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ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 141

[EPA-HQ-OW-2019-0583; FRL-10019-70-OW]

RIN 2040-AF93

Announcement of Final Regulatory Determinations for Contaminants on the Fourth Drinking Water Contaminant Candidate List

AGENCY: Environmental Protection Agency (EPA).

ACTION: Regulatory determinations.

SUMMARY: The U.S. Environmental Protection Agency (EPA or Agency) is announcing final regulatory determinations for eight of the 109 contaminants listed on the Fourth Contaminant Candidate List. Specifically, the Agency is making final determinations to regulate perfluorooctanesulfonic acid (PFOS) and perfluorooctanoic acid (PFOA) and to not regulate 1,1-dichloroethane, acetochlor, methyl bromide (bromomethane), metolachlor, nitrobenzene, and RDX. The Safe Drinking Water Act (SDWA), as amended in 1996, requires EPA to make regulatory determinations every five years on at least five unregulated contaminants. A regulatory determination is a decision about whether or not to begin the process to propose and promulgate a national primary drinking water regulation for an unregulated contaminant.

DATES: For purposes of judicial review, the determinations not to regulate in this document are issued as of [**INSERT DATE OF PUBLICATION IN THE *FEDERAL REGISTER***].

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SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

These final regulatory determinations will not impose any requirements on anyone. Instead, this action notifies interested parties of EPA's final regulatory determinations for eight unregulated contaminants and provides a summary of the major comments received on the March 10, 2020, preliminary determinations (USEPA, 2020a).

B. How Can I Get Copies of This Document and Other Related Information?

Docket: EPA has established a docket for this action under Docket ID No. EPA-HQ-OW-2019-0583. Publicly available docket materials are available either electronically at http://www.regulations.gov or in hard copy at the Water Docket, EPA/DC, EPA West, Room 3334, 1301 Constitution Ave., NW, Washington, DC. The telephone number for the Public Reading Room is (202) 566-1744, and the telephone number for the Water Docket is (202) 566-2426.

Electronic Access: You may access this *Federal Register* document electronically from the Government Printing Office under the "Federal Register" listings at *http://www.gpo.gov/fdsys/browse/collection.action?collectionCode=FR*.

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II. Purpose and Background

A. What is the Purpose of This Action?

The purpose of this action is to present a summary of EPA's final regulatory determinations for eight contaminants listed on the Fourth Contaminant Candidate List (CCL 4) (USEPA, 2016a). The eight contaminants are: perfluorooctanesulfonic acid (PFOS), perfluorooctanoic acid (PFOA), 1,1-dichloroethane, acetochlor, methyl bromide (bromomethane), metolachlor, nitrobenzene, and Royal Demolition eXplosive (RDX). The Agency is making final determinations to regulate two contaminants (PFOS and PFOA) and to not regulate the remaining six contaminants (1,1-dichloroethane, acetochlor, methyl bromide (bromomethane), metolachlor, nitrobenzene, and RDX). The Agency is not making any determination at this time on any other CCL contaminants, including strontium, 1,4-dioxane, and 1,2,3-trichloropropane. This action summarizes the statutory requirements for targeting drinking water contaminants for regulatory determination, provides an overview of the contaminants that the Agency considered for regulation, and describes the approach used to make the final regulatory determinations. In addition, this action summarizes the public comments received on the Agency's preliminary determinations announcement and the Agency's responses to those comments.

B. What are the Statutory Requirements for the Contaminant Candidate List (CCL) and Regulatory Determinations?

Section 1412(b)(1)(B)(i) of SDWA requires EPA to publish the CCL every five years after public notice and an opportunity to comment. The CCL is a list of contaminants which are not subject to any proposed or promulgated National Primary Drinking Water Regulations (NPDWRs) but are known or anticipated to occur in public water systems (PWSs) and may require regulation under SDWA. SDWA section 1412(b)(1)(B)(ii) directs EPA to determine, after public notice and an opportunity to comment, whether to regulate at least five contaminants from the CCL every five years.

Under Section 1412(b)(1)(A) of SDWA, EPA makes a determination to regulate a contaminant in drinking water if the Administrator determines that:

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

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

(c) In the sole judgment of the Administrator, regulation of such contaminant presents a meaningful opportunity for health risk reduction for persons served by public water systems.

