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Part IV

Environmental Protection Agency

40 CFR Part 141

National Primary Drinking Water Regulations; Synthetic Organic Chemicals, Inorganic Chemicals and Microorganisms; Proposed Rule

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ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 141

[OW-FRL-2909-7]

National Primary Drinking Water Regulations; Synthetic Organic Chemicals, Inorganic Chemicals and Microorganisms

AGENCY: Environmental Protection Agency (EPA).

ACTION: Proposed rulemaking.

SUMMARY: This proposed rule under the Safe Drinking Water Act (42 U.S.C. 300f *et seq.*) would establish Recommended Maximum Contaminant Levels (RMCLs) for synthetic organic chemicals (SOCs), inorganic chemicals (IOCs) and microbiological parameters in drinking water. Proposed RMCLs (goals) for substances considered to be probable human carcinogens are set at the zero level and RMCLs for substances not treated as probable human carcinogens are set based upon chronic toxicity or other data. SOCs, IOCs and microorganisms that are not included in this proposal may be considered for subsequent rulemaking under the Safe Drinking Water Act.

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

Public comments are solicited on each of the proposed RMCLs as well as on the regulatory approach being considered. Specific scientific and technical reviews and comments are requested on the support documents on analytical methods and health effects.

The notice and supporting documentation also contains calculations and information regarding risks from contaminants that EPA is proposing not to regulate. EPA has published nonregulatory Drinking Water Health Advisories on these contaminants. Public comment is also requested on the scientific basis for those calculations and whether EPA should develop Primary Drinking Water Regulations, or Health Advisories, or take no action.

DATES: Written comments should be submitted by March 13, 1986. A public hearing will be held in Washington, D.C. on January 28 and 29, 1986, beginning at 9:00 am in Conference Room 1, adjacent to the Washington Information Center, EPA, 401 M Street SW., Washington, DC.

ADDRESSES: Send written comments to Comment Clerk, Criteria and Standards Division, Office of Drinking Water (WH-550), Environmental Protection Agency, 401 M Street SW., Washington, DC 20460. A copy of the comments and supporting documents will be available for review during normal business hours at the EPA, Room 2904 (rear), 401 M Street SW., Washington, DC 20460. It is requested that anyone planning to attend the public hearing (especially those who plan to make statements) register in advance by calling or writing Ms. Arnetta Davis at 202/382-7575, EPA, WH-550, 401 M Street SW., Washington, DC 20460. Persons planning to make statements at the hearing are encouraged to submit written copies of their remarks at the time of the hearing.

Supporting documents cited in Section XI will be available for inspection at the Drinking Water Supply Branches in EPA's Regional Offices.

I. JFK Federal Bldg., Boston, MA 02203, Phone: (617) 223-6486, Jerome Healy
II. 26 Federal Plaza, Room 824, New York, NY 10278, Phone: (212) 264-1800, Walter Andrews
III. 6th & Walnut Sts., Philadelphia, PA 19106, Phone: (215) 597-9873, Bernie Sarnowski
IV. 345 Courtland Street, Atlanta, GA 30365, Phone: (404) 881-3781, Robert Jourdan
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VI. 1201 Elm St., Dallas, TX 75270, Phone: (214) 767-2620, James Graham
VII. 726 Minnesota Ave., Kansas City, KS 66101, Phone: (913) 236-2815, Gerald R. Foree
VIII. 1860 Lincoln St., Denver, CO 80295, Phone: (303) 293-2815, Marc Alston
IX. 215 Fremont St., San Francisco, CA 94105, Phone: (415) 974-8076, Leslie Ragle
X. 1200 Sixth Ave., Seattle, WA 98101, Phone: (206) 442-1225, Jerry Opatz.

Copies of draft health criteria, occurrence, analytical methods and health advisory documents will be available for a fee from the National Technical Information Service (NTIS), U.S. Department of Commerce, 5285, Port Royal Road, Springfield, Virginia 22161. The toll free number is 800/336-4700; local: 703/487-4650.

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

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I. Statutory Requirements

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

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

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

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

House Report No. 93-1185, July 10, 1974, at 20. In addition, a list of contaminants is to be included in the regulations for "any contaminant the level of which cannot be accurately enough measured in drinking water to establish a Recommended Maximum Contaminant Level and which may have any adverse effect upon the health of persons." 1412(b)(1)(B).

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

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

II. Regulatory Framework

Issuing Revised Primary Drinking Water Regulations is the second step in the evolution of the primary drinking water regulations mandated by the SDWA.

In the *first step*, the National Interim Primary Drinking Water Regulations were promulgated on December 24, 1975, with an effective date of June 24, 1977. Amendments were issued in 1976, 1979 and 1980. See 40 CFR Part 141. Maximum contaminant levels (MCLs) and monitoring and reporting requirements were set for numerous microbiological, inorganic, organic, and radionuclide contaminants (40 CFR Part 141, Subpart B).

Section 1412(e) of the SDWA directed EPA to arrange for the National Academy of Sciences (NAS) or an equivalent organization to conduct a study to assess the health effects of contaminants in drinking water and to provide proposals for RMCLs.

As the *second step*, section 1412(b)(1)(B) provided that EPA must propose and promulgate National Revised Primary Drinking Water

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Regulations (NPDWR) that would include RMCLs, MCLs and monitoring and reporting requirements for those contaminants that may have any adverse effect on human health.

Development of the NPDWR will be accomplished in four phases:

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

This notice is Phase II—proposal of RMCLs for certain inorganic, organic and biological contaminants. Because of the great number of possible substances to be considered, additional drinking water contaminants from these three groups will be considered in later iterations of NPDWR.

In general, the procedure for all four phases will be similar as is shown in Figure 1. Generally, it requires approximately one year for each major regulatory action (e.g., RMCL to proposal). The status of each Phase is provided below:

- Phase I:

ANPRM, March 4, 1982 47 FR 9350
Public Meeting, April 28, 1982
Four Public Workshops, June–August 1982
RMCL Proposal, June 12, 1984 49 FR 24320
Final RMCL rule/MCL Proposal, Scheduled for October 1985

- Phase II:

Microbiological Workshop, December 4, 1981
ANPRM, October 5, 1983 48 FR 45502
Four Public Workshops, September-December 1983
Public Meeting, December 13, 1983
Final RMCL rule/MCL Proposal, Scheduled for October 1986.

- Phase IIA:

Notice, December 1, 1981 46 FR 58345
ANPRM, October 5, 1983 48 FR 45502
RMCL Proposal, May 14, 1985 50 FR 20164
Final RMCL/MCL Proposal, Scheduled for November, 1985

- Phase III:

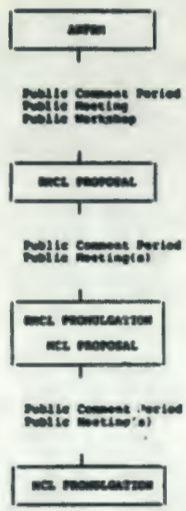
Radionuclides Workshop, May 1983
ANPRM, October 5, 1983 48 FR 45502
RMCL Proposal, Scheduled for December, 1985

- Phase IV, ANPRM Scheduled for 1986

Figure 1

NATIONAL PRIMARY DRINKING WATER REGULATIONS

REGULATORY PROCEDURE



III. Regulatory Background

The discussion in this section provides a brief overview of: (1) The Interim Regulations and the implementation experiences, (2) the National Academy of Sciences (NAS) role in development of the Revised Regulations, (3) areas that will be addressed in the Revised Regulations, and (4) summary of comments on the Phase II ANPRM for these contaminants.

A. Interim Regulations

As required by the SDWA, EPA acted quickly following the passage of the SDWA in publishing Interim Primary Drinking Water Regulations. Regulations were established initially for ten inorganic chemicals, six pesticides and two microbiological indicator contaminants. Amendments were later added on radionuclides, trihalomethanes and corrosion/sodium monitoring. The regulations set MCLs, monitoring and reporting requirements. Analytical methods were specified as well as public notification requirements (40 FR 59566, Radionuclides, 44 FR 68641, 45 FR 57332).

These regulations apply to some 60,000 community water systems and 160,000 non-community water systems. The distinction between community and non-community systems is that non-community systems serve a transient population (as opposed to residential). Only those MCLs in the Interim Regulations thought to have potentially acute health concerns were applied to the non-community systems; these included total coliforms, turbidity and nitrate.

RMCLs were not established in the Interim Regulations because the SDWA only specifies that RMCLs are to be set in establishing the Revised Regulations. The MCLs in the Interim Regulations were to:

"protect health to the extent feasible, using technology, treatment techniques, and other means which the Administrator determines are generally available (taking costs into consideration) on the date of enactment of this title." Section 1412(a)(2)

This mandate for Interim MCLs is similar to the MCLs in the Revised Regulations; however, the Revised Regulations are different in two fundamental respects. First, in the Revised Regulations, a goal (i.e., the RMCL) is established and then the MCL is set as close to the goal as is feasible. RMCLs are to be established for a comprehensive number of contaminants. This proposal deals with the RMCLs. Second, in setting MCLs, feasible is defined in the SDWA as using the *best* (emphasis added) technology, treatment techniques, and other means which the Administrator finds are generally available (taking costs into consideration).

B. National Academy of Sciences (NAS)

Under the SDWA, NAS is charged with assessing the following:

- Summary/evaluation of relevant publications and studies.
- Methodologies/assumptions used in estimating levels at which adverse effects occur.
- Methodologies/assumptions for estimating margin of safety for drinking water regulations.
- Develop proposals for RMCLs.
- List of contaminants the level of which cannot be determined in drinking water that pose a health risk.
- Research priorities.

While the NAS did not provide proposals of RMCLs, the NAS has provided guidance on the above items and on the toxicological effects of numerous drinking water contaminants. The five volumes of *Drinking Water and Health* include the following:

Volume I (1977)

- Safety and risk assessment procedures.

- Toxicity of organic, inor
microbial and radionuclide
contaminants.

Volume II (1980)

- Disinfection of drinking
- Chemistry of disinfectio
drinking water.
- Granular activated carb

Volume III (1980)

- Epidemiology studies.
- Problems of risk estime
- Toxicity of selected co
- Nutritional aspects of d
water contaminants.

Volume IV (1982)

- Distribution system pot
problems.
- Toxicity of inorganic an
chemicals.

Volume V (1983)

- Toxicity of selected con
- Epidemiology of arsenic
asbestos.

Because the NAS did not
proposed RMCLs, the Agen
developed the proposed RM
notice based upon its own e
which included the NAS rep
with other pertinent data.

C. Issues Being Addressed i Regulations

As directed by the SDWA
Revised Regulations will ad
variety of problems in drink
quality in public water syst
the United States. Much exp
been gained from the implem
the Interim Regulations and
comprehensive review and
the provisions of the Interim
is being undertaken during
of the Revised Regulations.
new problems in drinking w
have been discovered that
addressed in the Revised R
Under the requirements and
of the SDWA, the basic que
considered in the efforts inc

- For which contaminant
regulations be set?
- What levels for the RM
MCLs would be appropriat
- What monitoring and
requirements would be app

The Advance Notice of P
Rulemaking (ANPRM), issu
1984 (49 FR 45502), provide
with an opportunity to revi
and comment to the Agency
development stages. The A
followed by four public tec
workshops and a public me
which the regulatory and s
issues were discussed. The

• Toxicity of organic, inorganic, microbial and radionuclide contaminants.

Volume II (1980)

• Disinfection of drinking water.
• Chemistry of disinfection in drinking water.
• Granular activated carbon.

Volume III (1980)

• Epidemiology studies.
• Problems of risk estimation.
• Toxicity of selected contaminants.
• Nutritional aspects of drinking water contaminants.

Volume IV (1982)

• Distribution system potential health problems.
• Toxicity of inorganic and organic chemicals.

Volume V (1983)

• Toxicity of selected contaminants.
• Epidemiology of arsenic and asbestos.

Because the NAS did not provide proposed RMCLs, the Agency has developed the proposed RMCLs in this notice based upon its own evaluations which included the NAS reports along with other pertinent data.

C. Issues Being Addressed in Revised Regulations

As directed by the SDWA, the Revised Regulations will address a wide variety of problems in drinking water quality in public water systems across the United States. Much experience has been gained from the implementation of the Interim Regulations and a comprehensive review and revision of the provisions of the Interim Regulations is being undertaken during development of the Revised Regulations. In addition, new problems in drinking water quality have been discovered that will be addressed in the Revised Regulations. Under the requirements and definitions of the SDWA, the basic questions being considered in the efforts include:

• For which contaminants should regulations be set?
• What levels for the RMCLs and MCLs would be appropriate?
• What monitoring and reporting requirements would be appropriate?

The Advance Notice of Proposed Rulemaking (ANPRM), issued October 5, 1984 (49 FR 40502), provided the public with an opportunity to review the issues and comment to the Agency early in the development stages. The ANPRM was followed by four public technical workshops and a public meeting during which the regulatory and scientific issues were discussed. The public comments received on the ANPRM are discussed in this section, Part D.

This proposal addresses only the selection of contaminants for possible regulation and the proposed RMCLs for these substances. Public comments and workshop results addressing MCLs and monitoring and reporting requirements will be addressed in the MCL proposal which will be published with the promulgated RMCLs. However, to provide some perspective on approaches being considered for the regulations, a brief overview of problems that will be considered in the development of the RMCLs, MCLs, and monitoring and reporting requirements is provided below.

Implementation of the Interim Regulations has shown the following major areas of concern:

• Compliance by Small Systems

For the most part, compliance problems with the MCLs and monitoring and reporting requirements have generally been associated with small systems. Resources are often limited in small systems and when a drinking water problem is present, the small systems generally are least able to cope with it.

Variances may be granted when a system, "because of characteristics of the raw water sources which are reasonably available to the systems, cannot meet the requirements . . . despite application of the best technology, treatment techniques, or other means, which the Administrator finds are generally available (taking costs into consideration)." (Section 1415(a)(1)(A)). Variances do not have a fixed date in the law for the system to come into compliance but the system must be put on a compliance schedule requiring compliance as expeditiously as practicable.

The approach to variances in the Revised Regulations would be similar to that promulgated for the trihalomethane regulations (48 FR 8403, Feb. 28, 1983). The best generally available technology (GAT) under section 1415 would be defined for each regulated contaminant, taking cost into consideration and possibly categorizing by system characteristics such as size or water source. States would evaluate each case on a site-specific basis to determine if the identified GAT was appropriate and effective for that system. In addition to central treatment alternatives, decentralized options such as use of bottled water and point-of-use treatment devices are being considered as control measures to reduce contamination until the system can reach compliance with the NPDWR through other means.

• Strength of Evidence of Potential Health Effects

Because of the added costs of treatment, many systems remain out of compliance with several MCLs; some systems remain unconvinced that the net benefits of contaminant reduction is worth the costs. This issue often relates to the availability and strength of evidence of data on potential health effects.

Comprehensive assessments have been conducted of the available information on potential health effects of every contaminant in this proposal. State-of-the-art scientific methods have been utilized in determining the potential health effects. All of the accumulated/analyzed data are summarized and presented for public comment in the Health Effects Criteria Documents referenced in Section X of this notice.

• Monitoring Inflexibility

The Interim Regulations require monitoring to assess compliance with the MCLs at set frequencies for certain contaminants; for example, monitoring for inorganic compounds must be conducted at least once per year or once per three years for supplies using surface or ground water sources, respectively. While monitoring once a year or every three years does not seem to be overly demanding, this can be costly for small systems, and upon those States that conduct monitoring for certain of the systems (e.g., small systems) within their boundaries. States have reported that certain of these inorganic compounds have not been detected at significant levels in the drinking water in many systems and the probability of future contamination is very slight. Monitoring has shown that little change in concentrations occurs over time for certain contaminants, primarily inorganics in ground water. In addition, some contaminants such as the six pesticides in the Interim Regulations have been found only rarely since compliance monitoring requirements went into effect.

To provide for more efficient use of State and local resources, flexibility in monitoring requirements will be a general principle in development of the Revised Regulations. In addition, to assure detection and control of intermittent contaminants or those that are not distributed homogeneously, more specific monitoring requirements will be designed. States also will be provided authority to determine specific monitoring frequencies for systems beyond the federal minima. A three-

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tiered approach is being considered for determining whether and in what manner to regulate specific contaminants. Contaminants would be divided into three categories:

Tier I—Those which occur with sufficient frequency and which are of sufficient concern to warrant national regulation (MCLs) and consistent monitoring and reporting.

Tier II—Those which are of sufficient concern to warrant national regulation (MCLs) but which occur at limited or predictable frequency, justifying flexible national minimum monitoring requirements.

Tier III—Those which would not warrant development of a regulation but for which non-regulatory health guidance could be provided to States or water systems.

Additional descriptions of the categories can be found in the ANPRM (Oct. 5, 1983, 49 FR 45502).

• Public Notice

The SDWA is very specific in the area of public notice (Section 1414), and public notification requirements in the Interim Regulations are relatively inflexible. The specific media are stated in the law and regulations; however these do not appear always to be the most effective means of notice. The SDWA currently is under review for possible amendment in the area of public notification to allow some flexibility.

• Non-Community Water Systems

Non-community water systems (NCWS) serve transient populations as opposed to residential. Only three MCLs in the Interim Regulations apply to NCWS and these protect against acute health effects. Concerns have been raised about reported human exposure to contaminants in certain types of NCWS, such as schools or factories which consistently serve the same consumers. In addition, monitoring frequencies were similar for all sizes of NCWS regardless of the number of consumers served per day.

The Revised Regulations will address these two situations by considering redefinition of NCWS and possible application of other MCLs with potential chronic health concerns. In addition, monitoring requirements will be evaluated according to the size of the NCWS and other relevant factors.

• Monitoring and Determining Compliance to Accurately Detect Contaminants

In certain systems, problems with exceeding MCLs are experienced continually by a portion of the system; e.g., corrosion by-products or portions of a system using a specific well, well field, or surface water source. If results are averaged, the overall system may be technically in compliance and thus no action (including no public notification) would be required. However, those consumers in the particular problem portion of the distribution system are exposed continually to levels above the MCLs. Similarly, corrosion by-products (e.g., lead) are dependent upon the corrosive characteristics of the drinking water and the distribution system materials; monitoring may not detect any violations of MCLs if not sampled at the proper locations. Sampling techniques also can impact on the level of contamination detected.

These are areas of significant concern and the Revised Regulations will determine the most effective means of calculating compliance. Monitoring requirements will be redesigned to assess more effectively the occurrence of corrosion by-products and human exposure in various parts of distribution systems.

Other areas of concern that will be addressed in the Revised Regulations include:

• Numerous synthetic organic chemicals (SOCs) which have been found at increasing frequencies in drinking water across the country. Essentially, two separate groups of SOCs are being detected.

—Pesticides. Different pesticides have been detected in drinking water in various parts of the country. The extent of the problem is highly dependent upon the pesticide application mode and hydrogeological factors. Pesticide contamination of surface waters appears to be closely related to such factors as rates and times of application and rainfall and run-off. Thus, monitoring results can be misleading if sampling is not done at appropriate times and places.

—Organic Chemicals (industrial uses). Concern is focused primarily upon contamination of ground waters from improper disposal of hazardous wastes; numerous SOCs have been detected in ground water as well as in surface waters although introduction of controls to industrial and municipal effluents apparently has improved surface water quality in recent years.

• Inorganic chemicals have been found to be problems primarily in ground waters and are usually a result of natural contamination by geological formations. In addition, contamination for hazardous waste site run-off and leachates remains a concern. All inorganics in the Interim Regulations will be re-evaluated along with a number of other inorganics that have been detected in drinking water. Some of these include arsenic, barium, nitrate, nitrite, copper, sulfate, asbestos, and sodium.

• The microbiological quality of drinking water continues to be a principal concern in development and eventual implementation of the Revised Regulations. Despite compliance with the Interim Regulations by most water systems, waterborne outbreaks of disease are reported with increasing frequency. Much of this may be attributable to better reporting procedures. Major causes of outbreaks are deficiencies in (or lack of) treatment as well as distribution system problems. The specific etiology of the outbreaks is for the most part unknown; *Giardia lamblia* has been identified as a primary agent in many recent outbreaks in all parts of the country. Viruses also have been implicated in a large number of outbreaks. Controls on coliforms and turbidity alone, although highly effective, are not always sufficient to assure the biological safety of drinking water.

D. Public Comments

The ANPRM (October 5, 1983, 48 FR 45502) solicited comments on the technical and regulatory issues that are being examined concerning the development of National Revised Primary Drinking Water Regulations for organic, inorganic, and microbial contaminants in drinking water. See Appendix A for a summary of the public comments received.

IV. Regulatory Alternatives

Either of two philosophies could be followed in determining which substances are appropriate for regulations under the SDWA. The broader view would establish RMCLs and MCLs for as many substances as possible that may be of health concern in drinking water. RMCLs and MCLs would be set for substances on the basis of (1) sufficient health effects information and (2) the occurrence in drinking water or the potential for more widespread occurrence in drinking water.

A more limited view would be to establish RMCLs and MCLs only for those substances which actually occur in public drinking water supplies at or near levels that could result in sufficient public health risk of national scope from drinking water. The limited view would not examine the potential for widespread occurrence in drinking water and thus would not incorporate

an approach for long term anticipatory protection of water supplies; it would essentially wait for widespread contamination to occur before regulatory action would be taken.

The strength of the broad view is that it would provide a single, extensive listing of concentration limits that had been thoroughly and publicly reviewed and that would help to define "potable" drinking water regardless of source. The presence of such a listing may assist response to many situations involving contamination from many infrequently detected substances. Since decisions to act or not to act must be made for every contamination incident, this anticipatory and consistent approach might be the most cost/effective, avoiding case-by-case decisions on the safety of the drinking water. One additional consideration is the intensity of public interest and awareness that some chemical might be in drinking water; the public wants to know if the drinking water is safe; a federal standard provides guidance on this question.

Another situation where regulation may be appropriate under the broad view is for a chemical that has only been detected (in surveys conducted to date) at levels below those associated with potential health risks. The MCL would provide guidance that no action was necessary for these systems with less than that level; without regulations, these types of situations have met widely varying responses by States and public water systems. Regulations can provide a basis for rational and uniform responses to incidents of contamination.

The second, more limited approach to standard setting has the strength of allowing EPA to concentrate its resources on those substances which pose the greatest public health risks from drinking water, reflecting both extent of contamination and the size of populations at risk. Thus, the formal regulatory process would be reserved for the most significant current problems. States or other entities would be forced to address contaminants of more limited scope. This approach would likely lead to a multiplicity of State regulations and the need for a large number of non-regulatory EPA Drinking Water health advisories to fill the gap. Health advisories can be prepared more quickly by EPA because they are limited only to considerations of toxicology (they do not consider technology feasibility and costs) and they would not require rulemaking. On the other hand, health advisories have been adopted by some States as *de facto* standards.

The National Drinking Water Advisory Council (NDWAC) was requested to provide advice on the general philosophical approach to determining which contaminants should be selected for regulation; the Council recommended the following:

> The Council recommends that contaminants be considered for regulation on the basis of sufficient health effects information and the occurrence of contaminants in drinking water and the potential for more widespread occurrence in drinking water.
>
> The minority felt that contaminants should be considered for regulation on the basis of sufficient health effects information and the potential for occurrence in drinking water. It was contended that the aim of the Safe Drinking Water Act is to prevent the occurrence of contaminants in drinking water and waiting to regulate until the contaminant has been detected is not an approach considered to be protective of public health.

The approach followed in this proposal is the broad view that would set RMCLs and MCLs for contaminants of health concern that have been detected in drinking water or have the potential for more widespread occurrence. Public comments are requested on the appropriateness of this approach.

V. Factors in the Development of RMCLs

The SDWA authorizes EPA to establish RMCLs for "each contaminant which, in [the Administrator's] judgment . . . may have any adverse effect on the health of persons". Section 1412(b)(1)(B). A primary drinking water regulation is to be established for each contaminant for which an RMCL is established. Section 1412(b)(2). Presented below are discussions of (1) the factors used to select contaminants for regulation and (2) the methodology and basis for determining what levels are appropriate for the RMCLs.

A. Selection of Contaminants for Regulation

This section provides a discussion of the factors used to select the specific contaminants for which RMCLs are proposed at this time. SOCs, IOCs and microbials that were not included in this proposal will be reconsidered in later iterations of the Revised Regulations as additional data become available. The reader also is referred to the RMCL proposal for VOCs (49 FR 24330) for additional discussion on which factors are considered in selecting contaminants for regulation.

Other than the above directive of the SDWA, little additional guidance was provided upon which to determine how to select contaminants for regulation under the SDWA. Obviously, it is impossible to consider for regulation every chemical that may appear in drinking water and that theoretically may adversely affect health in some remote circumstances. What is needed is some priority of contaminants for regulation adversely affect health in some remote circumstances. What is needed is some priority of contaminants for regulation so that a reasonable number of contaminants of sufficient concern can be addressed in regulations that will advance the goals of the SDWA and provide definitive guidance to address potential human health effects of exposure to hazardous materials in drinking water. To accomplish this, EPA could adopt criteria from which to select contaminants for possible regulation under the SDWA. The most relevant criteria for selection of contaminants are: (1) The analytical ability to detect a contaminant in drinking water, (2) the potential health risk, and (3) the occurrence or potential for occurrence in drinking water.

A set of selection criteria have been developed which essentially expand the three primary factors listed above. Use of a specific formula to apply selection criteria is not believed to be appropriate because of the many variables associated with contaminants in drinking water; however, a decision-making "logic train" has been developed which incorporates the selection criteria and provides a framework from which to make appropriate determinations. Given the variability associated with exposure and human health aspects of drinking water contaminants and the directives of the SDWA, the decision criteria must remain flexible such that a case-by-case decision can be made for each contaminant. However, the decision criteria do set forth an operative framework. For each contaminant, the essential factors in the analysis are as follows:

- Is an analytical method available to detect the contaminant in drinking water? If EPA cannot ascertain whether the contaminant can be found in drinking water, a regulation may not be appropriate.
- Are there sufficient health effects data upon which to make a judgment on an RMCL or health advisory?
- Are there potential adverse health effects from exposure to the contaminant via ingestion?
- Does the contaminant occur in drinking water?

—Has the contaminant been detected in significant frequencies and in a widespread manner?

—If data are limited on the frequency and nature of contamination, is there

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a significant potential of drinking water contamination?

Each of these essential factors is discussed below.

Analytical Methods. Analytical methods must be available such that the presence of the chemicals in water can be validly determined and quantified within acceptable limits. If the level of a contaminant cannot be accurately enough measured to set an RMCL, the contaminant is to be listed. SDWA section 1412(b)(1)(B). This factor is also an important part in determining whether the substance can be regulated (i.e., whether the contaminant can be found in drinking water) and whether an MCL or a treatment technique regulation should be promulgated. The SDWA states that MCLs are appropriate if "it is economically and technologically feasible to ascertain the level of such contaminant in water in public water systems." SDWA section 1401. If not, a treatment technique is to be specified.

A number of factors are taken into consideration in evaluating if analytical methods are available, including such factors as:

- Method validity (reliability).
- Sampling techniques and preparation including volume of sample, preservation, and time of transport.
- Laboratory experience/availability/capabilities.
- Precision and accuracy.
- Detection limits.
- Costs of analysis.

The reliability of analytical methods used for compliance monitoring is critical for determining the MCL. The accuracy (lack of bias) and precision (good reproducibility) of the analytical methods is evaluated in order to determine what are reasonable levels of performance by analytical laboratories at levels considered for the MCLs. This evaluation is carried out in order to ensure that reasonable performance expectations for those laboratories which will be performing the actual analysis is considered, instead of an analysis based upon a single, best laboratory situation which is not representative of real world situations.

Health Effects. Consideration of the potential health effects of a chemical encompasses the (1) suitability of the available data for assessing the toxicity of the chemical and (2) the possibility of human health concern from exposure in drinking water. The human health concerns relate to acute and chronic toxicities, carcinogenic effects including effects in animals or humans, and other toxicological concerns such as whether or not a contaminant is a mutagen or teratogen. Assessment of these potential health effects also considers the International Agency for Research on Cancer (IARC) criteria and the EPA proposed guidelines for carcinogen risk assessment. In the absence of potential carcinogenic risks, most estimated allowable exposure levels will be considerably higher than usually found in drinking water.

Occurrence in Drinking Water. Consideration of occurrence data encompasses the frequency of occurrence, the level of occurrence and the extent of the population exposed. An examination of the available data with regard to how well it represents national occurrence is carried out, along with an evaluation of the quality of the data.

EPA has conducted a number of national sampling surveys to assess occurrence of certain contaminants in drinking water across the country. In addition, a number of States have conducted surveys of public water systems for certain contaminants. These surveys constitute the best sources of available data on occurrence of contaminants in drinking water. However, limitations of this information for certain contaminants are notable:

- Extent of sampling and sampling sites as being representative of national occurrence.

—EPA surveys conducted to date all have been limited to some extent by resource constraints. The surveys have not been statistically comprehensive such that complete knowledge on the extent of potential contamination is known.

—States are more limited in extent of sampling. While a few States have conducted comprehensive surveys for certain contaminants, most States have not. Reliability of these analytical data is usually unknown.

—Surveys usually are conducted one sample at one time which may not assess reliably the variations in surface water quality or variations in ground water quality due to pumping patterns.

—Also, surveys of ground water systems are seldom of every well in a system and some samples are taken in the distribution systems rather than at the well head.

- surveys analyzed for a limited number of contaminants. For both EPA and State surveys, a targeted list of contaminants is the basis for analysis. Many contaminants thus have not been looked for.
- Reliability of resultant data (quality assurance, limits of detection).

Analysis of SOCs and pesticides requires sophisticated techniques using highly sensitive instruments. The methods used have been developed only recently and quality assurance programs only recently have been a priority concern. Thus, existing data from past surveys may be questioned. In general, if a positive result is reported at a certain concentration, it can be assumed that the contaminant was in the sample but the level could be questioned. However, it cannot be said with confidence that if a sample was negative, the contaminant was not there. Of course, detection limits are also a significant limitation.

The extent and quality of the available data varies for each of the contaminants under consideration. Thus, EPA usually must base its decision on appropriate regulatory action (or no action) for certain contaminants on an imperfect data set.

In evaluating occurrence data, the most significant aspects are frequency of occurrence and the widespread (or limited) characteristics of the contamination. Because of the limitations in the survey data (noted previously), levels of contamination that have been detected are not believed to be necessarily representative of the quality of drinking water in all supplies across the country. Many of the contaminants in drinking water are man-made synthetic chemicals whose presence in drinking water indicates that a pollution incident has occurred. The levels detected to date may or may not be representative and levels may change due to pollution incidents. Levels for natural contaminants in ground water are generally more representative and would not be expected to change. Thus, because of the imperfect knowledge on the levels of contamination, frequency of occurrence is a more important factor than levels of occurrence in decisionmaking regarding for which contaminant standards should be set. Therefore, data are evaluated for frequency of occurrence, population exposed and the widespread (or limited) nature of contamination problems. A judgment is made for each contaminant based in part upon these factors.

The ideal data base would provide an estimate of the number of public water systems with contamination problems and population exposure and the widespread or limited nature of contamination problems across the country. Where comprehensive data such as these do exist (and human exposure to the contaminant may pose an adverse effect on the health of persons), this is the primary decision factor in determining if a regulation should be set for a certain contaminant. This decision factor is a judgment that

the contaminant occurs in drinking water with sufficient frequency to warrant national regulations. No guidance on what constitutes a sufficient frequency for regulatory action was provided in the SDWA, but frequencies of less than one percent of systems (i.e., less than 500 systems) could certainly be sufficient. Thus, regulations may be appropriate even where the frequency of occurrence was very low if the contamination problem was found in different parts of the country or in various regions of the country.

Potential for Contamination of Drinking Water. For contaminants that have been detected in drinking water but for which data are limited, an analysis of the potential for widespread drinking water contamination is conducted. Factors considered in this analysis in order of importance are the following:

(1) *Occurrence in Drinking Water Other Than Community Water Supplies.* Certain contaminants have been detected in private wells but not in public water systems, usually because of limited sampling programs. For the most part, this factor deals with pesticides which have been detected during certain studies of pesticide usage and drinking water contamination.

(2) *Direct or Indirect Additives.* Numerous contaminants are in drinking water as a result of direct addition as a water treatment chemical or indirectly through such actions as leaching from pipe coatings or corrosive actions on piping materials. Pesticides registered for use in or around drinking water fall into this category.

(3) *Ambient Surface Water or Ground Water.* Contaminants detected in surface waters or in ground waters through various water quality surveys or in sampling around hazardous waste sites have the potential for contaminating drinking water.

(4) *Present in Liquid or Solid Waste.* Contaminants known to be in industrial or municipal wastewater effluents or in waste ponds or known to be in solid waste being disposed in landfills have the potential to migrate to drinking water intakes.

(5) *Mobile to Surface Water (run-off) or Ground Water (leaching).* The physical/chemical characteristics of contaminants are examined to determine their potential for movement to a drinking water supply. This is essentially an analysis of the fate and transport of contaminants looking toward the potential for contamination of drinking water sources.

(6) *Widespread Dispersive Use Patterns.* This evaluation assesses the characteristics of the use of a contaminant and the locations of that use that would contribute to potential widespread contamination problems in drinking water.

(7) *Production Rates.* An assessment of the amount of contaminant being produced annually to assess if the potential exists for significant contamination.

While the above factors are listed in priority order, the last four factors generally are examined collectively to assess the overall potential for drinking water contamination.

From the list of inorganic, synthetic organic chemicals/pesticides and microbiological contaminants in the October 5, 1983, ANPRM, contaminants have been selected for inclusion in the Revised Regulations. Available data on each of the chemicals on analytical methods, health effects, occurrence and potential occurrence were evaluated using the selection criteria and framework outlined above. Table 1 summarizes the recommended regulatory or non-regulatory action for each contaminant in the ANPRM.

Table 1.—ANPRM IOCs/SOCs/Pesticides Recommended Actions

Inorganic Chemicals

- Include in Revised Regulations:

Arsenic
Asbestos
Barium
Cadmium
Chromium
Copper
Lead
Mercury
Nitrate
Nitrite
Selenium

- Develop Health Advisories:

Molybdenum
Nickel
Cyanide
Sodium
Sulfate
Silver

- Not included in Revised Regulations, nor is EPA developing Health Advisories at the present time. EPA will evaluate later.

Aluminum
Antimony
Beryllium
Thallium
Vanadium

- Does not currently appear to be a need for an RMCL or Health Advisory. Will examine in future if data warrant reconsideration.

Zinc

Synthetic Organic Chemicals/Pesticides

- Include in Revised Regulations:

Acrylamide
Alachlor
Aldicarb
Carbofuran
Chlordane
cis-1,2-Dichloroethylene
Dibromochloropropane
ortho-Dichlorobenzene
1,2-Dichloropropane
2,4-D
Epichlorohydrin
Ethylbenzene
Ethylene dibromide
Heptachlor
Heptachlor epoxide
Lindane
Methoxychlor
Monochlorobenzene
Polychlorinated biphenyls
Pentachlorophenol
Styrene
Toluene
Toxaphene
trans-1,2-Dichloroethylene
2,4,5-TP
Xylenes

- Develop Health Advisories:

Atrazine
meta-Dichlorobenzene
Dioxin (2,3,7,8-TCDD)
Endrin
Hexachlorobenzene

- Not included in Revised Regulations nor is EPA developing Health Advisories at the present time. EPA will evaluate later.

Adipates
Dalapon
Dibromomethane
Dinoseb
Diquat
Endothall
Glyphosate
Hexachlorocyclopentadiene
Polyaromatic hydrocarbons
Phthalates
Picloram
Simazine
1,1,2-Trichloroethane
Vydate

Microbial Contaminants

- Include in Revised Regulations:

Total coliforms
Turbidity
Giardia
Viruses

- Develop Health Advisory.

Legionella

- Monitoring for Heterotrophic Plate Count will be included within compliance monitoring requirements for total coliforms.

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B. Sources of Occurrence and Human Exposure Data

Data on the occurrence of synthetic organic chemicals in air, food and drinking water have been assembled from numerous published reports and supplemented by federal research studies and results from federal and state regulatory activity. The resulting summaries provide a view of potential human exposure indicating the relative exposure of each source of intake. These data are then factored into the determination of the RMCL, as the RMCL must consider intake from all sources, not just from drinking water. Food data, when available, come largely from compliance studies conducted by the U.S. Food and Drug Administration. The market basket studies and compliance program reports provide data on residue levels of selected chemicals in grain and vegetable crops, fish tissue, food animal tissue, spices and other products. FDA total dietary studies for adults, infants and toddlers were used when available. The majority of these data were developed during the 1970's. Data developed by USDA on residue levels in meats were used to supplement existing knowledge as were data from specific studies covering limited areas of the United States.

Data on the level of pesticides in air were developed by the EPA Office of Pesticide Programs and predecessor organizations. Air samples were collected in 1970–72 at sites in 16 U.S. cities and examined for pesticide content. Data on the level of non-pesticide organics in ambient air were extracted from a report for the EPA Office of Air and Radiation (Brodzinsky, R. and Singh, H.B. 1982. Volatile Organic Chemicals in the Atmosphere; An Assessment of Available Data. Prepared by SRI International, Menlo Park, California. Office of Research and Development, U.S. EPA Research Triangle Park, North Carolina. Contract No. 68-02-3452). Data from individual studies were summarized to provide insight to ambient air levels in urban and rural locations. Data on the level of inorganic chemicals in air were extracted from computerized information developed by the EPA Environmental Monitoring and Support Laboratories.

Drinking water data come largely from either (1) National Interim Primary Drinking Water Regulations (NIPDWR) compliance monitoring data, accessed through the Federal Reporting Data System (FRDS), or (2) special studies conducted by EPA's Office of Drinking Water (ODW). Six national surveys have been conducted by ODW since 1975. These include:

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

NORS was conducted in 1975 to determine the level of six SOCs in 80 cities across the country. These water supplies served 36 million individuals.

NOMS, conducted in 1976–1977, extended EPA's knowledge on the occurrence of volatile organic compounds in drinking water. One hundred and thirteen cities using surface water were included in this study.

NSP, conducted between June 1977 and March 1981, provided a broadened examination of SOCs in drinking water. The compounds sampled included 23 hydrocarbons, 6 aromatics, 22 pesticides, phenols and acids. One hundred and sixty-six water supplies, mostly using surface water, located in 33 States participated in the study.

Two different CWSS studies have been conducted. The 1969 CWSS provided information on the level of inorganics in drinking water. Over 950 cities throughout the United States participated in the study. A second CWSS was conducted in 1978 providing information on both inorganic and volatile organic contaminants.

The RWS was conducted in 1978 to examine the quality of rural water supplies. The level of both inorganic and volatile organic contaminants was determined for over 800 samples.

The GWSS, focusing on ground water supplies was conducted in 1980–1981. This study provided information on the occurrence of 34 SOCs in nearly 1,000 water supplies.

In addition, the National Inorganics and Radionuclides survey is currently underway and it is anticipated that data from this survey will be available for evaluation before promulgation of the RMCLs proposed herein.

Data on the occurrence of pesticides in drinking water comes from numerous special studies conducted by the EPA Office of Pesticides, U.S. Geological Survey and selected state agencies. Published information on the occurrence of pesticides is integrated into these summaries. Other sources of information include various State surveys and results of monitoring around hazardous waste sites by the Superfund program.

C. Evaluation of Health Effects and Determination of RMCLs

For those contaminants that "may have any adverse effect on the health of persons" (for which analytical methods are available), RMCLs are to be set at a level which:

> no known or anticipated adverse effects on the health of persons occur and which allows an adequate margin of safety. Section 1412(b)(1)(i)(B).

Summarized below are the approaches used to determine the RMCLs for non-carcinogens and carcinogens. Additional discussion is provided in the Phase I (Volatile Organic Chemicals) RMCL proposal (See 48 FR 24330).

1. Non-Carcinogens—AADIs

For toxic agents not considered to have carcinogenic potential, "no effect" levels for chronic/lifetime periods of exposure including a margin of safety are referred to commonly as ADIs or Acceptable Daily Intakes. These ADIs are considered to be exposure levels estimated to be without significant risk to humans when received daily over a lifetime.

The intent of a toxicological analysis is to identify the highest no-observed-adverse-effect-level (NOAEL) based upon assessment of available human or animal data (usually from animal experiments). To determine the ADI for regulatory purposes, the NOAEL is divided by (an) appropriate "uncertainty" or "safety" factor(s). This process accommodates for the extrapolation of animal data to the human, for the existence of weak or insufficient data and for individual differences in human sensitivity to toxic agents, among other factors.

ADIs traditionally are reported in mg/kg/day but for RMCL purposes, the "no effect" level needs to be measurable in terms of drinking water quality, i.e., mg/liter. An adjustment of the ADI to mg/liter is accomplished by factoring in an assumed weight of the consumer and the assumed amount of drinking water consumed per day. The "no effect level" in mg/l has been termed the Adjusted ADI (AADI). AADI's are calculated by:

- Determining the highest No-Observed-Adverse Effect Level (NOAEL), or the Lowest-Observed-Adverse Effect Level (LOAEL) in mg/kg body weight/day;
- Dividing by an appropriate safety or uncertainty factor (U.F.);
- Multiplying by the assumed weight of an adult (70 kg); and
- Dividing by the assumed amount of water consumed by an adult per day (2 liters/day).

The formula for this calculation is as follows:

$$AADI = \frac{(NOAEL \text{ in mg/kg/day})(70 \text{ kg})}{(U.F.)(2\ 1/\text{day})}$$

In the calculation of the AADIs, the NOAELs are determined based upon data from ingestion studies. If ingestion data are not available, inhalation studies are used and conversion factors applied to convert the exposure. In some cases, however, inhalation will not be used if the data are not determined to be adequate.

The AADIs are calculated based upon the 70 kg adult except for those chemicals where the child has been shown to be the more sensitive subpopulation. In these cases, the AADI is based upon the 10 kg child. Comment is requested on this approach.

In this proposal, AADIs determined from less than lifetime studies are termed provisional AADIs. The AADIs which are based upon lifetime studies will not have the prefix "provisional".

In the proposal, AADIs have been calculated for some chemicals with evidence of carcinogenicity as well as for non-carcinogens. The purpose for calculating AADIs for chemicals with evidence of carcinogenicity is to demonstrate that non-carcinogenic endpoints, as well as carcinogenic endpoints, may occur from exposure to these compounds and these endpoints may be used to identify chronic toxicity levels. However, the derivation of AADIs does not imply that these levels are acceptable for lifetime consumption of the carcinogens. These levels have been derived for the purpose of presenting a broader perspective on the overall toxicity of the chemical.

The uncertainty factor(s) applied in the derivation of the AADI is used in order to estimate the comparable "no effect" level for a large heterogenous human population. In such a population, there may be individuals particularly sensitive to the toxicant and the possibility must be considered that humans are some sensitive to the toxic effects of the chemicals than are animals. The use of uncertainty factor(s) accounts for intra- and inter-species variability, the small number of animals tested compared to the size of the exposed population, sensitive subpopulations and the possibility of synergistic action between chemicals. Uncertainty factors allow for the extrapolation of data to the human population with an added margin of safety to account for the factors discussed above.

The determination of ADIs in the United States began with the examination of food additives. The application of an uncertainty factor of 100 was proposed (Lehman and Fitzhugh, 1954. One Hundred-fold Margin of Safety. Assoc. Food Drug Off. 2 Bull. 18:33–35) as a means of accounting for intra-or inter-species variability to the toxicity of the chemical, sensitive subpopulations within the human population and possible synergism between the intentional and unintentional contaminants in the human diet.

This initial use of the 100-fold uncertainty factor was expanded further in order to better account for the available information. For example, the FDA recommended the use of an uncertainty factor of 1000 instead of 100 in situations where chronic data were unavailable but subchronic data were available in two species. The additional 10-fold uncertainty factor was due to the added uncertainty when estimating an ADI from shorter term toxicity data. The use of a 2000-fold uncertainty factor was recommended by the FDA if subchronic data were available for only one species.

The National Academy of Sciences (*Drinking Water and Health*, 1977. Vol. I) also expanded upon the concept of uncertainty factors by recommending a similar approach when estimating ADIs for contaminants in drinking water. The NAS guidelines are as follows:

- An uncertainty factor of 10 used when good acute or chronic human exposure data are available and supported by acute or chronic data in other species.
- An uncertainty factor of 100 used when good acute or chronic data are available for one species, but human data are not.
- An uncertainty factor of 1000 used when acute or chronic data in all species are limited or incomplete.

The EPA (1980. Guidelines and Methodology used in the Preparation of Health Effects Assessment Chapters of the Consent Decree Water Quality Criteria. 45 FR 79347) recommended uncertainty factors for estimating ADIs of chemicals in ambient waters. The same guidelines outlined above for the NAS were applied, with the addition of the application of an uncertainty factor of between 1 and 10 when an ADI was estimated from a LOAEL (if a NOAEL was unavailable) in order to adjust the LOAEL into the range of a NOAEL.

The guidelines outlined by the NAS (1977) and further modified by the EPA (1980) for the derivation of the water quality criteria (see Table 2) are used in determining the appropriate uncertainty factor to be applied in the derivation of the AADIs. These guidelines were developed specifically for water contaminants and have had widespread use and are generally accepted in the scientific community. These uncertainty factors have been modified by scientific judgment in instances where more information is known about a specific chemical. As concluded by a member of EPA's Science Advisory Board,

> Safety factors do not pretend to have mathematical precision. When you have very little information, order-of-magnitude safety factors may be about the best you can do in assessing the risk from low levels of exposure. The use of an elaborate mathematical model in such situations may convey a misleading impression of precision. On the other hand, in situations where you do have more information, where there is, for example, a known mechanism for bioaccumulation or enzyme measurements showing how much of the enzyme is being metabolized, then I think it is vital that such information be taken into account in the assessment process. In such situations it would be wrong to ignore the data and to use a simple procedure that mechanically selects a certain power of ten as the appropriate safety factor.

For several of the inorganic chemicals uncertainty factors have been applied in the derivation of AADIs which differ from the traditional 10, 100 or 1,000 factors. These intermediate uncertainty factors have been applied according to the guidelines (Table 2), when the scientific judgment is that the data appear to fall between categories. These intermediate uncertainty factors may be classified according to the available data and the characteristics of the chemicals. The following is a summary of the data involved which dictated the application of an intermediate uncertainty factor.

- The AADI was based upon a study in which the animals were not exposed for a full lifetime.
- The intermediate uncertainty factor was applied to account for the additional uncertainty involving a less than-lifetime study.
- The chemical is an essential nutrient at low levels and toxic at higher levels.

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The application of the traditional uncertainty factor results in a concentration of the chemical being less than that considered necessary for good health. An intermediate uncertainty factor was applied which did not result in the allowable exposure level being below the nutritionally essential level.

• The AADI was based upon a study in which the inorganic chemical was given via food rather than drinking water.

Data suggest that certain chemicals ingested via food may be absorbed less efficiently than via water. Thus, an intermediate uncertainty factor was applied to account for the potential of higher absorption via drinking water.

• The endpoints used to derive the AADI are much more sensitive than the traditional endpoints used in AADI calculations.

An intermediate uncertainty factor was applied which, according to scientific judgment, would provide an adequate margin of safety for the particular chemical.

TABLE 2.—GUIDELINES ON THE USE OF UNCERTAINTY FACTORS

Uncertainty factor	Guideline
10	Uncertainty factor of 10 used with valid experimental results on appropriate durations of exposure in humans.
100	Uncertainty factor of 100 used when human data are not available and extrapolating from valid results of long-term studies in animals.
1,000	Uncertainty factor of 1,000 used when human data are not available and extrapolating from studies in animals of less than chronic exposure.
1-10	Additional uncertainty factor between 1 and 10 when using a LOAEL instead of a NOAEL.
Intermediate uncertainty factor.	Other uncertainty factor used, according to scientific judgment, when justified.

An intermediate uncertainty factor was applied, since the traditional 10-fold uncertainty factor is applied in order to account for sensitive subpopulations within the human population.

Another issue is the rate of absorption of chemicals through the gastrointestinal tract. Numerous factors affect the absorption of a chemical, including the animal used, the presence of other chemicals and bacteria, whether the chemical was administered via food or water and the previous dietary intake. However, the absorption rates of chemicals from the gastrointestinal tract generally have been shown to be comparable in laboratory animals and humans.

The absorption rate assumed for each chemical in the proposal has been determined on a case-by-case basis. The available data, including animal and human studies, were examined for each chemical and a summary of the data is presented in the background Health Effects Criteria Documents. The absorption rate assumed in the AADI calculations was determined using a best estimate based upon the available data.

2. Calculation of the RMCLs

To determine the RMCL, the contribution from other sources of exposure, including air and food should be taken into account. When sufficient data are available on the relative contribution of other sources, the RMCL is determined as follows:
RMCL = (AADI)—(contribution from food)—(contribution from air). This calculation assures that the total exposure from drinking water, food and air does not exceed the ADI.

However comprehensive data are usually not available on exposures from air and food. In these cases the RMCL is determined as follows: RMCL = (AADI) (Percentage Drinking Water Contribution).

The percentage drinking water contribution often used in this proposal is a 20 percent contribution for organic chemicals. For inorganic chemicals, the actual contribution from other sources was often available and this data was factored in the RMCL. The NIPDWR used 20 percent as the drinking water exposure factor for pesticides. This exposure factor is judgmental and is adjusted when mitigating information exists. A wide range of environmental exposure distributions occurs across urban and rural populations and differences exist due to age and occupation. Use of a 20 percent contribution is considered to be reasonably conservative and protective. The World Health Organization (WHO), in "Guidelines for Drinking Water Quality" (1984), assigned as little as 1 percent of the ADI to drinking water where the chemical was known to bioaccumulate to a high degree, while greater proportions were assigned where the chemical bioaccumulated to a lesser degree. In "Drinking Water and Health" (1977), the National Academy of Sciences provided projections of 1 percent and 20 percent as illustrations of drinking water contributions.

3. Short-term Assessments

In addition to the RMCLs, short-term risk assessments (health advisories) have been developed for inorganic and organic contaminants for non-carcinogenic endpoints of toxicity. These evaluations are considered to be exposure levels which would not result in adverse health effects over a roughly specified short-time period (usually one-day, ten-days and longer-term (several months to several years)). If data are available for all exposure durations, all three numbers are derived. For certain chemicals, there are inadequate data to derive a specific exposure level. For the chemicals where there are inadequate data to derive a ten-day number, a ten-day number is derived by dividing the one-day number by 10. However, if this number is inconsistent with other levels determined by using data of higher quality, the divided number is not used. If there are inadequate data to derive a longer-term number, a divided number is not used, and a longer-term number is not derived. The longer-term number must be derived from a subchronic study rather than an acute study; only acute and short-term studies are used to calculate the one-day and ten-day numbers.

The toxicological assessment methods used to develop the assessments are the same as outlined for the RMCLs, i.e., the identification of a NOAEL based upon human or animal data and dividing the NOAEL by an appropriate uncertainty factor(s). For RMCLs, a 70 kg adult consuming 2 liters of water per day is used in the calculations to adjust the ADI for drinking water purposes. For these short-term assessments, the 70 kg adult consuming 2 liters of water per day and the 10 kg child consuming 1 liter of water per day are both used to calculate the numbers in terms of the protected population. Both the adult and the child are used in order to provide flexibility for those officials applying the number to use the value that is felt to best fit the needs of the specific situation. Both values are calculated to present a broader perspective than would be available with one number.

The assessments are developed as guidance values for short-term exposure situations, such as spills or accidents, and usually are issued separately by EPA as Health Advisories. They are not assessments which are used to develop RMCLs or MCLs. The purpose of providing the assessments in this proposal is to elicit comment on these scientific assessments which will be then converted into formal Health Advisories. The following is a list of Health Advisories which are available from the National Technical Information Service at the address listed at the beginning of the proposal.

Health Advisories

Acrylamide
Alachlor
Aldicarb
Arsenic

Barium
Benzene
Cadmium
Carbofuran
Carbon tetrachloride
Chlordane
Chlorobenzene (Monochlorobenzene)
Chromium
Cyanide
2,4-D
DBCP
Dichlorobenzenes (o,m,p)
1,2-Dichloroethane
1,1-Dichloroethylene
cis-1,2-Dichloroethylene
trans-1,2-Dichloroethylene
Dichloromethane
Dichloropropane
p-Dioxane
EDB (Ethylene Dibromide)
Endrin
Epichlorohydrin
Ethylbenzene
Ethylene glycol
Heptachlor/Heptachlor epoxide
Hexachlorobenzene
n-Hexane
Lead
Legionella
Lindane
Mercury
Methoxychlor
Methyl ethyl ketone
Nickel
Nitrate/Nitrite
Oxamyl
Polychlorinated biphenyls
Pentachlorophenol
Styrene
TCDD (Dioxin)
Tetrachloroethylene
Toluene
Toxaphene
2,4,5-TP
1,1,1-Trichloroethane
Trichloroethylene
Vinyl chloride
Xylenes

4. Carcinogens

Evaluations of the toxicology of substances which may possess carcinogenic potential is a two-phase process. In the first phase, the toxicological data base for non-carcinogenic endpoints of toxicity was evaluated in the same manner as described above for "non-carcinogens" and AADIs are determined. In the second phase, assessment made of the evidence of the carcinogenic potential (e.g., long-term bioassays in rodents and human epidemiology) as well as information which provides indirect evidence (e.g., mutagenicity and other short-term test results). The objectives of this assessment are (1) to determine the level or strength of evidence that the substance is an animal or human carcinogen, and (2) to provide an upper bound estimate of the possible risks of human exposure to the substances in drinking water.

An issue that is considered in assessing carcinogenicity is inhalation vs. ingestion data. For all chemicals, the total data base is examined, considering data on both ingestion and inhalation exposure. If the data show the chemical to be carcinogenic through ingestion exposure, then the chemical will be considered a potential carcinogen and evaluated based upon the carcinogenicity data.

A second situation consists of instances where the chemical has been shown to be carcinogenic by inhalation exposure but not by ingestion exposure (e.g., negative data exists on exposure by the ingestion route and there is an adequate basis to toxicologically distinguish between the routes of exposure). In these instances the chemical will not be considered a potential carcinogen via drinking water and the RMCL will be based upon non-carcinogenic effects. A third case consists of chemicals which have been demonstrated to have carcinogenic effects via inhalation exposure and the data are either not available or equivocal via ingestion exposure. In these situations, the RMCL will be determined on a case-by-case basis by examining the applicability of the inhalation data to drinking water exposure.

5. Evidence of Carcinogenicity

Several groups of scientists have attempted to classify chemicals on the basis of available evidence for carcinogenicity. These include the IARC, the NAS Safe Drinking Water Committee and EPA via its recently proposed risk assessment guidelines for carcinogenicity (49 FR 46294).

The IARC is responsible for a program on the Evaluation of Carcinogenic Risk of Chemicals to Humans, which involves the preparation and publication of monographs providing a qualitative assessment of the carcinogenic potential of individual chemicals and complex mixtures. The assessments are made by independent, international working groups of experts in cancer research. The program has existed since 1971 and has evaluated over 585 chemicals to date.

Criteria used for evaluating carcinogenic risk to humans were first established in 1971 and were used by the IARC for the preparation of the first 16 volumes of the monographs. These criteria consisted of the terms "sufficient evidence" and limited evidence" of carcinogenicity, referring to the amount of evidence available and not to the potency of the carcinogenic effect.

The later monographs and IARC Supplement 4, used revised criteria to evaluate or reevaluate the chemicals carcinogenic risk to humans. An overall evaluation of carcinogenicity for humans was made on the basis of the combined evidence from humans and experimental systems. The degrees of evidence for carcinogenicity were characterized as follows:

1. Assessment of evidence for carcinogenicity from studies in humans:

(a) Sufficient evidence, which indicates that there is a causal relationship between the agent and human cancer.

(b) Limited evidence, which indicates that a causal interpretation is credible, but that alternative explanation such as chance, bias or confounding, could not be adequately excluded.

(c) Inadequate evidence which indicates that one of three conditions prevailed: (1) there were few pertinent data (2) the available studies, while showing evidence of association, did not exclude chance, bias or confounding (3) studies were available which did not show evidence of carcinogenicity.

2. Assessment of evidence for carcinogenicity from studies in experimental animals:

(a) Sufficient evidence, which indicates that there is an increased incidence of malignant tumors (1) in multiple species or strains or (2) in multiple experiments (preferably with different routes of administration or using different dose levels or (3) to an unusual degree with regard to incidence, site or type of tumor, or age at onset.

(b) Limited evidence, which means that the data suggest a carcinogenic effect but are limited because (1) the studies involve a single species, strain or experiment or (2) the experiments are restricted by inadequate dosage levels, inadequate duration of exposure to the agent, inadequate period of follow-up, poor survival, too few animals, or inadequate reporting or (3) the neoplasms produced often occur spontaneously and, in the past, have been difficult to classify as malignant by histological criteria alone (e.g., lung and liver tumors in mice).

(c) Inadequate evidence, which indicates that because of major qualitative or quantitative limitations, the studies cannot be interpreted as showing either the presence or absence of a carcinogenic effect; or that within the limits of the tests used, the chemical is not carcinogenic.

(d) No data, which indicates that data were not available to the Working Group.

The categories sufficient evidence and limited evidence refer only to the strength of the experimental evidence that these

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chemicals are carcinogenic and not to the extent of their carcinogenic activity nor to the mechanism involved.

3. Assessment of data from short-term tests:

(a) Sufficient evidence, when there were at least three positive results in at least two of three test systems measuring DNA damage, mutagenicity or chromosomal effects. When two of the positive results were for the same genetic effects, they had to be derived from systems of different biological complexity.

(b) Limited evidence, when there were at least two positive results, either for different endpoints or in systems representing two levels of biological complexity.

(c) Inadequate evidence, when there were generally negative or only one positive test result. Up to two positive test results were considered inadequate if they were accompanied by two or more negative test results.

The IARC then placed the chemicals in one of three groups to reflect carcinogenic risk to humans. These groups are quite broad and are not defined by strict rules concerning the assessment of evidence from the criteria outlined above. The IARC placed the chemicals in the categories based on evaluation of the criteria along with scientific judgment on other properties of the compound which would affect its potential carcinogenic risk to humans. These groups are defined as follows:

Group 1—The chemical, group of chemicals, industrial process or occupational exposure is carcinogenic to humans. This category was used only when there was sufficient evidence from epidemiological studies to support a causal association between the exposure and cancer.

Group 2—The chemical, group of chemicals, industrial process or occupational exposure is probably carcinogenic to humans. This category includes exposures for which, at one extreme, the evidence for human carcinogenicity is almost "sufficient" as well as exposures for which, at the other extreme, it is inadequate. To reflect this range, the category was divided into higher (Group A) and lower (Group B) degrees of evidence. Usually, category 2A was reserved for exposures for which there was at least limited evidence for carcinogenicity to humans. The data from studies in experimental animals played an important role in assigning studies to category 2, and particularly those in Group B, thus, the combination of sufficient evidence in animals and inadequate data in humans usually resulted in a classification of 2B.

In some cases, the Working Group considered that the known chemical properties of a compound and the results from short term tests allowed its transfer from Group 3 to 2B or from Group 2B to 2A.

Group 3—The chemical, group of chemicals, industrial process or occupational exposure cannot be classified as to its carcinogencity to humans.

The EPA has proposed guidelines for carcinogen risk assessment (49 FR 46294) which contain a classification system for chemicals using the degree of evidence of carcinogenicity. The categorization scheme places chemicals into five groups:

Group A: Human carcinogen (sufficient evidence from epidemiological studies)

Group B: Probable human carcinogen
- Group B1: At least limited evidence of carcinogenicity to humans
- Group B2: Usually a combination of sufficient evidence in animals and inadequate data in humans

Group C: Possible human carcinogen (limited evidence of carcinogenicity in animals in the absence of human data)

Group D: Not classified (inadequate animal evidence of carcinogenicity)

Group E: No evidence of carcinogenicity for humans (no evidence of carcinogenicity in at least two adequate animal tests in different species or in both epidemiological and animal studies)

The NAS (*Drinking Water and Health*, 1977, Vol. I) classified chemicals in four categories based upon the strength of the experimental evidence. These categories are: human carcinogens, suspected human carcinogens, animal carcinogens and suspected animal carcinogens.

In the Phase I RMCL proposal, EPA considered three main options for setting RMCLs for carcinogens. These options were: (1) Set the RMCLs at zero, (2) set the RMCLs at the analytical detection limit, and (3) set the RMCLs at a non-zero level based upon a claculated negligible contribution to lifetime risk. In addition, EPA requested comment on the strength of evidence of carcinogenicity and how this could be factored into the RMCL determinations.

RMCLs could be proposed at zero, based upon the inability of scientists to demonstrate experimentally a threshold of effects for "carcinogens". This leads to the assumption that since no threshold dose can be demonstrated for carcinogens, any exposure theoretically would represent some finite level of risk for carcinogens. Depending upon the potency of the specific carcinogen, such a risk could be vanishingly small at very low doses. The House Report which accompanied the Safe Drinking Water Act of 1974, suggested that RMCLs for non-threshold toxicants (i.e., carcinogens) should be zero.

RMCLs could be set at the analytical detection limit. Since RMCLs at zero theoretically are unattainable, basing the levels upon defined state-of-the-art analytical detection limits would provide measurable goals for carcinogens in drinking water. The analytical detection limit is, for all practical purposes, the functional equivalent of zero and thus would present the same philosophy as a zero RMCL.

RMCLs could be set based upon a calculated negligible contribution to lifetime risk using mathematical models which would estimate the number of excess cancer cases occurring in a population as the result of a chemical of specified concentration being present in the drinking water. EPA would select an excess cancer risk level low enough to be considered a "virtually safe" level. Such a level is not really very different from zero and could be argued to fit the requirement that the RMCL be set at the no-effect level with an adequate margin of safety.

EPA has evaluated these three approaches and based upon our analysis and the public comments received on the Phase I proposal, a three-category approach based upon strength of evidence of carcinogenicity will be used to set the RMCLs. This approach is summarized in Table 3. Category I includes those chemicals which have sufficient human or animal evidence of carcinogenicity to warrant their regulation as probable human carcinogens. The RMCLs for Category I chemicals will be proposed at zero. Category II includes those substances for which some limited inconclusive evidence of carcinogenicity exists from animal data. These will not be regulated as human carcinogens. However, RMCLs will reflect the fact that some possible evidence of carcinogenicity in animals exists. Thus, they will be treated more conservatively than Category III substances. Category III includes substances with inadequate or no evidence of carcinogenicity. RMCLs will be calculated based upon AADIs.

