J. Does Anchorage Have an Inspection and Maintenance (I/M) Program in Place That Meets EPA Requirements in Section 182(a)(2)(B) of the Act?

Yes. Anchorage's I/M program was initially implemented in 1985. Since then, Anchorage has continued to improve its performance. Improved program elements include: test equipment and procedures, quality assurance and quality control procedures, vehicle repair requirements and enforcement. The Anchorage I/M program, improvements and amendments, have been adopted through previous SIP revisions (51 FR 8203, September 15, 1986; 54 FR 31522, July 31, 1989; 60 FR 17232, April 5, 1995; 64 FR 72940, December 29, 1999, 67 FR 822, January 8, 2002).

K. Are There Controls on Stationary Sources of CO as Required by Section 172(c)(5) of the Act?

Yes. Section 172(c)(5) of the Act requires States with nonattainment areas to include in their SIPs a permit program for the construction and operation of new or modified major stationary sources in nonattainment areas. In a separate, prior action, we approved the new source review permit program for Alaska. (See 60 FR 8943, February 16, 1995.)

L. Has Anchorage Implemented an Oxygenated Fuel Program as Described in Section 187(b)(3)?

Yes. In a separate, prior action, we approved the oxygenated gasoline program for Anchorage (61 FR 24712, May 16, 1996).

III. Summary of EPA's Proposal

We are proposing approval of the following elements of the Anchorage CO Attainment Plan, as submitted on January 4, 2002:

A. Procedural requirements, under section 110(a)(1) of the Act;

B. Base year emission inventory, periodic emission inventory and commitments under sections 187(a)(1) and 187(a)(5) of the Act;

C. Attainment demonstration, under section 187(a)(7) of the Act;

D. The TCM programs under 182(d)(1) and 108(f)(1)(A) of the Act

E. Contingency measures under section 187(a)(3) of the Act.

F. RFP demonstration, under sections 171(1) and 172(c)(2) of the Act; and

H. The conformity budget under section 176(c)(2)(A) of the Act and § 93.118 of the transportation conformity rule (40 CFR part 93, subpart A).

IV. Administrative Requirements

Under Executive Order 12866 (58 FR 51735, October 4, 1993), this proposed action is not a ''significant regulatory action'' and therefore is not subject to review by the Office of Management and Budget. For this reason, this action is also not subject to Executive Order 13211, ''Actions Concerning Regulations That Significantly Affect Energy Supply, Distribution, or Use'' (66 FR 28355, May 22, 2001). This proposed action merely proposes to approve state law as meeting Federal requirements and imposes no additional requirements beyond those imposed by state law. Accordingly, the Administrator certifies that this proposed rule will not have a significant economic impact on a substantial number of small entities under the Regulatory Flexibility Act (5 U.S.C. 601 *et seq.*). Because this rule proposes to approve pre-existing requirements under state law and does not impose any additional enforceable duty beyond that required by state law, it does not contain any unfunded mandate or significantly or uniquely affect small governments, as described in the Unfunded Mandates Reform Act of 1995 (Public Law 104–4).

This proposed rule also does not have tribal implications because it will not have a substantial direct effect on one or more Indian tribes, on the relationship between the Federal Government and Indian tribes, or on the distribution of power and responsibilities between the Federal Government and Indian tribes, as specified by Executive Order 13175 (65 FR 67249, November 9, 2000). This action also does not have Federalism implications because it does not have substantial direct effects on the States, on the relationship between the national government and the States, or on the distribution of power and responsibilities among the various levels of government, as specified in Executive Order 13132 (64 FR 43255, August 10, 1999). This action merely proposes to approve a state rule implementing a Federal standard, and does not alter the relationship or the distribution of power and responsibilities established in the Clean Air Act. This proposed rule also is not subject to Executive Order 13045 ''Protection of Children from Environmental Health Risks and Safety Risks'' (62 FR 19885, April 23, 1997), because it is not economically significant.

In reviewing SIP submissions, EPA's role is to approve state choices, provided that they meet the criteria of the Clean Air Act. In this context, in the absence of a prior existing requirement for the State to use voluntary consensus standards (VCS), EPA has no authority to disapprove a SIP submission for failure to use VCS. It would thus be inconsistent with applicable law for EPA, when it reviews a SIP submission, to use VCS in place of a SIP submission that otherwise satisfies the provisions of the Clean Air Act. Thus, the requirements of section 12(d) of the National Technology Transfer and Advancement Act of 1995 (15 U.S.C. 272 note) do not apply. This proposed rule does not impose an information collection burden under the provisions of the Paperwork Reduction Act of 1995 (44 U.S.C. 3501 *et seq.*).

List of Subjects in 40 CFR Part 52

Environmental protection, Air pollution control, Carbon monoxide, Incorporation by reference, Intergovernmental relations, Reporting and recordkeeping requirements.

Dated: May 22, 2002.

Elbert Moore,

Acting Regional Administrator, Region 10.

[FR Doc. 02–13698 Filed 5–31–02; 8:45 am]

BILLING CODE 6560–50–P

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 141

[FRL–7221–8]

RIN 2040–AD61

Announcement of Preliminary Regulatory Determinations for Priority Contaminants on the Drinking Water Contaminant Candidate List

AGENCY: Environmental Protection Agency.

ACTION: Notice of preliminary regulatory determination.

SUMMARY: The Safe Drinking Water Act (SDWA), as amended in 1996, directs the Environmental Protection Agency (EPA) to publish a list of contaminants (referred to as the Contaminant Candidate List, or CCL) to assist in priority-setting efforts. SDWA also directs the Agency to select five or more contaminants from the current CCL and determine by August 2001 whether or not to regulate these contaminants with a National Primary Drinking Water Regulation (NPDWR). Today's action presents the preliminary regulatory determinations for nine contaminants and describes the supporting rationale for each.

DATES: Comments must be received on or before August 2, 2002.

ADDRESSES: Please send your comments to the W–01–14 Comments Clerk. Submit electronic comments to: *ow-docket@epa.gov.* Written comments should be mailed to: Water Docket (MC–4101), U.S. Environmental Protection Agency, 1200 Pennsylvania Avenue, NW., Washington, DC, 20460. Hand deliveries should be delivered to EPA's Water Docket at East Tower Basement (EB Room 57), Waterside Mall, 401 M Street, SW., Washington, DC, 20460. You may contact the docket at (202) 260–3027 between 9 a.m. and 3:30 p.m. Eastern Time, Monday through Friday. Comments may be submitted electronically. *See* **SUPPLEMENTARY INFORMATION** for file formats and other information about electronic filing and docket review.

FOR FURTHER INFORMATION CONTACT: For information regarding today's action, contact Karen Wirth, Office of Ground Water and Drinking Water, EPA, 1200 Pennsylvania Avenue, NW. (MC 4607M), Washington, DC 20460; telephone 202–564–5246, e-mail: *wirth.karen@epa.gov.* General information may also be obtained from the EPA Safe Drinking Water Hotline, phone: (800) 426–4791 or its local number (703) 412–3330, e-mail: *hotline.sdwa@epa.gov.* The Hotline is open Monday through Friday, excluding Federal holidays, from 9:00 a.m. to 5:30 p.m. Eastern Time.

SUPPLEMENTARY INFORMATION:

Submission of Comments

EPA will accept written or electronic comments (please do not send both). EPA prefers electronic comments. Commenters should use a separate paragraph for each issue discussed. No facsimiles (faxes) will be accepted. Commenters who want EPA to acknowledge receipt of their comments should also send a self-addressed, stamped envelope. If you submit written comments, please submit an original and three copies of your comments and enclosures (including references).

Electronic comments must be submitted in WordPerfect 8 (or an older version) or ASCII file format. Compressed or zipped files will not be accepted. You may file electronic comments on this action online at many Federal Depository Libraries.

The Agency's response-to-comments document for the final decision will address the comments received on this action. The response-to-comments document will be made available in the docket.

Obtaining Docket Materials

The docket is available for inspection from 9:00 a.m. to 4:00 p.m. Eastern Time, Monday through Friday, excluding legal holidays, at the Water Docket, East Tower Basement (EB Room 57), Waterside Mall, USEPA, 401 M Street, SW; Washington, D.C. For access to docket (Docket Number W–01–03) materials, please call (202) 260–3027 between 9:00 a.m. and 3:30 p.m., Eastern Time, Monday through Friday, to schedule an appointment.

Abbreviations and Acronyms

<—Less than
>—Greater than
μ—Microgram, one-millionth of a gram
μg/L—Micrograms per liter
AIDS—Acquired immunodeficiency syndrome
ATSDR—Agency for Toxic Substances and Disease Registry
AWWA—American Water Works Association
AWWARF—American Water Works Association Research Foundation
BW—Body weight for an adult, assumed to be 70 kilogram (kg)
CASRN—Chemical Abstract Services Registry Number
CCL—Contaminant Candidate List
CDC—Centers for Disease Control and Prevention
CFR—Code of Federal Regulations
CMR—Chemical Monitoring Reform
DASH—Dietary Approaches to Stop Hypertension
DW—Drinking water consumption, assumed to be 2 L/day
EPA—U.S. Environmental Protection Agency
FR—Federal Register
g/day—Grams of contaminant per day g/L—Grams of the contaminant per liter
G6PD—Glucose-6-phosphate dehydrogenase
GAE—Granulomatous amoebic encephalitis
HIV—Human immunodeficiency virus
HRL—Health reference level
IOC—Inorganic compound
IRIS—Integrated Risk Information System
kg—Kilogram
L—Liter
LD_{50}—Lethal Dose 50; the dose at which 50% of the test animals died; a calculated value (LD_{50})
LOAEL—Lowest-observed-adverse-effect level
MCLG—Maximum contaminant level goal
mg—Milligram, one-thousandth of a gram
mg/kg—Milligrams of contaminant per kilogram body weight
mg/L—Milligrams of the contaminant per liter
mg/m^3—Milligrams per cubic meter
NAS—National Academy of Sciences
NDWAC—National Drinking Water Advisory Council
NIH—National Institute of Health
NIRS—National Inorganic and Radionuclide Survey
NOAEL—No-observed-adverse-effect level
NPDWR—National Primary Drinking Water Regulation
NRC—National Research Council
NTP—National Toxicology Program
OW—Office of Water
PWS—Public Water System
RfD—Reference dose
RSC—Relative source contribution
SDWA—Safe Drinking Water Act
SDWIS/FED—Safe Drinking Water Information System, Federal version
SOC—Synthetic organic compound
TRI—Toxic Release Inventory
UCM—Unregulated Contaminant Monitoring
UF—Uncertainty factor
URIS—Unregulated Contaminant Information System
U.S.—United States of America
USGS—United States Geological Survey
VOC—Volatile organic compound
WHO—World Health Organization

Table of Contents

I. Background and Summary of Today's Action

A. What Is the Purpose of Today's Action?

Section 1412(b)(1)(A) of the SDWA, as amended in 1996, directs EPA to make determinations by August 2001 of whether or not to regulate at least five contaminants from EPA's Contaminant Candidate List of unregulated contaminants. For those contaminants that EPA determines to regulate, EPA has 24 months to propose Maximum Contaminant Level Goals (MCLGs) and National Primary Drinking Water Regulations (NPDWRs) and has 18 months following proposal to publish final MCLGs and promulgate NPDWRs. Today's action presents EPA's preliminary regulatory determinations for nine CCL contaminants together with the determination process, rationale, and supporting technical information for each.

The contaminants discussed in today's action include: Three inorganic compounds (IOCs) (manganese, sodium, and sulfate); three synthetic organic compounds (SOCs) (aldrin, dieldrin, and metribuzin); two volatile organic compounds (VOCs) (hexachlorobutadiene and naphthalene); and one microbial contaminant, *Acanthamoeba.*

B. What Is EPA's Preliminary Determination, and What Happens Next?

EPA's preliminary determination is that no regulatory action is appropriate for the contaminants *Acanthamoeba,* aldrin, dieldrin, hexachlorobutadiene, manganese, metribuzin, naphthalene, sodium, and sulfate.

EPA will make final determinations on these contaminants after a 60-day comment period and a public meeting. The public meeting will be held in the spring of 2002 in the Washington, D.C. area, to provide an information exchange with stakeholders on issues related to today's action. Further information about this meeting will be given in a future **Federal Register** Notice and will be available from the Drinking Water Hotline at 1–800–426–4791.

EPA is making preliminary regulatory determinations on CCL contaminants that have sufficient information to support a regulatory determination at this time. The Agency continues to conduct research and/or to collect occurrence information on the remaining CCL contaminants. EPA has been aggressively conducting research to fill identified data gaps and recognizes that stakeholders may have a particular interest about the planned timing for future regulatory determinations for other contaminants on the CCL. The Agency is not precluded from taking action when information becomes available and will not necessarily wait until the end of the next regulatory determination cycle before making other regulatory determinations.

C. What Is the CCL?

SDWA, as amended in 1996, directs EPA to publish a list of contaminants to assist in priority setting for the Agency's drinking water program. This list is called the Contaminant Candidate List or CCL. Section 1412(b)(1)(B) states that the EPA Administrator shall publish a list of contaminants which '' * * * are not subject to any proposed or promulgated national primary drinking water regulation, which are known or anticipated to occur in public water systems, and which may require regulation under this title [SDWA].''

The CCL was developed with considerable input from the scientific community and stakeholders. A draft CCL requesting public comment was published on October 6, 1997 (62 FR 52193). The first CCL was published on March 2, 1998 (63 FR 10273). The SDWA requires that a new CCL will be published every five years thereafter (e.g., February 2003). The 1998 CCL contained 60 contaminants, including 50 chemicals or chemical groups and 10 microbiological contaminants or microbial groups. Many of these contaminants lacked some of the information necessary to support a regulatory determination and were identified as having data needs. CCL contaminants were divided into categories to represent next steps and data needs associated with each contaminant. The categories were: (1) Regulatory determination priorities (i.e., no data needs); (2) health effects research priorities; (3) treatment research priorities; (4) analytical methods research priorities; and (5) occurrence priorities. Twenty contaminants were classified as regulatory determination priorities on the 1998 CCL because EPA believed in 1998 that there were sufficient data to evaluate both exposure and risk to public health, and to support a determination of whether or not to proceed to promulgation of a NPDWR.

Since the March 1998 CCL, EPA found that there was insufficient information to support a regulatory determination for 12 of the 20 priority contaminants (see Table 1). In addition, sodium was added to the list of eight remaining regulatory determination priorities primarily as a means of reassessing the current guidance level. Thus, EPA is now presenting preliminary regulatory determinations for nine priority contaminants that have sufficient information to support a regulatory determination at this time: *Acanthamoeba,* aldrin, dieldrin, hexachlorobutadiene, manganese, metribuzin, naphthalene, sodium, and sulfate.