If after considering public comment on a preliminary determination, the Agency makes a determination to regulate a contaminant, EPA will initiate the process to propose and promulgate an NPDWR. In that case, the statutory time frame provides for Agency proposal of a regulation within 24 months and action on a final regulation within 18 months of proposal. When proposing and promulgating drinking water regulations, the Agency must conduct a number of analyses.

C. What Contaminants did EPA Consider for Regulation?

On March 10, 2020, EPA published preliminary regulatory determinations for eight contaminants on the fourth Contaminant Candidate List (CCL 4) (85 FR 14098) (USEPA, 2020a). The eight contaminants are PFOS, PFOA, 1,1-dichloroethane, acetochlor, methyl bromide, metolachlor, nitrobenzene, and RDX. The Agency is making final regulatory determinations to regulate two contaminants (i.e., PFOS and PFOA) and to not regulate six contaminants (i.e., 1,1-dichloroethane, acetochlor, methyl bromide, metolachlor, nitrobenzene, and RDX).

Information on the eight contaminants with regulatory determinations can be found in the *Final Regulatory Determination 4 Support Document* (USEPA, 2021a). More information is available in the Public Docket at *www.regulations.gov* (Docket ID No. EPA–HQ–OW–2019–

0583) and also on EPA's Regulatory Determination 4 website at https://www.epa.gov/ccl/regulatory-determination-4.

III. What Process did EPA Use to Make the Regulatory Determinations?

A. How EPA Identified and Evaluated Contaminants for the Fourth Regulatory Determination

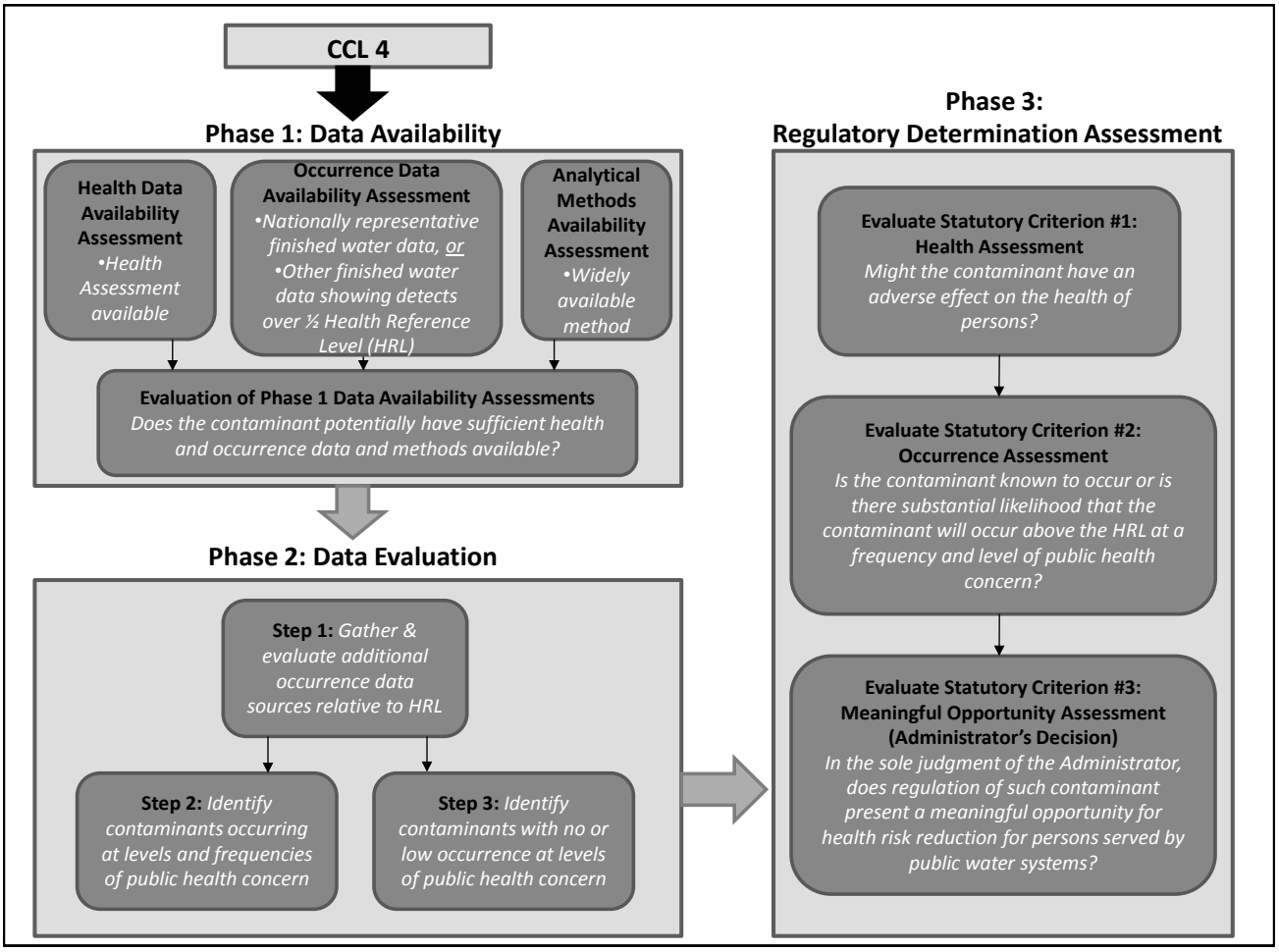
This section summarizes the process the Agency followed to identify and evaluate contaminants for the Fourth Regulatory Determination. For more detailed information on the process and the analyses performed, please refer to the "Protocol for the Regulatory Determination 4" found in Appendix E of the *Final Regulatory Determination 4 Support Document* (USEPA, 2021a) and the *Federal Register* publication for the preliminary regulatory determinations (USEPA, 2020a).