Table 3

Three-Category Approach for Setting RMCLs

Category I—Strong evidence of carcinogenicity.
- EPA Group A or Group B
- IARC Group 1, 2A or 2B

Category II—Equivocal evidence of carcinogenicity.
- EPA Group C
- IARC Group 3

Category III—Inadequate or no evidence of carcinogenicity in animals.
- EPA Group D or E
- IARC Group 3

The method for determining the RMCLs for Category II chemicals is more complex than for the other categories. To be placed in Category II, chemicals are not considered to be probable carcinogens via ingestion although some data is available that causes concern. Thus, these substances should be treated more conservatively than Category III "non-carcinogens," yet less conservatively than Category I chemicals. Two options are available for setting the RMCLs for Category II chemicals; the first option involves basing the RMCL upon the AADI which is based upon non-carcinogenic endpoints of toxicity using an uncertainty factor according to the adequacy of the data and toxicological principles. To account for the possible evidence of carcinogenicity, an additional factor would be applied (e.g., AADI divided by a factor of 10 or some other value). A value of 10 is commonly applied for the contaminants proposed for regulation today. Traditionally, for every additional factor contributing to uncertainty, a factor of 10 has been included. Equivocal evidence of carcinogenicity is such an additional uncertainty factor. However, a factor other than 10 will be applied if the properties of the chemical dictate an alternate factor. The second option involves basing the RMCLs upon a lifetime risk calculation in the range of 10^{-5} to 10^{-6} using a conservative method. This risk range is commonly considered to be protective and in the future, if additional data led to reconsideration of a chemical's carcinogenicity, the RMCL would still be set at a level that would represent an extremely low nominal risk. EPA will use both approaches to set the RMCLs for Category II chemicals. The first option, basing the RMCL upon the AADI, will be used if sufficient valid chronic toxicity data are available. If sufficient data are not available, the RMCL will be based upon a risk calculation.

The following is a summary of the classification of the SOCs and IOCs based upon the classification system outlined in EPA's proposed guidelines for carcinogen risk assessment.

IOCs

The classification of the inorganic chemicals for carcinogenicity by the weight of evidence approach in the proposed EPA carcinogen risk assessment guidelines takes into account the total evidence regardless of exposure route (includes inhalation and ingestion exposure). By this approach, asbestos, arsenic and chromium are in Group A (sufficient evidence in humans), nickel and cadmium are in Group B1 (limited evidence in humans), lead is in Group B2 (sufficient evidence in animals), and barium, nitrate/nitrite, sodium, cyanide, copper, mercury, selenium, silver, molybdenum, and sulfates are in Group D (inadequate evidence).

The carcinogenicity data for the inorganic chemicals indicate that the carcinogenic potential from ingestion should be distinguished from that by other routes. Arsenic, in humans, has sufficient evidence for carcinogenicity by inhalation and by the oral route, however other factors such as the potential that it is an essential element were also considered in developing the proposed RMCL. Although lead is technically listed in Group B2, the test dosages that induced cancer in animals were beyond the lethal dose in humans and thus the RMCL for lead will be based upon other sensitive endpoints. Asbestos is a proven carcinogen by inhalation, but the evidence with oral exposure is limited even after extensive studies have been performed. The evidence for the carcinogenicity of chromium, cadmium, and nickel is inadequate by the oral route but sufficient by the inhalation route.

In this proposal, the RMCLs for those chemicals in Group D are set based upon chronic toxicity data (Regulatory Group III). In addition, the RMCLs for chromium, cadmium, arsenic and lead are also being set based upon chronic toxicity data, for the reasons outlined above. The RMCL for asbestos is set based upon an excess risk level (Regulatory Category II) due to the weak oral carcinogenicity data. An RMCL is not proposed for nickel due to problems with the limited toxicological data base, which is now being expanded by new studies.

SOCs

DBCP, dioxin, epichlorohydrin, hexachlorobenzene, alachlor, toxaphene, acrylamide, EDB, chlordane, heptachlor, heptachlor epoxide and PCBs have been classified in EPA's proposed Group B2; sufficient evidence of carcinogenicity in animals.

1,2-Dichloropropane, styrene, monochlorobenzene, and lindane have been classified in EPA's proposed Group C; limited evidence of carcinogenicity in animals.

Pentachlorophenol, cis-1,2-dichloroethylene, trans-1,2-dichloroethylene, o-dichlorobenzene, m-dichlorobenzene, 2,4-D, 2,4,5-TP, ethylbenzene, methoxychlor, toluene, xylene, atrazine, and simazine have been classified in EPA's proposed Group D; inadequate animal evidence of carcinogenicity.

Endrin and carbofuran have been classified in EPA's proposed Group E; no evidence of carcinogenicity for humans.

Table 4 presents a classification of the SOCs and inorganics into the three category approach for setting RMCLs. As noted in the table, there are six chemicals which are classified in Category II (limited evidence of carcinogenicity): styrene, 1,2-dichloropropane, monochlorobenzene, lindane, asbestos and arsenic. Table 4A presents the rationale for the placement of each chemical in its respective category. Table 5 presents the RMCL options for the Category II contaminants. See Section VIII for a further discussion of the RMCLs for the individual chemicals. Comment is requested on the three-category approach for setting RMCLs and on the proposed classifications of chemicals in each category.

TABLE 4.—PRELIMINARY CLASSIFICATION OF SOCS AND INORGANICS INTO THREE CATEGORY APPROACH

Chemical	Category		
	I [1]	II [2]	III [3]
DBCP	X		
Dioxin	X		
Epichlorohydrin	X		
Hexachlorobenzene	X		
Alachlor [4]	X		
Toxaphene	X		
Acrylamide	X		
EDB	X		
Chlordane [4]	X		
Heptachlor [4]	X		
PCBs	X		
Heptachlor epoxide	X		
Lindane		X	
Styrene		X	
1,2-Dichloropropane		X	
Monochlorobenzene		X	
Pentachlorophenol			X
Aldicarb			X
cis-1,2-Dichloroethylene			X
trans-1,2-Dichloroethylene			X
o-Dichlorobenzene			X
m-Dichlorobenzene			X
2,4-D			X
Endrin			X
Ethylbenzene			X
Methoxychlor			X
Toluene			X
2,4,5-TP			X
Xylene			X
Atrazine			X
Simazine			X
Carbofuran			X
All the inorganics except arsenic and asbestos			X
Asbestos (ingestion only, fiber's > 10 μm)		X	
Arsenic		X	

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[1] Category I—Strong evidence of carcinogenicity; zero RMCL.

[2] Category II—Equivocal evidence of carcinogenicity; RMCL based on cancer risk or the AADI with additional factor.

[3] Category III—Inadequate or no evidence of carcinogenicity, RMCL based on AADI.

[4] Classified by IARC in Group 3. However, re-examination has resulted in the classification in EPA's Group B2 and thus these chemicals have been placed in the "probable" carcinogen category.

TABLE 4A.—CLASSIFICATION OF SOCS AND IOCS BASED ON PROPOSED EPA GUIDELINES

Chemical	EPA classification	Basis
A. SOCs		
DBCP	B2	Carcinogenic in rats/mice. Carcinomas of forestomach in rats and mice of both sexes, positive in short tests.
2,3,7,8-TCDD	B2	Carcinogenic in rats/mice. Cancer in liver, thyroid, tongue, etc. Inadequate evidence in humans.
Epichlorohydrin	B2	Carcinogenic in rats. Cancer in forestomach (oral), subcutaneous injection sites, and nasal turbinates (inhalation). Mutagen. No human evidence.
Hexachlorobenzene	B2	Carcinogenic in rats/hamsters/mice. Liver cancer. No human evidence.
Alachlor	B2	Carcinogenic in rats/mice. Both sexes and dose responsive.
Toxaphene	B2	Do.
Acrylamide	B2	Carcinogenic in two species, tumors at multiple sites.
EDB	B2	Carcinogenic in rats/mice by gavage, inhalation. Dermal in mice only.
Chlordane	B2	Carcinogenic in both sexes of mice. Liver hepatocellular carcinoma.
Heptachlor	B2	Carcinogenic in mice. Liver hepatocellular carcinoma.
Heptachlor Epoxide	B2	Carcinogenic in mice and rats. Hepatocellular carcinoma.
PCB	B2	Certain PCB's carcinogenic in mice and rats (oral). Produces benign and malignant neoplasms.
Lindane	C	Marginal tumors of the liver of both sexes in mice. Carcinogenic metabolite.
1,2 dichloropropane	C	Limited evidence of carcinogenicity in mice. Evidence in rats equivocal (based on NTP draft report).
Styrene	C	Carcinogenic in rats. Alveolar/bronchiolar adenomas and carcinomas in both sexes of rats (oral). However, these studies are not conclusive.
Monochlorobenzene	C	Increased occurrence of neoplastic nodules of the liver in high dose male rats. (Based on NTP draft report).
Pentachlorophenol	D	Negative in studies in rats and mice, mouse data not really solid. Negative for mutagenicity. No human evidence.
Cis and Trans-1,2-dichloroethylene.	D	Not tested.
o-Dichlorobenzene	D	Negative results in both rats and mice NTP (draft report) gavage studies. Some caution should be used since high dose may have been below MTD.
m-Dichlorobenzene	D	Not tested.
2,4-D	D	Inadequate animal data to classify for carcinogenicity.
2,4,5-TP	D	Do.
Ethylbenzene	D	Not tested.
Methoxychlor	D	Inadequate animal evidence. Inconclusive results.
Toluene	D	Negative in one CIIT bioassay (inhalation) up to 300 ppm. MTD was not reached. Negative in microbial bioassay.
Xylene	D	Insufficient information to determine whether or not xylene itself is carcinogenic.
Alachlor	D	Inadequate data to classify.
Simazine	D	Do.
Endrin	E	Negative results in studies, including NCI, 1978, bioassay.
Carbofuran	E	Negative in 2 species and negative in short-term tests.
Aldicarb	E	Negative results in several studies including the NCI bioassay.
B. IOCs		
Asbestos	A	By inhalation, carcinogenic in humans and animals. By ingestion of intermediate (> 10 μm length) range chrysotile asbestos, limited evidence in animals-benign polyps in male rats. However, the available epidemiologic/experimental data are inadequate to conclude that the chemical is carcinogenic via ingestion.
Arsenic	A	Carcinogenic in humans by inhalation and ingestion. However, this chemical has potential essential nutrient value.
Chromium	A (based on data for Cr^{+6}).	Carcinogenic in humans by inhalation and rodents by intratracheal instillation. However, regulating as "D" since there is inadequate evidence to conclude that the chemical is carcinogenic via ingestion.
Cadmium	B1	Limited evidence in humans exposed to cadmium fumes, cancer in rats exposed to cadmium chloride aerosol, injection site tumors in animals given cadmium salts. However, regulating as "D" since there is inadequate evidence to conclude that the chemical is carcinogenic via ingestion.
Nickel	B1 (based on subsulfide and carbonyl).	Limited evidence in humans by inhalation, sufficient evidence in animals by inhalation and injection. However, regulating as "D" since there is inadequate evidence to conclude that the chemical is a carcinogenic via ingestion.
Lead	B2	Sufficient evidence in animals. Kidney tumors by oral route in rats. However, insufficient basis to regulate as human carcinogen via ingestion.
Barium	D	Inadequate data to classify.
Nitrate/Nitrite	D	Do.
Sodium	D	Do.
Cyanide	D	Do.
Copper	D	Do.
Selenium	D	Do.
Silver	D	Do.
Molybdenum	D	Do.
Sulfates	D	Do.
Mercury	D	Do.

TABLE 5.—RMCL OPTIONS FOR CATEGORY II CONTAMINANTS

Chemical	AADI with added factor of 10 (μg/l) contributing 20 percent drinking water contribution	10^{-5} cancer risk (CAG μg/l)	10^{-6} cancer risk (CAG)(μg/l)
1,2-Dichloropropane	NA	6	0.6
Lindane	0.2	0.20	0.020

TABLE 5.—RMCL OPTIONS FOR CATEGORY II CONTAMINANTS—Continued

Chemical	AADI with added factor of 10 (μg/l) contributing 20 percent drinking water contribution	10^{-5} cancer risk (CAG μg/l)	10^{-6} cancer risk (CAG)(μg/l)
Monochlorobenzene	60	24(NAS)	2.4(NAS)
Styrene	140	NA	NA

TABLE 5.—RMCL OPTIONS FOR CATEGORY II CONTAMINANTS—Continued

Chemical	AADI with added factor of 10 (μg/l) contributing 20 percent drinking water contribution	10^{-5} cancer risk (CAG μg/l)	10^{-6} cancer risk (CAG)(μg/l)
Asbestos (medium and long fibers)	NA	71,[illegible] f/l	7,[illegible] f/l

NA = Not available.

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VI. Microbiological RMCLs

The Interim Primary Drinking Water Regulations, along with the predecessor U.S. Public Health Service standards, have led to widely improved drinking water quality over the last several decades. Compliance with the regulations for total coliform and turbidity is being achieved by most public water systems. No longer do widespread epidemics of typhoid fever and other waterborne diseases occur in the U.S. population. Yet, waterborne disease outbreaks are at significant levels and the trend in the reported number of outbreaks is increasing. Recognition of waterborne illness is difficult and studies indicate that a relatively small percentage of actual cases is being reported. Some of the outbreaks are caused by fecal agents such as *Salmonella* and *Shigella* whose presence should be indicated by accompanying total coliforms bacteria. Many outbreaks are caused by agents such as *Giardia* and viruses for which total coliform bacteria are inadequate as an indicator. Thus, even though total coliform and turbidity regulations are being met by most systems and the microbiological safety of drinking water has improved greatly over the years, significant problems continue to exist and additional regulatory controls appear necessary.

Because total coliforms and turbidity are satisfactory indicators for a number of organisms which have adverse health effects (pathogens), RMCLs will be proposed for both. In addition, RMCLs will also be proposed for *Giardia* and viruses because they have adverse health effects and their presence is not identified by conventional indicators (e.g., total coliforms). The proposed RMCLs appear in Table 6. As discussed later, an RMCL is not being proposed at this time for heterotrophic bacteria (i.e., standard or heterotrophic plate count (HPC)) because there are not yet sufficient data to correlate toxicity with HPC and because they are likely to be controlled through the mechanisms for controlling total coliforms and turbidity. In addition, an RMCL is not being proposed for legionellae because the role of public water supplies in the etiology of the disease has not been determined, and because control of legionellae is more effective at locations of susceptible populations.

In addition, EPA is considering proposing a treatment technique regulation which would require: (1) Surface water systems to practice filtration and disinfection and (2) ground water systems to use disinfection. Variances could be issued for systems that can demonstrate that the raw water quality is such that installation of these technologies would not be needed to protect public health or that alternative technologies are at least equally effective. Summaries of the outbreaks of disease, availability of analytical methods, and human health concerns are provided below.

TABLE 6.—PROPOSED RMCLS FOR MICROBIOLOGICAL PARAMETERS

Parameter	Proposed RMCL
Total Coliforms	Zero
Turbidity	0.1 NTU [2]
Giardia [1]	Zero
Viruses [1]	Zero

[1] Analytical methods do exist but they are not considered to be technically and economically available for *Giardia* or viruses for use in compliance monitoring. Thus, a treatment technique regulation will be proposed.
[2] NTU=Nephelometric Turbidity Unit.

A. Interim Regulations and Recent Outbreaks of Waterborne Disease

The Interim Regulations rely on the measurement of total coliforms and turbidity as indicators of water treatment efficiency, deterioration of water quality in the distribution system, and fecal pollution. Applicable to both community and non-community systems, specific MCLs of the Interim Regulations are shown in Table 7.

Monitoring requirements for coliforms depend upon the size of system, and range from 500 samples per month for systems serving more than 4.7 million persons to one sample per month (or per 3 months in some cases) for systems serving 25 to 1000 persons. Turbidity monitoring is required daily for systems using surface water supplies.

From 1971–1983, there were 427 reported outbreaks of waterborne disease in the United States, involving over 100,000 individuals. Forty outbreaks and 21,000 cases occurred in 1983 alone. Many outbreaks, perhaps the great majority, are not reported to the Centers for Disease Control (CDC), which keeps records on the incidence of reportable diseases. This is because only a few types of waterborne diseases are required to be reported and also because disease outbreaks are often not recognized in a community or, if recognized, are not traced to the drinking water source. In Colorado, an EPA-funded effort to improve the outbreak reporting system indicated that only about one-quarter of the actual outbreaks were being recognized and reported. As recognition of waterborne illness has improved, the trend in the reported number (although not necessarily the actual number) of disease outbreaks and cases has increased.

TABLE 7.—INTERIM REGULATIONS: MICROORGANISMS

Parameter	MCL
1. Total Coliforms:	
a. Membrane filter technique:	
Monthly average MCL	1/100 ml.
Single sample MCLs:	
<20 samples/month	<4/100 ml once/month.
≥20 samples/month	4/100 ml in 5 pct of samples.
b. Multiple-tube fermentation procedure (10 ml portions):	
Monthly average MCL	10 pct of tubes.
Single sample MCLs:	
<20 samples/month	3 or more tubes in one sample/mo.
≥20 samples/month	3 or more tubes in 5 pct of samples/mo.
c. Multiple-tube fermentation procedure (100 ml portions):	
Monthly average MCL	60 pct of tubes.
Single sample MCLs:	
<5 samples/month	5 tubes in one sample/mo.
≥5 samples/month	5 tubes in 20 pct of samples/mo.
2. Turbidity:	
Monthly average	1 NTU (up to 5 NTU).
2-consecutive day average.	5 NTU.

From 1971–1980, 50 percent of the outbreaks reported were in non-community water systems, 39 percent in community systems, and 11 percent in private systems. Although most of the outbreaks were from non-community systems, about 75 percent of the illness occurred from outbreaks in community systems. Between 1971–1980, the major causes of outbreaks in community water systems were treatment deficiencies (49%) and contamination in the distribution system (32%). Almost all outbreaks (83%) and illnesses (89%) in non-community systems were a result of using ground water without treatment or using ground water with inadequate treatment, primarily interrupted and inadequate disinfection.

Most known agents of waterborne disease cause acute gastrointestinal disorders, especially diarrhea and cramps. During the period 1971–1983, the most commonly identified pathogen was the protozoan *Giardia lamblia*. During these years, there were 77 reported outbreaks of waterborne giardiasis involving nearly 23,000 cases. A number of bacteria also have recently been implicated in waterborne disease. These include *Salmonella* species, *Shigella species*, *Campylobacter jejuni*, *Yersinia enterocolitica*, and enteropathogenic *E. coli*. Viral agents implicated in recent waterborne illnesses include Norwalk and Norwalk-like agents, rotaviruses, and the hepatitis A agent. In about half the waterborne outbreaks the causative

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agent has not been found. There is growing suspicion that many of these may be due to viruses. Unfortunately, the unavailability of suitable analytical techniques have impaired efforts to resolve this issue.

B. Total Coliform Bacteria

Total coliform bacteria have been used for decades as the primary measurement of the microbiological quality of drinking water. Total coliform bacteria are a measure of the efficiency of water treatment, disclose deterioration in the distribution system and signal the possible presence of fecal contamination. Analytical methodology for total coliform monitoring has existed for decades. Current EPA drinking water regulations specify the use of either the multiple-tube fermentation technique or the membrane filter technique. Both enjoy widespread acceptance and are recognized as suitable methods by *Standard Methods for the Examination of Water and Wastewater.* EPA is also considering additional analytical methods, such as Clark's Presence-Absence test (see *Standard Methods,* 6th ed.) which detects coliforms but does not quantify them.

Coliforms are usually present in fecally-polluted water and are often associated with disease outbreaks. Although coliforms are not usually pathogenic themselves, their presence in drinking water indicates the likely presence of pathogens (e.g., *Salmonella* and *Shigella*). However, use of total coliforms has a number of drawbacks including the following:

- Given their ubiquitous nature, coliforms are often found in the absence of fecal contamination.
- There are reports of pathogen occurrence and disease outbreaks where coliforms were not detected.
- Coliforms are inadequate for predicting the presence of pathogens/toxins not associated with fecal contamination such as atypical mycobacteria, *Legionella,* and algal toxins.
- They also may not adequately predict the presence of enteric viruses, *Giardia* and some other organisms, because they are less resistant to treatment (e.g., disinfection) than these organisms.

The qualitative use of total coliforms can be scientifically justified, but there is an absence of scientific data in the literature supporting a particular value for coliform density, below which the water can be considered safe. Pathogens and outbreaks have been associated with coliform densities ranging from zero to very high levels. This is not surprising; pathogens vary in their: (1) Association with fecal contamination, (2) die-off times in water, and (3) resistance to disinfection or other treatment. Although the total coliform indicator is an inexact tool, fecal pathogens in ambient water are usually accompanied by substantially higher levels of total coliforms. Since higher levels of pathogens should roughly translate to greater risk of disease, coliform densities indirectly serve as a measure of the risk of waterborne disease. Treatment which provides coliform-free water as measured by the standard tests should reduce pathogens to minimal levels, even though large volume risk-free water is probably unattainable.

Despite these and other limitations, total coliforms are still the single most useful indicator of drinking water quality. Because total coliforms most directly measure pathogens which widely occur in drinking water, EPA is proposing an RMCL for total coliforms. (To assure a safe drinking water supply, control of total coliforms should be used in combination with other microbiological parameters and protective measures, e.g., turbidity, filtration and disinfection.) The discontinuance of total coliforms as an indicator of treatment efficiency would substantially undermine the ability to predict the potential presence of pathogens, even if turbidity monitoring is retained. Turbidity removals, for example, cannot measure disinfection effectiveness.

The object of an RMCL for total coliforms is to recommend that as a goal the consumer will not be exposed to a sufficient dose of a pathogen to result in disease. Since the relationship between coliform and pathogen levels is highly variable, the proposed RMCL for total coliforms should be zero, i.e., no detectable coliforms/100 ml as measured by the Most Probable Number (MPN), Membrane Filter (MF), and other EPA approved analytical methods in order to assure maximum protection within the limits of the measurement technique. This RMCL includes an adequate margin of safety. While the RMCL is being proposed at zero, the concept of "presence-absence" is being considered as the basis for the enforceable MCLs. This would involve measurement of total coliforms to determine only if coliforms were present or absent without quantification. For example, MCL regulations could specify that 95 percent of samples examined over a given time period have no coliforms present. Public comment is requested on setting an RMCL of zero for total coliforms and on the use of the Presence-Absence concept as the basis of the total coliform MCL.

C. Turbidity

Turbidity in water is a non-specific measure of suspended material in drinking water and is measured by determining the degree of light scattering caused by particulates in a sample. Turbidity has been used for decades as an indicator of drinking water quality indicating the presence of such particulates as clay, silt, finely divided organic and inorganic matter, and microorganisms. Analytical methodology for turbidity monitoring is available. Currently, the only EPA-approved technique is the Nephelometric Method. This is also recognized as an acceptable technique by *Standard Methods for the Examination of Water and Wastewater.*

Turbidity is an imprecise measure because particulate characteristics vary from system to system and seasonally within the same system, and some particulate types are more significant than others for human health. The more important particulates appear to be larger than 0.03 μm in diameter and are usually organic materials. Turbidity is of concern in drinking water because of the following characteristics:

- Turbidity can reduce the efficiency of disinfection.

—Certain particles protect adsorbed pathogenic organisms against disinfection. In general, inorganic particles such as clays and water flocculating agents appear to have little, if any, protective effect. In contrast, organic particulate matter, whether cell debris, sewage solids, or living or dead organisms such as nematodes or crustaceans, can provide marked protection to microorganisms associated with them.

—A significant disinfectant demand can chemically interfere with disinfection during treatment or maintenance of a disinfectant residual in the distribution system.

- The turbidity can be caused by particles that are toxic themselves or that adsorb toxic inorganic or organic substances from the water.
- Higher turbidity levels can also interfere with total coliform analyses by the membrane filtration procedure.
- Turbidity is a measure of the efficiency of drinking water coagulation and filtration processes. Removal of turbidity usually provides concommitant removal of microbial pathogens, especially those which aggregate on particulates, as well as particulate

er which is either toxic itself or
rbs toxic contaminants.

ess, turbidity represents many
cerns in drinking water quality but
nct relationships between levels of
idity and the safety of the drinking
r are not quantifiable. However, as
ndard sanitation principle, the
r the turbidity of treated water, the
likely the water will contain
aminants deleterious to human
th. For this reason and because
idity is common in drinking water
ces, EPA is proposing an RMCL for
idity.

s with the total coliform RMCL, the
ct of the turbidity RMCL is to
vide maximum assurance that the
sumer will not be exposed to a
ficient pathogen dose to result in
ase (including a margin of safety).
this basis, the proposed health goal
urbidity is 0.1 NTU. This is primarily
ed upon the premise that the lower
urbidity levels, the less the
obility that aggregated
roorganisms are entering finished
king water. Significantly, there
ears to be some correlation between
idity removal and *Giardia* removal
iencies in filtered water. Setting the
CL at 0.1 NTU represents the lowest
l for which sufficient data are
lable (and is essentially the
ytical level of detection). Lower
idity can be measured, but only
ugh careful measurements under
rolled conditions. An RMCL for
idity of zero would not be
opriate since even distilled water
turbidity of about 0.05 NTU. In
tion, additional benefits of
eving very low turbidities include
following:

Several studies have demonstrated
substantial removal of *Giardia*
s by coagulation and rapid granular
ia filtration. However, this would
suggest that a drinking water with a
turbidity level would de facto be
from *Giardia*. *Giardia* can be
ent in low turbidity unfiltered
ers.

Removal of turbidity particles for
removal will also substantially
ce levels of other pathogenic
nisms and toxic particulate matter,
as bacteria, pathogenic viruses and
amoeba cysts.

The total coliform group, while the
le best indicator of water quality, is
equate by itself to measure drinking
r quality. The measurement of
idity complements coliform
itering since particles may interfere
coliform analysis and particles may
ate toxic materials even in the
nce of coliforms. There are cases on record where outbreaks were accompanied by sudden rises of turbidity in filtered coliform-free water. In addition, turbidity is monitored at least daily and results are available in a much shorter time than for coliforms.

Other organizations support turbidity controls including the American Water Works Association (AWWA) and the World Health Organization (WHO).

• The AWWA policy is as follows.

Today's consumer expects a sparkling, clear water. The goal of less than 0.1 unit of turbidity insures satisfaction in this respect. There is evidence that freedom from disease organisms is associated with freedom from turbidity, and that complete freedom from taste and odor requires no less than such clarity. Improved technology in the modern treatment processes make this a completely practical goal.

The WHO guidelines for turbidity state that "turbidity must always be low, preferrably below 1 NTU and always less than 5 NTU."

Public comments are requested on setting an RMCL for turbidity in drinking water. Specifically:

• Is a level of 0.1 NTU appropriate for the RMCL? If not, what other level and upon what basis?

D. Giardia

The protozoan *Giardia lamblia* causes more waterborne disease outbreaks in the United States than any other single identified etiologic agent. Symptoms of giardiasis include diarrhea, fatigue, abdominal cramps, and possibly other gastrointestinal symptoms, and these may persist for several days to several months. For these reasons, EPA is proposing an RMCL for *Giardia*.

Between 1971–1983, there were 77 reported outbreaks of waterborne giardiasis with nearly 23,000 cases. The trend in outbreaks has been on the increase, probably due to greater recognition and reporting of giardiasis outbreaks in recent years. Waterborne giardiasis represented 15.7 percent of the total reported cases of giardiasis in 1980 and 2.4 percent in 1981. These percentages, however, may substantially under-represent the actual situation, since the route of transmission for many cases is unknown and may in fact be waterborne. In Pennsylvania recently, there were four separate out breaks of waterborne giardiasis, with about 700 cases. Over 350,000 individuals were being served by public water systems under boil water orders (i.e., water provided by a public water system is not safe to drink unless boiled first). In three outbreaks, the systems provided only chlorination. In the remaining outbreak, coagulation, filtration and disinfection were used, but the system appeared to be overloaded and improperly operated. In all four situations, the systems were in compliance with total coliform and turbidity MCLs.

The populations most at risk are those consuming surface waters which are either untreated or only chlorinated. *Giardia* are effectively controlled by filtration (including coagulation) and disinfection. Disinfection alone is not normally sufficient. It is estimated that over 65 million people are served by surface water systems that either provide only disinfection or no treatment at all.

Cyst recovery and analysis procedures have been published in the literature, but they are considered insensitive, tedious and expensive. The most commonly used method for recoverl and detection of *Giardia* from water was developed by EPA. This involves filtering large volumes (up to 2,000 liters) of water through an Orlon fiber filter, separating the cysts from the fiber, and examining the cyst concentrate microscopically. This procedure appears in *Standard Methods for the Examination of Water and Wastewater* (15th ed.), as a tentative method. Beyond the difficulties in recovery and detection, it is not yet possible to distinguish between viable and non-viable cysts. This method is therefore not considered available for the monitoring of waterborne *Giardia*. Promising research is being conducted on *Giardia* recovery and analysis techniques.

The proposed RMCL is based upon a human study. In this investigation, male volunteers were fed human-source *Giardia* cysts contained in gelatin capsules. Those receiving one cyst were not infected, while infections did occur in the group receiving ten cysts. A major deficiency of this study is that cyst viability was not established; thus it is possible that a sizable fraction of the ingested cysts were non-viable. In addition, only two individuals were tested at the ten cyst level (both, however, were infected). Recent unpublished data with mice support the low infective dose for *Giardia*. In four experiments, the average dose at which 50 percent of the animals were infected (ID_{50}) were 1.4, 2.6, 3.8 and 17.6 cysts. Since some of these cysts may have been non-viable, the ID_{50} dose may actually be lower than the results indicate.

On the basis of these studies and inclusion of a safety factor, the RMCL is proposed at zero viable cysts. Because of the insensitivity of the currently available analytical procedures, the potential for intermittent contamination

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and thus difficulty in monitoring, and lack of technologically and economically feasible methods, treatment requirements are being considered (instead of setting an MCL). However, the inability to measure these contaminants alone does not prevent establishment of an RMCL. In this case, it is reasonably clear that any *Giardia lamblia* cyst can have an adverse health effect. Therefore EPA is setting an RMCL of zero. Treatment techniques could include filtration and disinfection for all water systems using surface water. Ground water is not nearly as vulnerable to cyst penetration since the aquifer and the overlying soil usually serve as an effective barrier. Thus, filtration of ground water will not be proposed. Variances under section 1415 may be considered in those circumstances where the system is able to demonstrate to the State by a sanitary survey or other means to be specified, that the treatment is not needed to protect the health of persons because of the quality of the raw water. The treatment technique requirements will be proposed when the MCL for other contaminants are proposed. Criteria for determining if a variance would be appropriate will be included in the proposal.

Public comments are requested on this approach of setting an RMCL of zero for *Giardia* and treatment requirements to control *Giardia* in drinking water. Specific comments are also requested on the following:

- Are analytical methods available (i.e., is it economically and technologically feasible) to ascertain the level of *Giardia* in drinking water?
- What specific criteria should be included in the regulations that would allow States to evaluate if a variance should be issued to a particular system?

E. Pathogenic Viruses

Between 1978–1982, there were 18 reported waterborne disease outbreaks in the U.S. caused by viruses, with over 5,700 cases. These values are probably far too low to represent the actual number of outbreaks because there is evidence that most of the waterborne disease outbreaks of unknown origin are caused by viruses. Viruses are a class of infectious agents which are extremely small (smaller than bacteria) and reproduce only within cells of a suitable living host such as humans. They contain genetic material surrounded by a protein coat. Pathogenic viruses, by definition, are those that adversely effect health. The lack of adequate tools for the recovery and analysis of most pathogenic waterborne viruses undoubtedly contributes to this under-reporting.

The most important of the waterborne pathogenic viruses are the Norwalk and Norwalk-like agents, rotavirus, and the hepatitis A agent. Except for the latter, their predominant health effect is acute gastroenteritis. Hepatitis A agent results in hepatitis. All have been implicated in recent waterborne disease outbreaks. While serological techniques are available for identifying these agents (e.g., radioimuno-assay, immune electron microscopy, and others), there are no satisfactory culture procedures currently available. Promising research on culturing rotaviruses and hepatitis A agent is presently being conducted.

The classical enteroviruses (poliovirus, echovirus, and coxsackievirus) are common in ambient water and are occasionally found in drinking water, but they have not been implicated in recent outbreaks. Nevertheless, their health effects may be severe (e.g., meningitis, paralysis, myocarditis, diarrhea).

Unlike many of the other agents, recovery and analysis procedures for these enteroviruses do exist, some of which are described in *Standard Methods for the Examination of Water and Wastewater* (15th ed). Viral assay, however, is beyond the capability of most water microbiology laboratories and must be done by a trained virologist working in specifically equipped viral laboratory facilities.

The minimum infective dose for some strains of waterborne viruses is very low. There is evidence that in some instances, as little as one tissue culture infective dose is able to infect a person. Data from a recent study where healthy human volunteers were infected by Echovirus 12 suggest that 1 percent of a population could be infected by 17 plague-forming units of this virus. Because of this, the goal should be to have no viruses in potable water. This is supported by the WHO. Thus, the proposed RMCL goal for human pathogenic viruses in drinking water is zero. This RMCL goal would include the enteroviruses in addition to any others for which analytical methods are not yet available. Because EPA concludes that one virus may have adverse effects, it is able to propose an RMCL, despite the fact that the measurement is difficult.

Because routine, validated procedures for detection of the most important waterborne viruses (hepatitis A agent, rotaviruses, and Norwalk and Norwalk-like agents) are not yet technically feasible, and because of the potential for intermittent contamination which presents difficulties in effective monitoring, a treatment technique requirement is being considered for proposal in the next rulemaking. This could include coagulation, filtration and disinfection for water systems using surface water and disinfection for systems using ground water. Alternatively, an easily measurable surrogate for waterborne viral pathogens could be selected as the proper regulatory approach; however, the only techniques currently under study are classical enteroviruses and coliphage and these are not considered sufficient to provide protection against the myriad of pathogens potentially present in various drinking waters. A variance could be considered in those circumstances where a system is able to demonstrate to the satisfaction of the State, by an on-site sanitary survey and other means to be specified, that treatment would not be needed to protect public health because of the quality of the raw water supply. The treatment technique requirements, if any, will be proposed when the MCL for other contaminants are proposed. Criteria to be used by public water systems and States in determining if a variance would be appropriate will be included in the proposal.

Public comments are requested on this approach of setting an RMCL of zero for viruses and setting treatment requirements for control of viruses in drinking water. Comments are also requested on the following:

- Are analytical methods available (i.e., is it economically and technologically feasible) to ascertain the level of pathogenic viruses in drinking water?
- What specific criteria should be specified in the regulations that would allow States to provide variances to certain systems?
- Are satisfactory methods available for recovery and analysis of classical enteroviruses or coliphage and are they representative surrogates for pathogenic viruses? If so, should an RMCL and MCL or a treatment technique be established for classical enteroviruses or coliphage?

F. Legionellae

Legionellae are bacteria that have been identified as the cause of legionellosis. It has been estimated that 50,000–100,000 cases of this disease occur annually within the United States, and are caused primarily by 1 of the 28 currently recognized species of the genus *Legionella*. The number of cases attributable to drinking water is unknown. Most people who have developed Legionnaires Disease, the pneumonia form of legionellosis, were

patients that were immunosuppressed, or were individuals who appeared to be more susceptible because of an underlying illness, heavy smoking, alcoholism, or were over 50 years old. In contrast, while some apparently healthy individuals have developed Legionnaries Disease, outbreaks involving healthy people have been limited mostly to the milder non-pneumonia form of the disease called Pontiac Fever.

Legionellae are abundant in ambient water, and may survive water treatment, especially since they are relatively resistant to chlorine. Even conventional treatment (i.e., filtration and disinfection) probably cannot prevent the passage of a small number of legionellae into the distribution system. They may also be introduced into drinking water via broken or corroded piping, repair of existing mains, installation of new mains, back siphonage, and cross connections. When the legionellae enter hot water tanks, they can settle to the botom and, under certain circumstances, will proliferate. If they proliferate, plumbing fixtures such as aerators, water fitings, and showerheads may be seeded, resulting in colonization and growth at these sites. They are often found in the hot water plumbing of hospitals, hotels, and other buildings, especially in hot water tanks and showerheads where the hot water temperature does not exceed 120°F. In many hospitals, the hot water temperature is maintained at this temperature and below to prevent patient scalding and to reduce energy costs.

There is good epidemiologic evidence from several hospitals that Legionnaires Disease is transmitted by aerosols of potable water from showerheads containing legionellae. Other sources implicated in disease transmission are aerosols from cooling towers and whiripools. Inhalation of aerosolized potable water is probably the primary route of infection, although ingestion is a possibility.

Analytical tools exist for recovery and enumeration of these organisms from plumbing systems. However, these are not yet very efficient for selectively recovering and enumerating legionellae from drinking water.

Because of the ubiquity of legionellae in ambient water, their proliferation primarily at warmer temperatures, their relative resistance to chlorination and ability to colonize in plumbing systems, control is probably more appropriate at locations where susceptible populations reside, rather than at the waterworks. More significant, however, is the fact that virulence factors have not yet been identified for the organisms in this genus. There is great variation in virulence, and the mere presence of large populations of legionellae in the drinking water does not necessarily represent a health threat. For all these reasons, setting national drinking water regulations for legionellae appears to be inappropriate at this time, and an RMCL is not being proposed.

EPA has prepared guidance for hospitals and other high risk locales; this guidance is summarized below and is available from EPA at the address cited at the beginning of this notice. In order to reduce legionellae levels in drinking water, storage reservoirs should be managed to minimize the addition of organic matter and growth of algae and protoza. Moreover, newly repaired or constructed components of the water distribution system must be thoroughly flushed and disinfected before being put into operation. Even after flushing and disinfection, one cannot assume legionellae have been controlled since design factors in the distribution system may impede the efficiency of these measures.

In order to control legionellae growth in hot water plumbing, several approaches may be considered. Most of the published data have examined the effectiveness of chlorine and/or heat. The maintenance of free chlorine has been found effective for controlling legionellae. Shock chlorination is also effective, but unless free chlorine is maintained within a system, the organism may reappear. Control can probably be achieved if free chlorine levels in the hot water are maintained above 2 mg/l, but at these levels corrosion of pipes may occur. Successful eradication has been reported with 3 mg/l chlorine in the hot water system for 10 days, followed by a 1.5 mg/l maintenance level. With this treatment, only a slight increase in corrosivity was found. Undoubtedly, the level of chlorine found effective will depend, in part, on the design criteria of the plumbing system. A pertinent facet in controlling legionellae is the difficulty of maintaining a chlorine residual in hot water. This problem can be minimized by using either a closed recirculating system or a continuous-flow proportional chlorinator on the hot water system.

Heat shock (160 °F for 1 hour) may eradicate legionellae in hot water tanks and is a temporary measure which must be carried out routinely to be effective. Maintenance of hot water at 130 °F or higher apparently controls the organism, while lower temperatures may not. If legionellae are controlled by heat, care must be taken to prevent scalding, especially in health care situations.

Legionellae may be controlled by ultraviolet radiation. Data on ozone treatment are incomplete at this time.

In addition to disinfection and heat, other procedures may be effective in controlling legionellae. Hot water tanks should be designed to give uniform temperatures throughout. Dead-end piping should be eliminated. Hot or cold water tanks used intermittently should be disconnected from the system, drained, flushed, and disinfected before being reconnected. Hot water tanks should be drained regularly or at least be bled to remove accumulated sludge that may serve as a substrate for growth of legionellae and other micro-organisms. Taps and showers in unused areas of health care facilities should at least be flushed before patients are exposed to them. Finally, faucet sieves and aerators, and rubber washers and gaskets in the plumbing system should be used with caution, especially in institutions housing compromised individuals and where hot water is maintained at temperatures lower than 130 °F.

Public comments are requested on whether it is appropriate to propose an RMCL and primary drinking water regulations for legionellae or whether the appropriate action is to provide guidance.

G. Heterotrophic Bacteria

Heterotrophic bacteria are those which use organic nutrients for growth. This group includes virtually all of the bacterial pathogens as well as many other innocuous bacteria. The population density of these bacteria in water is often measured by the Standard Plate Count (SPC) procedure, as described in *Standard Methods for the Examination of Water and Wastewater*. The 16th edition of this book changes the SPC designation to Heterotrophic Plate Count (HPC).

Primary reasons for considering a regulation for heterotrophic bacteria include the following:

- Many heterotrophs in water are opportunistic pathogens (30% in one study). There is some evidence that numerous hospital-acquired infections have been caused by waterborne opportunistic pathogens. Thus, a higher bacterial density, as measured by the HPC procedure, may reflect a higher level of pathogens in the water which are able to cause disease in the compromised population.
- High HPC densities, or sudden increases of HPC density, may indicate water quality deterioration. Thus, this

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group or organisms can be used to provide warning of a filter bed breakthrough, monitor the effectiveness of disinfection throughout the distribution system provide a method for monitoring the condition of point-of-use water filters and provide other monitoring data. In some of these areas, HPC monitoring can be more informative than total coliform monitoring. For example, in research studies of point-of-use filters, coliforms are rarely found in the effluents. The growth or potential growth of opportunistic pathogens or even primary pathogens could be better signalled by higher HPC densities in the effluent. Thus, HPC monitoring can be a useful supplement to coliform monitoring.

• The NAS stated that HPC is a valuable procedure for assessing the bacterial quality of water, and that a density less than 500/ml is attainable by water systems (*Drinking Water and Health*, 1977, Vol. I).

• Higher HPC densities may interfere with total coliform analysis. Several published articles have indicated that non-coliform bacterial densities greater than 500 colonies/ml can suppress coliform growth with both the membrane filter and multiple tube fermentation procedures. There is also strong evidence that drinking water samples containing high non-coliform bacterial densities resulting from regrowth during sample transit may reduce the chances of detecting total coliforms.

Although there is some evidence that high heterotrophic bacterial densities are of concern, the Agency believes that it would be premature to propose an RMCL, given the lack of conclusive data regarding the link between opportunistic pathogens in drinking water and nosocomial (hospital-acquired) infections. Instead of proposing an RMCL, MCL and monitoring/reporting requirements, the Agency is considering incorporating a level of HPC control into the total coliform monitoring requirements since high densities of HPC interfere with coliform analysis. The monitoring requirements for total coliforms could specify that a certain percentage of the samples collected for coliform analyses would also be analyzed for HPC; if HPC were greater than some level (e.g., 500 colonies/ml), the total coliform analyses would not be valid. This would provide a measure of quality assurance not previously required in the Interim Regulations. Monitoring for HPC would also indicate treatment effectiveness and possible deterioration of water quality.

Public comments are requested on the following:

• Should an RMCL and MCL be set for HPC? Upon what basis and what level?

• Should HPC be included in the monitoring requirements for total coliforms such that HPC could not exceed a certain density in order to have valid total coliform results? What level would be appropriate?

H. Treatment Technique Requirements

Discussed below are possible treatment regulations that EPA is considering for the next rulemaking. The regulations are not being proposed at the present time and are presented for the purpose of public comment.

1. Mandatory Filtration and Disinfection of Surface Water for Microorganism

EPA is considering a regulation requiring the filtration and disinfection of all surface waters before distribution to consumers. This regulation would provide protection against a myriad of disease-producing microorganisms which can frequently be found in ambient water used as drinking water supplies. As discussed previously, a search in finished drinking water for each of these primary and opportunistic pathogens would not be technologically or economically feasible. The concentration of pathogens in water would usually be sufficiently small so as to require large-volume samples. Many are so fastidious that only highly specialized laboratory techniques can be used to recover and detect them, if they can be detected at all. This is true of the hepatitis A agent, rotavirus, Norwalk and Norwalk-like agents, and *Giardia*. Unfortunately, total coliform bacteria are inadequate as an indicator for the presence of these organisms. Thus, EPA may propose a treatment requirement in the next rulemaking.

Of the approximately 14,000 community water systems in the U.S. which use surface water, about 79 percent practice full conventional treatment and about 98 percent employ at least disinfection. Between 1971 and 1980, there were 19 reported waterborne disease outbreaks with over 11,000 cases in localities served by surface waters receiving chlorination only. This represented 35 percent of the total number of outbreaks and 44 percent of the cases associated with surface water supplies, even though disinfected-only surface water systems represent less than 17 percent of the total number of surface water systems. During that same period, 6 disease outbreaks (11% of the total) and over 200 cases were associated with untreated surface water supplies. These data strongly suggest that filtration and disinfection of surface waters substantially reduces the potential for waterborne disease outbreaks. As previously discussed, recent studies suggest that actual disease outbreaks may be much higher than reported.

Rapid granular filtration, in use since the 1890s, can remove between 90-99.9 percent of the bacteria and protozoan cysts and 90-99 percent of the viruses from source water. Slow sand filtration, in continual use from the early 19th century, can remove 90-99.9 percent of the bacteria, and viruses and 90-99 percent of protozoan cysts. A third type of filter, diatomaceous earth (diatomite), used since World War II, can remove 90-99 percent of the bacteria, 95 percent of the viruses, and 99 percent of the cysts. Percentage removals for filtration without pretreatment (i.e., coagulation, flocculation, and settling) are extremely variable, ranging from 0-60 percent for bacteria and cysts and 0-50 percent for viruses.

Besides providing a barrier to organisms, especially for *Giardia*, viruses, and other pathogens relatively resistant to disinfection, filtration reduces the level of particulate matter that may protect microorganisms from disinfectants, reduces the level of toxic particulate matter, and reduces short-term fluctuations in water quality. Formation of trihalomethanes and other by-products of disinfection are also reduced by filtration which reduces the quantity of precursors that can react with the disinfectant.

Filtration and disinfection reduce contamination continuously and deal with the possible events of periodic contamination. Without treatment, monitoring for contamination is necessarily intermittent and the microbiological quality of the drinking water is not known for more than a day or more and sometimes much longer. This is especially true of small systems where total coliform monitoring is quite limited. In 1982, about 24 percent of the public water systems violated coliform monitoring requirements at least intermittently and 14 percent violated turbidity monitoring requirements. Furthermore, as noted previously, total coliform monitoring is not a adequate indicator of all possible pathogens.

Any filtration and disinfection of surface supplies would not necessarily require all systems to adopt these treatment methods. The SDWA (section 1415) allows systems to receive a variance from the requirements if certain conditions are met. All systems would be required to install filtration and disinfection unless the system demonstrates to the satisfaction of the

State that the treatment technique is not necessary to protect the health of persons because of the quality of the raw water source of the system. Criteria would be provided in the regulation which the system must meet in order to receive a variance.

The practice of filtering surface water is supported by a number of professional groups such as the AWWA: "The American Water Works Association (AWWA) strongly supports the practices of filtration of surface water used as sources of public water supply, disinfection of public water supplies, including the maintenance of residual disinfection in the distribution system, . . ." (AWWA, 1983. AWWA Officers and Committee Directory, Policy Statements and Official Documents. p. 74). In addition, a workshop convened by the EPA's Office of Drinking Water, in conjunction with the American Society for Microbiology, to advise EPA on a variety of drinking water issues, strongly recommended the filtration of surface waters (Assessment of Microbiology and Turbidity Standards for Drinking Water, Dec. 2–4, 1981, July 1983, EPA 570–9–83–001).

Public comment is requested on the following:

- Should a treatment technique requirement be established such that system using surface waters would be required to use filtration and disinfection? Upon what basis?
- What specific filtration and disinfection technologies should be included in the definition of a "filtration" and "disinfection"? For example, direct filtration? slow sand filtration?
- Should these treatment requirements apply to non-community drinking water systems?
- What criteria should be specified that would provide guidance in the issuance of variances?

2. Mandatory Disinfection of Ground Water

EPA may also propose, in the next rulemaking, a treatment regulation requiring the disinfection of all ground waters before distribution to the consumer. Many of the same microorganisms that occur in surface waters are also found in ground waters. Because a search for each pathogen is not technically or economically feasible, and because the presence of some are not adequately signalled by the presence of coliforms, a treatment technique regulation may be proposed. Filtration of ground water supplies, while encouraged, may not be proposed as a requirement because the soil acts as a natural filter, thereby usually reducing microbial and particulate contamination of the underlying water.

The number of reported disease outbreaks and cases associated with untreated ground water supplies are substantially greater than those for treated ground water supplies. According to published data, communities served by untreated ground water have had 3.7 times as many cases of illnesses. Between 1971–1982, untreated well water was associated with 110 disease outbreaks and over 8500 cases of illnesses. If untreated spring water is added to this total, the values are 126 outbreaks and over 9800 cases. In 1982, untreated ground water was responsible for 28 percent of all reported waterborne disease outbreaks and 10 percent of all waterborne illnesses. The etiological agents implicated in these outbreaks were the hepatitis A agent, *Yersinia*, and *Giardia*; in 5 outbreaks the agent was not identified.

Adequate disinfection reduces contamination continuously and deals with periodic contamination. Similar to surface waters, monitoring for contamination is necessarily intermittent, especially for small systems. Moreover, in 1982, about 24 percent of the utilities violated coliform monitoring requirements at least intermittently and 14 percent violated turbidity monitoring requirements.

A variety of disinfectants are available. Currently, the best are chlorine (as hypochlorous acid), ozone, and chlorine dioxide. All three have excellent biocidal activities against bacteria and viruses. For inactivation of protozoan cysts, ozone is excellent, chlorine has only moderate biocidal activity, and no published data are yet available for chlorine dioxide. Chlorine and chlorine dioxide residuals can persist in the distribution system, ozone residuals cannot. Besides these three disinfectants, others are being used or have been suggested for use. These include chloramines, iodine, bromine, and ultraviolet light. A treatment regulation will recommend the types of disinfectants appropriate for use, the range of acceptable disinfectant concentrations, minimum contact times, and possibly the minimum and maximum residual concentrations in the distribution system. Variances would be considered in those cicumstances where a system is able to demonstrate to the satisfaction of the State that the source water is of sufficiently good quality to obviate the need for disinfection. Like the surface water regulation, criteria to assist in making variance determinations would be provided if a regulation is proposed.

Public comment is requested on the following:

- Should a treatment technique requirement be established such that systems using ground water would be required to provide disinfection? Upon what basis?
- What specific disinfection technology should be included in the regulation?
- Should these treatment requirements apply to non-community drinking water systems?
- What criteria should be specified for the issuance of variances?

VII. Inorganic RMCLs

The Interim Regulations contain MCLs for the following ten inorganic chemicals:

Contaminant	MCL, mg/l
Arsenic	0.05
Barium	1
Cadmium	0.010
Chromium	0.05
Fluoride	1.4 to 2.4, depending on climate.
Lead	0.05
Mercury	0.002
Nitrate (as N)	10
Selenium	0.01
Silver	0.05

Monitoring and reporting requirements were also included in the Interim Regulations for sodium and corrosion.

The ANPRM (48 FR 45502) listed 23 IOCs under consideration for Primary Drinking Water Regulations. RMCLs are proposed for 11 IOCs (one of which was not listed in the ANPRM—nitrite), one IOC (fluoride) will be included in a separate proposal, and 6 IOCs (cyanide, molybdenum, nickel, silver, sodium, and sulfate) have been determined inappropriate for regulation based upon limited health effects data and/or occurrence in drinking water. Five IOCs (antimony, beryllium, thallium, vanadium and aluminum) will be addressed at a later date and one IOC (zinc) has been determined inappropriate for regulation based upon EPA and the National Academy of Sciences (1977 and 1980) reviews.

For the 11 inorganic chemicals for which RMCLs are proposed, the Administrator has determined that human exposure to these IOCs in drinking water may have an adverse effect upon the health of persons.

Table 8 presents the proposed RMCLs for the 11 IOCs. Table 9 summarizes the short-term assessments for those chemicals for which RMCLs are proposed and Table 10 summarizes the short-term assessments and provisional

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AADIs for those chemicals for which RMCLs are not proposed.

Presented in this section are (1) a discussion of analytical methods available for measurement of IOCs and (2) separate discussions for each IOC on (a) the occurrence in drinking water and the relative contributions from drinking water, air and food, and (b) the potential health effects of exposure. In this notice, EPA is presenting a summary of those analytical methods that appear to be available. In the MCL proposal, EPA will propose methods that have been determined to be economically and technologically feasible.

In the discussion of health effects, information on 1-day exposure, 10-day exposure and chronic toxicity effects is included. In addition, a summary of health-related guidelines prepared by other groups and organizations is presented for each IOC. Levels that have been calculated by the WHO, the NAS and EPA's Office of Water Regulations and Standards (Water Quality Criteria) have been included. In several instances, these values differ from the proposed AADIs. This is due to several different factors, including the use of different uncertainty factors, different interpretations of data and varying assumptions. In some cases, new data may have become available resulting in the derivation of an AADI which differs from the earlier calculated values. This section closes with a description of the toxicological basis for the proposed RMCL. This includes calculations of Adjusted Acceptable Daily Intakes (AADI's) for threshold toxicants and, in addition, a risk assessment for substances that are being proposed for regulation as potential human carcinogens. Issues are identified for which public comments are requested on each of the IOCs. The information presented here is summarized from the supporting documents on analytical methods, occurrence, and health effects referenced in Section X.

A. Availability of Analytical Methods

Analytical methods are available for the determination of all the IOCs for which RMCLs are proposed in this notice with the exception of asbestos. Preliminary assessments have been conducted of existing methodologies to determine their suitability in terms of performance, cost, complexity, and other factors such as the availability of trained personnel to conduct the analyses. Specific analytical methods for each contaminant will be proposed as part of the MCL proposal along with specific criteria for the determination of acceptable performance for those laboratories conducting compliance analyses.

TABLE 8.—PROPOSED RMCLS FOR IOCS

IOC	Provisional AADI [4] (mg/l)	Proposed RMCL [5] (mg/l)
Arsenic	0.10	0.050 [1]
Asbestos (medium and long fibers).		7.1 million fibers/liter [6]
Barium	1.6	1.0
Cadmium	0.010	0.005
Chromium	0.17	0.12
Copper	1.3	1.3 [2]
Lead		0.020
Mercury	0.005	0.002
Nitrate	10.0	10.0 [3]
Nitrite	1.0	1.0 [3]
Selenium	0.100	0.045

[1] Based on NAS recommendation and data which suggest that arsenic may be an essential nutrient.
[2] Based on acute toxicity.
[3] Based on acute toxicity for infants up to 3 to 6 months of age.
[4] The AADIs were determined from studies of less than lifetime duration (approximately 2 years for an animal study).
[5] Based upon Provisional AADI with drinking water contribution factored in.
[6] Based upon classification in Category II as a possible carcinogen using a 10^{-6} cancer in risk level. Limited to fibers longer than 10 um.

TABLE 9.—SHORT-TERM ASSESSMENTS FOR IOCS FOR WHICH RMCLS ARE ALSO PROPOSED

IOC	1-Day (mg/l)		10-day (mg/l)		Longer-term [1] (mg/l)	
	Child	Adult	Child	Adult	Child	Adult
Arsenic	0.05	0.05	0.05	0.05	0.05	0.05
Asbestos	NA	NA	NA	NA	NA	NA
Barium	NA	NA	NA	NA	NA	NA
Cadmium	0.043	0.150	0.005	0.020	NA	NA
Chromium (VI)	NA	NA	1.4	5.0	0.24	0.84
Copper	1.3	1.3	NA	NA	NA	NA
Lead	NA	NA	NA	NA	NA	NA
Mercury	NA	NA	NA	NA	NA	NA
Nitrate	NA	NA	[2] 10	111	NA	NA
Nitrite	NA	NA	[2] 1	11	NA	NA
Selenium	NA	NA	0.045	0.164	NA	NA

NA=Not available. Adequate dose-response data are not available. Assessment derived for the next longer duration of exposure is considered to be protective in these cases.
[1] The longer-term numbers are not discussed in the individual *FR Notice* sections but the rationale for the numbers are discussed in the Health Advisory documents.
[2] Based on a 4 kg infant. For rationale see the Health Advisory for nitrate/nitrite.

TABLE 10.—SHORT-TERM ASSESSMENTS AND PROVISIONAL AADIS FOR IOCS FOR WHICH RMCLS ARE NOT PROPOSED

IOC	1-day (mg/l)		10-day (mg/l)		Provisional AADI (mg/l)	Guidance level (mg/l)
	Child	Adult	Child	Adult		
Aluminum	NA	NA	NA	NA	NA	[4] 0.05
Cyanide	NA	NA	0.22	0.75	0.75	0.75
Molybdenum	2.7	8.0	0.27	0.90	0.10	NA
Nickel	NA	NA	1.0	3.5	0.020	[4] 0.150
Silver	NA	NA	NA	NA	0.090	0.050
Sodium						[5] 20
Sulfate						[4] 400
						[6] 250

NA=Not Available. Adequate dose-response data are not available. Assessment derived for the next longer exposure duration is considered to be protective in these cases.
[4] For high risk individuals on sodium restricted diets. EPA will also prepare secondary standards based upon aesthetic quality.
[5] For protection of the infant as a sensitive subpopulation.
[6] Based on taste and odor.
[7] Based upon problems of pipe precipitation in the distribution system. Aluminum would be a candidate for a secondary standard.
[8] Provisional AADI with data on female exposure factored in.

Table 11 provides a listing of several analytical methodologies for the IOCs and estimated detection limits.

Two method validation studies have been conducted for the furnace atomic absorption (AA) and the inductively coupled plasma (ICP) atomic emission spectrometry techniques. Analytes include all the metals listed in Table 11

analyzable by these techniques. Other performance related data are being gathered from ongoing performance evaluation studies conducted by EPA's Environmental Monitoring and Support Laboratory in Cincinnati, Ohio (EMSL-CIN) for all IOCs included in this RMCL proposal, with the exception of asbestos. These data will be the basis for the analytical methods and performance criteria which will be proposed when the MCLs are proposed.

TABLE 11.—ANALYTICAL METHODS FOR IOC'S

IOC and Methodology	Approximate detection limits (µg/l)	References		
		EPA	16th edition Std. methods	ASTM
Aluminum:				
AA Flame	100.0	202.1	303 C or D	D 857-70 D or E.
AA Furnace	3.0	202.2	304	
ICP	45.0	200.7		
Arsenic:				
AA; gaseous hydride	2.0	206.3	303 E	D 2972-70 B.
AA Furnace	1.0	206.2	304	
ICP	3.0	200.7		
Asbestos: Transmission Electron Microscopy				
Barium:				
AA Flame	100.0	208.1	303 C	
AA Furnace	2.0	208.2	304	
ICP	0.4	200.7		
Cadmium:				
AA Flame	5.0	213.1	303 A or B	D 3557-70 A or B.
AA Furnace	0.1	213.2	304	
ICP	1.0	200.7		
Chromium:				
AA Flame	50.0	218.1	303 A or B	D 1687-77 D.
AA Furnace	1.0	218.2	304	
ICP	0.6	200.7		
Copper:				
AA Flame	20.0	220.1	303 A or B	D 1688-77 D, E.
AA Furnace	1.0	220.2	304	
ICP	0.6	200.7		
Cyanide:				
Distillation; Colorimetric	20.0	335.2	412 B or D	
Automated Electrode	50.0		412 E	
Lead:				
AA Flame	100.0	239.1	303 A or B	D 3559-70 A or B.
AA Furnace	1.0	239.2	304	
ICP	5.0	200.7		
Mercury:				
Manual Cold Vapor Technique	0.2	245.1	303 F	D 3223-79.
Automated Cold Vapor Technique	0.2	245.2		
Molybdenum:				
AA Flame	100.0	246.1	303 C	
AA Furnace	1.0	246.2	304	
ICP	0.6	200.7		
Nickel:				
AA Flame	40.0	249.1	303 A or B	D 1658-77 C or D.
AA Furnace	1.0	249.2	304	
ICP	15.0	200.7		
Nitrate:				
Colorimetric Brucine	100.0	352.1		
Spectrometric; Cadmium Reduction	10.0	353.3	418 C	D 3867-70 B.
Automated Hydrazine Reduction	10.0	353.1		
Automated Cadmium Reduction	50.0	353.2	418 F	D 3867-70 A.
Nitrite: Spectrophotometric	10.0	354.1	419	
Selenium:				
AA; gaseous hydride	2.0	270.3	303 E	D 3859-70.
AA Furnace	2.0	270.2	304	
ICP	1.0	200.7		
Silver:				
AA Flame	10.0	272.1	303 A	D 3886-62 A or B.
AA Furnace	0.2	272.2	304	D 3886-62 C.
ICP	75.0	200.7		
Sulfate:				
Turbidimetric	1,000.0	375.4	428 C	D516-68B.
Colorimetric; Automated	3,000.0	375.2		
Sodium:				
AA Flame	2.0	273.1		
AA Furnace	0.2	273.2		
Flame Photometry	10.0		325 B	D 1428-64 A.

B. Proposed RMCLs

1. Arsenic

Arsenic occurs naturally and is commonly found as arsenic sulphide or as an impurity in various minerals and as arsenate or arsenite. In particular, arsenic is found in ores of copper, lead, zinc, iron, manganese, uranium, and gold. Most of the arsenic produced is a by-product of the smelting of copper, lead, and zinc ores. Areas with elevated levels of arsenic in geologic materials are found throughout the United States, mostly concentrated in the eastern States. Some coals, particularly from eastern States, have a high arsenic content.

Arsenic has been found in both ground water and surface waters; generally ground waters tend to contain higher arsenic levels than surface waters. Arsenic in water can result from both natural processes and industrial activities, including smelting operations, use of arsenical pesticides, and industrial waste disposal.

Analytical Methods. Analytical methods available for analyzing elemental arsenic in drinking water

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include the atomic absorption, gaseous hydride, furnace atomic absorption and inductively coupled plasma atomic emission spectrometry techniques.

Human Exposure. Arsenic enters the atmosphere as a result of the smelting of non-ferrous ores, mostly copper; glass production plants, coal burning facilities, and arsenical compound production plants may also emit arsenic to the air. Data collected by EPA and others show the level of arsenic in air ranges from 0.0005–1.5 ug/m^3; the median value is typically less than 0.01 ug/m^3. Respiratory intake of arsenic on a daily basis is approximately 0.12 ug of which 0.03 ug would be absorbed, assuming 30 percent absorption and based on a 1981 national average air value of 0.006 ug/m^3 of air and a daily ventilation rate of 20 m^3.

Arsenic, primarily in an organic form is a normal constituent of food. Seafoods tend to have particularly high arsenic levels. Meat, fish, poultry, grain, and cereals also contain arsenic. The FDA estimated in its 1979 market basket survey that adults consume 61.5 ug/day of arsenic in the diet.

Sixty-three community water supplies in the United States have been reported to exceed 50 ug/l of arsenic. Limited data from two federal surveys indicate that water supplies using ground water had arsenic present above the detection limit more frequently and at higher concentrations than supplies using surface water. Of 330 ground water supplies sampled in the two surveys, 55 (16.7%) had levels above 5 ug/l; of the 115 surface water supplies, only 2 (1.7%) had levels above 5 ug/l.

Health Effects. Arsenic compounds have been shown to produce acute and chronic toxic effects which include systemic irreversible damage. The trivalent (+3) compounds are the most toxic and tend to accumulate in the body. Chronic animal studies have shown body weight changes, decreased blood hemoglobin, hepatic damage and kidney damage.

There is evidence that arsenic is an essential element in certain animal species and potentially in humans. Arsenic at low concentrations in the diet enhances some parameters of growth and development in animals and it has been suggested that arsenic may be an essential element for humans (NAS, 1980. *Drinking Water and Health,* Vol. III).

EPA's Risk Assessment Forum is currently evaluating the nutritional requirements for arsenic. A comprehensive literature search examining the available data on the possible essentiality of arsenic is being carried out.

The NAS (*Drinking Water and Health,* 1983. Vol. V) reevaluted the toxicity of arsenic and stated that,

> Research should also be designed to evaluate the possible essentiality of arsenic for humans—a requirement that has been demonstrated in four mammalian species. In the absence of new data, the conclusion reached in the third volume of *Drinking Water and Health* remains valid, i.e. 'If 0.05 mg/kg of dietary [total] arsenic is also a nutritionally desirable level for people, then the adequate human diet should provide a daily intake of approximately 25 to 50 ug. The current American diet does not meet this presumed requirement' (National Research Council, 1980). The unresolved status of this issue is further reason for maintaining the current MCL for arsenic.