TABLE 1.—1998 PRIORITY CONTAMINANTS WHICH ARE NOW JUDGED TO LACK INFORMATION SUFFICIENT TO SUPPORT A REGULATORY DETERMINATION

Chemical contaminant	Research needs
Boron	Treatment technology and finalization of a health risk assessment (reference dose—RfD).
Bromobenzene	Non-cancer health effects data including subchronic toxicity tests, immunotoxicity, neurotoxicity, and structure-activity analyses. Further work to identify an appropriate treatment technology.
1,1-dichloroethane	Health effects data—cancer, reproductive, developmental, and pharmacokinetic studies. Further work to identify an appropriate treatment technology.
1,3-dichloropropene	Occurrence information using revised sample preservation method.
2,2-dichloropropane	Health effects data—mutagenicity and carcinogenicity screening tests, and structure-activity analysis. Further work to identify an appropriate treatment technology.
p-isopropyltoluene	Health effects data—subchronic, chronic, cancer, neurodevelopmental, reproductive, and developmental. Evaluate related findings on cumene and other alkylbenzenes.
Metolachlor, s-metolachlor, and metolachlor degradation products: ethane sulfonic acid, and oxanilic acid.	Analysis of health effects of metolachlor degradation degradates and occurrence information.

TABLE 1.—1998 PRIORITY CONTAMINANTS WHICH ARE NOW JUDGED TO LACK INFORMATION SUFFICIENT TO SUPPORT A REGULATORY DETERMINATION—Continued

Chemical contaminant	Research needs
Organotins	Non-cancer health effects data—developmental and reproductive toxicity, neurotoxicity, and immunotoxicity. Pharmacokinetic studies and structure-activity analysis recommended. Further work needed to identify appropriateness of treatment technology and analytical methods. Additional occurrence information.
1,1,2,2-tetrachloroethane	Non-cancer health effects data—developmental and reproductive toxicity, neurotoxicity, and immunotoxicity. Carcinogenicity studies. Further work to identify an appropriate treatment technology.
Triazines & degradation products	Analytical methods data and occurrence information. Finalize list of degradates to evaluate.
1,2,4-trimethylbenzene	Health effects data—neurotoxicity screening tests. Further work to identify an appropriate treatment technology.
Vanadium	Health effects data on neurotoxicity and toxicokinetics of inhalation and oral routes. Further work to identify an appropriate treatment technology.

The Agency continues to conduct research and/or to collect occurrence information for all other contaminants on the CCL. The overall research approach is closely aligned with the 1983 National Research Council (NRC) risk assessment/risk management paradigm, which involves a systematic evaluation of data on health effects, exposure, and risk management options (NRC 1983) and is detailed in the Draft CCL Research Plan (USEPA 2001a). The plan was drafted in close consultation with outside stakeholders including the American Water Works Association (AWWA), the AWWA Research Foundation (AWWARF), other governmental agencies, universities, as well as other public and private sector groups. EPA and the AWWARF jointly sponsored a conference, in late September of 1999, to review all aspects of the proposed CCL Research Plan and to make suggestions for future research activities. The three-day meeting was attended by representatives from the water utility industry, State and Federal health and regulatory agencies, professional associations, academia, and public interest groups. The recommendations and results from this meeting have been incorporated into the draft research plan (USEPA 2001a).

EPA's Science Advisory Board reviewed the research plan in August of 2000 and again in June of 2001. The plan is targeted for completion in 2002. It will be available to the public at that time and will be posted on EPA's web site. Implementation of the research plan will require the coordinated efforts of both governmental and non-governmental entities. EPA intends to make all aspects of CCL research planning, implementation, and communication a collaborative process.

D. Does Today's Action Apply to My Public Water System?

Today's action itself does not impose any requirements on anyone. Instead, it notifies interested parties of EPA's preliminary determination not to regulate nine CCL contaminants.

II. What Criteria and Approach Did EPA Use To Make the Preliminary Regulatory Determinations?

Section 1412(b)(1)(A) of SDWA directs that EPA shall publish a MCLG and promulgate a NPDWR for a contaminant if the Administrator determines that (i) the contaminant may have adverse effects on the health of persons; (ii) the contaminant is known to occur, or there is substantial likelihood that the contaminant will occur, in public water systems with a frequency, and at levels of public health concern; and (iii) in the sole judgment of the Administrator, regulation of such contaminant presents a meaningful opportunity for health risk reduction for persons served by public water systems.

This section presents the decision-making framework for selecting contaminants from a CCL for future action. It also discusses criteria that EPA used for making the preliminary regulatory determinations announced in today's action.

The process of making preliminary regulatory determinations benefitted from substantial expert input and reflects major recommendations and themes suggested by different groups including stakeholders, the NRC, and the National Drinking Water Advisory Council (NDWAC).

A. Recommended Criteria and Approaches

The Agency held a stakeholders meeting on November 16–17, 1999. The purpose of the meeting was to provide an update and to seek comment from stakeholders on the following: The regulatory determination process, specific factors to consider when making regulatory determinations, the draft CCL research plan, and the process for developing future CCLs. Participants at the meeting included representatives of public water utilities, State drinking water programs, public health and environmental groups, local government, the private sector, EPA and other Federal agencies. EPA intends to hold an additional stakeholders meeting in the spring of 2002 to solicit input on the preliminary regulatory determinations that are outlined in today's action.

1. The National Research Council's Recommended Approach

EPA asked the NRC for assistance in developing a scientifically sound approach for deciding whether or not to regulate contaminants on the current and future CCLs. In response to the request, the NRC's Committee on Drinking Water Contaminants published the report, Setting Priorities for Drinking Water Contaminants (NRC 1999). This report evaluated various existing schemes for setting priorities among environmental contaminants and recommended a framework to guide EPA in deciding which contaminants on the CCL to regulate.

The recommended framework applies to both chemical and microbial contaminants and would proceed as follows: (1) Gather and analyze health effects, exposure, treatment, and analytical methods data for each contaminant; (2) conduct a preliminary risk assessment for each contaminant based on the available data; and (3) issue a decision document for each contaminant describing the outcome of the preliminary risk assessment. The NRC notes that in using this decision framework, EPA should keep in mind the importance of involving all interested parties, recognize that the

process requires considerable expert judgment to address uncertainties from gaps in information about exposure potential and/or health effects, evaluate the many different effects that contaminants can cause, and interpret available data in terms of statutory requirements.

2. The National Drinking Water Advisory Council's Recommended Criteria and Approach

One of the formal means by which EPA works with its stakeholders is through the NDWAC. The Council comprises members from the general public, State and local agencies, and private groups concerned with safe drinking water. It advises the EPA Administrator on key aspects of the Agency's drinking water program. The NDWAC provided specific recommendations to EPA on a protocol to assist the Agency in its efforts to make regulatory determinations for current and future CCL contaminants. These recommendations were the result of a working group formed by the NDWAC charged with developing regulatory determination criteria and protocols. Separate but similar protocols were developed for chemical and microbial contaminants. These protocols are intended to provide a consistent approach to evaluating contaminants for regulatory determinations.

The NDWAC protocol uses the three statutory requirements of SDWA section 1412(b)(1)(A)(i)–(iii) (specified in section II of today's action) as the foundation for guiding EPA in making regulatory determination decisions. For each statutory requirement, evaluation criteria were developed and are summarized later in this section for the chemical contaminants only.

To address whether a contaminant may have adverse effects on the health of persons (a statutory requirement in section 1412(b)(1)(A)(i)), the NDWAC recommended that EPA characterize the health risk and estimate a health reference level for evaluating the occurrence data for each contaminant.

To evaluate the known or likely occurrence of a contaminant, (required by statute 1412(b)(1)(A)(ii)), the NDWAC recommended that EPA consider: (1) The actual and estimated national percent of public water systems (PWSs) reporting detections above half the health reference level; (2) the actual and estimated national percent of PWSs with detections above the health reference level; and (3) the geographic distribution of the contaminant.

To address whether regulation of a contaminant presents a meaningful opportunity for health risk reduction (a statutory requirement in section 1412(b)(1)(A)(iii)), the NDWAC recommended that EPA consider estimating the national population exposed above half the health reference level and the national population exposed above the health reference level.

B. EPA's Criteria and Approach

EPA developed its evaluation approach based on the recommendations from NRC and NDWAC. For the nine contaminants addressed in today's action, EPA evaluated the following: the adequacy of current analytical and treatment methods; the best available peer reviewed data on health effects; and approximately seven million analytical data points on contaminant occurrence. For those contaminants with adequate monitoring methods, as well as health effects and occurrence data, EPA employed an approach to assist in making preliminary regulatory determinations that follows the themes recommended by the NRC and NDWAC to satisfy the three SDWA requirements under section 1412(b)(1)(A)(i)–(iii). The process was independent of many of the more detailed and comprehensive risk management factors that will influence the ultimate regulatory decision making process. Thus, a decision to regulate is the beginning of the Agency regulatory development process, not the end.

Specifically, as described in section III.A. of today's action, EPA characterized the human health effects that may result from exposure to a contaminant found in drinking water. Based on this characterization, the Agency estimated either a health reference level (HRL) or a benchmark value for each contaminant.

As described in section III.B., for each contaminant EPA estimated the number of PWSs with detections greater than one-half the HRL (>½ HRL) and greater than the HRL (>HRL); the population served at these benchmark values; and the geographic distribution using a large number of State occurrence data (approximately seven million analytical points) that broadly reflect national coverage. If a benchmark value was used instead of a HRL, the same process was carried out with ½ the benchmark value and the full benchmark value. Use and environmental release information, as well as ambient water quality data were used to augment the State data and to evaluate of the likelihood of contaminant occurrence.

The findings from these evaluations were used to determine if there was adequate information to evaluate the three SDWA statutory requirements and to make a preliminary determination of whether to regulate a contaminant.

EPA prepared Regulatory Determination Support Documents that are available for review and comment in the EPA Water Docket. These documents present summary information and data on a contaminant's physical and chemical properties, uses and environmental release, environmental fate, health effects, occurrence, and exposure. The documents discuss in detail the rationale used to support the preliminary regulatory determination.

As a parallel effort during the comment period, EPA intends to have the Science Advisory Board review the analysis, the approach used for making regulatory determinations, and the preliminary regulatory determinations.

III. What Analysis Did EPA Use To Support the Preliminary Regulatory Determinations?

Sections III.A. and B. of today's action outline the evaluation steps EPA used to support the preliminary determinations.

A. Evaluation of Adverse Health Effects

The purpose of this section is to discuss the health effects information evaluated, the approach used to derive a HRL for evaluating the occurrence data, and to briefly describe the support documents that provide detailed information on adverse health effects and their dose response.

As discussed previously, section 1412(b)(1)(A)(i) directs EPA to determine whether each candidate contaminant has an adverse effect on public health. The potential for adverse health effects for each contaminant are presented in section IV.B. of today's action.

For those contaminants considered to be human carcinogens or likely to be human carcinogens, EPA evaluated data on the mode of action of the chemical to determine the method of low dose extrapolation. When this analysis indicates that a low dose extrapolation is needed and when data on the mode of action are lacking, EPA uses a default low dose linear extrapolation to calculate risk specific doses. These are estimated oral exposures associated with risk levels that range from one cancer in ten thousand (10^{-4}) to one cancer in a million (10^{-6}). These risk specific doses are combined with drinking water consumption data to estimate drinking water concentrations corresponding to this risk range, which are then used as HRLs for these contaminants. Of the nine contaminants discussed in today's action, only aldrin,

dieldrin, and hexachlorobutadiene had data to consider them to be likely or possible human carcinogens. They are also the only contaminants for which linear low dose extrapolation was done. The Agency selected the 10^{-6} risk specific concentration as the HRL for these three contaminants.

For those chemicals not considered to be carcinogenic to humans, EPA generally calculates a reference dose (RfD). An RfD is an estimate of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. It can be derived from a ''no-observed-adverse-effect level (NOAEL),'' ''lowest-observed-adverse-effect level (LOAEL),'' or benchmark dose, with uncertainty factors generally applied to reflect limitations of the data used.

The Agency uses an uncertainty factor (UF) to address uncertainty resulting from incompleteness of the toxicological database. Generally, the UFs are factors ranging from 3 to 10-fold that are multiplied together and used in deriving the RfD from experimental data. UFs are intended to account for: (1) The variation in sensitivity among the members of the human population (*i.e.,* intraspecies variability); (2) the uncertainty in extrapolating animal data to humans (*i.e.,* interspecies variability); (3) the uncertainty in extrapolating from data obtained in a study with less-than-lifetime exposure to lifetime exposure (*i.e.,* extrapolating from subchronic to chronic exposure); (4) the uncertainty in extrapolating from a LOAEL rather than from a NOAEL; and (5) the uncertainty associated with extrapolation from animal data when the data base is incomplete.

For manganese, metribuzin and naphthalene EPA derived the HRLs using the RfD approach as follows: HRL = (RfD × BW)/DW × RSC.

Where:

RfD = Reference Dose
BW = Body weight for an adult, assumed to be 70 kilograms (kg)
DW = Drinking water consumption, assumed to be 2 L/day (90th percentile)
RSC = The relative source contribution, or the level of exposure believed to result from drinking water when compared to other sources (*e.g.,* air). The RSC is assumed to be 20% unless noted otherwise.

The HRL for sulfate was not established using the RfD approach. The available data do not provide the necessary dose-response information to support the derivation of an RfD for sulfate. However, 500 milligram/liter (mg/L) is a concentration at which adverse effects did not occur in any of the reported studies. This value was used as the HRL. Further details on the sulfate HRL are included in section IV.B.8.

In the case of sodium, the benchmark value used to evaluate the occurrence data is not designated as an HRL because of the lack of suitable dose-response data and the considerable controversy regarding the role of sodium in the etiology of hypertension. The benchmark value for sodium of 120 mg/L was derived from the recommended daily dietary intake of 2.4 grams/day (g/day). Additional information regarding the sodium benchmark value is included in section IV.B.7.

Monitoring data are not available from PWSs for Acanthamoeba. Accordingly, an HRL was not established.

EPA has prepared Health Effects Support Documents for each contaminant that are available for review and comment at the EPA Water Docket. These documents address the following: exposure from drinking water and other media; toxicokinetics; hazard identification; dose-response assessment; and an overall characterization of risk from drinking water. The Acanthamoeba health effects support document addresses the details of the following: occurrence in water and soil, exposure, populations at risk, association with contact lenses and poor hygiene, symptoms of keratitis eye infections, incidence, diagnosis and treatment of granulomas amoebic encephalitis (GAE), risk factors and prevention.

EPA used the best available peer reviewed data and analyses in evaluating adverse health effects. Health effects information is available for aldrin, dieldrin, hexachlorobutadiene, manganese, metribuzin, and naphthalene in the Integrated Risk Information System (IRIS) database. IRIS is an electronic EPA data base (*www.epa.gov/iris/index.htm*) containing peer reviewed information on human health effects that may result from exposure to various chemicals in the environment. These chemical files contain descriptive and quantitative information on hazard identification and dose response, RfDs for chronic noncarcinogenic health effects; as well as slope factors and unit risks for carcinogenic effects. In all cases, the IRIS information was supplemented with more recent data from peer reviewed publications. In cases where the new data impacted the IRIS evaluation, the Office of Water (OW) Health Effects Support Documents are being independently peer reviewed.

