the information available was not sufficient to establish specific limits for these chemicals. Moreover, the concentrations of these chemicals, where tested, have been below those which would constitute a known health hazard. The Committee believes that pollution of water supplies with such contaminants can become significant and urges that the problem be kept under closer surveillance. Further, the Committee recommends that regulatory actions be taken to minimize concentrations of such chemicals in drinking water.

In view of the accelerating pace of new developments affecting water quality, the Committee recommends that a mechanism be established for continual appraisal and appropriate revision of the Standards. It also recommends that the Public Health Service intensify its continuing studies toward the development of basic information on the relationship of the biological, chemical, physical, and radiological aspects of water quality to health.

The following pages contain the Drinking Water Standards recommended by the Committee, the membership of which is listed in appendix F.

CONTENTS

	Page
Drinking water standards, 1962.....	1
Definition of terms.....	1
Adequate protection by natural means.....	1
Adequate protection by treatment.....	1
Certifying authority.....	1
Coliform group.....	1
Health hazard.....	2
Pollution.....	2
Reporting agency.....	2
Standard sample.....	2
Water supply system.....	2
Source and protection.....	2
Source and treatment.....	2
Sanitary survey.....	2
Water supply system approval.....	2
Responsibility.....	3
Bacteriological quality.....	3
Sampling.....	3
Frequency and location.....	3
Minimum number.....	3
Laboratories.....	3
Laboratory inspection.....	4
Special samples.....	5
Limits.....	5
Standard 10 ml portions.....	5
Standard 100 ml portions.....	5
Membrane filter.....	6
Physical characteristics.....	6
Sampling.....	6
Limits.....	6
Chemical characteristics.....	6
Sampling.....	6
Limits.....	7
Recommended.....	7
Grounds for rejection of supply.....	8
Fluorides.....	8
Radioactivity.....	8
Sampling.....	8
Limits.....	9
Mixtures of nuclides.....	9

recommended analytical methods	9
Standard methods	9
Barium	10
Carbon chloroform extract	10
Radioactivity	10
Selenium	10
Organisms of the coliform group	10
Appendix	11
A—Source and protection	11
B—Microbiology	11
C—Physical characteristics	21
D—Chemical characteristics	21
Alkyl benzene sulfonate	22
Arsenic	25
Barium	27
Cadmium	29
Carbon chloroform extract	31
Chloride, sulfate, and dissolved solids	32
Chromium	36
Copper	39
Cyanide	39
Fluoride	41
Iron	42
Lead	43
Manganese	46
Nitrate	47
Phenols	51
Selenium	52
Silver	52
Zinc	55
E—Radioactivity	56
F—Membership of Advisory Committee, Technical Subcommittee and Task Force on Toxicology	60

PUBLIC HEALTH SERVICE DRINKING WATER STANDARDS—1962

Standards promulgated by the Public Health Service, U.S. Department of Health, Education, and Welfare, Effective April 5, 1962, for potable water used by carriers subject to the Federal Quarantine Regulations

(Superseding Standards adopted Feb. 6, 1946)¹

1. DEFINITION OF TERMS

The terms used in these Standards are as follows:

1.1 *Adequate protection by natural means* involves one or more of the following processes of nature that produces water consistently meeting the requirements of these Standards: dilution, storage, sedimentation, sunlight, aeration, and the associated physical and biological processes which tend to accomplish natural purification in surface waters and, in the case of ground waters, the natural purification of water by infiltration through soil and percolation through underlying material and storage below the ground water table.

1.2 *Adequate protection by treatment* means any one or any combination of the controlled processes of coagulation, sedimentation, absorption, filtration, disinfection, or other processes which produce a water consistently meeting the requirements of these Standards. This protection also includes processes which are appropriate to the source of supply; works which are of adequate capacity to meet maximum demands without creating health hazards, and which are located, designed, and constructed to eliminate or prevent pollution; and conscientious operation by well-trained and competent personnel whose qualifications are commensurate with the responsibilities of the position and acceptable to the Reporting Agency and the Certifying Authority.

1.3 *Certifying Authority* means the Surgeon General of the U.S. Public Health Service or his duly authorized representatives. Reference to the Certifying Authority is applicable only for those water supplies to be certified for use on carriers subject to the Public Health Service Regulations—(42 CFR Part 72).

1.4 The *coliform group* includes all organisms considered in the coliform group as set forth in *Standard Methods for the Examination of Water and Wastewater*, current edition, prepared and published

¹ Public Health Reports 61: 371-384, March 15, 1946.

Works Association, and Water Pollution Control Federation.

1.5 *Health hazards* mean any conditions, devices, or practices in the water supply system and its operation which create, or may create, a danger to the health and well-being of the water consumer. An example of a health hazard is a structural defect in the water supply system, whether of location, design, or construction, which may regularly or occasionally prevent satisfactory purification of the water supply or cause it to be polluted from extraneous sources.

1.6 *Pollution*, as used in these Standards, means the presence of any foreign substance (organic, inorganic, radiological, or biological) in water which tends to degrade its quality so as to constitute a hazard or impair the usefulness of the water.

1.7 *Reporting Agencies* means the respective official State health agencies or their designated representatives.

1.8 *The standard sample* for the bacteriological test shall consist of:

1.81 For the bacteriological fermentation tube test, five (5) standard portions of either:

(a) ten milliliters (10 ml)

(b) one hundred milliliters (100 ml)

1.82 For the membrane filter technique, not less than fifty milliliters (50 ml).

1.9 *Water supply system* includes the works and auxiliaries for collection, treatment, storage, and distribution of the water from the sources of supply to the free-flowing outlet of the ultimate consumer.

2. SOURCE AND PROTECTION

2.1 The water supply should be obtained from the most desirable source which is feasible, and effort should be made to prevent or control pollution of the source. If the source is not adequately protected by natural means, the supply shall be adequately protected by treatment.

2.2 Frequent sanitary surveys shall be made of the water supply system to locate and identify health hazards which might exist in the system. The manner and frequency of making these surveys, and the rate at which discovered health hazards are to be removed, shall be in accordance with a program approved by the Reporting Agency and the Certifying Authority.

2.3 Approval of water supplies shall be dependent in part upon:

(a) Enforcement of rules and regulations to prevent development of health hazards;

(b) Adequate protection of the water quality throughout all parts of the system, as demonstrated by frequent surveys;

(c) Proper operation of the water supply system under the responsible charge of personnel whose qualifications are acceptable to the Reporting Agency and the Certifying Authority;

(d) Adequate capacity to meet peak demands without development of low pressures or other health hazards; and

(e) Record of laboratory examinations showing consistent compliance with the water quality requirements of these Standards.

2.4 For the purpose of application of these Standards, responsibility for the conditions in the water supply system shall be considered to be held by:

(a) The water purveyor from the source of supply to the connection to the customer's service piping; and

(b) The owner of the property served and the municipal, county, or other authority having legal jurisdiction from the point of connection to the customer's service piping to the free-flowing outlet of the ultimate consumer.

3. BACTERIOLOGICAL QUALITY

3.1 *Sampling.*

3.11 Compliance with the bacteriological requirements of these Standards shall be based on examinations of samples collected at representative points throughout the distribution system. The frequency of sampling and the location of sampling points shall be established jointly by the Reporting Agency and the Certifying Authority after investigation by either agency, or both, of the source, method of treatment, and protection of the water concerned.

3.12 The minimum number of samples to be collected from the distribution system and examined each month should be in accordance with the number on the graph in Figure I, for the population served by the system. For the purpose of uniformity and simplicity in application, the number determined from the graph should be in accordance with the following: for a population of 25,000 and under—to the nearest 1; 25,001 to 100,000—to the nearest 5; and over 100,000—to the nearest 10.

3.13 In determining the number of samples examined monthly, the following samples may be included, provided all results are assembled and available for inspection and the laboratory methods and technical competence of the laboratory personnel are approved by the Reporting Agency and the Certifying Authority:

(a) Samples examined by the Reporting Agency.

(b) Samples examined by local government laboratories.

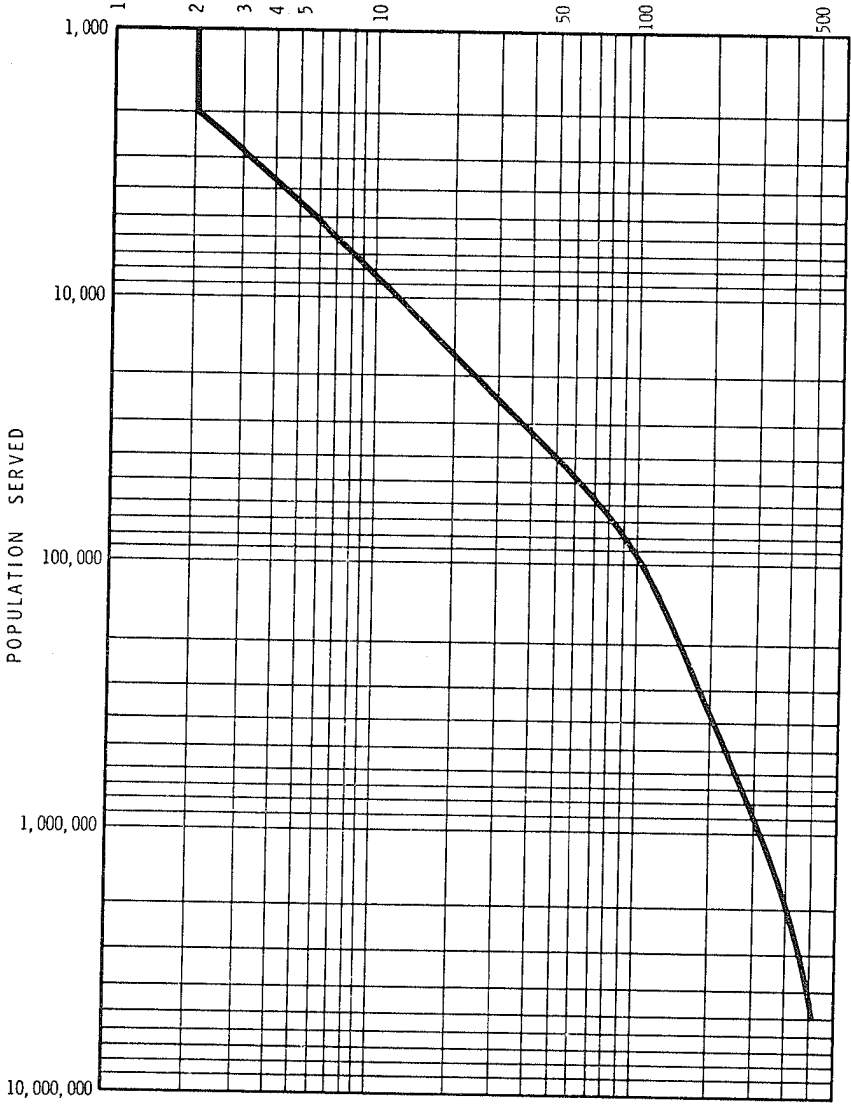


Figure 1

- (c) Samples examined by the water works authority.
- (d) Samples examined by commercial laboratories.

3.14 The laboratories in which these examinations are made and the methods used in making them shall be subject to inspection at any time by the designated representatives of the Certifying Authority and the Reporting Agency. Compliance with the

specified procedures and the results obtained shall be used as a basis for certification of the supply.

3.15 Daily samples collected following a bacteriologically unsatisfactory sample as provided in sections 3.21, 3.22, and 3.23 shall be considered as special samples and shall not be included in the total number of samples examined. Neither shall such special samples be used as a basis for prohibiting the supply, provided that: (1) When waters of unknown quality are being examined, simultaneous tests are made on multiple portions of a geometric series to determine a definitive coliform content; (2) Immediate and active efforts are made to locate the cause of pollution; (3) Immediate action is taken to eliminate the cause; and (4) Samples taken following such remedial action are satisfactory.

3.2 *Limits.*—The presence of organisms of the coliform group as indicated by samples examined shall not exceed the following limits:

3.21 When 10 ml standard portions are examined, not more than 10 percent in any month shall show the presence of the coliform group. The presence of the coliform group in three or more 10 ml portions of a standard sample shall not be allowable if this occurs:

- (a) In two consecutive samples;
- (b) In more than one sample per month when less than 20 are examined per month; or
- (c) In more than 5 percent of the samples when 20 or more are examined per month.

When organisms of the coliform group occur in 3 or more of the 10 ml portions of a single standard sample, daily samples from the same sampling point shall be collected promptly and examined until the results obtained from at least two consecutive samples show the water to be of satisfactory quality.

3.22 When 100 ml standard portions are examined, not more than 60 percent in any month shall show the presence of the coliform group. The presence of the coliform group in all five of the 100 ml portions of a standard sample shall not be allowable if this occurs:

- (a) In two consecutive samples;
- (b) In more than one sample per month when less than five are examined per month; or
- (c) In more than 20 percent of the samples when five or more are examined per month.

When organisms of the coliform group occur in all five of the 100 ml portions of a single standard sample, daily samples from the same sampling point shall be collected promptly and examined

show the water to be of satisfactory quality.

3.23 When the membrane filter technique is used, the arithmetic mean coliform density of all standard samples examined per month shall not exceed one per 100 ml. Coliform colonies per standard sample shall not exceed 3/50 ml, 4/100 ml, 7/200 ml, or 13/500 ml in:

- (a) Two consecutive samples;
- (b) More than one standard sample when less than 20 are examined per month; or
- (c) More than five percent of the standard samples when 20 or more are examined per month.

When coliform colonies in a single standard sample exceed the above values, daily samples from the same sampling point shall be collected promptly and examined until the results obtained from at least two consecutive samples show the water to be of satisfactory quality.

4. PHYSICAL CHARACTERISTICS

4.1 *Sampling.*—The frequency and manner of sampling shall be determined by the Reporting Agency and the Certifying Authority. Under normal circumstances samples should be collected one or more times per week from representative points in the distribution system and examined for turbidity, color, threshold odor, and taste.

4.2 *Limits.*—Drinking water should contain no impurity which would cause offense to the sense of sight, taste, or smell. Under general use, the following limits should not be exceeded:

Turbidity	5 units
Color	15 units
Threshold Odor Number	3

5. CHEMICAL CHARACTERISTICS

5.1 *Sampling.*

5.11 The frequency and manner of sampling shall be determined by the Reporting Agency and the Certifying Authority. Under normal circumstances, analyses for substances listed below need be made only semiannually. If, however, there is some presumption of unfitness because of the presence of undesirable elements, compounds, or materials, periodic determinations for the suspected toxicant or material, should be made more frequently and an exhaustive sanitary survey should be made to determine the source of the pollution. Where the concentration of a substance is not expected to increase in processing and distribution, available and acceptable source water analyses performed in accordance with standard methods may be used as evidence of compliance with these Standards.

5.12 Where experience, examination, and available evidence indicate that particular substances are consistently absent from a water supply or below levels of concern, semiannual examinations for those substances may be omitted when approved by the Reporting Agency and the Certifying Authority.

5.13 The burden of analysis may be reduced in many cases by using data from acceptable sources. Judgment concerning the quality of water supply and the need for performing specific local analyses may depend in part on information produced by such agencies as: (1) The U.S. Geological Survey, which determines chemical quality of surface and ground waters of the United States and publishes these data in "Water Supply Papers" and other reports, and (2) The U.S. Public Health Service which determines water quality related to pollution (or the absence of pollution) in the principal rivers of the Nation and publishes these data annually in "National Water Quality Network." Data on pollution of waters as measured by carbon chloroform extracts (CCE) may be found in the latter publication.

5.2 *Limits.*—Drinking water shall not contain impurities in concentrations which may be hazardous to the health of the consumers. It should not be excessively corrosive to the water supply system. Substances used in its treatment shall not remain in the water in concentrations greater than required by good practice. Substances which may have deleterious physiological effect, or for which physiological effects are not known, shall not be introduced into the system in a manner which would permit them to reach the consumer.

5.21 The following chemical substances should not be present in a water supply in excess of the listed concentrations where, in the judgment of the Reporting Agency and the Certifying Authority, other more suitable supplies are or can be made available.

<i>Substance</i>	<i>Concentration in mg/l</i>
Alkyl Benzene Sulfonate (ABS)-----	0.5
Arsenic (As)-----	0.01
Chloride (Cl)-----	250.
Copper (Cu)-----	1.
Carbon Chloroform Extract (CCE)-----	0.2
Cyanide (CN)-----	0.01
Fluoride (F)-----	(See 5.23)
Iron (Fe)-----	0.3
Manganese (Mn)-----	0.05
Nitrate ¹ (NO ₃)-----	45.
Phenols-----	0.001
Sulfate (SO ₄)-----	250.
Total Dissolved Solids-----	500.
Zinc (Zn)-----	5.