In addition, the NAS Safe Drinking Water Committee stated that "it is therefore the opinion of this committee that 0.05 mg/l provides a sufficient margin of safety. . . ." Based on the specific recommendations of the NAS, EPA proposes that all Health Advisories for arsenic be 0.05 mg/l.

A provisional AADI was calculated based upon an animal study (Heywood, R. and R.J. Sortwell, 1979. Arsenic Intoxication in the Rhesus Monkey. Toxicol. Lett. 3:137–144) in which adolescent and infant Rhesus monkeys were exposed to arsenic for one year. A NOAEL of 3.74 mg/kg/day arsenate (2.8 mg As/kg/day) was selected, with an uncertainty factor of 1000 and consumption of 2 liters of water per day factored in. A provisional AADI of 0.10 mg/l was determined from this study.

Arsenic has been shown to be mutagenic in several test systems and to induce chromosomal aberrations in *in vivo* and *in vitro* systems. Carcinogenicity studies with laboratory animals have reported conflicting results. Several studies have reported an increased incidence of bronchiogenic carcinomas in rats exposed to an arsenic-containing pesticide through intratracheal exposure. In humans, tumors of the skin, lungs, genital organs and visual organs have been associated with arsenic exposure.

The IARC evaluated arsenic and stated that there is evidence that arsenite and arsenate cross the placenta in mammals and that sodium arsenate and arsenite have embryolethal effects and teratogenic potential in several mammalian species. The IARC classified arsenic compounds in Group 1; inadequate evidence for carcinogenicity in animals and sufficient evidence that inorganic arsenic compounds are skin and lung carcinogens in humans. They also stated that the data suggesting an increased risk for cancer at other sites are inadequate for evaluation. Arsenic has been classified in EPA's Group A, according to EPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon evidence of human carcinogenicity through inhalation and ingestion exposure.

EPA's Office of Research and Development commissioned a report (Andelman, et al. 1983. Feasibility Study to Resolve Questions of the Relationship of Arsenic in Drinking Water to Skin Cancer. Submitted to Office of Research and Development, U.S. EPA) to examine the available epidemiologic studies which looked at the relationship between arsenic exposure and skin cancer in the United States. The conclusion of the report was that the studies performed to date lacked sufficient power to definitively determine if arsenic causes skin cancer. However, the report stated that the precursors of skin cancer (increases in skin pigmentation and callous formation) were not seen in these studies and these precursor effects would normally be seen in cases of arsenic-induced skin cancer.

Based upon the specific recommendations of the NAS, EPA proposes that the RMCL for arsenic remain at the current MCL of 0.050 mg/l. This level is below the concentration at which toxicity is demonstrated and is in the range which may be essential for humans (*Drinking Water and Health,* 1983. Vol. V).

EPA considered using 0.10 mg/l as the proposed RMCL, based upon the provisional AADI. It was determined that the RMCL should not be based upon this data, because an insufficient number of animals (4 per dose group) were studied.

Zero or some other value for the RMCL based upon the carcinogenic potential for arsenic was also considered. However, studies of drinking water related arsenic exposure have not detected increased risks via drinking water in the USA. Because evidence suggests that arsenic may be an essential element, setting the RMCL at zero would not take into account the possible beneficial effects from arsenic exposure.

The WHO guideline for arsenic is 0.050 mg/l which is the same level that EPA is proposing for the RMCL. This value was based upon human data which suggests that a concentration of 50 µg/l of arsenic per liter is not associated with any adverse health effects. In addition, the WHO stated that at an arsenic concentration of 50 µg/l, the contribution made by water to the daily intake will normally be about

one-half to two-thirds and for very low dietary intakes of arsenic, the proportion provided by water may be somewhat higher. The WHO concluded that arsenic in drinking water will normally be the main source of inorganic arsenic.

EPA's CAG has calculated excess cancer risk values for arsenic (U.S. EPA. 1980. Ambient Water Quality Criteria for Arsenic. EPA 440/5-80-02). CAG calculated that concentrations of 22 ng/l, 2.2 ng/l, and 0.22 ng/l would result in an incremental increase of cancer risk over the lifetime at 10^{-5}, 10^{-6} and 10^{-7}, respectively. These values were calculated based upon a study of increased incidence of skin cancer in humans in Taiwan (Tseng, et al. 1968. Prevalence of Skin Cancer in an Endemic Area of Chronic Arsenicism in Taiwan. Jour. Natl. Cancer Inst. 40:453).

EPA is proposing to regulate arsenic because of the potential adverse health effects and its widespread occurrence. Based upon the NAS recommendations, an RMCL of 0.050 mg/l is proposed. Comments are requested on all of the issues outlined below, and comments are specifically requested on the appropriate level for the RMCL.

Questions for Comment:

1. How should the proposed nutritional essentiality of arsenic be considered in determining the RMCL?

2. Is 0.050 mg/l an appropriate level for the RMCL, or should the provisional AADI (0.10 mg/l) or another value be used?

3. Should arsenic's carcinogenic potential alone be used in setting the RMCL?

2. Asbestos

Asbestos is a commercial term applied to a group of highly fibrous, hydrated silicate minerals. These minerals separate into long, thin, strong fibers that are heat resistant and chemically inert, and which possess sufficient flexibility to be woven. Asbestos minerals belong to the serpentine or amphibole groups. The amphiboles are further divided into the orthorhombic crystal system and the monoclinic crystal system. Of the commercially mined and processed asbestos minerals, chrysotile (serpentine group) accounts for 95 percent of production.

There are literally thousands of recorded uses of asbestos minerals in the U.S. The major uses, however, are in production of cement products, floor tiles, paper products, paint, and caulking; in transportation-related applications; and in the production of textiles and plastics.

Analytical Methods. The analytical determination of asbestos in drinking water presents a unique problem. Asbestos is a generic designation for a group of minerals of various compositions. Therefore, the accurate determination of asbestos requires the identification, characterization and measurement by counting the number of particles possessing the required fibrous shape, crystalline structure and elemental composition. The best existing technique for this purpose consists of separation of fibers and quantitation by transmission electron microscopy and identification by X-ray diffraction. The accuracy of this method is highly sensitive to the quality of the water sample and to the presence of interfering substances. A major drawback is the initial capital outlay of approximately $200,000 for equipment, the analytical cost of approximately $500 per sample and requirements for specialized facilities and personnel. Thus, monitoring is not considered to be technologically and economically feasible for all types and lengths of asbestos fibers for compliance purposes in public water systems.

Three different approaches have been investigated to develop a simpler, faster and cheaper measurement method for asbestos. These approaches depend upon light scattering properties of particulates (turbidimetric and magnetic alignment-light scattering methods), or surface properties of chrysotile asbestos which is selectively extracted into iso-octane in the presence of a surfactant (two phase liquid separation method). The most promising method is the one based on magnetic alignment-light scattering.

Optical microscopy may be used to screen water samples to measure fibers above a certain length. Fibers with a minimum length of 5 micrometers and about 0.5 micrometers in width can be measured using this method, with the analytical costs estimated at $50–$100 per sample and an initial outlay of approximately $1,500 for an optical microscope. This technique characterizes fiber shapes only and does not discriminate between asbestos and non-asbestos types. However, the technique could be useful for screening to determine which samples should be analyzed more intensively using transmission electron microscopy or for surveillance after the fibers in a water supply had been characterized by x-ray diffraction or transmission electron microscopy.

Human Exposure. The NAS (1984) have reported an air concentration of asbestos of 0.0004 ug/cm³ as typical to an urban dweller, combining indoor and outdoor exposure. The daily exposure through inhalation of ambient air would be approximately 2000 fibers/day at a ventilation rate of 20 m³ for the adult male.

No information was available from the FDA on the occurrence of asbestos in food. Some wines have been shown to contain as much as 64 million fibers of asbestos per liter (MFL).

Levels of asbestos fibers in drinking water have been summarized by EPA (1983) for 409 cities in 47 States, Puerto Rico, and the District of Columbia. The distribution of reported asbestos concentrations is presented below:

Highest asbestos concentration, 10^6 fibers/l	Number of cities	Percentage
Below detection limits	117	29
<1	246	60
1 to 10	26	6
>10	40	10

In 1981, EPA summarized the results of a nationwide sampling for asbestos in drinking water from 109 systems. Samples were taken from a representative point in the distribution system of each utility. Levels above detection of 0.00 MFL were found in twelve of the 109 systems. Levels ranged from 0.365 to 1,071 MFL. These data and other data from various State studies indicate that asbestos occurs in various drinking water supplies across the country as a result of asbestos in the raw water supply or as a result of corrosion of asbestos-cement pipe in the distribution system.

Human exposure to asbestos in drinking water occurs primarily via ingestion but exposure via inhalation can occur as a result of the use of humidifiers and possibly showers. However, data are limited on these sources of exposure.

Health Effects. A wide range of effects have been observed following exposure via inhalation to asbestos particles. This is in contrast to exposure via ingestion where the only effects reported in animals at very high levels were changes in the mucosal lining cells of the ileum and changes in the colon, rectum and small intestine.

Inhalation studies have shown that various forms of asbestos have produced lung tumors and mesotheliomas in laboratory animals. The majority of asbestos ingestion studies have failed to produce carcinogenic effects in animals. The National Toxicology Program (NTP) investigated the carcinogenic potential of the ingestion of amosite and tremolite asbestos in rats. No toxicity or increase in neoplasia was observed in tremolite-exposed rats, while significant increases

in C-cell carcinomas of the thyroid and monocytic leukemia in male rats were observed in the amosite-exposed group.

The NTP concluded that: (1) The biological significance of the C-cell carcinoma in relation to amosite exposure is discounted because of a lack of significance when C-cell adenomas and carcinomas were combined and the positive effect was not observed in the amosite plus preweaning gavage group, and (2) the biological significance of an increased incidence of mononuclear cell leukemia is questionable because of a lack of statistical significance in the amosite group when evaluated using life table analysis.

Recently, NTP has reported the results on the toxicology and carcinogenesis of chrysotile asbestos in F344/N rats (NTP. 1984. Toxicology and Carcinogenesis Studies of Chrysotile Asbestos in F344/N Rats. Draft Report). In this report, the NTP has concluded that "there was some evidence of carcinogenicity" in male rats only that were exposed to one percent intermediate range (IR) chrysotile asbestos in the diet for the lifetime of the animals. This preliminary conclusion was based on the following observations: (1) A significant increase in benign epithelial neoplasms (adenomatous polyps) in the large intestine of IR chrysotile asbestos male rats (9/250; 3.6%) when compared with the incidence of epithelial neoplasms (benign and malignant combined) of the large intestine in the pooled male control groups of all the NTP oral asbestos lifetime studies (3/524; 0.6%), (2) the incidence of similar lesions in the small intestine or glandular stomach of five additional IR chrysotile male rats, and (3) the rare occurrence of epithelial neoplasm lesions in F344/N rats (1/1,727 for male rats and 0/1,777 for female) is standard.

There have been a number of epidemiological studies which showed gastrointestinal cancer to be associated with occupational exposure to asbestos. The consistency of an increased cancer risk at extrathoracic sites and its magnitude, either in absolute or relative terms, is less for cancer at other sites than for lung cancer. Nevertheless, many studies document significant cancer risks at various gastrointestinal sites. Even though these studies document definite evidence of association of observed gastrointestinal cancer risk and elevated lung cancer in workers, the question of whether the observed increased risk of gastrointestinal cancer is due to the ingestion of inhaled asbestos in the occupationally exposed workers is not resolved at this time. The Chronic Hazard Advisory Panel on Asbestos (U.S. CPSC Draft Report, 1983) has stated the following: "Lung cancer and mesothelioma constitute the majority of asbestos produced cancers. The association of these malignancies with asbestos exposure is firmly established. Some other forms of cancer, particularly digestive tract, oral pharyngeal, laryngeal, and kidney have, in some large studies, been found to be increased; there are disagreements among Panel members as to the strength of the evidence associating this group of cancers with asbestos exposure." A report prepared for the Health and Safety Commission of the United Kingdom examined the available evidence on the health effects of inhaled asbestos and concluded, "In particular, there are no grounds for believing that gastrointestinal cancers in general are peculiarly likely to be caused by asbestos exposure. The increase in relative risk for gastrointestinal sites is similar to that for other sites, and their selection for special attention appears to have been dictated largely by the findings in one study and the fact that they are common, so that a given observed relative risk may be statistically significant for these sites but not for others."

The IARC has classified asbestos in Group 1; sufficient evidence for carcinogenicity in animals and humans. This classification is based upon inhalation data. Asbestos has been classified in EPA's Group A, according to EPA's Proposed Guidelines for Carcinogen Assessment, based upon evidence of carcinogenicity in humans through inhalation exposure.

Several epidemiological studies have been carried out investigating potential associations between asbestos fibers in drinking water and gastrointestinal cancer. Marsh (Environ. Health Persp. 1983. 53:49–56) reviewed and evaluated thirteen epidemiological studies of ingested asbestos in five areas of the United States and Canada for the risk associated with ingestion of water containing asbestos. He concluded that even though one or more studies found association between asbestos in water supplies and cancer mortality (or incidence) due to neoplasms of various organs, no individual study or aggregation of studies exists that would establish risk levels from ingested asbestos.

EPA's CAG (1980) and the NAS (1983) have extrapolated the results of cohort studies of populations occupationally exposed via inhalation in order to estimate the potential risk associated with the ingestion of asbestos in drinking water. The CAG (U.S. EPA. 1980. Ambient Water Quality Criteria for Asbestos. EPA 440/5-80-022) and the NAS (*Drinking Water and Health*, 1983. Vol. V) considered much the same data of occupationally exposed workers with GI tract cancers, but use a slightly different method of calculating the excess cancer risk values. The estimated levels which would result in increased lifetime cancer risks of 10^{-5}, 10^{-6}, and 10^{-7} calculated by CAG (1980) are 300,000 fibers/1, 30,000 fibers/1 and 3,000 fibers/1, respectively. Corresponding numbers for males calculated by the NAS (1983) are 110,000 fibers/1, 11,000 fibers/1 and 1,100 fibers/1. The more restrictive levels calculated by the NAS compared to CAG are primarily due to the application of two different assumptions:

(1) The NAS assumed that 30 percent of the inhaled fibers were subsequently swallowed, where the CAG assumed that 100 percent would eventually be cleared and ingested.

(2) The NAS assumed a conversion factor of 50 for optical microscopy to transmission electron microscopy, where the CAG assumed a factor of 200.

The available information on the risk of developing gastrointestinal tract cancer associated with the ingestion of asbestos from drinking water is limited. Risk projections based upon ingestion studies would appear to be more appropriate than inhalation for exposures via drinking water. CAG (1984) has derived an estimate of the risk for asbestos by injection based upon a draft NTP (1984, draft report) ingestion study of chrysotile short range (98% <10 um) and intermediate range (65> 10um; with −14%> 100um) fibers in animals. The results of this study showed no evidence of carcinogenicity for the short-range fibers in either male or female rats and no evidence of carcinogenicity for the intermediate range fibers in the female rats. However, there was an increase in benign polyps of the large intestine for the male rats ingesting the intermediate range fibers (>10 um) at 1 percent of the diet. Although not statistically significant compared with the concurrent controls, the incidence of these neoplasms was highly significant when compared with the incidence of epithelial neoplasms (benign and malignant combined) of the large intestine in the pooled control groups of all the NTP oral asbestos lifetime studies. If indeed a cause/effect can be deduced from this experiment, CAG calculated, based upon the one-hit model, that 7.1×10^{7}, 7.1×10^{6} and 7.1×10^{5}

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fibers/l, would result in a lifetime excess cancer risk of 10^{-5}, 10^{-6} and 10^{-7}, respectively. These levels are calculated for the intermediate range chrysotile fibers only. As such, the levels are much more restrictive than they would have been had they incorporated the study using the short range chrysotile fibers. The short range chrysotile fiber study showed no effects with 50 times the number of fibers as the intermediate range study. If the short range study had been included with the data from the intermediate range study had been included with the data from the intermediate range study, levels would have been at least 10 times higher (i.e., less restrictive). Conversely, if the shorter fiber had been eliminated from the positive intermediate fiber study, levels would have been lower by a factor of about 2.5.

While significant efforts have been expended to determine the potential human health risks of exposure to asbestos via drinking water, questions still remain. Summarizing:

- Asbestos has carcinogenic effects via inhalation exposure in humans.
- Animal studies consisting of oral exposure to asbestos were negative except in one experiment in the recently reported NTP study (draft report, 1984).
- Epidemiology studies examining asbestos in drinking water have not found consistent credible correlations between carcinogenic effects and asbestos.
- Evidence exists which shows increased risk of gastrointestinal cancer from inhalation of asbestos fibers during occupational exposure. However, questions have been raised as to whether the gastrointestinal cancer was caused by swallowing inhaled fibers or due to other mechanisms.

EPA's Science Advisory Board (SAB) examined the question of the carcinogenic potential of ingested asbestos in 1984 but without access to the latest NTP report of benign adenomatous polyps in male rats. They concluded:

> Given the positive signal seen in some epidemiologic studies, plus well-documented evidence for the association between asbestos fiber inhalation and lung cancer, it is hard for the Committee to feel comfortable in dismissing the possibility of an increased risk of gastrointestinal cancer in humans exposed to asbestos fibers from drinking water. However, the Committee consensus is that current peer-reviewed evidence for humans and animals does not support the view that asbestos ingested in water causes organ-specific cancers.

The SAB reexamined the issue in 1985, considering whether the increased incidence of benign polyps reported in the NTP study was sufficient to conclude that carcinogenicity was demonstrated under the conditions of the study. The SAB concluded that the data were equivocal and reaffirmed their 1984 conclusions as summarized above.

EPA has considered two main regulatory options for asbestos in drinking water. The first option consists of not proposing a primary regulation for asbestos, due to the inconclusive nature of the data. Animal studies (except for the recently reported NTP study) have not shown a correlation between oral exposure to asbestos and carcinogenic effects and epidemiologic studies examining asbestos in drinking water have not shown a consistent relationship between asbestos and carcinogenic effects. Thus, EPA could conclude that sufficient evidence is not available to demonstrate that asbestos in drinking water is associated with organ-specific cancers and an RMCL is not warranted. As a suboption, EPA could prepare a health advisory based upon the data from the NTP study but it would be in the form of guidance rather than enforceable standard.

The second option consists of proposing an RMCL for asbestos fibers exceeding 10 um in length. The basis for this option is that asbestos has been shown to be a human carcinogen through inhalation exposure and data exists which suggests that asbestos may be associated with an increased risk of gastrointestinal cancer through occupational exposure. The results of the NTP study showed an association in male rats between ingestion of asbestos fibers greater than 10 μm in length and gastrointestinal tumors that may be indicative of carcinogenic effects. In addition, evidence exists to support the association between asbestos fiber length and carcinogenic effects: inhalation exposure to medium and long (>10um) fibers have been shown to result in mesotheliomas in rats while nonfibrous particulates have not been shown to cause tumors. Asbestos would be considered to have equivocal evidence of carcinogenicity in drinking water (Regulatory Category II) and the RMCL would be proposed at the 10^{-6} risk levels (i.e., 7.1×10^{6} fibers/liter). Comment would then be requested on whether there is an available analytical method for asbestos. If so, an MCL for long fibers would be proposed. If it is determined that an analytical method was not available, a treatment regulation would be proposed.

EPA is proposing an RMCL for asbestos (Option #2) at the present time and is soliciting public comment on the asbestos issue. An RMCL is being proposed based upon the widespread occurrence of asbestos in public water supplies, data showing that asbestos is carcinogenic in humans through inhalation exposure, and data showing that fiber lengths greater than 10 μm may be carcinogenic through ingestion exposure. EPA will reconsider this option based upon any new data and the public comments received.

Questions for Comment:

1. Are there sufficient data upon which to regulate asbestos as a possible human carcinogen by ingestion (Category II)?

2. Is there a sufficient basis for EPA to regulate asbestos fibers exceeding 10 μm in length based upon the recent NTP bioassay in male rats?

3. Is the analytical method technologically adequate to determine the level of asbestos fibers >10 μm in drinking water?

4. The RMCL for asbestos fibers exceeding 10 μm in length is proposed based upon the 10^{-6} risk level, as calculated by CAG from animal ingestion studies which considered the association between fiber length and carcinogenicity. The RMCL could also be based upon the risk levels derived by CAG from cohort studies of populations occupationally exposed via inhalation. These risk levels derived from occupational exposure do not consider fiber size in the analysis. Which of these risk calculations are more appropriate as the basis for the RMCL?

3. Barium

Barium is a naturally occurring metal found in many types of rock. Limestones, sandstones, and soils in the eastern United States may contain 300–500 ppm barium. Certain geologic formations in California, Arkansas, Missouri, and Illinois are known to contain barium levels about 1,000 times higher than those found in other portions of the United States. Areas associated with deposits of coal, petroleum, natural gas, oil shale, black shale, and peat may also contain high levels of barium. Principal areas where high levels of barium have been found in drinking water include parts of Iowa, Illinois, Kentucky, and Georgia.

The environmental release of barium is also associated with oil and gas drilling muds, coal fired power plants, fillers for automotive paints and specialty compounds used in bricks, tiles, and jet fuels.

Analytical Methods. Analytical methods available for analyzing barium in drinking water include the flame atomic absorption, furnace atomic absorption and inductively coupled

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plasma emission spectrometry techniques.

Human Exposure. Barium is present in ambient air from combustion of diesel fuel, aviation fuel, coal, as well as mining refining and manufacturing. Data collected by EPA show the mean level of barium in air ranges from 0.0015–0.95 $\mu g/m^3$. Medians and mean values for individual sites have generally been shown to be below 0.4 $\mu g/m^3$, which would result in an adult male having an approximate respiratory intake of 0.03–22 μg/day. The International Commission on Radiological Protection (ICRP) (1974) estimates respiratory intake to be 0.09–30 μg/day.

Little data are available on the level of barium in the U.S. food supply. It has been reported that nuts contain high levels of barium, with 1000 ppm in pecans and up to 10,000 ppm in brazil nuts. Studies on four individuals indicated the dietary intake of barium ranged from 440–1800 μg/day. The "average" value of 900 μg/day reportedly includes intake from beverages. The ICRP reports an "average" daily dietary intake of 750 μg/day for an adult male from food and fluids, of which 80 μg/day comes from drinking water. Based on these data, the diet contributes approximately 670 μg barium to the adult human intake each day.

Compliance monitoring indicates that 43 community water supplies in the United States contain more than 1 mg/l of barium (the current interim standard). Data on 132 ground water systems assembled between 1969–1980 show that approximately 14 percent of those systems contained levels of barium greater than 250 μg/l and 1–2 percent were over 1000 μg/l; data from surface water systems indicated that 14–15 percent of 28 systems contained levels of barium greater than 250 μg/l but no levels above the 500 μg/l level were found.

Health Effects. Acute exposure to barium in animals and humans results in a variety of cardiac, gastrointestinal and neuromuscular effects. Inadequate data were available to calculate shortterm assessments for barium.

The role of waterborne barium in the etiology of experimental and human "essential" hypertension remains a matter of controversy and conjecture. A provisional AADI was calculated based upon an experimental study showing chronic exposure to barium resulting in hypertension in rats (Perry, H.M., et al. 1983. Cardiovascular Effects of Chronic Barium Ingestion. In: Hemphill, D.D., ed. Proceedings of the 17th Annual Conference on Trace Substances in Environmental Health. Vol 17. Columbia, MO, University of Missouri Press, pp. 155–164). Exposure to 100 mg/l barium in drinking water for 1 to 16 months produced hypertensinogenic and cardiotoxic effects in rats. A provisional AADI of 1.8 mg/l was calculated from a LOAEL of 100 mg/l barium (5.1 mg/kg/day) with an uncertainty factor of 100, and assuming consumption of 2 liters of water per day. An uncertainty factor of 100 was applied, instead of the traditional 1000-fold uncertainty factor used with a LOAEL, based upon the minimized exposure of the rats to trace metals (i.e., calcium) in the experiment. This lack of calcium could contribute to the hypertensinogenic effects observed.

The current MCL for barium, under the National Interim Primary Drinking Water Regulations, is 1 mg/l. This value was based upon the threshold limit value (TLV) of 0.5 mg/m³ air, assuming that 75 percent of the barium inhaled is absorbed into the blood stream and that 90 percent is absorbed via the gastrointestinal tract.

In 1982, the NAS (*Drinking Water and Health*, 1982. Vol. IV) derived a revised chronic suggested no-adverse-response level (SNARL) for barium of 4.7 mg/l. This value was derived based upon the TLV of 0.5 mg/m³ air and assuming a 20 percent gastrointestinal absorption rate for barium in the adult human. However, animal studies indicate that young rats absorb up to 65 percent of an administered oral dose of barium compared to 7 percent absorption in adult rats. Thus, 4.7 mg/l does not appear to be applicable to the general population which would include young children who would also have an enhanced uptake efficiency over adults.

An epidemiological study of communities in Illinois demonstrated that male and female adults living in the high-barium (7.3 mg/l drinking water) community for more than ten years did not manifest significant differences in mean systolic/diastolic blood pressures (Brenniman, G.R., et al., 1981. High Barium Levels in Public Drinking Water and its Association with Elevated Blood Pressure. Arch. Environ. Health 36:28–32). However, several factors in this study have been questioned, such as confounding variables, uncontrolled parameters and data inconsistencies.

The IARC have not evaluated barium for possible carcinogenic effects. Barium has been classified in EPA's Group D, according to EPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon inadequate data from animal studies.

Barium exposure has been associated with hypertension and cardiotoxicity in animals and a provisional AADI of 1.8 mg/l has been determined. For this reason and because of the widespread occurrence of barium in drinking water, EPA is proposing to continue to regulate barium. Factoring in data on human exposure (0.7 mg/day via the diet and 0 mg/day via air), the RMCL is being proposed as 1.5 mg/l. This value is five-fold lower than the barium level (7.3 mg/l) which Brenniman, et al. (1981) found to produce no effects on blood pressure in humans. It is also lower than the chronic SNARL of 4.7 mg/l derived by NAS (1982). Thus, this RCML of 1.5 mg/l contains a several-fold safety factor and should be sufficiently protective against adverse effects.

Questions for Comment:

1. Is it appropriate to base the RMCL on the hypertensinogenic and cardiotoxic potential of barium in animals?

2. Should the epidemiological study by Brenniman, et al. (1981) be used for derivation of the RMCL without additional safety factors?

3. Should the chronic SNARL value for barium of 4.7 mg/l be used as the RMCL?

4. Cadmium

Cadmium is found in very low concentrations (usually >1 ppm) in most rocks, as well as in coal and petroleum and often in combination with zinc. Geologic deposits of cadmium can serve as sources to ground water and surface water, especially when in contact with soft, acidic waters.

Cadmium uses include electroplating, nickel-cadmium batteries, paint and pigments, and plastic stabilizers. It is introduced into the environment from mining and smelting operations and industrial operations, including electroplating, reprocessing cadmium scrap and incineration of cadmium-containing plastics. The remaining cadmium emissions are from fossil fuel use, fertilizer application and sewage sludge disposal. Cadmium may enter drinking water as a result of corrosion of galvanized pipe. Landfill leachates are also an important source of cadmium in the environment.

Analytical Methods. Analytical methods available for analyzing cadmium in drinking water include the flame atomic absorption, furnace atomic absorption and inductively coupled plasma atomic emission spectometry techniques.

Human Exposure. Cadmium is introduced into the atmosphere by both natural and anthropogenic means. Higher levels are found in areas where non-ferrous metal mining, smelting and refining occur. Also, cadmium levels in air are higher in urban than in rural

areas. Cigarettes contain high levels of cadmium, and thus tobacco smokers have an increased respiratory intake of cadmium. Data collected by EPA show mean levels of cadmium in air to typically range from 0.0005–0.01 μg/m³. Using 0.01 μg/m³ as a typical, high value and 20 m³/day as the ventilation rate, the respiratory intake for the adult male would be up to 0.20 μg/day.

Cadmium is present in low levels in most foods. The FDA examined 481 samples of 12 raw agricultural commodities and found cadmium to be present at or above trace (0.02 ppm) quantities in at least one sample of each product. Soybeans, peanuts, beans, wheat, and lettuce were found to have a high frequency of samples with elevated levels (>0.15 ppm). Shellfish are noted for high cadmium levels, reportedly ranging from 0.7–7.8 ppm. The FDA has estimated that daily dietary cadmium intake for the adult male is 27.2 μg (excluding beverages). Grains and cereals, potatoes, meat, fish and poultry were identified as major food categories contributing to cadmium intake.

Compliance monitoring indicates that there are currently 25 public water supplies with reported levels of cadmium above 10 μg/l, the current standard. Data on 757 ground water supplies obtained in federal surveys conducted between 1969–1980 show that about 27 percent have levels above 2 μg/l; then mean of the positives was about 3 μg/l. In the same federal survey, 19.7 percent of 117 surface water supplies had levels above 2.0 μg/l; the mean of the positives was 3.2 μg/l. None were found to exceed 10 μg/l.

Health Effects. Acute and chronic exposure to cadmium in animals and humans results in renal dysfunction, hypertension, anemia and altered liver microsomal activity. The kidney is considered to be the critical target organ in humans chronically exposed to cadmium by ingestion. The early clinical signs of renal injury include proteinuria, glucosuria and aminoaciduria.

A human study (Lauwerys, 1979. Cadmium in Man. In: Webb, ed. The Chemistry, Biochemistry and Biology of Cadmium. Elsevier/North Holland Biomedical Press, pp. 439–453) was selected for the derivation of short-term assessments for children and adults. From a NOAEL of 0.043 mg/kg/day based on the emetic effects following acute exposure in adult humans, with an uncertainty factor of 10, and assuming consumption of 1 liter (10 kg child) or 2 liters (70 kg adult) of water per day, 1-day assessments for a 10 kg child and 70 kg adult of 43 μg/l and 130 μg/l were determined, respectively

An animal study (Kotsonis and Klaassen, 1978. The Relationship of Metallothionein to them Toxicity of Cadmium after prolonged Oral Administration to Rats. Toxicol. Appl. Pharm. 46:39–54) was used for the derivation of 10-day assessments for children and adults. From a NOAEL of 0.08 mg/kg/day based on proteinuria following up to 24 weeks of cadium feeding in rats, with an uncertainty factor of 1,000 (an additional uncertainty factor of 10 was used due to the number of animals (6 rats) per dose group), and assuming consumption of 1 liter (10 kg/child) or 2 liters (70 kg/adult) of water per day, 10-day assessments for a 10 kg/child and a 70 kg/adult of 8 μg/l and 20 μg/l, respectively were calculated. These values would be very conservative because of the extended length of the test (24 weeks). These values would be considered protective of the child and adult for a one-day exposure.

A provisional AADI was calculated using renal dysfunction as an endpoint. The critical (threshold) concentration of cadmium in the renal cortex associated with renal dysfunction has been calculated to range from 50 μg/g to 300 μg/g. A value of 200 μg/g (Friberg, L., et al. 1974. Cadmium in the Environment, 2nd ed. Boca Raton, Florida: CRC Press Inc.; Kjellstrom, et al. 1984. Conceptual Problems in Establishing the Critical Concentration of Cadmium in Human Kidney Cortex. Env. Res. 33:264–285) has been selected as the basis of the NOAEL in the calculation of the provisional AADI. This value is probably the most widely accepted estimate of the critical concentration for renal dysfunction. Several models have been proposed to estimate the daily intake of cadmium required to reach the critical concentration in the renal cortex. Using a conservative model (Friberg, L., et al., 1974) which assumes 4.5 percent absorption of the daily oral dose and 0.01 percent excretion of the total body burden per day, a daily intake of 0.352 mg/day cadmium will be used as the LOAEL for renal effects in humans. A provisional AADI of 0.018 mg/l has been calculated using an uncertainty factor of 10 and consumption of 2 liters of water per day.

Cadmium and cadmium compounds have been shown to induce sarcomas at injection sites in animals when administered parenterally and cadmium chloride given by aerosol for 18 months can produce lung tumors in rats. The IARC have classified cadmium and certain cadmium compounds in Group 2B: limited evidence for carcinogenicity in humans, sufficient evidence for carcinogenicity in animals and inadequate evidence for activity in short-term tests. The IARC classification is based on exposure to cadmium via inhalation. However, no evidence has been found linking ingestion of cadmium with carcinogenicity in animals or humans. Cadmium has been classified in EPA's Group B1, according to EPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon evidence of carcinogenicity in humans through inhalation exposure. However, since cadmium has not been shown to be carcinogenic through ingestion exposure, the compound will be regulated based upon chronic toxicity data.

The NAS (*Drinking Water and Health*, 1982, Vol. IV) estimated a SNARL for cadmium of 0.005 mg/l based upon the same study which was used to calculate the 10-day assessment values (Kotsonis and Klaassen, 1978. The Relationship of Metallothionein to the Toxicity of Cadmium after Prolonged Oral Administration to Rats. Toxicol. Appl. Pharmacol. 46:39–54).

The current MCL for cadmium, under the National Interim Primary Drinking Water Regulations, is 0.010 mg/l. This level was based upon a cadmium level of 200 ppm in the renal cortex association with proteinuria, with 5 percent gastrointestinal absorption, rapid excretion of 10 percent of the absorbed dose and 0.05 percent daily excretion of the total body burden. It was estimated that it would take about 500–600 μg/day to cause proteinuria and with assumed diet of 75 μg/day, a drinking water level of 0.010 mg/l would provide a four-fold safety factor. The provisional AADI was also determined based upon renal dysfunction as an endpoint, but newer data were used to determine the critical concentration in the renal cortex and the daily intake of cadmium required to reach this critical concentration.

The WHO guideline for drinking water is 0.005 mg/l. This value was derived based upon the provisional, tolerable weekly intake of 0.4–0.5 person, established in 1972 by the Joint FAO/WHO Expert Committee on Food Additives. The WHO determined that a guideline value of 0.005 mg/l was suitable based upon the fact that at this concentration about one-quarter of the total cadmium absorbed might be derived from water. EPA's ambient water quality criteria for human health (U.S. EPA. 1980. Ambient Water Quality Criteria for Cadmium. EPA 440/5–80–025) is 0.010 mg/l considering ingestion of water and contaminated aquatic organisms. This value was based upon

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an analysis which concluded that water constitutes only a relatively minor portion of the total cadmium intake and thus the existing drinking water standard of 0.010 mg/l was determined to provide ample protection for human health.

Because of cadmium's potential adverse health effects and widespread occurrence in raw waters, EPA is proposing to continue to regulate it in the NPDWR. The NAS and the WHO have determined guidelines of 0.005 mg/l cadmium. A provisional AADI of 0.018 mg/l has been calculated. An RMCL of 0.005 mg/l is proposed based upon the NAS and WHO guidelines which would result in a theoretical allocation to drinking water of approximately 25 percent of the ADI.

Questions for Comment:

1. Is it appropriate to use a value of 200 μg/g as the critical concentration of cadmium in the human renal cortex?

2. Is the model used to estimate the daily intake of cadmium required to reach the critical concentrations in the renal cortex based upon reasonable assumptions?

3. In the drinking water criteria document on cadmium, an alternate study is cited (Perry, et al., 1977b, 1979) for the derivation of the RMCL. Comments are requested on the use of this study in the possible derivation of the RMCL.

4. Is the use of 25 percent drinking water contribution reasonable in determining the RMCL?

5. Chromium

Chromium is a naturally occurring metal that in drinking water forms compounds with valences of +3 and +6, with the trivalent state being the more common. The average chromium content of the earth's crust has been estimated to be 40 ppm, with a range of 10–200 ppm for most geologic materials. Chromium levels of 1,000–12,800 ppm have been reported for certain ultramafic rocks that are high in iron and magnesium and low in silica. The only commercial chromium ore is chromite, which occurs in small deposits in Washington, Oregon, California, Montana, Wyoming, Texas, Pennsylvania, Maryland, North Carolina, and Georgia.

Although chromium is not currently mined in the U.S., wastes from old mining operations may enter surface and ground water through runoff and leaching. Chromate wastes from plating operations may also be a source of water contamination. Fossil fuel combustion, waste incineration, cement plant emissions, chrome plating and other metallurgical and chemical operations may result in releases of chromium to the atmosphere.

Analytical Methods. Analytical methods available for analyzing chromium in drinking water include the flame atomic absorption, furnace atomic absorption and inductively coupled plasma atomic absorption emission spectrometry techniques.

Human Exposure. Data from EPA for the years 1977 to 1981 show that ambient air at most stations has mean values below 0.3 $\mu g/m^3$ (24-hour averages) and medium values below 0.2 $\mu g/m^3$. Twenty-four hour average concentrations in the U.S. for total chromium generally range between 0.005 and 0.187 $\mu g/m^3$. Maximum annual average ambient (total) chromium levels within 20 kilometers of emission sources have been predicted to range from 0.01 to 13.5 $\mu g/m^3$.

Nearly all foods contain some chromium, ranging from 20 to 590 μg/kg. The largest sources are meats, mollusks, crustaceans, vegetables and unrefined sugar. Dietary intake in humans has been estimated to range from 5 to 500 μg/day, with a typical value of approximately 100 μg/day.

Compliance monitoring data indicate that 17 ground water and one surface water supply have provided drinking water with levels greater than 50 μg/l. Twelve of the ground water supplies are small systems serving fewer than 500 people; only 3 serve more than 10,000 people and none serve more than 75,000 people. However, the one surface water supply serves more than 100,000 people.

In three national surveys conducted between 1969 and 1980, chromium was found to be present at levels above 5 μg/l in 77 of 795 ground water supplies sampled (9.7%). The mean of the positive values was 16 μg/l, with values ranging up to 49 μg/l. In surface water supplies, chromium was found at levels above 5 μg/l in 24 of 142 systems sampled (16.9%). The mean of the positives was 10 μg/l; none of the surface water supplies had values above 25 μg/l.

Health Effects. Chromium III (Cr III) and chromium VI (Cr VI) have greatly differing toxicity characteristics. Chromium III is a nutritionally essential element; the NAS (*Drinking Water and Health*, 1980, Vol. III) have estimated an adequate and safe intake level for adults of 0.05 to 0.20 mg/day via the diet. Chromium VI is much more toxic than Cr III and has been shown to produce liver and kidney damage, internal hemorrhage, and respiratory disorders. Also, subchronic and chronic exposure to Cr VI in the form of chromic acid can cause dermatitis and ulceration of the skin.

There are no suitable data available for calculating one-day assessment values for chromium III or chromium VI. However, the ten-day assessment for chromium VI of 1.4 mg/l for the 10 kg child and 5.0 for the 70 kg adult will be protective for one-day exposures.

An animal study (Gross and Heller, 1949, Chromates in Animal Nutrition, J. Ind. Hyg. Toxicol. 28:52–56) was selected to serve as the basis for calculations of the Chromium VI 10-day assessment for the 10 kg child and 70 kg adult. From a NOAEL of 14.4 mg/kg/day based on no-effects in rats following a 60-day exposure, with an uncertainty factor of 100, and assuming consumption of 1 liter (10 kg child) or 2 liters (70 kg adult) of water per day, ten-day assessments for the child and adult of 1.4 mg/l and 5.0 mg/l were calculated.

Suitable data were not available for calculating chromium III 10-day assessments for the child and the adult.

A provisional AADI for chromium (through the oral route of exposure) was determined on the basis of the effects of Cr VI and a separate AADI was not determined for Cr III for the following reasons:

1. Cr III and Cr VI are in dynamic equilibrium, the degree of oxidation in an aqueous medium depending on factors such as pH, dissolved oxygen, or presence of reducing agents. In ambient water (as opposed to sediments) there is slow oxidation of Cr III to Cr VI. The rate of this oxidation is accelerated by the addition of an oxidizing agent (MnO_2). It has been shown that water treatment involving chlorination will effectively transform Cr III to Cr VI. The normal presence of residual oxidizing capacity in treated water throughout the water distribution system will assure maintenance of dissolved chromium in the oxidized state; therefore, if chromium is present, drinking water at the point of consumption (i.e., the tap) is likely to contain substantial amounts of Cr VI.

2. Cr VI is more toxic than Cr III, since only the hexavalent species readily crosses cell membranes. The nonmutagenicity of Cr III, as contrasted with the mutagenicity of Cr VI, can be directly attributed to this fact, even though Cr III reacts with DNA in the cells. An AADI based on Cr VI, therefore, will be conservative with respect to any Cr III which might be present in water.

3. Even though orally ingested Cr VI is reduced in part to Cr III during passage through the stomach, reduction is incomplete, and there is greater absorption and greater tissue

accumulation of chromium following ingestion of Cr VI than Cr III.

A study in which rats were supplied drinking water containing up to 25 mg/l Cr VI for one year was used to calculate a provisional AADI (MacKenzie, R.D., et al. 1958. Chronic toxicity studies. II. Hexavalent and Trivalent Chromium Administered in Drinking Water to Rats. AMA Arch. Ind. Health 18:232-234). This study resulted in a NOAEL of 2.41 mg/kg/day. Using this NOAEL, an uncertainty factor of 500 based upon an animal study in which the rats were exposed to Cr VI for only 40 percent of a normal span, and consumption of 2 liters of water per day, a provisional AADI of 0.17 mg/l was determined.

The IARC have classified chromium and certain chromium compounds in Group 1 (chromium VI); sufficient evidence for carcinogenicity in humans and animals. The IARC classification is based on inhaled chromium VI. Chromium has been classified in EPA's Group A, according to EPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon positive inhalation data for chromium VI in humans and animals. However, since chromium has not been shown to be carcinogenic through ingestion exposure, the compound will be regulated based upon chronic toxicity data.

The current MCL for total chromium, under the National Interim Primary Drinking Water Regulations, is 0.05 > mg/l. This value was based upon an analysis which concluded that 0.05 mg/l chromium incorporates a reasonable factor of safety to avoid any hazard to human health.

The WHO guideline for drinking water is 0.05 mg/l for chromium, as Cr VI and also total chromium. This value was based upon the 1970 European standards for drinking water. EPA's ambient water quality criteria for human health (U.S. EPA. 1980. Ambient Water Quality Criteria for Chromium. EPA 440/5-80-035) considering ingestion of water and contaminated aquatic organisms for Cr III and Cr VI are 170 mg/l and 0.050 mg/l, respectively. These values have been updated (February 1984. 49 FR 4551) and the proposed revised values are 30 and 0.050 mg/l for Cr III and Cr VI, respectively. These values are based upon dose dependent reductions in organ weights of the liver and spleen for chromium III and the observation that the standard appears, through past experience, to be satisfactorily protective against chromium VI toxicity in humans.

Chromium exposure at high levels has been shown to result in chronic toxic effects in animals and humans by ingestion. EPA is proposing to continue regulation of this contaminant because of the potential adverse health effects and its widespread occurrence. An RMCL of 0.12 mg/l is proposed for total chromium (Cr III and Cr VI) in drinking water, based upon a provisional AADI of 0.17 mg/l with data on human exposure factored in (0.10 mg/day via the diet and 0 mg/day via air).

Question for Comment:

1. Should the RMCL calculated for chromium be based on total chromium (Cr III and Cr VI), or should separate RMCLs for the two valence states be calculated? Are the available analytical methods sensitive enough to differentiate readily between the two valence states?

6. Copper

Copper is ubiquitous in the earth's crust, occurring commonly as sulfides and oxides and occasionally as metallic copper. Weathering and dissolution of these natural copper minerals results in background levels of copper in natural surface waters at concentrations generally below 20 μg/l. The principal sources in drinking water include corrosion of brass and copper pipe by acidic waters, and other sources include use of copper salts as aquatic algicides, industrial effluents, atmospheric fallout and sewage treatment plant effluents. The major industrial sources include smelting and refining industries, copper wire mills, coal burning industries and iron and steel producing industries. Copper may enter natural water either directly from these sources or by atmospheric fallout of air pollutants produced by these industries.

Analytical Methods. Analytical methods available for analyzing copper in drinking water include the flame atomic absorption, furnace atomic absorption and inductively coupled plasma atomic emission spectrometry techniques.

Human Exposure. The principal source of elevated copper levels in air is copper dust generated by copper-processing operations. Coal-burning power plants are another source of copper emissions and tobacco smoke is a possible source.

In 1983, a National Air Sampling Network survey showed that the airborne copper concentrations were 0.01 and 0.257 μg/m³ in rural and urban communities, respectively. Even near copper smelters, where high levels (1 to 2 μg/m³) are reached, the dose of metal that would be acquired through inhalation of ambient air would comprise only about 1 percent of the total normal daily intake.

Copper is found in certain foods including shellfish (especially oysters) and organ meats (liver, heart, beef, or swine liver). Nuts, dried legumes, dried vine and stone fruits and cocoa are particularly rich in copper. The copper content of these items can range from 10 μg/g to as high as 400 μg/g. Dairy products, white sugar, and honey rarely contain more than 0.5 μg copper/g. The nonleafy vegetables and most fresh fruits and refined cereals generally contain up to 2 μg/g. Cheese, milk, beef, mutton, white and brown bread and many breakfast cereals (unless they are fortified) are relatively poor sources of copper, i.e., they have less than 50 μg copper/100 kcal. The refining of cereals for human consumption results in significant losses of copper, although this loss is not so severe as it is for iron, manganese and zinc.

A daily copper intake of 2 mg is considered to be adequate for health and normal copper metabolism. The normal daily adult intake of copper from food in the U.S. is reported to range from 2.0 to 4.0 mg per day. The reported average intake of copper in young children is 1.5 mg per day; the minimum dietary requirement is 0.10 μg/kg of body weight per day.

Water can be a significant source of copper intake depending upon the geographical location, the character of the water (i.e., hardness, pH, alkalinity), the temperature of the water and the presence of copper-containing pipes.

In 1957, the U.S. Department of Interior published the results of a five-year study of 350 finished drinking water systems. Analysis of results showed levels of copper to range from 1 to 1,000 μg/l, with a mean of 43 μg/l. In 1970, the U.S. Department of Health, Education and Welfare reported the average concentration of copper in drinking water supplies to be 134 μg/l, with a maximum concentration of 8,350 μg/l. Results of a survey of 83 water supplies by EPA Region V showed copper to range from < 5.0 to 200 μg/l in finished drinking water.

Higher copper levels have been observed in tissue samples of residents of cities with soft water. This might be due to corrosion of copper pipes and fittings, thereby increasing the intake of soluble copper. Another explanation may lie in the ability of calcium or magnesium ions in hard water to suppress the intestinal absorption of copper.

Health Effects. Copper has toxic effects at high dose levels and is an essential element at lower levels. Toxic effects resulting from acute exposure to copper in laboratory animals and

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humans include gastrointestinal disturbances, hemolytic anemia, renal damage, liver damage and glucose-6-phosphate dehydrogenase inhibition. Limited data are available on the chronic toxicity of copper. Efficient homeostatic mechanisms generally protect mammals from the adverse effects of dietary copper excess. In humans, individuals with Wilson's disease are at additional risk from the toxic effects of copper. Wilson's disease is an inborn error of copper metabolism in which copper accumulates in the liver, brain and kidney, resulting in hemolytic anemia, neurological abnormalities and corneal opacities. In addition, individuals with glucose-6-phosphate dehydrogenase (G6PD) deficiency are likely to be at increased risk to the toxic effects of copper.

Copper is regarded as an essential element in mammalian nutrition because it is required in many enzymatic reactions. Copper deficiency can result in decreased iron absorption and iron deficiency, and may also lead to reproductive abnormalities. The NAS (*Drinking Water and Health*, 1980, Vol. III) has recommended an adequate and safe intake level of 2-3 mg/day copper.

One-day assessments of 1.3 mg/l for the adult and the child were determined based upon a LOAEL of 5.3 mg/day based upon human clinical case studies (Chuttani, H.R., et al. 1965. Acute Copper Sulphate Poisoning. Am. J. Med. 39:849) in which 5.3 mg was the lowest oral dose at which gastro-intestinal effects were seen. An uncertainty factor of two was applied for the following reasons: (1) The effect noted was local gastrointestinal irritation and was not permanent, (2) 5.3 mg was the lowest value determined in the literature based upon a number of studies and thus is very conservative number, (3) copper is an essential element and the application of a larger uncertainty factor would bring the level below that considered necessary for human nutrition and, (4) copper absorption is controlled by a homeostatic mechanism and the compound does not tend to accumulate in the body. Ten-day values were not calculated due to inadequate data.

A provisional AADI was determined for copper based upon the compound's acute toxicity effects, since these are the effects of concern from exposure to copper. The same LOAEL and uncertainty factor used to derive the 1-day assessments were used to determine a provisional AADI, resulting in value of 1.3 mg/l.

Copper compounds have generally provided negative results in microbial mutation assays. Copper sulfate was observed to increase the frequency of recessive lethal mutations in *D. melanogaster* at high concentrations. Equivocal results have been obtained from carcinogenicity studies. Administration of copper compounds to mice by subcutaneous injection has been reported to induce tumor formation. Orally administered copper compounds were not found to increase tumor incidence in several studies. The IARC have not evaluated the carcinogenic potential of copper. Copper has been classified in EPA's Group D, according to EPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon inadequate data in humans and animals.

EPA's ambient water quality criteria for copper (U.S. EPA. 1980. Ambient Water Quality Criteria for Copper. EPA 440/5-80-036) is 1 mg/l, based upon taste and odor considerations. Copper is included in the National Secondary Drinking Water Regulations (EPA. National Secondary Drinking Water Regulations, 1979) as a secondary standard of 1.0 mg/l based upon taste and odor. The WHO has not proposed a guideline for copper based upon health effects; however they have proposed a guideline value of 1 mg/l, based upon the ability of copper to stain laundry and plumbing fixtures at concentrations above 1 mg/l.

Copper exposure at high levels may result in gastrointestinal disturbances and other acute toxic effects. For this reason and because occurrence is widespread, EPA is proposing an RMCL for copper. The RMCL will be based upon acute toxicity effects and a RMCL of 1.3 mg/l is proposed. Data on human exposure were not factored in the provisional AADI because the calculations were based upon acute effects.

Questions for Comment:

1. Is there sufficient health effects information upon which to base an RMCL, or should a health advisory be developed instead?

2. Is it appropriate to base the RMCL for copper upon acute, short-term effects?

7. Lead

Lead (Pb) is a relatively rare metal in the earth's crust, ranking 34th among the elements in crustal abundance with average concentrations of 15 ppm in the continental United States.

About 65 percent of the lead produced is used for the manufacture of storage batteries; approximately 10 percent is used for gasoline additives, the second largest use. The remainder is used for pigments, ammunition, solder, plumbing, cable coverings, caulking and bearings. Although lead can be released to all media, the atmosphere is the major initial recipient of lead emissions. The main source of lead in drinking water is leaching from lead piping and services and lead solders. Airborne lead from gasoline combustion may be one of the major contributors to total lead in drinking water.

Analytical Methods. Analytical methods available for analyzing lead in drinking water include flame atomic absorption, furnace atomic absorption, inductively coupled plasma atomic emission spectrometry techniques, X-ray fluorescence, colorimetric analyses and electrochemical techniques such as stripping voltometry.

Human Exposure. Human populations in the United States are exposed to lead in air, food, water and dust. In rural areas, Americans not occupationally exposed to lead are estimated to consume 40-60 μg Pb/day. This level of exposure is referred to as the baseline exposure for the American population because it is unavoidable except by drastic change in lifestyle or by regulation of lead in foods or ambient air. Forty-four percent of the baseline consumption of lead by children is estimated to result from consumption of 0.1 g of dust per day. Ninety percent of this dust lead is of atmospheric origin (U.S. EPA, 1984, Air Quality Criteria for Lead. EPA 600/8-23-028B).

Leaded gasoline combustion in vehicles accounts for about 90 percent of the total anthropogenic input of lead to the atmosphere. Atmospheric lead concentrations can range from 0.000076 μg/m³ in remote areas to 10 μg/m³ near point sources; EPA data show the average annual values in most areas to be below 1.0 μg/m³. The EPA has estimated respiratory intake of lead to be about 1 μg/day for the adult and 0.5 μg/day for a 2 year old child (U.S. EPA, 1985, Occurrence of Lead in Drinking Water, Food, Air; U.S. EPA, 1984 Air Quality Criteria for Lead. EPA-600/8-83-028B).

The route by which adults and older children in the baseline population of the U.S. receive the largest proportion of lead intake is through foods. Atmospheric lead may be added to food crops in the field or pasture, during the transportation to the market, during processing, and during kitchen preparation. Metallic lead, mainly from solder, may be added during processing and packaging. Other sources of lead, as yet undetermined, increase the lead content of food between the field and dinner table. American children, adult females and adult males are estimated to consume 19, 25, and 36 μg Pb/day, respectively, in milk and onbeverage

of lead carcinogenicity remains to be resolved in light of the other more extensive epidemiology studies which did not detect increased tumor incidence among lead-exposed human populations. The IARC have classified lead in Group 2: Inadequate evidence for carcinogenicity to humans, sufficient evidence of carcinogenicity to animals (for some salts), inadequate evidence for activity in short-term tests. Lead has been classified in EPA's Group B2, according to EPA's Proposed Guidelines for Carcinogen Risk Assessment based upon evidence of kidney tumors in rats by the oral route. However, the dosages that induced the kidney tumors in rats were very high (beyond the lethal dose in humans) and several extensive epidemiology studies did not show an association between lead exposure and increased tumor incidence in occupationally exposed workers. In view of the above issues, the RMCL for lead will not be set based upon carcinogenic endpoints, instead other sensitive endpoints will be the basis.

The current MCL for lead, under the National Interim Primary Drinking Water Regulations, is 0.05 mg/l. This level was based upon an estimation that this level in drinking water would contribute 25 to 30 percent of the lead normally ingested for a child and 20 percent of that in food for an adult.

The NAS (*Drinking Water and Health*, 1984 Vol. V) has stated that the current drinking water standard of 0.050 mg/l may not, in view of other sources of environmental exposure, provide a sufficient margin of safety, particularly for fetuses and young growing children. The WHO guidelines for drinking water (1984) suggest that lead should not exceed 0.050 mg/l. This level was based upon a provisional tolerable weekly intake of 3 mg of lead per person established in 1972 by the Joint FAO/WHO Expert Committee on Food Additives. Assuming a drinking water lead level of 0.050 mg/l, the 3 mg weekly intake would not be exceeded even with a daily diet containing 0.3 mg of lead and the WHO concluded that this allowed for some margin of safety. New data indicate that adverse health effects from lead occur at lower blood lead levels than believed in 1972 when the tolerable weekly intake was established.

EPA's ambient water quality criteria (U.S. EPA, 1980. Ambient Water Quality Criteria for Lead. EPA 440/5-80-057) for human health, which considers ingestion of water and contaminated aquatic organisms, is 0.050 mg/l. This value was based upon an analysis which concluded that the present drinking water standard of 0.050 mg/l may be viewed as representing the upper limit of acceptability.

Short-term exposures have not been determined for lead because, other than in cases of acute exposures to rather high lead levels, it generally takes a sustained exposure to lead for many days to produce body lead levels capable of inducing adverse effects.

At blood lead levels below 30 µg/dl, many of the different effects reported as being associated with lead exposure might be argued as separately not being of clear medical significance, although each are indicative of interference by lead with normal physiological processes. On the other hand, the collective impact of all of the observed effects (representing potentially impaired functioning and depleted reserve capacities of many different tissues and organs) may, at some point distinctly below 30 µg/dl, be seen as representing an adverse pattern of effects worthy of avoidance with some added margin of safety. The onset of signs of detectable heme synthesis impairment in many different organ systems, indications of increasing degrees of pyrimidine metabolism interference, signs of altered nervous system activity, and interference in vitamin-D metabolism start appearing at PbB levels around 10-15 µg/dl. All of these effects could be viewed as becoming sufficiently adverse to warrant avoidance when the various effects come to represent marked deviations from normal as PbB levels exceed 20-30 µg/dl.

An additional concern is one recent report that an increase of 5 µg/l in the blood lead levels produced a concomitant increase in diastolic blood pressure of approximately 2 mm Hg in white males, ages 40-59.

If one assumes that infants consuming formula reconstituted from tap water are the most sensitive subpopulation under consideration, and that a blood lead concentration of 15-20 µg/dl is the level of concern, it is possible to calculate the lead level in drinking water at which this group would be at risk. If 15 µg/dl were selected as the level of concern and using a conversion factor of 0.25 to convert from PbB to lead in drinking water (Ryu, et al. Dietary Intake of Lead and Blood Lead Concentration in Early Infancy. Am. J. Dis. Child. 137:895-891), the following calculation can be made: 15 µg/dl × 0.25 = 94 µg/l, (assuming consumption of 1 liter of water per day). Several alternatives are available in terms of the application of an uncertainty factor. One alternative is to assume an uncertainty factor of five, which would correspond to a drinking water lead level of 20 µg/l. If drinking water were the sole source of exposure. An uncertainty factor of five could be assumed instead of the traditional factor of 10 for human studies, since extremely subtle effects in a sensitive subpopulation (infants and children) were the endpoints upon which the level of concern was selected. A second alternative consists of applying an uncertainty factor of 10, resulting in a drinking water lead level of 10 µg/l.

A second option consists of using 20 µg/dl as the level of concern, converting this blood lead level to drinking water lead as was done in the first option (resulting in 125 µg/l), and applying an uncertainty factor. If an uncertainty factor of 10 was applied, this would correspond to a drinking water lead level of 13 µg/l. A second alternative consists of applying an uncertainty factor of 5, corresponding to a drinking water lead level of 25 µg/l.

A third option would be to set the RMCL at zero, since the health effects seen are often extremely subtle effects that may not have a threshold.

Exposure to lead results in effects on the nervous system, hematopoietic system and other physiological processes. Due to these effects and the significant occurrence potential in drinking water, EPA is proposing to continue to regulate lead. EPA is proposing an RMCL of 0.020 µg/l based upon the effects of lead in infants as a sensitive subpopulation. Comments are requested on all of the issues outlined below and on the additional options proposed for setting the RMCL.

Questions for Comment:

1. Is it most appropriate to base the RMCL upon a single health effect endpoint, or a constellation of effects?

2. What uncertainty factor should be included in setting the RMCL for lead?

3. To what extent should possible carcinogenic effects of lead be taken into account as a margin of safety consideration?

4. What is the most appropriate model to relate blood lead levels with drinking water levels?

5. Are women of child bearing age, the developing fetus, the neonate or the one to three year old child the most sensitive subpopulation(s)?

6. Which of the options presented for developing an RMCL for lead is most appropriate given currently available knowledge about lead health effects and exposures?

8. Mercury

Mercury exists in two basic forms: the inorganic salt and organic mercury

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compounds (methyl mercury). Mercury levels in coal range from 10–50,000 ppb, though generally it is in the range of 200–400 ppb, with soil concentrations ranging from 20–300 ppb. Most of the known areas with substantially elevated mercury in geologic materials are found in California, Nevada, Oregon and Washington.

The major use of mercury is in electrical equipment (batteries, lamps, switches, and rectifiers). It is also used in the chloralkali industry as a flowing cathode for electrolytic deposition of salt brine into chlorine, sodium hydroxide and hydrogen.

Mercury may also enter the environment from mining, smelting, and fossil fuel combustion. Natural processes such as volcanic activity, geothermal activity and volatilization from mineral deposits result in mercury entering the atmosphere.

Analytical Methods. Analytical methods available for analyzing mercury in drinking water include the manual cold vapor and automated cold vapor techniques.

Human Exposure. Mercury is introduced into the air from both natural and anthropogenic sources. Ambient levels of mercury range between 10–20 ng/m^3. In areas near coal-fired power plants, levels be as high as 1000 ng/m^3. Usually high concentrations of 10,000–15,000 ng/m^3 have been reported near rice fields where mercury fungicides have been, used and in the vicinity of mercury mines.

The average daily absorption of atmospheric mercury for an adult male is estimated to be approximately 320 ng, based upon an average atmospheric concentration of 20 ng/m^3, a ventilation rate of 20 m^3 and the assumption that 80 percent of the inhaled rate is absorbed.

Food is a primary source of exposure to mercury, and the major dietary source appears to be the consumption of fish. The FDA estimated the dietary intake of mercury to be 4.3 μg/day for the adult male (excluding beverages), of which 3.8 μg comes from meat, fish, and poultry categories.

Almost all mercury detected to date in drinking water is in the form of inorganic mercury. Compliance monitoring indicates that 12 ground water and 11 surface water supplies have reported levels above 2 μg/l. All of the ground water supplies serve less than 5,000 people whereas five of the surface water supplies serve more than 10,000 people.

There is only limited federal survey data on mercury. Of 106 ground water supplies sampled between 1978 and 1980, 32 (30%) were found to have levels above 0.5 μg/l, and 14 (13%) were found to have levels above 2 μg/l. In surface water supplies, mercury was observed at levels of 0.5 μg/l in 10 out of 21 supplies sampled (52%), with 5 supplies (18%) at levels above 2 μg/l. It is not clear why the federal surveys suggest a substantially higher frequency of occurrence above 2 μg/l than is indicated by the compliance monitoring results. The major source of federal survey data was the Rural Water Survey.

Health Effects. Inorganic mercury is poorly absorbed through the gastro-intestinal tract, does not penetrate cell membranes rapidly and is less toxic than methyl mercury. The principal target organ of inorganic mercury is the kidney. Methyl mercury compounds exert their toxicologic effect on the central nervous system. Contamination of fish and shellfish by methyl mercury compounds has resulted in Minamata Disease in Japan, characterized by mental disturbances, ataxia, disturbance in speech and hearing impairment.

Short-term assessment values were not calculated for mercury due to insufficient data. A provisional AADI was calculated for inorganic mercury based upon a study (Druet, et al. 1978. Immune Type Glomerulonephritis Induced by Mercuric Chloride in the Brown Norway Rat. Ann. Immunol. 129C:777–792) in which rats were exposed to inorganic mercury salts for 8 to 12 weeks through subcutaneous injection. The primary endpoints evaluated were antibody formation and proteinuria, with proteinuria seen at doses of 100 μg/kg and above, but not at 50 μg/kg. In the 50 μg/kg exposure group, a total of 36 doses totalling 1800 μg/kg were injected over the 84-day period. The NOAEL was determined to be 50 μg/kg/day and a factor of 0.739 to adjust for the percentage by weight of mercury in mercuric chloride was included in the development of the provisional AADI. An uncertainty factor of 1000 was applied because proteinurea, an adverse health effect, occurred at concentrations of mercury only twice the NOAEL. To compensate for the difference in absorption between subcutaneous and oral exposure, an absorption factor of 10 was used, resulting in a provisional AADI of 0.0055 mg/l. This value was based upon inorganic mercury because almost all mercury in drinking water is thought to be in the form of inorganic mercury.

The IARC have not evaluated the carcinogenic potential of mercury. Mercury has been classified in EPA's Group D, according to EPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon inadequate data in animals and humans.

The WHO guideline (1984) for mercury in drinking water is 0.001 mg/l and applies to all chemical forms of mercury. This level was based upon the determination that 2 liters of water containing 0.001 mg/l mercury would normally contribute less than 10 percent of the tolerable intake of mercury. EPA's ambient water quality criterion (U.S EPA. 1980. Ambient Water Quality Criteria for Mercury. EPA 440/5–80–058) for human health considering ingestion of water is 0.000144 mg/l. This was based upon the ingestion of water and contaminated aquatic organisms which contain methyl mercury.

