Summary of Nominations for the Fourth Contaminant Candidate List (CCL 4)

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Contents

Exhibits

Acronyms and Abbreviations

ATSDR	Agency for Toxic Substances and Disease Registry
AWWA	American Water Works Association
CASRN	Chemical Abstract Service Registry Number
CCL	Contaminant Candidate List
CCL 1	EPA's First Contaminant Candidate List
CCL 2	EPA's Second Contaminant Candidate List
CCL 3	EPA's Third Contaminant Candidate List
CCL 4	EPA's Fourth Contaminant Candidate List
CDC	Centers for Disease Control and Prevention
CFR	Code of Federal Regulations
EPA	United States Environmental Protection Agency
HA	Health Advisories
HPC	Heterotrophic Plate Count
MCL	Maximum Contaminant Level
MCLG	Maximum Contaminant Level Goal
MDEP	Massachusetts Department of Environmental Protection
MDH	Minnesota Department of Health
MTBE	Methyl tertiary butyl ether
NJDEP	New Jersey Department of Environmental Protection
NPDWR	National Primary Drinking Water Regulations
NRDC	National Resource Defense Council
OSHA	Occupational Safety and Health Administration
PCCL	Preliminary Contaminant Candidate List
PFOA	Perfluorooctanoic acid
PWSs	Public Water Systems
REDs	Reregistration Eligibility Decision
SDWA	Safe Drinking Water Act
U.S.	United States of America
USDA	United States Department of Agriculture
USGS	United States Geological Survey
WHO	World Health Organization

1.0 Introduction

Section 1412(b)(1) of the Safe Drinking Water Act (SDWA), as amended in 1996, requires EPA to publish the Contaminant Candidate List (CCL) every five years. The SDWA specifies that the list include contaminants that 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 the SDWA. EPA uses this list of unregulated contaminants to help identify priority contaminants for regulatory decision making and to prioritize research and data collection efforts. SDWA also requires the agency to consult with the scientific community, including the Science Advisory Board (SAB), and provide notice and opportunity for public comment prior to the publication of the Final CCL. In addition, SDWA directs the agency to consider the health effects and occurrence information for unregulated contaminants to identify those contaminants that present the greatest public health concern related to exposure from drinking water.

EPA published the third CCL (CCL 3), which listed 116 contaminants on October 8, 2009 (74 FR 51850 (USEPA, 2009a)). In developing the CCL 3, EPA implemented a multi-step process to select contaminants for the final CCL 3, which included the following key steps:

1) The identification of a broad universe of potential drinking water contaminants (CCL 3 Universe);
2) Screening the CCL 3 Universe to a Preliminary CCL (PCCL) using screening criteria based on the potential to occur in PWSs and the potential for public health concern;
3) Evaluation of the PCCL contaminants based on a more detailed review of the occurrence and health effects data using a scoring and classification system to identify a final list of 116 CCL 3 contaminants; and
4) Incorporating public input and expert review in the CCL 3 process.

Steps 1, 2 and 3 in the process are described in detail in the CCL 3 support documents:

- "Final Contaminant Candidate List 3 Chemicals: Identifying the Universe" (USEPA, 2009b);
- "Final Contaminant Candidate List 3 Chemicals: Screening to a PCCL" (USEPA, 2009c);
- "Final Contaminant Candidate List 3 Chemicals: Classification of the PCCL to the CCL" (USEPA, 2009d);
- "Final Contaminant Candidate List 3 Microbes: Identifying the Universe" (USEPA, 2009e);
- "Final Contaminant Candidate List 3 Microbes: Screening to the PCCL" (USEPA, 2009f); and
- "Final Contaminant Candidate List 3 Microbes: PCCL to CCL Process" (USEPA, 2009g).

These documents can be found on the EPA web site at: http://www.epa.gov/ccl/contaminant-candidate-list-3-ccl-3 or at http://www.regulations.gov (docket ID: EPA-HQ-OW-2007-1189).

After a Final CCL is published, SDWA section 1412(b)(1)(B)(ii) as amended in 1996, requires EPA at five year intervals to make determinations of whether to regulate or not to regulate no fewer than five contaminants from the CCL in a process called regulatory determination. This is a separate process from the listing of contaminants on the CCL. The 1996 SDWA Amendments specify three criteria to determine whether a contaminant may require regulation:

- the contaminant may have an adverse effect on the health of persons;
- the contaminant is known to occur or there is a substantial likelihood that the contaminant will occur in PWSs with a frequency and at levels of public health concern; and
- in the sole judgment of the Administrator, regulation of such contaminant presents a meaningful opportunity for health risk reduction for persons served by PWSs.

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].

On February 11, 2011, as a separate action, the agency issued a positive regulatory determination for perchlorate, a chemical listed in CCL 1, CCL 2 and CCL 3 (76 FR 7762 (USEPA, 2011)). In January 2016 (81 FR 13 (USEPA, 2016a)), the agency made final determinations not to regulate four contaminants: dimethoate; 1,3-dinitrobenzene; terbufos; and terbufos sulfone and delayed the final determination of strontium pending analysis of additional data. These six contaminants were not listed on the Draft CCL 4, pending their final determinations, and are also not included on the Final CCL 4.

In May 2012, EPA sought public input by requesting nominations of contaminants to be considered for inclusion on the CCL 4 (77 FR 27057 (USEPA, 2012)). EPA reviewed the nominations and supporting information provided by nominators to determine if any new data were provided that had not been previously evaluated for CCL 3. EPA also requested supporting information that has been made available since the development of the CCL 3 or existing information that was not considered in the development of the CCL 3, which shows that the nominated contaminant may have an adverse health effect on people and occurs or is likely to occur in public water systems. EPA reviewed the nominations and supporting information provided by nominators to determine if any new data were provided that had not been previously evaluated for CCL 3. The agency also collected additional data for the nominated contaminants, when it was available, from both CCL 3 data sources that had been updated and from new data sources that were not available at the time of CCL 3. A complete list of references provided by nominators can be found in Appendices 3, 4 and 5 of this document. A more detailed description

[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 C.F.R. 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 analytical methods and taking cost into consideration.

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

of the CCL data sources collected by EPA may be found in the support document "Data Sources for the Fourth Contaminant Candidate List (CCL 4)" (USEPA, 2016b).

The Draft CCL 4 was published on February 4, 2015 (80 FR 6076 (USEPA, 2015)), and includes 100 chemicals or chemical groups and 12 microbes. EPA conducted an abbreviated evaluation and selection process for the CCL 4. This abbreviated CCL 4 process includes a three pronged approach: (1) carrying forward CCL 3 contaminants (minus those with regulatory determinations), (2) seeking and evaluating nominations from the public for additional contaminants to consider and (3) evaluating any new data for those contaminants with previous negative regulatory determinations from CCL 1 or CCL 2 for potential inclusion on the CCL 4.

EPA requested comment on the Draft CCL 4 and on how to further improve upon the selection process developed for CCL 3 as a tool for future CCLs. The agency received 27 public comment letters on the Draft CCL 4. EPA considered all public comments and evaluated the data and information provided by commenters in determining the Final CCL 4. EPA used the same process used in the CCL 3 to screen and score any contaminants with new data or information provided by commenters. Based on these analyses, EPA is not listing three cancelled pesticides (disulfoton, fenamiphos, and molinate) on the Final CCL 4 that were included on the Draft CCL 4 because these chemicals are not known or anticipated to occur in PWSs and are not anticipated to require regulation. With the exception of these three pesticides, all of the contaminants listed on the Draft CCL 4 are listed on the Final CCL 4. EPA has responded to all public comments in the "Comment Response Document for the Fourth Drinking Water Contaminant Candidate List (Categorized Public Comments)" document that is available in the docket (USEPA 2016c).

This document describes EPA's request for contaminant nominations and summarizes the nominations received by EPA. In addition, it describes EPA's analysis of the nominated contaminants and reports on their status in the CCL 4. The specific contaminants nominated, the information provided by the nominators and the outcome of the nominated contaminants in the CCL 4 process are included in Appendices 1 through 7 of this document. More detailed information on the CCL 4 is available in the CCL 4 support documents found on the CCL 4 Web site at: https://www.epa.gov/ccl/draft-contaminant-candidate-list-4-ccl-4. The original nomination letters submitted via the docket and nominations submitted via the Web site can be found in the docket at http://www.regulations.gov (docket ID: EPA-HQ-OW-2012-0217).

2.0 Requesting Nominations

The agency sought nominations for contaminants to be considered for possible inclusion in the CCL 4 by framing the SDWA requirements in a series of questions to document the anticipated or known occurrence in PWSs and the adverse health effects of potential contaminants. The agency requested that the public respond to those questions and provide the documentation and rationale for including a contaminant for consideration in the CCL 4 process. The questions posed to the public were:

- What is the contaminant's name, Chemical Abstract Service Registry Number (CASRN), and/or common synonym (if applicable)?

- What factors make this contaminant a priority for the CCL 4 process (e.g., widespread occurrence; anticipated toxicity to humans; potentially harmful effects to susceptible populations (e.g., children); potentially contaminated source water (surface or ground water) and/or finished water; release to air, land and/or water; contaminant is manufactured in large quantities with a potential to occur in source waters)?
- What are the new significant health effects and occurrence data that are available since CCL 3 or existing information that was not considered in CCL 3, which you believe supports the CCL requirement(s) that a contaminant may have an adverse effect on the health of persons and is known or anticipated to occur in PWSs?
- Please provide complete citations, including author(s), title, journal and date. Contact information for the primary investigator would also be helpful.

Nominations were received via the EPA Web site and via the EPA docket (docket ID: EPA-HQ-OW-2012-0217). The agency compiled the information from the nominations process to identify the contaminants nominated, the rationale for the nomination and to compare the supporting data submitted to information gathered by EPA. Where new information was of sufficient quality, that information was used in the analysis. EPA analyzed the nominated contaminants using the CCL 3 process, which is described briefly in Section 1, to select the CCL 4.

3.0 Nominated Contaminants

EPA received nominations for 59 unique contaminants for the CCL 4 submitted by 10 organizations and individuals. These 59 nominations include 5 microbial and 54 chemical contaminants. Eight contaminants were nominated by more than one nominator. Aldicarb, bisphenol A, carbaryl, chlorpyrifos, *Toxoplasma gondii*, and microcystin-LR were each nominated by two separate nominators. Manganese and perfluorooctanoic acid (PFOA) were nominated by three different nominators each.

The agency did not require nominators to provide their name or an affiliated organization. Two nominators remained anonymous while providing documentation and rationale for the contaminants. Two other individuals identified themselves but did not provide an organization affiliation.

The organizations that nominated contaminants were:

- American Water Works Association (AWWA),
- Natural Resources Defense Council (NRDC),
- State of Massachusetts Department of Environmental Protection (MDEP),
- State of Minnesota Department of Health (MDH),
- State of New Jersey Department of Environmental Protection (NJDEP), and
- U.S. Department of Agriculture (USDA).

EPA received three general types of nominations:

- specific individual chemicals,
- specific individual organisms, and
- groups of contaminants (Heterotrophic Plate Count (HPC) was considered as a group).

The AWWA also provided a letter with recommendations for the CCL 4 process. The full text of this letter can be found at http://www.regulations.gov (docket ID: EPA-HQ-OW-2012-0217).

3.1 Chemical Nominations

There were a total of 54 unique chemical contaminant nominations for the CCL 4. The full list of chemical nominations and the supporting information provided by the nominators can be found in Appendix 1. The references provided by the nominators for chemical nominations can be found in Appendix 3.

3.1.1 Analysis of Nominated Chemical Contaminants

SDWA specifies that the CCL only includes those contaminants without any proposed or promulgated NPDWRs. There are two nominated contaminants covered under the existing NPDWR for beta photon emitters (strontium 90 and cesium 137) (40 CFR §141.66 (d)(1)); therefore, these will not be considered for CCL 4. Radon was also nominated but is not eligible for CCL 4 since a proposed NPDWR has been developed (64 FR 59245, November 2, 1999 (USEPA, 1999). Aldicarb was nominated but is not eligible for CCL 4 since it has an existing NPDWR (40 CFR §141.61(c)); (Note, in response to an administrative petition the agency issued an administrative stay of the effective date of the maximum contaminant levels (MCLs) for aldicarbs).

For the remaining 50 nominated chemicals, EPA reviewed the nominations and supporting information provided by nominators to determine if any new data were provided that had not been previously evaluated for CCL 3. In addition to the data provided by nominators, and the data EPA collected previously under CCL 3, EPA collected data for the nominated contaminants, when it was available, from both CCL 3 data sources that have been updated and from new data sources that were not available at the time of CCL 3. A list and description of these data sources can be found in the "Data Sources for the Fourth Contaminant Candidate List (CCL 4)" (USEPA, 2016b) support document. If new data were available, EPA screened and scored the nominated contaminants using the same process as was used in CCL 3. Five of the nominated chemicals were on CCL 3 and were carried forward to the Draft CCL 4 along with the other CCL 3 contaminants. The five chemicals are: perfluorooctanoic acid (PFOA), microcystin-LR, methyl tertiary butyl ether (MTBE), alpha-hexachlorocyclohexane and permethrin. These chemicals are also listed on the Final CCL 4. Microcystin-LR is included within the group of cyanotoxins in the Final CCL 3 and the Final CCL 4.

Forty of the nominated chemicals were previously included in the CCL 3 Universe, and were carried forward to the CCL 4 Universe. In addition to these forty, EPA has added three nominated chemicals to the CCL 4 Universe (octylphenol ethoxylate, oxacillin and virginiamycin) based on health effects and/or occurrence data that is newly available since the development of the CCL 3. Seven of the nominated chemicals did not have enough data in order

to be added to the CCL 4 Universe. EPA screened all of the nominated chemicals in the CCL 4 Universe according to the screening criteria developed for CCL 3 and based on that evaluation, twenty of the nominated chemicals were included in the PCCL 4. Eighteen of those 20 chemicals were also included in the PCCL 3 and EPA added two new chemicals (manganese and nonylphenol) to the PCCL 4. The data used to screen the nominated chemicals from the CCL 4 Universe to the PCCL 4 can be found in the "Screening Document for the Fourth Preliminarty Contaminant Candidate List (PCCL 4): (USEPA, 2016d). EPA further evaluated the nominated chemicals on the PCCL 4 based on the classification process developed in CCL 3 and determined that manganese and nonylphenol should be added to the CCL 4 (in addition to the chemicals carried forward from the CCL 3 to the CCL 4) based on new health and/or occurrence information that warrants further evaluation. The data which was used to further evaluate the nominated contaminants from the PCCL 4 and to select those that were included in the CCL 4 can be found in the "Contaminant Information Sheets (CISs) for the Final Fourth Contaminant Candidate List (CCL 4)" (USEPA, 2016e).

Manganese is an element that naturally occurs in oxide forms and in combinations with other elements in many minerals. Small amounts, found in foods, are an essential nutrient for humans and animals. Manganese ores are used in a variety of applications in the U.S. Its principal use is in steel production to improve hardness, stiffness and strength (ATSDR, 2012). In 2003 and as part of the CCL 1 Regulatory Determination process, EPA made a negative regulatory determination for manganese based on the health and occurrence data available at that time. However, EPA is continuing to evaluate the potential risks to children and infants per over 30 recent studies cited by the public during the nomination and comment period that indicate concern for neurological effects in children and infants exposed to excess manganese. These studies were not available at the time manganese was considered for Regulatory Determination 1 or CCL 3. In addition, new monitoring studies from USGS and drinking water monitoring information from several States support an earlier survey (i.e., the National Inorganics and Radionuclides Survey) that indicates manganese is known to occur in drinking water. EPA believes the new health effects information and additional occurrence data merit listing manganese on the CCL 4.

Nonylphenol is used in the preparation of lubricating oil additives, resins, plasticizers and antioxidants for plastics and rubber. Additionally, sixty percent of nonylphenol is used in the production of nonylphenol ethoxylates, which are found in detergents and used in the treatment of textiles. Nonylphenol was previously considered for CCL 3. It was included in the CCL 3 Universe, but was not included on the PCCL 3 or CCL 3. Updated health and occurrence data (since the development of the CCL 3) are now available for nonylphenol, and these data (as follows) were considered in evaluating nonylphenol for the Draft and Final CCL 4. Nonylphenol and some of its degradation products have been found to have estrogenic activity in rats and mice (WHO, 2004). Monitoring data are available from a USGS National Reconnaissance monitoring study of ambient water (Kolpin et al., 2002). EPA believes this updated health data and additional occurrence data show that nonylphenol is anticipated to occur in PWSs and has potential adverse health effects; therefore, it merits listing on the CCL 4.

Dicofol was nominated by the public and EPA considered adding it to the Draft CCL 4; however, both recent manufacturers of the pesticide ceased all production as of May 17, 2011,

and agreed to an EPA registration cancellation, which effectively prohibits all labeled uses of existing stocks after October 31, 2016. Use of dicofol has declined significantly in recent years. The chemical properties of dicofol indicate that it is has low mobility in water because it is expected to adsorb to organic matter in soil and sediment and it has moderately low solubility in water. Therefore, EPA did not list dicofol on the CCL 4 because it is not known or anticipated to occur in drinking water due to its low mobility. Additionally, the registration cancellation, which will prohibit use of existing stocks beyond October 2016, is expected to further lessen any potential occurrence in drinking water.

Exhibit 1 shows the nominated chemicals that were included in the Final CCL 4. In addition, Appendix 6 shows a list of the nominated chemicals and whether they were included in the CCL 4 Universe, PCCL 4 or Final CCL 4.

Exhibit 1. Nominated Chemicals Included in the Final CCL 4

Contaminant Name	CASRN
*alpha-hexachlorocyclohexane	319-84-6
manganese	7439-96-5
*methyl tertiary butyl ether (MTBE)	1634-04-4
*microcystin-LR	101043-37-2
nonylphenol	25154-52-3[1]
*perfluorooctanoic acid (PFOA)	335-67-1
*permethrin	52645-53-1

[1] The organization that nominated "nonylphenol" for CCL 4 provided the CASRN of 25451-52-3. The name "nonylphenol" does not allow for a definitive identification of chemical structure since nonylphenol can exhibit two forms of isomerism. There are at least five CASRNs known to be associated with "nonylphenol:" in addition to 25154-52-3 (which represents n-nonylphenol with the ortho-, meta-, or para-substitution unspecified), other CASRNs include: 104-40-5 (4-n-nonylphenol); 84852-15-3 (4-nonylphenol, branched); 91672-41-2 (2-nonylphenol, branched); and 139-84-4 (3-n-nonylphenol). None of these five CASRNs are adequately general enough to represent both forms of isomerism. For the sake of consistency, the CASRN provided by the nominator was selected and the additional possible CASRNs and structures are delineated here
*Indicates that this chemical was carried forward from CCL 3 to the CCL 4.

3.2 Microbial Nominations

Five unique microbial nominations were submitted by the public in response to EPA's request for nominations for contaminants to be considered for possible inclusion in the CCL 4 (77 FR 27057). The following organisms or group of organisms were nominated: Heterotrophic Plate Count bacteria, adenovirus, *Naegleria fowleri*, *Toxoplasma gondii* and *Vibrio chloerae.* *Toxoplasma gondii* was nominated by two different nominators. Adenovirus and *Naegleria fowleri* were included in the Final CCL 3 and are carried forward to the Final CCL 4. Additional information on the nominated microbes and the information submitted by the nominators can be found in Appendix 2.

3.2.1 Analysis of Nominated Microbial Contaminants

EPA reviewed the nominated microbial contaminants and the supporting information provided by nominators to determine if any new data were provided that had not been previously

evaluated. The agency also collected additional data for the nominated microbial contaminants, when it was available, from both CCL 3 data sources that had been updated and from literature searches covering the time between CCL 3 and CCL 4 (2007–2012). If new data were available, EPA screened and scored the microbial contaminants nominated for CCL 4 using the same process that was used for CCL 3. The new data did not change the CCL 3 scores or listing decisions for the nominated microbial contaminants.

The group of HPC bacteria was nominated for the CCL 4; however, available epidemiological evidence shows no relationship between gastrointestinal illness and HPC bacteria in drinking water (Calderon, 1988; Calderon and Mood, 1991; Payment et al., 1997; WHO, 2003). Thus, EPA considers the potential health risk of HPC bacteria in drinking water as likely negligible and is not including HPC on the CCL 4. In addition, HPC bacteria are addressed under the Surface Water Treatment Rule as a treatment technique where they can be monitored in lieu of a disinfectant residual.

Vibrio cholerae and *Toxoplasma gondii* remain on the PCCL 4 and *Naegleria fowleri* and adenovirus are listed on the Final CCL 4, along with the other microbes included on the Final CCL 3. A summary of the outcomes for the microbial contaminants for the CCL 3 and CCL 4 can be found in Appendix 7.

4.0 References

Note: The following references apply to Sections 1.0, 2.0 and 3.0 above. References provided by CCL 4 nominators are listed in Appendix 5 below.

Calderon, R.L. 1988. Bacteria Colonizing Point-of-Entry Granular Activated Carbon filters and their Relationship to Human Health. EPA CR-813978-01-0, US Environmental Protection Agency, Washington, DC.