The CCL 4 identified 109 contaminants that are currently not subject to any proposed or promulgated national drinking water regulation, are known or anticipated to occur in public water systems, and may require regulation under SDWA (USEPA, 2016a). Since some of the CCL 4 contaminants do not have adequate health and/or occurrence data to evaluate against the three statutory criteria (see section II.B of this document), as when EPA evaluated the previous CCLs, the Agency used a three-phase process to identify which of the contaminants are candidates for regulatory determinations. Priority was given to identifying contaminants known to occur or with substantial likelihood to occur at frequencies and levels of public health concern.

Because the regulatory determination process includes consideration of human health effects, the Agency's Policy on Evaluating Health Risks to Children (USEPA, 1995a) reaffirmed by Administrator Wheeler in a memorandum dated October 11, 2018 to Agency staff (USEPA, 2018a), applies to this document. The policy requires EPA to consistently and comprehensively address children's unique vulnerabilities. We have explicitly considered children's health in the RD 4 process by reviewing all the available children's exposure and health effects information.

The three phases of the Fourth Regulatory Determination process are (1) the Data Availability Phase, (2) the Data Evaluation Phase and (3) the Regulatory Determination Assessment Phase. The overall process is displayed in **Exhibit 1**.

Exhibit 1: The Three Phases of the Regulatory Determination 4 Process



The purpose of the first phase, the *Data Availability Phase,* is to screen out contaminants that clearly do not have sufficient data to support a regulatory determination. The Agency applies criteria to ensure that any contaminant that potentially has sufficient data to characterize the health effects and known or likely occurrence in drinking water will proceed to the *Data Evaluation Phase*, the second phase of the regulatory determination process. From the 109 CCL 4 contaminants, the Agency identified 25 CCL 4 contaminants to further evaluate in the second phase. These are known as the "short list."

During the second phase, the Agency evaluates the contaminants on the short list in greater depth and detail to identify those that have sufficient data (or are expected to have

sufficient data within the timeframe allotted for the second phase) for EPA to assess the three statutory criteria. As part of the second phase, the Agency specifically focuses its efforts on identifying those contaminants or contaminant groups that are occurring or have substantial likelihood to occur at levels and frequencies of public health concern, based on the best available peer reviewed data. If, during the first or second phase, the Agency finds that sufficient data are not available or not likely to be available to evaluate the three statutory criteria, then the contaminant is not considered a candidate for making a regulatory determination.