Exposure to inorganic mercury compounds at high levels results in renal effects. Because inorganic mercury is the form of mercury detected in drinking water, has widespread occurrence and may have adverse health effects, a revised regulation is proposed. An RMCL for inorganic mercury of 0.003 mg/l is proposed, based upon a provisional AADI of 0.0055 mg/l with data on human expoeure factored in (0.0043 mg/day via the diet and 0.001 mg/day via air).

Question for Comment:

1. Should a seperate RMCL be proposed for organic mercury compounds?

9. Nitrate and Nitrite

Nitrate is the more stable oxidized form of combined nitrogen in most environmental media. Most nitrogenous materials in natural waters tend to be converted to nitrate, and, therefore, all sources of combined nitrogen (particularly organic nitrogen and ammonia) should be considered as potential nitrate sources. Nitrates occur naturally in mineral deposits (generally sodium or potassium nitrate), in soils, seawater, freshwater systems, the atmosphere and in biota. Lakes and other static water bodies usually have less than 1.0 μg/l of nitrate/nitrogen. (Generally, the occurrence of nitrate is measured and reported in terms of its nitrogen content; 1.0 μg/l of nitrate/nitrogen is equivalent to 4.4 μg/l of nitrate.) Ground water levels of nitrate/nitrogen may range up to 20 μg/l or more, with higher levels characteristically occurring in shallow aquifers beneath areas of extensive development. Major sources of nitrates or nitrite in drinking water include fertilizer, sewage and feedlots.

Analytical Methods. Analytical methods available for analyzing nitrate and nitrite in drinking water include the colorimetric brucine spectrometric cadmium reduction, automated

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hydrazine reduction and automated cadmium reduction techniques.

Human Exposure. A daily respiratory intake ranging from 25 to 70 μg/day based upon reported urban and nonurban levels of nitrogen oxides has been determined.

Food is generally recognized as the major source of nitrate intake. The estimated dietary intake is approximately 250 μg/day, with 85 to 90 percent coming from vegetables and 9 percent from cured meats. Most vegetables contain nitrate. Beets, celery, lettuce, radishes, and spinach were reported to have levels ranging from 120 to 2500 ppm (dry weight). Much lower levels (10–24 ppm) were reported for tomatoes, potatoes and peas.

Compliance monitoring indicates that approximately 270 community public water supplies are providing water with nitrate/nitrogen levels above 10 μg/l (44 μg/l nitrate).

Three national surveys conducted between 1969 and 1980 indicated that 625 out of 1,679 (38%) ground water supplies had nitrate/nitrogen levels above 0.5 μg/l (nitrate levels of 1.25 μg/l). The mean nitrate/nitrogen levels above 0.5 μg/l was 1.8 μg/l, and values ranged up to about 22 μg/l. There were 20 ground water supplies with levels above 10 μg/l (1.6%).

In these same surveys, nitrate/nitrogen levels of 0.5 μg/l were found in 175 out of 620 surface water supplies sampled (42.5%), with a mean value of 1.8 μg/l. The nitrate/nitrogen values in surface water ranged up to 21 μg/l. There were 5 supplies (1.2%) with levels above 10 μg/l. The presence of nitrite in drinking water at significant levels is unusual and would indicate probable organic contamination (i.e., sewage), lack of disinfection (reducing conditions) and probable bacterial contamination.

Health Effects. The toxicity of nitrate in humans is due to the body's reduction of nitrate to nitrite. This reaction takes place in saliva of humans at all ages and in the gastrointestinal tract of infants during the first three months of life. The toxicity of nitrite is demonstrated by vasodilatory/cardiovascular effects at high dose levels and methemoglobinemia at lower dose levels. Methemoglobinemia is an effect in which hemoglobin is oxidized to methemoglobin, resulting in asphyxia.

Infants up to 3 months of age are the most susceptible subpopulation with regard to nitrate. This is due to the fact that in the adult and child, about 10 percent of ingested nitrate is transformed to nitrite, while 100 percent of ingested nitrate can be transformed to nitrite in the infant.

The effects of methemoglobinemia are rapidly reversible and there are, therefore, no cumulative effects. There are no suitable data for calculating one-day assessment values for either nitrate or nitrite. However, the ten-day assessment values for both nitrate and nitrite will be protective for one-day exposures.

A study by Craun, et al. (1981) was selected as the basis for calculating ten-day assessment values for nitrate in adults. Craun, et al. (1981) observed that nitrate levels up to 111 μg/l nitrate/nitrogen did not produce methemoglobin in one to eight year old children. The data of Craun, et al. (1981) are appropriate to use as the basis for a ten-day adult assessment of 111 μg/l nitrate-nitrogen because the child and the adult would be expected to have similar responses to nitrate. An uncertainty factor was not employed in the derivation of the number since the most sensitive subpopulation, infants, are considered separately and would not need to be protected under this number.

The ten-day adult assessment for nitrite can be calculated using the ten-day nitrate assessment and assuming a 10 percent conversion of nitrate to nitrite. Thus, the ten-day assessment for nitrite in adults is 11 μg/l nitrite nitrogen.

As the provisional AADI for both nitrate and nitrite is based upon short-term exposure in the infant, it is appropriate to use the provisional AADI as a 10-day assessment for the infant.

A provisional AADI was calculated for nitrate/nitrogen based upon the infant (4 kg) as a high risk subpopulation. The study used was an epidemiologic study in which 300 cases of infant methemoglobinemia were surveyed (Walton, G. 1951. Survey of Literature Relating to Infant Methemoglobinemia due to Nitrate Contaminated Water. Am. J. Pub. Health 41:986–996). No cases were associated with drinking water containing less than 10 μg/l of nitrate/nitrogen. Using 10 mg/l as a NOAEL, without the application of an uncertainty factor (large number of subjects exhibiting no signs of toxicity), a provisional AADI of 10 mg/l was determined for nitrate/nitrogen. This value would also be protective for the infant for short-term exposures (1- and 10-days). For nitrite/nitrogen, the same study was used, with an uncertainty factor of 10 applied to the NOAEL (10 mg/l), due to the demonstrated direct toxicity of this chemical, resulting in a provisional AADI of 1 mg/l. This value would also be protective for the infant for short-term exposures (1- and 10-days).

Many studies have demonstrated that nitrate/nitrite administered with nitrosatable compounds are carcinogenic in animals. However, the carcinogenic potential of nitrate/nitrite when administered in the absence of nitrosatable compounds has not been demonstrated. The IARC have not classified nitrate/nitrite for potential carcinogenicity. Nitrate/nitrite have been classified in EPA's Group D, according to EPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon inadequate data in animals and humans.

The current MCL for nitrate/nitrogen, under the National Interim Primary Drinking Water Regulations, is 10 mg/l. This level was based upon human case studies in which fatal poisonings in infants have occurred following ingestion of well waters containing nitrate concentrations greater than 10 mg/l nitrate/nitrogen.

The WHO guidelines are 10 mg/l for nitrate/nitrogen and 1 mg/l for nitrite/nitrogen. The basis is that undesirable increases in methemoglobin levels in blood occur at levels from 10 mg/l to 20 mg/l nitrate and the ingestion of nitrite leads to a more rapid onset of clinical effects and thus the guideline value should be correspondingly lower (1 mg/l).

Nitrate/nitrite compounds have demonstrated adverse toxic effects in infants. Due to potential toxicity and wide spread occurrence in water, revised drinking water regulations will be proposed. The RMCL will be based upon non-carcinogenic effects and RMCLs of 10 mg/l for nitrate/nitrogen and 1 mg/l for nitrite/nitrogen are proposed. Data on human exposure were not factored in the RMCL because the provisional AADI was based upon an epidemiologic study in which exposure via the air and diet is already taken into account.

Questions for Comment:

1. Should the carcinogenic potential of nitrosatable compounds influence the proposed RMCLs for nitrate/nitrogen and nitrite/nitrogen? If so, how?

2. The Criteria Document on Nitrate/Nitrite outlines an alternate methodology for calculation of short-term assessment values. Is this an appropriate approach for calculation of these numbers?

3. What uncertainty factors should be applied in the calculation of the AADIs for nitrate and nitrite?

10. Selenium

Selenium occurs in U.S. soils at concentrations ranging from 0.05 to 0.8 ppm with very little difference between

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the soils of eastern and western States. However, in the western States, the more alkaline soils tend to make selenium more water soluble and increased plant uptake and accumulation occurs. Sedimentary rocks have higher concentrations of selenium than do igneous rocks. Of the sedimentary rocks, shales frequently contain more selenium than limestones or sandstones.

Most of the commercial selenium produced in the U.S. is recovered from copper ores. Only 235 metric tons were produced in 1981; an additional 316 metric tons were imported. The reported consumption of selenium for 1981 was for electronic and photocopy applications, glass manufacture, pigments, chemicals, pharmaceuticals, fungicides and animal feed additives. Selenium releases to the environment result from coal (coal contains 1.0–3.3 ppm) burning, non-ferrous mining and smelting, and the remainder from selenium refining, glass manufacture and fuel oil combustion (1979 estimate).

Analytical Methods. Analytical methods available for analyzing selenium in drinking water include the gaseous hydride atomic absorption, furnace atomic absorption and inductively coupled plasma atomic emission spectrometry techniques.

Human Exposure. In general, selenium levels in the air in the U.S. appear to be below 5 ng/m³. One study showed that levels near a coal-fired power plant, a major source of selenium to air, were <3 ng/m³. These reported estimates of daily respiratory intake of selenium are 0.02, 0.07 and <1 µg.

The selenium content of foods ranges from undetected or trace levels (<0.001 ppm) to approximately 1.0 ppm. Higher levels (0.3–1.0 ppm) are reported for the meat, fish, poultry, and grains and cereal food classes. A recent FDA market basket survey reported that dietary intake for the adult male was 152 µg/day, of which 52 percent comes from grains and cereals, 38 percent from meat, fish and poultry and 10 percent from dairy products. Other studies have reported selenium intake through the diet ranging from 50 to 150 µg/day (*Drinking Water and Health*, 1980, Vol. III; Welsh, et al. 1981. Selenium in Self-Selected Diets of Maryland Residents. Jour. Am. Dietic Assn. 79:277–285).

Compliance monitoring indicate that 130 ground water and 6 surface water supplies contain selenium levels above 10 µg/l.

In the 1980 Community Water Supply Survey (CWSS), 654 out of 671 ground water supplies (97%) had selenium at levels ranging from 1 µg/l to 65 µg/l (mean of the positives was 2.7 µg/l). In the 1979 CWSS, 12 out of 258 ground water supplies (4.7%) had levels above 5 µg/l and 2 (0.8%) had levels above 10 µg/l. In the Rural Water Survey (RWS), 30 out of 71 ground water supplies (42.3%) had levels above 5 µg/l and 8 out of 71 (11.3%) had levels above 10 µg/l.

The 1980 CWSS reported that selenium was found in all 108 surface water supplies sampled, at levels ranging from 1.0 to 10 µg/l (mean value, 4.6 µg/l). In the 1979 CWSS, none of the 94 supplies sampled were found to have measurable levels of selenium; the minimum quantifiable levels ranged from 2.5 to 5 µg/l. In the RWS, 2 out of 21 surface supplies had levels above 5 µg/l.

Health Effects. Selenium has toxic effects at high dose levels and is nutritionally essential at low levels. Acute and chronic toxic effects have been observed in animals. In humans, little data exists on acute toxicity, while a recently completed study on endemic selenium intoxication in humans in China (Yang, G., et al. 1983. Endemic Selenium Intoxication of Humans in China. Amer. Jour. Clin. Nutr. 37:872–881) reported that chronic toxicity was observed in individuals consuming a daily average of 4.5 mg selenium through the diet, with a minimum selenium intake of 3.2 mg.

In animals, selenium deficiency results in congenital white muscle disease and other diseases. A level of 0.1 mg Se/kg food has been considered to be the general level of dietary requirements in animals. Selenium is considered to be an essential element for human nutrition, with an approximate intake level of 100–200 µg/day adequate to prevent deficiency in a 70 kg adult. The NAS (*Drinking Water and Health*, 1980, Vol. III) have estimated an adequate and safe intake of selenium for adults of 0.05 to 0.20 mg/day with correspondingly lower intakes for children and infants.

A 1-day assessment was not calculated for selenium due to insufficient data. The values calculated for the 10-day levels are considered protective for the 1-day exposure period. A 10-day assessment for the adult was calculated based upon a study (Halverson, et al. 1966. Toxicity of Selenium to Post-weanling Rats. Toxicol. Appl. Pharmacol. 9:477–484) in which rats were fed sodium selenite for 6 weeks and levels of 4.6 mg/kg feed (0.41 mg/kg/day) produced no adverse effects as compared with controls. A 10-day assessment for the adult of 144 µg/l was determined, using 0.41 mg/kg day as the NOAEL, an uncertainty factor of 100 based upon an animal study and consumption of 2 liters of water per day. A 10-day assessment for the child of 48 µg/l was calculated based upon the same study, assuming consumption of 1 liter of water per day.

A provisional AADI was calculated based upon a human study (Yang, et al. 1983. Endemic Selenium Intoxication of Humans in China. Amer. Jour. Clin. Nutr. 37:872–881) which examined selenium toxicity and deficiency effects in China. The minimum daily selenium intake level in an area of chronic selenosis was reported to be 3.20 mg. A provisional AADI of 0.160 mg/l was calculated using 3.20 mg/day as a LOAEL, consumption of two liters of water per day and an uncertainty factor of 10. This uncertainty factor was applied to a human study with a great deal of data. An uncertainty factor of 10 would traditionally be applied, however in this instance an uncertainty factor of 15 was applied due to data which suggests that selenium ingested in food may be absorbed less efficiently than selenium ingested in water. This information would support the use of an uncertainty factor greater than 10.

Naturally occurring selenium compounds (selenate and selenite) have not been shown to be carcinogenic in animals. On the contrary, many studies have shown these selenium compounds to result in the inhibition of tumors of various types, including tumors of the skin, liver, mammary glands, colon and lung. Selenium sulfide, a manufactured product not normally found in water, was found to be positive in the National Cancer Institute bioassay by gavage (NCI, 1980. Bioassay of Selenium Sulfide (gavage) for Possible Carcinogenicity. NCI Technical Support Series No. 194. NTP No. 80–17). The IARC has not classified selenium for potential carcinogenicity. Selenium has been classified in EPA's Group D, according to EPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon inadequate data in animals and humans.

The current MCL for selenium, under the National Interim Primary Drinking Water Regulation, is 0.01 mg/l. This level was based upon signs of selenium toxicity at an intake of 0.7–7 mg/day and an assumed selenium intake of 200 µg/day.

The WHO guideline (1984) for selenium is 0.01 mg/l. The WHO stated that current estimates of selenium intake range between 120 and 200 µg/day and the maximum daily selenium intake from drinking water should not exceed 10 percent of the recommended maximum daily dietary intake of 200 µg. Assuming an intake of 2 liters of water per day, the WHO determined a

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guideline value of 0.01 mg/l. EPA's ambient water quality criteria (U.S. EPA. 1980. Water Quality Criteria Document for Selenium. EPA 440/5-80-070) for human health is 0.010 mg/l in agreement with the NSDWR. Current data suggests that selenium is an essential nutrient and has anti-carcinogenic effects in animals. These guidelines (WHO and ambient water quality criteria) did not take the beneficial effects of selenium into account.

Selenium exposure at high levels results in chronic adverse health effects. EPA is proposing to continue regulation of selenium because of its potential adverse health effects and widespread occurrence. An RMCL of 0.005 mg/l is proposed, based upon a provisional AADI of 0.105 mg/l, with data on human exposure factored in (0.105 mg/day via the diet).

Questions for Comment:

1. Does the proposed RMCL for selenium reflect an adequate balance between the toxicity and deficiency effects of the compound?

2. Is it more appropriate to apply a traditional uncertainty factor of 10 for selenium, resulting in an RMCL of 10 µg/l?

C. IOC's for Which RMCLs Are Not Proposed

1. Aluminum

Aluminum is the third most abundant element in the earth's crust and occurs most commonly as aluminosilicates, such as clay, kaolin, mica and feldspar and as bauxite or cryolite. Uses of aluminum compounds and alloys include production of aircraft, utensils, and electrical conductors. The metal powder is used in explosives, pigments, paints and coatings and putties. Aluminum compounds (e.g., aluminum sulfate) are commonly used in drinking water treatment for coagulation.

Analytical Methods. Analytical methods available for analyzing aluminum in drinking water include the flame atomic absorption, furnace atomic absorption and inductively coupled emission spectrometry techniques.

Human Exposure. Levels of elemental aluminum in the ambient air over the continental U.S. have been reported to range from 0.16–0.0 ng/m³. Levels of aluminum in metropolitan areas were an order of magnitude greater than those in rural areas. Background atmospheric concentrations have been measured from 0.005–0.020 ng/m³.

Aluminum is naturally present in nearly all foods. Other significant sources of the metal in the diet are baking powder, food additives, antacids, and dietary supplements. In addition, aluminum cookware, utensils and storage containers can be sources of the metal in foods. Numerous studies are available on dietary intake of aluminum reported levels ranging from less than 1.59 to as much as 100 mg/day. The average adult intake from the diet, however, is calculated to be approximately 10 mg aluminum per day.

Aluminum commonly occurs in finished drinking water supplies, especially surface waters treated with alum. Use of this compound can increase (or decrease) the level of aluminum in finished waters.

In a recent EPA study (1983) of the occurrence of aluminum in drinking water, an analysis was conducted of randomly selected community water systems. Levels of aluminum (mg/l) in finished drinking water determined for various types of water systems and sizes are shown below:

	Levels	Medians
Ground Water—no coagulant	0.014–0.030	0.021
Surface Water—no coagulant	0.015–1.127	0.045
Surface Water—Alum coagulant	0.014–0.073	0.112
Surface Water—Iron coagulant	0.015–0.021	0.020

Median aluminum levels (mg/l) by population category (both ground water and surface water) were as follows:

Number of individuals served	Concentrations of aluminum	
	Medians	Ground medians
25 to 500	<0.014–0.224	<0.014
501 to 3,300	<0.014–0.204	0.023
3,301 to 10,000	<0.014–0.223	0.040
> 10,000	<0.014–0.202	0.020

A review of earlier surveys of finished drinking water supplies across the U.S. (1962, 1969, 1977) indicates mean levels of aluminum ranging from 0.073–0.104 µg/l. Levels of the metal in individual samples ranged from 0.000–2.4 µg/l.

Health Effects. In general, aluminum is considered to possess low acute toxicity for the normal individual following oral exposure. Oral LD$_{50}$s for aluminum chloride in several animal species ranged from 380 to 700 mg/kg. Few data exist from animal studies following subchronic or long-term oral exposure. These suggest primarily that effects may be seen at relatively high doses on phosphorus balance, and that the adverse sequelae are, in fact, the result of this change (e.g., osteomalacia). No evidence of fetotoxicity or teratogenicity has been observed in animals following oral exposure. On the other hand, there is a report of decreased sperm count and motility in rats exposed to 2.5 mg/kg by gavage for 6 months. Studies to evaluate the potential of aluminum or its salts to induce mutagenic or carcinogenic effects have yielded negative results.

In the human, deposition of aluminum in the brain has been suggested as an etiological factor in the neurologic disorders, Alzheimer's disease and dialysis dementia in patients with chronic renal failure. This hypothesis has not been confirmed and the question of the relationship between aluminum and certain neurological disorders is one that is receiving a great deal of attention by the medical research community at the present time. In addition, the Agency is pursuing lines of investigation in experimental studies which should shed light on the dose-response relationships in animals following exposure to aluminum salts in drinking water. Since, at the present time, there are no adequate dose-response data available from which to estimate an AADI for ionic aluminum in drinking water, EPA is proposing not to establish an RMCL and primary regulation. Aluminum will be reevaluated at a later date, after the results of the animal studies become available.

A guidance level of 0.05 mg/l is recommended for aluminum such that treatment is fully effective in removing coagulated materials to avoid settling of particles in the distribution system. This value is not based upon health and has been recommended by the American Waters Works Association as a goal for potable water. The WHO has recommended a guideline value of 0.2 mg/l based upon water discoloration.

2. *Cyanide.* Cyanides are organic or inorganic compounds that contain the cyanide moiety, —CN. Organic compounds having this functional group are referred to as nitriles. Cyanides are formed readily in many industrial processes and can be found in a variety of effluents. Cyanide commonly occurs in water as hydrocyanic acid (HCN), the cyanide ion (CN^-), simple cyanides, metallocyanide complexes or as simple chain and complex ring organic molecules.

Cyanide production in the U.S. in 1978 exceeded 700 million pounds. The major industrial users of cyanide in the U.S. are the producers of steel, plastics, synthetic fibers and chemicals and the electroplating and metallurgical industries. In addition to these industries, cyanide wastes are discharged into the environment from the pyrolysis of a number of synthetic and natural materials and from chemical, biological, and clinical laboratories. Cyanide in drinking water is oxidized by chlorine at basic pH to cyanate.

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Analytical Methods. Analytical methods available for analyzing cyanide in drinking water include colorimetric and automated electrode techniques with distillation.

Human Exposure. No information is available on levels of cyanide in the ambient air.

No data are available on the occurrence of cyanide in foods. Except for certain naturally occurring nitriles in plants, it is unlikely that cyanide would be found in foods of the U.S.

Cyanide is relatively uncommon in most U.S. water supplies. In 2,595 water samples, the Community Water Supply Survey (CWSS) of 969 U.S. public water supply systems in 1976 revealed the highest cyanide concentration found was 8 µg/l and the average concentration was 0.09 µg/l. However, cyanide has been found in surface waters and is present in waste waters. It is soluble in water and used in large quantities across the country. Although it is biologically and chemically degradable, it has appeared in some drinking water systems.

Health Effects. Cyanides are readily absorbed from the lungs, the gastro-intestinal tract and the skin by animals and humans. The toxic effects of cyanide occur due to the combination of cyanide with cytochrome in the cell, with resulting hypoxia. Cyanide may be detoxified in the liver by rhodanese, an enzyme in the liver which metabolizes cyanide to form a less toxic compound, thiocyanate. The major route of cyanide elimination from the body is via urinary excretion of thiocyanate.

No suitable data were identified for the calculation of a one-day assessment. Although there are data available to calculate the 10-day assessment value, it is recommended that the 1-day and 10-day levels be set at the AADI of 0.75 mg/l for the 70 kg adult consuming 2 liters of water per day. Using the same data base, the 1-day and 10-day assessment value for the 10 kg child consuming 1 liter of water is calculated to be 230 µg/l.

A provisional AADI was calculated based upon a two-year study in which rats were administered diets containing 0, 100 or 200 mg/kg hydrogen cyanide (Howard, J.W. and Hansel, R.F. 1955. Chronic Toxicity for Rats of Food Treated with Hydrogen Cyanide. J. Agric. Food Chem. 3:323-329). Average doses due to volatilization were calculated to be 76 mg/kg/diet hydrogen cyanide (73 mg/kg/diet cyanide) for the low dose group and 187 mg/kg/diet hydrogen cyanide (180 mg/kg/diet cyanide) for the high dose group. The estimated daily doses were 3.8 and 4.6 mg/kg body weight cyanide for the low-dose males and females, respectively, and 7.5 and 10.8 mg/kg body weight cyanide for the high dose males and females, respectively. At these dose levels, no treatment-related toxic effects including histopathologic lesions were observed. A provisional AADI of 0.75 mg/l cyanide was calculated from the NOAEL of 10.8 mg/kg cyanide with an uncertainty factor of 300 (an additional uncertainty factor of 5 was included because cyanide was given in the diet rather than in drinking water) and assuming human consumption of 2 liters of water per day.

Potassium cyanide was negative for mutagenicity in *Salmonella typhimurium* and *Bacillus subtilis*. The IARC have not classified cyanide for potential carcinogenic effects. Cyanide has been classified in EPA's Group D, according to EPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon inadequate data in animals and humans.

The WHO guideline for drinking water is 0.1 mg/l. This value was based upon 4.7 mg cyanide/day which has been recorded to not be harmful to humans. Assuming consumption of 2 liters of water per day, the WHO concluded that cyanide concentrations of 2.35 mg/l could be consumed in water. Allowing for a safety factor, the WHO considered a guideline value of 0.1 mg/l to be reasonable. EPA's ambient water quality criteria (U.S. EPA. 1980. Ambient Water Quality Criteria for Cyanide. EPA 440/5-80-037) for human health considering ingestion of water and contaminated aquatic organisms is 0.2 mg/l. This value was updated (February 1984. 49 FR 4551) and the proposed revised value is 3.77 mg/l based upon the same study used to derive the provisional AADI. The difference in the two numbers is due to the addition of an additional uncertainty factor of 5 in the derivation of the provisional AADI since cyanide was given in the diet rather than drinking water.

Cyanide has rarely been detected in drinking water supplies, and when it has been detected it has been at levels so far below the provisional AADI that an RMCL for cyanide would almost certainly be useless. Thus, EPA has decided not to propose an RMCL for cyanide at the present time.

Questions for Comment:

1. Are there sufficient health effects and exposure information upon which to base an RMCL?

2. Is the assumption of 100 percent contribution from drinking water reasonable?

3. Is it appropriate to apply an additional uncertainty factor of 5 to account for the absorption differences between cyanide exposure via food and drinking water?

4. Should EPA set an RMCL for cyanide despite the fact that it has been detected at levels far below the provisional AADI?

3. Molybdenum

Molybdenum occurs naturally as molybdenum sulfide and as molybdenite salts. Uses include the manufacture of special steel for tools, boiler plate, and propeller shafts, tungsten, x-ray tubes, filaments and non-ferrous alloys. Molybdenum is also used as an additive in lubricants.

Analytical Methods. The analytical methods available for analyzing molybdenum in drinking water are the flame atomic absorption, furnace atomic absorption and inductively coupled plasma atomic emission spectrometry techniques.

Human Exposure. Molybdenum is considered an essential trace element in humans. The NAS has estimated the dietary intake of molybdenum to range from 0.1 to 0.48 mg/day. In a separate study, an average daily intake of 0.18 mg/day was estimated based on an analysis of 300 samples of 40 different foods, collected from 5 supermarkets over a 3-year period.

In an early study (1964) of finished water supplies of the 100 largest cities in the United States, levels of molybdenum were reported to range from not detected to 68 µg/l (median = 1.4 µg/l). In a later 5-year study (1970) of 350 finished waters, 29.9% had measurable levels of molybdenum; concentrations ranged from 3 to 1,034 µg/l (mean = 85.9 µg/l). Results of a recent and extensive study (1976) of tap waters of the U.S. showed molybdenum in 30 percent of the samples, with levels ranging from 1.1 to 52.7 µg/l (mean = 8.0 µg/l). According to the NAS, molybdenum in drinking water, except from highly contaminated sources (e.g., molybdenum mining wastewater) is not likely to constitute a significant portion of the total human daily intake of the element.

Health Effects. Molybdenum is readily absorbed through the gastrointestinal tract, with the highest concentrations in the liver, kidneys and bone. There is no apparent bioaccumulation of molybdenum in animal or human tissues. A steady state concentration is reached when intake is increased, and when the dose is withdrawn, tissue concentrations return to normal levels. The principal route of excretion is via the urine.

Acute toxic effects from exposure to molybdenum consists of damage to the

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liver, kidneys and sometimes adrenals and spleen. Ruminants are more sensitive to molybdenum than are monogastric animals. Subchronic toxic effects in animals consist of a decreased growth rate, male infertility, weight loss and bone or joint abnormalities in forelegs.

Molybdenum is an essential element at low doses and has toxic effects at high dose levels. The NAS (*Drinking Water and Health*, 1980, Vol. III) have estimated an adequate and safe intake level of 0.15 to 0.50 mg/day for adults and children 11 years and older.

One-day assessments of 2.7 mg/l (child) and 9.5 mg/l (adult) have been calculated for molybdenum based upon a study (Fairhall, et al., 1945. The Toxicity of Molybdenum. Pub. Hlth. Serv. Bull.) in which a single intraperitoneal dose of 20 mg molybdenum (80 mg/kg ammonium molybdate) did not show any apparent effects. Other studies have shown that the LD^{50} for orally ingested ammonium molybdate is about three times higher than for molybdenum trioxide and calcium molybdate, the forms commonly found in water. Thus, a NOAEL of 27 mg/kg was used, calculated by dividing the NOAEL for ammonium molybdate (80 mg/kg) by three to account for the potential differential toxicity. An uncertainty factor of 100 was applied based upon a NOAEL from an animal study, with an assumed water consumption of 1 liter per day (child) and 2 liters per day (adult).

Sufficient data were not available from which to derive 10-day assessments. Ten-day numbers were calculated by dividing the 1-day numbers by 10, resulting in values of 0.27 and 0.95 mg/l for children and adults, respectively.

A provisional AADI was calculated for molybdenum based upon a human study (Chappell, W.R., et al., 1979. Human Health Effects of Molybdenum in Drinking Water. Cincinnati, OH. U.S. EPA–600A–79–009) in which blood molybdenum levels were within normal ranges and no adverse effects were noted when drinking water levels were 0.200 mg/l or less. Using 0.200 mg/l as a NOAEL, an uncertainty factor of 2 based upon a human study with no adverse effects noted and consumption of 2 liters of water per day, a provisional AADI of 0.10 mg/l was determined. Data on human exposure were not factored in the AADI because the calculations were based upon an epidemiological study in which exposure via other sources were necessarily taken into consideration.

The only molybdenum compound that has been shown to produce tumors in animals is an inorganic pigment which contains a lead chromate, sulfate and molybdenum compound. This compound was shown to produce tumors by subcutaneous injection; however since both lead and chromate are present in the compound it is possible that the effect was due to these metals and not molybdenum. The IARC have not classified molybdenum for potential carcinogenicity. Molybdenum has been classified in EPA's Group D, according to EPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon inadequate data in animals and humans.

The WHO has not recommended a limit for molybdenum in drinking water. The NAS also has not recommended a level of molybdenum in drinking water other than the safe intake level for nutritional effects. According to the NAS (1980), "Our understanding of chronic molybdenum toxicity or deficiency is presently extremely limited. This topic should be studied. Further studies should also be conducted to determine the interaction of molybdenum with other elements and nutrients in humans."

EPA has decided not to propose an RMCL at this time for molybdenum because of the inadequate data on toxicity of the compound. A provisional AADI of 0.10 mg/l was determined based upon an epidemiological study in which only one dose was examined and no effects were noted. This study did not determine at what level effects may have been seen. Animal studies examining chronic toxicity are not available and thus an AADI could not be calculated based upon these effects. Acute animal studies are available but acute studies are not an adequate basis for determining an AADI.

Question for Comment:

1. Should a Health Advisory be developed for molybdenum or is there sufficient health effects information upon which to base an RMCL?

4. Nickel

Elemental nickel is not commonly found in nature as the pure metal, but occurs as sulfides, arsenides, antimonides, and oxides or silicates. The pure metal is very low in solubility. Nickel salts and many nickel compounds, however, are soluble. In the aquatic environment, nickel is most likely to occur as a divalent cation and may often absorb to or complex with mineral or organic compounds.

The major use of nickel is in the manufacture of stainless steel, nickel-chrome resistance wire and in alloys for electronic and space applications.

Analytical Methods. Analytical methods available for analyzing total nickel in drinking water include the flame atomic absorption, furnace atomic absorption and inductively coupled plasma emission spectrometry techniques.

Human Exposure. Levels of nickel in the ambient air of 237 urban and 47 nonurban localities in the U.S., for the period 1970 to 1974, were determined by EPA. Arithmetic means for each of these years ranged from 0.009 to 0.015 $\mu g/m^3$ for urban areas and 0.002 to 0.003 $\mu g/m^3$ for nonurban areas (detection limit, 0.001 $\mu g/m^3$). For any given year, the urban values were 3 to 4 times that of the nonurban areas.

Reported dietary levels of nickel for U.S. consumers range from 165 to 900 μg/day. Average values range from 400 to 900 μg/day. Nickel is common in a wide variety of foods. Food processing methods, however, may add additional levels through leaching from stainless steel processing equipment.

A summary of levels of nickel in 380 U.S. drinking water supplies for the years 1962 to 1967 was prepared by the U.S. Department of Interior. Levels of nickel ranged from 1 to 450 μg/l, with a mean of 34.2 μg/l. The frequency of detection was 4.6 percent.

Levels of nickel have been reported for 969 public water supplies in 8 metropolitan areas for the years 1969 to 1970. The average value was 4.8 μg/l and the maximum was 75 μg/l.

Health Effects. The absorption of dietary nickel from the gastrointestinal tract appears to be quite low with the majority of nickel excreted in the feces. Laboratory studies have demonstrated depressed body weight gain, alterations in hematology parameters, cytochrome oxidase activity and iron contents of organs following high dose oral exposure.

Insufficient data are available for the derivation of 1-day assessments for nickel. The available short-term (1–15 days) toxicity studies of nickel using the oral route of exposure reported only acute effects (LD_{50}).

The 10-day assessments for the 10 kg child and 70 kg adult were based on an animal study (Whagner, 1973. Effects of Dietary Nickel on Enzyme Activities and Mineral Content in Rats. Toxicol. Appl. Pharmacol. 25:323–331). From a NOAEL of 10 mg Ni/kg/day based on effects on weight, hematologic parameters and cytochrome oxidase activity in rats following 6 week oral ingestion, with an uncertainty factor of 100, assuming consumption of 1 liter (10 kg child) or 2 liters (70 kg adult) of water per day, 10-day values for a child and an adult of 1.0 mg Ni/l and 3.5 mg Ni/l, respectively, were calculated.

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A provisional AADI was calculated based upon a two-year feeding study in which rats were given various doses of nickel in their food (Ambrose, A.M., et al. 1976. Long Term Toxicological Assessment of Nickel in Rats and Dogs. J. Food Sci. Technol. 13:181–187). Based on a number of parameters (body weight, organ-to-body weight ratios, hematologic values, gross and histologic pathology), this study revealed a NOAEL of 5 mg Ni/kg day. A disadvantage of this study is that nickel was given in food, and absorption of metals from the gastrointestinal tract may be decreased by the presence of food (Reber, I. and Kostial, K. 1981. Bioavailability of Cadmium in Rats Fed Various Diets. Arch. Toxicol. 47:83–90; Foulkes, E. 1984. Nickel Absorption Studies in Rats; preliminary report). Using a NOAEL of 5 mg/kg/day, an uncertainty factor of 100, an absorption efficiency of 0.20 (assumed difference in absorption of nickel in food vs. water) and consumption of 2 liters of water per day, a provisional AADI of 0.350 mg/l was calculated. The available data indicate that the estimated intake of nickel from food and air are 400 μg/day and 0.6 μg/day (negligible) respectively. Factoring in this data on human exposure, a guidance level of 0.150 mg/l has been determined.

Two other studies investigating the effects of nickel ingestion on reproduction in rats (Ambrose, A.M., et al. 1976; Schroeder, H.A. and Mitchner, M. 1971. Toxic Effects of Trace Elements on the Reproduction of Mice and Rats. Arch. Environ. Health 23:102–106) were not selected for the derivation of an AADI for nickel due to several experimental design flaws.

Nickel chloride was negative for mutagenicity in *Escherichia coli* and *Bacillus subtilis*, while nickel chloride and nickel sulfate have been shown to be mutagenic in eukaryotic test systems.

The chemical form and route of exposure are important factors in determining the carcinogenic potential of nickel. Metallic nickel, nickel subsulfide and nickel carbonyl which are insoluble nickel compounds, have been shown to produce tumors through inhalation exposure in animals. Intravenous injection of nickel carbonyl has also been shown to result in liver and kidney sarcomas in animals. In humans, it has been demonstrated that the incidence of respiratory tract cancers in nickel refinery workers is significantly elevated. However, nickel has not been shown to be carcinogenic through oral exposure. The results of several studies suggest that 5 mg/l nickel in drinking water is not carcinogenic in rats and mice. Data are not available concerning the potential carcinogenic effects of ingested nickel compounds in humans. The IARC has classified nickel in Group 2A; sufficient evidence for carcinogenicity in animals and limited evidence for carcinogenicity in humans. This assessment was based upon inhalation exposure. Nickel has been classified in EPA's Group B1, according to EPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon the subsulfide and carbonyl compounds which have been shown to have limited evidence of carcinogenicity in humans and sufficient evidence in animals.

EPA's ambient water quality criteria for nickel (U.S. EPA. 1980. Ambient Water Quality Criteria for Nickel. EPA 440/5-80-060) considering ingestion of water and contaminated aquatic organisms is 0.0134 mg/l. This value was revised (February 1984, 49 FR 4551) and the proposed revised value is 0.632 mg/l based upon the same study which was used to determine the provisional AADI.

EPA has decided not to propose drinking water regulations for nickel due to inadequate toxicological data. Data problems with the provisional AADI and the data base on nickel include: (a) lack of data on the absorption of nickel; (b) lack of data on reproductive effects; and (c) the study by Ambrose, et al. (1976) requires additional statistical analyses.

Question for Comment:

1. Should a Health Advisory be developed for nickel, or are there sufficient health effects information upon which to base an RMCL?

5. Silver

Silver is a relatively rare metal with average concentrations in the earth's crust ranging from 0.07 to 0.08 ppm. In soils, the average concentration is 0.1 ppm; coal can contain slightly elevated silver concentrations, typically ranging from about 0.1 to 2 ppm. Silver is recovered primarily from gold-silver and silver-lead deposits. Background levels of silver in surface water are generally below 3 μg/l.

The major commercial uses of silver are in photography, electric/electronic components, sterling and electroplate, and alloys and solder. Environmental releases can occur during ore mining and processing, product fabrication and disposal. However, because of the great economic value of silver, recovery practices are typically used to minimize losses.

Analytical Methods. Analytical methods available for analyzing silver in drinking water include the flame atomic absorption, furnace atomic absorption and inductively coupled plasma atomic emission spectrometry techniques.

Human Exposure. There are little data on silver levels in air. Silver in ground level aerosols from various U.S. locations was found to range from 0.04 to 4.3 ng/m³, generally ranging around 1 ng/m³. Assuming a ventilation rate of 20 m³/day for the adult male, respiratory intake at levels of 1 ng/m³ would be approximately 0.02 μg/day.

The silver content of various foods has been reported to range from <10 ppb in meats to 2 ppm in seafood. The ICRP estimated the dietary intake of silver for the adult male to be 70 μg/day (including fluids).

Compliance monitoring indicate that 12 ground water supplies and one surface water supply have silver levels above 50 μg/l. Drinking water data are also available on silver from three national surveys. In the 1980 Community Water Supply Survey (CWSS), silver was found in 320 out of 677 ground water supplies (49%) at levels ranging from 0.1 to 9 μg/l. In the 1978 Community Water Supply Survey (CWSS), silver was observed in 8 out of 81 ground water supplies (10%); however, the minimum quantifiable levels was 30 μg/l. Positive values ranged from 30 to 40 μg/l. In the Rural Water Survey (RWS), silver was found in 8 out of 71 supplies sampled (11%) with a range of positive values from 20 to 80 μg/l; the minimum quantifiable level for the RWS was 20 μg/l.

In surface water supplies, silver was observed in the 1980 CWSS in 58 out of 108 supplies sampled (54%) at values ranging from 0.1 to 4 μg/l. In the 1978 CWSS, silver was found to range from 0.04 to 4.3 ng/m³, generally ranging around 1 ng/m³.

Point of use water treatment devices containing silver impregnated granular activated carbon could be the principal source of silver in drinking water at the consumer's tap.

Health Effects. The only adverse effect resulting from chronic exposure to low levels of silver in animals and humans is argyria, a blue-gray discoloration of the skin and internal organs. Argyria is markedly disfiguring and is a permanent, non-reversible effect. Argyria is the result of silver deposition in the dermis and at basement membranes of the skin and other internal organs.

One-day and 10-day assessments were not calculated for silver due to inadequate data. A provisional AADI was determined based upon several clinical reports in which humans developed argyria as a result of i.v. and oral exposure to silver (Gaul and Staud,

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1985. Clinical Spectroscopy. Seventy Cases of Generalized Argyrosis Following Organic and Colloidal Silver Medication. J. Am. Med. Assoc. 104:1387–1390; Blumberg, H. and T.N. Carey. 1984. Argyrosis: Detection of Unsuspected and Obscure Argyria by the Spectrographic Demonstration of High Blood Silver. J. Am. Med. Assoc. 102:1821–1824; and East, et al. 1980. Silver Retention, Total Body Silver and Tissue Silver Concentration in Argyria Associated with Exposure to an Anti-smoking Remedy Containing Silver Acetate. Clin. Exp. Dermatol. 5:205–211). Calculations were carried out to determine the level of silver in drinking water, apportioned over a lifetime, which would not result in argyria. The average ADI of the calculations from the 3 studies was determined to be 182 µg/day (average of 169 µg/day and 195 µg/day). This was calculated by standardizing the observed body weight to the 70 kg adult and multiplying this by the estimated total dose necessary to cause argyria and apportioning this over a lifetime (70 years). An uncertainty factor of two was used instead of the standard 10-fold uncertainty factor for the following reasons: the 10-fold uncertainty factor is applied to humans to account for possible sensitive individuals in the general population. The studies used in the ADI calculations involved sensitive individuals and thus an uncertainty factor less than 10 is warranted. In addition, the ADI calculations are extremely conservative because the estimated dose which caused argyria in 2 to 3 years is being apportioned over a lifetime of 70 years. An uncertainty factor less than 10 would be sufficiently protective in this instance. Consumption of 2 liters of water per day was factored in, resulting in a provisional AADI of 0.090 mg/l.

There is no evidence that exposure to silver results in mutagenic or carcinogenic effects. The IARC have not classified silver for potential carcinogenicity. Silver has been classified in EPA's Group D, according to EPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon inadequate data in animals and humans.

The current MCL for silver, under the National Interim Primary Drinking Water Regulations, is 0.05 mg/l. This level was based upon one gram of silver resulting in argyria. EPA's ambient water quality criteria for silver (U.S. EPA. 1980. Water Quality Criteria for Silver. EPA 440/5-80-071) is 0.050 mg/l, in agreement with the NIPDWR. The WHO has not set a guideline for silver in drinking water.

The only adverse effect from exposure to silver is argyria, which does not impair the functioning of the body or other physiological problems. Thus, argyria is considered a cosmetic effect and not an adverse health effect and an RMCL is not proposed.

Question for Comment:

1. Is it appropriate to consider argyria to be a cosmetic effect or is there evidence that argyria is an adverse health effect? Should an RMCL be proposed for silver? Should a secondary regulation be proposed for silver?

6. Sulfate

Sulfate (SO^{2-}) is a divalent anion found in almost all natural waters. It also occurs frequently in rainfall, particularly from air masses of metropolitan areas where sulfate is released from combustion of fossil fuels. An important terrestrial source is evaporite sediment, from which magnesium, sodium and calcium sulfate may be leached. Metallic sulfides such as iron pyrites are found in both sedimentary and igneous rocks. Sulfates are released through oxidation in the weathering process. Household waste including detergents add sulfate to sewage. Industrial effluents from tanneries, steel mills, sulfate-pulp mills, and textile plants are other significant sources to surface water.

Human Exposure. Data on levels of sulfate in the ambient air are available from EPA's Office of Air Quality Planning and Standards. A summary of atmospheric concentrations (1975) for selected locations in the U.S. is presented below.

	Number of samples	Concentration SO_4 (µg/m³)	
		Arithmetic mean	Maximum
California (Oakland)	25	4.5	10.2
Ohio (Cincinnati)	25	10.2	22.0
Texas (Lubbock)	10	5.0	25.4
Virginia (Richmond)	25	10.5	27.5

Data were not available on the occurrence of sulfate in foods.

The CWSS, conducted in 1970, examined 969 drinking water supplies in the U.S. Levels of sulfate were reported to range from <1 to 770 mg/l (median = 4.8 mg/l). Of the sampled water supplies, 3 percent had concentrations exceeding 250 mg/l. Analysis of the interstate carrier water supply systems (1975) involved examination of 625 finished drinking water supplies. In 3.4 percent of the sampled systems, levels of sulfate exceeded 250 mg/l. The maximum measured concentration was 978 mg/l.

Analytical Methods. Analytical methods available for analyzing sulfate in drinking water include the turbidimetric and automated colorimetric techniques.

Health Effects. The only adverse effects in animals and humans noted from exposure to high levels of sulfate are diarrhea and dehydration. Soluble sulfate salts are absorbed from the intestine and distributed throughout the body, with large doses being incompletely absorbed, resulting in cathartic effects. Sulfate is continuously excreted in the urine.

In humans, the concern is for transient situations as individuals will become acclimated to high levels of sulfate in a short period of time and diarrhea and dehydration do not occur after the initial acclimation period. Infants appear to be more sensitive to sulfate than adults, with several cases of diarrhea and gastroenteritis reported in infants consuming formula containing sulfate at levels ranging from 630–1150 mg/l.

In areas of the country with high sulfate concentrations in their drinking water supplies, no adverse health effects in older children and adults have been associated with exposure to sulfate over lifetime exposure. In addition, insufficient toxicological data are available to calculate an AADI based upon short-term effects in adults or infants. Thus, a provisional AADI will not be calculated for sulfate. Instead, a guidance level of 400 mg/l, to protect infants, was derived based upon case histories which suggest that parents should not use water from wells with sulfate concentrations greater than 400 to 500 mg/l for preparation of infant formula.

In addition, 250 mg/l is presented as a guidance level based upon aesthetic considerations. This value would serve to limit the intake and protect the high risk population (infants).

The current EPA secondary drinking water standard for sulfate is 250 mg/l based upon aesthetic effects. The WHO guidline for sulfate is 400 mg/l based upon taste considerations. The U.S. Army has recommended limits for sulfate of 300 or 100 mg/l, based upon personnel who consume up to 15 liters of water per day (100 mg/l) and those who consume up to 5 liters of water per day (300 mg/l) (Scofield, R. and Hatch, D. Criteria and Recommendations for Standards for Sulfate in Military Field Water Supplies. Univ. of Calif.).

An RMCL and primary drinking water regulations will not be developed for sulfate as there are not sufficient data at the present time on which to set a level for health protection. However,

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exposure to sulfate at high levels does present a health concern for transient exposure situations and EPA will reconsider an RMCL in the future based upon new data and research results, as available.

Question for Comment:

1. It is appropriate to derive an advisory for sulfate based upon the high risk population or should an RMCL be developed?

7. Sodium

Sodium (atomic number 11, atomic weight 22.99) is a light silvery-white alkali metal that composes 2.36 percent (by weight) of the earth's crust. Sodium does not occur as the free element in nature but in the form of halides, silicates, and carbonates. Sodium is the principal cation in the hydrosphere. It is derived geologically from the leaching of surface and underground deposits of salts (e.g., sodium chloride) and from the decomposition of sodium aluminum silicates and similar minerals.

Analytical Methods. Analytical methods for analyzing sodium in drinking water include the direct aspiration atomic absorption, furnace atomic absorption and flame photometry.

Human Exposure. While there is considerable information on the sodium content of foods, few studies are available of total daily sodium-ion intake for healty adults. Data that have been reported are based on measurement of sodium excretion in urine over 12 and 24 hour periods. Reported mean 24-hour levels range from 1,800 to 6,600 mg. A recent estimate for infants is 69 to 92 mg/kg/day.

The sodium ion is a major constituent of natural waters. Human activities also contribute sodium to water supplies, primarily through the use of sodium chloride as a deicing agent, and the use of washing products. A survey of 2,100 finished water supplies was conducted from 1963 to 1969 by the U.S. Public Health Service. Levels of sodium ion were found to range from 0.4 to 1,900 mg/l. Of the supplies having sodium ion concentrations, 42 percent exceeded 20 mg/l. Levels in 5 percent of the supplies were greater than 250 mg/l. In a later study (1975) of interstate carrier water supply systems, sodium ion concentrations in 630 systems were found to range from <1 to 402 mg/l. A total of 42 percent had levels exceeding 20 mg/l; 3 percent had levels greater than 200 mg/l.

Health Effects. The first epidemiologic study of the relationship of sodium in drinking water and blood pressure in the United States was undertaken by Calebrese and Tothill (1977. Elevated Pressure and High Sodium Levels in the Public Drinking Water. 32:200–202; Ibid. 1978. 34:157–159). These investigators compared blood pressure distributions among tenth graders in two Massachusetts communities exposed to low (8 mg/l) and high (107 mg/l) levels of sodium in drinking water. Students living in the high-sodium community exhibited higher blood pressures than their counterparts in the low-sodium community. In the Netherlands (Hofman, et al. 1980. Increased Blood Pressure in School Children Related to High Sodium Levels in Drinking Water. J. Epidemiol. Comm. Hlth. 34:179–181), a study reported findings which supported the hypothesis that sodium intake influences blood pressure.

Various epidemiologic studies that failed to confirm an association between drinking water sodium and blood pressure also have been reported (Pomrehn, et al. 1983. Community Differences in Blood Pressure Levels and Drinking Water Sodium. Am. J. Epidemiol. 118:60–71; Punsar, et al. 1978. Coronary Heart Disease and Drinking Water. J. Chron. Dis. 28:259–287; Bierenbaum, et al. 1975. Possible Toxic Water Factor in Coronary Heart Disease. Lancet. 1:1009–1010; Hallenbeck, et al. 1981. High Sodium in Drinking Water and Total Sodium Intake on Blood Pressure. Am. J. Epidemiol. 114:817–826; Faust, 1982. Effects of Drinking Water and Total Sodium Intake on Blood Pressure. Am. J. Clin. Nutr. 35:1459–1467; Armstrong, et al. 1982. Water Sodium and Blood Pressure in Rural School Children. Arch. Environ. Hlth. 37:235–245). Willett assessed epidemiologic data relating to the hypothesis that sodium in drinking water causes a clinically significant elevation of blood pressure among school children in Massachusetts and concluded that the data should be interpreted with extreme caution (1981. Drinking Water Sodium and Blood Pressure: A Cautious View of the 'Second Look'. Am. J. Pub. Hlth. 71:729–732). Based on the available studies, it appears that insufficient evidence is available to conclude whether or not sodium in drinking water causes an elevation of blood pressure in the general population.

It has been estimated that food accounts for approximately 90 percent of the daily intake of sodium whereas drinking water contributes up to the remaining 10 percent.

In order to afford protection to a segment of the U.S. population on a sodium-restricted diet, in 1969, the American Heart Association (AHA) recommended a level of 5 mg of sodium per 8 ounces of water or 20 mg/l. The basis for AHA's recommended level of sodium was a nutritionally adequate diet requiring 500 mg sodium per day. Of the 500 mg, 440 mg would be naturally occurring in food. An additional 60 mg would be from non-nutritional intake such as drugs, water, and incidential intake sources.

In 1979, the National Interim Primary Drinking Water Regulations did not contain an MCL for sodium because the available data did not support any particular level of sodium in drinking water. In lieu of the absence of an MCL for sodium, EPA supported the AHA's recommended level of 20 mg sodium/l in drinking water. In 1980, EPA amended the National Interim Primary Drinking Water Regulations, requiring community water systems to monitor and report sodium levels in finished drinking water.

EPA is not proposing an RMCL for sodium due to insufficient data showing an association between sodium in drinking water and hypertension in the general population and because of the normally minor contribution of drinking water to the total dietary intake of sodium. EPA is suggesting a guidance level for sodium of 20 mg/l in drinking water for the high risk population as recommended by the AHA, since drinking water meeting this goal would not present a sodium-related hazard to those segments of the population thought to be at high risk (e.g., individuals with genetic predisposition to hypertension, pregnant women, hypertensive patients). EPA will reconsidered the development of an RMCL for sodium if additional data become available. A secondary standard based upon aesthetic effects will be prepared for sodium.

Questions for Comment:

1. Are there sufficient data available to develop an RMCL instead of a health advisory for sodium in drinking water?

2. Does the proposed health advisory for sodium provide protection to high risk populations?

8. Antimony, Beryllium, Thallium and Vanadium

Antimony, beryllium, thallium and vanadium were included in the list of IOCs under consideration for Revised Regulations in the ANPRM. Preliminary analysis indicated limited potential for drinking water exposure causing a significant risk from these substances. Data collection efforts on occurrence/human exposure and potential health effects have not yet been completed on these substances and these four IOCs will be considered in later Phases of the Revised Regulations.

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9. Zinc

Zinc was also included in the list of IOCs under consideration for Revised Regulations in the ANPRM. The Agency has not identified any adverse health effects that are caused by zinc. The NAS Safe Drinking Water Committee (*Drinking Water and Health*, 1977, Vol. I) concluded that, "zinc is an essential nutrient for humans. There is evidence of borderline deficiencies of the element in children in the United States as well as in other parts of the world . . . . The possibility of detrimental health effects arising from zinc consumed in food and drinking water is extremely remote."

Thus, EPA has concluded that potential adverse health effects will not arise from zinc in drinking water and this compound is not being considered for regulation at the present time.

VIII. Synthetic Organic Chemicals: RMCL's

The ANPRM (48 FR 45502) listed a total of 43 synthetic organic chemicals (SOCs) that were being considered for inclusion in the NPDWR. Inclusion of specific SOCs on the list was based upon the occurrence or potential occurrence of the SOC in drinking water and the potential health effects of exposure to that SOC. Inclusion in the list did not necessarily mean that regulations would be developed for the SOC but that these were the SOCs currently being considered; other SOCs not listed could also be considered and included in the NPDWR. Selection of SOCs for the NPDWR is based upon an analysis of occurrence and potential occurrence, the significance of potential human exposure, associated health effects of exposure and other pertinent factors.

EPA is today proposing to regulate 28 of the 43 SOCs in the ANPRM; five of the SOCs were determined to be inappropriate for regulation due to such factors as lack of potential occurrence in drinking water, lack of actual occurrence data, or insufficient health effects data. Short- and longer-term toxicology assessments have been developed for those five SOCs for which regulations are not appropriate; these assessments may be converted to formal Health Advisories. In addition, 12 SOCs of the 43 SOCs will be reconsidered in later phases of the Revised Regulation development as additional data become available (see Table 1).

RMCLs are proposed for 28 SOCs for which the Administrator has determined that: (1) Analytical methods are available, (2) exposure to any of these SOCs "may have any adverse effect upon the health of persons" and, (3) they occur or are likely to occur in drinking water.

Below are: (1) A summary of the availability of analytical methods, and (2) summaries per SOC of analytical methods, occurrence/exposure and toxicology.

In the MCL proposal, EPA will propose the analytical methods that have been determined to be economically and technologically feasible. In the toxicology discussion for each SOC, the acute and chronic toxic effects of exposure along with any carcinogenicity data are summarized. When data are available, adjusted acceptable daily intakes (AADIs) based on non-carcinogenic effects are determined for long term exposure to the SOCs. In addition, short-term exposure is also considered and short-term assessments are determined for 1-day and 10-day exposures. These assessments are provided for both SOCs for which RMCLs are proposed and for those SOCs for which regulations do not appear to be appropriate. A summary of health-related guidelines prepared by other groups and organizations is provided for each SOC. Values that have been calculated by the World Health Organization (WHO), the National Academy of Sciences (NAS), EPA's Office of Water Regulations and Standards (OWRS, Water Quality Criteria) and EPA's Office of Pesticide Programs (OPP) have been included. In several instances, these values differ from the proposed AADIs. This is due to several factors, including the use of different uncertainty factors, and reinterpretation of data and varying assumptions. In addition, new data may have become available over the years which has resulted in the derivation of an AADI which differs from older calculated values. Taste and odor threshold values also have been included for certain contaminants.

A summary of the RMCLs and AADIs is presented in Table 12. Risk estimates have been projected using calculation models for SOCs for which data are available and are summarized in Table 13. Short-term assessments and provisional AADIs for SOCs for which RMCLs are not proposed are summarized in Table 14 and short-term assessments for SOCs for which RMCLs are proposed are summarized in Table 15.

A. Availability of Analytical Methods

EPA approved analytical methods are available for most of the SOCs being considered in this RMCL proposal. These methods may involve gas chromatography (GC), gas chromatography/mass spectrometry (GC/MS), and and high pressure liquid chromatography (HPLC).

Purge and trap methods are available for those SOCs that are volatile. Those compounds which are methylene chloride extractable may be analyzed by 600 series methods (i.e., EPA methods for analysis of priority pollutants). Other compounds may be analyzed by newer methods developed recently but not yet approved by the Agency.

Since a number of the 600 series methods have been recently applied to drinking water samples in addition to waste effluent samples, multi-laboratory method validation data are available for many of the compounds in this proposal. Multi-laboratory data from performance evaluation studies are also available for some compounds, using reagent water. For the newer methods, only single laboratory, single operator performance data are available.

TABLE 12.—PROPOSED RMCL'S AND AADI'S FOR SOC'S PROPOSED FOR REGULATION

SOC	Safety factor	AADI [1] (mg/l)	Proposed RMCL (mg/l)
Acrylamide	100	* 0.007	0
Alachlor	NA	NA	0
Aldicarb, aldicarb sulfoxide and aldicarb sulfone	100	* 0.042	0.009
Carbofuran	100	0.18	0.036
Chlordane	100	0.00	0
cis-1,2-Dichloroethylene	1,000	0.35	0.07
DBCP	NA	NA	0
1,2-Dichloropropane	NA	NA	0.000
o-Dichlorobenzene	1,000	3.12	0.62
2,4-D	100	* 0.35	0.07
EDB	NA	NA	0
Epichlorohydrin	100	0.075	0
Ethylbenzene	1,000	* 3.4	0.68
Heptachlor	1,000	* 0.0005	0
Heptachlor epoxide	1,000	* 0.001	0
Lindane	1,000	* 0.01	0.0002
Methoxychlor	100	1.7	0.34
Monochlorobenzene	1,000	* 2.0	0.08
PCBs	NA	NA	0
Pentachlorophenol	100	1.1	0.22
Styrene	1,000	* 7.0	0.14
Toluene	100	10.1	2.0
2,4,5-TP	NA	0.35	0.052
Toxaphene	NA	NA	0
trans-1,2-Dichloroethylene	1,000	0.35	0.07
Xylene	1,000	* 2.2	0.44

[1] Does not consider carcinogenicity potential.

* These AADIs are termed "provisional" as they were determined from studies of less than lifetime duration (approximately 2 years for an animal study).

Note.—NA = Not available.

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TABLE 13.—RISK ESTIMATES FOR SOC'S FOR WHICH DATA WERE AVAILABLE

SOC	Projected upper limit, carcin. lifetime cancer risk (10^{-6}), consumption in drinking water (µg/l)		EPA Classification	IARC Classification
	CAG	NAG		
Acrylamide	NA	NA	D[1]	Not classified.
Alachlor	[2] 0.15	NA	D[1]	Group 3.
Chlordane	0.02	0.005	D[1]	Group 3.
DBCP	0.0003	NA	D[1]	Group 2B.
1,2-Dichloropropane	0.38	NA	C	Not classified.
Diquat	[2] 2.2	NA	D[1]	Group 2B.
EDB	0.00005	0.11	D[1]	Group 2B.
Epichlorohydrin	2.54	NA	D[1]	Group 2B.
Heptachlor	0.00104	NA	D[1]	Group 3.
Heptachlor epoxide	0.00065	NA	D[1]	Not classified.
Hexachlorobenzene	0.02	0.04	D[1]	Group 2B.
Lindane	0.005	0.005	C	Group 3.
Methoxychlor	NA	2.35	C	Not classified.
PCBs	0.0079	0.16	D[1]	Group 2B.
Styrene	NA	NA	C	Group 3.
Toxaphene	0.02	NA	D[1]	Group 2B.

[1] Calculated by EPA's Office of Pesticide Programs.
[2] Times 10^{-3}.
Note.—NA—Not available.

TABLE 14.— SHORT-TERM ASSESSMENTS AND PROVISIONAL AADI'S FOR SOC'S FOR WHICH RMCLS ARE NOT PROPOSED

SOC	1 day (mg/l)		10-Day (mg/l)		Provisional AADI (mg/l)
	Child	Adult	Child	Adult	
Acrylamide	NA	NA	NA	NA	[1] 0.75
m-Dichlorobenzene	0.9	31.2	0.9	31.2	NA
Diquat	[2] 1.0	[2] 3.5	[2] 1	[2] 3.5	[2] 3.5
Endrin	0.02	0.07	0.0005	0.02	0.0010
Hexachlorobenzene	0.05	0.17	0.05	0.17	0.005

[1] Calculated by the NAS.
[2] Times 10^{-3} mg/l.
[3] Times 10^{-3} mg/l.
[4] Times 10^{-3} l.
Note.—NA—Not available.

TABLE 15.—DRAFT SHORT-TERM ASSESSMENTS FOR SOCS FOR WHICH RMCLS ARE PROPOSED

SOC	1-day (mg/l)		10-day (mg/l)		Longer-term (mg/l)[1]	
	Child	Adult	Child	Adult	Child	Adult
Acrylamide	1.5	5.25	0.3	1.0	0.02	0.04
Alachlor	15	52.5	15	52.5	NA	NA
Aldicarb, aldicarb sulfoxide and aldicarb sulfone	0.012	0.042	0.012	0.042	0.012	0.042
Carbofuran	0.05	0.175	NA	NA	0.05	0.18
Chlordane	NA	NA	0.005	0.22	NA	NA
cis-1,2-Dichloroethylene	4.0	14	1.0	3.5	1.0	3.5
DBCP	0.2	0.7	0.05	0.17	NA	NA
1,2-Dichloropropane	NA	NA	0.09	0.31	NA	NA
o-Dichlorobenzene	NA	NA	NA	NA	9.0	31.2
2,4-D	1.1	3.85	0.3	1.1	NA	NA
EDB	NA	NA	0.005	0.027	NA	NA
Epichlorohydrin	NA	NA	0.14	0.5	0.002	0.016
Ethylbenzene	21	72	2.1	7.2	NA	NA
Heptachlor	NA	NA	0.01	0.036	NA	NA
Heptachlor epoxide	NA	NA	NA	NA	NA	NA
Lindane	NA	NA	1.2	4.2	0.002	0.12
Methoxychlor	6.4	22.4	2.0	7.0	NA	NA
Monochlorobenzene	NA	NA	1.6	6.3	9	30.5
PCBs	NA	NA	NA	NA	NA	NA
Pentachlorophenol	1.0	3.5	0.3	1.1	NA	NA
Styrene	27	94.5	20	70	20	70
Toluene	15	53	6	21	NA	NA
2,4,5-TP	NA	NA	0.2	0.75	NA	NA
Toxaphene	0.5	1.75	0.05	0.20	NA	NA
trans-1,2-Dichloroethylene	2.7	9.5	1.0	3.5	1.0	3.5
Xylene	12	42	NA	NA	7.0	27.3

Note.—NA—Not available. Adequate dose-response data were not available. Assessment derived for the next longer duration of exposure is considered to be protective in these cases.
[1] The longer-term number are not discussed in the section on the individual chemicals in the FR Notice but are discussed in the Health Advisory documents.

Table 16 lists the analytical methods presently available for the SOCs. There is no standardized EPA method for acrylamide. The analysis of acrylamide has been reported in the published literature using bromination of the double bond followed by gas chromatography analysis using an electron capture detector. Further research on this approach or an alternate procedure is required to develop a suitable analytical method for the determination of acrylamide in drinking water. Additional research is required on the development of multi-contaminant monitoring methods for the SOCs, including the use of capillary GC and capillary GC/MS techniques.