Calderon, R.L. and Mood, E.W. 1991. Bacteria Colonizing Point-of-Use Granular Activated Carbon Filters and their Relationship to Human Health. EPA CR 811904-01-0, US Environmental Protection Agency, Washington, DC.

Kolpin, D. W., E. T. Furlong, M. T. Meyer, E. M. Thurman, S. D. Zaugg, L. B. Barber, and H. T. Buxton. 2002. Pharmaceuticals, Hormones, and Other Organic Wastewater Contaminants in U.S. Streams, 1999-2000: A National Reconnaissance. Environmental Science and Technology, v. 36, no. 6

Payment, P., J. Siemiatycki, L. Richardson, G. Renaud, E. Franco, and M. Prevost. 1997. A prospective epidemiological study of gastrointestinal health effects due to the consumption of drinking water. Int. J. Environ. Health Res. (7): 5-31.

USEPA, 1999. National Primary Drinking Water Regulations; Radon-222; Proposed Rule. Federal Register. Vol. 64, No. 211. p. 59245, November 2, 1999.

USEPA. 2009a. Drinking Water Contaminant Candidate List 3--Final. Federal Register. Vol. 747. No 194. p. 51850. October 8, 2009.

USEPA. 2009b. Final Contaminant Candidate List 3 Chemicals: Identifying the Universe. EPA 815-R09-006. August, 2009.

USEPA. 2009c. Final Contaminant Candidate List 3 Chemicals: Screening to a PCCL. EPA 815-R-09-007. August, 2009.

USEPA. 2009d. Final Contaminant Candidate List 3 Chemicals: Classification of the PCCL to the CCL. EPA. 815-R-09-008. August, 2009.

USEPA. 2009e. Final Contaminant Candidate List 3 Microbes: Identifying the Universe. EPA. 815-R-09-004. August, 2009.

USEPA. 2009f. Final Contaminant Candidate List 3 Microbes: Screening to the PCCL. EPA 815-R-09-0005. August, 2009.

USEPA. 2009g. Final Contaminant Candidate List 3 Microbes: PCCL to CCL Process. EPA 815-R-09-009. August, 2009.

USEPA. 2011. Drinking Water: Regulatory Determination on Perchlorate. Federal Register. Vol. 76, No. 29. p. 7762, February 11, 2011.

USEPA. 2012. Request for Nominations of Drinking Water Contaminants for the Fourth Contaminant Candidate List. Federal Register. Vol. 77. No 89. p. 27057. May 8, 2012

USEPA. 2015. Drinking Water Contaminant Candidate List 4—Draft. Federal Register. Vol. 80 No. 23, p 6076, February 4, 2015.

USEPA. 2016a. Announcement of Final Regulatory Determinations for Contaminants on the Third Drinking Water Contaminant Candidate List. Federal Register. Vol. 81 No. 1, p 13, January 4, 2016.

USEPA. 2016b. Data Sources for the Fourth Contaminant Candidate List (CCL 4). EPA 815-R-16-007. November, 2016.

USEPA. 2016c. Comment Response Document for the Fourth Drinking Water Contaminant Candidate List (Categorized Public Comments). EPA 815-R-16-004. November, 2016.

USEPA. 2016d. Screening Document for the Fourth Preliminary Contaminant Candidate List 4 (PCCL 4). EPA 815-R-16-008. November, 2016.

USEPA. 2016e. Contaminant Information Sheets (CISs) for the Final Fourth Contaminant Candidate List (CCL 4). EPA 815-R-16-003. November, 2016.

WHO. 2003. Emerging Issues in Water and Infectious Disease Series: Heterotrophic Plate Counts and Drinking-water Safety. ed. J. Bartram, J. Cotruvo, M. Exner, C. Fricker, and A. Glasmacher. IWA Publishing, London, UK. p. 119 – 122.

WHO. 2004. Integrated Risk Assessment: Nonylphenol Case Study. ed. D. Bontje, J. Hermens, T. Vermeire, and T. Damstra. 63 pp. December, 2004. Available on the Internet at:http://www.who.int/ipcs/methods/Nonylphenol.pdf.

5.0 Appendices

The appendices that follow provide tabulated summaries that present a list of the chemical and microbial contaminants nominated for consideration in CCL 4.

Appendix 1 lists the chemical nominations, provides the chemical abstracts service registry number (CASRN) for each chemical contaminant, common name, nominating individual or organization, health effects information provided with the nomination, occurrence information provided with the nomination and additional information provided with the nomination. For the purpose of developing this appendix, EPA separated original text submitted with the nomination for each contaminant and placed it into the health effects information, occurrence information or additional information columns, as appropriate. EPA maintained the text submitted with each nomination verbatim; however, footnote numbers submitted in nominators' letters have been removed for clarity. The footnotes generally refer to references or comments and can be found in the original letters located in the docket.

Appendix 2 provides the same type of information as Appendix 1 for the microbial contaminants.

Appendix 3 lists the references provided with chemical nominations including: CASRN, contaminant name, nominating organization or individual and references cited. The references cited in Appendix 3 are in an abbreviated citation format (e.g., Fiore et al., 1986).

Appendix 4 lists the same type of information as Appendix 3 for the microbial nominations.

Appendix 5 includes the complete list of full references provided with CCL 4 nominations for both microbial and chemical nominations. The original nomination letters and the nominations submitted via the Web site can be found in the docket at http://www.regulations.gov (docket ID: EPA-HQ-OW-2012-0217). The original documents contain all tables referenced in Appendix 1 and 2.

Appendix 6 shows the outcome of the nominated chemicals in the CCL 4 process (i.e., whether the nominated chemical was included in the CCL 4 Universe, PCCL 4 or Final CCL 4). It also denotes the status of the nominated chemicals in the CCL 3 process. An "X" denotes that a chemical was included in that stage of the process. Note that nominated contaminants with an NPDWR or proposed NPDWR were not eligible for CCL 4, as explained in Section 3.1.1 above.

Appendix 7 shows the outcome of the nominated microbes in the CCL 4 process (i.e., whether the nominated microbe was included in the CCL 4 Universe, PCCL 4 or Final CCL 4). It also denotes the status of the nominated microbe in the CCL 3 process. An "X" denotes that the microbe was included in that stage of the process.

Appendix 1. Chemical Contaminants Nominated[1]

CASRN	Common Name	Nominator	Health Effects Information Provided with Nomination	Occurrence Information Provided with Nomination	Additional Information Provided with Nomination
77439-76-0	3-chloro-4-dichloromethyl-5-hydroxy-2(5H)-furanone)	AWWA	None given	None given	None given
116-06-03	Aldicarb	AWWA	None given	None given	None given
116-06-3	Aldicarb	Natural Resources Defense Council	Aldicarb is an N-methyl carbamate insecticide that causes reversible red blood cell and plasma cholinesterase inhibition. This pesticide is classified as Toxicity Category 1 because of its high toxicity through all routes of exposure (oral, dermal and inhalation). Symptoms of acute aldicarb exposure observed in animal studies include decreased motor activity, lacrimation, tremors, salivation, pinpoint pupils, and decreased grip strength. A rat study by EPA/ORD demonstrated that young animals are more susceptible to aldicarb-induced brain cholinesterase inhibition than adults. Although it is generally believed that acute high level exposure to aldicarb will not cause chronic health effects, one case study by Grendon et al. (1994) in Washington State documented long-term health problems in men and sheep resulting from a single poisoning incident. EPA has not assessed the risks of chronic exposure to aldicarb in its 2006 Revised Human Health Risk Assessment (HRA). The Agency reasoned that since cholinesterase inhibition due to aldicarb exposure is reversed in less than 24 hours, such an assessment is unnecessary and chronic exposure can be treated as a series of acute exposures. However, EPA mentioned in the Revised HRA that effects such as pale kidneys and hydroceles in the oviducts occurred in dams in a developmental study, symptoms that suggest chronic damage not seen in acute single-exposure cases. In addition, some studies suggest that chronic exposure to aldicarb may have longer-term effects on the immune and nervous systems. Fiore et al (2006) analyzed immune function in two groups of women, one exposed to aldicarb at environmental concentrations in groundwater at levels below 61 ppb (23 subjects), and an unexposed group (27 subjects). No women in either group had known reasons for immune problems. The researchers found a significant association between aldicarb exposure and abnormalities in T-cell subset ratios. Hajoui et al. (1992) also found changes in the percentages of certain T-cell subsets after subchronic, but not chronic exposure.The results of a rat study by Smulders et al. (2003) suggest that exposure to carbamates such as aldicarb may also lead to chronic changes in the nervous system resulting from the inhibition of neuronal nicotinic acetylcholine receptors. A similar study of the carbamates fenoxycarb, carbaryl, and S-ethyl N,N-dipropylthiocarbamate (EPTC), which have the same mechanism of action, showed that increasing the pesticide dose or the length of exposure reduced the rate of reversal of acetylcholine receptor inhibition. Therefore, two mechanisms, cholinesterase inhibition and acetylcholine receptor inhibition may lead to chronic neurotoxicity from exposure to carbamate pesticides such	EPA placed aldicarb under Special Review in 1984 due to concerns about groundwater contamination. Aldicarb degradation in groundwater is slow. This chemical is persistent and mobile in soil, and degrades in the environment to aldicarb sulfoxide and aldicarb sulfone, both of which are cholinesterase inhibitors. In 1991 EPA established MCLs of 0.003 ppb for aldicarb, 0.004 ppb for aldicarb sulfoxide and 0.002 ppb for aldicarb sulfone, but these MCLs never went into effect. Instead, EPA issued a 7 ppb health advisory for each of the aldicarb species and for combined aldicarb residues. EPA based its drinking water risk assessment in the HRA on the highest aldicarb concentrations in groundwater found in eight regions where aldicarb was used. The concentrations ranged from 0 to 24 ppb. The region with no aldicarb detections was removed from the analysis. Surface water concentrations, on the other hand, were derived from models for lack of sufficient monitoring data.	None given
			as aldicarb. This raises concerns about chronic low-level exposure such as may result from aldicarb contamination of drinking water.		

CASRN	Common Name	Nominator	Health Effects Information Provided with Nomination	Occurrence Information Provided with Nomination	Additional Information Provided with Nomination
			Acute dietary exposure estimates from food alone exceeded the level of concern for children 1 to 2 years old (159% of the acute Population Adjusted Dose, or aPAD), and children 3 to 5 years old (129% aPAD), so that any additional exposures from drinking water would increase these risks of concern. The highest exposure from groundwater calculated for the regions where this pesticide was detected was 945% aPAD for the 95th percentile of the most exposed population sub-group. For the general U.S. population and other sub-groups, exposure ranged from 20% aPAD to 393% aPAD. It is clear from EPA's own analysis that aldicarb is a water contaminant that poses health risks of concern at levels found in food and drinking water. Given that food exposure alone exceeds levels of concern for children, drinking water exposure creates an additional unacceptable risk. EPA must move to establish a protective MCL for aldicarb.		
68555-24-8	Alkylphenol mono- to tri-oxylates	Natural Resources Defense Council	Alkylphenols were first reported to be estrogenic in the 1930s. In 1991, publication of the effects of nonylphenol on cultured human breast cancer cells led to health concerns. Estrogenic effects have also been shown in the mouse. Estrogenic effects are present at tissue concentrations of 0.1 µM for octylphenol and 1 µM for nonylphenol. A recombinant yeast screen using the human estrogen receptor has shown similar results.	An estimated 450,000,000 pounds of alkylphenol polyethoxylates (APEs) are produced annually in the United States, and about half that amount is estimated to be released to wastewater. Alkylphenol polyethoxylates do not break down effectively in sewage treatment plants or in the environment. Instead they degrade to alkylphenols and alkylphenol ethoxylates, which persist for longer. Nonylphenol and its ethoxylates, and other alkylphenols, have been detected in wastewater and in waterways.	None given
26787-78-0	Amoxicillin	Natural Resources Defense Council	Widespread exposure to antibiotics is contributing to the growth of bacterial resistance, and this problem is of grave concern. In the past several decades almost every bacteria that can cause infections in humans has developed resistance to at least one antibiotic, and some are resistant to multiple antibiotics. The Centers for Disease Control and Prevention (CDC) have identified antibiotic resistance as one of the most pressing public health problems to face our nation. Infections caused by bacteria with resistance to at least one antibiotic have been estimated to kill over 60,000 hospitalized patients each year. Antibiotic resistance is caused by a number of factors including repeated and improper use of antibiotics in both humans and animals. Scientists also agree that exposure to low levels of antibiotics actually promotes bacterial resistance by exerting selective pressure for genes that promote resistance. Beta-lactam antibiotics are a broad class of antibiotics which include penicillin derivatives, cephalosporins, monobactams, carbapenems and Beta-lactamase inhibitors. Methicillin, a form of penicillin, had been relied upon as an common effective treatment for Staphylococcus aureus infections but now many strains of S. aureus bacteria are resistant to methicillin (MRSA or methicillin-resistant Staphylococcus aureus.) Unfortunately, MRSA is resistant to much of the entire class of penicillin-like antibiotics called beta-lactams. Therefore, EPA must include penicillin, amoxicillin, oxacillin and methicillin on the CCL4.	Antibiotics are found in wastewater because the body does not completely metabolize all drugs, so both the metabolized and unmetabolized drug are excreted by humans into wastewater. For example,when amoxicillin is ingested, 60-75% of the antibiotic is excreted unchanged into the urine. This antibiotic, now in the environment, may encounter other bacteria and promote resistance. It is unknown how much of an impact current low levels of antibiotics in drinking water are having on the problem of bacterial resistance. However, the potential has been recognized for many years.	None given

CASRN	Common Name	Nominator	Health Effects Information Provided with Nomination	Occurrence Information Provided with Nomination	Additional Information Provided with Nomination
86-50-0	Azinphos-methyl	Natural Resources Defense Council	Azinphos-methyl (CAS # 86-50-0) is an organophosphate pesticide classified as toxicity category 1 for oral exposure. Exposure to azinphos-methyl causes plasma, red blood cell and brain cholinesterase inhibition, with symptoms including headache, nausea, vomiting, dizziness, anxiety, muscle tremors and weakness. Studies by Souza et al. (2004, 2005) found that azinphos-methyl affected human placental enzymatic activity, which may have adverse consequences for fetal development. Exposure to organophosphate pesticides (OPs) such as azinphos-methyl has been associated with lower performance on neurobehavioral tests in exposed adults. Children are more vulnerable than adults to the neurotoxic effects of OPs and may suffer developmental effects from low-level chronic exposures.	Azinphos-methyl has a high potential to pollute surface waters due to runoff and spray drift. Data on environmental concentrations of azinphos-methyl in the United States are limited, but studies in South Africa suggest that under certain conditions azinphos-methyl may also reach high concentrations (>40 ppb) in groundwater. EPA indicated in its drinking water assessment in the Interim Reregistration Eligibility Decision (IRED) document for azinphos-methyl that the estimated environmental concentration (EEC) of this pesticide in surface water is 16 ppb at typical application rates in peaches. This concentration is over three times the acute drinking water level of comparison (DWLOC) the agency calculated for infants less than a year old (5 ppb), and over twice the DWLOC for children 1-6 years (6 ppb). The highest annual mean concentrations in surface water according to monitoring data and EPA models ranged from 0.27 ppb to 7.2 ppb. The latter concentration exceeds the chronic DWLOC the agency calculated for infants less than a year old (7 ppb). While EPA argued in the IRED that the phase-out of azinphos –methyl use on peaches will eliminate drinking water risks of concern, EPA is still allowing the use of azinphos¬methyl on apples (the most frequently treated crop) at application rates equal to or higher than those for peaches (1.0-1.5 lb ai/A per application, 4.5 lb ai/A per year maximum on apples vs. 1.125 lbs ai/A per application, 4.5 lbs ai/A per year maximum on peaches). Furthermore, the total amount of azinphos-methyl used on apples (890,000 lb active ingredient) is over seven times the amount used on peaches (120,000 lb). Therefore, the EPA assessment indicates that azinphos-methyl poses a risk to drinking water supplies. While EPA has issued a four-year limited registration for azinphos-methyl use on apples and seven other crops, the Agency has stated that these registrations may be extended, thus creating the need to regulate azinphos-methyl as a drinking water contaminant.	None given
1405-89-6	Bacitracin zinc	Natural Resources Defense Council	Widespread exposure to antibiotics is contributing to the growth of bacterial resistance, and this problem is of grave concern. In the past several decades almost every bacteria that can cause infections in humans has developed resistance to at least one antibiotic, and some are resistant to multiple antibiotics. The Centers for Disease Control and Prevention (CDC) have identified antibiotic resistance as one of the most pressing public health problems to face our nation. Infections caused by bacteria with resistance to at least one antibiotic have been estimated to kill over 60,000 hospitalized patients each year. Antibiotic resistance is caused by a number of factors including repeated and improper use of antibiotics in both humans and animals. Scientists also agree that exposure to low levels of antibiotics actually promotes bacterial resistance by exerting selective pressure for genes that promote resistance.	Antibiotics are found in wastewater because the body does not completely metabolize all drugs, so both the metabolized and unmetabolized drug are excreted by humans into wastewater. For example, when amoxicillin is ingested, 60-75% of the antibiotic is excreted unchanged into the urine. This antibiotic, now in the environment, may encounter other bacteria and promote resistance. It is unknown how much of an impact current low levels of antibiotics in drinking water are having on the problem of bacterial resistance. However, the potential has been recognized for many years. Large animal feeding operations generate a large amount of waste that can potentially contaminate groundwater and waterways contributing to antibiotic resistance and contamination of waterways with steroid hormones. As occurs in humans, some portion of the antibiotics administered to livestock will pass unchanged through their bodies and will be excreted in their waste. It has been estimated that	None given

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			Massive quantities of antibiotics are used in agriculture both to treat infections and as food additives to promote growth and to compensate for conditions that contribute to infection. Animals raised in Concentrated Animal Feeding Operations (CAFOs) are at increased risk for infection due to close confinement and stress. In fact, it has been estimated that 70% of the antibiotics used in the U.S. are for animal husbandry. Improper use and overuse of antibiotics in livestock and poultry can cause resistance in strains of bacteria that can infect humans. Furthermore, half of the antibiotics used in livestock are in the same classes of drugs that are used in humans. As a result the U.S. Institute of Medicine (IOM) and the World Health Organization (WHO) both stated that the widespread use of antibiotics in agriculture is contributing to antibiotic resistance in humans.	between 25-75% of antibiotics are excreted unchanged in feces and can persist in the soil after land application. Manure is applied in large quantities as fertilizer in farm fields. In addition to potentially contaminating the food supply with antibiotic resistant bacteria, antibiotics in manure can persist in soil and promote the development of more antibiotic resistant bacteria. Animal waste and its associated contaminants can enter waterways through groundwater contamination, overflow of waste lagoons into surface water or by over-application of manure as fertilizer in farm fields. A recently published study found evidence of fecal contamination and increased levels of antibiotic resistant bacteria downstream of a swine concentrated feeding operation. Other studies have found antibiotic resistance in groundwater underlying a swine waste lagoon. As such, antibiotics that are used both for human medical needs and in large-scale agriculture operations at low levels in animal feed to promote animal growth must be included on the CCL4 and must be regulated. These antibiotics include bacitracin zinc, spiramycin, tylosin, and virginiamycin. Notably, these antibiotics were all banned for agricultural use in the European Union in 1998.	
25057-89-0	Bentazone	AWWA	None given	None given	None given
85-68-7	Benzyl butyl phthalate	Natural Resources Defense Council	Phthalates are endocrine disruptors that have been found to cause developmental and reproductive abnormalities in animal studies. Furthermore, studies have found an association between phthalate metabolite concentrations and obesity, insulin resistance and thyroid hormone levels in humans. Stahlhut et al.(2007) analyzed urinary concentrations of seven phthalate metabolites in 1,443 adult men and found a statistically significant positive association between concentrations of the metabolites MBzP, MEHHP, MEOHP and MEP and abdominal obesity. Concentrations of MBP, MBzP and MEP were positively and significantly associated with insulin resistance. In a different study, baby boys exposed to the phthalates, DMP, DEP or BBP, in their mother's breast milk were found to have hormonal disturbances at 3 months of age. Studies have found that di-isononyl phthalate (DiNP) is an anti-androgenic endocrine disruptor with developmental and reproductive toxicity. Exposures in pregnant rats have been shown to adversely affect development of the male reproductive tract. Adverse effects include a cluster of outcomes that has been called "phthalate e syndrome" and includes underdeveloped or absent reproductive organs, retained nipples, cryptorchidism, decreased anogenital distance (AGD), hypospadias, and decreased or abnormal sperm. DINP does not bind to the androgen receptor and these effects are likely mediated through interference with testosterone synthesis. Phthalates are ubiquitous in the U.S. population. Although significant exposure can occur due to consumer products, the contribution of drinking water to	Phthalates enter the environment as a result of releases from industrial facilities that manufacture or use these compounds, from waste disposal sites, through the use of phthalate-containing products by consumers and through discharge of municipal wastewaters containing phthalates. Phthalates have been found in numerous hazardous waste sites: diethyl phthalate (DEP) has been identified at 348 sites, dibutyl phthalate (DBP) at 602 sites, dimethyl phthalate (DMP) at 167 sites, benzyl butyl phthalate (BzBP) at 413 sites, dicyclohexyl phthalate (DCHP) at one site, and di-n-octyl phthalate (DnOP) at 433 sites, among other phthalates. Phthalates have been detected in environmental water samples across the United States, which raises concerns about drinking water as a route of exposure. The maximum concentrations found for some of these phthalates are particularly alarming. The following data were found in a search for stream water samples analyzed for phthalates in the EPA STORET database. Results are for the period January 2000 through June 2012: Benzyl butyl phthalate (BzBP) was present in 789 (19.3 %) out of 4077 stream water samples analyzed for this chemical, with a maximum concentration of 1000 ug/L.	None given

