AUTHENTICATED
U.S. GOVERNMENT
INFORMATION
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NATIONAL ARCHIVES AND RECORDS ADMINISTRATION
1985

FEDERAL REGISTER

Vol. 79 Monday,
No. 202 October 20, 2014

Part II

Environmental Protection Agency

40 CFR Part 141
Announcement of Preliminary Regulatory Determinations for Contaminants on the Third Drinking Water Contaminant Candidate List; Proposed Rule

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 141

[EPA–HQ–OW–2012–0155; FRL–9917–87–OW]

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

AGENCY: Environmental Protection Agency (EPA).

ACTION: Request for public comment.

SUMMARY: The Safe Drinking Water Act (SDWA), as amended in 1996, requires the Environmental Protection Agency (EPA) to make regulatory determinations every five years on at least five unregulated contaminants. A regulatory determination is a decision about whether or not to begin the process to propose and promulgate a national primary drinking water regulation (NPDWR) for an unregulated contaminant. These unregulated contaminants are chosen from the Contaminant Candidate List (CCL), which SDWA requires the agency to publish every five years. EPA published the third CCL (CCL 3) in the **Federal Register** on October 8, 2009. This notice presents the preliminary regulatory determinations and supporting rationale for 5 of the 116 contaminants listed on CCL 3. The agency is making preliminary determinations to regulate one contaminant (i.e., strontium) and to not regulate four contaminants (i.e., 1,3-dinitrobenzene, dimethoate, terbufos and terbufos sulfone). EPA seeks comment on these preliminary determinations.

DATES: Comments must be received on or before December 19, 2014, 60 days after publication in the **Federal Register**.

ADDRESSES: Submit your comments, identified by Docket ID No. EPA–HQ–OW–2012–0155, by one of the following methods:

- *www.regulations.gov:* Follow the online instructions for submitting comments.
- *Mail:* Water Docket, Environmental Protection Agency, Mailcode: [28221T], 1200 Pennsylvania Ave. NW., Washington, DC 20460.
- *Hand Delivery:* EPA Docket Center, [EPA/DC] EPA West, Room 3334, 1301 Constitution Ave. NW., Washington, 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–2012–0155. EPA's policy is that all comments received will be included in the public docket without change and may be made available online at *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 *www.regulations.gov.* The *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 email comment directly to EPA without going through *www.regulations.gov* your email 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 information about EPA's public docket visit the EPA Docket Center homepage at *http://www.epa.gov/epahome/dockets.htm.* For additional instructions on submitting comments, go to Section I.B of the **SUPPLEMENTARY INFORMATION** section of this document.

Docket: All documents in the docket are listed in the *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 *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 Water Docket is (202) 566–2426.

FOR FURTHER INFORMATION CONTACT: Zeno Bain, Standards and Risk Management Division, Office of Ground Water and Drinking Water, Office of Water (Mailcode 4607M), Environmental Protection Agency, 1200 Pennsylvania Ave. NW., Washington, DC 20460; telephone number: (202) 564–5970; email address: *bain.zeno@epa.gov.* For general information, contact the Safe Drinking Water Hotline, telephone number: (800) 426–4791. The Safe Drinking Water Hotline is open Monday through Friday, excluding legal holidays, from 10 a.m. to 4 p.m. Eastern time.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this action apply to me?

Neither these preliminary regulatory determinations nor the final regulatory determinations, when published, impose any requirements on anyone. Instead, this action notifies interested parties of EPA's preliminary regulatory determinations for five unregulated contaminants for comment.

B. Tips for Preparing Your Comments

When submitting comments, remember to:

- Identify the rulemaking by docket number and other identifying information (subject heading, **Federal Register** date and page number).
- Explain why you agree or disagree and suggest alternatives.
- Describe any assumptions and provide any technical information and/or data that you used.
- Provide specific examples to illustrate your concerns, and suggest alternatives.
- Explain your views as clearly as possible.
- Make sure to submit your comments by the comment period deadline identified.

ABBREVIATIONS USED IN THIS DOCUMENT

Abbreviation	Meaning
µg/L	Micrograms per liter.
ADAF	Age Dependent Adjustment Factor.
AM	Assessment Monitoring.
AMWA	Association of Metropolitan Water Agencies.
ATSDR	Agency For Toxic Substances And Disease Registry.
AWWA	American Water Works Association.
BATs	Best Available Technologies.

ABBREVIATIONS USED IN THIS DOCUMENT—Continued

Abbreviation	Meaning
BMD	Benchmark Dose.
BMDL	Benchmark Dose (95% Lower Confidence Bound).
BW	Body Weight.
CARC	Cancer Assessment Peer Review Committee.
CAS	Chemical Abstracts Service.
CASRN	Chemical Abstract Service Registry Number.
CBI	Confidential Business Information.
CCL	Contaminant Candidate List.
CCL 1	First Contaminant Candidate List.
CCL 2	Second Contaminant Candidate List.
CCL 3	Third Contaminant Candidate List.
CCR	Consumer Confidence Report.
CFR	Code of Federal Regulations.
ChE	Cholinesterase.
CMR	Chemical Monitoring Reform.
CSF	Cancer Slope Factor.
CUSIUR	Chemical Update System/Inventory Update Rule.
cVOC	Carcinogenic Volatile Organic Compounds.
CW	Concentration in Water.
CWS	Community Water System.
CWSS	Community Water System Survey.
DBP	Disinfection Byproduct.
DBP ICR	Disinfection Byproduct Information Collection Rule.
DDE	1,1-Dichloro-2,2-bis(p-chlorophenyl)ethylene.
DSMRT	Distribution System Maximum Residence Time.
DWI	Drinking Water Intake.
DWS	Drinking Water Strategy.
EFSA	European Food Safety Authority.
ELCD	Electrolytic Conductivity Detection.
EPA	Environmental Protection Agency.
EPCRA	Emergency Planning And Community Right-To-Know Act.
EPTC	S-Ethyl propylthiocarbamate.
EPTDS	Entry Point to the Distribution System.
ESA	Ethanesulfonic Acid.
EWG	Environmental Working Group.
F	Fraction of a 70 year lifetime applicable to the age period.
FFQ	Food Frequency Questionnaire.
FIFRA	Federal Insecticide, Fungicide, And Rodenticide Act.
FR	**Federal Register**.
GAC	Granular Activated Carbon.
GAO	Government Accountability Office.
GC	Gas Chromatography.
GW	Ground Water.
HA	Health Advisory.
HRL	Health Reference Level.
ICR	Information Collection Rule.
IOC	Inorganic Compound.
IREDs	Interim Eligibility Decisions.
IRIS	Integrated Risk Information System.
Kg	Kilogram.
LOAEL	Lowest Observed Adverse Effect Level.
MCLG	Maximum Contaminant Level Goal.
MDL	Method Detection Limit.
mg/L	Milligrams per liter.
mg/kg/day	Milligrams per kilogram per day.
MDBP	Microbial Disinfection Byproduct.
MOA	Mode of Action.
MRL	Minimum Reporting Limit.
MS	Mass Spectrometry.
MTBE	Methyl Tertiary Butyl Ether.
NAS	National Academy of Sciences.
NAWQA	National Water Quality Assessment.
NCFAP	National Center for Food and Agricultural Policy.
NCI	National Cancer Institute.
NCOD	National Drinking Water Contaminant Occurrence Database.
NDBA	N-Nitroso-di-n-butylamine.
NDEA	N-Nitrosodiethylamine.
NDMA	N-Nitrosodimethylamine.
NDPA	N-Nitroso-di-n-propylamine.
NDPhA	N-Nitrosodiphenylamine.
NDWAC	National Drinking Water Advisory Council.
NIRS	National Inorganics And Radionuclides Survey.
NMEA	N-Nitrosomethylethylamine.
NOAEL	No Observed Adverse Effect Level.
NPDES	National Pollutant Discharge Elimination System.
NPDWR	National Primary Drinking Water Regulation.
NPYR	N-Nitrosopyrrolidine.
NRC	National Research Council.
NREC	National Reconnaissance of Emerging Contaminants.
NTP	National Toxicology Program.
OA	Oxanilic Acid.
OPP	Office of Pesticides Program.
OW	Office of Water.
PCCL	Preliminary Contaminant Candidate List.
PCE	Tetrachloroethylene.
PDP	Pesticide Data Program.
PFOA	Perfluorooctanoic Acid.
PFOS	Perfluorooctanesulfonic Acid.
PHA	Provisional Health Advisory.
PID	Photoionization Detection.
PMP	Pesticide Monitoring Program.
PWS	Public Water System.
QA	Quality Assurance.
RD 1	Regulatory Determinations 1.
RD 2	Regulatory Determinations 2.
RD 3	Regulatory Determinations 3.
RED	Reregistration Eligibility Decision.
RfD	Reference Dose.
RL	Reporting Limit.
RSC	Relative Source Contribution.
SAP	Scientific Advisory Panel.
SDWA	Safe Drinking Water Act.
SEPW	U.S. Senate Committee on Environment and Public Works.
SS	Screening Survey.
SSCTs	Small System Compliance Technologies.
STORET	Storage And Retrieval (STORET) Data System.
SW	Surface Water.
SY	Six Year Review.
SY3	Six Year Review 3.
TCE	Trichloroethylene.
TPTH	Triphenyltin Hydroxide.
TRED	Tolerance Reassessment Progress And Risk Management Decision.
TRI	Toxic Release Inventory.
TT	Treatment Technique.
UCM	Unregulated Contaminant Monitoring.
UCMR 1	First Unregulated Contaminant Monitoring Regulation.
UCMR 2	Second Unregulated Contaminant Monitoring Regulation.
UCMR 3	Third Unregulated Contaminant Monitoring Regulation.
UF	Uncertainty Factor.
USDA	United States Department of Agriculture.
USGS	United States Geological Survey.
VOC	Volatile Organic Compound.
WHO	World Health Organization.

Table of Contents

II. Purpose and Background

This section briefly summarizes the purpose of this action, the statutory requirements, and previous activities related to the CCL and regulatory determinations.

A. What is the purpose of this action?

The purpose of this action is to present and request comment on EPA's preliminary regulatory determinations for five unregulated contaminants. The five contaminants include: Dimethoate, 1,3-dinitrobenzene, strontium, terbufos, and terbufos sulfone. The agency is making preliminary determinations to regulate one contaminant (strontium) and to not regulate the remaining four contaminants (dimethoate, 1,3-dinitrobenzene, terbufos, and terbufos sulfone). EPA seeks comment on these preliminary determinations. The agency is also presenting and requesting comment on the process used for this round of regulatory determinations (i.e., RD 3), the supporting information, and the rationale used to make these preliminary decisions.

B. Background on the CCL and Regulatory Determinations

1. Statutory Requirements for CCL and Regulatory Determinations. Section 1412(b)(1)(B)(i) of the 1996 Safe Drinking Water Act Amendments (SDWA) requires EPA to publish the CCL every five years. The CCL is a list of contaminants which are not subject to any proposed or promulgated national primary drinking water regulations (NPDWRs), are known or anticipated to occur in public water systems (PWSs), and may require regulation under SDWA. SDWA section 1412(b)(1)(B)(ii) directs EPA to determine whether to regulate at least five contaminants from the CCL every five years. For EPA to make a determination to regulate a contaminant, SDWA requires the Administrator to determine that:

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

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

(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 these three statutory criteria are met and makes a final determination to regulate a contaminant, the agency has 24 months to publish a proposed Maximum Contaminant Level Goal [1] (MCLG) and NPDWR.[2] 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) and Regulatory Determinations (RD 1). EPA published the final CCL 1, which contained 60 chemical and microbiological contaminants, in the **Federal Register** (FR) on March 2, 1998 (63 FR 10273; USEPA, 1998). The agency made and published the final regulatory determinations for 9 of the 60 CCL 1 contaminants in the FR on July 18, 2003. The agency determined that NPDWRs were not necessary for any of these nine contaminants: *Acanthamoeba,* aldrin, dieldrin, hexachlorobutadiene, manganese, metribuzin, naphthalene, sodium, and sulfate (68 FR 42898; USEPA, 2003a). The agency posted information about *Acanthamoeba* [4] on the EPA Web site and issued health advisories [5] for manganese, sodium, and sulfate.

3. The Second Contaminant Candidate List (CCL 2) and Regulatory Determinations (RD 2). The agency published the final CCL 2 in the FR on February 24, 2005, (70 FR 9071; USEPA, 2005a) and carried forward the 51 remaining chemical and microbial contaminants listed on CCL 1. The agency made and published the final regulatory determinations for 11 of the 51 CCL 2 contaminants in the FR on July 30, 2008. The agency determined that NPDWRs were not necessary for any of these 11 contaminants: boron, the dacthal mono- and di-acid degradates, 1,1-dichloro-2,2-bis(p-chlorophenyl)ethylene (DDE), 1,3-dichloropropene (Telone), 2,4-dinitrotoluene, 2,6-dinitrotoluene, s-ethyl propylthiocarbamate (EPTC), fonofos, terbacil, and 1,1,2,2-tetrachloroethane (73 FR 44251; USEPA, 2008a). The agency issued new or updated health advisories [6] for boron, dacthal degradates, 2,4-dinitrotoluene, 2,6-dinitrotoluene and 1,1,2,2-tetrachloroethane.

4. The Third Contaminant Candidate List (CCL 3) and Regulatory Determinations (RD 3). The agency published the final CCL 3, which listed 116 contaminants, in the FR on October 8, 2009 (74 FR 51850; USEPA, 2009a). In developing CCL 3, EPA improved and built upon the process that was used for CCL 1 and CCL 2. The new CCL 3 process was based on substantial expert input and recommendations from the National Academy of Science's (NAS) National Research Council (NRC) and the National Drinking Water Advisory Council (NDWAC) as well as input from the public. Based on these consultations and input, EPA developed a multi-step process to select candidates for the final

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[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 non-enforceable health goals.'' (40 CFR 141.2; 42 U.S.C. 300g–1)

[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. The MCL is the highest level of a contaminant that is allowed in drinking water and is set as close to the MCLG as feasible using the best available treatment technology and taking cost into consideration.

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

[4] Consumer information about *Acanthamoeba* for people who wear contact lenses can be found at *http://water.epa.gov/action/advisories/acanthamoeba/index.cfm*.

[5] The health advisories for CCL 1 can be found at *http://water.epa.gov/drink/standards/hascience.cfm*.

[6] The health advisories for CCL 2 can be found at *http://water.epa.gov/drink/standards/hascience.cfm*.

CCL 3, which included the following key steps:

(a) Identification of a broad universe of ~7,500 potential drinking water contaminants (the CCL 3 Universe);

(b) screening the CCL 3 Universe to a preliminary CCL (PCCL) of ~600 contaminants based on the potential to occur in PWSs and the potential for public health concern; and

(c) evaluation of the PCCL contaminants based on a more detailed review of the occurrence and health effects data to identify a final list of 116 CCL 3 contaminants.

The development of the CCL, regulatory determinations, and any subsequent rulemaking should be viewed as a progression where each process builds upon the previous process, including the collection of data and analyses conducted. The agency's improvements in developing CCL 3 provide an excellent foundation for RD 3 by enhancing EPA's ability to identify contaminants of concern for drinking water.

While this notice focuses on the preliminary regulatory determinations for 5 of the 116 CCL 3 contaminants, it is important to note that the agency made and published a final determination to regulate one CCL 3 contaminant, perchlorate, on February 11, 2011 (76 FR 7762; USEPA, 2011a). Additional information about CCL 3 and the perchlorate final determination can be found in the October 8, 2009 (74 FR 51850; USEPA, 2009a) and February 11, 2011 (76 FR 7762; USEPA, 2011a) **Federal Register** notices, respectively. Sections III and IV in this notice provide more detailed information about the approach and outcome used for RD 3 and the contaminant-specific regulatory determinations.

5. The Drinking Water Strategy. In March 2010, EPA announced the agency's new Drinking Water Strategy (DWS),[7] which is aimed at finding ways to strengthen the protection of public health from contaminants in drinking water. The new vision is intended to streamline decision-making, expand protection under existing laws, and promote cost-effective new technologies to meet the needs of rural, urban, and other water-stressed communities. The four principles underlying the DWS are:

(a) Address contaminants as groups rather than one at a time so that enhancement of drinking water protection can be achieved cost-effectively.

(b) Foster development of new drinking water technologies to address health risks posed by a broad array of contaminants.

(c) Use the authority of multiple statutes to help protect drinking water.

(d) Partner with States to develop shared access to all PWSs monitoring data.

The first principle (i.e., addressing contaminants as groups) has a direct bearing on RD 3 and how to designate the contaminants for analysis, determination and subsequent regulation; that is, should they be considered individually or as a group. Although the agency has previously regulated contaminants as groups (e.g., total trihalomethanes, total haloacetic acids, gross alpha radionuclides, gross beta and photon emitters, etc.), all of the determinations for RD 1 and RD 2 were made on individual contaminants. As part of the DWS, the agency identified several factors to evaluate which contaminants might effectively be regulated as a group and considered these factors in evaluating contaminant groups for RD 3. All the factors do not have to be met, but the more factors that are met, the more suitable it may be to regulate the contaminants as a group. These factors include whether the contaminants in the group:

(a) Have a similar health endpoint,

(b) can be measured by the same analytical methods,

(c) can be treated using the same technology or treatment technique approach and/or

(d) have been shown to occur individually (and possibly co-occur if data are available).

EPA conducted extensive national outreach to solicit input from stakeholders on the DWS and how best to address groups of contaminants. Stakeholders generally agreed that while public health protection is of paramount importance, the grouping factors previously listed were some of the other important factors to consider in evaluating which contaminants would work best in a group regulation. Several CCL 3 contaminants (as well as non-CCL 3 contaminants) belong to contaminant groups that underwent consideration for regulation during the RD 3 process.

In February 2011,[8] the agency decided to address carcinogenic volatile organic compounds (cVOCs) as a group in a separate and concurrent regulatory process (which the agency expects to release in late 2014). Some of the cVOCs being considered include unregulated cVOCs listed on CCL 3 (e.g., 1,2,3-trichloropropane). While the cVOC group is being evaluated in a separate regulatory process, the same factors used to group cVOCs (i.e., similar health endpoint, measured by the same analytical method, similar treatment technique approach, etc.) were used to evaluate groups of contaminants for RD 3 as well (e.g., nitrosamines, chloroacetanilides, etc.). Although EPA evaluated the nitrosamines and chloroacetanilides groups as part of the RD 3 process, in the end, EPA decided not to make any preliminary determinations for these groups under RD 3.

The SDWA requires EPA to review each existing NPDWR at least once every six years and revise them, if appropriate. The purpose of the review, called the Six Year Review (SY), is to identify those NPDWRs for which current health effects assessments, changes in technology, and/or other factors provide a health or technical basis to support a regulatory revision that will maintain or provide for greater protection of the health of persons. In contrast, the RD process is intended to address currently unregulated contaminants. The agency will review the existing Microbial Disinfection Byproduct (MDBP) regulations as part of the third Six Year Review (SY3). Because chlorate and nitrosamines are disinfection byproducts (DBPs) that can be introduced or formed in public water systems partly because of disinfection practices, the agency believes it is important to evaluate these unregulated DBPs in the context of the review of the existing DBP regulations. DBPs need to be evaluated collectively, because the potential exists that the chemical disinfection used to control a specific DBP could affect the concentrations of other DBPs. Therefore, the agency is not making a regulatory determination for chlorate and nitrosamines at this time. The agency expects to complete the review of these DBPs by the end of 2015.