TABLE 16.—ANALYTICAL METHODS FOR SOCS

Compound	Method[1]
Acrylamide	No standardized EPA method.
Alachlor	Solvent extraction GC.
Aldicarb	HLPC; 531.
Aldicarb sulfoxide	Solvent extraction GC; 610.
Aldicarb sulfone	Solvent extraction HPLC; 531.
Carbofuran	Solvent extraction GC; 531.
Chlordane[2]	Solvent extraction GC; 505. Solvent extraction GC/MS; 525.
DBCP	Purge & Trap GC; 502.1. Purge & Trap GC/MS; 524.
cis- and trans-1,2-Dichloroethylene.	Purge & Trap GC; 502.1.
trans-1,2-Dichloroethylene	Purge & Trap GC; 524.
o-Dichlorobenzene	Purge & Trap GC; 502.1. Purge & Trap GC/MS; 524.
m-Dichlorobenzene	Purge & Trap GC; 502.1. Purge & Trap GC/MS; 524.
1,2-Dichloropropane	Purge & Trap GC; 502.1. Purge & Trap GC; 524. Purge & Trap GC/MS; 524. Purge & Trap GC/MS; 524.
2,4-D	Derivatization GC; 515.
EDB	Purge & Trap GC; 502.1.
Endrin	Solvent extraction GC; 505.
Epichlorohydrin	Direct injection GC.
Ethylbenzene	Purge & Trap GC; 502.
Heptachlor	Solvent extraction GC; 505. Solvent extraction GC/MS; 525.
Hexachlorobenzene	Solvent extraction GC; 612.
Lindane	Solvent extraction GC; 505.
Methoxychlor	Solvent extraction GC; 505.
Monochlorobenzene	Purge & Trap GC; 502.1. Purge & Trap GC/MS; 524.
Pentachlorophenol	Solvent extraction GC; 515. Solvent extraction GC-MS; 525.
PCBs[3] PCB-1242	Solvent extraction GC; 505.
Simazine	Solvent extraction GC; 507.
Styrene	Purge & Trap GC; 502.1.
Toluene	Purge & Trap GC; 502.
Toxaphene[2]	Solvent extraction GC; 505.
2,4,5-TP	Derivatization GC; 515.
2,3,7,8-TCDD	Solvent extraction GC/MS; 613.
Xylenes: para-, meta-, ortho-.	Purge & Trap GC; 502.1.

[1] multiple peak response.
[2] PCBs included are 1260, 1254, 1248, 1242, 1232, 1221, and 1016.
[3] Series 500 methods are EPA methods for analysis of priority pollutants. Series 600 methods are EPA methods for analysis of drinking water contaminants.
NA—not available. FID—flame ionization detector. ECD—electron capture detector.

B. Proposed RMCLs

1. Acrylamide

Acrylamide (propenamide, CAS #79-06-1) is used primarily as a starting material for the manufacture of water soluble polymers employed to enhance oil and water recovery from wells, as flocculents in potable and waste water treatment, food processing, in paper making, dye application, adhesives, soil conditioners and permanent press fabrics. Acrylamide is extremely soluble in water (2.15×10^6 mg/l) and is soluble in alcohol, ether and acetone.

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Analytical Methods. No EPA approved analytical method is presently available for acrylamide, although a research method is now available.

Human Exposure. Acrylamide monomer is a common contaminant of polyacrylamide used in food production and as a coagulant aid in the water treatment process. Polyacrylamide may be contaminated with up to 0.05 percent acrylamide monomer, which upon leaching, could yield a 0.5 µg/day intake by humans from drinking water alone. Technical grades of polyacrylamide, having higher levels of acrylamide monomer, are used in drilling new potable water wells. These acrylamide residues may remain in the surrounding soils. Technical grades of polyacrylamide are used in mine management and reclamation and have been reported to have contaminated local water supplies with acrylamide monomer. No monitoring data are available to describe the frequency or level of human exposure via food, air or drinking water. While monitoring for acrylamide has been limited, the potential for occurrence in drinking water exists because of its use as an additive in drinking water treatment processes.

Health Effects. The principal toxic effect from exposure to acrylamide (monomer) over any duration of time and by any route in animals is peripheral neuropathy. Subchronic studies have demonstrated a variety of effects, including atrophy of skeletal muscles in the hind quarters, testicular atrophy and weakness in the limbs. Long-term exposure to acrylamide also has been shown to result in neurotoxic effects and weakness in the hind quarters. Case reports suggest that similar effects occur in the human following exposure via the dermal, oral or inhalation routes. Recent evidence shows that acrylamide is carcinogenic in mice and rats when administered by one of several routes: oral, topical or intraperitoneal.

No adequate dose-response data representing the oral route of exposure are available from which to develop short-term assessments. However, in the light of substantial chemical disposition evidence showing that acrylamide is absorbed rapidly and completely by virtually any route of exposure, it is considered acceptable to use data generated following exposure via these other routes. The 1-day assessments were derived from a NOAEL of 15 mg/kg identified in an animal study (Miller, et al. 1983. Altered Retrograde Axonal Transport of Nerve Growth Factor After Single and Repeated Doses of Acrylamide. Toxicol. Appl. Pharmacol. 69:95–101). While measuring the rate of retrograde axonal transport of indicated nerve growth factor in rats treated with single intraperitoneal doses, the authors showed that significant inhibition of transport caused at or above doses of 25 mg/kg, while no significant changes were seen at or below 15 mg/kg. The 1-day assessments were derived by applying an uncertainty factor of 100 to the NOAEL of 15 mg/kg, yielding a value for the 10 kg child of 1.5 mg/l which assumes consumption of 1 liter of water per day and a value for the 70 kg adult of 5.25 mg/l which assumes consumption of 2 liters of water per day.

The 10-day assessments were derived from a NOAEL identified in a drinking water study (Garshnabi, et al. 1978. Results of Palatability (12-day) and Tolerance (21-day) Studies on Acrylamide Monomer Administered in the Drinking Water of Rats. Dow Chemical Company. Unpublished report). The monomer was administered in drinking water at levels of 0, 1, 3, 10 or 30 mg/kg bw/day for 21 consecutive days. Based upon histological examination of peripheral nerves at both the light and electron microscopic levels, it was determined that effects occurred at the two higher doses, while no significant changes were apparent at the two lower doses. A NOAEL of 3 mg/kg/day thus was identified. The 10-day values were derived by applying an uncertainty factor of 100 to the NOAEL, yielding a level of 0.3 mg/l for the 10 kg child which assumed consumption of 1 liter of water per day and a level of 1.05 mg/l for the 70 kg adult which assumes consumption of 2 liters of water per day.

The study selected for the derivation of a provisional AADI was a subchronic study in which rats were administered daily doses of 0, 0.05, 0.2, 1, 5 or 20 mg/kg in their drinking water (Burch, et al. 1980. J. Environ. Path Tox. 4:157–182). The parameters measured were gross neuropathy as observed by the limb splaying method, ultrastructural examination of peripheral motor nerves, hematology, clinical chemistry and body and organ weights. On the basis of the most sensitive measure of toxicity, the ultrastructural examination, it was concluded that 0.2 mg/kg was the NOAEL. Using this NOAEL, an uncertainty factor of 100 based upon an animal study with the NOAEL identified, an uncertainty factor of 10 to convert from subchronic to chronic exposure and consumption of 2 liters of water per day, a provisional AADI of 0.007 mg/l was determined.

Acrylamide did not elicit mutagenic activity in the Ames test (both with and without microsomal activation) or in the hepatocyte culture DNA repair assay (Bull, et al. 1984. Carcinogenic Effects of Acrylamide in Sencar and A/J mice. Cancer Res. 44:107–111; Miller, et al. 1984. Lack of Genotoxicity of Acrylamide Using the Hepatocyte Primary Culture (HPC)/DNA Repair Test. Abstract No. 138. Presented at the 1984 meeting of the Society of Toxicology, Atlanta, GA. The Toxicologist 4(1):35). Chromosome aberrations were noted in the spermatogonia of mice exposed to 75 mg/kg/day in the diet for 2–3 weeks, but bone marrow cells were not affected (Shiraishi, Y. 1978. Chromosome Aberrations Induced by Monomeric Acrylamide in Bone Marrow and Germ Cells of Mice. Mut. Res. 57:313–324). Both the marrow cells and spermatogonia showed a striking decrease in mitotic index following a single intraperitoneal dose of 50 to 150 mg/kg.

Only one published study (Bull, et al. 1984. Carcinogenic Effect of Acrylamide in Sencar and Strain A/J Mice. Cancer Research. 44:107–111) is available which addresses the carcinogenic effects of acrylamide. This study showed that acrylamide acted as a tumor initiator in the skin of the female Sencar mouse when administered orally, topically or by intraperitoneal injection. Acrylamide also increased the yield of lung adenomas in strain A/J mice when given orally. In addition, preliminary data from another study show that acrylamide causes a significant increase in tumor incidences at several sites in both male and female rats exposed to acrylamide in their drinking water. A quantitative risk assessment has not been performed on acrylamide, pending submission of the data to the Agency for review. Acrylamide has been classified in EPA's Group B2, according to EPA's Proposed Guidelines for Carcinogen Risk Assessment based upon the positive results in studies in mice and rats.

The data strongly suggest that acrylamide monomer is carcinogenic in animal species. Because of these potential adverse health effects and the fact that acrylamide is likely to be occurring in water supplies, due to its use as an additive in the drinking water treatment process, EPA is proposing to regulate this contaminant. The RMCL will be based upon carcinogenic effects and an RMCL of zero is proposed.

2. Alachlor

Alachlor [(2-chloro-2',6' diethyl-n-(methoxymethyl) acetanilide); CAS # 15972-60-8] is a herbicide used primarily

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on corn and soybeans. Alachlor is slightly soluble in water and can enter water systems by runoff from agricultural fields into surface water or by leaching downward through soil to ground water.

Analytical Methods. Analytical methods available for analyzing alachlor in drinking water include the solvent extraction-gas chromatography technique.

Human Exposure. Alachlor is registered primarily for use on beans, corn, cotton, peanuts, peas, sorghum, soybeans, sunflowers and woody ornamentals. Tolerances for alachlor have been established for eggs, milk, and the fat, meat and meat byproducts of cattle, goats, hogs, horses, poultry and sheep.

Estimates of dietary exposure to alachlor have been calculated based upon the estimated level of each food item for which a tolerance has been set in the typical diet. These calculations suggest a total dietary exposure of 4×10^{-4} mg/kg/day.

There are no data to indicate the presence or absence of alachlor in ambient air at production and use sites, or in urban air.

Five regional studies conducted in the midwestern United States provide evidence of the occurrence of alachlor in surface and ground water. During the spring and summer of 1981, one study showed alachlor to be present in 80 percent of the samples (293) taken from 12 different streams. The maximum concentration observed was 104 µg/l. During 1982–1984, alachlor was also shown to be present in surface water sources, at a maximum concentration of 75 µg/l.

In 1980, 2 out of 14 wells sampled in Nebraska were found to contain alachlor levels of approximately 0.04 µg/l. Recent results from drinking water wells in Iowa (1982–1984) showed levels of alachlor up to 16 µg/l, with levels typically less than 3 λg/l. Drinking water samples of tap water from Ohio have also detected alachlor at similar levels. In Maryland, 4 out of 30 wells found to contain alachlor at levels approximately 0.4 µg/l.

Other drinking water supplies have been found to have detectable levels of alachlor. Selected surface water supplies in one State have been reported to contain as much as 14.3 µg/l alachlor during spring and summer months. In some instances the drinking water level differed little from the level found in raw water. Alachlor was detected in 4 out of 104 samples from NSP for organics. Levels ranged from 0.1–0. 9 µg/l.

Health Effects. Alachlor exhibits relatively low acute toxicity by the oral (rat LD_{50} = 0.93 g/kg), dermal (rabbit LD_{50} = 11.3 g/kg), or inhalation (rabbit LC_{50} > 5.1 ml/l) routes of exposure (Monsanto. 1978a. Acute Oral Rat, Acute Dermal Rabbit. Unpublished study received 1978; CDL:241273; Monsanto. 1981c. Acute Inhalation LD_{50} Rat. Unpublished study received 1981; CDL:246053). Although alachlor is a skin sensitizer and causes ocular lesions upon chronic exposure, the technical product has only slight skin and eye irritation potential after an acute exposure (Monsanto. 1978b. Primary Eye and Primary Dermal Irritation Rabbit. Unpublished study received 1978; CDL:241273; Monsanto. 1984a. Dermal Sensitization Guinea Pig. Unpublished study received 1984; CDL:252772).

A two year rat feeding study in the Long-Evans strain of rat showed alachlor to be toxic at all doses tested; 14.0, 42.0 and 129.0 mg/kg/day (Monsanto. 1982. Environmental Fate of Microencapsulated Alachlor: Vol. I and II. Unpublished study received 1982. CDL:070841). The principal toxic effects of concern were heptatotoxicity and an ocular lesion, referred to as the uveal degeneration syndrome (UDS). UDS is characterized in its mildest form by free floating irideal and chorod'al pigment in the ocular chamber and pigment deposition on the cornea and lens. In its most severe form, the syndrome is characterized by bilateral degeneration of the iris and diminution of the size of the ocular globe with secondary total cataract formation.

A follow-up two-year feeding study in the same strain of rat was conducted at 0.5, 2.5 and 15.0 mg/kg/day (Stout. 1983. A Chronic Study of Alachlor Administered in Feed to Long-Evans Rats. Unpublished study received 1984. CDL:252468). There was a small increase at the high dose in animals exhibiting the initial stage of UDS, specifically molting of retinal pigmentation.

No duration specific data are available to derive a one-day health advisory; therefore, it is recommended that the ten-day health advisory be applied for the one-day assessment.

The ten-day assessment is derived from a teratogenicity study in the rat reported by Rodwell and Tracher, 1980 (Teratology study in rats. IRDC No. 401–050; IR–79–020. Unpublished study including submitter study, received Oct. 16, 1980, under EPA Reg. No. 524–365, prepared by International Research and Development Corporation, submitted by Monsanto Agricultural Products Co., St. Louis, MO. CDL: 252570). No teratogenic effects were observed at 400 mg/kg/day; however, this level did produce maternal and fetotoxicity. A maternal and fetotoxic NOAEL was established at 150 mg/kg/day when alachlor was administered to rats on day 6 through 15 of gestation. Using the NOAEL of 150 mg/kg/day, a 10 day health advisory for a 10 kg child is 15 mg/l and for a 70 kg adult is 52.5 mg/l.

Alachlor feeding studies have demonstrated oncogenic effects which include lung tumors in mice, and stomach, thyroid, and nasal turbinate tumors in rats. Two chronic feeding studies were conducted in the Long Evans strain of rat with alachlor. In the first study, the technical material was stabilized with epichlorohydrin during the first year of the study (Daly. 1981b. An Eighteen-Month Chronic Feeding Study of Alachlor in Mice. Unpublished study received 1981. CDL:07168–A, 070169) and fed to 90 animals/sex at dose levels of 14, 42, and 129 mg/kg/day. During the second year of this study, alachlor stabilized with epoxidized soybean oil was the test material throughout the study.

Dose-related responses were observed for tumors of the nasal turbinate of both sexes for the mid and high doses. Also, increases were observed in the incidence of malignant stomach tumors (described by the authors as neoplasms pluripotent in ability to form a mixed carcinomasarcoma-type tumor) in the high dose of both sexes ($p < 0.001$). In addition, thyroid follicular tumors (adenomas plus carcinomas) appeared to increase in both sexes at the high-dosage level with the increase being significant ($p < 0.001$) in males. The incidence of the nasal turbinate, stomach and thyroid tumors as well as other tumors, i.e., liver and brain, are considered of potential biological significance.

In the second two-year feeding study (Stout. 1983a. A Chronic Study of Alachlor Administered in Feed to Long-Evans Rats. Unpublished study received 1984. CDL:252466–7), throughout which epoxidized soybean oil (1.28%) was used as a stabilizer in the test material, three treatment groups of 50 males and 50 female Long-Evans rats received 0.5, 2.5, and 15 mg/kg/day. Data from an additional study run concurrently with the previously discussed study have recently been submitted to EPA (Stout. 1983. A Chronic Study of Alachlor Administered in Feed to Long-Evans Rats. Unpublished study received 1984. CDL:252468). This additional study used a fourth treatment group, 126 mg/kg/day, that was exposed to the new technical material (without epichlorohydrin as a stabilizer). A group was treated for five to five and one-half months and then put on a control diet

for the remainder of the two year period. This study indicates that the tumor response observed in the earlier study cannot be explained by the presence of epichlorohydrin in the test material and suggests that partial lifetime exposure (approximately one-fourth of the lifespan of the animals) resulted in a similar tumor incidence as a lifetime exposure.

The IARC have classified alachlor in Group 3; inadequate evidence for carcinogenicity in humans and inadequate evidence for carcinogenicity in animals. This classification was carried out before the results of the alachlor feeding studies were available. Alachlor has been classified in EPA's Group B2, according to EPA's Proposed Guidelines for Risk Assessment, based upon positive results in feeding studies in mice and rats.

EPA's Office of Pesticide Programs has derived estimates of risk of daily exposure to alachlor in drinking water based upon the incidence of tumors of the nasal epithelium, stomach and thyroid from an ingestion study in rats. The estimated lifetime cancer risk rates are shown in Table 13.

The available data indicate that alachlor has carcinogenic effects in animals. Alachlor has been detected in public water systems and is highly mobile in the environment. Thus, EPA is proposing to regulate this contaminant and an RMCL will be based upon carcinogenicity; an RMCL of zero is proposed.

3. Aldicarb, Aldicarb Sulfoxide and Aldicarb Sulfone

Aldicarb [2-methyl-2-(methylthio)propanal-o-([methylamino] carbonyl)oxime; CAS #116-06-3] also known as Temik, is a registered pesticide used to control insects, mites and nematodes. Aldicarb's high aqueous solubility is one reason that water serves as a pathway for its movement in the environment. Aldicarb is not tightly bound to either organic material or clays. Although the vapor pressure of aldicarb is low, aldicarb may enter air directly during and following application, spillage, or disposal. EPA estimated that 3.5 million pounds of aldicarb were used in 1979. Commercial agricultural applications account for 90 percent of the estimated annual domestic usage. Aldicarb also is used on ornamentals (commercial field grown and nursery plantings, greenhouse crops, and potted plants).

Analytical Methods. Analytical methods available for analyzing aldicarb and its degradation products in drinking water include the high pressure liquid chromatography technique.

Human Exposure. The data obtained on levels of aldicarb in food in the U.S. were insufficient for use in estimating typical dietary intake of aldicarb. However, a worst-case estimate of dietary intake from food residues on raw agricultural commodities could approach 108 μg/day for a 70 kg adult (1.5 μg/kg/day).

Residues are approved for specific crops. In a 1982 study of citrus fruit, no detectable aldicarb residues were found in any samples of oranges. One grapefruit sample contained 50 μg/kg aldicarb. Aldicarb residues, ranging from a trace to 470 μg/kg, were identified in 78 percent of samples of potatoes analyzed in 1979. Ninety-four percent of samples analyzed in 1980 contained detectable residues ranging from 50–530 μg/kg.

No data were available on levels of aldicarb in ambient air.

Aldicarb has been detected in ground water used as drinking water. Of the 8404 samples collected from wells on Long Island, New York, as of 1981, 29 percent contained total aldicarb residues greater than the detection limit of 1 μg/l. Wells near a farm in northern California showed aldicarb residues of up to 24 μg/l. Three out of nine wells sampled in southern New Jersey contained aldicarb concentrations of 3, 4, and 50 μg/l; a water sample collected near citrus groves in Florida was reported to contain 3.5 μg/l aldicarb. Data on water samples from wells in Wisconsin, Florida, Maine, Virginia and North Carolina indicated that samples from approximately 4 percent of the wells studied had aldicarb concentrations in excess of 10 μg/l. Aldicarb residues also have been found in ground water in New Jersey, Rhode Island (recent USGS study), Missouri and Massachusetts. Aldicarb also has been detected in surface waters.

Health Effects. Animal studies have demonstrated that aldicarb, as well as its sulfoxide and sulfone metabolites, are absorbed readily by mammalian and non-mammalian species. The parent compound and its sulfoxide metabolites are potent cholinesterase inhibitors; the sulfone is substantially less so at equivalent doses. Laboratory studies have found that aldicarb is excreted rapidly from the body, primarily via the urine.

The principal toxic effect of aldicarb and its sulfoxide and sulfone metabolites is cholinesterase inhibition as measured in plasma, erythrocyte and brain. This inhibition has been demonstrated to be transient in nature, when not fatal, due to the spontaneous recovery of the inhibited enzyme.

Given the nature of the primary toxicity of aldicarb and its metabolites (rapidly-reversible cholinesterase inhibition), the same NOAEL can be used as the basis for the derivation of allowable levels over virtually any duration of exposure. This NOAEL (0.125 mg/kg/day) was identified in a study in which rats were administered doses of aldicarb sulfoxide at levels of 0, 0.125, 0.25, 0.5 or 1.0 mg/kg bw or aldicarb sulfone at levels of 0, 0.2, 0.6, 1.8, 5.4 or 16.2 mg/kg bw in the diet for periods of 3 or 6 months (C.S. Weil and C.P. Carpenter. 1963a,b. Temik sulfoxide. Temik sulfone. Results of Feeding in the Diet of Rats for Six Months and Dogs for Three Months. Mellon Institute Report 31–141 and 31–142. EPA Pesticide Petition No. 9F0798). The results of the study demonstrated a substantial reduction of cholinesterase activity at the three highest dosage levels of both compounds when measured immediately after cessation of feeding. A NOAEL of 0.125 mg/kg bw was determined for the more toxic sulfoxide.

From the NOAEL of 0.125 mg/kg/day, a provisional AADI of 0.042 mg/l for the 70 kg adult can be derived by applying an uncertainty factor of 100, appropriate for use with a NOAEL derived from animal data, and assuming consumption of 2 liters of water per day. The provisional AADI (0.042 mg/l) is also appropriate for use as 1-day and 10-day assessments for the 70 kg adult. The 1-day and 10-day assessment for the child (assuming a 10 kg child consuming 1 liter of water per day) is 0.012 mg/l based upon the same study.

Conclusive evidence on the mutagenicity of aldicarb is not currently available, although the few studies done to date do not suggest mutagenic potential. Aldicarb has not been shown to be carcinogenic in animals. The National Cancer Institute (NCI) conducted a bioassay in which rats and mice were fed 2 or 6 ppm aldicarb in the diet for 103 weeks (0.1 or 0.3 mg/kg, bw, respectively) (NCI. 1979. Bioassay of Aldicarb for Possible Carcinogenicity. NCI–CG–TR–13h U.S. HEW PHS, National Institutes of Health). The conclusions of this study were that no tumors could be attributed solely to aldicarb administration. Two 2-year feeding studies in rats also reported that aldicarb did not produce a statistically significant increase in tumors when compared to controls (C.S. Weil and C.P. Carpenter. 1965. Two-year Feeding Study of Compound 21149 in the Diet of Rats. Unpublished report; C.S. Weil, 1972. Aldicarb (A), Aldicarb Sulfoxide (AsO), Aldicarb Sulfone (AsO_2) and a

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1:1 Mixture of ASO:ASO$_2$. Two year Feeding in the Diet of Rats. Unpublished report). Aldicarb has been classified in EPA's Group E, according to EPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon negative results in several animal studies.

The Food and Agricultural Organization/World Health Organization has proposed ADIs for aldicarb residues of 0-0.001 mg/kg/day in 1979 and 0-0.005 mg/kg/day in 1982. The NAS (1977; 1983) proposed an ADI of 0.001 mg/kg/day based upon two-year feeding studies in rats and dogs and a suggested-no-adverse-response-level (SNARL) of 7 µg/l using the same studies with an uncertainty factor of 1000. The SNARL is protective for a 70 kg adult for whom drinking water contributes 20 percent of the daily exposure to aldicarb residues. EPA's Office of Pesticide Programs has established an ADI of 0.003 mg/kg/day (46 FR 57047) based upon the same study used to derive the provisional AADI.

Ordinarily, an RMCL is proposed for the parent compound. In this case, however, the RMCL is proposed for total aldicarb residues (the parent compound as well as the sulfoxide and sulfone degradation products). The reason for this is that the residues of aldicarb found most often in water samples are the sulfoxide and sulfone, with relatively little of the parent compound being present. In addition, the analytical methodology most commonly used to determine aldicarb residues in water samples includes oxidation of the residues to the sulfone, followed by identification/quantification of the residues as sulfone. Toxicologically, the RMCL is based upon data from studies on the sulfoxide. This is because the sulfoxide is slightly more potent that the parent compound and significantly more potent than the sulfone as an inhibitor of cholinesterase, the end-point of toxicity considered to be the most sensitive measure of an effect.

EPA is proposing to regulate this contaminant based on its toxicology, occurrence in water and potential occurrence in drinking water supplies. The RMCL for aldicarb residues is based upon the effect of cholinesterase inhibition. The proposed RMCL of 0.009 mg/l is considered protective of the 70 kg adult for whom there is assumed a 20 percent contribution to exposure from drinking water. This RMCL is also considered to be protective of the 10 kg child over durations of exposure of less-than-lifetime and for whom drinking water constitutes a greater contribution to total exposure.

Questions for Comment:

1. Is it appropriate to propose an RMCL for aldicarb residues to be protective of the 70 kg adult, rather than the 10 kg child? The most sensitive end-point of toxicity (cholinesterase inhibition) is an acute, rapidly-reversible phenomenon which is the basis for the derivation of allowable exposure levels over all durations of exposure.

2. In the allocation of an RMCL for aldicarb residues, it was assumed that drinking water could contribute 20 percent of an individual's daily exposure to these residues. Is this appropriate, in light of the potential for significant exposure via non-water sources for the 70 kg adult, but more limited potential for the 10 kg child?

4. Carbofuran

Carbofuran (2,3-dihydro-2,2-dimethyl-7-benzofuranol-methylcarbamate; CAS # 1563-66-2) is an insecticide and nematocide. EPA estimated that about 11 million pounds were used in the U.S. during 1980. Eighty-four percent of the carbofuran is used on corn. Technical carbofuran has an aqueous solubility of 700 mg/l and is mobile in water.

Analytical Methods. Analytical methods available for analyzing carbofuran in drinking water include the solvent extraction-gas chromatography/mass spectrometry and the solvent extraction-high pressure liquid chromatography techniques.

Human Exposure. Data obtained on levels of carbofuran in foods were insufficient for use in estimating typical dietary intake levels. However, carbofuran residues are permitted on several crops. Peanuts from Arkansas have been reported to contain up to 25 µg/kg carbofuran. If all crops contained the maximum residues permitted, the daily intake for adults would be approximately 530 µg/day.

No data were available on levels of carbofuran in ambient air.

Carbofuran has been found in ground water samples from New York and Wisconsin at levels of 1-50 µg/1. It also has been detected in ground water in three other States.

Health Effects. Like other members of the class of carbamate pesticides, carbofuran is a potent inhibitor of cholinesterase. It is expected to be absorbed readily and rapidly by all likely routes of human exposure: oral, dermal and inhalation. Carbofuran's oxidative metabolites, 3-hydroxycarbofuran and 3-ketocarbofuran, also presses significant cholinesterase inhibitory properties. On the other hand, the hydrolytic metabolites, 3-keto-7-phenol and 7-hydroxycarbofuran phenol do not. Excretion of carbofuran metabolites is relatively rapid and the compound(s) would not be expected to accumulate significantly in mammalian tissues following repeated exposures.

The principal adverse health effect occurring following exposure to carbofuran is the rapid inhibition of cholinesterase activity. The effect has been demonstrated to be transient in nature, when not fatal, due to the spontaneous recovery of the inhibited enzyme at sites in the central and peripheral nervous systems. Other acute effects on the immune system and blood parameters have been reported. These, too, appear to be reversible once exposure is terminated. In addition, at doses above those at which inhibition of cholinesterase is noted, aspermia and testicular degeneration in dogs and some minimal decreases in rat pup survival also have been observed.

A study by FMC Corporation (1977. Industrial Hygiene Studies, final report. MRI Project No. 4230-B. EPA Accession No. 241303) was selected as the basis for the calculation of 1-day assessments for the 10 kg child and the 70 kg adult. Adult male human volunteers were administered a single oral dose of carbofuran shortly after eating breakfast. Of the three doses employed (0.05, 0.10 or 0.25 mg/kg bw), only the lowest dose was without effect, as identified by a lack of a statistically and biologically significant depression of red blood cell cholinesterase activity levels. To the NOAEL of 0.05 mg/kg, an uncertainty factor of 10 was applied, consistent with accommodation for intraspecies variability. This resulted in a 1-day assessment for the 10 kg child of 0.05 mg/l and for the 70 kg adult, 0.18 mg/l. No adequate dose-response data exist from which the 10-day assessment could be derived. Since, however, the end-point of toxicity that is of concern in this case is a rapidly-reversible, transient effect, the 1-day assessments for the 10 kg child and the 70 kg adult also can serve as the 10-day assessment for each individual.

The one-year dietary study in beagle dogs was selected to serve as the basis for the derivation of the AADI (FMC Corporation, Agricultural Chemical Group. 1983. One-year Chronic Oral Study in Beagle Dogs with Carbofuran. Study No. FMC A81-605/Toxigenics 410-0715. EPA Accession No. 250740-250744). Groups of animals were administered daily doses of 0, 10, 20 or 500 ppm carbofuran in the feed. These dietary levels corresponded to approximate daily doses of 0, 0.25, 0.5 or 12.5 mg/kg bw/day. From the results of this study, it was determined that the middle dose (0.50 mg/kg/day) was the

NOAEL, based upon the absence of a biologically significant depression of cholinesterase activity or reproductive effect in the males. Applying an uncertainty factor of 100 to the NOAEL of 0.50 mg/kg/day and assuming consumption of 2 liters of water psy day, as ADI of 0.005 mg/kg/day and an AADI of 0.18 mg/l was calculated for the 70 kg adult. This AADI is supported further by evidence from the 2-year rat feeding study (FMC Corporation, Agricultural Chemical Group. 1980. Two-year Dietary and Carcinogenicity Study in Rats. Carbofuran Technical Report No. Act. 130.91. EPA Accession No. 244491). In this study, groups of rats were fed 0, 10, 20 or 100 mg carbofuran/kg diet for 2 years. A NOAEL Of 1 mg/kg/day (the middle treatment dose) was identified, to which an uncertainty factor of 200 was applied to protect against cholinesterase depression and systemic effects. Again assuming consumption of 2 liters of water per day, and AADI of 0.18 mg/1 was calculated for the 70 kg adult. Both of these studies which were used to determine the provisional AADIs are long-term studies (1-year and 2-years) with many dose levels. These studies are more appropriate for determining a lifetime number than the human data (used to determine the short-term assessment) which consisted of one dose applied in a single exposure.

The mutagenic potential of carbofuran has been tested in a number of short-term assays. The majority of the results presented no evidence of mutagenicity. Two studies yielded equivocal results. The carcinogenic potential of carbofuran was evaluated in lifetime dietary studies in the rat and the mouse (FMC Corporation. Agricultural Chemical Group. 1980. Two-year Dietary Toxicity and Carcinogenicity Study in Rats. Carbofuran Technical Report No. ACT 130.51. EPA Accession No. 244491; Ibid. 1980. Two-year Dietary Toxicity and Carcinogenicity Study in Mice. Carbofuran Technical Report No. ACT 130.52. EPA Accession No. 244489). In neither study were there statistically increased tumor incidences attributable to exposure to the compound. Carbofuran has been classified in EPA's Group E, according to EPA's Proposed Guidelines for Risk Assessment, based upon the negative results in studies in rats and mice.

EPA is proposing to regulate carbofuran because of its potential adverse effects on health and potential for occurrence in drinking water. Exposure to carbofuran at sufficient levels results in a rapidly-reversible inhibition of cholinesterase activity, as well as possible reproductive and immune effects. An RfdCL of 0.036 mg/l is based upon the AADI of 0.18 mg/l and an assumed drinking water contribution of 20 percent.

5. Chlordane

Chlordane (1,2,4,5,6,7,8,8-octachloro-2,3,3a,4,7,7a-hexahydro-4,7methano-1H-indene; CAS # 57-74-9) is a broad spectrum insecticide. Technical chlordane is a mixture of stereo-isomers and other chlorinated analogs, including heptachlor. The water solubility of chlordane is 150-220 μg/l at 22 °C. Chlordane is relatively non-volatile with a vapor pressure of 1×10^{-5} mm Hg at 25 °C.

Chlordane is currently the most extensively used insecticide for subterranean termite control in the United States. Prior to the 1977 cancellation of registrations for agricultural and home garden use, chlordane was used for the control of soil insects and ants.

Analytical Methods. Analytical methods available for analyzing chlordane in drinking water include the solvent extraction chromatography and solvent extraction-gas chromatography/mass spectrometry techniques.

Human Exposure. In the FDA FY 77 compliance program report on pesticides and metals, chlordane was detected in 2.8 percent of foods sampled. Chlordane occurred in 1.8 percent of raw agricultural product samples, 9.3 percent of fish and marine animal samples, 0.2 percent of processed food samples, 1.4 percent of processed animal feed samples, and 2.5 percent of egg and egg product samples. In the FDA FY 79 total dietary study for adults, no chlordane was detected in any of the samples.

The USDA reported chlordane residues in violation of maximum allowable levels (300 ug/kg on raw agricultural commodities) in 0.1 percent of fat samples of various animal species intended for human consumption during the years 1982-1983.

Chlordane has been detected in ambient air at levels as high as 204 ng/m³. In the Surburban Air Sampling Program in 1975, 15 samples were collected at three suburban locations. Nine samples were positive for chlordane with a maximum value of 59 ng/m³.

Chlordane has occasionally been reported in wells near areas treated for termite control. In addition, chlordane was detected at low levels in the New Orleans Water Supply Study conducted by EPA. Five wells in New Jersey contained chlordane above 0.01 μg/l (range 0.01-0.02 μg/l). Chlordane has been detected in drinking water in a total of five States. One state found that 49 percent of the systems analyzed (87 ground systems) were positive. In the 1975 Region V Survey, one sample contained gamma-chlordane at a level of 0.004 μg/l (detection limit not reported).

Contamination of public water systems has been reported in several cases which occurred from back syphonage from tank filling operations during pesticide applications.

Chlordane has been identified at three hazardous waste sites designated in complaints and consent decrees under the Comprehensive Emergency Response Compensation and Liability Act of 1980 (CERCLA) and the National Contingency Plan (40 CFR Part 300). The concentration of chlordane ranged from unknown amounts migrating in water, 782-2300 mg/l in sediments, to 101 mg/l in soil.

Health Effects. The principal non-carcinogenic effects of chlordane from both acute and chronic exposure include neurotoxicity, induction of hepatic microsomal enzyme activity and liver effects.

Sufficient dose response data were not available to derive 1-day assessments. However, it should be noted that the ten-day assessment would also be protective for the one-day exposure. Ten-day assessments were based upon a study in which rats were given by gastric intubation doses of 0, 6.25, 12.5, 25.0, 50.0, 100.0 or 200 mg/kg chlordane for 15 days (Ambrose, et al. 1953. Toxicological and Pharmacological Studies on Chlordane. Arch. Ind. Hyg. Occup. Med. 7:197). The minimal histopathological changes such as presence of abnormal intracytoplasmic bodies of various diameters were evident at a dose level of 6.25 mg/kg. Using 6.25 mg/kg as the LOAEL, an uncertainty factor of 100 based upon an animal study and consumption of 1 liter (child) or 2 liters of water (adult) per day, 10-day assessments of 0.063 mg/l for a 10 kg child and 0.22 mg/l for a 70 kg adult were calculated.

An AADI for chlordane was derived based upon a two-year feeding study in dogs where 0.075 mg/kg/day (3 mg/kg in diet) was identified as the NOAEL (Vettorazzi, 1975. Toxicological Decisions and Recommendations Resulting from the Safety Assessment of Pesticide Residues in Food. Crt. Rev. Toxicol. 4:125). Using 0.075 mg/kg/day as the NOAEL, an uncertainty factor of 100 based upon an animal study and consumption of 2 liters of water per day, an AADI of 0.03 mg/l was calculated.

Chlordane was shown to be mutagenic in studies with transformed human cells in culture. A National

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Cancer Institute study (NCI. 1977a. Bioassay of Chlordane for Possible Carcinogenicity. NCI Carcinogenesis. Tech. Rep. Ser. No. 8) examined the carcinogenic effects of chlordane. Male and female mice were fed diets containing analytical-grade chlordane for 80 weeks, with the results showing a highly significant dose-dependent incidence of hepatocellular cacinoma in both male and female mice. Hepatic nodules and liver hyperplasia were also produced in rats. A study examining the reproductive effects of chlordane concluded that chlordane in the diet (16 mg/kg) of male and female rats from weaning appears to interfere with fertility and survival of the litters (Ambrose, et al. 1953a. Toxicological and Pharmacological Studies on Chlordane. Arch. Ind. Hyg. Occup. Med. 7:197). The IARC classified chlordane in Group 3; inadequate evidence for carcinogenicity in humans, limited evidence for carcinogenicity in animals and inadequate evidence for activity in short-term tests. Chlordane has been classified in EPA's Group B2, according to EPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon studies showing positive results in mice and female rats.

EPA's CAG and the NAS have calculated risk estimates (see Table 13) based upon the NCI study.

The WHO recommended guideline value (1984) is 3 µg/l for chlordane. This was based upon an acceptable daily intake of 0.001 mg/kg/day, with the assumption that not more than one percent of the ADI would be derived from drinking water.

A detection and odor threshold value of 0.005 mg/l has been reported in the literature for chlordane.

The available data indicate that chlordane has carinogenic effects in animals. For this reason and because of the occurrence of this contaminant in a number of drinking water supplies, EPA is proposing a primary regulation for chlordane. The RMCL will be based upon carcinogenic effects and an RMCL of zero is proposed.

6. Dibromochloropropane

Dibromochloropropane (1,2-dibromo-3-chloropropane (DBCP); CAS #96-12-8) is a soil fumigant used for nematode control on crops. DBCP is moderately soluble in water (approximately 1 g/l). Recent information suggests that DBCP may readily leach into aquifers used for drinking water.

Util 1977, DBCP was used in commercial agricultural applications. It was also used for non-crop applications, including commercial turf. Presently all uses have been cancelled.

Analytical Methods. Analytical methods available for analyzing DBCP in drinking water include the purge and trap-gas chromatography and the purge and trap-chromatography/mass spectrometry techniques.

Human Exposure. DBCP residues have been detected in U.S foods. In 1978, the FDA detected residues in carrots grown in soil fumigated with DBCP. Levels ranged from 20–1,500 µg/kg. Residues were detected in broccoli, cabbage, cauliflower and cucumbers in the range of 10–1,120 µg/kg. Additionally, residues were detected in peanut kernels in the range of 10–40 µg/kg.

The FDA compliance program report for FY 79 reported residues of DBCP in samples of domestic and imported fish. Seven (0.5%) out of 1,515 fish samples contained DBCP in excess of the detection limit.

DBCP has been detected in ambient air. One composite study of volatile organic chemicals in the atmosphere from locations nationwide showed the pressure of DBCP in ambient air samples in one location. A mean concentration of 6.4 ng/m³ in seven air samples was calculated for DBCP (median, 1.8 ng/m³).

Several regional studies have documented the presence of DBCP in ground water. Positive samples have been detected in Hawaii, California, Arizona, South Carolina and Maryland, with concentrations typically ranging from 0.02–20 µg/l. One of these States found 62 out of 92 samples to be positive, in ground water. DBCP is reported to be mobile in runoff and through soils.

DBCP has been identified at one hazardous waste site designated in complaints and consent decrees under the Comprehensive Emergency Response Compensation and Liability Act of 1980 (CERCLA) and the National Contingency Plan (40 CFR Part 300). The concentration of DBCP ranged from 0.08 mg/l in drinking water, 95 mg/l in ground water, to 2600 mg/l in soil. DBCP was also detected in ground water at a hazardous waste site in Colorado and a site in California.

Health Effects. DBCP is absorbed by the gastrointestinal tract, the lungs and the skin and is widely distributed throughout the body. Effects of acute, oral exposure to DBCP in rats include impaired renal function, hepatocellular necrosis, loss of spermatogenic elements in the testes and testicular and epididymal atrophy. Similar effects have been observed for subchronic oral exposure, while chronic exposure in animals has resulted in high incidences of toxic tubular nephropathy. Antifertility effects in men have been associated with exposure to DBCP.

One-day assessments were calculated based on a study in rates (Khuwe, W.M. 1985. Initial and Residual Toxicity following Acute Exposure of Developing Male Rats to Dibromochloropropane. Toxicol. Appl. Paramacol. 79:54–63) where single day, subcutaneous doses of DBCP induced renal lesions in 6-day-old rats. Using a 20 mg/kg/day LOAEL, an uncertainty factor of 1000, and consumption of 1 liter (child) or 2 liters (adult) of water per day, 1-day assessments of 0.2 mg/l for a 10 kg child and 0.7 mg/1 for a 70 kg adult were calculated.

Ten-day assessments were calculated based on a NOAEL of 0.5 mg/kg/day for increased kidney weights in a 90-day feeding study with DBCP in rats (Torkelson, T.R. et al. 1961. Toxicologic Investigations of 1,2-dibromo-3-chloropropane. Toxicol. Appl. Pharmacol. 3:545). Using an uncertainty factor of 100 and consumption of 1 liter (child) or 2 liters (adult) of water per day, 10-day assessments of 0.05 mg/l for a 10 kg child and 0.175 mg/l for a 70 kg adult were calculated.

A provisional AADI was not determined for DBCP due to insufficient data. The taste and odor threshold for DBCP in water has been reported to be 0.01 mg/l in the literature.

The NAS (*Drinking Water and Health*, 1977 and 1981. Vols. I and IV) did not calculate a Suggested-No-Adverse-Response-Level (SNARL) for DBCP. They felt that it would be premature to calculate a SNARL for pure DBCP since the contaminants in technical DBCP could be responsible for toxicological effects in animals and humans.

DBCP has been shown to result in reverse mutations in *Salmonella typhimarium*, recessive lethal mutations in *Drosophila melanogaster*, dominant lethal mutations in rats and chromosomal damage in rats and cultured Chinese hamster cells.

DBCP has been studied for carcinogenicity in mice and rats by oral and inhalation exposure and in mice by dermal application. A National Cancer Institute bioassay (NCI. 1977. Bioassay of Dibromochloropropane for Possible Carcinogenicity. NTIS PB279–472) reported highly significant dose-related incidences in rats of squamous-cell carcinoma of the forestomach of males and females and mammary adenocarcinoma in females receiving gavage doses of 10.7 and 20.7 mg/kg/day. Significantly increased incidences of squamous-cell carcinoma of the forestomach of male and female mice

were found at doses of 78.6 to 156.4 mg/kg/day.

A chronic dietary carcinogenicity study (Hazelton Laboratories America, Inc. 1977. One Hundred Four-Week Dietary Study in Rats; 1,2-dibromo-3-chloropropane (DBCP). Final report. Unpublished report submitted to Dow Chemical Co., Midland, MI. Oct. 29, 1977) reported that male and female rats, at a dose of 2.0 mg/kg/day, as estimated in U.S. EPA, 1979, 44 FR 65135, exhibited significantly increased incidences of carcinoma of the renal tubules, hepatocellular carcinomas and squamous-cell carcinoma of the stomach. DBCP has been shown to result in increased incidences of nasal cavity tumors in mice through inhalation exposure.

The IARC have classified DBCP in Group 2B; inadequate evidence for carcinogenicity in humans and sufficient evidence for carcinogenicity in animals. DBCP has been classified in EPA's Group B2, according to EPA's Proposed Guidelines for Carcinogenic Risk Assessment, because there are results in studies in animals (rats and mice).

Based upon the tumor incidence for liver, kidney and stomach in rats from the chronic dietary carcinogenicity study discussed above, CAG has estimated the possible carcinogenic risk of lifetime exposure to DBCP for a person consuming 2 liters of water per day. This risk estimate is shown in Table 13.

The Shell Oil Company (Health Effects Evaluation and Risk Assessment of DBCP submitted to Criteria and Standards Division, Office of Drinking Water, EPA, Dec. 1983) has derived risk estimates based on the tumor incidence for liver, kidney and stomach in rats from the same study. Using the geometric mean of five models (probit, logit, Weibull, gamma multi-hit and multi-stage) and three dose scaling factors (mg/kg, ppm, and mg/m^3) for each data set, the Shell Oil Company calculated risks from lifetime exposure to DBCP in drinking water. These risk estimates are as follows:

- 4.6×10^{-6} from lifetime exposure to DBCP based on male rat data.
- 2.2×10^{-14} from lifetime exposure to DCBP based on female rat data.
- 3.7×10^{-6} from one-seventh lifetime exposure to 100 ppb DBCP based on male rat data.
- 1.5×10^{-7} from two-seventh lifetime exposure to 100 ppb DBCP based on male rat data.

The available data indicate that DBCP has non-carcinogenic and carcinogenic effects in animals. Based on potential adverse health effects and occurrence and potential occurrence in drinking water, EPA is proposing to regulate DBCP. The RMCL will be based upon carcinogenic effects and an RMCL of zero is proposed.

Question for Comment:

1. Is there adequate evidence indicating that the contaminants in DBCP account for the toxicity of the compound?

7. o-, m-Dichlorobenzene

o- and m-Dichlorobenzene (CAS #s 95-50-1, 541-73-1) are solvents with low vapor pressures. o-Dichlorobenzene is used primarily in the production of organic chemicals, including pesticides and dyes. It also has direct solvent and pesticidal uses.

Releases of o-dichlorobenzene to air were estimated to be between 118 and 208 kkg in 1983. Industrial losses of m-dichlorobenzene were reported to be 0.165–0.608 kkg/year in 1983. Isomers of dichlorobenzene appear to vaporize rapidly from surface waters, despite their low vapor pressures, and are expected to degrade slowly in the environment.

Analytical Methods. Analytical methods available for analyzing o- and m-dichlorobenzene in drinking water include the purge and trap-gas chromatography and the purge and trap-gas chromatography/mass spectrometry techniques.

Human Exposure. Data on dichlorobenzene in the U.S. food supply are limited. Ortho- and m-dichlorobenzene have been reported in fish from the Great Lakes (0.3–3.0 μg/kg). In one study reported in 1980, 42 samples of milk from nursing mothers in New Jersey, Pennsylvania, Louisiana and West Virginia were found to contain a mean level of 9 μg/ml of dichlorobenzene isomers. All samples were positive for dichlorobenzene with a maximum level of 68 μg/ml. The highest levels reported in the study were found in samples from New Jersey and Louisiana.

Data indicating the level of o- and m-dichlorobenzene in ambient air are available from studies conducted in many States. From the median values of these data, it is estimated that rural/remote, urban/suburban and source dominated dichlorobenzene in ambient air approximate 0.0, 6.8 and 350 ng/m^3, respectively. The estimated respiratory intake for the adult male is expected to vary between zero and 6.2 μg/kg/day. Respiratory intake for formula-fed infants could vary between 0 and 4.3 μg/kg/day.

Median values of data also indicatre that rural/remote, urban/suburban, source dominated and maximum levels of m-dichlorobenzene in ambient air approximate 0.0, 0.036, 0.56 and 16 μg/m^3, respectively. The estimated respiratory intakes for the adult male and formula-fed infant can vary between 0–5.3 and 0–3.7 μg/kg/day, respectively.

Using data for surface water and ground water supplies, it is estimated that 99.3 percent of the population served by public drinking water systems are receiving water with no o-dichlorobenzene or levels less than 0.5 μg/l and that 0.7 percent may be exposed to levels of o-dichlorobenzene in drinking water at or above 0.5 μg/l. The vast majority of cases of drinking water contamination is in surface water supplies. m-Dichlorobenzene was not detected in the Ground Water Supply Survey. o-Dichlorobenzene has been detected in wastewaters and hazardous wastes, is mobile in runoff and soils and large amounts are produced and used across the country.

Health Effects. The principal toxic effects of o-dichlorobenzene in humans and other animals from acute and longer-term exposures include central nervous system depression, blood dyscrasias, lung, kidney and liver damage. Similar data are not available for m-dichlorobenzene. However, based upon the results of a few studies in short-term assay systems, the meta-isomer appears to be similar in toxicity to the ortho-isomer (apparently, not showing greater toxicity). Therefore, the short-term assessments developed for o-dichlorobenzene also will be used for m-dichlorobenzene.

No satisfactory does-response data exist from which to derive a 1-day assessments for either o- or m-dichlorobenzene. It is recommended that, for this duration of exposure, the 10-day level be applied.

The 10-day assessments were derived from results of mouse and rat subchronic gavage studies (Battelle-Columbus, 1978c. Subchronic Toxicity: Ortho-dichlorobenzene. $B_6C_3F_1$ mice. Unpublished report; Battelle-Columbus, 1978i. Subchronic toxicity: Ortho-dichlorobenzene. Fischer 344 rats. Unpublished report). Treated mice and rats received single doses of 0, 30, 60, 125, 250 or 500 mg/kg/day in oil, five days/week, for 90 days. A NOAEL of 125 mg/kg/day was identified for the mouse as well as the rat. Using this NOAEL, applying an uncertainty factor of 100 based upon an animal study in which a no-effect level was identified, a factor of 5/7 to account for conversion from a 5 day/week dosing regimen to a 7 day/week and assuming consumption of 1 liter of water per day, a 10-day assessment for the 10 kg child of 8.9 mg/

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l was derived. A 10-day assessment of 31.2 mg/l for the 70 kg adult, who is assumed to drink 2 liters of water per day, also was derived.

The data base selected for derivation of the provisional AADI for o-dichlorobenzene was the subchronic gavage studies in rats and mice described above for the derivation of the 10-day assessments. Using the NOAEL of 125 mg/kg/day, an uncertainty factor of 1000 based upon an animal study of less than lifetime exposure, a factor of 5/7 to account for conversion from a 5 day/week dosing regimen to 7 day/week and consumption of 2 liters of water per day, a provisional AADI of 3.12 mg/l was determined.

The dichlorobenzenes possess mutagenic activity in certain test systems. Neither were positive in the Ames/*Salmonella* or *E. coli* WP2 mutagenicity assay systems. However, m-dichlorobenzene, both with and without metabolic activation, increased mitotic recombination in *S. cerevisiae*. The orthoisomer was shown to produce abnormal mitotic division in the union, *Allium cepa*. Both o- and m-dichlorobenzene were shown to interact with and damage bacterial DNA in the *E. coli* W3110 polA$^+$/p3478 pol A$^-$ differential toxicity assay system.

o-Dichlorobenzene has been tested by gavage for carcinogenic potential in rats and mice in the NTP Bioassay program (NTP, 1982. Draft Technical Report on the Carcinogenesis Bioassay of 1,2-dichlorobenzene (CAS #95-50-1) in F344/N rats and $B_6C_3F_1$ Mice (Gavage Study). Draft report). A draft report of the results suggests that o-dichlorobenzene was not carcinogenic under the test conditions. This is a preliminary assessment and no final determination has been made on the carcinogenicity of o-dichlorobenzene. o-Dichlorobenzene has been classified in EPA's Group D, according to EPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon the preliminary results of NTP bioassay. The IARC classified o-dichlorobenzene in Group 3; inadequate evidence of carcinogenicity in humans, inadequate evidence for carcinogenicity in animals and inadequate evidence of activity in short-term tests. m-Dichlorobenzene has not been tested for carcinogenicity and thus has also been classified in EPA's Group D.

EPA's ambient water quality criterion (U.S. EPA. 1980. Ambient Water Quality Criteria for Dichlorobenzene. 440/5-80-039) for o-dichlorobenzene is 2.6 mg/l, based upon the NOAEL of 16.8 mg/kg identified in the 1958 Hollingsworth, et al., study (Toxicity of o-dichlorobenzene: Studies on Animals and Industrial Experience. AMA Arch. Ind. Hlth. 17(1):180-187). The WHO proposed drinking water guideline for the ortho- isomer is 0.3 μg/l, based upon organoleptic considerations. The NAS Safe Drinking Water Committee recommended a tentative chronic SNARL of 0.3 mg/l for the orthoisomer, using the lowest dose (60 mg/kg) in the carcinogenicity bioassay (NTP, 1982. See above) and assuming that a 70 kg adult consumes 2 liters of water per day, an uncertainty factor of 1000 and a factor of 5/7, with 20 percent contribution of exposure from water (*Drinking Water and Health*, 1983. Vol. III).

The ambient water quality criteria for o-dichlorobenzene was based upon an older study with a lower NOAEL than was used to develop the provisional AADI, while the WHO guideline did not consider health effects in the derivation of the number. The NAS SNARL was based upon a chronic study which only examined one parameter, while the subchronic study upon which the provisional AADI was based examined many parameters.

The odor thresholds for the dichlorobenzenes in water range from 0.01 to 0.03 mg/l.

Exposure to ortho-dichlorobenzene at high dose levels results in a variety of toxic effects, including central nervous system depression, kidney and liver damage. Because there is also sufficient occurrence potential in drinking water, a Revised Regulation will be proposed and the RMCL will be based upon non-carcinogenic effects. An RMCL of 0.62 mg/l is proposed for ortho-dichlorobenzene based upon a provisional AADI of 3.12 mg/l and assuming 20 percent contribution to total exposure via drinking water. An RMCL is not being proposed for meta-dichlorobenzene due to the lack of long-term compound-specific toxicity data. The only data available on the similarity of toxicity between ortho- and meta-dichlorobenzene consists of data from short-term systems. This data was not considered to be sufficient to propose an RMCL for the meta- isomer based upon the toxicity of orthodichlorobenzene. In addition, the potential for contamination of drinking water supplies by meta-dichlorobenzene appears to be remote and lower than the potential for either the ortho- or the para-isomer.

Question for Comment:

1. Would an appropriate approach be to develop an RMCL for m-dichlorobenzene based upon the toxicity data on o-dichlorobenzene?

8. cis- and trans-1,2-Dichloroethylenes

1,2-Dichloroethylene (CAS #s 156-59-2, 540-59-0) also known as acetylene chloride, is a solvent consisting of a mixture of cis- and trans-isomers. Their proportion varies depending on production conditions. The cis- and trans-isomers have vapor pressures of 180 and 265 mm Hg at 20°C, respectively, and water solubilities of 3.5 and 6.3 g/l, respectively. 1,2-Dichloroethylene is used as a low-temperature extraction solvent for organic materials, and as a chemical intermediate in the synthesis of other compounds. The principal source of these dichloroethylenes in drinking water appears to be from in situ transformations from other chlorinated hydrocarbons.

Analytical Methods. Analytical methods available for analyzing cis- and trans-1,2-dichloroethylene in drinking water include the purge and trap-gas chromatography and the purge and trap-gas chromatography/mass spectrometry techniques.

Human Exposure. No data are available on levels of 1,2-dichloroethylene in the U.S. food supply.

Little data are available on the level of trans-1,2-dichloroethylene in air. Limited sampling at one site resulted in the detection of levels around 4 μg/m^3. Ambient air monitoring for the cis-isomer is available from 10 States, dominated, and maximum levels of cis-1,2-dichloroethylene in ambient air approximate 0.0, 0.27, 1.2 and 27 μg/m^3, respectively. Using these data, estimated respiratory intake for the adult male will vary between zero in rural areas to 8.9 μg/kg/day following exposure at maximum levels. Respiratory intake for formula-fed infants would vary between zero and 6.2 μg/kg/day.

Using combined data for surface water and ground water supplies, up to 2.2 percent of the U.S. population is estimated to be receiving water containing >0.5 μg/l of 1,2-dichloroethylene, and 0.2 percent of the population could be receiving drinking water with 1,2-dichlorethylene levels greater than 20 μg/l.

Health Effects. The principal toxic effects of cis- and trans-1,2-dichloroethylene following acute exposure are upon the liver and kidney. In addition, these compounds possess general anesthetic and narcotic properties at exposure levels above those at which liver and kidney effects are seen. No data are available following longer-term exposures.

One-day assessments for cis-1,2-dichloroethylene were derived from an animal study (Jenkins, L.J., Jr., M.J. Trabelus and S.D. Murphy. 1972. Biochemical Effects of 1,1-Dichloroethylene. Toxicol. Appl.

Pharmacol. 23:501–510). In this study, the authors identified what is interpreted to be a LOAEL. In measuring levels of three liver enzymes and two plasma enzymes, all indicators of liver function, the investigators showed that a single 400 mg/kg oral dose to the rat produced a significant change only in liver alkaline phosphatase, while the levels of the other four enzymes were not affected significantly. The 1-day assessment for the 10 kg child of 4.0 mg/l is derived from the LOAEL of 400 mg/kg, using an uncertainty factor of 1000 since a NOAEL was not determined and an assumed consumption of one liter of water per day. A comparable 1-day level for the 70 kg adult would be 14 mg/l, assuming the ingestion of 2 liters of water per day.

No satisfactory dose-response data are available from which to derive 10-day assessments for cis-1,2-dichloroethylene. Evaluation of the available toxicological data on cis-1,2-dichloroethylene and 1,1-dichloroethylene suggests that a ten-day assessment can be derived from data in the 90-day drinking water study in rats with 1,1-dichloroethylene (Rampy, et al., 1977). A NOAEL of 100 ppm (10 mg/kg/bw) was identified in this study. Applying an uncertainty factor of 100 and assuming consumption of 1 liter of water per day, the ten-day assessment for the 10 kg child would be 1 mg/l. A comparable ten-day level for the 70 kg adult would be 3.5 mg/l, assuming the ingestion of 2 liters of water per day.

One-day assessments for trans-1,2-dichloroethylene were derived from an animal study (Freundt, K.J., G.B. Liebalt and R. Liberwirth. 1977. Toxicology Studies on trans-1,2-Dichloroethylene. Toxicol. 10:131–139). This study identified a NOAEL of 200 ppm inhaled by mature female rats over a single 8-hour exposure period. This exposure resulted in slight liver effects in one of six animals, as observed histologically. No changes were observed in several serum biochemical parameters. A total absorbed dose of 27.2 mg/kg was estimated by converting ppm into mg/m^3 (200×3.97) and multiplying by 8 hours' exposure, assuming that 30 percent of the exposure dose was absorbed, then dividing by 70 kg. The 1-day assessment for the 10 kg child of 2.7 mg/l was derived by applying an uncertainty factor of 100 to the total absorbed dose (in mg/kg) and assuming consumption of 1 liter of water per day. A 1-day assessment of 9.45 mg/l for the 70 kg adult would be derived in the same manner, but, assuming a consumption of 2 liters of water per day.

No satisfactory dose-response data are available from which to derive 10-day assessments for trans-1,2-dichloroethylene. Thus, it has been determined that, in the absence of compound-specific data, the data for 1,1-dichloroethylene used above for the calculation of the 10-day assessments for the cis-isomer are appropriate for the trans-isomer, as well. Therefore, the 10-day assessment for the 10 kg child would be 1 mg/l, and, for the 70 kg adult, 3.5 mg/l.

An AADI for the 1,2-isomers of dichloroethylene could not be developed from compound-specific data since these data do not exist at this time. Two options are available. The first is to propose no AADI at all. The second is to apply an AADI developed from data on 1,1-dichloroethylene. The available information from shorter term exposures to all three of these compounds suggests that the non-carcinogenic toxicity induced by the 1,2-isomers is likely to be no more severe than that of 1,1-dichloroethylene. Since the endpoints of non-carcinogenic toxicity to all three dichloroethylenes are essentially identical, applying the AADI developed for 1,1-dichloroethylene to the 1,2-isomers may even result in an added margin of safety.

The study selected for the derivation of the AADI was a two-year chronic toxicity/oncogenicity study in which male and female rats were given concentrations of 0, 50, 100 or 200 mg/l 1,1-dichloroethylene in their drinking water (Quast, et al., 1983. A Chronic Toxicity and Oncogenicity Study in Rats and Subchronic Toxicity Study in Dogs on Ingested Vinylidene Chloride. Fund Appl. Toxicol. 3:55–62). In the highest dose group, significant microscopic changes were noted in the livers of animals of both sexes. Minimal hepatocellular swelling and fatty changes were detected in female rats at all dose levels. An AADI of 0.35 mg/l was determined using a LOAEL of 100 mg/l (or 10 mg/kg), and uncertainty factor of 1000 based upon an animal study with the NOAEL not identified, and consumption of 2 liters of water per day.

Both cis- and trans-1,2-dichloroethylene were non-mutagenic when assayed with *E. coli* K12 at similar concentrations use for 1,1-dichloroethylene at which the latter was found to be mutagenic (Griem, et al. 1975. Mutagenicity *In Vitro* and Potential Cercinogenicity of Chlorinated Ethylenes as a Function of Metabolic Oxirane Formation. Biochem. Pharmacol. 24:2013–2017). Cis-, but not trans-1,2-dichloroethylene was found to be mutagenic in the host-mediated assay using *Salmonella* tester strains in mice (Cerna, M. and H. Kyenova. 1977. Mutagenic Activity of Chloroethylenes Analyzed by Screening System Tests. Mut. Res. 46:214 Abst.). The same authors observed that the cis- isomer also produced chromosomal aberrations in mouse bone marrow cells following intraperitoneal injections.

No long-term studies have been carried out on the carcinogenic potential of cis- and trans-1,2-dichloroethylene and thus both isomers have been classified in EPA's Group D, according to EPA's Proposed Guidelines for Carcinogenic Risk Assessment.

Exposure to cis- and trans-1,2-dichloroethylene at high dose levels results in liver and kidney effects. Since there is widespread occurrence of these contaminants, EPA proposes to regulate them. The proposed RMCL of 0.07 mg/l is based upon an AADI of 0.35 mg/l for both cis- and trans-1,2-dichloroethylene effects derived from data on 1,2-dichloroethylene assuming 20 percent of the exposure is via drinking water.

Question for Comment:

1. In the absence of compound-specific data, is it reasonable to develop an AADI for cis- and trans-1,2-dichloroethylene from toxicity data on 1,1-dichloroethylene?

9. 1,2-Dichloropropane

1,2-Dichloropropane (propylene dichloride; CAS #78–87–5) is a solvent and a pesticide. United States production of 1,2-dichloropropane was approximately 77 million pounds in 1980. Primary uses of 1,2-dichloropropane include: soil fumigation for nematodes and other insects; solvent for metal degreasing, fats, oils, waxes, gums, and resins; intermediate for perchloroethylene and carbon tetrachloride; lead scavenger for anti-knock fluids; and in dry cleaning fluids.

Analytical Methods. Analytical methods available for analyzing 1,2-Dichloropropane in drinking water include the purge and trap-gas chromatography and the purge and trap-gas chromatography/mass spectrometry techniques.

Human Exposure. No data were available on the levels of 1,2-dichloropropane in ambient air or in food.

1,2-Dichloropropane was found in 6 of 466 randomly sampled drinking water systems in the GWSS. The mean level found was 3.7 ug/l. In non-randomly selected samples, 1,2-dichloropropane was observed in 7 out of 479 systems with a mean concentration of 3.7ug/l. High concentrations have been reported

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in cases of contaminated ground water. 1,2-Dichloropropane was found in 66 out of 410 drinking water wells in several counties throughout California between 1979 and 1983. 1,2-Dichloropropane has also been detected in wells in New York.