B. Evaluation of National Occurrence and Exposure

As noted previously in today's action, section 1412(b)(1)(A)(ii) directs EPA to determine whether each candidate for regulation is known to occur, or is substantially likely to occur, in PWSs with a frequency, and at levels, of public health concern. A substantial amount of State finished drinking water occurrence data for unregulated contaminants are provided under the Agency's Unregulated Contaminant Monitoring (UCM) program. These data form part of the Agency's basis for its estimates of national occurrence. The UCM program was initiated in 1987 to fulfill a SDWA requirement of the 1986 amendments that PWSs monitor for specified ''unregulated'' contaminants to gather scientific information on their occurrence for future regulatory decision making purposes. An additional EPA study conducted in the mid-1980s, the National Inorganic and Radionuclide Survey (NIRS), provides a statistically representative sample of the national occurrence of many regulated and unregulated inorganic contaminants in ground water CWSs.

EPA prepared a report entitled Analysis of National Occurrence of the 1998 Contaminant Candidate List (CCL) Regulatory Determination Priority Contaminants in Public Water Systems (USEPA 2001b) that provides detailed reviews of the State monitoring data for each CCL regulatory determination priority contaminant. This report includes detailed information regarding how the data were assessed for quality, completeness, and representativeness, how the data were aggregated into national cross-sections, and presents summary occurrence findings. In EPA's contaminant-specific Regulatory Determination Support Documents described earlier (*see* section II.B. of today's action), additional information is included that presents an analysis of the occurrence data for special trends as well as populations served by PWSs with detections. EPA also reviewed information on the use, environmental release, and ambient occurrence of each contaminant to augment the State drinking water data (UCM and supplemental State monitoring data) and aid in the evaluation of occurrence. Summary descriptions of these data and analyses for each regulatory determination priority contaminant are presented in section IV. of today's action.

Section III.B. describes how the drinking water data sets were used to evaluate the occurrence of the regulatory determination priority

contaminants, including data sources, data quality, and analytical methods. Also included are summary descriptions of the ambient occurrence data, as well as the use and environmental release information that were considered.

The primary drinking water occurrence data for the regulatory determination priority contaminants are from the UCM program and the NIRS (*see* Table 2). The sources of these data, their quality, national aggregation, and the approach used to estimate a given contaminant's occurrence are discussed in the following sections.

TABLE 2.—PRIMARY DRINKING WATER OCCURRENCE DATA SOURCES USED IN THE REGULATORY DETERMINATION PROCESS

Contaminant	UCM round 1 cross section	UCM round 2 cross section	NIRS
Aldrin		X	
Dieldrin		X	
Hexachlorobutadiene	X	X	
Manganese			X
Metribuzin		X	
Naphthalene	X	X	
Sodium			X
Sulfate		X	

1. The Unregulated Contaminant Monitoring Program

Occurrence data for most of the regulatory determination priority contaminants (aldrin, dieldrin, hexachlorobutadiene, metribuzin, naphthalene, and sulfate) are from the monitoring results of the UCM program. This program was implemented in two phases, or ''rounds.'' The first round of UCM monitoring began in 1987, and the second in 1993. EPA reviewed and edited the data for the purposes of this analysis.

a. UCM Rounds 1 and 2. The 1987 UCM (52 FR 25720, July 8, 1987) contaminants include 34 VOCs including the regulatory determination priority contaminants hexachlorobutadiene and naphthalene. The UCM (1987) contaminants were first monitored during the period 1988–1992. This period is referred to as ''Round 1'' monitoring. The Round 1 data were put into a database called the Unregulated Contaminant Information System (URIS).

The 1993 UCM contaminants included 34 VOCs (including naphthalene and hexachlorobutadiene), 13 SOCs, and sulfate (52 FR 25720, July 8, 1987). Aldrin, dieldrin, and metribuzin were among the 13 SOCs monitored. Monitoring for the UCM (1993) contaminants began in 1993 and continued through 1999. This is referred to as ''Round 2'' monitoring. The UCM (1987) contaminants (the 34 VOCs monitored in Round 1) were also included in the Round 2 monitoring. As with other monitoring data, PWSs reported these results to the States. During the past several years, States have submitted Round 2 data to EPA's Safe Drinking Water Information System (Federal version; SDWIS/FED) database.

The details of the actual individual monitoring periods are complex. The timing and procedures for required monitoring are outlined in the report entitled Analysis of National Occurrence of the 1998 Contaminant Candidate List (CCL) Regulatory Determination Priority Contaminants in Public Water Systems (USEPA 2001b). Round 1 and Round 2 data were analyzed separately because they represent different time periods, include different States (only eight States are represented in the data from both rounds), and only two CCL priority contaminants are common to both rounds.

b. Development of occurrence data cross-sections. The Round 1 database contains contaminant occurrence data from 38 States, Washington, D.C. and the United States (U.S.) Virgin Islands. The Round 2 database contains data from 34 States and Tribes. Therefore, neither database contains data from all States. Also, data from some of the States in the databases are incomplete. As a result, unadjusted national results could be skewed to low-occurrence or high-occurrence settings (*e.g.,* some States only reported detections). To address this lack of representativeness, national cross-sections from the Round 1 and Round 2 State data were established using a similar approach developed for the EPA report entitled A Review of Contaminant Occurrence in Public Water Systems (USEPA 1999a). The cross-section approach in this report was developed to support occurrence analyses for EPA's Chemical Monitoring Reform (CMR) evaluation, and was supported by scientific peer reviewers and stakeholders.

For SOCs and VOCs on the CCL, two national cross-sections were developed from the UCM data. The Round 1 national cross-section consists of data from 24 States with approximately 3.3 million analytical data points from approximately 22,000 unique PWSs. The Round 2 national cross-section consists of data from 20 States with approximately 3.7 million analytical data points from slightly more than 27,000 unique PWSs. The actual number of systems and records varies for each contaminant according to the number of reported records for a particular contaminant. The support document, Analysis of National Occurrence of the 1998 Contaminant Candidate List (CCL) Regulatory Determination Priority Contaminants in Public Water Systems (USEPA 2001b), provides a summary description of how the national cross-sections for the Round 1 and Round 2 data sets were developed.

All samples in the Round 1 and Round 2 State data sets were taken from finished drinking water, representing the product delivered to the public. Data were limited to samples with confirmed water source and sampling type information. Only routine monitoring samples were used; ''special'' samples, ''investigation'' samples (investigating a contaminant problem, that would likely bias the results), and samples of unknown type were excluded from the data set. Various quality control and review checks were made of the results, including follow-up questions to the States providing the data to clarify potential reporting inconsistencies, records with invalid codes, or use of analytical units. The State data sets were then compiled into single database in a unified format.

While the national cross-sections of States provides a good picture of

national occurrence, there are limitations in the data in that the original monitoring data were not collected by a statistical random sample. Since the data sets do not include the entire U.S., they cannot capture all local variations in contaminant occurrence. However, EPA believes the cross-sections do provide a reasonable estimate of the overall distribution, including the central tendency, of contaminant occurrence across the U.S.

c. Occurrence analysis. The summary descriptive statistics presented in section IV of today's action for each contaminant generally include the following: The number of samples, the total number of systems, the percent of samples with at least one observed detection that has a concentration above the HRL (the HRL is an estimated health effect level used for the purposes of this analysis), and the 99th percentile concentration and median concentration of the observed detections. As described in section III. A, in the case of sodium, the benchmark was used to evaluate the occurrence data rather than a designated HRL. The 99th percentile concentration is commonly used to characterize upper bound data to avoid maximum values that are often problematic outlier observations. Because most of the regulatory determination priority contaminants have very low occurrence (<1% of samples with detections), these statistics are presented for the detections only. One exception is sulfate, for which the median and 99th percentile concentrations are presented for all samples (i.e., the entire universe of samples) because of its relatively high occurrence. The percentages of PWSs, and population served, having at least one detected concentration above >½HRL and >HRL are also presented. As noted, the occurrence values and summary statistics presented are the actual data from the aggregated State cross-sections. EPA considered this the most straightforward and accurate way to present the data that were available for the determination process. EPA extrapolated values for national occurrence (based on the actual cross-section data). However, because the State data used for the cross-section are not a statistical sample, national extrapolations can be problematic, especially for contaminants with such low occurrence as was the case for many of these CCL contaminants. National extrapolations based on peak concentrations, such as the percent of systems with at least one observed concentration above the HRL, may also be misleading, since peak concentrations are highly variable from one location to another. For these reasons, the nationally extrapolated estimates of occurrence and exposure are not presented in today's action and are not used as the basis for the preliminary regulatory determinations. However, to provide additional perspective, the nationally extrapolated occurrence and exposure values are presented in the support documents and are available for review and comment.

At this phase of consideration, more involved statistical modeling of the data was not performed. The presentation of the actual results of the cross-section analysis provides a straight-forward presentation and demonstrates the integrity of the data available for stakeholder review. As noted, however, the cross-section analysis should provide a reasonable estimate of the central tendency of occurrence for these contaminants because of the large number of States included with complete monitoring data sets for the intended purposes (Round 1 consists of approximately 3.3 million analytical data points from 22,000 PWSs in 24 States; and Round 2 consists of approximately 3.7 million analytical data points from 27,000 PWSs in 20 States) that are representative of the range of pollution potential indicators and spatial/hydrogeologic diversity in the nation. EPA believes that the current approach is appropriate and protective but is seeking comments on the necessity of applying a further, more rigorous statistical modeling effort that could be conducted on the cross-section data. This additional effort could use probabilistic modeling to estimate the distribution of mean contaminant concentrations in PWSs in the U.S. Because this approach is based on estimating mean concentrations, instead of peaks as in the current approach, the results would be more statistically robust and more suitable to national extrapolation. This approach allows for better quantification of estimation error. It would also allow an assessment of systems with mean, rather than peak concentrations which exceed the HRL and ½ the HRL, which may be more appropriate for chronic health effects. However, EPA does not believe that such an undertaking would fundamentally change the conclusions drawn from the data for these nine contaminants or the resulting preliminary regulatory determinations. The approach is currently being peer reviewed for use by the Agency to review and revise, if necessary, existing NPDWRs (*i.e.,* the ''six-year review''). The model is described in the report entitled, Occurrence in Estimation Methodology and Occurrence Findings Report for Six-Year Regulatory Review (USEPA 2001c).

d. Comparison to the Six-Year Review. EPA is using a similar methodology for occurrence analysis for the six-year review of existing NPDWRs. For this effort, EPA compiled a separate and different contaminant occurrence database and constructed a cross-section that consists of 13 million compliance monitoring results from approximately 41,000 PWSs in 16 States. Also, as for the CCL, contaminant occurrence is reported in terms of the number of PWSs having at least one sample concentration above the levels of regulatory interest. For the six-year review effort, however, the Agency has also performed the more detailed statistical modeling as previously described, in order to estimate, for a certain number of the regulated contaminants, the number of PWSs with mean concentrations over time that exceed the levels of interest. This effort is driven by the underlying nature of the data and the type of data analysis it can support (i.e., the data base has a significant number of detections) as contrasted with the CCL data set.

2. National Inorganic and Radionuclide Survey and Supplementary IOC Occurrence Data

The NIRS database includes 36 IOCs (including 10 now-regulated IOCs), two regulated radionuclides, and four unregulated radionuclides. Manganese and sodium were two of the IOCs monitored. The NIRS provides contaminant occurrence data from 989 community water systems served by ground water. The NIRS does not include surface water systems. The selection of CWSs included in NIRS was designed so that the contaminant occurrence results are statistically representative of national occurrence at CWSs using ground water sources (the survey was focused on ground water systems, in part, because ground water has a higher occurrence and concentrations of naturally occurring IOCs). Most of the NIRS data are from smaller systems (based on population served) and each of the 989 statistically randomly selected CWSs was sampled at a single time between 1984 and 1986.

The NIRS data were collected from ground water CWSs in 49 States. Data were not available for the State of Hawaii. NIRS data were designed to be stratified based on system size (population served by the system), and uniform analytical detection limits were employed.

The summary descriptive statistics presented in section IV of today's action

for manganese and sodium are derived from NIRS data analyses and generally include the total number of systems and samples, the percent systems with detections, the 99th percentile concentration of all samples, the 99th percentile concentration of samples with detections, and the median concentration of samples with detections. The percentages of PWSs, and population served, with detections >½ HRL and >HRL are also presented. Because the NIRS data were collected in a statistically designed sample survey, these summary statistics are representative of national occurrence in ground water PWSs. The actual values for the NIRS analyses are also reported, similar to the treatment for the cross-section data.

One limitation of the NIRS study is a lack of occurrence data for surface water systems. To provide perspective on the occurrence of the CCL determination priority contaminants in surface water systems relative to ground water systems, additional State monitoring data were reviewed. These State ground water and surface water PWS occurrence data were available to EPA from an independent review of the occurrence of regulated contaminants in PWSs and published in the report A Review of Contaminant Occurrence in Public Water Systems (USEPA 1999a). The review contains data from Alabama, California, Illinois, New Jersey, and Oregon for manganese (approximately 38,700 samples from 5,500 systems total) and sodium (approximately 36,000 samples from 6,500 PWSs total). The data were subject to the same quality review and editing process as the Round 1 and Round 2 data described previously. The data analysis, and presentation of results, were similar as well. However, because State surface water and ground water data were available from only a few States for manganese and sodium, the State data were analyzed individually. National cross-sections could not be developed for them.

3. Supplemental Data

EPA collected supplemental data for each contaminant, including use and environmental release information (*e.g.,* EPA's Toxic Release Inventory, academic and private sector publications) and ambient water quality data (*i.e.,* source water existing in surface waters and aquifers before extraction and treatment as drinking water), to augment the drinking water data and better characterize the contaminant's presence in the environment. Data from the U.S. Geological Survey's National Water Quality Assessment program, the most comprehensive and nationally consistent data describing ambient water quality in the U.S. were included when available. A detailed discussion of the supplemental data collected for each contaminant can be found in the respective Regulatory Determination Support Document.

IV. Preliminary Regulatory Determinations

A. Summary

The Agency is soliciting public comment on whether a preliminary determination that nine contaminants do not meet all three SDWA requirements is appropriate and thus no NPDWRs should be considered for those nine contaminants, identified by chemical abstract service registry number (CASRN) in Table 3.

TABLE 3.—PRELIMINARY REGULATORY DETERMINATIONS

Contaminant	CASRN	Preliminary Regulatory Determination
Acanthamoeba.	N/A	Do not regulate.
Aldrin	309–00–2	Do not regulate.
Dieldrin	60–57–1 ...	Do not regulate.
Hexachlorobutadiene.	87–68–3 ...	Do not regulate.
Manganese	7439–96–5	Do not regulate.
Metribuzin ...	21087–64–9.	Do not regulate.
Naphthalene	91–20–3 ...	Do not regulate.
Sodium	7440–23–5	Do not regulate.
Sulfate	14808–79–8.	Do not regulate.

As previously stated, EPA is only making regulatory determinations on CCL contaminants that have sufficient information to support a regulatory determination at this time. The Agency continues to conduct research and/or to collect occurrence information on the remaining CCL contaminants. EPA has been aggressively conducting research to fill identified data gaps and recognizes that stakeholders may have a particular interest in the timing of future regulatory determinations for other contaminants on the CCL. Stakeholders may be concerned that regulatory determinations for such contaminants should not necessarily wait until the end of the next regulatory determination cycle.