CASRN	Common Name	Nominator	Health Effects Information Provided with Nomination	Occurrence Information Provided with Nomination	Additional Information Provided with Nomination
			overall exposure deserves to be examined. The NHANES measured the concentrations of 12 phthalate metabolites in the urine of over 2,500 children		
			and adults. The highest average concentration was 163 ug/g creatinine for MEP, a metabolite of the plasticizer diethyl phthalate (see Table 3). [See the CDC report cited below] The Centers for Disease Control and Prevention (CDC) did not report what percentage of samples had detectable concentrations of each of the metabolites, but nine of the metabolites were found in the urine of at least half of the individuals. [See Table 3. CDC report of phthalate concentrations in urine; NHANES 1999 – 2000 and 2001 - 2002 located on page 27 of the NRDC Nomination Letter and Table 4 Frequency of detection of phthalate metabolites in human urine samples, United States located on pages 28 and 29 of the NRDC Nomination Letter.]		
80-05-7	Bisphenol A	Anonymous 201	Weak endocrine disruptor - principal concern is for potential reproductive and developmental effects in early life stages. EPA has considered exposures to children from drinking water and from the use of BPA in consumer products. EPA also examined potential ecological impact from the presence of BPA in the environment.	None given	None given
80-05-7	Bisphenol A	Natural Resources Defense Council	A number of recent studies have revealed that early life exposures to low-doses of BPA result in adverse effects later in life. The developing fetus is especially vulnerable. Although many of these studies were done in laboratory animals, the exposures occurred at concentrations currently found in the human population. Recent research finds low levels of BPA exposure causes harm in the mammary gland, prostate tissue, and brain. In rats, in utero exposure to BPA causes long-term effects on development of mammary tissue, causing preneoplastic lesions, increased susceptibility to cancer and increased sensitivity to a chemical known to cause breast cancer. Perinatal exposure to low levels of BPA causes precancerous prostate lesions (prostatic intraepithelial neoplasia) in rats. The effect appears to result from the failure in exposed animals of a gene to become hypermethylated as the rats age. Experiments with mice reveal that chronic adult exposure to BPA causes insulin resistance, a common problem in humans that can lead to Type II diabetes and heart disease. Recent human studies continue to find links between BPA and cardiovascular disease, obesity and metabolic changes affecting insulin levels, which could lead to diabetes. BPA has been shown to cause aneuploidy in mouse oocytes. Meiotic aneuploidy is the most common cause of miscarriage in women. In 2007, a group of 38 scientists issued a consensus statement expressing their concern that current levels of BPA exposure were contributing to the human health conditions of neurobehavioral problems, obesity, infertility and reproductive cancers. In addition, the U.S. National Toxicology Program has issued a draft report expressing “some concern” that BPA could cause neurobehavioral abnormalities, early onset puberty, and reproductive cancers, especially in fetuses, infants and children who are exposed.	BPA is produced at over one million pounds per year and is frequently found in the environment. BPA releases to the environment in the U.S. totaled 1.4 million pounds in 2006, including 3,410 pounds released directly to water and 108,805 pounds released to the air. BPA is a water contaminant. A study in Germany found BPA in surface water (0.0005 to 0.41 ug/L), in sewage effluents (0.018 to 0.702 ug/L), in sediments (0.01 to 0.19 mg/kg) and in sewage sludge (0.004 to 1.363mg/kg dw). Cousins et al. (2002) reviewed previously published monitoring data for the United States and found a median reported water concentration of 0.5 ug/l (below the detection limit of the studies) and a 90th percentile of 4.4 ug/l. The same study also suggested a half-life for BPA of 4.5 days in surface water, indicating that BPA can be transported hundreds of kilometers in rivers before levels fall below detection limits. In December, 2011, the International Chemical Secretariat in the E.U. reported that many drinking water pipes are being restored by relining them with epoxy resin that contains BPA, and that this BPA is leaching into the drinking water. Anecdotally, this practice seems to also be occurring in the U.S. This is another important source of exposure in the drinking water – and suggests that levels of BPA are even higher than articles suggest.	Bisphenol A - (4,4'-(1-ethylethylidene)bisphenol or 4,4'-Isopropylidenediphenol), (CASRN 80-05-7), is a monomer used as the building block of polycarbonate plastics and other plastics including epoxy resins. BPA is found in a wide variety of everyday consumer products, such as the coating of food and drink packaging, dental sealants, baby bottles, water bottles, microwave ovenware and eating utensils. As these products age, the polycarbonate polymer breaks down, releasing the BPA monomer.
1689-84-5	Bromoxynil	AWWA	None given	None given	None given
63-25-2	Carbaryl	AWWA	None given	None given	None given

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63-25-2	Carbaryl	Natural Resources Defense Council	Carbaryl (CAS # 63-25-2) is an N-methyl carbamate pesticide that acts as a neurotoxic acetylcholinesterase inhibitor and a "likely" carcinogen according to the Office of Pesticide Programs Cancer Assessment Review Committee. The systemic effects of carbaryl include headache, dizziness, weakness, shaking, nausea, stomach cramps, diarrhea, and sweating. Effects may also include loss of appetite, weakness, weight loss, and general malaise. Carbaryl is particularly toxic to the developing nervous system of fetuses, infants, and young children. Exposure to elevated levels of carbaryl may cause developmental neurotoxicity and "significant changes in some of the morphometric measurements of the brain". Given the limitations in the monitoring data that the Agency [EPA] has acknowledged, and the fact that the highest EEC estimated by EPA models was 55 times the acute DWLOC for children 1 to 2 years old, it is clear that carbaryl presents risks of concern from drinking water exposure and should be regulated as a drinking water contaminant by establishing an MCL.	Approximately 3.9 million pounds of carbaryl active ingredient are used annually in the U.S. When EPA issued its Revised Risk Assessment for carbaryl in 2003, its water assessment did not consider non-agricultural sources of carbaryl, which constitute a total of 40% of carbaryl use by weight, and which are the dominant sources of carbaryl pollution in surface water. Despite ignoring non-agricultural uses, the carbaryl health risk assessment in the Interim Reregistration Eligibility Decision (IRED) found that acute surface water risks presuming maximum label application rates exceeded the drinking water level of concern (DWLOC) for children and the general population when combined with estimated food exposures. U.S. Geological Survey National Water Quality Assessment (NAWQA) monitoring data presented in the carbaryl assessment demonstrated that streams draining urban areas had both higher concentrations of carbaryl and more frequent detections, when compared with streams draining agricultural or mixed land use areas. It is clear that contamination of water is predominantly from non-agriculture uses of carbaryl, and that by not considering these uses, the Agency dramatically underestimated the amount of carbaryl in drinking water (Estimated Environmental Concentration, or EEC), which is likely to be two-times higher than EPA estimates. Twenty-one percent of surface water samples in the NAWQA database contained detectable levels of carbaryl.EPA discussed in its IRED the limitations of existing monitoring data: "Carbaryl is fairly mobile, but is not likely to persist or accumulate in the environment. As such, it is difficult for monitoring studies to detect peak concentrations that can occur. EPA determined that currently available monitoring studies for carbaryl are limited in this regard, and did not use them to define peak values for carbaryl."As a result of these data limitations, EPA used models to estimate drinking water EECs for currently registered uses in the carbaryl IRED. The Agency reported that the acute drinking water EECs ranged from 23 to 410 ppb for acute exposure, and from 1.3 to 23 ppb for chronic exposure, which exceeded the acute DWLOC for children 1-2 years old (7.4 ppb) and for the general population (200 ppb). This is especially concerning, given that these calculations are likely to underestimate risk by excluding non-agricultural uses of carbaryl, which comprise 40% of total carbaryl used. Therefore, it is likely that actual EEC's are even higher, possibly 40% higher, than what the Agency calculates. The high toxicity of carbaryl, coupled with the high exceedances of acceptable levels in drinking water, make this level of risk to infants and children unacceptably high.	None given
10045-97-3	Cesium 137	Anonymous 197	Cs-137 is prevalent in atmosphere due to melt down underway in Japan of four reactors with 40 years of spent fuel on site. Cs-137 interferes with endocrine function and fetal development.	There are 23 nuclear power plants of exact design to the fatal power plants in Japan, failure due to earthquakes near population centers and water sources.	Monitoring existing conditions leads to rate of change analysis when done on a predictable time frame.
1897-45-6	Chlorothalonil	AWWA	None given	None given	None given
2821-88-2	Chlorpyrifos	AWWA	None given	None given	None given

CASRN	Common Name	Nominator	Health Effects Information Provided with Nomination	Occurrence Information Provided with Nomination	Additional Information Provided with Nomination
2921-88-2	Chlorpyrifos	Natural Resources Defense Council	With chlorpyrifos and other developmental neurotoxic chemicals, risk to the fetus, infant, and child comes primarily from the timing of exposure. Even a very small dose, for even a short duration, during a developmental period of vulnerability will result in permanent neural dysfunction. There is no demonstrated reliable threshold of safety for this highly toxic chemical, as indicated in the IRED, where a no-effect level could not be determined for developmental neurotoxicity. However, there is demonstrated evidence of neuropathology and increased vulnerability of fetuses when exposed to chlorpyrifos. EPA has acknowledged this susceptibility in the chlorpyrifos Human Health Risk Assessment: In conclusion, the weight of the evidence raises concern for an increase in both the sensitivity and susceptibility of the fetus or young animal to adverse biochemical, morphological, or behavioral alterations from chlorpyrifos treatment during brain development. With respect to cholinesterase inhibition, an increase in sensitivity of the young compared to adults was seen all along the dose response curve, even at relatively low doses." Using the PRZM/EXAMS screening model, EPA estimated that 90-day average and peak chlorpyrifos concentrations were 6.7 and 40 ppb respectively. Meanwhile, acute DWLOCs for infants less than a year old, children 1-6 years and females 13 to 50 years ranged from 0.9 to 9 ppb. Chronic DWLOCs for these population groups ranged from 0.2 to 0.72 ppb. EPA's modeling estimates therefore show that chlorpyrifos exposure in drinking water has the potential to expose vulnerable groups of the population to unacceptable levels of this chemical.	Chlorpyrifos (CAS # 2921-88-2) is an organophosphate pesticide used at approximately 21 to 24 million pounds active ingredient (a.i.) annually in the United States. Most chlorpyrifos is used in agriculture on crops such as corn and cotton, but other uses include golf courses, road medians, food processing plants, manufacturing plants, ship holds, railroad boxcars, and non-structural wood treatments. Chlorpyrifos is applied aerially, by chemigation, groundboom, hand wand, airblast sprayer, and other methods. Although EPA said in the IRED that the drinking water risk is below the level of concern, the Agency noted that there have been cases of high levels of drinking water well contamination associated with localized applications of chlorpyrifos as a subterranean termiticide. This was addressed, EPA said, by eliminating all termiticidal uses. However, despite EPA's assertions that only termiticidal use leads to water contamination problems, USGS and others have found contamination of ground and surface water with chlorpyrifos and its metabolites, and EPA's own modeling shows that it is likely that in certain areas of heavy use, chlorpyrifos (and its metabolites) present significant water risks. There is no evidence that the water risks of chlorpyrifos and its metabolites are limited to termiticidal use.There is extensive evidence of the potential of chlorpyrifos to contaminate surface and groundwater. Combined USGS data for state, local, national, and multi-state studies that measured chlorpyrifos concentrations in surface water detected the pesticide at 7 of 108 (6%) sites sampled. Chlorpyrifos has medium runoff potential due to its relatively low water solubility, 2 mg/L. A chlorpyrifos flux as a percentage of use of 0.15 has been measured in the Minnesota River. Chlorpyrifos is also used in non-agricultural settings and can drift or runoff directly into surface water bodies in areas of high population density.Data from the Mid-Continent Pesticide Study show that chlorpyrifos was present in the ground water in 4.2% of the wells sampled. Chlorpyrifos has been detected in 0.6% of wells sampled, according to the U.S. EPA's Pesticides in Ground Water Database. Long (1989) detected chlorpyrifos in the ground water of 30% of 56 sites examined beneath pesticide mixing and loading facilities in Illinois. The maximum concentration detected was 0.5 ppb.Water monitoring sample sites are not necessarily correlated with chlorpyrifos use sites, and in particular, may miss sites where multiple fields are treated with chlorpyrifos resulting in pooled runoff into a common water source. In fact, the IRED states, "it is not clear that they [monitoring data] represent the most vulnerable groundwater where chlorpyrifos is used most intensively" (IRED p.18). Monitoring of surface water is likely to be subject to the same problem. Levels of chlorpyrifos in pooled runoff sites are likely to be many times higher than single field sites. Similarly, data collection is not timed to correspond with worst-case scenarios, such as closely following chlorpyrifos applications, or following large storm runoff events, and thus most often misses these highly toxic environmental exposures.	None given

CASRN	Common Name	Nominator	Health Effects Information Provided with Nomination	Occurrence Information Provided with Nomination	Additional Information Provided with Nomination
84-74-2	Dibutyl phthalate	Natural Resources Defense Council	Phthalates are endocrine disruptors that have been found to cause developmental and reproductive abnormalities in animal studies.Furthermore, studies have found an association between phthalate metabolite concentrations and obesity, insulin resistance and thyroid hormone levels in humans. Stahlhut et al. (2007) analyzed urinary concentrations of seven phthalate metabolites in 1,443 adult men and found a statistically significant positive association between concentrations of the metabolites MBzP, MEHHP, MEOHP and MEP and abdominal obesity. Concentrations of MBP, MBzP and MEP were positively and significantly associated with insulin resistance. In a different study, baby boys exposed to the phthalates, DMP, DEP or BBP, in their mother's breast milk were found to have hormonal disturbances at 3 months of age. Studies have found that di-isononyl phthalate (DiNP) is an anti-androgenic endocrine disruptor with developmental and reproductive toxicity. Exposures in pregnant rats have been shown to adversely affect development of the male reproductive tract. Adverse effects include a cluster of outcomes that has been called "phthalate e syndrome" and includes underdeveloped or absent reproductive organs, retained nipples, cryptorchidism, decreased anogenital distance (AGD), hypospadias, and decreased or abnormal sperm. DINP does not bind to the androgen receptor and these effects are likely mediated through interference with testosterone synthesis. Phthalates are ubiquitous in the U.S. population. Although significant exposure can occur due to consumer products, the contribution of drinking water to overall exposure deserves to be examined. The NHANES measured the concentrations of 12 phthalate metabolites in the urine of over 2,500 children and adults. The highest average concentration was 163 ug/g creatinine for MEP, a metabolite of the plasticizer diethyl phthalate (see Table 3). [See the CDC report cited below.] The Centers for Disease Control and Prevention (CDC) did not report what percentage of samples had detectable concentrations of each of the metabolites, but nine of the metabolites were found in the urine of at least half of the individuals. [See Table 3. *CDC report of phthalate concentrations in urine; NHANES 1999 – 2000 and 2001 - 2002* located on page 27 of the NRDC Nomination Letter and Table 4 Frequency of detection of phthalate metabolites in human urine samples, United States located on pages 28 and 29 of the NRDC Nomination Letter.]	Phthalates enter the environment as a result of releases from industrial facilities that manufacture or use these compounds, from waste disposal sites, through the use of phthalate-containing products by consumers and through discharge of municipal wastewaters containing phthalates. Phthalates have been found in numerous hazardous waste sites: diethyl phthalate (DEP) has been identified at 348 sites, dibutyl phthalate (DBP) at 602 sites, dimethyl phthalate (DMP) at 167 sites, benzyl butyl phthalate (BzBP) at 413 sites, dicyclohexyl phthalate (DCHP) at one site, and di-n-octyl phthalate (DnOP) at 433 sites, among other phthalates. Phthalates have been detected in environmental water samples across the United States, which raises concerns about drinking water as a route of exposure. The maximum concentrations found for some of these phthalates are particularly alarming. The following data were found in a search for stream water samples analyzed for phthalates in the EPA STORET database. Results are for the period January 2000 through June 2012: Dibutyl phthalate (DBP) was found in 828 (19.9%) of the 4160 stream water samples for that period, with a maximum concentration of 2,760 ug/L.	None given
1918-00-9	Dicamba	AWWA	None given	None given	None given
62-73-7	Dichlorvos	Natural Resources Defense Council	Dichlorvos (CAS # 62-73-7), or DDVP, is an organophosphate insecticide widely used in agriculture. Like other organophosphates, dichlorvos is an acetylcholinesterase inhibitor. DDVP exposure may cause symptoms such as nausea, vomiting, dizziness, muscle spasms, and seizures. According to a 2000 EPA Cancer Assessment review, there is suggestive evidence that dichlorvos may cause cancer. The National Toxicology Program has stated that there is "clear evidence" of carcinogenic activity of dichlorvos in a mice study. One study has linked dichlorvos exposure to leukemia in children under 15. Another study has also found an association between dichlorvos exposure and leukemia in adult men. Furthermore, EPA has determined that "dichlorvos	Dichlorvos is soluble in water and may enter surface waters in runoff. However, no data on its occurrence in surface waters has been collected; there is also little data on dichlorvos in groundwater. Two other pesticides, naled and trichlorfon, degrade to dichlorvos in the environment and represent additional inputs of dichlorvos to water. However, monitoring data on these two pesticides is also very limited. Given the lack of monitoring data, EPA used IR-PCA PRZM/EXAMS models to calculate estimated drinking water concentrations (EDWCs) of dichlorvos in surface water. The models produced estimates that were below the EPA level of concern. However; the complete lack of	None given

CASRN	Common Name	Nominator	Health Effects Information Provided with Nomination	Occurrence Information Provided with Nomination	Additional Information Provided with Nomination
			has been shown to be a direct acting mutagen by common *in vitro* bacterial genetic toxicity assays and *in vitro* mammalian test systems."		
				monitoring data raises questions about whether an exclusive reliance on modeling results is appropriate for a neurotoxic and potentially carcinogenic pesticide such as dichlorvos. EPA should collect data monitoring data for dichlorvos by requiring such data from the registrants or commissioning its own studies to better assess drinking water risks and set an MCL if necessary.	
115-32-2	Dicofol	Natural Resources Defense Council	Animal studies have found that dicofol causes toxicity in the liver, adrenal glands, kidneys, thyroid, reproductive organs, heart and stomach. Liver and thyroid effects occurred at relatively low doses (100 ppm and 10 ppm, respectively). Dicofol is a possible human carcinogen. Dicofol has shown endocrine disruptor activity in vivo and in vitro. This chemical has been shown to interfere with blastocyst implantation in rats. The first problem with the assessment is related to the way EPA calculated the Reference Dose (RfD). EPA is supposed to apply an additional safety factor of 10x to the RfD calculation to protect infants and children, who may have increased susceptibility to health effects from chemical exposures compared to adults. The Agency reduced the FQPA safety factor of 10x to 3x based on the lack of increased pre-natal or post-natal susceptibility to dicofol in developmental toxicity studies. However, EPA stated that a developmental neurotoxicity study was necessary because dicofol produced neurotoxicity in rats and such a study might identify an endpoint for dietary risk. Despite lacking such a study, EPA improperly reduced the safety factor to 3x. If the 10x factor had been applied as mandated by the Food Quality Protection Act, a more protective acute RfD of 0.015 mg/kg day-1 would have been chosen instead of the 0.05 mg/kg day-1 dose EPA used in its assessment. Had EPA applied the 10x safety factor, dicofol exposure from food alone would have exceeded the acute RfD and the EPA level of concern for all population groups (see Table 1) [See Table 1. Comparison of acute dietary exposure values from food at the 99.9th percentile located on page 16 of the NRDC nomination letter]. This would have resulted in a DWLOC of zero (0), so that any drinking water exposure would have been of concern. The unwarranted reduction of the FQPA safety factor also affected the outcome of the chronic dietary exposure assessment. As shown in Table 2 [See Table 2. Chronic dietary food exposure and risk estimate from Dicofol (in food alone) located on page 17 of the NRDC nomination letter.], if the 10x factor had been applied, chronic exposures from food alone for infants and children 1 to 6 years old would have exceeded the level of concern. Therefore, any drinking water exposure would have been of concern as well. [See Table 2. Chronic dietary food exposure and risk estimate from Dicofol (in food alone) located on page 17 of the NRDC nomination letter.]	Dicofol is an organochlorine pesticide used in agriculture, primarily on cotton and citrus crops. Approximately 860,000 pounds of active ingredient are used every year.EPA used its SCI-GROW model to estimate dicofol concentrations in groundwater and calculated a 90-day average peak concentration of 0.069 ppb. An overall mean surface water concentration of 0.5 ppb was estimated with the PRZM-EXAMS model. Both concentrations were below the Drinking Water Levels of Comparison (DWLOCs) for children and the general U.S. population for both acute and chronic exposure. However, there are some important shortcomings in EPA's assessment of dicofol exposure and risk. Another shortcoming in the EPA assessment is that the Agency relied on models to estimate environmental concentrations in surface and groundwater, but did not have a robust set of monitoring data. EPA should require the collection of surface and groundwater monitoring data in areas where dicofol is applied. The Agency should use these data to corroborate its exposure estimates and make a regulatory determination for dicofol under the SDWA.	None given
84-61-7	Dicyclohexyl phthalate	Natural Resources Defense Council	Phthalates are endocrine disruptors that have been found to cause developmental and reproductive abnormalities in animal studies. Furthermore, studies have found an association between phthalate metabolite concentrations and obesity, insulin resistance and thyroid hormone levels in humans. Stahlhut et al. (2007) analyzed urinary concentrations of seven phthalate metabolites in 1,443 adult men and found a statistically significant	Phthalates enter the environment as a result of releases from industrial facilities that manufacture or use these compounds, from waste disposal sites, through the use of phthalate-containing products by consumers and through discharge of municipal wastewaters containing phthalates. Phthalates have been found in numerous hazardous waste sites: diethyl phthalate (DEP) has been identified at	None given