1,2-Dichloropropane has been identified at one hazardous waste site in complaints and consent decrees under the Comprehensive Emergency Response Compensation and Liability Act of 1980 (CERCLA) and the National Contingency Plan (40 CFR Part 300). The concentration of 1,2-dichloropropane ranged from 74 ug/l in soil to 1800 ug/l in runoff.

Health Effects. The principal target organ for 1,2-dichloropropane toxicity appears to be the liver. Centrilobular necrosis, liver congestion and hepatic fatty changes have been reported in animals. Effects on the kidneys and lungs have also been reported in animal studies. There are insufficient toxicological data available in the scientific literature to calculate a 1-day assessment for 1,2-dichloropropane. The 10-day assessment is recommended to be applied to a 1-day exposure.

Ekshtat, et al. (1975. Study of the Cum ulative Properties of Substances at Different Levels of Activity. Uch.Zap.-Mosk. Nauchno-Issled. Inst. Gig. 22:46–48.) reported the results of orally administered 1,2-dichloropropane at dose levels of 8.8, 44 or 220 mg/kg for 20 days. The investigators observed disturbances in the animals' protein formation, hepatic enzyme levels and lipid metabolism. The NAS (1979) in a request from the Office of Drinking Water provided a 7-day Suggested-No-Adverse-Response-Level (SNARL) for 1,2-dichloropropane based on the Ekshtat, et al. (1975) study in rats. A LOAEL of 8.8 mg/kg/day was used to derive a 7-day level for a 70 kg adult consuming 2 liters water/day. The NAS SNARL can be used as an interim ten-day assessment as well. The 10-day level is derived using 8.8 mg/kg/day as the LOAEL divided by an uncertainty factor of 1000 (because data are limited and incomplete). For a 70 kg adult consuming 2 liters of water per day, the 10-day number is 0.3 mg/1. For a 10 kg child consuming 1 liter of water per day, the 10-day number is 0.09 mg/l.

There are insufficient data available to calculate a provisional AADI for 1,2-dichloropropane.

Mutagenicity studies have shown that at high concentrations 1,2-dichloropropane induces base-pair substitutions in *Salmonella typhimurim* and induces sister chromatid exchanges or chromosome aberrations in Chinese hamster ovary cells, with or without S9 metabolic activation. In a carcinogenesis bioassay of 1,2-dichloropropane conducted by the National Toxico. gy Program (NTP Draft Report, 1982), rats were administered 1,2-dichloropropane in corn oil by gavage. In their draft report, the NTP reported significant increases in non-neoplastic liver lesions (foci of clear cell change, centrilobular necrosis) and mammary gland adenocarcinomas in female rats, but treatment related non-neoplastic or neoplastic effects were not observed in male rats.

In a NTP mice study (NTP Draft Report, 1982), non-neoplastic liver lesions were observed in male mice only, but hepatocellular adenomas were increased in both male and female mice. Under the conditions of these studies, 1,2-dichloropropane was considered to be carcinogenic for male and female mice and the effects of 1,2-dichloropropane in rats were considered equivocal. It should be noted that these results are reported in the draft NTP report and the study is currently being audited. The final results may change when the audit is completed and reported by the NTP.

EPA's CAG have derived estimates of risk of daily exposure to 1,2-dichloropropane based upon the NTP mice study. As previously noted, these results are reported in the draft NTP report and the final results may change. The CAG risk estimates are summarized in Table 13.

The EPA (1980) concluded that data regarding the toxicity of 1,2-dichloropropane were insufficient for the derivation of an ambient water quality criterion for the protection of human health.

1,2-Dichloropropane has been classified in EPA's Group C, according to EPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon one positive study (NTP Draft Report, 1982) in mice. Thus, 1,2-dichloropropane has been placed in Regulatory Category II and the RMCL will be set at a non-zero level based upon the 10^{-5} risk level. The reason for using a risk level is because the chronic toxicity data are extremely limited and an AADI has not been established for 1,2-dichloropropane. The 10^{-5} level was used because of the quality of the bioassay data which have not been validated.

Exposure to 1,2-dichloropropane has been shown to result in adverse health effects. For this reason and because there is sufficient occurrence in drinking water, a regulation will be proposed. 1,2-Dichloropropane is classified as a possible carcinogen and an RMCL of 6 μg/l is proposed, based upon the 10^{-5} cancer risk level as calculated by EPA's CAG.

Questions for Comment:

1. Are there data available on the toxicology of 1,2-dichloropropane which may be used for the derivation of a provisional AADI?

2. Should 1,2-dichloropropane be classified in EPA's Group C? What should be the basis for the RMCL for this compound?

10. 2,4-D

2,4-D (2,4-Dichlorophenoxyacetic acid; CAS #94-75-7; current MCL is 0.1 mg/l) is a systemic herbicide used to control broadleaf weeds. 2,4-D is sold as a variety of salts, esters and other derivatives which are very soluble in water. 2,4-D and its derivatives undergo both chemical and biological degradation when released to the environment. Soil residues break down in approximately six weeks and repeated application usually does not lead to accumulation. Phenoxy herbicides undergo photolysis and bacterial degradation.

Nearly 60 percent of the domestically available 2,4-D is used on agricultural crop sites. The remainder is used on range and pasture land, industrial and commercial sites, lawns and turf, forests and in water. 2,4-D is currently registered for aquatic weed control in ponds, lakes, reservoirs, marshes, bayous, drainage ditches, canals, rivers and streams.

Analytical Methods. Analytical methods available for analyzing 2,4-D in drinking water include the derivatization gas chromatography technique.

Human Exposure. In the 1970–73 FDA total dietary studies for adults, four samples of leafy vegetables contained residues of 2,4-D in the range of 10–130 ug/kg, and one potato composite sample was found to contain 14 ug/kg. In the 1975–76 total dietary studies for infants and toddlers, one positive sugar and adjunct sample (25 ug/kg) was identified in the toddler food analysis. No residues were found in the infant food analysis. FDA's compliance reports showed that 9 out of 11 food samples were positive for 2,4-D in FY 78, 5 out of 39 samples were positive in FY 80, and 1 out of 10 samples was positive in FY 82.

2,4-D has been reported in ambient air samples from cities in New York and Utah at levels as high as 4 ug/m^3. Thirty percent of the ambient ash air samples taken in western Canada in 1976 were found to contain 2,4-D levels above 0.1 ug/m^3.

2,4-D has been detected in drinking water in four States. In the NORS, one

large surface water system was found to contain detectable levels of 2,4-D (0.04 ug/l. In the NSP, once surface water system contained 2,4-D (1.1 ug/l). None of the surface water systems sampled uring the RWS contained 2,4-D in excess of the minimum quantification limit of 0.01 ug/l. National compliance reports for surface water systems sampled for 2,4-D were below the MCL. Concentrations of approximately 0.025 ug/l 2,4-D have been occasionally detected in public water supplies by State agencies. 2,4-D was detected in 2.4% of surface water monitoring stations in the USGS/EPA Pesticide Monitoring Network.

2,4-D has been detected in many surface and ground waters. The compound has been detected in waste waters and hazardous wastes, is mobile and widely used on many crops.

There are insufficient data available to make reliable estimates of total human intake of 2,4-D from drinking water, food, and air. Assuming that drinking water concentrations vary from 0–10 ug/l, and assuming that air levels range from 0.001–0.05 ug/m^3, the estimated total intake from these two sources could range from 0.00033–0.45 ug/kg/day for the adult male and from 0/00023–0/54 ug/kg/day for the formula fed infant.

Health Effects. Short-term exposure to 2,4-D at high doses by the oral route or injection by various routes result in progressive symptoms so muscular incoordination, hindquarter paralysis, stupor, coma and death in animals.

One-day assessments, were derived from an animal study (Hill, E.V. and H. Carlisle, 1947. Toxicity of 2,4-D for Experimental Animals. J. Ind Hyg. Toxicol. 29:88–95). In this experiment, groups of six guinea pigs that were administered 10 doses of 50 or 100 mg/day 2,4-D sodium salt by gavage for 12 days did not develop characteristic evidence of intoxication (i.e)., muscular signs) or mortality. If it is assumed that the guinea pigs weighed 0.3 kg (the reported approximate weight in the single dose studies), the NOAEL of 50 mg/day corresponds to a daily dose of 138.9 mg/kg/day; the equivalent dose of 2,4-D acid is 126.3 mg/kg/day. Although symptoms or signs of intoxication were not specifically associated with this exposure, these criteria of toxicity are still too insensitive to justify using 126.3 mg/kg/day as an animal NOAEL. Using 126.3 mg/day as a LOAEL, an uncertainty factor of 1000 based upon an animal study with a LOAEL and consumption of 1 liter (child) and 2 liters (adult) of water per day, 1-day assessments of 1.1 mg/l and 3.85 mg/l were determined for the child and the adult, respectively.

An animal study (Rowe, V.K. and T.A. Hymas, 1954. Summary of Toxicological Information on 2,4-D and 2,4,5-TP Herbicides and on Evaluating the Hazards to Livestock Associated with Their Use. Am. J. Vet. Res. 15:622–629) was used to develop 10-day assessments. In this study, investigators administered 0, 100, 300 or 1000 ppm 2,4-D in the diet to group of five young female rats for 114 days. If it is assumed that young rats consume 10 percent of their body weight in food per day, the corresponding daily doses would be 0, 10, 30 and 100 mg/kg/day. No effects (same parameters as in the 4 week gavage study) were found at 10 or 30 mg/kg/day, but 100 mg.kg/day produced "excessive mortality" with depressed growth rate, slightly increased liver weights and slight cloudy swelling of the liver. Using a NOAEL of 30 mg/kg/day, an uncertainty factor of 1000 based upon an animal study and consumption of 1 liter (child) and 2 liters (adult) of water per day, 10-day assessments of 0.30 mg/l and 1.1 mg/1 were determined for the child and adult, respectively.

The NAS (*Drinking Water and Health*, 1977. Vol. I) has calculated an ADI for 2,4-D based upon a 2-year feeding study (Hanson, et al. 1977. Toxicology and Applied Pharmacology 20:22–129) in dogs in which 12.5 mg/kg/day was selected as the NOAEL. Using this NOAEL, an uncertainty factor of 1000 based upon an animal study and assumption of an average daily intake of 2 liters of water per day, an ADI of 0.0125 mg/kg/day was calculated.

Inconsistencies and inadequacies in the 2,4-D toxicity data base have been recognized by both EPA and its Scientific Advisory Panel. For example, the inconsistencies consist of varying effects seen at different dose levels in different studies. In order to resolve the inconsistencies and inadequacies, the Agency has taken action under FIFRA 3(c)(2)(B) to require 2,4-D registrants to update and complete the 2,4-D toxicity data base. Some of the required studies have been completed and some are ongoing. As a result of the Panel's recommendations, and the 3(c)(2)(B) notice, a 90-day range-finding study was performed as a preliminary to a 2-year feeding study. The range-finding study (Document Accession No. 251473) identified a NOAEL of 1 mg/kg body weight with the liver and kidney as target organs. Preliminary data from the chronic study support the 1 mg/kg NOAEL. Assuming that at the end of the 2-year experiments there is no change in the NOAEL, an uncertainty factor of 100 can be applied to derive a provisional AADI of 0.35 mg/l.

2,4-D may have mutagenic activity in certain systems; however, the general lack of positive genotoxic effects *in vivo* for mammalian assays may indicate that sufficient levels of 2,4-D are not able to reach the target tissues. No information is available on mammalian mutagenicity testing conducted with esters of 2,4-D; these forms could theoretically show higher levels of penetration into target cells.

Available data from laboratory animals have not provided a sufficient demonstration of carcinogenicity of 2,4-D, although increased tumor production is suggested. This questions cannot be adequately resolved until more compelling evidence is available from well designed bioassays. 2,4-D has been classified in EPA's Group D, according to EPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon inadequate data from animal studies.

EPA's MCL for 2,4-D in drinking water, under the National Interim Primary Drinking Water Regulations, is 0.1 mg/l. This standard is based upon a NOAEL of 8 mg/kg/day, an uncertainty factor of 500 and the assumption that 20 percent of the total intake is via drinking water. New studies have determined a NOAEL at 1 mg/kg/day.

Exposure to 2,4-D at high dose levels results in kidney damage and skeletal muscle changes. It has also been detected in drinking water. Therefore, an RMCL will be proposed based upon an AADI of 0.35 mg/l for non-carcinogenic effects, assuming 20 percent of total exposure is via drinking water. An RMCL of 0.07 mg/l is proposed.

11. Epichlorohydrin

Epichlorohydrin (1-chloro-2,3-epoxypropane; CAS #106-89-8) is a halogenated alkyl epoxide. It is soluble in water (6.6 x 10^4 mg/l) and organic solvents. A large fraction of the epichlorohydrin supply is used to make glycerin. Other applications include: use as a major raw material for epoxy and phenoxy resins and flocculants (sometimes used in the production of potable water and foods), solvent for resins, gums, cellulose esters and ethers, paints, varnishes, nail enamels and lacquers (sometimes used to coat interiors of water tanks and pipes); cement for celluloid; curing for propylene-based rubbers; and use as a high wet-strength resin for the paper industry.

Analytical Methods. Analytical methods available for analyzing

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epichlorohydrin in drinking water include the direct injection gas chromatography technique.

Human Exposure. No ambient monitoring data are available on human intake of epichlorohydrin from ambient air, food or drinking water supplies. However, epichlorohydrin emissions and public exposure levels were estimated by EPA's Office of Air Quality Planning and Standards, based upon plants that manufacture or use epichlorohydrin in the production of glycerin, epoxy and polyamide resins, elastomers and surfactants. A human exposure model was employed, based upon a 50 km radius around each epichlorohydrin facility, which estimated than approximately 50 persons are exposed to a concentration of 8 $\mu g/m^3$ or greater, approximately 1,000 persons are exposed to a concentration of 1 $\mu g/m^3$ or greater and approximately 70,000 persons are exposed to a concentration of 0.1 $\mu g/m^3$ or greater.

Epichlorohydrin is a contaminant of polymers used in the clarification and storage of potable water and in food processing. It has been detected in waste and is considered mobile in water.

Health Effects. Epichlorohydrin is rapidly absorbed following oral, dermal or inhalation exposures. Epichlorohydrin accumulates in the kidneys, liver, pancreas, spleen and adrenals and is excreted via the urine and respiratory tract.

Following acute exposure to epichlorohydrin, toxic effects in the central nervous system, lungs, liver and kidneys have been observed. At the site of application epichlorohydrin is a strong irritant. The major target organs for toxicity following chronic exposure to epichlorohydrin are the nasal turbinates, lungs, kidneys, male reproductive organs and the central nervous system.

A short-term assessment for a 10-day exposure was calculated based on a study in rats (Van Esch, G.J. 1981. Ryksinstitute Voor De Volksgezondheid Bilthoven Rapport. 627805 006) where antifertility effects were observed in males given oral doses of epichlorohydrin for 10 days before mating and through parturition of an F_{1a} generation. Using a NOAEL of 2 mg/kg, an uncertainty factor of 100, and consumption of 1 liter (child) or 2 liters (adults) of water per day, a 10-day assessment of 0.14 mg/l for a 10 kg child and 1-day and 10-day assessments of 0.5 mg/l for a 70 kg adult was calculated. This assessment is also considered protective for a 1-day exposure for the child and adult.

The NAS (*Drinking Water and Health*, 1980. Vol. III) has cited a threshold for odor perception of epichlorohydrin of 0.5 to 1.0 mg/l and a threshold for irritant action of 0.1 mg/l. A human study (Fassin, 1983. Biological Effects of Epichlorohydrin and Its Hygienic Significance as an Atmospheric Pollutant Gig. Hyg. Sanit. 31:357–363) reported an odor threshold of 0.3 mg/m^3 for the most sensitive subjects and a subthreshold level of 0.2 mg/m^3.

A provisional AADI was calculated for epichlorohydrin based upon an inhalation study in which male rats were exposed to 100 ppm epichlorohydrin 6 hours/day for 30 days and 10 or 30 ppm epichlorohydrin 6 hours/day, 5 days/week for the lifetime of the rats and inflammation changes in the respiratory tract and kidney lesions were observed (Laskin, S., A.R. Sellakumar, M., Kuschner, N. Nelson, S. LaMendola, G.M. Rusch, et al., 1980. Inhalation Carcinogenicity of Epichlorohydrin in Noninbred Sprague-Dawley rats. J. Natl. Cancer Inst. 65:751–757). Using 2.16 mg/kg/day, as derived from the 10 ppm exposure level, as the LOAEL, an uncertainty factor of 1000 and consumption of 2 liters of water per day, a provisional AADI of 0.076 mg/l was calculated.

The NAS (*Drinking Water and Health*, 1980. Vol. III) calculated 1-day and 7-day SNARLs for epichlorohydrin but did not calculate an ADI or cancer risk numbers.

Epichlorohydrin has been found to be mutagenic in a number of prokaryotic systems and eukaryotic cell cultures. Negative results were reported for the mouse dominant lethal assay and micronucleus assay.

Epichlorohydrin has been found to be carcinogenic following oral and inhalation exposures. Oral exposure produced a dose-dependent increase in forestomach tumors characterized as squamous and basal cell hyperplasia, squamous cell papillomas or squamous cell carcinomas Inhalation exposure produced squamous cell carcinomas in the nasal cavities. The IARC have classified epichlorohydrin in Group 2B; inadequate evidence for carcinogenicity in humans, sufficient evidence for carcinogenicity in animals and sufficient evidence for activity in short-term tests. Epichlorohydrin has been classified in EPA's Group B2, according to EPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon positive results in several sites in rats.

EPA's CAG has derived estimates of risk from daily exposure to epichlorohydrin in drinking water based upon the incidence of tumors of the forestomach in a study in which rats were given drinking water containing 0, 375, 750, or 1500 mg/l epichlorohydrin for 61 weeks (Kawabata, A., 1981. Studies on the Carcinogenic Activity of Epichlorohydrin by Oral Administration in Male Wistar Rats. J. Amer. Med. Assoc. 32:279–283). The CAG risk estimate is shown in Table 13.

The CAG has also calculated risk estimates (95 percent confidence limits) from lifetime exposure to 10 $\mu g/l$ epichlorohydrin using several different models. These estimates are as follows:

Dose	95% upper confidence limit of additional risks			
	Multi-stage model	One-hit model	Weibull model	Log-Probit model
10 $\mu g/l$	2.6×10^{-4}	3.4×10^{-4}	0	0

The available data indicate that epichlorohydrin has carcinogenic effects in animals. For this reason and because it is likely to be occurring in drinking water due to its use in coatings for drinking water pipes, regulation is proposed. The RMCL will be based upon carcinogenic effects and an RMCL of zero is proposed.

12. Ethylbenzene

Ethylbenzene (CAS # 100-41-4) is a flammable liquid, soluble in water (152 mg/l) and in alcohol, benzene, ether, acetone and other organic solvents. In 1982, the U.S. production totaled 6,658,241,630 pounds. The primary use of ethylbenzene is in the production of styrene.

Analytical Method. Analytical method available for analyzing ethylbenzene in drinking water include the purge and trap-gas chromatography technique.

Human Exposure. No data are available on the levels of ethylbenzene in foods.

Limited data are available on the levels of ethylbenzene in ambient air. Ethylbenzene levels in ambient air have been reported between 2 $\mu g/m^3$-5 $\mu g/m^3$ in various areas.

Ethylbenzene has been observed in 3 out of 466 randomly selected drinking water ground water systems. The mean was 0.87 $\mu g/l$. Ethylbenzene also was detected in 3 out of 479 ground water systems selected non-randomly (0.6%). All three positives were from systems serving less than 10,000 people. The mean was 0.78 $\mu g/l$.

Ethylbenzene has been identified at one hazardous waste site designated in complaints and consent decrees under the Comprehensive Emergency Response Compensation and Liability

Act of 1980 (CERCLA) and the National Contingency Plan (40 CFR Part 300). The concentration of ethylbenzene ranged from 23 mg/l in drinking water to 13 mg/l in an aquifer.

Health Effects. Ethylbenzene is absorbed readily from the gastrointestinal tracts, lungs and skin and can be expected to accumulate in adipose tissue. Urinary excretion of metabolites of ethylbenzene is the predominant route of elimination.

Ethylbenzene is not severely toxic after acute exposure. The major effects following acute and chronic exposure include liver and kidney pathologies and nervous system disorders.

No adequate dose-response data exist via the oral route of exposure from which to derive 1-day assessments. Therefore, the derivation of the 1-day levels was based upon a 100 ppm (435 mg/m^3) NOAEL identified in humans following a single 8-hour inhalation exposure (Bardodej and Bardodejova, 1970. Biotransformation of Ethylbenzene, Styrene and Alpha-Methyl Styrene in Man. Amer. Ind. Hyg. Assoc. J. 31(2):209–309). A total absorbed dose of 20.7 mg/kg was determined, assuming a human respiratory inhalation volume of 20 m^3/day and an absorption efficiency of 50 percent for a 70 kg adult. From this total absorbed dose, a 1-day assessment of 20.7 mg/l was derived for the 10 kg child, assuming consumption of 1 liter of water per day. A 1-day assessment of 72.5 mg/l for the 70 kg adult was derived, assuming consumption of 2 liters of water per day, applying an uncertainty factor of 10, appropriate for use with adequate human data.

Because of the lack of appropriate exposure duration data, the 10-day assessments are derived from the 1-day levels simply by dividing the 1-day numbers by 10 to give estimated 10 values. The resulting 10-day assessment for the 10 kg child is 2.07 mg/l and for the 70 kg adult, 7.25 mg/l.

A provisional AADI was determined based upon a laboratory study in which rats were treated orally at four dose levels, 5 days/weeks for 6 months (Wolf, et al. 1956. Toxicological Studies of Certain Alkylated Benzenes and Benzene. Arch. Ind. Health. 14:387–398). No observable effects were reported in the groups exposed at 13.8 and 136 mg/kg/day, while histopathological changes were evident in groups treated at 408 and 680 mg/kg/day. A NOAEL of 136 mg/kg/day was identified, with an uncertainty factor of 1000 based upon an animal study of significantly less than lifetime duration and a factor of 5/7 to convert from a 5 day/week dosing regimen to a 7 day/week exposure pattern, consumption of 2 liters of water per day factored in, resulting in a provisional AADI of 2.4 mg/l.

Ethylbenzene does not appear to be a mutagen, based upon testing in a limited number of essays with *Salmonella typhimurium* strains. The carcinogenicity of ethylbenzene has not been tested adequately. A long-term carcinogenicity bioassay has been initiated by the National Cancer Institute, but data are not yet available. Ethylbenzene has been classified in EPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon inadequate evidence in animals.

EPA's ambient water quality criterion (U.S. EPA. 1980. Ambient Water Quality Criteria for Ethylbenzene. 440/5–80–048) for ethylbenzene of 1.4 mg/l, based upon the TLV and an uncertainty factor of 1000. The provisional AADI was based upon actual toxicological data with an identified NOAEL, in contrast to the ambient water quality criterion which was based on the TLV which is not necessarily based on a NOAEL or toxicology data.

The taste and odor thresholds for ethylbenzene in water are 0.1 and 0.2 mg/l, respectively (Fazzalari, 1978. Odor and Taste Threshold Data, DS–48A. American Society for Testing and Materials. Philadelphia, PA. p. 71).

Ethylbenzene exposure at high dose levels results in liver and kidney effects and the contaminant has been detected in drinking water. Thus, regulation is proposed and an RMCL of 0.68 mg/l is proposed based upon a provisional AADI of 3.4 mg/l for non-carcinogenic effects assuming 20 percent of the exposure is via drinking water.

13. Ethylene Dibromide

Ethylene dibromide (1,2-dibromoethane, EDB; CAS # 106–93–4) is a pesticide. Most uses of EDB were canceled in 1984. Citrus quarantine and several very minor uses remain registered. EDB is water soluble (4,500 mg/l) and highly volatile. Recent data suggest that EDB has a half-life of approximately eight years at 20 °C under neutral conditions. More than 90 percent of the annual total production of EDB is used as an additive in leaded gasoline. In addition, EDB has been used as a soil fumigant for soybeans, cotton, peanuts, pineapples and many other fruit and vegetable crops. EDB had been used in post-harvest fumigation of stored grain and spot fumigation of grain milling machinery. Other uses have included the fumigation of field logs and the quarantine fumigation of citrus and other fruits and vegetables, application to beehives and storage vaults and for termite control.

Analytical Methods. Analytical methods available for analyzing EDB in drinking water include the purge and trap-gas chromatography technique.

Human Exposure. Ethylene dibromide has been detected in up to 60 percent of raw grain and 6 percent of grain products at levels as high as 5400 ppb. EDB residues also have been detected on up to 5 percent of the citrus products entering the U.S. food supply.

Ethylene dibromide has been reported in ambient air associated with the use of leaded gasoline. The following median concentrations were estimated for levels of EDB in ambient air: rural/remote areas, 0.0 ng/m^3; urban/suburban areas, 200 ng/m^3; and source dominated areas, 1,500 ng/m^3. Concentrations of EDB in typical urban areas ranged from 80–460 ng/m^3. The highest value detected was 240,000 ng/m^3.

EDB has been found in drinking water and ground water in Hawaii, California, South Carolina, Washington, Connecticut, Massachusetts, Georgia and Florida. Two small community water supplies in one State contained 0.1 and 2.0 μg/l of EDB. Four out of 100 wells sampled in another State contained EDB at levels ranging from 0.02–0.10 μg/l. In addition, levels of EDB ranging from 0.02–580 μg/l were detected in 25 samples of water collected from wells in a third State.

Health Effects. EDB has a high degree of acute toxicity when administered to animals orally, dermally or by inhalation. The target organs affected include the lung, liver, spleen, kidney as well as the central nervous system. Repeated exposure to EDB also may affect the live. stomach and adrenal cortex along with significant reproductive system toxicity. The testis is particularly sensitive to atrophy and antispermatogenic effects.

No appropriate dose-response data are available on non-carcinogenic effects following exposure to ethylene dibromide from which a 1-day assessment or an AADI could be derived. However, there are data from which a 10-day assessment can be derived. A LOAEL of 7.5 mg/kg/day was identified from a study by Eljack and Hrudka (1979) in which rams were given EDB subcutaneously for 12 consecutive days. Applying a series of uncertainty factors which total 10000 to accommodate for intra- and inter-species extrapolation (100), conversion of a LOAEL to a NOAEL (10) and the potential that the human is closer in sensitivity to the bull than the ram (10), and assuming that the 10 kg child drinks

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1 liter of water per day, a 10-day assessment of 0.008 mg/l is derived. The equivalent 10-day assessment for a 70 kg adult would be 0.027 mg/l.

Since there are no duration-specific data available from which to derive a 1-day assessment, it is recommended that the 10-day assessment be used for 1-day as well.

EDB has been shown to be mutagenic *in vitro* in bacteria and eukaryotic cells. Limited evidence suggests that EDB may cause clastogenic effects such as sister chromatid exchanges, chromosomal aberrations and polyploidy.

EDB has been demonstrated to be a potent carcinogen in rats and mice exposed via gavage and inhalation. The IARC have classified EDB in Group 2B; inadequate evidence for carcinogenicity in humans, sufficient evidence for carcinogenicity in animals and sufficient evidence for activity in short-term tests. EDB has been classified in EPA's Group B2, according to EPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon positive results in studies in rats and mice.

Administration of EDB to rats and mice by gavage for 49–61 weeks produced significantly increased incidences of squamous cell carcinomas of the forestomach, hemangiosarcomas of the circulatory system, hepatocellular carcinomas and liver neoplastic nodules. (NCI, 1978. Bioassay of 1,2-Dibromoethane for Possible Carcinogenicity. NCI Carcino. Tech. Rep. Ser. Co. PB 2888-438). Chronic inhalation exposures produced significantly increased incidences of nasal cavity tumors in rats of both sexes, alveolar/bronchiolar carcinomas or adenomas in mice and nasal cavity tumors in female mice. (NTP, 1982. Carcinogenesis Bioassay of 1,2-Dibromoethane in F344 Rats and $B_6C_3F_1$ Mice (Inhalation Study) NTP-80-38). Significantly increased incidences of circulatory system hemangiosarcomas, pituitary adenomas, tunica vaginalis mesotheliomas and subcutaneous fibrosarcomas also were found in the study.

The NAS (*Drinking Water and Health*, 1980. Vol. III) calculated excess cancer risk estimates for EDB. This estimate was based upon the results of a report (Olson, et al. 1973. Induction of Stomach Cancer in Rats and Mice by Halogenated Aliphatic Fumigants. J. Natl. Cancer Inst. 51:1993–1995) which describes the results of the bioassay conducted by NCI. EPA's CAG (1983) also has estimated lifetime cancer risks for EDB in ground water. These risks were calculated from a model that is essentially the one-hit model with "Weibull" timing. These estimated risks assume consumption of 2 liters of water per day by a 69 kg adult, over a 70 year lifetime and are derived from the gastric tumor response in male rats in the National Cancer Institute gavage study. Subsequently, CAG (1984) modified these estimates to determine risks posed in drinking water to a 70 kg adult, consuming 2 liters of water per day from age one to age 70.

The NAS (1980) and CAG (1984) risk estimates are shown in Table 13.

The available data indicate that EDB has carcinogenic effects in animals. It has also been found in drinking water and therefore it will be regulated. The RMCL will be based upon its carcinogenicity and an RMCL of zero is proposed.

14. Heptachlor and Heptachlor Epoxide

Heptachlor (1,4,5,6,7,8,8'-heptachloro-3a,4,7,7a-tetrahydro-4,7-methanoindene; CAS #76-44-8; epoxide CAS #1024-57-3) is an insecticide. Technical grade heptachlor is a waxy substance which contains chlordane and has a water solubility of 0.056 mg/l at 25 °C. Heptachlor rapidly oxidizes to the epoxide 1,4,5,6,7,8,8'-heptachloro-2,3-epoxy-3a,7,7a-tetrahydro-4,7-methanoindene, which is more stable and more persistent. Most registrations of heptachlor were suspended in 1978.

Prior to 1974, heptachlor was widely used for control of termites, ants, soil insects in agriculture and various insects and on gardens, lawns, turf, and ornamentals. Heptachlor was used extensively for the protection of corn crops in Ohio, Iowa, Missouri, Illinois and Indiana.

In 1974, EPA issued a Notice of Intent to cancel all registered uses of heptachlor except for subsurface control of termites and dipping of roots and tops of non-food plants. In 1978, EPA issued a final order cancelling all registrations of heptachlor subject to the 1974 Notice of Intent to Cancel. Currently, heptachlor is registered only for subsurface control of subterranean termites and dipping of roots and tops of non-food plants.

Analytical Methods. Analytical methods available for analyzing heptachlor in drinking water include the solvent extraction-gas chromatography and solvent extraction-gas chromatography/mass spectrometry techniques.

Human Exposure. In FDA's compliance program report for fiscal year 1977 on pesticides and metals, heptachlor epoxide was detected in 4.7 percent of the samples tested. In the FDA fiscal year 1979 total dietary study for adults, heptachlor epoxide, the metabolite of heptachlor, was detected in 85 percent of the dairy products samples, 20 percent of the meat, fish and poultry samples, 5 percent of the oils and fats samples, 5 percent of the root vegetable samples, and 5 percent of the oils and fats samples. The range of positive values for heptachlor epoxide was 0.2–2 μg/kg. In the infant study, heptachlor epoxide was detected in 50 percent of the whole milk samples, 80 percent of the other dairy and substitutes samples, and 70 percent of the meat, fish, and poultry samples. The range of positive values was 0.2–1 μg/kg for heptachlor epoxide. In the toddler studies for fiscal year 1979, heptachlor epoxide was detected in 80 percent of the whole milk samples, 69 percent of the other dairy and substitutes samples, 70 percent of the meat, fish, and poultry samples, and 10 percent of the oils and fats samples. The range of positive values of heptachlor epoxide in this study was 0.2–20 μg/kg. The U.S. Department of Agriculture reports that 4.7 percent of fat tissue samples from various animals intended for human consumption were found to contain heptachlor in the range of 100–6,000 μg/kg, although the majority of positive values were in the range of 10–100 μg/kg.

Milk supplies have on occasion been found to contain heptachlor epoxide above FDA's action level. Samples of human milk collected in the mid-1970s were found to contain heptachlor epoxide. In 1980-1982, the Oahu milk supply was extensively contaminated by heptachlor epoxide. The levels exceeded 1 ppm in the milk fat and typically exceeded the action level.

In a 1970–72 EPA study of levels of heptachlor in the ambient air of 16 U.S. cities, the maximum level measured was 27.9 ng/m³. The mean value for all positive samples was 1.0 ng/m³. Heptachlor was detected in 42 percent of the 2,470 samples collected during the monitoring period.

The results of the Suburban Air Monitoring Program conducted from April to June 1975, showed that all five samples collected from a city in Florida were found to contain heptachlor with a mean concentration of 2.1 ng/m³ and a maximum value of 3.8 ng/m³. All five samples collected from a city in Mississippi were found to contain residues of heptachlor. The sample mean was reported as 10.9 ng/m³, with a maximum reported value of 22.1 ng/m³.

The maximum level of heptachlor detected in analysis of weekly air samples in the Mississippi Delta during 1972–73 was reported as 0.6 ng/km³.

Heptachlor has been reported in drinking water. Three wells in one State were found to contain heptachlor; the

range of positive values was 0.01–1.0 µg/l. A rural water supply study conducted in another State showed that 62.5 percent of the samples taken in one county and 45.5 percent the samples taken in another county had concentrations of heptachlor above 0.01 µg/l.

Two out of 22 tap water samples collected in another State reportedly were found to contain heptachlor at levels between 0.005–0.25 µg/l.

Heptachlor has been identified at two hazardous waste sites designated in complaints and consent decrees under the Comprehensive Emergency Response Compensation and Liability Act of 1980 (CERCLA) and the National Contingency Plan (40 CFR Part 300). The concentration of heptachlor ranged from undetermined in migrating ground water, 38 µg/l in soil to 4800 µg/l in sediments.

Health Effects. Heptachlor epoxide is the major metabolite of heptachlor and is distributed to tissues of animals, with the highest levels detected in adipose tissue. Symptoms of acute heptachlor intoxication include central nervous system disturbances such as tremors, convulsions, paralysis and hypothermia. Lower doses result in microsomal enzyme induction, hyperplasia, hepatic vein thrombosis and cirrhosis in mice.

Ten-day assessments for heptachlor were derived based upon a feeding study (Enan, et al. 1982. Effects of Some Chlorinated Hydrocarbon Insecticides on Liver Function in White Rats. Meded. Fac. Landbouwwet. Rijksuniv. Gent. 47(1):447) in rats for 14 days where a level of 1.0 mg/kg/day (10 ppm) resulted in definitive adverse effects (i.e., evidence of liver damage and altered liver function). Using 1.0 mg/kg/day as the NOAEL, an uncertainty factor of 1000 based upon an animal study and consumption of 1 liter (child) or 2 liters of water (adult) per day, 10-day assessments of 0.01 mg/l for a 10 kg child and 0.035 mg/l for a 70 kg adult were calculated.

There are insufficient toxicological data available to derive a 1-day assessment for heptachlor; however it should be noted that the 10-day numbers would also be protective for the 1-day exposure period. Insufficient data are available to derive 1-day or 10-day numbers for heptachlor epoxide.

A provisional AADI for heptachlor was derived based upon a feeding study (Witherup, et al. 1955. The Physiological Effects of the Introduction of Heptachlor into the Diet of Experimental Animals in Varying Levels of Concentration. Unpublished report cited in Epstein, 1976. Sci. Total Environ. 6:103) in rates for 110 weeks where 0.075 mg/kg/day (1.5 ppm) was identified as a lowest effect level causing increased liver weights of animals. Using 0.075 mg/kg/day as the LOAEL, an uncertainty factor of 1000 based upon an animal study and consumption of 2 liters of water per day, a provisional AADI of 0.0025 mg/l was calculated.

For heptachlor epoxide, a provisional AADI was derived based upon a two-year oral study in dogs (Unpublished. IRDC. 1971. Two-year study Oral Study in Beagle Dogs) where 0.025 mg/kg/day (1 ppm) was identified as the NOAEL. Using 0.025 mg/kg/day as the NOAEL, an uncertainty factor of 1000 for an animal study of less than lifetime duration and consumption of 2 liters of water per day, a provisional AADI of 0.001 mg/l was calculated.

Heptachlor has been tested for mutagenicity in a number of systems. Negative results have been obtained in the bacterial assay in fruit flies, for unscheduled DNA synthesis in rats, mouse and hamster primary hepatocyte cultures and for the dominant lethal assay in mice. Positive results were reported for unscheduled DNA synthesis in transformed human fibroblasts with S-9 activation and in the dominant lethal assay in rats (Cerey, et al. 1973. Effects of Heptachlor on Dominant Lethality and Bone-Marrow in Rats. Mutat. Res. 21:28).

Heptachlor has been studied for carcinogenicity in mice and rats. In one study, statistically significant increased incidences of hepatic carcinoma were determined in a study of heptachlor epoxide at 0, 0.5, 2.5, 5.0, 7.5 and 10.0 mg/kg diet in rats for 108 weeks (Witherup, et al. 1959. The Physiological Effects of the Introduction of Heptachlor Epoxide in Varying Levels of Concentration into the Diet of CFN Rats. Unpublished report cited in Epstein, 1976. Sci. Total Environ. 6:103). In a National Cancer Institute bioassay (NCI. 1977. Bioassay of Heptachlor for Possible Carcinogenicity. NCI Carcinogenesis Tech. Rep. Ser. No. 8), male mice received dietary concentrations of 0, 6.1 and 13.6 mg heptachlor/kg diet and female mice received diets containing 0, 9.0 and 18.0 mg/kg diet. The incidence of hepatocellular carcinomas was statistically significant in the males, while a highly significant dose-related trend was also observed between high- and low-dose females. Heptachlor was not carcinogenic in male and female rats similarly treated with concentrations of 25.7 to 77.9 mg/kg diet.

EPA's CAG has derived estimates of risk of daily exposure to heptachlor in drinking water based upon the incidence of hepatocellular carcinomas in male mice in the National Cancer Institute bioassay. The NAS (*Drinking Water and Health*, 1977. Vol. I) has also estimated cancer risks. The CAG and NAS lifetime risk estimates are shown in Table 13. The IARC have classified heptachlor in Group 3; inadequate evidence of carcinogenicity in humans and limited evidence of carcinogenicity in animals. Heptachlor has been classified in EPA's Group B2, according to EPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon positive results in studies in mice.

EPA's CAG has also derived risk estimates from daily exposure to heptachlor epoxide in drinking water based upon a feeding study in female rats (Witherup, et al. 1959. The Physiological Effects of the Introduction of Heptachlor Epoxide in Varying Levels of Concentration into the Diet of CFN Rats. Unpublished report cited in Epstein, 1976. Sci. Total Environ. 6:103). The CAG risk estimates are shown in Table 13. The IARC have not classified heptachlor epoxide for carcinogenicity. Heptachlor epoxide has also been classified in EPA's Group B2, based upon positive results in studies in mice and rats.

The WHO recommended an ADI value of 0.5 µg/kg bw for heptachlor or heptachlor epoxide. This recommendation was established by the Joint FAO/WHO Expert Committee on Food Additives. A guideline value in drinking water (1984) of 0.1 µg/l was also recommended by the WHO, based upon this level as one percent of the ADI. A detection and odor threshold value of 0.02 mg/l has been reported in the literature for heptachlor.

The available data indicate that heptachlor and heptachlor epoxide have carcinogenic effects in animals. Since heptachlor epoxide is considered to be a major metabolite of heptachlor and because they have been detected in drinking water, they are proposed for regulation. An RMCL of zero is proposed for both heptachlor and heptachlor epoxide.

Question for Comment:

1. Should a single RMCL or two individual RMCLs be proposed for heptachlor and its metabolite heptachlor epoxide?

15. Lindane

Lindane (gamma is isomer of 1,2,3,4,5,6-hexachlorocyclohexane; CAS # 58-89-9; current MCL is 4 µg/l) is an insecticide registered for commercial and home use. Lindane is the active ingredient in several prescribed shampoos used for the elimination of head lice. Similar preparations are

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available for the elimination of fleas and lice on pets and farm animals.

Lindane is slightly soluble in water (7.8 mg/l at 25° C). Despite a low vapor pressure, lindane will volatilize to the atmosphere from soil or water. It is persistent in soils (half-life greater than 100 days), though it does undergo rapid biotransformation under anaerobic conditions.

Analytical Methods. Analytical methods available for analyzing lindane in drinking water include the solvent extraction gas chromatography technique.

Human Exposure. In 1977, the FDA reported finding lindane residues in 2.1 percent of the food samples tested. In the 1979 total diet study, FDA reported lindane in 12.7 percent of adult food samples with concentrations ranging from trace to 8.0 μg/kg in sugar, meat, fish, poultry, grains, cereal, vegetables and garden fruit. In the same year, 2 percent of the infant food samples and 14.7 percent of toddler food samples were positive for lindane, with concentrations ranging from 0.2 to 2 μg/kg and from 0.2 to 5 μg/kg, respectively.

Tolerances for residues of lindane established by the EPA's Office of Pesticide Programs (OPP) in or on raw agricultural commodities are given below:

- Seven parts per million in or on the fat of meat from cattle, goats, horses, and sheep.
- Four parts per million in or on the fat of meat from hogs.
- Three parts per million in or on cucumbers, lettuce, melons, mushrooms, pumpkins, squash and tomatoes.
- One part per million in or on apples, apricots, asparagus, avocados, broccoli, brussel sprouts, cabbage, cauliflower, celery, cherries, collards, eggplants, grapes, guavas, kale, kohlrabi, mangos, mustard greens, nectarines, okra, onions (dry bulb only), peaches, pears, pepper, pineapples, plums (fresh prunes), quinces, spinach, strawberries and Swiss chard.
- A 0.001 part per million (negligible residue) in or on pecans.

The USDA notes that 0.1 percent of swine samples violated allowable lindane concentrations in a 1980–1983 monitoring study. No lindane residues in excess of the tolerance were found in other meat products examined.

In a 1970–1972 EPA study of 16 U.S. cities, lindane was detected in 66 percent of the ambient air samples. The positive mean value was 0.9 ng/m³ and the maximum value reported was 11.7 ng/m³. In a 1980 EPA study monitoring 10 locations in the U.S., 0.8 percent of the samples contained detectable levels of lindane, with a mean level of 0.1 ng/m³ and a maximum level of 1.5 ng/m³.

Low levels of lindane have occasionally been found in drinking water. It has been found in drinking water in at least four States. In the National Organics Reconnaissance Survey (NORS), two out of eight surface water systems contained lindane (0.01 μg/l and trace).

National compliance data show that no surface or ground public water systems in the U.S. were reported to be in violation of the MCL for lindane.

In the Rural Water Survey (RWS) 1 out of 71 ground water systems exceeded the minimum quantification limit (0.002 μg/l) for lindane. In the NORS, water samples from the two ground water systems sampled contained lindane, but the levels were below the minimum quantifiable concentration.

Local conditions can lead to drinking water contamination. A rural water supply study in one State reported that 58.3 percent of samples contained levels of lindane greater than the detection limit of 0.01 μg/l.

Lindane has been identified at five hazardous waste sites designated in complaints and consent decrees under the Comprehensive Emergency Response Compensation and Liability Act of 1980 (CERCLA) and the National Contingency Plan (40 CFR Part 300).

Health Effects. Acute exposure of animals to lindane results in neurological and behavioral effects. Subchronic and chronic studies have shown a variety of effects, including liver hypertrophy, kidney tubular degeneration and interstitial nephritis. The liver and the kidney appear to be the primary target organs for lindane toxicity.

Insufficient data are available to derive a one-day assessment for lindane. A 10-day assessment was based upon a study (Muller, et al. 1981. Electroneurophysiological Studies on Neurotoxic Effects of Hexachlorocyclohexane isomers and gamma-pentachlorocyclohexane. Bull. Environ. Contam. Toxicol. 27:704–708) in which decreased motor conduction velocity was detected in the tail nerve of rats fed lindane for 30 days at 25.6 mg/kg. Ten-day values of 1.2 mg/l for a 10 kg child and 4.3 mg/l for a 70 kg adult were calculated from a NOAEL of a 12.3 mg/kg, with an uncertainty factor of 100 based upon an animal study and consumption of 1 liter (child) or 2 liters (adult) of water per day.

In the absence of an appropriate toxicological data base for a one-day assessment, the ten-day values of 1.2 mg/l for the child and 4.3 mg/l for the adult are recommended for use for 1-day assessments.

A provisional AADI was calculated for lindane based upon a feeding study (Research Consulting Co. Ltd., 1983. OPP Support Document) in which male and female rats were fed pure lindane in the diet at levels of 0, 0.2, 0.8, 4, 20 and 100 ppm for 84 consecutive days. Liver hypertrophy, kidney tubular degeneration, interstitial nephritis and basophilic tubules and other effects were seen at the 20 and 100 ppm levels. Effects were rare and very mild at the 4 ppm level. Using 0.8 mg/kg/day (4 ppm) as the NOAEL, an uncertainty factor of 1000 based upon an animal study of short duration and consumption of 2 liters of water per day, a provisional AADI of 0.01 mg/l was calculated.

Lindane has been tested for carcinogenicity in laboratory animals. Thorpe and Walker (1973. Food and Cosmet. Toxicol. 11:433–442) reported a significant increase in liver tumors in the treated mice relative to the controls (96% in treated males and 95% in treated females, compared to 24% and 23%, respectively, in the controls). In addition, there was evidence of tumor metastases to the lungs in both sexes. In 1977, the NCI (Technical Rep. Ser. No. 14) published the results of a lifetime bioassay of lindane in mice. The incidence of hepatocellular carcinomas in low dose (80 ppm) males was significant when compared to pooled controls, but not in the high dose (160 ppm) males. The evidence indicates that lindane is carcinogenic to mice when administered orally, producing liver tumors. The IARC have classified lindane in Group 3; inadequate evidence for carcinogenicity in humans, limited evidence for carcinogenicity in animals and inadequate evidence for activity in short-term tests. EPA's Office of Pesticide Programs has stated, "Using the new Proposed Guidelines for Carcinogen Risk Assessment (cited above), the CAG has classified lindane as 'B2–C' (i.e., in between the lower half of the 'B' category of 'probable' and the 'C' category of 'possible' carcinogen classifications) based upon evidence that lindane gives rise to malignant liver tumors in two strains of mice, plus supportive evidence of pre-cancerous liver lesions in shorter term studies." This weight of evidence classification also included consideration that, on the one hand, at least one study was positive in both sexes and a lindane metabolite has been shown to be oncogenic, while, on the other hand, and leaning the classification toward class "C", lindane has not been shown to be mutagenic after extensive testing and

none of the oncogenicity studies showed a dose-response. Therefore, the weight of evidence appears to be closer to a Category C carcinogen than to Category B2 carcinogen.

Based upon the carcinogenic effects observed in the liver of mice fed lindane and using one of several models available, the NAS (*Drinking Water and Health*, 1977, Vol. I) estimated the possible carcinogenic risk of lifetime exposure to lindane for a person consuming two liters of water per day. EPA's CAG has also derived estimates of possible risk of daily exposure to lindane in drinking water. CAG also based the risk estimate on the liver carcinogenicity observed in mice. The NAS and CAG risk estimates are shown in Table 13.

The WHO proposed an ADI of 0.01 mg/kg bw and a guideline value in drinking water of 3 µg/l. The ADI was established by the Joint FAO/WHO Expert Committee on Food Additives and the guideline in drinking water was set at a level of one percent of the ADI.

EPA's MCL for lindane in drinking water, under the National Interim Primary Drinking Water Regulations, is 0.004 mg/l. This standard is based upon a NOAEL of 15 ppm (0.9 mg/kg), with an uncertainty factor of 300 and the assumption that 20 percent of the total intake is via drinking water. New data indicate that lindane is carcinogenic in the mouse and this data was not taken into account in the establishment of the interim MCL.

The available data do not permit definitive decisions on the oncogenic potential of lindane in the rat. The Office of Pesticide Programs is requiring that a rat oncogenicity bioassay be performed (OPP, Sept. 30, 1985). Until definitive decision on the oncogenic potential of lindane is established, lindane will be classifed in EPA's Group C for the reasons outlined by the Office of Pesticide Programs, above. Lindane has been placed in Regulatory Category II and the RMCL has been set based upon the provisional AADI of 0.01 mg/l with an additional factor of 10, with 20 percent drinking water contribution factored in. The proposed RMCL is based upon a feeding study (Research Consulting., Ltd. 1983. OPP Support Document) in rats. Using 0.9 mg/kg/day (4 ppm) as the NOAEL, an uncertainty factor of 1000 based upon an animal study of short duration and consumption of 2 liters of water per day, a provisional AADI of 0.01 mg/l was calculated. An additional factor of 10 was applied because an order of magnitude uncertainty factor was determined to be sufficiently protective for this chemical. Alternatively, the RMCL could have been set based upon 1) the AADI approach using an additional uncertainty factor less than 10 due to the nature of the data, 2) the risk calculation approach (10^{-5} risk), or 3) the risk calculation approach (10^{-6} risk).

An RMCL and primary regulation will be proposed for lindane because of the potential adverse effects and occurrence in drinking water. Lindane has been classified as a possible carcinogen and the proposed RMCL of 0.0002 mg/l for lindane is based on a provisional AADI of 0.01 mg/l for non-carcinogenic effects and an additional factor of 10, assuming 20 percent contribution from drinking water.

Questions for Comment:

1. Is it appropriate to propose an AADI for lindane based on a shorter than lifetime feeding study in animals (Research Consulting Co. Ltd. 1983)?

2. In the background document on lindane, alternate studies (Fitzhugh, et al. 1960. The Chronic Toxicities of Technical Benzene Hexachloride and its Alpha, Beta and Gamma Isomers. J. Pharmacol. Expo. Ther. 100:59, and Rivett, et al. 1978. Effects of Feeding Lindane to Dogs for Periods of up to Two Years. Toxicology. 9:273) are cited in the consideration of an AADI. Comments are requested on their appropriateness in the derivation of an AADI.

3. Is it more appropriate to classify lindane in EPA's Group B2 or Group C? On what basis?

4. If lindane is classified in EPA's Group C, should the RMCL be based upon the AADI approach or the risk calculation approach? If the AADI approach is used, should an extra uncertainty factor of 10 be applied or some lesser value? If the risk approach is used should be RMCL be based upon the 10^{-5} or 10^{-6} risk level?

18. Methoxychlor

Methoxychlor [(2,2-bis (p-methoxyphenyl) 1,1,1-trichloroethane; CAS # 72-43-5; current MCL is 100 µg/l], a chemical closely related to DDT, has been used as an insecticide for approximately 40 years. Technical methoxychlor is a mixture containing 88 percent of the p,p'-isomer. In 1977, domestic consumption of methoxychlor was 10 million pounds. Methoxychlor has been widely used for home and garden applications, as well as on domestic animals, tree, and in waters. The p,p'-isomer is soluble in water to 0.26 mg/l at 25°C. The half life for methoxychlor in water is estimated to be 46 days and thus it is not considered to be persistent.

Analytical Methods. Analytical methods available for analyzing methoxychlor in drinking water include the solvent extraction-gas chromatography technique.

Human Exposure. In the FDA FY 79 total dietary studies for adults, methoxychlor was detected in 1 out of 10 dairy products sampled at 6 µg/kg. Methoxychlor was also detected in 1 out of 10 whole milk samples at a level of 2 µg/kg in the dietary study for infants. Residues of methoxychlor ranging from 2-6 µg/kg were reported in a whole milk sample, a sample of the dairy and other substitutes, and in a fruit and fruit juice sample from toddler studies. Compliance reports for FY 78 reveal that methoxychlor was detected in 1 percent of domestic food samples tested. In addition, methoxychlor levels in violation of the USDA maximum allowable level were detected in 0.2 percent of samples of calf tissues for 1982-1983.

The tolerance levels set by EPA's Office of Pesticide Programs for residues of methoxychlor in or on raw agricultural commodities are given below:

- One hundred parts per million in or on alfalfa, clover, cowpeas, grass for forage, peanuts and soybeans.
- Fourteen parts per million in or on various fruits and vegetables.
- Seven parts per million in or on sweet potatoes and yams from preharvest and postharvest application.
- Three parts per million in or on the fat of meat from cattle, goats, hogs, horses or sheep.
- Two parts per million in or on the following grains from storage-bin treatment: barley, corn, oats, rice, rye, sorghum grain and wheat.
- A 1.25 ppm in milk fat reflecting negligible residues in milk.
- One part per million in or on potatoes.
- One part per million inor on horseradish.

No data are availaable on levels of methoxychlor in ambient air.

Methoxychlor has not been detected during national drinking water monitoring surveys. Compliance monitoring has not reported methoxychlor in excess of the MCL. However, methoxychlor has been detected in drinking water in high use areas. One state study on the effects of forest runoff on the quality of water supply systems observed an ambient concentration of methoxychlor of 50 µg/l. In one county, 46 percent of the samples of rural water supplies contained detectable levels of methoxychlor (mean of 0.033 µg/l); in another, 64 percent of the samples were positive (mean of 0.023 µg/l).

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Health Effects. Methoxychlor exhibits a wide range of toxicity when administered at high dose levels to animals. Central nervous system effects were observed in dogs, chronic nephritis and cystic tubular nephropathy in swine and rats, fatty changes in the livers of swine and marked testicular atrophy in rats and swine. Histopathologic changes in the kidneys, liver or reproductive organs were not observed at dietary levels below 1000 mg/kg.

One-day assessments for methoxychlor were based upon a study investigating the effects of methoxychlor following a single oral dose of 640 mg/kg in rats (Morgan and Hickenbottom, 1978. Relative Sensitivities of Various Biochemical, Toxicological and Pathological Techniques in Demonstrating Sublethal Lesions in the Rat Following Oral Administration of Low Levels of Methoxychlor. Toxicol. Appl. Pharmacol. 45:237). The effects observed were decreased lactate and glycogen phosphorylase and increased glucose-6-phosphatase. Using 640 mg/kg/day as the LOAEL, an uncertainty factor of 1000 based upon an animal study and consumption of 1 liter (child) or 2 liters of water (adult) per day, 1-day assessments of 6.4 mg/l for a 10 kg child and 22.4 mg/l for a 70 kg adult were calculated.

Ten-day assessment for methoxychlor were based upon a study (Stein, 1966. Comparative Methoxychlor Toxicity in Dogs, Swine, Rats, Monkeys and Man. Ind. Med. Surg. 37:540) in which volunteers were given methoxychlor at levels of 0.5, 1.0 or 2.0 mg/kg/day. No effects were reported for routine biochemical and histological examination of the organs. Using 2.0 mg/kg/day as a NOAEL, an uncertainty factor of 10 and consumption of 1 liter (child) or 2 liters of water (adult) per day, 10-day assessments of 2.0 mg/l for a 10 kg child and 7.0 mg/l for a 70 kg adult were calculated.

An AADI was calculated for methoxychlor based upon a feeding study in rats where male and female rats (FDA (unpublished), 1951; Lehman, 1965), fed diets containing 10, 25, 100, 500 or 2000 mg/kg/diet methoxychlor for 2 years. Growth retardation occurred at the highest dosage level in males, and at dosage levels of 200 mg/kg/diet and above for females. Using 5 mg/kg/day (100 mg/kg/diet) as the NOAEL, an uncertainty factor of 100 based upon an animal study and consumption of 2 liters of water per day, an AADI of 1.7 mg/l was calculated.

The evidence from studies with experimental animals and *in vitro* assays indicates that methoxychlor is not a carcinogen, mutagen or teratogen under the conditions of the experiments. A National Cancer Institute bioassay (NCI. 1978. Bioassay of Methoxychlor for Possible Carcinogenicity. NCI-CG-TR-35) reported inconclusive results. Male and female rats were exposed to technical grade methoxychlor in the diet for 78 weeks. It was concluded by the investigators that methoxychlor was not carcinogenic under the conditions of the reported assay. Re-examination of the data by an independent pathologist indicated that methoxychlor was carcinogenic in the studies; however, recent reviews by EPA's CAG in 1984 of the NCI bioassay program recommended that the results be considered neither positive or negative, but inconclusive. Methoxychlor has been classified in EPA's Group D, according to EPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon inconclusive results in animal tests.

Negative results were obtained from a number of mutagenicity studies in bacteria and yeast, from assays of methoxychlor induced DNA damage in DNA repair-deficient strains, from recessive lethal assays in *Drosophila melanogaster* and in assays of unscheduled DNA synthesis in mammalian cell cultures.

EPA's MCL for methoxychlor in drinking water, under the National Interim Primary Drinking Water Regulations, is 0.1 mg/l. This level was based upon human studies which identified a NOAEL of 2 mg/kg/day, with an uncertainty factor of 100 due to short-term human data (2 months) and 20 percent contribution from drinking water. The NAS (*Drinking Water and Health*, 1977. Vol. I) has calculated an ADI for methoxychlor of 0.1 mg/kg/day based on a 2-year study (Hodge, et al., 1952. Chronic Oral Toxicity Tests of Methoxychlor in Rats and Dogs. J. Pharmacol. Exp. Ther. 104:60) in rats, using a NOAEL of 10 mg/kg/day and an uncertainty factor of 100. The AADI is based upon a lower identified NOAEL in a newer study compared with the NAS' ADI.

The WHO has established an ADI for humans of 0 to 0.1 mg methoxychlor/kg bw (Joint Meeting of the FAO Working Party of Experts on Pesticide Residues an . the WHO Expert Committee on Pesticide Residues, 1975). The basis of the ADI was not identified. The WHO has also recommended a guideline value (1984) of 30 μg/l for methoxychlor based upon drinking water as one percent of the ADI.

A detection odor threshold value of 4.7 mg/l in water for methoxhchlor is reported in the literature.

Methoxychlor has been shown to exhibit chronic toxic effects at high dose levels and has been detected in drinking water. The RMCL will be based upon non-carcinogenic effects; assuming 20 percent contribution from drinking water and based upon an AADI of 1.7 mg/l; and RMCL of 0.34 mg/l is proposed.

17. Monochlorobenzene

Monochlorobenzene (chlorobenzene CAS #108-90-7) is a solvent with a vapor pressure of 12.4 mm Hg at 25 °C. It is nearly insoluble in water. In 1981, 129,500 kkg of monochlorobenzene were produced in the U.S.

Monochlorobenzene is used as a solvent in cold cleaning operations and pesticide manufacturing. These uses accounted for half of its production in 1978. Releases of monochlorobenzene to air from production and use processes in 1978 where estimated to be 79,500 kkg. Data are not available on releases to land and water. Quantities of monochlorobenzene entering surface waters are expected to volatilize to ambient air. Little informationis available on the fate of monochlorobenzene in the atmosphere.

Analytical Methods. Analytical methods available for analyzing monochlorobenzene in drinking water include the purge and trap-gas chromatography and the purge and trap-gas chromatography/mass spectrometry techniques.

Human Exposure. Insufficient data are available on levels of monochlorobenzene in foods to determine a daily dietary intake.

Ambient air monitoring data are available from over 800 site locations in 18 States. From median values of these data, the estimated rural/remote, urban/suburban and source dominated levels of monochlorobenzene in ambient air approximate 0.0, 1500 and 140 ng/m^3, respectively. Therefore, respiratory intake for the adult male is expected to vary from 0 to 32 μg/kg/day. Respiratory intake for formula-fed infants could vary between 0 and 22 μg/kg/day.

Drinking water monitoring data indicated that 99.9 percent of the public drinking water systems contain either no monochlorobenzene or levels less than 0.5 μg/l; 0.1 percent are estimated to have levels of monochlorobenzene in drinking water ranging from 0.5 to 5 μg/l. All exposure to monochlorobenzene in drinking water at levels above 0.5 μg/l is projected to come from ground water sources. Monochlorobenzene has been detected in drinking water in three States.

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Monochlorobenzene has been identified at five hazardous waste sites designated in complaints and consent decrees under the Comprehensive Emergency Response Compensation and Liability Act of 1980 (CERCLA) and the National Contingency Plan (40 CFR Part 300). The concentration of monochlorobenzene was not given for four sites; however, ground water migration was mentioned as a problem at two sites, one of which had a concentration in ground water of 100 mg/l.

Health Effects. Acute, high dose effects of monochlorobenzene include sedation, anesthesia and death due to respiratory failure. Chronic exposure to these compounds may result in blood dyscrasias and lung, liver and kidney damage. The principal target organs of monochlorobenzene are the central nervous system, liver and kidney.

No adequate dose-response data are available from which to derive a 1-day assessment for either the 10 kg child or the 70 kg adult. It is recommended that, for this duration of exposure, the 10-day assessment be applied.

The 10-day assessment was derived from inhalation data in the teratology study in rats and rabbits (Hayes, et al., 1982. Monochlorobenzene inhalation teratology study in rats and rabbits. Unpublished report. Toxicology Research Laboratory. Dow Chemical Company. 115 pp.). Animals were exposed at 0, 75, 210 or 500 ppm, 6 hr/day on days 6–15 (rat) or days 6–16 (rabbit) or pregnancy. No toxicity, fetal or maternal, was observed in the rats at any dose. In the rabbit dams, increased relative and absolute liver weights were observed at the two higher doses. Using the NOAEL of 75 ppm (18 mg/kg/day), a 10-day assessment for the 10 kg child of 1.8 mg/l was derived. A comparable assessment for the 70 kg adult would be 0.3 mg/l.

The study selected for the derivation of the provisional AADI was a subchronic study in which rats and mice were administered monochlorobenzene five times weekly by gavage in corn oil at doses of 0, 60, 125, 250, 500 or 750 mg/kg (Battelle-Columbus, 1978c. Chlorobenzene. Subchronic Toxicity Study $B_6C_3F_1$ Mice. Unpublished report; Battelle-Columbus, 1978d. Chlorobenzene. Subchronic Toxicity Study—Fischer 344 Rats. Unpublished report). This study showed significantly increased liver/body weight ratios in mice of both sexes at 500 and 750 mg/kg, and a slight increase in males at 125 mg/kg. Both male and female rats showed an increase in liver/body weight ratios at 250 and 500 mg/kg. Mice and rats at the three highest doses all exhibited hepatic necrosis, nephrosis and lymphoid necrosis. A NOAEL of 125 mg/kg/day was identified from this study. Using this NOAEL, an uncertainty factor of 100 based upon an animal study with the NOAEL identified, and uncertainty factor of 10 to convert from subchronic to chronic exposure and consumption of 2 liters of water per day, a provisional AADI of 3.0 mg/l was determined.

Using the same NOAEL of 125 mg/kg/day and an uncertainty factor of 100 based upon an animal study identify a NOAEL and adjusting for exposure, a 10-day assessment of 20 mg/l for a 70 kg adult and 9 mg/l for a 10 kg child can be derived. These levels are also protective for 1-day exposures in the adult and child.

Monochlorobenzene has been shown to cause mutagenic effects in higher plants and certain microorganisms. Monochlorobenzene was tested for its carcinogenic potential in rats and mice in the NTP bioassay program (NTP, 1983. Draft NTP Technical Report of the Carcinogenicity Bioassay of Chlorobenzene (CAS #108-90-7) in F344/N Rats and $B_6C_3F_1$ Mice). The results of the study showed that monochlorobenzene administration increased the occurrence of neoplastic nodules of the liver in the high dose (120 mg/kg) male rats. Carcinogenic effects were not observed in female rats or mice of either sex. On the basis of these preliminary data, the NAS (*Drinking Water and Health*, 1983. Vol. V) derived an estimate of risk of daily exposure to monochlorobenzene in drinking water. This risk estimate is shown in Table 13.