¹ In areas in which the nitrate content of water is known to be in excess of the listed

concentrations listed shall constitute grounds for rejection of the supply:

<i>Substance</i>	<i>Concentration in mg/l</i>
Arsenic (As) -----	0.05
Barium (Ba) -----	1.0
Cadmium (Cd) -----	0.01
Chromium (Hexavalent) (Cr ⁺⁶) -----	0.05
Cyanide (CN) -----	0.2
Fluoride (F) ----- (See 5.23)	0.05
Lead (Pb) -----	0.01
Selenium (Se) -----	0.01
Silver (Ag) -----	0.05

5.23 *Fluoride*.—When fluoride is naturally present in drinking water, the concentration should not average more than the appropriate upper limit in Table I. Presence of fluoride in average concentrations greater than two times the optimum values in Table I shall constitute grounds for rejection of the supply.

Where fluoridation (supplementation of fluoride in drinking water) is practiced, the average fluoride concentration shall be kept within the upper and lower control limits in Table I.

TABLE I.

Annual average of maximum daily air temperatures ¹	Recommended control limits— Fluoride concentrations in mg/l		
	Lower	Optimum	Upper
50.0–53.7 -----	0.9	1.2	1.7
53.8–58.3 -----	0.8	1.1	1.5
58.4–63.8 -----	0.8	1.0	1.3
63.9–70.6 -----	0.7	0.9	1.2
70.7–79.2 -----	0.7	0.8	1.0
79.3–90.5 -----	0.6	0.7	0.8

¹ Based on temperature data obtained for a minimum of five years.

In addition to the sampling required by paragraph 5.1 above, fluoridated and defluoridated supplies shall be sampled with sufficient frequency to determine that the desired fluoride concentration is maintained.

6. RADIOACTIVITY

6.1 *Sampling*.

6.11 The frequency of sampling and analysis for radioactivity shall be determined by the Reporting Agency and the Certifying Authority after consideration of the likelihood of significant amounts being present. Where concentrations of Ra²²⁶ or Sr⁹⁰ may vary considerably, quarterly samples composited over a period of three months are recommended. Samples for determina-

tion of gross activity should be taken and analyzed more frequently.

6.12 As indicated in paragraph 5.1, data from acceptable sources may be used to indicate compliance with these requirements.

6.2 *Limits.*

6.21 The effects of human radiation exposure are viewed as harmful and any unnecessary exposure to ionizing radiation should be avoided. Approval of water supplies containing radioactive materials shall be based upon the judgment that the radioactivity intake from such water supplies when added to that from all other sources is not likely to result in an intake greater than the radiation protection guidance² recommended by the Federal Radiation Council and approved by the President. Water supplies shall be approved without further consideration of other sources of radioactivity intake of Radium-226 and Strontium-90 when the water contains these substances in amounts not exceeding 3 and 10 $\mu\mu\text{c}$ /liter, respectively. When these concentrations are exceeded, a water supply shall be approved by the certifying authority if surveillance of total intakes of radioactivity from all sources indicates that such intakes are within the limits recommended by the Federal Radiation Council for control action.

6.22 In the known absence³ of Strontium-90 and alpha emitters, the water supply is acceptable when the gross beta concentrations do not exceed 1,000 $\mu\mu\text{c}$ /liter. Gross beta concentrations in excess of 1,000 $\mu\mu\text{c}$ /liter shall be grounds for rejection of supply except when more complete analyses indicates that concentrations of nuclides are not likely to cause exposures greater than the Radiation Protection Guides as approved by the President on recommendation of the Federal Radiation Council.

7. RECOMMENDED ANALYTICAL METHODS

7.1 Analytical methods to determine compliance with the requirements of these Standards shall be those specified in *Standard Methods for the Examination of Water and Wastewater*, Am. Pub. Health Assoc., current edition and those specified as follows.

² The Federal Radiation Council, in its Memorandum for the President, Sept. 13, 1961, recommended that "Routine control of useful applications of radiation and atomic energy should be such that expected average exposures of suitable samples of an exposed population group will not exceed the upper value of Range II (20 $\mu\mu\text{c}$ /day of Radium-226 and 200 $\mu\mu\text{c}$ /day of Strontium-90)."

³ Absence is taken here to mean a negligibly small fraction of the above specific limits, where the limit for unidentified alpha emitters is taken as the listed limit for Radium-226.

Samples, Water Supply Paper 1404, Rainwater, p. 11. and Thatcher, L. L., U.S. Geological Survey, Washington, D.C.

7.3 Carbon Chloroform Extract (CCE)—*Manual for Recovery and Identification of Organic Chemicals in Water*, Middleton, F. M., Rosen, A. A., and Burttschell, R. H., Robert A. Taft Sanitary Engineering Center, Public Health Service, Cincinnati, Ohio, *Tentative Method for Carbon Chloroform Extract (CCE) in Water*, J. Am. Water Works A. 54: 223-227, Feb. 1962.

7.4 Radioactivity—*Laboratory Manual of Methodology, Radio-nuclide Analysis of Environmental Samples, Technical Report R59-6*, Robert A. Taft Sanitary Engineering Center, Public Health Service, Cincinnati, Ohio; and *Methods of Radiochemical Analysis Technical Report No. 173*, Report of the Joint WHO-FAO Committee, 1959, World Health Organization.

7.5 Selenium—*Suggested Modified Method for Colorimetric Determination of Selenium in Natural Water*, Magin, G. B., Thatcher, L. L. Rettig, S., and Levine, H., J. Am. Water Works Assoc. 52, 1199 (1960).

7.6 Organisms of the coliform group—All of the details of techniques in the determination of bacteria of this group, including the selection and preparation of apparatus and media, the collection and handling of samples and the intervals and conditions of storage allowable between collection and examination of the water sample, shall be in accordance with *Standard Methods for the Examination of Water and Wastewater*, current edition, and the procedures shall be those specified therein for:

7.61 The Membrane Filter Technique, Standard Test, or

7.62 The Completed Test, or

7.63 The Confirmed Test, procedure with brilliant green lactose bile broth,⁴ or

7.64 The Confirmed Test, procedure with Endo or eosin methylene blue agar plates.⁴

⁴The Confirmed Test is allowed, provided the value of this test to determine the sanitary quality of the specific water supply being examined is established beyond reasonable doubt by comparisons with Completed Tests performed on the same water supply.

APPENDIX

BACKGROUND USED IN DEVELOPING THE 1962 DRINKING WATER STANDARDS

The Public Health Service Drinking Water Standards of 1962 have been predicated upon the best and latest information available at the time of their promulgation. The concepts and rationale included in this Appendix were used in making this revision and should enable those whose responsibility it is to interpret, apply, or enforce the Standards to do so with understanding, judgment, and discretion.

A—Source and Protection

B—Microbiology

C—Physical Characteristics

D—Chemical Characteristics

E—Radioactivity

F—Membership of Advisory Committee.

Technical Subcommittee, and Task Force
on Toxicology

A—SOURCE AND PROTECTION OF SUPPLY

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 which will be in full compliance with the provisions of these Standards and will be esthetically acceptable to the consumer.

Production of water supplies which poses no threat to the consumer's health depends upon continuous protection. Because of human frailties associated with this protection, priority should be given to selection of the purest source. Polluted sources should be used only when other sources are economically unavailable and then only when the provision of personnel, equipment, and operating procedures can be depended upon to purify and otherwise protect the drinking water supply continuously.

Well waters obtained from aquifers beneath impervious strata, and not connected with fragmented or cavernous rock, are usually considered sufficiently protected to preclude need for purification. How-

and the resulting hazards require special surveillance. An illustration of such pollution is the presence of chemical pollutants originating either from sewage or industrial effluents. Surveillance of the safety of these water supplies should include chemical, physical, radiological, and biological examination.

Surface waters are subjected to increasing pollution and although some surface waters may be sufficiently protected to warrant their use as a supply without coagulation and filtration, they are becoming rare. Surface waters should never be used without being disinfected. Because of the increasing hazards of pollution, the use of surface waters without coagulation and filtration must be accompanied by intensive surveillance of the quality of the raw water and the disinfected supply in order to assure constant protection. This surveillance should include sanitary survey of the source and water handling, as well as biological, radiological, physical, and chemical examination of the supply.

The degree of treatment should be determined by the health hazards involved and the quality of 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, 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. When used, the source should be under continuous surveillance to assure adequacy of treatment in meeting the hazards of changing pollution conditions.

The adequacy of treatment should be judged, in part, upon a record of the quality of water produced by the treatment plant and the relation of this quality to the requirements of these Standards. 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 upon the protection of the water in the distribution system as well as protection of the source and by treatment. Minimum protection in the distribution system should include programs which result in the *provision* of sufficient and safe materials and equipment to treat and distribute the water; *disinfection*

¹ See reference to relationship of chlorine residual and contact time required to kill viruses, in section on Microbiology.

of water mains, storage facilities, and other equipment after each installation, repair, or other modification which 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 supply 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.

B—MICROBIOLOGY

BACTERIOLOGICAL QUALITY

The bacteriological requirements for drinking water as specified by the 1946 Drinking Water Standards have been discussed extensively (1).¹

Coliform Group

Of the two bacteriological examinations—(a) agar plate count for 24 hours at 35° C, and (b) quantitative estimation of the coliform group which have come to be recognized generally—the test for organisms of the coliform group is almost universally conceded to be the most significant. The plate count at 35° C or (20° C) incubation temperature is not required in the definition of a safe standard for potable waters but is useful as a routine quality control test in the various water treatment procedures and as a method for estimating the sanitary conditions of basins, filters, etc.

It does not seem advisable to repeat extensive discussions (1, 2, 3) of the principles involved in the quantitative interpretation of fermentation tests according to the "most probable number" concept in multiple portions of equal volume and in portions constituting a geometric series.

Discussions of the principles involved in the quantitative interpretation of membrane filter procedure results and as compared to the "most probable number" concept are available in the literature (4, 5, 6).

¹ Footnotes cited will be found at end of Microbiology Section.

The coliform group, as specified in U.S. Public Health Service Drinking Water Standards (1)³ is defined in *Standard Methods* (2): "The coliform group includes all of the aerobic and facultative anaerobic, Gramnegative, nonspore-forming, rod-shaped bacilli which ferment lactose with gas formation within 48 hours at 35° C."

The coliform group includes organisms that differ in biochemical and serologic characteristics and in their natural sources and habitats. *Escherichia coli* is characteristically an inhabitant of human and animal intestines (3-6). *Aerobacter aerogenes* and *Aerobacter cloacae* are frequently found on various types of vegetation (7-9) and in materials used in joints and valves of pumps and in pipelines (10-11). The intermediate-aerogenes-cloacae (I.A.C.) subgroups may be found in fecal discharges but usually in smaller numbers than *Esch. coli*. *Aer. aerogenes* and intermediate types of organisms are commonly present in soil (12-14) and in waters polluted sometime in the past. Another subgroup comprises plant pathogens (15) and other organisms of indefinite taxonomy about whose habitat information is limited. All the subgroups may be found in sewage and in polluted waters. *Esch. coli* is therefore frequently referred to as "fecal coli"; the I.A.C. group as "nonfecal". It must be remembered, however, that these terms are only relative.

Survival Times

Available information indicates that 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 *Esch. 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 recent from 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.

² This article, authored by Paul W. Kabler and Harold F. Clark, was published in J. Am. Water Works A. and is reprinted as a part of this appendix by permission of the AWWA.

³ References cited in this article will be found at the end of the article.

Differentiation of Organisms

Because various members of the coliform group normally grow in diverse natural habitats, attempts have been made to differentiate the population in polluted waters according to their original sources. In his pioneer work, MacConkey (23, 24) defined the aerogenes group in terms of certain fermentation characteristics, ability to produce indole, and reaction in the Voges-Proskauer test. Rogers, Clark, and Davis, (25) Clark and Lubs, (26) Koser, (27) and others contributed to the development of techniques and laboratory data that differentiated the coliform group on the basis of indole production, methyl red and Voges-Proskauer reactions, and citrate utilization (IMViC tests) into the *Esch. coli*, aerogenes, intermediate, and irregular subgroups. Hajna and Perry (28) and Vaughn, Levine, and Smith (29) further developed the Eijkman (30) test to distinguish organisms of fecal origin from those of nonfecal origin by increased temperature incubation. Clark and associates (31, 32) have reported additional data indicating the usefulness of such tests in sanitary investigations.

Sanitary Significance

Information on the sanitary significance of the various types of coliform organisms is incomplete. In relation to untreated waters, however, the present position may be thus stated:

Fecal coliform organisms (*Esch. coli*) may be considered indicators of recent fecal pollution. No satisfactory method is currently available for differentiating fecal coliform organisms of human and animal origin. Therefore, it is necessary to consider all fecal coliform organisms as indicative of dangerous contamination.

In the absence of fecal coliform organisms, the presence of I.A.C. group organisms in untreated waters may be the result of relatively less recent fecal pollution, soil runoff water, or infrequently, fecal pollution containing only the I.A.C. group.

In general terms, the presence of fecal coliform organisms indicates recent and possibly dangerous pollution. The presence of I.A.C. organisms suggests less recent pollution or reveals the existence of defects in water treatment or distribution.

Summary

The presence of any type of coliform organism in treated drinking water suggests either inadequate treatment or access of undesirable materials to the water after treatment. Although there are some differences between strain and subgroup organisms with regard to survival under natural conditions and resistance to chlorination, in

terns in the same order of magnitude. The presence of coliform organisms (as defined earlier) in treated water calls for definitive action for their elimination.

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 indication of protection provided, the coliform group is preferred to fecal coliform organisms (*Esch. coli*). Whether these considerations can be extended to include rickettsial and viral organisms has not been definitely determined.

REFERENCES

1. Drinking Water Standards—1946. Jour. AWWA. 38: 361 (March 1946)
Pub. Health Repts., 61: 371 (1946).
2. Standard Methods for the Examination of Water and Wastewater. APHA, AWWA, and WPCF, New York 11th ed., 1960).
3. Escherich, T. Intestinal Bacteria in Newborn and Suckling Children. Fortschr. Med. (Ger.), 3: 515, 547 (1885).
4. Rogers, L. A., Clark, W. M., and Evans, A. C. The Characteristics of Bacteria of the Colon Type Found in Bovine Feces. J. Infectious Diseases, 15: 99 (1915).
5. Parr, L. W. The Occurrence and Succession of Coliform Organisms in Human Feces. Am. J. Hyg., 27: 67 (1938).
6. Foote, H. B. The Possible Effects of Wild Animals on the Bacterial Pollution of Water. Jour. AWWA, 29: 72 (January 1937).
7. Rogers, L. A., Clark, W. M., and Evans, A. C. The Characteristics of Bacteria of the Colon Type Occurring on Grains, J. Infectious Diseases, 17: 137 (1915).
8. Thomas, S. B., and McQuillin, J. Coli-aerogenes Bacteria Isolated From Grass. J. Appl. Bacteriol., 15: 41 (1952).
9. Thomas, S. B., and Hobson, P. M. Coli-aerogenes Bacteria Isolated From Ears and Panicles of Cereal Crops. J. Appl. Bacteriol., 18: 1 (1955).
10. Caldwell, E. L., and Parr, L. W. Pump Infection Under Normal Conditions in Controlled Experimental Fields. Jour. AWWA, 25: 1107 (August 1933).
11. Rapp, W. M., and Weir, P. Cotton Caulking Yarn. Jour. AWWA, 26: 743 (June 1934).
12. Chen, C. C., and Retger, L. F. A Correlation Study of the Colon-aerogenes Group of Bacteria, With Special Reference to the Organisms Occurring in the Soil. J. Bacteriol., 5: 253 (1920).
13. Bardsley, D. A. The Distribution and Sanitary Significance of *E. coli*, *B. lactis Aerogenes*, and Intermediate Types of Coliform Bacilli in Water, Soil, Feces, and Ice Cream. J. Hyg., 34: 38 (1934).
14. Frank, N., and Skinner, C. E. Coli-aerogenes Bacteria in Soil. J. Bacteriol. 42: 143 (1941).
15. Elrod, R. P. The *Erwinia*-Coliform Relationship. J. Bacteriol., 44: 433 (1942).
16. Parr, L. W. Viability of Coli-aerogenes Organisms in Cultures and in Various Environments. J. Infectious Diseases, 60: 291 (1937).