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			positive association between concentrations of the metabolites MBzP, MEHHP, MEOHP and MEP and abdominal obesity. Concentrations of MBP,	348 sites, dibutyl phthalate (DBP) at 602 sites, dimethyl phthalate (DMP) at 167 sites, benzyl butyl phthalate (BzBP) at 413 sites,	
			MBzP and MEP were positively and significantly associated with insulin resistance. In a different study, baby boys exposed to the phthalates, DMP, DEP or BBP, in their mother's breast milk were found to have hormonal disturbances at 3 months of age. Studies have found that di-isononyl phthalate (DiNP) is an anti-androgenic endocrine disruptor with developmental and reproductive toxicity. Exposures in pregnant rats have been shown to adversely affect development of the male reproductive tract. Adverse effects include a cluster of outcomes that has been called "phthalate e syndrome" and includes underdeveloped or absent reproductive organs, retained nipples, cryptorchidism, decreased anogenital distance (AGD), hypospadias, and decreased or abnormal sperm. DINP does not bind to the androgen receptor and these effects are likely mediated through interference with testosterone synthesis. Phthalates are ubiquitous in the U.S. population. Although significant exposure can occur due to consumer products, the contribution of drinking water to overall exposure deserves to be examined. The NHANES measured the concentrations of 12 phthalate metabolites in the urine of over 2,500 children and adults. The highest average concentration was 163 ug/g creatinine for MEP, a metabolite of the plasticizer diethyl phthalate (see Table 3). [See the CDC report cited below.] The Centers for Disease Control and Prevention (CDC) did not report what percentage of samples had detectable concentrations of each of the metabolites, but nine of the metabolites were found in the urine of at least half of the individuals. [See Table 3. *CDC report of phthalate concentrations in urine; NHANES 1999 – 2000 and 2001 - 2002* located on page 27 of the NRDC Nomination Letter and Table 4 Frequency of detection of phthalate metabolites in human urine samples, United States located on pages 28 and 29 of the NRDC Nomination Letter.]	dicyclohexyl phthalate (DCHP) at one site, and di-n-octyl phthalate (DnOP) at 433 sites, among other phthalates. Phthalates have been detected in environmental water samples across the United States, which raises concerns about drinking water as a route of exposure. The maximum concentrations found for some of these phthalates are particularly alarming.	
84-66-2	Diethyl phthalate	Natural Resources Defense Council	Phthalates are endocrine disruptors that have been found to cause developmental and reproductive abnormalities in animal studies. Furthermore, studies have found an association between phthalate metabolite concentrations and obesity, insulin resistance and thyroid hormone levels in humans. Stahlhut et al. (2007) analyzed urinary concentrations of seven phthalate metabolites in 1,443 adult men and found a statistically significant positive association between concentrations of the metabolites MBzP, MEHHP, MEOHP and MEP and abdominal obesity. Concentrations of MBP, MBzP and MEP were positively and significantly associated with insulin resistance. In a different study, baby boys exposed to the phthalates, DMP, DEP or BBP, in their mother's breast milk were found to have hormonal disturbances at 3 months of age. Studies have found that di-isononyl phthalate (DiNP) is an anti-androgenic endocrine disruptor with developmental and reproductive toxicity. Exposures in pregnant rats have been shown to adversely affect development of the male reproductive tract. Adverse effects include a cluster of outcomes that has been called "phthalate e syndrome" and includes underdeveloped or absent reproductive organs, retained nipples,	Phthalates enter the environment as a result of releases from industrial facilities that manufacture or use these compounds, from waste disposal sites, through the use of phthalate-containing products by consumers and through discharge of municipal wastewaters containing phthalates. Phthalates have been found in numerous hazardous waste sites: diethyl phthalate (DEP) has been identified at 348 sites, dibutyl phthalate (DBP) at 602 sites, dimethyl phthalate (DMP) at 167 sites, benzyl butyl phthalate (BzBP) at 413 sites, dicyclohexyl phthalate (DCHP) at one site, and di-n-octyl phthalate (DnOP) at 433 sites, among other phthalates. Phthalates have been detected in environmental water samples across the United States, which raises concerns about drinking water as a route of exposure. The maximum concentrations found for some of these phthalates are particularly alarming. The following data were found in a search for stream water samples analyzed for phthalates in the EPA STORET database. Results are for the period January 2000 through June 2012:	None given

CASRN	Common Name	Nominator	Health Effects Information Provided with Nomination	Occurrence Information Provided with Nomination	Additional Information Provided with Nomination
			cryptorchidism, decreased anogenital distance (AGD), hypospadias, and decreased or abnormal sperm. DINP does not bind to the androgen receptor		
			and these effects are likely mediated through interference with testosterone synthesis. Phthalates are ubiquitous in the U.S. population. Although significant exposure can occur due to consumer products, the contribution of drinking water to overall exposure deserves to be examined. The NHANES measured the concentrations of 12 phthalate metabolites in the urine of over 2,500 children and adults. The highest average concentration was 163 ug/g creatinine for MEP, a metabolite of the plasticizer diethyl phthalate (see Table 3). [See the CDC report cited below.] The Centers for Disease Control and Prevention (CDC) did not report what percentage of samples had detectable concentrations of each of the metabolites, but nine of the metabolites were found in the urine of at least half of the individuals. [See Table 3. CDC report of phthalate concentrations in urine; NHANES 1999 – 2000 and 2001 - 2002 located on page 27 of the NRDC Nomination Letter and Table 4 Frequency of detection of phthalate metabolites in human urine samples, United States located on pages 28 and 29 of the NRDC Nomination Letter.]	Diethyl phthalate (DEP) was detected in 840 (20.1%) of the 4174 stream water samples analyzed. The maximum concentration found was 1000 ug/L.	
28553-12-0	Di-isononyl phthalate	Natural Resources Defense Council	Phthalates are endocrine disruptors that have been found to cause developmental and reproductive abnormalities in animal studies. Furthermore, studies have found an association between phthalate metabolite concentrations and obesity, insulin resistance and thyroid hormone levels in humans. Stahlhut et al. (2007) analyzed urinary concentrations of seven phthalate metabolites in 1,443 adult men and found a statistically significant positive association between concentrations of the metabolites MBzP, MEHHP, MEOHP and MEP and abdominal obesity. Concentrations of MBP, MBzP and MEP were positively and significantly associated with insulin resistance. In a different study, baby boys exposed to the phthalates, DMP, DEP or BBP, in their mother's breast milk were found to have hormonal disturbances at 3 months of age. Studies have found that di-isononyl phthalate (DiNP) is an anti-androgenic endocrine disruptor with developmental and reproductive toxicity. Exposures in pregnant rats have been shown to adversely affect development of the male reproductive tract. Adverse effects include a cluster of outcomes that has been called "phthalate e syndrome" and includes underdeveloped or absent reproductive organs, retained nipples, cryptorchidism, decreased anogenital distance (AGD), hypospadias, and decreased or abnormal sperm. DINP does not bind to the androgen receptor and these effects are likely mediated through interference with testosterone synthesis.	Phthalates enter the environment as a result of releases from industrial facilities that manufacture or use these compounds, from waste disposal sites, through the use of phthalate-containing products by consumers and through discharge of municipal wastewaters containing phthalates. Phthalates have been found in numerous hazardous waste sites: diethyl phthalate (DEP) has been identified at 348 sites, dibutyl phthalate (DBP) at 602 sites, dimethyl phthalate (DMP) at 167 sites, benzyl butyl phthalate (BzBP) at 413 sites, dicyclohexyl phthalate (DCHP) at one site, and di-n-octyl phthalate (DnOP) at 433 sites, among other phthalates. Phthalates have been detected in environmental water samples across the United States, which raises concerns about drinking water as a route of exposure. The maximum concentrations found for some of these phthalates are particularly alarming. The following data were found in a search for stream water samples analyzed for phthalates in the EPA STORET database. Results are for the period January 2000 through June 2012: Di-isononyl phthalate (DiNP) – No data available. [The Institute for Health and Consumer Protection (IHCP) of the European Chemicals Bureau estimated a half life in surface water for DINP of 50 days. According to the IHCP, 7 percent of the DINP in the influent in sewage treatment plants will be released in the effluent. See European	None given

CASRN	Common Name	Nominator	Health Effects Information Provided with Nomination	Occurrence Information Provided with Nomination	Additional Information Provided with Nomination
			Phthalates are ubiquitous in the U.S. population. Although significant exposure can occur due to consumer products, the contribution of drinking water to overall exposure deserves to be examined. The NHANES measured the concentrations of 12 phthalate metabolites in the urine of over 2,500 children and adults. The highest average concentration was 163 ug/g creatinine for MEP, a metabolite of the plasticizer diethyl phthalate (see Table 3). [See the CDC report cited below.] The Centers for Disease Control and Prevention	Commission Joint Research Centre, Institute for Health and Consumer Protection, 1,2-Benzenedicarboxylic acid, Di-C8-10-Branched Alkyl Esters, C9-Rich and Di-"Isononyl" Phthalate (DINP), CAS Nos: 68515-48-0 and 28553-12-0, EINECS Nos: 271-090-9 and 249-079-5, Summary Risk Assessment Report, 2003. http://ecb.jrc.it/DOCUMENTS/Existing-	
			(CDC) did not report what percentage of samples had detectable concentrations of each of the metabolites, but nine of the metabolites were found in the urine of at least half of the individuals. [See Table 3. CDC report of phthalate concentrations in urine; NHANES 1999 – 2000 and 2001 - 2002 located on page 27 of the NRDC Nomination Letter and Table 4 Frequency of detection of phthalate metabolites in human urine samples, United States located on pages 28 and 29 of the NRDC Nomination Letter.]	Chemicals/RISK_ASSESSMENT/SUMMARY/dinpsum046.pdf. Given the widespread use and high production volumes of DINP, these releases could pose risks for water quality.]	
131-11-3	Dimethyl phthalate	Natural Resources Defense Council	Phthalates are endocrine disruptors that have been found to cause developmental and reproductive abnormalities in animal studies. Furthermore, studies have found an association between phthalate metabolite concentrations and obesity, insulin resistance and thyroid hormone levels in humans. Stahlhut et al. (2007) analyzed urinary concentrations of seven phthalate metabolites in 1,443 adult men and found a statistically significant positive association between concentrations of the metabolites MBzP, MEHHP, MEOHP and MEP and abdominal obesity. Concentrations of MBP, MBzP and MEP were positively and significantly associated with insulin resistance. In a different study, baby boys exposed to the phthalates, DMP, DEP or BBP, in their mother's breast milk were found to have hormonal disturbances at 3 months of age. Studies have found that di-isononyl phthalate (DiNP) is an anti-androgenic endocrine disruptor with developmental and reproductive toxicity. Exposures in pregnant rats have been shown to adversely affect development of the male reproductive tract. Adverse effects include a cluster of outcomes that has been called "phthalate e syndrome" and includes underdeveloped or absent reproductive organs, retained nipples, cryptorchidism, decreased anogenital distance (AGD), hypospadias, and decreased or abnormal sperm. DINP does not bind to the androgen receptor and these effects are likely mediated through interference with testosterone synthesis. Phthalates are ubiquitous in the U.S. population. Although significant exposure can occur due to consumer products, the contribution of drinking water to overall exposure deserves to be examined. The NHANES measured the concentrations of 12 phthalate metabolites in the urine of over 2,500 children and adults. The highest average concentration was 163 ug/g creatinine for MEP, a metabolite of the plasticizer diethyl phthalate (see Table 3). [See the CDC report cited below.] The Centers for Disease Control and Prevention (CDC) did not report what percentage of samples had detectable concentrations of each of the metabolites, but nine of the metabolites were found in the urine of at least half of the individuals. [See Table 3. CDC report	Phthalates enter the environment as a result of releases from industrial facilities that manufacture or use these compounds, from waste disposal sites, through the use of phthalate-containing products by consumers and through discharge of municipal wastewaters containing phthalates. Phthalates have been found in numerous hazardous waste sites: diethyl phthalate (DEP) has been identified at 348 sites, dibutyl phthalate (DBP) at 602 sites, dimethyl phthalate (DMP) at 167 sites, benzyl butyl phthalate (BzBP) at 413 sites, dicyclohexyl phthalate (DCHP) at one site, and di-n-octyl phthalate (DnOP) at 433 sites, among other phthalates. Phthalates have been detected in environmental water samples across the United States, which raises concerns about drinking water as a route of exposure. The maximum concentrations found for some of these phthalates are particularly alarming. The following data were found in a search for stream water samples analyzed for phthalates in the EPA STORET database. Results are for the period January 2000 through June 2012: Dimethyl phthalate (DMP) was present in 587 (15.9%) of 3687 stream water samples, with a maximum of 2,500 ug/L.	None given

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			of phthalate concentrations in urine; NHANES 1999 – 2000 and 2001 - 2002 located on page 27 of the NRDC Nomination Letter and Table 4 Frequency of detection of phthalate metabolites in human urine samples, United States located on pages 28 and 29 of the NRDC Nomination Letter.]		
117-84-0	Di-n-octyl phthalate	Natural Resources Defense Council	Phthalates are endocrine disruptors that have been found to cause developmental and reproductive abnormalities in animal studies. Furthermore, studies have found an association between phthalate metabolite concentrations and obesity, insulin resistance and thyroid hormone levels in humans. Stahlhut et al. (2007) analyzed urinary concentrations of seven phthalate metabolites in 1,443 adult men and found a statistically significant positive association between concentrations of the metabolites MBzP, MEHHP, MEOHP and MEP and abdominal obesity. Concentrations of MBP, MBzP and MEP were positively and significantly associated with insulin resistance. In a different study, baby boys exposed to the phthalates, DMP, DEP or BBP, in their mother's breast milk were found to have hormonal disturbances at 3 months of age. Studies have found that di-isononyl phthalate (DiNP) is an anti-androgenic endocrine disruptor with developmental and reproductive toxicity. Exposures in pregnant rats have been shown to adversely affect development of the male reproductive tract. Adverse effects include a cluster of outcomes that has been called "phthalate e syndrome" and includes underdeveloped or absent reproductive organs, retained nipples, cryptorchidism, decreased anogenital distance (AGD), hypospadias, and decreased or abnormal sperm. DINP does not bind to the androgen receptor and these effects are likely mediated through interference with testosterone synthesis. Phthalates are ubiquitous in the U.S. population. Although significant exposure can occur due to consumer products, the contribution of drinking water to overall exposure deserves to be examined. The NHANES measured the concentrations of 12 phthalate metabolites in the urine of over 2,500 children and adults. The highest average concentration was 163 ug/g creatinine for MEP, a metabolite of the plasticizer diethyl phthalate (see Table 3). [See the CDC report cited below.] The Centers for Disease Control and Prevention (CDC) did not report what percentage of samples had detectable concentrations of each of the metabolites, but nine of the metabolites were found in the urine of at least half of the individuals. [See Table 3. CDC report of phthalate concentrations in urine; NHANES 1999 – 2000 and 2001 - 2002 located on page 27 of the NRDC Nomination Letter and Table 4 Frequency of detection of phthalate metabolites in human urine samples, United States located on pages 28 and 29 of the NRDC Nomination Letter.]	Phthalates enter the environment as a result of releases from industrial facilities that manufacture or use these compounds, from waste disposal sites, through the use of phthalate-containing products by consumers and through discharge of municipal wastewaters containing phthalates. Phthalates have been found in numerous hazardous waste sites: diethyl phthalate (DEP) has been identified at 348 sites, dibutyl phthalate (DBP) at 602 sites, dimethyl phthalate (DMP) at 167 sites, benzyl butyl phthalate (BzBP) at 413 sites, dicyclohexyl phthalate (DCHP) at one site, and di-n-octyl phthalate (DnOP) at 433 sites, among other phthalates. Phthalates have been detected in environmental water samples across the United States, which raises concerns about drinking water as a route of exposure. The maximum concentrations found for some of these phthalates are particularly alarming. The following data were found in a search for stream water samples analyzed for phthalates in the EPA STORET database. Results are for the period January 2000 through June 2012: Di-n-octyl phthalate (DnOP) was found in 129 out of 2469 stream water samples, with a maximum concentration of 20 ug/L.	None given
115-29-7	Endosulfan	Natural Resources Defense Council	Endosulfan is an organochlorine insecticide and acaricide. Technical grade endosulfan is made of both alpha and beta stereoisomers whose toxicity is manifested through blockage of inhibitory GABA (gamma amino butyric acid) gated chloride channels, resulting in over-stimulation of the central nervous	In 2010, EPA's pesticide office announced that it was cancelling all uses of endosulfan in the U.S. However, because endosulfan is persistent, past uses of this pesticide continue to contaminate our water. According to the EFED risk assessment for the RED on	None given

CASRN	Common Name	Nominator	Health Effects Information Provided with Nomination	Occurrence Information Provided with Nomination	Additional Information Provided with Nomination
			system. Endosulfan is a recognized neurotoxin and endocrine disruptor, making even extremely low-dose exposures of very great concern, especially to vulnerable populations such as children and fetuses. Endosulfan is similar in its acute oral toxicity to the related insecticides aldrin and dieldrin, except that it is slightly more toxic than these substances in female laboratory animals. Inhalation of endosulfan dust by humans has been	endosulfan, monitoring data show widespread contamination of surface water. EPA modeled surface water contamination and calculated acute estimated environmental concentrations ranging from 4.49 ppb to 23.86 ppb. Chronic EECs ranged from 0.53 ppb to 1.5 ppb. The acute and chronic EEC for endosulfan in groundwater was 0.012 ppb. EPA concluded in the RED that "residues of endosulfan in drinking water are of concern" for acute exposure for infants less than	
			associated with slight nausea, confusion, excitement, flushing, and dry mouth. Nine employees who had been working with 50-percent water-wettable endosulfan powder for only a few days had convulsions. Endosulfan is a significant endocrine disruptor and reproductive toxicant. This pesticide increases the rate of testosterone breakdown and excretion. In immature rats, endosulfan causes significant dose-related decreases in sperm counts, and causes sperm deformities at low exposure levels. In fish, endosulfan elevates levels of thyroxine and suppresses levels of triiodothyronine, probably by inhibiting the conversion of thyroxine to T3. The developing brain is potentially most severely affected by this pesticide via altered levels of critical neurotransmitters such as dopamine, noradrenaline and serotonin; the altered neurotransmitter levels are associated with deficits in learning and memory.	one year old and for children 1-6 years old. EPA determined that exposure from food alone created risks of concern for children 1 to 6 years old and set a DWLOC of zero ppb for this population.	
2164-17-2	Fluometuron	AWWA	None given	None given	None given
319-84-6	Hexachlorocyclohexane (alpha isomer)	AWWA	None given	None given	None given
165800-03-3	Linezolid	Natural Resources Defense Council	Widespread exposure to antibiotics is contributing to the growth of bacterial resistance, and this problem is of grave concern. In the past several decades almost every bacteria that can cause infections in humans has developed resistance to at least one antibiotic, and some are resistant to multiple antibiotics. The Centers for Disease Control and Prevention (CDC) have identified antibiotic resistance as one of the most pressing public health problems to face our nation. Infections caused by bacteria with resistance to at least one antibiotic have been estimated to kill over 60,000 hospitalized patients each year. Antibiotic resistance is caused by a number of factors including repeated and improper use of antibiotics in both humans and animals. Scientists also agree that exposure to low levels of antibiotics actually promotes bacterial resistance by exerting selective pressure for genes that promote resistance. Linezolid resistance in Staphylococcus aureus was reported in 2003. Community-acquired MRSA (CA-MRSA) has now emerged as an epidemic that is responsible for rapidly progressive, fatal diseases including necrotizing pneumonia, severe sepsis and necrotizing fasciitis. Outbreaks of CA-MRSA infections have been reported in correctional facilities, among athletic teams, among military recruits, in newborn nurseries, and among active homosexual men. Therefore, linezolid must be included on the CCL4.	Antibiotics are found in wastewater because the body does not completely metabolize all drugs, so both the metabolized and unmetabolized drug are excreted by humans into wastewater. For example, when amoxicillin is ingested, 60-75% of the antibiotic is excreted unchanged into the urine. This antibiotic, now in the environment, may encounter other bacteria and promote resistance. It is unknown how much of an impact current low levels of antibiotics in drinking water are having on the problem of bacterial resistance. However, the potential has been recognized for many years.	None given