If sufficient data are available for a contaminant to characterize the potential health effects and known or likely occurrence in drinking water, the contaminant is evaluated against the three statutory criteria in the *Regulatory Determination Assessment Phase*, which is the third phase of the process. Of the 25 contaminants that were evaluated under Phase 2, 10 were designated for evaluation against the three statutory criteria in Phase 3.

Of the 10 CCL4 contaminants that were evaluated in Phase 3, the Agency did not make preliminary regulatory determinations for two contaminants (1,4-dioxane and 1,2,3-trichloropropane); see Section IV of this document for discussion about these contaminants. Additionally, in Section IV of this document, EPA discusses continuing with its previous 2016 decision to defer a final determination for strontium (a CCL3 contaminant for which the Agency made a preliminary positive determination in the third regulatory determination (RD 3)) in order to further consider additional studies related to strontium exposure.

Of the eight remaining CCL 4 contaminants (PFOS, PFOA, 1,1-dichloroethane, acetochlor, methyl bromide, metolachlor, nitrobenzene, and RDX) evaluated in Phase 3 against the three statutory criteria, including an evaluation of level and frequency of occurrence in drinking water, the size of the population exposed to concentrations of health concern, and information on sensitive populations and lifestages[1] (e.g., pregnant women, infants and children), the Agency made preliminary regulatory determinations to regulate PFOS and PFOA and to not

[1] https://www.epa.gov/children/childhood-lifestages-relating-childrens-environmental-health

regulate the remaining six contaminants. These preliminary determinations, with their supporting analyses and documentation, were published in the *Federal Register* on March 10, 2020, for public comment (USEPA, 2020a). The public comment period was initially intended to run through May 11, 2020. In response to stakeholder requests, on April 30, 2020, EPA extended the comment period by 30 days to June 10, 2020.

B. Consideration of Public Comments

EPA received comments from approximately 11,600 organizations and individuals on the March 10, 2020, *Federal Register* document including 12 states (California, Colorado, Connecticut, Indiana, Massachusetts, Michigan, Missouri, New Hampshire, New Mexico, South Carolina, West Virginia, and Wisconsin). Comments on specific contaminants, and EPA's responses, are briefly summarized in the sections below. The Agency prepared a response-to-comments document for this action (USEPA, 2021b) that is available in the Public Docket at *www.regulations.gov* under Docket ID No. EPA-HQ-OW-2019-0583. The response-to-comments document is organized in a manner similar to this document and generally contains more detailed responses to the public comments received than those found in this document.

IV. EPA's Findings on Specific Contaminants

After considering the public comments, EPA is making final regulatory determinations to regulate PFOS and PFOA and to not regulate 1,1-dichloroethane, acetochlor, methyl bromide, metolachlor, nitrobenzene, and RDX.

This document provides a brief description of the Agency findings on these contaminants. Details on the background, health and occurrence information, and analyses used to evaluate and make final determinations for these contaminants can be found in the *Final Regulatory Determination 4 Support Document* (USEPA, 2021a) and the *Federal Register* publication for the preliminary regulatory determination (USEPA, 2020a).

For each contaminant, the Agency reviewed the available human and toxicological data, derived a health reference level (HRL),[2] analyzed data on occurrence in drinking water, and estimated the population likely exposed to concentrations of the contaminant at levels of health concern in public water systems. The Agency also considered whether information was available on sensitive populations. The Agency used the findings to evaluate the contaminants against the three SDWA statutory criteria. **Table 1** gives a summary of the health and occurrence information for the eight contaminants with final determinations under RD 4.