17. Platt, A. E. The Viability of *Bact. coli* and *Bact. aerogenes* in Water: A Method for the Rapid Enumeration of These Organisms. *J. Hyg.*, 35: 437 (1935).
18. Taylor, C. B. The Ecology and Significance of the Different Types of Coliform Bacteria Found in Water. *J. Hyg.*, 42: 23 (1942).
19. Tonney, F. O., Greer, F. E., and Danforth, T. F. The Minimal "Chlorine Death Point" of Bacteria. *Am. J. Public Health*, 18: 1259 (1928).
20. Heathman, L. S., Pierce, G. O., and Kabler, P. W. Resistance of Various Strains of *E. typhi* and *Coli-aerogenes* to Chlorine and Chloramine. *Pub. Health Repts.*, 51: 1367 (1936).
21. Betterfield, C. T., *et al.* Influence of pH and Temperature on the Survival of Coliforms and Enteric Pathogens When Exposed to Free Chlorine. *Pub. Health Repts.*, 58: 1837 (1943).
22. Kabler, P. W. Relative Resistance of Coliform Organisms and Enteric Pathogens in the Disinfection of Water With Chlorine. *Jour. AWWW*, 43: 553 (July 1951).
23. MacConkey, A. Lactose-Fermenting Bacteria in Feces. *J. Hyg.*, 5: 333 (1905).
24. MacConkey, A. Further Observations on the Differentiation of Lactose-Fermenting Bacteria, With Special Reference to Those of Intestinal Origin. *J. Hyg.*, 9: 86 (1909).
25. Rogers, L. A., Clark, W. M., and Davis, B. J. The Colon Group of Bacteria. *J. Infectious Diseases*, 14: 411 (1914).
26. Clark, W. M., and Lubs, W. A. The Differentiation of Bacteria of the Colon-Aerogenes Family by the Use of Indicators. *J. Infectious Diseases*, 17: 160 (1915).
27. Koser, S. A. Differential Tests for Colon-Aerogenes Group in Relation to Sanitary Quality of Water. *J. Infectious Diseases*, 35: 14 (1924).
28. Hajna, A. A., and Perry, C. A. A Comparison of the Eijkman Test With Other Tests for Determining *E. coli*. *J. Bacteriol.*, 30: 479 (1935).
29. Vaughn, R. H., Levine, M., and Smith, H. A. A Buffered Boric Acid Lactose Medium for Enrichment and Presumptive Identification of *Escherichia coli*. *Food Research*, 16: 10 (1951).
30. Eijkman, C. Fermentation Analysis at 46° C as an Aid in Drinking Water Examination. *Zentr. Bakteriolog. Parasitenk. (Ger.)*, 37: 742 (1904).
31. Clark, H. F., *et al.* The Coliform Group. I. The Boric Acid Lactose Broth reaction of Coliform IMViC Types. *Appl. Microbiol.*, 5: 396 (1957).
32. Geldreich, E. E., *et al.* The Coliform Group. II. Reactions in EC Medium at 45° C. *Appl. Microbiol.*, 6: 347 (1958).

Fecal Streptococci as Indicators of Pollution

Fecal streptococci appear to be characteristic of fecal pollution, being consistently present in both the feces of all warm-blooded animals and in the environment associated with animal discharges (7, 8, 9). They do not multiply in streams or surface waters to yield overgrowths as sometime occur with the coliform group. So far as is currently known, they are rare in soil or on vegetation not subject to continued fecal pollution (10). Therefore, the presence of fecal streptococci in a water indicates fecal pollution with the density equal to those originally present or reduced by natural purification processes.

the source of the fecal pollution can be estimated. For example: predominating strains of *Streptococcus fecalis* indicate human fecal pollution; *S. bovis* and *S. acidominimus* predominate in bovine excrement but are rarely present in human feces (about 0.4 percent of *Streptococcus* density); while in porcine excretal material, the species are about one-third *S. fecalis* (atypical types), one-third *S. bovis* and one-fourth *S. acidominimus*. Thus, it may be possible to separate human from other animal pollution and further studies of various animal excrement may permit further interpretations.

Improved methods and media are urgently needed for the analysis of streptococcal group. Investigations on the distribution of the various species of streptococci in nature should be diligently pursued. Azide Dextrose—EVA—(14, 15, 16, 17) multiple-tube procedure yields good results with the streptococci species present in humans but is relatively inefficient for the analysis of fecal streptococci present in other animals. The Slanetz MF (18) procedure yields a few more species. The KF streptococcus (19) medium and biochemical test procedures appear to offer promise of a more complete enumeration of fecal streptococci.

The streptococcus group in potable waters which are not chlorinated or which are in surface waters to be treated, appears to have certain advantages as indicator organisms in the interpretation of the type of pollution present. However, they do not appear to have any advantage over the coliform group in the examination of adequately chlorinated potable water.

Enteric Viruses in Water

Enteric viruses (infectious hepatitis (20), poliomyelitis, Coxsackie, and ECHO) should be considered as waterborne infectious agents. Epidemiological evidence indicates that treated water from a public supply is not a frequent carrier of such organisms. Clarke and Chang (21) have recently reviewed both the published reports on outbreaks of infectious hepatitis and poliomyelitis and laboratory evidence on the resistance of various enteric viruses.

An estimated 20,000 to 40,000 cases of infectious hepatitis were reported in Delhi, India (1955-56) (22), attributable to treated municipal water supply. The outbreak was not accompanied by noticeable increase of typhoid fever and other intestinal diseases. This indicates that, in practice, the virus of infectious hepatitis is more resistant to chlorine (chloramine) than are vegetative bacteria. On the strength of epidemiological evidence, poliomyelitis outbreak in Edmonton, Canada (23) was attributed to the drinking (treated)

water supply. Kelly and Sanderson showed (1958) (24) that inactivation of enteric viruses (Polio virus I:MK 500 and Mahoney and Coxsackie B5) in water at pH 7, and 25° C requires a minimum free residual chlorine of 0.3 mg/1 for at least 30 minutes. At higher pH levels or lower temperatures, either more chlorine or longer contact time is required. The same authors (1960) (25) showed that for the same viruses in water at 25° C and a pH of 7, a concentration of at least 9 mg/1 combined residual chlorine is necessary to inactivate with a contact period of 30 minutes; of 6 mg/1 with a 1-hour contact time; 0.5 mg/1 with a contact period of more than 7 hours.

Sabin found 10^6 TcD₅₀ of polio virus per gram of feces in human stools. Neefe *et al.* estimated there were 10^4 to 10^5 infectious doses of infectious hepatitis virus per gram of feces from human cases. Other estimations of viral content in feces have been in the same order of magnitude or less. Human feces normally contain 10^6 to 10^{10} coliform bacteria per gram. An estimated mean value is 10^8 coliforms per gram. Because nearly all feces contain coliform organisms and only a relatively small portion (2 to 20 percent) contribute pathogenic virus (26, 27, 28), domestic sewage normally contains approximately 10,000 times as many coliforms as virus. Virus populations in sewage and polluted waters are subject to die-aways due to aging, adsorption, and sedimentation, dilution, and various undetermined causes. It is likely, therefore, that the virus content of polluted surface waters, wells, etc., is quite low when judged on the basis of the coliform-virus ratio. This relatively low virus content may account for the apparent paucity of virus infections attributed to such sources. The possibility of waterborne epidemics remains, and the efficacy of various water treatment processes including high free chlorine dosages and increased contact times should be further investigated.

Virology techniques have not yet been developed to a point where virus enumerations can be recommended as a routine procedure in microbiological examination of drinking water. Development of methodologies to permit such examination is currently under investigation but may require extended periods of study before perfection. The objectives of a research program under which several laboratories could cooperate should include the accumulation of sufficient data and the development of methodologies on which to base standards. In the interim, control laboratories having access to facilities for virus isolation and identification should be encouraged to utilize the best available procedures for evaluating the occurrence of enteroviruses in treated waters.

1. U.S. Public Health Service. Manual of recommended water-sanitation practice. Public Health Service Pub. No. 525, Washington, D.C., U.S. Government Printing Office. 1946.
2. Anon. Report of the advisory committee on official water standards, appendix III. Coli densities as determined from various types of samples. Pub. Health Rep. 40: 704-716 (1925).
3. Woodward, R. L. How probable is the most probable number? J. Am. Water Works A. 49: 1060-1068, August 1957.
4. Geldreich, E. E., Kabler, P. W., Jeter, H. L., and Clark, H. F. A delayed incubation membrane filter test for coliform bacteria in water. Am. J. Pub. Health 45: 1463-1474, November 1955.
5. Thomas, H. A., Jr., Woodward, R. L., and Kabler, P. W. Use of molecular filter membrane for water potability control. J. Am. Water Works A. 48: 1391-1402, November 1956.
6. Clark, H. F., Kabler, P. W., and Geldreich, E. E. Advantages and limitations of the membrane filter procedure. Water and Sewage Works 104: 385-387, September 1957.
7. Sherman, J. M. The streptococci. Bact. Rev. 1: 3-97, December 1937.
8. Hajna, A. A., and Perry, C. A. Comparative study of presumptive and confirmative media for bacteria of the coliform group and for fecal streptococci. Am. J. Pub. Health 33: 550-556, May 1943.
9. Winter, C. E., and Sandholzer, L. A. Studies on the fecal streptococci. Fishery Leaflet No. 201. Washington, D.C. Fish and Wildlife Service, Department of Interior.
10. Dible, J. H. The enterococcus and the faecal streptococci; their properties and relations. J. Path. Bact. 24: 3 (1921).
11. Cooper, K. E., and Ramadan, F. M. Studies in the differentiation between human and animal pollution by means of faecal streptococci. J. Gen. Microbiol. 12: 180-189, April 1955.
12. Barnes, E. M., and Ingram, M. The identity and origin of faecal streptococci in canned hams. Ann. Inst. Pasteur Lille 7: 115-119 (1955).
13. Moore, B. Streptococci and food poisoning. J. Appl. Bact. 18: 606-618, December 1955.
14. Litsky, W., Mallmann, W. L., and Fifield, C. W. Comparison of the most probable numbers of escherichia coli and enterococci in river waters. Am. J. Pub. Health 45: 1049-1053, August 1955.
15. Litsky, W., Mallmann, W. L., and Fifield, C. W. A new medium for the detection of enterococci in waters. Am. J. Pub. Health 43: 873-879, October 1953.
16. Litsky, W., Rosenbaum, M. J., and France, R. L. A comparison of the most probable numbers of coliform bacteria and enterococci in raw sewage. Appl. Microbiol. 1: 247-250, September 1953.
17. Croft, C. C. A comparative study of media for detection of enterococci in water. Am. J. Pub. Health 49: 1379-1387, October 1959.
18. Slanetz, L. W., and Bartley, C. H. Numbers of enterococci in water, sewage, and feces determined by the membrane filter technique with an improved medium. J. Bact. 74: 591-595, November 1957.
19. Kenner, B. A., Clark, H. F., and Kabler, P. W. Fecal streptococci. II. Quantification of streptococci in feces. Am. J. Pub. Health 50: 1553-1559, October 1960.

20. Neefe, J. R., and Stokes, J., Jr. An epidemic of infectious hepatitis apparently due to a waterborne agent. *J. A.M.A.* 128: 1063-1075, August 11, 1945.
21. Clarke, N. A., and Chang, S. L. Enteric viruses in water. *J. Am. Water Works A.* 51: 1299-1317, October 1959.
22. Viswanathan, R. Epidemiology. *Indian J. Med. Res.* 45: 1-29, January 1957 (Supplementary Number).
23. Little, G. M. Poliomyelitis and water supply. *Canad. J. Pub. Health* 45: 100-102, March 1954.
24. Kelly, S., and Sanderson, W. W. The effect of chlorine in water on enteric viruses. *Am. J. Pub. Health* 48: 1323-1334, October 1958.
25. Kelly, S., and Sanderson, W. W. The effect of chlorine in water on enteric viruses. II. The effect of combined chlorine on poliomyelitis and Coxsackie viruses in sewage. *Am. J. Pub. Health* 50: 14-20, January 1960.
26. Kelly, S., Winsser, J., and Winkelstein, W., Jr. Poliomyelitis and other enteric viruses in sewage. *Am. J. Pub. Health* 47: 72-77, January 1957.
27. Melnick, J. L., Emmons, J., Coffey, J. H., and Schoof, H. Seasonal distribution of Coxsackie viruses in urban sewage and flies. *Am. J. Hyg.* 59: 164-184, March 1954.
28. Bancroft, P. M., Engelhard, W. E., and Evans, C. A. Poliomyelitis in Huskerville (Lincoln) Nebraska—Studies indicating a relationship between clinically severe infection and proximate fecal pollution of water. *J. A.M.A.* 164: 836-847, June 22, 1957.

C—PHYSICAL CHARACTERISTICS

Turbidity, color, and odor requirements are easily attained during general use by properly designed and operated treatment plants and distribution systems. Failure to meet these requirements is an indication of either inadequate treatment facilities or improper operation of the system. Supplies used without treatment should also meet these requirements. It should not be implied that these turbidity limits represent acceptable effluent standards for water treatment plants. Such plants should routinely produce water with a turbidity of less than one unit.

Although these tests do not directly measure the safety of the water, they are related to consumer acceptance of the water. The levels of 5 units of turbidity, 15 units of color, and a threshold odor number of 3 are levels at which these characteristics become objectionable to a considerable number of people. Experience has shown that under such circumstances, many people turn to alternate supplies which may be less safe.

D—CHEMICAL CHARACTERISTICS

INTRODUCTION

In its report, the Advisory Committee defined guidelines which were used in developing the standards. The following pages present de-

limits.

In general, "grounds for rejection" 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. The Committee has attempted to set 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 of man.

The limits, which should not be exceeded when more suitable water supplies can be made available, are based on factors which render a supply less desirable for use. These considerations relate to materials which impart objectionable taste and odor to water, render it economically or aesthetically inferior, or are toxic to fish or plants. In one instance (Carbon Chloroform Extract), the limit is expected also to have utility as a generalized procedure for limiting toxic exposure to organic chemicals.

The Drinking Water Standards are regarded as a standard of quality which is generally attainable by good water quality control practices. Poor practice is an inherent health hazard. It has been the policy of the Committee to set limits which 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 which might enter a public water supply. While the Committee is fully cognizant of the need for continued attention to chemical contaminants of water, the Standards are limited to recognized need. Standards for innumerable substances would require an impossible burden of analytical examination.

ALKYL BENZENE SULFONATE (Anionic Surfactant)

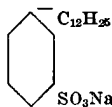
The surfactant is a synthetic organic chemical having high residual affinity at one end of its molecule and low residual affinity at the other. Its vigorous surface activity justifies not only its name but its use as a principal ingredient of modern household detergents. Surfactants may be divided into two broad chemical classifications, ionic and non-ionic. Ionic types may be either anionic (-) or cationic (+). Alkyl benzene sulfonate is a typical anionic surfactant.

Contamination of drinking water supplies with surfactants results from their disposal, as household and industrial wastes, into sources of raw water. Such contamination is appearing in supplies from both surface and ground waters. Other potential sources of human intake

of surfactants are inadequately rinsed cooking and household utensils and dinnerware and food.

More than 75 percent of the surfactants in household detergents are of the anionic type. Alkyl aryl sulfonates account for almost three-quarters of these, the remainder being mostly alkyl sulfates. Next in extent of such use are the nonionics, the cationics making up only a small percentage (1). Hence, the anionic group comprises the specific materials of this type most apt to be present in raw water supplies if any at all are present (2). The principal agent in this anionic group is the sodium salt of the sulfonation product of dodecylbenzene, an alkyl aryl sulfonate, termed alkyl benzene sulfonate or simply ABS (3). It is largely for this reason that the degree of detergent contamination is established currently in terms of the concentration of alkyl benzene sulfonate (ABS), for which quantitative determination can be made by practical and reasonably satisfactory laboratory procedures.

In general, commercial ABS is produced by condensing polypropylene (typically the tetrapolymer) with benzene, followed by a distillation cut to yield a reproducible product. ABS is thus a controlled mixture of isomers and homologues of dodecylbenzene, which upon sulfonation may be represented by the following typical structure:



Concentrations of anionic surfactants found in drinking waters have ranged from 0 to 2.6 mg/1 in well water supplies and from 0 to 5 mg/1 in river water supplies. In one instance, a municipal water supply contained 5 mg/1 when a period of drought necessitated use of an impounded, highly purified sewage treatment plant effluent as a raw water supply (4).

In a study (5) made for the purpose, 10 percent of those using water containing less than 1 mg/1 anionic sulfonated detergents complained of an off-taste, whereas all those using water containing 1.5 mg/1 complained of an off-taste. Frothing was also a common complaint occurring most frequently at concentrations of 1 mg/1 and above. The off-taste has been described as oily, fishy, or perfume-like (5). ABS itself is essentially odorless. The odor and taste characteristics are likely to rise from the degradation of products of other wastes rather than from ABS. The concentration of ABS in municipal sewage is of the order of 10 mg/1. Thus waters containing ABS are likely to be at least 10 percent of sewage origin for each mg ABS/1 present.

From the basic toxicologic point of view, there are two reports which are especially pertinent to the present consideration.

Committee of the Food and Nutrition Board, National Research Council, published a comprehensive report in 1956 (6) bearing on the question of surfactants in food. Reviewing extensively the acute and chronic toxicity studies which have been reported on these chemicals, they found that there appears to be little specific relationship of toxicity to surface activity (reduction of interfacial tension). In conclusion, it was stated that:

(a) There are no toxic effects common to all surfactants.

(b) Surface activity *per se* is not a measure of toxicity.

(c) The safety of each surfactant used in food must be determined separately.

The report pointed out that surfactants may occur fortuitously in some foods in amounts of a few parts per million and that: "It appears probably that the interfacial tension existing in the digestive tract of a healthy human is so low that it will not be further lowered by the small amounts of synthetic surfactants which may be present in food."