CASRN	Common Name	Nominator	Health Effects Information Provided with Nomination	Occurrence Information Provided with Nomination	Additional Information Provided with Nomination
330-55-2	Linuron	Natural Resources Defense Council	Linuron (CAS # 330-55-2) is an urea-based herbicide used primarily on soybeans (79 percent of usage). It has been shown to cause non-malignant testicular and liver tumors in animals. Investigation of the testicular tumors revealed that this herbicide acts by blocking the function of male androgens. In animals, at relatively low doses, linuron is a recognized anti-androgen. This chemical has been shown in laboratory studies to decrease male sex organ weights, cause testicular atrophy, delay puberty, and increase estrogen levels	About 400,000 pounds of linuron are used in U.S. agriculture each year. This herbicide persists for 1-5 months in soil, and has been shown to run off of fields into surface and groundwater supplies. EPA concluded in its Reregistration Eligibility Decision (RED) that linuron exceeded the Levels of Concern (LOC) for groundwater quality. EPA also expressed "moderate concerns" for drinking water supply systems relying on surface water sources.	Since linuron is not regulated under the Safe Drinking Water Act (SDWA), water supply systems are not required to sample or analyze for it. This is a particular problem because EPA admits that drinking water
			in males. Casting further doubt on EPA's estimates of the risks of linuron in drinking water sources is the fact that the model used for surface water assessment was not tested against any data whatsoever. The exposure estimates (18 ppb) for infants and children exceed EPA's chronic DWLOC (6 ppb) by three-fold. This result is of particular concern in light of the serious flaws in the drinking water risk assessment that conspire to underestimate the actual risk. EPA admits that "residues of linuron and its metabolites in drinking water may represent a chronic human health risk..."	Several factors in EPA's drinking water exposure assessment raise concerns about groundwater contamination. In the groundwater portion of the assessment, data were present for only four states: Georgia, Missouri, Virginia, and Wisconsin. In Georgia linuron was found in groundwater in concentrations up to 5 ppb. EPA later cast doubt on the reliability of the data and removed it from consideration in the final RED, basing its decision on new information received from the State of Georgia. Valid groundwater detections in Missouri (up to 1.9 ppb), Virginia (up to 1.31 ppb in 4 of 8 wells) and Wisconsin (up to 2.7 ppb) may seriously underestimate linuron levels throughout the country because these three states are not among the 16-20 states where linuron is most heavily used. The sixteen states listed on page 3 of EPA's Overview of Linuron Risk Assessment appear to account for well over 80% of linuron use in the United States, so the complete absence of any data on groundwater in any of these states is a critical data gap. The USGS has also reported on areas where linuron is most heavily used on a per-acre basis. The USGS maps indicate that Indiana, Ohio, Michigan, Delaware, and Maryland are heavy use states. These states are not among the ones from which groundwater data are available. Strangely, only one of these (Michigan) is listed by EPA as among heavy use states.	treatment is unlikely to remove linuron and its degradates. The Agency must move rapidly to collect more data on linuron in water and must make a high priority of regulating linuron under the SDWA.
121-75-5	Malathion	AWWA	None given	None given	None given
7439-96-5	Manganese	Massachusetts Department of Environmental Protection	There has been an accumulating body of work since US EPA's last review of manganese suggesting an association between drinking water exposures in school age children and a variety of subtle neurological effects (see selected references in Appendix B). Effects in one of the more recent studies have been seen at manganese water concentrations below the current US EPA lifetime Health Advisory (HA) value, suggesting that the validity of that research finding be critically examined and that possibly the basis for the current HA be revisited. The effect — exposure duration relationship deserves attention in view of the fact that some children may have altered neurological function after exposures to manganese in water at concentrations greater than the lifetime HA level but after less than lifetime durations of exposure. New federal guidance could contribute towards providing protective guidance for sensitive subgroups for less than lifetime exposures.	Occurrence: From sampling across Massachusetts, we have manganese in groundwaters serving as sources of drinking water for public and private water supplies at concentrations above current health-based guidance concentrations (see Appendix A for examples);	We see a clear need for national level drinking water guidance for manganese which reflects emerging science. There is currently no clear, up-to-date, national uniform message about the health risks from ingestion of manganese in drinking water, resulting in states having to handle manganese issues individually. Our experience has been that manganese in drinking water is not perceived as a potential health issue, but rather purely an aesthetic one. We believe this to not be the case and strongly support the

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			Identifying protective toxicity values for ingestion of manganese is particularly challenging because it is an essential element and there appears to be differential bioavailability of manganese between water and food stuffs. Present drinking water guidance (applicable to the entire population except for infants) is based upon a safe, no effect level of manganese derived from adult dietary intake studies. The recent studies with children suggest that they		inclusion of manganese on the CCL4 list. Doing so would raise the prominence of this issue. Given the complexity of manganese's toxicity (exposure route and chemical form specific, essentiality versus toxicity,
			should be evaluated as a susceptible subgroup of the population and that toxicity should be factored into setting manganese drinking water exposure limits, possibly along with considerations of essentiality. While infants have been singled out as of special concern by US EPA in its existing HA, we are especially concerned about bottle-fed infants due to their apparently low nutritional requirement for Mn in early life, their immature homeostatic mechanism for controlling Mn absorption and excretion, and potentially high levels of Mn in infant formulas.		differential life stage, susceptibility, emerging science not currently reflected in US EPA guidance), we believe that an examination of manganese toxicity for drinking water is in order. The current US EPA HA gives no guidance to the states in terms of what advice they should offer to regulated entities or private well owners, or what regulatory stance they should take with exceedances of HA levels, leaving states to craft their own positions on this issue. National leadership with a national primary drinking water standard would provide some uniformity in how manganese health risks are communicated and dealt with.
7439-96-5	Manganese	Minnesota Department of Health	Since US EPA's last review of manganese, a body of research has accumulated suggesting an association between drinking water exposures in school age children and a variety of subtle neurological effects (see manganese references in Appendix B). In two recent epidemiology studies, effects have been seen at manganese water concentrations below the current US EPA lifetime HA value, suggesting that the basis for the current HA should be revisited (Bouchard et al., 2011, Khan et al., 2011). The relationship between exposure duration and health effects deserves attention in light of the fact that some children have exhibited altered neurological function after exposures to manganese in water at concentrations greater than the lifetime HA level but after less than lifetime durations. New federal guidance could contribute towards providing protective guidance for sensitive subgroups for less than lifetime exposures.	Manganese is commonly detected in groundwater in the United States at concentrations greater than the lifetime Health Advisory (HA) value of 300 ug/L. Twelve percent of 4,976 groundwater samples taken throughout the United States by the US Geological Survey from 1992 – 2003 exceeded the HA for manganese (Ayotte, Gronberg, & Apodaca, 2011). Manganese is found in groundwater throughout Minnesota, including groundwater that serves as source of drinking water for public and private water supplies, at concentrations above current health-based guidance concentrations (See Appendix A - Minnesota Department of Health nomination letter). Appendix A. Occurrence Information for Manganese in Groundwater in Minnesota Over 4000 groundwater well samples were collected from throughout Minnesota and analyzed for manganese. Almost two-thirds (61%) of wells which might serve as water sources for public supplies or private residences had manganese concentrations greater than the	We are nominating manganese for CCL4 because it is frequently detected in public and private wells, and there is some recent evidence of health effects at concentrations below the current EPA health advisory value. Also, we have concerns about manganese exposures among sensitive populations such as infants and children, and for less than lifetime exposure durations. The current US EPA HA gives no guidance to the states in terms of what advice they should offer to regulated entities or private well owners, or what regulatory stance they should take when of

CASRN	Common Name	Nominator	Health Effects Information Provided with Nomination	Occurrence Information Provided with Nomination	Additional Information Provided with Nomination
			Identifying protective toxicity values for ingestion of manganese is particularly challenging because it is an essential element and there appears to be differential bioavailability of manganese between water and foodstuffs. Present drinking water guidance (applicable to the entire population except for infants) is based upon a safe, no effect level of manganese derived from adult dietary intake studies. The recent studies with children suggest that they should be evaluated as a susceptible subgroup of the population.	Secondary Maximum Contaminant Level of 0.050 mg/L. Twenty-one percent had concentrations greater than the lifetime health advisory value (See Table 1- Minnesota Department of Health nomination letter).	HA levels are exceeded, leaving states to craft their own positions on this issue. National leadership with a national primary drinking water standard would provide some uniformity in how
			While infants have been singled out as a special concern by US EPA in its existing HA, we are especially concerned about bottle-fed infants due to their apparently low nutritional requirement for Mn in early life, their naturally high blood Mn concentrations at birth, their immature homeostatic mechanism for controlling Mn absorption and excretion, and potentially high levels of Mn in infant formulas.		manganese health risks are communicated and dealt with.
7439-96-5	Manganese	NJ Department of Environmental Protection	Manganese in drinking water is of current interest to a number of states. State and EPA FSTRAC members have formed a partner group to evaluate recent health effects information relevant to drinking water exposure to manganese. The current EPA Health Advisory for manganese is based on the assumption that manganese exposure from drinking water is much lower than from the diet, and is not based on health effects. This manganese Health Advisory is several-fold higher than the secondary standard for manganese that is based on aesthetic effects. However, manganese occurs in NJ and other states in both public water supplies and private wells at levels which result in much higher exposures than those assumed by EPA in their comparison to dietary exposures. Also, several recent studies suggest that manganese by the oral route may cause neurodevelopmental effects. There is a need for an updated health assessment for manganese in drinking water based on current health effects data. This health assessment could be used for an updated Health Advisory or as the basis for a proposed MCLG.	None given	None given
61-32-5	Methicillin	Natural Resources Defense Council	Widespread exposure to antibiotics is contributing to the growth of bacterial resistance, and this problem is of grave concern. In the past several decades almost every bacteria that can cause infections in humans has developed resistance to at least one antibiotic, and some are resistant to multiple antibiotics. The Centers for Disease Control and Prevention (CDC) have identified antibiotic resistance as one of the most pressing public health problems to face our nation. Infections caused by bacteria with resistance to at least one antibiotic have been estimated to kill over 60,000 hospitalized patients each year. Antibiotic resistance is caused by a number of factors including repeated and improper use of antibiotics in both humans and animals. Scientists also agree that exposure to low levels of antibiotics actually promotes bacterial resistance by exerting selective pressure for genes that promote resistance. Beta-lactam antibiotics are a broad class of antibiotics which include penicillin derivatives, cephalosporins, monobactams, carbapenems and Beta-lactamase inhibitors. Methicillin, a form of penicillin, had been relied upon as an common	Antibiotics are found in wastewater because the body does not completely metabolize all drugs, so both the metabolized and unmetabolized drug are excreted by humans into wastewater. For example, when amoxicillin is ingested, 60-75% of the antibiotic is excreted unchanged into the urine. This antibiotic, now in the environment, may encounter other bacteria and promote resistance. It is unknown how much of an impact current low levels of antibiotics in drinking water are having on the problem of bacterial resistance. However, the potential has been recognized for many years.	None given

CASRN	Common Name	Nominator	Health Effects Information Provided with Nomination	Occurrence Information Provided with Nomination	Additional Information Provided with Nomination
			effective treatment for Staphylococcus aureus infections but now many strains of S. aureus bacteria are resistant to methicillin (MRSA or methicillin-resistant Staphylococcus aureus.) Unfortunately, MRSA is resistant to much of the entire class of penicillin-like antibiotics called beta-lactams. Therefore, EPA must include penicillin, amoxicillin, oxacillin and methicillin on the CCL4.		
298-00-0	Methyl parathion	AWWA	None given	None given	None given
1634-04-4	Methyl tertiary butyl ether	NJ Department of Environmental Protection	A recent chronic cancer bioassay of MTBE by the drinking water exposure route in rats (Dodd et al., 2011) should be considered by USEPA. Previously, a chronic inhalation study in mice and rats (an exposure route that is not as relevant to drinking water exposure, Bird et al., 1997) and an oral gavage study in rats from the Ramazzini Institute in Italy, which USEPA has decided not to consider this study because of issues related to the pathology evaluations (http://www.epa.gov/iris/ramazzini.htm) were the only studies available as the basis for the assessment of the carcinogenic potential of MTBE in drinking water. The recent Dodd et al. (2011) study suggests that MTBE in drinking water may cause brain tumors in rats and should be considered by EPA.	None given	None given
101043-37-2	Microcystin-LR	Minnesota Department of Health	Liver toxicity has long been identified as the most sensitive toxicological endpoint for microcystin-LR. As part of the CCL3 process, EPA derived a draft RfD of 0.000003 mg/kg-d based on hepatotoxicity, using an estimated NOAEL of 3 ug/kg-d in mice from ingestion of water containing 20 ug/L microcystin-LR (Ueno et al., 1999). However, a more recent study reports male reproductive effects in mice exposed to lower doses of microcystin-LR in drinking water. (Chen et al., 2011). Significant decreases in testosterone and sperm motility and count were observed at doses as low as approximately 0.64 ug/kg-d. (This dose is estimated based on ingestion of water containing 3 ug/L microcystin-LR.) In addition to the Chen et al. study, there are a limited number of intraperitoneal injection studies in mice, rats and rabbits and in vitro studies in Sertoli cells which reported male reproductive effects on sperm, testes and Sertoli cells (Li, Y., J. Sheng, et al., 2008; Liu, Y., P. Xie, et al., 2010; Wang, X., F. Ying, et al.,2012). A recent oral study reported altered reproductive function and disruption in spermatogenesis in medaka fish (Trinchet, I., C. Djediat, et al., 2011) Because the Chen et al. study identifies a new toxicological endpoint at a dose level nearly five-fold lower than that used in EPA's draft RfD, and some supporting data also indicate potential reproductive toxicity, we believe microcystin-LR is worthy of consideration for updated guidance at the federal level. Because of the episodic nature of microcystin "outbreaks" in surface water (see Occurrence section), we believe that short-term exposures and effects should be given special consideration for this chemical.	Microcystin –LR is largely a surface water contaminant, and is commonly detected in lakes in temperate climates (Ohio Environmental Protection Agency, 2012; Boyer et al., 2005; Graham et al., 2004). Surface water is used as a drinking water source in many locations in the United States. Contamination of a surface water body with microcystin-LR is likely to be episodic in nature, exhibiting both seasonal variation (Billam et al., 2006) and aquatic concentrations that are highly sensitive to total phosphorus, total nitrogen, and other chemical parameters (Graham et al., 2004). Therefore, we believe that short-term exposures and effects should be given special consideration for this chemical. The use of "recycled" wastewater for drinking water is increasingly being viewed as a water supply management option in some areas of the United States (City of San Diego, 2012; Barringer, 2012). Wastewater, including treated wastewater, provides nutrients that can promote the growth of cyanobacteria in surface water (Ho et al., 2010). This indicates a potential future route of microcystin exposure via drinking water.	None given
101043-37-2	Microcystin-LR	Minnesota Department of Health	Liver toxicity has long been identified as the most sensitive toxicological endpoint for microcystin-LR. As part of the CCL3 process, EPA derived a draft RfD of 0.000003 mg/kg-d based on hepatotoxicity, using an estimated NOAEL of 3 ug/kg-d in mice from ingestion of water containing 20 ug/L microcystin-LR (Ueno et al., 1999). However, a more recent study reports male reproductive effects in mice exposed to lower doses of microcystin-LR in drinking water. (Chen et al.,	Microcystin –LR is largely a surface water contaminant, and is commonly detected in lakes in temperate climates (Ohio Environmental Protection Agency, 2012; Boyer et al., 2005; Graham et al., 2004). Surface water is used as a drinking water source in many locations in the United States. Contamination of a surface water body with microcystin-LR is likely to be episodic in nature, exhibiting both seasonal variation (Billam et al.,	We are nominating microcystin-LR for CCL4 because its oral RfD, while already low, may need to be revised downward in light of new toxicological data. Also, there is some concern that the human exposure to microcystin-LR in drinking water may

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			2011). Significant decreases in testosterone and sperm motility and count were observed at doses as low as approximately 0.64 ug/kg-d. (This dose is estimated based on ingestion of water containing 3 ug/L microcystin-LR.) In addition to the Chen et al. study, there are a limited number of intraperitoneal injection studies in mice, rats and rabbits and in vitro studies in	2006) and aquatic concentrations that are highly sensitive to total phosphorus, total nitrogen, and other chemical parameters (Graham et al., 2004). Therefore, we believe that short-term exposures and effects should be given special consideration for this chemical. The use of "recycled" wastewater for drinking water is increasingly	increase due to more favorable conditions for algal growth in lakes and reservoirs (i.e., nutrients, temperature, wastewater releases), and new
			Sertoli cells which reported male reproductive effects on sperm, testes and Sertoli cells (Li, Y., J. Sheng, et al., 2008; Liu, Y., P. Xie, et al., 2010; Wang, X., F. Ying, et al.,2012). A recent oral study reported altered reproductive function and disruption in spermatogenesis in medaka fish (Trinchet, I., C. Djediat, et al., 2011) Because the Chen et al. study identifies a new toxicological endpoint at a dose level nearly five-fold lower than that used in EPA's draft RfD, and some supporting data also indicate potential reproductive toxicity, we believe microcystin-LR is worthy of consideration for updated guidance at the federal level.	being viewed as a water supply management option in some areas of the United States (City of San Diego, 2012; Barringer, 2012). Wastewater, including treated wastewater, provides nutrients that can promote the growth of cyanobacteria in surface water (Ho et al., 2010). This indicates a potential future route of microcystin exposure via drinking water.	efforts to recycle wastewater into drinking water.
25154-52-3[2]	Nonylphenol	Natural Resources Defense Council	Alkylphenols were first reported to be estrogenic in the 1930s. In 1991, publication of the effects of nonylphenol on cultured human breast cancer cells led to health concerns. Estrogenic effects have also been shown in the mouse. Estrogenic effects are present at tissue concentrations of 0.1 µM for octylphenol and 1 µM for nonylphenol. A recombinant yeast screen using the human estrogen receptor has shown similar results.	An estimated 450,000,000 pounds of alkylphenol polyethoxylates (APEs) are produced annually in the United States, and about half that amount is estimated to be released to wastewater. Alkylphenol polyethoxylates do not break down effectively in sewage treatment plants or in the environment. Instead they degrade to alkylphenols and alkylphenol ethoxylates, which persist for longer. Nonylphenol and its ethoxylates, and other alkylphenols, have been detected in wastewater and in waterways.	None given
9016-45-9	Nonylphenol ethoxylate	Natural Resources Defense Council	Alkylphenols were first reported to be estrogenic in the 1930s. In 1991, publication of the effects of nonylphenol on cultured human breast cancer cells led to health concerns. Estrogenic effects have also been shown in the mouse. Estrogenic effects are present at tissue concentrations of 0.1 µM for octylphenol and 1 µM for nonylphenol. A recombinant yeast screen using the human estrogen receptor has shown similar results.	An estimated 450,000,000 pounds of alkylphenol polyethoxylates (APEs) are produced annually in the United States, and about half that amount is estimated to be released to wastewater. Alkylphenol polyethoxylates do not break down effectively in sewage treatment plants or in the environment. Instead they degrade to alkylphenols and alkylphenol ethoxylates, which persist for longer. Nonylphenol and its ethoxylates, and other alkylphenols, have been detected in wastewater and in waterways.	None given
27193-28-8	Octylphenol	Natural Resources Defense Council	Alkylphenols were first reported to be estrogenic in the 1930s. In 1991, publication of the effects of nonylphenol on cultured human breast cancer cells led to health concerns. Estrogenic effects have also been shown in the mouse. Estrogenic effects are present at tissue concentrations of 0.1 µM for octylphenol and 1 µM for nonylphenol. A recombinant yeast screen using the human estrogen receptor has shown similar results.	An estimated 450,000,000 pounds of alkylphenol polyethoxylates (APEs) are produced annually in the United States, and about half that amount is estimated to be released to wastewater. Alkylphenol polyethoxylates do not break down effectively in sewage treatment plants or in the environment. Instead they degrade to alkylphenols and alkylphenol ethoxylates, which persist for longer. Nonylphenol and its ethoxylates, and other alkylphenols, have been detected in wastewater and in waterways.	None given