Table 1. Summary of the Health and Occurrence Information and the Final Determinations for the Eight Contaminants Receiving a Final Determination under RD 4

RD 4 contaminant	Health reference level (HRL), µg/L	Occurrence Findings from Primary Data Sources					Final determination
		Primary database	PWSs with at least 1 detection > ½ HRL	Population served by PWSs with at least 1 detection > ½ HRL	PWSs with at least 1 detection > HRL	Population served by PWSs with at least 1 detection > HRL	
PFOS	0.07	UCMR 3 AM	95 / 4,920 (1.93%)	10,427,193 / 241 M (4.32%)	46 / 4,920 (0.93%)	3,789,831 / 241 M (1.57%)	Regulate.
PFOA	0.07	UCMR 3 AM	53 / 4,920 (1.07%)	3,652,995 / 241 M (1.51%)	13 / 4,920 (0.26%)	490,480 / 241 M (0.20%)	Regulate.
1,1-Dichloroethane	1,000	UCMR 3 AM	0 / 4,916 (0.00%)	0 / 241 M (0.00%)	0 / 4,916 (0.00%)	0 / 241 M (0.00%)	Do not regulate.
Acetochlor	100	UCMR 1 AM UCMR 2 SS	0 / 3,869 (0.00%) – UCMR 1 0 / 1,198 (0.00%) – UCMR 2	0 / 226 M (0.00%) – UCMR 1 0 / 157 M (0.00%) – UCMR 2	0 / 3,869 (0.00%) – UCMR 1 0 / 1,198 (0.00%) – UCMR 2	0 / 226 M (0.00%) – UCMR 1 0 / 157 M (0.00%) – UCMR 2	Do not regulate.
Methyl Bromide (Bromomethane)	100	UCMR 3 AM	0 / 4,916 (0.00%)	0 / 241 M (0.00%)	0 / 4,916 (0.00%)	0 / 241 M (0.00%)	Do not regulate.
Metolachlor	300	UCMR 2 SS	0 / 1,198 (0.00%)	0 / 157 M (0.00%)	0 / 1,198 (0.00%)	0 / 157 M (0.00%)	Do not regulate.
Nitrobenzene	10	UCMR 1 AM	2 / 3,861 (0.05%)	255,358 / 226 M (0.11%)	2 / 3,861 (0.05%)	255,358 / 226 M (0.11%)	Do not regulate.
RDX	30 (noncancer) 0.4 (cancer)	UCMR 2 AM	0 / 4,139 (0.00%) > 15 µg/L 3 / 4,139 (0.07%) > 0.2 µg/L	0 / 229 M (0.00%) > 15 µg/L 96,033 / 229 M (0.04%) > 0.2 µg/L	0 / 4,139 (0.00%) > 30 µg/L 3 / 4,139 (0.07%) > 0.4 µg/L	0 / 229 M (0.00%) > 30 µg/L 96,033 / 229 M (0.04%) > 0.4 µg/L	Do not regulate.

[2] An HRL is a health-based concentration against which the Agency evaluates occurrence data when making decisions about preliminary regulatory determinations. An HRL is not a final determination on establishing a protective level of a contaminant in drinking water for a particular population; it is derived prior to development of a complete health and exposure assessment and can be considered a screening value. See Section E.5.1 of the *Final Regulatory Determination 4 Support Document* for information about how HRLs are derived (USEPA, 2021a).

A. PFOS and PFOA

1. Description

Per- and polyfluoroalkyl substances (PFAS) are a class of synthetic chemicals that have been manufactured and in use since the 1940s (AAAS, 2020; USEPA, 2018b). PFAS are most commonly used to make products resistant to water, heat, and stains and are consequently found in industrial and consumer products like clothing, food packaging, cookware, cosmetics, carpeting, and fire-fighting foam (AAAS, 2020). PFAS manufacturing and processing facilities, facilities using PFAS in production of other products, airports, and military installations have been associated with PFAS releases into the air, soil, and water (USEPA 2016b; USEPA 2016c). People may potentially be exposed to PFAS through the use of certain consumer products, through occupational exposure, and/or through consuming contaminated food or contaminated drinking water (Domingo and Nadal, 2019; Fromme et al. 2009).

Perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA) are part of a subset of PFAS referred to as perfluorinated alkyl acids (PFAA) and are two of the most widely studied and longest-used PFAS. Due to their widespread use and persistence in the environment, most people have been exposed to PFAS, including PFOA and PFOS (USEPA 2016b; USEPA 2016c). PFOA and PFOS have been detected in up to 98% of serum samples taken in biomonitoring studies that are representative of the U.S. general population (CDC, 2019). Following the voluntary phase-out of PFOA by eight major chemical manufacturers and processors in the United States under EPA's 2010/2015 PFOA Stewardship Program and reduced manufacturing of PFOS (last reported in 2002 under Chemical Data Reporting), serum concentrations have been declining. The National Health and Nutrition Examination Survey (NHANES) data exhibited that 95th-percentile serum PFOS concentrations have decreased over

75%, from 75.7 µg/L in the 1999-2000 cycle to 18.3 µg/L in the 2015-2016 cycle (CDC, 2019; Jain, 2018; Calafat et al., 2007; Calafat et al., 2019).

