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Drinking Water: Regulatory Determinations Regarding Contaminants on the Second Drinking Water Contaminant Candidate List—Preliminary Determinations; Proposed Rule

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 141

[EPA–HQ–OW–2007–0068 FRL–8301–3]

RIN 2040–AE58

Drinking Water: Regulatory Determinations Regarding Contaminants on the Second Drinking Water Contaminant Candidate List—Preliminary Determinations

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: The Safe Drinking Water Act (SDWA), as amended in 1996, requires the Environmental Protection Agency (EPA) to make regulatory determinations on at least five unregulated contaminants and decide whether to regulate these contaminants with a national primary drinking water regulation (NPDWR). SDWA requires that these determinations be made every five years. These unregulated contaminants are typically chosen from a list known as the Contaminant Candidate List (CCL), which SDWA requires the Agency to publish every five years. EPA published the second CCL (CCL 2) in the **Federal Register** on February 24, 2005 (70 FR 9071 (USEPA, 2005a)). This action presents the preliminary regulatory determinations for 11 of the 51 contaminants listed on CCL 2 and describes the supporting rationale for each. The preliminary determination is that an NPDWR is not appropriate for any of the 11 contaminants considered for regulatory determinations. The Agency seeks comment on these 11 preliminary determinations. While the Agency has not made a preliminary determination for perchlorate, this action provides an update on the Agency's evaluation of perchlorate. The Agency requests public comment on the information and the options that the Agency is considering in evaluating perchlorate and welcomes the submission of relevant, new information and/or data that may assist the Agency in its regulatory determination.

DATES: Comments must be received on or before July 2, 2007.

ADDRESSES: Submit your comments, identified by Docket ID No. EPA–HQ–OW–2007–0068, by one of the following methods:

<bullet≤ *http://www.regulations.gov:* Follow the online instructions for submitting comments.

<bullet≤ *Mail:* Water Docket, Environmental Protection Agency, Mailcode: 2822T, 1200 Pennsylvania Ave., NW., Washington, DC 20460.

<bullet≤ *Hand Delivery:* Water Docket, EPA Docket Center (EPA/DC). Such deliveries are only accepted during the Docket's normal hours of operation, and special arrangements should be made for deliveries of boxed information.

Instructions: Direct your comments to Docket ID No. EPA–HQ–OW–2007–0068. EPA's policy is that all comments received will be included in the public docket without change and may be made available online at *http://www.regulations.gov,* including any personal information provided, unless the comment includes information claimed to be Confidential Business Information (CBI) or other information whose disclosure is restricted by statute. Do not submit information that you consider to be CBI or otherwise protected through *http://www.regulations.gov.* The *http://www.regulations.gov* Web site is an ''anonymous access'' system, which means EPA will not know your identity or contact information unless you provide it in the body of your comment. If you send an e-mail comment directly to EPA without going through *http://www.regulations.gov* your e-mail address will be automatically captured and included as part of the comment that is placed in the public docket and made available on the Internet. If you submit an electronic comment, EPA recommends that you include your name and other contact information in the body of your comment and with any disk or CD–ROM you submit. If EPA cannot read your comment due to technical difficulties and cannot contact you for clarification, EPA may not be able to consider your comment. Electronic files should avoid the use of special characters, any form of encryption, and be free of any defects or viruses. For additional instructions on submitting comments, go to Unit I.B of the **SUPPLEMENTARY INFORMATION** section of this document.

Docket: All documents in the docket are listed in the *http://www.regulations.gov* index. Although listed in the index, some information is not publicly available, *e.g.*, CBI or other information whose disclosure is restricted by statute. Certain other material, such as copyrighted material, will be publicly available only in hard copy. Publicly available docket materials are available either electronically in *http://www.regulations.gov* or in hard copy at the Water Docket, EPA/DC, EPA West, Room 3334, 1301 Constitution Ave., NW., Washington, DC. The Public Reading Room is open from 8:30 a.m. to 4:30 p.m., Monday through Friday, excluding legal holidays. The telephone number for the Public Reading Room is (202) 566–1744, and the telephone number for the EPA Docket Center is (202) 566–2426.

FOR FURTHER INFORMATION CONTACT: Wynne Miller, Office of Ground Water and Drinking Water, Standards and Risk Management Division, at (202) 564–4887 or e-mail *miller.wynne@epa.gov.* For general information contact the EPA Safe Drinking Water Hotline at (800) 426–4791 or e-mail: *hotline-sdwa@epa.gov.*

SUPPLEMENTARY INFORMATION:

Abbreviations and Acronyms

a. i.—active ingredient
<—less than
<=—less than or equal to
≤—greater than
≤=—greater than or equal to
[mu]—microgram, one-millionth of a gram
[mu]g/g—micrograms per gram
[mu]g/kg—micrograms per kilogram
[mu]g/L—micrograms per liter
ATSDR—Agency for Toxic Substances and Disease Registry
AWWARF—American Water Works Association Research Foundation
BMD—bench mark dose
BMDL—bench mark dose level
BW—body weight for an adult, assumed to be 70 kilograms (kg)
CASRN—Chemical Abstract Services Registry Number
CBI—confidential business information
CDC—Centers for Disease Control and Prevention
ChE—cholinesterase
CCL—Contaminant Candidate List
CCL 1—EPA's First Contaminant Candidate List
CCL 2—EPA's Second Contaminant Candidate List
CFR—Code of Federal Regulations
CMR—Chemical Monitoring Reform
CWS—community water system
1,3-DCP—1,3-dichloropropene
DCPA—dimethyl tetrachloroterephthalate (dacthal)
DDE—1,1-dichloro-2,2-bis(p-chlorophenyl)ethylene
DDT—1,1,1-trichloro-2,2-bis(p-chlorophenyl)ethane
DNT—dinitrotoluene
DW—dry weight
DWEL—drinking water equivalent level
DWI—drinking water intake, assumed to be 2 L/day
EPA—United States Environmental Protection Agency
EPCRA—Emergency Planning and Community Right-to-Know Act
EPTC—s-ethyl dipropylthiocarbamate
ESA—ethane sulfonic acid
FDA—United States Food and Drug Administration
FQPA—Food Quality Protection Act
FR—Federal Register
FW—fresh weight
g—gram
g/day—grams per day

HRL—health reference level
IOC—inorganic compound
IRIS—Integrated Risk Information System
kg—kilogram
L—liter
LD_{50}—an estimate of a single dose that is expected to cause the death of 50 percent of the exposed animals; it is derived from experimental data.
LOAEL—lowest-observed-adverse-effect level
MAC—*mycobacterium avium intercellulare*
MCL—maximum contaminant level
MCLG—maximum contaminant level goal
mg—milligram, one-thousandth of a gram
mg/kg—milligrams per kilogram body weight
mg/kg/day—milligrams per kilogram body weight per day
mg/L—milligrams per liter
mg/m^3—milligrams per cubic meter
MRL—minimum or method reporting limit (depending on the study or suvey cited)
MTBE—methyl tertiary butyl ether
MTP—monomethyl-2,3,5,6-tetrachloroterephthalate
N—number of samples
NAS—National Academies of Sciences
NAWQA—National Water Quality Assessment (USGS Program)
NCEH—National Center for Environmental Health (CDC)
NCFAP—National Center for Food and Agricultural Policy
NCI—National Cancer Institute
NCWS—non community water system
ND—not detected (or non detect)
NDWAC—National Drinking Water Advisory Council
NHANES—National Health and Nutrition Examination Survey (CDC)
NIRS—National Inorganic and Radionuclide Survey
NIS—sodium iodide symporter
NOEL—no-observed-effect-level
NOAEL—no-observed-adverse-effect level
NPS—National Pesticide Survey
NQ—not quantifiable (or non quantifiable)
NRC—National Research Council
NPDWR—National Primary Drinking Water Regulation
NTP—National Toxicology Program
OA—oxanilic acid
OW—Office of Water
OPP—Office of Pesticide Programs
PCR—Polymerase Chain Reaction
PGWDB—pesticides in ground water data base
PWS—public water system
RED—Reregistration Eligibility Decision
RfC—reference concentration
RfD—reference dose
RSC—relative source contribution
SAB—Science Advisory Board
SDWA—Safe Drinking Water Act
SOC—synthetic organic compound
SVOC—semi-volatile organic compound
T3—triiodothyronine
T4—thyroxine
TDS—Total Diet Study (FDA)
Tg-DNT—technical grade DNT
TPA—2,3,5,6-tetrachchloroterephthalic acid
TRI—Toxics Release Inventory
TSH—thyroid stimulating hormone
TT—treatment technique
UCM—Unregulated Contaminant Monitoring
UCMR 1—First Unregulated Contaminant Monitoring Regulation
UF—uncertainty factor
US—United States of America
USDA—United States Department of Agriculture
USGS—United States Geological Survey
UST—underground storage tanks
VOC—volatile organic compound

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I. General Information

A. Does This Action Impose Any Requirements on My Public Water System?

None of these preliminary regulatory determinations or the final regulatory determinations, when published, will impose any requirements on anyone. Instead, this action notifies interested parties of the availability of EPA's preliminary regulatory determinations for 11 of the 51 contaminants listed on CCL 2 and seeks comment on these preliminary determinations. This action also provides an update on the Agency's review of perchlorate and methyl tertiary butyl ether (MTBE).

B. What Should I Consider as I Prepare My Comments for EPA?

You may find the following suggestions helpful for preparing your comments:

1. Explain your views as clearly as possible.
2. Describe any assumptions that you used.
3. Provide any technical information and/or data you used that support your views.
4. If you estimate potential burden or costs, explain how you arrived at your estimate.
5. Provide specific examples to illustrate your concerns.
6. Offer alternatives.
7. Make sure to submit your comments by the comment period deadline.
8. To ensure proper receipt by EPA, identify the appropriate docket identification number in the subject line on the first page of your response. It would also be helpful if you provided the name, date, and **Federal Register** citation related to your comments.

II. Purpose, Background and Summary of This Action

This section briefly summarizes the purpose of this action, the statutory requirements, previous activities related to the Contaminant Candidate List and regulatory determinations, and the approach used and outcome of these preliminary regulatory determinations.

A. What Is the Purpose of This Action?

The Safe Drinking Water Act (SDWA), as amended in 1996, requires EPA to publish a list of currently unregulated contaminants that may pose risks for drinking water (referred to as the Contaminant Candidate List, or CCL) and to make determinations on whether to regulate at least five contaminants from the CCL with a national primary drinking water regulation (NPDWR)

(section 1412(b)(1)). The 1996 SDWA requires the Agency to publish both the CCL and the regulatory determinations every five years. The purpose of this action is to present (1) EPA's preliminary regulatory determinations for 11 candidates selected from the 51 contaminants listed on the second CCL (CCL 2), (2) the process and the rationale used to make these determinations, and (3) a brief summary of the supporting documentation. This action also includes a request for comment(s) on the Agency's preliminary determinations.

The 11 regulatory determination contaminants candidates discussed in this action are boron, the dacthal mono- and di-acid degradates, 1,1-dichloro-2,2-bis(p-chlorophenyl)ethylene (DDE), 1,3-dichloropropene, 2,4-dinitrotoluene, 2,6-dinitrotoluene, s-ethyl propylthiocarbamate (EPTC), fonofos, terbacil, and 1,1,2,2-tetrachloroethane.

B. Background on the CCL and Regulatory Determinations

1. Statutory Requirements for CCL and Regulatory Determinations. The specific statutory requirements for the CCL and regulatory determinations can be found in SDWA section 1412(b)(1). The 1996 SDWA Amendments require EPA to publish the CCL every five years. The CCL is a list of contaminants that are not subject to any proposed or promulgated NPDWRs, are known or anticipated to occur in public water systems (PWSs), and may require regulation under SDWA. The 1996 SDWA Amendments also direct EPA to determine whether to regulate at least five contaminants from the CCL every five years (within three and one-half years after publication of the final list). In making regulatory determinations, SDWA requires EPA to publish a Maximum Contaminant Level Goal [1] (MCLG) and promulgate an NPDWR [2] for a contaminant if the Administrator determines that:

(a) The contaminant may have an adverse effect on the health of persons;

(b) the contaminant is known to occur or there is a substantial likelihood that the contaminant will occur in public water systems with a frequency and at levels of public health concern; and

(c) 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.

If EPA determines that all three of these statutory criteria are met and makes a final determination that a national primary drinking water regulation is needed, the Agency has 24 months to publish a proposed MCLG and NPDWR. After the proposal, the Agency has 18 months to publish and promulgate a final MCLG and NPDWR (SDWA section 1412(b)(1)(E)).[3]

2. The First Contaminant Candidate List (CCL 1). Following the 1996 SDWA Amendments, EPA sought input from the National Drinking Water Advisory Council (NDWAC) on the process that should be used to identify contaminants for inclusion on the CCL. For chemical contaminants, the Agency developed screening and evaluation criteria based on recommendations from NDWAC. For microbiological contaminants, NDWAC recommended that the Agency seek external expertise to identify and select potential waterborne pathogens. As a result, the Agency convened a workshop of microbiologists and public health experts who developed criteria for screening and evaluation and subsequently developed an initial list of potential microbiological contaminants.

The first CCL process benefited from considerable input from the NDWAC, the scientific community, and the public through stakeholder meetings and the public comments received on the draft CCL published on October 6, 1997 (62 FR 52193 (USEPA, 1997a)). EPA published the final CCL, which contained 50 chemical and 10 microbiological contaminants, on March 2, 1998 (63 FR 10273 (USEPA, 1998a)). A more detailed discussion of how EPA developed CCL 1 can be found in the 1997 and the 1998 **Federal Register** notices (62 FR 52193 (USEPA, 1997a) and 63 FR 10273 (USEPA, 1998a)).

3. The Regulatory Determinations for CCL 1. EPA published its preliminary regulatory determinations for a subset of contaminants listed on CCL 1 on June 3, 2002 (67 FR 38222 (USEPA, 2002a)). The Agency published its final regulatory determinations on July 18, 2003 (68 FR 42898 (USEPA, 2003a)). EPA identified 9 contaminants from the 60 contaminants listed on CCL 1 that had sufficient data and information available to make regulatory determinations. The 9 contaminants were *Acanthamoeba*, aldrin, dieldrin, hexachlorobutadiene, manganese, metribuzin, naphthalene, sodium, and sulfate. The Agency determined that a national primary drinking water regulation was not necessary for any of these 9 contaminants. The Agency issued guidance on *Acanthamoeba* and health advisories for magnesium, sodium, and sulfate.

The decision-making process that EPA used to make its regulatory determinations for CCL 1 was based on substantial expert input and recommendations from different groups including stakeholders, the National Research Council (NRC) and NDWAC. In June 2002, EPA consulted with the Science Advisory Board (SAB) Drinking Water Committee and requested its review and comment on whether the protocol EPA developed, based on the NDWAC recommendations, was consistently applied and appropriately documented. SAB provided verbal feedback regarding the use of the NRC and NDWAC recommendations in EPA's decision criteria for making its regulatory determinations. SAB recommended that the Agency provide a transparent and clear explanation of the process for making regulatory determinations. The Agency took SAB's recommendation into consideration and further explained the CCL 1 regulatory determination evaluation process in the July 18, 2003 (68 FR 42898 (USEPA, 2003a)) notice and in the supporting documentation.

EPA has used the same approach to develop the regulatory determinations discussed in this action. While this action includes a short description of the decision process used to make regulatory determinations (section II.C), a more detailed discussion can be found in the 2002 and the 2003 **Federal Register** notices (67 FR 38222 (USEPA, 2002a) and 68 FR 42898 (USEPA, 2003a)).

4. The Second Contaminant Candidate List (CCL 2). The Agency published its draft CCL 2 **Federal Register** notice on April 2, 2004 (69 FR 17406 (USEPA, 2004a)) and the final CCL 2 **Federal Register** notice on February 24, 2005 (70 FR 9071 (USEPA, 2005a)). The CCL 2 carried forward the 51 remaining chemical and microbial contaminants that were listed on CCL 1.

5. The Regulatory Determinations for CCL 2. This current action discusses EPA's preliminary determinations for 11 of the 51 contaminants listed on the CCL 2.

[1] The MCLG is the ''maximum level of a contaminant in drinking water at which no known or anticipated adverse effect on the health of persons would occur, and which allows an adequate margin of safety. Maximum contaminant level goals are nonenforceable health goals'' (40 CFR 141.2).

[2] An NPDWR is a legally enforceable standard that applies to public water systems. An NPDWR sets a legal limit (called a maximum contaminant level or MCL) or specifies a certain treatment technique (TT) for public water systems for a specific contaminant or group of contaminants.

[3] The statute authorizes a nine month extension of this promulgation date.

C. Summary of the Approach Used To Identify and Evaluate Candidates for Regulatory Determination 2

Figure 1 provides a brief overview of the process EPA used to identify which CCL 2 contaminants are candidates for regulatory determinations and the SDWA statutory criteria considered in making the regulatory determinations.

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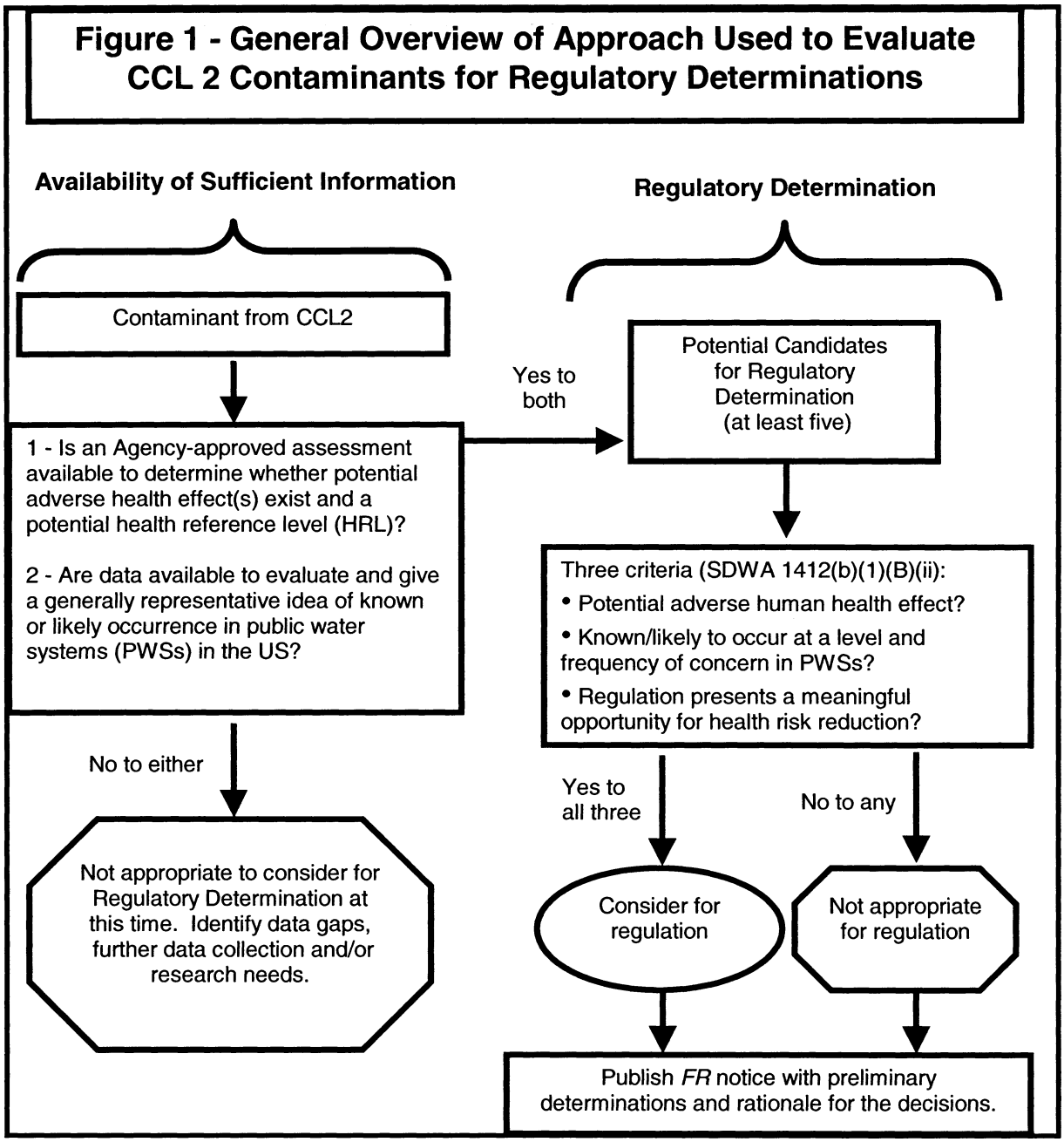

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In identifying which CCL 2 contaminants are candidates for regulatory determinations, the Agency considered whether sufficient information and/or data were available to characterize the potential health effects and the known/likely occurrence in and exposure from drinking water. With regards to sufficient health effects information/data, the Agency considered whether an Agency-approved health risk assessment [4] was available to identify any potential adverse health effect(s) and derive an estimated level at which adverse health effect(s) are likely to occur. With regards to sufficient occurrence information/data, the Agency considered whether information/data were available to

[4] Health information used for the regulatory determinations process includes but is not limited to health assessments available from the Agency's Integrated Risk Information System (IRIS), the Agency's Office of Pesticide Programs (OPP) in a Reregistration Eligibility Decision (RED), the National Academy of Sciences (NAS), and/or the Agency for Toxic Substances and Disease Registry (ATSDR).

evaluate and give a generally representative idea of known and/or likely occurrence in public water systems. If sufficient information/data were available to characterize adverse human health effects and known/likely occurrence in public water systems, the Agency identified the contaminant as a potential candidate for regulatory determinations. In addition to information/data for health and occurrence, EPA also considered the availability and adequacy of analytical methods (for monitoring) and treatment.

If EPA chose a contaminant as a candidate for regulatory determination, the Agency used an approach similar to the first regulatory determination process to answer the three statutory criteria (listed in section II.B.1).

For the current regulatory determination process, the Agency considered the following in evaluating each of the three statutory criteria.

(1) First statutory criterion—Is the contaminant likely to cause an adverse effect on the health of persons? The Agency evaluated the best available, peer-reviewed assessments and studies to characterize the human health effects that may result from exposure to the contaminant when found in drinking water. Based on this characterization, the Agency estimated a health reference level (HRL) for each contaminant. Section III.A provides more detailed information about the approach used to evaluate and analyze the health information.

(2) Second statutory criterion—Is the contaminant known or likely to occur in public water systems at a frequency and level of concern? To evaluate known occurrence in PWSs, the Agency compiled, screened, and analyzed data from several occurrence data sets to develop representative occurrence estimates for public drinking water systems. EPA used the HRL estimates for each contaminant as a benchmark against which to conduct an initial evaluation or screening of the occurrence data. For each contaminant, EPA estimated the number of PWSs (and the population served by these PWSs) with detections greater than one-half the HRL (≤ 1/2 HRL) and greater than the HRL (≤ HRL). To evaluate the likelihood of a contaminant to occur in drinking water, the Agency considered information on the use and release of a contaminant into the environment and supplemental information on occurrence in water (*e.g.*, ambient water quality data, State ambient or finished water data, and/or special studies performed by other agencies, organizations and/or entities). Section III.B provides more details on the approach used to analyze the occurrence information/data.

(3) Third statutory criterion—In the sole judgment of the Administrator, does regulation of the contaminant present a meaningful opportunity for health risk reduction for persons served by public water systems? EPA evaluated the potential health effects and the results of the occurrence and exposure estimates (i.e., the population exposed and the sources of exposure) at the health level of concern to determine if regulation presents a meaningful opportunity for health risk reduction. EPA has made a preliminary determination regarding the meaningful opportunity for health risk reduction for 11 contaminants based upon the population exposed to these contaminants at levels of concern.

If the answers to all three statutory criteria are affirmative for a particular contaminant, then the Agency makes a determination that a national drinking water regulation is necessary and proceeds to develop an MCLG and a national primary drinking water regulation for that contaminant. It should be noted that this regulatory determination process is independent of the more detailed analyses needed to develop a national primary drinking water regulation. Thus, a decision to regulate is the beginning of the Agency regulatory development process, not the end.

If the answer to any of the three statutory criteria is negative, then the Agency makes a determination that a national drinking water regulation is not necessary for that contaminant.

D. What Are EPA's Preliminary Determinations and What Happens Next?

EPA has made preliminary determinations that no regulatory actions are appropriate for the 11 contaminants evaluated for this second round of regulatory determinations. EPA will make final determinations on these 11 contaminants after a 60-day comment period. EPA is making preliminary regulatory determinations only on those CCL 2 contaminants that have sufficient information to support such a determination at this time. The Agency continues to conduct research and/or to collect information on the remaining CCL 2 contaminants to fill identified data gaps. 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.

E. Supporting Documentation for EPA's Preliminary Determinations

For this action, EPA prepared several support documents that are available for review and comment in the EPA Water Docket and at *http://www.regulations.gov*. These support documents include:

<bullet≤ A comprehensive regulatory support document entitled, ''Regulatory Determinations Support Document for Selected Contaminants from the Second Drinking Water Contaminant Candidate List'' (CCL 2) (USEPA, 2006a). This support document summarizes the information and data on the physical and chemical properties, uses and environmental release, environmental fate, potential health effects, occurrence and exposure estimates, the preliminary determination for each contaminant candidate, and the Agency's rationale for its determination. The technical health and occurrence support documents listed next served as the basis for the health information and the drinking water occurrence estimates summarized in this comprehensive regulatory support document.

<bullet≤ Technical health support documents. These documents address exposure from drinking water and other media, toxicokinetics, hazard identification, and dose-response assessment, and provide an overall characterization of the risk from drinking water for the contaminants considered for regulatory determination. These documents are listed in the reference section as ''USEPA, 2006j'' through ''USEPA, 2006r.''

<bullet≤ Technical occurrence support documents (USEPA, 2006b and USEPA, 2006c). These documents include more detailed information about the sources of the data, how EPA assessed the data quality, completeness, and representativeness, and how the data were used to generate estimates of drinking water contaminant occurrence in support of these regulatory determinations. Section III.B.3 provides more information about the title and content of these technical support documents.

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

Sections III.A and B of this action outline the health effects and occurrence/exposure evaluation process EPA used to support these preliminary determinations.

A. Evaluation of Adverse Health Effects

Section 1412(b)(1)(A)(i) of SDWA requires EPA to determine whether each

candidate contaminant may have an adverse effect on public health. This section describes the overall process the Agency used to evaluate health effects information, the approach used to estimate a contaminant HRL (a benchmark against which to conduct the initial evaluation of the occurrence data), and the approach used to identify and evaluate information on hazard and dose-response for the contaminants under consideration. More specific information about the potential for adverse health effects for each contaminant is presented in section IV.B of this action.

There are two different approaches to the derivation of an HRL. One approach is used for chemicals that cause cancer and exhibit a linear response to dose and the other applies to noncarcinogens and carcinogens evaluated using a non-linear approach.

1. Use of Carcinogenicity Data for the Derivation of a Health Reference Level. For those contaminants considered to be likely or probable 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 linear low dose extrapolation is appropriate or when data on the mode of action are lacking, EPA uses a low dose linear extrapolation to calculate risk-specific doses. The risk-specific doses are the estimated oral exposures associated with lifetime excess risk levels that range from one cancer in ten thousand (10^{-4}) to one cancer in a million (10^{-6}). The risk-specific doses (expressed as mg/kg of body weight per day) are combined with adult body weight and drinking water consumption data to estimate drinking water concentrations corresponding to this risk range. EPA generally used the one-in-a-million (10^{-6}) cancer risk in the initial screening of the occurrence data for carcinogens evaluated using linear low dose extrapolation. Five of the eleven contaminants discussed in this action had data available to classify them as likely or probable human carcinogens. These five are also the only contaminants for which low dose linear extrapolations were performed. These five are p,p-dichlorodiphenyldichloroethylene (DDE), 1,3-dichloropropene (1,3-DCP or Telone), 2,4-dinitrotoluene, 2,6-dinitrotoluene, and 1,1,2,2-tetrachloroethane. The remaining 6 contaminants have not been identified as known, likely or probable carcinogens.

2. Use of Non-carcinogenic Health Effects Data for Derivation of an HRL. For those chemicals not considered to be carcinogenic to humans, EPA generally calculates a reference dose (RfD). A 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 either a ''no-observed-adverse-effect level'' (NOAEL), a ''lowest-observed-adverse-effect level'' (LOAEL), or a benchmark dose, with uncertainty factors applied to reflect limitations of the data used.