6. Outreach for RD 3 (Stakeholder Meetings and Expert Review).

EPA sought external advice and expert input for RD 3 by convening two public stakeholder meetings and conducting an Expert Review panel. On March 3, 2011, EPA held an Environmental Justice (EJ) Stakeholder meeting in Washington, DC to solicit input on RD 3 and environmental justice issues. Approximately 90 stakeholders participated (either by phone or in person) including representatives of children's advocacy groups, environmental organizations, community action groups, the drinking water industry, and State drinking water and public health programs. Stakeholders did not identify any EJ

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[7] More information about the DWS can be found at *water.epa.gov/lawsregs/rulesregs/sdwa/dwstrategy/.*

[8] *http://water.epa.gov/lawsregs/rulesregs/sdwa/dwstrategy/.*

issues specific to RD 3. On June 16, 2011, EPA held another public Stakeholder Meeting in Washington, DC, to disseminate information on the progress of RD 3 and solicit input from stakeholders, the public, and other interested groups. Forty-six participants attended including representatives from States, environmental and public health organizations, drinking water systems, chemical manufacturers, local governments, and academia. EPA presented and discussed: (a) The approach used to narrow the contaminants listed on CCL 3 and identify potential candidates for RD 3 (with a focus on those occurring at levels of health concern in drinking water) and (b) the background, health, and occurrence information for a ''short list'' of 32[9] contaminants being evaluated as potential RD 3 candidates. Stakeholders asked questions and provided comments about the approach as well as the health and occurrence information presented on several contaminants. One stakeholder provided additional health information on the chloroacetanilides and submitted a letter requesting that EPA regulate these compounds with an NPDWR (USEPA, 2011b). A summary of the June 16, 2011, meeting is provided in the docket for this action (USEPA, 2011c).

In May 2011, the Government Accountability Office (GAO) released a report entitled, ''EPA Should Improve Implementation of Requirements on Whether to Regulate Additional Contaminants'' (GAO, 2011). Specifically for regulatory determinations, GAO recommended that the agency develop criteria to identify contaminants of greatest public health concern and be more transparent, clear, and consistent by developing policies/guidance to interpret the SDWA criteria and make determinations (i.e., include thresholds for positive findings, factors for determining adequacy of occurrence/health data to make determinations, an approach for evaluating health effects on sensitive subpopulations, a process for presenting key information in documents, etc). In response to questions regarding the GAO report at a July 2011 U.S. Senate Committee on Environment and Public Works (SEPW) hearing,[10] EPA committed to consulting with an independent panel of scientists on the RD 3 process to determine how SDWA criteria 1 and 2 are evaluated,[11] how the best available science is used to make decisions, how the contaminants of greatest public health risk are assessed, and how vulnerable populations (especially children) are considered. EPA also committed to making the process used for regulatory determinations publicly available and to review the process every five years as EPA conducts the regulatory determination cycle.

To implement the commitment, EPA convened a panel of experts in October 2011 to provide an independent review of the approach used for RD 3, which EPA described in a draft of the document entitled, ''Protocol for the Regulatory Determinations 3'' (USEPA, 2014a). The Expert Review panel included seven experts representing one or more of the following areas of expertise: health effects evaluation, drinking water occurrence/exposure information evaluation, State drinking water perspective, PWS perspective, and/or some familiarity with the RD 3 process (including the Contaminant Candidate List). The review involved a three-week paper review of the October 2011 Draft RD 3 Protocol document and an in-person meeting held in Washington DC, on October 26 and 27, 2011. Panel members were encouraged to provide comments as individuals based upon their expertise and background, not as representatives of any respective organizational affiliation. The information and input provided by the expert reviewers assisted the agency in revising and clarifying the approach used for the RD 3 process. A summary of the October 26–27, 2011, meeting and the expert reviewers' comments (USEPA, 2011d), as well as the protocol document (USEPA, 2014a), are provided in the docket for this action.

III. Approach and Overall Outcome for RD 3

This section describes (a) the approach EPA uses to identify and evaluate contaminants for the agency's third round of Regulatory Determinations (RD 3) along with the overall outcome of applying this approach, (b) the supporting RD 3 documentation, and (c) the technical analyses and sources of health and occurrence information.

A. Summary of the Approach and Overall Outcome for RD 3

The three phases of the RD 3 Process are (1) the Data Availability Phase, (2) the Data Evaluation Phase, and (3) the Regulatory Determination Assessment Phase. Figure 1 provides a brief overview of the process EPA uses to identify which CCL 3 contaminants are candidates for regulatory determinations and the SDWA statutory criteria considered in making the regulatory determinations. For more detailed information on the three phases of the RD 3 process please refer to the ''Protocol for the Regulatory Determinations 3'' (USEPA, 2014a).

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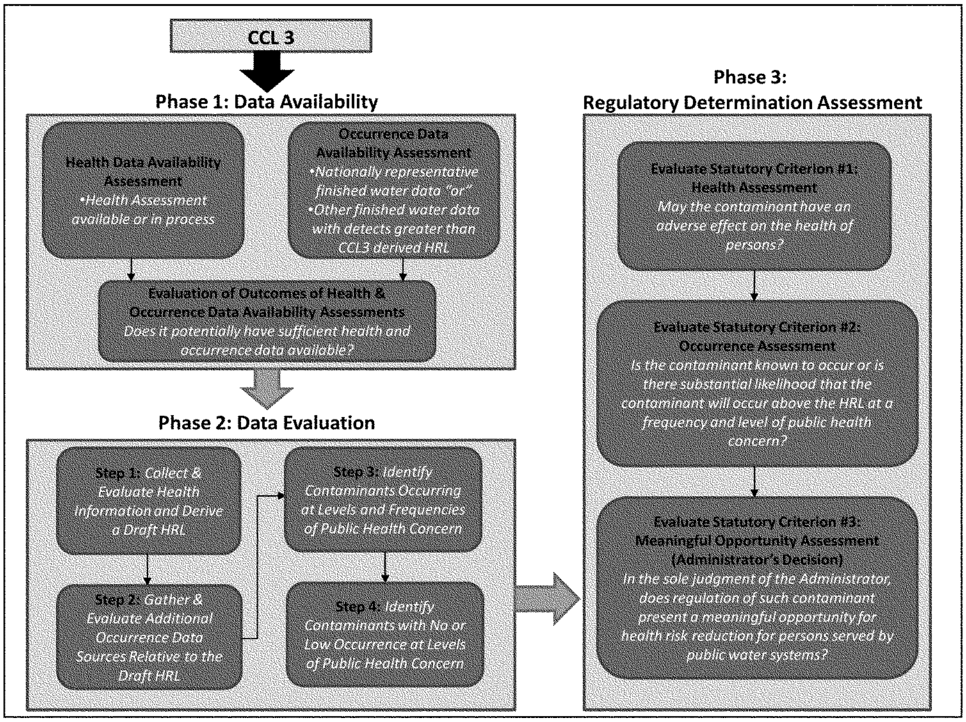
[9] Subsequent to the June 2011 stakeholder meeting and before the October 2011 Expert Review, EPA identified two additional contaminants for the shortlist, bringing the total to 34. In response to the Expert Review comments, an additional contaminant was added to the short list, bringing the final total to 35 CCL 3 contaminants.

[10] The U.S. Senate Committee on Environment and Public Works full committee hearing, entitled ''Oversight Hearing on the Environmental Protection Agency's Implementation of the Safe Drinking Water Act's Unregulated Drinking Water Contaminants Program'' can be found at (*http://www.epw.senate.gov/public/index.cfm?FuseAction=Hearings.Hearing&Hearing_ID=fc5a8756-802a-23ad-454a-b9eeb7bf1c36*).

[11] Under the statute, SDWA criterion 3 of Section 1412(b)(1)(A) is solely the Administrator's decision.

Figure 1: The Three Primary Phases of the RD 3 Process



1. Phase 1 (Data Availability Phase)

In Phase 1, the *Data Availability Phase,* the agency identifies contaminants that may have sufficient health and occurrence data to proceed to Phase 2 and be listed on a ‘‘short list’’ for further evaluation. With regard to sufficient health effects data used to identify potential adverse health effect(s), the agency considers whether a peer-reviewed health risk assessment is available or in process from one of the following sources: (a) The agency's Integrated Risk Information System (IRIS); (b) the agency's Office of Water (OW); (c) the agency's Office of Pesticide Programs (OPP); (d) the National Academy of Sciences (NAS); (e) the Agency for Toxic Substances and Disease Registry (ATSDR); and/or (f) the World Health Organization (WHO). For a non-EPA health assessment (i.e., NAS, ATSDR, WHO) to be utilized for regulatory determinations, the health assessment must use comparable methods, standards, and guidelines to an EPA health assessment. If a health assessment is not available from one of these sources, then the contaminant is not considered for RD 3.

In regard to sufficient occurrence data, the agency considers the availability of nationally representative finished water data and whether other finished water data are available that indicate known and/or likely occurrence in PWSs. Occurrence data from the following sources, administered or overseen by EPA, is considered nationally representative: (a) The Second Unregulated Contaminant Monitoring Regulation (UCMR 2); (b) the First Unregulated Contaminant Monitoring Regulation (UCMR 1) Assessment Monitoring; (c) the Unregulated Contaminant Monitoring (UCM) program; and/or (d) the National Inorganics and Radionuclides Survey (NIRS).

If nationally representative data are not available, EPA identifies and evaluates other finished water data, which may include other national assessments as well as regional, State, and more localized finished water assessments. These other national finished water data include assessments that are geographically distributed across the nation but not intended to be statistically representative of the nation. These other finished water data include the following sources for consideration in the regulatory determination process: (a) Finished water assessments for Federal agencies (e.g., EPA and the United States Geological Survey (USGS));[12] (b) state-level finished water monitoring data; (c) research performed by institutions and universities (e.g., scientific literature); and/or (d) other supplemental finished water monitoring surveys (e.g., Pesticide Monitoring Program (PMP), National Reconnaissance of Emerging Contaminants (NREC), and other targeted surveys or localized State/Federal monitoring surveys).

EPA prefers to have nationally representative data available when making regulatory determinations but may also use these other sources of finished water occurrence data to evaluate the contaminant and determine if there is ‘‘substantial likelihood that the contaminant will occur in PWSs with a frequency and at levels of public health concern.’’ If there is sufficient occurrence in these other finished water data sources, EPA uses this information to address the occurrence-related aspects of the statutory criteria when deciding to regulate a contaminant. However, it is difficult to determine that a contaminant is not occurring or not likely to occur based on these other sources of finished water data because the data are limited in scope and the contaminant could be occurring in other parts of the country that were not monitored.

EPA also considers the availability of analytical methods for monitoring, and whether the contaminant is part of a contaminant group based on factors defined by the Drinking Water Strategy

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[12] These may be assessments that are geographically distributed across the nation but not intended to be statistically representative of the nation. Examples include EPA's Disinfection By Product Information Collection Request and various USGS water quality surveys.

(DWS) (see section II.B.5). After conducting the health and occurrence data availability assessments, the agency identifies those contaminants and contaminant groups that meet the following Phase 1 data availability criteria:

(a) A peer-reviewed health assessment is available or in process, and

(b) A widely available analytical method for monitoring is available, and

(c) Either nationally representative finished water occurrence data are available, or other finished water occurrence data shows occurrence at levels >½ CCL 3 health reference level (HRL).[13]

If a contaminant meets these three criteria, it is placed on a ''short list'' and proceeds to Phase 2. EPA also evaluated whether the contaminant could be considered as part of a group using the DWS factors discussed earlier in section II.B.5. After evaluating the 116 CCL 3 contaminants in Phase 1, the agency identified 35 CCL 3 contaminants and two non-CCL 3 contaminants (listed in Table 1) to evaluate further in Phase 2. The non-CCL 3 contaminants were included because they are part of a larger group (nitrosamines) that also includes a number of CCL 3 contaminants.

TABLE 1—CONTAMINANTS PROCEEDING FROM PHASE 1 TO PHASE 2

1, 1, 1, 2-Tetrachloroethane[1,3]
1, 2, 3-Trichloropropane[1,3]
1, 3-Dinitrobenzene[1]
1, 4-Dioxane[2]
Acephate[2]
Acetochlor[1,3]
Acetochlor ethanesulfonic acid (ESA)[1,3]
Acetochlor oxanilic acid (OA)[1,3]
Alachlor ethanesulfonic acid (ESA)[1,3]
Alachlor oxanilic acid (OA)[1,3]
Chlorate[2]
Cobalt[1]
Dimethoate[1]
Disulfoton[4]
Diuron[4]
Methyl bromide (Bromomethane)[1]
Methyl tert-butyl ether[1]
Metolachlor[1,3]
Metolachlor ethanesulfonic acid (ESA)[1,3]
Metolachlor oxanilic acid (OA).[1,3]
Molinate.[1]
Molybdenum.[1]
Nitrobenzene.[1,3]
N-Nitroso-di-n-butylamine (NDBA).[1,3,5]
N-Nitrosodiethylamine (NDEA).[1,3]
N-Nitrosodimethylamine (NDMA).[1,3]
N-Nitroso-di-n-propylamine (NDPA).[1,3]
N-Nitrosodiphenylamine (NDPhA).[3]
N-Nitrosomethylethylamine (NMEA).[1,3,5]
N-Nitrosopyrrolidine (NPYR).[1,3]
Perfluorooctanesulfonic acid (PFOS).[2]
Perfluorooctanoic acid (PFOA).[2]
RDX.[1]
Strontium.[1]
Terbufos.[2,3]
Terbufos sulfone.[1,3]
Vanadium.[1]

[1] Has nationally representative finished water data and available or in process health assessment.
[2] Has other finished water data (occurrence at levels >½ CCL 3 HRL) and available or in process health assessment.
[3] Component of a contaminant group and will be further evaluated in Phase 2.
[4] One exception to the criterion of having available nationally representative drinking water data applies to contaminants monitored in the UCMR 1 Screening Survey (SS). As noted in section 5, the UCMR 1 SS is a statistically defined, national sample of 300 PWSs. Because this survey only includes 300 systems, the agency identified and compiled additional supplemental data to compliment the UCMR 1 SS data for these contaminants that proceed to Phase 2 for further evaluation.
[5] A non-CCL 3 contaminant that is part of the nitrosamine group.

The remaining 81 CCL 3 contaminants (listed in Table 2) did not meet either or both of the Phase 1 data availability criteria above and were not considered further for RD 3.

TABLE 2—CONTAMINANTS NOT PROCEEDING FROM PHASE 1 TO PHASE 2

Has nationally representative finished water data but no health assessment

1,1-Dichloroethane
3-Hydroxycarbofuran
Chloromethane (Methyl chloride)
Germanium
Halon 1011 (Bromochloromethane).
n-Propylbenzene.
sec-Butylbenzene.
Tellurium.

Has available or in process health assessment and other finished drinking water data but no occurrence at levels >½ CCL 3 HRL

1-Butanol
Acrolein
alpha-Hexachlorocyclohexane
Bensulide
Benzyl chloride
Captan
Formaldehyde.
Methamidophos.
Oxydemeton-methyl.
Oxyfluorfen.
Permethrin.
Profenofos.

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[13] See section III.C for a discussion about how EPA derives an HRL. EPA developed the CCL 3 HRLs using the most recent health data available during the CCL 3 process. EPA uses ½ CCL 3 HRL as a conservative value to identify contaminants with potential occurrence of concern during Phase 1 of the RD process. The CCL 3 HRLs for the 116 contaminants can be found at (*http://water.epa.gov/scitech/drinkingwater/dws/ccl/upload/Final-CCL-3-Contaminant-Information-Sheets.pdf*). After updating, completing and peer-reviewing health assessments as necessary, the final HRL used for RD 3 may be different than the CCL 3 HRL.

TABLE 2—CONTAMINANTS NOT PROCEEDING FROM PHASE 1 TO PHASE 2—Continued

Dicrotophos	Tebuconazole.
Ethoprop	Tribufos.
Ethylene glycol	Vinclozolin.
Ethylene thiourea (Maneb)	Ziram.
Fenamiphos	
Has other finished drinking water data but no health assessment	
17-alpha-Estradiol	Estriol.
Acetaldehyde	Estrone.
Aniline	Ethinyl Estradiol (17-alpha-ethynyl estradiol).
Butylated hydroxyanisole	HCFC-22.
Cyanotoxins (Anatoxin-a, Cylindrospermopsin, Microcystin-LR)	Hexane.
Equilenin	Mestranol.
Equilin	Norethindrone (19-Norethisterone).
Erythromycin	*Naegleria fowleri*.*
Estradiol (17-beta-Estradiol)	
Does not have nationally representative or other finished water data	
1,3-Butadiene	Quinoline.
2-Methoxyethanol	Tebufenozide.
2-Propen-1-ol	Thiodicarb.
4,4′-Methylenedianiline	Thiophanate-methyl.
Acetamide	Toluene diisocyanate.
Clethodim	Triethylamine.
Cumene hydroperoxide	Triphenyltin hydroxide (TPTH).
Dimethipin	Urethane.
Ethylene oxide	*Campylobacter jejuni*.
Hydrazine	*Escherichia coli (0157)*.
Methanol	*Helicobacter pylori*.
Nitroglycerin	Hepatitis A virus.
N-Methyl-2-pyrrolidone	*Salmonella enteric*.
o-Toluidine	*Shigella sonnei*.
Oxirane, methyl-	
Does not have a widely available analytical method for occurrence monitoring	
Adenovirus	*Legionella pneumophila*.
Caliciviruses	*Mycobacterium avium*.
Enterovirus	
Not within scope of this RD 3 since regulatory determination made in February 2011	
Perchlorate	

* Does not have a widely available analytical method for occurrence monitoring.

2. Phase 2 (Data Evaluation Phase)

Contaminants that meet the minimum health and occurrence data availability requirements in Phase 1 are advanced to the Phase 2 evaluation. In addition to health and occurrence information data assessed in Phase 1, the agency collects additional health and occurrence data and more thoroughly evaluates this information to identify a list of contaminants that should proceed to Phase 3. The agency uses the following steps to develop this list: (a) Derive a draft HRL[14] (See section III.C) for each contaminant, (b) compare all occurrence data against the draft HRL (along with the analytical method minimum reporting limit (MRL)), (c) identify contaminants that occur at levels and frequencies of public health concern, and (d) identify contaminants that have no or low occurrence at levels of public health concern.

Using the available health effects assessments, the agency derives a draft HRL and then evaluates this HRL value (along with the analytical method MRL), against the concentration values compiled for the nationally representative or other finished water occurrence information identified in Phase 1. The agency also gathers additional occurrence data and information on monitoring in ambient or source water (relative to the draft HRL and the analytical method MRL), production, use, release to the environment, and persistence and mobility. In Phase 2, the agency specifically focuses its efforts to identify those contaminants or contaminant groups that are occurring or have substantial likelihood to occur at levels and frequencies of public health concern. To identify such contaminants, the agency considers the following information:

(a) How many samples (# and %) have detections > draft HRL and ½ draft HRL in the nationally representative and other finished water occurrence data?

(b) How many systems (# and %) have detections > draft HRL and ½ draft HRL in the nationally representative and other finished water occurrence data? and

(c) Is the contaminant associated with a contaminant group that is of public

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[14] HRLs are not final determinations about the level of a contaminant in drinking water that is necessary to protect any particular population and are derived prior to development of a complete exposure assessment. HRLs are risk derived concentrations against which to evaluate the occurrence data to determine if contaminants occur at levels of potential public health concern.

health concern and is being considered as part of the DWS?[15]

(d) Are there uncertainties or limitations with the data and/or analyses, such as the age of the dataset, limitation of the detection limit (i.e., MRL > draft HRL) and/or representativeness of the data (e.g., limited to a specific region) that may cause misestimation of occurrence in finished water at levels and frequency of public health concern?

After identifying contaminants that are occurring at levels and frequencies of public health concern to proceed to Phase 3, the agency evaluates the remaining contaminants on the ''short list'' to determine which contaminants have no or low occurrence at levels of health concern that could also proceed to Phase 3 by considering the following factors:

(a) Does the contaminant have nationally representative finished water data showing no or low # or % of detections > draft HRL?[16]

(b) If a contaminant has other finished water data in addition to nationally representative finished water data, does it support no or low potential for occurrence in drinking water?

(c) Does additional occurrence information of known quality support low or no occurrence or potential for occurrence in drinking water? For example, is the occurrence in ambient/source water at levels below the draft HRL? Are releases to the environment or use/production decreasing over time?

(d) There are no critical information/data gaps after evaluating the available health or occurrence data; and

(e) The contaminant is not included or evaluated with a group of contaminants based on the factors defined by the DWS.

After evaluating these factors and whether a contaminant appears to have sufficient data to evaluate the statutory criteria for regulatory determination, the agency determines if the contaminant should proceed to Phase 3. After evaluating the ''short list'' contaminants (listed in Table 1), the agency identified 10 CCL 3 contaminants and 2 non-CCL 3 contaminants (listed in Table 3) that were within one of the following Phase 2 data evaluation categories to proceed to Phase 3:

(a) A contaminant or part of a contaminant group occurring or likely to occur at levels and frequencies of public health concern, or

(b) A contaminant not occurring or likely to occur at levels and frequencies of public health concern and no data gaps.