In this regard, it is important to recognize that the Agency is not precluded from monitoring, conducting research, developing guidance, or regulating contaminants not included on the CCL to address an urgent threat to public health (see SDWA section 1412(b)(1)(D)); or taking action on CCL contaminants when information becomes available. As previously mentioned, the Agency continues to conduct research and/or to collect occurrence information for contaminants on the CCL (except the nine mentioned in today's action) and may proceed with regulatory determination prior to the end of the next regulatory determination cycle. EPA solicits comment on which of the remaining CCL contaminants stakeholders believe should have the highest priority for future regulatory determinations and their reasons in support of such comments.

The following sections summarize the data and rationale used by the Agency to reach these preliminary decisions.

B. Contaminant Profiles

This section discusses the following background information for each regulatory priority contaminant: The available human and toxicological data; how the drinking water data sets were used to evaluate occurrence in PWSs; and the population served at levels of public health concern. The findings from these evaluations were used to determine if the three SDWA statutory requirements were satisfied for each contaminant, and in making preliminary determinations whether to regulate the contaminants. Table 4 presents summary statistics describing the occurrence of the regulatory determination priority contaminants. Monitoring data are not available from PWSs for Acanthamoeba, therefore, summary statistics are not represented in Table 4. In reviewing these statistics it is important to keep in mind that they are based on peak rather than mean concentrations at the sampled systems. In general, the percentages of systems with mean concentrations exceeding the HRL and ½ the HRL would be lower.

TABLE 4.—OCCURRENCE SUMMARY FOR THE CHEMICAL REGULATORY DETERMINATION PRIORITY CONTAMINANTS

Contaminant	Actual cross-section and NIRS data			
	Systems >½HRL	Systems >HRL	Population >½HRL	Population >HRL
Aldrin (R2) ..... HRL = 0.002 µg/L	0.02% ..... (2 of 12,165)	0.02% ..... (2 of 12,165)	0.02% ..... (8,700 of 47.7 M)	0.02% (8,700 of 47.7 M)
Dieldrin (R2) ..... HRL = 0.002 µg/L	0.09% ..... (11 of 11,788)	0.09% ..... (11 of 11,788)	0.07% ..... (32,200 of 45.8 M)	0.07% (32,200 of 45.8 M)
Hexachlorobutadiene ..... (R1 & R2) HRL = .9 µg/L	Round 1: 0.16% ..... (20 of 12,284)	Round 1: 0.11% ..... (14 of 12,284)	Round 1: 0.57% ..... (407,600 of 71.6 M)	Round 1: 0.37% (262,500 of 71.6 M)
	Round 2: 0.08% ..... (18 of 22,736)	Round 2: 0.02% ..... (4 of 22,736)	Round 2: 2.3% ..... (1.6 M of 67.1 M)	Round 2: 0.005% (3,100 of 67.1 M)
Manganese (NIRS) ..... HRL = 300 µg/L	6.1% ..... (60 of 989)	3.2% ..... (32 of 989)	4.6% ..... (68,100 of 1.5 M)	2.6% (39,000 of 1.5 M)
Metribuzin (R2) ..... HRL = 91 µg/L	0% ..... (0 of 13,512)	0% ..... (0 of 13,512)	0% ..... (0 of 50.6 M)	0% (0 of 50.6M)
Naphthalene ..... (R1 & R2) HRL = 140 µg/L	Round 1: 0.01% ..... (2 of 13,452)	Round 1: 0.01% ..... (2 of 13,452)	Round 1: 0.007% ..... (5,600 of 77.2 M)	Round 1: 0.007% (5,600 of 77.2 M)
	Round 2: 0.01% ..... (2 of 22,923)	Round 2: 0% ..... (0 of 22,923)	Round 2: 0.002% ..... (1,700 of 67.5 M)	Round 2: 0% (0 of 67.5 M)
Sodium (NIRS) ..... Benchmark = 120,000 µg/L	22.6% ..... (224 of 989)	13.2% ..... (131 of 989)	18.5% ..... (274,300 of 1.5 M)	8.3% (123,600 of 1.5 M)
Sulfate (R2) ..... HRL = 5000,000 µg/L	4.97% ..... (819 of 16,495)	1.8% ..... (295 of 16,495)	10.2% ..... (5.2 M of 50.4 M)	0.9% (446,200 of 50.4 M)

1. Acanthamoeba

After reviewing the best available public health and occurrence information, EPA has made a preliminary determination not to regulate Acanthamoeba with a National Primary Drinking Water Regulation (NPDWR). EPA's finding is that Acanthamoeba does have adverse effects on the health of persons primarily as a result of infections affecting the eye, lung, brain, and skin. EPA has no national monitoring data for Acanthamoeba occurrence in PWSs. The Agency, however, believes that filtration practices commonly used to treat drinking water in the U.S. have a high removal rate for Acanthamoeba cysts. Moreover, EPA finds that the disease incidence for Acanthamoeba is extremely low and that exposure to Acanthamoeba-related infections are not typically produced by ingestion of drinking water, inhalation during showering, or other standard uses of drinking water. Rather, Acathamoeba related infections are typically associated with poor hygiene practices among contact lens wearers. Thus, EPA finds that regulation of Acanthamoeba does not present a meaningful opportunity for health risk reduction for persons served by PWSs. The Agency believes issuing guidance targeted to individuals at risk is a more appropriate action at this time. Detailed information supporting EPA's finding and tentative determination is provided in the Health Effects Support Document for Acanthamoeba, and is summarized later in this section.

a. Background. Acanthamoeba is a common free-living microbe found in water, soil, and air. The protozoa exists in two stages: an active infective trophozoite form, and a dormant cyst form. The cyst stage also has potential to cause infection as it reverts to a trophozoite under appropriate conditions (Ferrante 1991). The cysts are resistant to inactivation by the levels of chlorine routinely used to disinfect municipal drinking water, swimming pools, and hot tubs and can survive for many years in the environment. However, because the cysts are fairly large (larger than *Giardia* and *Cryptosporidium*), they are very likely removed by filtration practices commonly used to treat drinking water.

b. Health effects. Acanthamoeba species have been associated with human infections affecting the eye, lung, brain, and skin. There are two major clinically distinct human infections: Acanthamoeba keratitis and GAE.

Acanthamoeba keratitis infection is a chronic ulceration and perforation of the cornea. Infection occurs predominantly in individuals who wear soft contact lenses and is thought to be a consequence of improper storage, handling, and disinfection of the lenses or lense case (Stehr-Green *et al.* 1989, Seal *et al.* 1992); wearing lenses in hot tubs and during swimming; and the formation of bacterial biofilms on contact lenses and lens storage cases (Schaumberg, *et al.* 1998). Acanthamoeba keratitis does not result from ingestion of contaminated drinking water.

GAE can be caused by some species of Acanthamoeba. GAE is diagnosed more frequently in people with compromised immune systems including individuals with human immunodeficiency virus (HIV) and acquired immunodeficiency syndrome (AIDS) (Martinez and Visvesvera 1997). Reports indicate that possible routes of entry of Ancanthamoeba in immunocompromised individuals may be through the respiratory tract and skin lesions. Once inside the body, it spreads throughout the bloodstream to other parts of the body, and the central nervous system and may cause personality changes, cranial nerve palsies, nausea and headaches (Martinez and Visvesvera 1997, Marshall *et al.* 1997).

c. Occurrence and exposure. i. Acanthamoeba occurrence. Members of the genus Acanthamoeba are widespread in nature and have been isolated worldwide from brackish and sea water, tap water, bottled water, airborne dust, swimming pools, hot springs, thermal effluents of power plants, ocean sediments, vegetables, and hot tubs. *Acanthamoeba* has also been recovered from the nose and throat of humans with impaired respiratory function and from apparently healthy persons, suggesting that the amoeba is

commonly inhaled. There are no monitoring data for *Acanthamoeba* under the UCMR or other programs. There is a published report on a presumed *Acanthamoeba* contamination of municipal drinking water supply occurring after a flooding incident in Iowa during 1993–1994 (Meier *et al.* 1998). The report suggests that increase in the incidence of *Acanthamoeba* keratitis in areas affected by flooding was associated with a higher than normal concentration of *Acanthamoeba* in surface water supplies. However, the overall risk of keratitis in the U.S., even with the Iowa flooding, is less than the 1:10,000 risk of infection per year that EPA has set as a goal for surface water supplies.

ii. *Acanthamoeba* keratitis disease incidence. The Centers for Disease Control and Prevention (CDC) published a survey identifying 208 cases of *Acanthamoeba* keratitis (between 1973 and 1988) in the U.S. based on requests made to their laboratories for analysis of samples from individuals affected with ocular keratitis and from a limited survey of eye health care practitioners in four States. The data indicate that keratitis has been reported from 34 States and the District of Columbia. While most cases were reported from California, Texas, Florida, and Pennsylvania (Stehr-Green *et al.* 1989), there were no distinct regional patterns of occurrence. Because keratitis is not a disease which is required to be reported to CDC, these reports may underestimate a national occurrence.

Between 1973 and 1996 an estimated 700 *Acanthamoeba* keratitis cases have occurred in the U.S. (Martinez and Visvesvera 1997, Stehr-Green et al. 1989). There appears to be an increased keratitis incidence over the past decade that may be attributed to the increase in the number of contact lens wearers. The available published data on incidence from 1985 to 1987 (Schaumberg et al. 1998) was used to conservatively estimate incidence at 1.65 to 2.01 cases per million contact-lens wearers. This would forecast a total of 64 cases per year for the U.S. contact-lens wearing population (about 34 million people wear contact lenses). The estimated number of Acanthamoeba keratitis cases is small compared to the population at risk.

iii. GAE Disease Incidence. GAE is not a reportable disease in the U.S. Between 1957 and 1998 about 110 cases of GAE have been reported world-wide; 64 of the 110 cases were reported in the U.S., of which 30 cases were diagnosed in AIDS patients. GAE has been reported to occur predominantly in patients who are immunocompromised, those with diabetes or alcoholism, and those receiving radiation therapy (Visvesvera and Stehr-Green 1990). Based on an EPA demographic distribution of sensitive population groups, there are approximately two million people in the U.S. who are considered immunocompromised from cancer chemotherapy, genetic factors, and HIV/AIDS (CDC 1997 and USEPA 1998a). Diabetics are also more vulnerable to GAE (Visvesvera and Stehr-Green 1990). Because the number of diabetics in the U.S. is about eight million (USEPA 1998a), the total population group more vulnerable to GAE because of preexisting disease is about 10 million. Note that cases in these populations are more likely to be diagnosed since the individuals are under a degree of medical surveillance not typical of the general population. The number of cases of GAE is very small when compared to the population of the U.S. even considering the more vulnerable subgroups.

d. *Preliminary determination.* The Agency has made the preliminary determination not to regulate *Acanthamoeba* with a NPDWR since regulation would not present a meaningful opportunity for health risk reduction for the people served by public drinking water systems. Several species of *Acanthamoeba* infect humans and can be found worldwide in a range of environmental media (*e.g.,* soil, dust, and fresh water). Because of this, it is assumed that finished drinking water may be a source of exposure. However, *Acanthamoeba* keratitis is not known to be produced by ingestion of drinking water, inhalation during showering, or other standard uses of drinking water. Rather, keratitis is associated with poor hygiene practices among contact lens wearers. GAE has been reported in a very small number of individuals known to be at risk for developing this disease; there have been a total of 64 U.S. cases which is a low incidence even considering the possible vulnerability of an estimated number of immunocompromised and diabetic individuals of 10 million. Reports indicate that the possible routes of entry of *Acanthamoeba* in immunocompromised individuals are through the respiratory tract and from skin lesions. Thus, it is unlikely that any of the 64 U.S. cases were associated with ingestion of *Acanthamoeba* in drinking water.

EPA does not believe that there is an opportunity for meaningful public health protection through issuance of a drinking water regulation for *Acanthamoeba.* An effective means to protect public health is to identify those groups of individuals who may be at risk or more sensitive than the general population to the harmful effects of *Acanthamoeba* in drinking water and target them with protective measures (*e.g.,* encourage contact lens wearers to follow manufacturers' or health care practitioners' instructions for cleaning and rinsing their contact lens). EPA intends to release a guidance document addressing the risks of *Acanthamoeba* infection.

2. Aldrin and Dieldrin

After reviewing the best available public health and occurrence information, EPA has made a preliminary determination not to regulate the contaminants aldrin and dieldrin with National Primary Drinking Water Regulations (NPDWRs). EPA's findings are that aldrin and dieldrin may have adverse effects on the health of persons, and both are classified by EPA as likely to be carcinogenic to humans. EPA also finds that aldrin and dieldrin occur in PWSs, but not at a frequency or level of public health concern. Aldrin at >½ health reference level (HRL) was found at approximately 0.02% of PWS surveyed, affecting approximately 0.02% of the population served; dieldrin at >½ HRL was found at approximately 0.09% of PWS surveyed, affecting approximately 0.07% of the population served. As discussed later, EPA does not consider exposure to aldrin and dieldrin to be widespread nationally. Most uses of these compounds were canceled in 1987. Thus, EPA finds that regulating aldrin and dieldrin would not present a meaningful opportunity for health risk reduction for persons served by PWSs.

Detailed information supporting our findings and preliminary determinations is provided in the Health Effect Support Document for Aldrin and Dieldrin, the Analysis of National Occurrence of the 1998 Contaminant Candidate List (CCL) Regulatory Determination Priority Contaminant in Public Water Systems, and the Regulatory Determination Support Document for Aldrin and Dieldrin. This information is summarized later in this section.

a. Background. Aldrin and dieldrin (CASRNs 309–00–2 and 60–57–1, respectively) are the common names of two structurally similar insecticides. They are discussed together in today's action because aldrin readily changes to dieldrin in the body and in the environment, and they cause similar adverse health effects.

The Shell Chemical Company was the sole U.S. manufacturer and distributor of aldrin and dieldrin; although neither

compound has been produced in the U.S. since 1974 (ATSDR 1993). From 1950–1970, aldrin and dieldrin were popular pesticides used for crops such as corn and cotton. Because of concerns about damage to the environment and the potential harm to human health, EPA banned most uses of aldrin and dieldrin in 1974 except for the control of termites. In 1987, EPA banned all uses.

b. Health effects. EPA issued health advisories for aldrin and dieldrin in 1992 and 1988, respectively. These chemicals caused liver tumors in mice, but not in rats, and are classified as Group B2, probable human carcinogens, under the 1986 cancer guidelines. Under EPA's 1999 proposed Guidelines for Carcinogen Risk Assessment (USEPA 1999b), aldrin and dieldrin are classified as likely to be carcinogenic to humans.

In animals, oral exposure to aldrin and dieldrin has produced a variety of dose-dependent systemic, neurological, immunological, endocrine, reproductive, developmental, genotoxic and tumorigenic effects over a collective dose range of at least three orders of magnitude (<0.05–50 mg/kg body weight), depending on the specific endpoint and the duration of exposure.