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9036-19-5	Octylphenol ethoxylate	Natural Resources Defense Council	Alkylphenols were first reported to be estrogenic in the 1930s. In 1991, publication of the effects of nonylphenol on cultured human breast cancer cells led to health concerns. Estrogenic effects have also been shown in the mouse. Estrogenic effects are present at tissue concentrations of 0.1 µM for octylphenol and 1 µM for nonylphenol. A recombinant yeast screen using the human estrogen receptor has shown similar results.	An estimated 450,000,000 pounds of alkylphenol polyethoxylates (APEs) are produced annually in the United States, and about half that amount is estimated to be released to wastewater. Alkylphenol polyethoxylates do not break down effectively in sewage treatment plants or in the environment. Instead they degrade to alkylphenols and alkylphenol ethoxylates, which persist for longer. Nonylphenol and its ethoxylates, and other alkylphenols, have been detected in wastewater and in waterways.	None given
66-79-5	Oxacillin	Natural Resources Defense Council	Widespread exposure to antibiotics is contributing to the growth of bacterial resistance, and this problem is of grave concern. In the past several decades almost every bacteria that can cause infections in humans has developed resistance to at least one antibiotic, and some are resistant to multiple antibiotics. The Centers for Disease Control and Prevention (CDC) have identified antibiotic resistance as one of the most pressing public health problems to face our nation. Infections caused by bacteria with resistance to at least one antibiotic have been estimated to kill over 60,000 hospitalized patients each year. Antibiotic resistance is caused by a number of factors including repeated and improper use of antibiotics in both humans and animals. Scientists also agree that exposure to low levels of antibiotics actually promotes bacterial resistance by exerting selective pressure for genes that promote resistance. Beta-lactam antibiotics are a broad class of antibiotics which include penicillin derivatives, cephalosporins, monobactams, carbapenems and Beta-lactamase inhibitors. Methicillin, a form of penicillin, had been relied upon as an common effective treatment for Staphylococcus aureus infections but now many strains of S. aureus bacteria are resistant to methicillin (MRSA or methicillin-resistant Staphylococcus aureus.) Unfortunately, MRSA is resistant to much of the entire class of penicillin-like antibiotics called beta-lactams. Therefore, EPA must include penicillin, amoxicillin, oxacillin and methicillin on the CCL4.	Antibiotics are found in wastewater because the body does not completely metabolize all drugs, so both the metabolized and unmetabolized drug are excreted by humans into wastewater. For example, when amoxicillin is ingested, 60-75% of the antibiotic is excreted unchanged into the urine. This antibiotic, now in the environment, may encounter other bacteria and promote resistance. It is unknown how much of an impact current low levels of antibiotics in drinking water are having on the problem of bacterial resistance. However, the potential has been recognized for many years.	None given
multiple CAS #s	Penicillin	Natural Resources Defense Council	Widespread exposure to antibiotics is contributing to the growth of bacterial resistance, and this problem is of grave concern. In the past several decades almost every bacteria that can cause infections in humans has developed resistance to at least one antibiotic, and some are resistant to multiple antibiotics. The Centers for Disease Control and Prevention (CDC) have identified antibiotic resistance as one of the most pressing public health problems to face our nation. Infections caused by bacteria with resistance to at least one antibiotic have been estimated to kill over 60,000 hospitalized patients each year. Antibiotic resistance is caused by a number of factors including repeated and improper use of antibiotics in both humans and animals. Scientists also agree that exposure to low levels of antibiotics actually promotes bacterial resistance by exerting selective pressure for genes that promote resistance.	Antibiotics are found in wastewater because the body does not completely metabolize all drugs, so both the metabolized and unmetabolized drug are excreted by humans into wastewater. For example, when amoxicillin is ingested, 60-75% of the antibiotic is excreted unchanged into the urine. This antibiotic, now in the environment, may encounter other bacteria and promote resistance. It is unknown how much of an impact current low levels of antibiotics in drinking water are having on the problem of bacterial resistance. However, the potential has been recognized for many years.	None given

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			Beta-lactam antibiotics are a broad class of antibiotics which include penicillin derivatives, cephalosporins, monobactams, carbapenems and Beta-lactamase inhibitors. Methicillin, a form of penicillin, had been relied upon as an common effective treatment for Staphylococcus aureus infections but now many strains of S. aureus bacteria are resistant to methicillin (MRSA or methicillin-resistant Staphylococcus aureus.) Unfortunately, MRSA is resistant to much of the entire class of penicillin-like antibiotics called beta-lactams. Therefore, EPA must include penicillin, amoxicillin, oxacillin and methicillin on the CCL4.		
335-67-1	Perfluorooctanoic acid	Eileen Murphy	http://www.c8sciencepanel.org/publications.html The C8 Panel has been reporting detrimental effects at relatively low exposure levels, particularly in children.	In areas where monitoring for PFOA (and other perfluorinated chemicals) occurs, it is detected at some level. Where there are known sources, levels are higher. However, PFOA is often detected in areas with no obvious source.	None given
335-67-1	Perfluorooctanoic Acid	Natural Resources Defense Council	None given	None given	None given
335671	Perfluorooctanoic Acid	NJ Department of Environmental Protection	Post et al. (2012) summarizes many recent toxicology and epidemiology studies relevant to the assessment of potential health effects of PFOA in drinking water. The studies cited in Post et al. (2012) should be considered by USEPA. Two additional very recent publications showing associations of PFOA exposure and kidney and testicular cancer in communities with drinking water exposure (C8 Science Panel, 2012), and with hypertension and elevated homocysteine (a marker for risk of heart disease; Min et al., 2012) in the general population should also be considered by USEPA. While the C8 Science Panel (2012) report is not a peer-reviewed publication, several publications on the cancer incidence study are cited as “in press” in the report; these publications are expected to be available in the near future and should be considered by USEPA.	The following information is a summary of information discussed in Post et al. (2012, citation below): Unlike most other commonly detected organic drinking water contaminants, PFOA and other perfluorinated chemicals do not degrade in the environment and persist indefinitely. PFOA and other perfluorinated compounds are highly water soluble, unlike most other persistent organic pollutants (e.g. PCBs, dioxins, chlordane) which bind preferentially to soil and sediments and are not highly water soluble. For this reason, drinking water is a major exposure route, while drinking water is not a major exposure route for these other persistent organic pollutants (e.g. PCBs, dioxins, chlordane). PFOA bioaccumulates from drinking water to serum with a serum:drinking water ratio of about 100:1 after ongoing exposure, and exposure to even relatively low drinking water concentrations substantially increases total exposure in humans. PFOA persists in humans with a serum half-life of several years. Exposure to PFOA in drinking water by breast-fed and formula-fed infants, a potentially susceptible subpopulation for PFOA's developmental effects, is higher than in adults using the same drinking water source. The review by Post et al. (2012) summarizes many recent studies of the environmental fate and transport, sources, and occurrence of PFOA in source waters (groundwater and surface water) and drinking water. These studies should be considered by USEPA.	None given
52645-53-1	Permethrin	AWWA	None given	None given	None given
732-11-6	Phosmet	Natural Resources Defense Council	Phosmet is a neurotoxicant that causes red blood cell, plasma, serum and brain cholinesterase inhibition. It also shows mutagenic activity. Phosmet interferes with human placental enzymatic activity, which may affect fetal development. EPA has stated in its Interim Reregistration Eligibility Decision (IRED) for phosmet that there is “suggestive evidence of carcinogenicity” based on increased incidence of liver adenomas and carcinomas in male mice, and of mammary gland tumors in females.	Phosmet is an organophosphate pesticide used primarily on apples, peaches, walnuts, almonds and pears. Approximately 1.25 million pounds of active ingredient are applied every year.Phosmet is mobile in runoff and has the potential to contaminate drinking water sources. EPA has done a drinking water risk assessment for phosmet as part of the pesticide reregistration process.	

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			The EPA IRED used modeling estimates to assess phosmet exposure through drinking water due to the limited amount of monitoring data available. Estimated environmental concentrations ranged from 0.4 to 140 ppb. While EPA concluded that drinking water exposure through surface and groundwater was not of concern, there are several flaws in the EPA analysis that undermine that conclusion, as explained below. The IRED drinking water assessment should not be relied upon to decide whether to regulate phosmet under the SDWA.		
			EPA determines whether the drinking water risks of a pesticide are of concern as part of its dietary risk assessment for that chemical. For drinking water risk to remain below the Agency's level of concern, the sum of food and drinking water exposures must be less than the Population Adjusted Dose (PAD). [The PAD is a term that expresses the dietary risk of a chemical, and reflects the Reference Dose, either acute or chronic, that has been adjusted to account for the FQPA safety factor (i.e., RfD/FQPA safety factor)]. A risk estimate that is less than 100% of the acute or chronic PAD does not exceed the Agency's risk concern.)The IRED risk summary for phosmet indicates that dietary risk, acute and chronic, is below the Agency's level of concern. However, the Agency had initially determined that acute dietary exposures were of great concern for infants and children, with up to 2000% of the acute Reference Dose (aRfD) consumed.. Such an exceedance in exposure from food alone means that any additional exposure from drinking water would create additional unacceptable risks. The assessment of food exposure was subsequently revised using a newly submitted acute neurotoxicity study by the registrant, reviewed by the Agency in February 1999, and by the hazard identification assessment review committee (HIARC) in July, 1999. This new acute neurotoxicity study in rats was used to raise the No Observable Adverse Effects Level (NOAEL) to 4.5 mg/kg/day, from the 1.1 value that had been used, based on a chronic toxicity study in rats. The result was a four-fold change, which also resulted in an increase in the PAD. The HIARC executive summary of the study states that "no effects of treatment were seen in the 3.0 or 4.5 mg/kg group", which is in agreement with the registrants conclusions. However, the study DER finds some critical problems with this study: -"Extremely high variability was noted in the data from the motor activity testing, raising questions about the sensitivity of the procedures used in this study. For example, an increase in subsession activity approaching 300% above control levels was not found to be statistically significantly different from controls." -"There was also some large variability in some of the blood cholinesterase measurements (especially for the red blood cells), such that decreases of 25% were not statistically significant. Again, it is possible that true differences caused by exposure to phosmet might be obscured by the high variability of the measure." In fact, the DER states that "the smallest statistically significant change detected in blood measures was 40% (DER p. 10) -"no information is available regarding the dose response curves for cholinesterase inhibition or behavioral effects. This is especially relevant since		

CASRN	Common Name	Nominator	Health Effects Information Provided with Nomination	Occurrence Information Provided with Nomination	Additional Information Provided with Nomination
			similar levels of inhibition (60-75%) were seen in brain and red blood cell cholinesterase at the high dose, with brain inhibition persisting throughout the study." NRDC suggests that this study is not sufficient to establish a NOAEL, since the variability in cholinesterase inhibition was so great that the study design did not provide any statistical power to detect treatment effects. Therefore, the increase in the PAD that made it possible for food and drinking water exposures to remain below the level of concern was not scientifically supported.Notwithstanding EPA's previous determination that phosmet in drinking water does not pose risks of concern (which, as noted above, was based on a flawed study), phosmet should be regulated as a water contaminant under the SDWA and an MCL should be established.		
57-83-0	Progesterone	Natural Resources Defense Council	While each of these compounds [See Table 5 Concentrations of reproductive hormones in U.S. streams (USGS, 2002) located on page 29 of the NRDC letter] is generally found at low concentrations, the potential effects on human health of mixtures of these compounds are unknown. Based on the individual effects of these chemicals, possible risks include defects of the reproductive system in individuals exposed during critical stages of development (e.g. testosterone).	The U.S. Geological Survey (USGS) conducted a study of 139 streams in 30 states that found widespread presence of estrogenic compounds, ovulation inhibitors and other reproductive hormones in surface water near urbanized and agricultural areas (see Table 5). [See Table 5 *Concentrations of reproductive hormones in U.S. streams (USGS, 2002)* located on page 29 of the NRDC letter] Wastewater treatment plants, the likely sources of most of these chemicals, do not treat sewage for these pollutants. Furthermore, drinking water treatment plants do not generally test or treat water for these contaminants, so the frequency of occurrence of these chemicals in treated drinking water and the degree of human exposure are not known. Additional monitoring of water sources and drinking water are necessary to determine the full extent of the contamination, to assess risks to human health, and to determine acceptable levels of exposure and appropriate regulatory action. EPA properly included many of these reproductive hormones on CCL3, although it has not made a final regulatory determination on any of them. However, considering that progesterone and testosterone also occur at similar concentrations and similar frequency as some of the hormones that were include, they should also be added to the CCL4.	None given
10043-92-2	Radon	NJ Department of Environmental Protection	Radon is a known human carcinogen. In New Jersey and other states where radon is prevalent in groundwater, the cancer risk from radon in drinking water is higher than for most other drinking water contaminants that are regulated based on their carcinogenicity. For example, the average level in NJ public water supplies is 921 pCi/L, and the lifetime cancer risk at this level (from inhalation plus ingestion) is 7 x 10-4.	Radon occurs widely in drinking water using groundwater sources in New Jersey and some other states. In New Jersey, the concentration of radon ranged from nondetectable levels to 41,000 pCi/L with an average concentration of 921 pCi/L in public water supplies, and from 50 pCi/L to 170,000 pCi/L with an average concentration of 5,040 pCi/L in private wells.	None given
8025-81-8	Spiramycin	Natural Resources Defense Council	Widespread exposure to antibiotics is contributing to the growth of bacterial resistance, and this problem is of grave concern. In the past several decades almost every bacteria that can cause infections in humans has developed resistance to at least one antibiotic, and some are resistant to multiple antibiotics. The Centers for Disease Control and Prevention (CDC) have identified antibiotic resistance as one of the most pressing public health	Antibiotics are found in wastewater because the body does not completely metabolize all drugs, so both the metabolized and unmetabolized drug are excreted by humans into wastewater. For example, when amoxicillin is ingested, 60-75% of the antibiotic is excreted unchanged into the urine. This antibiotic, now in the environment, may encounter other bacteria and promote resistance. It	None given

CASRN	Common Name	Nominator	Health Effects Information Provided with Nomination	Occurrence Information Provided with Nomination	Additional Information Provided with Nomination
			problems to face our nation. Infections caused by bacteria with resistance to at least one antibiotic have been estimated to kill over 60,000 hospitalized patients each year. Antibiotic resistance is caused by a number of factors including repeated and improper use of antibiotics in both humans and animals. Scientists also agree that exposure to low levels of antibiotics actually promotes bacterial resistance by exerting selective pressure for genes that promote resistance.	is unknown how much of an impact current low levels of antibiotics in drinking water are having on the problem of bacterial resistance. However, the potential has been recognized for many years. Large animal feeding operations generate a large amount of waste that can potentially contaminate groundwater and waterways contributing to antibiotic resistance and contamination of waterways with steroid hormones. As occurs in humans, some portion of the	
			Massive quantities of antibiotics are used in agriculture both to treat infections and as food additives to promote growth and to compensate for conditions that contribute to infection. Animals raised in Concentrated Animal Feeding Operations (CAFOs) are at increased risk for infection due to close confinement and stress. In fact, it has been estimated that 70% of the antibiotics used in the U.S. are for animal husbandry. Improper use and overuse of antibiotics in livestock and poultry can cause resistance in strains of bacteria that can infect humans. Furthermore, half of the antibiotics used in livestock are in the same classes of drugs that are used in humans. As a result the U.S. Institute of Medicine (IOM) and the World Health Organization (WHO) both stated that the widespread use of antibiotics in agriculture is contributing to antibiotic resistance in humans.	antibiotics administered to livestock will pass unchanged through their bodies and will be excreted in their waste. It has been estimated that between 25-75% of antibiotics are excreted unchanged in feces and can persist in the soil after land application. Manure is applied in large quantities as fertilizer in farm fields. In addition to potentially contaminating the food supply with antibiotic resistant bacteria, antibiotics in manure can persist in soil and promote the development of more antibiotic resistant bacteria. Animal waste and its associated contaminants can enter waterways through groundwater contamination, overflow of waste lagoons into surface water or by over-application of manure as fertilizer in farm fields. A recently published study found evidence of fecal contamination and increased levels of antibiotic resistant bacteria downstream of a swine concentrated feeding operation. Other studies have found antibiotic resistance in groundwater underlying a swine waste lagoon. As such, antibiotics that are used both for human medical needs and in large-scale agriculture operations at low levels in animal feed to promote animal growth must be included on the CCL4 and must be regulated. These antibiotics include bacitracin zinc, spiramycin, tylosin, and virginiamycin. Notably, these antibiotics were all banned for agricultural use in the European Union in 1998.	
121831-99-0	Strontium 90	Anonymous 197	Public Health goals recommend a reasonable standard of 0.35 pCi/L based upon carcinogenic potency of 5.59 x 10E-11 pCi/L for Sr-90 in drinking water.	There are 23 nuclear power plants of exact design to the fatal power plants in Japan, failure due to earthquakes near population centers and water sources.	Monitoring existing conditions leads to rate of change analysis when done on a predictable time frame.
58-22-0	Testosterone	Natural Resources Defense Council	While each of these compounds (see Table 5) [See Table 5 Concentrations of reproductive hormones in U.S. streams (USGS, 2002) located on page 29 of the NRDC letter] is generally found at low concentrations, the potential effects on human health of mixtures of these compounds are unknown. Based on the individual effects of these chemicals, possible risks include defects of the reproductive system in individuals exposed during critical stages of development (e.g. testosterone).	The U.S. Geological Survey (USGS) conducted a study of 139 streams in 30 states that found widespread presence of estrogenic compounds, ovulation inhibitors and other reproductive hormones in surface water near urbanized and agricultural areas (see Table 5). [See Table 5 *Concentrations of reproductive hormones in U.S. streams (USGS, 2002)* located on page 29 of the NRDC letter] Wastewater treatment plants, the likely sources of most of these chemicals, do not treat sewage for these pollutants. Furthermore, drinking water treatment plants do not generally test or treat water for these contaminants, so the frequency of occurrence of these chemicals in treated drinking water and the degree of human exposure are not known. Additional monitoring of water sources and drinking water are necessary to determine the full extent of the contamination,	None given

CASRN	Common Name	Nominator	Health Effects Information Provided with Nomination	Occurrence Information Provided with Nomination	Additional Information Provided with Nomination
				to assess risks to human health, and to determine acceptable levels of exposure and appropriate regulatory action. EPA properly included many of these reproductive hormones on CCL3, although it has not made a final regulatory determination on any of them. However, considering that progesterone and testosterone also occur at similar concentrations and similar frequency as some of the hormones that were include, they should also be added to the CCL4.	
52-68-6	Trichlorfon	Natural Resources Defense Council	Like the other organphosphates, trichlorfon is a neurotoxicant and cholinesterase inhibitor. Trichlorfon exposure is associated with kidney, lung and gastrointestinal abnormalities in animal studies. Anemia has also been reported, as well as benign pheochromocytomas. A statistically significant increase in mononuclear cell leukemia was also observed. Incidences of alveolar/bronchiolar adenomas, renal tubular adenomas and alveolar/bronchiolar carcinomas, while not statistically significant, occurred with frequencies "well outside of the historical control range for all three tumor types." While EPA decided to classify trichlorfon in Group E for carcinogenicity arguing that the statistically significant increases in tumors in the studies were seen in the lower but not the higher doses, we argue that the evidence remains suggestive given that separate studies found significant increases in the same types of tumors. In the studies analyzed by EPA during the reregistration process, trichlorfon also showed developmental toxicity in animals (decreased fetal body weight, delayed or reduced ossification) and mutagenic activity in an in vitro cytogenetic study in mammalian cells.	Trichlorfon is an organophosphate insecticide with agricultural non-food and feed crop uses (e.g. agricultural non-cultivated areas, ornamental trees, etc.), as well as indoor and outdoor residential use. Usage volume data for these registered uses is not available. Trichlorfon is highly mobile in soil, but EPA did not assess its groundwater contamination potential during the reregistration process for lack of appropriate data. Trichlorfon can enter surface waters in ground spray and runoff. Well samples from Georgia in the EPA Pesticides in Ground Water Database showed trichlorfon detections in 12 of 179 wells with concentration up to 10 ppb. EPA did not consider these samples useful, citing analytical uncertainties. Trichlorfon has a half-life in soil of 1 to 27 days, depending on soil type, which increases its potential to contaminate surface waters. However, the trichlorfon RED does not address drinking water risks. Despite the cancellation of feed and food crop uses, trichlorfon still has registered agricultural and residential outdoor uses that pose a risk of surface and possibly groundwater contamination. The scarcity of monitoring data on environmental concentrations should not lead to an assumption of negligible risk. The known toxicity of trichlorfon, its mobility and extended half-life in soil all make it a likely water contaminant in high use areas. EPA should require the collection of monitoring data for these areas to enable the Agency to assess water contamination risks and make a regulatory decision concerning trichlorfon.	None given
101-20-2	Triclocarban	Natural Resources Defense Council	Triclocarban is a possible endocrine disruptor An animal study indicates that triclocarban exposure enhanced the effects of testosterone both in vitro and in vivo in male rats.	Triclocarban (Urea, N-(4-chlorophenyl)-N'-(3,4-dichlorophenyl) 3,4,4'-Trichlorocarbanilide), an antimicrobial pesticide also known as TCC, has been widely detected in effluent from wastewater treatment plants (WWTPs) in the United States. TCC has also been frequently detected in environmental water samples. The half-life of TCC in sediment is 540 days. One study predicted the magnitude and frequency of TCC contamination nationwide based on experimental and modeling data to be 1150 ng/L and 60%, respectively; much higher than previously recognized by EPA (240 ng/L, 30%). Another study in the Greater Baltimore area found an average TCC level of 6.75 ug/L in wastewater samples, while river water samples had concentrations of up to 5.6 ug/L. These concentrations are higher than the Predicted Environmental	None given