TABLE 3—CONTAMINANTS PROCEEDING FROM PHASE 2 TO PHASE 3

Chlorate[1][3]
Dimethoate[2]
1,3-Dinitrobenzene[2]
N-Nitroso-di-n-butylamine (NDBA)[1][4]
N-Nitrosodimethylamine (NDMA)[1]
N-Nitroso-di-n-propylamine (NDPA)[1]
N-Nitrosodiethylamine (NDEA).[1]
N-Nitrosomethylethylamine (NMEA).[1][4]
N-Nitrosopyrrolidine (NPYR).[1]
Strontium.[1][3]
Terbufos.[2]
Terbufos Sulfone.[2]

[1] A contaminant or part of a contaminant group occurring or likely to occur at levels and frequencies of public health concern.

[2] A contaminant not occurring or likely to occur at levels and frequencies of public health concern and no data gaps.

[3] The UCMR 3 includes sampling at both the entry point to the distribution system (EPTDS) and distribution system maximum residence time (DSMRT) for this contaminant (77 FR 26071, May 2, 2012). For some contaminants, including disinfection byproducts and inorganics, occurrence values may differ between the EPTDS and the DSMRT due to dynamics within the distribution system such as contaminant degradation, formation, accumulation and release.

[4] A non-CCL 3 contaminant that is part of the nitrosamine group.

Note that the agency does not have a threshold or a bright line for occurrence in drinking water that triggers whether a contaminant is of public health concern. There are a number of factors to consider in developing thresholds, some of which include the health effect(s), the potency of the contaminant, the level at which the contaminant is found in drinking water, how frequently the contaminant is found, the geographic distribution (national, regional, or local occurrence), other possible sources of exposure, and potential impacts on sensitive populations or lifestages, etc. Given the many possible combinations of factors and the constantly evolving science, EPA believes it is better to analyze each contaminant and characterize and present the best available information that helps identify whether the occurrence of a contaminant is of public health concern. In the end, the determination of whether there is a meaningful opportunity for health risk reduction by regulation of a contaminant in drinking water is a highly contaminant-specific one that takes into consideration a large number of factors.

The remaining 25 CCL 3 contaminants (listed in Table 4) did not proceed to Phase 3 and were not considered for RD 3 because of one or more of the following critical health, occurrence, and/or other data gaps:

(a) An updated health assessment is needed, but was not completed by fall 2011;

(b) A health assessment is in process, but was not completed by fall 2011;

(c) Critical health effects gap (e.g., lack of data to support quantification for the oral route of exposure);

(d) Lacked nationally representative occurrence data;

(e) Insufficient other finished water occurrence data to demonstrate occurrence at levels and frequencies of public health concern (although it may have some levels of public health concern);

(f) Individual contaminants that were part of a group but lacked a widely available analytical method for occurrence monitoring; and

(g) Critical occurrence data gap (e.g., inconsistent results and/or trends in occurrence data, significant uncertainty in occurrence analyses and/or data).

Table 4 identifies the health, occurrence, and/or other data gaps that prevented the following 25 contaminants from moving forward for RD 3. The agency continues to conduct research, collect information or find other avenues to fill the data and information gaps identified in Table 4.

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[15] Carcinogenic Volatile Organic Compounds (including 1,2,3-trichloropropane) are being evaluated in a separate regulatory effort.

[16] Note that the non-national data tend to be limited in scope and EPA does not use these data alone to support a determination that the contaminant is not or is not substantially likely to ''occur in PWSs with a frequency and at levels of public health concern,'' which would therefore be a decision ''not to regulate'' (*i.e.*, negative determination).

TABLE 4—DATA AND RATIONALE SUMMARY OF THE 25 CONTAMINANTS NOT PROCEEDING TO PHASE 3

No.	Contaminant	Health data available	Occurrence data available	Rationale
1	1,4-Dioxane	Yes	No[1]	Occurrence data gaps (no nationally representative finished water data or sufficient other finished water data).
2	Acephate	Yes	No	Occurrence data gaps (no nationally representative finished water data or sufficient other finished water data).
3	Acetochlor	No	Yes	Health data gap (no health assessment for the degradates) and no detections in nationally representative finished water data.
4	Acetochlor ethanesulfonic acid (ESA).	No	Yes	Health data gap (no health assessment for the ESA degradate) and no or low detections based on nationally representative finished water data.
5	Acetochlor oxanilic acid (OA)	No	Yes	Health data gap (no health assessment for the OA degradate) and no or low detections based on nationally representative finished water data.
6	Alachlor ethanesulfonic acid (ESA).	No	Yes	Health data gap (no health assessment for the ESA degradate) and no or low detections based on nationally representative finished water data.
7	Alachlor oxanilic acid (OA)	No	Yes	Health data gap (no health assessment for the OA degradate) and no or low detections based on nationally representative finished water data.
8	Cobalt	No	Yes[2]	Health data gap (health assessment not updated by fall 2011) and no detections in nationally representative or other finished water data at levels of public health concern.
9	Disulfoton	Yes	No	Occurrence data gap (no nationally representative finished water data and no detections in other finished water data).
10	Diuron	Yes	No	Occurrence data gap (no nationally representative finished water data and no detections in other finished water data).
11	Methyl Bromide	No	Yes[1]	Health data gap (health assessment not updated by fall 2011).
12	Methyl tert-butyl ether	No	Yes	Health data gap (IRIS health assessment not completed by fall 2011) and no or low detections based on nationally representative finished water data.
13	Metolachlor	No	Yes	Health data gap (no health assessment for degradates) and few detections in nationally representative finished water data.
14	Metolachlor ethanesulfonic acid (ESA).	No	Yes	Health data gap (no health assessment for ESA degradate) and no or low detections based on nationally representative finished water data.
15	Metolachlor oxanilic acid (OA)	No	Yes	Health data gap (no health assessment for OA degradate) and no or low detections based on nationally representative finished water data.
16	Molinate	No	Yes	Health data gap (OPP health assessment not completed by fall 2011 due to cancellation of molinate) and no detections in nationally representative or other finished water data at levels of public health concern.
17	Molybdenum	No	Yes	Health data gap (health assessment not updated by fall 2011) and no detections in nationally representative or other finished water data at levels of public health concern.
18	N-Nitrosodiphenylamine (NDPhA).	Yes	No	Health data gap (health assessment not updated by fall 2011) and occurrence data gaps (no EPA approved analytical method for monitoring).
19	Perfluorooctanesulfonic acid (PFOS).	No	No[1]	Health data gap (health assessment not completed by fall 2011) and occurrence data gaps (limited other finished water data available).
20	Perfluorooctanoic acid (PFOA)	No	No[1]	Health data gap (health assessment not completed by fall 2011) and occurrence data gaps (limited other finished water data available).
21	RDX	No	Yes	Health data gap (IRIS health assessment not updated by fall 2011) and no detections in nationally representative or other finished water data at levels of public health concern.
22	Vanadium	No	Yes[2]	Health data gap (health assessment not updated by fall 2011) and no to low detections in nationally representative finished water data at levels of public health concern.
23	1,1,1,2-Tetrachloroethane			Will be evaluated and considered for the Carcinogenic Volatile Organic Compounds (cVOCs) group rule addressed in a separate process.
24	1,2,3-Trichloropropane		(1)	Will be evaluated and considered for the Carcinogenic Volatile Organic Compounds (cVOCs) group rule addressed in a separate process.

TABLE 4—DATA AND RATIONALE SUMMARY OF THE 25 CONTAMINANTS NOT PROCEEDING TO PHASE 3—Continued

No.	Contaminant	Health data available	Occurrence data available	Rationale
25	Nitrobenzene			Will be evaluated and considered for the Carcinogenic Volatile Organic Compounds (cVOCs) group rule addressed in a separate process.

[1] The UCMR 3 includes sampling at the EPTDS for this contaminant (77 FR 26071, May 2, 2012).

[2] The UCMR 3 includes sampling at both the EPTDS and DSMRT for this contaminant (77 FR 26071, May 2, 2012). For some contaminants, including disinfection byproducts and inorganics, occurrence values may differ between the EPTDS and the DSMRT due to dynamics within the distribution system such as contaminant degradation, formation, accumulation and release.

3. Phase 3 (Regulatory Determination Assessment Phase)

Phase 3, the Regulatory Determination Assessments Phase, involves a complete evaluation of the statutory criteria for each contaminant or group of contaminants that proceed from Phase 2 and have sufficient information and data for making a regulatory determination. In this phase, the agency evaluates the following statutory criteria:

(a) Statutory Criterion #1—The contaminant may have an adverse effect on the health of persons. To evaluate statutory criterion #1, EPA completes any health assessment that needs to be updated and externally peer-reviewed, and derives a final HRL. The derivation of the final HRL, further described in the section III.C.1, Evaluation of Adverse Health Effects, takes into account many of the key elements that are considered when evaluating criterion #1, which includes the mode of action, the critical health effect(s), the dose-response for critical health effect(s), impacts on sensitive populations(s) or lifestages, the RfD, and/or the cancer slope factor. HRLs are not final determinations about the level of a contaminant in drinking water that must not be exceeded to protect any particular population and are derived prior to the development of a complete exposure assessment. HRLs are risk derived concentrations against which to evaluate the occurrence data to determine if contaminants may occur at levels of potential public health concern. With this information, EPA determines whether the contaminant ‘‘may have an adverse effect.’’ While CCL 3 contaminants are generally expected to meet statutory criterion #1 because their adverse health effects were analyzed as part of the determination to list them on the CCL, the availability of a final HRL is derived as part of the first statutory criterion and is necessary to evaluate the second statutory criterion.

(b) Statutory Criterion #2—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. EPA compares the occurrence data for each contaminant to the final peer-reviewed HRL to determine if the contaminant occurs at a frequency and levels of public health concern. The types of occurrence data used at this stage are described in section III.C.2, Evaluation of Contaminant Occurrence and Exposure. The agency considers the following factors when identifying contaminants or contaminant groups that are occurring at frequencies and levels of public health concern:

- How many samples (# and %) have detections > final HRL in the nationally representative and other finished water occurrence data?
- How many systems (# and %) have detections > final HRL in the nationally representative and other finished water occurrence data?
- Is the contaminant associated with a contaminant group that is of public health concern and is being considered as part of the DWS?
- Is the geographic distribution of the contaminant occurrence national, regional, or localized?
- In addition to the number of systems, what type of systems does the contaminant occur in? Does the contaminant occur in large or small systems? Does the contaminant occur in surface or ground water systems?
- Are there significant uncertainties or limitations with the data and/or analyses, such as the age of the dataset, limitation of the detection limit (i.e., MRL > final HRL) and/or representativeness of the data (e.g., limited in scope to a specific region)?

Additional, less important factors that the agency considers when identifying contaminants or contaminant groups that are of public health concern also include:

- How many samples (# and %) have detections >½ final HRL[17] in the nationally representative and other finished water occurrence data?
- How many systems (# and %) have detections >½ final HRL in the nationally representative and other finished water occurrence data?
- How many samples (# and %) have detections > final HRL and ½ final HRL in the ambient/source water occurrence data?
- How many monitoring sites (# and %) have detections > final HRL and ½ final HRL in the ambient/source water occurrence data?
- Are production and use trends for the contaminant increasing or decreasing?
- How many pounds are discharged annually to surface water and/or released to the environment?
- Do the environmental fate and transport parameters indicate that the contaminant would persist and/or be mobile in water?
- Are there other uncertainties or limitations with the data and/or analyses for these additional factors that should be considered?
- Is the contaminant introduced by water treatment processes (e.g., disinfection byproducts)?

If a contaminant is known to occur or substantially likely to occur at a frequency and level of health concern in public water systems based on the factors listed above, then the agency answers ‘‘yes’’ to the second statutory criterion.

(c) Statutory Criterion #3—In the sole judgment of the Administrator, regulation of the contaminant presents a meaningful opportunity for health risk reduction for persons served by public water systems. EPA evaluates the population exposed at the health level of concern along with several other factors to determine if regulation presents a meaningful opportunity for health risk reduction. EPA considers the following factors in evaluating statutory criterion #3:

- Based on the occurrence information for statutory criterion #2 (and the potential number of systems impacted), what is the national population exposed or served by

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[17] Note that the ½ HRL threshold is based on a recommendation from the NDWAC working grouping that provided recommendations on the first regulatory determinations effort. (USEPA, 2000b)

systems with levels ≥ HRL and ½ HRL (provide actual and estimated # and %)?

- What is the nature of the health effect(s) identified in statutory criterion #1 and are there sensitive populations that may be impacted (either qualitative or quantitative[18])?
- For non-carcinogens, are there other sources of exposure that should be considered (i.e., what is the relative source contribution)?
- What is the geographic distribution of occurrence (e.g., local, regional, national)?
- Are there any uncertainties and/or limitations in the health and occurrence information or analyses that should be considered?
- What other factors or other pieces of information should be considered that may have direct bearing on any decision to regulate the contaminant (e.g., treatment, analytical methods,[19] etc.)?

After evaluating these factors, if the Administrator determines that there is a meaningful opportunity to reduce risk by regulating the contaminant in drinking water, then the agency answers ''yes'' to the third statutory criterion.

If the agency answers ''yes'' to all three statutory criteria in Phase 3 for a particular contaminant, then the agency makes a ''positive'' preliminary determination and requests public comment.

If after the public comment period, the agency answers ''yes'' to all three statutory criteria, the agency then makes a ''positive'' final determination that regulation is necessary and proceeds to develop an MCLG and NPDWR. The agency has 24 months to publish a proposed MCLG and NPDWR and an additional 18 months to publish a final MCLG and promulgate a final NPDWR. It should be noted that this regulatory determination process is distinct from the more detailed analyses needed to develop a national primary drinking water regulation. Thus, a decision to regulate is the beginning of the agency's regulatory development process, not the end.

If a contaminant has sufficient information and the agency answers ''no'' to any of the three statutory criteria, based on the available data, then the agency considers making a ''negative'' determination that an NPDWR is not necessary for that contaminant at that time. The agency may decide to develop a Health Advisory (HA), which provides non-regulatory concentration values for drinking water contaminants at which adverse health effects are not anticipated to occur over specific exposure durations (one-day, ten-days, several years, and a lifetime). HAs serve as informal technical guidance to assist Federal, State, and local officials, and managers of public or community water systems (CWSs) in protecting public health when emergency spills or contamination situations occur.

While a negative determination is considered a final agency action for this round of regulatory determinations, the contaminant is reconsidered for inclusion on the next CCL. If new health or occurrence information becomes available on contaminants with negative regulatory determinations, the agency considers whether the contaminant(s) should be listed on the next CCL and further evaluated in the next regulatory determinations process.

Of the twelve contaminants that proceeded to Phase 3, the agency is not making preliminary regulatory determinations for seven contaminants at this time. The seven contaminants include chlorate and the six nitrosamines (i.e., NDBA, NDMA, NDPA, NDEA, NPYR, and NMEA). As discussed in section V, chlorate and the six nitrosamines are DBPs and the agency plans to consider these contaminants as part of the regulatory review of existing MDBP regulations. DBPs need to be evaluated collectively, because the potential exists that the control of one DBP could affect the concentrations of other DBPs or the necessary treatment. After evaluating the five remaining CCL 3 contaminants in Table 3 (i.e., dimethoate, 1,3-dinitrobenzene, strontium, terbufos, and terbufos sulfone) against the three SDWA criteria and considering the factors listed for each, the agency is making preliminary regulatory determinations for these five CCL 3 contaminants. Table 5 provides a summary of the five contaminants evaluated for Phase 3 and the preliminary regulatory determination outcome. The agency seeks comment on the preliminary determination to regulate one contaminant (i.e., strontium) and to not regulate the remaining four contaminants (i.e., dimethoate, 1,3-dinitrobenzene, terbufos, and terbufos sulfone). Section IV.B of this notice provides a more detailed summary of the information and the rationale used by the agency to reach its preliminary decisions for these five contaminants.

TABLE 5—CONTAMINANTS EVALUATED IN PHASE 3 AND THE REGULATORY DETERMINATION OUTCOME

No.	RD 3 contaminants	Preliminary determination outcome
1	Dimethoate	Do not regulate.
2	1,3-Dinitrobenzene	Do not regulate.
3	Strontium	Regulate.
4	Terbufos	Do not regulate.
5	Terbufos Sulfone	Do not regulate.

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[18] If appropriate and if available, the agency quantitatively takes into account exposure data applicable to sensitive populations or lifestages when deriving HRLs for regulatory determinations. When data is not available on sensitive populations, the derivation of the RfD typically includes an uncertainty factor to account for the weakness in the database. See section III.C.1. Sensitive populations are also qualitatively considered by providing national prevalence estimates for a particular sensitive population if available.

[19] If the agency decides to regulate a contaminant, SDWA requires that EPA issue a proposed regulation within two years of the final determination (with the possibility of a 9 month extension). As part of the proposal, the agency must list the best available technologies (BATs), small system compliance technologies (SSCTs), and approved analytical methods if it proposes an enforceable MCL. Alternatively, if EPA proposes a treatment technique (TT) instead of an MCL, the agency must identify the TT. EPA must also prepare a health risk reduction and cost analysis. This analysis includes an extensive evaluation of the treatment costs and monitoring costs at both system level and aggregated at the national level. To date, treatment information and approved analytical methods have not been a significant factor in regulatory determinations but are important considerations for regulation development.

B. 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. These support documents include:

- The comprehensive regulatory support document entitled, ''Regulatory Determination 3 Support Document'' (USEPA, 2014b), 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 determinations, and the agency's rationale for these determinations.
- A separate health effects support document for strontium, entitled ''Health Effects Support Document for Strontium'' (USEPA, 2014c), that addresses exposure from drinking water and other media, toxicokinetics, hazard identification, and dose-response assessment, and provides an overall characterization of the risk from drinking water containing strontium. For the contaminants with negative determinations, the agency refers the reader to the IRIS or OPP assessments for more detailed information regarding health effects (USEPA, 1990a, 1990b, 2003c). These documents serve as the basis for the health information provided in the regulatory support documents.
- A comprehensive technical occurrence support document for UCMR 2 entitled, ''Occurrence Data from the Second Unregulated Contaminant Monitoring Rule (UCMR 2)'' (USEPA, 2014d). This occurrence support document includes more detailed information about UCMR 2, 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.
- A comprehensive protocol document, entitled ''Protocol for the Regulatory Determination 3'' (USEPA, 2014a). This protocol document describes the approach implemented by the agency to evaluate 116 CCL 3 contaminants in a three phase process and select the contaminants for preliminary determinations for RD 3. The protocol underwent expert review and the comments received were addressed by the agency.

C. Analyses Used To Support the Preliminary Regulatory Determinations

Sections III.C.1 and 2 of this action outline the health effects and occurrence/exposure evaluation process EPA used to support these preliminary determinations.

1. 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 uses to evaluate health effects, hazard and dose-response information, and the approach for deriving the health reference level (HRL) for the contaminants under consideration for regulatory determinations. HRLs are not final determinations about the level of a contaminant in drinking water that must not be exceeded to protect any particular population. HRLs are derived prior to the development of a complete exposure assessment. HRLs are risk derived concentrations against which to evaluate the occurrence data to determine if contaminants occur at levels of potential public health concern. More specific information about the potential for adverse health effects for each contaminant is presented in section IV.B of this action.

In evaluating contaminants for regulatory determination, Section 1412 (b)(1)(C) of SDWA also requires the agency to consider among other factors of public health concern, the effect of such contaminants upon subgroups that comprise a meaningful portion of the general population ''such as infants, children, pregnant women, the elderly, individuals with a history of serious illness, or other subpopulations'' that are identifiable as being at greater risk of adverse health effects compared to the general population. If appropriate and if available, the agency quantitatively takes into account data from sensitive populations and lifestages when deriving HRLs for regulatory determinations.

There are two general 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 non-carcinogens and carcinogens evaluated using a non-linear approach. The derivation of HRLs for carcinogens and non-carcinogens are described below.

a. Derivation of an HRL for Carcinogens

For those contaminants that are considered to be likely or probable human carcinogens by a mutagenic or unknown mode of action (MOA), the agency calculates a toxicity value that defines the relationship between dose and response (i.e., the cancer slope factor or CSF).