In general, animal studies have provided only mixed evidence that exposures to aldrin and dieldrin at moderate-to-high levels can result in adverse reproductive or developmental effects such as reduced fertility or litter size, reduced pup survival, fetotoxicity, or teratogenicity. Various in vivo and in vitro studies have provided evidence that aldrin and dieldrin may be weak endocrine disruptors (ATSDR 2000a), that is to say, they may weakly disrupt the hormones responsible for the maintenance of normal body function and the regulation of developmental processes.

EPA derived the RfD of 3×10^{-5} mg/kg/day for aldrin by dividing the LOAEL for liver toxicity from a lifetime study on rats of 0.025 mg/kg/day by an uncertainty factor (UF) of 1,000 (USEPA 1988, see section III.A. of today's action). The UF is a product of three 10-fold factors that account for the variation in sensitivity among the members of the human population, the uncertainty in extrapolating animal data to humans, and the uncertainty in extrapolating from a LOAEL rather than from a NOAEL.

EPA derived the RfD of 5×10^{-5} mg/kg/day for dieldrin by dividing the NOAEL for liver toxicity from a lifetime study on rats of 0.005 mg/kg/day by a UF of 100 (10 to extrapolate from rats to humans, and 10 to protect sensitive humans) (USEPA 1990).

The most sensitive endpoint of concern is cancer for both aldrin and dieldrin. The Agency used a linearized multi-stage model to extrapolate from effects seen at high doses in animal studies to predict tumor response at low doses. This model is based on the biological theory that a single exposure to a carcinogen can initiate tumor formation, and it assumes that a threshold does not exist for carcinogenicity. Based on this approach, it is estimated that aldrin and dieldrin carcinogenic potencies are 17 per mg/kg-day and 16 per mg/kg-day, respectively. Using these cancer potencies, the concentrations associated with a specific risk levels for both contaminants are 0.2, 0.02, and 0.002 µg/L at the theoretical cancer risk of 10^{-4}, 10^{-5}, and 10^{-6}, respectively (*i.e.*, 1 case in 10,000; 1 case in 100,000; and 1 case in 1,000,000) (USEPA 1993a and 1993b). EPA adopted the dose level of 0.002 µg/L for both contaminants as the HRL, or the benchmark against which to evaluate the occurrence data.

Potential susceptibility of life-stages and other sensitive populations. Aldrin and dieldrin are found as residues in food and mother's milk; however, no long-term studies demonstrating adverse effects on children are available. Although these chemicals are thought to be weak endocrine disruptors the HRL should adequately protect sensitive individuals from this and other adverse effects because cancer is assumed to be the most sensitive endpoint of concern.

No other sensitive subpopulations were identified that may be affected by exposure to these contaminants.

c. Occurrence and exposure. For most people, exposure to aldrin and dieldrin occurs when people eat contaminated foods. Contaminated foods might include fish or shellfish from contaminated lakes or streams, root crops, dairy products, and meats. Exposure to aldrin and dieldrin also occurs when you drink water, breathe air, or touch contaminated soil at hazardous waste sites containing these contaminants.

Aldrin was monitored under Round 2 of the Unregulated Contaminant Monitoring (UCM). Cross-section occurrence estimates are very low with only 0.006% of the samples (2 out of 31,083) showing detections at 0.58 µg/L and 0.69 µg/L.

The cross-section analysis shows that 0.02% of the reporting PWSs (2 out of 12,165) experienced detections of aldrin at both >½ HRL and >HRL, affecting 0.02% of the population served (8,600 out of 47.8 million people).

Dieldrin was also monitored under Round 2 of the UCM. The cross-section occurrence estimates are also very low with only 0.064% of samples (19 out of 29,603) showing detections. For samples with detections, the median and the 99th percentile concentrations are 0.16 µg/L and 1.36 µg/L, respectively.

The cross-section analysis shows that 0.09% of the reporting PWSs (11 out of 11,788) have detections of dieldrin at both >½ HRL and >HRL, affecting 0.07% of the population served (32,000 out of 45.8 million).

To augment SDWA drinking water data analysis, and to provide additional coverage of the corn belt States where aldrin and dieldrin use as agricultural insecticides was historically high but not represented in the Round 2 data, independent analyses of SDWA drinking water data from the States of Iowa, Illinois, and Indiana were undertaken. There were no detections of aldrin in Iowa or Indiana surface or ground water PWSs (Hallberg et al. 1996, USEPA 1999a). While Illinois had no detections in ground water, aldrin was detected in 2 out of 109 (1.8%) surface water PWSs, the maximum concentrations of aldrin was 2.4 µg/L. A survey of Illinois community water supply wells during the mid-1980s also showed very low occurrence of aldrin.

Dieldrin was not reported in Iowa surface or ground water PWSs (Hallberg et al. 1996). While Illinois and Indiana also had no detections of the compound in ground water PWSs, dieldrin was detected in surface water PWSs in those States (USEPA 1999a). Dieldrin occurrence was relatively low in both States: 2 out of 109 (1.8%) surface water systems showed detections in Illinois and 1 out of 47 (2.1%) surface water systems showed detections in Indiana. For Illinois and Indiana surface water PWSs, the maximum concentrations of dieldrin were 0.1 µg/L and 0.04 µg/L, respectively (USEPA 1999a).

Even the data from all Round 2 reporting States, including States with incomplete or potentially skewed data, show very low occurrence of aldrin and dieldrin. Approximately 0.21% (32 out of 15,123) of the reporting PWSs have detections of aldrin at both >½ HRL and >HRL, affecting approximately 291,000 of the population served (out of 59 million). For dieldrin, approximately 0.21% (31 out of 14,725) of the reporting PWSs have detections at both >½ HRL and >HRL, affecting about 212,000 of the population served (out of 57 million).

d. Preliminary determination. The Agency has made a preliminary determination not to regulate aldrin or dieldrin with a NPDWR. Since the

contaminants occur in PWSs at a very low frequency and at low levels, a regulation would not present a meaningful opportunity for health risk reduction for the people served by public drinking water systems. EPA recognizes that aldrin and dieldrin are probable human carcinogens, but the chemicals have been banned for most uses since 1974, and have relatively low levels of occurrence in drinking water supplies. It is likely that there will be so few people exposed to aldrin and dieldrin in their drinking water that a national regulation to control these two pesticides in drinking water would not provide a meaningful opportunity to reduce risk.

EPA will work closely with those few States that show aldrin and dieldrin contamination and encourage them to work with affected systems to evaluate site specific protective measures and to consider State-level regulation.

3. Hexachlorobutadiene

After reviewing the best available public health and occurrence information, EPA has made a preliminary determination not to regulate hexachlorobutadiene with a National Primary Drinking Water Regulation (NPDWR). EPA's finding is that hexachlorobutadiene may have adverse effects on the health of persons. It is classified by EPA as likely to be carcinogenic to humans. EPA also finds that hexachlorobutadiene occurs in PWSs, but not at a frequency or level of public health concern. Hexachlorobutadiene at >½ health reference level (HRL) was found at approximately 0.16% of PWS surveyed in Round 1 cross section samples and 0.08% of Round 2 cross section samples, affecting approximately 0.57% of the population served in Round 1 and 2.3% in Round 2. (The Round 2 affected population percentage is strongly influenced by a >½ HRL detection at one PWS serving 1.5 million people.) Thus, EPA finds that regulating hexachlorobutadiene with a NPDWR would not present a meaningful opportunity for health risk reduction for persons served by PWSs.

Detailed information supporting our finding and tentative determination is provided in the Health Effects Support Document for Hexachlorobutadiene, the Analysis of National Occurrence of the 1998 Contaminant Candidate List (CCL) Regulatory Determination Priority Contaminant in Public Water Systems, and the Regulatory Determination Support Document for Hexachlorobutadiene. These findings are summarized later in this section.

a. Background. Hexachlorobutadiene (CASRN 87–68–3) is a VOC that is relatively insoluble in water (solubility of 2–2.55 mg/L) and has never been manufactured as a commercial product in the U.S. However, significant quantities of the chemical are generated in the U.S. as a waste by-product from the chlorination of hydrocarbons, and lesser quantities are imported mostly from Germany as a commercial product. Hexachlorobutadiene is mainly used to make rubber compounds. It is also used as a solvent, to make lubricants, in gyroscopes, as a heat transfer liquid, and as a hydraulic fluid.

Eight million pounds of hexachlorobutadiene were generated as a waste by-product in the U.S. in 1975, with 100,000 pounds released into the environment. By 1982, the annual U.S. by-product generation of the chemical increased to 28 million pounds. In contrast, the annual import rate of hexachlorobutadiene dropped from 500,000 pounds per year imported annually in the late 1970's, to 145,000 pounds per year imported in 1981 (ATSDR 1994, Howard 1989).

Hexachlorobutadiene is listed by EPA as a toxic release inventory (TRI) chemical. Air emissions constitute most of the on-site releases. Also, over a 10-year period (1988–1998), surface water discharges generally increased, peaked in 1992–93, and then decreased significantly through the late-1990s. The TRI data for hexachlorobutadiene are reported from eight States (USEPA 2001d).

b. Health effects. There are no reliable data of human health effects following exposure to hexachlorobutadiene. Hexachlorobutadiene is classified by EPA as a Group C, Possible Human Carcinogen, (USEPA 1991) in accordance with EPA's 1986 Guidelines for Carcinogen Risk Assessment (USEPA 1986), and is considered likely to be a carcinogen to humans by the 1999 Proposed Guidelines for Carcinogen Risk Assessment (USEPA 1999b). Studies in animals show the selective effect of hexachlorobutadiene on the proximal tubule of the kidney. Subchronic (NTP 1991) and chronic (Kociba *et al.* 1977) studies in rodents present a clear picture of dose-related renal (kidney) damage at 2 mg/kg/day and above. Progressive events over time include changes in kidney weight, altered renal function (as shown by increased excretion of coproporhyrin), renal tubular degeneration and regeneration, hyperplasia (abnormal growth of cells), and renal tumor formation. Developmental effects were also observed in the offspring of hexachlorobutadiene exposed female rats (Harleman and Seinen 1979). However, these effects were observed at higher doses than for renal toxicity. Pups with lower birth weights and reduced growth were reported at maternal dose of 8.1–15 mg/kg/day in rats (Badaeva 1983, Harleman and Seinen 1979).

Only one study of lifetime oral exposure to hexachlorobutadiene has been reported in peer reviewed literature (Kociba *et al.* 1977). At the highest dose of 20 mg/kg/day in the study, benign and malignant tumors were seen in approximately 23% (9/39) of the male rats, and 15% (6/40) of the female rats. This dose exceeded the maximum tolerated dose at which increased mortality, severe renal toxicity, and significant weight loss were also observed. There were no tumors found in rats at the second highest dose of 2 mg/kg/day. The conclusion from the dose response analysis is that hexachlorobutadiene is a weak carcinogen with its demonstrated carcinogenicity only at a cytotoxic dose.

EPA divided the NOAEL for damage to kidney cells (specifically, renal tubular epithelial cell degeneration and regeneration) in rats from the Kociba *et al.* (1977) study and in mice from the National Toxicology Program (NTP 1991) study of 0.2 mg/kg/day by an uncertainty factor (UF) of 1000 (see section III.A. of today's action). The UF is a product of four factors, and rounded from 900 to 1000, that account for: the uncertainty in extrapolating animal data to humans (UF=10), the variation in sensitivity among the members of the human population (UF=10), using a minimum effect NOAEL, that may be a minimal LOAEL (UF=3), and the uncertainty associated with extrapolation from an incomplete animal data base (UF=3, the data base lacks chronic oral exposure studies and 2-generation reproductive toxicity studies) to arrive at an RfD of 2×10^{-4} mg/kg/day (USEPA 1998b). The RfD was used to develop the HRL of 1 µg/L as a benchmark against which to evaluate the occurrence data as described in section III.A. of today's action.

The nonlinear approach for low dose extrapolation (i.e., point of departure of 0.054 mg/kg/day divided by a margin of exposure 300), gives a result equal to the RfD. Thus, the RfD of 2×10^{-4} mg/kg/day which protects against damage to kidney tubule cells will also be protective against tumor formation in the kidney.

Potential susceptibility of life-stages and other sensitive populations. Individuals with preexisting kidney damage may be more sensitive to

adverse health effects from hexachlorobutadiene. Studies in animals showed that young rats and mice were more sensitive to the acute effects of hexachlorobutadiene (Hook *et al.* 1983, Lock *et al.* 1984), suggesting that infants may also be more susceptible to hexachlorobutadiene toxicity, perhaps as a result of immature organ systems.

c. Occurrence and exposure. Most exposure to hexachlorobutadiene comes from breathing it in workplace air. People living near hazardous waste sites containing hexachlorobutadiene may be exposed to it by breathing air or by drinking contaminated water.

Hexachlorobutadiene was monitored under both Rounds 1 and 2 of the Unregulated Contaminant Monitoring (UCM). The cross-section occurrence estimates are low for Round 1 and Round 2 with only 0.13% (54 of 42,839) and 0.05% (43 of 93,585) of all samples showing detections, respectively. For Round 1 cross-section samples with detections, the median and the 99th percentile concentrations are 0.25 µg/L and 10 µg/L, respectively. For Round 2 cross-section samples with detections, the median and the 99th percentile concentrations are 0.30 µg/L and 1.5 µg/L, respectively.

For Round 1, the cross-section analysis shows that 0.16% of the reporting PWSs (20 out of 12,284) had detections >½ HRL, affecting 0.57% of the population served (407,000 out of 71.6 million). The percentage of reporting PWSs with detections >HRL is 0.11% (14 out of 12,284), affecting 0.37% of the population served (263,000 out of 71.6 million).

For Round 2, the cross-section analysis shows that 0.08% of the reporting PWSs >½ HRL (18 out of 22,736), affecting 2.3% of the population served (1.6 out of 67 million). The percentage of the reporting PWSs with detections >HRL is 0.02% (4 out of 22,736), affecting 0.005% of the population served (3,350 out of 67 million).

The Round 1 cross-section estimates of PWSs affected by hexachlorobutadiene are influenced by the State of Florida. Florida reports 5.4% of its PWSs experienced detections >HRL, a value considerably greater than the next highest State (1.5%). In addition, only 13% of the PWSs in Florida (112 out of 855 PWSs) provided data, suggesting that only systems experiencing problems submitted data for hexachlorobutadiene, thereby biasing Florida's results for occurrence measures.

The large values for the Round 2 cross-section estimates of population served with detections >½ HRL are influenced by the inclusion of one PWS serving a very large population (1.5 million people). While the percentages of systems with detections of hexachlorobutadiene >½ HRL are low for both rounds, the difference in population served is larger.

d. Preliminary determination. The Agency has made a preliminary determination not to regulate hexachlorobutadiene with a NPDWR since the contaminant occurs in PWSs at a very low frequency and at very low levels and would therefore not present a meaningful opportunity for health risk reduction for persons served by public drinking water supplies. Monitoring data indicate that hexachlorobutadiene is infrequently detected in public water supplies. It is important to note that when hexachlorobutadiene is detected, it very rarely exceeds the HRL or even a value of one-half the HRL.