2. In a report on an investigation dealing with the chronic and subacute toxicity for rats of several surface-active agents, among which was sodium alkyl aryl sulfonate, Fitzhugh and Nelson (7) declared that: "The toxic effects of the surface-active agents studied in the experiments were produced by irritation of the gastrointestinal tract (10,000 ppm or more in the diet). To an extent which depended on the concentration of the surface-active agents in the diet, this irritation prevented proper nutrition. In severe cases of irritation, death resulted.

It is recommended that alkyl benzene sulfonate (ABS) in drinking water be limited to 0.5 mg/1, inasmuch as higher concentrations may cause the water to exhibit undesirable taste and foaming. Concentrations of ABS above 0.5 mg/1 are also indicative of questionably undesirable levels of other sewage pollution.

An ABS concentration of 0.5 mg/1 in drinking water, in terms of a daily adult human intake of 2 liters, would give a safety factor of the order of 15,000, calculated on the results of subacute (6) and 2-year (8) tests on rats fed diets containing ABS. In these rat studies, it was found that levels of ABS in the diet of 0.5 percent and below produced no discernible physiological, biochemical, or pathological deviations from normal.

Human experience (6 subjects) with oral doses of purified ABS of 100 mg (equivalent to 2 liters of water containing 50 mg ABS/1) daily for 4 months led to no significant evidence of intolerance (9).

LITERATURE CITATIONS

1. Schwartz, A. M., Perry, J. W., and Berch, J. Surface active agents and detergents, Vol. II. New York, N.Y., Interscience Publishers, Inc., 1958, ch. 1.
2. Task Group Report. Determination of synthetic detergent content of raw water supplies. *J. Am. Water Works A.* 50: 1343-1352, October 1958.
3. Task Group Report. Effects of synthetic detergents on water supplies. *J. Am. Water Works A.* 49: 1355-1358, October 1957.
4. Metzler, D. F., Culp, R. L., Stollenburg, H. A., Woodward, R. L., Walton, G., Chang, S. L., Clark, W. A., Palmec, C. M., and Middleton, F. M. Emergency use of reclaimed water for potable supply at Chanute, Kansas. *J. Am. Water Works A.* 50: 1021-1051, August 1958.
5. Flynn, J. M., Andreoli, A., and Guerrera, A. A. Study of synthetic detergents in ground water. *J. Am. Water Works A.* 50: 1551-1562, December 1958.
6. Food Protection Committee. The relation of surface activity to the safety of surfactants in foods. National Academy of Sciences—National Research Council Pub. 463. Washington, D.C., 1956.
7. Fitzhugh, O. G., and Nelson, A. A. Chronic oral toxicities of surface active agents. *J. Am. Pharm. A. (Sc. Ed.)* 37: 29-32 (1948).
8. Tusing, T. W., Painter, O. E., and Opdyke, D. L. Chronic toxicity of sodium alkylbenzene sulfonate by food and water administration to rats. *Toxicol. Appl. Pharm.* 2: 464-473, July 1960.
9. Freeman, S., Burrill, M. W., Li, T. W., and Ivy, A. C. The enzyme inhibitory action of an alkyl aryl sulfonate and studies on its toxicity when ingested by rats, dogs, and humans, *Gastroenterology* 4: 332-343 (1945).

ARSENIC

The widespread use of inorganic arsenic in insecticides and its presence in animal foods, tobacco, and other sources, make it necessary to set a limit on the concentration of arsenic in drinking water.

Normal human blood contains approximately 0.064 mg of arsenic per 100 ml, whereas urine may contain from trace amounts up to 5 mg per day. Arsenic is found in many foods in varying amounts, occurring naturally in some foods and introduced in others as in pork and turkey and appears in poultry feeds or as a pesticide spray. Shellfish and crustaceans may contain up to 170 ppm (1), but it is suspected that assimilation of arsenic from this source is limited. Vegetables and fruits (and wine) may contain varying small amounts. The tolerance for arsenic on sprayed fruits and vegetables set by the Food and Drug Administration is 3.5 ppm (2). Neither trivalent nor pentavalent arsenic is known to be an essential or beneficial element, and the body is not known to be dependent on a daily intake.

The toxicity of arsenic is well-known and the ingestion of as little as 100 mg usually results in severe poisoning. Chronic poisoning from arsenic may be insidious and pernicious. A considerable proportion is retained at low intake levels. A single dose may require ten days for

its cumulative effects (3, 4).

Both trivalent and pentavalent arsenic are easily absorbed from the gastrointestinal tract and lung, and become distributed throughout the body tissues and fluids. The toxicity of the pentavalent form is believed to be due to its reduction to the trivalent state. Inorganic arsenicals are potent inhibitors of the intracellular SH enzymes involved in cellular oxidations. The concentration of arsenic in kidney, liver, and the walls of the intestine can lead to serious consequences (4).

Recent evidence supports the view that arsenic may be carcinogenic. 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 (5, 6, 7). 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/1 (8).

Arsenic concentrations of from 2 to 4 mg/1 are reported not to interfere with the self-purification of streams (9), nor have arsenic concentrations of 3 to 14 mg/1 been harmful to mayfly nymphs and 10 to 20 mg/1 to dragon and damselflies (10). Bass have tolerated 6 mg/1 for 232 hours (11). A concentration of 15 mg/1 proved toxic to crappies and blue gills (11), and 20 mg/1 (as sodium arsenite) proved harmful to minnows after 36 hours exposure (12).

The U.S. Public Health Service Drinking Water Standards for 1946 established an arsenic limit of 0.05 mg/1. In light of our present knowledge concerning the potential health hazard from the ingestion of inorganic arsenic, the concentration of arsenic in drinking water should not exceed 0.01 mg/1 and concentrations in excess of 0.05 mg/1 are grounds for rejection of the supply.

LITERATURE CITATIONS

1. Monier-Williams, G. W. Trace elements in food. New York, N.Y., John Wiley & Sons, 1949, pp. 162-206.
2. National Agricultural Chemicals Association. Official FDA tolerances. A. New and Pesticide Rev. 19., Washington, D.C. Nat. Agri. Chem. Assoc., December 1960 (Special issue) 19 pp.
3. Sollman, T. H. A manual of pharmacology, Ed. 8., Philadelphia, Pa., W. B. Saunders Co., 1957, pp. 1198-1227.
4. Drill, V. A. Pharmacology in Medicine. Ed. 2., New York, N.Y. McGraw-Hill, 1958, pp. 794-795.
5. Hill, A. B., Fanning, E. L., Perry, F., Bowler, R. G., Buckell, H. M., Druett, H. A., and Schilling, R. S. F. Studies in the incidence of cancer in a factory handling inorganic compounds of arsenic. Brit. J. Indust. Med. 5: 1-15, (1948).

6. Doll, R. Occupational lung cancer: A review. *Brit. J. Indust. Med.* 16: 181-190 (1959).
7. Merewether, E. R. A. *Industrial medicine and hygiene*. London, Butterworth & Co., 1956, Vol. 3, pp. 196-205.
8. Neubauer, O. Arsenical cancer: A review. *Brit. J. Cancer*. L: 192-251 (1947).
9. Federation of Sewage Works Association, Committee on Research, Section A. A critical review of the literature of 1943 on sewage and waste treatment and stream pollution. *Sewage Works J.* 16: 222-277 (March 1944).
10. Podubsky, V. and Stedrousky, E. Toxicity of some metals on fish and river crabs. *Annals of the Czechoslovak. Academy of Agric.* 21: 207-219 (1948).
11. Warrick, L. F., Wurth, H. E., and Van Horn, W. Control of microorganisms and aquatic vegetation. *Water Works and Sewerage* 90: 267-272 (July 1943).
12. Grindley, J. Toxicity to rainbow trout and minnows of some substances known to be present in waste water discharged to rivers. *Ann. Appl. Biol.* 33: 103-112 (1946).

BARIUM

Reference to a limiting concentration for barium in the Public Health Service Drinking Water Standards of 1946 is confined to "salts of barium . . . shall not be added for water-treatment purposes." No reference to barium is made in the International Drinking Water Standards of 1958. Barium occurs naturally in some mineral springs as the carbonate salt.

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 produces 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 deposition 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

peatedly 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 standard may be set. A rational basis for a water standard may be derived from the threshold limit of 0.5 mg Ba/m³ air set by the American Conference of Governmental Industrial Hygienists (10) by procedures that have been discussed (11). By making reasonable assumptions as to retention of inhaled barium dusts and absorption from the intestine (and including a safety factor) 1 mg/l is derivable as a limit that should constitute a "no effect" level in water. Concentrations of barium in excess of 1 mg/l are grounds for rejection of the supply because of the seriousness of the toxic effects of barium on the heart, blood vessels, and nerves.

LIMITS AND RANGES RELATIVE TO BARIUM STANDARD

1. Average U.S. urban air concentration..... 0.025 ug Ba/m³ (12)
2. Surface and ground waters..... Not usually present
3. Concentrations harmful to fish..... 400 mg/l (13)
4. Concentrations harmful to Daphnia Magna..... 30 mg/l (14)
5. Barium content of Brazil nuts (Only food with barium in considerable amounts)..... 0.06-0.3% (15)
6. Concentrations of various natural anions required to reach solubility product of barium salts:

	<i>Solubility product moles/l at 25° C</i>	<i>Milligrams anion re- quired per liter to attain solubility product at 1 mg barium</i>
BaSO ₄	1 × 10 ⁻¹⁰	1.3 SO ₄
BaCO ₃	8 × 10 ⁻⁹	66 CO ₃
BaF ₂	1.7 × 10 ⁻⁶	9000 F

The solubility of relatively insoluble barium salts such as the sulfate may be increased in the presence of iron, magnesium, and aluminum salts, so that in the presence of the latter, calculations of solubility from the solubility product may not apply.

LITERATURE CITATIONS

1. Sollman, T. H. A manual of pharmacology. Ed. S. Philadelphia, Pa., W. B. Saunders Co., 1957, pp. 665-667.
2. Lorente de NÓ, R., and Feng, T. P. Analysis of effect of barium upon nerve with particular reference to rhythmic activity. J. Cell Comp. Physiol. 28: 397-464, December 1946.
3. Gotsev, T. Blutdruck and Herztätigkeit. III Mittheilung: Kreislaufwirkung von barium. Naunyn-Schiedeberg Arch. Exper. Path. 203: 264-277, August 24, 1944.
4. Fite, F. Granuloma of lung due to radiographic contrast medium. AMA Arch. Path. 59: 673-676, June 1955.

5. Kay S. Tissue reaction to barium sulfate contrast medium. *AMA Arch. Path.* 57: 279-284, April 1954, Ibid Kay S., and Chay. *Sun Hak: Results of intraperitoneal injection of barium sulfate contrast medium* 59: 388-392, March 1955.
6. Arnolt, R. I. Fijacion y determinación quimica del bario en organos *Rev. Col. Farm. Nac. (Rosario)* 7: 75-81, June 1940.
7. Gerlach, W., and Muller, R. Occurrence of strontium and barium in human organs and excreta. *Arch. Path. Anat. (Virchows)* 294: 210 (1934).
8. Sowden, E. M., and Stitch, S. R. Trace elements in human tissue. Estimation of the concentrations of stable strontium and barium in human bone. *Biochem. J.* 67: 104-109, September 1957.
9. Bauer, G. C. H., Carlsson, A., and Lindquist, B. A comparative study of metabolism of 140 Ba and 45 Ca in rats. *Biochem. J.* 63: 535-542, August 1956.
10. American Conference of Governmental Industrial Hygienists. Threshold limit values for 1958. *A.M.A. Arch. Indust. Health* 18: 178-182 (1958).
11. Stokinger, H. E., and Woodward, R. L. Toxicologic methods for establishing drinking water standards. *J. Am. Water Works A.* 50: 515-529, April 1958.
12. U.S. Public Health Service. Air Pollution measurements of the national air sampling network. Cincinnati 26, Ohio., Robert A. Taft Sanitary Engineering Center, 1958.
13. Ohio River Valley Water Sanitation Comm. Subcommittee on Toxicities. Metal finishing industries action committee rept. no. 3. Cincinnati, Ohio, Ohio River Valley Water Sanitation Commission, 1950.
14. Anderson, B. G. Apparent thresholds of toxicity to *Daphnia magna* for chlorides of various metals when added to Lake Erie water. *Trans. Am. Fish Soc.* 78: 96-113 (1948).
15. Seaber, W. M. Barium as a normal constituent of brazil nuts. *Analyst* 58: 575-580, October 1933.

CADMIUM

As far as is known, cadmium is biologically a nonessential, non-beneficial element. On the other hand, cadmium is recognized to be an element of high toxic potential. Slight cognizance has been taken of this in water quality control as evidenced by the fact that only the USSR, and in the United States, North Dakota, have set a permissible water standard for cadmium, 0.1 mg/l by the former and 0.4 mg/l as a tentative value by the latter. Recognition of the serious toxic potential of cadmium when taken by mouth is based on: (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) long-term oral toxicity studies in animals.

The possibility of cadmium being a water contaminant has been reported in 1954 (4); 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

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/1 cadmium (1). This is commonly considered the emetic threshold concentration for cadmium. It has been stated (5) 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 (6). A dietary relation to cadmium toxicity has been reported by others (7).

Fifty ppm 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 (8).

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/1. 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/1. 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/1 (9).

Thus, all levels of dietary cadmium so far tested have shown cadmium accumulation in the soft tissues down to and including 0.1 mg/1 (in drinking water). Because the presence of minute amounts (5×10^{-6} M) of cadmium in rat liver mitochondria has been shown (10) to interfere with an important pathway of metabolism (uncoupled oxidative phosphorylation), and because suspicion has been cast on the presence of minute amounts of cadmium in the kidney as responsible for adverse renal arterial changes in man (3), concentrations of cadmium in excess of 0.01 mg/1 in drinking water are grounds for rejection of the supply.

Further evidence that a concentration of 0.01 mg/1 can be tolerated is found in a study made on long-continued cadmium absorption, without history of symptoms, in individuals whose drinking water had an average cadmium content of 0.047 mg/1 (11).

LIMITS AND RANGES RELATIVE TO CADMIUM WATER STANDARD

U.S. average urban air concentration (1954-56) (12)-----	0.005 ug Cd/m ³
U.S. range urban air concentration (1954-56) (12)-----	0-0.599 ug Cd/m ³
Cd concentration lethal to minnows (13)-----	1,000 mg/1
Cd concentration lethal to stickleback (14)-----	0.20 mg/1
Cd concentration in tobacco-----	Not known
Cd concentration in foods-----	Not known

LITERATURE CITATIONS

1. Frant, S., and Kleeman, I. Cadmium "Food Poisoning." J.A.M.A., 117, 86 (1941).
2. Cangelosi, J. T. Acute Cadmium Metal Poisoning. U.S. Nav. Med. Bull., pp. 39 and 408 (1941).
3. Schroeder, H. A. Trace Metals and Chronic Diseases in Advances in Internal Medicine. Vol. VIII, 1956, Year Book Publishers, Inc.
4. Lieber, M., and Welsch, W. F. Contamination of Ground Water by Cadmium. J.A.W.W.A., 46, p. 51 (1954).
5. Potts, A. M., Simon, F. P., Tobias, J. M., Postel, S., Swift, M. N., Patt, H. M., and Gerald, R. W. Distribution and Fate of Cadmium in the Body. Arch. Ind. Hyg. 2, p. 175 (1950).
6. Fitzhugh, O. G., and Meiller, F. H. Chronic Toxicity of Cadmium. J. Pharm., 72 p. 15 (1941).
7. Wilson, R. H., and De Eds, F. Importance of Diet in Studies of Chronic Toxicity. Arch. Ind. Hyg. 1, p. 73 (1950).
8. Ginn, J. T., Volker, J. F. Effect of Cd and F on Rat Dentition. Proc. Soc. Exptl. Biol. Med. 57, p. 189 (1944).
9. Decker, L. E., Byerrum, R. U., Decker, C. F., Hoppert, C. A., and Langham, R. F. Chronic Toxicity Studies, I. Cadmium Administered in Drinking Water to Rats. A.M.A. Arch. Ind. Health, 18, p. 228 (1958).
10. Jacobs, E. E., Jacob, M., Sanadi, D. R., and Bradley, L. B. Uncoupling of oxidative phosphorylation by cadmium ion. J. Biol. Chem. 233, p. 157 (1956).
11. Princi, F. Metals-cadmium in Oxford loose-leaf medicine. Vol. IV, Chap. XI, New York, 1949.
12. Air Pollution measurements of the national air sampling network, 1953-57. Robt. A. Taft San. Eng. Center, Cincinnati, Ohio, 1958.
13. Carpenter, K. E. The Lethal Action of Soluble Metallic Salts on Fishes. Brit. J. Exp. Biol. 4, p. 378 (1927).
14. Ohio River Valley Water Sanitation Comm. Subcommittee on Toxicities, Metal finishing industries action comm. rept. 3 (1950).

CARBON CHLOROFORM EXTRACT

The use of Carbon Chloroform Extract (CCE) (1) as a practical measure of water quality and as a safeguard against the intrusion of

discussed elsewhere (2). It is proposed as a technically practical procedure which will afford a large measure of protection against the presence of undetected toxic materials in finished drinking water.