(1) MOA: Unknown

In cases where the data on the mode of action are lacking, EPA typically uses a default low dose linear extrapolation to calculate a CSF. The unit risk is the estimated upper-bound excess lifetime cancer risk from a continuous exposure to a chemical at a concentration of 0.001 mg/L in drinking water. The exposure estimate assumes an adult body weight of 70 kg and the 90th percentile adult drinking water intake of 2 L/day.

$$\text{Unit Risk } (\mu g/L)^{-1} = \text{CSF} \times [(\text{DWI} \times \text{CW})/\text{BW}]$$

Where:

CSF = Cancer Slope Factor $(mg/kg/day)^{-1}$

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

CW = Unit risk concentration in drinking water of 0.001 mg/L (1 μg/L)

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

The cancer HRL is the concentration of a contaminant in drinking water corresponding to an excess estimated lifetime cancer risk of one-in-a-million (1×10^{-6}), calculated as follows:

$$\text{HRL } (\mu g/L) = \text{Risk Level of } 10^{-6} \div \text{Unit Risk } (\mu g/L)^{-1}$$

As noted above, HRLs are not final determinations about the level of a contaminant in drinking water that must not be exceeded to protect any particular population. Rather, HRLs are risk derived concentrations against which to evaluate the occurrence data during the RD process to determine if contaminants occur at levels of *potential* public health concern.

(2) MOA: Mutagenic

If the chemical has a mutagenic mode of action, low dose linear extrapolation is used to calculate the CSF as described in the preceding paragraph. The U.S. EPA's 2005 Guidelines for Carcinogen Risk Assessment (USEPA, 2005b) requires that the potential increased cancer risk due to early-life exposure be taken into account for chemicals with a mutagenic mode of action. When chemical-specific data to quantify the increased risk are lacking, Age Dependent Adjustment Factors (ADAFs) are applied to estimate age-adjusted unit risks. The age-adjusted unit risk is determined by using the sum of the unit risks for each of the three ADAF developmental groups (birth to <2 yrs; 2 yrs to <16 yrs; 16 yrs to 70 yrs). The age-adjusted unit risks include a ten-fold adjustment for early life (birth to <2 yrs) exposures, a three-fold adjustment for childhood/adolescent (2 yrs to <16 yrs) exposures, and no additional adjustment for exposures later in life (16 yrs to 70 yrs), in conjunction with age-specific drinking water intake values derived from the U.S. EPA's 2011 Exposure Factors Handbook (USEPA, 2011e), and the fraction of a 70 year lifetime applicable to each age period. The increase in risk during early life results from active tissue growth resulting in limited time for repair of DNA replication errors. The age-adjusted unit risk is the upper-bound excess lifetime cancer risk estimated to result from continuous postnatal exposure to a chemical at a concentration of 0.001 mg/L in drinking water.

$$\text{Age-Adjusted Unit Risk } (\mu g/L)^{-1} = \boldsymbol{\Sigma}(CSF \times ADAF \times DWI/BWR \times CW \times F)$$

Where:

CSF = Cancer Slope Factor (mg/kg/day) $^{-1}$

ADAF = The Age Dependent Adjustment Factor for the age group birth to two-years (ADAF = 10), two years to sixteen years (ADAF = 3), and sixteen to seventy years (ADAF = 1)

DWI/BWR = Drinking Water Intake Body Weight Ratio (DWI/BWR) expressed as liters per kg body weight for the age-specific group (90th percentile, consumers only)[20]

CW = Unit risk concentration in drinking water of 0.001 mg/L (1 μg/L)

F = The fraction of a 70 year lifetime applicable to the age period: 2/70 for birth to two years, 14/70 for two years to sixteen years and 54/70 for sixteen years to seventy years

The cancer HRL is the concentration of a contaminant in drinking water corresponding to an excess estimated lifetime cancer risk of one-in-a-million (1×10^{-6}), calculated as follows:

$$\text{HRL } (\mu g/L) = \text{Risk Level of } 10^{-6} \div \text{Age-Adjusted Unit Risk } (\mu g/L)^{-1}$$

The six nitrosamines discussed in section V had data available to classify them as known or likely human carcinogens with a mutagenic mode of action. Low-dose linear extrapolations and ADAFs were applied to all four of the CCL 3 nitrosamines: NDMA, NDPA, NDEA and NYPR, as well as the two non-CCL 3 nitrosamines, NMEA and NDBA. The five contaminants for which the agency is making preliminary regulatory determinations (dimethoate, 1,3-dinitrobenzene, strontium, terbufos and terbufos sulfone) are non-carcinogens and were therefore evaluated using the RfD approach (discussed in the following section).

b. Derivation of an HRL for Non-Carcinogens

EPA generally calculates a reference dose (RfD) for those chemicals considered to be non-carcinogenic or not likely to be carcinogenic to humans. An RfD is an estimate of a daily oral exposure to the human population (including sensitive populations or lifestages) that is likely to be without an appreciable risk of deleterious effects during a lifetime. The RfD can be derived from either a no-observed-adverse-effect level (NOAEL), a lowest-observed-adverse-effect level (LOAEL), or the 95% lower confidence bound on a benchmark dose (BMD), known as a BMDL, with uncertainty factors applied to reflect limitations of the data used. In addition, if the critical health endpoint has high quality data associated with exposure for a specific developmental group or period of sensitivity, age-specific drinking water intake to body weight ratio values from the Exposure Factors Handbook (USEPA, 2011e) may be included in deriving an HRL from the RfD.

The agency uses uncertainty factors (UFs) to address uncertainty resulting from incompleteness of the toxicological database (e.g., lacking sensitive population data). The individual UFs (usually applied as integers of one, three, or ten) are multiplied together and used to derive the RfD from experimental data. Individual UFs are intended to account for:

(1) Variation in sensitivity among the members of the human population (i.e., intraspecies variability);

(2) uncertainty in extrapolating animal data to humans (i.e., interspecies variability);

(3) 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) uncertainty in extrapolating from an LOAEL rather than from an NOAEL; and/or

(5) uncertainty associated with an incomplete database.

For chlorate, dimethoate, 1,3-dinitrobenzene, strontium,[21] terbufos, and terbufos sulfone, EPA derived the HRLs using the RfD approach as follows:

$$\text{HRL (mg/L)} = [(RfD \times BW)/DWI] \times RSC$$

Where:

RfD = Reference Dose (mg/kg-day)

BW = Body Weight for an adult, assumed to be 70 kilograms (kg); for a child, assumed to be 10 kg

DWI = Drinking Water Intake for an adult, assumed to be 2 L/day (90th percentile); for child, assumed to be 1L/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). In all cases, a 20% RSC is used for HRL derivation because (1) HRLs are developed prior to a complete exposure assessment and (2) 20% is the most conservative RSC used in the derivation of an MCLG for drinking water.

c. Sources of Data/Information for Health Effects

EPA uses the best available peer-reviewed data and analyses in evaluating adverse health effects. Peer-reviewed health-risk assessments are available for all chemicals considered for regulatory determinations from the agency's Integrated Risk Information System (IRIS) Program,[22] the agency's Office of Pesticide Programs (OPP),[23] the National Academy of Sciences (NAS), the Agency for Toxic Substances and Disease Registry (ATSDR),[24] and/or the World Health Organization (WHO).[25] For a non-EPA health

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[20] The drinking water intake values were derived from the data in the U.S. EPA's Exposure Factors Handbook (USEPA, 2011e). The procedure used for the data normalization is described in the OW Policy paper for determining lifetime cancer risks involving early life exposures (USEPA, 2012c).

[21] Because the critical health endpoint had dose-response data associated with exposure during a specific period of sensitivity (i.e., sensitive population), EPA used age-specific drinking water intake to body weight ratio values (DWI/BWR) from the Exposure Factors Handbook (USEPA, 2011e) to derive the HRL for strontium.

[22] IRIS is an electronic EPA data base (*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.

[23] The OPP is required under the Federal Insecticide Fungicide and Rodenticide Act (FIFRA) to periodically review the health effects data on all registered pesticides and reregister them for continued use. The results of the reregistration analysis are published in the Reregistration Eligibility Decision (RED) documents. Copies of the REDs are located at the following EPA Web site (*http://www.epa.gov/oppsrrd1/reregistration/status.htm*).

[24] ATSDR establishes oral minimal risk levels for non-neoplastic endpoints for acute (14 days or less), intermediate (15—364 days), and chronic (365 days or more) exposure durations. Minimal risk levels for oral chronic exposure are similar to EPA's RfDs. However, ATSDR and EPA use different approaches when the database is limited to subchronic studies and no adequate chronic study is available. ATSDR derives an intermediate duration minimal risk level that protects against exposures up to 10% of a lifetime, and it does not incorporate an uncertainty factor to account for using a less-than-lifetime study. ATSDR does not perform quantitative cancer assessments or assign formal cancer classifications or descriptors.

[25] WHO establishes a ''guideline value'', a drinking water concentration that uses different default assumptions than EPA for estimating water concentration from doses, including a 60 kg adult body weight, daily water consumption of 2 L/day, and a data derived or default RSC of 10%. WHO develops one guideline value that is based either on cancer or non cancer.

assessment (i.e., NAS, ATSDR, WHO) to be considered for regulatory determinations, the health assessment must use comparable methods, standards, and guidelines to an EPA health assessment. Table 6 summarizes the sources of the health assessment data for each chemical under consideration for RD 3.

The agency performs a literature search for studies published after the available health assessment is completed to determine if new information suggests a different outcome. The agency collects and evaluates 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 indicate that a change to the existing RfD or cancer assessment is needed, the EPA Office of Water prepares and independently peer-reviews an ''OW Assessment'' of the data. EPA updates all quantitative cancer assessments conducted under the Guidelines for Carcinogen Risk Assessment (USEPA, 1986) using the Guidelines for Carcinogen Risk Assessment (USEPA, 2005b), the Supplemental Guidance for Assessing Susceptibility from Early-life Exposures to Carcinogens (USEPA, 2005c), and the Exposure Factors Handbook (USEPA, 2011e). These guidelines include considerations for contaminants with a mutagenic mode of action and potential risks due to early childhood exposure.

TABLE 6—SOURCES AND DATES OF EPA HEALTH RISK ASSESSMENTS

Chemical	IRIS (date)	OPP RED (date)	OW Assessment (date)
Dimethoate		2007	
1,3-Dinitrobenzene [1]	1988		
Strontium	1992		2012
Terbufos		2006	
Terbufos Sulfone [2]		2006	

[1] The agency also reviewed a non-EPA source (ATSDR, 1995) for 1,3-dinitrobenzene to corroborate the IRIS assessment.
[2] The OPP RED for the parent compound (terbufos) was used.

As noted in section III.B, EPA prepared a technical Health Effects Support Document for strontium (USEPA, 2014c). This document addresses the exposure from drinking water and other media, toxicokinetics, hazard identification, and dose-response assessment, and provides an overall characterization of risk from drinking water. For the contaminants with a preliminary negative determination (i.e., a decision not to regulate), refer to the EPA health risk assessments online from OPP or IRIS for additional health effect information.

2. Evaluation of Contaminant Occurrence and Exposure

EPA uses data from many sources to evaluate occurrence and exposure from drinking water contaminants. The following comprise the primary sources of finished drinking water occurrence data discussed in this **Federal Register** notice:

- the Unregulated Contaminant Monitoring Regulation (UCMR 1 and 2),
- the National Inorganic and Radionuclide Survey (NIRS), and
- Disinfection Byproducts Information Collection Rule (DBP ICR).

Several of the primary sources of finished water occurrence data are designed to be statistically representative of the nation. These data sources include UCMR 1, UCMR 2, and NIRS.[26] The DBP ICR is geographically distributed across the country and national in scope but is not intended to be statistically representative of the nation.

The agency also evaluates supplemental sources of information on occurrence in drinking water, occurrence in ambient and source water, and information on contaminant use and release to augment and compliment these primary sources of drinking water occurrence data. Section III.C.2.a. of this action provides a brief summary of the primary sources of finished water occurrence data, and sections III.C.2.b and II.C.2.c provide brief summary descriptions of some of the supplemental sources of occurrence information and/or data. These descriptions do not cover all the reports that EPA reviews and evaluates. For individual contaminants EPA reviews additional published reports and peer-reviewed studies that may provide the results of monitoring efforts in limited geographic areas. A summary of the occurrence data and the results or findings for each of the contaminants considered for regulatory determination is presented in section IV.B, the contaminant profiles section, and the data are described in further detail in the support documents for the RD 3 process (see USEPA, 2014a, b, c and d).

a. Primary Sources of Finished Drinking Water Occurrence Data

As previously mentioned, the primary national sources of the drinking water occurrence data discussed in this **Federal Register** notice are UCMR 1, UCMR 2, NIRS, and the DBP ICR. The following sections provide a brief summary of these data sources. Table 7 in section IV lists the primary data source/finding used to evaluate each of the five contaminants considered for regulatory determinations. The contaminant-specific discussions in section IV provide more detailed information about the primary data source findings as well as any supplemental occurrence information.

(1) The Unregulated Contaminant Monitoring Regulation (UCMR 1 and UCMR 2)

The UCMR is currently EPA's primary vehicle for collecting monitoring data on the occurrence of unregulated contaminants in PWSs. The UCMR is designed to collect nationally representative occurrence data and is developed in coordination with the CCL and Regulatory Determination process and the National Drinking Water Contaminant Occurrence Database (NCOD). The UCMR sampling is limited by statute to 30 contaminants during any five year cycle (SDWA section 1445(a)(2)) and the PWSs and State primacy agencies are required to report the data to EPA. EPA published the list and requirements for the first Unregulated Contaminant Monitoring Regulation cycle (i.e., UCMR 1) in September 17, 1999 (64 FR 50556, September 17, 1999, USEPA, 1999; see also 65 FR 11372, March 2, 2000, USEPA, 2000a; and 66 FR 2273, January 11, 2001, USEPA, 2001a), and the monitoring was conducted primarily during 2001–2003. UCMR 2 was published on January 4, 2007 (72 FR 367; USEPA, 2007a), with monitoring

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[26] NIRS is designed to be statistically representative of groundwater systems and does not include surface water systems.

conducted during 2008–2010. (The complete analytical monitoring lists are available at: *http://water.epa.gov/lawsregs/rulesregs/sdwa/ucmr/*.)

The UCMR was designed as a three-tiered approach for monitoring contaminants related to the availability of analytical methods and related analytical laboratory capacity. Assessment Monitoring (AM), the largest sampling tier, typically relies on analytical methods that are in common use in drinking water laboratories. The Screening Survey (SS), the second tier, uses newly developed analytical methods that may not be as commonly used in drinking water laboratories. The SS has involved a smaller number of PWSs because laboratory capacity is expected to be limited. The third tier, Pre-Screen Testing was designed to address contaminants with analytical methods that are in an early stage of development and the analyses would be limited to a few special laboratories. The expectation was that it would only involve the limited number of systems determined to be most vulnerable to the targeted contaminants. No Pre-Screen Testing was conducted during UCMR 1 or UCMR 2.

EPA designed the AM sampling frame to ensure that sample results would support a high level of confidence and a low margin of error (see USEPA, 1999 and 2001b, for UCMR design details). AM is required for all large PWSs, those serving more than 10,000 people (i.e., a census of all large systems) and a national statistically representative sample of 800 small PWSs, those serving 10,000 or fewer people (for a total sample of approximately 4,000 systems). PWSs that purchase 100% of their water were not required to participate.

Each system conducts UCMR assessment monitoring for one year (during the three-year monitoring period). The rules require quarterly monitoring for surface water systems and twice-a-year, six-month interval monitoring for ground water systems. At least one sampling event must occur during a specified vulnerable period. Differing sampling points within the PWS may be specified for each contaminant related to the contaminants source(s).

The objective of the UCMR 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 two systems from each State for spatial coverage. The UCMR AM program includes systems from all 50 States, the District of Columbia, four U.S. Territories, and Tribal lands in five EPA Regions. With contaminant monitoring data from all large PWSs—a census of large systems—and a statistical, nationally representative sample of small PWSs, the UCMR AM program provides a robust dataset for evaluating national drinking water contaminant occurrence.

UCMR 1 AM was conducted by approximately 3,090 large systems and 797 small systems. Approximately 33,800 samples were collected for each contaminant. In UCMR 2, sampling was conducted by over 3,300 large systems and 800 small systems, and resulted in over 32,000 sample results for each contaminant.

As noted, in addition to AM, SS monitoring was required for contaminants. For UCMR 1, the SS was conducted at 300 PWSs (120 large and 180 small systems) selected at random from the pool of systems required to conduct AM. Samples from the 300 PWSs from throughout the nation provided approximately 2,300 analyses for each contaminant. While the statistical design of the SS is national in scope, the uncertainty in the results for contaminants that have low occurrence is relatively high. Therefore, EPA looked for additional data to supplement the SS data for regulatory determinations.

For the UCMR 2 SS, EPA improved the design to include a census of all systems serving more than 100,000 people (approximately 400 PWSs—but the largest portion of the national population served by PWSs) and a nationally representative, statistically selected sample of 320 PWSs serving between 10,001 and 100,000 people, and 480 small PWSs serving 10,000 or fewer people (72 FR 367, January 4, 2007, USEPA, 2007a). With approximately 1,200 systems participating in the SS, sufficient data were generated to provide a confident national estimate of contaminant occurrence and population exposure. In UCMR 2, the 1,200 PWSs provided more than 11,000 to 18,000 analyses (depending on the sampling design for the different contaminants).

As previously noted, the details of the occurrence data and the results or findings for each of the contaminants considered for regulatory determination is presented in Section IV.B, the contaminant profiles section, and is described in further detail in the support documents for the RD 3 process (USEPA, 2014a and 2014b). The national design, statistical sampling frame, any new analytical methods, and the data analysis approach for the UCMR program has been peer-reviewed at different stages of development (see, USEPA, 2001b, 2008c, 2014d, for example.)

(2) National Inorganics and Radionuclides Survey (NIRS)

EPA conducted the NIRS to provide a statistically representative sample of the national occurrence of 36 selected inorganic compounds (IOCs) and radionuclides in CWSs served by ground water. The sample was stratified by system size and 989 ground water CWSs were selected at random representing 49 States (all except Hawaii) as well as Puerto Rico. 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 selected CWSs was sampled at a single time between 1984 and 1986.

One limitation of the NIRS is a lack of occurrence data for surface water systems. EPA also reviews additional finished water data from State datasets and other sources, as well as data from ambient and source surface waters, to augment the NIRS data. Information about NIRS monitoring and data analysis is available in 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, 2008b).

(3) Disinfection Byproducts Information Collection Rule (DBP ICR)

The DBP ICR (61 FR 24353, May 14, 1996 (USEPA, 1996)) required PWSs serving at least 100,000 people to monitor and collect data on DBPs from July 1997 to December 1998. The DBP ICR data were collected from 296 water systems that provided extensive information on the occurrence of DBPs and on water treatment methods. The DBP ICR data were collected as part of a national project to support development of national disinfection by-products and microbial drinking water standards. EPA used the data to identify national and regional patterns and overall water quality, not to reach system-by-system or treatment plant-by-treatment plant conclusions. Additional details on the data collection process for the DBP ICR, along with an independent analysis of the data, can be found in a report sponsored by the Microbial/Disinfection Products Council (McGuire et al., 2002).

The DBP ICR provided a census of the largest systems that serve the largest proportion of the population served by PWSs at that time. It has previously been vetted for use in regulatory development, and EPA determined it can be used in the regulatory determination process.

b. Supplemental Sources of Finished Drinking and Ambient Water Occurrence Data

The agency evaluates several sources of supplemental information related to contaminant occurrence in finished water and ambient and source waters to augment the primary drinking water occurrence data. Some of these sources were part of other agency information gathering efforts or submitted to the agency in public comment or suggested by stakeholders during previous CCL and Regulatory Determination efforts. These supplemental data are useful to evaluate the likelihood of contaminant occurrence in drinking water and/or to more fully characterize a contaminant's presence in the environment and potentially in source water, and to evaluate any possible trends or spatial patterns that may need further review. The descriptions that follow do not cover all the reports that EPA used. For individual contaminants EPA reviewed additional published reports and peer-reviewed studies that may have provided the results of monitoring efforts in limited geographic areas. A more detailed discussion of the supplemental sources of information/data that EPA evaluated and the occurrence data for each contaminant can be found in the comprehensive regulatory determination support documents (USEPA, 2014a and 2014b).