CASRN	Common Name	Nominator	Health Effects Information Provided with Nomination	Occurrence Information Provided with Nomination	Additional Information Provided with Nomination
				Concentrations (PEC) calculated by the TCC Consortium in a report submitted to EPA in 2003 as part of the High Production Volume Chemical program, which estimated PECs from 0.0013 to 0.050 jtg/L. The actual measurements from the Greater Baltimore area study also exceed the TCC Consortium's Predicted No Effect Concentration of 0.146 ~g/L.	
				A study of a 684 million liter per day typical activated sludge WWTP found a concentration of 6.1 ± 2.0 ug/L in the influent and 0.17 ± 0.03 ug/L in the effluent. Approximately 127 ± 6 g/d exited the plant in the effluent, a clear indication that conventional wastewater treatment may leave considerable levels of TCC in the water. Because of this, TCC concentrations tend to be higher downstream of WWTPs. The most important sources of triclocarban to the aquatic environment were estimated to be activated sludge treatment plants (contributing 39-67%), followed by trickling filters (31- 54%), combined sewer overflows (2-7%) and sanitary sewer overflows (<0.2%). Given these data, triclocarban should be added to the CCL4.	
3380-34-5	Triclosan	Natural Resources Defense Council	The chemical structure of triclosan is similar to other endocrine disrupting compounds and potential breakdown products of triclosan include dioxins. Recently, low levels of triclosan were found to interfere with the metamorphosis of frogs. Exposure to as little as 0.15 µg/L triclosan caused an earlier metamorphosis than normal, with effects on the tadpole brain and tail. Triclosan activates the human pregnane X receptor (hPXR), which is involved in the enzymatic metabolism of steroids and xenobiotics.	Triclosan (5-chloro-2-(2,4-dichlorophenoxy)-phenol), is a broad spectrum antimicrobial pesticide that is widely used in personal care products such as soaps, toothpastes, cosmetics, skin creams and deodorants; kitchen accessories such as cutting boards and utensils; and in textiles such as sportswear, shoes and carpets. Approximately three quarters of Americans between the ages of six to over 65 have triclosan in their urine. Triclosan has even been detected in human blood plasma and breast milk. Triclosan is produced at over one million pounds per year. Triclosan is one of the most frequently detected chemicals in streams across the U.S. Wild Atlantic bottlenose dolphins have been found with triclosan in their bodies. Triclosan has been found in wastewater treatment effluent and drinking water sources. Triclosan was detected in Louisiana sewage treatment plant effluent at 10-21 ng/l. Boyd (2004) reported triclosan concentrations of ND – 29 ng/l in two stormwater canals in New Orleans. Triclosan has also been detected in raw and finished drinking water samples from Southern California.	None given
1401-69-0	Tylosin	Natural Resources Defense Council	Widespread exposure to antibiotics is contributing to the growth of bacterial resistance, and this problem is of grave concern. In the past several decades almost every bacteria that can cause infections in humans has developed resistance to at least one antibiotic, and some are resistant to multiple antibiotics. The Centers for Disease Control and Prevention (CDC) have identified antibiotic resistance as one of the most pressing public health problems to face our nation. Infections caused by bacteria with resistance to at least one antibiotic have been estimated to kill over 60,000 hospitalized patients each year. Antibiotic resistance is caused by a number of factors including repeated and improper use of antibiotics in both humans and animals. Scientists also agree	Antibiotics are found in wastewater because the body does not completely metabolize all drugs, so both the metabolized and unmetabolized drug are excreted by humans into wastewater. For example, when amoxicillin is ingested, 60-75% of the antibiotic is excreted unchanged into the urine. This antibiotic, now in the environment, may encounter other bacteria and promote resistance. It is unknown how much of an impact current low levels of antibiotics in drinking water are having on the problem of bacterial resistance. However, the potential has been recognized for many years. Large animal feeding operations generate a large amount of waste that can potentially contaminate groundwater and waterways	None given

CASRN	Common Name	Nominator	Health Effects Information Provided with Nomination	Occurrence Information Provided with Nomination	Additional Information Provided with Nomination
			that exposure to low levels of antibiotics actually promotes bacterial resistance by exerting selective pressure for genes that promote resistance. Massive quantities of antibiotics are used in agriculture both to treat infections and as food additives to promote growth and to compensate for conditions that contribute to infection. Animals raised in Concentrated Animal Feeding	contributing to antibiotic resistance and contamination of waterways with steroid hormones As occurs in humans, some portion of the antibiotics administered to livestock will pass unchanged through their bodies and will be excreted in their waste. It has been estimated that between 25-75% of antibiotics are excreted unchanged in feces and can persist in the soil after land application. Manure is applied in large	
			Operations (CAFOs) are at increased risk for infection due to close confinement and stress. In fact, it has been estimated that 70% of the antibiotics used in the U.S. are for animal husbandry. Improper use and overuse of antibiotics in livestock and poultry can cause resistance in strains of bacteria that can infect humans. Furthermore, half of the antibiotics used in livestock are in the same classes of drugs that are used in humans. As a result the U.S. Institute of Medicine (IOM) and the World Health Organization (WHO) both stated that the widespread use of antibiotics in agriculture is contributing to antibiotic resistance in humans. [Quotes from the Healthcare Without Harm factsheet. A*ntibiotic Resistance and Agricultural Overuse of Antibiotics*. 2005. http://www.noharm.org/us/food/issue *U.S. Institute of Medicine/National Academy of Science:* "Clearly, a decrease in antimicrobial use in human medicine alone will have little effect on the current [antibiotic-resistant] situation. Substantial efforts must be made to decrease inappropriate overuse in animals and agriculture as well." *World Health Organization*: "There is clear evidence of the human health consequences due to resistant organisms resulting from non-human usage of antimicrobials. These consequences include infections that would not have otherwise occurred, increased frequency of treatment failures (in some cases death) and increased severity of infections."]	quantities as fertilizer in farm fields. In addition to potentially contaminating the food supply with antibiotic resistant bacteria, antibiotics in manure can persist in soil and promote the development of more antibiotic resistant bacteria. Animal waste and its associated contaminants can enter waterways through groundwater contamination, overflow of waste lagoons into surface water or by over-application of manure as fertilizer in farm fields. A recently published study found evidence of fecal contamination and increased levels of antibiotic resistant bacteria downstream of a swine concentrated feeding operation. Other studies have found antibiotic resistance in groundwater underlying a swine waste lagoon. As such, antibiotics that are used both for human medical needs and in large-scale agriculture operations at low levels in animal feed to promote animal growth must be included on the CCL4 and must be regulated. These antibiotics include bacitracin zinc, spiramycin, tylosin, and virginiamycin. Notably, these antibiotics were all banned for agricultural use in the European Union in 1998.	
1404-90-6	Vancomycin	Natural Resources Defense Council	Widespread exposure to antibiotics is contributing to the growth of bacterial resistance, and this problem is of grave concern. In the past several decades almost every bacteria that can cause infections in humans has developed resistance to at least one antibiotic, and some are resistant to multiple antibiotics. The Centers for Disease Control and Prevention (CDC) have identified antibiotic resistance as one of the most pressing public health problems to face our nation. Infections caused by bacteria with resistance to at least one antibiotic have been estimated to kill over 60,000 hospitalized patients each year. Antibiotic resistance is caused by a number of factors including repeated and improper use of antibiotics in both humans and animals. Scientists also agree that exposure to low levels of antibiotics actually promotes bacterial resistance by exerting selective pressure for genes that promote resistance. Beta-lactam antibiotics are a broad class of antibiotics which include penicillin derivatives, cephalosporins, monobactams, carbapenems and Beta-lactamase inhibitors. Methicillin, a form of penicillin, had been relied upon as an common effective treatment for Staphylococcus aureus infections but now many strains of S. aureus bacteria are resistant to methicillin (MRSA or methicillin-resistant	Antibiotics are found in wastewater because the body does not completely metabolize all drugs, so both the metabolized and unmetabolized drug are excreted by humans into wastewater. For example, when amoxicillin is ingested, 60-75% of the antibiotic is excreted unchanged into the urine. This antibiotic, now in the environment, may encounter other bacteria and promote resistance. It is unknown how much of an impact current low levels of antibiotics in drinking water are having on the problem of bacterial resistance. However, the potential has been recognized for many years.	None given

CASRN	Common Name	Nominator	Health Effects Information Provided with Nomination	Occurrence Information Provided with Nomination	Additional Information Provided with Nomination
			Staphylococcus aureus.) Unfortunately, MRSA is resistant to much of the entire class of penicillin-like antibiotics called beta-lactams. Therefore, EPA must include penicillin, amoxicillin, oxacillin and methicillin on the CCL4. Infections among hospital patients (nosocomial infections) from enterococci bacteria are very common. Such infections that result in human disease can		
			be fatal, particularly those caused by strains of vancomycin-resistant enterococci (VRE). During 2004, VRE caused about one of every three infections in hospital intensive-care units, according to the Centers CDC. As of 2007, the U.S. had reported seven cases of vancomycin-resistant *Staphylococcus aureus* (VRSA) infection. Therefore, vancomycin must be included on the CCL3.		
11006-76-1	Virginiamycin	Natural Resources Defense Council	Widespread exposure to antibiotics is contributing to the growth of bacterial resistance, and this problem is of grave concern. In the past several decades almost every bacteria that can cause infections in humans has developed resistance to at least one antibiotic, and some are resistant to multiple antibiotics. The Centers for Disease Control and Prevention (CDC) have identified antibiotic resistance as one of the most pressing public health problems to face our nation. Infections caused by bacteria with resistance to at least one antibiotic have been estimated to kill over 60,000 hospitalized patients each year. Antibiotic resistance is caused by a number of factors including repeated and improper use of antibiotics in both humans and animals. Scientists also agree that exposure to low levels of antibiotics actually promotes bacterial resistance by exerting selective pressure for genes that promote resistance. Massive quantities of antibiotics are used in agriculture both to treat infections and as food additives to promote growth and to compensate for conditions that contribute to infection. Animals raised in Concentrated Animal Feeding Operations (CAFOs) are at increased risk for infection due to close confinement and stress. In fact, it has been estimated that 70% of the antibiotics used in the U.S. are for animal husbandry. Improper use and overuse of antibiotics in livestock and poultry can cause resistance in strains of bacteria that can infect humans. Furthermore, half of the antibiotics used in livestock are in the same classes of drugs that are used in humans. As a result the U.S. Institute of Medicine (IOM) and the World Health Organization (WHO) both stated that the widespread use of antibiotics in agriculture is contributing to antibiotic resistance in humans. [Quotes from the Healthcare Without Harm factsheet. A*ntibiotic Resistance and Agricultural Overuse of Antibiotics*. 2005. http://www.noharm.org/us/food/issue *U.S. Institute of Medicine/National Academy of Science:* "Clearly, a decrease in antimicrobial use in human medicine alone will have little effect on the current [antibiotic-resistant] situation. Substantial efforts must be made to decrease inappropriate overuse in animals and agriculture as well."	Antibiotics are found in wastewater because the body does not completely metabolize all drugs, so both the metabolized and unmetabolized drug are excreted by humans into wastewater. For example, when amoxicillin is ingested, 60-75% of the antibiotic is excreted unchanged into the urine. This antibiotic, now in the environment, may encounter other bacteria and promote resistance. It is unknown how much of an impact current low levels of antibiotics in drinking water are having on the problem of bacterial resistance. However, the potential has been recognized for many years. Large animal feeding operations generate a large amount of waste that can potentially contaminate groundwater and waterways contributing to antibiotic resistance and contamination of waterways with steroid hormones. As occurs in humans, some portion of the antibiotics administered to livestock will pass unchanged through their bodies and will be excreted in their waste. It has been estimated that between 25-75% of antibiotics are excreted unchanged in feces and can persist in the soil after land application. Manure is applied in large quantities as fertilizer in farm fields. In addition to potentially contaminating the food supply with antibiotic resistant bacteria, antibiotics in manure can persist in soil and promote the development of more antibiotic resistant bacteria. Animal waste and its associated contaminants can enter waterways through groundwater contamination, overflow of waste lagoons into surface water or by over-application of manure as fertilizer in farm fields. A recently published study found evidence of fecal contamination and increased levels of antibiotic resistant bacteria downstream of a swine concentrated feeding operation. Other studies have found antibiotic resistance in groundwater underlying a swine waste lagoon. As such, antibiotics that are used both for human medical needs and in large-scale agriculture operations at low levels in animal feed to promote animal growth must be included on the CCL4 and must be regulated. These antibiotics include bacitracin zinc, spiramycin, tylosin, and virginiamycin. Notably, these antibiotics were all banned for agricultural use in the European Union in 1998.	None given

CASRN	Common Name	Nominator	Health Effects Information Provided with Nomination	Occurrence Information Provided with Nomination	Additional Information Provided with Nomination
			World Health Organization: "There is clear evidence of the human health consequences due to resistant organisms resulting from non-human usage of antimicrobials. These consequences include infections that would not have otherwise occurred, increased frequency of treatment failures (in some cases death) and increased severity of infections."]		

[1]For the purpose of developing this appendix, EPA separated original text submitted with the nomination for each contaminant and placed it into the health effects information, occurrence information or additional information columns, as appropriate. EPA maintained the text submitted with each nomination verbatim.

[2] The organization that nominated "nonylphenol" for CCL 4 provided the CASRN of 25451-52-3. The name "nonylphenol" does not allow for a definitive identification of chemical structure since nonylphenol can exhibit two forms of isomerism. There are at least five CASRNs known to be associated with "nonylphenol:" in addition to 25154-52-3 (which represents n-nonylphenol with the ortho-, meta-, or para-substitution unspecified), other CASRNs include: 104-40-5 (4-n-nonylphenol); 84852-15-3 (4-nonylphenol, branched); 91672-41-2 (2-nonylphenol, branched); and 139-84-4 (3-n-nonylphenol). None of these five CASRNs are adequately general enough to represent both forms of isomerism. For the sake of consistency, the CASRN provided by the nominator was selected and the additional possible CASRNs and structures are delineated here.

Appendix 2. Microbial Contaminants Nominated

Common Name	Nominator	Health Effects Information Provided withNomination	Occurrence Information Provided with Nomination	Additional Information Provided with Nomination
Adenovirus	NJ Dept. of Environmental Protection	Adenoviruses (Ads) are a common cause of childhood gastroenteritis. Persons with weakened immune systems are most susceptible to infection, with high rates of severe illness and mortality. As with many other pathogens, infants are most susceptible among persons with normal immune system function (Post et al, 2011). Some serotypes are shed in feces but cause respiratory (e.g., Ads 1 through 7) or eye disease (e.g., Ads 3, 7, and 14). These types can also spread via aerosol, direct contact, or sexual routes (Jiang, 2006; Langley, 2005). Ads 12, 18, and 31 may cause diarrhea on occasion, but infection usually results in apparent illness in infants. Other serotypes are associated with GI illness but have not been proven to cause illness. However, there is probably sufficient evidence that Ad 40 and Ad 41 are waterborne pathogens that can cause diarrhea in infants. Recent evidence has also shown a possible connection between infection with Adenovirus 36 and obesity in humans, including a potential waterborne route of exposure (Atkinson, 2012). Ads can be shed in the feces for months to years following infection.	Although data from Borchardt (2008) show low concentrations of adenovirus in drinking water and a lack of association between the presence of adenoviruses and enteric disease. And although the waterborne transmission route for adenoviruse-based disease has not been definitively proven, Borchardt (2008) and others (e.g., Katayama et al, 2008; Rodriguez et al, 2008) have shown that adenoviruses are among the most common virus groups detected in water.	What is the correlation, or co-occurrence, of adenovirus and other viruses in the Borchardt (2008) and other studies? The EPA may wish to also consider monitoring for adenoviruses, not as pathogens themselves, but as a potential "viral indicator" of the presence of other pathogenic human enteric viruses (HEV). However, if adenoviruses are monitored, the NanoCeram filter may not be appropriate for this group of viruses (Gibbons et al, 2010; as cited in the proposed UCMR3 [USEPA, 2011]). Also, with regard to the Ground Water Rule (GWR)(USEPA, 2006), because adenoviruses are the most UV-resistant group of microbes, and because the GWR UV dose requirements are based on inactivating adenoviruses, the EPA may wish to generate additional data on the presence of adenoviruses in GW. Such data could be generated in conjunction with epidemiological studies similar to those of Borchardt (2008) but in other locations in the US. If such studies confirmed the findings of Borchardt (2008), who observed a lack of association of gastrointestinal illness with adenovirus concentrations, then perhaps the GWR UV dose requirements could be reduced. Reduced UV dose requirements would result in substantial cost savings for many public water systems.
Heterotrophic Plate Count Bacteria	NJ Dept. of Environmental Protection	None provided	There is evidence that HPC counts in a well significantly above average for all sources could serve as a trigger for fecal indicator monitoring in ground water, but the data was limited and a definitive conclusion could not be drawn (Atherholt et al, 2003). Other investigators have shown that HPC may be a useful GW indicator (Butscher et al, 2011, Goeppert and Goldscheider, 2011). HPC testing is also very useful for QC purposes including negative control counts and for the ease of determining quantifiable and reproducible (similar) counts in replicates of field samples.	The proposed UCMR3 stated that aerobic spores would be monitored. It is not clear why aerobic spores would be monitored as pathogen indicators. Anaerobic spores (e.g., spores of Clostridia) are a more fecal-specific indicator than are aerobic spores (presumably from soil-borne Bacillus spp.), but it has been shown that anaerobic spores are poor indicators of fecal contamination compared to other indicators such as coliform or enterococcus bacteria (Francy et al, 2000 & 2004; Atherholt et al, 2003; Butscher et al, 2011). If aerobic spores are to be employed, not as a fecal indicator, but as an indicator of surface water influence, we suggest monitoring for heterotrophic plate count (HPC) bacteria instead.

Common Name	Nominator	Health Effects Information Provided withNomination	Occurrence Information Provided with Nomination	Additional Information Provided with Nomination
Naegleria fowleri	NJ Dept. of Environmental Protection	The disease, primary amoebic meningoencephalitis, is fatal following exposure of susceptible individuals with death occurring within 72 hours after symptoms (similar to viral and bacterial meningitis) first appear.	Although in the US, there is a 14-year average of just 2 cases of primary amoebic meningoencephalitis per year, 11 of 143 wells (8%), with an average water temperature of 29 oC were found to contain Naegleria.	Because 11 of 143 wells (8%), with an average water temperature of 29°C were found to contain Naegleria (Blair et al, 2008), the EPA may wish to consider conducting a summer monitoring survey of "warm water" wells in the US (with a suitable control group of "cold water" wells).
Toxoplasma gondii	J. Jones	Ocular and congenital illness	Waterborne Toxoplasma gondii has been implicated in other countries and could contaminate drinking water that is not filtered.	None given
Toxoplasma gondii	US Dept. of Agriculture	Many outbreaks causing serious disease in humans detailed.	[See (de Moura et al., 2006) in Appendix 5]	None given
Vibrio cholerae	Natural Resources Defense Council	This bacterium is known to cause outbreaks of cholera, an acute diarrheal illness caused by intestinal infection with the bacterium Vibrio cholerae, with serious and occasionally fatal human consequences.	As recently as April 2011, an outbreak of Vibrio cholerae O75 was reported in Florida. Ten cases were identified in the outbreak. Their occurrence is likely to expand as climate change continues, which makes it appropriate to include this pathogen in the CCL4.	None given

Appendix 3. References Provided with Chemical Nominations

CASRN	Contaminant Name	Nominating Individual	Organization Name	Abbreviated References Cited in the Nomination
77439-76-0	3-chloro-4-dichloromethyl-5-hydroxy-2(5H)-furanone)	Thomas W. Curtis	AWWA	None given
116-06-03	Aldicarb	Thomas W. Curtis	AWWA	None given
116-06-3	Aldicarb	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(Fiore et al., 1986) (Grendon and Baum, 1994) (Hajoui et al., 1992) (Smulders et al., 2003) (Smulders et al., 2004) (USEPA, 1984) (USEPA, 1988) (USEPA, 2006) (USEPA, 2006a)
68555-24-8	Alkylphenol mono- to tri-oxylates	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(Gatidou et al., 2006) (Ying et al., 2002)
26787-78-0	Amoxicillin	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(CDC, 2012) (Chee-Sanford et al., 2001) (Gilchrist et al., 2007) (Health Care Without Harm, 2005) (Mellon et al., 2000) (Sapkota et al., 2007a) (USA Today, 2000) (Wallinga et al., 2006) (Wallinga, 2005)
86-50-0	Azinphos-methyl	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(Dabrowski et al., 2006) (Loewy et al., 2003) (Loewy et al., 2006) (Rohlman et al., 2005) (Rothlein et al., 2006) (Souza et al., 2004) (Souza et al., 2005) (USEPA, 2001a) (USEPA, 2006a)
1405-89-6	Bacitracin zinc	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(CDC, 2012) (Chee-Sanford et al., 2001) (Gilchrist et al., 2007) (Health Care Without Harm, 2005) (Mellon et al., 2000) (Sapkota et al., 2007a) (USA Today, 2000) (Wallinga et al., 2006) (Wallinga, 2005)
25057-89-0	Bentazone	Thomas W. Curtis	AWWA	None given