EPA's ambient water quality criteria (U.S. EPA. 1980. Ambient Water Quality Criteria for Chlorinated Benzenes 440/5-80-028) for monochlorobenzene are 488 μg/l based upon toxicity data and 20 μg/l based upon organoleptic data. The level based upon toxicity data (488 μg/l) was calculated based upon a NOAEL from animals of 14.4 mg/kg/day and an uncertainty factor of 1000. The provisional AADI was based upon newer data than as used to derive the water quality criteria level. the WHO drinking water quality guideline for monochlorobenzene (1984) is 0.3 μg/l, based upon organoleptic considerations.

Monochlorobenzene has been classified in EPA's Group C, according to EPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon the preliminary data from the NTP study. Monochlorobenzene has been placed in Regulatory Category II and the RMCL has been set based upon the provisional AADI of 3.0 mg/l with an additional factor of 10, with 20 percent drinking water contribution factored in. The RMCL is proposed based upon chronic toxicity data because the bioassay data (NTP study) are preliminary and an inadequate basis for extrapolation. An additional factor of 10 was applied because an order of magnitude uncertainty factor was determined to be sufficiently protective for this chemical.

An RMCL and primary regulation will be proposed for monochlorobenzene because of the potential adverse effects and occurrence in drinking water. Monochlorobenzene has been classified as a possible carcinogen and the proposed RMCL of 0.08 mg/l for monochlorobenzene is based on a provisional AADI of 3.0 mg/l for non-carcinogenic effects and an additional factor of 10, assuming 20 percent contribution from drinking water.

Questions for Comment:

1. Should monochlorobenzene be classified in EPA's Group C?

2. What should be the basis for the RMCL for this compound?

3. The 10-day assessments, based upon a study done in rabbits, are lower than the Longer-term assessments or the AADI which were derived from studies performed with rats and mice. It is apparent that these species exhibit differing sensitivities to the chemical. That the rabbit were used for the 10-day assessment is in keeping with the philosophy of using information from the most sensitive species tested. However, data on the rabbit were not available for longer durations of exposure. Is it appropriate to use the rabbit data for the 10-day assessment or should data from the comparable study in the rat have been used? Should the Longer-term assessment and/or the AADI be modified to accommodate for the lack of data in the more sensitive species and, thus, perhaps, an inadequate margin of safety when attempting to identify a level of exposure for the human that would be protective?

18. Polychlorinated Biphenyls

Polychlorinated biphenyls (PCBs) are a class of colorless and stable chemicals. They contain a biphenyl nucleus with two or more substituent chlorine atoms. Technical PCBs are mixed isomers from 10 classes of chlorobiphenyls containing 209 possible isomers. In general, PCBs are insoluble in water and are soluble in many common organic solvents.

Prior to 1971, mixtures containing up to 68 percent chlorine were used in a number of applications, including plasticizers, heat transfer fluids, hydraulic fluids, compressor lubricants, wax extenders, waterproofing aids (in surface coatings), printing inks,

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carbonless copy paper, as well as ingredients in lacquers, paints, varnishes and special adhesives. In 1974, PCBs were a major component of capacitors and of transformers and millions of pounds were manufactured. After 1976, the manufacture of PCBs was discontinued in the U.S.

Analytical Methods

Analytical methods available for analyzing PCBs in drinking water include the solvent extraction gas chromatography technique.

Human Exposure. The FDA estimated the total adult intake of PCBs to be 0.93 μg/day. The major sources are dairy products (0.37 μg/day); meat, fish, and poultry (0.52 μg/day); and fats and oils (0.03 μg/day). Trends in daily intake for the years 1977, 1978, and 1979 are 0.016, 0.027 and 0.014 μg/kg body weight, respectively.

No information was available on human intake of PCBs from ambient air.

National data on the level of PCBs in drinking water comes from the National Organics Monitoring Survey (NOMS) conducted in 1976–77. PCBs were found in 6 percent of finished ground water supplies at levels of 0.1 μg/l. PCBs were detected in approximately 2 percent of finished surface water systems; quantified levels were less than or equal to 1.4 μg/l. In a report published in 1978, one State indicated that PCBs were observed in 32 out of 163 ground water supplies sampled; concentrations as high as 1.27 μg/l were detected.

PCBs have been identified at ten hazardous waste sites designated in complaints and consent decrees under the Comprehensive Emergency Response Compensation and Liability Act of 1980 (CERCLA) and the National Contingency Plan (40 CFR Part 300). The concentration of PCBs ranged from no level given for three sites, 4–20 μg/l in runoff, 11.7–200 μg/l in sediments, 127–444,000 μg/l in soil, 200μg/l in creek water, 7–175 μg/l in leachate, 60–215 pounds in an underground oil-water layer, to 28 μg/l in pond effluent.

Health Effects. Short and longer term exposure to PCBs in animals in a variety of physiological and morphological alterations to the liver including liver enlargement, fatty infiltration, centrilobular necrosis and effects on liver prophyrin metabolism. The major biochemical effects of PCBs are the inductive effect on hepatic mixed-function oxidase enzymes and modification of prophyrin metabolism. Short-term assessments or a provisional AADI have not been determined for PCBs.

PCBs have been studied for mutagenic effects using *Salmonella typhimurimum*, in the presence of a microsomal activation system. The results showed 4-chlorobiphenyl to be significantly mutagenic in this assay. A National Cancer Institute study (NCI, 1978. NCI Carcinogenesis Technical Report Series No. 38) examined the effects of PCBs ingested through the diet for 2 years. The results showed a high incidence of hepatocellular proliferative lesions as well as adenofibrosis; however, the incidence of hepatocellular adenomas and carcinomas was not found to be significant. In another study (Kimbrough, et al. 1975. Journal NCI. 55:1453), feeding of PCBs at a concentration of 100 mg/kg for 21 months resulted in neoplastic nodules and hepatocellular carcinomas. The IARC classified PCBs in Group 2B; inadequate evidence for carcinogenicity in humans, sufficient evidence for carcinogenicity in animals and inadequate evidence for activity in short-term tests. PCBs have been classified in EPA's Group B2, according to EPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon the positive results in studies in mice and rats.

EPA's CAG and the NAS have derived estimates of risk of daily exposure to PCBs based upon a long-term study (Kimbrough, et al. 1975 Journal NCI. 55:1453), in which rats were fed PCBs (Arochlor 1260) at a dose of 100 mg/kg body weight. The CAG and NAS risk estimates are shown in Table 13.

The available data suggests that PCBs (Arochlor 1260) have carcinogenic effects in animals. For this reason and because of the occurrence and potential occurrence of PCBs in drinking water, an RMCL and a primary regulation will be proposed. An RMCL of zero is proposed for PCBs as a class of compounds, based upon the carcinogenic effects noted for Arochlor 1260.

Questions for Comment:

1. Is there adequate evidence to base the RMCL for all PCBs upon carcinogenic effects?

2. PCBs include several chlorinated biphenyls and unknown contaminants. Variability in the health effects of several PCB isomers have also been reported. Is setting regulations for PCBs as a class of compounds appropriate? What alternative approach would be better?

19. Pentachlorophenol

Pentachlorophenol (PCB or penta) has been used as a herbicide, defoliant, insecticide, fungicide and wood preservative. Eighty percent of the pentachlorophenol produced is used in the treatment of wood. Pentachlorophenol is also used in termite prevention on farms. The only currently registered agricultural uses are seed treatment or seed crop uses. Pentachlorophenol is slightly soluble in water (18 mg/l at 25 °C). Pentachlorophenol has been used in wood products that come in contact with drinking water.

In July 1984, the Agency issues a notice of intent to cancel registrations for pesticides containing pentachlorophenol and/or its salts unless labeling was amended. EPA restricted wood preservative use to certified applicators; added protective measures to reduce exposure; prohibited application in homes and to most wood intended for indoor use or in contact with food, feed, or water; prohibited use of pentachlorophenol treated logs for construction of log homes; and required a limit on the hexadioxin contaminant. This Agency action has been challenged and administrative proceedings are underway. EPA has also issued a data call-in for non-chronic data such as mutagenicity, metabolism and air monitoring studies. In late 1984, the Agency also initiated actions to cancel virtually all registrations of pentachlorophenol for non-wood preservative uses.

Analytical Methods. Analytical methods available for analyzing pentachlorophenol in drinking water include the solvent extraction-gas chromatography technique.

Human Exposure. Although not detected in adult diet studies, pentachlorophenol was detected in 10 percent of oils and fat samples in the FDA infant diet study in 1979 at a concentration of 18 μg/kg. Additional data were obtained on the estimated total intake of pentachlorophenol for adults in 1974–1979 and for infants in 1975–1979. The average total intakes for adults, infants and toddlers over the years studied were 0.010, 0.005 and 0.009 μg/kg/day, respectively. The FDA toddler diet study of 1979 detected pentachlorophenol in 10 percent of the oils and fats samples at a concentration of 24 μg/kg.

Pentachlorophenol has been detected in rivers and streams (0.01–16 μg/l) and limited data show it may occur in drinking water from surface supplies. Pentachlorophenol was detected in the NSP in two surface water systems (1.3 and 12 μg/l). None of the 12 ground water systems examined for pentachlorophenol in the NSP contained levels in excess of the quantification limit of 1.0 μg/l.

Pentachlorophenol has been identified at one hazardous waste site designated

in complaints and consent decrees under the Comprehensive Emergency Response Compensation and Liability Act of 1980 (CERCLA) and the National Contingency Plan (40 CFR Part 300). The concentration of pentachlorophenol was not given.

Health Effects. Pentachlorophenol is rapidly absorbed following oral exposure, with the major sites of tissue deposition being the livers, kidneys, brain, spleen and fat. It is rapidly excreted following a single dose, while during chronic exposure the biological half-life appears to be approximately 20 days. The major route of excretion is via the urine.

The major targets of pentachlorophenol toxicity are the liver, kidneys and central nervous system. Pentachlorophenol produces pigmentation, increases liver weight and induces hepatic enzymes in the liver. The major renal effects have been reported to be increased kidney weight and pigmentation, while effects on the central nervous system include capillary congestion and chromatolysis of the nerve cells. Pentachlorophenol is fetotoxic and has adverse effects on reproduction.

One-day assessment were calculated based on a study in rats (Nishimura, et al. 1982. Effects of Pentachlorophenol on the Levels of Hepatic Glycogen. Sangyo Isaku. 24(4):388–389) where a single oral dose of pentachlorophenol was shown to increase liver weights. Using a NOAEL of 10 mg/kg/day, an uncertainty factor of 100 and consumption of 1 liter (child) or 2 liters (adult) of water per day, a 1-day assessments of 1.0 mg/l for a 10 kg child and 3.5 mg/l for a 70 kg adult were calculated.

Ten-day assessments were calculated based on a NOAEL of 3 mg/kg/day for liver pathology in rats in a 90-day feeding study with pentachlorophenol (Johnson, et al. 1973. Chlorinated Dibenzodioxins and Pentachlorophenol. Environ. Health Persper. Exp. Issue No. 5:171–175) and for reproductive effects in a one-generation reproduction study in rats fed pentachlorophenol in the diet (Schwetz, et al. 1978. Results of 2-year Toxicity and Reproduction Studies on Pentachlorophenol in Rats. In: T.R. Rao, ed. Pentachlorophenol: Chemistry, Pharmacology and Environmental Toxicology, Plenum Press, NY pp. 301–309). Using an uncertainty factor of 100 and consumption of 1 liter (child) or 2 liters (adult) of water per day, 10-day assessments of 0.3 mg/l for a 10 kg child and 1.1 mg/l for a 70 kg adult were calculated.

An AADI was calculated for pentachlorophenol based upon a 24-month feeding study in which rats were fed doses of 0, 1, 3, 10 or 30 mg commercial pentachlorophenol/kg bw/day (Schwetz, B.A., J.F. Quast, P.A. Keeler, L.G. Humiston and R.J. Kociba. 1978. Results of 2-Year Toxicity and Reproduction Studies on Pentachlorophenol in Rats. In: K.R. Rao, ed. Pentachlorophenol: Chemistry, Pharmacology and Environmental Toxicology. Plenum Press. N.Y. pp. 301–309). At the highest dose level, increased serum glutamic pyruvic transaminase activity and pigmentation of the liver and kidney were observed in both males and females. At a dose of 10 mg/kg bw/day, pigmentation of the liver and kidney was observed in the females, but not in the males. An AADI of 1.1 mg/l was calculated from a NOAEL of 3 mg/kg/day, with an uncertainty factor of 100 based upon an animal study and consumption of 2 liters of water per day.

Commercial grades of pentachlorophenol used in toxicity studies with animals commonly contain the following nonphenolic contaminants: hexachloro-p-dioxin, heptachloro-p-dioxin, octachloro-p-dioxin, hexachlorodibenzofuran, heptachlorodibenzofuran, and octachlorodibenzofuran. These contaminants affect the toxicity of the pentachlorophenol being studied and would result in toxic effects being noted which could not be attributed to pentachlorophenol. 2,3,7,8-Tetrachlorodibenzo-p-dioxin was not detected in commercial pentachlorophenol used in the toxicity tests described above.

The NAS (*Drinking Water and Health*, 1977. Vol. I) has calculated an ADI of 0.021 mg/l based on a 90-day feeding study in rats (Johnson, R.L., P.J. Gehring, R.J. Kociba and B.A. Schwetz, 1973. Chlorinated Dibenzodioxins and Pentachlorophenol. Environ. Health Persper. Exp. Issue No. 5, Sept. 1973, pp. 171–175). This value was determined before the results of the 24-month rats study which is more appropriate for an AADI calculation, due to the length of the study.

Pentachlorophenol has not been shown to be mutagenic in *Salmonella typhimurium*, *Escherichia coli* or *Serratia marcescens*. Two oral carcinogenicity studies have been carried out on pentachlorophenol. There were no significant differences in tumor response between treated and control animals in either of these studies. The National Toxicology Program is currently testing pentachlorophenol for carcinogenic activity. No data from these studies are currently available. The IARC has classified pentachlorophenol in Group 3; inadequate evidence for carcinogenicity in humans, inadequate evidence for carcinogenicity in animals and inadequate evidence for activity in short-term tests. Pentachlorophenol has been classified in EPA's Group D, according to EPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon inadequate data in animal studies.

The EPA ambient water quality criterion for Pentachlorophenol (U.S. EPA. 1980. Ambient Water Quality Criteria for pentachlorophenol. EPA 440/5-80-065) is either 1.01 mg/l based on toxicity data or 0.03 mg/l based on the organoleptic properties of pentachlorophenol. The 1.01 mg/l value is based on the same study used for the AADI calculation and also considers bioconcentration by fish. In an EPA Position Document 4 on Wood Preservative Pesticides, the Agency regulatory position to restrict usage of pentachlorophenol considered, with respect to health risk, teratogenicity and fetotoxicity of pentachlorophenol and oncogenicity of its hexachlorodibenzo-p-dioxin (HxCDD) and hexachlorobenzene contaminants.

Odor thresholds of 857 μg/l at 30 °C and 1600 μg/l at 20–22 °C and a taste threshold of 30 μg/l have been reported in the literature for pentachlorophenol. The WHO guideline for pentachlorophenol (1984) is 10 μg/l. This value is based upon an ADI of 3 μg/kg body weight with 10 percent of the ADI attributable to drinking water. The ADI was based upon an uncertainty factor of 1000 applied to unspecified animal data.

Although commercial pentachlorophenol contains residues of hexa- and octachlorodioxin, the RMCL is based upon the parent pentachlorophenol. This is because data indicates that the mobility of the dioxin contaminants is often less than the mobility of the parent compound and thus the parent compound would be the chemical of concern in drinking water. The potential exposure to the dioxin contaminant will be dealt with separately (see section on 2,3,7-tetrachlorodibenzo-p-dioxin).

Exposure to pentachlorophenol at sufficient levels results in effects on the liver and kidney. Since pentachlorophenol has been detected in drinking water and is likely to be occurring in drinking water, an RMCL and primary regulation will be proposed. An RMCL of 0.2 mg/l is proposed based upon an AADI of 1 mg/l and an assumed drinking water contribution of 20 percent.

Questions for Comment:

1. Are additional data available on exposure through food, air and other sources for pentachlorophenol?

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2. Is there adequate toxicological evidence that the presence of detected non-phenolic contaminants in commercial pentachlorophenol would affect the AADI?

20. Styrene

Styrene (vinyl benzene or ethenyl benzene; CAS # 100-42-5) is slightly soluble in water (0.32 mg/l) and soluble in many organic solvents. In 1982, the total U.S. production of styrene equaled 5,842,037 pounds. Styrene is used extensively for the manufacture of plastics, including polystyrene, rubber modified impact polystyrene, acrylonitrile-butadiene-styrene (ABS), and styrene-acrylonitrile copolymer (SAN). It is also used in the manufacture of synthetic rubber, resins, and insulators. Some resins manufactured from styrene are used in the treatment of potable water.

Analytical Method. Analytical methods available for analyzing styrene in drinking water include the purge and trap-gas chromatography technique.

Human Exposure. No information is available on the occurrence of styrene in food. Low level contamination of some foods is expected because polymers and resins of styrene used in packaging commonly contain a detectable level of the monomer.

Data on exposure to styrene through inhalation of ambient air are limited. Styrene has been detected in ambient air in source dominated areas at approximately 2.3 $\mu g/m^3$. This level could lead to a respiratory intake for adults of approximately 52 μg/day.

Nearly 1000 drinking water samples from ground water and 100 surface water supplies have been tested in national monitoring surveys to determine the presence or absence of styrene. Styrene was not detected in any of these samples.

Styrene has been identified at one hazardous waste site designated in complaints and consent decrees under the Comprehensive Emergency Response Compensation and Liability Act of 1980 (CERCLA) and the National Contingency Plan (40 CFR Part 300). The concentration of styrene was not given.

Health Effects. Styrene is readily absorbed from the gastrointestinal tract and distributed throughout the body, accumulating in adipose tissue. Styrene is metabolized to styrene oxide, a highly reactive intermediate. The acute toxicity of styrene is relatively low, with reduced weight gain, increased kidney and liver weights and lung congestion exhibited following non-lethal exposures. Repeated inhalation exposures at very high dose levels in animals have been reported to result in alterations in hepatic enzyme activity and biochemical changes in the brain.

One-day assessments are based upon a study (Srivastava, et al. 1982. Hepatic Effects of Orally Administered styrene in Rats. J. Appl. Toxicol. 2(4)219–222) in which the NOAEL for increased sensitivity of dopamine receptors in the brain was determined to be 270 mg/kg/day. An uncertainty factor of 100 was applied and the consumption of drinking water was based upon 2 liters/day for the adult and 1 liter/day for the 10 kg child. The one-day number is 94.5 mg/l for the adult and 27 mg/l for the 10 kg child.

Ten-day assessments are based upon minor hepatotoxicity in rats (Agrawal, et al. 1982. Effects of Styrene on Dopamine Receptors. Bull. Environ. Contam. Toxicol. 29(4):400–403). A NOAEL of 200 mg/kg day was determined, with an uncertainty factor of 100 and consumption of 1 liter (child) or 2 liters (adult) of water per day. Ten-day numbers of 70 mg/l for the adult and 20 mg/l for the 10 kg child were determined.

A provisional AADI was calculated from a study where beagle dogs were given styrene in a peanut oil suspension by gavage 7 days/wk for 560 days (Quast, J.F., R.P. Kalnins, K.J. Olson, et al., 1978. Results of a toxicity study in dogs and teratogenicity studies in rabbits and rats administered monomeric styrene. Tox. Appl. Pharm. 45:293–294). Dose levels were 200, 400 or 600 mg/kg bw/day. The controls received peanut oil only. At the two higher dose levels, minimal histopathogolic effects were noted in the liver (increased iron deposits within the reticuloendothelial cells) as well as hematologic effects that included increased Heinz bodies in erythrocytes and a decreased packed cell volume. At the lowest dose level (200 mg/kg/day), these effects were not noted. Using 200 mg/kg/day as a NOAEL in both species and an uncertainty factor of 1000 based on an animal study of less than lifetime exposure and consumption of 2 liters of water per day, a provisional AADI of 7 mg/l was determined.

Styrene has not been shown to be mutagenic in *Salmonella typhimurium* in the absence of metabolic activation, while positive results have been obtained in yeast, fruit flies and cultured mammalian cells with metabolic activation. Styrene produced positive results in cultured mammalian cells, isolated human lymphocytes and when tested *in vivo* in rats and mice.

The NAS (*Drinking Water and Health*, 1977, Vol. I) has calculated an ADI of 0.133 mg/kg/day based upon a study in rats (Wolf, M.A., et al. 1956. Toxicological Studies of Certain Alkylated Benzenes and Benzene. Arch. Ind. Health. 14:387–389). Using a NOAEL of 0.133 mg/kg/day, with an uncertainty factor of 1000 and the assumption that 20 percent of total intake is via drinking water, a value of 0.9 mg/l was determined.

Styrene has been tested for carcinogenicity in several animal studies. In one study (Ponomarkov, V.F. and Tomatis, L., 1980. Effects of Long Term Oral Administration of Styrene to Mice and Rats. Scand. J. Work Environ. Health. 4 (Suppl. 2)127–135), an increased incidence of lung tumors was found in one strain of mice, but this dose resulted in excessive early mortality. Elevated tumor incidences were not observed in another strain of mice or rats. In the National Cancer Institute bioassay of styrene, a statistically significant increase in alveolar/bronchiolar adenomas and carcinomas was seen in male and female rats at a dose of 300 mg/kg/day (NTP, NCI No. 1985, 1979). In an inhalation study (Jersey, G.M., et al. 1978. Two year Chronic Inhalation Toxicity and Carcinogenicity Study on Monomeric Styrene in Rats. Dow Chemical Study for MCA. Dec. 6, 1978), the incidence of leukemia/lymphosarcomas was slightly elevated in female rats. The IARC has classified styrene in Group 3; inadequate evidence for carcinogenicity in humans, limited evidence for carcinogenicity in animals and sufficient evidence for activity in short-term tests.

Styrene has been classified in EPA's Group C, according to EPA's Proposed Guidelines for Carcinogen Risk Assessment based upon several positive animal studies which are limited due to poor survival of the animals. Styrene has been placed in Regulatory Category II and the RMCL is proposed based upon the provisional AADI of 7.0 mg/l with an additional factor of 10, with 20 percent drinking water contribution factored in. The RMCL is based upon chronic toxicity data because the bioassay data are very limited and inadequate for extrapolation purposes. An additional factor of 10 was conservatively applied based upon the equivocal evidence of carcinogenicity.

The odor threshold for styrene is between 10 and 60 ppm while the taste threshold is between 0.005 and 0.773 mg/l.

Exposure to styrene at high dose levels results in adverse health effects in animals. Because styrene is likely to be occurring in drinking water, EPA will propose an RMCL and primary regulation. An RMCL of 0.14 mg/l is proposed, based upon an AADI of 7 mg/

l with an additional factor of 10 and 20 percent drinking water contribution.

Question for Comment:

1. Should styrene be classified in EPA's Group C? What should be the basis for the RMCL for this compound?

21. Toluene

Toluene (methyl benzene; CAS #108-88-3) is an aromatic solvent. It is slightly soluble in water (534.8 mg/l) and is soluble in alcohol, benzene, ether, acetone, and other organic solvents. Much of the toluene produced is blended directly into aviation gasoline. Toluene also is used as a starting material in the production of benzene and other chemicals, and is used as a solvent for paints, coatings, gums, oils, and resins.

Analytical Method. Analytical methods available for analyzing toluene in drinking water include the purge and trap-gas chromatography technique.

Human Exposure. No information was available on the intake of toluene from food.

Data compiled for EPA's Office of Air Quality Planning and Standards indicate that the mean ambient air levels of toluene are 0.3 $\mu g/m^3$ for rural/remote areas, 2.6 $\mu g/m^3$ for urban/suburban areas, and 23 $\mu g/m^3$ for source dominated areas. Using the value of 3.6 $\mu g/m^3$, the mean respiratory intake of toluene for adults is calculated to be 83 μg/day.

In the CWSS, toluene was measured in two ground water systems, at concentrations of 0.305 and 0.98 $\mu g/l$. Three surface water supplies had measurable concentrations of toluene—0.52, 0.72 and 1.62 $\mu g/l$. Toluene was detected in six randomly selected systems tested during the GWSS. Levels ranged from 0.5–2.9 $\mu g/l$.

In the NSP, approximately 20 percent of the surface water supplies sampled contained detectable levels of toluene in finished water. Measured concentrations ranged from 0.1 to 1.4 $\mu g/l$, with a mean of 0.295 $\mu g/l$. A level of 0.1 $\mu g/l$ was measured in the one ground water system in which toluene was detected.

State agencies have also detected toluene in drinking water. Levels as high as 2,500 $\mu g/l$ have been reported.

Health Effects. Acute exposure to high levels of toluene in animals results in central nervous system depression and effects on the lungs, liver and kidney. Toxic effects following chronic exposure are similar to those seen following acute exposure, predominantly on the kidneys and lungs (if via inhalation).

No adequate dose-response data developed via the oral route of exposure exist from which to derive 1-day assessments. Thus, their derivation was based upon a 100 ppm (377 mg/m^3) NOAEL identified in humans following single inhalation exposures of up to 8 hours (von Oettingen, et al. 1942a. The Toxicity and Potential Dangers of Toluene, with Special Reference to its Maximal Permissible Concentration. U.S. Public Health Service Pub. Hlth. Bull. No. 278. 80 pp; von Oettingen, et al. 1942b. The Toxicity and Potential Dangers of Toluene—Preliminary report. J.A.M.A. 118:379–384; Carpenter, et al. 1944. Studies on the Inhalation of 1,3-Butadiene; with a Comparison of its Narcotic Effect with Benzol, Toluol and Styrene, with a Note on the Elimination of Styrene by the Human. J. Ind. Hyg. Toxicol. 26:69–78; Ogata, et al. 1970. Urinary Excretion of Hippuric Acid and m- or p-Methylhippuric acid in the Urine of Persons Exposed to Vapors of Toluene and m- or p-Xylene as a Test of Exposure. Brit. J. Ind. Med. 27(1): 43–40). A total absorbed dose of 18 mg/kg was determined, assuming a human respiratory inhalation volume of 20 m^3/day and an absorption efficiency of 50 percent for a 70 kg adult. From this total absorbed dose, a 1-day number of 18 mg/l was derived for the 10 kg child, assuming consumption of 1 liter of water per day. A 1-day number of 63 mg/l was derived for the 70 kg adult, assuming consumption of 2 liters of water per day, applying an uncertainty factor of 10, appropriate for use with adequate human data.

Because of the lack of appropriate exposure duration data, the 10-day assessments were derived from the 1-day assessments by dividing each 1-day number by 3 to give estimated 10-day values. The use of a 3-fold uncertainty factor, rather than the more usual 10-fold factor, is substantiated by the nature of the kinetic and toxic properties of the compound, i.e., rapid uptake and excretion and little bio-accumulation potential. The resulting 10-day number for the 10 kg child is 6 mg/l and for the 70 kg adult, 21 mg/l.

An AADI has been determined for toluene based upon a laboratory study in which rats were exposed to toluene via inhalation at 0, 113, 337 or 1130 mg/m^3 for 6 hours/day, 5 days/week for up to 2 years (C.I.I.T., 1980. Unpublished. A Twenty-four Month Inhalation Toxicology Study in Fisher-344 Rats Exposed to Atmospheric Toluene). No dose-related adverse effects were identified from this study and a NOAEL of 1130 mg/m^3 was identified. Using this NOAEL, an uncertainty factor of 100 based upon an animal study and assuming 50 percent pulmonary absorption, an AADI of 10.1 mg/l was determined.

Toluene has not been shown to be mutagenic in the Ames *Salmonella typhimurium* assay, with or without rat liver S-9 homogenate, and has been shown to be ineffective in increasing the reversion of an *E. coli* strain to tryptophan-independence.

Limited data are available on the carcinogenic effects of toluene. Only one long-term bioassay of toluene has been conducted; this study (C.I.I.T., 1980. Unpublished) concluded that toluene is not carcinogenic following inhalation in rats. The National Toxicology Program is conducting 2-year carcinogenicity bioassays on toluene in which the compound is being administered by inhalation and gavage to rats and mice. Toluene has been classified in EPA's Group D, according to EPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon negative results in an inhalation study and inadequate data through ingestion exposure.

EPA's ambient water quality criterion (U.S. EPA. 1980. Ambient Water Quality Criteria for Toluene. 440/5-80-075) for toluene is 14.3 mg/l based upon a 6-month oral study in rats (Wolf, et al. 1956. Toxicological studies of Certain Alkylated Benzenes and Benzene. Arch. Ind. Health. 14:387). The study used to derive the AADI is a lifetime study and was thus determined to be more appropriate for the derivation of the AADI than the 6-month study described above.

The odor threshold for toluene in drinking water is 1 mg/l.

Exposure to toluene at high dose levels results in central nervous system depression and other systemic effects; it has occurred in many drinking water supplies. Thus, a primary regulation will be proposed. The RMCL is proposed at 2.0 mg/l based upon an AADI of 10.1 mg/l for non-carcinogenic effects assuming 20 percent contribution from drinking water.

22. Toxaphene

Toxaphene (a mixture of C_{10} chlorinated camphenes with an approximate overall empirical formula of $C_{10}H_{16}Cl_8$; current MCL is 5 $\mu g/l$) is a persistent, broad spectrum insecticide. This product was used extensively on food and fiber crops for many years, but current registered uses are limited.

The solubility of toxaphene in water is approximately 0.4 mg/l. The EPA Toxaphene Work Group reported that toxaphene is highly persistent and accumulates in the environment.

Analytical Methods. Analytical methods available for analyzing toxaphene in drinking water include the

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solvent extraction-gas chromatography technique.

Human Exposure. In FDA's compliance program report for FY 77 on pesticides and metals, toxaphene was detected in 3.3 percent of samples tested. In the FY 79 total dietary study, the FDA detected toxaphene levels ranging from 10 to 59 μg/kg in 10 percent of the garden fruits included in the dietary study for adults. Toxaphene was detected in 30 percent of the oils and fat samples for infants; concentrations ranged from 40 to 173 μg/kg. Residues of toxaphene ranging from 30 to 77 μg/kg were also reported in 10 percent of the vegetable samples and 60 percent of the oils and fat samples for toddlers.

Recent USDA activity (1982-83) failed to detect toxaphene residues in animal meat products.

Ambient air studies conducted between 1970-1978 reported maximum toxaphene levels as high as 8.7 μg/m³. However, because of substantially reduced used rates since these studies, actual levels at this time are probably considerably lower.

Toxaphene has been detected in streams and rivers and in drinking water in two States and in twenty-seven systems tested by EPA. None of the surface or ground water systems analyzed during the RWS contained detectable levels of toxaphene. Compliance data from the National Interim Primary Drinking Water Regulation did not report toxaphene in excess of the MCL.

Health Effects. Acute exposure to toxaphene results in a variety of central nervous system effects, including salivation, hyper excitablity, behavioral changes and convulsions. The kidney, liver and testes are also affected by acute exposure to toxasphene. The critical target organ in chronic or subchronic exposure is the liver. One-day assessments were calculated based upon kidney and liver pathology as the critical endpoints. Using a LOAEL of 4 mg/kg/day, an uncertainty factor of 100 and consumption of one liter (child) or two liters (adult) of water per day, 1-day numbers of 0.5 mg/l for the child and 1.75 mg/l for the adult were calculated (Lackey, R.W. 1949. Observations on the Acute and Chronic Toxicity of Toxaphene in the Dog. J. Industrial Hygiene Toxicology. 31:117-129).

Ten-day numbers were based on the significant results of the Lackey (1949) study. Minimal kidney and liver pathology were reported in dogs exposed to toxaphene at 4 mg/kg bw/day for up to 44 days. These effects were not observed at higher doses in other species. It is uncertain if these effects occur within 10 days of exposure. Occasional convulsions were also noted at this level. Using the LOAEL of 4 mg/kg/day, the ten-day health advisory is 0.08 mg/l for a 10 kg child and 0.28 mg/l for a 70 kg adult, using an uncertainty factor or 500 and 2000, respectively.

The NAS (*Drinking Water and Health*, 1977. Vol. I) has calculated an ADI of 0.00123 mg/kg/day for toxaphene. This was based upon a 2-year rat study in which 1.25 mg/kg/day was selected as the NOAEL and an uncertainty factor of 1000.

Toxaphene has been shown to be mutagenmic in the *Salmonella* minrosomal reverse mutation assay, with mutagenicity decreased by the addtion of active function oxidases. The National Cancer Institute carried out a study (NCI. 1979. Bioassay of Toxaphene for Possible Carcinogenicity; NCI Carinogenesis Technical Report Series No. 37, DHEW Publication No. (NIH) 79-832) on the carcinogenicity of toxaphene in which male and female mice and rats were fed various doses of toxaphene added to the diet as an acetone solution, 2 percent corn oil. It was concluded that under the conditions of the bioassay, toxaphene was carcinogenic in male and female mice, causing incresed incidences of hepatocellular carcinomas in a dose related manner. The results also suggested that toxaphene was carcinogenic for the thyroid of male and female rats. In a separate study, with male and female mice (NCI, 1979. DHEW 79-832), increased incidences of hepatocelluar carcinoma in male mice were observed after 18 months of toxaphene ingestion in the diet. The IARC has classified toxaphene in Group 2; inadequate evidence for carcinogencity in humans and adequate evidence for carcinogenicity in animals. Toxaphene has been classified in EPA's Group B2, according to EPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon the positive results in studies in rats and mice.

EPA's CAG have derived estimates of risk of daily exposure to toxaphene in drinking water based upon the incidence of hepatocellular carcinoma from an ingestion study in mice. Table 13 summarizes the CAG risk estimate.

EPA's MCL for toxaphene in drinking water, under the National Interim Primary Drinking Water Regulations, is 0.005 mg/l. This standard is based on the reported organoleptic effects of toxaphene at concentrations greater than 5 μg/l (Sigworth, E.A. 1965. Indentifiation and Removal of Herbicides and Pesticides. J.Am. Water Works Assoc. 57:1016).

EPA's ambient water quality criteria for human health, considering ingestion of contaminated water and aquatic organisms is suggested as concentration levels of 7.1 ng/l, 0.71 ng/l and 0.071 ng/l equivalent to calculated excess cancer risks of 10^{-5}, 10^{-6}, and 10^{-7}, respectively.

Toxaphene has been reported to have an organoleptic threshold of 0.14 mg/l and the WHO has not determined a guideline for toxaphene in drinking water.

The available data indicates that toxaphene is a potent carcinogen in animals. For this reason and because there is some occurrence in drinking water, an RMCL and a primary drinking water regulation will be proposed. The RMCL will be based upon carcinogenic effects and an RMCL of zero is proposed.

23. 2,4,5-TP

2,4,5-TP [2-(2,4,5,-Trichlorophenoxy) propionic acid or silvex; CAS #93-72-1; current MCL is 10 μg/l] is a herbicide that has been used for weed and brush control on rangeland and rights of way, pastures, commercial or ornamental turf, home weed control and weed control in and along canals and other waterways.

2,4,5,-TP is soluble in water (140 mg/l at 25 °C). The environmental persistence of 2,4,5-TP is expected to be relatively short. Phenoxy acid herbicides undergo photolysis and bacterial degradation.

In 1979, EPA issued an emergency suspension order covering the use of 2,4,5-TP for weed and brush control in forests, rights of way, pastures, irrigation canals, and other waterways, turfs and homes. The suspension was never lifted, and all registrations for herbicides containing 2,4,5-TP are now canceled. While there is still limited use of existing stocks of certain 2,4,5-TP products (non-suspended uses only), the existing stocks period has expired for most products.

Analytical Methods. Analytical methods available for analyzing 2,4,5-TP in drinking water include the derivatization-gas chromatography technique.

Human Exposure. No comprehensive data are available on the levels of 2,4,5-TP in the U.S. food supply. Data on levels of 2,4,5-TP on apples in 1975 indicate the presence of residues in unwashed fruit of 97 μg/kg initially, decreasing to 36 μg/kg after 4 months of storage.

No data are available on levels of 2,4,5-TP in air.

2,4,5-TP has been found in drinking water in three States. Of eight surface systems sampled in the NORS, one large system was found to contain 0.02 μg/l 2,4,5-TP. In the NSP, 2,4,5-TP was not detected in any of the 105 surface water

systems sampled. None of the 21 surface water systems sampled during the RWS contained 2,4,5-TP in excess of the minimum quantification limit of 0.1 μg/l.

Concentrations of 2,4,5-TP ranging from 0.03 to 0.09 μg/l were found in a USGS survey of finished drinking water collected from 15 surface water systems in Florida.

National compliance reports show that one medium-sized surface system violated the MCL for 2,4,5-TP.

Finished drinking water samples from 3 out of 127 water supplies sampled in Florida contained 2,4,5-TP in the range of 0.04 to 0.08 μg/l. A USGS survey of ground water supplies in Florida also detected 2,4,5-TP at levels ranging from 0.04μg/l to 0.30 μg/l.

2,4,5-TP has been found in hazardous waste and in waste water. The compound is mobile and is widely used.

Health Effects. 2,4,5-TP is contaminated to varying extents with 2,3,7,8-TCDD, a highly toxic polychlorinated dibenzo-p-dioxin. Substantial differences in the toxicity of 2,4,5-TP have been reported, probably based upon the degree of contamination of the compound.

Single, oral exposure to 2,4,5-TP at high doses causes a variety of physiological and biochemical effects including depression, posterior quarter muscle weakness, irritation of the stomach and minor liver and kidney damage in mammals. Subchronic exposure to 2,4,5-TP in animals has produced histopathologic changes in the liver and kidney, while chronic studies have noted adverse effects such as mild degeneration and necrosis of hepatocytes accompanied by elevation of SGOT and SGPT.

The available data are inadequate to estimate a 1-day assessment for 2,4,5-TP. Two unpublished studies by Dow Chemical (1962, 1983) that described the toxicity of 2,4,5-TP to rats and dogs were examined. No effects were reported in dogs administered 2 mg/kg/day 2,4,5-TP in the diet for 89 days. Only one dose level was employed in this study, and the available description of this unpublished work provided no details on the extensiveness of pathologic examination. In a companion study, rats administered 5 mg/kg/day of the sodium salt of PGBE ester of 2,4,5-TP for 90 days had an increase in relative weight of the liver and kidney with "histopathologic changes" in both.

Of these two studies, the study in the dog that identified a NOAEL of 2 mg/kg/day was used to calculate a 10-day assessment. Using the NOAEL of 2 mg/kg/day, an uncertainty factor of 100 based upon an animal study and consumption of 1 liter (child) of 2 liters (adult) of water per day, 10-day numbers of 0.2 mg/l and 0.7 mg/l were determined for the child and adult, respectively.

The NAS (*Drinking Water and Health*, 1977, Vol. I) has calculated an ADI for 2,4,5-TP based upon a two-year feeding study (Mullison, 1986. South Weed Conf. Proc. 19th Annual Meeting, Jacksonville, Florida, pages 420–435) in dogs in which 0.75 mg/kg/day was selected as the NOAEL. Using this NOAEL and an uncertainty factor of 1000, the NAS calculated an ADI of 0.0075 mg/kg/day.

The AADI will be determined based upon the same study used by the NAS to calculate the ADI (Mullison, 1986. South Week Conf. Proc. 19th Annual Meeting, Jacksonville, Florida, pp. 420–435). Using 0.75 mg/kg/day as the NOAEL, an uncertainty factor of 100 and consumption of 2 liters of water per day, an AADI of 0.26 mg/l has been determined.

A mutagenicity assay (Anderson, et al., 1972. J. Agric. Food Chem. 20(3):649) on 2,4,5-TP found that the compound did not cause point mutations in histidine-requiring mutants of *Salmonella typhimurium*. Limited data are available on the potential carcinogenicity of 2,4,5-TP. In one study (Innes, 1969, J. Natl. Cancer Inst. 42:1101), chronic oral exposure to 2,4,5-TP did not significantly increase the incidence of tumors at any site in mice exposed for 80–81 weeks, while another study (Gehring and Besto, 1978. Ecol. Bull. Stockholm. 27:122) also showed no increase in tumor incidence in rats or dogs exposed to 2,4,5-TP for 2 years. However, these studies did not employ the recent NCI bioassay procedure recommending the use of the maximum tolerated dose and half of the maximum tolerated dose and thus it is difficult to reach a conclusion on the carcinogenicity of the compound. 2,4,5-TP has been classified in EPA's Group D, according to EPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon inadequate data from animal studies.

EPA's MCL for 2,4,5-TP in drinking water, under the National Interim Primary Drinking Water Regulations, is 0.01 mg/l. This standard is based upon a NOAEL of 0.9 mg/kg/day, with an uncertainty factor of 500 and the assumption that 20 percent of the total intake is via drinking water.

Exposure to 2,4,5,-TP at high dose levels results in a variety of chronic adverse health effects. Because this contaminant also has been detected in several drinking water systems, an RMCL and a primary regulation will be proposed. An RMCL of 0.052 mg/l is proposed based upon the AADI (0.26 mg/l) with 20 percent drinking water contribution.

24. Xylene

For the purposes of this proposal, xylene is considered to be the mixture (CAS # 1330-20-7) of three isomers (ortho-, CAS # 95-47-6; meta-, CAS # 108-38-3; and para-, CAS # 2169-42-3). Xylene isomers are slightly soluble in water and are soluble in alcohol, ether and many other organic liquids.

Xylene is used in aviation and automobile gasoline, protective coatings, in the synthesis of many organic chemicals, pharmaceuticals and vitamins, and as solvents for many preparations, including pesticides.

Analytical Methods. Analytical methods available for analyzing xylene in drinking water include the purge and trap-gas chromatography technique.

Human Exposure. No information was available on the human intake of xylene from food. The median level of o-xylene in ambient air reportedly ranges from 0.4 $\mu g/m^3$ in rural and remote areas to 3 $\mu g/m^3$ in source dominated areas. Median levels of m-and p-xylene (combined) range from 0.4 $\mu g/m^3$ to 73 $\mu g/m^3$ in source dominated areas. Based on these data, estimated maximum daily intake for adults in rural and source dominated areas, respectively, would approach 8 μg/day and 1.7 μg/day for all isomers (combined).

The results of the GWSS indicated that approximately 3 percent of the ground water systems contained detectable levels of xylene while the CWSS indicated that xylene was in 6 percent of the surface water supplies. The maximum detected level of contamination reported in federal and state surveys was 750 μg/l in ground water and 5.2 μg/l in surface water supplies.

Health Effects. The principal toxic effects of xylene are central nervous system disturbances, such as changes in numerative ability reaction time, short-term memory and electroencephalographic patterns. Xylene also affects the liver at very high concentrations. A 1-day number was calculated for xylene based upon an inhalation study in human volunteers (Gamberale, et al. 1978. Exposure to Xylene and Ethylbenzene. III. Effects on Central Nervous Functions. Scan. J. Work Environ. Health. 4:204). In this study, a NOAEL was determined at an inhalation concentration of 1300 mg/m^3 as for approximately one hour. Using 1300 mg/m^3 the NOAEL for a 1-day exposure, an uncertainty factor of 10 and consumption of 1 liter (child) or 2 liters of water (adult) per day, 1-day

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numbers of 12 mg/l for a 10 kg child and 42 mg/l for a 70 kg adult were calculated.

Insufficient ingestion toxicological data are available to calculate 10-day numbers. However, 10-day numbers were calculated based upon a study (Carpenter, et al. 1975. Petroleum Hydrocarbon Toxicity Studies. V. Animal and Human Response to Vapors of Mixed Xylenes. Toxicol. Appl. Pharmacol. 33:543) in rats exposed by inhalation to mixed xylene at 2000 mg/m^3 for 6 hours/day (5 days/week) for up to 13 weeks. No significant effects on blood chemistry or tissue histology were reported as a result of the xylene exposure. Using 200 mg/m^3 as a NOAEL, an uncertainty factor of 100, and consumption of 1 liter (child) or 2 liters (adult) of water per day, 10-day numbers of 7.8 mg/l for a 10 kg child and 27 mg/l for a 70 kg adult were calculated.

A provisional AADI for xylene is based on an inhalation study (Jenkins, et al. 1970. Long-term Inhalation Studies on Benzene Toluene, o-Xylene and Cumene on Experimental Animals. Toxicol. Appl. Pharmacol. 16:818) in rats, guinea pigs, monkeys and dogs. In this study, animals were exposed at a dose level of 377 mg/m^3 continuously for 90 days. No statistically significant effects were observed with respect to body weight, hematology and histopathological examination of treated animals. Using 337 mg/m^3 as the NOAEL, an uncertainty factor of 1000 based upon an animal study with few animals per dose level, and consumption of 2 liters of water per day, a provisional AADI of 2.2 mg/l was calculated.

The estimated concentration for detection by taste and odor in surface water is 0.3 to 1.0 mg/l (Middleton, et al. 1958. J. Am. Water Works Assoc. 50:21).

Little data are available on the carcinogenicity of xylene. One study examined the dermal effects of xylene and concluded that xylene was not a skin tumorigen. A long-term carcinogenicity bioassay is presently being conducted by the National Toxicology Program. Mice and rats have been treated, but data from this study are not yet available. Xylene has been classified in EPA's Group D, according to EPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon inadequate data from animal studies.

Exposure to xylene at high dose levels has been shown to result in chronic toxic effects. For this reason and because there has been significant occurrence of this contaminant in water, an RMCL and a primary regulation will be proposed. An RMCL of 0.44 mg/l is proposed, based upon an AADI of 2.2 mg/l assuming 20 percent drinking water contribution.

C. RMCLS Not Proposed

1. Atrazine

Atrazine (6-chloro-N-ethyl-N'-(1-methylethyl)-1,3,5-triazine-2,4-diamine; CAS # 1912-24-9) is a herbicide and a plant-growth regulator. It is slightly soluble in water under normal conditions (33 mg/l at 27 °C). Its vapor pressure is low (1.4×10^{-6} mm Hg at 30 °C). Domestic use of atrazine is estimated to range between 100 and 500 million pounds annually. About 98 percent of the domestically supplied atrazine is used on corn and soybeans.

Analytical Methods. Analytical methods available for analyzing atrazine in drinking water include the solvent extraction-gas chromatography technique.

Human Exposure. Very little data are available on atrazine levels in food. The 1971 and 1972 National Soils Monitoring Program reported that 1 percent of grain and vegetable samples from areas where atrazine is used contained atrazine in excess of the minimum detection level of 10 μg/kg. Atrazine residues are permitted on certain crops. If all foods contained atrazine residues up to the tolerance level, an adult might have a daily atrazine residue intake of up to 77 μg/day.

No information is available on the level of atrazine in ambient air.

One large surface water supply system sampled in the NORS contained 0.1 μg/l atrazine in its finished drinking water. During the NSP, 29 percent of the surface water systems contained atrazine in excess of the quantification limit of 0.1 μg/l. The range of positive values was 0.1–2.9 μg/l.

Atrazine was detected in six samples from drinking water supplies drawn from Northwestern Ohio rivers. Atrazine levels ranged from 0.087–15.9 μg/l; the average reported value was 6.76 μg/l.

Atrazine concentrations were detected during a study comparing the effectiveness of various types of water treatment for the removal of herbicides. Average concentrations of atrazine for the three supplies were 0.90, 2.1 and 3.31 μg/l, respectively. Peak concentrations were 1.22, 5.2 and 7.84 μg/l, respectively. Atrazine was found in a major water supply on the Mississippi River at 4.7–5.1 μg/l.

Two ground water systems analyzed in the NORS contained traces of atrazine. Ground water supplies in three midwestern States tested positive for atrazine, with concentrations typically in the range of 0.8 μg/l.

Health Effects. Insufficient toxicological data are available on the short-term effects of atrazine to calculate 1-day or 10-day assessments.

Atrazine appears to have low chronic toxicity in animals. In a 2-year chronic feeding study at 100 mg/l in the diet of rats, no gross or microscopic signs of toxicity were observed. The NAS (*Drinking Water and Health*, 1977, Vol. I) calculated an AADI for atrazine based upon an 80 week study (Innes, J.R.M., et al. 1969. Bioassay of Pesticides and Industrial Chemicals for Tumorigenicity in Mice. A preliminary note. J. Nat. Cancer Inst. 42: 1101–1114) in mice in which a dose of 21.5 mg/kg/day was shown to result in an incidence of hepatomas of 4.24 percent in controls and 5.8 percent in atrazine treated animals. An ADI of 0.0215 mg/kg/day was determined based upon 21.5 mg/kg/day as a LOAEL and an uncertainty factor of 1,000 based on animal study. Based upon the NAS ADI, a provisional AADI of 0.75 mg/l was calculated, assuming consumption of 2 liters of water per day.

Atrazine has not been shown to be mutagenic in standard assays with microorganisms and studies on the carcinogenicity of the compound have shown inconclusive results. The IARC has not evaluated the carcinogenicity of atrazine. Atrazine has been classified in EPA's Group D, according to EPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon inadequate evidence from animals studies.

Only the Innes study is available on the health effects of atrazine and this study is inadequate to serve as the basis for the RMCL. Other studies submitted to EPA's Office of Pesticides Programs have been seriously questioned because of unscientific laboratory techniques. Due to the paucity of the toxicological data, an RMCL and primary regulation are not being proposed for atrazine. Atrazine will be reexamined for regulation when the toxicology data base is expanded.

2. 2,3,7,8-Tetrachlorodibenzo-p-dioxin

2,3,7,8-Tetrachlorodibenzo-p-Dioxin (2,3,7,8-TCDD; CAS # 826-00-2) is not manufactured purposefully; it is formed as a contaminant or impurity during chemical production or chemical pyrolysis. 2,4,5-Trichlorophenol (2,4,5-TCP), a chemical formed from 1,2,4,5-tetrachlorobenzene, is contaminated with 2,3,7,8-TCDD. 2,4,5-TCP is, in turn, used in the production of several herbidices, including 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) esters, and Silvex. Therefore, 2,3,7,8-

TCDD may be a contaminant of these herbicides. Additionally, TCDD may be formed as an impurity during the pyrolysis of chlorinated phenols, chlorinated benzenes, and polychlorinated diphenyl ethers.

Analytical Methods. Analytical methods available for analyzing 2,3,7,8-TCDD in drinking water include the solvent extraction-gas chromatography mass spectrometry technique.

Human Exposure. In theory, 2,3,7,8-TCDD could occur in food as a result of contamination of plant crops by herbicides such as Silvex or 2,4,5-T, consumption by livestock of 2,3,7,8-TCDD-contaminated forage or concentration of residues through the food chain. However, data on actual occurrence in food are rare. TCDD has been reported at levels of 4–70 ppt in the fat of cattle that had grazed on land treated with 2,4,5-T. 2,3,7,8-TCDD has also been detected in several species of commercial and non-commercial fish in several rivers and lakes in the United States. Levels of TCDD reported in fish and shellfish range from 1–700 ppt. The estimated maximum daily intake of 2,3,7,8-TCDD for individuals who regularly consume contaminated fish from the Great Lakes region might range from 0.39–8.4 ng/day.

Data on ambient air levels of 2,3,7,8-TCDD are limited. TCDD has been found in ambient air under special conditions. Air levels, ranging from 0.09 ng/m^3 to 0.07 ng/m^3, have been reported following agricultural application of Silvex. In addition, 2,3,7,8-TCDD was detected at an average level of 1,100 ppt in air at a disposal site near Jacksonville, Arkansas. Atmospheric emissions from municipal incinerators and from certain fires have been reported; however, data on ground level concentrations of 2,3,7,8-TCDD were not available.

Dioxin has not been detected in drinking water, as the physical/chemical characteristics of the compound suggest that it is relatively immobile and thus would not be expected to be found in drinking water. Data are not available showing occurrence in surface or ground waters.

Dioxin has been identified at more than 32 hazardous waste sites designated in complaints and consent decrees under the Comprehensive Emergency Response Compensation and Liability Act of 1980 (CERCLA) and the National Contingency Plan (40 CFR Part 300). The concentration of dioxin in the soil at one site was reported at 0.5 mg/kg and at levels of 20 mg/l in non-aqueous phase liquids in the dump.

In December 1983, EPA announced a National Dioxin Strategy to determine the extent of dioxin (primarily 2,3,7,8-TCDD) contamination throughout the country. The strategy provides a systematic framework under which the Agency will (1) study the nature of dioxin contamination throughout the U.S. and the risks to people and the environment, (2) clean-up dioxin-contaminated sites that threaten public health, (3) find ways to prevent future contamination, and (4) find ways to destroy or dispose of existing dioxins.

Health Effects. 2,3,7,8-TCDD is readily absorbed by mammals following either oral or dermal exposure and is rapidly distributed to tissues with a high lipid content. The liver represents a major site of accumulation in many species. Metabolism of 2,3,7,8-TCDD occurs slowly, with the polar metabolites excreted in the urine.

Characteristic non-carcinogenic effects resulting from exposure to 2,3,7,8-TCDD include thymic atrophy and weight loss. In certain species, liver damage is a major pathological effect.

One-day assessments were calculated based on a study in rats (Turner, J.N. and D.N. Collins. 1983. Liver Morphology in Guinea Pigs Administered either Pyrolysis Products of a Polychlorinated Biphenyl Transformer Fluid or 2,3,7,8-tetrachloro-dibenzo-p-dioxins. Toxicol. Appl. Pharmacol. 67:417–429) where a single day oral dose of 2,3,7,8-TCDD induced liver changes in female guinea pigs. Using a LOAEL of 0.1 μg/kg, an uncertainty factor of 1,000 and consumption of liter (child) or 2 liters (adult) of water per day, 1-day numbers of 1.0×10^{-3} μg/l for a 10 kg child and 3.5×10^{-3} μg/l for a 70 kg adult were calculated.

Ten-day assessments were calculated by dividing the one-day assessments by ten. Consequently, 10-day numbers of 1.0×10^{-4} μg/l for a 10 kg child and 3.5×10^{-4} μg/l for a 70 kg adult were calculated.

A provisional AADI has been calculated that is consistent with that developed by the Agency as indicated by the EPA, Ambient Water Quality Criteria for 2,3,7,8-Tetrachlorodibenzo-p-dioxin (U.S. EPA, 1984. Ambient Water Quality Criteria for 2,3,7,8-Tetrachlorodiobenzo-p-dioxin. EPA 440/5–80–007) where it concluded that the 0.001 ug/kg dose in a three-generation reproduction study in rats by Murray, et al. (Murray, F.J., F.A. Smith, K.B. Nitockle, C.G. Huniston, R.J. Kociba, and B.A. Schwetz. 1979. Three-generation Reproduction Study of Rats Given 2,3,7,8-Terachloro-p-dioxin (TCDD) in the diet. Toxicol. Appl. Pharmacol. 50:24–251), in conjunction with reproductive effects noted at 0.0015 ug/kg in a limited study with monkeys by Schantz, et al. (Schantz, S.L., D.A. Barsotti and J.R. Allen, 1979. Toxicological Effects Produced in Non-human Primates Chronically Exposed to Fifty Parts per Trillion 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD). Toxicol. Appl. Pharmacol. 48:A160), represents a LOAEL for adverse reproductive effects. A LOAEL of 0.001 ug/kg was selected, with an uncertainty factor of 1,000 based upon an animal study and consumption of 2 liters of water per day, resulting in an AADI of 3.5×10^{-6} mg/l.

The NAS (*Drinking Water and Health*, 1977. Vol. I) calculated an ADI of 0.0001 ug/kg day based on a 13 week oral treatment study in rats (Kociba, R.J., P.A. Keeler, C.N. Park, and P.J. Gehring. 1976. 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD): Results of a 13-week Study in Rats. Toxicol. Appl. Pharmacol. 35:553–574) using a NOAEL of 0.01 ug/kg/day, an uncertainty factor of 100 and consumption of 2 liters of water per day.

Mutagenicity tests have shown conflicting results with inconclusive evidence as to the mutagenicity of 2,3,7,8-TCDD. Animal studies have demonstrated the compound to be a potent animal carcinogen. Oral administration of 2,3,7,8-TCDD, either in the diet or by gavage, results in the production of hepatocellular carcinomas in female rats and both sexes of mice. In the National Toxicology Program bioassay (NTP, 1980. Bioassay of 2,3,7,8-Tetrachlorodibenzo-p-dioxin for Possible Carcinogenicity (Gavage Study). Carcinogenesis Testing Program. NCI, NIH, Bethesda, MD and NTP, Research Triangle Park, NC. Pub. No. 82–1765), rats and mice were dosed twice weekly by gavage with 2,3,7,8-TCDD in a corn oil-acetone solution. In male rats, a dose-dependent increase in the incidence of follicular-cell adenomas or carcinomas of the thyroid was observed. In female rats, observed increases in the incidence of subcutaneous tissue fibrosarcomas, adrenal cortical adenomas and hepatocellular carcinomas were observed only in the high dose group. Other studies have reported squamous cell carcinomas in both sexes of rats and follicular-cell adenomas of the thyroid in both male and female mice.

A number of reports and epidemiological studies have attempted to relate 2,3,7,8-TCDD exposure to human health effects. 2,3,7,8-TCDD has been implicated as the caustive agent for chloracne, hyperpigmentation, altered liver function and porphyria cutanea tarda in humans. In addition, questions have been raised regarding a possible relationship between 2,3,7,8-

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TCDD exposure and cancer. The available studies do not establish a definite relationship between 2,3,7,8-TCDD and the development of tumors in humans, although an association has been suggested with soft-tissue sarcomas, lymphomas, and stomach cancer. The IARC have classified 2,3,7,8-TCDD in Group 2B; inadequate evidence for carcinogenicity in humans, sufficient evidence for carcinogenicity in animals and inadequate evidence for activity in short-term tests. 2,3,7,8-TCDD has been classified in EPA's Group B2, according to EPA's Proposed Guidelines for Carcinogens Risk Assessment, based upon positive results in studies in rats and mice.

EPA's CAG has derived estimates of risk from exposure to 2,3,7,8-TCDD in drinking water based upon a carcinogenicity study consisting of lifetime feeding of 2,3,7,8-TCDD in female rats (Kociba, R.J., D.G. Keyes, J.E. Berger, et al. 1977. Results of a Two-year Chronic Toxicity and Oncogenicity Study of 2,3,7,8-Tetrachlorodibenzo-p-dioxin in Rats. Toxicol. Appl. Pharmacol. 46:279–303). A q^*_1 of 1.56×10^5 (mg/kg/day)$^{-1}$ was calculated by the multi-stage model for this risk assessment. This is the geometric mean of $2q^*_1$ calculations based on the review of tissue slides by two independent pathologists and corrected for early mortality. The OHEA risk estimate is shown in Table 13.

CAG (U.S. EPA, Health Assessment Document for Polychlorinated Dibenzo-p-dioxin, May 1984. EPA-600/8-84-014A) has also derived risk estimates for 2,3,7,8-TCDD based on the same study using other models. The comparison for a risk estimate from exposure to a 2,3,7,8-TCDD level of 10^{-8} ug/kg/day is given below:

Dose (µg/kg/day)	95 percent upper confidence limit of additional risks		
	Multi-stage model/one-hit model [1]	Weibull	Log-probit
10^{-11}	1.5×10^{-9}	9.7×10^{-9}	7.7×10^{-10}
10^{-12}	1.5×10^{-10}	1.3×10^{-9}	4.4×10^{-11}

[1] Both models gave identical results.
[2] Based upon Kociba analysis of pathology, corrected for early mortality.
[3] Based upon Squire analysis of pathology, corrected for early mortality.

The EPA water quality criterion for 2,3,7,8-TCDD for carcinogenic endpoints (U.S. EPA. 1984. Ambient Quality Criteria for 2,3,7,8-Tetrachlorodibenzo-p-dioxin. EPA 440/5-80-007) is 1.3×10^{-9} µg/l based on an estimated human lifetime cancer risk of 1×10^{-6} and assuming daily consumption of 2 liters of water and 6.5 g of fish and shellfish. The study and statistical model used for this risk assessment were the same as those used for the risk assessment for 2,3,7,8-TCDD previously described. The FDA has issued a Health Advisory (FDA, 1983. Statement by S.A. Miller, Director, Bureau of Foods, FDA, before the Subcommittee on Natural Resources, Agriculture Research and Environment, U.S. House of Representatives, June 30) in which fish containing >50 ppt 2,3,7,8-TCDD should not be consumed and fish containing <25 ppt 2,3,7,8-TCDD do not pose a serious health concern. A tolerance of 0.05 ppm for hexachlorophene methylenebis (2,3,6-trichlorophenol) in or on feedstock cottonseed has been set with the condition that the technical grade material contain no more than 0.1 ppm. 2,3,7,8-TCDD (40 CFR 180.302).

2,3,7,8-TCDD has not been detected in drinking water supplies. The compound is not mobile in runoff or soils and has not been found in ground water or surface water that is a potential source of drinking water. Due to the limited occurrence and potential for occurrence in drinking water, an RMCL and primary regulation is not being proposed for 2,3,7,8-TCDD.

Question for Comment:

1. Are there any data to suggest that 2,3,7,8-TCDD is known or likely to be found in drinking water supplies?

3. Endrin

Endrin (1,2,3,4,10,10-hexachloro-6,7-epoxy-1,4,4a, 5,6,7,8,8a-octa-hydro-1,4-endo,endo-5,8-dimethanoaphthalene; CAS # 72-20-8; current MCL is 0.2 µg/l) is a commercially used insecticide and rodenticide. The solubility of endrin in water is 0.25 mg/l. Endrin is persistent and is concentrated through the aquatic food chains.

Endrin was only widely used in the U.S. The EPA issued a notice of rebuttable presumption against registration and continued registration (RPAR) of endrin-containing products in 1976. It included three risk presumptions—risk of significant population reductions of non-target organisms, acute toxicity to wildlife, and teratogenicity. After review, the Agency determined that the offsetting economic, social or environmental benefits were not great enough, and endrin was cancelled for a number of uses and registration for new uses of endrin were denied. Endrin is presently registered only for the control of cutworms, grasshoppers and moles; however, the sole Endrin manufacturer has ceased production for use in the U.S. Environmental concentrations of endrin appear to be decreasing due to these use restrictions.

Analytical Methods. Analytical methods available for analyzing endrin in drinking water include the solvent extraction-gas chromatography technique.

Human Exposure. In FDA's compliance program report for FY 77 on pesticides and metals, endrin was detected in 2.1 percent of the samples tested. In the FDA FY 79 total dietary study for adults and infants, no endrin was detected in any of the foods sampled. One residue was detected in an oils and fats sample at a level of 1 µg/l for the toddler diet. According to the USDA, in 1982–1983, 0.3 percent of the fat tissue from various animal species intended for human consumption contained endrin levels ranging from 0.01–0.10 µg/kg.

Tolerances for residues of endrin as set by EPA's Office of Pesticide Programs, in or beets, broccoli, brussels sprouts, cabbage, cauliflower, cottonseed, cucumbers, eggplant, pepper, potatoes, squash and tomatoes are 10 ppm.

Ambient air studies between 1970–1975 reported endrin levels as high as 39.3 ng/m³.

Endrin is rarely detected in drinking water. The compound has been detected in three surface water supplied drinking water systems in one State. None of the surface or ground water systems analyzed during the NSP or the RWS contained detectable levels of endrin. National compliance reports with the NIPDWR show that no system sampled reported endrin in excess of the MCL of 0.2 mg/l.