The Agency uses uncertainty factors (UFs) to address uncertainty resulting from incompleteness of the toxicological database. The individual UFs (usually applied as integers of 1, 3, or 10) are multiplied together and used to derive the RfD from experimental data. Individual 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/or

(5) the uncertainty associated with an incomplete database.

For boron, the dacthal (DCPA) mono and di acid degradates, s-ethyl dipropylthiocarbamate (EPTC), fonofos and terbacil, EPA derived the HRLs using the RfD approach as follows:

$$HRL = [(RfD \times BW)/DWI] \times RSC$$

Where:

RfD = Reference Dose

BW = Body Weight for an adult, assumed to be 70 kilograms (kg)

DWI = Drinking Water Intake, assumed to be 2 L/day (90th percentile)

RSC = Relative Source Contribution, or the level of exposure believed to result from drinking water when compared to other sources (*e.g.*, food, ambient air). A 20 percent RSC is being used to estimate the HRL and screen the occurrence data because it is the lowest and most conservative RSC used in the derivation of an MCLG for drinking water. For each of the 6 aforementioned non-carcinogenic compounds for which the Agency has made a preliminary regulatory determination in this action, EPA used the RfD in conjunction with a 20 percent RSC to derive a conservative HRL estimate and perform an initial screening of the drinking water occurrence data. Since the initial screening of the occurrence data at this conservative HRL value resulted in a preliminary negative determination for each of these 6 compounds, the Agency determined that it was not necessary to further evaluate the RSC in making the regulatory determination.

As discussed in section IV.B.2 and 3, the HRL for the two dacthal degradates is based on the HRL value derived for the DCPA parent following the guidance provided by EPA's Office of Pesticide Programs.

3. Sources of Data/Information for Health Effects. EPA used the best available peer-reviewed data and analyses in evaluating adverse health effects. Peer-reviewed health-risk assessments were available for all chemicals considered for regulatory determinations from the Agency's Integrated Risk Information System (IRIS) Program[5] and/or the Office of Pesticide Programs (OPP) Reregistration Eligibility Decisions (RED).[6] Table 1 summarizes the sources of the health assessment data for each chemical under regulatory determination consideration. The Agency performed a literature search for studies published after the IRIS or OPP health-risk assessment was completed to determine if new information suggested a different outcome. The Agency collected and evaluated any peer-reviewed publications identified through the literature search for their impact on the RfD and/or cancer assessment. In cases where the recent data indicated that a change to the existing RfD or cancer assessment was needed, the updated OW assessment, as described in the health effects support document, was independently peer-reviewed. All quantitative cancer assessments conducted under the Guidelines for Carcinogen Risk Assessment (51 FR 33992 (USEPA, 1986)) were updated using the Guidelines for Carcinogen Risk Assessment (USEPA, 1999a) as directed in the November 2001 (66 FR 59593 (USEPA, 2001a)) **Federal Register** notice.

In March 2005, EPA updated and finalized the Cancer Guidelines and a Supplementary Children's Guidance,

[5] IRIS is an electronic EPA database (*http://www.epa.gov/iris/index.html*) 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.

[6] The OPP is required under the Federal Insecticide Fungicide and Rodenticide Act (FIFRA) to review all pesticides registered prior to 1984 and determine whether to reregister them for continued use. The results of the reregistration analysis are included in the REDs. Copies of the REDs are located at the following Web site: *http://cfpub.epa.gov/oppref/rereg/status.cfm?show=rereg.*

which include new considerations for mode of action and added guidelines related to potential risks due to early childhood exposure (USEPA, 2005b; USEPA, 2005c). EPA updated the earlier assessments (based on the 1986 Guidelines) for DDE, the dinitrotoluenes (2,4 and 2,6 as a mixture), and 1,1,2,2-tetrachloroethane following the 1999 Guidelines. None of these chemicals have been determined to have a mutagenic mode of action, which would require an extra factor of safety for children's health protection. Therefore, conducting the cancer evaluation using the 2005 Cancer Guidelines would not result in any change from the assessment updated following the 1999 Guidelines.

The cancer assessment for 1,3-dichloropropene was done by OPP and IRIS (USEPA, 1998b and 2000a) under the Proposed Guidelines for Carcinogen Risk Assessment (61 FR 17960 (USEPA, 1996a)). The Administrator (USEPA, 2005d) has directed that current completed assessments can be considered to be scientifically sound based on the guidance used when the assessment was completed until a new assessment is performed by one of the responsible program offices.

TABLE 1.—SOURCES AND DATES OF EPA HEALTH RISK ASSESSMENTS

Chemical	IRIS	Date	OPP RED	Date
Boron	X	2004		
Dacthal and its mono- and di-acid degradates	X	1994	X	1998
1,3-Dichloropropene	X	2000	X	1998
DDE	X	1988		
2,4-Dinitrotoluene	X	1990/1992		
2,6-Dinitrotoluene	* X	1990		
EPTC	X	1990	X	1999
Fonofos	X	1991	** X	1996
Terbacil	X	1989	X	1998
1,1,2,2-Tetrachloroethane	X	1986		

* Applies to a mixture of 98 percent 2,4-dinitrotoluene and 2 percent 2,6-dinitrotoluene.
** Health Risk Assessment; RED not completed due to pesticide cancellation.

As noted in section II.E, EPA has prepared several technical health effects support documents for the contaminants considered for this round of regulatory determinations. These documents address the exposure from drinking water and other media, toxicokinetics, hazard identification, and dose-response assessment, and provide an overall characterization of risk from drinking water.

B. Evaluation of Contaminant Occurrence and Exposure

EPA used data from several sources to evaluate occurrence and exposure for the 11 contaminants considered in these regulatory determinations. The major or primary sources of the drinking water occurrence data used to support these determinations include the following sources:

<bullet≤ The first Unregulated Contaminant Monitoring Regulation (UCMR 1),

<bullet≤ The Unregulated Contaminant Monitoring (UCM) program, and

<bullet≤ The National Inorganic and Radionuclide Survey (NIRS).

In addition to these primary sources of occurrence data, the Agency also evaluated supplemental sources of occurrence information. Section III.B.1 of this action provides a brief summary of the primary sources of drinking water occurrence data and section III.B.2 provides brief summary descriptions of the supplemental sources of occurrence information and/or data. A summary of the occurrence data and the results or findings for each of the 11 contaminants considered for regulatory determination is presented in Section IV.B, the contaminant profiles section.

1. Primary Data Sources. As previously mentioned, the primary sources of the drinking water occurrence data used to support this action are the UCMR 1, the UCM program, and NIRS. The following sections provide a brief summary of the data sources and the approach used to estimate a given contaminant's occurrence. Table 2 lists the primary data sources the Agency used for each of the 11 contaminants considered for regulatory determinations.

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

Number	Contaminant	Primary data sources				
		UCMR 1		UCM		NIRS
		List 1 assessment monitoring	List 2 screening survey	Round 1 cross section	Round 2 cross section	
1	Boron					[1] X
2	Dacthal mono- and					
3	di-acid degradates	X				
4	DDE	X				
5	1,3-Dichloropropene	[2] X		X	X	
6	2,4-Dinitrotoluene	X				
7	2,6-Dinitrotoluene	X				
8	EPTC	X				
9	Fonofos		X			
10	Terbacil	X				

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

Number	Contaminant	Primary data sources				
		UCMR 1		UCM		NIRS
		List 1 assessment monitoring	List 2 screening survey	Round 1 cross section	Round 2 cross section	
11	1,1,2,2-Tetrachloroethane			X	X	

[1] For boron, EPA also considered the results of a study funded by AWWARF (Frey *et al.*, 2004).
[2] 1,3-Dichloropropene was sampled as a UCM Round 1 and 2 analyte but due to sample degradation concerns the contaminant was re-analyzed using the samples provided by the small systems that participated in the UCMR 1 List 1 Assessment Monitoring.

a. *The Unregulated Contaminant Monitoring Regulation.* In 1999, EPA developed the UCMR program in coordination with the CCL and the National Drinking Water Contaminant Occurrence Database (NCOD) to provide national occurrence information on unregulated contaminants (September 17, 1999, 64 FR 50556 (USEPA, 1999b); March 2, 2000, 65 FR 11372 (USEPA, 2000b); and January 11, 2001, 66 FR 2273 (USEPA, 2001b)). EPA used data from the UCMR 1 program to evaluate occurrence for 9 of the 11 contaminants considered for these regulatory determinations. These 9 contaminants include the dacthal mono- and di-acid degradates, DDE, 1,3-dichloropropene, 2,4-dinitrotoluene, 2,6-dinitrotoluene, EPTC, fonofos, and terbacil.

EPA designed the UCMR 1 data collection with three parts (or tiers) primarily based on the availability of analytical methods. Occurrence data for 8 of the 9 contaminants listed in the preceding paragraph are from the first tier of UCMR (also known as UCMR 1 List 1 Assessment Monitoring). Occurrence data for fonofos are from the second tier of UCMR 1 (also known as the UCMR 1 List 2 Screening Survey). EPA has not collected data as part of the third tier due to the lack of adequate analytical methods.

The UCMR 1 List 1 Assessment Monitoring was performed for a specified number of chemical contaminants for which analytical methods have been developed. EPA required all large[7] PWSs, plus a statistically representative national sample of 800 small [8] PWSs to conduct Assessment Monitoring.[9] Approximately one-third of the participating small systems were scheduled to monitor for these contaminants during each calendar year from 2001 through 2003. Large systems could conduct one year of monitoring anytime during the 2001–2003 UCMR 1 period. EPA specified a quarterly monitoring schedule for surface water systems and a twice-a-year, six-month interval monitoring schedule for ground water systems. The objective of the UCMR 1 sampling approach for small systems was to collect contaminant occurrence data from a statistically selected, nationally representative sample of small systems. The small system sample was stratified and population-weighted, and included some other sampling adjustments such as allocating a selection of at least 2 systems from each State. With contaminant monitoring data from all large PWSs and a statistical, nationally representative sample of small PWSs, the UCMR 1 List 1 Assessment Monitoring program provides a contaminant occurrence data set suitable for national drinking water estimates.

In total, 370,312 sample results have been collected under the UCMR 1 List 1 Assessment Monitoring program at approximately 3,083 large systems and 797 small systems. Approximately 33,600 samples were collected for each contaminant. The UCMR 1 List 1 Monitoring program included systems from all 50 States, the District of Columbia, 4 U.S. Territories, and Tribal lands in 5 EPA Regions. An additional 3,719 samples were collected for 1,3-DCP at all small systems that conducted UCMR 1 List 1 Assessment Monitoring.

In addition to the UCMR 1 List 1 Assessment Monitoring, EPA required monitoring for selected contaminants (including fonofos) for which analytical methods were developed but not widely used. Known as the UCMR 1 List 2 Screening Survey, EPA randomly selected 300 public water systems (120 large and 180 small systems) from the pool of systems required to conduct UCMR 1 List 1 Assessment Monitoring. In total, 29,765 sample results have been collected under the UCMR 1 List 2 Screening Survey from the participating large and small systems. Approximately 2,300 samples were collected for each contaminant. The UCMR 1 List 2 Screening Survey included systems from 48 States, 2 U.S. Territories, and Tribal lands in 1 EPA Region. EPA used the occurrence data from this survey to evaluate fonofos.

EPA analyzed the UCMR 1 List 1 Assessment Monitoring and List 2 Screening Survey data to generate the following initial occurrence and exposure summary statistics:

<bullet≤ The total number of systems and the total population served by these systems,

<bullet≤ The number and percentage of systems with at least 1 observed detection that has a concentration greater than ½ the HRL and greater than the HRL (or in some cases greater than or equal to the minimum reporting limit or MRL), and

<bullet≤ The number of people and percentage of the population served by systems with at least one observed detection greater than ½ the HRL and greater than the HRL (or in some cases greater than or equal to the MRL).[10]

The initial UCMR 1 summary occurrence statistics for dacthal mono- and di-acid degradates, DDE, 1,3-dichloropropene, 2,4-dinitrotoluene, 2,6-dinitrotoluene, EPTC, fonofos, and terbacil are presented in section IV.B of this action.

b. *The Unregulated Contaminant Monitoring Program Rounds 1 and 2.* In 1987, EPA initiated the UCM program to fulfill a 1986 SDWA Amendment that required monitoring of specified unregulated contaminants to gather information on their occurrence in drinking water for future regulatory decision-making purposes. EPA used data from the UCM program to evaluate

[7] Systems serving more than 10,000 people.

[8] Systems serving 10,000 people or fewer.

[9] Large and small systems that purchase 100% of their water supply were not required to participate in the UCMR 1 Assessment Monitoring or the UCMR 1 Screening Survey.

[10] EPA's support documents (USEPA, 2006a and 2006b) provide summary statistics for the median and 99th percentile concentrations of all analytical detections and detailed occurrence results based on UCMR data according to source water type (surface versus ground water), system size, and State.

occurrence for 2 of the 11 contaminants considered for these regulatory determinations. These two contaminants are 1,3-dichloropropene and 1,1,2,2-tetrachloroethane.

EPA implemented the UCM program in two phases or rounds. The first round of UCM monitoring generally extended from 1988 to 1992 and is referred to as UCM Round 1 monitoring. The second round of UCM monitoring generally extended from 1993 to 1997 and is referred to as UCM Round 2 monitoring.

UCM Round 1 monitored for 34 volatile organic compounds (VOCs), including 1,3-dichloropropene and 1,1,2,2-tetrachloroethane (52 FR 25720 (USEPA, 1987)). UCM Round 2 monitored for 13 synthetic organic compounds (SOCs), sulfate and the same 34 VOCs from UCM Round 1 monitoring (57 FR 31776 (USEPA, 1992a)).

The UCM Round 1 database contains contaminant occurrence data from 38 States, Washington, DC, and the U.S. Virgin Islands. The UCM Round 2 database contains data from 34 States and several Tribes. Due to incomplete State data sets, national occurrence estimates based on raw (unedited) UCM Round 1 or Round 2 data could be skewed to low-occurrence or high-occurrence settings (e.g., some States only reported detections). To address potential biases in the data,[11] EPA developed national cross-sections from the UCM Round 1 and Round 2 State data using an approach similar to that used for EPA's 1999 Chemical Monitoring Reform (CMR), the first Six Year Review, and the first CCL Regulatory Determinations. This national cross-section approach was developed to support occurrence analyses and was supported by scientific peer reviewers and stakeholders. This approach identified 24 of the original 38 States from the UCM Round 1 database and 20 of the original 34 States from the UCM Round 2 data base for the national cross-section.

Because UCM Round 1 and Round 2 data represent different time periods and include occurrence data from different States, EPA developed separate national cross-sections for each data set. The UCM Round 1 national cross-section consists of data from 24 States, with approximately 3.3 million total analytical data points from approximately 22,000 unique PWSs. The UCM 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 UCM Round 1 and 2 national cross-sections represent significantly large samples of national occurrence data. Within each cross-section, the actual number of systems and analytical records for each contaminant varies. The support document, ''The Analysis of Occurrence Data from the Unregulated Contaminant Monitoring (UCM) Program and National Inorganics and Radionuclides Survey (NIRS) in Support of Regulatory Determinations for the Second Drinking Water Contaminant Candidate List'' (USEPA, 2006c), provides a description of how the national cross-sections for the Round 1 and Round 2 data sets were developed.

EPA constructed the national cross-sections in a way that provides a balance and range of States with varying pollution potential indicators, a wide range of the geologic and hydrologic conditions, and a very large sample of monitoring data points. While EPA recognizes that some limitations exist, the Agency believes that the national cross-sections do provide a reasonable estimate of the overall distribution and the central tendency of contaminant occurrence across the United States.

EPA analyzed the UCM Round 1 and 2 National Cross-Section data to generate the following initial occurrence and exposure summary statistics:

<bullet≤ The total number of systems and the total population served by these systems,

<bullet≤ The number and percentage of systems with at least 1 observed detection that has a concentration greater than ½ the HRL and greater than the HRL (or in some cases greater than or equal to the MRL), and

<bullet≤ The number of people and percentage of the population served by systems with at least 1 observed detection that has a concentration greater than ½ the HRL and greater than the HRL (or in some cases greater than or equal to the MRL).[12]

The initial UCM summary occurrence statistics for 1,3-dichloropropene and 1,1,2,2-tetrachloroethane are presented in section IV.B of this action.

c. *National Inorganic and Radionuclide Survey.* In the mid-1980's, EPA conducted the NIRS to provide a statistically representative sample [13] of the national occurrence of inorganic contaminants in community water systems (CWSs) served by ground water. EPA used data from NIRS, as well as a supplemental survey, to evaluate occurrence for boron.

The NIRS database includes 36 radionuclides and inorganic compounds (IOCs), including boron. The NIRS provides contaminant occurrence data from 989 ground water CWSs covering 49 States (all except Hawaii) and does not include surface water systems. The survey focused on ground water systems, in part because IOCs tend to occur more frequently and at higher concentrations in ground water than in surface water. Each of the 989 randomly selected CWSs was sampled at a single time between 1984 and 1986.

EPA analyzed the NIRS data to generate the following occurrence and exposure summary statistics for boron:

<bullet≤ The total number of systems and the total population served by these systems,

<bullet≤ The number and the percentage of systems with at least 1 detection that has a concentration greater than ½ the HRL and greater than the HRL,

<bullet≤ The number of people and percentage of the population served by systems with at least 1 observed detection that has a concentration greater than ½ the HRL and greater than the HRL.[14]

Similar to the treatment of the UCM cross-section data, the actual values for the NIRS analyses of boron are reported in section IV.B. Because the NIRS data were collected in a randomly designed sample survey, these summary statistics are representative of national occurrence in ground water CWSs.

One limitation of the NIRS is a lack of occurrence data for surface water systems. To provide perspective on the occurrence of boron in surface water systems relative to ground water systems, EPA reviewed and took into consideration a recent boron occurrence survey funded by American Water Works Association Research Foundation (AWWARF) (Frey *et al.*, 2004). A short description of the AWWARF study is provided in the supplemental section

[11] The potential bias in the raw UCM data are due to lack of representativeness (since not all States provided UCM data) and incompleteness (since some States that provided data had incomplete data sets).

[12] EPA's support documents (USEPA, 2006a and 2006c) provide summary statistics for the median and 99th percentile concentrations of all analytical detections and detailed occurrence results based on the UCM Round 1 and 2 Nationals Cross-Sectons according to source water type (surface versus ground water), system size, and State.

[13] NIRS was designed to provide results that are statistically representative of natioal occurrence at CWSs using ground water sources and is stratified based on system size (population served by the system). Most of the NIRS data are from smaller systems (92 percent from systems serving 3,300 persons or fewer).

[14] EPA's support documents (USEPA, 2006a and 2006c) provide the number and percentage of 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.

(section III.B.2) and the results of the AWWARF survey are presented in section IV.B of this action.

d. *Presentation of Occurrence Data and Analytical Approach.* As noted previously, the occurrence values and summary statistics presented in this action are the actual data from the UCMR 1, UCM, and NIRS data sets. These occurrence values represent direct counts of the number and percent of systems, and population served by systems, with at least 1 analytical detection above some specified concentration threshold. EPA considered this to be the most straightforward and accurate way to present these data for the regulatory determination process.

While both UCMR 1 and UCM data could support more involved statistical modeling to characterize occurrence based on mean (rather than peak) concentrations, EPA chose not to perform this step for the regulatory determinations proposed in this action. EPA believes that presenting the actual results of the occurrence monitoring is straight-forward and the use of an analysis based on peak concentrations provides conservative estimates of occurrence and potential exposure from drinking water. Given that the preliminary determinations for the 11 contaminants discussed in this action are negative, it is not necessary to go beyond the conservative (peak concentration) approach used for this analysis.

2. Supplemental Data. The Agency evaluated several sources of supplemental occurrence information to augment the primary drinking water occurrence data, to evaluate the likelihood of contaminant occurrence, and/or to more fully characterize a contaminant's presence in the environment. Sections II.B.2.a through II.B.2.f provide brief descriptions of the main supplemental information/data sources cited in this action. Summarized occurrence findings from these supplemental sources are presented in Section IV.B, the contaminant profiles section. While the following descriptions cover the more commonly referenced supplemental sources of information/data, they do not include every study and survey cited in the contaminant discussions. A more detailed discussion of the supplemental sources of information/data that EPA evaluated for each contaminant can be found in the comprehensive regulatory determination support document (USEPA, 2006a).

a. *USGS NAWQA Information/Data.* The United States Geological Survey (USGS) collects long-term and nationally consistent data describing water quality in ground water and surface water. In 1991, USGS implemented the National Water-Quality Assessment (NAWQA) Program for 10-year cyclical data collection and data analyses. During the first cycle (1991–2001), the NAWQA program monitored 51 major watersheds and aquifers (study units), which supply more than 60% of the nation's drinking water and water used for agriculture and industry in the U.S. (Hamilton *et al.*, 2004). NAWQA has collected data from over 6,400 surface water and 7,000 ground water sampling points. USGS National Synthesis teams prepare comprehensive analyses of data on topics of particular concern. EPA evaluated information/data from the following USGS National Synthesis reports/projects:

(1) The NAWQA Pesticide National Synthesis Project. In 2003, USGS posted the preliminary results from the first cycle of monitoring for pesticides in streams and ground water. USGS considers these results to be provisional. The results and the data can be accessed at *http://ca.water.usgs.gov/pnsp/.* Data are presented separately for surface water and ground water, as well as bed sediments and biota. In each case, results are subdivided by land use category. Land use categories include agricultural, urban, mixed (deeper aquifers of regional extent in the case of ground water), and undeveloped. In this action, the NAWQA pesticide data for surface water are referenced as Martin *et al.* (2003) and the ground water data are referenced as Kolpin and Martin (2003).

(2) The National Survey of MTBE and Other VOCs in Community Drinking Water Sources (part of the VOC National Synthesis Project). In 2003, USGS published the survey findings for MTBE, other ether gasoline oxygenates, and other volatile organic compounds (VOCs) in source water used by CWSs in the United States. The survey was funded by AWWARF and performed by USGS in collaboration with the Metropolitan Water District of Southern California and the Oregon Health and Science University. USGS performed the survey in two independent stages designed to provide representative sampling of all CWSs in the United States (Random Source-Water Survey) and to improve understanding of the temporal variability of MTBE and other compounds in selected water sources (Focused Source-Water Survey). Participating water utilities provided samples that were analyzed for 66 VOCs. The random survey design selected 954 CWSs to be nationally representative of surface and ground waters sources used by CWSs. The focused survey studied source waters from 134 CWSs suspected or known to contain MTBE. The reports/results and data sets from the survey can be accessed at *http://sd.water.usgs.gov/nawqa/vocns/nat—survey.html.* The random survey results can be found in the USGS Water Resources Investigations Report 02–4079, referenced as Grady (2003). The focused survey results can be found in the USGS Water Resources Investigations Report 02–4084, referenced as Delzer and Ivahnenko (2003a).

b. *USGS National Highway Runoff Data and Methodology Synthesis.* In addition to the NAWQA project, USGS has prepared additional surveys of national contaminant occurrence. For the National Highway Runoff Data and Methodology Synthesis, USGS conducted a review of 44 studies of semi-volatile organic compounds (SVOCs) and VOCs in runoff conducted since 1970. The USGS Synthesis sought to evaluate data quality parameters for comparison between and among these studies, including documentation of sampling protocols and methods, limits of reporting and detection, and protocols of quality-control and quality-assurance. The complete USGS report is Open-File Report 98–409 and is referenced as Lopes and Dionne (1998).

c. *Toxics Release Inventory.* EPA established the Toxics Release Inventory (TRI) in 1987 in response to section 313 of the Emergency Planning and Community Right-to-Know Act (EPCRA). EPCRA section 313 requires facilities to report to both EPA and the States annual information on toxic chemical releases from facilities that meet reporting criteria. EPCRA section 313 also requires EPA to make this information available to the public through a computer database. This database is accessible through TRI Explorer, which can be accessed at *http://www.epa.gov/triexplorer.* In 1990 Congress passed the Pollution Prevention Act, which required that additional data on waste management and source reduction activities be reported under TRI. The TRI database details not only the types and quantities of toxic chemicals released to the air, water, and land by facilities, but also provides information on the quantities of chemicals sent to other facilities for further management (USEPA, 2002b and 2003b).

Facilities are required to report releases and other waste management activities related to TRI chemicals if they manufacture, process, or otherwise use more than established threshold quantities of these chemicals. Currently

for most chemicals, the thresholds are 25,000 pounds for manufacturing and processing and 10,000 pounds for use. Although TRI can provide a general idea of release trends, it is far from exhaustive and should not be used to estimate general public exposure to a chemical (USEPA, 2002b and 2003b).

d. *Pesticides in Ground Water Database.* The Pesticides in Ground Water Database (PGWDB) is a compilation of data from ground water studies conducted by Federal, State, and local governments, the pesticide industry, and other institutions between 1971 and 1991 (USEPA, 1992b). Data from 68,824 wells in 45 states are included. The vast majority of the wells (65,865) were drinking water wells. Monitoring was conducted for 258 pesticides and 45 degradates. Not all studies tested for every compound.

e. *The National Pesticide Survey.* In 1990, EPA completed a national survey of pesticides in drinking water wells. The purpose of the National Pesticide Survey (NPS) was to determine the national occurrence frequencies and concentrations of select pesticides in the nation's drinking water wells, and to improve EPA's understanding of how pesticide occurrence in ground water correlates with patterns of pesticide usage and ground water vulnerability. The survey included approximately 1,300 CWS wells and rural domestic wells. Sampling was conducted between 1988 and 1990. Wells were sampled for 101 pesticides, 25 pesticide degradates, and nitrate. The survey targeted areas representing a variety of pesticide usage levels and ground water vulnerability. The survey was designed to provide a statistically reliable estimate of pesticide occurrence in the nation's drinking water wells (USEPA, 1990a).

f. *The AWWARF Boron Study.* The American Water Works Research Foundation funded a survey to evaluate the occurrence of boron (as well as hexavalent chromium) in drinking water sources (Frey *et al.*, 2004). The AWWARF study recruited 189 PWSs representing 407 source waters in 41 states. Of the 407 source water sample kits distributed in 2003, approximately 342 were returned. Of these 342 samples, 341 were analyzed for boron. Approximately 67 percent (or 228) represented ground water sources and 33 percent (or 113) represented surface water sources. The results of the AWWARF survey for boron are presented in section IV.B of this action.

3. Supporting Documentation for Occurrence. As mentioned in section II.E, EPA prepared several technical occurrence documents to support this action. These technical occurrence documents include the following:

<bullet≤ ''The Analysis of Occurrence Data from the Unregulated Contaminant Monitoring (UCM) Program and National Inorganics and Radionuclides Survey (NIRS) in Support of Regulatory Determinations for the Second Drinking Water Contaminant Candidate List'' (USEPA, 2006c), which this action refers to as the ''UCM and NIRS Occurrence Report.''

<bullet≤ ''The Analysis of Occurrence Data from the First Unregulated Contaminant Monitoring Regulation (UCMR 1) in Support of Regulatory Determinations for the Second Drinking Water Contaminant Candidate List'' (USEPA, 2006b), which this action refers to as the ''UCMR 1 Occurrence Report.''

The ''UCM and NIRS Occurrence Report'' provides more detailed information about the UCM and the NIRS data, how EPA assessed the data quality, completeness, and representativeness, and how the data were used to generate estimates of contaminant occurrence. The ''UCMR 1 Occurrence Report'' provides more detailed information about the UCMR 1 data, how EPA assessed the data quality, completeness, representativeness, and how the data were used to generate estimates of contaminant occurrence.

The comprehensive regulatory support document (USEPA, 2006a) provides a summary of the results from the drinking water occurrence analyses discussed in the aforementioned technical support documents, as well as information on production and use, environmental releases, and/or occurrence in ambient water, potential health effects, the Agency's preliminary determination, and the rationale for the determination.

IV. Preliminary Regulatory Determinations

A. Summary of the Preliminary Regulatory Determination

The Agency has made a preliminary determination that each of the 11 contaminants listed in Table 3 do not meet all three of the SDWA criteria (discussed in section II.C) and thus do not warrant regulation with an NPDWR. Table 3 also summarizes the primary information used to make these regulatory determinations. Section IV.B of this action provides a more detailed summary of the information and the rationale used by the Agency to reach its preliminary decisions. The Agency solicits public comment on the preliminary determinations for these 11 contaminants.