The most desirable condition is one in which the water supply delivered to the consumer contains no organic residues. Residual organic matter in the treated water clearly represents man-made or natural pollutants which have not been removed in water treatment or material such as lubricants inadvertently introduced by the water plant. In view of a general inability to clearly define the chemical and toxicological nature of this material, it is most desirable to limit it to the lowest obtainable level. Analysis of data available indicates that water supplies containing over 200 micrograms CCE/1 of water represent an exceptional and unwarranted dosage of the water consumer with ill-defined chemicals. It is recommended that 200 ug CCE/1 be the limiting concentrations in drinking water.

LITERATURE CITATIONS

1. Middleton, F. M. Nomenclature for referring to organic extracts obtained from carbon with chloroform or other solvents. *J. Am. Water Works A.* 53: 749, 1961.
2. Ettinger, M. B. A proposed toxicological screening procedure for use in the water works. *J. Am. Water Works A.* 52: 689-694, 1960.

CHLORIDE, SULFATE, AND DISSOLVED SOLIDS

The importance of chloride, sulfate, and dissolved solids as they affect water quality hinges upon their taste and laxative properties. There is evidence that excessive amounts of these constituents cause consumer reactions which may result in individual treatment or rejection of the supply. Therefore, limiting amounts for these chemical constituents have been included in the Standards. The bases for developing these limits are described below.

Taste

The literature contains a number of reports on the taste threshold of various salts. Whipple, (1) using a panel of 10 to 20 persons, found the range of concentration of various salts detected as shown in Table 1. Richter and MacLean (2) studied the response of a larger panel to sodium chloride in distilled water. Table 2 summarizes their results.

Lockhart, Tucker, and Merritt (3) also studied the taste threshold of the ions in distilled water by studying the effect of ions in water on the flavor of brewed coffee. Using a triangular test with panels of 18 or more, they found results which are summarized in Table 3.

In the Triangular taste test, the panel members are asked to taste three samples. Two of the samples may contain either the salt being tested or distilled water, while the third is different from the other two. The panel member is asked to identify the odd one. Using this test procedure, the threshold concentration is arbitrarily defined as the concentration at which the number of correct separations is 50 percent above the chance probability of one-third correct separations, i.e., when two-thirds of the panel make the separations correctly.

The results shown in Table 1 and Table 3 are in surprisingly good agreement, considering the difference in methods used. The Richter and MacLean study found taste thresholds considerably below those of the other two studies. They support reasonably well the recommended limits of 250 mg/1 for chloride and sulfates and 500 mg/1 for total solids.

It should be emphasized that there may be a great difference between a detectable concentration and an objectionable concentration of the neutral salts. The factor of acclimatization is particularly important. More than 100 public supplies in the United States provide water with more than 2,000 mg/1 of dissolved solids. Newcomers and casual visitors would certainly find these waters almost intolerable and, although some of the residents use other supplies for drinking, many are able to tolerate if not to enjoy these highly mineralized waters.

Relatively little information is available on consumer attitudes toward mineralized water. In this connection, the findings of a survey made by the California State Department of Public Health (4) showed that in five communities where the public supplies were highly mineralized, about 40 percent of the families surveyed purchased bottled water and about 50 percent stated they were dissatisfied with the water. These supplies had dissolved solids contents in the range of 500 to 1,750 mg/1. Calcium, sulfate, and magnesium were the dominant ions present, with sulfate concentrations in the range of 300 to 700 mg/1.

The taste threshold for magnesium is said to be 400-600 mg/1 (5).

Laxative Effects

Both sodium sulfate and magnesium sulfate are well known laxatives. The laxative dose for both Glauber salt ($\text{Na}_2\text{SO}_4 \cdot 10\text{H}_2\text{O}$) and Epsom salt ($\text{MgSO}_4 \cdot 7\text{H}_2\text{O}$) is about two grams. Two liters of water with about 300 mg/1 of sulfate derived from Glauber salt, or 390 mg/1 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.

mation on the laxative effects of water as related to mineral quality. This has been obtained by having individuals submitting water samples for mineral analysis complete a questionnaire which 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 (6) and Moore (7) 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/1 of sulfate showed a laxative effect and those with less than 600 mg/1 generally did not. If the water was high in magnesium, the effect was shown at lower sulfate concentrations 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/1 and by the average person when magnesium was 500-1,000 mg/1.

Moore analyzed the data as shown in Table 4. When sulfates plus magnesium exceed 1,000 mg/1 or dissolved solids exceed 2,000 mg/1, a majority of those who gave a definite reply indicated a laxative effect.

Other Effects

Highly mineralized water affects the quality of coffee brewed with it. Lockhart, Tucker, and Merritt (3) found that from 400 to 500 mg/1 of chlorides or 800 mg/1 of sulfate as $MgSO_4$ affected the taste of coffee. Gardner (8) studied the effect of ions in water on the brewing time of drip coffee and hence on the quality of the product since prolonged contact with the grounds makes the coffee bitter. Sodium had a distinct deleterious effect.

At high enough mineral concentration, water becomes completely unusable for drinking. These concentrations are in the range above 5,000 mg/1 and need not be considered here.

Conclusion

It is recommended that waters containing more than 250 mg/1 of chlorides or sulfates and 500 mg/1 of dissolved solids not be used if other less mineralized supplies are available. This is influenced primarily by considerations of taste. Cathartic effects are commonly experienced with water having sulfate concentrations of 600 to 1,000 mg/1, particularly if much magnesium or sodium is present. Although waters of such quality are not generally desirable, it is recognized that a considerable number of supplies with dissolved solids in excess of the recommended limits are used without any obvious ill effects.

LITERATURE CITATIONS

1. Whipple, G. C. The value of pure water. New York, N.Y., John Wiley & Sons, 1907, 84 pp.
2. Richter, C. P., and MacLean, A. Salt taste threshold of humans. *Am. J. Physiol.* 126: pp. 1-6 (1939).
3. Lockhart, E. E., Tucker, C. L., and Merritt, M. C. The effect of water impurities on the flavor of brewed coffee. *Food Research*, 20: pp. 598-605 (1955).
4. Unpublished report. California State Department of Public Health, Bureau of Sanitary Engineering.
5. Moore, E. W. The desalting of saline waters. Washington, D.C., Bulletin of the Committee on Sanitary Engineering and Environment, National Research Council, May 24, 1950, Appendix D, pp. 347-363.
6. Peterson, N. L. Sulfates in drinking water. Official Bulletin North Dakota Water and Sewage Works Conference, 18: pp. 6-7, 11, April-May 1951.
7. Moore, E. W. Physiological effects of the consumption of saline drinking water. Bulletin of Subcommittee on Water Supply, National Research Council, Jan. 10, 1952, Appendix B, pp. 221-227.
8. Gardner, D. G. Effect of certain ion combinations commonly found in potable water on rate of filtration through roasted and ground coffee. *Food Research*, 23: pp. 76-84 (1958).

TABLE 1.—Range of concentration of various salts detected by taste in drinking water by panel of 10 to 20 persons

Salt	Concentration detected—mg/l			
	Median		Range	
	Salt	Anion	Salt	Anion
KCL.....	525	250	350-600	167-286
NaCl.....	300	182	200-450	121-274
CaCl ₂	250	160	150-350	95-224
MgCl ₂	500	372	200-750	149-600
Sea water.....		1 300		1 150-600
NaSO ₄	350	237	250-550	169-372
CaSO ₄	525	370	250-900	177-635
MgSO ₄	525	419	400-600	320-479

¹ In terms of mg/l chloride.

Source: Whipple, G. C., The value of pure water. Wiley (1907).

TABLE 2.—Taste threshold concentrations of panel of 53 adults for NaCl

	Concentrations mg/l					
	Mean		Median		Range	
	NaCl	Cl	NaCl	Cl	NaCl	Cl
Difference from distilled water noted....	160	97	100	61	70- 600	42- 364
Salt taste identified.....	870	530	650	395	200-2, 500	120-1, 215

	Threshold concentration—mg/l		
	Salt	Cation	Anion
NaCl.....	345	135	210
KCl.....	650	340	310
CaCl ₂	347	125	222
MgSO ₄	500	100	400
NaHCO ₃	1,060	290	770

Source: Lockhart, E. E., Tucker, C. L., and Merritt, M. C. The effect of water impurities on the flavor of brewed coffee, *Food Research*, 20, 593-605 (1955).

TABLE 4.—*Solids and ion concentration of wells as related to presence or absence of laxative effects*

Determination	Range mg/l	Number of wells in range	Laxative		Effects present not stated	Percent of yes answers ¹
			Yes	No		
Total dissolved solids.....	0-1,000	51	5	37	9	12
	1,000-2,000	72	12	45	15	21
	2,000-3,000	62	25	21	16	54
	3,000-4,000	30	13	11	6	54
	over 4,000	33	14	4	15	78
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

¹ 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.

Source: Moore, Edward W., *Physiological effects of the consumption of saline drinking water*, a progress report to the Subcommittee on Water Supply of the Committee on Sanitary Engineering and Environment. National Research Council (1952).

CHROMIUM

The limit of 0.05 mg/l for chromium as hexavalent chromium ion appearing in the U.S. Public Health Service 1946 Drinking Water Standards was based on the lowest amount analytically determinable at the time it was established. At present, the level of chromate ion that can be tolerated by man for a lifetime without adverse effects on health is unknown. A family of four individuals is known to have drunk water for periods of 3 years at a level as high as 1 mg chromate/l without known effects on their health, as determined by a single medical examination (1). The family continued to drink the water which, when sampled later, contained

25 mg/1. No continued medical observation of these individuals was made.

When inhaled, chromium is a known cancerigenic agent for man (2, 3). It is not known whether cancer will result from ingestion of chromium in any of its valence forms. According to Fairhall (4), trivalent chromium salts show none of the toxicity of the hexavalent form, particularly the highly insoluble salts. Trivalent chromium moreover, is believed not to be of concern in drinking water supplies.

Chromium is not known to be either an essential or beneficial element in the body.

The most recent study by MacKenzie, Byerrum, et al. (5) was designed to determine the toxicity of chromate ion (and chromic ion) at various levels in the drinking water of rats. This study, like a number of previous ones, showed no evidence of toxic response after 1 year at levels from 0.45 to 25 mg/1 by the tests employed, viz., body weight, food consumption, blood changes, and mortality. However, significant accumulation of chromium in the tissues occurred abruptly at concentrations above 5 mg/1. Unfortunately, no study was made of the effect of chromate on a cancer-susceptible strain of animal. It would appear, however, from this and other studies of toxicity (6, 7, 8), that a concentration of 0.05 mg/1 is sufficiently low to cause no effect on health.

The possibility of dermal effects from bathing in water containing 0.05 mg/1 would likewise seem remote, although chromate is a recognized and potent sensitizer of the skin (9).

Chromium is not known to be a common or significant element in food sources. That which may be found in small quantities in foods is in trivalent form, is usually adventitious, and arises chiefly from cooking in stainless-steel ware. Neither the amounts nor the assimilability are known to be of any hygienic significance (8, 11).

LIMITS AND RANGES RELATIVE TO CHROMIUM WATER STANDARDS

Threshold range for color (12)-----	1.4-11 mg/1
Threshold range for taste (12)-----	1.4-25 mg/1
U.S. urban air conc'n range (1954-56) (13)-----	0-0.29 ug/m ³
Average urban air conc'n (1954-56) (13)-----	0.007 ug/m ³
Chromium content of cigarette tobacco (14)-----	1.4 ug/cigarette
Chromium in foods cooked in stainless-steel ware (9)-----	0-0.35 mg/100 g
Chromate conc'n toxic to fish (15, 16, 17)-----	5-200 mg/1
Chromate conc'n toxic to <i>Daphnia Magna</i> (17)-----	0.05 mg/1
Chromate conc'n range in surface water (12)-----	0-2.3 mg/1

(Approximate Values)

Food, cooked in stainless-steel ware-----	10-25 ug
Water -----	2 ug
Air -----	0.3 ug
Cigarettes -----	10-15 ug

LITERATURE CITATIONS

1. Davids, H. W., and Lieber, M. Underground water contamination by chromium wastes. *Water and Sewage Works* 98: 528-534, December 1951.
2. Machle, W., and Gregorius, F. Cancer of the respiratory system in the United States chromate-producing industry. *Pub. Health Rep.* 63: 1114-1127, August 29, 1942.
3. U.S. Public Health Service. Health of workers in the chromate industry. *Pub. Health Service Pub. No. 192.* Washington, D.C., U.S. Government Printing Office, 1953.
4. Fairhall, L. T. *Industrial toxicology.* Ed. 2, Baltimore, Md., Williams and Wilkins. 1957, pp. 21-23.
5. MacKenzie, R. D., Byerrum, R. U., Decker, C. F., Hoppert, C. A., and Langham, R. F. Chronic toxicity studies II hexavalent and trivalent chromium administered in drinking water to rats. *A.M.A. Arch. Indust. Health* 18: 232-234, September 1958.
6. Gross, W. G., and Heller, V. G. Chromates in animal nutrition. *J. Indust. Hyg. & Toxicol* 28: 52-56 (1946).
7. Brard, M. D. Study of toxicology of some chromium compounds. *J. Pharm. et Chim.* 21: 5-23 (1935).
8. Conn, L. W., Webster, H. L., and Johnson, A. H. Chromium toxicology. Absorption of chromium by the rat when milk containing chromium lactate was fed. *Fed. Am. J. Hyg.* 15: 760-765 (1932).
9. Denton, C. R., Keenan, R. G., and Birmingham, D. G. The chromium content of cement and its significance in cement dermatitis. *J. Invest. Derm.* 23: 184 (1954).
10. Monier-Williams, G. W. *Trace elements in food.* Ed. 2. New York, John Wiley & Sons, 1950, pp. 439-443.
11. Titus, A. C., Elkins, H. B., Finn, H. G., Fairhall, L. T., and Drinker, C. K. Contamination of food cooked or stored in contact with nickel-chromium-iron alloys. *J. Indust. Hyg.* 12: 306-313, October 1930.
12. Cass, J. S., and Foldes, P. E. Interim Report on chromium. Physiologic aspects of "Water Quality Criteria" with regard to man. Kettering Laboratory, Cincinnati, Ohio (1957).
13. U.S. Public Health Service, Division of Air Pollution. Air Pollution measurements of the national air sampling network. *Robt. A. Taft Sanitary Eng. Center, Cincinnati, Ohio* (1958).
14. Cogbill, E. C., and Hobbs, M. E. Transfer of metallic constituents of cigarettes to the main-stream smoke. *Tobacco Sci.* 144: 68-73 (1957).
15. Southgate, B. A. Treatment and disposal of industrial waste waters. London, England, H. M. Stationery Office, 1948.
16. Ellis, M. M. Detection and measurement of stream pollution. *Bur. Fisheries Bul. No. 22: 365-437* (1937).

17. Ohio River Valley Water Sanitation Commission. Subcommittee on toxicities, metal finishing industries action committee, Report No. 3, Cincinnati, Ohio (1950).

COPPER

In the Public Health Service 1942 Drinking Water Standards, the permissible concentration of copper in drinking water was raised from 0.2 mg/l to 3.0 mg/l.

Copper is an essential and beneficial element in human metabolism, and it is well known that a deficiency in copper results in nutritional anemia in infants. The daily requirement for adults has been estimated to be 2.0 mg (1). The children of preschool age require about 0.1 mg daily for normal growth. The average daily urinary excretion is in the order of 1.0 mg, the remainder being eliminated in the feces. Since the normal diet provides only a little more than is required, an additional supplement from water would ensure an adequate intake. The distribution of copper in the body is fairly uniform, except for the liver where it appears to accumulate.

Copper imparts some taste to water but individuals vary in the acuity of their taste perception and the detectable range varies from 1-5 mg/l (2). Small amounts are generally regarded as nontoxic but large doses may produce emesis and prolonged oral administration may result in liver damage.

Inasmuch as copper does not constitute a health hazard but imparts an undesirable taste to drinking water, it is reasonable to establish the concentration of 1.0 mg/l as the recommended limit.

LITERATURE CITATIONS

1. Sollmann, T. H. A manual of pharmacology, Ed. 8. Philadelphia, Pa., W. B. Saunders Co., 1957, pp. 1299-1302.
2. Cohen, J. M., Kamphake, L. J., Harris, E. K., and Woodward, R. L. Taste threshold concentrations of metals in drinking water. J. Am. Water Works A. 52: 660-670 (1960).

CYANIDE

The U.S. Public Health Service Drinking Water Standards for 1946 contain no limit for cyanide. Since 1946, standards have been developed for cyanide by other agencies as shown in the following tabulation.

<i>Standard set by</i>	<i>Limit for cyanide mg/l</i>
International Standards for Drinking Water, Geneva (1958)-----	0.01
Netherlands (1959)-----	0.01
USSR Standard (1951)-----	0.2
Ohio Water Pollution Control Board (1952)-----	0.15
Adv. Bd. Lake Erie-Ontario Sect. I.J.C. (1953)-----	0.1
N.Y. Water Pollution Control Bd. (1952)-----	0.1
Pacific N.W. River Basin (1952)-----	0.05

and not for man, as is shown by a comparison that follows of the safe, toxic, and lethal doses for fish and for man. Cyanide in reasonable doses (10 mg or less) is readily converted to thiocyanate in the body. Usually lethal toxic effects occur only when the detoxifying mechanism is overwhelmed.