4. Manganese

After reviewing the best available public health and occurrence information, EPA has made a preliminary decision not to regulate manganese with a National Primary Drinking Water Regulation (NPDWR). EPA's finding is that manganese is essential for normal physiological functioning in humans and all animal species, however, several diseases are associated with both deficiencies and excess intake of manganese. Nonetheless, manganese is generally considered to have low toxicity when ingested orally. EPA also finds that manganese occurs in PWSs, with 6.1% of reporting ground water PWSs having detections above the >½ health reference level (HRL) and 3.2% having detections above the HRL. But, because the toxicity of manganese by oral ingestion is low, EPA finds that regulation of manganese in drinking water does not present a meaningful opportunity for health risk reduction for persons served by PWSs.

Detailed information supporting our finding and tentative determination is provided in the Health Effects Support Document for Manganese, the Analysis of National Occurrence of the 1998 Contaminant Candidate List (CCL) Regulatory Determination Priority Contaminant in Public Water Systems, and the Regulatory Determination Support Document for Manganese. These findings are summarized later in this section.

a. Background. Manganese (CASRN 7439–96–5) is a naturally occurring element that constitutes approximately 0.1% of the earth's crust. It does not occur in the environment in its pure metal form, but is ubiquitous as a component of more than 100 minerals including many silicates, carbonates, sulfides, oxides, phosphates, and borates (ATSDR 2000b). Manganese occurs naturally at low levels in soil, water, and food, and is essential for normal physiological functioning in humans and all animal species.

EPA established a National Secondary Drinking Water Standard for manganese at 0.05 mg/L to prevent clothes from staining and to minimize taste problems. Secondary standards are non-enforceable Federal guidance for aesthetic effects (such as color, taste, or odor) or cosmetic effects (such as skin or tooth discoloration) and are provided as a guideline for States and PWSs.

b. Health effects. Manganese is needed for normal growth and function; however, several diseases are associated with both deficiencies and excess intake of manganese.

There is no information available on the carcinogenic effects of manganese in humans, and animal studies have reported mixed results. EPA considers manganese to be not classifiable with respect to carcinogenicity; Group D according to the Guidelines for Carcinogen Risk Assessment (1999b). Data from oral exposure suggest that manganese has a low developmental toxicity.

There are several reports of toxicity to humans exposed to manganese by inhalation. Inhaled manganese can lead to neurological symptoms (*e.g.,* tremor, gait disorders, etc.) as seen in miners exposed to manganese dusts or fumes. Much less is known about oral intake of manganese. The major source of manganese intake in humans (with the exception of possible occupational exposure) is dietary ingestion; however, manganese is not considered to be very toxic when ingested with food, and reports of adverse effects are rare.

An epidemiological study performed in Peloponnesus, Greece (Kondakis *et al.* 1989) showed that lifetime consumption of drinking water containing naturally high concentrations of manganese oxides may lead to neurological symptoms and increased manganese retention as reflected in the concentration of manganese in hair for people over 50 years old. For the group consuming the highest concentration (around 2 mg/L) for more than 10 years, the authors suggested that some neurologic impairment might be present. The study raises concerns about possible adverse neurological effects following chronic ingestion from drinking water at doses within ranges deemed essential. However, the study did not examine

manganese intake data from other routes/sources (*i.e.,* dietary intake, inhalation from air, etc.), precluding its use as a basis for the RfD.

Another long-term drinking water study in Germany (Vieregge *et al.* 1995) found no neurological effects in people older than 50 years of age who drank water containing 0.3 to 2.16 mg/L of manganese for more than 10 years. However, this study also lacks exposure data from other routes and sources, and the manganese concentration range in water is very wide. Thus, the study cannot be used for quantitative assessment.

A small Japanese community (total 25 individuals) ingested high levels of manganese in contaminated well water (leaked from dry cell batteries buried near the wells) over a three-month period (Kawamura *et al.* 1941). Manganese intake was not determined at the time of intoxication, but was assayed months later; it was estimated to be close to 29 mg/L (*i.e.,* 58 mg/day or 1.45 mg/kg/day). Symptoms included lethargy, increased muscle tonus (tension), tremor, mental disturbances, and even death. Autopsies revealed macroscopic and microscopic changes in the brain tissue. In contrast, six children (1 to 10 years old) were not as affected as were the adults by this exposure. The elderly were more severely affected. Some effects may have resulted from factors other than manganese exposure.

In various surveys, manganese intakes of adults eating western type and vegetarian diets ranged from 0.7 to 10.9 mg per day (Freeland-Graves 1994, Gibson 1994). Depending on individual diets, a normal intake may be well over 10 mg/day, especially from a vegetarian diet. Thus, from the dietary surveys taken together, EPA concluded that an appropriate RfD for manganese is 10 mg/day (0.14 mg/kg/day) (USEPA 1996). The Agency applied an uncertainty factor (UF) of 1 (see section III.A. of today's action) because the information used to determine the RfD was considered to be complete—it was taken from many large human populations consuming normal diets over an extended period of time with no adverse health effects. EPA derived a HRL for evaluating the occurrence data of 0.30 mg/L. The HRL is based on the dietary RfD and application of a modifying factor of 3 for drinking water as recommended by IRIS (USEPA 1996) (see the description of an RfD in section III.A. of today's action) and allocation of an assumed 20% relative source contribution from water ingestion. The modifying factor accounts for concerns raised by the Kondakis study (1989); the potential for higher absorption of manganese in water compared to food; consideration of fasting individuals; and the concern for infants with potentially higher absorption and lower excretion rates of manganese.

Potential susceptibility of life-stages and other sensitive populations. There are no data to indicate that children are more sensitive to manganese than adults. Because manganese is an essential nutrient in developing infants, the potential adverse effects from manganese deficiency may be of greater concern than potential toxicity from over-exposure. Potential sensitive sub-populations include the elderly, pregnant women, iron-deficient individuals and individuals with impaired liver and bile duct function.

c. Occurrence and exposure. Manganese has been detected in ground water PWS samples collected through the National Inorganics and Radionuclide Survey (NIRS). Approximately 68% (671 of 989) of the systems that were sampled, showed manganese above detection levels. However, for samples with detections, the median and the 99th percentile concentrations are 0.01 mg/L and 0.72 mg/L, respectively. NIRS samples show that 6.1% of the reporting ground water PWSs had detections >½ HRL (60 out of 989), affecting about 4.6% of the population served (68,200 out of 1.5 million). The percentage of reporting ground water PWSs with detections >HRL is 3.2% (32 out of 989) affecting 2.6% of the population served (39,000 out of 1.5 million).

d. Preliminary determination. The Agency has made a preliminary determination not to regulate manganese with a NPDWR because it is generally not considered to be very toxic when ingested with the diet and because drinking water accounts for a relatively small proportion of manganese intake. Thus, regulation would not present a meaningful opportunity for health risk reduction for persons served by PWSs.

5. Metribuzin

After reviewing the best available public health and occurrence information, EPA has made a preliminary determination not to regulate metribuzin with a National Primary Drinking Water Regulation (NPDWR). EPA's finding is that metribuzin is not classifiable as a human carcinogen, but there may be other adverse health effects related to metabolic activity from chronic exposure to high doses. EPA also finds that metribuzin has a very low occurrence in PWSs. Only one sample out of 34,507, in Round 2 of the Unregulated Contaminant Monitoring (UCM), was reported as having a detection and the concentration of that sample was below ½ health reference level (HRL). Because metribuzin has such low occurrence, EPA finds that the regulation of metribuzin in drinking water does not present a meaningful opportunity for health risk reduction for persons served by PWSs.

Detailed information supporting our findings and preliminary determinations is provided in the Health Effect Support Document for Metribuzin, the Analysis of National Occurrence of the 1998 Contaminant Candidate List (CCL) Regulatory Determination Priority Contaminant in Public Water Systems, and the Regulatory Determination Support Document for Metribuzin. These findings are summarized later in this section.

a. Background. Metribuzin (CASRN 21087–64–9) is an SOC that does not volatilize readily, yet is very soluble in water. Metribuzin is relatively persistent in the environment and degrades primarily through exposure to sunlight.

Metribuzin is used as an herbicide on crops and has limited non-agricultural utility. Applications are primarily targeted to soybeans, potatoes, alfalfa, and sugar cane, and the geographic distribution of use largely reflects the distribution of these crops across the U.S. In terms of use, the herbicide is ranked 200th out of approximately 1,150 active ingredients used in agricultural pesticides (USGS 1999). According to the U.S. Department of Agriculture's Agricultural Resources Management Study, the amount of metribuzin used annually and the number of acres treated appears to be modestly declining over the 10-year survey period (1990–1999).

b. Health effects. Metribuzin is not classifiable as to human carcinogenicity (Group D) (USEPA 1998c). This classification is based on the lack of evidence of carcinogenicity in the following studies: (1) A mouse study in which there were no increases in tumor incidences at dosing levels up to 438 mg/kg/day in the diet for males and 567 mg/kg/day for females in the diet; (2) a rat study in which there were no statistically significant increases in tumor incidence at dosing levels up to 14.36 mg/kg/day for males and 20.38 mg/kg/day for females; and (3) a rat study which indicated no evidence for carcinogenicity at dosing levels up to 42.2 mg/kg/day for males and 53.6 mg/kg/day for females (USEPA 1998c).

Acute exposures to metribuzin, as reflected in high LD_{50} values, are

indicative of low toxicity (USEPA 1998c). Subchronic studies in rats and dogs suggest that metribuzin causes decreased body weight gain, increased organ weight (liver, thyroid and brain) and small decreases in blood serum activities. Chronic effects of metribuzin exposure at high doses, in rats and dogs, include changes in body weight gain, mortality, elevated liver enzyme activity and histopathological changes in the liver. There are a few studies available on metribuzin exposure and reproductive and developmental effects. Developmental studies in rabbits and rats show that maternal toxicity occurs at or above doses of 1.3 mg/kg/day in the diet (USEPA 1998c). In general, effects to the fetus occur only as a result of maternal toxic effects. Similarly, in reproductive studies in rats, systemic toxicity was observed at mid- and high-doses (7.5 mg/kg/day and 37.5 mg/kg/day) in both parental animals and pups. Effects were expressed as slightly decreased body weights, decreased body weight gain and exaggerated liver cell growth (USEPA 1998c). Metribuzin exposure can also produce some endocrine effects in vivo as seen in the principal study used to derive the RfD.

A few inhalation studies are available on metribuzin exposure and the effects are comparable to the existing oral exposure studies. At high exposure (720 mg/m^3), increases in organ weights as well as liver enzyme activities were reported (USEPA 1998c).

The RfD for metribuzin is 0.013 mg/kg/day based on a two-year feeding study in rats where statistically significant increases in blood levels of T4 (thyroxine), decreases in blood levels of T3 (triiodothyronine), increased absolute and relative weight of the thyroid and decreased lung weight were observed at 1.3 mg/kg/day (LOAEL). However, these effects were of marginal biological significance and the 1.3 mg/kg/day dose was regarded as a NOAEL in the derivation of the RfD. The Agency applied an uncertainty factor (UF) of 100 (see section III.A. of today's action). The UF is a product of two 10-fold factors that account for the variation in sensitivity among the members of the human population and the uncertainty in extrapolating animal data to humans (USEPA 1998c).

EPA derived a HRL for evaluating the occurrence data of 91 µg/1 using the RfD approach (described in section III.A. of today's action).

Potential susceptibility of life-stages and other sensitive populations. There is no evidence to suggest that children, or any other population subgroup, would be more sensitive than others when exposed to metribuzin. In addition, the UF applied for variation in sensitivity for humans adequately protects sensitive subgroups of the population.

c. *Occurrence and exposure.* Metribuzin has been monitored under Round 2 of the UCM program. The cross-section shows that only 1 out of 34,507 samples had detections from the 13,512 PWSs sampled (0.10 µg/L). No cross-section PWSs had detection >½ HRL or >HRL.

The heaviest use of metribuzin is across the nation's corn-soybean production area. These States are not well represented in the Round 2 database. Therefore, additional data from the Midwest corn belt were also evaluated. Drinking water data from Iowa, Indiana, Illinois, and Ohio also show very low occurrence of metribuzin.

d. Preliminary determination. The Agency has made a preliminary determination not to regulate metribuzin with a NPDWR because it is not known to occur in PWSs at levels of public health concern. Monitoring data indicate that metribuzin is infrequently detected in public water supplies. When metribuzin is detected, it very rarely exceeds the HRL or a value of one-half of the HRL.

6. Naphthalene

After reviewing the best available public health and occurrence information, EPA has preliminarily determined not to regulate naphthalene with a National Primary Drinking Water Regulation (NPDWR). EPA's finding is that there is inadequate data to support a conclusion about carcinogenicity of naphthalene by the oral route of exposure. But, there may be other adverse health effects from exposure to naphthalene such as hemolytic anemia from very high doses of naphthalene (e.g. ingestion of mothballs). EPA also finds that naphthalene has a very low occurrence in PWSs. Naphthalene at >½ health reference level (HRL) was found at approximately 0.01% of public water supplies surveyed in Round 1 and Round 2 cross section samples, affecting less than 0.007% of the population served. Because naphthalene has such a low occurrence level, EPA finds that the regulation of naphthalene in drinking water does not present a meaningful opportunity for health risk reduction for persons served by PWSs.

Detailed information supporting our findings and preliminary determination is provided in the Health Effect Support Document for Naphthalene, the Analysis of National Occurrence of the 1998 Contaminant Candidate List (CCL) Regulatory Determination Priority Contaminant in Public Water Systems, and the Regulatory Determination Support Document for Naphthalene. These findings are summarized later in this section.

a. Background. Naphthalene (CASRN 91–20–3) is a VOC that is naturally present in fossil fuels such as petroleum and coal and is formed when wood or tobacco are burned. Naphthalene is produced in commercial quantities from either coal tar or petroleum. Most of naphthalene use (60%) is as an intermediary in the production of phthalate plasticizers, resins, phthaleins, dyes, pharmaceuticals, and insect repellents. Crystalline naphthalene is used as a moth repellent and as a solid block deodorizer for diaper pails and toilets.

Naphthalene production in the U.S. dropped from 900 million pounds per year in 1968 to 354 million pounds per year in 1982. Approximately seven million pounds of naphthalene were imported and nine million pounds were exported in 1978. By 1989, imports had dropped to four million pounds, and exports increased to 21 million pounds (ATSDR 1995).

b. Health effects. In inhalation studies (NTP 1992, 2000), rats and mice exposed to naphthalene developed tumors of the respiratory tract (nose, lungs). This appears to be a route-specific effect. Naphthalene is currently categorized as Group C, a possible human carcinogen, based on inadequate data in humans and limited evidence in animals (NTP 1992) via the inhalation route. According to the proposed 1999 cancer guidelines for carcinogen risk assessment, the carcinogenic potential of naphthalene cannot be determined via the oral or inhalation routes. A recent finding of clear evidence for nasal tumors in male and female mice (NTP 2000) suggests a need to reevaluate the carcinogenicity of naphthalene via the inhalation route of exposure.