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Table 3. Summary of the Health and Occurrence Information and the Preliminary Determinations for the 11 Contaminants Considered Under CCL Regulatory Determinations 2

#	Contaminant and Its Chemical Abstract Registry Number (CASRN)	Preliminary Determination	Health Reference Level (HRL)	Occurrence Findings from Primary Data Sources (UCMR 1, UCM Round 1 and 2 Cross Sections, NIRS)				
				Database	PWSs with at least 1 detection > ½ HRL	Population served by PWSs with at least 1 detection > ½ HRL	PWSs with at least 1 detection > HRL	Population served by PWSs with at least 1 detection > HRL
1	Boron (7440-42-8)	Do not regulate[1]	1,400 µg/L	NIRS	4.3% (43 of 989)	2.9% (42.7K of 1.48M)	1.7% or (17 of 989) [1]	0.4% (6.4K of 1.48M)
2 3	Dacthal di acid degradate[2] (2136-79-0) Dacthal mono acid degradate[3] (887-54-7)	Do not regulate	70 µg/L [4]	UCMR 1[5]	0.05% (2 of 3,868)	0.33% (739K of 225M)	0.03% (1 of 3,868)	<0.01% (500 of 225M)
4	DDE[6] (72-55-9)	Do not regulate	0.2 µg/L	UCMR 1	-------[7]	-------[7]	0.03%[7] (1 of 3,867) [8]	0.01% (18K of 226M) [8]
5	1,3-Dichloropropene (Telone) (542-75-6)	Do not regulate	0.4 µg/L	UCM Rd1 UCM Rd2 UCMR 1	0.16% (15 of 9,164) [9] 0.30% (50 of 16,787) [9] -------[7]	0.86% (436K of 51M) [9] 0.42% (193K of 46M) [9] -------[7]	0.16% (15 of 9,164) [9] 0.23% (38 of 16,787) [9] 0.00% (0 of 796) [8]	0.86% (436K of 51M) [9] 0.33% (152K of 46M) [9] 0.00% (0 of 2.8M) [8]
6	2,4-Dinitrotoluene (121-14-2)	Do not regulate	0.05 µg/L	UCMR 1	-------[7]	-------[7]	0.03% (1 of 3,866) [8]	0.02% (38K of 226M) [8]
7	2,6-Dinitrotoluene (606-20-2)	Do not regulate	0.05 µg/L	UCMR 1	-------[7]	-------[7]	0.00% (0 of 3,866) [8]	0.00% (0 of 226M) [8]
8	EPTC[10] (759-94-4)	Do not regulate	175 µg/L	UCMR 1	0.00% (0 of 3,866)	0.00% (0 of 226M)	0.00% (0 of 3,866)	0.00% (0 of 226M)
9	Fonofos (944-22-9)	Do not regulate	10 µg/L	UCMR 1	0.00% (0 of 295)	0.00% (0 of 41M)	0.00% (0 of 295)	0.00% (0 of 41M)
10	Terbacil (5902-51-2)	Do not regulate	90 µg/L	UCMR 1	0.00% (0 of 3,866)	0.00% (0 of 226M)	0.00% (0 of 3,866)	0.00% (0 of 226M)
11	1,1,2,2-Tetrachloroethane (79-34-5)	Do not regulate	0.4 µg/L	UCM Rd1 UCM Rd2	0.22% (44 of 20,407) [9] 0.07% (18 of 24,800) [9]	1.69% (1.6M of 95M) [9] 0.51% (362K of 71M) [9]	0.20% (41 of 20,407) [9] 0.07% (17 of 24,800) [9]	1.63% (1.5M of 95M) [9] 0.08% (56K of 71M) [9]

Footnotes: (1) EPA also considered the results of an AWWARF study of PWSs indicating that surface water sources are unlikely to contain boron at levels > the HRL of 1,400 µg/L (Frey *et al.*, 2004). (2) 2,3,5,6-tetrachchloroterephthalic acid (TPA). (3) monomethyl-2,3,5,6-tetrachloroterephthalate (MTP). (4) Using the dacthal parent HRL since it includes the toxicity for the degradates. (5) Degradates monitored in aggregate and converted to the parent equivalent. (6) 1,1-dichloro-2,2-bis(p-chlorophenyl)ethylene. (7) Not reported since MRL > ½ the HRL. (8) Shows results > MRL, rather than > HRL, since MRL is greater than the HRL. In all cases the MRL is within the 10^{-4} to 10^{-6} risk range. (9) The MRLs used in UCM varied from below the ½ HRL to above the HRL. However, even the highest MRLs used are within the 10^{-4} to 10^{-6} risk range. (10) s-ethyl dipropylthiocarbamate..

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B. Contaminant Profiles

This section provides further details on the background, health, and occurrence information that the Agency used to evaluate each of the 11 candidate contaminants considered for regulatory determination. For each candidate, the Agency evaluated the available human and toxicological data, derived a health reference level, and evaluated the potential and/or likely occurrence and exposed population for the contaminant in public water systems. The Agency used the findings from these evaluations to determine whether the three SDWA statutory requirements were satisfied.

As discussed in section II.E, the Agency has also prepared a regulatory support document (USEPA, 2006a) that provides more details on the background, health, and occurrence information/analyses used to evaluate and make preliminary determinations for these 11 candidates.

1. Boron

a. *Background.* Boron, a metalloid, tends to occur in nature in the form of borates (e.g., boric acid, borax, boron oxide). Man-made releases are typically in the form of borates or boron halides (e.g., boron trichloride, boron trifluoride). Boron compounds are used in the production of glass, ceramics, soaps, fire retardants, pesticides, cosmetics, photographic materials, and high energy fuels (USGS, 2004; ATSDR, 1992).

Natural processes such as the weathering of rocks, volcanic activity, and geothermal steam contribute to the release of boron in the environment. Releases to the environment from human activities occur through the production, use, and disposal of boron-containing compounds (e.g., industrial emissions, fertilizer and herbicide runoff, hazardous waste deposits, and municipal sewage) (HSDB, 2004a; ATSDR, 1992).

Although quantitative data are not available on the man-made releases of most borates in the United States, two boron halide compounds, boron trichloride and boron trifluoride, are listed as Toxics Release Inventory (TRI) chemicals. TRI data for boron trichloride and boron trifluoride are reported for the years 1995 to 2003 (USEPA, 2006d). The TRI data show boron trichloride releases from facilities in 6 States and indicate that air emissions account for all of the total releases of boron trichloride (on- and off-site), which generally fluctuated in the range of hundreds of pounds per year during the period of record. The TRI data show boron trifluoride releases from facilities in 14 States and indicate that air emissions also account for nearly all of the boron trifluoride releases, which ranged in the tens of thousands of pounds annually.

b. *Health Effects.* The Institute of Medicine (IOM, 2001) of the National Academies categorizes boron as a possible trace mineral nutrient for humans. Boron is essential for plant growth and deficiency studies in animals and humans have provided some evidence that low intakes of boron affects cellular function and the activity of other nutrients. It may interact with Vitamin D and calcium homeostasis, influence estrogen metabolism, and play a role in cognitive function (IOM, 2001). Iyengar *et al.* (1988) reported an average dietary intake of 1.5 mg/day for male adults based on the Food and Drug Administration (FDA) Total Diet Study (TDS).

Some human oral data are available from cases where boron was ingested as a medical treatment. When the amount ingested was less than 3.68 mg/kg, subjects were asymptomatic, while doses of 20 and 25 mg/kg resulted in nausea and vomiting. Case reports and surveys of accidental poisonings indicate that the lethal doses of boron range from 15 to 20 grams (approximately 200 to 300 mg/kg) for adults, 5 to 6 grams (approximately 70 to 85 mg/kg) for children, and 2 to 3 grams (approximately 30 to 45 mg/kg) for infants (USEPA, 2004b).

The primary adverse effects seen in animals after chronic exposure to low doses of boron generally involve the testes and developing fetus. Chronic effects of dietary boron exposure in two-year studies included testicular atrophy and spermatogenic arrest in dogs, decreased food consumption,

suppressed growth, and testicular atrophy in rats, and decreased survival, testicular atrophy, and interstitial cell hyperplasia in mice. Although researchers observed some increases in tumor incidences in the liver and in subcutaneous tissues in mice, based on comparisons to historic controls, these tumors were determined not to be associated with exposure to boron from boric acid (USEPA, 2004b). Boron is not considered mutagenic and the Agency determined that there are inadequate data to assess the human carcinogenic potential for boron (USEPA, 2004c).

In developmental studies with rats, mice, and rabbits, oral exposure to boric acid resulted in decreased pregnancy rate, increased prenatal mortality, decreased fetal weights, and increased malformations in fetuses and pups. However, these reproductive effects were associated with maternal toxicity including changes in maternal organ weights, body weights, weight gain, and increased renal tubular dilation and/or regeneration (Price *et al.*, 1990, 1994, 1996; Heindel *et al.*, 1992, 1994; Field *et al.*, 1989). Reproductive effects in males were noted in the subchronic and chronic studies described in the preceding paragraphs.

The EPA RfD for boron is 0.2 mg/kg/day (USEPA, 2004c) based on developmental effects in rats from two studies (Price *et al.*, 1996; Heindel *et al.*, 1992). The RfD was derived using the benchmark dose (BMD) method (bench mark dose level or BMDL from Allen *et al.*, 1996). EPA calculated the HRL of 1.4 mg/L or 1,400 [mu]g/L for boron using the RfD of 0.2 mg/kg-day and a 20 percent screening relative source contribution.

EPA also evaluated whether health information is available regarding the potential effects on children and other sensitive populations. Studies in rats, mice, and rabbits identify the developing fetus as potentially sensitive to boron. Price *et al.* (1996) identified a LOAEL of 13.3 mg/kg-day and an NOAEL of 9.6 mg/kg-day in the developing fetus, based on decreased fetal body weight in rats. Accordingly, boron at concentrations greater than the HRL might have an effect on prenatal development. Individuals with severely impaired kidney function might also be sensitive to boron exposure since the kidney is the most important route for excretion.

c. *Occurrence Analyses.* The National Inorganics and Radionuclides Survey (NIRS) included boron as an analyte. Using data from NIRS, EPA performed an initial evaluation of occurrence and exposure at levels greater than 700 [mu]g/L (½ the HRL) and greater than 1,400 [mu]g/L (the HRL for boron). The NIRS data indicate that approximately 4.3 percent (or 43) of the 989 ground water PWSs sampled had detections of boron at levels greater than 700 [mu]g/L, affecting approximately 2.9 percent of the population served (or 42,700 people from 1.48 million). Approximately 1.7 percent (or 17) of 989 ground water PWSs sampled had detections of boron at levels greater than 1,400 [mu]g/L, affecting approximately 0.4 percent of the population served (6,400 people from 1.48 million) (USEPA, 2006a and 2006c).

Because NIRS did not contain data for surface water systems, the Agency evaluated the results of a survey funded by the American Water Works Association Research Foundation (Frey *et al.*, 2004) to gain a better understanding of the potential occurrence of boron in surface water systems. The AWWARF study recruited 189 PWSs representing 407 source waters that covered 41 states. Of these 407 PWS source water samples, 342 were returned and 341 were analyzed for boron. Of these 341 samples, approximately 67 percent (or 228) represented ground water sources and 33 percent (or 113) represented surface water sources. None of the 113 surface water sources exceeded the boron HRL of 1,400 [mu]g/L and the maximum concentration observed in surface water was 345 [mu]g/L. Extrapolation of the data indicates that 95 percent of the ground water detections had boron levels less than 1,054 [mu]g/L; the maximum observed concentration in ground water was approximately 3,300 [mu]g/L. Seven of the 228 ground water sources (from 5 systems) had boron concentrations greater than 1,400 [mu]g/L (Seidel, 2006).

d. *Preliminary Determination.* The Agency has made a preliminary determination not to regulate boron with an NPDWR. While boron was found at levels greater than the HRL (and ½ the HRL) in several of the ground water systems surveyed by NIRS, it was not found at levels greater than the HRL (or ½ the HRL) in the surface waters sources evaluated in the AWWARF study. Taking this surface water information into account, the Agency believes that the overall national occurrence and exposure from both surface and ground water systems together is likely to be lower than the values observed for the NIRS ground water data. Because boron is not likely to occur at levels of concern when considering both surface and ground waters systems, the Agency believes that a national primary drinking water regulation does not present a meaningful opportunity for health risk reduction.

The Agency encourages those States with public water systems that have boron at concentrations above the HRL to evaluate site-specific protective measures and to consider whether State-level guidance (or some other type of action) is appropriate. The Agency also plans to update the Health Advisory for boron to provide more recent health information. The updated Health Advisory will provide information to any States with public water systems that may have boron above the HRL.

2 and 3. Mono- and Di-Acid Degradates of Dimethyl Tetrachloroterephthalate (DCPA)

a. *Background.* Dimethyl tetrachloroterephthalate (DCPA), a synthetic organic compound (SOC) marketed under the trade name ''Dacthal,'' is a pre-emergent herbicide historically used to control weeds in ornamental turf and plants, strawberries, seeded and transplanted vegetables, cotton, and field beans. As of 1990, more than 80 percent of its use was for turf, including golf courses and home lawns (USEPA, 1990b). On July 27, 2005, in response to concerns about groundwater contamination (especially for one of the DCPA degradates), the Agency published a **Federal Register** notice announcing that the registrant for Dacthal had voluntarily terminated a number of uses for products containing DCPA (70 FR 43408; USEPA, 2005f). The only uses retained were those for use on sweet potatoes, eggplant, kale and turnips.

DCPA is not especially mobile or persistent in the environment. Biodegradation and volatilization are the primary dissipation routes. Degradation of DCPA forms two breakdown products, the mono-acid degradate (or monomethyl tetrachloroterephthalate or MTP) and the di-acid degradate (tetrachloroterephthalic acid or TPA). The di-acid, which is the major degradate, is unusually mobile and persistent in the field, with a potential to leach into water (USEPA, 1998c).

Several studies and reports provide estimates of the amount of DCPA used during the 1990s in the United States. The Agency estimated that 1.6 million pounds of DCPA active ingredient a.i. were used annually in the early 1990s (USEPA, 1998c). USGS estimated that approximately 998 thousand pounds of DCPA a.i. were used annually circa 1992 (Thelin and Gianessi, 2000). The National Center for Food and Agricultural Policy (NCFAP, 2004) estimates that approximately 1.7 million

pounds of DCPA a.i. were used in 1992 and approximately 600 thousand pounds a.i. were used in 1997 (NCFAP, 2004). The NCFAP data suggest a decrease in the use of DCPA from the early to the late 1990s.

b. *Health Effects.* Currently, no subchronic or chronic studies are available to assess the toxicological effects of MTP (the mono-acid degradate) and 3 studies in rats (30 and 90-day feeding studies and a one-generation reproductive study) are available for TPA (the di-acid degradate). The effects of exposure were mild (weight loss and diarrhea) and occurred at doses greater than or equal to 2,000 mg/kg/day. No reproductive effects were observed.

The present toxicity database for MTP and TPA is not sufficient to derive RfDs for these two chemicals. However, since the available data indicate that neither MTP nor TPA are more toxic than their parent compound, DCPA, the Agency suggests that the RfD for the DCPA parent would be protective against exposure from these two DCPA metabolites (USEPA, 1998c). Both compounds are formed in the body from the DCPA parent and therefore, the toxicity of these degradates is reflected in the toxicity of the parent. The RfD for DCPA is 0.01 mg/kg/day based on a chronic rat study (ISK Biotech Corporation, 1993) with a NOAEL of 1.0 mg/kg/day and an uncertainty factor of 100 for rat to human extrapolation and intra-species variability.

No carcinogenicity studies have been performed using either TPA or MTP. Based on the cancer data for the parent and lack of mutagenicity for TPA and DCPA, the Agency (USEPA, 2004d) concludes that TPA is unlikely to pose a cancer risk. Klopman *et al.* (1996) evaluated the carcinogenic potential of TPA based on its chemical and biological properties, as well as by a variety of computational tools, and determined that it did not present any substantial carcinogenic risk. There was suggestive evidence that DCPA could be carcinogenic based on an increased incidence of thyroid and liver tumors in rats. The presence of hexachlorobenzene and dioxin as impurities in the material tested could have contributed to the cancer risk.

Using the DCPA RfD of 0.01 mg/kg/day (USEPA, 1994) and a 20 percent screening relative source contribution, the Agency calculated an HRL of 0.07 mg/L or 70 [mu]g/L for DCPA and used this HRL for TPA and MTP.

EPA also evaluated whether health information is available regarding the potential effects on children and other sensitive populations. There are no data that identify a particular sensitive population for DCPA exposure. Results of a single developmental study indicate that exposure to pregnant dams with doses less than or equal to 2,500 mg/kg/day of TPA via gavage did not have an adverse effect on the fetus. EPA did not identify any data that suggest gender-related differences in toxicity or sensitivity in the elderly.

c. *Occurrence.* EPA included the DCPA mono- and di-acid degradates (MTP and TPA) as analytes in the UCMR 1. The analysis results reported for UCMR 1 are the sum of both the mono- and di-acid degradates. EPA converted the analysis result for the degradates to the parent DCPA equivalent and performed an initial evaluation of occurrence and exposure at levels greater than 35 [mu]g/L (½ the HRL) and greater than 70 [mu]g/L (the HRL). As previously discussed, EPA used the HRL derived for the DCPA parent because it includes the toxicity for the mono- and di-acid degradates. While the UCMR 1 data indicate that the DCPA degradates were the most commonly reported analytes in the monitoring survey (detected at an MRL of 1 [mu]g/L in 772 samples from 175 of the 3,868 PWSs sampled), very few systems exceeded the health level of concern. PWSs with detections were found in 24 States and 1 Territory. The UCMR 1 data indicate that approximately 0.05 percent (or 2) of the 3,868 PWSs sampled had a detection of the DCPA degradates at levels greater than 35 [mu]g/L, affecting approximately 0.33 percent of the population served (or 739,000 people from 225 million). Approximately 0.03 percent (or 1) of the 3,868 PWSs sampled have a detection of the DCPA degradates at levels greater than 70 [mu]g/L, affecting less than 0.01 percent of the population served (or 500 people from 225 million) (USEPA, 2006a and 2006b).

EPA also evaluated several sources of supplemental occurrence information for the DCPA parent, the mono-acid degradate and/or the di-acid degradate. These supplemental sources include:

<bullet≤ The National Pesticide Survey (NPS),

<bullet≤ The provisional pesticide results from the 1992–2001 USGS NAWQA survey of ambient surface and ground waters across the U.S., and

<bullet≤ Studies performed by the DCPA or dacthal registrant.

As part of the National Pesticide Survey, EPA collected samples from approximately 1,300 community water systems and rural drinking water wells between 1988 and 1990. The NPS included monitoring for the DCPA parent and the di-acid degradate. The DCPA parent was not detected in any wells (using a detection limit of 0.06 [mu]g/L). While the di-acid degradate was detected in 49 of 1,347 wells (using a detection limit of 0.1 [mu]g/L), the maximum reported concentration of 7.2 [mu]g/L did not exceed the HRL of 70 [mu]g/L (USEPA, 1990a).

The USGS NAWQA program included the DCPA parent and the mono-acid degradate as analytes in its 1992–2001 monitoring survey of ambient surface and ground waters across the United States. EPA evaluated the results of the provisional data, which are available on the Web (Martin *et al.*, 2003; Kolpin and Martin, 2003). While the USGS detected the DCPA parent in both surface and ground waters, at least 95 percent of the samples from the various land use settings were less than or equal to 0.007 [mu]g/L. The estimated maximum surface water concentration, 40 [mu]g/L (agricultural setting), and the estimated maximum ground water concentration, 10 [mu]g/L (agricultural setting), are both less than 70 [mu]g/L (the DCPA HRL). While the USGS detected the mono-acid degradate in both surface waters and ground waters, at least 95 percent of the samples from the various land use settings were less than 0.07 [mu]g/L (the reporting limit for the mono-acid degradate). The maximum surface water concentration, 0.43 [mu]g/L (agricultural setting), and the maximum ground water concentration, 1.1 [mu]g/L (agricultural setting), are both less than 70 [mu]g/L (the DCPA HRL, which includes the toxicity of the degradates).

Beginning in 1992, the registrant for DCPA performed two small-scale ground water occurrence studies in New York and California over a period of 17 and 22 months, respectively. The registrant monitored for the DCPA parent and both of its degradates. The average reported values, which are the sum of the parent and its degradates, were 50.36 [mu]g/L in New York and 12.75 [mu]g/L in California. Neither average value exceeded the HRL of 70 [mu]g/L (USEPA, 1998c).

d. *Preliminary Determination.* The Agency has made a preliminary determination not to regulate the DCPA mono-acid degradate and/or the DCPA di-acid degradate with an NPDWR. Because these degradates appear to occur infrequently at health levels of concern in PWSs, the Agency believes that a national primary drinking water regulation does not present a meaningful opportunity for health risk reduction. While the Agency recognizes that these degradates have been detected in the PWSs monitored under the UCMR 1, only 1 PWS had a detect above the HRL.

The Agency encourages those States with public water systems that have detects for these degradates to evaluate site-specific protective measures and to consider whether State-level guidance (or some other type of action) is appropriate. The Agency also plans to update the Health Advisory for the DCPA parent to include the mono and di acid degradates, as well as any recent health information related to these compounds. The updated Health Advisory will provide information to any States with public water systems that may have DCPA degradates at levels above the HRL.

4. 1,1-Dichloro-2,2-bis(*p*-chlorophenyl) ethylene (DDE)

a. *Background.* DDE is a primary metabolite of DDT,[15] a pesticide once used to protect crops and eliminate disease-carrying insects in the U.S. until it was banned in 1973. DDE itself has no commercial use and is only found in the environment as a result of contamination and/or breakdown of DDT. While DDE tends to adsorb strongly to surface soil and is fairly insoluble in water, it may enter surface waters from runoff that contains soil particles contaminated with DDE. In both soil and water, DDE is subject to photodegradation, biodegradation, and volatilization (ATSDR, 2002).

b. *Health Effects.* DDE is not produced as a commercial product. This has limited the numbers of conventional studies that have been performed to assess toxicological properties. Limited data on DDE, mostly from a National Cancer Institute (NCI) bioassay, suggest that the liver is the primary target organ in mammalian species. However, the NCI study did not evaluate a full array of noncancer endpoints. There is an RfD of 0.0005 mg/kg/day for the parent pesticide DDT based on a NOAEL of 0.05 mg/kg/day from a dietary subchronic study (USEPA, 1996b). In this study, liver lesions were identified at a LOAEL of 0.25 mg/kg/day. Data on DDT identify effects on the nervous and hormonal systems as adverse effects that might also be seen with DDE because it is one of DDT's primary metabolites. The limited data for DDE suggest that any effects on the nervous system are less severe than those seen with DDT. Endocrine effects from DDE are discussed in this section.

Based on animal studies DDE is likely to be carcinogenic to humans. This classification is based on increases in the incidence of liver tumors, including carcinomas, in two strains of mice and in hamsters after dietary exposure to DDE. Some epidemiological studies suggest a possible association of the levels of DDE in serum with breast cancer. However, other studies with similar methodologies do not show any association. DDE was mutagenic in mouse lymphoma L5178Y and Chinese hamster V79 cells but negative in the Ames assay. In the 1988 IRIS, EPA calculated an oral slope factor of 0.34 $(mg/kg/day)^{-1}$ for DDE (USEPA, 1988a). For this regulatory determination, EPA calculated an oral slope factor from the same data set (from the 1988 IRIS) using the EPA 1999 Cancer Guidelines (USEPA, 1999a). The revised slope factor is 1.67×10^{-1} $(mg/kg/day)^{-1}$ resulting in a one-in-a-million cancer-risk (HRL) of 0.2 [mu]g/L.

There are some indications that DDE has an adverse impact on the immune system (Banerjee *et al.*, 1996). Oral exposures to 22 mg/kg/day for 6 weeks suppressed serum immunoglobin levels and antibody titers. Inhibition of leucocytes and macrophage migration were observed at the cellular level. Considerable evidence exists that DDE can act as an endocrine disruptor since it binds to the estrogen and androgen receptors. DDE has a stronger affinity for the androgen receptor than for the estrogen receptor. It competes with testicular hormones for the androgen receptor leading to receptor-related changes in gene expression (Kelce *et al.*, 1995).

EPA evaluated whether health information is available regarding the potential effects on children and other sensitive populations. Children and adolescents may be sensitive populations for exposure to DDE due to its endocrine disruption properties. Some data suggest that DDE can delay puberty in males (ATSDR, 2002).

c. *Occurrence.* EPA included DDE as an analyte in the UCMR 1. Because the HRL for DDE (0.2 [mu]g/L) is lower than the minimum reporting limit (MRL) used for monitoring (0.8 [mu]g/L), EPA used the MRL value to evaluate occurrence and exposure. The MRL is within the 10^{-4} to the 10^{-6} cancer risk range for DDE. In evaluating the UCMR 1 data, EPA found that approximately 0.03 percent (or 1) of the 3,867 PWSs sampled had a detection of DDE at the MRL of 0.8 [mu]g/L, affecting approximately 0.01 percent of the population served (or 18,000 people from 226 million) (USEPA, 2006a and 2006b).

The USGS NAWQA program included DDE as an analyte in its 1992–2001 monitoring survey of ambient surface and ground waters across the United States. EPA evaluated the results of the provisional data, which are available on the Web (Martin *et al.*, 2003; Kolpin and Martin, 2003), as a supplemental source of occurrence information. While the USGS detected DDE in both surface and ground waters, 95 percent of the samples from the various land use settings were less than 0.006 [mu]g/L (the USGS reporting limit). The maximum surface water concentration, 0.062 [mu]g/L (agricultural setting), and the maximum ground water concentration, 0.008 [mu]g/L (agricultural setting), are both less than 0.2 [mu]g/L (the DDE HRL).

d. *Preliminary Determination.* The Agency has made a preliminary determination not to regulate DDE with an NPDWR. Because DDE appears to occur infrequently at levels of concern in PWSs, the Agency believes that a national primary drinking water regulation does not present a meaningful opportunity for health risk reduction. DDE was detected in only one of the PWSs monitored under the UCMR 1 at a level greater than the MRL (0.8 [mu]g/L), a concentration that is within the 10^{-4} to the 10^{-6} cancer risk range. In addition, ambient water data from the USGS indicate that the maximum concentrations detected in surface and ground water were less than the HRL of 0.2 [mu]g/L.

EPA recognizes that DDE is listed as a probable human carcinogen. For this reason, the Agency encourages those States with public water systems that might have DDE above the HRL to evaluate site-specific protective measures and to consider whether State-level guidance (or some other type of action) is appropriate.

5. 1,3-Dichloropropene (1,3-DCP; Telone)

a. *Background.* 1,3-Dichloropropene (1,3-DCP), a synthetic volatile organic compound, is used as a pre-plant soil fumigant to control nematodes and other pests in soils to be planted with all types of food and feed crops. 1,3-DCP is typically injected 12'' to 18'' beneath the soil surface and can only be used by certified handlers (USEPA, 1998b). To mitigate risks to drinking water, 1999 labeling requirements restrict the use of 1,3-DCP:

<bullet≤ In areas with shallow ground water and vulnerable soils in certain northern tier States (ND, SD, WI, MN, NY, ME, NH, VT, MA, UT, and MT);

<bullet≤ In fields within 100 feet of a drinking water well; and

<bullet≤ In areas overlying karst [16] geology.

[15] 1,1,1-trichloro-2,2-bis(p-chlorophenyl)ethane.

[16] Karst is a type of typography that is formed by the dissolution and collapse of soluble rocks (typically limestone and dolomite). According to the Karst Waters Institute, as excerpted by USGS (2006), common geological characteristics of karst regions that influence human use of its land and water resources include ground subsidence,

Estimates of national annual use during the 1990s vary widely, from approximately 23 to 40 million pounds of active ingredient a.i. Based on information from a 1991 data call-in and other sources, EPA estimates that approximately 23 million pounds of 1,3-DCP a.i. were used annually from 1990 to 1995 (USEPA, 1998b). NCFAP (2004) estimates that approximately 40 million pounds a.i. were used in 1992 and approximately 35 million pounds a.i. were used in 1997.