(1) Individual States' Data

To support the second Six-Year Review of regulated contaminants (see USEPA, 2009b), EPA issued an ICR to collect compliance monitoring data from PWSs for the time period covering 1998–2005. After issuing the ICR, EPA received monitoring data from 45 States plus Region 8 and Region 9 Tribes. Six States and Region 9 Tribes also provided monitoring data for unregulated contaminants along with their compliance monitoring data. EPA further collected additional unregulated contaminant data from two additional States that provide monitoring data through their Web sites. EPA reviews these datasets during the RD 3 process. These datasets vary from State to State in the contaminants included, the number of samples, and the completeness of monitoring. They are reviewed and used to augment the national data and assess if they provide supportive observations or any unique occurrence results that might warrant further review.

(2) Community Water System Survey (CWSS)

EPA periodically conducts the CWSS to collect data on the financial and operating characteristics from a nationally representative sample of CWSs. As part of the CWSS, all systems serving more than 500,000 people receive the survey. In the 2000 and 2006 CWSS, these very large systems were asked questions about the occurrence and concentration of unregulated contaminants in their raw and finished water. The 2000 CWSS (USEPA, 2002a, 2002b) requested data from 83 very large CWSs and the 2006 CWSS (USEPA, 2009c, 2009d) requested data from 94 very large CWSs. Not all systems answered every question or provided complete information on the unregulated contaminants. Because reported results are incomplete, they are illustrative, not representative, and are only used as supplemental information.

(3) United States Department of Agriculture (USDA) Pesticide Data Program (PDP)

Since 1991, the USDA PDP has gathered data on pesticide residues in food. In 2001 the program expanded to include sampling of pesticide residues in treated drinking water, and in 2004 some sampling of raw water was incorporated as well (USDA, 2004). The CWSs selected for sampling tend to be small and medium-sized water surface water systems (serving under 50,000 people) located in regions of heavy agriculture. The sampling frame is designed to monitor in regions of interest for at least two years to reflect the seasonal and climatic variability during growing seasons. PDP works with EPA and the American Water Works Association (AWWA) to identify specific water treatment facilities where monitoring data are collected. The number of sites and samples have varied among different sampling periods. EPA reviewed the PDP data on the occurrence of select contaminants in untreated and treated water (USDA, 2004).

(4) United States Geological Survey (USGS) Pilot Monitoring Program (PMP)

In 1999, USGS and EPA conducted the PMP to provide information on pesticide concentrations in small drinking water supply reservoirs in areas with high pesticide use (Blomquist et al., 2001). The study was undertaken, in part, to test and refine the sampling approach for pesticides in such reservoirs and related drinking water sources. Sampling sites represent a variety of geographic regions, as well as different cropping patterns. Twelve water supply reservoirs considered vulnerable to pesticide contamination were included in the study. Samples were collected quarterly throughout the year and at weekly or biweekly intervals following the primary pesticide-application periods. Water samples were collected from the raw water intake and from the finished drinking water prior to entering the distribution system. At some sites, samples were also collected at the reservoir outflow.

(5) United States Geological Survey (USGS) National Water Quality Assessment (NAWQA)

The USGS instituted the National Water Quality Assessment (NAWQA) program in 1991 to examine ambient water quality status and trends in the United States. The NAWQA program is designed to apply nationally consistent methods to provide a consistent basis for comparisons over time nationally and among significant watersheds and aquifers across the country. These occurrence assessments serve to facilitate interpretation of natural and anthropogenic factors affecting national water quality. The NAWQA program monitors the occurrence of chemicals such as pesticides, nutrients, VOCs, trace elements, and radionuclides, and the condition of aquatic habitats and fish, insects, and algal communities. For more detailed information on the NAWQA program design and implementation, please refer to Leahy and Thompson (1994), Hamilton et al. (2004), and NRC (2002).

The NAWQA program has been designed in ten-year cycles to enable national coverage that can be used for trends and causal assessments. In the Cycle 1 monitoring period, which was conducted from 1991 through 2001, NAWQA collected data from over 6,400 surface water and 7,000 ground water sampling points. Cycle 2 monitoring covers the period from 2002 through 2012, with various design changes from Cycle 1 (see Hamilton et al., 2004).

EPA, with the cooperation of USGS, performed a summary analysis of all Cycle 1 water monitoring data for the CCL 3 and Regulatory Determination process. The surface water data consisted of stream samples; all surface water data were included in the EPA summary analysis. For ground water, all well data were used and data from springs and drainage systems were excluded.

For RD 3, EPA used and evaluated many USGS NAWQA reports to review causal or spatial factors that USGS may have presented in their interpretations. In particular, EPA evaluated many reports from the Pesticide National Synthesis Programs (e.g., Gilliom et al., 2007) and the VOC National Synthesis (e.g., Delzer and Ivahnenko, 2003). While there is overlap in the data used in the USGS reports and the EPA analysis, the USGS reports can provide unique observations related to their synthesis of additional data.

For RD 3, EPA also supplemented these data with information from recent special USGS reports that also used additional data from other programs, particularly reports that focused on contaminant occurrence in source waters for PWSs, such as: Organic Compounds in Source Water of Selected Community Water Systems (Hopple et al., 2009 and Kingsbury et al., 2008), and Water Quality in Public-Supply Wells (Toccalino et al., 2010).

(6) Storage and Retrieval (STORET) Data System

EPA's STORET database contains raw biological, chemical, and physical data from surface and ground water sampling conducted by Federal, State and local agencies, Indian Tribes, volunteer groups, academics, and others. A wide variety of data relating to water quality from all 50 States as well as multiple territories and jurisdictions of the United States are represented in this data system. These are primarily ambient water data, but in some cases they include finished drinking water data. STORET data have quality limitations. There are few restrictions on submission of data based on analytical methods, quality assurance (QA) practices, etc. For more general STORET data information, please refer to: *http://www.epa.gov/storet/index.html*. EPA reviewed STORET ground water data from wells and surface water data from lakes, rivers/streams, and reservoirs.

c. Supplemental Production, Use and Release Data

The agency reviews various sources of information to assess if there are changes or trends in a contaminant's production, use, and release that may affect its presence in the environment and potential occurrence in drinking water. The cancellation of a pesticide or a clear increase in production and use of a contaminant are trends that can inform the regulatory determination process. A more detailed discussion of the supplemental sources of information/data that EPA evaluated and the occurrence data for each contaminant can be found in the comprehensive regulatory determination support documents (USEPA, 2014a and 2014b). Several sources are described in more detail below.

(1) Chemical Update System/Inventory Update Rule (CUS IUR)

The IUR regulation requires manufacturers and importers of certain chemical substances, included on the Toxic Substances Control Act (TSCA) Chemical Substance Inventory, to report site and manufacturing information and the amount of chemicals produced or imported in amounts of 25,000 pounds or more at a single site. Additional information on domestic processing and use must be reported for chemicals produced or imported in amounts of 300,000 pounds or more at a single site. Prior to the 2003 TSCA Amendments (i.e., reporting from 2002 or earlier), information was collected for only organic chemicals that were produced or imported in amounts of 10,000 pounds or more, and was limited to more basic manufacturing information such as production volume. Because of changes in reporting rules, contaminants may have reports for some years but not others (USEPA, 2010a).

(2) Toxic Release Inventory (TRI)

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. 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, 2002c, 2003b). Currently, for most chemicals the reporting thresholds are 25,000 pounds for manufacturing and processing and 10,000 pounds for use. Both the number and type of facilities required to report has increased over time.

Although TRI can provide a general idea of release trends, it has limitations because of the reporting changes over time. Finally, TRI data are meant to reflect ''releases'' and should not be used to estimate general public exposure to a chemical (USEPA, 2002c).

(3) Pesticide Usage Estimates

For the regulatory determinations process, the agency reviews various sources of information about pesticide usage. SDWA directs EPA to consider pesticides in the CCL process. Pesticide use and manufacturing information is considered confidential business information and therefore, accurate measures of production and use are not publically available. As a result, the agency reviews various estimates of use as supplemental information in the deliberative process.

Occasionally, EPA presents estimations of annual U.S. usage of individual pesticides in its pesticide reregistration documents (e.g., Reregistration Eligibility Decisions or (REDs), Interim Reregistration Eligibility Decisions (IREDs), Tolerance Reassessment Progress and Risk Management Decisions (TREDs)). EPA also periodically issues Pesticides Industry Sales and Usage reports. The reports provide contemporary and historical information on U.S. pesticide production, imports, exports, usage, and sales, particularly with respect to dollar values and quantities of active ingredient. The most recent report presents data from the years 2000 and 2001 (USEPA, 2004).

The National Center for Food and Agricultural Policy (NCFAP), a private non-profit institution, has also produced national pesticide use estimates based on USDA State-level statistics and surveys for commercial agriculture usage patterns and State-level crop acreage. The database contains estimates of pounds applied and acres treated in each State for 220 active (pesticide) ingredients and 87 crops. The majority of the chemicals monitored are herbicides, but the database also follows significant numbers of fungicides and insecticides (NCFAP, 2000).

The USGS produced usage estimates and maps for over 200 pesticides used in United States crop production, providing spatial insight to the regional use of many pesticides (USGS, 2007). These pesticide use estimates were generated by the USGS through State-level estimates of pesticide usage rates for individual crops that were compiled by the CropLife Foundation and the Crop Protection Research Institute, combined with county-level data on harvested crop acreage obtained from the 2002 Census of Agriculture.

IV. Contaminant-Specific Discussions for the RD 3 Preliminary Regulatory Determinations

A. Summary of the Preliminary Regulatory Determination

Based on EPA's evaluation of the three SDWA criteria (discussed in section II.B.1), the agency is making preliminary determinations to regulate one contaminant and to not regulate four contaminants. Table 7 summarizes the primary health and occurrence information used to make these preliminary regulatory determinations. Section IV.B of this notice provides a more detailed summary of the information and the rationale used by the agency to reach its preliminary decisions for these five contaminants.

TABLE 7—SUMMARY OF THE HEALTH AND OCCURRENCE INFORMATION AND THE PRELIMINARY DETERMINATIONS FOR THE FIVE CONTAMINANTS CONSIDERED FOR REGULATORY DETERMINATIONS 3

No.	RD 3 contaminants	Health reference level (HRL)	Occurrence findings from primary data sources					Preliminary determination
			Primary 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	Dimethoate	15.4 µg/L	UCMR 2	0% (0 of 4138).	0% (0 of 229M).	0% (0 of 4138).	0% (0 of 229M).	Do not regulate.
2	1,3-Dinitrobenzene.	0.7 µg/L	UCMR 2	0% (0 of 4137).	0% (0 of 229M).	0% (0 of 4137).	0% (0 of 229M).	Do not regulate.
3	Strontium	1,500 µg/L	NIRS	14.3% (141 of 989).	16.6% (246K of 1.5M).	7.0% (69 of 989).	10.7% (158.5K of 1.5M).	Regulate.
4	Terbufos	0.35 µg/L	UCMR 1	0% (0 of 295)	0% (0 of 41M)	0% (0 of 295)	0% (0 of 41M)	Do not regulate.
5	Terbufos Sulfone.	0.35 µg/L	UCMR 2	0.02% (1 of 4138).	0.01% (44.6K of 229M).	0.02% (1 of 4138).	0.01% (44.6K of 229M).	Do not regulate.

B. Contaminant Profiles

This section provides further information on the background, health, and occurrence data that the agency uses to evaluate each of the five candidate contaminants considered for regulatory determinations. For each candidate, the agency evaluates the available human and toxicological data, derives a health reference level, and evaluates the potential and/or likely occurrence and exposed population for the contaminant in public water systems. The agency also considers whether information is available on sensitive populations. The agency uses the findings from these evaluations to determine whether the three SDWA statutory criteria are satisfied. The agency also prepares a regulatory support document (USEPA, 2014b) that provides more details on the background, health, and occurrence information/analyses used to evaluate and make preliminary determinations for these five contaminants.

1. Dimethoate

a. Background

Dimethoate is an organophosphate pesticide, commonly used as an insecticide on field crops (e.g., wheat, alfalfa, corn, and cotton), orchard crops, vegetable crops, and in forestry. Synonyms for dimethoate include dimethogen, dimeton, dimevur, and cygon (HSDB, 2009; USEPA, 2007b). EPA has estimated that the total annual average domestic use of dimethoate is approximately 1.8 million pounds (USEPA, 2007b). EPA's most recent Pesticide Industry Sales and Usage reports indicate that the amount of dimethoate active ingredient (a.i.) used in the United States was between 1 and 2 million pounds in 1999 and 2001, and less than 1 million pounds in 2005 and 2007 (USEPA, 2004: USEPA, 2011f). TRI data from the years 1997 to 2010 show that annual releases to various sources range from tens of pounds to tens of thousands of pounds, with the larger releases occurring only occasionally and in no clear pattern (USEPA, 2012a). For example, reported on-site air emissions were in the range of tens of pounds for 1997–2005 but increased to the range of thousands of pounds in 2006–2010. The only reported non-zero release by underground injection was in 2004 and was over 28,000 pounds. Reported on-site releases to surface water and land were low or non-existent in most years, but peaked suddenly at nearly 20,000 pounds in 1998 (land) and over 2,000 pounds in 2004 (surface water). Dimethoate is considered highly mobile and relatively non-persistent in the environment (USEPA, 2007b).

b. Statutory Criterion #1 (Adverse Health Effects)

Dimethoate meets the SDWA statutory criterion #1 for regulatory determinations; it may have an adverse effect on the health of persons. Dimethoate belongs to a group of pesticides called organophosphates, which share a common MOA. Organophosphates affect the proper function of the nervous system by inhibiting cholinesterase (ChE), an important enzyme involved in neurotransmission. Inhibition of ChE in the brain, plasma, and red blood cells is the most sensitive endpoint described in numerous studies with adult and juvenile animals, following oral, dermal, or inhalation exposures of dimethoate or its primary toxic metabolite omethoate (USEPA, 2007b). As discussed in the 2007 OPP assessment, the U.S. EPA's Cancer Assessment Review Committee (CARC) classified dimethoate as a Group C carcinogen (a possible human carcinogen) in 1991, with concurrence from the FIFRA Scientific Advisory Panel (SAP) on the agency's classification in 1992 (USEPA, 2007b).

The 2007 OPP assessment established a chronic oral RfD for dimethoate of 0.0022 mg/kg/day based on a 2-year feeding study in rats with inhibition of brain ChE as the critical effect (USEPA, 2007b). The RfD was derived using the BMD method and based on the lower 95% confidence limit (BMDL) of 0.22 mg/kg/day, with application of a composite UF of 100 (i.e., intraspecies and interspecies variability). EPA calculated a non-cancer HRL of 15.4 µg/L for dimethoate using the RfD of 0.0022 mg/kg/day for a 70 kg adult ingesting 2 L of drinking water per day and an RSC of 20%. The chronic RfD and subsequent HRL of 15.4 µg/L for dimethoate are considered to be protective of any potential cancer risk or acute ChE effects (USEPA, 1990a, 2007b). The OPP RED (USEPA, 2007b) presents more detailed information about the potential health effects for dimethoate.

c. Statutory Criterion #2 (Occurrence at Frequency and Levels of Public Health Concern)

Dimethoate does not meet the SDWA statutory criterion #2 for regulatory determinations; it does not occur with a frequency and at levels of public health concern in public water systems based on EPA's evaluation of the following occurrence information.

The primary data for dimethoate are recent (2008–2010) nationally-representative drinking water monitoring data, generated through EPA's UCMR 2. Dimethoate was not detected in any of the 32,013 UCMR 2 samples collected by 4,138 PWSs (serving ~ 230 million people) at levels greater than the ½ HRL (7.7 μg/L), the HRL (15.4 μg/L), or the MRL (0.7 μg/L) (USEPA, 2014d).

The State of California reported results from testing more than 20,000 finished drinking water samples from over 2,000 PWSs and dimethoate was detected in two samples from two different PWSs. The detected concentrations (1 μg/L and 2 μg/L) were less than the ½ HRL (7.7 μg/L) and the HRL (15.4 μg/L) (see USEPA, 2014b). The USDA PDP monitored for dimethoate in finished water from 2001 to 2009 and had only two detections in 3,555 samples; both detected concentrations were less than the ½ HRL and the HRL (USDA, 2012). The USGS PMP monitored for dimethoate in finished water in 1999 and had no detections greater than ½ the HRL or the HRL in any of the 221 samples (Blomquist et al., 2001).

Dimethoate occurrence data for ambient water are consistent with those for finished drinking water. The USGS PMP also monitored for dimethoate in ambient water in 1999 and had no detections greater than the ½ HRL (7.7 μg/L) or the HRL (15.4 μg/L) in any of the 317 samples (Blomquist et al., 2001). Ambient water data from a two-phase USGS study conducted between 2002 and 2005 by Hopple et al. (2009) and Kingsbury et al. (2008) reported no detections in the 221 Phase 1 groundwater samples. Only two detections were reported from 146 Phase 1 surface water samples at nine PWSs. The highest concentration detected was 0.009 μg/L, which is less than the ½ HRL and the HRL. In Phase 2, there were no detections of dimethoate from 48 raw and finished water groundwater samples (Hopple et al., 2009; Kingsbury et al., 2008). Ambient water data in STORET included no measured results above 0.44 μg/L in 5,299 samples from 798 sites (USEPA, 2012b). Ambient water data reported by the California Department of Pesticide Regulation included no measured results above 2.4 μg/L (USEPA, 2007b).

d. Statutory Criterion #3 (Meaningful Opportunity)

EPA finds that dimethoate does not meet the SDWA statutory criterion #3 for regulatory determinations; regulation of dimethoate does not present a meaningful opportunity health risk reduction for persons served by PWSs based on the estimated exposed population, including sensitive populations. The estimated population exposed to dimethoate at levels of public health concern is 0%; it was not found to occur at levels above the HRL (or the ½ HRL) in 4,138 PWSs and 32,013 samples from the UCMR 2 monitoring. In addition, other supplementary sources of finished water and ambient water data indicate that the occurrence of dimethoate in PWSs is likely to be low to non-existent. As a result, the agency finds that an NPDWR for dimethoate does not present a meaningful opportunity for health risk reduction.

EPA also evaluated whether health information is available regarding the potential health effects on children and other sensitive populations. The database for dimethoate includes a 3-generation reproductive study in mice, developmental (teratology) studies in rats and rabbits, and a neurodevelopmental toxicity study (USEPA, 1990a, 2007b). The critical effect of ChE inhibition is a more sensitive endpoint compared to the reproductive and developmental endpoints (USEPA, 2007b); therefore no sensitive populations were identified or characterized. The OPP RED (USEPA, 2007b) presents more detailed information about the potential health effects and sensitive populations for dimethoate.

e. Preliminary Regulatory Determination

The agency is making a preliminary determination to not regulate dimethoate with an NPDWR after evaluating health, occurrence, and other related information against the three SDWA statutory criteria. While data suggests that dimethoate may have an adverse effect on human health, the occurrence data indicate that dimethoate is not occurring or not likely to occur in PWSs with a frequency and at levels of public health concern. Therefore, the agency finds that an NPDWR would not present a meaningful opportunity to reduce health risk for persons served by PWSs. The Regulatory Determinations 3 Support Document (USEPA, 2014d) and the Occurrence Data from the Second Unregulated Contaminant Monitoring Regulation (UCMR 2) (USEPA, 2014a) present additional information and/or analyses supporting the agency's evaluation of dimethoate.

2. 1,3-Dinitrobenzene

a. Background

1,3-Dinitrobenzene is a nitro aromatic compound that is used as an industrial chemical and formed as a by-product in the manufacture of munitions as well as in the production of other substances (HSDB, 2009). There are no known natural sources of 1,3-dinitrobenzene. Annual production and importation of 1,3-dinitrobenzene in the United States was last reported by CUS–IUR in 1986 to be between 10–50 million pounds (USEPA, 2010b). TRI data indicate 19,858 pounds were released to the environment by industry in 2008 and 10,595 pounds in 2010 (USEPA, 2012a). 1,3-dinitrobenzene appears to be moderately persistent in environmental media and moderately mobile in soil and water, though in soils with high clay content it will be less mobile (USEPA, 2014b).

b. Statutory Criterion #1 (Adverse Health Effects)

1,3-dinitrobenzene meets the SDWA statutory criterion #1 for regulatory determinations; it may cause adverse effect on the health of persons. 1,3-dinitrobenzene has demonstrated adverse health effects in many rodent and occupational studies. Occupational studies indicate that methemoglobinemia, hemolytic anemia, and cyanosis are seen in workers who experience an acute reaction to 1,3-dinitrobenzene (Hajjar et al., 1992). The EPA IRIS assessment (USEPA, 1990b) of the carcinogenicity of 1,3-dinitrobenzene currently lists it as Group D (not classifiable as to human carcinogenicity).