CASRN	Contaminant Name	Nominating Individual	Organization Name	Abbreviated References Cited in the Nomination
85-68-7	Benzyl butyl phthalate	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(ATSDR) (CDC, 2005) (Gray et al., 2000) (Gray et al., 2006) (Main et al., 2006) (Meeker et al., 2007) (Stahlhut et al., 2007) (USEPA)
80-05-7	Bisphenol A	Anonymous 201	None given	None given
80-05-7	Bisphenol A	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(Adewale et al., 2011a) (Alonso-Magdalena et al., 2006) (Ayyanan et al., 2011b) (Calafat et al., 2008a) (Carwile and Michels, 2011) (ChemSec, 2012) (Cousins et al., 2002) (Durando et al., 2007) (Fromme et al., 2002) (Hoet al., 2006) (Hunt et al., 2003) (Melzer et al., 2012) (Murray et al., 2007) (National Institute of Environmental Health Sciences, 2008) (Prins et al., 2011) (Raloff, 2012) (Sorianoet al., 2012) (USEPA) (vom Saal et al., 2007)
1689-84-5	Bromoxynil	Thomas W. Curtis	AWWA	None given
63-25-2	Carbaryl	Thomas W. Curtis	AWWA	None given
63-25-2	Carbaryl	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(Tarplee, 1999) (Tarplee, 2001) (USEPA, 2004a)
10045-97-3	Cesium 137	Anonymous 197	None given	None given
1897-45-6	Chlorothalonil	Thomas W. Curtis	AWWA	None given
2921-88-2	Chlorpyrifos	Thomas W. Curtis	AWWA	None given
2921-88-2	Chlorpyrifos	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(Barbas and Resek, 1996) (Becker et al., 1989) (Burkart and Kolpin, 1993) (Goss, 1992) (Larson, et al., 1997) (Long, 1989) (Makris et al., 1998) (USEPA, 2000a) (USEPA, 2002) (USEPA, 2002a)

CASRN	Contaminant Name	Nominating Individual	Organization Name	Abbreviated References Cited in the Nomination
84-74-2	Dibutyl phthalate	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(ATSDR) (CDC, 2005) (Gray et al., 2000) (Gray et al., 2006) (IHCP, 2003) (Main et al., 2006) (Meeker et al., 2007) (Stahlhut et al., 2007) (USEPA)
1918-00-9	Dicamba	Thomas W. Curtis	AWWA	None given
62-73-7	Dichlorvos	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(Brown et al., 1990) (Leissand Savitz, 1995) (National Toxicology Program, 1989) (USEPA, 2000b) (USEPA, 2000c) (USEPA, 2006b)
115-32-2	Dicofol	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(Hoekstra et al. 2005) (Ishihara et al., 2003) (Jadaramkunti and Kaliwal, 2001) (Thibaut and Porte, 2004) (USEPA, 1998)
84-61-7	Dicyclohexyl phthalate	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(ATSDR) (CDC, 2005) (Gray et al., 2000) (Gray et al., 2006) (Main et al., 2006) (Meeker et al., 2007) (Stahlhut et al., 2007)
84-66-2	Diethyl phthalate	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(ATSDR) (CDC, 2005) (Gray et al., 2000) (Gray et al., 2006) (Main et al., 2006) (Meeker et al., 2007) (Stahlhut et al., 2007) (USEPA)
28553-12-0	Di-isononyl phthalate	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(ATSDR) (CDC, 2005) (Gray et al., 2000) (Gray et al., 2006) (IHCP, 2003) (Main et al., 2006) (Meeker et al., 2007) (Stahlhut et al., 2007) (USEPA)
131-11-3	Dimethyl phthalate	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(ATSDR) (CDC, 2005) (Gray et al., 2000) (Gray et al., 2006) (Main et al., 2006) (Meeker et al., 2007) (Stahlhut et al., 2007) (USEPA)

CASRN	Contaminant Name	Nominating Individual	Organization Name	Abbreviated References Cited in the Nomination
117-84-0	Di-n-octyl phthalate	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(ATSDR) (CDC, 2005) (Gray et al., 2000) (Gray et al., 2006) (Main et al., 2006) (Meeker et al., 2007) (Stahlhut et al., 2007) (USEPA)
115-29-7	Endosulfan	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(Association of American Pesticide Control Officials, Inc, 1969) (Lakshmana and Raju, 1994) (OSHA, 1989) (Sinha et al., 1991) (Sinha et al., 1997) (State of California: Department of Industrial Relations) (USEPA, 2002b) (Willey and Kron, 2001) (Wilson and LeBlanc, 1998)
2164-17-2	Fluometuron	Thomas W. Curtis	AWWA	None given
319-84-6	Hexachlorocyclohexane (alpha isomer)	Thomas W. Curtis	AWWA	None given
165800-03-3	Linezolid	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(CDC, 2012) (Chee-Sanford et al., 2001) (Gilchrist et al., 2007) (Health Care Without Harm, 2005) (Mellon et al., 2000) (Sapkota et al., 2007a) (USA Today, 2000) (Wallinga et al., 2006) (Wallinga, 2005)
330-55-2	Linuron	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(Cook, 1993) (EXTOXNET, 1996) (Gray et al., 1999) (USEPA, 1995) (USEPA, 2002c) (USGS, 1992)
121-75-5	Malathion	Thomas W. Curtis	AWWA	None given

CASRN	Contaminant Name	Nominating Individual	Organization Name	Abbreviated References Cited in the Nomination
7439-96-5	Manganese	Michael S. Hutcheson	Massachusetts Department of Environmental Protection	(Ayotte, et al., 2001) (Bouchard et al., 2007) (Bouchard et al., 2011) (Boyes, 2010) (Brown and Foos, 2009) (Claus et al., 2010) (Deveau, 2010) (Dorman and Wong, 2006) (Erikson et al., 2007) (Fordahlet al., 2012) (Golub et al., 2005) (Kern et al., 2010) (Khan et al., 2011) (Khan et al., 2012) (Kim et al., 2009) (Ljung et al., 2009) (Ljung, 2007) (Menezes-Filho et al., 2009) (Moreno et al., 2009) (Parvez et al., 2011) (Riojas-Rodriguez et al., 2010) (Roels et al., 2012) (Santamaria and Sulsky, 2010) (Santamaria, 2008) (USEPA, 1979) (USEPA, 2004b) (Wasserman et al., 2006) (Wasserman et al., 2011) (Yoon et al., 2011) (Yoon et al., 2009)

CASRN	Contaminant Name	Nominating Individual	Organization Name	Abbreviated References Cited in the Nomination
7439-96-5	Manganese	James Kelly	Minnesota Department of Health	(Anoka County, 2004) (Ayotte, et al., 2001) (Bouchard et al., 2007) (Bouchard et al., 2011) (Boyes, 2010) (Brown and Foos, 2009) (Claus et al., 2010) (County Geologic Atlas) (Deveau, 2010) (Dorman and Wong, 2006) (Erikson et al., 2007) (Fong, et al., 1998) (Fordahlet al., 2012) (Golub et al., 2005) (GWMAP) (Kern et al., 2010) (Khan et al., 2011) (Khan et al., 2012) (Kim et al., 2009) (Lively et al., 1992) (Ljung et al., 2009) (Ljung, 2007) (MARS data set) (Menezes-Filho et al., 2009) (Minesota, 2011) (Moreno et al., 2009) (Parvez et al., 2011) (Riojas-Rodriguez et al., 2010) (Santamaria and Sulsky, 2010) (Santamaria, 2008) (Smith and Nemetz, 1995) (State of Minnesota) (USEPA, 1979) (USEPA, 2004) (Wall and Regan, 1994) (Wasserman et al., 2006) (Wasserman et al., 2011) (Yoon et al., 2011) (Yoon et al., 2009)
7439-96-5	Manganese	Gloria B. Post	NJ Department of Environmental Protection	(Bouchard et al., 2011) (Khan et al., 2011) (Wasserman et al., 2011)
61-32-5	Methicillin	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(CDC, 2012) (Chee-Sanford et al., 2001) (Gilchrist et al., 2007) (Health Care Without Harm, 2005) (Mellon et al., 2000) (Sapkota et al., 2007a) (USA Today, 2000) (Wallinga et al., 2006) (Wallinga, 2005)
298-00-0	methyl parathion	Thomas W. Curtis	AWWA	None given
1634-04-4	Methyl tertiary butyl ether	Gloria B. Post	NJ Department of Environmental Protection	(Bird et al., 1997) (Dodd et al., 2011) (Ramazzini Institute Study)

CASRN	Contaminant Name	Nominating Individual	Organization Name	Abbreviated References Cited in the Nomination
101043-37-2	Microcystin-LR	Christopher Greene	Minnesota Department of Health	(Barringer, 2012) (Billam et al., 2006) (Boyer et al., 2005) (Chen et al., 2011) (City of San Diego, 2012) (Graham et al., 2004) (Ho L, et al., 2010) (Li et al., 2008) (Liu et al., 2010) (Ohio EPA, 2012) (Trinchet et al., 2011) (Ueno et al., 1999) (Wang et al., 2012)
101043-37-2	Microcystin-LR	James Kelly	Minnesota Department of Health	(Boyer et al., 2005) (Chen et al., 2011) (City of San Diego, 2012) (Graham et al., 2004) (Ho L, et al., 2010) (Li et al., 2008) (Liu et al., 2010) (Ohio EPA, 2012) (Trinchet et al., 2011) (Ueno et al., 1999) (Wang et al., 2012)
25154-52-3	Nonylphenol	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(Gatidou et al., 2006) (Ying et al., 2002)
9016-45-9	Nonylphenol ethoxylate	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(Gatidou et al., 2006) (Ying et al., 2002)
27193-28-8	Octylphenol	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(Gatidou et al., 2006) (Ying et al., 2002)
9036-19-5	Octylphenol ethoxylate	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(Gatidou et al., 2006) (Ying et al., 2002)
66-79-5	Oxacillin	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(CDC, 2012) (Chee-Sanford et al., 2001) (Gilchrist et al., 2007) (Health Care Without Harm, 2005) (Mellon et al., 2000) (Sapkota et al., 2007a) (USA Today, 2000) (Wallinga et al., 2006) (Wallinga, 2005)
multiple CAS #s	Penicillin	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(CDC, 2012) (Chee-Sanford et al., 2001) (Gilchrist et al., 2007) (Health Care Without Harm, 2005) (Mellon et al., 2000) (Sapkota et al., 2007a) (USA Today, 2000) (Wallinga et al., 2006) (Wallinga, 2005)

CASRN	Contaminant Name	Nominating Individual	Organization Name	Abbreviated References Cited in the Nomination
68141-02-6	Perfluoro octanoic acid	Eileen Murphy	None given	(C8 Science Panel, 2012) (Post et al., 2009) None given (C8 Science Panel, 2012) (Min et al., 2012) (Post et al., 2012)
52645-53-1	Permethrin	Thomas W. Curtis	AWWA	None given
732-11-6	Phosmet	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(Cappon, 1998) (Hasegawa et al., 1993) (Raffaele, 1999) (Raffaele, 2002) (Souza et al., 2005) (Swartz, 1999) (Taylor, 1999) (USEPA, 2001b) (Vlckova et al., 1993)
multiple CAS #s	Phthalates[2]	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(Adibiet al., 2003) (Blount et al., 2000) (Meeker et al., 2007) (Silva et al., 2007) (Stahlhut RW, et al., 2007) (Wolff et al., 2007)
57-83-0	Progesterone	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(Hotchkiss et al., 2007) (Kolpin et al., 2002)
10043-92-2	Radon	Gloria B. Post	NJ Department of Environmental Protection	(New Jersey Drinking Water Quality Institute, 2009)
8025-81-8	Spiramycin	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(CDC, 2012) (Chee-Sanford et al., 2001) (Gilchrist et al., 2007) (Health Care Without Harm, 2005) (Mellon et al., 2000) (Sapkota et al., 2007a) (USA Today, 2000) (Wallinga et al., 2006) (Wallinga, 2005)
121831-99-0	Strontium 90	Anonymous 197	None given	None given
58-22-0	Testosterone	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(Hotchkiss et al., 2007) (Kolpin et al., 2002)
52-68-6	Trichlorfon	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(USEPA, 1997) (USEPA, 1997)
101-20-2	Triclocarban	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(Chen et al., 2008) (Halden and Paull, 2004) (Halden and Paull, 2005) (Heidler et al., 2006) (Sapkota et al., 2007b) (TCC Consortium, 2002)

CASRN	Contaminant Name	Nominating Individual	Organization Name	Abbreviated References Cited in the Nomination
3380-34-5	Triclosan	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(Boyd et al., 2003) (Boyd et al., 2004) (Calafat et al., 2008b) (Dayan, 2007) (Fair et al., 2009) (Greyshock and Vikesland, 2006) (Hovander et al., 2002) (Jacobs et al., 2005) (Kolpin, et al., 2002) (Latch et al., 2005) (Loraine and Pettigrove, 2006) (Veldhoen et al., 2006)
1401-69-0	Tylosin	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(CDC, 2012) (Chee-Sanford et al., 2001) (Gilchrist et al., 2007) (Health Care Without Harm, 2005) (Mellon et al., 2000) (Sapkota et al., 2007a) (USA Today, 2000) (Wallinga et al., 2006) (Wallinga, 2005)
1404-90-6	Vancomycin	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(CDC, 2012) (Chee-Sanford et al., 2001) (Gilchrist et al., 2007) (Health Care Without Harm, 2005) (Mellon et al., 2000) (Sapkota et al., 2007a) (USA Today, 2000) (Wallinga et al., 2006) (Wallinga, 2005)
11006-76-1	Virginiamycin	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(CDC, 2012) (Chee-Sanford et al., 2001) (Gilchrist et al., 2007) (Health Care Without Harm, 2005) (Mellon et al., 2000) (Sapkota et al., 2007a) (USA Today, 2000) (Wallinga et al., 2006) (Wallinga, 2005)

[1] The organization that nominated "nonylphenol" for CCL 4 provided the CASRN of 25451-52-3. The name "nonylphenol" does not allow for a definitive identification of chemical structure since nonylphenol can exhibit two forms of isomerism. There are at least five CASRNs known to be associated with "nonylphenol:" in addition to 25154-52-3 (which represents n-nonylphenol with the ortho-, meta-, or para-substitution unspecified), other CASRNs include: 104-40-5 (4-n-nonylphenol); 84852-15-3 (4-nonylphenol, branched); 91672-41-2 (2-nonylphenol, branched); and 139-84-4 (3-n-nonylphenol). None of these five CASRNs is adequately general enough to represent both forms of isomerism. For the sake of consistency, the CASRN provided by the nominator was selected and the additional possible CASRNs and structures are delineated here.

[2] The Natural Resources Defense Council nomination letter contained several references that were included in a general discussion of phthalates. The references included in this general discussion are included under this listing. See the individual phthalates listings above for specific references.

Appendix 4. References Provided with Microbial Nominations

Contaminant Name	Nominating Individual	Organization Name	Abbreviated References Cited in the Nomination
Adenovirus	Thomas B. Atherholt	NJ Department of Environmental Protection	(Atkinson, 2012) (Borchart et al., 2008) (Gibbons et al., 2010) (Jiang, 2006) (Katayama et al., 2008) (Langley, 2005) (Post et al., 2011) (Rodriguez et al., 2008) (USEPA, 2006) (USEPA, 2011)
HPC Heterotrophic Plate Count Bacteria	Thomas B. Atherholt	NJ Department of Environmental Protection	(Atherholt et al., 2003) (Butscher et al., 2011) (Francy et al., 2000) (Francy et al., 2004) (Goeppert et al., 2011)
Naegleria fowleri	Thomas B. Atherholt	NJ Department of Environmental Protection	(Blair et al., 2008) (Post et al., 2011)
Toxoplasma gondii	J. Jones	None given	None given
Toxoplasma gondii	Jitender P.Dubey	U.S. Department of Agriculture	(de Moura et al., 2006) (Jones et al., 2010)
Vibrio cholerae	Mae C. Wu & Jennifer Sass	Natural Resources Defense Council	(Natural Resources Defense Council, 2010) (Onifade et al., 2011)

Appendix 5. Complete List of References Provided with CCL 4 Nominations

Note: References are cited as they were received from the nominating individual or organization.

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Appendix 6: Outcome of Nominated Chemicals in the CCL 4 Process

CASRN	Common Name – Registry Name	NPDWR or Proposed NPDWR	CCL 3 Universe	CCL 4 Universe	PCCL 3	PCCL 4	Final CCL 3	Final CCL 4[1]
54	**54**	**4**	**40**	**43**	**18**	**20**	**5**	**7**
77439-76-0	3-chloro-4-dichloromethyl-5-hydroxy-2(5H)-furanone		X	X				
319-84-6	alpha-Hexachlorocyclohexane		X	X	X	X	X	X
116-06-3	Aldicarb	X						
68555-24-8	Alkylphenol mono- to tri-oxylates							
26787-78-0	Amoxicillin							
86-50-0	Azinphos-methyl		X	X	X	X		
1405-89-6	Bacitracin zinc							
25057-89-0	Bentazone		X	X	X	X		
85-68-7	Benzyl butyl phthalate		X	X	X	X		
80-05-7	Bisphenol A		X	X	X	X		
1689-84-5	Bromoxynil		X	X				
63-25-2	Carbaryl		X	X	X	X		
10045-97-3	Cesium 137	X						
1897-45-6	Chlorothalonil		X	X	X	X		
2921-88-2	Chlorpyrifos		X	X				
84-74-2	Dibutyl phthalate		X	X				
1918-00-9	Dicamba		X	X				
62-73-7	Dichlorvos		X	X	X	X		
115-32-2	Dicofol		X	X	X	X		
84-61-7	Dicyclohexyl phthalate		X	X				
84-66-2	Diethyl phthalate		X	X				
28553-12-0	Di-isononyl phthalate		X	X				
131-11-3	Dimethyl phthalate		X	X				
117-84-0	Di-n-octyl phthalate		X	X				
115-29-7	Endosulfan		X	X	X	X		
2164-17-2	Fluometuron		X	X	X	X		
165800-03-3	Linezolid							
330-55-2	Linuron		X	X	X	X		
121-75-5	Malathion		X	X	X	X		
7439-96-5	Manganese		X	X		X		X

CASRN	Common Name – Registry Name	NPDWR or Proposed NPDWR	CCL 3 Universe	CCL 4 Universe	PCCL 3	PCCL 4	Final CCL 3	Final CCL 4[1]
61-32-5	Methicillin							
298-00-0	Methyl parathion		X	X				
1634-04-4	Methyl tertiary butyl ether (MTBE)		X	X	X	X	X	X
101043-37-2	Microcystin-LR		X	X	X	X	X	X
25154-52-3[2]	Nonylphenol		X	X		X		X
9016-45-9	Nonylphenol ethoxylate		X	X				
27193-28-8	Octylphenol		X	X				
9036-19-5	Octylphenol ethoxylate			X				
66-79-5	Oxacillin			X				
(multiple CASRNs)	Penicillin		X	X				
335-67-1	Perfluorooctanoic acid (PFOA)		X	X	X	X	X	X
52645-53-1	Permethrin		X	X	X	X	X	X
732-11-6	Phosmet		X	X	X	X		
57-83-0	Progesterone		X	X				
10043-92-2	Radon	X						
8025-81-8	Spiramycin							
121831-99-0	Strontium 90	X						
58-22-0	Testosterone		X	X				
52-68-6	Trichlorfon		X	X				
101-20-2	Triclocarban		X	X				
3380-34-5	Triclosan		X	X				
1401-69-0	Tylosin		X	X				
1404-90-6	Vancomycin							
11006-76-1	Virginiamycin			X				

[1]These seven contaminants were also listed on the Draft CCL 4

[2]The organization that nominated "nonylphenol" for CCL 4 provided the CASRN of 25451-52-3. The name "nonylphenol" does not allow for a definitive identification of chemical structure since nonylphenol can exhibit two forms of isomerism. There are at least five CASRNs known to be associated with "nonylphenol:" in addition to 25154-52-3 (which represents n-nonylphenol with the ortho-, meta-, or para-substitution unspecified), other CASRNs include: 104-40-5 (4-n-nonylphenol); 84852-15-3 (4-nonylphenol, branched); 91672-41-2 (2-nonylphenol, branched); and 139-84-4 (3-n-nonylphenol). None of these five CASRNs are adequately general enough to represent both forms of isomerism. For the sake of consistency, the CASRN provided by the nominator was selected and the additional possible CASRNs and structures are delineated here.

Appendix 7: Outcome of Nominated Microbes in the CCL 4 Process

Microbe	CCL 3 Universe	CCL 4 Universe	PCCL 3	PCCL 4	Final CCL 3	Final CCL 4[1]
Adenovirus	x	x	x	x	x	x
Naegleria fowleri	x	x	x	x	x	x
Toxoplasma gondii	x	x	x	x		
Vibrio cholerae	x	x	x	x		
Heterotrophic plate count (HPC)						

[1]These two microbes were also listed on the Draft CCL 4