2. Agency Findings

The Agency is making a determination to regulate PFOA and PFOS with a NPDWR. EPA has determined that PFOA and PFOS may have adverse health effects; that PFOA and PFOS occur in public water systems with a frequency and at levels of public health concern; and that, in the sole judgment of the Administrator, regulation of PFOA and PFOS presents a meaningful opportunity for health risk reduction for persons served by public water systems.

a) Adverse Health Effects

The Agency finds that PFOA and PFOS may have adverse effects on the health of persons. In 2016, EPA published health assessments (Health Effects Support Documents or HESDs) for PFOA and PFOS based on the Agency's evaluation of the peer reviewed science available at that time. The lifetime Health Advisory (HA) of 0.07 µg/L is used as the HRL for Regulatory Determination 4 and reflect concentrations of PFOA and PFOS in drinking water at which adverse health effects are not anticipated to occur over a lifetime. Studies indicate that exposure to PFOA and/or PFOS above certain exposure levels may result in adverse health effects, including developmental effects to fetuses during pregnancy or to breast-fed infants (e.g., low birth weight, accelerated puberty, skeletal variations), cancer (e.g., testicular, kidney), liver effects (e.g., tissue damage), immune effects (e.g., antibody production and immunity), and other effects (e.g., cholesterol changes). Both PFOA and PFOS are known to be transmitted to the fetus via the placenta and to the newborn, infant, and child via breast milk. Both compounds were also associated with tumors in long-term animal studies (USEPA, 2016d; USEPA, 2016e; NTP, 2020). For specific details on the potential for adverse health effects and approaches used to identify and evaluate information on hazard and dose-response, please see (USEPA, 2016b; USEPA, 2016c; USEPA, 2016d; USEPA, 2016e).

b) Occurrence

EPA has determined that PFOA and PFOS occur with a frequency and at levels of public health concern at PWSs based on the Agency's evaluation of available occurrence information. In accordance with SDWA 1412(b)(1)(B)(ii)(II), EPA has determined monitoring data from the third Unregulated Contaminant Monitoring Rule (UCMR 3) are the best available occurrence information for PFOA and PFOS regulatory determinations. UCMR 3 monitoring occurred between 2013 and 2015 and are currently the only nationally representative finished water dataset for PFOA and PFOS. Under UCMR 3, 36,972 samples from 4,920 PWSs were analyzed for PFOA and PFOS. The minimum reporting level (MRL) for PFOA was 0.02 µg/L and the MRL for PFOS was 0.04 µg/L. A total of 1.37% of samples had reported detections (greater than or equal to the MRL) of at least one of the two compounds. To examine the occurrence of PFOS and PFOA in aggregate, EPA summed the concentrations detected in the same sample to calculate a total PFOS/PFOA concentration. EPA notes that the reference doses (RfDs) for both PFOA and PFOS are based on similar developmental effects and are numerically identical; when these two chemicals co-occur at the same time and location in drinking water sources, EPA has recommended considering the sum of the concentrations (USEPA, 2016d; USEPA, 2016e) and has done so for this regulatory determination. The maximum summed concentration of PFOA and PFOS was 7.22 µg/L and the median summed value was 0.05 µg/L. Summed PFOA and PFOS concentrations exceeded one-half the HRL (0.035 µg/L) at a minimum of 2.4% of PWSs (115 PWSs) and exceeded the HRL (0.07 µg/L) at a minimum of 1.3% of PWSs (63 PWSs [3]). Since UCMR 3 monitoring occurred, certain sites where elevated levels of PFOA and PFOS were detected may have installed treatment for PFOA and PFOS, may have chosen to blend water from multiple sources, or may have otherwise remediated known sources of contamination. Those 63 PWSs serve a total population of approximately 5.6 million people and