1,3-Dichloropropene is listed as a TRI chemical and releases are reported from facilities in 17 States over a time period covering 1988 to 2003 (although not all States had facilities reporting releases every year) (USEPA, 2006e). Air emissions appear to account for most of the on-site (and total) releases and generally declined between 1988 and 2003. A sharp decrease in air emissions is evident between 1995 and 1996. Surface water discharges are minor compared to air emissions and no obvious trend is evident between 1988 and 2003. Reported underground injection, releases to land, and off-site releases are generally insignificant.

b. *Health Effects.* Chronic and subchronic exposures to 1,3-DCP at doses of 12.5 mg/kg/day and above in animal dietary studies indicate that 1,3-DCP is toxic to organs involved in metabolism (liver), excretion of conjugated metabolites (e.g., urinary bladder and the kidney) and organs along the portals of entry (e.g., forestomach for oral administration; mucous membrane of the nasal passage and lungs for inhalation exposure). Exposure to 1,3-DCP has not been shown to cause reproductive or developmental effects. Neither reproductive nor developmental toxicity were observed in a two-generation reproductive study in rats or in developmental studies in rats and rabbits at maternal inhalation concentrations up to 376 mg/m^3 (USEPA, 2000a). Even concentrations that produced parental toxicity did not produce reproductive or developmental effects (USEPA, 2000a).

An RfD of 0.03 mg/kg/day for 1,3-DCP (USEPA, 2000a) has been established using a benchmark dose (BMD) analysis based on a two-year chronic bioassay (Stott *et al.*, 1995) in which chronic irritation (forestomach hyperplasia) and significant body weight reduction were the critical and co-critical effects, respectively. A reference concentration (RfC) of 0.02 mg/m^3 was derived from a two-year bioassay (Lomax *et al.*, 1989), which observed histopathology in the nasal epithelium.

Under the proposed cancer risk assessment guidelines, the weight of evidence for evaluation of 1,3-DCP's ability to cause cancer suggest that it is likely to be carcinogenic to humans (USEPA, 2000a). This characterization is supported by tumor observations in chronic animal bioassays for both inhalation and oral routes of exposure.

The oral cancer slope factors calculated from chronic dietary, gavage and inhalation data ranged from 5 x 10^{-2} to 1 x 10^{-1} (mg/kg/day)$^{-1}$. Due to uncertainties in the delivered doses in some studies, EPA (IRIS) recommended using the oral slope factor of 1 x 10^{-1} (mg/kg/day)$^{-1}$ from an NTP (1985) study. Using this oral slope factor, EPA calculated an HRL of 0.4 [mu]g/L at the 10^{-6} cancer risk level.

EPA also evaluated whether health information is available regarding the potential effects on children and other sensitive populations. No human or animal studies are available that have examined the effect of 1,3-DCP exposure on juvenile subjects. Therefore, its effects on children are unknown. Developmental studies in rats and rabbits show no evidence of developmental effects and therefore it is unlikely that 1,3-DCP causes developmental toxicity.

c. *Occurrence.* EPA included 1,3-DCP as an analyte in the UCM Round 1 and UCM Round 2 surveys. The MRLs for UCM Round 1 ranged from 0.02 to 10 [mu]g/L and the MRLs for UCM Round 2 ranged from 0.08 to 1 [mu]g/L. EPA also analyzed for 1,3-DCP using the samples from the small systems that were included in the UCMR 1 survey. The MRL used for the UCMR 1 survey was 0.5 [mu]g/L. Because some of these reporting limits exceeded the thresholds of interest, the occurrence analyses may result in an underestimate of systems affected (USEPA, 2006a, 2006b and 2006c). However, the MRL values used for UCM Round 1 and UCM Round 2 as well as UCMR 1 are within the 10^{-4} to the 10^{-6} cancer risk range.

The UCM Round 1 Cross Section data indicate that approximately 0.16 percent (or 15) of the 9,164 PWSs sampled had detections of 1,3-DCP at levels greater than 0.2 [mu]g/L (½ the HRL), affecting approximately 0.86 percent of the population served (or 438,000 of 51 million). The UCM Round 1 Cross Section data also indicate the same values when the data are analyzed using 0.4 [mu]g/L (the HRL). That is, 0.16 percent (or 15) of 9,164 PWSs sampled had detections greater than 0.4 [mu]g/L (the HRL), affecting approximately 0.86 percent of the population served (or 438,000 of 51 million people). The 99th percentile of all detections is 2 [mu]g/L and the maximum reported value is 2 [mu]g/L.

The UCM Round 2 Cross Section data indicate that approximately 0.30 percent (or 50) of the 16,787 PWSs sampled had detections of 1,3-DCP at levels greater than 0.2 [mu]g/L (½ the HRL), affecting approximately 0.42 percent of the population served (or 193,000 of 46 million). The UCM Round 2 Cross Section data indicate that approximately 0.23 percent (or 38) of the 16,787 PWSs sampled had detections of 1,3-DCP at levels greater than 0.4 [mu]g/L (the HRL), affecting approximately 0.33 percent of the population served (or 152,000 of 46 million). The 99th percentile of all detections is 39 [mu]g/L and the maximum reported value is 39 [mu]g/L.

Because the sample preservative used may have resulted in potential underestimates of occurrence for the UCM Rounds 1 and 2 data, EPA subsequently analyzed for 1,3-DCP using the samples provided by 796 of the small systems included in the recent UCMR 1 survey. None of the 3,719 samples from these 796 small systems (serving a population of 2.8 million) had detects of 1,3-DCP at levels greater than 0.5 [mu]g/L (the minimum reporting limit used for the analysis of 1,3-DCP and a level that is slightly higher than the HRL).

EPA also evaluated several sources of supplemental information, which included:

<bullet≤ The National Pesticide Survey,

<bullet≤ The Pesticides in Ground Water Database,

<bullet≤ A well water survey submitted by the registrant of Telone (1,3-DCP),

<bullet≤ The USGS VOC National Synthesis Random Source Water Survey, and

<bullet≤ The USGS VOC National Synthesis Focused Source Water Survey.

As part of the National Pesticide Survey, EPA collected samples from approximately 1,300 community water systems and rural drinking water wells between 1988 and 1990. The NPS included *cis* and *trans* 1,3-DCP as analytes in the monitoring survey. Neither compound was detected in the survey using a minimum reporting limit of 0.010 [mu]g/L (USEPA, 1990a).

The Pesticides in Ground Water Database (USEPA, 1992b) indicates that 1,3-DCP was found in 6 of 21,270 ground water wells sampled in 7 States. The 6 wells with positive detections for 1,3-DCP included 3 wells in California (at concentrations ranging from 0.890 to 31.0 [mu]g/L), 2 wells in Florida (at concentrations of 0.279 to 7.83 [mu]g/L), and 1 well in Montana (at concentrations of 18 to 140 [mu]g/L). While most or all of these 6 wells had

sinkhole collapse, ground water contamination, and unpredictable water supply.

concentrations greater than the HRL for 1,3-DCP, the overall percentage of positive wells detections was less than 0.1 percent.

In 1998, the registrant for Telone (1,3-DCP) submitted a private well water study to the Agency. The well water survey covered 5 regions where Telone was used intensively and evaluated 518 wells (5,800 samples) for the presence of 1,3-DCP. Of the 518 wells, 65 had detectable levels of 1,3-DCP and/or its metabolites at levels greater than 0.015 [mu]g/L (the detection limit for 1,3-DCP was 0.015 [mu]g/L and the metabolites were 0.023 [mu]g/L). None of the wells exceeded 0.2 [mu]g/L (a level half the EPA-derived HRL for 1,3-DCP) (USEPA, 2004e and 2004f).

For the Random Source Water Survey, the USGS collected samples from 954 source waters that supply community water systems between 1999 and 2000. For the Focused Source Water Survey, the USGS collected 451 samples from 134 source waters that supply community water systems between 1999 and 2001. The USGS included 1,3-DCP as an analyte in both surveys. The USGS did not detect 1,3-DCP in any of the source water samples from the Random Source Water Survey using a reporting limit of 0.2 [mu]g/L (a level that is one-half the HRL for 1,3-DCP). In addition, the USGS did not detect 1,3-DCP in any of the source water samples in the Focused Source Water Survey using a detection limit of 0.024 [mu]g/L for cis-1,3-dichloropropene and 0.026 [mu]g/L for trans-1,3-dichloropropene (levels that are about 16 times lower than the HRL for 1,3-DCP) (Ivahnenko *et al.*, 2001; Grady, 2003; Delzer and Ivahnenko, 2003a).

d. *Preliminary Determination.* The Agency has made a preliminary determination not to regulate 1,3-DCP with an NPDWR. Because 1,3-DCP appears to occur infrequently at health levels of concern in PWSs, the Agency believes that a national primary drinking water regulation does not present a meaningful opportunity for health risk reduction. While 1,3-DCP was detected in the UCM Round 1 (late 1980s) and the UCM Round 2 (mid 1990s) surveys, it was not detected in a subsequent evaluation of 796 small systems from the UCMR 1 survey. In addition, the USGS did not detect 1,3-DCP in two occurrence studies performed between 1999 and 2001 using monitoring levels that were lower than the HRL. EPA believes the 1999 pesticide labeling requirements, which are intended to mitigate risks to drinking water, may be one reason for the lack of occurrence of 1,3-DCP at levels of concern in subsequent monitoring surveys.

EPA recognizes that 1,3-dichloropropene is listed as a probable human carcinogen. For this reason, the Agency encourages those States with public water systems that may have 1,3-dichloropropene above the HRL to evaluate site-specific protective measures and to consider whether State-level (or some other type of action) is appropriate. The Agency also plans to update the Health Advisory document for 1,3-DCP to provide more recent health information. The updated Health Advisory will provide information to any States with public water systems that may have 1,3-DCP above the HRL.

6 and 7. 2,4- and 2,6-Dinitrotoluenes (2,4- and 2,6-DNT)

a. *Background.* 2,4- and 2,6-dinitrotoluene (DNT), semi-volatile organic compounds, are two of 6 isomers of dinitrotoluene. Dinitrotoluenes are used in the production of polyurethane foams, automobile air bags, dyes, ammunition, and explosives, including trinitrotoluene or TNT (HSDB, 2004b and 2004c; ATSDR, 1998). Neither 2,4- nor 2,6-DNT occur naturally. They are generally produced as individual isomers or as a mixture called technical grade DNT (tg-DNT). Technical grade DNT primarily contains a mixture of 2,4-DNT and 2,6-DNT with the remainder consisting of the other isomers and minor contaminants such as TNT and mononitrotoluenes (HSDB, 2004b).

No recent quantitative estimates of DNT production or use are available. The Hazardous Substances Data Bank (HSDB, 2004b) cites a 1980 EPA Ambient Water Quality Criteria Document that places combined 2,4- and 2,6-DNT production at 272,610,000 pounds in 1975.

Both 2,4-DNT and 2,6-DNT are listed as TRI chemicals. TRI data for 2,4-DNT are reported from facilities in 21 States over a time period covering 1988 to 2003. Total releases nationally in 2003 were 14,899 lbs. Releases of all kinds (off-site releases and on-site air, surface, underground injection, and land releases) declined in the early 1990s, and then peaked again around 1999–2001. On-site air emissions and surface water releases of 2,4-DNT were generally the most consistent (least fluctuating) types of releases, with surface water releases generally declining over the period on record (USEPA, 2006f).

TRI data for 2,6-DNT are reported from facilities in 10 States over a time period covering 1988 to 2003 (with no more than 9 States having reporting facilities in any one year). Total reported releases for 2003 were 10,937 lbs. Trends for 2,6-DNT are similar to those for 2,4-DNT. The TRI data for 2,6-DNT show a trend of declining releases in the late 1980s and early 1990s, and a subsequent peak around 1999–2001. On-site air emissions and surface water discharges are the most consistent types of release for 2,6-DNT and surface water discharges exhibit a declining trend (USEPA, 2006f).

In addition, TRI lists mixed DNT isomer releases as a separate category over the same time period (1990–2003). TRI releases of mixed isomers were reported from facilities in 9 States, with no more than 7 States having reporting facilities in any one year. Total releases in 2003 were 13,790 lbs. Underground injections made up the bulk of on-site releases during the 1990s, but diminished thereafter. Air emissions remained relatively constant. Surface water discharges and releases to land were generally insignificant but peaked in 2003. Off-site releases varied widely. Total releases peaked in 1993 and 1997, and generally diminished in recent years (USEPA, 2006f).

b. *Health Effects.* In experimental animal studies, 2,4- and 2,6-DNT appear to be acutely toxic at moderate to high levels (LD_{50}'s [17] ranging from 180 to 1,954 mg/kg) when administered orally. In subacute studies (4 weeks) conducted by Lee *et al.* (1978), dogs, rats, and mice were fed 2,4-DNT and studied for toxic effects. A NOAEL of 5 mg/kg/day was established; decreased body weight gain and food consumption, neurotoxic signs, and lesions in the brain, kidneys, and testes occurred at 25 mg/kg/day (the highest dose tested).

Subchronic studies in mice, rats, and dogs that administered 2,4- and 2,6-DNT in the diet produced similar effects in all species. All species exposed to 2,4-DNT exhibited methemoglobinemia, anemia, bile duct hyperplasia sometimes accompanied by hepatic degeneration, and depressed spermatogenesis. Neurotoxicity and renal degeneration occurred in dogs at a dose level of 20 mg/kg/day of 2,6-DNT (Lee *et al.*, 1976). At a dose level of 25 mg/kg/day of 2,4-DNT, male and female dogs developed impaired muscle movement and paralysis, methemoglobinemia, aspermatogenesis, hemosiderosis of the spleen and liver, cloudy swelling of the kidneys, and lesions of the brain (Ellis *et al.*, 1985).

[17] LD_{50} = An estimate of a single dose that is expected to cause the death of 50% of the exposed animals. It is derived from experimental data.

These doses were determined to be LOAELs for these studies.

2,4-DNT has been shown to cause reproductive effects in rats, mice, and dogs (Ellis *et al.*, 1979; Lee *et al.*, 1985; Hong *et al.*, 1985; Ellis *et al.*, 1985). Ellis *et al.* (1979) observed effects in rats following dietary exposure after a dose of 35 mg/kg/day but not 5 mg/kg/day over 3 generations. Male mice fed 2,4-DNT for 13 weeks exhibited testicular degeneration and atrophy and decreased spermatogenesis at 95 mg/kg/day (Hong *et al.*, 1985). In another reproductive study, dogs exhibited mild to severe testicular degeneration and reduced spermatogenesis (Ellis *et al.*, 1985) when administered 2,4-DNT in capsules at 25 mg/kg/day. There are currently no studies of the reproductive or developmental toxicity of 2,6-DNT although a subchronic study in dogs identified atrophy of spermatogenic cells in males suggesting a one- or two-generation study as a data need for 2,6-DNT.

Some studies evaluated the effects of DNT in the form of a technical mixture (tg-DNT). In a study by Price *et al.* (1985), the teratogenic potential of tg-DNT (containing approximately 76 percent 2,4-DNT and 19 percent 2,6-DNT) was investigated in rats. The study was conducted in two phases to evaluate the possible teratogenicity of DNT as well as DNT effects on postnatal development. For the first phase, rats were administered 0, 14, 35, 37.5, 75, 100, or 150 mg/kg/day of DNT in corn oil by gavage. In the postnatal phase, rats were administered 14, 35, 37.5, 75, or 100 mg/kg/day of DNT in corn oil by gavage. The NOAEL and LOAEL for developmental toxicity were 14 and 35 mg/kg/day, respectively, based on significant increases in relative liver and spleen weight in the fetuses of dams administered DNT at levels of 35 mg/kg/day or greater. No teratogenic toxicity was seen in the study rats.

In chronic exposures, oral dietary administration of 2,4-DNT to dogs primarily affected the nervous system, erythrocytes, and biliary tract (Ellis *et al.*, 1979, 1985). Based on neurotoxicity, hematologic changes, and effects on the bile ducts in dogs, the LOAEL was determined to be 1.5 mg/kg/day and the NOAEL was 0.2 mg/kg/day. EPA established an RfD of 0.002 mg/kg/day for 2,4-DNT (USEPA, 1992c) based on this study. An uncertainty factor of 100, to account for interspecies and intraspecies variability, was applied to derive the RfD.

EPA established an RfD of 0.001 mg/kg/day for 2,6-DNT (USEPA, 1992c). This RfD was also based on neurotoxicity, Heinz body formation, biliary tract hyperplasia, liver and kidney histopathology, and death in beagle dogs that were fed gelatin capsules containing 2,6-DNT daily for up to 13 weeks (Lee *et al.*, 1976). The NOAEL for this study was 4 mg/kg/day, and an uncertainty factor of 3,000 (100 for inter- and intra-species variability, 10 for the use of a subchronic study, 3 to account for the limited database) was applied to derive the RfD.

DNT is likely to be carcinogenic to humans (classified as a B2 carcinogen; USEPA, 1990c). This is based on significant increases in hepatocellular carcinoma and mammary gland tumors in female rats fed DNT (98 percent 2,4-DNT with 2 percent 2,6-DNT) in the diet in a two-year study (Ellis *et al.*, 1979). The tumor incidence in the female rats was used to establish a slope factor of 6.67×10^{-1} according to the 1999 EPA guidelines. Concentrations of 5 [mu]g/L, 0.5 [mu]g/L, and 0.05 [mu]g/L are associated with carcinogenic risks of 10^{-4}, 10^{-5}, and 10^{-6} respectively. There were no studies found in the literature that evaluated the effects of 2,4- or 2,6-DNT on children. There is evidence that the pups and fetuses from dams administered tg-DNT had significant increases in relative liver and spleen weights (Price *et al.*, 1985). DNT toxicity may be different in children, compared to adults, since it undergoes bioactivation in the liver and by the intestinal microflora (ATSDR, 1998). Newborns may be more sensitive to DNT-related methemoglobinemia because an enzyme that protects against increased levels of methemoglobin is inactive for a short duration immediately after birth (Gruener 1976; ATSDR, 1998). However, there are no experimental data on differences in children's responses to 2,4-/2,6-DNT.

c. *Occurrence.* EPA included both 2,4- and 2,6-DNT as analytes in the UCMR 1. Because the HRL for both 2,4- and 2,6-DNT (0.05 [mu]g/L) is lower than the minimum reporting limit used for monitoring (MRL of 2 [mu]g/L), EPA used the MRL to evaluate occurrence and exposure. The MRL is within the 10^{-4} to the 10^{-6} cancer risk range for either 2,4- or 2,6-DNT. In evaluating the UCMR 1 data, EPA found that 1 of the 3,866 PWSs sampled (or 0.03 percent) detected 2,4-DNT at the MRL of 2 [mu]g/L, affecting 0.02 percent of the population served (or 38,000 people from 226 million). None of the 3,866 PWSs sampled (serving 226 million) detected 2,6-DNT at the MRL of 2 [mu]g/L (USEPA, 2006a and 2006b).

EPA also evaluated the results of a USGS review of 3 highway and urban runoff studies (Lopes and Dionne, 1998). These studies showed no detects for either 2,4- or 2,6-DNT using a reporting limit of 5 [mu]g/L (a value within the 10^{-4} to 10^{-6} risk range).

d. *Preliminary Determination.* The Agency has made a preliminary determination not to regulate 2,4- or 2,6-DNT with an NPDWR. Because 2,4- and 2,6-DNT appear to occur infrequently at levels of concern in PWSs, the Agency believes that a national primary drinking water regulation does not present a meaningful opportunity for health risk reduction. 2,4-DNT was detected only once at a minimum reporting level that is within the 10^{-4} to the 10^{-6} cancer risk range, while 2,6-DNT was not detected at this same level in any of the PWSs monitored under the UCMR 1.

EPA recognizes that 2,4- and 2,6-DNT are listed as probable human carcinogens. For this reason, the Agency encourages those States with public water systems that may have either 2,4- or 2,6-DNT above the HRL to evaluate site-specific protective measures and to consider whether State-level guidance (or some other type of action) is appropriate. The Agency's original Health Advisories for 2,4- and 2,6-DNT were developed for military installations. Because the Agency recognizes that 2,4- and 2,6-DNT may still be found at some military sites, the Agency has updated the Health Advisories to reflect recent health effects publications. The Health Advisories are available for review in the docket. The updated Health Advisories will provide information to any States with public water systems that may have either 2,4- or 2,6-DNT above the HRL.

8. s-Ethyl dipropylthiocarbamate (EPTC)

a. *Background.* EPTC, a synthetic organic compound, is a thiocarbamate herbicide used to control weed growth during the pre-emergence and early post-emergence stages of weed germination. First registered for use in 1958, EPTC is used across the U.S. in the agricultural production of a number of crops, most notably corn, potatoes, dried beans, alfalfa, and snap beans. EPTC is also used residentially on shade trees, annual and perennial ornamentals, and evergreens (USEPA, 1999c).

Estimates of EPTC usage in the United States suggest a decline from approximately 17 to 21 million pounds active ingredient in 1987 to approximately 7 to 9 million pounds active ingredient in 1999. TRI data from 1995 to 2003 indicate that most on-site industrial releases of EPTC tend to be releases to air and underground injections. Surface water discharges are

minimal in comparison (USEPA, 2006g). Total releases for 2003 were 2,183 lbs.

Environmental fate data indicate that EPTC would not be persistent under most environmental conditions. Volatilization into the atmosphere and degradation by soil organisms appear to be the primary dissipation routes. EPTC has a low affinity for binding to the soil so the potential to leach to ground water does exist. If EPTC reaches ground water, volatilization is less likely to occur (USEPA, 1999c).

b. *Health Effects.* In acute animal toxicity studies, EPTC was shown to be moderately toxic via oral and dermal routes and highly toxic via inhalation exposures. EPTC is a reversible cholinesterase (ChE) inhibitor. Similar to other thiocarbamates, it does not produce a consistent ChE inhibition profile. There was no consistent pattern observed in any of the toxicity studies with regard to species, duration of treatment, or the type of ChE enzyme measured. Typically, studies showed inhibition of plasma ChE with dose-related decreases in red blood cell and brain ChE activity. Some studies have shown that brain ChE activity was inhibited without any effect on either plasma or erythrocyte ChE activities. Other studies illustrated erythrocyte ChE inhibition with no effect on either plasma or brain ChE (USEPA, 1999c). In a primary eye irritation study in rabbits, technical grade EPTC was shown to be slightly irritating (USEPA, 1999c).

In subchronic and chronic studies performed in both rats and dogs, there was a dose-related increase in the incidence and severity of cardiomyopathy, a disorder of the heart muscle (Mackenzie, 1986; USEPA, 1999c). An increase in the incidence and severity of degenerative effects (neuronal and/or necrotic degeneration) in both the central and peripheral nervous system was observed in rats and dogs following exposure to EPTC (USEPA, 1999c).

EPA derived an RfD of 0.025 mg/kg/day for EPTC (USEPA, 1990d; USEPA, 1999c). This value was calculated using a NOAEL of 2.5 mg/kg/day from a study by Mackenzie (1986). An uncertainty factor of 100 was applied for inter- and intraspecies differences. The critical effect associated with the RfD is cardiomyopathy (disease of the heart muscle). In the reregistration of EPTC, the application of a ten-fold Food Quality Protection Act (FQPA) factor was recommended in order to be protective against residential exposures of infants and children. The Agency derived the HRL for EPTC using the RfD of 0.025 mg/kg/day and a 20 percent relative source contribution. The HRL is calculated to be 0.175 mg/L or 175 [mu]g/L.

The Agency used long-term studies in mice and rats and short-term studies of mutagenicity to evaluate the potential for carcinogenicity (USEPA, 1990d). Based on these data and using EPA's 1999 Guidelines for Carcinogen Risk Assessment, EPTC is not likely to be carcinogenic to humans (USEPA, 1999a).

EPA also evaluated whether health information is available regarding the potential effects on children and other sensitive populations. Data do not suggest increased pre- or post-natal sensitivity of children and infants to EPTC exposure. In animal studies, adverse developmental effects (i.e., decreased fetal body weight and decreased litter size) were only seen at doses that were toxic to the mother (USEPA, 1999c). Results from both developmental and reproductive studies indicate that there are only minimal adverse effects. The behavior patterns of children that lead to heightened opportunities for exposure in the indoor environment and the need for a developmental neurotoxicity study lead OPP to recommend the application of a ten-fold FQPA factor for EPTC. However, EPA did not apply this factor in the screening analysis because it does not apply to programs other than the pesticide registrations.

c. *Occurrence.* EPA included EPTC as an analyte in the UCMR 1. None of the 3,866 PWSs sampled (serving a population of 226 million) had detects of EPTC at the MRL of 1 [mu]g/L. Hence, these data indicate that no occurrence and exposure is expected at levels greater than 87.5 [mu]g/L (½ the HRL) and greater than 175 [mu]g/L (the HRL) (USEPA, 2006a and 2006b).

EPA also evaluated several sources of supplemental information, which included:

<bullet≤ The National Pesticide Survey,

<bullet≤ The Pesticides in Ground Water Database, and

<bullet≤ The provisional pesticide results from the 1992–2001 USGS NAWQA survey of ambient surface and ground waters across the U.S.

As part of the National Pesticide Survey, EPA collected samples from approximately 1,300 community water systems and rural drinking water wells between 1988 and 1990. The NPS included EPTC as an analyte in the monitoring survey. EPTC was not detected using a minimum reporting limit of 0.15 [mu]g/L (USEPA, 1990a).

The Pesticides in Ground Water Database (USEPA, 1992b) indicates that EPTC was found in 2 of 1,752 ground water wells that were sampled in 10 States. Both contaminated wells were in Minnesota. The detected concentrations ranged from 0.01 to 0.33 [mu]g/L. All of these positive detections are less than the HRL of 175 [mu]g/L, as well as 87.5 [mu]g/L (½ the HRL).

The USGS NAWQA program included EPTC as an analyte in its 1992–2001 monitoring survey of ambient surface and ground waters across the United States. EPA evaluated the results of the provisional data, which are available on the Web (Martin *et al.*, 2003; Kolpin and Martin, 2003). While the USGS detected EPTC in both surface and ground waters, 95 percent of the samples from the various land use settings were less than or equal to 0.018 [mu]g/L. The estimated maximum surface water concentration, 29.6 [mu]g/L (mixed land use settings), and the maximum ground water concentration, 0.45 [mu]g/L (agricultural settings), are both less than 175 [mu]g/L (the EPTC HRL).

d. *Preliminary Determination.* The Agency has made a preliminary determination not to regulate EPTC with an NPDWR. Because EPTC does not appear to occur at health levels of concern in PWSs, the Agency believes that a national primary drinking water regulation does not present a meaningful opportunity for health risk reduction. While EPTC has been found in ambient waters, it was detected only at levels less than the HRL (as well as ½ the HRL) and it was not found in the UCMR 1 survey of public water supplies.

9. Fonofos

a. *Background.* Fonofos, an organophosphate, is a soil insecticide used to control pests such as corn rootworms, cutworms, symphylans (i.e., garden centipedes), and wireworms. Primarily used on corn crops, fonofos was also used on other crops such as asparagus, beans, beets, corn, onions, peppers, tomatoes, cole crops, sweet potatoes, peanuts, peas, peppermint, plantains, sorghum, soybeans, spearmint, strawberries, sugarcane, sugar beets, white (Irish) potatoes, and tobacco (USEPA, 1999d).

Fonofos was scheduled for a reregistration decision in 1999. However, before the review was completed, the registrant requested voluntary cancellation. The cancellation was announced in the **Federal Register** on May 6, 1998 (63 FR 25033 (USEPA, 1998d)), with an effective date of November 2, 1998, plus a one-year grace period to permit the exhaustion of existing stocks (USEPA, 1999d).

NCFAP data indicate that fonofos use declined significantly during the 1990s (NCFAP, 2004). According to NCFAP,

approximately 3.2 million pounds of fonofos a.i. were applied annually around 1992 and approximately 0.4 million pounds a.i. were applied annually around 1997. The U.S. Geological Survey (USGS) estimates an average of 2.7 million pounds a.i. were used annually around 1992 (Thelin and Gianessi, 2000).

Fonofos is moderately persistent in soil and its persistence depends on soil type, organic matter, rainfall, and sunlight. Since fonofos adsorbs moderately well to soil, it is not readily leached or transported to ground water but it can be transported to surface waters in runoff. Fonofos is rapidly degraded by soil microorganisms (Extoxnet, 1993). Fonofos tends to volatilize from wet soil and water surfaces, but the process is slowed by adsorption to organic material in soil, suspended solids, and sediment (HSDB, 2004d).

b. *Health Effects.* Fonofos (like many organophosphates) is toxic to humans and animals. Case reports and acute oral toxicity studies in animals indicate that oral exposure to fonofos induces clinical signs of toxicity that are typical of cholinesterase inhibitors. In humans, accidental exposures produced symptoms of acute intoxication, nausea, vomiting, salivation, sweating, muscle twitches, decreased blood pressure and pulse rate, pinpoint pupils, profuse salivary and bronchial secretions, cardiorespiratory arrest, and even death in 1 exposed individual (Hayes, 1982; Pena Gonzalez *et al.*, 1996).