The primary adverse biological effects from exposure to 1,3-dinitrobenzene are on red blood cells, spleen, and testes. The RfD for 1,3-dinitrobenzene is 0.0001 mg/kg/day (Cody et al., 1981). The RfD was derived from a NOAEL of 0.4 mg/kg/day in a subchronic oral study in rats where increased spleen weight was identified as the critical effect (Cody et al., 1981). A composite UF of 3,000 (intraspecies variability, interspecies variability, subchronic to chronic duration, and lack of chronic, developmental, and multigenerational reproductive toxicity studies) was applied to the NOAEL to obtain the RfD. EPA calculated a non-cancer HRL of 0.7 µg/L for 1,3-dinitrobenzene using the RfD of 0.0001 mg/kg/day for a 70 kg adult ingesting 2 L of drinking water per day and an RSC of 20%.

The current EPA oral RfD value is supported by a more recent 1,3-dinitrobenzene assessment that was conducted by ATSDR, in which an oral intermediate duration minimal risk level of 0.0005 mg/kg/day for splenic hemosiderosis in male rats was established using a LOAEL of 0.54 mg/kg/day (Linder et al., 1986; dose adjusted for a 7-day/week exposure) and a composite UF of 1,000 (intraspecies variability and interspecies variability, LOAEL to NOAEL). Based on EPA assumptions and a composite UF of 3,000 (intraspecies variability, interspecies variability, LOAEL to NOAEL and subchronic to chronic duration) applied to the LOAEL of 0.54 mg/kg/day, the resultant HRL value of 1 µg/L supports the HRL value of 0.7 µg/L derived from the IRIS RfD (ATSDR, 1995). The IRIS assessment (USEPA, 1990b) presents more detailed information about the potential health effects for 1,3-dinitrobenzene.

c. Statutory Criterion #2 (Occurrence at Frequency and Levels of Public Health Concern)

1,3-dinitrobenzene does not meet the SDWA statutory criterion #2 for regulatory determinations; it does not occur with a frequency and at levels of public health concern in public water systems based on EPA's evaluation of the following occurrence information.

The primary data for 1,3-dinitrobenzene are recent (2008–2010) nationally-representative drinking water monitoring data generated through EPA's UCMR 2 (USEPA, 2014d). UCMR 2 is the only dataset with finished water data for this contaminant. UCMR 2 collected 32,017 samples from 4,137 PWSs and 1,3-dinitrobenzene was not detected above the MRL (0.8 µg/L), which is only slightly higher than the HRL (0.7 µg/L).

Findings from the available ambient water data for 1,3-dinitrobenzene are consistent with the results in finished water. Ambient water data in STORET included no measured results above 0.33 µg/L in 143 samples from 70 sites (USEPA, 2012b). It should be noted that some occurrence above the HRL may have gone undetected since reporting levels are not documented.

d. Statutory Criterion #3 (Meaningful Opportunity)

EPA finds that 1,3-dinitrobenzene does not meet the SDWA statutory criterion for regulatory determinations; regulation of 1,3-dinitrobenzene does not present a meaningful opportunity for health risk reduction for persons served by PWSs based on the estimated exposed population, including sensitive populations. The estimated population exposed to 1,3-dinitrobenzene at or above the MRL is 0%; it was not found to occur in finished drinking water at levels > MRL (0.8 µg/L), which is only slightly higher than the HRL (0.7 µg/L), in 32,017 samples and 4,137 PWSs from the UCMR 2 monitoring. As a result, the agency finds that an NPDWR for 1,3-dinitrobenzene does not present a meaningful opportunity for health risk reduction.

EPA also evaluated whether information is available regarding the potential health effects on children and other sensitive populations. Individuals with a genetic predisposition to methemoglobinemia (estimated prevalence in the general population = 1% or 1 per 100) and/or hemosiderosis, neonates, and those co-exposed to other hemolytic agents, could be more sensitive to exposure to 1,3-dinitrobenzene (ATSDR, 1995; Jaffe and Hultquist, 1989). Males having sperm production complications could also have increased sensitivity to 1,3-dinitrobenzene exposure (Hajjar et al., 1992). There is currently no multigenerational animal study available for 1,3-dinitrobenzene, and no data available from studies of 1,3-dinitrobenzene developmental toxicity (Hajjar et al., 1992). However, the RfD incorporated a UF for this database deficiency. The IRIS assessment (USEPA, 1990b) presents more detailed information about the potential health effects and sensitive populations for 1,3-dinitrobenzene.

e. Preliminary Regulatory Determination for 1,3-dinitrobenzene

The agency is making a preliminary determination to not regulate 1,3-dinitrobenzene with an NPDWR after evaluating health, occurrence, and other related information against the three SDWA statutory criteria. While data suggest that 1,3-dinitrobenzene may have an adverse effect on human health, the occurrence data indicate that 1,3-dinitrobenzene is not occurring or not likely to occur in PWSs with a frequency and at levels of public health concern. Therefore, the agency has determined that an NPDWR for 1,3-dinitrobenzene would not present a meaningful opportunity to reduce health risk for persons served by PWSs. The Regulatory Determinations 3 Support Document (USEPA, 2014b) and the Occurrence Data from the Second Unregulated Contaminant Monitoring Regulation (UCMR 2) (USEPA, 2014d) present additional information and analyses supporting the agency's evaluation of 1,3-dinitrobenzene.

3. Strontium

a. Background

Strontium is a naturally occurring element (atomic number 38) and a member of the alkaline earth metals (ANL, 2007). There are several radioactive strontium isotopes formed by nuclear fission of uranium or plutonium. The best known is ^{90}Sr, a legacy from above ground testing of the atomic bomb (half-life 29 years). Since drinking water contamination by radioactive isotopes, including beta particle emitters, is covered under the existing radionuclides rule, this FR notice deals primarily with the stable ^{88}Sr isotope which represents 83% of total environmental strontium (ATSDR, 2004).

Strontium mineral mining ceased in the United States in 1959. The United States imports both strontium minerals for refining and refined strontium containing compounds (USGS, 2009). Imports of strontium minerals and compounds were approximately 31,000 to 38,500 metric tons from 1994 to 2001 and have declined since 2001 (ATSDR, 2004; USGS, 2009). In the United States, total consumption of strontium minerals and compounds was 16,700 metric tons of strontium content in 2004 and approximately 7,750 metric tons in 2008 (USGS, 2009).

Historically, the most important commercial use of strontium has been in the faceplate glass of cathode-ray tube televisions to block x-ray emissions (ATSDR, 2004). Conversely, flat panel televisions incorporating LCD or Plasma displays are not capable of emitting x-radiation; therefore, they do not require strontium (FDA, 2011). As flat panel technology has become widespread in the United States in the last decade, demand for strontium for this application has fallen (USGS, 2009). In 2008, approximately 30% of commercial strontium consumption was in pyrotechnics and signals (as strontium nitrate and other compounds), 30% in ferrite ceramic magnets (as strontium ferrite), 10% in master alloys (as strontium metal), 10% in pigments and fillers (as strontium chromate), 10% in electrolytic production of zinc (as strontium carbonate), and 10% in other applications such as fluorescent lights (strontium phosphate), toothpaste (strontium chloride), and medicines (strontium chloride and strontium peroxide). The feed material for most applications is strontium carbonate (ATSDR, 2004; HSDB, 2010; USGS, 2009). Strontium can exist in oxidation states 0 and +2; under normal environmental conditions it is found in the +2 oxidation state in various ionic or salt forms. Strontium is considered to have moderate or moderate-to-low mobility in soils. The mobility of strontium in water can increase with increased salt concentrations due to a decrease in sorption to sediments (USEPA, 20104b). The Regulatory Determination 3 Support Document (USEPA, 2014b) for this notice presents more detailed background information on strontium.

b. Statutory Criterion #1 (Adverse Health Effects)

Strontium meets the SDWA statutory criterion #1 for regulatory determinations; it may have an adverse effect on the health of persons. The primary target of strontium exposure is the bone. The chemical similarity of strontium to calcium allows it to exchange imperfectly for calcium in a variety of biological processes; the most important of these is the substitution of calcium in bone, affecting skeletal development. Due to the MOA for strontium toxicity, strontium uptake into bone is affected by the intake of nutrients related to bone formation, such as calcium, phosphorous, and vitamin D (Clarke, 2008; Grynpas and Marie, 1990; Marie et al., 1985). The decreased calcification in bones results in increased width of the epiphyseal cartilage, changes in the pattern of calcification, abnormally long metaphyses, reduction in bone mineralization, and active osteoclasts in young rats (Marie and Hott, 1986; Matsumoto, 1976; Neufeld and Boskey, 1994; Storey, 1961). Due to this effect on growing bones, infants, children, and adolescents are of particular concern as a sensitive population.

A study based on decreased bone calcification rate in male weanling rats (i.e., comparable to the sensitive time period in humans), which administered strontium chloride in drinking water for nine weeks (Marie et al., 1985), was identified by EPA as the critical study for RfD determination. The RfD was established by using the BMD method and based on the lower 95% confidence limit (BMDL) of 328 mg/kg/day for a one standard deviation decrease in bone calcification compared to control. Using a composite UF of 1000 (10 for intraspecies variability, 10 for interspecies variability, and 10 for database uncertainties) the RfD for strontium is calculated to be 0.3 mg/kg/day. This RfD is supported by additional studies reporting bone effects on weanling rats at similar dose levels (Grynpas and Marie, 1990; Storey, 1961). EPA calculated a non-cancer HRL of 1500 µg/L for strontium using the RfD of 0.3 mg/kg/day, a default RSC of 20% and age-specific exposure factors (i.e., drinking water intake expressed as liters per kg of body weight) for the sensitive population of birth through 18 years to reflect the most active period of bone growth and development (see section IV.B.10.d.3).

EPA released an IRIS assessment for strontium in 1992 and developed an RfD of 0.6 mg/kg/day based on the Storey, 1961 study. The IRIS assessment was completed before the 1998 changes to the IRIS program wherein the agency develops and peer reviews a detailed Toxicological Review before posting an IRIS summary. The point of departure for the 1992 IRIS RfD of 0.6 mg/kg/day is a NOAEL of 190 mg Sr/kg-day with a composite UF of 300 (10 for interspecies variability, 3 for intraspecies variability, and 10 for database uncertainties). This would yield an HRL of 3000 µg/L, using the same age-specific exposure adjustment factors described above. If the age-specific exposure adjustment factors were not used, the HRL would be 2000 µg/L based on the OW assessment, or 4000 µg/L based on the IRIS assessment. As noted in section III.C.1.c, EPA evaluates the existing data and performs a literature search for studies published after the available health assessment is completed to determine if new information suggests a different outcome. In cases where the review suggests that a change the existing RfD or cancer assessment is needed, the EPA Office of Water prepares and independently peer-reviews an OW Assessment of the data. In the case of strontium and because newer information provided additional support for the 1985 Marie et al. study, EPA chose to use the BMDL of 328 mg/kg/day from Marie et al., (over the 1961 Storey study) for the following reasons: (a) Marie et al., (1985) reported the doses rather than estimated the doses; (b) the study duration was longer (63 days for Marie as compared to 20 days for Storey); (c) the monitoring of the bone effects for Marie et al., (1985) was more quantitative than the photomicrographs evaluated by Storey (1961); (d) dosing was provided via drinking water, the medium of interest (rather than a weakly soluble $SrCO3$ in the diet); (e) Marie et al., (1985) reported the strain of rats and the age of the animals, at the time that dosing was initiated and completed; (f) the data were amenable to dose-response modeling to identify the BMD and BMDL; and (g) the dietary calcium provided in the Storey study was three times higher than that in the Marie study, making those rats less at risk due to the calcium and strontium competition for uptake, as verified by a comparison of serum data from the two studies. The OW assessment uses a 10x uncertainty factor for intraspecies variability, rather than the 3x factor used in the 1992 IRIS assessment because it is not clear if the window of vulnerability was adequately captured, since the weanling rats were exposed only for 28–63 days, a period that did not include exposure during gestation, lactation, and through young adulthood. EPA requests comment on its revised RfD calculation and on its proposal to use the OW assessment in lieu of the RfD from the 1992 IRIS assessment.

There is inadequate information to assess the carcinogenic potential of strontium due to the lack of adequate studies of chronic duration. The Health Effects Support Document (USEPA, 2014c) for this determination presents more detailed analysis of the health effects of strontium.

c. Statutory Criterion #2 (Occurrence at Frequency and Levels of Public Health Concern?)

Strontium meets the SDWA statutory criterion #2 for regulatory determinations; it does occur with a frequency and at levels of public health concern in public water systems based on EPA's evaluation of the following occurrence information.

EPA used the National Inorganics and Radionuclides Survey (NIRS) (USEPA, 2008b) as the primary data source to evaluate the occurrence of strontium in PWSs. It provides contaminant occurrence data from 989 CWSs served by ground water sources. Each of these randomly selected PWSs was sampled a single time between 1984 and 1986. Of the 989 systems in NIRS, 980 (99%) had detectable levels of strontium ranging from 1.53 to 43,550 µg/L. The mean concentration was 603 µg/L. Approximately 7.0% (69 of 989) of the NIRS PWSs detected strontium at a level greater than the HRL (1500 µg/L) and 14.3% (141 of 989) detected strontium at a level greater than ½ HRL (750 µg/L). Extrapolated by the total number of ground water CWSs found nationally, this represents 2,798 and 5,718 groundwater CWSs that could have strontium at a level greater than the HRL and the ½ HRL, respectively. These figures are summarized in Table 8 (USEPA, 2014b).

TABLE 8—ESTIMATES OF POPULATION EXPOSED TO STRONTIUM, OBSERVED AND EXTRAPOLATED FROM NIRS

Threshold	National Inorganics and Radionuclides Survey (NIRS)		Extrapolation of NIRS data to groundwater systems nationwide	
	Systems	Population	Systems	Population
Systems with Detectable Concentrations	99.1% (980 of 989)	99.9% (1.481M of 1.482M)	99.1% (39.7K of 40.1K)	99.9% (93.0M of 93.1M)
Systems detecting strontium above one half the HRL (>750 µg/L)	14.3% (141 of 989)	16.6% (246K of 1.5M)	14.3% (5.7K of 40.1K)	16.6% (15.4M of 93.1M)
Systems detecting strontium above the HRL (>1500 µg/L)	7.0% (69 of 989)	10.7% (159K of 1.5M)	7.0% (2.8K of 40.1K)	10.7% (10.0M of 93.1M)

As a point of reference to the earlier IRIS assessment, if EPA used the HRL derived from this assessment of 3000 µg/L, 30/989 systems (3%) would have finished water samples that exceed the HRL using the NIRS data, compared to 69/989 (7%) using the HRL of 1500 µg/L derived from the more recent OW assessment.

Finished water data, analyzed between 1998 and 2005, from Ohio and Illinois are also consistent with the NIRS data. The State of Illinois reported results from testing 21 drinking water samples from 19 PWSs and strontium was detected in all 21 samples (100%) from all 19 systems (100%). Approximately 23.8% (5 of 21) of samples from five systems (26.3%) had strontium at levels greater than the ½ HRL (750 µg/L) and approximately 23.8% (5 of 21) of samples from five systems (26.3%) had strontium at levels greater than the HRL (1500 µg/L) (USEPA, 2012b). The State of Ohio reported results from testing 77 samples from 32 PWSs and strontium was detected in 75 samples (97.4%) from 30 different systems (93.8%). Approximately 27.3% (21 of 77) of samples from 10 systems (31.3%) had strontium at levels greater than the ½ HRL and approximately 23.4% (18 of 77) of samples from seven systems (21.9%) had strontium at levels greater than the HRL (USEPA, 2014b).

Although there are limited surface water data available for strontium, the available data are consistent and demonstrate high occurrence in surface waters. Ambient water data for strontium are also consistent with high occurrence in finished water, which is expected since it is a naturally occurring element. The NAWQA Quality of Public Supply Wells (Toccalino et al., 2010) study collected water samples from source (untreated) groundwater public supply wells in 41 states. Each well was sampled once from 1993–2007 and 100% of samples (503 of 503) had a strontium detection. Of the detections, 25.1% (126 of 503) were above the ½ HRL (750 µg/L) and 12.1% (61 of 503) were above the HRL (1500 µg/L). Additional occurrence information on strontium can be found in the Regulatory Determinations 3 Support Document (USEPA, 2014b).

d. Statutory Criterion #3 (Meaningful Opportunity?)

EPA makes a preliminary finding that strontium meets the SDWA statutory criterion #3 for regulatory determinations; regulation of strontium in drinking water presents a meaningful opportunity for health risk reduction based on the estimated exposed population, potential impacts on sensitive populations and estimated exposure from other sources (e.g., food).

1. National Population Exposed: In the NIRS dataset 989 ground water systems were sampled serving a population of 1.48 million. The NIRS data indicates that the population exposed to strontium at a level greater than the HRL (1500 µg/L) is 158,557 (11%) and the ½ HRL (750 µg/L) is 245,870 (17%) (USEPA, 2012b). EPA also performed national extrapolations generated by multiplying the NIRS findings of system/population percentages and the national system/population inventory numbers for PWSs developed from EPA's Safe Drinking Water Information System, the CWSS, and UCMR. Out of the 93.1 million people served by 40,106 ground water CWSs in the nation, the national extrapolation indicates that 10.0 million may be exposed to concentrations greater than the HRL (1500 ug/L) and 15.4 million may be exposed to concentrations greater than the ½ HRL (750 ug/L). The system and population estimates are summarized in Table 8.

Strontium occurs naturally and is abundant in the environment. Its occurrence in water at concentrations >HRL may be a reflection of the geologic and geochemical setting of the source waters for PWSs. The NIRS drinking water data showed that strontium was detected in one or more systems sampled in all 48 continuous states, Alaska and Puerto Rico (Hawaii was not included in NIRS). The occurrence data (e.g., NIRS) show that PWSs with strontium at concentrations greater than the HRL and the ½ HRL occur in 26 states and 34 states, respectively (USEPA, 2014b).

2. Exposure from media other than water: EPA has determined that there is a meaningful opportunity to regulate strontium in drinking water to reduce the public's overall exposure after evaluating the available exposure data from media other than water. Although strontium is known to occur in food, air, and soil, data on levels in those media are limited as are estimates of intake from those sources. Therefore, EPA used the default 20% RSC to calculate the HRL. This section provides a summary of the available exposure data.

An FDA Total Diet Study by Pennington and Jones (1987) collected 234 individual foods in 1984 from three cities in one region of the country and indicated dietary intakes of 493 µg/day for young children (6 to 11 months), 928 to 1,388 µg/day for 14 to 16 year old adolescents, and 979–1,489 µg/day for adults. The FDA Total Diet Study foods are prepared with distilled water and do not reflect any contributions from the cooking water during preparation of foods that absorb water such as rice and pasta. Thus, the strontium in many foods will be impacted by the strontium levels in the local water supply. Using the mean of the detected water concentrations from the NIRS dataset (603 µg/L), the estimated water intake for young children (90th percentile water intake of 1L/day) is 603 µg/day and 1,206 µg/day for adults (90th percentile water intake of 2L/day). The estimated strontium intakes from air and soil are very low compared with those from food and drinking water. The estimated air exposure for children is 0.1 µg/day and for adults is 0.3 µg/day (Dzubay and Stevens, 1975). The estimated exposure from soil is 24 µg/day for children and 12 µg/day for adults (Shacklette and Boerngen, 1984). No data were identified on consumer products, such as toothpaste that contain strontium as an ingredient or impurity.