[3] Sum of PFOA + PFOS results rounded to 2 decimal places in those cases where a laboratory reported more digits.

are located in 25 states, tribes, or U.S. territories (USEPA, 2019a). Data from more recent state monitoring (discussed below) demonstrate occurrence in multiple geographic locations consistent with UCMR 3 monitoring and support the Agency's final determination that PFOA and PFOS occur with a frequency and at levels of public health concern in finished drinking water across the United States. The *Final Regulatory Determination 4 Support Document* presents a sample-level summary of the results for PFOA and PFOS individually and includes discussion on state monitoring efforts as well as uncertainties in occurrence data (USEPA, 2021a).

Consistent with the Agency's commitment in the PFAS Action Plan (the Agency's first multi-media, multi-program, national research, management, and risk communication plan to address a challenge like PFAS) to present information about additional sampling efforts for PFAS in water systems, the Agency has supplemented its Unregulated Contaminant Monitoring Regulation (UCMR) data with data collected by states who have made their data publicly available at this time (USEPA, 2019b). A summary of these occurrence data were presented in the preliminary Regulatory Determination 4 Federal Register document. Subsequent to the preliminary announcement, based on comments and information received on the proposed determination, the Agency collected additional data from additional states. The finished water data available from fifteen states collected since UCMR 3 monitoring showed that there were at least 29 PWSs where the summed concentrations of PFOA and PFOS exceeded the EPA HRL. The Agency notes that some of these data are from targeted sampling efforts and thus may not be representative of levels found in all PWSs within the state or represent occurrence in other states. The state data demonstrate occurrence in multiple geographic locations and support EPA's finding that PFOA and PFOS occur with a frequency and at levels of public health concern in drinking water systems across the United States. The Final Regulatory Determination 4 Support Document presents a detailed discussion of state PFOA and PFOS occurrence information (USEPA, 2021a). EPA acknowledges that there may be other states with occurrence data

available and that additional states have or intend to conduct monitoring of finished drinking water. As such, EPA will consider any new or additional state data to inform the development of the proposed NPDWR for PFOA and PFOS.

c) Meaningful Opportunity

Considering the population exposed to PFOA and PFOS including sensitive populations and lifestages, the potential adverse human health impacts of these contaminants, the environmental persistence of these substances, the persistence in the human body and potential for bioaccumulation of these substances, the availability of validated methods to measure and treatment technologies to remove PFOA and PFOS, the detections that exceeded the HRL and ½ the HRL, and significant public concerns (particularly those expressed in comments submitted by state and local government agencies) on the challenges that these contaminants pose for communities nationwide, the Agency has determined that regulation of PFOA and PFOS presents a meaningful opportunity for health risk reduction for persons served by PWSs, including sensitive populations such as infants, children, and pregnant and nursing women.

PFOA and PFOS are both generated as degradation products of other perfluorinated compounds (e.g., fluorotelomer alcohols), and due to their strong carbon-fluorine bonds, are resistant to metabolic and environmental degradation (USEPA, 2016b; USEPA, 2016c). Due to this underlying chemical structure, PFOA and PFOS are extremely persistent in the environment, including resistance to chemical, biological, and physical degradation processes. While most U.S. manufacturers have voluntarily phased out production and manufacturing of both PFOS and PFOA, their environmental persistence and formation as degradation products from other compounds may still contribute to their release in the environment. Upon exposure to the human body, there is a potential for bioaccumulation and toxicity at environmentally relevant concentrations as studies show it can take years to leave the human body (NIEHS, 2020; USEPA, 2016b; USEPA, 2016c).

Adverse effects observed following exposures to PFOA and PFOS include effects in humans on serum lipids, birth weight, and serum antibodies. Some of the animal studies show common effects on the liver, neonate development, and responses to immunological challenges. Both compounds were also associated with tumors in long-term animal studies (USEPA, 2016d; USEPA, 2016e). In determining that regulation of PFOA and PFOS presents a meaningful opportunity for health risk reduction for sensitive populations, EPA noted that both PFOA and PFOS are associated with developmental toxicity in animals, with reduced birth weight in humans, and have been shown to be transmitted to the fetus via the placenta and to the newborn, infant, and child via breast milk (USEPA, 2016b; USEPA, 2016c).