In animals, clinical signs of exposure included tremors, salivation, diarrhea, and labored breathing (USEPA, 1996c). Chronic exposure studies also indicated that oral administration of fonofos inhibits cholinesterase (Banerjee *et al.*, 1968; Cockrell *et al.*, 1966; Hodge, 1995; Horner, 1993; Miller, 1987; Miller *et al.*, 1979; Pavkov and Taylor, 1988; Woodard *et al.*, 1969). Cholinesterase inhibition is one of the critical effects associated with the RfD, which was verified by EPA (USEPA, 1991) at 0.002 mg/kg/day. EPA derived the RfD of 0.002 mg/kg/day using a NOAEL of 0.2 mg/kg/day (Hodge, 1995) and a 100-fold uncertainty factor to account for inter- and intraspecies differences.

Fonofos is classified as an unlikely human carcinogen (Group E) because there is no evidence of carcinogenic potential in the available long-term feeding studies in rats and mice (Banerjee *et al.*, 1968; Pavkov and Taylor, 1988; Sprague and Zwicker, 1987). In addition, fonofos does not appear to be mutagenic (USEPA, 1996c).

EPA evaluated whether health information is available regarding the potential effects on children and other sensitive populations. In the available developmental studies with rabbits (Sauerhoff, 1987) and mice (Minor *et al.*, 1982; Pulsford, 1991), no developmental effects were observed at oral doses as high as 1.5 mg/kg/day in the rabbit (highest dose tested) nor in mice at doses as high as 2.0 mg/kg/day (Minor *et al.*, 1982; Pulsford, 1991). However, in mice, effects were noted at higher dose levels. These effects included an increase in the incidence of variant sternebrae ossifications (at 6 mg/kg/day or greater) and a slight dilation of the fourth brain ventricle in offspring (at 4 mg/kg/day or greater). No developmental neurotoxicity study with fonofos is available for further assessment of this endpoint. In a three-generation reproduction study in rats (Woodard *et al.*, 1968), no treatment-related adverse effects were observed at the 2 dose levels used in this study, 0.5 and 1.58 mg/kg/day.

The Agency believes that the current RfD is adequately protective of children. The current fonofos RfD of 0.002 mg/kg/day is 1000-fold lower than the NOAEL observed in the Woodard *et al.* (1968) developmental studies.

Using the RfD of 0.002 mg/kg/day for fonofos and a 20 percent screening relative source contribution, the Agency derived an HRL of 0.014 mg/L and rounded to 0.01 mg/L (or 10 [mu]g/L).

c. *Occurrence.* EPA included fonofos as an analyte in the UCMR 1 List 2 Screening Survey. None of the 2,306 samples from the 295 PWSs sampled (serving a population of 41 million) contained detects for fonofos at the MRL of 0.5 [mu]g/L. Hence, these data indicate that no occurrence and exposure is expected at levels greater than 5 [mu]g/L (½ the HRL) and greater than 10 [mu]g/L (the HRL) (USEPA, 2006a and 2006b).

The USGS NAWQA program included fonofos as an analyte in its 1992–2001 monitoring survey of ambient surface and ground waters across the United States. EPA evaluated the results of the provisional data, which are available on the Web (Martin *et al.*, 2003; Kolpin and Martin, 2003). While the USGS detected fonofos in both surface and ground waters, 95 percent of the samples from the various land use settings were less than 0.003 [mu]g/L (the reporting limit). The maximum surface water concentration, 1.20 [mu]g/L (agricultural setting), and the maximum ground water concentration, 0.009 [mu]g/L (agricultural setting), are both less than 10 [mu]g/L and less than 5 [mu]g/L (the fonofos HRL and ½ the HRL).

d. *Preliminary Determination.* The Agency has made a preliminary determination not to regulate fonofos with an NPDWR. Because fonofos does not appear to occur at health levels of concern in PWSs, the Agency believes that a national primary drinking water regulation does not present a meaningful opportunity for health risk reduction. While fonofos has been found in ambient waters, it was detected only at levels less than the HRL (as well as ½ the HRL) and it was not found in UCMR 1 Screening Survey of public water supplies. Fonofos was voluntarily cancelled in 1998 and the Agency expects any remaining stocks and releases into the environment to decline. In addition, since fonofos tends to bind strongly to soil, any releases to the environment are not likely to contaminant source waters.

10. Terbacil

a. *Background.* Terbacil, a synthetic organic compound, is a selective herbicide used to control broadleaf weeds and grasses on terrestrial food/feed crops (e.g., apples, mint, peppermint, spearmint, and sugarcane), terrestrial food (e.g., asparagus, blackberry, boysenberry, dewberry, loganberry, peach, raspberry, youngberry, and strawberry), terrestrial feed (e.g., alfalfa, forage, and hay) and forest trees (e.g., cottonwood) (USEPA, 1998e).

In 1998, EPA estimated that agricultural usage of terbacil consumed approximately 221,000 to 447,000 pounds of active ingredient annually and non-agricultural usage consumed approximately 9,000 to 14,000 pounds. These estimates are based on data collected mostly between 1990 and 1995, and in some cases as early as 1987 (USEPA, 1998e). According to NCFAP (2004), approximately 298,000 pounds of terbacil a.i. were applied annually in agriculture around 1992 and approximately 342,000 pounds a.i. were applied around 1997.

Terbacil is listed as a TRI chemical and data are reported from one or more facilities in a single state, Texas, for the time period covering 1995 to 1997. During this three-year period, all reported releases were on-site releases to surface water that varied between 3,000 to 10,000 pounds annually (USEPA, 2006h).

Terbacil is considered a persistent and potentially mobile herbicide in terrestrial environments. Because of its low affinity to soils, it can potentially leach into ground and/or surface waters (USEPA, 1998e; Extoxnet, 1994).

b. *Health Effects.* In acute and subchronic toxicity studies, terbacil is practically non-toxic (Haskell Laboratories, 1965a and 1965b). Terbacil does not cause dermal sensitivity in

rabbits or guinea pigs and causes mild conjunctival eye irritation in rabbits (Henry, 1986; Hood, 1966). In rats exposed subchronically to dietary terbacil, effects were seen at a LOAEL of 25 mg/kg/day and included increased absolute and relative liver weights, vacuolization, and enlargement of liver cells (Wazeter *et al.*,1964; Haskell Laboratories, 1965c).

A primary target organ in rats following exposure to terbacil is the liver. Chronic effects of dietary terbacil exposure in two-year studies included increases in thyroid-to-body weight ratios, slight increases in liver weights and elevated alkaline phosphatase levels in beagle dogs, significant decreases in body weight in rats, increases in serum cholesterol levels and increases in liver to body weight ratios in rats (Wazeter *et al.*,1967a; Malek, 1993). In beagle dogs, effects were seen at or above 6.25 mg/kg/day (NOAEL = 1.25 mg/kg/day). In rats, effects (*i.e.*, decreases in body weight, increases in liver weights and cholesterol levels) were seen at higher levels (LOAELs = 56 mg/kg/day for males and 83 mg/kg/day for females).

Terbacil is not considered to be a developmental or reproductive toxicant. In developmental studies, maternal effects were generally seen prior to or at the same levels as developmental effects. Haskell Laboratories (1980) reported maternal effects (*i.e.*, decreased body weight) and significant decreases in the number of live fetuses per litter due to early fetal resorption at a LOAEL of 62.5 mg/kg/day in rats. In rabbits administered terbacil via gavage, the maternal and developmental LOAELs were equal (600 mg/kg/day). Maternal toxicity was based on the death of the dams and developmental toxicity was based on a decrease in live fetal weights (Solomon, 1984). No reproductive effects were seen in a three-generation study where terbacil was administered to male and female rats at dose levels of 2.5 and 12.5 mg/kg/day (Wazeter *et al.*, 1967b).

Terbacil is not mutagenic. Terbacil was tested and found negative in a chromosomal aberration study in rat bone marrow cells, found negative in a gene mutation assay (with and without S9 activation), and found negative for DNA synthesis when tested up to cytotoxic levels in rats (Cortina, 1984; Haskell Laboratories,1984). Terbacil shows no evidence of carcinogenicity and is unlikely to be carcinogenic to humans (Group E) (USEPA, 1998e).

The RfD of 0.013 mg/kg/day for terbacil (USEPA, 1998e) is calculated from a two-year chronic study in beagle dogs. The LOAEL of 6.25 mg/kg/day was based on increased thyroid-to-body weight ratios, slight increases in liver weights, and elevated alkaline phosphatase levels with a NOAEL of 1.25 mg/kg/day. In deriving the RfD, the Agency applied an uncertainty factor of 100 to account for interspecies and intraspecies differences. Using the RfD of 0.013 mg/kg/day and applying a 20 percent screening relative source contribution, the Agency derived an HRL of 0.090 mg/L (or 90 g/L) for terbacil.

EPA also evaluated whether health information is available regarding the potential effects on children and other sensitive populations. In the case of terbacil, the Agency determined that there was no need to apply an FQPA factor to the RfD in order to protect children (USEPA, 1998e). Other potentially sensitive subpopulations have not been identified.

c. *Occurrence.* EPA included terbacil as an analyte in UCMR 1. None of the 3,866 PWSs sampled (serving a population of 226 million) had detects for terbacil at the MRL of 2 g/L. Hence, these data indicate that no occurrence and exposure is expected at levels greater than 45 g/L (½ the HRL) and greater than 90 [mu]g/L (the terbacil HRL) (USEPA, 2006a and 2006b).

EPA also evaluated several sources of supplemental information, which included:

<bullet≤ The National Pesticide Survey,

<bullet≤ The Pesticides in Ground Water Database, and

<bullet≤ The provisional pesticide results from the 1992–2001 USGS NAWQA survey of ambient surface and ground waters across the U.S.

As part of the National Pesticide Survey, EPA collected samples from approximately 1,300 community water systems and rural drinking water wells between 1988 and 1990. The NPS included terbacil as an analyte in the monitoring survey. Terbacil was not detected using a minimum reporting limit of 1.7 [mu]g/L (USEPA, 1990a).

The Pesticides in Ground Water Database (USEPA, 1992b) indicates that terbacil was found in 6 of the 288 ground water wells tested for this contaminant in 6 States. Terbacil was found in 1 ground water well in Oregon (at a concentration of 8.9 [mu]g/L) and 5 ground water wells in West Virginia (with concentrations ranging from 0.3 to 1.2 [mu]g/L). All of the positive detections are less than the HRL of 90 [mu]g/L, as well as 45 [mu]g/L (½ the HRL).

The USGS NAWQA program included terbacil as an analyte in its 1992–2001 monitoring survey of ambient surface and ground waters across the United States. EPA evaluated the results of the provisional data, which are available on the Web (Martin *et al.*, 2003; Kolpin and Martin, 2003). While the USGS detected terbacil in both surface and ground waters, 95 percent of the samples from the various land use settings were less than 0.034 [mu]g/L (the USGS reporting limit). The maximum surface water concentration, 0.54 [mu]g/L (agricultural setting), and the maximum ground water concentration, 0.891 [mu]g/L (mixed land use setting), are both less than 90 [mu]g/L and less than 45 [mu]g/L (the terbacil HRL and ½ the HRL).

d. *Preliminary Determination.* The Agency has made a preliminary determination not to regulate terbacil with an NPDWR. Because terbacil does not appear to occur at health levels of concern in PWSs, the Agency believes that a national primary drinking water regulation does not present a meaningful opportunity for health risk reduction. Terbacil has been found in ambient waters but the levels were less than the HRL (as well as ½ the HRL). It was not found in the UCMR 1 survey of public water supplies.

11. 1,1,2,2-Tetrachloroethane

a. *Background.* 1,1,2,2-Tetrachloroethane, a volatile organic compound, is not known to occur naturally in the environment (IARC, 1979). Prior to the 1980s, 1,1,2,2-tetrachloroethane was synthesized for use in the production of other chemicals, primarily chlorinated ethylenes. 1,1,2,2-Tetrachloroethane was also once used as a solvent to clean and degrease metals, in paint removers, varnishes, lacquers, and photographic films, and for oil/fat extraction (Hawley, 1981). Commercial production of 1,1,2,2-tetrachloroethane in the U.S. ceased in the 1980s when other processes to generate chlorinated ethylenes were discovered (ATSDR, 1996).

Production of 1,1,2,2-tetrachloroethane in the U.S. was approximately 440 million pounds in 1967 (Konietzko, 1984). Production declined to an estimated 34 million pounds by 1974 (ATSDR, 1996). Although U.S. commercial production ceased in the 1980s, 1,1,2,2-tetrachloroethane is still generated as a byproduct and/or intermediate in the production of other chemicals. TRI data indicate that environmental releases have generally declined from a high of about 175,000 pounds in 1988 to a low of 3,500 pounds in 2003. Most releases took the form of air emissions, though surface water discharges were also documented nearly every year (USEPA, 2006i).

Volatilization from water or soil surfaces to the atmosphere appears to be the primary dissipation route for 1,1,2,2-tetrachloroethane. In subsurface soils and ground water, 1,1,2,2-tetrachloroethane is subject to biodegradation by soil organisms and/or chemical hydrolysis by water (ATSDR, 1996).

b. *Health Effects.* Data on the toxicity of 1,1,2,2-tetrachloroethane in humans are limited, consisting of one experimental inhalation study, a few case reports of suicidal or accidental ingestion, and dated occupational studies. In most cases, there was no quantification of the exposure. Respiratory and mucosal effects, eye irritation, nausea, vomiting, and dizziness were reported by human volunteers exposed to 1,1,2,2-tetrachloroethane vapors under controlled chamber conditions (Lehmann and Schmidt-Kehl, 1936). Effects from non-lethal occupational exposures included gastric distress (i.e., pain, nausea, vomiting), headache, loss of appetite, an enlarged liver, and cirrhosis (Jeney *et al.*, 1957; Lobo-Mendonca, 1963; Minot and Smith, 1921).

There have been a variety of animal studies in rats and mice using both the inhalation and oral exposure routes. Recent studies by the National Toxicology Program (NTP, 2004) provide a detailed evaluation of the short-term and subchronic oral toxicity of 1,1,2,2-tetrachloroethane and confirm many of the observations from earlier studies. In rats and mice exposed orally, the liver appears to be the primary target organ. The RfD (10 [mu]g/kg/day) for 1,1,2,2-tetrachloroethane was derived from the BMDL for a 1 standard deviation change in relative liver weight, a biomarker for liver toxicity. A 1,000-fold uncertainty factor was applied in the RfD determination.

A National Cancer Institute (1978) bioassay of 1,1,2,2-tetrachloroethane found clear evidence of carcinogenicity in male and female B6C3F1 mice based on a dose-related statistically significant increase in liver tumors. There was equivocal evidence for carcinogenicity in Osborn Mendel rats because of the occurrence of a small number of rare-for-the species neoplastic and preneoplastic lesions in the livers of the high dose animals. The Agency used the slope factor of 8.5×10^{-2} for the tumors in female mice to derive the HRL of 0.4 [mu]g/L for use in the analysis of the occurrence data for 1,1,2,2-tetrachloroethane. Information on the reproductive effects of 1,1,2,2-tetrachloroethane is limited. There is a single one-generation inhalation study that does not follow a standard methodology and examined a small number of rats (5 females and 7 males) exposed via inhalation to 1 dose (13.3 mg/m^3). There were no statistically significant differences in the percentage of females having offspring, number of pups per litter, average birth weight, sex ratio, or post natal offspring mortality (Schmidt *et al.*, 1972). Effects on sperm in male rats were seen after oral (27 mg/kg/day; NTP, 2004) and inhalation (13 mg/m^3; Schmidt *et al.*, 1972) exposures. Similar effects were seen in mice but at higher doses. Fetal toxicity did not occur in the absence of maternal toxicity.

Developmental range-finding studies conducted for NTP (1991a and b) found that 1,1,2,2-tetrachloroethane was toxic to the dams and pups of Sprague Dawley rats and CD–1 Swiss mice. Rats were more sensitive than mice. The NOAEL in the rats for both maternal toxicity and associated fetal toxicity was 34 mg/kg/day with a LOAEL of 98 mg/kg/day. In mice, the NOAEL was 987 mg/kg/day and the LOAEL was 2,120 mg/kg/day.

EPA also evaluated whether health information is available regarding the potential effects on children and other sensitive populations. Individuals with preexisting liver and kidney damage would likely be sensitive to 1,1,2,2-tetrachloroethane exposure. Low intake of antioxidant nutrients (e.g., Vitamin E, Vitamin C, and selenium) could be a predisposing factor for liver damage. In addition, individuals with a genetically low capacity to metabolize dichloroacetic acid (the primary metabolite of 1,1,2,2-tetrachloroethane) may be at greater risk than the general population as a result of 1,1,2,2-tetrachloroethane exposure.

c. *Occurrence.* EPA included 1,1,2,2-tetrachloroethane as an analyte in the UCM Round 1 and UCM Round 2 surveys. EPA evaluated the UCM Round 1 Cross Section and the UCM Round 2 Cross Section data at levels greater than 0.2 [mu]g/L (½ the HRL) and greater than 0.4 [mu]g/L (the HRL) (USEPA, 2006a and 2006c). The MRLs for UCM Round 1 ranged from 0.1 to 10 [mu]g/L and the MRLs for UCM Round 2 ranged from 0.1 to 2.5 [mu]g/L. Because some of the reporting limits exceeded the thresholds of interest, the occurrence analyses may result in an underestimate of systems affected. However, all the MRL values used for UCM Round 1 and UCM Round 2 are within the 10^{-4} to the 10^{-6} cancer risk range.

Analysis of UCM Round 1 Cross Section data indicates that approximately 0.22 percent (or 44) of the 20,407 PWSs sampled had detections of 1,1,2,2-tetrachloroethane at levels greater than 0.20 [mu]g/L (½ the HRL), affecting approximately 1.69 percent of the population served (or 1.6 million of 95 million). The UCM Round 1 Cross Section data indicate that approximately 0.20 percent (or 41) of the 20,407 PWSs sampled had detections of 1,1,2,2-tetrachloroethane at levels greater than 0.4 [mu]g/L (the HRL), affecting approximately 1.63 percent of the population served (or 1.5 million of 95 million). The 99th percentile of all detects is 112 [mu]g/L and the maximum reported value is 200 [mu]g/L.

Analysis of the UCM Round 2 Cross Section data indicate that approximately 0.07 percent (or 18) of the 24,800 PWSs sampled had detections of 1,1,2,2-tetrachloroethane at levels greater than 0.2 [mu]g/L (½ the HRL), affecting approximately 0.51 percent of the population served (or 362,000 of 71 million). The UCM Round 2 Cross Section data indicate that approximately the same percentage and number of the PWSs sampled (0.07 percent or 17 of the 24,800) had detections of 1,1,2,2-tetrachloroethane at levels greater than 0.4 [mu]g/L (the HRL), affecting approximately 0.08 percent of the population served (or 56,000 of 71 million). The 99th percentile of all detects is 2 [mu]g/L and the maximum reported value is 2 [mu]g/L.

EPA also evaluated several sources of supplemental information, which included the USGS VOC National Synthesis Random Source Water Survey and the Focused Source Water Survey. For the Random Source Water Survey, the USGS collected samples from 954 source waters that supply community water systems between 1999 and 2000. For the Focused Source Water Survey, the USGS collected 451 samples from 134 source waters that supply community water systems between 1999 and 2001. The USGS included 1,1,2,2-tetrachloroethane as an analyte in both surveys and did not detect it in any of the source water samples using a reporting limit of 0.2 [mu]g/L (a level that is less than the 1,1,2,2-tetrachloroethane HRL). In addition, USGS did not detect 1,1,2,2-tetrachloroethane when using a detection level of 0.026 [mu]g/L (a level that is over 10 times lower than the 1,1,2,2-tetrachloroethane HRL) in the focused survey (Ivahnenko *et al.*, 2001, Grady, 2003, Delzer and Ivahnenko, 2003a).

d. *Preliminary Determination.* The Agency has made a preliminary determination not to regulate 1,1,2,2-tetrachloroethane with an NPDWR. Because 1,1,2,2-tetrachloroethane appears to occur infrequently at health levels of concern in PWSs, the Agency

believes that a national primary drinking water regulation does not present a meaningful opportunity for health risk reduction. While 1,1,2,2-tetrachloroethane was detected in both the UCM Round 1 and the UCM Round 2 surveys, the percentage of detections had decreased by the time the UCM Round 2 survey was performed in the mid-1990's. In addition, the USGS did not detect 1,1,2,2-tetrachloroethane in two subsequent monitoring surveys of source waters that supply community water systems using a reporting limit that is less than the 1,1,2,2-tetrachloroethane HRL. The Agency believes that this decrease in detections occurred because commercial production of 1,1,2,2-tetrachloroethane ceased in the mid-1980's. Hence, the Agency does not expect 1,1,2,2-tetrachloroethane to occur in many public water systems today.

EPA recognizes that 1,1,2,2-tetrachloroethane is listed as a likely human carcinogen. For this reason, the Agency encourages those States with public water systems that may have 1,1,2,2-tetrachloroethane above the HRL to evaluate site-specific protective measures and to consider whether State-level guidance (or some other type of action) is appropriate. The Agency also plans to update the Health Advisory document for 1,1,2,2-tetrachloroethane to provide more recent health information. The updated Health Advisory will provide information to any States with public water systems that may have 1,1,2,2-tetrachloroethane at levels above the HRL.

V. What Is the Status of the Agency's Evaluation of Perchlorate?

At this time, the Agency is not making a preliminary determination as to whether a national primary drinking water regulation is needed for perchlorate. However, the Agency has placed a high priority on making a regulatory determination for perchlorate and will publish a preliminary determination as soon as possible. EPA is not able to make a preliminary determination at this time because, in order to evaluate perchlorate against the three SDWA statutory criteria, the Agency believes additional information may be needed to more fully characterize perchlorate exposure and determine whether regulating perchlorate in drinking water presents a meaningful opportunity for health risk reduction. This is particularly true if the Agency uses food exposure data to first calculate a relative source contribution (RSC) and corresponding health reference level (HRL) below the drinking water equivalent level (DWEL)[18] in order to determine whether regulating perchlorate would present a meaningful opportunity for health risk reduction. However, the Agency is considering several other approaches, discussed below, for making this statutory determination and is requesting public comment on the strengths and limitations of these approaches.

The following sections explain why EPA is not making a preliminary regulatory determination for perchlorate at this time, and discusses the information the Agency has collected to date (that may be relevant to making a preliminary regulatory determination), the additional information the Agency is soliciting in this action, and options for additional analyses that the Agency may conduct to support a regulatory determination. Sections V.A through V.D provide a summary of the available and relevant information/data that the Agency has collected and reviewed regarding the sources of perchlorate in the environment, its potential health effects, and its occurrence in drinking water, food, human urine, breast milk, and amniotic fluid. Section V.E explains the Agency's basis for not making a preliminary regulatory determination for perchlorate at this time and Section V.F. presents the options the Agency is considering to better characterize perchlorate exposure and the alternate approaches that EPA is considering for making a preliminary regulatory determination. This action provides an opportunity for the public to submit other relevant data that may further characterize exposure to perchlorate through the consumption of foods and/or through other pathways and to comment on these alternate approaches. The Agency in particular seeks comment on the use of urine biomonitoring data in estimating perchlorate exposure. The Agency will consider any relevant information/data provided in response to this action as the Agency determines whether to regulate perchlorate with a national primary drinking water regulation and how best to proceed to address perchlorate.

A. Sources of Perchlorate

Perchlorate (ClO_4^-) is an anion commonly associated with the solid salts of ammonium, magnesium, potassium, and sodium perchlorate. Perchlorate salts are highly soluble in water, and because perchlorate sorbs poorly to mineral surfaces and organic material, perchlorate can be mobile in surface and subsurface aqueous environments. Although commonly known as a man-made chemical, perchlorate also may be derived from natural processes.

While perchlorate has a wide variety of industrial uses, it is primarily used in the form of ammonium perchlorate as an oxidizer in solid fuels used to power rockets, missiles, and fireworks. Approximately 90 percent of perchlorate is manufactured for this application (Wang *et al.*, 2002). Perchlorate can also be present as an ingredient or as an impurity in road flares, lubricating oils, matches, aluminum refining, rubber manufacturing, paint and enamel manufacturing, leather tanning, paper and pulp processing (as an ingredient in bleaching powder), and as a dye mordant.

Perchlorate can also occur naturally in the environment. Chile possesses caliche ores rich in sodium nitrate ($NaNO_3$), which are also a natural source of perchlorate (Schilt, 1979 and Ericksen, 1983). These Chilean nitrate salts (saltpeter) have been mined and refined to produce commercial fertilizers, which before 2001 accounted for about 0.14 percent of U.S. fertilizer application (USEPA, 2001d). The USEPA (2001d) conducted a broad survey of fertilizers and other raw materials and found that all products surveyed were devoid of perchlorate except for those known to contain or to be derived from mined Chilean saltpeter.

Perchlorate has also been found in other geologic materials. Orris *et al.* (2003) measured perchlorate at levels exceeding 1,000 parts per million (ppm or mg/kg) in several samples of natural minerals, including potash ore from New Mexico and Saskatchewan (Canada), playa crust from Bolivia, and hanksite from California.

Texas Tech University Water Resources Center conducted a large-scale sampling program to determine the source and distribution of perchlorate in northwest Texas groundwater (Jackson *et al.*, 2004; Rajagopalan *et al.*, 2006). Perchlorate was detected at concentrations greater than 0.5 g/L in 46 percent of public wells and 47 percent of private wells. Jackson *et al.* (2004) hypothesized that atmospheric production and/or surface oxidative weathering is the source of the perchlorate. In related research, Dasgupta *et al.* (2005) detected perchlorate in many rain and snow samples and demonstrated that perchlorate is formed by a variety of simulated atmospheric processes suggesting that natural, atmospherically-

[18] DWEL = [(Reference Dose x Body Weight of 70 kg) / Drinking Water Intake of 2 L per day].

derived perchlorate exists in the environment. Barron *et al.* (2006) developed a method for the rapid determination of perchlorate in rainwater samples, with a detection limit between 70 and 80 ng/L. Of the ten rainwater samples collected in Ireland in 2005, perchlorate was detected in 4 samples at concentrations between 0.075 and 0.113 g/L, and in 1 other sample at 2.8 g/L. Kang *et al.* (2006) conducted seven-day experiments to determine if it was possible to produce perchlorate by exposing various chlorine intermediates to UV radiation in the form of high intensity UV lamps and/or ambient solar radiation. Perchlorate formation was demonstrated in aqueous salt solutions with initial concentrations of hypochlorite, chlorite, or chlorate between 100 and 10,000 mg/L.

After a limited investigation, the Massachusetts Department of Environmental Quality (MA DEP, 2005) found that perchlorate may be present in sodium hypochlorite solutions used in water and wastewater treatment plants, and that the level of occurrence depends upon storage conditions and the initial purity of the stock solution (MA DEP, 2005). According to MA DEP (2005), the Town of Tewksbury conducted a small study to evaluate the impact of storage conditions (temperature and light) on a new shipment of sodium hypochlorite stock solution. Tewksbury found that the perchlorate concentration in the new stock solution increased from 0.2 g/L to levels ranging from 995 to 6,750 g/L depending on the storage conditions. Accounting for the large dilution factor (*e.g.*, 20,000 to 1 ratio) used in chlorination processes at drinking water treatment plants, MA DEP (2005) concluded that ''absent additional efforts to minimize breakdown of hypochlorite solutions, it would appear that low levels of the perchlorate ion (0.2 to 0.4 g/L) detected in a drinking water supply disinfected with sodium hypochlorite solutions could be attributable to the chlorination process.''

It is not clear at this time what proportion of perchlorate found in public water supplies or entering the food chain comes from these various anthropogenic and natural sources. The significance of different sources probably varies regionally. A study by Dasgupta *et al.* (2006) analyzes the three principal sources of perchlorate and their relative contributions to the food chain. These are its use as an oxidizer including rocket propellants, Chilean nitrate used principally as fertilizer, and that produced by natural atmospheric processes.