3. Sensitive populations: Children are expected to be a sensitive population, since they are actively growing and strontium can substitute for calcium in growing bone. This means that changes in bone structure and homeostasis may have more severe and/or a long-term impact than similar changes in adults. These effects would be expected to have the greatest impact during periods of rapid growth in the developing fetus, during childhood and adolescence, particularly if their calcium intake is insufficient (Abrams et al., 2000; Lee et al., 1996; Matkovic et al., 2005; Storey, 1961). The estimated populations of pregnant women (and thus fetuses) and of children (<17 years old) are 6 and 75 million, respectively (O'Day et al., 1998). The RfD was based on changes in bone growth in weanling rats (i.e., the sensitive population). As a result, the data do not include the risk during prenatal development and lactation so these factors were considered when selecting the UFs used to derive the RfD. Age-specific exposure factors (USEPA, 2012c) were also used to reflect the sensitive population (birth through 18 years) in derivation of the HRL. Exposures from drinking water at or below the HRL (1500 µg/L) are expected to be protective of the sensitive population, assuming that 80% of exposure comes from other sources such as air, soil and food.

The toxic effects of strontium result from strontium ions substituting for calcium ions, therefore calcium deficiency would be expected to result in increased risk among sensitive populations. In this respect, it is important to note that recent NHANES data indicate that about 50% of females, nine years and older, fail to receive adequate calcium from diet and supplements on a daily basis (IOM, 2010). Groups with higher risks of becoming calcium deficient include: Adolescent girls, postmenopausal women, amenorrheic women, female athletes, vegans, and individuals with lactose intolerance or cow's milk allergies (IOM, 2010; NIH, 2011a).

The major route of elimination of strontium is via the kidneys, therefore individuals with impaired renal function are another sensitive population. This population may potentially have impaired strontium clearance, as has been shown in renal failure patients. There are approximately 20 million people (10%) above the age of 20 with chronic kidney disease (CDC, 2010) and 548,000 people with kidney end-stage renal disease (USRDS, 2010), who may be at an increased risk. People with disorders affecting the normal equilibrium between the breakdown of old bone and the formation of new bone (such as Paget's disease) might also be sensitive to strontium exposure (D'Haese et al., 1999, 2000; Schrooten et al., 1998, 2003; Tothill et al., 1983). According to the National Institute of Arthritis and Musculoskeletal and Skin Diseases, there are approximately 1 million people (1.2 people per 100 men and women age 45 to 74) diagnosed with Paget's disease of the bone (NIH, 2011b). The Health Effects Support Document (USEPA, 2014c) for strontium presents more detailed information about the potential health effects and sensitive populations. Because the RfD includes an uncertainty factor of 10 for intraspecies variability, the RfD is also expected to be protective of these sensitive populations.

d. Preliminary Regulatory Determination

At this time, the agency is making a preliminary determination to regulate strontium with an NPDWR after evaluating the available health, occurrence, and other related information against the three SDWA statutory criteria. Specifically, it is EPA's preliminary determination that (a) strontium may have an adverse effect on the health of persons, (b) it is known to occur or there is substantial likelihood that strontium will occur in public water systems with a frequency and at levels of public health concern, (c) regulation of strontium with an NPDWR presents a meaningful opportunity to reduce health risks for persons served by PWSs.

It is important to note that the agency included strontium in UCMR 3. As of January 2014, a preliminary analysis (USEPA, 2014e) of the first nine months of the UCMR 3 monitoring data indicate that 4.9% (70 of 1,423) of systems, 3.8% (175 of 4,547) of entry points, and 3.9% (274 of 7,061) of samples have detects of strontium at levels greater than the HRL of 1500 µg/L. While EPA believes the occurrence data from NIRS (in concert with the supplemental information discussed earlier) are sufficient to make the regulatory determination, the agency believes the additional monitoring results from UCMR 3 will assist EPA in making the final regulatory determination for strontium and in developing the proposed NPDWR. As noted in section III.A.3, this regulatory determination process is distinct from the more detailed analyses needed to develop a national primary drinking water regulation. To inform the agency, the EPA plans to conduct more extensive field testing of treatment technologies to assess the effectiveness of strontium removal in PWSs prior to promulgating a national primary drinking water regulation. Thus a decision to regulate is the beginning of the agency's regulatory development process, not the end. As the agency collects additional information about drinking water and other sources of exposure (and performs more detailed analyses), this information will inform the agency's opinion as to whether strontium should be regulated. The agency asks the public to submit any data or information that may be useful in evaluating drinking water and other sources of exposure (e.g., food, food prepared in drinking water, air, soil, etc.).

4 and 5. Terbufos and Terbufos Sulfone

a. Background

Terbufos is a phosphorodithioate pesticide (i.e., an organophosphate) used as an insecticide-nematicide to control a variety of insect pests, primarily used on corn and sugar beets (USEPA, 2006c). Terbufos sulfone is a degradate of terbufos. EPA's most recent Pesticide Industry Sales and Usage report states that between 5 and 7 million pounds of terbufos active ingredient were used in 1999 and between 3 and 5 million pounds of active ingredient were used in 2001 (USEPA, 2004). There are no industrial release data available for terbufos from TRI. As a pesticide degradate, terbufos sulfone is neither produced nor used commercially. Total toxic residues of terbufos and degradates are highly mobile and persistent in the environment, with terbufos sulfone being more mobile and substantially more persistent than terbufos (USEPA, 2006c).

b. Statutory Criterion #1 (Adverse Health Effects?)

Terbufos and its degradate, terbufos sulfone, meet the SDWA statutory criterion #1 for regulatory determinations; they may cause an adverse effect on the health of persons. Terbufos and terbufos sulfone belong to a group of pesticides called organophosphates, which share a common mechanism of toxicity. Organophosphates affect the proper function of the nervous system by inhibiting ChE, an essential enzyme in neurotransmission. There has been no evidence that terbufos is carcinogenic in animal studies (Rapp, 1974; Silverman et al., 1986) and it is classified as a class D carcinogen (inadequate evidence of carcinogenicity) (USEPA, 1988). Overall, health effects information for the terbufos sulfone degradate is lacking; there are no long-term studies or cancer classification for terbufos sulfone.

The 2006 OPP RED assessment (USEPA, 2006c) established an oral RfD for terbufos of 0.00005 mg/kg/day, derived from the NOAEL of 0.005 mg/kg/day for ChE inhibition in the 28-day and 1-year dog studies by Shellenberger (1984) and Shellenberger and Billups (1986). A composite UF of 100 (interspecies and intraspecies variability) was applied to the NOAEL to obtain the RfD. EPA calculated a non-cancer HRL of 0.35 µg/L for terbufos using the RfD of 0.00005 mg/kg/day for a 70 kg adult ingesting 2 L of drinking water per day and an RSC of 20%. The agency has not developed an RfD for terbufos sulfone because subchronic and chronic studies are not available. However, Bailey (1988) conducted a 14-day study of both terbufos and its sulfone degradate in dogs. The NOAEL based on ChE activity for terbufos sulfone was greater than the LOAEL of 2.5 mg/kg/day for the same endpoint following 14-day dosing with the parent compound terbufos. This suggests that the terbufos sulfone degradate is less toxic than its parent, and that the use of the terbufos HRL of 0.35 µg/L for the degradate, terbufos sulfone, is acceptable. The OPP RED (USEPA, 2006c) presents more detailed information about the health effects for terbufos and terbufos sulfone.

c. Statutory Criterion #2 (Occurrence at frequency and levels of public health concern?)

Terbufos and terbufos sulfone do not meet the SDWA statutory criterion #2 for regulatory determinations; they do not occur with a frequency and at levels of public health concern in public water systems based on EPA's evaluation of the following occurrence information.

The primary data for terbufos sulfone are nationally-representative finished water monitoring data generated through EPA's UCMR 2 (2008–2010) (USEPA, 2014d). UCMR 2 collected 32,012 finished water samples from 4,138 PWSs (serving ~ 230 million people) and terbufos sulfone was detected in only one sample, at a concentration of 0.42 µg/L. The MRL was 0.4 µg/L, which is slightly higher than the HRL (0.35 µg/L) (USEPA, 2012d). The primary data for terbufos are from the UCMR 1 screening survey (2001–2003) (USEPA, 2008c). The UCMR 1 screening survey collected 2,301 finished water samples from 295 PWSs. Terbufos was not detected at levels at or above the MRL (0.5 µg/L), which is slightly higher than the HRL (0.35 µg/L) (USEPA, 2008c). Finished water data for terbufos and terbufos sulfone from California, Iowa, USDA, and USGS are also consistent with the UCMR 1 and UCMR 2 data. The State of California reported no detections of terbufos in 191 samples from 23 PWSs (see USEPA, 2014b). The State of Iowa reported no detections of terbufos sulfone from 13 wells (see USEPA, 2014b). The USDA PDP monitored for terbufos (2,597 samples) and terbufos sulfone (2,923 samples) in finished water from 2001 to 2009 and reported no detections at or above method reporting levels ranging from 0.005 µg/L to 0.1 µg/L (USDA, 2012: USEPA, 2014b). The USGS PMP monitored for terbufos in finished water in 1999 and reported no detections, at or above their method reporting level of 0.013 µg/L (Blomquist et al., 2001).

Terbufos and (very limited) terbufos sulfone occurrence data for ambient water from EPA, STORET, and several USGS programs or studies are consistent with those for finished water. The USGS NAWQA Program (1992–2001) reported no groundwater detections above the ½ HRL (0.175 µg/L) or the HRL (0.35 µg/L) for terbufos in 20,960 samples at 7,118 sites. NAWQA reported surface water detections for terbufos in 28 of 14,480 samples (0.19%) at 20 of 1,907 sites (1.05%). Of the 28 surface water detections for terbufos, only four samples (0.03%) at four sites (0.21%) were above the ½ HRL and only one sample (0.01%) at one site (0.05%) was above the HRL (Gilliom et al., 2007). The NAWQA Carbonate Aquifer Study (1993–2005; Lindsey et al., 2008) and the NAWQA Domestic Well Water Quality Study (1991–2004; DeSimone, 2009) reported no detections for terbufos above the ½ HRL or the HRL in 1,027 and 2,539 samples, respectively. The NAWQA National Synthesis Program (1992–2001) reported no groundwater detections for terbufos above the ½ HRL or the HRL and one surface water detection (0.56 µg/L), from agricultural sites, above the HRL (Gilliom et al., 2007).

Ambient water data from a two phase USGS study conducted between 2002 and 2005 by Hopple et al. (2009) and Kingsbury et al. (2008) reported no terbufos detections in the 221 Phase 1 groundwater samples nor the 146 Phase 1 surface water samples. In Phase 2, there were no detections of terbufos from 48 raw and 48 finished groundwater samples. Ambient water data from a USGS study conducted between 1993 and 2007 by Toccalino et al. (2010) reported no terbufos detections in 898 groundwater samples.

Terbufos ambient data reported in EPA's OPP RED for Terbufos (USEPA, 2006c) document included 20 detections in 4,563 groundwater samples from 13 States. The detections ranged from 0.01 to 20 µg/L, a range that extends both above and below the ½ HRL (0.175 µg/L) and the HRL (0.35 µg/L). The USGS PMP monitored for terbufos in ambient water in 1999 and reported no detections (Blomquist et al., 2001).

Terbufos ambient data are reported in STORET from 17 States (USEPA, 2012b). No groundwater detections were reported in 699 samples at 441 sites. STORET reported surface water detections in 457 of 5,826 samples (7.84%) at 138 of 625 sites (22.1%). Of the 457 surface water detections, only 23 samples (0.39%) at 14 sites (2.24%) were above the ½ HRL and only two samples (0.03%) at two sites (0.32%) were above the HRL.

d. Statutory Criterion #3 (Meaningful Opportunity?)

Terbufos and terbufos sulfone do not meet the SDWA statutory criterion #3 for regulatory determinations; regulation of terbufos and terbufos sulfone do not present a meaningful opportunity for health risk reduction based on the estimated population exposed, including sensitive populations. The estimated population exposed to terbufos at or above the MRL is 0%; the compound was not found to occur in finished water at levels greater than or equal to the MRL (0.4 µg/L), which is slightly higher than the HRL (0.35 µg/L), in 2,301 samples from 295 PWSs in UCMR 1 (USEPA, 2008c). The estimated population exposed to terbufos sulfone at a level of public health concern (based on the HRL for terbufos) is 44,600 (0.02% of the population served by PWSs); there was only one detection greater than the HRL in 4,138 PWSs (1 of 32,012 samples in UCMR 2) (USEPA, 2014d). As a result, the agency finds that an NPDWR does not present a meaningful opportunity for health risk reduction.

EPA also evaluated whether health information is available regarding the potential health effects on children and other sensitive populations. Developmental studies with terbufos in rats and rabbits did not find any developmental effects (USEPA, 2003c). There are no data on reproductive and developmental effects for terbufos sulfone. No sensitive populations were identified or characterized. The OPP RED (USEPA, 2006c) presents more detailed information about the potential health effects and sensitive populations for terbufos and terbufos sulfone.

e. Preliminary Regulatory Determination

The agency is making preliminary determinations to not regulate terbufos and terbufos sulfone with NPDWRs after evaluating health, occurrence, and other related information against the three SDWA statutory criteria. While the data suggests that terbufos and terbufos sulfone may have adverse effects on human health, the occurrence data indicate there is no substantial likelihood that terbufos or terbufos sulfone will occur in PWSs with a frequency and at levels of public health concern. Therefore, the agency finds that NPDWRs for terbufos and terbufos sulfone would not present meaningful opportunities to reduce health risk for persons served by PWSs. The Regulatory Determinations 3 Support Document (USEPA, 2014b) presents additional information and/or analyses supporting the agency's evaluation of terbufos and terbufos sulfone.

V. What is the Status of the Agency's Evaluation of Chlorate and Nitrosamines?

The agency will review the existing MDBP regulations as part of the SY3. Because chlorate and nitrosamines are DBPs that can be introduced or formed in public water systems partly because of disinfection practices, the agency believes it is important to evaluate these unregulated DBPs in the context of the review of the existing DBP regulations. DBPs need to be evaluated collectively, because the potential exists that the chemical disinfection used to control a specific DBP could affect the concentrations of other DBPs. Therefore, the agency is not making a regulatory determination for chlorate and nitrosamines at this time. The agency expects to complete the review of these DBPs by the end of 2015.

A. Chlorate

The following sections provide the background, health and occurrence information/data that the agency has collected to date for chlorate. If the public has any additional health and occurrence information that may be useful as the agency evaluates chlorate in the context of the existing MDBP rules, please provide this information to the docket.

1. Background

The chlorate anion (ClO_3^-) forms a variety of salts (e.g., sodium chlorate, calcium chlorate, potassium chlorate, and magnesium chlorate) collectively known as chlorates, which are powerful oxidizers. Chlorate compounds (especially sodium chlorate) are used as herbicides and to generate chlorine dioxide (ClO_2) as a bleaching agent (USEPA, 2006a). Disinfection practices are the most important source of chlorate in drinking water; this includes formation as a DBP from use of chlorine dioxide and its presence in hypochlorite disinfectants as an impurity (USEPA, 2006a).

Chlorate can be formed during decomposition of hypochlorite (ClO^-) solutions, which are used as a disinfectant and/or oxidant in water treatment. Hypochlorite solutions that are more aged are generally less effective and require higher doses to achieve the treatment (disinfection) objectives, which can result in more chlorate to be introduced into the chlorinated water. In addition to being a DBP (along with chlorite) formed from the use of chlorine dioxide as a disinfectant, chlorate ion may also be present as an impurity in the chlorine dioxide (Gates et al., 2009; USEPA, 2006a). Chlorate can also form by the reaction of chlorite with free chlorine applied as a residual disinfectant in the distribution system (Gallagher et al., 1994). In addition, chlorite can be oxidized by a strong oxidant (such as ozone) to produce chlorate in the water (von Gunten, 2003). Chlorate salts readily dissolve in water and are highly mobile because of the absence. In the absence of redox reactions, the chlorate ion would be expected to partition predominantly into water and to be highly mobile in water. However, under most environmental conditions chlorate is subject to redox reactions, which are expected to reduce the concentration of chlorate in the water column (USEPA, 2006a).

2. Health Effects Information

Acute ingestion of high levels of sodium chlorate has resulted in acute kidney failure and hemolysis among other effects based on numerous case reports of individuals accidently ingesting high levels of chlorate compounds (USEPA, 2006b; WHO, 2005). A population-based case-control study of chlorate as a DBP at concentrations >200 µg/L identified significantly increased odds ratios for obstructive urinary defects, cleft palate, and spina bifida (Righi et al., 2012). The median chlorate exposure for the study population was 280 µg/L. In a case-control study of the same population in Italy, Aggazzotti et al. (2004) found no association between preterm births and exposure to chlorate.

The animal studies provide clear and consistent evidence that subchronic and chronic exposure to chlorate results in effects on blood and thyroid. Subchronic studies in rats have reported decreased hemoglobin, hematocrit, and red blood cell (RBC) counts (Abdel-Rahman et al., 1984; Barrett, 1987; McCauley et al., 1995) and thyroid colloid depletion, follicular cell hypertrophy and hyperplasia (Hooth et al., 2001).

A chronic study based on increased thyroid gland follicular cell hypertrophy in male rats (NTP, 2005a) was identified as the critical study for establishing an RfD of 0.03 mg/kg/day (USEPA, 2006b). The RfD was derived by using the BMD method and based on the lower 95% confidence limit (BMDL) of 28 mg/L as sodium chlorate (22 mg/L as chlorate), corresponding to 0.9 mg/kg/day (USEPA, 2006b), with a composite UF of 30 for intraspecies (i.e., sensitive populations) and interspecies variability (i.e., thyroid hormone differences between humans and rats). EPA calculated a non-cancer HRL of 210 µg/L for chlorate using the RfD of 0.03 mg/kg/day for a 70 kg adult ingesting 2 L of drinking water per day and an RSC of 20%.

A cancer risk assessment was not conducted for chlorate because sodium chlorate is classified as not likely to be carcinogenic to humans at doses that do not alter thyroid hormone homeostasis under the USEPA (2005b) Cancer Guidelines. The RfD is protective against acute alterations in thyroid homeostasis and therefore considered to also be protective of tumorigenicity as well as other chronic and subchronic adverse health effects discussed in the literature (Hooth et al., 2001; Khan et al., 2005; NTP, 2005a).

EPA also evaluated whether health information is available regarding sensitive populations. According to the OPP RED, there was no pre- or postnatal sensitivity or susceptibility observed in the submitted developmental studies in rats and rabbits or the 2-generation reproduction study in rats. However, there is a concern for developing offspring because of the effects of inorganic chlorate on thyroid function in rats (USEPA, 2006a). Chlorate is one of a number or inorganic ions that may interfere with iodine uptake by the thyroid, but chlorate is not highly potent in this respect (Van Sande et al., 2003).

Chlorate may also cause hemolysis, thus persons with low red blood cell counts such as those with anemia may be particularly sensitive to sodium chlorate. Data from the 1994 National Health Interview Survey (O'Day et al.,

1998) indicate that there were about 5 million people in the U.S. who suffered from some form of anemia. About 3 to 5% of the population may have an inherited glucose-6-phosphate dehydrogenase (G6PD) deficiency, with males more sensitive than females (Luzzatto and Mehta, 1989), and about 1% may have a form of hereditary methemoglobinemia (Jaffe and Hultquist, 1989). Each one of these conditions is a contributor to low red blood cell counts within the population. Individuals co-exposed to other ions that decrease iodine uptake by the thyroid or have low RBC counts may be more sensitive to chlorate exposure.

3. Occurrence Data and Information

a. Drinking Water

The 1997–1998 DBP ICR is currently the best available data source for characterizing the national occurrence baseline for chlorate. The DBP ICR, which included monitoring data for 296 water systems serving 100,000 people or more (representing a total population of 130 million), required water systems that use hypochlorite solutions or chlorine dioxide for disinfection to monitor for chlorate (USEPA, 1996). Subsequently, 82 water systems serving approximately 40 million people monitored and reported chlorate occurrence under the DBP ICR (using an MRL of 20 µg/L). Table 9 presents the number and percentage of samples and systems (along with the population served) that measured chlorate at levels exceeding the specified threshold concentrations (i.e., HRL and ½ HRL). These samples were associated with 41.5% (34 of 82) of the ICR systems using hypochlorite solutions or chlorine dioxide for disinfection and 11.5% (34 of 296) of all of the ICR systems. EPA assumes there was no occurrence of chlorate among the ICR systems that were not required to monitor for it, since they use disinfection techniques not expected to produce chlorate. Approximately 51.1% (878 of 1,719) of ICR samples from the finished water or distribution system of the systems required to monitor had chlorate at levels greater than the ½ HRL (105 µg/L) and 19.3% (332 of 1,719) had chlorate at levels greater than the HRL (210 µg/L). The samples greater than the ½ HRL were associated with 73.2% (60 of 82) of the ICR systems using hypochlorite solutions or chlorine dioxide for disinfection and 20.3% (60 of 296) of all ICR systems (including those that were not required to monitor for chlorate). The samples greater than the HRL were associated with 41.5% (34 of 82) of the ICR systems using hypochlorite solutions or chlorine dioxide for disinfection and 11.5% (34 of 296) of all ICR systems (including those that were not required to monitor for chlorate) (McGuire et al., 2002).