Oral toxicity of cyanide for man

Dosage	Response	Literature citations
2.9-4.7 mg/day.....	Noninjurious.....	(5)
10 mg, single dose.....	Noninjurious.....	(6)
19 mg in water.....	Calculated from threshold limit for air to be safe.....	(7)
50-60 mg, single dose.....	Fatal.....	(8)

Toxicity of cyanide for fish

Cyanide in mg/l	Time of exposure	Fish species	Response	Literature citations
0.05.....	120 hours.....	Trout.....	Death.....	(1)
0.1-0.2.....	1-2 days.....	do.....	do.....	(2)
0.126.....	170 minutes.....	do.....	Overturned.....	(3)
0.176.....	Bluegills, Sunfish.....	Toxic limit.....	(9)
1.0.....	20 minutes.....	Trout.....	Death.....	(1)
10.0.....	90 minutes.....	Carp.....	do.....	(4)
0.02.....	27 days.....	Trout.....	Survival.....	(1)
0.4.....	96 hours.....	Bluegills.....	do.....	(3)
0.5.....	96 hours.....	Bullheads.....	do.....	(3)

Because proper treatment will reduce cyanide levels to 0.01 mg/l or less, it is recommended that concentrations in water be kept below 0.01 mg CN/l.

For the protection of the health of human populations, concentrations above 0.2 mg CN/l constitute ground for rejection of the supply. This limit should provide a factor of safety of approximately 100 and is set at this level because of the rapidly fatal effect of cyanide. Proper chlorination under neutral or alkaline conditions will reduce cyanide to a level below the recommended limit. The acute oral toxicity of cyanogen chloride, the chlorination product of hydrogen cyanide, is approximately one-twentieth that of hydrogen cyanide (9).

LITERATURE CITATIONS

1. Karsten, A. Effect of cyanide on Black Hills trout. Black Hills Eng. 22: 145-174 (1934). Abs. in J. Am. Water Works A. 28: 660 (1936).
2. Southgate, B. A. Treatment and disposal of industrial waste waters. London, England, H. M. Stationery Office, 1948.
3. Ohio River Valley Water Sanitation Commission, Subcom. on Toxicities: metal finishing industries action committee report no. 3, Cincinnati, Ohio, Ohio River Valley Water Sanitation Commission, 1950.

4. Symons, C. E., Simpson, R. W. Report on fish destruction in the Niagara River in 1937. *Trans. Am. Fish. Soc.* 68: 246 (1938).
5. Smith, O. M. The detection of poisons in public water supplies. *Water Works Eng.* 97: 1293-1312, Nov. 1, 1944.
6. Bodansky, M., and Levy, M. D. I: Some factors influencing the detoxication of cyanides in health and disease. *Arch. Int. Med.* 31: 373-389 (1923).
7. Stokinger, H. E., and Woodward, R. L. Toxicologic methods for establishing drinking water standards. *J. Am. Water Works A.* 50: 515-529, April 1958.
8. Anon. The Merck Index. Ed. 6. Rahway, N.J., Merck & Co., Inc. 1952, p. 508.
9. Spector, W. S. Handbook of toxicology. Tech. Rept. No. 55-16, Wright-Patterson Air Force Base, Ohio, Wright Air Devel. Center, Air Res. and Devel. Command, April 1955.

FLUORIDE

Fluoride in drinking water will prevent dental caries. When the concentration is optimum, no ill effects will result and caries rates will be 60-65 percent below the rates in communities using water supplies with little or no fluoride (1, 2).

Excessive fluoride in drinking water supplies produces objectionable dental fluorosis which increase with increasing fluoride concentration above the recommended upper control limits.¹ In the United States, this is the only harmful effect observed to result from fluoride found in drinking water (3, 4, 5, 6, 7, 8, 9). Other expected effects from excessively high intake levels are: (a) bone changes when water containing 8-20 mg fluoride per liter (8-20 ppm) is consumed over a long period of time (5); (b) crippling fluorosis when 20 or more mg of fluoride from all sources is consumed per day for 20 or more years (10); (c) death when 2,250-4,500 mg of fluoride (5,000-10,000 mg sodium fluoride) is consumed in a single dose (5).

The optimum fluoride level 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 (11, 12, 13, 14). Many communities with water supplies containing less fluoride than the concentration shown as the lower limit for the appropriate air temperature range¹ have provided fluoride supplementation (15, 16, 17). 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 (18, 19).

LITERATURE CITATIONS

1. Dean, H. T., Arnold, F. A., Jr., and Elvove, E. Domestic water and dental caries. V. Additional studies of the relation of fluoride in domestic waters to dental caries experience in 4,425 white children, age 12 to 14 years, of 13 cities in 4 States. *Pub. Health Rep.* 57: 1155-1179, Aug. 7, 1942.

¹ See Table 1, p. 8 of the Drinking Water Standards.

- dental caries. 11. A study of 2,882 white children, aged 12 to 17 years, of 8 suburban Chicago communities, including lactobacillus acidophilus studies of 1,761 children. Pub. Health Rep. 56: 761-792, Apr. 11, 1941.
3. Moulton, F. R., Editor. Fluorine and dental health. A.A.A.S. Pub. No. 19, Washington, D.C., 1946, pp. 6-11, 23-31.
 4. Dean, H. T. Chronic endemic dental fluorosis (mottled enamel). J.A.M.A. 107: 1269-1273 (1936).
 5. Shaw, J., Editor. Fluoridation as a public health measure. A.A.A.S. Pub. No. 38, Washington, D.C., 1954, pp. 79-109.
 6. Heyroth, F. F. Toxicologic evidence for the safety of fluoridation of public water supplies. Am. J. Pub. Health 42: 1568-1575 (1952).
 7. McClure, F. J. Fluorine in food and drinking water. J. J. Am. Diet. A. 29: 560-564 (1953).
 8. U.S. Public Health Service. Natural fluoride content of communal water supplies in the United States. Public Health Service Pub. No. 655, Washington, D.C., U.S. Government Printing Office, 1959.
 9. Leone, N. C., Shimkin, M. B., Arnold, F. A., Stevenson, C. A., Zimmermann, E. R., Geiser, P. B., and Lieberman, J. E. Medical aspects of excessive fluoride in a water supply. Pub. Health Rep. 69: 925-936, October 1954.
 10. Roholm, K. Fluorine intoxication. A clinical-hygienic study. London, H. K. Lewis & Co., Ltd. (1937).
 11. Galagan, D. J., and Lamson, G. G. Climate and endemic dental fluorosis. Pub. Health Rep. 68: 497-508, May 1953.
 12. Galagan, D. J. Climate and controlled fluoridation. J. Am. Dent. A. 47: 159-170, August 1953.
 13. Galagan, D. J., Vermillion, J. R., Nevitt, G. A., Stadt, A. M., and Dart, R. E. Climate and fluid intake. Pub. Health Rep. 72: 484-490, June 1957.
 14. Galagan, D. J., and Vermillion, J. R. Determining optimum fluoride concentrations. Pub. Health Rep. 72: 491-493, June 1957.
 15. Pelton, W. J., and Wisan, J. M. Dentistry in public health. Philadelphia, Pa., W. B. Saunders Co., 1949, pp. 136-162.
 16. Arnold, F. A., Jr., Dean, H. T., Jay, P., and Knutson, J. W. Effect of fluoridated public water supplies on dental caries prevalence. Pub. Health Rep. 71: 652-658, July 1956.
 17. Anon. Status of fluoridation in community water supplies. Pub. Health Rep. 74: 427, May 1959.
 18. Dean, H. T., and McKay, F. S. Production of mottled enamel halted by a change in common water supply. Am. J. Pub. Health 29: 590-596, June 1939.
 19. Dean, H. T., McKay, F. S., and Elvove, E. Mottled enamel survey of Bouxite, Ark., 10 years after a change in the common water supply. Pub. Health Rep. 53: 1736-1748, Sept. 30, 1938.

IRON

Both iron and manganese are highly objectionable constituents in water supplies for either domestic or industrial use. The domestic consumer complains of the brownish color which iron imparts to laundered goods. Iron appreciably affects the taste of beverages (*1*).

The taste which iron imparts to water may be described as bitter and astringent. Individuals vary in their acuity of taste perception,

and it is difficult to establish a level which would not be detectable for the majority of the population. A study by the Public Health Service (2) indicates that the taste of iron may be readily detected at 1.8 mg/1 in spring water and at 3.4 mg/1 in distilled water.

The daily nutritional requirement is 1 to 2 mg but intake of larger quantities is required as a result of poor absorption. Diets contain 7 to 35 mg per day and average 16 (3). The amount of iron permitted in water by quality control to prevent objectionable taste or laundry staining (as much as 0.3 mg/1) constitutes only a small fraction of the amount normally consumed and is not likely to have any toxicologic significance.

Whereas the U.S. Public Health Service 1946 Drinking Water Standards set a limit of 0.3 mg/1 for iron and manganese combined, it is recommended that a limit be established for each and that the concentration of iron be limited to 0.3 mg/1.

LITERATURE CITATIONS

1. Riddick, T. M., Lindsay, N., and Tomassi, A. Iron and manganese in water supplies. *J. Am. Water Works A.* 50: 688-696, May 1958.
2. Cohen, J. M., Kamphake, L. J., Harris, E. K., and Woodward, R. L. Taste threshold concentrations of metals in drinking water. *J. Am. Water Works A.* 52: 660-670, May 1960.
3. Sollman, T. H. *A manual of pharmacology.* Ed. 8, Philadelphia, Pa., W. B. Saunders Co., 1957, pp. 1247-1267.

LEAD

Lead taken into the body can be seriously injurious to health, even lethal, if taken in by either brief or prolonged exposure. Prolonged exposure to relatively small quantities may result in serious illness or death. Lead taken into the body in quantities in excess of certain relatively low "normal" limits is a cumulative poison. Poisoning may result from an accumulation in the body of lead absorbed in sufficient quantities from any one or all of three common sources: food, air, and water, including that used in cooking and in beverages. A fourth, but variable source of intake is inhaled tobacco smoke. Except in certain occupational conditions, absorption of lead through the skin is not of general public health importance.

The total amount of lead taken into the body from these sources as modified by absorption and elimination, determines whether the sources of exposure have been excessive and produce poisoning, or may be tolerated without effect throughout a lifetime.

The daily intake of lead that may be tolerated without effect throughout each decade of life is not precisely known, but a value may be determined from the following information.

1. The amount of lead ingested in food and beverage by adults in the United States has been shown by

2.0 mg/day with a mean value of about 0.32 mg/day. At these levels, excretion keeps pace with intake, and if any accumulation of lead occurs it is intermittent and of no hygienic significance.

2. When, under experimental condition, the daily intake of lead from all sources amounted to 0.5–0.6 mg over a long period of time (1 year or more), a small amount is retained in normal healthy adults but produced no detectable deviation from normal health. Indirect evidence from industrial workers exposed to known amounts of lead for long periods was consistent with these findings (2).

3. Appreciable increases in the daily intake of lead above 0.6 mg daily result in body accumulation at rates that increase as the daily dose increases. Extrapolations from data from balance experiments over a 5-year period indicate, but do not prove, that an intake appreciably in excess of 0.6 mg/day will result in the accumulation of a dangerous quantity of lead in the body during a lifetime.

4. The intake of lead from food sources is probably approaching an irreducible minimum; on the other hand, the number of sources and the extent of lead exposure are increasing. The atmosphere is one of these. Over the past decade, the amount of atmospheric lead in many cities has increased more than tenfold, from a few tens of micrograms (ug) per cubic meter (m^3) of air to more than 15 ug/ m^3 in some cities on repeated occasions (3). The national average for urban atmosphere is presently 1.4 ug/ m^3 . Wide variations in these values exist throughout the nation because the sources are largely unregulated and are increasing at different rates in different areas from vehicular traffic. If the average daily intake of air of an adult is 20 cubic meters, then the daily addition to the body burden of lead from the atmosphere could be of the order of several micrograms to a few tens of micrograms, depending on the location. This assumes a modest 10 percent retention of that which the individual inhales.

5. The amount of lead in cigarette tobacco smoke has been reported (4) to be as high as 0.3 ug/puff. In a heavy smoker, a few micrograms per day could be added to the lead body burden assuming 10 percent retention of the total smoke inhaled.

Foods contain lead in widely varying amounts because of the natural and unavoidable content of lead in foods, the inevitable contamination with lead that results incidentally from processing and packaging, and the residue from insecticidal spraying and dusting. Certain foods, in particular those which are more seriously and unavoidably contaminated, are required by law to contain by analysis no more than a prescribed concentration of lead. The foods under regulation make up a relatively small portion of the average normal diet. Conse-

quently, only partial control is exercised over the lead intake from food sources. The foods that contribute the greater portion of the diet contain concentrations of lead which are considered to be normal (that is, natural or incidental) but in any case unavoidable (under 0.2 ppm, and usually well under 0.1 ppm). The total intake of lead from these foods is governed by the quantity and quality of the food ingested, and by contamination with lead in the handling and preparation of the food.

The lead concentration in surface and in ground drinking water sources in the United States in 1940 ranged from traces to 0.04 mg/1, averaging 0.01 mg/1. It is now not uncommon to find the lead content of water in urban supplies to be from one-half to one-fifth this value, provided the water is not stored in tanks painted with oil-base lead paint (Type I) or provided that the piping and fixtures are not of lead or lead alloys. However, a principal source of lead in municipal drinking waters is lead pipe and goosenecks in house services and plumbing systems. The practice of using lead pipe is still permitted by many plumbing codes. Normal adults in the temperate zone drink quantities of water, ranging from less than 1 to more than 3 liters/day, the average being taken as 2 liters. This is in addition to the water used in cooking and in other beverages. Thus, water can contribute a substantial proportion of the total daily intake of lead, depending upon the concentration of lead therein, the environmental temperature, and physical exertion.

Inasmuch as three of the four sources of lead intake in the human body—ingested foodstuffs, inhaled atmosphere, and tobacco smoke—are for the most part unregulated in their lead content, and because the total daily intake of lead which results in progressive retention of lead in the human body appears to be less than twice the average normal intake of lead in adults in the United States, concentrations of lead in drinking water greater than 0.05 mg/1 constitute grounds for rejection of the supply.

In consonance with this limit is the reported finding that bacterial decomposition of organic matter is inhibited by lead concentrations at or above 0.1 mg/1 (5). Lead in soft water is highly toxic to certain fish (6); 0.1 mg/1 is toxic to small sticklebacks, larger fish are somewhat less susceptible to lead. Calcium ion at a concentration of 50 mg/1 removes the toxic effect of 1 mg/1 lead for fish (7).

LIMITS AND RANGES OF LEAD AFFECTING HEALTH

Physiologically safe in water :

Lifetime -----	0.05 mg/1
Short period, a few weeks-----	2-4 mg/1

Toxic.....	8-10 mg/1, several weeks.
Lethal.....	Unknown, but probably more than 15 mg/1, several weeks.

LITERATURE CITATIONS

1. Kehoe, R. A., Cholak, J., Hubbard, D. M., Bamburg, K., McNary, R. R., and Story, R. V. Experimental studies on the ingestion of lead compounds. *J. Indust. Hyg. Tox.* 22: 381-400 (1940).
2. Kehoe, R. A. Exposure to lead. *Occup. Med.* 3: 156-171 (1947).
3. Tabor, E. C. National air sampling network data. Columbia, Mo., U. of Missouri Bull. 60: 9-15 (1957).
4. Cogbill, E. C., and Hobbs, M. E. Transfer of metallic constituents of cigarettes to the main-stream smoke. *Tobacco Science* 144: 68-73, May 10, 1957.
5. Kalabina, M. M., Vis, K. A. M., Razumov, A. S. and Rogovskaja, Ts. I. Effects of toxic substances in effluents from nonferrous metal industries on the micro-organisms and biogeochemical processes. *Abs. in Gigena (U.S.S.R.)* 9: 10/11: 1, 1944. *Water Poll. Abs.* 21: 47 Abs. No. 281, February 1948.
6. Doudoroff, P., and Katz, M. Critical review of literature on the toxicity of industrial wastes and their components to fish, II the metals as salts. *Sewage and Indust. Wastes* 25: 802-839, July 1953.
7. Ohio River Valley Water Sanitation Comm., Subcommittee on Toxicities. Metal finishing industries action committee report, number 3, Cincinnati, Ohio, Ohio River Valley Water Sanitation Commission (1950).

MANGANESE

There are two reasons for limiting the concentration of manganese in drinking water: (a) to prevent esthetic and economic damage, and (b) to avoid any possible physiologic effects from excessive intake.

It has been reported that minute amounts of manganese cause difficulty in water quality control. The domestic consumer finds that it produces a brownish color in laundered goods and impairs the taste of beverages including coffee and tea (1,2).