B. Health Effects

Perchlorate can interfere with the normal functioning of the thyroid gland by competitively inhibiting the transport of iodide into the thyroid. Iodide is an important component of two thyroid hormones, T4 and T3, and the transfer of iodide from the blood into the thyroid is an essential step in the synthesis of these two hormones. Iodide transport into the thyroid is mediated by a protein molecule known as the sodium (Na+)—iodide (I-) symporter (NIS). NIS molecules bind iodide with very high affinity, but they also bind other ions that have a similar shape and electric charge, such as perchlorate. The binding of these other ions to the NIS inhibits iodide transport into the thyroid, which can result in intrathyroidal iodide deficiency and consequently decreased synthesis of T4 and T3. There is compensation for iodide deficiency, however, such that the body maintains the serum concentrations of thyroid hormones within narrow limits through feedback control mechanisms. This feedback includes increased secretion of thyroid stimulating hormone (TSH) from the pituitary gland, which has among its effects the increased production of T4 and T3 (USEPA, 2005e). Sustained changes in thyroid hormone and TSH secretion can result in thyroid hypertrophy and hyperplasia (abnormal growth or enlargement of the thyroid) (USEPA, 2005e).

In January 2005, the National Research Council (NRC) of the National Academies of Science (NAS) published ''Health Implications of Perchlorate Ingestion,'' a review of the current state of the science regarding potential adverse health effects of perchlorate exposure and mode-of-action for perchlorate toxicity (NRC, 2005). Based on recommendations of the NRC, EPA chose data from the Greer *et al.* (2002) human clinical study as the basis for deriving a reference dose (RfD) for perchlorate (USEPA, 2005e). Greer *et al.* (2002) report the results of a well-controlled study that measured thyroid iodide uptake, hormone levels, and urinary iodide excretion in a group of 24 healthy adults administered perchlorate doses orally over a period of 14 days. Dose levels ranged from 0.007 to 0.5 mg/kg/day in the different experimental groups. No significant differences were seen in measured serum thyroid hormone levels (T3, T4, total and free) in any dose group. The statistical no observed effect level (NOEL) for perchlorate-induced inhibition of thyroid iodide uptake was 0.007 mg/kg/day. Although the NRC committee concluded that hypothyroidism is the first adverse effect in the continuum of effects of perchlorate exposure, NRC recommended that ''the most health-protective and scientifically valid approach'' was to base the perchlorate RfD on the inhibition of iodide uptake by the thyroid (NRC, 2005). NRC concluded that iodide uptake inhibition, although not adverse, is the key biochemical event in the continuum of possible effects of perchlorate exposure and would precede any adverse health effects of perchlorate exposure. The lowest dose (0.007 mg/kg/day) administered in the Greer *et al.* (2002) study was considered a NOEL (rather than a NOAEL) because iodide uptake inhibition is not an adverse effect but a biochemical change (USEPA, 2005e). A summary of the data considered and the NRC deliberations can be found in the NRC report (2005) and the EPA Integrated Risk Information System (IRIS) summary (USEPA, 2005e).

The NRC recommended that EPA apply an intraspecies uncertainty factor of 10 to the NOEL to account for differences in sensitivity between the healthy adults in the Greer *et al.* (2002) study and the most sensitive population, fetuses of pregnant women who might have hypothyroidism or iodide deficiency. Because the fetus depends on an adequate supply of maternal thyroid hormone for its central nervous system development during the first trimester of pregnancy, iodide uptake inhibition from low-level perchlorate exposure has been identified as a concern in connection with increasing the risk of neurodevelopmental impairment in fetuses of high-risk mothers (NRC, 2005). The NRC (2005) viewed the uncertainty factor of 10 as conservative and health protective given that the point of departure is based on a non-adverse effect (iodide uptake inhibition) that precedes the adverse effect in a continuum of possible effects of perchlorate exposure. NRC concluded that no uncertainty factor was needed for the use of a less-than chronic study, for deficiencies in the database, or for interspecies variability. To protect the most sensitive human population from chronic perchlorate exposure, EPA derived an RfD of 0.0007 mg/kg/day with a ten-fold total uncertainty factor from the NOEL of 0.007 mg/kg/day (USEPA, 2005e).

Blount *et al.* (2006b) recently published a study examining the relationship between urinary levels of perchlorate and serum levels of TSH and total T4 in 2,299 men and women (ages 12 years and older), who participated in CDC's 2001–2002

National Health and Nutrition Examination Survey (NHANES).[19] Blount *et al.* (2006b) evaluated perchlorate along with covariates known or likely to be associated with T4 or TSH levels to assess the relationship between perchlorate and these hormones, and the influence of other factors on this relationship. These covariates included sex, age, race/ethnicity, body mass index, serum albumin, serum cotinine (a marker of tobacco smoke exposure), estimated total caloric intake, pregnancy status, post-menopausal status, premenarche status, serum C-reactive protein, hours fasting before sample collection, urinary thiocyanate, urinary nitrate, and use of selected medications. The study found that perchlorate was a significant predictor of thyroid hormones in women, but not men. After finding evidence of gender differences, the researchers focused on further analyzing the NHANES data for the 1,111 women participants. They divided these 1,111 women into two categories, higher-iodide and lower-iodide, using a cut point of 100 [mu]g/L of urinary iodide based on the World Health Organization (WHO) definition of sufficient iodide intake.[20] Hypothyroid women were excluded from the analysis. According to the study authors, about 36 percent of women living in the United States have urinary iodide levels less than 100 [mu]g/L (Caldwell *et al.*, 2005). For women with urinary iodide levels less than 100 [mu]g/L, the study found that urinary perchlorate is associated with a decrease in (a negative predictor for) T4 levels and an increase in (a positive predictor for) TSH levels. For women with urinary iodide levels greater than or equal to 100 [mu]g/L, the researchers found that perchlorate is a significant positive predictor of TSH but not a predictor of T4. The study found that perchlorate was not a significant predictor of T4 or TSH in men. The researchers state that perchlorate could be a surrogate for another unrecognized determinant of thyroid function. Also, the study reports that while large doses of perchlorate are known to decrease thyroid function, this is the first time an association of decreased thyroid function has been observed at these low levels of perchlorate exposure. Of note is that the vast majority of the participants in this group had urinary levels of perchlorate corresponding to estimated dose levels that are below the RfD of 0.0007 mg/kg/day. The clinical significance of the variations in T4/TSH levels, which were generally within normal limits, has not been determined. The researchers noted several limitations of the study (e.g., assumption that urinary perchlorate correlates with perchlorate levels in the stroma and tissue and preference for measurement of free T4 as opposed to total T4) and recommended that these findings be confirmed in at least one more large study focusing on women with low urine iodide levels. It is also not known whether the association between perchlorate and thyroid hormone levels is causal or mediated by some other correlate of both, although the relationship between urine perchlorate and total TSH and T4 levels persisted after statistical adjustments for some additional covariates known to predict thyroid hormone levels (e.g., total kilocalorie intake, estrogen use, and serum C-reactive protein levels). A planned follow-up study will include additional measures of thyroid health and function (e.g., TPO-antibodies, free T4). As EPA proceeds towards a regulatory determination for perchlorate, the Agency will continue to review any new findings/studies on perchlorate and their relationship to thyroid function as they become available.

C. Occurrence in Water, Food, and Humans

1. Sources of Perchlorate. Section V.A. summarizes the potential sources of perchlorate in the environment.

2. Studies on Perchlorate Occurrence in Public Drinking Water Systems and/or Drinking Water Sources. EPA included perchlorate as an analyte in the 1999 Unregulated Contaminant Monitoring Regulation (UCMR 1) and collected drinking water occurrence data for perchlorate from 3,858 public water systems (PWSs) between 2001 and 2005. EPA analyzed the available UCMR 1 data on perchlorate at concentrations greater than or equal to 4 [mu]g/L, the minimum reporting limit (MRL) for EPA Method 314.0.[21] The Agency found that approximately 4.1 percent (or 160) of 3,858 PWSs that sampled and reported under UCMR 1 had at least 1 analytical detection of perchlorate (in at least 1 entry/sampling point) at levels greater than or equal to 4 [mu]g/L. These 160 systems are located in 26 states and 2 territories. Of these 160 PWSs, 8 are small systems (serving 10,000 or fewer people) and 152 are large systems (serving more than 10,000 people). Approximately 1.9 percent (or 637) of the 34,193 samples collected (by these 3,858 PWSs) had positive detections of perchlorate at levels greater than or equal to 4 [mu]g/L. The maximum reported concentration of perchlorate was 420 [mu]g/L, which was found in a surface water sample from a PWS in Puerto Rico. The average concentration of perchlorate for those samples with positive detections for perchlorate was 9.85 [mu]g/L and the median concentration was 6.40 [mu]g/L.

These 160 PWSs (with at least 1 analytical detection for perchlorate at levels greater than or equal to 4 [mu]g/L) serve approximately 7.5 percent (or 16.8 million) of the 225 million people served by the 3,858 PWSs that sampled and reported results under UCMR 1. The 16.8 million population-served value represents the total number of people served by the 160 PWSs with at least one detect. Not all people served by these systems necessarily have perchlorate in their drinking water. Some of these 160 public water systems have multiple entry points to the distribution system and not all of the entry points sampled had positive detections for perchlorate in the UCMR 1 survey. An alternative approach to the system-level assessment of populations served is to use an assessment at the entry (sampling) point level.[22] EPA does not have population-served values for each entry point at the system level. However, an assessment can be performed by assuming that each entry (or sampling) point at a public water system serves an equal proportion of the total population-served by the system. In other words, for the alternative assessment, the population served by each system is assumed to be equally distributed across all entry (or sampling) points at each system. For example, if a system serves a million people and has 5 entry points, it is assumed that each entry point serves 200,000 people. Using this approach and counting only

[19] While CDC researchers measured urinary perchlorate concentration for 2,820 NHANES participants, TSH and total T4 serum levels were only available for 2,299 of these participants.

[20] WHO notes that the prevalence of goiter begins to increase in populations with a median iodide intake level below 100 [mu]g/L (WHO, 1994).

[21] EPA Method 314.0 was the analytical method approved and used for UCMR 1 at the time of data collection.

[22] EPA acknowledges that uncertainties exist in the population-served estimates for this alternative assessment since the population for a system is assumed to be equally distributed across the entry points for that system. Because the actual population-served by an entry point is not known, this alternative approach has an equal chance of underestimating or overestimating the actual population-served by entry points with positive detections for perchlorate. In addition, this approach could underestimate the population served that is potentially exposed to perchlorate and overestimate the level of exposure because it can not incorporate the effects of mixing of water between different entry points within the distribution system. This is because the approach cannot account for the dilution that may occur when water that has no detections of perchlorate is mixed within the distribution system with water that has positive detections for perchlorate.

the population served for the entry points with positive detections (concentrations greater than or equal to 4 [mu]g/L), the total population served by these entry points with perchlorate detections is approximately 5 million. Section V.E provides the number of systems and population-served estimates for other thresholds of interest.

The California Department of Health Services (CA DHS) began monitoring for perchlorate in 1997. In 1999, CA DHS began requiring monitoring for perchlorate for drinking water sources that were identified as vulnerable to perchlorate contamination under California's own State monitoring program (i.e., Unregulated Chemicals for which Monitoring is Required). About 60 percent (or 7,100) of all drinking water sources in California (about 12,000) were monitored for perchlorate under the State monitoring program. Between June 2001 and June 2006, CA DHS (2006) reports that 284 (about 4%) of the approximately 7,100 water sources that monitored had at least 2 or more positive detections for perchlorate at concentrations greater than or equal to 4 [mu]g/L (the reporting limit). These 284 sources supply water for 77 drinking water systems (CA DHS, 2006) and represent active and standby sources (and exclude inactive, destroyed, and abandoned sources, and monitoring and agricultural wells) (CA DHS, 2006).

In 2005, the State of Massachusetts's Department of Environment Protection (MA DEP) reported monitoring results for 85 percent (379 of 450) of its community water systems and 86 percent (212 of 250) of its non-transient, non-community water systems. MA DEP found that 9 (1.5%) of the 591 public water systems detected perchlorate at levels greater than or equal to 1 [mu]g/L (the reporting limit used for a modified version of EPA Method 314.0). MA DEP found that the occurrence of perchlorate for these water systems could be traced to the use of blasting agents, military munitions, fireworks, and, to a lesser degree, sodium hypochlorite disinfectant (MA DEP, 2005).

3. Studies on Perchlorate Occurrence in Foods, Plants, Beverages, and Dietary Supplements. The Food and Drug Administration (FDA), the United States Department of Agriculture (USDA), and researchers from academia and industry have studied perchlorate in foods. Some of these studies are described briefly in this section, and also summarized in Table 4. EPA has concluded that the sampling results described in this section and Table 4 are too limited to characterize food-borne exposure to perchlorate on a national scale. The sampling data are limited in the types of foods sampled, sample sizes, geographic coverage, and/or analytical method adequacy and many were targeted to foods or areas known or likely to have elevated levels of perchlorate. Section V.F of this action describes the limitations of the food sampling data and also describes plans for including perchlorate as part of the FDA's Total Diet Study. EPA requests that commenters provide the Agency with any additional data that may further characterize the concentrations of perchlorate in foods commercially available in the U.S. When providing data to the Agency, please describe the specific locations where the samples were collected, including geographic location, type of location (e.g., grocery store, farmer's market, commercial field, home garden), and the methodologies used to select, collect, prepare, and analyze the samples. Please include available laboratory data reports as well as all relevant quality assurance/quality control information.

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Table 4. Summary Data on Perchlorate Occurrence in Food Items

Food Item	Data Reference	Units	N	MRL	Range of Detections	Reported Mean[s]	Rate of Detection (percent)	Sample Locations
Iceberg Lettuce	FDA (2004)[a]	µg/kg FW	38	1	<MRL – 71.6	7.76	79%[b]	AZ, CA, FL, NJ
	Sanchez *et al.* (2005a)[c]	µg/kg FW	44	~20	<MRL - 26	NA	86%	AZ, CA
	Sanchez *et al.* (2005a)[f]	µg/kg FW	24	25-30	ND - 24	10	NA	AZ, CA
	Sanchez *et al.* (2005b)[f]	µg/kg FW	63	20-40	ND - 31	7.4	NA	See note[m]
Romaine Lettuce	FDA (2004)[a]	µg/kg FW	40	1	<MRL - 129	11.9	95%[b]	AZ, CA, FL, NJ, TX
	Sanchez (2004)[e]	µg/kg FW	7	20 - 50	<MRL - 81	NA	100%	AZ, CA
	Sanchez *et al.* (2005a)[d]	µg/kg FW	24	25-30	ND - 20	13	NA	AZ, CA
	Sanchez *et al.* (2005b)[e]	µg/kg FW	84	20-40	ND - 100	17.1	NA	See note[m]
Green Leaf Lettuce	FDA (2004)[a]	µg/kg FW	25	1	1.00– 27.4	10.7	100%	AZ, CA, NJ, TX
	Sanchez (2004)[e]	µg/kg FW	3	20 - 50	46-64	NA	100%	AZ, CA
	Sanchez *et al.* (2005a)[e]	µg/kg FW	24	25-30	ND - 102	33	NA	AZ, CA
	Sanchez *et al.* (2005b)[e]	µg/kg FW	69	20-40	ND - 195	16.5	NA	See note[m]
Red Leaf Lettuce	FDA (2004)[a]	µg/kg FW	25	1	<MRL – 52.0	11.6	92%[b]	AZ, CA, TX
	Sanchez *et al.* (2005a)[e]	µg/kg FW	24	25-30	ND - 81	27	NA	AZ, CA
	Sanchez *et al.* (2005b)[e]	µg/kg FW	67	20-40	ND - 104	14.5	NA	See note[m]
Butterhead Lettuce	Sanchez *et al.* (2005a)[e]	µg/kg FW	24	25-30	ND - 104	29	NA	AZ, CA
	Sanchez *et al.* (2005b)[e]	µg/kg FW	45	20-40	ND - 98	17.2	NA	See note[m]
Arugula	Sanchez *et al.* (2005b)[e]	µg/kg FW	9	20-40	ND - 195	55.8	NA	See note[m]
Spinach	Sanchez *et al.* (2005b)[e]	µg/kg FW	10	20-40	ND - 628	85.1	NA	See note[m]
Bottled Water	FDA (2004)	µg/L	51	0.5	<MRL – 0.56	NA	4%[b]	CA, CO, GA, MD, MN, MO, NC, NE, PA, SC, TX, WI
Dairy Milk	FDA (2004)	µg/L	104	3	<MRL – 11.3	5.76	97%[b]	AZ, CA, GA, KS, LA, MD, MO, NJ, NC, PA, SC, TX, VA, WA
	Kirk *et al.* (2005)	µg/L	47	~1[g]	ND – 11.0	2.0	98%	AK, AZ, CA, FL, HI, KS, ME, NH, NM, NY, PA
	Kirk *et al.* (2003)	µg/L	7	0.5[g]	1.7 – 6.4	NA	100%	TX
Melon	Sanchez (2004)[h]	µg/kg FW	25	20 - 50	ND - <MRL	NA	48%	AZ, CA
	Jackson *et al.* (2005)[i]	µg/kg FW	1	NA	1600	NA	100%	KS

Table 4. Summary Data on Perchlorate Occurrence in Food Items

Food Item	Data Reference	Units	N	MRL	Range of Detections	Reported Mean[s]	Rate of Detection (percent)	Sample Locations
Cucumber	Jackson *et al.* (2005)[n]	µg/kg FW	2	NA	40 - 770	NA	100%	TX, KS
Tomato	Sanchez (2004)	µg/kg FW	8	20 - 50	ND - <MRL	NA	37%	AZ, CA
	Jackson *et al.* (2005)	µg/kg FW	2	NA	42 - 220	NA	100%	KS
Pepper	Sanchez (2004)	µg/kg FW	10	20 - 50	ND - <MRL	NA	30%	AZ, CA
Carrot	Sanchez (2004)	µg/kg FW	10	20 - 50	ND	NA	0%	CA
Onion	Sanchez (2004)	µg/kg FW	10	20 - 50	ND	NA	0%	CA
Sweet Corn	Sanchez (2004)	µg/kg FW	18	20 - 50	ND	NA	0%	AZ, CA
Squash	Sanchez (2004)	µg/kg FW	10	20 - 50	ND	NA	0%	AZ, CA
Wheat	Sanchez (2004)[j]	µg/kg FW	NA	20 - 50	ND	NA	0%	AZ
	Jackson *et al.* (2005)[k]	µg/kg FW	12	NA	710 – 4400[l]	NA	100%	TX
Alfalfa	Sanchez (2004)[o]	µg/kg FW	10	20 - 50	109 - 668	NA	100%	AZ, CA
	Jackson *et al.* (2005)[p]	µg/kg FW	3	NA	NA	2900	100%	TX
Soy Milk	Kirk *et al.* (2005)	µg/L	1	~1[g]	0.7	NA	100%	TX
Lemon	Sanchez *et al.* (2006)	µg/kg FW	33	~2.5	ND – 14.8	2.3	NA	AZ, CA
Grapefruit	Sanchez *et al.* (2006)	µg/kg FW	15	~2.5	ND – 16.2	3.3	NA	AZ, CA
Orange	Sanchez *et al.* (2006)	µg/kg FW	28	~2.5	ND – 37.6	7.4	NA	AZ, CA
Seaweed	Martinelango *et al.* (2006a)[q]	µg/kg DW	13	NA	29 - 878	NA	100%	Atlantic Ocean (ME)
Beer	Aribi *et al.* (2006)	µg/L	144	NA	0.005 – 21.096	NA	100%	47 countries (including USA)
	Aribi *et al.* (2006)	µg/L	8	NA	0.364 – 2.014	0.662[r]	100%	USA
Wine	Aribi *et al.* (2006)	µg/L	77	NA	0.029 – 50.25	NA	100%	22 countries (including USA)
	Aribi *et al.* (2006)	µg/L	12	NA	0.197 – 4.593	2.09[r]	100%	USA

Notes:
N = number of samples; MRL = minimum reporting limit; ND = not detected; FW = fresh weight; DW = dry weight; NA = not available from (or not appropriate for) the cited study.
[a] Outermost leaves of each lettuce head were removed prior to sample analysis.
[b] Rate of detection is based on number of samples for which perchlorate was quantifiable (not just detectable).
[c] Samples are of "edible head" (trimmed of frame and wrapper leaves).
[d] Samples are "bulk" (partial removal of stem core and partial severing of upper and outer leaf blade margins).
[e] Samples preparation included minimal trimming.
[f] Samples have had multiple layers of their outer wrapper leaves removed
[g] Value reported as the "limit of detection."
[h] Samples include cantaloupe, casaba, honey dew, galia, and watermelon.
[i] Sample of cantaloupe from a home garden in Morris County, KS.
[j] Durum wheat.
[k] Whole wheat head, including seed (endosperm), bran, germ, and chaff.
[l] Represents the range of average values (3 samples, each) of 4 commercial growing fields in Gaines County, TX. In partitioned samples, perchlorate in the whole grain (not including the chaff) measured 1300 µg/kg FW in 1 sample and was not detected in 2 samples of wheat endosperm.
[m] Study was restricted to foods outside the lower Colorado River region. Sample locations were not presented for each food item, however, the complete list of regions sampled is CA, CO, MI, NJ, NM, NY, OH, and Quebec.
[n] Samples were collected from home gardens in Gaines County, TX, and Morris County, KS.
[o] Six of the 10 alfalfa samples were sent to FDA for confirmatory analysis by IC-MS/MS. The FDA results ranged from 121 to 382 µg/kg FW.
[p] Samples were collected from a single commercial growing field in Gaines County, TX.
[q] Samples of 11 different commercially available species were collected.
[r] Value provided is the median (not the mean).
[s] When comparing means from the studies it is important to note that the different studies likely treated non-detects differently. Some studies treated non detects as one-half the MRL and others treated non-detects as zero.

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a. *FDA Targeted Sampling.* The FDA released data on perchlorate in milk, lettuce, and bottled water in November 2004. To analyze food samples, FDA used ion chromatography (IC)-tandem mass spectrometry (MS/MS), referred to as IC–MS/MS. The quantitation limits for perchlorate in these analyses were 0.5 [mu]g/L for bottled water, 1 [mu]g/kg by fresh weight (FW) for lettuce, and 3 [mu]g/L for dairy milk. The mean concentration of perchlorate in 128 lettuce samples collected in 5 states (AZ, CA, FL, NJ, TX) was 10.3 [mu]g/kg FW (FDA, 2004), and ranged from not quantifiable (NQ) to 129 [mu]g/kg FW. The mean concentrations of perchlorate in several varieties of lettuce are reported in Table 4. The mean concentration of perchlorate in 104 dairy milk samples collected in 14 states (AZ, CA, GA, KS, LA, MD, MO, NJ, NC, PA, SC, TX, VA, WA) was 5.76 [mu]g/L (FDA, 2004), with a range from NQ to 11.3 [mu]g/L. FDA (2004) detected perchlorate in 2 of the 51 bottled water samples representing 34 distinct sources collected in 12 states (CA, CO, GA, MD, MN, MO, NC, NE, PA, SC, TX, WI) at levels of 0.56 [mu]g/L and 0.45 [mu]g/L.

b. *Other Published Studies.* Sanchez (2004) and Sanchez *et al.* (2005a) report the results of an analysis of agricultural products sampled from the lower Colorado River region of Arizona and California, the Imperial Valley of California, and the Coachella Valley of California, where irrigation water is known or suspected to contain perchlorate. The studies were partially supported by the U.S. Department of

Agriculture—Agricultural Research Service (USDA–ARS). Samples of iceberg, romaine, and leaf lettuce, carrots, onions, sweet corn, squash, melons, tomatoes, peppers, broccoli, cauliflower, cabbage, durum wheat, and alfalfa were analyzed for perchlorate using ion chromatography (IC) as the primary analytical method. For these analyses, the fresh-weight method reporting limit was not identified in most cases, but was reported to range from 20 to 50 [mu]g/kg FW, depending on the moisture content of the samples (Sanchez, 2004). Sanchez *et al.* (2005a) report that the method reporting level for iceberg lettuce was approximately 20 [mu]g/kg FW and for other types of lettuce was 25–30 [mu]g/kg FW. Perchlorate in the irrigation water ranged from 1.5 to 8.0 [mu]g/L over the period of the survey (Sanchez *et al.*, 2005a).

Sanchez *et al.* (2005a) analyzed 44 samples of iceberg lettuce heads that had been trimmed of frame and wrapper leaves, which are usually removed before the lettuce is consumed. Perchlorate was quantified in 5 of the samples (ranging from 23 to 26 [mu]g/kg FW),[23] perchlorate was not detectable in 6 samples, and the results of the remaining samples were less than the method reporting limit, which the authors defined as ''a detectable peak among duplicates and/or replicates but below a level that can be quantitated.'' Perchlorate concentrations in 10 samples of romaine and green leaf lettuce ranged from less than the method reporting limit to 81[mu]g/kg FW (Sanchez, 2004).

As shown in Table 4, Sanchez (2004) also detected perchlorate in samples of melons, tomatoes, and peppers, but at levels below the method reporting limit. Perchlorate was not detected in carrots, onions, sweet corn, squash, and durum wheat. Concentrations of perchlorate in 10 samples of alfalfa ranged from 109 to 668 [mu]g/kg FW. Six of the 10 alfalfa samples were sent to FDA for confirmatory analysis by IC–MS/MS. The FDA results were generally lower than those of the corresponding samples by Sanchez (2004), ranging from 121 to 382 [mu]g/kg FW.

Sanchez *et al.* (2006) conducted studies to evaluate the uptake and distribution of perchlorate in citrus trees and the occurrence of perchlorate in lemons, grapefruit, and oranges grown in southern California and southwestern Arizona. Five whole lemon trees irrigated with Colorado River water were harvested for destructive sampling. Sanchez *et al.* (2006) estimate that the irrigation water had an average perchlorate concentration of 6 [mu]g/L. Most of the sample analysis was conducted using IC–MS/MS, having an MRL of approximately 25 [mu]g/kg by dry weight (DW). In samples of tree trunks, roots, and branches, perchlorate was close to or below the MRL. Perchlorate was much higher in the leaves than the fruit (peel and pulp), with mean concentrations of 1,835 and 128 [mu]g/kg DW, respectively.

Citrus samples were collected during 2004–2005 from the lower Colorado River Valley, the University of Arizona Research Farm, the Coachella Valley, and Los Angeles County. All analyses of fruit pulp were conducted using IC–MS/MS with an approximate MRL of 2.5 [mu]g/kg FW. For the 86 citrus samples collected, the perchlorate concentration in the fruit pulp ranged from below detection to 37.6 [mu]g/kg FW. Mean concentrations in lemons (33 samples), grapefruit (15 samples), and oranges (28 samples) were 2.3, 3.3, and 7.4 [mu]g/kg FW, respectively.

Sanchez *et al.* (2005b) surveyed perchlorate occurrence in lettuce and other leafy vegetables produced outside the lower Colorado River region. Samples were analyzed by IC, with a minimum reporting level of approximately 20 to 40 [mu]g/kg FW, depending on the leafy vegetable type. Results of some of the more heavily sampled food items are presented in Table 4.

While not shown in Table 4, Sanchez *et al.* (2005b) performed additional analysis by partitioning the leafy vegetable samples by type of culture. Perchlorate was detected in 70 of 268 samples of conventionally-grown leafy vegetables and 72 of 170 samples of organically-grown leafy vegetables. The range of perchlorate concentrations was ND to 104 [mu]g/kg FW in conventional leafy vegetables and ND to 628 [mu]g/kg FW in organic leafy vegetables. Sanchez *et al.* (2005b) analyzed the results using regression analysis and estimated that the median perchlorate concentration in organically-grown samples was 2.2 times higher than in conventionally-grown samples. The regression analysis also suggested that variation among sampling locations was greater than variation among lettuce types.

Researchers at Texas Tech University analyzed samples of dairy and soy milk using IC and/or IC/MS analytical methods with detection limits of 1 [mu]g/L or better (Kirk *et al.*, 2005). In a study of perchlorate in dairy milk, Kirk *et al.* (2005) found mean perchlorate levels of 2.0 [mu]g/L in 47 retail dairy milk samples from 11 states (AK, AZ, CA, FL, HI, KS, ME, NH, NM, NY, PA), with a range from not detected (ND) to 11.0 [mu]g/L. A single sample of soy milk was analyzed and reported to contain 0.7 [mu]g/L perchlorate (Kirk *et al.*, 2005). An earlier study by Kirk *et al.* (2003) found perchlorate ranging from 1.7 [mu]g/L to 6.4 [mu]g/L in 7 dairy milk samples purchased in a city in Texas.