The data on naphthalene's ability to cause cancer by the oral route of exposure are inadequate to support a conclusion about its carcinogenicity by this route. The tumor data from the only long term oral exposure study (Schmahl 1955) indicates that naphthalene was not carcinogenic by the oral route, but the published study did not present quantitative data on tumor incidence. Most of the studies of naphthalene's ability to damage DNA are negative.

Naphthalene can cause methemoglobinemia in humans, and humans are more sensitive to this effect than rats and mice. Methemoglobinemia is a condition where some of the red blood cells are chemically changed so

that they are not able to carry oxygen. It often leads to changes in the affected red blood cells so that they are broken down by the spleen (hemolysis) and removed from the bloodstream causing what is called hemolytic anemia. In the case of naphthalene, most of the data on methemoglobinemia and hemolysis come from cases in which large amounts of naphthalene (*e.g.,* mothballs) were ingested causing significant hemolysis and requiring medical attention.

In animal studies, high doses of naphthalene lead to cataracts in certain strains of rabbits, rats, and mice. The data on cataracts in humans are very limited and are confounded by exposure to other contaminants in addition to naphthalene. In the respiratory tract, naphthalene causes irritation, inflamation, and an increase in the number of cells (hyperplasia).

To calculate the RfD, EPA divided the NOAEL of 71 mg/kg/day for impaired weight gain in rats from the Battelle Columbus Laboratory study (1980) by an uncertainty factor (UF) of 3,000 (see section III.A. of today's action) to arrive at an RfD of 0.02 mg/kg-day (USEPA 1998d). The UF is a product of four factors that account for: the variation in sensitivity among the members of the human population (UF=10), the uncertainty in extrapolating animal data to humans (UF=10), the uncertainty in extrapolating from data obtained in a study with less-than-lifetime exposure to lifetime exposure (UF=10), and the uncertainty associated with extrapolation from an incomplete animal data set (UF=3, the data set lacks chronic oral exposure studies and 2-generation reproductive toxicity studies). The RfD of 0.02 mg/kg/day was used to develop the HRL of 140 µg/L as a benchmark against which to evaluate the occurrence data as described in section III.A. of today's action.

Potential susceptibility of life-stages and other sensitive populations. Newborn infants with one or two copies of a defective gene for the enzyme, glucose-6-phosphate dehydrogenase (G6PD) are most sensitive to the hemolytic effects of naphthalene. There is evidence of naphthalene toxicity in infants who reportedly were exposed by dermal contact with diapers or clothing that had been stored with naphthalene mothballs or naphthalene flakes (ATSDR 1995). However, inhalation of the naphthalene vapors was likely a contributing route of exposure in each case (ATSDR 1995, EPA 1998d). Adults with the G6PD defect are also susceptible to naphthalene, but to a lesser extent than infants. In infants, production of the enzyme methemoglobin reductase is delayed rendering them more sensitive than adults to methemoglobinemia. Based on the available data the 10-fold UF for intraspecies differences (*i.e.,* sensitivity among the members of the human population) used in developing the RfD will adequately protect individuals who are sensitive to naphthalene.

c. Occurrence and exposure. The major source of human exposure to naphthalene is through the use of mothballs containing naphthalene. This exposure can be from breathing the vapors or handling the mothballs. People also may be exposed by breathing tobacco smoke and air near industries that produce naphthalene. Usually naphthalene is not found in water because it evaporates or biodegrades quickly. When it is found in water, it is usually at levels lower than 0.01 mg/L (ATSDR 1995).

Naphthalene was monitored under both Rounds 1 and 2 of the Unregulated Contaminant Monitoring (UCM). For Round 1 samples with detections, the median and the 99th percentile concentrations are 1.0 µg/L and 900 µg/L, respectively. There are indications that two ground water systems in one cross-section State had outlier values (i.e., atypically high values not consistent with the rest of the data) and, thus, the 99th percentile value is suspect. Excluding these outliers from the analyses, no other State that contributed Round 1 monitoring data had any detections that exceeded the HRL (140 µg/L). For Round 2 samples with detections, the median and the 99th percentile concentrations are 0.73 µg/L and 73 µg/L, respectively.

For Round 1, the cross-section analysis shows that 0.01% of the reporting PWSs (1 out of 13,452) had detections at both >½ HRL and >HRL, affecting 0.007% of the population served (5,400 out of 77.2 million).

For Round 2, the cross-section analysis shows that 0.01% of the reporting PWSs had detections >½ HRL (2 out of 22,923), affecting 0.002% of the population served (1,300 out of 67.5 million). No Round 2 PWSs had detections >HRL.

d. Preliminary determination. The Agency has made a preliminary determination not to regulate naphthalene with a NPDWR because it is not known to occur in PWSs at levels of public health concern. Monitoring data indicate that naphthalene is infrequently detected in public water supplies. When naphthalene is detected, it very rarely exceeds the HRL or a value of one-half of the HRL.

7. Sodium

After reviewing the best available public health and occurrence information, EPA has made a preliminary determination not to regulate sodium with a National Primary Drinking Water Regulation (NPDWR). Sodium is essential for normal physiological functioning in humans and all animal species; however, in humans several disorders are associated with excess intake of sodium, in particular, high blood pressure. EPA finds that sodium occurs in PWSs. Sodium at >½ benchmark value (60 mg/L) was found at approximately 22.6% of PWS in the National Inorganic and Radionuclides Survey (NIRS) samples. Sodium at > the benchmark value (120 mg/L) was found at 13.2% of PWS. EPA believes that the contribution of drinking water to daily sodium intake is very small when compared to the total dietary intake and that short-term excursions beyond the benchmark values pose no adverse health risk for most individuals, including the majority of persons with hypertension. Because sodium in drinking water is a very small contributor to daily dietary intake and because the levels at which sodium intake can contribute to increasing the blood pressure of individuals with normal blood pressures is not clearly established, EPA does not believe that a NPDWR presents a meaningful opportunity for public health protection. Concurrent with today's action, EPA intends to issue an updated advisory to provide guidance to communities that may be exposed to drinking water with elevated levels of sodium chloride and other sodium salts, so that those individuals with restricted sodium intake may take appropriate actions.

Detailed information supporting our finding and preliminary determination is provided in the Draft Drinking Water Advisory: Consumer Acceptability Advice and Health Effects Analysis on Sodium, Analysis of National Occurrence of the 1998 Contaminant Candidate List (CCL) Regulatory Determination Priority Contaminants in Public Water Systems, and Regulatory Determination Support Document for Sodium. These documents are available for review and comment at the EPA Water Docket.

a. Background. Sodium (CASRN 7440–23–5) is the sixth most abundant element on Earth and is widely distributed in soils, plants, water, and foods. Most of the world has numerous deposits of sodium-containing minerals. The sodium ion is ubiquitous in water,

due to the high solubility of many sodium salts. Ground water typically contains higher concentrations of minerals and salts than do surface waters. In addition to naturally occurring sources of sodium, it is used in deicing roads, water treatment chemicals, and domestic water softeners; sewage effluents can also contribute significant quantities of sodium to water.

Research indicates that the lower level of the taste threshold for sodium chloride in water is 30–60 mg/L (Pangborn and Pecore 1982). Individuals who are sensitive to the taste of sodium chloride can detect the taste in water at a concentration of 30 mg/L and recognize that taste as salty at a concentration of 60 mg/L. Accordingly, a moderate amount of sodium can be tolerated without any adverse impact on the aesthetic acceptability of the water. The taste threshold for sodium is influenced by a number of factors. It increases with the age of the consumer, in the presence of other dissolved minerals, and in waters with low chloride concentrations.

Sodium consumption and source contribution of drinking water. Sodium is a normal component of the body, and adequate levels of sodium are required for good health. Food is the main source of daily human exposure to sodium, primarily in the form of sodium chloride (table salt). Most of the sodium in our diet is added to food during food processing and preparation. Various studies have reported dietary intakes of sodium that range from 1,800 to 5,000 mg/day (Abraham and Carroll 1981, Dahl 1960, Pennington *et al.* 1984). Discretionary sodium intake is variable and can be quite large. The Food and Drug Administration has found that most American adults tend to eat between 4,000 and 6,000 mg/day. Sodium-restricted diets range from below 1,000 to 3,000 mg/day (Kurtzweil 1995). The NRC recommended daily dietary intake for sodium is 2,400 mg/day.

Drinking water generally accounts for a relatively small proportion of total sodium intake. An estimated 75% of dietary sodium comes from the sodium in processed foods, 15% is from discretional use of table salt during cooking and serving of foods, and 10% is from sodium naturally present in foods (Sanchez-Castillo *et al.* 1987). Drinking water is not considered in dietary intake surveys.

b. Health end points. The primary health effect of concern from long term exposures to excess sodium is increased blood pressure (hypertension). A large body of evidence suggests that excessive sodium intake may contribute to age-related increases in blood pressure (NAS 1977, WHO 1979). High blood pressure is a multi-factorial disorder with dietary sodium as one of a number of factors influencing its incidence.

Frost *et al.* (1991) conducted an analysis of 14 published studies (12,773 subjects) from the U.S., Europe, and Asia, which measured blood pressure and sodium intake. The analysis indicated that there is a significant positive association between blood pressure and dietary sodium within populations. Elliot (1991) performed a similar analysis of 14 studies in 16 populations (12,503 subjects) relating 24-hour urinary sodium excretion and blood pressures. This analysis also showed a significant positive correlation between urinary sodium and both systolic and diastolic blood pressure for both males and females.

Sullivan (1991) analyzed data on 183 subjects to determine sodium sensitivity, which was defined as an increase of mean blood pressure of more than five percent when progressing from low- to high-sodium intake. Using this criterion, sodium sensitivity was detected in 15% of Caucasian subjects with normal blood pressure, 29% of Caucasian borderline hypertensive subjects, 27% of African-American subjects with normal blood pressure and 50% of African-American borderline hypertensive subjects.

Recent controlled studies of borderline hypertensive subjects called the Dietary Approaches to Stop Hypertension (DASH) trials demonstrated decreases in blood pressure with a diet that combined a moderate sodium intake (3,000 mg/day) with a high fruit and vegetable diet (DASH diet). The DASH diet was (two to three times) higher in potassium, calcium, magnesium, and fiber than the control diet. It reduced average blood pressures compared with the control diet in this clinical study (Vogt et al. 1999). When the study was repeated with differing degrees of salt restriction, small but additional decreases in blood pressure were observed for subjects on the sodium restricted DASH diet as opposed to subjects on the control diet (Sacks *et al.* 2001). These results add to the weight-of-evidence that sodium is not the only factor in the diet to consider when managing blood pressure.

Some clinical studies on the effect of decreased sodium intake on blood pressure have not detected convincing evidence of a protective effect of low sodium intake on the risk of cardiovascular disease (Muntzel and Drueke 1992, Salt Institute 2000, NIH 1993, Callaway 1994, Kotchen and McCarron 1998, McCarron 1998). Thus, it has been difficult to clearly define the role of sodium in the development of hypertension. Experts at the National Heart, Lung and Blood Institute, the scientific experts at the American Heart Association, American Society of Hypertension, and the European and International Societies of Hypertension do not feel that universal salt reduction is warranted for individuals with normal blood pressure (Taubes 1998). However, the National Institutes of Health, National Academy of Sciences, American Heart Association and U.S. Department of Agriculture all recommend restricting daily dietary sodium intake to 2.4 g/day or less, even though present average intake of most people exceed this value. The current outdated EPA guidance level for sodium in drinking water is 20 mg/L. It was developed to protect those individuals restricted to a total sodium intake of 500 mg/day (EPA, 1976). The recently updated guidance document, Draft Drinking Water Advisory: Consumer Acceptability Advice and Health Effects Analysis on Sodium, is available for review and comment at the EPA Water Docket. It is based on current health effects and occurrence data, includes the taste effects of sodium in drinking water, and allows EPA to provide appropriate guidance to water suppliers.

Ingestion of sodium ion is not believed to cause cancer. However, some studies suggest that sodium chloride may enhance risk of gastrointestinal tract cancer caused by other chemicals. Sodium salts have generally produced inconclusive results in in vitro or in vivo genotoxicity tests.

Very high doses of sodium chloride (1,667 mg/kg) have been observed to cause reproductive effects in various strains of pregnant rats. Effects on the pregnant rats have included decreases in pregnancy rates and maternal body weight gain. Effects in offspring have included increased blood pressure and high mortality. No studies on developmental effects from exposure to sodium were identified.

Benchmark Value. In the case of sodium, the value used to evaluate the occurrence data is not designated as an health reference level (HRL) because of the lack of suitable dose-response data and the considerable controversy regarding the role of sodium in the etiology of hypertension. Instead a benchmark value is used. The benchmark value for sodium was derived from the recommended daily dietary intake of 2.4 g/day (NRC 1989). It is important to note that the recommended intake is not related

directly to dose-response information and is lower than most estimates of the present average daily intake of the U.S. population. A relative source contribution of 10% was applied in recognition that foods and other discretional use of table salt are the major source of sodium exposure. This results in a benchmark value of 120 mg/L, assuming 2 liters of water per day (i.e., 2,400 mg/day/2L x 10% = 120 mg/L). The ½ benchmark value coincides with the upper limit of the concentration at which those who are sensitive to the taste of sodium chloride in water are able to detect the salt taste. The EPA derived benchmark value of 120 mg/L was used as a means for evaluating the occurrence data. This value is more conservative than the values used for evaluating the other regulatory determination contaminants in today's action. It was derived from the NRC dietary guideline (NRC 1989) for adults of 2,400 mg/day for sodium from salt rather than from the highest NOAEL in a toxicological study or even average dietary intake.

Potential susceptibility of life-stages and other sensitive populations. Several studies have shown that children are more sensitive than adults to the acute effects of high sodium intake (Elton et al. 1963, DeGenaro and Nyhan 1971). This increased sensitivity is associated with a lower ability of the immature kidney to control sodium levels compared to the adult. The elderly may be sensitive to the hypertensive effects of sodium because they have a higher incidence of cardiovascular disease (including high blood pressure) than younger subjects (Sowers and Lester 2000). African-Americans may also be more susceptible to sodium-induced adverse health effects due to high prevalence of hypertension and increased salt sensitivity characteristics in this population (Sullivan 1991, Svetkey *et al.* 1996). Individuals with decreased kidney function or kidney insufficiency are more sensitive to high sodium intake compared to individuals with healthy kidneys.

c. Occurrence and exposure. Sodium was detected in 100% (989 of 989) of the ground water PWS samples collected through the National Inorganics and Radionuclides Survey (NIRS). The median and the 99th percentile concentrations of all samples are 16.4 mg/L and 517 mg/L, respectively.

Analysis of NIRS samples shows 22.6% of the reporting ground water PWSs have detections > ½ the benchmark level (60 mg/L) (224 out of 989) affecting approximately 18.5% of the population served (274,000 out of 1.5 million people). The percentage of reporting ground water PWSs with detections > the benchmark level (120 mg/L) is 13.2% (131 out of 989), affecting approximately 8.3% of the population served (123,000 out of 1.5 million people).