From the health standpoint, there are no data to indicate at what level manganese would be harmful when ingested (3,4). The principal toxic effects which have been reported are the results of inhalation of manganese dust or fumes. It has been estimated that the daily intake of manganese from a normal diet is about 10 mg (5). In animals, at least, it has been shown to be an essential nutrient, since diets deficient in manganese interfere with growth, blood, and bone formation and reproduction. Hepatic cirrhosis has been produced in rats when treated orally with very large doses. As far as is known, the neurologic effects of manganese have not been reported from oral ingestion in man or animal (6).

The principal reason for limiting the concentration of manganese is to provide water quality control and thus reduce the esthetic and economic problems (1, 3, 8).

The U.S. Public Health Service Drinking Water Standards (1946) state that iron and manganese together should not exceed 0.3 mg/1. In a survey of 13 States reporting on levels of manganese giving rise to water quality problems, only three States recommended levels as high as 0.2 mg/1, two permitted 0.15 mg/1 and four each permitted 0.1 mg/1 and 0.05 mg/1 respectively. Domestic complaints arise when the level of manganese exceeds 0.15 mg/1 regardless of iron content. Griffin (8), in reviewing the significance of manganese as chairman of the task group on "Manganese Deposition in Pipelines", quoted the belief of certain water utility men that water to consumers should be free of manganese. For some industries, this is imperative. However, Griffin believes that concentration of manganese could be tolerated by the average consumer at 0.01-0.02 mg/1.

In view of the above and the difficulty of removing manganese to residual concentrations much less than 0.05 mg/1, and measuring such concentrations, manganese concentrations should be limited to a maximum of 0.05 mg/1.

LITERATURE CITATIONS

1. Griffin, A. E. Manganese removal with chlorine and chlorine dioxide. *J. New England Water Works Association* 72: pp. 321-327, September 1958.
2. Riddick, T. M., Lindsey, N. L., and Tomassi, A. Iron and Manganese in water supplies. *J. Am. Water Works A.* 50: pp. 688-702, May 1958.
3. Cotzias, G. C. Manganese in health and disease. *Physiol. Rev.* 38: pp. 503-532 (1958).
4. Drill, V. A. *Pharmacology in medicine*. Ed. 2. New York, N.Y. McGraw-Hill, 1958. pp. 709, 787, 794.
5. Sollmann, T. H. *A manual of pharmacology*. Ed. 8. Philadelphia, Pa., W. B. Saunders Co., 1957, pp. 1278-1281.
6. von Oettingen, W. F. Manganese its distribution, pharmacology and health hazards. *Physiol. Rev.* 15: pp. 175-201 (1935).
7. Cohen, J. M., Kamphake, L. J., Harris, E. K., and Woodward, R. L. Taste threshold concentrations of metals in drinking water. *J. Am. Water Works A.* 52: pp. 660-670, May 1960.
8. Griffin, A. E. Significance and removal of manganese in water supplies. *J. Am. Water Works A.* 52: pp. 1326-1334, October 1960.

NITRATE

Serious and occasionally fatal poisonings in infants have occurred following ingestion of well waters shown to contain nitrate (NO_3). 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.

in Minnesota alone (1). Wastes from chemical fertilizer plants and field fertilization may be sources of pollution. The causative factor producing serious blood changes in infants was first reported in 1945 in polluted water containing 140 mg/1 nitrate nitrogen (NO_3 -N) and 0.4 mg/1 nitrite (NO_2) ion in one case; in the second case, 90 mg/1 nitrate nitrogen and 1.3 mg/1 nitrite ion (2). Since this report, many instances of similar occurrences have been recorded not only in this country but in Canada, Great Britain, Belgium, Germany, and other countries.

The International Drinking Water Standards of 1958 took cognizance of the problem in noting that ingestion of water containing nitrates in excess of 50 mg/1 (as nitrate) may give rise to infantile methemoglobinemia but have included no limit. Taylor (3), in England, has suggested a limit of 20 mg/1 nitrate nitrogen. Bosch, et al. (1), consider nitrate nitrogen concentrations in excess of 10-20 mg/1 capable of producing cyanosis in infants. Various South American countries have recommended maximum permissible levels of from 0.5-228 mg/1 nitrate (NO_3) (0.1-51 mg/1 nitrate nitrogen) (4).

Cases of infantile nitrate poisoning have been reported to arise from concentrations ranging from 15-250 or more mg/1 nitrate nitrogen (usually with traces of nitrite ion) in instances in which the water was analyzed up to 1952, according to Campbell (5). Campbell himself reported a case from ingesting water with 26.2 mg/1 as nitrate nitrogen (116 mg/1 nitrate ion).

According to methods of analysis commonly employed for nitrate in water, the presence of appreciable amounts of chloride would result in an erroneously low value for nitrate, and the presence of considerable amounts of organic matter would give an erroneously high value for nitrate. Insufficient attention has been given this important factor in evaluating permissible safe levels of nitrate in water.

Nitrate poisoning appears to be confined to infants during their first few months of life; adults drinking the same water are not affected but breast-fed infants of mothers drinking such water may be poisoned (6). Cows drinking water containing nitrate may produce milk sufficiently high in nitrate to result in infant poisoning (5). Both man and animals can be poisoned by nitrate if the concentration is sufficiently great.

Among the more acceptable hypotheses for the specificity of nitrate poisoning of infants is the following: the gastric, free acidity of infants is low (a pH of 4 or greater), permitting the growth of nitrate-reducing flora in a portion of the gastrointestinal tract from

which nitrite absorption can occur. It is also stated that foetal hemoglobin forms methemoglobin more readily than the adult form.

According to a recent study from Germany (8), the primary causes of toxicity are an elevated nitrate concentration and the presence of an unphysiologic amount of nitrite-forming bacteria, especially in the upper portion of the digestive tract. Members of the coliform group and the genus *Clostridium* are capable of reducing nitrate to nitrite. In infants whose diet is mainly carbohydrate, it is believed that the coliform organisms are the group responsible; organisms capable of reducing nitrite to nitrogen are not normally present in the infant. Careful measurement of a number of other constituents in 23 offending well waters, nitrite, ammonia, chloride, and organic substances, failed to reveal a casual relation of these substances to the injury.

There are no reports of methemoglobinemia in infants fed water from public water supplies in the United States, although levels of nitrate in some may be routinely in excess of 45 mg/l. This may indicate that well water for analysis has often been improperly sampled or that some other as yet unknown factor is involved. Practically nothing is known of the variation in nitrate concentration in the same well. Because samples associated with injury are taken after injury occurs, it is conceivable that this delay has resulted in failure to measure truly injurious concentrations.

Sodium nitrate has been fed to rats for a lifetime without adverse effects at levels below 1 percent (10,000 ppm) in the diet (9); two dogs tolerated for 105 and 125 days, respectively, 2 percent nitrate in the diet without effects on blood or other adverse effects.

Nitrite is equally dangerous in water supplies. Although concentrations that occur naturally are generally of no health significance, nevertheless, they may enter water supplies inadvertently as a result of intentional addition to private supplies as anticorrosion agents.

A limit of 200 ppm of nitrite (or nitrate) in "corned" products has been set by Federal regulation on the basis that 100g corned beef could convert maximally from 10-40g hemoglobin to methemoglobin (1.4-5.7 percent of total hemoglobin). Adult human blood normally contains on the average of 0.7 percent methemoglobin; the blood of "heavy" smokers may contain 7-10 percent carboxyhemoglobin, another blood pigment conversion product incapable of transporting oxygen. Carbon monoxide in urban atmosphere adds perceptibly to the total inactive pigment. The summated blood pigment conversion products represent about the maximum tolerated without headache.

Because of the great difference in molecular weight between sodium nitrite, 69, and hemoglobin, 64,000, small increments of nitrite produce large quantities of methemoglobin (1g nitrite converts 460-

in infants whose blood volume is small, their total blood hemoglobin is decreasing after birth (from 17–20g to 10.5–12g), and their foetal hemoglobin is more readily converted to methemoglobin.

An instance of nitrite poisoning of children has been reported (10). The children ate frankfurters and bologna containing nitrite considerably in excess of the 200 ppm permitted.

Evidence in support of the recommended limit for nitrate is given in detail by Walton (7) in a survey of the reported cases of nitrate poisoning of infants in this country to 1951. The survey shows that no cases of poisoning were reported when the water contained less than 10 mg/1 nitrate nitrogen. Walton notes, however, that in many instances the samples for analysis were not obtained until several months after the occurrence of the poisoning.

In light of the above information and because of the uncertainty introduced by tardy analyses, the frequent lack of attention to possible interfering factors in the analysis, the health of the infant, and the uncertain influence of associated bacterial pollution, 10 mg nitrate nitrogen (or 45 mg nitrate) per liter of water is a limit which should not be exceeded.

At present there is no method of economically removing excessive amounts of nitrate from water. It is important, therefore, for health authorities in areas in which nitrate content of water is known to be in excess of the recommended limit to warn the population of the potential dangers of using the water for infant feeding and to inform them of alternative sources of water that may be used with safety.

LIMITS AND RANGES RELATED TO NITRATE WATER STANDARD

Average concentration adult human blood: 10 ug nitrate/100 ml (0.1 ppm).

Average daily urinary nitrate excretion: 500 mg (mainly from vegetables).

Strained baby foods: 0 (squash, tomatoes)—833 ppm nitrate (spinach).

Green Vegetables: 50 ppm nitrate (asparagus, dry weight), 3,600 ppm nitrate (spinach, dry weight).

Limit of nitrite (or nitrate) permitted in meat (or fish) products by Federal regulation: 200 ppm.

LITERATURE CITATIONS

1. Bosch, H. M., Rosenfield, A. B., Huston, R., Shipman, H. R., and Woodward, R. L. Methemoglobinemia and Minnesota well supplies. *J. Am. Water Works A.* 42: pp. 161–170, July 1950.
2. Comly, H. H. Cyanosis in infants caused by nitrates in well water. *J.A.M.A.* 129: 112–116 (1945).
3. Taylor, E. W. Examination of water and water supplies. Ed. 7. Philadelphia, Pa., The Blakiston Company, 1958, 841 pp.

4. Caballero, P. J. Discussion sobre las normas de calidad para aqua potable. *Organo Oficial de la Asociacion Interamericana de Ingenieria Sanitaria* 3: pp. 53-64. July 1949-June 1950.
5. Campbell, W. A. B. Methemoglobinemia due to nitrates in well water. *Brit. Med. J.*, 2: pp. 371-373 (1952).
6. Donahoe, W. E. Cyanosis in infants with nitrates in drinking water as cause. *Pediat.* 3: pp. 308-311 (1949).
7. Walton, G. Survey of literature relating to infant methemoglobinemia due to nitrate-contaminated water. *Am. J. Pub. Health* 41: pp. 986-996, August 1951.
8. Horn, K. Über gesundheitsstörungen durch nitrathaltiges trinkwasser vornehmlich bei saughlinger unter berücksichtigung der orthshygienischen Verhältnisse. *Stadtehygiene* 9: pp. 2-21-25 (1958).
9. Lehman, A. J. Nitrates and nitrites in meat products. *A. Food & Drug Officials, U.S.* 22: pp. 136-138, July 1958.
10. Orgeron, J. D., Martin, J. D., Caraway, C. T., Martine, R. M., and Hauser, G. H. Methemoglobin from eating meat with high nitrite content. *Pub. Health Rep.* 72: pp. 189-192, March 1957.

PHENOLS

The term "phenols" is understood to include cresols and xlenols. Both the International Drinking Water Standards and those of the U.S. Public Health Service of 1946 recommended a limit of 1 ug/1 of phenol in water. This limit is set because of the undesirable taste often resulting from chlorination of waters containing extremely low concentrations of phenol. Phenol concentrations of 5 mg/1 or more are injurious to fish, whereas 1 mg/1 or less will not seriously affect most fish. Concentrations from 15-1,000 mg/1 in the drinking water were reported (1) without observable effect on rats for extended periods; 5,000 mg/1 appeared likewise to exert no effect on digestion, absorption, or metabolism, but 7,000 mg/1 arrested growth and resulted in many stillbirths. Thus, concentrations injurious to health are far removed from those which impart unpleasant taste or affect fish. Phenol is largely detoxified in the mammalian body by conjugation to far less toxic substances (2).

Although additional information has been developed (3) since the 1946 Standard was set, its nature indicates no need of a change in the former limit for phenols 0.001 mg/1 (1 ug/1).

LITERATURE CITATIONS

1. Heller, V. G., and Pursell, L. Phenol-contaminated waters and their physiologic action. *J. Pharm. Exp. Ther.* 63: pp. 99-107 (1938).
2. Williams, R. T. Detoxication mechanism. New York, N.Y., John Wiley & Sons, Inc., 1959. pp. 278-317.
3. Burttschell, R. H., Rosen, A. A., Middleton, F. M., and Ettinger, M. B. Chlorine derivatives of phenol causing taste and odor. *J. Am. Water Works A.* 51: pp. 205-214, February 1959.

The presence of selenium in water has heretofore been a matter of regional importance (1). The fact that it is now recognized as being toxic to both man and animals makes it essential that limits be set for all water intended for human consumption.

Selenium is known to produce "alkali disease" in cattle, and its effects, like those of arsenic, may be permanent (1,2). Recent reports indicate also that selenium may increase the incidence of dental caries in man (3). Of greater importance in limiting the concentration of selenium is its potential carcinogenicity (4). Rats fed a diet containing varying concentrations of selenium (3 to 40 mg/1) showed toxic effects at all levels, the outstanding pathologic lesion being hepatic cell tumors.

From very limited information (5) concentrations of selenium in water considered safe for man have been found toxic for fish.

In view of the potential seriousness of above reported effects, it is recommended that the limits for selenium be lowered from its present value of 0.05 mg/1 to 0.01 mg/1 and concentrations in excess of this lower value be used as grounds for rejection of the supply.

LITERATURE CITATIONS

1. Dudley, H. C. Toxicology of selenium: I. A study of the distribution of selenium in acute and chronic cases of selenium poisoning. *Am. J. Hyg.* 23: pp. 169-186 (1936).
2. Drill, V. A. *Pharmacology in Medicine*. Ed. 2., New York, N.Y., McGraw-Hill, 1958, pp. 794-795.
3. Hadjimarkos, D. M., and Bonhorst, C. W. The trace element selenium and its influence on dental caries susceptibility. *J. Pediat.* 52: pp. 274-278 (1958).
4. Fitzhugh, O. G., Nelson, A. A., and Bliss, E. I. The chronic oral toxicity of selenium. *J. Pharm. Exp. Ther.* 80: pp. 289-299 (1944).
5. Ellis, M. M. Pollution and aquatic life. *Am. Wildlife* 26: pp. 38, 45-46 (1937).

SILVER

The need to set a water standard for silver (Ag) arises from its intentional addition to waters for disinfection. The chief effect of silver in the body is cosmetic, which consists of a permanent blue-grey discoloration of the skin, eyes, and mucous membranes which is as unsightly and disturbing to the observer as to the victim. The amount of colloidal silver required to produce this condition (argyria, argyrosis), and which would serve as a basis of determining the water standard, is not known, 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 taken by mouth or by injection. 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 is 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/1 (nature of the silver salt unstated) showed pathologic changes in kidneys, liver, and spleen at 400, 700, and 1,000 ug/1.

A study (9) of the resorption of silver through human skin using radio-silver 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.

Great uncertainty, however, currently surrounds any evaluation of the amount of silver introduced into the body when silver-treated

vegetables belonging to the family Brassicaceae, such as cabbage, turnip, cauliflower, and onion, would combine with residual silver in the cooking water. The silver content of several liters of water could thus be ingested.

Despite these uncertainties and the present lack of appropriate drinking water studies, it is possible to derive a tentative drinking water standard for silver by using silver deposited in excess of 1g in the integument of the body as an end point that must not be exceeded. Assuming that all silver ingested is deposited in the integument, it is readily calculated that 10 ug/1 could be ingested for a lifetime before 1g silver it attained from 2 liters water intake per day; 50 ug/1 silver could be ingested approximately 27 years without exceeding silver deposition of 1g.

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 increased absorbability of silver as silver-bound sulfur components of food cooked in silver-treated waters, the intake for which absorption was reported in 1940 to amount to 60–80 ug per day (10); and because of the above calculation, a concentration in excess of 50 ug/1 is grounds for rejection of the supply.