Jackson *et al.* (2005) conducted limited sampling of edible and forage vegetation in 1 Texas county and in 1 Kansas home garden. In Texas, wheat and alfalfa were sampled from commercial fields irrigated with groundwater containing perchlorate from an unknown source, and a cucumber was sampled from an irrigated home garden. In Kansas, cantaloupe, cucumber, and tomatoes were sampled from an irrigated home garden near a slurry explosives site. Researchers used IC for sample analysis but did not report fresh-weight detection limits. Perchlorate was detected in all 12 samples of winter wheat heads (whole, including the chaff) at a mean concentration of 2,000 [mu]g/kg FW but perchlorate was not detected in wheat endosperm (2 samples)[24]. The mean perchlorate concentration in 3 samples of alfalfa was 2,900 [mu]g/kg FW. A cucumber sample from a Texas home garden contained 40 [mu]g/kg FW perchlorate; a sample of irrigation water from this garden contained 20.7 [mu]g/L perchlorate. In the Kansas home garden, the cucumber sample contained 770 [mu]g/kg FW perchlorate, the cantaloupe sample contained 1,600 [mu]g/kg FW perchlorate, and 2 samples of tomato contained 42 and 220 [mu]g/kg FW perchlorate. The reported concentration of perchlorate in irrigation water for the Kansas home garden was 81 [mu]g/L. EPA notes that the perchlorate levels in irrigation water samples associated with these two home gardens were significantly higher than in the vast majority of surface and ground water samples in the US.

Aribi *et al.* (2006) developed an analytical method for perchlorate that uses ion chromatography with suppressed conductivity and electrospray ionization tandem mass

[23] Sanchez (2004) presents somewhat different results. Specifically, of the 44 samples of ''edible head'' lettuce, perchlorate was quantified in one of the samples (26 [mu]g/kg), perchlorate was not detectable in 6 samples, and the remaining sampling results were qualified as <MRL, which the author defined as ''represents a seemingly detectable peak but below a level that can be quantitated.''

[24] A wheat kernel (seed) has three major parts—the bran, the germ, and the endosperm. The majority of the wheat kernel is the endosperm, which is the portion of the kernel that is retained in refined (white) wheat flours. Whole wheat flours contain endosperm, wheat bran, and wheat germ in approximately the same proportions as in the wheat kernel. Wheat flours do not contain the chaff (husk).

spectrometry (IC–ESI–MS/MS). The method was used to measure perchlorate in samples of various food products, including fresh/canned fruits and vegetables, wine, beer, and other beverages. Most samples were purchased in grocery and liquor stores in greater Toronto, Canada, between January 2005 and February 2006. Produce samples originated from many different parts of the world and all samples contained measurable amounts of perchlorate. However, the survey was limited to only a few samples of each food. Products from California, Chile, Costa Rica, Guatemala, and Mexico had the highest levels of perchlorate. Products from Canada and China had the lowest levels of perchlorate. The highest detection was in cantaloupe from Guatemala (463.50 [mu]g/kg FW). Analysis of raw asparagus (39.900 [mu]g/kg FW) and cooked asparagus (24.345 [mu]g/kg FW) demonstrated that perchlorate can remain in food processed at a high temperature. Perchlorate concentrations in 8 samples of produce from the U.S. ranged from 0.094 [mu]g/kg FW (for blueberries) to 19.29 [mu]g/kg FW (for green grapes).

Aribi et al. (2006) analyzed 77 samples of wine and 144 samples of beer from many parts of the world. All samples contained measurable amounts of perchlorate. The wine sample with the single highest concentration of perchlorate, 50.250 [mu]g/L, was from Portugal. Overall, wine samples from Chile contained the highest concentrations of perchlorate, ranging from 5.358 to 38.88 [mu]g/L in 8 samples. Twelve samples of wine from the U.S. contained perchlorate concentrations ranging from 0.197 to 4.593 [mu]g/L. Results from analysis of beer samples varied substantially among countries, with an overall range from 0.005 [mu]g/L (Ireland) to 21.096 [mu]g/L (France). Concentrations of perchlorate in 8 beer samples from the U.S. ranged from 0.364 to 2.014 [mu]g/L.

Snyder *et al.* (2006) measured perchlorate in dietary supplements and flavor enhancing ingredients collected from various vendors in Las Vegas, NV, and Seattle, WA. Analyses were performed using LC–MS/MS with a limit of detection between 2 and 5 [mu]g/kg. Perchlorate was detected in 20 of 31 analyzed supplements, with detectable concentrations ranging from 10 to 2,420 [mu]g/kg. Based on manufacturers' recommended intake of the supplements, the resulting daily oral doses of perchlorate would range from 0.03 to 18 [mu]g/day. Twelve of the supplements tested were prenatal or children's vitamins. The highest level of perchlorate (2,420 [mu]g/kg or 0.018 mg/day at the recommended daily dose) was found in a prenatal vitamin; in the remaining prenatal and children's vitamins perchlorate did not exceed 28 [mu]g/kg. The study noted that ''vitamin and mineral supplements are typically formulated to include the Recommended Daily Allowance (RDA) of iodine, a factor that would provide protection against any possible impacts of microgram levels of perchlorate found in these supplements.'' Perchlorate was also detected at 740 [mu]g/kg in a sample of kelp granules (a flavor enhancer), which equates to 2.2 [mu]g perchlorate per serving.

Martinelango *et al.* (2006a) measured perchlorate in seaweed, which is often used as a source of iodide in food and nutritional supplements. Martinelango *et al.* (2006a) collected samples of 11 different species of seaweed growing off the coast of northeastern Maine. Perchlorate was detected in all species, with concentrations ranging from 29 to 878 [mu]g/kg DW. The iodide content in the samples was much higher, ranging from 16 to 3,134 mg/kg DW. Martinelango *et al.* (2006a) found that samples of *Laminaria* species concentrated iodide more selectively than perchlorate. Laminaria is a genus of large brown seaweeds that are commonly used in kelp tablets. Martinelango *et al.* (2006a) also analyzed 4 seaweed samples that had been washed with deionized water and found that a single wash removed 38 to 73 percent of the perchlorate and 34 to 44 percent of the iodide.

D. Occurrence Studies on Perchlorate in Human Urine, Breast Milk, and Amniotic Fluid

Recently researchers have used the results of the analysis of urine samples to estimate human exposure to perchlorate. Ingested perchlorate is not metabolized by humans and is excreted largely in the urine (Merrill *et al.*, 2005). The CDC's National Center for Environmental Health (NCEH) developed a sensitive and selective analytical method to analyze perchlorate in human urine (Valentin-Blasini *et al.*, 2005). The method uses ion chromatography coupled with electrospray ionization tandem mass spectrometry (IC/MS/MS) and achieves an MRL of 0.025 [mu]g/L in human urine. The authors report that the method is robust enough to process first-morning-void urine samples, which are samples of the first voiding of urine upon waking.

Valentin-Blasini *et al.* (2005) analyzed urine samples from 61 healthy adult donors who lived in the area of Atlanta, Georgia. The urine samples were provided anonymously, without associated donor information. Perchlorate was detected in all of the urine samples, with concentrations ranging from 0.66 to 21 [mu]g/L. The authors cited dietary exposure as a potential source of perchlorate because perchlorate was found only at low levels (0.1—0.2 [mu]g/L) in area tap water samples (Valentin-Blasini *et al.*, 2005).

Valentin-Blasini *et al.* (2005) also analyzed the urine samples for creatinine, which is a metabolic breakdown product in muscles that is eliminated from the body in urine at a predictable rate. When adjusted for urinary creatinine content, the reported range of perchlorate in the samples is 1.0 to 35 [mu]g of perchlorate per gram of creatinine. The median perchlorate concentration was 3.2 [mu]g/L (7.8 [mu]g/g creatinine). The researchers stated that only 1 sample from the Atlanta population contained perchlorate at a level slightly in excess of the amount expected to be excreted by an individual exposed to perchlorate at the reference dose of 0.0007 mg/kg/day (Valentin-Blasini *et al.*, 2005). Specifically, assuming that perchlorate is excreted uniformly in urine throughout the day, a urinary excretion level of 34 [mu]g perchlorate per gram creatinine would be associated with a daily perchlorate intake of 0.0007 mg/kg/day, for a 70 kg male that excretes creatinine at a typical rate of 1.44 grams per day (g/day). These assumptions are imprecise for individual exposure assessment but allow for spot urine perchlorate excretion to be related to the reference dose for toxicological perspective. Estimating perchlorate exposure from a single spot urine sample (as opposed to a sample collected continuously over a period of time) is imprecise due to the episodic nature of perchlorate exposure and the short half-life of perchlorate in the human body. The precision of estimated individual perchlorate exposure can be improved by more precise estimation of 24-hour creatinine excretion based on sex, height, weight, and age as described by Mage *et al.* (2004). In addition, imprecision stemming from the episodic nature of perchlorate exposure can be reduced with increased sampling.

The analytical method developed by Valentin-Blasini *et al.* (2005) was further used by Blount *et al.* (2006a) to evaluate urine samples from 27 volunteers with differing dietary habits. Blount *et al.* (2006a) collected first-morning-void urine specimens from volunteers living in the Atlanta area. The study volunteers self-assessed their consumption of milk, dairy products, and green/leafy vegetables within the 16 hours before the sample was collected.

The samples were grouped into 2 categories (''one or fewer servings'' and ''three or more servings'') based on total consumption of these selected foods. Total daily perchlorate exposure was calculated using a bodyweight of 70 kg and a creatinine excretion rate of 1.44 g/day, assuming that each first-morning void urine sample was representative of that individual's daily perchlorate exposure. Each volunteer also collected a drinking water sample from home and work. Blount *et al.* (2006a) analyzed drinking water samples with the same method used for urine analysis and estimated exposure from drinking water based on a body weight of 70 kg and daily consumption of 2 liters of water per day. The mean creatinine-adjusted urinary perchlorate level was 1.8 times higher for individuals who identified themselves as consuming three or more servings of milk, dairy products, and/or green/leafy vegetables (6.13 versus 3.45 [mu]g/g creatinine). There were no significant differences in the perchlorate levels in the drinking water samples of the 2 diet groups, which ranged from <0.05 to 0.25 [mu]g/L with a median of 0.10 [mu]g/L. Using a median drinking water level of 0.10 [mu]g/L, Blount *et al.* (2006a) estimated that the perchlorate dose from drinking water was 0.003 [mu]g/kg/day. Compared to this drinking water estimate, the total perchlorate dose estimate based on mean urinary perchlorate excretion was 24 times higher (0.071 [mu]g/kg/day) and 42 times higher (0.126 [mu]g/kg/day) for the low-consumption and high-consumption diet groups, respectively. The overall range of perchlorate found in urine was 0.94 to 17 [mu]g/g creatinine with a median of 4.2 [mu]g/g creatinine.

In the largest study of its kind, Blount *et al.* (2006c) measured perchlorate in urine samples collected from a nationally representative sample of 2,820 U.S. residents, ages 6 years and older, as part of the 2001–2002 NHANES. Blount *et al.* (2006c) detected perchlorate at concentrations greater than 0.05 [mu]g/L in all 2,820 urine samples tested, with a median concentration of 3.6 [mu]g/L (3.38 [mu]g/g creatinine) and a 95th percentile of 14 [mu]g/L (12.7 [mu]g/g creatinine). Only 0.7% of the study participants had an estimated perchlorate dose in excess of 0.0007 mg/kg/day. Women of reproductive age (15–44 years) had a median urinary perchlorate concentration of 2.9 [mu]g/L (2.97 [mu]g/g creatinine) and a 95th percentile of 13 [mu]g/L (12.1 [mu]g/g creatinine). The demographic with the highest concentration of urinary perchlorate was children (6–11 years), who had a median urinary perchlorate concentration of 5.2 [mu]g/L (5.79 [mu]g/g creatinine). Blount *et al.* (2006c) estimated a total daily perchlorate dose for each adult and found a median dose of 0.066 [mu]g/kg/day (about one tenth of the RfD) and a 95th percentile of 0.234 [mu]g/kg/day (about one third of the RfD). Eleven adults (0.7%) had estimated perchlorate exposure in excess of the RfD (0.7 [mu]g/kg/day). The highest estimated exposure was 3.78 [mu]g/kg/day. Because of daily variability in diet and perchlorate exposure, and the short residence time of perchlorate in the body, these single sample measurements may overestimate long-term average exposure for individuals at the upper end of the distribution and may underestimate the long-term average exposure for individuals at the lower end of the distribution. Daily perchlorate dose is not presented for children and adolescents due to the limited validation of formulas for these age groups (Blount *et al.*, 2006c).

Valentin-Blasini *et al.* (2005) and T[eacute]llez *et al.* (2005) analyzed urine samples of pregnant women in 3 cities in Chile and found higher median levels of urinary perchlorate in cities with higher concentrations of perchlorate in tap water. Based on an assessment of drinking water intake, the researchers determined that, in all 3 cities, there was an additional source of perchlorate for the study participants that may be explained by dietary (food) intake (T[eacute]llez *et al.*, 2005). This gap between estimated perchlorate exposure and perchlorate intake from tap water consumption ranged from 21.7 [mu]g/day to 33.8 [mu]g/day in the 3 Chilean cities (T[eacute]llez *et al.*, 2005).

Martinelango *et al.* (2006b) developed a method to measure perchlorate in human urine with a limit of detection of 0.080 [mu]g/L, and reported analytical results of 9 spot urine samples from male and female volunteers. Perchlorate was present in all samples analyzed, at concentrations ranging from 2.2 to 14.9 [mu]g/L, with a median value of 8.1 [mu]g/L.

Other studies have investigated perchlorate in human breast milk. Kirk *et al.* (2005) analyzed 36 breast milk samples from 18 states (CA, CT, FL, GA, HI, MD, ME, MI, MO, NC, NE, NJ, NM, NY, TX, VA, WA, WV) and found perchlorate concentrations in all samples ranging from 1.4 to 92.2 [mu]g/L in all samples, with a mean concentration of 10.5 [mu]g/L. T[eacute]llez *et al.* (2005) report maternal parameters for participants from the study in Chile. Breast milk samples indicated that a significant amount of perchlorate leaves the body of the nursing mother through breast milk, in addition to urine. However, the breast milk perchlorate levels were highly variable and no significant correlations could be established between breast milk perchlorate and either urine perchlorate or breast milk iodide concentrations for the individuals evaluated in these Chilean cities (T[eacute]llez *et al.*, 2005). Kirk *et al.* (2006) evaluated variations of iodide, thiocyanate and perchlorate in human milk samples. These authors suggest that if the overall intake of iodide is sufficient, it is unlikely that milk with an occasional low iodide or high perchlorate content would pose a major risk to infants. However, their limited data (evaluating only 10 women) show that the milk of some women may not supply infants with adequate iodide and they suggest that it may be important to base risk assessments for perchlorate exposure on the iodide to perchlorate ratio or the ratio of iodide to a ''selectively-weighted sum of iodide uptake inhibiting agents.''

Blount and Valentin-Blasini (2006) developed a sensitive and selective method for quantifying iodide, perchlorate, thiocyanate, and nitrate in human amniotic fluid. The analytical limit of detection for perchlorate was calculated to be 0.020 [mu]g/L. Samples of amniotic fluid at 15 to 20 weeks gestation were collected from 48 healthy women in an Eastern U.S. city for analysis. Perchlorate was found in all samples tested and exhibited a log-normal distribution. The perchlorate concentrations ranged from 0.057 to 0.71 [mu]g/L with a median value of 0.18 [mu]g/L.

E. Status of the Preliminary Regulatory Determination for Perchlorate

As stated earlier, the Agency is not making a preliminary regulatory determination for perchlorate in this notice. The Agency believes that additional information is needed on the sources of human exposure if it decides to base its determination regarding health risk reduction potential on a health reference level (HRL) derived from the RfD and the relative source contribution (RSC) for drinking water. Under this approach, the Agency would use the RfD and RSC to estimate an HRL and then use this HRL as a benchmark against which to conduct an evaluation of the occurrence data. In conducting such an assessment for the 6 non-carcinogens discussed previously in this action, EPA used a 20 percent RSC, which is the lowest and most conservative RSC used to estimate an HRL. Since the initial screening of the occurrence data against the HRL resulted in a preliminary negative determination, the Agency found that it

was not necessary to further evaluate the RSC for these contaminants. In the case of perchlorate, the Agency is not at the point of being able to make either a negative or a positive determination using this approach because it is not yet clear what an appropriate RSC for perchlorate is. If EPA were to use a default RSC of 20% for perchlorate, the resulting HRL would be 5 [mu]g/L. Approximately 3.16% of the 3,858 PWSs in the UCMR1 data set had at least one detect of perchlorate greater than or equal to 5 [mu]g/L. Given this level of occurrence at the default-derived HRL, the Agency believes a better informed RSC and HRL would be needed to use this approach to determine whether regulation of perchlorate in drinking water presents a meaningful opportunity for health risk reduction.

Table 5 shows the number of systems and population served that would exceed the HRL under various RSC scenarios and the sensitivity of this estimate to relatively small changes in the estimated RSC. For example, increasing the RSC from 20 to 30 percent would lower the estimated number of systems impacted by about a third and the estimated population served by about half. Hence, the choice of an appropriate RSC and resulting HRL could impact EPA's determination of whether regulation of perchlorate represents a meaningful opportunity for health risk reduction if it uses this approach.

EPA recognizes that system-level population estimates shown in Table 5 may be conservative because some systems have multiple entry points to the distribution system and not all entry points had a positive detection for perchlorate in the UCMR 1 survey. Hence, to derive a less conservative population estimate (last column in Table 5), EPA assumed that the population for each system is equally distributed over all of the entry (or sampling) points and estimated a population-served value based on entry points that had at least 1 analytical detection for perchlorate at levels greater than each of the HRL thresholds.

TABLE 5.—UCMR 1 OCCURRENCE AND POPULATION ESTIMATES FOR PERCHLORATE AT VARIOUS HRL THRESHOLDS [a]

RSC scenarios (percent)	Estimated HRL thresholds based on various RSC scenarios [b]	PWSs with at least 1 detection ≤ threshold of interest	PWS entry or sample points with at least 1 detection ≤ threshold of interest [c]	Population served by PWSs with at least 1 detection ≤ threshold of interest [d]	Population estimate for entry or sample points having at least 1 detection ≤ threshold of interest [e]
20	5 [mu]g/L	3.16% (122 of 3,858)	1.88% (281 of 14,984)	14.6 M	4.0 M
30	7 [mu]g/L	2.13% (82 of 3,858)	1.14% (171 of 14,984)	7.2 M	2.2 M
40	10 [mu]g/L	1.35% (52 of 3,858)	0.65% (97 of 14,984)	5.0 M	1.5 M
50	12 [mu]g/L	1.09% (42 of 3,858)	0.42% (63 of 14,984)	3.6 M	1.2 M
60	15 [mu]g/L	0.80% (31 of 3,858)	0.29% (44 of 14,984)	2.0 M	0.9 M
70	17 [mu]g/L	0.70% (27 of 3,858)	0.24% (36 of 14,984)	1.9 M	0.8 M
80	20 [mu]g/L	0.49% (19 of 3,858)	0.16% (24 of 14,984)	1.5 M	0.7 M
100	25 [mu]g/L	0.36% (14 of 3,858)	0.12% (18 of 14,984)	1.0 M	0.4 M

Footnotes:

[a] These data represent summary statistics for the 3,858 public water systems that have sampled for perchlorate as a part of the UCMR 1 survey.

[b] HRL threshold = [(RfD of 0.0007 mg/kg/day x 70 kg BW for pregnant female) / (2 L DWI)] x the RSC scenario. Each HRL threshold value is converted from mg/L to [mu]g/L units and then rounded to the nearest whole number.

[c] The entry/sample-point-level population served estimate is based on the system entry/sample points that had at least 1analytical detection for perchlorate greater than the HRL threshold of interest. The UCMR 1 small system survey was designed to be representative of the nation's small systems, not necessarily to be representative of small system entry points.

[d] The system-level population served estimate is based on the systems that had at least 1analytical detection for perchlorate greater than the HRL threshold of interest.

[e] Because the population served by each entry/sample point is not known, EPA assumed that the total population served by a particular system is equally distributed across all entry/sample points. To derive the entry/sample point-level population estimate, EPA summed the population values for the entry/sample points that had at least 1 analytical detection greater than the threshold of interest.

Table 5 also includes information on the effects of using an RSC of 100% (that is, using an HRL set at the DWEL of 24.5 [mu]g/L, rounded to a whole number). Crawford-Brown *et al.* (2006), in an estimate of risk variability from perchlorate exposure through community water systems, noted that the subjects in the original 2002 Greer *et al.*, study (on which the RfD of .0007 mg/L was based) presumably had other sources of perchlorate exposure outside of the study and suggested that it may be appropriate to view their results as reflecting the effects of incremental exposure to perchlorate above the background levels already in food and water rather than the effects of total exposure, as is implicitly assumed when the HRL is derived using an RSC to account for other sources of exposure. Use of an RSC to derive the HRL is clearly appropriate when the RfD or cancer slope factor is derived from animal studies with carefully controlled exposure. Crawford-Brown *et al.* suggest, however, that an RSC is not necessary for perchlorate because there is no reason to assume that the background exposure of the study subjects was different than that of the general population. EPA notes that the sample size in the Greer study was small and EPA is not aware of data on their background exposure to perchlorate or how representative it may be. EPA requests comment on whether information is available on the background exposure of subjects in the Greer study and whether it should consider the background exposure of these subjects in determining an HRL for perchlorate.

While several States have recommended guidelines or public health goals for perchlorate, EPA recognizes that at least 1 state, Massachusetts,[25] has already promulgated a final drinking water standard for perchlorate, that other States may set drinking water standards in the future, and that these standards

[25] Massachusetts promulgated a final drinking water standard of 2 [mu]g/L for perchlorate on July 28, 2006. For more information about the final standard, see *http://www.mass.gov/dep/public/press/pchl0706.htm* (MA DEP, 2006).

could impact national occurrence estimates once these standards are fully implemented.

F. What Are the Potential Options for Characterizing Perchlorate Exposure and Proceeding With the Preliminary Regulatory Determination for Perchlorate?

While the Agency recognizes that food and other pathways may be important sources of perchlorate exposure, the Agency believes the currently available food data (summarized in section V.C.3) are inadequate to develop a better informed RSC (and HRL). First, some of the existing data are limited in their sample numbers, geographic coverage, and analytical method adequacy. Second, the current studies provide little or no data for several food groups (*e.g.*, meat, poultry, fish, eggs, root and tuber vegetables, brassica vegetables, bulb vegetables, tree fruits, legumes, and cereal grains) that account for about half of the diet (by mass) for females of reproductive age (mid-teens to mid-forties).

This section presents and requests comment on data EPA might use to estimate an RSC based on food-borne exposure as well as on several other options that the Agency is considering to better characterize perchlorate exposure and assist the Agency in making its regulatory determination for perchlorate. These options could serve as a supplement or an alternative to developing an HRL based on a better informed RSC derived from food concentration and consumption data. The Agency specifically seeks comment on the use of urine biomonitoring data in estimating perchlorate exposure. If the Agency decides to use any of the approaches discussed in V.F.2, EPA will need to determine what statistics (*e.g.*, mean, median, percentile, etc.) are most appropriate for consideration in a regulatory determination. The Agency will also conduct a peer review, as appropriate, of any new methodology it decides to use.

The Agency also invites the public to submit relevant data that may further characterize exposure to perchlorate through consumption of foods and/or through other pathways. The Agency will consider any new, relevant information/data provided in response to this action as the Agency determines whether to regulate perchlorate with a national primary drinking water regulation.

1. Use of Food Concentration and Consumption Data to Estimate an RSC. In the past, the Agency has relied on dietary exposure information from the FDA Total Diet Study (TDS) to determine the RSC allowed for drinking water and to set health goals (*i.e.*, Maximum Contaminant Level Goals) for several inorganic compounds (*e.g.*, antimony, cadmium, chromium, and selenium). Under the TDS, foods are sampled at retail outlets, prepared as they would be consumed, and analyzed for a variety of analytes (*e.g.*, nutrients, pesticides, industrial chemicals). Approximately 280 foods, covering a broad spectrum of the diet, are currently sampled in each sampling event. Sampling events (known as ''market baskets'') occur about 4 times per year, with each event being confined to 1 of the 4 regions of the country. The dietary intake of the analyzed compounds can be calculated for the U.S. population by multiplying the concentrations found in TDS foods by the consumption amounts for each food. FDA compiles food consumption amounts for the total U.S. population by gender and by age group.[26]

FDA is including perchlorate as an analyte in the 2006 TDS. EPA believes that a comprehensive dietary intake estimate for perchlorate will be useful in evaluating dietary exposure relative to drinking water. When sufficient quantitative exposure data are available (such as the data published by FDA in conjunction with the TDS), EPA can use the procedure used previously for several regulated inorganic compounds (*i.e.*, chromium and selenium) to calculate the relative source contribution for perchlorate. In these cases where dietary intake values were available, EPA subtracted the dietary intake value from the Drinking Water Equivalent Level DWEL and used the remainder as the allowance for water. This procedure assures that total exposure does not exceed the RfD.

The Agency invites the public to submit relevant data that may further characterize exposure to perchlorate through consumption of foods and/or through other pathways. This information may help the Agency in the evaluation of currently available food data and the 2006 TDS.

2. Use of Urinary Biomonitoring Data to Evaluate Exposure to Perchlorate. Researchers at CDC's National Center for Environmental Health (NCEH) have conducted a large national study of total perchlorate exposure through analysis of urine samples collected for NHANES 2001–2002 (Blount *et al.*, 2006b and 2006c). The use of urinary perchlorate excretion to estimate perchlorate exposure has been demonstrated in Valentin-Blasini *et al.* (2005), Tollez *et al.* (2005), and Blount *et al.* (2006c). While this would be the first time the Agency has used biomonitoring data to assist EPA in making a preliminary regulatory determination for a CCL contaminant, the Agency believes that estimating perchlorate exposure among large populations using urinary perchlorate excretion data may be appropriate for the following reasons:

<bullet≤ Perchlorate is not metabolized in the body and is excreted unchanged primarily via the renal pathway (Merrill *et al.*, 2005),

<bullet≤ Perchlorate does not bioaccumulate, that is, it is excreted essentially completely (Merrill *et al.*, 2005),

<bullet≤ Perchlorate has a short half-life in the human body (approximately 8 hours), simplifying the estimation of daily exposure (Greer *et al.*, 2002), and

<bullet≤ A methodology exists that allows estimation of daily perchlorate intake from all sources (*e.g.*, water, food) using standard creatinine adjustment factors to account for variations in urine concentration (Mage *et al.*, 2004).

The Agency could use the 2001–2002 NHANES urine data in several ways as described in the following paragraphs. The Agency welcomes comment from the public on these approaches, as well as suggestions for other analyses that may inform the preliminary regulatory determination for perchlorate.

One potential approach is to use the 2001–2002 NHANES urine data to directly determine whether regulation of perchlorate in drinking water presents a meaningful opportunity for health risk reduction. More specifically, we could use the urine data (as in Blount *et al.*, 2006b and c) to evaluate whether total exposure from food and water is likely to result in an appreciable risk of adverse health effects for the U.S. population. If the Agency concluded that total exposure, as estimated from the urine data, does not pose an appreciable risk, even at the upper end of the exposure distribution, then it would follow logically that reducing this exposure by regulating drinking water would not present a meaningful opportunity for health risk reduction. As summarized above, Blount *et al.* (2006c) estimated a median total daily perchlorate dose for adults of 0.066 [mu]g/kg/day (about one tenth of the RfD) and a 95th percentile dose of 0.234 [mu]g/kg/day (about one third of the RfD). Only eleven adults (0.7%) had an estimated dose in excess of the RfD (0.7 [mu]g/kg/day). EPA requests comment on whether or not these data provide an adequate basis to support a regulatory

[26] Information about FDA's TDS design, food list, analytes, and analytical results can be found at *http://www.cfsan.fda.gov/comm/tds-toc.html.* (FDA, 2006)

determination for perchlorate. EPA also requests comment on the relevance, if any, to a regulatory determination for perchlorate, of the Blount et al (2006b) study, which showed an association between T4/TSH levels in women and urinary perchlorate concentrations at levels below the RfD (see Section V.B).