Health Effects. At high dose levels, endrin has been shown to accumulate in liver, brain, kidneys, and fat, while at lower dose levels endrin is quickly metabolized and eliminated from the body. The major toxicant in mammals is considered to be the metabolite 12-ketoendrin.

One-day assessment were calculated for endrin based upon a study in squirrel monkeys (Revzin, 1966. Effects of Chronic Endrin Administration on Brain Electrical Activity in the Squirrel Monkey. Fed. Proc. 27:697) where 0.2 mg/kg endrin was administered daily for 7 days and alterations in the EEG were recorded. No effects were noted at this dose for shorter term exposure. Using 0.2 mg/kg/day as the NOAEL for 1-day exposure, an uncertainty factor of 100 and consumption of 1 liter (child) or 2 liters of water (adult) of water per day, 1-day numbers of 0.02 mg/l for a 10 kg child and 0.07 mg/l for a 70 kg adult were calculated.

Ten-day assessment were calculated based upon a study (Nelson, et al. 1966. Serum Alkaline Phosphatase Levels, Weight Changes, and Mortality Rates of

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Rats Fed Endrin. J. Agric. Food & Chem. 4:966) in rats exposed for 1 or 2 weeks to 5 ppm (0.05 mg/kg/day) endrin in the diet. The body weight of the exposed animals was decreased relative to controls. Using 0.05 mg/kg/day as a NOAEL, an uncertainty factor of 100, and consumption of 1 liter (child) or 2 liters of water per day (adult), 10-day numbers of 0.005 mg/l for a 10 kg child and 0.018 mg/l for a 70 kg adult were calculated.

A provisional AADI for endrin was derived based upon a feeding study (Treon, et al. 1955. Toxicity of Endrin for Laboratory Animals. J. Agric. Food & Chem. 3:842) in which dogs were exposed for 18.7 months to 1, 3, or 4 ppm endrin in the diet. Based on measured food intake, the daily dose varied from 0.045 to 0.12 mg/kg bw for the 1 ppm group, 0.12 to 0.25 mg/kg bw for the 3 ppm group and 0.15 to 0.21 mg/kg bw for the 4 ppm group. Increases in heart and kidney weights were noted at 3 and 4 ppm but not at 1 ppm. Using 0.045 mg/kg bw (1 ppm) as the NOAEL, an uncertainty factor of 1000 based upon an animal study with few animals per dose level, and consumption of 2 liters of water per day, a provisional AADI of 0.002 mg/l was calculated.

Endrin was not shown to be mutagenic in microbial systems with or without microsomal activation. The potential carcinogenic effects of endrin were evaluated in several animal studies. The results were negative in four studies, including the National Cancer bioassay (NCI. 1979. Bioassay of Endrin for Possible Carcinogenesis. Tech. Rep. Ser. 12, NCR-CG-TR-12). The only study (Deichmann, et al. 1970. Tumorigenicity of Aldrin, Dieldrin and Endrin in the Albino Rat. Ind. Med. 39:428) reporting positive results was a rat study in which endrin was administered at concentrations of 0, 0.1, 5, 20 or 25 ppm. The total number of malignant tumors was increased in all exposed groups. No more than 2 tumors/group, however, were reported for one site. Moreover, no dose response was apparent with the greatest number of tumors occurring in the 0.1 ppm group. The NAS (*Drinking Water and Health*, 1977, Vol. I) have stated that there are insufficient data to permit a statistical extrapolation of cancer risk for endrin and that endrin is a suspect animal carcinogen. The IARC have not addressed the carcinogenicity of endrin. Endrin has been classified in EPA's Group E, according to EPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon the negative results from four studies including the NCI bioassay.

EPA's ambient water criterion for endrin (U.S. EPA. 1980. Ambient Water Quality Criteria for Endrin. EPA/440/5-80-047) for human health is 0.001 mg/l. This was based upon the same study which was used to derive the provisional AADI and a value of 0.001 mg/l was recommended because it was the maximum allowable concentration proposed by the Public Health Service for drinking water. The WHO (1973) established as a guideline a maximum intake of 2 μg/kg/day, or 138.2 μg/day, for a 69.1 kg person. The WHO has not established a drinking water guideline for endrin.

The MCL, under the National Interim Primary Drinking Water Regulations, is 0.002 mg/l. The provisional AADI is slightly higher than the interim MCL due to the fact that the provisional AADI is calculated based on the measured food intake of a dog, while the MCL was calculated based upon the assumed food consumption of a dog, both based upon the same study (Treon, et al. 1955. Toxicity of Endrin for Laboratory Animals. J. Agric. Food & Chem. 3:842).

Endrin is rarely detected in drinking water and has been cancelled for new uses. The compound is not considered to be very mobile and thus EPA is not proposing an RMCL for endrin.

Question for Comment:

1. Is there sufficient occurrence and potential for occurrence in drinking water to propose an RMCL foe endrin?

4. Hexachlorobenzene

Hexachlorobenzene (CAS # 118-74-1) has a vapor pressure of 1.09×10^{-5} mm Hg (20 °C). It has a very low aqueous solubility (6 ug/l at 25 °C). Hexachlorobenzene is no longer produced in the United States. However, approximately 2 to 5 metric tons of hexachlorobenzene are generated annually as a waste byproduct of chlorinated solvent and pesticide production. The primary use of hexachlorobenzene in 1972 was as a fungicide. The majority of these formulations are no longer produced. Other industrial uses have included dye manufacturing, an intermediate in organic synthesis, porosity controller in the manufacture of electrodes, a wood preservative and an additive in pyrotechnic compositions for the military. Since hexachlorobenzene is no longer produced in the United States, commercial uses of hexachlorobenzene have virtually ceased.

Analytical Methods. Analytical methods available for analyzing hexachlorobenzene in drinking water include the solvent extraction-gas chromatography technique.

Human Exposure. Hexachlorobenzene has been a contaminant of concern because of its occurrence in human tissue and the milk from nursing mothers. The compound has been found in adipose tissue and milk of cattle raised in the vicinity of industrialized regions in Louisiana, and in adipose tissue of sheep in western Texas and California. Hexachlorobenzene has also been found in fish and birds.

Hexachlorobenzene has been found in ambient air around production sand waste disposal sites.

Limited information is available on levels of hexachlorobenzene in finished drinking water supplies. Hexachlorobenzene was detected in a Regional survey in two finished water supplies at levels of 4 and 6 ng/l. In the NSP, the compound was not detected.

Health Effect. Hexachlorobenzene is readily absorbed and distributed to tissues that have high lipid content. The adipose tissue accumulates the greatest concentrations of hexachlorobenzene, although bone marrow and skin also accumulate the compound. Hexachlorobenzene is metabolized slowly into other chlorinated benzenes, chlorinated phenols and other minor metabolities. The excretion of hexachlorobenzene is characterized by an initial rapid phase followed by a very slow phase.

Chronic toxicity studies in animals have shown a significant increase in liver and kidney weights in hexachlorobenzene-treated animals, as well as hepatic and renal lesions. Increased prophyrin-levels in the liver and the urine have been reported in several species.

Porphyria cutanea tarda (PCT) has been associated with exposure of humans to hexachlorobenzene. An epidemic of hexachlorobenzene-induced PCT occurred in Turkey, from exposure during 1955 to 1959 to contaminated seed wheat used for food. It has been estimated that 0.05 to 0.2 g of hexachlorobenzene was consumed per day. PCT is a disease of disturbed porphyrin metabolism manifested by cutaneous lesions and hyperpigmentation. Follow-up studies conducted 20 to 25 years after the onset of porphyria showed that a few patients still had active porphyria, whereas greater than 50 percent exhibited hyperpigmentation, scarring and other signs of hexachlorobenzene toxicity. Hexachlorobenzene residues were also found in the blood, fat or breast milk of some patients.

One-day and 10-day assessments were calculated based on a study (Kuiper-Goodman, et al. 1977. Subacute

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Toxicity of Hexachlorobenzene in the Rat. Toxicol. Appl. Pharmacol. 40:529–549) where porphyrin, liver lesions and other toxic effects were found in rats fed hexachlorobenzene for 15 weeks and held to 48 weeks. Using a NOAEL of 0.5 mg/kg, an uncertainty factor of 100 and consumption of 1 liter (child) or 2 liters (adult) of water per day, 1-day and 10-day assessments of 0.050 mg/l for a 10 kg child and 0.175 mg/l for a 70 kg adult were calculated.

An AADI was calculated for hexachlorobenzene based upon a 130 week feeding study in rats (Arnold, et al. 1983. Long-term Toxicology of Hexachlorobenzene in the Rat. In preparation). A NOAEL of 1.6 ppm (0.094 mg/kg/day) was selected based upon liver and kidney lesions and increased mortality at higher doses. An uncertainty factor of 100 was applied and consumption of 2 liters of water per day, resulting in an AADI of 0.029 mg/l.

Hexachlorobenzene has not been shown to be mutagenic in the *Salmonella* histidine reversion assay, but was reported mutagenic in a yeast *S. cerevisiae* assay. Hexachlorobenzene has been shown to be carcinogenic in animal studies, showing an increased incidence of malignant tumors of the liver in hamsters and rats, as well as inducing hepatomas in mice, rats and hamsters.

The IARC has classified hexachlorobenzene in Group 2B; inadequate evidence for carcinogenicity in humans and sufficient evidence for carcinogenicity in animals. Hexachlorobenzene has been classified in EPA's Group B2, according to EPA's Proposed Guidelines for Carcinogen Risk Assessment, based upon positive results in studies in rats, mice and hamsters.

The NAS (*Drinking Water and Health*, 1983. Vol. V) calculated upper 95 percent limits of cancer risk based on carcinogenicity data in a study with male and female mice. In the external review draft of the EPA Health Assessment Document for Chlorinated Benzenes (April 1984, EPA-600/8-84-015A), the CAG has calculated risk estimates based on hepatocellular carcinomas in a lifetime dietary feeding study with female rats using them 95 percent upper limit of the multi-stage model. The study used by CAG is more recent and the CAG number gives a more conservative risk estimate. The NAS and CAG risk estimates are shown in Table 13.

Point estimates 95 percent upper limit estimates based on several models were also calculated by the CAG. Using a lifetime exposure to 0.0 mg hexachlorobenzene/kg body weight/day, these estimates are as follows:

Assumption of human equivalent dose	Models	Risk at 0.01 mg/kg/day
Body weight basis	Multi-stage	2.7×10^{-4} (a) (2.2×10^{-4}) (b)
	Probit	3.6×10^{-4} (1.2×10^{-4})
	Weibull	1.2×10^{-4} (2.5×10^{-4})
	One-hit	2.7×10^{-4} (2.2×10^{-4})
Surface area basis	Multi-stage	1.7×10^{-4} (1.4×10^{-4})
	Probit	5.2×10^{-4} (4.1×10^{-4})
	Weibull	5.0×10^{-4} (1.3×10^{-4})
	One-hit	1.7×10^{-4} (1.4×10^{-4})

(a) 95% upper limit.
(b) Point estimate

The WHO guideline for hexachlorobenzene (1984) is 0.01 μg/l based upon a risk of one additional case of cancer per 100,000 population, assuming a daily consumption of 2 liters of drinking water.

Hexachlorobenzene has rarely been detected in drinking water and the compound is not considered to mobile. For these reasons, EPA is not proposing a primary regulation for the compound.

Question for Comment:

1. Is there sufficient occurrence and potential for occurrence in drinking water to propose an RMCL for hexachlorobenzene?

5. Simazine

Simazine (6-chloro-N,N'-diethyl-1,3,5-triazine-2,4-diamine; CAS # 122-34-9) is a herbicide applied to field crops and on non-agricultural sites. Irrigation or rainfall moves the chemical into the root zone of weeds where it is active. It may also be used as an algacide or to control submerged weeds. Because simazine has a low vapor pressure, there is little tendency for simazine to enter air directly during and following application, spillage or disposal. In the U.S., approximately 61 percent of simazine is applied in commercial agricultural operations; 39 percent is applied to non-agricultural sites (aquatic applications included).

Analytical Method. Analytical methods available for analyzing simazine in drinking water include the solvent extraction-gas chromatography technique.

Human Exposure. There are few data on the levels of simazine in the U.S. food supply. Six domestic food samples were analyzed for simazine in the FDA FY 77 pesticides and metals program. None of these samples contained simazine levels in excess of the quantitation limits (between 10 and 100 μg/kg).

No data were found on the levels of simazine in air.

Simazine has been reported in drinking water from surface supplies. In the NSP, conducted from June 1977-March 1981, 12 percent of finished drinking water samples collected from surface water systems contained simazine; levels ranged from 0.1–4.4 μg/l.

Levels of simazine ranging from 0.026–0.883 μg/l were detected in five drinking water samples collected from a surface water supply in one State during peak periods of pesticide usage and maximum pesticide export.

Simazine concentrations reported for drinking water samples collected from three treatment plants in another State in 1983 ranged from 0.077–0.30 μg/l; peak concentrations ranged from 0.13–0.63 μg/l for the three reports.

Simazine has also been found in ground water. One of the 12 ground water systems sampled in the NSP contained 1 μg/l. Six out of 166 wells in California were found to be contaminated at levels between 0.5–3.5 μg/l.

Health Effects. There is a lack of data on the health effects of simazine in animals and humans. In one study (U.S. EPA, 1976. Draft Report, Initial Scientific and Microeconomic Review, Simazine. Office of Pesticide Programs, EPA Contract No. 68-01-1904), simazine fed to rats for 2 years at 1.0, 10 and 100 mg/l did not produce any difference between treated and control animals in gross appearance or behavior.

The NAS (*Drinking Water and Health*, 1977. Vol. I) calculated an ADI for simazine based upon an 80 week study (EPA, No. 68-01-1904) in mice in which a dose of 215 mg/kg/day was shown to result in an incidence of hepatomas of 4.2 percent in controls and 5.6 percent in simazine treated animals. An ADI of 0.215 mg/kg/day was determined based upon 215 mg/kg/day as a LOAEL and an uncertainty factor of 1,000 based upon an animal study.

Simazine was negative in mutagenicity studies with four strains of *Salmonella typhimurium* and the results of carcinogenicity studies on the compound have been inconclusive. The IARC has not evaluated the carcinogenicity of simazine. Simazine has been classified in EPA's Group D, according to EPA's Proposed guidelines for Carcinogen Risk Assessment, based upon inadequate data from animal studies.

Due to inadequate toxicology data, an RMCL and primary regulation will not be proposed for simazine. The study used by the NAS to calculate an ADI

has since been found to be invalid and no new data are available to determine an AADI.

6. Other SOCs

Adipates, dalapon, dibromomethane, dinoseb, diquat, endothall, glyphosate, hexachlorocyclopentadiene, PAHs, phthalates, picloram, 1,1,2-trichloroethane and vydate were included in the list of SOCs under consideration for Revised Regulations in the ANPRM. Data collection efforts on occurrence/human exposure and potential health effects have not yet been completed on these substances and these SOCs will be considered in later phases of the Revised Regulations.

IX. Impact of This Regulation

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

The report will include an analysis of the impacts of the various alternatives on the water supply industry vis-a-vis capital costs of technology, operating and maintenance costs and the feasibility of financing new treatments. Additionally impacts on the consumer and the nation as a whole will be analyzed.

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

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

There are no paperwork burdens associated with this regulation, and there are no information collection requirements subject to the provisions of the Paperwork Reduction Act (144 U.S.C. 3501 *et seq.*).

X. Public Docket

All supporting materials pertinent to the development of this proposal are included in the Public Docket located at EPA headquarters, Washington, DC. The Public Docket is available to the public and the public should contact the Drinking Water Regulations Docket Manager for access. It would have been desirable to publish in this notice the list of supporting materials, but the Public Docket is voluminus and a listing of the documents in this notice would be much too long. However, references on occurrence of chemicals in drinking water, analytical methods and health effects criteria document are cited below as these documents provide summaries of data used in determining the proposed RMCLs. Other materials in the Public Docket include such documents as the following:

- Public comments on the ANPRM.
- Transcript of the December 13, 1983, Public Meeting.
- Report and background materials for the four public workshops, Fall 1983.
- Transcripts and minutes of NDWAC Meetings.
- Summaries of meetings, telephone calls from outside EPA.
- Letters to/from public.
- Technical Reports.
- Other supporting materials.

For each inorganic and organic chemical for which RMCLs are proposed, a health effects criteria document has been prepared and is available to the public. For example, a typical reference listing would be as follows:

EPA, Office of Drinking Water, Criteria and Standards Division, Draft Health Effects Criteria Document for *Lead*, September 1984.

Similarly, documents summarizing the occurrence of inorganic and organic chemicals in drinking water have been prepared for each chemical for which RMCLs are proposed. Individual documents have been prepared for inorganic chemicals whereas one document including all organic chemicals has been prepared. A typical reference listing for the inorganics occurrence document would be as follows:

EPA, Office of Drinking Water, Criteria and Standards Division, Draft Occurrence/Exposure of *Lead* in Drinking Water, September 1984.

The SOC occurrence document is referenced as follows:

EPA, Office of Drinking Water, Criteria and Standards Division, Draft Occurrence/Exposure of Synthetic Organic Chemicals (including pesticides) in Drinking Water, September 1984.

Summaries of the analytical methods, occurrence in drinking water and health effects in a single document have been prepared for each microbial contaminant for which RMCLs are proposed. A typical reference listing would be as follows:

EPA, Office of Drinking Water, Criteria and Standards Division, "Analytical Methods, Occurrence, and Health Effects of *Total Coliforms* in Drinking Water, September 1984.

Other pertinent references available in the public docket include the following:

"Standard Methods for the Examination of Water and Wastewater," 15th Edition, American Public Health Association, American Water Works Association, Water Pollution Control Federation, 1975.

Annual Book of ASTM Standards, Part 31 Water, American Society for Testing and Materials, 1916 Race Street, Philadelphia, Pennsylvania 19103.

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

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

EPA, EMSL. "Methods for Chemical Analysis of Water and Wastes" (EPA 600/4-79-020, March 1979). Available from ORD Publications, CERI, EPA, Cincinnati, Ohio 45268.

National Academy of Sciences, "Drinking Water and Health, Volume I (1977), II (1980), III (1981), IV (1981), V (1983).

IARC, 1979. IARC Monographs on the evaluation of the carcinogenic risk of chemicals to humans: some halogenated hydrocarbons. Vol. 20: 14-15.

IARC, Approaches to Classifying Chemical Carcinogens According to Mechanism of Action, Technical Report No. 83/001. April 1983.

NCI, "Policy of Risk Assessment of the Health Effects of Hazardous Exposures to Populations," Subcommittee on Environmental Carcinogens, National Cancer Advisory Board. 1983.

Assessment of Microbiology and Turbidity Standards for Drinking Water (Workshop Proceedings), EPA, ODW, 1983.

Evaluation of the Microbiology Standards for Drinking Water. NTIS, Accession No. PB 297119.

49 Federal Register, 46294 (November 23, 1984). EPA's Proposed Guidelines for Carcinogen Risk Assessment.

The above health effects criteria documents and summary documents for

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the microbials are available for viewing at EPA headquarters or in any of the ten EPA regional offices as listed in the beginning of the notice. Copies of these documents can be obtained for a fee at NTIS, U.S. Department of Commerce, 5285 Port Royal Road, Springfield, Virginia 22161. The toll free telephone number is 800/336-4700; local 703/487-4650.

XI. Request for Public Comment

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

• Is the regulatory approach in this proposal appropriate under the SDWA, i.e., set regulations for contaminants (1) that pose a health risk to consumers in drinking water, and (2) that have the potential for occurring in drinking water (e.g., pesticides registered for use in or near drinking water supplies) or that are known to occur on a regional or national basis but not necessarily at (a) high frequencies or (b) high levels.

This approach would result in a comprehensive list of regulated contaminants which would provide a standard by which to assess drinking water when contamination is found. Inherent in this approach is full public participation in the standard setting process. The alternate regulatory approach which would set regulations for contaminants that pose a health risk in drinking water but only for those contaminants detected at relatively high frequencies at levels near the level of health concern. Health Advisories would be provided as needed for those contaminants for which regulations were not developed.

• Do the proposed RMCLS represent a level such that "no known or anticipated adverse effect would result with an adequate margin of safety"?

—Is the three-category approach for setting RMCLs an acceptable method for factoring strength of evidence in the RMCL determinations?

—Are the classifications of the chemicals scientifically acceptable?

—For non-carcinogens, is the approach and actual studies used for computing the AADIs scientifically acceptable? Are the safety factors used in the ADI calculcation for each contaminant scientifically acceptable? Where data are not available, is providing for an assumed contribution of 20 percent from drinking water appropriate?

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

—For compounds with equivocal evidence of carcinogenicity, should the RMCLs be set based upon AADIs, cancer risk levels or some other method?

• The term "Acceptable Daily Intake" or "ADI" has been criticized as connotating an "acceptable" level of exposure to which any level greater than the ADI is "unacceptable". This is contrary to the views of most scientists who generally interpret the ADI as a "ballpark figure" which represents a level of exposure which is not likely to result in adverse effects in humans. It is viewed as a soft estimate in that exposures somewhat higher than the ADI are generally not expected to result in adverse effects; only if the ADI is significantly exceeded would one expect such negative consequences.

In order to help prevent misinterpretation, the term "ADI" could be replaced with the term "reference dose". This term was derived based upon the process of generating the ADI in which the NOAEL based upon animal data is divided by an uncertainty factor to generate a human dose. This derived human dose then serves as a point of reference, a benchmark against which other human doses are compared.

Comment is requested on the use of the term "Reference Dose" to replace ADI.

• The term "Adjusted Acceptable Daily Intake" or "AADI" has been used to represent a total allowable exposure as measured in mg/l drinking water, but is derived directly from the ADI which is measured in mg/kg body weight/day. This modification of units of measurement for a concept that is traditionally expressed in mg/kg/day has led to some confusion and criticism, particulary from the toxicological community. It has been proposed that a more appropriate term be created, such as "Drinking Water Equivalent Level (DWEL) or "Drinking Water ADI" (DWADI). These terms would allow a shift to a medium-specific unit (mg/l drinking water) and define a concentration in drinking water which, during the entire lifetime of the human, would be estimated to be without appreciable risk.

Comment is requested on the use of the terms "Drinking Water Equivalent Level" or "Drinking Water ADI" to replace AADI.

• How should exposure to multiple contaminants in drinking water be addressed to in the Revised Regulations? Because of concerns of synergistic effects should a RMCL and MCL be set for total SOCs or total IOCs?

• Are the levels proposed for RMCLs for microbial contaminants appropriate?

• Is the approach being proposed for *Giardia* and viruses which would set RMCL and MCLs, and monitoring but also require a treatment technique regulation of filtration and disinfection for surface waters and disinfection for ground waters appropriate? The public water system would have a choice of meeting the MCLs vs. the treatment technique requirement. What criteria would be appropriate upon which to base a variance from the treatment technique requirements for surface water systems? for ground water systems?

• Should certain types of non-community systems, such as schools and factories, be required to meet the MCLs that apply to community water systems?

• Are the cited analytical methods cited available (i.e., economically and technologically feasible) to ascertain the level of those contaminants for which RMCLs are proposed?

A public hearing will be held at EPA in Washington, DC, on January 28 and 29, 1986, in Conference Room 1, adjacent to the Washington Information Center for the interested public to comment and provide information and data on these proposed regulations.

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

Lists of Subjects in 40 CFR 141

Chemicals, Intergovernmental relatives, Radiation protection, Reporting and record keeping requirements, Water supply.

Dated: October 10, 1985.

Lee Thomas,

Administrator.

Appendix A.—Summary of Public Comments Pertinent to the Proposed Recommended Maximum Contaminant Levels (RMCLS) for Organic, Inorganic and Microbial Contaminants in Drinking Water

The following is a summary and discussion of the principal public comments to EPA's proposed rule for the establishment of RMCLs for organic, inorganic, and microbial contaminants in drinking water. EPA specifically solicited comments on the following issues in its October 5, 1983, Advance Notice of Proposed Rulemaking (ANPRM):

1. Is the three-tiered approach appropriate? What criteria should be used to determine which contaminants should be in each category?

2. For which contaminants should regulations be set? Into which category should the contaminants be included: Category I, Category II, of Category III as described? What levels for the RMCLs would be appropriate?

3. Should a treatment technique requirement of disinfection be set for all ground water systems? Should a treatment technique requirement of filtration be set for surface water systems?

4. Is using the ADI an appropriate method for establishing RMCLs for non-carcinogens?

5. What safety factors should be used in conjunction with chronic toxicity data in setting RMCLs for non-carcinogens?

6. What approach should be used to set RMCLs for carcinogens?

7. Is waiving certain MCLs when susceptible populations are not affected an appropriate approach?

8. Should separate or different MCLs be considered for certain contaminants for non-community water systems?

9. Are analytical methods available for contaminants under consideration?

EPA received 133 written comments during the 60+ day public comment period and three statements were presented at the public meeting held in Washington, D.C., on December 13, 1983. The statements presented at the public meeting have been incorporated with the written comments submitted. These 135 comments included 62 industries and industry associations, 33 water utilities, 11 state governments and state organizations, 6 public interest groups, 4 private citizens and 11 from other groups including some federal government agencies.

The following discussion summarizes comments pertinent to the RMCL proposal (i.e., primarily occurrence, health effects, and analytical methods) received on the ANPRM for revised drinking water regulations. Comments on such items as monitoring and GAT are pertinent to MCLs and will be summarized in the MCL proposal.

(1) Is the three-tiered approach appropriate? What criteria should be used to determine which contaminants should be in each category?

Thirty-four comments addressed this issue. The majority of commenters favored the three-tiered approach because this is a sound regulatory approach which offers States monitoring flexibility and permits States to optimize available resources. Most of these commenters agreed with the criteria for each category, as stated in the ANPRM. A few commenters suggested some recommendations to this approach, as outlined in the ANPRM, which included:

- retain the distinction between community and non-community water supplies;
- additional categories should be added in the future, if necessary;
- Category I should read, "have an adverse effect," not "*may* have an adverse effect";
- monitoring frequency for Category II chemicals should be established by the State using guidelines set by EPA;
- monitoring should be based on geographical areas within a State, type and source of supply, historical data, and system size; and
- a national monitoring program should be carried out before categorizing the chemicals.

A few commenters did not favor the three-tiered approach. One commenter felt "the concept is needlessly complicated and may result in a lengthy, unreasonable, and superfluous debate over which category a chemical should be in." They suggested only two categories—MCL or no MCL. Their reasoning was that primacy agencies could adjust monitoring requirements. Furthermore, they recommended EPA put a time limit on the applicability of health advisories so they will either be updated, establish a MCL if necessary or withdraw the health advisory and provide reasons for doing so. Some of the other commenters who did not favor this approach were also concerned about the health advisory program but for different reasons. They felt that the health advisories were being misused by other EPA program offices and they go beyond the statutory authority of the Safe Drinking Water Act (SDWA), which does not provide for a program of quasi-regulatory guidance to state and local officials. Their recommendations were for health advisories to receive independent scientific peer review and for the SDWA to be amended to formalize the health advisory process.

(2) For which contaminants should regulations be set? Into which category should the contaminants be included: Tier I, Tier II or Tier III as described? What levels for the RMCLs would be appropriate?

The majority of written comments received addressed which contaminants should regulations be set, specifically MCLs. The comments received did not distinguish between RMCLs and MCLs, however, it is appropriate to discuss for which contaminants should regulations be set for even though the comments pertain to MCLs, not RMCLs. Concerning occurrence data, five commenters felt EPA should broaden its data base. A single, large scale survey to determine the occurrence of the various constituents was suggested. Occurrence studies should be broadened to include statistically valid, geographically randomized sample of every system size, including ground and surface water sources. It was also proposed that EPA adopt a chemical data reporting system for all substances identified under other PEA programs. EPA should then provide this information to the primacy agencies.

Sixteen comments addressed the tier in which the contaminants should be included: Tier I, Tier II or Tier III. The majority of commenters felt that the turbidity and total coliform standards should be in Tier I to protect against waterborne disease. The inorganic chemicals currently regulated by EPA were placed predominantly in Tier II to allow for monitoring flexibility, but a few commenters recommended deleting a few of the MCLs and developing health advisories instead (Tier III). The several contaminants listed in the ANPRM that were already included in the secondary drinking water regulations were all placed in Tier III along with several other inorganic chemicals. A few commenters felt that the synthetic organic chemicals should either be in Tier II or III, depending on occurrence and adverse health effects information. The distribution of chemicals for Tier I, II and III was as follows:

Chemical	Tier		
	I	II	III
Arsenic		6	2
Barium		11	2
Cadmium		10	
Chromium		10	2
Lead	2	6	
Mercury		6	2
Nitrate	2	6	
Silver		5	1
Selenium		11	2
Coliforms	9		

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Chemical	Tier		
	I	II	III
Turbidity	6	1	
Aluminum			8
Antimony			4
Asbestos			8
Beryllium			1
Copper			8
Cyanide			3
Molybdenum			4
Nickel			1
Sodium			6
Sulfate			3
Thallium			1
Vanadium			3
Zinc			6
Viruses			1
Legionella			1
Giardia			1
SOCs	1	7*	

*II or III.

In order to present a summary of the comments which addressed which contaminants should regulations be set, the discussion will be summarized per chemical.

Inorganics

Arsenic

Eleven commenters addressed the present arsenic MCL of 0.05 mg/l. The majority of commenters felt the standard should remain the same because it appears that both valences are toxic and there is no available analytical method to differentiate. However, three commenters felt that due to differing toxicities, separate MCLs for trivalent and pentavalent arsenic should be established, if an acceptable means of analysis is available to differentiate the two compounds.

Two commenters felt the MCL should be deleted and EPA should prepare a Health Advisory. Their reasoning is that studies have been done in their cities because they have levels of arsenic in their drinking water above the MCL. The results of these studies have shown no ill-effects from ingesting these arsenic levels over many years.

Barium

Twenty-five commenters addressed the present barium MCL of 1.0 mg/l. One commenter submitted detailed scientific information on barium, which has been considered by EPA. The majority of commenters felt the barium MCL should be raised to 4.7 mg/l, which is the level the National Academy of Sciences recommended in *Drinking Water and Health*, Vol. 4. These commenters supported this position because an absorption factor of 20 percent was used, even though conservative, rather than 80 percent which was used for the present MCL. These commenters felt that, "there is no evidence in humans that gastrointestinal absorption rates are significantly different for adults or children". They also questioned using the new-born rat as a model for children. Furthermore, they felt that the possible role of barium and cardiovascular disease may not be supported since they are based on intravenous studies.

Six commenters felt that the standard should remain the same, whereas, two commenters felt the standard should be deleted. No reasons were stated for either position.

Cadmium

Twelve commenters addressed the present cadmium MCL of 0.010 mg/l. The majority of commenters felt the MCL should remain the same. One commenter mentioned leaching of cadmium due to corrosion of galvanized pipe as the mode of exposure in drinking water. Two estimates of the relative source contribution of cadmium from drinking water were 10 percent and 6.1 percent. One commenter felt the MCL should be raised but did not specify a level.

Chromium

Twenty-two comments addressed the present MCL for total chromium of 0.05 mg/l. Some commenters submitted detailed scientific information on chromium which has been considered by EPA. The majority of commenters felt the MCL should be changed either by establishing a separate MCL for trivalent (Cr III) and hexavalent chromium (Cr VI) or establishing an MCL for only hexavalent chromium. Their reasoning was that the two valence states can be distinguished analytically and an MCL for Cr(VI) is warranted based on its toxicity while Cr(III) is non-toxic and considered an essential nutrient. This position follows NAS' recommendation in *Drinking Water and Health*, Vol. IV. Two commenters suggested screening for total chromium and when this exceeds the numerical standard, analyzing for hexavalent chromium to determine whether the water meets the standard. These commenters further stated that total chromium in drinking water is not of significant concern. Reasons for this opinion included: (1) Chromium is extremely insoluble and immobile in the natural environment, (2) it is rarely found at concentrations above 0.05 mg/l, (3) only the trivalent form occurs in natural waters since the hexavalent form is quickly reduced, and (4) traces of Cr(VI) would be quickly converted to Cr(III) due to the acidic nature of the stomach. Most of these commenters felt that Cr(III) does not oxidize to Cr(VI) during chlorination. However, one commenter stated that "chlorination will oxidize Cr(III) to Cr(VI) since it is the thermodynamically favored species under oxidative conditions in solution".

A few commenters felt there should be no change to the present MCL for chromium since Cr(III) oxidizes to Cr(VI) and no easy procedure exists to analyze the two species separately.

Two commenters felt that the chromium standard should be deleted altogether. No reasons were stated.

Lead

Twenty-one comments addressed the present MCL for lead of 0.05 mg/l. The majority of commenters felt the present MCL should remain the same since this level protects the health of children and adults. They felt the MCL should not be lowered because the belief that even very low levels of lead in the blood have adverse effects on children is erroneous. Most of these commenters agree with EPA's statement in the ANPRM that, "while lead in drinking water may be the result of contamination of the water source, it most frequently results from corrosion in the distribution system". They favored reducing lead levels by implementing corrosion control programs such as special monitoring activities where corrosion in copper, galvanized and lead piping is suspected of being a problem. Furthermore, they suggested that regulations for corrosion control should be determined by the primary agency based on local conditions. A study in Carroll County, Maryland, found the current MCL was exceeded 24 percent of the time.

Most of the plumbing in this study was copper with lead soldered joints. One commenter noted that cigarettes and alcohol consumption were important sources of lead exposure: 9 percent due to cigarettes, 8 percent due to alcohol, 6 percent due to drinking water, and 77 percent due to other influences.

A number of commenters felt the lead standard should be lowered since "lead is bioaccumulative and the health effects of lead, particularly on the central nervous system is irreversible". Most of these commenters agreed with NAS' recommendation that the MCL should be lowered to 0.025 mg/l. One commenter felt the RMCL should be zero because lead is non-essential and any amount is undesirable and probably harmful and the MCL should be 0.002 mg/l. Another commenter suggested the RMCL should be 0.01 mg/l in order to protect the young and developing nervous systems of infants and young children.

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Conversely, one commenter felt the MCL should be raised. No reasons were stated.

Mercury

Eleven comments addressed the present MCL for mercury of 0.002 mg/l. The majority of commenters felt the standard should remain the same and recommended analytical methods be developed to differentiate between organic and inorganic mercury. Conversely, two commenters felt the standard should be deleted. No reasons were given.

Nitrate

Fifteen comments addressed the present nitrate-nitrogen MCL of 10 mg/l. The majority of commenters felt the MCL should remain the same since it is a reasonable standard especially concerning the flexibility afforded for non-community systems. A few other commenters felt that the same provision which applies to non-community water systems should also apply to community water systems since "water related cases of methemoglobinemia that have been reported, have seldom if ever involved community public water systems". Two commenters questioned the nitrate standard because there is little information available to support either retaining or revising the present MCL. They recommended that, "primacy agencies have more flexibility in resolving problems where infants are likely to consume the water". Another commenter stated that nitrate contamination can be minimized by modifying the well construction, as stated in the ANPRM. This information should be disseminated to the consumer.

One commenter believed that the MCL should be raised to 20 mg/l because nitrate is expensive and difficult to remove. They recommended using premixed formula or bottled water and issuing advisories to local physicians, health departments and hospitals, in order to protect children and pregnant women.

Selenium

Eighteen comments addressed the present MCL for selenium of 0.01 mg/l. The majority of commenters felt either the MCL should be raised or deleted. Their reasoning was that selenium has anticarcinogenic potential and the scientific basis for the present MCL appears at best questionable. Also, there is no economical way for small systems to remove selenium at present MCL levels. Conversely, a number of commenters felt the MCL should remain the same and placed in Tier II to allow for monitoring flexibility.

Silver

Eighteen comments addressed the silver MCL of 0.05 mg/l. Some commenters submitted detailed scientific information on silver, which has been considered by EPA. The majority of commenters felt the MCL should be deleted because of minimal occurrence and because of the lack of adverse health effects. A few commenters stated that silver does not occur at elevated levels in drinking water as a result of photographic discharges. Furthermore, "silver is rapidly and almost completely incorporated into sludge during secondary wastewater treatment". Some of these commenters feel that argyria is not an adverse health effect but a cosmetic effect. One commenter cited the MCL reasoning as, "the need to set a standard for silver arises from its intentional addition to water as a disinfectant. The chief effect of silver in the body is cosmetic". They supported NAS' recommendation in *Drinking Water and Health*, Vol. 1 to delete the MCL due to limited occurrence. Some commenters suggested preparing a health advisory for silver.

Five commenters felt there should be no change to the silver MCL. They felt it should be in Tier II allowing the States flexibility in monitoring due to silver's infrequent occurrence.

Aluminum

Twenty-two commenters addressed the possibility of establishing a MCL for aluminum. Three commenters submitted detailed scientific information on aluminum, which has been considered by EPA. The majority of commenters felt aluminum does not warrant an MCL because drinking water is not a significant source of aluminum exposure and there are no clearly demonstrated adverse health effects for the general population. Most of these commenters felt aluminum should be in Tier III so as to protect the sensitive subpopulation of dialysis patients. However, one commenter stated, "normal dietary sources of aluminum are an unlikely source of concern relative to the dementia cited in the ANPRM". Another commenter suggested reporting requirements similar to sodium, on an annual basis to local health officials to afford protection to dialysis patients. Two commenters felt that regulations under the SDWA are not an appropriate vehicle to deal with senile dementia and dialysis encephalopthy. A number of commenters raised the point that, "any limitations placed on aluminum in drinking water should recognize the importance of the use of alum in the treatment of drinking water". These commenters stressed that aluminum salts used in water treatment are the most common source of this metal in drinking water.

Conversely, a few commenters felt that an MCL was justified based on the adverse health effects. One commenter suggested adopting the WHO guideline of 0.2 mg/l.

Antimony

Fourteen comments addressed the possibility of establishing an MCL for antimony. All of the commenters felt that an MCL was not warranted based on lack of occurrence and adverse health effects. A few commenters recommended developing a health advisory for antimony (Tier III).

Asbestos

Twelve commenters addressed the possibility of establishing an MCL for asbestos. All of the commenters felt that an MCL is not justified. One commenter observed that all but a small percentage of U.S. drinking water contains asbestos concentrations below on million fibers per liter. This exposure is due to natural erosion. This commenter stated that exposure to asbestos in food and air is estimated to be 1,000 to 10,000 times that due to drinking water. The above commenters felt that there is no evidence available to demonstrate a health risk from ingested asbestos. One commenter stated that "if there is a causal relationship between asbestos in drinking water and cancer, it is extremely weak". Two commenters stated, "if additional research demonstrates an adverse health effect from ingested asbestos, control can be provided by requiring properly designed and operated filtration plants for surface water supplies and distribution of non-corrosive water". A number of these commenters felt asbestos should be in Tier III.

Beryllium

Six commenters addressed the possibility of establishing an MCL for beryllium and they all agreed that an MCL is not warranted. One commenter stated that there is no evidence that ingested beryllium poses a human cancer risk nor any other adverse health effects. Furthermore, beryllium is present in natural surface waters at concentrations generally less than 1.0 μg/l with average concentrations of generally less than 0.2 μg/l. Beryllium is relatively insoluble and is rapidly absorbed by clays.

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Copper

Fifteen comments addressed the possibility of establishing an MCL for copper. The majority of commenters felt an MCL is not warranted and copper should remain as a secondary regulation. A few of these commenters recommended copper be placed in Tier III. Conversely, one commenter felt an MCL should be established because there is evidence of adverse health effects from exposure to elevated levels in a public water supply system in their State.

Cyanide

Eight comments addressed the possibility of establishing an MCL for cyanide in a general fashion. All of the commenters felt that a MCL was not required but recommended a health advisory be set (Tier III).

Molybdenum

Thirteen comments addressed the possibility of establishing an MCL for molybdenum. All of the commenters felt an MCL was not justified due to lack of adverse health effects. One commenter stated that a failure in the ANPRM was the omission of discussing the question of molybdenum deficiency particularly as it relates to cancer. This commenter explained the limitation of NAS' adequate and safe intakes, that it is a range, not a specific limit. A few of the above commenters recommended molybdenum for Tier III.

Nickel

Thirteen comments addressed the possibility of establishing an MCL for nickel. All of the commenters stated that an MCL is not warranted since there are no adverse health effects and nickel in drinking water contributes a very small portion of the daily intake. One commenter stated that it is seldom observed in fresh water.

Sodium

Eighteen comments addressed the possibility of establishing an MCL for sodium. Presently, there are monitoring and reporting requirements for sodium to protect the sensitive subpopulation on sodium restricted diets. All of the commenters stated that an MCL is not required by recommended developing a health advisory (Tier III). These commenters stated that food is the major source of sodium, not drinking water. One commenter noted that drinking water sodium levels seldom exceed 200 mg/l. Another commenter recommended that point-of-use treatment devices are the most practical, effective and economical means for removing sodium. They also suggested that EPA do research on the role of chloride and hypertension. Some treatments add sodium to drinking water, such as soda ash addition and ion exchange softening.

Sulfates

Fourteen comments addressed the possibility of establishing an MCL for sulfates. Presently, there is a secondary maximum contaminant level of 250 mg/l. All of the commenters recommended that an MCL was not warranted and should remain as a secondary regulation since the sole attributable adverse health effect is as a laxative. Two commenters noted that elevated levels of sulfate are not uncommon. Levels as high as 2,000 to 3,000 mg/l have been reported. However, one commenter stated that "EPA's 1975 Interstate Water Carrier Analysis found that even the highest concentration of sulfate detected was below the level at which adverse health effects occur; 90 percent less than, 250 mg/l". This commenter supported NAS' contention that there are no observable adverse health effects at 500 mg/l.

Thallium

Five comments addressed the possibility of establishing an MCL for thallium in a general fashion. All of the commenters felt that an MCL was not warranted; no reasons were stated.

Vanadium

Twelve comments addressed the possibility of establishing an MCL for vanadium. They all felt that an MCL was not justified and that a health advisory should be developed (Tier III). One commenter stated that vanadium is "poorly absorbed when ingested and there is a lack of evidence of any chronic oral toxicity".

Zinc

Seventeen comments addressed the possibility of establishing an MCL for zinc. Presently, there is a secondary maximum contaminant level of 5 mg/l. All of the commenters felt an MCL for zinc was not warranted. Most of the above commenters suggested developing a health advisory (Tier III) and/or maintaining the secondary drinking water regulation. One commenter stated that there is no evidence of chronic zinc toxicity and acute symptoms do not apparently occur in water at levels less than 40 mg/l. Another commenter noted that "at levels well below toxicity, it would cause taste problems making the water unfit to drink".

Synthetic Organic Chemicals (SOCs)

Twenty-two comments addressed the issue as to whether or not to establish RMCLs for synthetic organic chemicals. The majority of comments dealt generally with synthetic organic chemicals, not specific chemicals. Most of the commenters felt that regulations should be set for SOCs and put in Tier II so primacy agencies would have monitoring flexibility to account for wide variations of occurrence. Other commenters recommended developing health advisories (Tier III) until sufficient occurrence and health effects data are available. One commenter noted that the only pesticides likely to intrude into ground water are highly polar, water-soluble, soil-incorporated compounds such as aldicarb, and conditions can be evolved to limit their leaching. A few commenters suggested that a random, nationwide survey should be conducted and funded by EPA to determine the frequency of occurrence and concentration levels to see whether a contaminant is placed in Tier II or III. Another commenter noted that, "the availability of health advisories, in the absence of a drinking water standard help present a more accurate picture of the health issues to the public, however, in many cases, the absence of a drinking water standard may actually result in the installation of treatment facilities for levels of contamination which actually do not exceed the Health Advisory level. Were formal MCLs established, it is unlikely that removal would occur unless the MCL were exceeded". In regard to the six pesticides currently regulated by EPA, one commenter stated that, "there are virtually no cases of non-compliance".

PAHs

Two commenters specifically addressed PAHs. One commenter stated that "the control of PAHs could best be implemented by limiting or discontinuing the use of coal-tar products in water distribution and storage systems. The other commenter recommended PAHs for either Tier II or III due to their widespread use in the water supply industry and their high toxicity.

Acrylamide

Two commenters felt it was inappropriate to establish an MCL for acrylamide. One of the two commenters want the phrase in the ANPRM of "total acrylamide" to be replaced with "acrylamide monomer". They recommended that EPA delay establishing an RMCL for acrylamide

until they complete an c
chronic toxicity study. A
commenter stated that "
acrylamide is regulated
this standard does not p
health, then the FDA sta
adjusted rather than est
additional regulations".

Adipates

Two commenters felt
adipates was not warra
are no ingestion related
effects and results from
data indicate that adipe
persist in the water colu

Glyphosate

Two commenters did
MCL for glyphosate. On
was against using regist
compound for aquatic u
determining criterion. "T
potential for presence in
not of itself present a sig
to the public health. Env
treatability and toxic pr
glyphosate are very favo
against the need for an
Furthermore, analytical
very expensive and requ
degree of skill."

DBCP

One commenter did n
MCL for DBCP because
felt that "there are no ad
effects below 100 ppb a
below 80 ppb. In additio
there is no increase in t
due to DBCP occupation
which is orders of magni
than drinking water exp
health effects informatio
submitted by this comm
been considered by EPA

Phthalate Esters

Two commenters did
MCL for phthalate ester
limited occurrence and
toxicity to warrant regul
phthalates are bio-degra

Alachlor

One commenter did n
establishing an MCL for
occurrence in drinking w

Xylenes

One commenter did n
MCL for xylene. No rea

Toluene

Two commenters felt
was not warranted for t
according to EPA's Heal
Document and the Scien

until they complete an oncogenicity/chronic toxicity study. Another commenter stated that "residual acrylamide is regulated by FDA and if this standard does not protect public health, then the FDA standard should be adjusted rather than establishing additional regulations".

Adipates

Two commenters felt that an MCL for adipates was not warranted since there are no ingestion related adverse health effects and results from unpublished data indicate that adipates do not persist in the water column.

Glyphosate

Two commenters did not support an MCL for glyphosate. One commenter was against using registration of a compound for aquatic use as the single determining criterion. "The mere potential for presence in raw water does not of itself present a significant hazard to the public health. Environmental fate, treatability and toxic properties of glyphosate are very favorable and argue against the need for an MCL. Furthermore, analytical methodology is very expensive and requires a high degree of skill."

DBCP

One commenter did not support an MCL for DBCP because the commenter felt that "there are no adverse health effects below 100 ppb and certainly not below 50 ppb. In addition, in humans, there is no increase in tumor formation due to DBCP occupational exposure which is orders of magnitude higher than drinking water exposure". Detailed health effects information was submitted by this commenter, which has been considered by EPA.

Phthalate Esters

Two commenters did not support an MCL for phthalate esters because of limited occurrence and insufficient toxicity to warrant regulation. Also, phthalates are bio-degradable in water.

Alachlor

One commenter did not favor establishing an MCL for alachor because occurrence in drinking water is low.

Xylenes

One commenter did not support an MCL for xylene. No reasons were given.

Toluene

Two commenters felt that an MCL was not warranted for toluene because according to EPA's Health Assessment Document and the Science Advisory Board, "no adverse health effects are likely in humans at current exposures".

Microbials

Total Coliforms

The MCL for total coliforms of <1/100 ml monthly average and 4/100 ml per single sample was addressed by twenty-three comments. A number of issues were raised in the ANPRM, including:

- Is the total coliform test still appropriate as an indicator?
- Is the presence/absence test appropriate?
- Are check samples appropriate?

All of the commenters agreed that the total coliforms test is still an appropriate indicator of the microbiological quality of drinking water and should remain as an MCL. Two of these commenters felt that an RMCL for total coliforms is inappropriate since this is an indicator of water quality, not contaminants per se, but favored a coliform MCL. However, most of these commenters felt the MCL should be simplified. A recommendation supported by many was the presence/absence test. Some commenters noted that the presence/absence test has merit but is not without problems. One commenter stated they "prefer the presence/absence test over estimates of the most probable number; however, the changes in procedures should be accompanied by a requirement to take and test more samples and to conform to other statistical constraints needed to assure that the method is reliable. Furthermore, at times, coliforms and other bacteria remain viable in water, but fail to grow and they are not detectable by standard analytical methods". It should be noted that a few commenters were against using the presence/absence test because there is not enough data to support this test for its use either in conjunction with, or as a substitute for existing procedures.

Another argument for simplifying the present coliforms MCL came from small water supply systems who said that "monthly averages are not only complicated but meaningless for small systems. The significance of the exact number of bacteria is not understood".

Most of the commenters felt that a "check" sample is imperative when a positive coliform sample occurs. A number of these commenters recommended that "check" sample results should be included in calculating compliance, unlike the current practice.

In addition, a few commenters suggested measuring only for fecal coliforms, not total coliforms since it would better reflect the conditions of the water system.

Turbidity

Twenty-two comments addressed the turbidity MCL of 1 turbidity unit (TU) (up to 5 TU). All of the commenters supported inclusion of turbidity in the Revised Regulations to insure the microbiological quality of drinking water. The majority of these commenters recommended turbidity for Tier I and retention of the same level for the MCL. Two commenters states that an RMCL for turbidity is inappropriate since turbidity is an indicator of water quality, not contaminants per se, but favored an MCL. A few commenters recommended either lowering the MCL to 0.2 TU for protection against *Giardia* and other cysts or establish a treatment goal of 0.2 TU as guidance.

Standard Plate Count

Twenty-four comments addressed the possibility of establishing an MCL for standard plate count (SPC). An overwhelming majority felt that an MCL was not warranted for the following reasons:

- A significantly large number of waterborne illnesses are not correlated to bacteria other than those specifically recognized as pathogenic,
- SPC is a good operational tool, best used as a guideline or screening mechanism, and
- SPC was recommended to be used in conjunction with the total coliforms test.

Conversely, a few commenters supported establishing an MCL for standard plate count. They recommended an MCL in the range of 100 to 500 colony-forming units (CFU)/ml. Their reasoning was based on the following:

- Confirmation of >500 CFU/ml should cause the water to be non-potable and require treatment,
- In one public water system a good correlation exists between SPC and chlorine residuals. In areas with low to non-existent chlorine residuals, high SPCs are seen although no coliforms are present. The reverse is true in areas with sufficient residual—no coliforms and low SPC, and
- Interference of total coliform analysis.

Giardia

Eighteen comments addressed the possibility of establishing an MCL for *Giardia*. The majority of commenters did not favor establishing an MCL because the analytical methodology is not economically or techically feasible. In addition, a few of these commenters felt *Giardia* does not constitute a significant threat to the general

population. One of the above commenters stated, "efforts should be made to develop improved testing procedures for *Giardia*; if so, then an MCL for *Giardia* should be established".

Some of the commenters who did not favor a drinking water standard for *Giardia*, mentioned that filtration followed by disinfection is effective against *Giardia*. However, they opposed mandatory filtration as the only alternative to protect against *Giardia*, but recommended source control (e.g., beaver removal). One commenter further stated that "filter treatment is neither the cheapest nor the most effective means of control of *Giardia* cysts in drinking water supplies for a required treatment method for Giardiasis control".

Conversely, two commenters felt an MCL should be established even though *Giardia* is difficult to enumerate but rather easy on a presence/absence basis. One commenter stated that, "25% of the cases of waterborne disease reported between 1981–80 were due to *Giardia*". Another commenter felt that *Giardia* should be monitored on a monthly basis for all untreated or disinfected only water supplies. Furthermore, "when an analytical method is available, then an MCL of 0 cysts should be established for finished water regardless of treatment".

The issue of mandatory filtration will be further discussed later in this section.

Viruses

Nineteen comments addressed the possibility of establishing an MCL for viruses. All of the commenters did not favor an MCL for viruses because analytical methods are not available which are timely and cost-effective. They felt more research was needed in this area. Proper disinfection should be emphasized but these commenters did not recommend mandatory disinfection.

Legionella

Eighteen comments addressed the possibility of establishing an MCL for *Legionella*. Every commenter felt that an MCL was not warranted since the relationship beween *Legionella* and public water systems has not been established. One commenter noted that, "an MCL for *Legionella* is not warranted because it is a problem that originates with a user's substandard operation of a hot water system". Another commenter further stated that, the best defense is education of homeowners and building supervisors". In addition, another commenter felt that an MCL was not warranted because the primary route of infection is via the respiratory system, and not enteric. *Legionella* is considerably resistant to chlorination and an MCL would just impose heavy costs with essentially no benefits. One State noted that they have had several *Legionella* outbreaks which have been due to an appurtenance in internal plumbing; therefore, control should be directed to appurtenances rather than through drinking water standards.

(3) Should a treatment technique requirement of disinfection be set for all ground water systems?

Twenty-two comments addressed the possibility of establishing a treatment technique requirement of disinfection for all ground water systems. The majority of commenters opposed mandatory disinfection of ground water systems. They felt that mandatory disinfection is a State discretion issue. Furthermore, these commenters recommend physical protection of the source, periodic sanitary surveys and good well isolation and construction. Most of the above commenters urged EPA to focus its efforts on bacterial standards and not on treatment techniques. One commenter felt that properly sited and constructed ground water systems which are regularly monitored for coliforms will not pose a significant added health risk. One State who strongly opposed mandatory disinfection stated that, "it is politically difficult to accomplish the installation of chlorination equipment on all ground water supplies with even further difficulties anticipated in continuing the treatment once started".

Conversely, some commenters favored mandatory disinfection for all ground water systems. However, a few of these commenters felt that States should be allowed the flexibility to waive for systems based on periodic sanitary surveys and good well construction.

• Should a treatment technique requirement of filtration be set for surface water systems?

Nineteen comments addressed the possibility of establishing a treatment technique requirement of filtration for surface water systems. The majority of commenters favored mandatory filtration for surface water systems to insure the microbiological quality of the drinking water. One commenter felt filtration should be a Tier II standard, with the provision that variances would be allowed if suitable monitoring and watershed characteristics could demonstrate no health risk. A few of these commenters mentioned that mandatory filtration seems prudent particularly in light of the growing concern over infectious agents, such as *Giardia*. One commenter further stated that "the SDWA provides for treatment requirements where monitoring techniques are not practical or available, such as *Giardia*, which can be removed by filtration followed by disinfection". This commenter also supported mandatory disinfection for all public water systems, as a Tier I standard to afford protection against (1) unexpected changes in raw water quality, (2) introduction of contamination in the distribution system, and (3) viruses and *Legionella* which can pass filtration.

The minority opinion who did not favor mandatory filtration for all surface water sytems felt this was a State discretion issue and recommended that it was more appropriate to establish and enforce proper MCLs to insure the microbiological quality of drinking water and then vary monitoring in accordance with the type of source and treatment provided. Furthermore, most of those commenters were against mandatory filtration as the only alternative to protect against *Giardia*. They recommended source control and periodic sanitary surveys. One commenter also recommended "disinfection of surface water supplies and an aggressive monitoring program in the watershed and distribution system," which is consistent with the Microbiology Workshop conclusion, "all surface water sources should be pretreated by such processes as coagulation, sedimentation and filtration or their equivalent prior to disinfection, unless it can be shown on the basis of a sanitary survey that such treatment is not necessary". Another commenter stated that "the establishment of treatment standards other than disinfection is not justified when there are means available to determine water quality".

(4) Is using the ADI an appropriate method for establishing RMCL for non-carcinogens?

Five comments addressed this issue of the ADI approach for non-carcinogens. All of the commenters felt that the ADI approach was quite valuable and appropriate. Two commenters emphasized that the contribution of drinking water to the total exposure should be considered.

(5) What safety factors should be used in conjunction with chronic toxicity data in setting RMCLs for non-carcinogens?

Four commenters addressed the issue of safety factors for non-carcinogens. The commenters recommendations were varied. Two commenters suggested that EPA use a range of factors so that the exact magnitude of uncertainty depends on consideration such as the species tested, and the quality and quantity of

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the test data. Another commenter stated that "a safety factor of 100 should be used to account for differing sensitivities within the heterogeneous population (e.g., young vs. old); variability in body weights and the amount of water ingested daily; and uncertainties in extrapolation". Another commenter felt, "NAS' safety factors are appropriate only when epidemiological data are lacking. If historical data are available to allow a lower safety factor (e.g., NO_3) or where a benefit can be prescribed (e.g., fluoride) then a lower safety factor should be applied".

(6) What approach should be used to set RMCLs for carcinogens?

Eight commenters addressed how RMCLs should be set for carcinogens. Four commenters felt that setting RMCLs at zero for non-threshold carcinogens was appropriate, however RMCLs must be clearly defined as a health goal. A common sentiment among some of these commenters was that zero provides little foundation for the establishment of MCLs. Furthermore, these commenters felt that RMCLs serve no purpose and are impractical because they are confusing to the public and the press, if not the regulated community. Despite the shortcomings of RMCLs, a few commenters made recommendations on how to improve the understanding of RMCLs. One commenter recommended that EPA, States and the American Water Works Association develop educational material to explain the differences between RMCLs and MCLs. Another commenter suggested that RMCLs and MCLs should be proposed and promulgated simultaneously.

Another suggestion as to how RMCLs should be set for carcinogens was expressed by one commenter to, "evaluate each compound and based on strength of the scientific data and comparative carcinogenicity of each compound, establish an RMCL at a level determined to have a minimal or insignificant health risk. These risk levels should be consistent with past determinations of "acceptable" risks to society, possibly 10^{-6}".

Two commenters were opposed to using risk models because there are such a variety of models which argue against the selection of only one for risk assessments. One commenter suggested EPA should consider a combination of mathematical models and the safety factor approach. Another commenter said, "due to the inadequacies in today's science, EPA should state that they are unable to determine safe levels for carcinogens".

One commenter recommended that animal bioassay results would have to be considered "sufficient" evidence of carcinogenicity before they can be considered for use in human cancer risk. They suggested using IARC's classification scheme which states that "limited" evidence in animals is "inadequate" to evaluate the carcinogenicity in humans.

(7) Is waiving certain MCLs when susceptible populations are not affected an appropriate approach?

Five comments addressed the issue whether to waiver certain MCLs when susceptible populations are not affected. The commenters were split on this issue, three felt this was an appropriate approach, particularly concerning nitrate.

Two commenters did not favor this approach. One commenter stated that, "waivers based on population would be very subjective and lead to the setting of undesirable precedents. Gathering the data to make determinations for a 'susceptible population' would be too timely and costly and populations are too mobile. Furthermore, failing to account for synergistic effects from other exposures would make any waiver meaningless in terms of protecting public health".

(8) Should separate or different MCLs be considered for certain contaminants for non-community water systems?

Two commenters addressed this issue. One commenter favored the existing procedures for non-community water systems to comply with the MCLs for coliforms, turbidity and nitrates. The other commenter recommended "changing the definitions for community and non-community to more properly reflect the difference between transient and non-transient populations because the current approach of resident vs. non-resident does not properly protect the health of persons using drinking water from systems such as schools and factories which are non-community systems that may have excessive concentrations of other contaminants, such as lead. Community systems should apply to non-transient populations such as schools, day-care facilities, and factories not just residential. Non-community systems should apply to true transient populations such as hotels, campgrounds, gas stations, restaurants, etc".

(9) Which analytical methods are most reliable?

Only a few comments were received which addressed the availability of analytical methods for contaminants under consideration. The following discussion will be summarized per chemical.

Inorganic Chemicals

Asbestos

One commenter noted that the only available analytical method for asbestos in water, transmission electron microscopy, is very expensive ($300–$600 per sample). This method is neither precise nor accurate enough to use for enforcement purposes.

Chromium

One commenter noted that there are two methods to separate dissolved Cr (III) from Cr (IV). The first was developed by Martin and Riley at U.S. EPA, Environmental Monitoring and Support Laboratory (EMSL). The colorimetric method (*Standard Methods*, 15th ed. #312.B) is also valuable for distinguishing the hexavalent form. They also mentioned that Krull, Panaro and Gerstner (*J. Chrom. Science*, In press, 1983) have developed a method for speciation of Cr (VI) and Cr (III) in water using HPLC-Direct Coupled Plasma Emission Spectroscopy.

Another commenter recommended that the Atomic Absorption method be used to screen for total chromium, but that Cr (VI) should be the basis for the MCL.

Pesticides

One commenter noted the analytical method for glyphosate in water requires a high degree of technical skill and is very expensive. Costs are about $300 per sample. National cost for each public water system to monitor two times per year would approach $100 million.

Another commenter noted that while electron capture detection has excellent characteristics for insecticides and herbicides regulated by the SDWA, it has generally poor detection ability for the currently used phosphorous and nitrogen containing pesticides. Specific detectors are required such as, thermionic detection to detect even high levels of atrazine, simazine and tabufos.

PART 141—(AMENDED)

For the reasons set out in the preamble, it is proposed that the following be added to proposed Subpart F, Part 141, Subchapter D, Chapter I of Title 40, *Code of Federal Regulations* as proposed on May 14, 1985 (50 FR 20175).

1. The authority section of Part 141 continues to read as follows:

Authority: 42 U.S.C. 300g–1, 300g–3, 300j–4, and 300j–9.

2. It is proposed that Subpart F, 40 CFR 141.50 is amended by adding (a) (6) through (15) and (b) table entries (4) through (18) to read as follows:

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§ 141.50 Recommended maximum contaminant levels for organic contaminants.

(a) * * *

* * * * *

(6) Acrylamide.
(7) Alachlor.
(8) Chlordane.
(9) DBCP.
(10) EDB.
(11) Epichlorohydrin.
(12) Heptachlor.
(13) Heptachlor epoxide.
(14) PCBs.
(15) Toxaphene.

(b) * * *

Contaminant	RMCL in mg/l
* * * * *	*
(4) Aldicarb, aldicarb sulfoxide and aldicarb sulfone	0.009
(5) Carbofuran	0.036
(6) 2,4-D	0.07
(7) o-Dichlorobenzene	0.62
(8) cis-1,2-Dichloroethylene	0.07
(9) trans-1,2-Dichloroethylene	0.07
(10) Ethylbenzene	0.68
(11) Lindane	0.0002
(12) Methoxychlor	0.34
(13) Monochlorobenzene	0.08
(14) Pentachlorophenol	0.22
(15) Styrene	0.14
(16) Toluene	2.0
(17) 2,4,5-TP	0.052
(18) Xylene	0.44

3. It is proposed that proposed Subpart F, § 141.51 be amended by adding (b) table entries (2) through (12) to read as follows:

§ 141.51 Recommended maximum contaminant levels for inorganic contaminants

(a) [Reserved]

(b) * * *

Contaminant	RMCL in mg/l
* * * * *	*
(2) Arsenic	0.05
(3) Asbestos (medium and long fibers)	[1]7.1
(4) Barium	1.5
(5) Cadmium	0.005
(6) Chromium (total)	0.12
(7) Copper	1.3
(8) Lead	0.020
(9) Mercury	0.002
(10) Nitrate (as N)	10.0
(11) Nitrite (as N)	1.0
(12) Selenium	0.045

[1] Million fibers per liter.

4. It is proposed to add a new § 141.52 to 40 CFR Part 141 to read as follows:

§ 141.52 Recommended maximum contaminant levels for microbial contaminants.

(a) Recommended maximum contaminant levels are zero for the following microorganisms: total coliforms, *Giardia*, and viruses.

(b) Recommended maximum contaminant levels for the following microorganisms parameters are as indicated: Turbidity 0.1 Nephelometric Turbidity Unit.

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