Since the DBP ICR was completed in 1998, these data likely underestimate current (2012) chlorate occurrence among the systems serving 100,000 people or more for the following two reasons: (1) Some of these systems may have changed the disinfectant type from chlorine gas to chlorine dioxide for compliance with the existing Stage 1 or Stage 2 DBP rules; and/or (2) some systems may have switched from chlorine gas to hypochlorite solution due to a security concern (i.e., a concern of safety of transportation and storage for chlorine gas). Disinfection surveys conducted by the AWWA Disinfection Systems Committee in 1998 and 2007 have confirmed that chlorine dioxide and hypochlorite use has increased (AWWA Disinfection Systems Committee, 2008a, 2008b; Connell et al., 2000a, 2000b).

TABLE 9—SUMMARY OF CHLORATE MONITORING RESULTS UNDER THE DBP ICR

Chlorate threshold	Of DBP ICR PWSs that monitored for chlorate, samples and PWSs with at least one detection > threshold *		Of all DBP ICR PWSs, PWSs with at least one detection > threshold and estimated population served **	
	Number (percentage) of DBP ICR samples with detection > threshold	Number (percentage) of DBP ICR PWSs with at least one detection > threshold	Number (percentage) of DBP ICR PWSs with at least one detection > threshold *	Population served by DBP ICR PWSs with at least one detection > threshold **
HRL (210 µg/L)	332 of 1,719 (19.3%)	34 of 82 (41.5%)	34 of 296 (11.5%)	11.8 of 130 million (9.1%)
½ HRL (105 µg/L)	878 of 1,719 (51.1%)	60 of 82 (73.2%)	60 of 296 (20.3%)	31.7 of 130 million (24.4%)

* 82 PWSs that used hypochlorite or chlorine dioxide were required to monitor for chlorate during the DBP ICR monitoring period, based on their potential to form chlorate. Number and percentage of samples and PWSs are based on those 82 PWSs that monitored for chlorate.

** The number and percentage of PWSs and population served > threshold is based on all 296 systems. EPA assumes that the 214 systems not required to monitor do not have chlorate concentrations above the thresholds.

Finished water data for chlorate from California collected between 2001 and 2007 show lower occurrence compared to the DBP ICR. The State of California reported results from testing more than 1,200 drinking water samples from 45 PWSs and chlorate was detected in 945 samples (78.4%) from 24 different systems (53.3%) (Ranalli, B., 2013). Approximately 41.6% (501 of 1,205) of samples from 17 systems (37.8%) had chlorate at levels greater than the ½ HRL (105 µg/L) and approximately 12.4% (149 of 1,205) of samples from 10 systems (22.2%) had chlorate at levels greater than the HRL (210 µg/L) (Ranalli, B., 2013).

It is important to note that the agency included chlorate in the UCMR 3, which is currently in process. UCMR 3 will provide a national dataset of chlorate occurrence in drinking water and will update the occurrence data provided by the DBP ICR.

Ambient water data for chlorate are limited, but chlorate could be present in areas where it is used as an herbicide or discharged from paper plants where it is used as a bleaching agent. Since chlorate is a DBP, higher concentrations are expected in finished water than in ambient water.

b. Exposure from media other than water

There is very little quantitative information available on the occurrence of chlorate in food, air, and soil or other products resulting in residential exposures. Without reliable estimates of intakes, it is not possible to estimate the contribution of drinking water to total exposure. However, based on modeling results, the agency estimated that the chlorate intake from food (as a result of sodium chlorate use as a pesticide) for the overall population is approximately 3 µg/kg-day, with somewhat higher intakes for children under five years old of approximately 5 to 8 µg/kg-day (USEPA, 2006a). Additional food exposure from use of sanitizing solutions in food preparation plants (e.g., equipment and contact surfaces) and processing (e.g., bleaching agent) may also be a source of exposure (21 CFR section 178.1010). Intake for adults from dietary supplements containing chlorate may range from 0.001 to 0.29 µg/kg-day.

B. Nitrosamines Group (6 Nitrosamines)

The following sections provide the background, health and occurrence information/data that the agency has collected to date for nitrosamines. If you have any additional health and occurrence information that may be useful as the agency evaluates nitrosamines in the context of the regulatory review of existing MDBP rules, please provide this information to the docket.

1. Background

Nitrosamines are a class of nitrogen-containing organic compounds that share a common nitrosamino functional group (HSDB, 2010). EPA included five nitrosamine compounds on the CCL 3: N-nitrosodimethylamine (NDMA), N-nitrosodiethylamine (NDEA), N-nitrosodi-n-propylamine (NDPA), N-nitrosopyrrolidine (NPYR), and N-nitrosodiphenylamine (NDPhA). EPA monitored six nitrosamines under UCMR 2 using EPA Analytical Method 521, four of which are CCL 3 compounds (i.e., NDMA, NDEA, NDPA, NPYR), and two non-CCL 3 nitrosamines [i.e., N-nitrosomethylethylamine (NMEA) and N-nitrosodi-n-butylamine (NDBA)]. The fifth CCL 3 nitrosamine compound, NDPhA, was not monitored under UCMR 2 due to lack of a reliable analytical method. Although other nitrosamines (e.g., N-nitrosomorpholine, N-nitrosopiperidine) have been identified in finished water (Mitch et al., 2009), they were also not included in UCMR 2 for similar analytical reasons. The nitrosamines from the UCMR 2 thus comprise the list of six nitrosamines that moved forward to the data evaluation phase of regulatory determination and are the focus of the information that follows below.

All six nitrosamines may be produced in small quantities for research purposes, but only one (NDEA) is currently produced commercially in the United States. NDEA is used as an additive in gasoline and in lubricants, as an antioxidant, and as a stabilizer in plastics, though no data are available about quantities used (HSDB, 2010). NDMA was once used in the production of rocket fuel, as a solvent, and as a rubber accelerator. It was also used or proposed for use as an antioxidant, an additive for lubricants, and a softener for copolymers (ATSDR, 1989). There are no production data on any of the nitrosamine compounds from EPA's Inventory Update Reporting (IUR) program.

NDMA can be formed as an unintended byproduct of manufacturing processes that involve the use of nitrite or nitrate and amines, including tanneries, fish processing plants, foundries, and pesticide, dye, rubber or tire manufacturing plants (ATSDR, 1989). Nitrosamines have been found in tobacco products, cured meats, ham, bacon, beer, whiskey, fish, cheese, soybean oil, toiletries, household cleaners, pesticides, rubber baby bottle nipples and pacifiers (ATSDR, 1989; Drabik-Markiewicz et al., 2009; Fine et al., 1977; NTP, 2011; Pérez et al., 2008; Yurchenko and Mölder, 2007).

NDMA is commonly present in municipal sewage sludge (ATSDR, 1989). NPYR has also been detected in municipal sewage sludge (HSDB, 2010). ATSDR (1989) cites several studies indicating that nitrosamine formation in sewage sludge appears to be the result of biological and chemical transformation of alkylamines in the presence of nitrite. In addition, nitrosamines may form in air, soil, water, sewage, food, animal systems and other media where precursors (e.g., amines and nitrite) are present (HSDB, 2010). NDMA can be produced endogenously in humans from the interaction of nitrates and nitrites with amines in the stomach (Mirvish 1975, 1992; Tricker et al., 1994).

As described in the following occurrence section, nitrosamines in finished water are commonly considered as DBPs because most of the literature indicates that the main source of nitrosamines in finished water is associated with water treatment, particularly from disinfection with chloramines. NDMA is the predominant species of nitrosamines found in finished water; other nitrosamines are detected less frequently. Based on their physical and chemical properties, the nitrosamines appear to be moderately to very mobile in the environment (the exception being NDBA, which is of low mobility). The nitrosamines are subject to a variety of removal mechanisms when present in soil and water, including volatilization (particularly NDMA), photodegradation, and microbial degradation, although the rates and extent of biodegradation are highly variable (HSDB, 2010).

2. Health Effects Information

As the more thoroughly studied nitrosamine compared to the other nitrosamine compounds, NDMA provides epidemiological case-control and other evidence that human nitrosamine exposure is associated with an increased risk of several types of cancer, including cancer of the stomach, esophagus, oral cavity, and pharynx (La Vecchia et al., 1995; Larsson et al., 2006; Loh et al., 2011; Straif et al., 2000). In accordance with the most recent Guidelines for Carcinogen Risk Assessment (USEPA, 2005b), EPA has categorized the six nitrosamine compounds as likely to be carcinogenic to humans based on sufficient evidence of carcinogenicity in animal studies with multiple tumor types (predominately liver and esophageal) in multiple animal species (e.g., rats, mice, and hamsters) (Clapp et al., 1968, 1971; Druckrey et al., 1967; Lijinsky, 1987a, 1987b; Peto et al., 1991a, 1991b). All of the six nitrosamines have been determined to cause cancer through a mutagenic MOA because of DNA adduct formation leading to errors in DNA replication, altered cell proliferation and ultimately tumors (Diaz Gomez et al., 1986; Goto et al., 1999; Jarabek et al., 2009; Souliotis et al., 1998). The mutagenic MOA is supported by positive findings from mutagenicity and genotoxicity *in vitro* and *in vivo* studies (Gollapudi et al., 1998; Kushida et al., 2000; Martelli et al., 1988, Robbiano et al., 1996, Tinwell et al., 1994).

With a mutagenic MOA, Age Dependent Adjustment Factors (ADAFs) are used to account for the potential increased cancer risk due to early-life exposure for infants and children (USEPA, 2005c). The age-adjusted unit risk is determined by summing up each of the time-weighted unit risks for the three ADAF developmental groups. The age-adjusted unit risks include a ten-fold adjustment for birth to <2 years, a three-fold adjustment for 2 years to <16 years, and no additional adjustment for exposures later in life, in conjunction with age-specific drinking water intake values (USEPA. 2012c), and the fraction of a 70-year lifetime applicable to each age period. The main cancer risk values used to derive the HRLs are further explained in section III.C.1 and are also summarized for nitrosamines in Table 10 below.

TABLE 10—EPA DERIVED RISK VALUES AND HRLS FOR THE SIX INDIVIDUAL NITROSAMINES

Nitrosamines	Studies for establishing a slope factor	Cancer slope factor $(mg/kg/day)^{-1}$	[1] Age-adjusted unit risk $(\mu g/L)^{-1}$	[2] HRL (μg/L)	[3] HRL (ng/L)
NDBA	Liver and esophageal tumors in rats (Druckrey et al., 1967).	0.4	3.0×10^{-5}	3×10^{-2}	30
NDEA	Liver and esophageal tumors in rats (Peto et al., 1991a,b).	30	2.3×10^{-3}	4×10^{-4}	0.4
NDMA	Liver tumors in rats (Peto et al., 1991a,b)	21	1.6×10^{-3}	6×10^{-4}	0.6
NDPA	Liver and esophageal tumors in rats (Druckrey et al., 1967).	2	1.5×10^{-4}	7×10^{-3}	7
NMEA	Liver tumors in rats (Druckrey et al., 1967)	4	3.0×10^{-4}	3×10^{-3}	3
NPYR	Liver tumors in rats (Peto et al., 1984)	7	5.3×10^{-4}	2×10^{-3}	2

[1] Based on the recommendations of the U.S EPA's 2005 *Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens,* the Unit Risk applicable to exposures beginning in early-life was adjusted with ADAFs and age-specific drinking water intakes resulting in a lifetime value of unit risk for exposure to 1 μg/L of a contaminant. The calculation for Age-Adjusted Unit Risk = Σ(CSF × ADAF × DWI/BWR × CW × F). The risk calculations for each individual nitrosamine can be found in the HESDs.

[2] The cancer HRL is determined by dividing the population risk level, one-in a million (10^{-6}), by the age-adjusted unit risk.

[3] The nitrosamine HRL values are converted to ng/L units by multiplying the μg/L values by 1000.

As shown in table 10, the available data indicate a range of cancer risk values for the individual nitrosamines. Moreover, when multiple nitrosamines from this group are present in finished water together, their individual cancer risks are additive (Berger et al., 1987).

EPA also evaluated whether health information is available regarding sensitive populations. The fetus, newborns, and infants may be potentially sensitive to the carcinogenic effects of nitrosamines due to the mutagenic MOA and evidence of transplacental carcinogenicity (Althoff et al., 1977; Donovan and Smith, 2008). Studies have found that younger rats were more susceptible to the development of liver tumors compared to rats exposed later in life to nitrosamines (Gray et al., 1991; Peto et al., 1984; Vesselinovitch et al., 1984). EPA's *Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens* (USEPA, 2005c) indicates that potential increased cancer risk due to early-life exposure should be taken into account for such compounds when there is the potential for greater susceptibility for structural changes to DNA leading to tumors when the exposures occur in infancy or childhood. Thus, the *Supplemental Guidance* (USEPA, 2005c) recommends using CSF estimates from chronic studies with ADAFs when chemical-specific data that quantify the potential increased risk are lacking. All of the HRLs are based on lifetime exposure and include application of ADAFs, which adjust for the increased risk from early life exposure (see section III.C.1).

In addition, habitual consumers of alcoholic beverages may be more susceptible to carcinogenic effects of nitrosamines because alcohol increases the metabolism of nitrosamines via a metabolic pathway that leads to the formation of mutagenic DNA adducts. Co-exposure to ethanol has been shown to exacerbate the cancer effects of nitrosamines in animal studies (Anderson et al., 1993; Kamataki et al., 2002; McCoy et al., 1986). There are approximately five million people in the U.S. who suffer from alcoholism (O'Day et al., 1998) that may have an increased risk if co-exposed to nitrosamines (Amelizad et al., 1989; Verna et al., 1996).

3. Occurrence Data and Information

The data collected under UCMR 2 (USEPA, 2014d) are currently the best available data for characterizing the national occurrence baselines for the six nitrosamines. Under UCMR 2, PWSs were required to collect a sample at each entry point to the distribution system as well as at the maximum residence time locations within the distribution system associated with each entry point, and to report the disinfectant type in use at these locations at the time that the samples were being taken. The agency was unable to measure at the HRL for some of the nitrosamines. Therefore, Table 11 presents all of the monitoring results for each of the six nitrosamines relative to the MRLs.

TABLE 11—SUMMARY OF UCMR 2 MONITORING RESULTS FOR SIX NITROSAMINES

Nitrosamines considered under RD 3	MRL	Percentages (number) of samples with detection	Percentages (number) of UCMR 2 PWSs with at least one detection	Percentages (number) of actual UCMR 2 population served with at least one detection*
Nitrosamine Group	2 to 7 ng/L	10.6% (1,907 of 18,053)	28.6% (343 of 1,198)	46.43% (73 of 157 million)
NDBA	4 ng/L	0.05% (9 of 18,043)	0.4% (5 of 1,198)	1.07% (1.7 of 157 million)
NDEA	5 ng/L	0.3% (46 of 18,038)	2.2% (26 of 1,198)	7.14% (11.2 of 157 million)
NDMA	2 ng/L	10.2% (1,841 of 18,040)	27.0% (324 of 1,198)	41.54% (65.3 of 157 million)
NDPA	7 ng/L	0% (0 of 18,049)	0% (0 of 1,198)	0% (0 of 157 million)
NMEA	3 ng/L	0.02% (3 of 18,043)	0.3 (3 of 1,198)	0.003% (0.004 of 157 million)
NPYR	2 ng/L	0.2% (41 of 18,043)	1.8% (21 of 1,198)	4.73% (7.4 of 157 million)

* The population-served values have been adjusted to include both the population served directly by a system and also the estimated attributable proportion of the population served by other systems that purchase water from the system. These adjustments are described in the UCMR 2 support document.

Finished water data for the nitrosamines from California (Ranalli, B., 2013) are consistent with the UCMR 2 data. The State of California reported NDMA detections in 23.8% (24 of 101) of PWSs and NDEA detections in 7.1% (1 of 14) of PWSs. There were no NPYR, NDPA, NMEA, or NDBA detections reported. Reporting levels are not known. For California data on NDMA and NDEA, the minimum reported detections were 1 ng/L and 30 ng/L, respectively. NDBA, NDPA, NMEA, and NPYR had no detections and thus no minimum reported value in the dataset (Ranalli, B., 2013). While ambient water data for the nitrosamines are limited, because they are DBPs, it is expected that in general there would be higher concentrations in finished water than in ambient water.

V. What about the remaining CCL 3 contaminants?

For the remaining CCL 3 contaminants, the agency lacked adequate health and/or occurrence information needed to address the three SDWA statutory criteria to make a regulatory determination. Table 2 and Table 4 of this notice provide information about the data or information gap(s) that prevented the contaminant from moving forward for this regulatory determination effort. The agency continues to conduct research, collect information or find other avenues to fill the data and information gaps identified in Table 2 and 4. One mechanism the agency plans to continue to use to fill occurrence gaps for several of these contaminants is the UCMR.

VI. EPA's Next Steps

EPA intends to carefully evaluate and respond to the public comments received on the five preliminary determinations and issue its final regulatory determinations in 2015. If the agency makes a final determination to regulate any of the contaminants, EPA will begin the process to propose an NPDWR within 24 months and promulgate a final NPDWR within 18 months following the proposal.

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Dated: October 3, 2014.

Gina A. McCarthy,

Administrator.

Appendix: HRL Derivation With Age-Related Exposure Factors

DERIVATION OF THE HEALTH REFERENCE LEVEL (HRL) FOR STRONTIUM USING AGE-SPECIFIC EXPOSURE FACTORS

Age range	DWI/BWR (L/kg/day)	Age-specific fractions of a 19-year exposure duration	Time-weighted DWI/BWR (L/kg/day)
Birth to <1 month	0.235	0.004	0.001
1 to <3 months	0.228	0.009	0.002
3 to <6 months	0.148	0.013	0.002
6 to <12 months	0.112	0.026	0.003
1 to <2 years	0.056	0.053	0.003
2 to <3 years	0.052	0.053	0.003
3 to <6 years	0.043	0.158	0.007
6 to <11 years	0.035	0.263	0.009
11 to <16 years	0.026	0.263	0.007
16 to <18 years	0.023	0.105	0.002
18 to <21 years #	0.026	0.053	0.001
Summation of the Time-Weighted DWI/BWRs =			**0.040 L/kg/day**[*]
Reference Dose =			**0.3 mg/kg/day**
RSC =			**20%**
[+]**HRL = (0.3 mg/kg/day ÷ 0.040 L/kg/day) × .20 =**			**1.500 mg/L**
Final child specific HRL:			**1500 µg/L**

[*] Rounded; # includes 18th year; DWI/BWR = drinking water intake to body weight ratio; HRL= health reference level; RSC = relative source contribution.

[+] HRL = (RfD/**Σ**(DWI/BWR × F)) × RSC.

The age-specific data on drinking water intakes in units of L/kg/day from birth through age 3 are from Table 3–19 in the EPA Exposures Factors Handbook (USEPA, 2011e) and from Table 3–38 for ages 3 to <19 . The exposure duration adjustment was calculated by dividing the age-specific fraction of a 19 year exposure by the total exposure in months or years as appropriate (e.g., birth to <1 month = (1/12)/19 years = 0.00439; 6 to <11 years = 5/19 years = 0.26316). The time-weighted DWI/BWR values are the product of the age-specific DWI/BWR multiplied by the age-specific fraction of a 19 year exposure. The time-weighted DWI/BWRs are summed to obtain the normalized value.

[FR Doc. 2014–24582 Filed 10–17–14; 8:45 am]

BILLING CODE 6560–50–P