EPA could also use the 2001–2002 NHANES urine data to qualitatively evaluate the importance of the water contribution to overall exposure. For this approach, the Agency could merge data from the 2001–2002 NHANES and UCMR 1 and compare the total perchlorate exposure values (based on the urine data) for the population of individuals whose drinking water contains perchlorate at various concentration levels, ranging from non-detect to the upper end of the occurrence distribution. The intent of this analysis would be to permit the Agency to determine whether total perchlorate exposure (as measured in urine) is meaningfully correlated with concentrations in local public drinking water supplies, though EPA would only use these results qualitatively because it is not possible to match up individual urine samples with individual drinking water exposures. However, the results could be useful in determining at least qualitatively the potential significance of drinking water exposure for total exposure. If there were not a significant correlation between public water system perchlorate occurrence and individual exposure as measured through biomonitoring, this might suggest that there is not a meaningful opportunity for health risk reduction through regulation of drinking water.

The Agency could also potentially use the 2001–2002 NHANES urine data to derive an RSC to use for drinking water. This could potentially be done in several different ways as follows.

a. *Use of Urinary Biomonitoring Total Exposure Value to Estimate an RSC.* One possible approach to estimating an RSC for water would be to use the urine data to estimate total perchlorate exposure, then subtract this exposure value from the reference dose and allow the remainder as the exposure limit for water. The allowed remainder divided by the RfD would be the RSC for drinking water. This approach would yield a conservative RSC value because the exposure used to represent food would actually correspond to both food and drinking water exposure, whereas, if it were possible to estimate the exposure from food alone, the relative amount allowed for water would be larger (resulting in a higher RSC and higher health reference value). As discussed in Section V.D, Blount *et al.* (2006c) estimated a total daily perchlorate dose for adults from urine data and found a median dose of 0.066 [mu]g/kg/day (about one tenth of the RfD) and a 95th percentile of 0.234 [mu]g/kg/day (about one third of the RfD). If EPA were to use the estimated 95th percentile total dose from the Blount study as if it represented the exposure from food alone, this would suggest a residual screening-level RSC of about 70% allocated to water. One possible limitation of this approach is that the Blount study estimates exposure for adults only. Therefore, an RSC developed based upon this data would not necessarily be representative of children. EPA requests comment on using this approach as the basis for deriving a screening-level RSC.

b. *Use of the Urine Data and UCMR 1 to Deduce Exposure from Other Sources and Derive the RSC.* Alternately, for those NHANES survey subjects served by public drinking water systems with positive detections for perchlorate (based on UCMR 1), EPA could estimate the expected perchlorate dose contributed by drinking water (using individual water consumption data from the NHANES survey combined with UCMR 1 data for the area in which they live) and subtract it from the total perchlorate dose (based on urinary perchlorate excretion data) to calculate the amount contributed by food. Subtraction of this calculated food contribution from the RfD would yield the amount allowed for drinking water, which could be divided by the RfD to calculate an RSC. One limitation of this methodology would be the assumption that subjects in the NHANES study are uniformly consuming drinking water that contains perchlorate at the concentration indicated in the UCMR 1 data for their area.

c. *Use of Urinary Biomonitoring Data from Exclusive Bottled Water Drinkers to Estimate an RSC.* The 2001–2002 NHANES data includes urinary perchlorate data for populations who exclusively drink bottled water. As noted in section V.C.3.a, FDA (2004) tested 51 samples of bottled water from 34 distinct sources in 12 states and detected perchlorate in 2 samples (at levels of 0.56 [mu]g/L and 0.45 [mu]g/L). These levels are well below the MRL for the UCMR 1 data and would not contribute significant amounts of perchlorate relative to the RfD. If the population of exclusive bottled water drinkers is sufficiently representative of the U.S. population, these data potentially could be used to estimate the contribution of perchlorate exposure coming from food and allow the Agency to estimate an RSC for drinking water. The RSC value could be derived by subtracting the estimated perchlorate exposure for exclusive bottled water drinkers from the RfD of 0.0007 mg/kg/day, using the remainder as the allowance for drinking water. One limitation of this methodology is that the perchlorate concentration of the bottled water used by this NHANES population is not known. Hence, we would have to assume that the bottled water concentration data collected by FDA (2004) is representative of the perchlorate concentration in the bottled water used by the NHANES exclusive bottled water population. Another limitation of this approach is that it would not subtract out the fraction of the drinking water intake that comes from water used for cooking purposes (since bottled water is probably not used by most subjects in cooking and household food preparation). It would thus produce a conservative (health protective) estimate of the RSC as it would overestimate the fraction of total exposure coming from food.

G. Next Steps

After the Agency evaluates and thoroughly reviews public comments and any new information/data on perchlorate obtained following this notice, and performs the necessary analyses, the Agency intends to move expeditiously to publish a preliminary regulatory determination for perchlorate. Depending on how quickly the Agency is able to complete the necessary analyses and determine the best approach for making this determination, EPA may be able to publish the preliminary determination in time to include a final determination for perchlorate as part of the final CCL 2 regulatory determination, which is due by July, 2008. If not, the Agency will publish its final determination for perchlorate as soon thereafter as possible. EPA does not intend to wait until the CCL 3 regulatory determination cycle to complete its determination for perchlorate.

VI. What About the Remaining CCL 2 Contaminants?

As previously stated, EPA is only making regulatory determinations on CCL 2 contaminants that have sufficient information to support a regulatory determination at this time. Section V discusses the status of EPA's review of perchlorate. For the 30 remaining chemicals and the 9 microbial pathogens, the Agency lacks adequate information in the areas of health effects or occurrence or both.

The Agency continues to conduct research and/or to collect information

on the remaining CCL 2 contaminants to fill identified data gaps. 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 conducting research, monitoring, developing guidance or health advisories, and/or making a determination prior to the end of the next cycle. In addition, the Agency is not precluded from regulating a contaminant at any time when it is necessary to address an urgent threat to public health, including any contaminant not listed on the CCL.

Because the focus of this action is to announce and solicit public comment on the Agency's preliminary determinations for 11 of the 51 CCL 2 contaminants, this action primarily provides information on these 11 contaminants. The Agency recognizes that the public may have a particular interest in metolachlor, methyl tertiary butyl ether (MTBE), and the microbial contaminants. Therefore, this action includes some additional information for these contaminants in the following sections and requests public comment on any further data, information and/or analyses that the Agency should be aware of.

A. Metolachlor

1. Background. Metolachlor is a broad spectrum herbicide used for general weed control in many agricultural food and feed crops (primarily corn, soybeans and sorghum), on lawns and turf, ornamental plants, trees, shrubs and vines, rights of way, fencerows and hedgerows, and in forestry. Metolachlor appears to be moderately persistent to persistent and depending on the type of soil, can be highly mobile. Degradation of metolachlor in the environment is dependent on microbially-mediated and abiotic processes. Metolachlor has at least 5 major degradates. Two of the more common degradates are metolachlor ethane sulfonic acid (ESA) and metolachlor oxanilic acid (OA).

2. Health. The Agency established an RfD for metolachlor of 0.1 mg/kg/day based on an NOAEL of 9.7 mg/kg/day and a UF of 100 (USEPA, 1995). The Agency derived the NOAEL from a one-year chronic feeding study in beagle dogs where the critical effect was decreased body weight gain. Metolachlor shows some evidence of causing developmental toxicity effects in rats but none in rabbits. The doses associated with the developmental effect in rats are greater than the NOAEL and therefore the NOAEL would be protective against developmental toxicity.

Metolachlor has been evaluated for carcinogenic activity in both rats and mice. No treatment-related cancer effects were observed in 2 studies using mice. In studies using rats, metolachlor caused a significant increase in liver nodules and carcinomas in high dose females. Negative results from mutagenicity studies suggest that tumors may result from a nonmutagenic mode of action. In 1991, a peer review committee recommended that metolachlor be classified as a possible human carcinogen based on increases in liver tumors in the female rat. However, a peer review conducted in July 1994 recommended that the evidence for cancer was suggestive and should not be quantified. This recommendation was supported by negative mutagenicity data and recent metabolism data indicating that the formation of the metabolite presumed to be the ultimate carcinogen is very low (USEPA, 1995).

3. Occurrence. EPA included metolachlor as an analyte in the UCM Round 2 survey. EPA evaluated the UCM Round 2 Cross Section data and found that metolachlor was detected at or above the reporting limit of 0.1 [mu]g/L in 0.83% of the 12,953 systems that sampled for metolachlor (USEPA, 2006a).

The USGS NAWQA program included metolachlor as an analyte in its 1992–2001 monitoring survey of ambient surface and ground waters across the United States. EPA evaluated the results of the provisional data, which are available on the Web at *http://ca.water.usgs.gov/pnsp/* (Martin *et al.*, 2003; Kolpin and Martin, 2003). While the USGS detected metolachlor in both surface and ground waters, 95 percent of the samples from the various land use settings were less than 1.38 [mu]g/L. The maximum surface water concentration is 77.6 [mu]g/L (agricultural setting) and the maximum estimated ground water concentration is 32.8 [mu]g/L (agricultural setting).

4. Consideration of the ESA and OA degradates. While EPA has health and occurrence information for metolachlor itself, the Agency believes it is prudent to also consider the occurrence and exposure of the ESA and OA degradates as well. At this time, there is no finished water occurrence and exposure information for these 2 degradates from a nationally representative sample of PWSs. However, a few small-scale studies indicate that the ESA and the OA degradates may be occurring at greater frequencies and at higher concentrations than the metolachlor parent (Phillips *et al.*, 1999a and 1999b; Rheineck and Postle, 2000). In order to gather more information about the occurrence of the ESA and OA degradates in finished water (along with the metolachlor parent), the Agency has added these degradates and their parent to the second unregulated contaminant monitoring regulation (UCMR 2; 70 FR 49093; USEPA, 2005g). While EPA awaits the results of the UCMR 2 survey, the Agency is planning to update the health advisory for metolachlor to include the ESA and OA degradates. The Agency requests comment from the public as to whether updating the health advisory to include these degradates will be useful for States and public water utilities.

In addition, the Agency requests answers to the following questions and any available data:

<bullet≤ Are States collecting data on the co-occurrence of metolachlor and its degradates in source waters on a state-wide basis? In drinking water on a state-wide basis?

<bullet≤ If available, are States willing to provide data on the co-occurrence of metolachlor and its ESA and OA degradates in community and public water systems? What analytical method and reporting limit were used to gather these data?

<bullet≤ Do States have any information on the number of PWSs impacted by metolachlor and/or its degradates?

<bullet≤ Have States seen an increase or decrease in the number of PWSs impacted by metolachlor and/or its degradates?

<bullet≤ How many systems have taken wells or sources offline due to impacts from metolachlor and/or its degradates?

B. Methyl tertiary-butyl ether

1. Background

Methyl tertiary-butyl ether (MTBE) is a volatile organic compound synthesized for use as a gasoline additive. First used as an octane enhancer to improve engine performance, MTBE is also used to reduce emissions that form carbon monoxide and ozone. Leaking underground storage tanks, gasoline distribution facilities, and even recreational boating can release MTBE into the environment.

In 1997, EPA issued a drinking water advisory of 20 to 40 [mu]g/L based on taste and odor (USEPA, 1997b). EPA is currently revising its health risk assessment for MTBE, and thus, will not be making a regulatory determination for MTBE as part of this action. The IRIS Chemical Assessment Tracking System *http://cfpub.epa.gov/iristrac/index.cfm* has the most up-to-date information on

the status of the MTBF health risk assessment and interested members of the public should check that Web site to find out the latest schedule.

The Agency collected data on MTBE occurrence as part of the UCMR 1 survey. In addition, EPA evaluated several sources of supplemental occurrence information described in the supporting documentation for this action entitled ''Regulatory Determinations Support Document for Selected Contaminants from the Second Drinking Water Contaminant Candidate List (CCL 2)'' (USEPA, 2006a). Section VI.B.2 provides a summary of some of the data and information on MTBE occurrence collected to date.

2. Occurrence Information

a. *UCMR 1.* EPA collected sampling results for MTBE from over 98.9 percent (3,068 of 3,100) of the large PWSs and over 99.5 percent (796 of 800) of the small systems required to sample under UCMR 1. Based on these data, 19 public water systems (0.49 percent of the 3,864 sampled) in 14 states (CA, CT, GA, IL, MA, MO, NH, NJ, NM, NY, PA, SD, TN, and WV) reported MTBE occurrence in drinking water. These 19 systems reported MTBE in 26 samples at the minimum reporting level of 5 [mu]g/L or above, representing approximately 0.33 percent (or 754 thousand of 226 million) of the population served by the public water systems that sampled for MTBE. (USEPA, 2006a)

Of the PWSs reporting detections at or above 5 [mu]g/L (the MRL), 15 were ground water systems and 4 were surface water systems. One small ground water system (49 [mu]g/L) and 3 large ground water PWSs (48 [mu]g/L, 36 [mu]g/L, and 33.2 [mu]g/L) reported MTBE at levels greater than 20 [mu]g/L (the lower end of the taste and odor threshold). One large surface water system (33 [mu]g/L) reported MTBE at levels greater than 20 [mu]g/L. The remaining 14 systems had detects between 5 [mu]g/L and 20 [mu]g/L (USEPA, 2006a).

b. *USGS studies/surveys/reviews.* In 2003, the USGS reported results of national source water sampling (previously introduced in section III.B.2.a.(2)). USGS sampling included a random study of a representative sample of untreated source waters (known as the ''Random Survey'') and a study of source waters from areas known or suspected of having MTBE (known as the ''Focused Survey''). In the Random Survey, USGS found that none of the source waters exceeded 20 [mu]g/L, and the three highest concentration sources ranged from 6 [mu]g/L to 19.5 [mu]g/L (Grady, 2003). Of the areas known or suspected of having MTBE in the Focused Survey, USGS found that 5 percent (e.g., ground waters for 7 of the 134 systems) had concentrations greater than 20 [mu]g/L (Delzer and Ivahnenko, 2003a).

USGS also reviewed the literature for national, regional, and State MTBE information (Delzer and Ivahnenko, 2003b), including 13 state-wide assessments. This information is summarized in Table 6. USGS noted that because study objectives varied, information varied in terms of reporting levels, sampling frequencies, and sources (e.g., ambient water, public and homeowner wells, treated drinking water).

Previously, USGS (Grady and Casey, 2001) studied MTBE occurrence in the drinking water of 12 States (New England and the Mid-Atlantic). The study found less than 1 percent of the CWSs had drinking water samples at or above 20 [mu]g/L, while 7.8 percent of the CWSs had MTBE at 1 [mu]g/L or higher.

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Table 6. Summary of MTBE State-wide Assessments (Delzer and Ivahnenko, 2003b)

State Survey Summary	Reporting Limit (RL)	Detection Frequency	Median Detected Concentration	Maximum Detected Concentration
Alabama: 2000 survey of 575 PWSs. Sampling at 1,053 sources (87 surface water sources, 27 springs, 939 wells)	0.5 - 2.0 µg/L	wells: 0.53% springs: 0% surface water sources: 0%	wells: NA springs: NA surface water sources: NA	wells: 8.4 µg/L springs: NA surface water sources: NA
California: partial survey of PWS source waters, covering 105 of 245 surface water sources (3,000 samples) and 2,988 of 13,919 PWS wells in 1996-1997; supplemented by information from DHS database (50,748 samples collected between 1989 and 2001)	NA DHS database: NA	surface water sources: 46.7% wells: 1.2% DHS database: 1.1%	surface water sources: NA wells: NA DHS database: 3.6 µg/L	surface water sources: >14 µg/L (26%) wells: NA DHS database: 610 µg/L
Connecticut: 1999 annual report on organics testing at PWSs (total number of PWSs not reported)	0.5 - 2.0 µg/L	NA (detected in 57 sources waters in 40 towns)	2.7 µg/L	110 µg/L
Florida: 8,739 samples collected from 1,692 public water supplies since early 1990s.	NA	4.9% of samples, 1.2% of PWSs (89% of the detects were from 2 PWSs)	1.4 µg/L	166 µg/L
Illinois: monitoring since 1994 at approximately 80% of the State's 1,200 CWSs, most of which (92%) utilize ground water	0.5 - 1.0 µg/L	2.7% of active systems, plus 3 systems that abandoned wells following MTBE contamination	NA	NA
Iowa: 530 samples collected from 235 PWS wells in "vulnerable bedrock regions" in 1999; plus sampling of water supplies in several cities since the 1990s	Bedrock project: 15 µg/L cities: NA	bedrock project: 8 sample detections < 15 µg/L cities: NA	bedrock project: < 15 µg/L cities: NA	bedrock project: < 15 µg/L cities: 63 µg/L in Alvord's water supply before well abandoned
Kansas: 27,935 samples from 1,122 PWS wells, collected 1996 - 2000	NA	1.6% of wells	NA	1,250 µg/L
Maine: survey of 793 of 830 public water supplies and 951 private household water supplies in 1998	0.1 µg/L	public supplies: 15.8% (6% had concentrations ~ 1-35 µg/L) private supplies: 15.8%; (6.6% had concentrations ~1-35 µg/L)	public supplies: NA private supplies: NA	public supplies: < 35 µg/L private supplies: > 35 µg/L (1.1% of supplies)
Maryland: 1,084 PWSs surveyed since 1995; data also collected on private wells contaminated by LUSTs	0.5 µg/L	PWSs: 7.8% private wells: NA	PWSs: NA private wells: NA	PWSs: >20 µg/L (11 systems) private wells: NA
Michigan: 31,557 samples from 18,046 CWS, NCWS, and private wells from 1987 through 1999	1.0 µg/L	2.9% of samples and 3.0% of wells	NA	>240 µg/L (29 samples)
Missouri: MO has monitored MTBE in 1,685 PWSs since 1994	5 µg/L	0.1% of monitored PWSs statewide (2 PWSs)	NA	NA
New Jersey: samples from about 400 CWSs from 1997 to 1998; plus a random sampling of 104 domestic wells	PWSs: 0.5 µg/L private wells: 0.1 µg/L	PWSs: 14.8% private wells: 35.6%	PWSs: NA private wells: 0.48 µg/L	PWSs: 8.4 µg/L private wells: 30.2 µg/L
Wisconsin: 2,271 wells (mostly private) sampled since 1990	12 µg/L	4.4% of wells (96 private wells and 3 public wells)	NA	1,700 µg/L (private well)

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c. *New England Interstate Water Pollution Control Commission (NEIWPCC).* In 2003, the NEIWPCC surveyed the States under a grant from EPA's Office of Underground Storage Tanks (UST). Twenty-six States estimated that they had public wells that were contaminated by MTBE at some level, and of those, 5 States (ME,

NH, NJ, DE, and MD) estimated having detectable levels of MTBE in at least 100 public water supply wells. Thirteen States did not know the answer, 8 States did not respond, and 3 States reported that no PWS wells were impacted. The survey established no reporting level to define ''contamination.'' Only 3 States documented the basis for their estimates (projected from several studies, raw and treated water analyses, and a survey of funded petroleum spill projects) (NEIWPCC, 2003).

d. *California Department of Health Services.* In 2000, California developed a drinking water standard of 13 [mu]g/L for MTBE (CA DHS, 2000). According to California's annual compliance reports, there were no violations of the 13 [mu]g/L standard by public water systems in 2002 and 2003, and 2 violations at 2 public water systems (serving almost 14,000 people) in 2004 (CA DHS, 2002; CA DHS, 2003; CA DHS, 2004).

e. *Other Sources of Data.* In April 2005, the Environmental Working Group (EWG, 2005) released a report, Like Oil and Water, on their Web page. In response to Freedom of Information Act requests, 29 State agencies submitted data to EWG. EPA informally evaluated the data posted by EWG to determine if this information might be useful in projecting state-wide occurrence. While EPA found the report interesting, the data as reported on the Web lacked some of the information needed to assess the representativeness and the quality of the data. For example, States submitted different time periods of monitoring data (*e.g.*, Alaska submitted 7 months of data for 1 system during the 2000 timeframe and Illinois submitted data that spanned 1990 to 2002). States did not report monitoring results for every system. Also, the data do not indicate if the samples came from source water or finished water, from ground water or surface water, the analytical method used for analysis nor the reporting level, the frequency of the sampling (*e.g.*, annual, quarterly), number of samples from each water system, number of non-detects, etc.

3. Request for Additional MTBE Occurrence Information

As discussed earlier, EPA is not making a regulatory determination for MTBE; however, EPA is presenting this information because of ongoing interest in MTBE. And as noted earlier, additional information is presented in the regulatory support document for this action (*i.e.*, USEPA, 2006a). While the Agency waits for the final health risk assessment, EPA will continue to collect and evaluate occurrence information. The Agency requests any data, information, or analyses that may be available on the following topics:

<bullet≤ Are there additional occurrence data for MTBE in community and non-community public water systems on a state-wide or more local basis? As noted in the previous section, the State data submitted to EWG lack some elements needed to assess the quality of the data, as required in EPA's guidance for information quality guidelines (USEPA, 2003c), and project state-wide occurrence.

<bullet≤ What analytical method and reporting limit were used to gather these data?

<bullet≤ Has there been an increase or decrease in the number of impacted PWSs? Over what time frame?

<bullet≤ For those PWSs whose water supplies have been impacted, has there been an increase or a decrease in the concentration of MTBE?

<bullet≤ How many systems have taken wells or sources offline, consolidated with other PWSs, or added customers due to impacts from MTBE?

<bullet≤ What treatments are being used in the field? What range of treatment effectiveness is being achieved?

<bullet≤ Is the listing of State bans for MTBE shown in Table 7 complete? Have state-wide bans decreased MTBE contamination in drinking water?

TABLE 7.—STATE ACTIONS BANNING MTBE (STATE-WIDE)

[Adapted from USEPA, 2004g and McCarthy and Tiemann, 2005]

State	Effective date	Extent of MTBE ban
Arizona	January 1, 2005	0.3% max volume in gasoline.
California	December 31, 2003	complete ban in gasoline.
Colorado	April 30, 2002	complete ban in gasoline.
Connecticut	January 1, 2004	complete ban in gasoline.
Illinois	July 24, 2004	0.5% max volume in gasoline.
Indiana	July 24, 2004	0.5% max volume in gasoline.
Iowa	July 1, 2000	0.5% max volume in gasoline.
Kansas	July 1, 2004	0.5% max volume in gasoline.
Kentucky	January 1, 2006	0.5% max volume in gasoline.
Maine	January 1, 2007	0.5% max volume in gasoline.
Michigan	June 1, 2003	complete ban in gasoline.
Minnesota	July 2, 2005	complete ban in gasoline. (following partial ban in 2000).
Missouri	July 1, 2005	0.5% max volume in gasoline.
Montana	January 1, 2006	no more than trace amounts in gasoline.
Nebraska	July 13, 2000	1% max volume in gasoline.
New Hampshire	January 1, 2007	0.5% max volume in gasoline.
New Jersey	January 1, 2009	0.5% max volume in gasoline.
New York	January 1, 2004	complete ban in gasoline.
North Carolina	January 1, 2008	0.5% max volume in gasoline.
Ohio	July 1, 2005	0.5% max volume in gasoline.
Rhode Island	June 1, 2007	0.5% max volume in gasoline.
South Dakota	July 1, 2001	0.5% max volume in gasoline.
Vermont	January 1, 2007	0.5% max volume in gasoline.
Washington	January 1, 2004	0.6% max volume in gasoline.
Wisconsin	August 1, 2004	0.5% max volume in gasoline.

C. Microbial Contaminants

1. Evaluation of Microbial Contaminants for Regulatory Determination. The 9 microbial contaminants listed on CCL 2 include:

<bullet≤ Four virus groups—Caliciviruses, Echoviruses, Coxsackieviruses, and Adenoviruses

<bullet≤ Four bacteria/bacterial groups-*Aeromonas hydrophila; Helicobacter pylori; Mycobacterium avium intercellulare (or MAC);* and Cyanobacteria (called blue-green algae[27]), fresh water algae, and the associated toxins

<bullet≤ One group of protozoa—Microsporidia (*Enterocytozoon bieneusi* and *Septata intestinalis,* now renamed *Encephalitozoon intestinalis*).

In addition to considering if the Agency had sufficient information to address the three statutory criteria listed in section II.B.1 (i.e., adverse health effects, known/likely occurrence, and meaningful opportunity for health risk reduction), the Agency also considered whether sufficient information was available to determine whether current treatment requirements adequately controlled for any of the 9 microbial contaminants. After consideration of these factors, the Agency determined that none of the 9 microbial contaminants have sufficient information at this time to address the three statutory criteria to make a regulatory determination. Table 8 identifies the specific areas for which information is insufficient.

TABLE 8.—INFORMATION GAPS FOR THE MICROBIAL CONTAMINANTS

Health effects	Treatment	Analytical methods	Occurrence
Microsporidia	*Aeromonas*	*Aeromonas*	*Aeromonas.*
Some Cyanotoxins	*MAC*	*MAC*	*MAC.*
	Adenoviruses	*Helicobacter*	*Helicobacter.*
	Caliciviruses	Microsporidia	Adenoviruses.
	Coxsackieviruses	Some Cyanotoxins	Caliciviruses.
	Echoviruses		Coxsackieviruses.
	Microsporidia		Echoviruses.
	Some Cyanotoxins		Microsporidia.
	Helicobacter		Some Cyanotoxins.

2. Research and Other Ongoing Activities. EPA has supported an active research program to fill the information gaps on the CCL 2 microorganisms. While several examples of the ongoing research activities are listed below, further information on these and other projects can be found on EPA's Drinking Water Research Information Network (DRINK). DRINK is a publicly-accessible, Web-based system that tracks over 1,000 ongoing research projects and can be accessed at: *http://www.epa.gov/safewater/drink/intro.html.*

a. *Virus.* For the CCL virus groups (or surrogates), the Agency has initiated treatment studies that simulate realistic conditions where viruses may be protected in aggregates. EPA also plans to conduct virus removal/inactivation studies in drinking water treatment plants and/or pilot plants. In order to assess the effectiveness of treatment and to perform monitoring studies, methods development for viruses is also in progress.

b. *Bacteria.* For Aeromonas spp., EPA recently completed a one-year UCMR 1 survey of 293 public water systems. The Agency is currently attempting to characterize and distinguish pathogenic from non-pathogenic strains, as well as develop methods to detect Aeromonas virulence factors. For *H. pylori,* the Agency is in the process of developing a culture method and method for its identification. For *MAC,* preliminary drinking water surveys have been conducted using a culture method followed by genetic detection. EPA is also conducting further research into methods development and the characterization of virulence factors for this organism.

EPA has funded projects to evaluate the effect of disinfectants on cyanotoxins, and on the removal of algal cells and cyanotoxins in a pilot scale treatment plant. EPA is developing analytical methods for potential use for future monitoring and has available analytical chemistry standards for the toxins of most concern in the United States—microcystin, cylindrospermopsin, and anatoxin-a. EPA has conducted several small-scale preliminary occurrence surveys for cyanotoxins using a screening method followed by confirmation by instrumental analysis. A number of health effects studies are also in progress on several high priority cyanotoxins. These include behavioral studies in mice, acute and subacute effects in neonatal mice, and biomarkers of human exposure. Risk assessments are being conducted at EPA on the cyanotoxins to determine reference doses where possible. The Agency has organized and participated in several workshops on cyanotoxins to assess the state-of-the-science.

As an interim measure to assist public water utilities, the Agency is planning to develop an information sheet that discusses pertinent information on cyanobacteria and some of its key toxins. The document will discuss the state of the knowledge on the prevention and treatment of cyanobacteria and its toxins, as well as the available information on the potential health effects of some of the toxins. EPA requests comment from the public as to whether such a document would be useful for public water utilities.

c. *Protozoa.* EPA has several ongoing projects to evaluate the susceptibility of microsporidia to chlorine and chloramine disinfectants. EPA has sponsored methods-related projects for microsporidia, which have included the use of fluorescent gene probes, real-time PCR, concentration methods, and immunomagnetic separation. Ongoing monitoring at EPA has revealed that microsporidia are present in ground water. EPA has funded work to determine exposure to microsporidia, and to determine strains (animal and human) of Enterocytozoon bieneusi found in water. EPA also held a workshop in 2003 on microsporidia to assess the state-of-the-science.

VII. EPA's Next Steps

EPA intends to respond to the public comments it receives on the 11

[27] Cyanobacteria are called blue-green algae even though they are technically bacteria.

preliminary determinations and subsequently issue its final regulatory determinations. Although the preliminary determinations for all 11 contaminants are not to regulate, if after consideration of public comments, the Agency determines that a national primary drinking water regulation is warranted for any of these 11 contaminants, the regulation would then need to be formally proposed within 24 months of the determination and promulgated 18 months following the proposal.[28]

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