Additional SDWA data from the States of Alabama, California, Illinois, New Jersey, and Oregon, including both ground water and surface water PWSs, were examined through independent analyses and also show substantial sodium occurrence. These data add an additional perspective to the NIRS estimates that only include data for ground water systems. The supplemental State data show that all five States reported almost 100% detections in both ground water and surface water systems. For all PWSs in the five States, the median concentrations of all samples ranged from 5.26 to 31 mg/L and 99th percentile concentrations of all samples ranged from 150 to 370 mg/L. Surface water PWS detection frequencies > the benchmark value are slightly lower than those for ground water.

d. Preliminary determination. The Agency has made a preliminary determination not to regulate sodium with a NPDWR since the relatively small amount of sodium in drinking water is not projected to cause adverse health effects in most individuals. This preliminary decision is based on the minor impact of sodium in drinking water. Drinking water generally accounts for a relatively small proportion of total sodium intake. Thus, restriction of the amount of sodium in drinking water would not present a meaningful opportunity for health risk reduction for persons served by PWSs.

Sodium intake is a matter of concern for salt-sensitive individuals with hypertension. However, blood pressure is greatly influenced by other nutrients in the diet, lifestyle, and behavioral factors in addition to sodium itself, and is best treated under medical supervision giving consideration to the multiple factors that contribute to the blood pressure problems.

EPA's Draft Drinking Water Advisory: Consumer Acceptability Advice and Health Effects Analysis for Sodium provides guidance to communities that may be exposed to elevated concentrations of sodium chloride or other sodium salts in their drinking water. The advisory provides appropriate cautions for individuals on low-sodium or sodium-restricted diets. It is based on current health effects and occurrence data, includes the taste effects of sodium in drinking water, and allows EPA to provide appropriate guidance to water suppliers.

EPA presently requires periodic monitoring of sodium at the entry point to the distribution system. Monitoring is to be conducted annually for surface water systems and every three years for ground water systems (as defined in 40 CFR 141.41). The water supplier must report sodium test results to local and State public health officials by direct mail within three months of the analysis, unless this responsibility is assumed by the State. This requirement provides the public health community with information on sodium levels in drinking water to be used in counseling patients and is the most direct route for gaining the attention of the affected population.

8. Sulfate

After reviewing the best available public health and occurrence information, EPA has made a preliminary determination not to regulate sulfate with a National Primary Drinking Water Regulation (NPDWR). EPA's finding is that sulfate may have adverse health affects on persons, primarily as a laxative effect following high acute exposures. EPA also finds that sulfate occurs in PWSs. Approximately 87% of the Round 2 Unregulated Contaminant Monitoring (UCM) samples showed detections of sulfate. Sulfate at >½ health reference level (HRL) was found at 4.97% of PWS surveyed in the Round 2 cross section samples, affecting 10.2% of the population served; at >HRL, it was found at 1.8% of the PWS, affecting 0.9% of the population served. EPA finds that the weight of evidence suggests that the risk of adverse health effects to the general population is limited, of short duration, and only occurs at high concentrations. Hence, the regulation of sulfate in drinking water does not present a meaningful opportunity for health risk reduction for persons served by PWSs. EPA is issuing a Drinking Water Advisory, with today's action, to provide guidance to communities that may be exposed to drinking water with high sulfate concentrations.

Detailed information supporting our finding and preliminary determination is provided in the Draft Drinking Water Advisory: Consumer Acceptability Advice and Health Effects Analysis on Sulfate, the Analysis of National Occurrence of the 1998 Contaminant Candidate List (CCL) Regulatory Determination Priority Contaminant in Public Water Systems, and the Regulatory Determination Support

Document for Sulfate. These findings are summarized later in this section.

a. Background. EPA was required by the 1986 SDWA amendments to issue a proposed and final standard for sulfate. EPA grouped sulfate with 23 other organic and IOCs in the ''Phase V'' regulatory package that was proposed in 1990 (55 FR 30371, July 25, 1990). The notice stated that the adverse health effect from ingesting high levels of sulfate is diarrhea and associated dehydration. Because local populations usually acclimate to high sulfate levels, the impact is primarily on infants, transient populations (e.g., business travelers, visitors, and vacationers), and new residents.

In the 1990 notice, EPA proposed alternative MCLG levels for sulfate of 400 mg/L and 500 mg/L. Given the high cost of the rule, the relatively low risk, and the need to explore alternative regulatory approaches targeted at the transient consumer, EPA deferred the final regulatory decision on sulfate. A new schedule was established, in connection with litigation, that required EPA to finalize its regulatory action for sulfate by May 1996. In December of 1994, EPA re-proposed the MCLG at 500 mg/L. Before the rule was promulgated, SDWA, as amended in 1996, directed EPA to determine by August 2001 whether to regulate sulfate in drinking water. In addition, section 1412(b)(12)(B) of SDWA directs EPA and the CDC to conduct a study, discussed in more detail later in this section, to establish a reliable dose-response relationship for the adverse human health effects from exposure to sulfate in drinking water, including the health effects that may be experienced by sensitive subpopulations (i.e., infants and travelers). SDWA specifies that the study be conducted using the best available peer-reviewed science in consultation with interested States, and completed by February 1999.

Sulfate (SO_4^{-2}, CASRN 14808–79–8) exists in a variety of inorganic salts. Sulfate salts such as sodium, potassium and magnesium are very water soluble and are often found in natural waters. Sulfate salts of metals such as barium, iron, or lead have very low water solubility.

Sulfate is found in soil, sediments and rocks and occurs in the environment as a result of both natural processes and human activities. Sulfate is used for a variety of commercial purposes, including pickle liquor (sulfuric acid) used in the steel and metal industries and as a reagent in the manufacturing of products such as copper sulfate (a fungicide/algicide). Specific data on the total production of all sulfates are not available, but production is expected to be in the thousands of tons per year.

Sulfate may enter surface or ground water as a result of discharge or disposal of sulfate-containing wastes. In addition, sulfur oxides produced during the combustion of fossil fuels are transformed to sulfuric acid in the atmosphere. Through precipitation (acid rain), sulfuric acid can enter surface waters, lowering the pH and raising sulfate levels.

Sulfate is present in the diet. A number of food additives are sulfate salts and most (such as copper sulfate and zinc sulfate) are approved for use as nutritional supplements.

EPA established a National Secondary Drinking Water Regulation for sulfate at 250 mg/L based on aesthetic effects (i.e., taste and odor) in 1979 (40 CFR part 43.3). This value was adopted from the 1962 Public Health Service Drinking Water Standards. The taste threshold for sulfate is reported to range from 200 to 900 mg/L depending on the specific sulfate salt. The threshold for unpleasant taste for sodium sulfate is about 800 to 1,000 mg/L, based on the results of a study by Heizer et al. (1997) and a study conducted under a cooperative agreement by the CDC and EPA (USEPA 1999c).

b. Health effects. Sulfate induces a laxative effect following high acute exposures (Anderson and Stothers 1978, Fingl 1980, Schofield and Hsieh 1983, Stephen *et al.* 1991, Cocchetto and Levy 1981, Gomez *et al.* 1995, Heizer *et al.* 1997). The concentrations of sulfate that induced these effects varied, but all occurred at concentrations >500 mg/L. A sulfate intake sufficient to produce a laxative effect when taken in one dose (5,400 mg) did not have the same effect when divided into four sequential hourly doses (Cocchetto and Levy 1981).

Chronic exposure to sulfate may not have the same laxative effect as an acute exposure since humans appear to develop a tolerance to drinking water with high sulfate concentrations (Schofield and Hsieh 1983). It is not known when this acclimation occurs; however in adults, acclimation is thought to occur within one to two weeks (USEPA 1999c).

Evidence indicates that sulfate concentrations do not exert adverse reproductive or developmental effects at concentrations as high as 5,000 mg/L (Andres and Cline 1989).

Although several studies (Peterson 1951, Moore 1952, Cass 1953) have been conducted on the long-term exposure of humans to sulfate in drinking water, none of them can be used to derive the relationship between a quantified exposure and adverse health effects (a dose-response characterization).

As required by SDWA, and discussed previously in this section, EPA and the CDC completed a study, ''Health Effects from Exposure to High Levels of Sulfate in Drinking Water Study'', (CDC and USEPA 1999b) in January 1999. The overall purpose of the Sulfate Study was to examine the association between consumption of tap water containing high levels of sulfate and reports of osmotic diarrhea (an increase in stool volume) in susceptible populations (infants and transients). Specifically, the CDC researchers designed field investigations of infants naturally exposed to high levels of sulfate in the drinking water provided by PWSs and an experimental trial of exposure in adults.

The CDC investigators were unable to study infants receiving their first bottles containing tap water with high levels of sulfate because the population of infants exposed to sulfate through their formula was not large enough to support the statistical requirements of such a study (USEPA 1999b). In the study of adult volunteers representing a transient population, the investigators did not find an association between acute exposure to sodium sulfate in tap water and reports of diarrhea. A total of 105 adult participants were randomly assigned to five sulfate-exposure groups (0, 250, 500, 800, and 1,200 mg/L) and were exposed to sulfate in bottled water over a period of six days. There was no significant dose-response association between acute exposure to sodium sulfate in water and reports of diarrhea. However, there was a weak (not statistically significant) increase in reports of increased stool volume at the highest dose level when it was compared to the combined lower doses.

As a supplement to the Sulfate Study, the CDC, in coordination with EPA, convened an expert workshop (USEPA 1999d), open to the public, in Atlanta, Georgia, on September 28, 1998 (64 CFR 7028). The expert scientists reviewed the available literature and the Sulfate Study results. They favored a health advisory for sulfate-containing drinking water at levels greater than 500 mg/L (USEPA 1999d). The most sensitive endpoint was considered by the panelists to be osmotic diarrhea. The panel noted that none of the reported data for humans identify laxative effects at concentrations of 500 mg/L or below. In most situations where laxative effects were observed at concentrations below 800 mg/L, the water contained other osmotically active contaminants such as magnesium or had been mixed with powdered infant formula. These data

suggest that the total concentration of osmotically active contaminants needs to be significantly higher than the 500 mg/L health-based advisory. The Agency used an HRL of 500 mg/L for evaluating the occurrence data, based on the recommendations of the CDC and EPA Panel (USEPA 1999d).

Potential susceptibility of life-stages and other sensitive populations. A potential sensitive population for dehydration resulting from diarrhea are infants receiving formula made with unfiltered tap water containing sulfate. Other groups include transient populations (i.e., tourists, hunters, students, and other temporary visitors) and people moving from areas with low sulfate drinking water concentrations into areas with high concentrations.

The health-based advisory value of 500 mg/L will protect against sulfate's laxative effects, even in formula-fed infants, in the absence of high concentrations of other osmotically active chemicals in the water. In situations where the water contains high concentrations of total dissolved solids and/or other osmotically active ions, laxative-like effects may occur if the water is mixed with concentrated infant formula or powdered nutritional supplements. In such situations, an alternate low-mineral-content water source is advised.

c. Occurrence and exposure. Sulfate was monitored under Round 2 of the UCM program. The State cross-section occurrence estimate is very high with 87% of the samples (35,221 of 40,484) showing detections. The median and the 99th percentile concentrations of all samples are 24 mg/L and 560 mg/L, respectively.

The Round 2 cross-section analysis shows that approximately 5% of the reporting PWSs have detections >½ HRL (820 out of 16,495 PWSs), affecting about 10.2% of the population served (5.1 million out of 50.4 million people). The percentage of the reporting PWSs with detections >HRL is approximately 1.8% (300 out of 16,495 PWSs), affecting about 0.9% of the population served (448,300 out of 50.4 million people).

Additional data from the States of Alabama, California, Illinois, Montana, New Jersey, and Oregon were examined. Of these States three had 99th percentile concentrations that exceeded the suggested HRL. A comparison between the 20-State cross-section data and the supplemental State data shows very similar results for sulfate detection frequencies in PWSs.

d. Preliminary determination. The Agency has made a preliminary determination not to regulate sulfate with a NPDWR since regulation would not present a meaningful opportunity for health risk reduction for persons served by public drinking water systems. This preliminary decision is based on the weight of evidence suggesting that the risk of adverse health effects to the general population is limited and acute (a short duration laxative-related response) and occurs at high drinking water concentrations (>500 mg/L, and in many cases >1,000 mg/L). In addition, people either develop a tolerance for high concentrations of sulfate in drinking water, or they decrease the amount of water they drink at one time, most likely because of the taste of the water (the taste threshold is 250 mg/L).

EPA intends to issue an advisory to provide guidance to communities that may be exposed to drinking water contaminated with high sulfate concentrations.

V. Specific Requests for Comment, Data or Information

EPA is requesting public comment on today's action. EPA intends to respond to the public comments it receives and issue final regulatory determinations in late 2002. If the Agency determines that regulations are warranted, the regulations would then need to be formally proposed within 24 months of the determination to regulate, and promulgated 18 months following the proposal.

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Dated: May 24, 2002.

Christine Todd Whitman,

Administrator.

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FEDERAL COMMUNICATIONS COMMISSION

47 CFR PART 73

[DA 02–1158, MB Docket No. 02–110, RM–10406]

Radio Broadcasting Services; Rose Hill and La Grange, NC

AGENCY: Federal Communications Commission.

ACTION: Proposed rule.

SUMMARY: This document requests comments on a petition filed by Conner Media, Inc. requesting the substitution of Channel 284C3 for Channel 284A at Rose Hill, North Carolina, reallotment of Channel 284C3 from Rose Hill, North Carolina, to La Grange, North Carolina, and modification of the license for Station WZUP to specify operation on Channel 284C3 at La Grange, North Carolina, as its community of license. The coordinates for Channel 284C3 at Rose Hill are 35–16–00 and 77–58–00. In accordance with Section 1.420(i) of the Commission's Rules, we shall not accept competing expressions of interest in the use of Channel 284C3 at La Grange.

DATES: Comments must be filed on or before July 8, 2002, and reply comments on or before July 23, 2002.

ADDRESSES: Federal Communications Commission, 445 Twelfth Street, SW, Washington, DC 20554. In addition to filing comments with the FCC, interested parties should serve the petitioner's counsel, as follows: Peter Gutmann, Pepper & Corazzini, 1776 K Street, NW, Suite 200, Washington, DC 20006.

FOR FURTHER INFORMATION CONTACT: Kathleen Scheuerle, Media Bureau, (202) 418–2180.

SUPPLEMENTARY INFORMATION: This is a summary of the Commission's Notice of Proposed Rule Making, MB Docket No. 02–110, adopted May 1, 2002, and released May 17, 2002. The full text of this Commission decision is available for inspection and copying during regular business hours at the FCC's Reference Information Center, Portals II, 445 12th Street, SW, Room CY–A257, Washington, DC, 20554. The complete text of this decision may also be purchased from the Commission's duplicating contractor, Qualex International, Portals II, 445 12th Street, SW, Room CY–B402, Washington, DC, 20554, telephone 202–863–2893, facsimile 202–863–2898, or via e-mail *qualexint@aol.com.* Provisions of the Regulatory Flexibility Act of 1980 do not apply to this proceeding. Members