LITERATURE CITATIONS

1. Hill, W. B., and Pillsbury, D. M. *Argyria*. The pharmacology of silver. Baltimore, Md., Williams & Wilkins, 1939, 172 pp.
2. *Ibid.*, *Argyria Investigation—Toxicologic Properties of Silver*, Am. Silver Producers Res. Proj. Report. Appendix II (1957).
3. Bell, C. D., Cookey, D. B., and Nickel, W. R. Amalgam tattoo-localized argyria. *A.M.A. Arch. Derm. Syph.* 66: pp. 523–525 (1952).
4. Joseph, M., and Van Deventer, J. B., *Atlas of Cutaneous Morbid Histology*. W. T. Kliner & Co., Chicago, 1906.
5. Scott, K. G., and Hamilton, J. G. The metabolism of silver in the rat with radiosilver used as indicator. *U. of Cal. Publ. in Pharm.* 2: pp. 241–262 (1950).
6. Wyckoff, R. C., and Hunter, F. R. Spectrographic analysis of human blood. *Arch. Biochem.* 63: pp. 454–460 (1956).
7. Aub, J. C., and Fairhall, L. T. Excretion of silver in urine. *J.A.M.A.* 118: p. 319 (1942).
8. Just, J., and Szniolis, A. Germicidal properties of silver in water. *J. Am. Water Works A.* 28: pp. 492–506, April 1936.
9. Norgaard, O. Investigations with Radio Ag¹¹¹ into the resorption of silver through human skin. *Acts Dermatovener* 34: pp. 415–419 (1954).
10. Kehoe, R. A., Cholak, J., and Story, R. V. Manganese, lead, tin, copper and silver in normal biological material. *J. Nutr.* 20: pp. 85–98 (1940).

ZINC

Limits for concentrations of zinc in drinking waters have been established as follows: (a) USPHS Drinking Water Standards (1946), 15 mg/1; (b) Ohio and North Dakota, 1 mg/1; (c) International Drinking Water Standards (1958), permissible—5 mg/1 and excessive—15 mg/1; (d) various South American Countries, 5 to 15 mg/1.

Zinc is an essential and beneficial element in human metabolism (1). The daily requirement for preschool-age children is 0.3 mg Zn/kg. Total zinc in the adult averages 2g. Zinc content of human tissues ranges from 10–200 ppm wet weight, the retina of the eye and the prostate containing the largest concentrations (500–1,000 ppm). Three percent of all blood zinc is in the white blood cells. The daily adult human intake averages 10–15 mg; excretion of zinc averages about 10 mg daily in the feces and 0.4 mg in the urine. Zinc deficiency in animals lead to growth retardation that is overcome by adequate dietary zinc. The activity of several body enzymes is dependent on zinc.

A group of individuals stationed at a depot used a drinking water supply containing zinc at 23.8 to 40.8 mg/1 and experienced no known harmful effects. Communities have used waters containing from 11–27 mg/1 without harmful effects (2, 3). Another report (4) stated spring water containing 50 mg/1 was used for a protracted period without noticeable harm. On the other hand, another supply containing approximately 30 mg/1 was claimed to cause nausea and fainting.

Zinc salts act as gastrointestinal irritants. Although the illness is acute, it is transitory. The emetic concentration range in water is 675–2,280 mg/1. In tests performed by a taste panel, 5 percent of the observers were able to distinguish between water containing 4 mg/1 (when present as zinc sulfate) and water containing no zinc salts (5). Soluble zinc salts at 30 mg/1 impart milky appearance to water, and at 40 mg/1, a metallic taste (6).

Inasmuch as zinc in water does not cause serious effects on health but produces undesirable esthetic effects, it is recommended that concentrations of zinc be kept below 5 mg/1.

Cadmium and lead are common contaminants of zinc used in galvanizing. Assuming that zinc is dissolved from galvanized water pipe no less than cadmium, dissolution of zinc to produce 5 mg/1 would be accompanied by something less than the allowable 0.01 mg cadmium per liter when cadmium contamination of the zinc is as high

creased by something less than the allowable 0.05 mg/1 when lead contamination of the zinc is as high as 0.6 percent.

LIMITS AND RANGES RELATIVE TO ZINC WATER STANDARD

Food (7)—Milk, 4 mg/1

Egg (Hen)—1 mg

Cd content of galvanized pipe: 0.014–0.04 percent. Average 0.03 percent.

Pb content of galvanized pipe: 0.24–0.6 percent. Average 0.45 percent.

Urban air concentration: Average 2 ug/m³ (8).

Concentrations toxic to fish: 0.3–4 mg/1, depending on degree of water hardness (9).

Drinking water containing 50 mg/1 (as Sulfate) was not harmful to rats which used it for 6 weeks (3).

LITERATURE CITATIONS

1. Vallee, B. L. Zinc and its biologic significance, *Arch Indust. Health.* 16: pp. 147–154, July 1957.
2. Anderson, E. A., Reinhard, C. E., and Hammel, W. D. The corrosion of zinc in various waters. *J. Am. Water Works A.* 26: pp. 49–60, January 1934.
3. Bartow, E., and Weigle, O. M. Zinc in water supplies. *Indust. Eng. Chem.* 24: pp. 463–465 (1932).
4. Hinman, J. J., Jr. Desirable characteristics of a municipal water supply. *J. Am. Water Works A.* 30: pp. 484–494, March 1938.
5. Cohen, J. M., Kamphake, L. J., Harris, E. K., and Woodward, R. L. Taste threshold concentrations of metals in drinking water. *J. Am. Water Works A.* 52: pp. 660–670, May 1960.
6. Kehoe, R. A., Cholak, J., and Largent, E. J. The hygienic significance of the contamination of water of certain mineral constituents. *J. Am. Water Works A.* 36: pp. 645–657, June 1944.
7. Sollmann, T. H. A manual of pharmacology, Ed. 8: Philadelphia, Pa., W. B. Saunders Co., 1957, pp. 1302–1305.
8. U.S. Public Health Service. Air pollution measurements of the national air sampling network. Cincinnati, Ohio, Robt. A. Taft San. Eng. Center (1958).
9. Jones, J. R. E. The relative toxicity of salts of lead, zinc, and copper to the Stickleback (*Gasterosteus aculeatus* L.) and the effect of calcium on the toxicity of lead and zinc salts. *Exper. Biol. J.* 15: pp. 394–407 (1938).

E—RADIOACTIVITY

The effects of radiation on human beings are viewed as harmful and any unnecessary exposure to radiation should be avoided. In this discussion we are concerned with radiation from radioactive materials in the environment, particularly in water, food, and air.

The development of the nuclear industry has been attended by a small, unavoidable increase of radioactivity in the environment. Nuclear weapons testing causes an increase of radioactivity from fallout. Exposure of human beings to environmental sources of radiation

should be minimized insofar as is technically and economically feasible.

The Federal Radiation Council (1) has provided guidance for Federal agencies conducting activities designed to limit exposure of individuals of population groups to radiation from radioactive materials deposited in the body as a result of their occurrence in the environment.

The following recommendation of the Federal Radiation Council is considered especially pertinent in applying these Standards: (2)

“There can be no single permissible or acceptable level of exposure without regard to the reason for permitting the exposure. It should be general practice to reduce exposure to radiation, and positive effort should be carried out to fulfill the sense of these recommendations. It is basic that exposure to radiation should result from a real determination of its necessity.”

The Federal Radiation Council criteria (1) (3) (4) have been observed in establishing the limits for radioactivity in the Drinking Water Standards. It should be noted that these Federal Radiation Council guides apply to normal peacetime operations.

The Federal Radiation Council guides are predicated upon three ranges of daily intake of radioactivity. For each range, a measure of control action was defined, which represented a graded scale of control procedures. These are shown by the following table:

TABLE I.—*Graded scales of action*

Ranges of transient rates of daily intake	Graded scale of action
Range I.....	Periodic confirmatory surveillance as necessary.
Range II.....	Quantitative surveillance and routine control.
Range III.....	Evaluation and application of additional control measures as necessary.

The Federal Radiation Council (4) further defined the action to be taken by stating that: “Routine control of useful applications of radiation and atomic energy should be such that expected average exposures of suitable samples of an exposed population group will not exceed the upper value of Range II.” Furthermore, they recommended, with respect to Range III, that “Control actions would be designed to reduce the levels to Range II or lower and to provide stability at lower levels.”

The radionuclide intake ranges recommended are the sum of radioactivity from air, food and water. Daily intakes were prescribed with the provision that dose rates be averaged over a period of one

eral Radiation Council (1) are shown in the following table:

TABLE II.—*Ranges of transient rates of intake (micromicrocures per day) for use in graded scale of actions summarized in Table I*

Radionuclides	Range I	Range II	Range III
Radium-226.....	0-2	2-20	20-200
Iodine-131 ¹	0-10	10-100	100-1000
Strontium-90.....	0-20	20-200	200-2000
Strontium-89.....	0-200	200-2000	2000-20,000

¹ In the case of Iodine-131, the suitable sample would include only small children. For adults, the RPPG for the thyroid would not be exceeded by rates of intake higher by a factor of 10 than those applicable to small children.

The Advisory Committee, in considering limits which should be established for drinking water, recommended limits for only two of the above nuclides, Radium-226 (3 uuc per liter) and Strontium-90 (10 uuc per liter). Iodine-131 is not found in significant quantities in public water supplies frequently enough to call for routine monitoring and Strontium-89 levels are not likely to be significant unless Strontium-90 levels also are high.

In the case of Radium-226, above-average levels of intake generally occur only in unusual situations where the drinking water contains naturally occurring Radium-226 in greater than average amounts, as in the case of certain ground waters, or from the pollution of the supply by industrial discharges of waste containing radium. With this in mind, a limit of 3 uuc/liter has been set for Radium-226 in drinking water. If one assumes a daily intake of such drinking water of about 2 liters per day, this would result in a daily intake from water of 6 uuc which falls in the lower portion of Range II in the above table. If there is evidence that Radium-226 from sources other than water is greater than usual, levels may have to be reduced below the above limit using the guides established by the Federal Radiation Council.

The principal source of Strontium-90 in the environment to date has been due to fallout from weapon tests, and human intake of Strontium-90 to date has been primarily from food. In recognition of this fact, the limit for Strontium-90 in water has been set at 10 uuc/liter, a limit substantially higher than the highest level found in public water supplies to date.

The Standards recognized the need to provide guidance for those situations where the limits are exceeded. In these instances, the Standards provide for the continued acceptance of the water supply if radioactivity from all other sources in addition to that from the water does not exceed intake levels recommended by the Federal Radiation Council for control action (the upper limit of Range II). It is essen-

tial in such instances for the certifying authority to determine with reasonable confidence that this latter condition is met.

Although a great variety of radionuclides may be present in drinking water, it has not been considered necessary to establish limits for general application to water supplies for other than the above two at this time. If significant concentrations of radioactivity are found in drinking water, an effort should be made to determine the radionuclides present and, where appropriate, to reduce their concentrations as much as feasible.

In assessing the hazard of radionuclides for which limits have not been set in these Standards, or for which guidance has not yet been provided by the Federal Radiation Council, it is suggested that the values (MPC_w for the 168-hour week) in table I, of the report of the International Commission on Radiological Protection (6) or the National Committee on Radiation Protection (7), adjusted by a factor appropriate for exposure of the general population, be used. When mixtures of radionuclides are present the permissible concentration of any single nuclide must be reduced by an amount determined through applicable calculations in these reports.

In these Standards an upper limit of 1,000 μmC per liter of gross beta activity (in the absence¹ of alpha emitters and Strontium-90) has been set. If this limit is exceeded the specific radionuclides present must be identified by complete analysis in order to establish the fact that the concentrations of nuclides will not produce exposures above the recommended limits established in the Radiation Protection Guides. (3)

LITERATURE CITATIONS

1. Federal Radiation Council, Radiation Protection Guidance for Federal Agencies, Memorandum for the President, Sept. 13, 1961. Reprint from the *Federal Register* of Sept. 26, 1961.
2. Federal Radiation Council, Radiation Protection Guidance for Federal Agencies, Memorandum for the President, May 13, 1960. Reprint from the *Federal Register* of May 18, 1960.
3. Federal Radiation Council, Report No. 1, Background Material for the Development of Radiation Protection Standards, May 13, 1960. Government Printing Office, 1960.
4. Federal Radiation Council, Report No. 2, Background Material for the Development of Radiation Protection Standards, September 1961. Government Printing Office, 1961.
5. Radiological Health Data, Public Health Service monthly publication, May 1962. Government Printing Office.

¹ "Absence" is intended to mean a negligibly small fraction of the limits established for these nuclides and the limit for unidentified alpha emitters is taken as the listed limit for Radium-226.

on permissible dose for internal radiation. New York, N.Y., Pergamon Press, Inc., 1959.

7. National Committee on Radiation Protection: Maximum permissible body burden and maximum permissible concentrations of radionuclides in air and in water for occupational exposure. National Bureau of Standards Handbook No. 69, Washington, D.C., U.S. Government Printing Office, June 4, 1959.

F—MEMBERSHIP OF ADVISORY COMMITTEE, TECHNICAL SUBCOMMITTEE AND TASK FORCE ON TOXICOLOGY

Membership of Advisory Committee¹

O. C. Hopkins, *Chairman, U.S. Public Health Service*, Washington, D.C.

George W. Burke, Jr., *Secretary, U.S. Public Health Service*, Washington, D.C.

REPRESENTATIVES OF FEDERAL ORGANIZATIONS

Food and Drug Administration: L. M. Beacham, Jr., Deputy Director, Division of Food, U.S. Department of Health, Education, and Welfare, Washington, D.C.
U.S. Geological Survey: S. K. Love, Chief, Quality of Water Branch, U.S. Department of the Interior, Washington, D.C.

REPRESENTATIVES OF SCIENTIFIC ASSOCIATIONS AND INDUSTRY

Air Transport Association of America: K. L. Stratton, Medical Director, American Airlines, LaGuardia Airport Station, Flushing 71, N.Y.
American Chemical Society: T. E. Larson, Head, Chemistry Section, State Water Survey Division, Urbana, Ill.
American Dental Association: Robert A. Downs, Chief, Public Health Dentistry Section, State Department of Public Health, Denver, Colo.
American Medical Association: W. D. Stovall, Director, State Laboratory of Hygiene, Madison, Wis.
American Public Health Association: Daniel A. Okun, Department of Sanitary Engineering, School of Public Health, University of North Carolina, Chapel Hill, N.C.
American Society of Civil Engineers: Thomas R. Camp, Camp, Dresser, and McKee, Consulting Engineers, 18 Tremont Street, Boston 8, Mass.
American Water Works Association: Oscar Gullans, Chief Filtration Engineer, So. District Filtration Plant, 3300 East Cheltenham Place, Chicago, Ill.
Association of American Railroads: R. S. Glynn, Director, Sanitation Research, Association of American Railroads, 3140 South Federal Street, Chicago, Ill.
Association of State and Territorial Public Health Laboratory Directors: F. R. Hassler, Director of Laboratories, State Department of Health, Oklahoma City, Okla.
Conference of State Sanitary Engineers: E. C. Jensen, Chief, Division of Engineering and Sanitation, State Department of Health, Seattle, Wash.
National Committee on Radiation Protection: John B. Hursh, Professor, Department of Radiation Biology, University of Rochester, Rochester, N.Y.

¹ Official positions and addresses at time of appointment.

Society of American Bacteriologists: Charles C. Croft, Assistant Chief of Laboratories, State Department of Health, Columbus, Ohio.

Water Pollution Control Federation: F. W. Gilcreas, Professor of Sanitary Science, Department of Civil Engineering, University of Florida, Gainesville, Fla.

MEMBER AT LARGE

Henry J. Ongerth, Supervising Sanitary Engineer, State Department of Public Health, 2151 Berkeley Way, Berkeley, Calif.

Technical Subcommittee, Officers of the Public Health Service

O. C. Hopkins, Deputy Chief, Division of Water Supply and Pollution Control, Washington, D.C. (Co-chairman).

Richard Woodward, Chief, Engineering Section, Water Supply and Pollution Research Branch, Cincinnati, Ohio (Co-chairman).

George W. Burke, Jr., Chief, Evaluation and Review Section, Technical Services Branch, Division of Water Supply and Pollution Control, Washington, D.C.

Paul Kabler, Chief, Microbiology Section, Water Supply and Pollution Research Branch, Cincinnati, Ohio.

M. B. Ettinger, Chief, Chemistry and Physics Section, Water Supply and Pollution Research Branch, Cincinnati, Ohio.

H. G. Magnuson, Chief, Occupational Health Program, Division of Special Health Services, Washington, D.C.

H. E. Stokinger, Chief, Toxicologic Services, Occupational Health Field Headquarters, Cincinnati, Ohio.

James G. Terrill, Assistant Chief, Division of Radiological Health, Washington, D.C.

Malcolm Hope, Chief, General Engineering Program, Division of Engineering Services, Washington, D.C.

Floyd Taylor, Chief, Water Supply Activity, Division of Engineering Services, Washington, D.C.

Task Force on Toxicology

H. E. Stokinger, Chief, Toxicologic Services, Occupational Health Field Headquarters, Public Health Service, Cincinnati, Ohio (Chairman).

Kenneth P. DuBois, Professor, Department of Pharmacology, University of Chicago, Chicago 37, Ill.

Harvey Haag, Professor of Pharmacology, Medical College of Virginia, Richmond 19, Va.

Wayland J. Hayes, Jr., Chief of Toxicology, Communicable Disease Center, Public Health Service, Atlanta, Ga.

Harry Hays, Director, Advisory Center on Toxicology, National Research Council, Washington, D.C.

Arnold J. Lehman, Director, Division of Pharmacology, Food and Drug Administration, Washington, D.C.