Drinking water analytical methods are available to measure PFOA, PFOS, and other PFAS in drinking water. EPA has published validated drinking water laboratory methods for detecting a total of 29 unique PFAS in drinking water, including EPA Method 537.1 (18 PFAS) and EPA Method 533 (25 PFAS).

Available treatment technologies for removing PFAS from drinking water have been evaluated and reported in the literature (e.g., Dickenson and Higgins, 2016). EPA's Drinking Water Treatability Database (USEPA, 2020b) summarizes available technical literature on the efficacy of treatment technologies for a range of priority drinking water contaminants, including PFOA and PFOS. In summary, conventional treatment (comprised of the unit processes coagulation, flocculation, clarification, and filtration) is not considered effective for the removal of PFOA and PFOS. Granular activated carbon (GAC), anion exchange resins, reverse osmosis and nanofiltration are considered effective for the removal of PFOA and PFOS.

d) Summary of Public Comments on PFOA and PFOS and Agency Responses

EPA received many comments on the Agency's evaluation of the first statutory criterion under section 1412(b)(1)(A) of SDWA. Most commenters agreed with EPA's finding that PFOA and PFOS may have adverse effects on the health of persons. Most commenters also state that

there is “strong evidence” and “substantial scientific evidence” for EPA’s finding of adverse health effects of PFOA and PFOS. One commenter disagreed with EPA’s evaluation of the first statutory criterion, arguing that the body of scientific evidence does not show adverse effects from PFAS in humans. EPA also received numerous comments relating to the Agency’s 2016 Lifetime Health Advisory for PFOA and PFOS, the corresponding HESD and the HRL used to support the preliminary regulatory determination. Numerous commenters encouraged EPA to update and “improve its health reference level” and “revise the PFOA and PFOS hazard assessments” prior to making a final regulatory determination.

EPA acknowledges commenters’ suggestions to consider and evaluate newer studies; however, EPA disagrees with recommendations to establish new HRLs prior to a final regulatory determination. Consistent with SDWA section 1412(b)(3)(A)(i), EPA is using the 2016 PFOA and PFOS Lifetime Health Advisory as the basis in deriving an HRL which the Agency has concluded represent the best available peer reviewed scientific assessment at this time. Based upon the 2016 EPA HESDs for PFOA and PFOS, and other supporting studies cited in the record, EPA finds that PFOA and PFOS may have an adverse effect on the health of persons. Consistent with commenters’ recommendations, EPA has initiated the first steps of a systematic literature review of peer-reviewed scientific literature for PFOA and PFOS published since 2013 with the goal of identifying any new studies that may be relevant to human health assessment. An annotated bibliography of the identified relevant studies as well as the protocol used to identify the relevant publications can be found in Appendix D of the Final Regulatory Determination 4 Support Document (USEPA, 2021a), available in the docket for this document. Additional analyses of these new studies is needed to confirm relevance, extract the data to assess the weight of evidence, and identify critical studies in order to inform future decision making.

EPA also received comments on the Agency’s evaluation of the second statutory criterion under section 1412(b)(1)(A) of SDWA. Many commenters supported EPA’s preliminary

determination that PFOA and PFOS meet the second statutory occurrence criterion under SDWA. Several commenters stated that while they are supportive of using UCMR 3 data as the basis of nationwide drinking water occurrence for PFOA and PFOS, solely relying on these monitoring data may be an inaccurate reflection of PFOA and PFOS exposure. The Agency also received comments and information on actions taken by a number of states to monitor PFOA, PFOS, and other PFAS in PWSs, particularly in locations that were not previously required to conduct UCMR monitoring. Some commenters suggested that PFOA and PFOS UCMR 3 occurrence information used by EPA in making the Preliminary Determination for PFOA and PFOS is not reflective of the actual occurrence of PFOS and PFOS within public water systems. These commenters stated that UCMR 3 monitoring excludes small public water systems and was conducted with high minimum reporting levels. Three commenters did not support EPA's preliminary determination that PFOA and PFOS meet the second statutory criterion under SDWA. These commenters expressed concern that the data EPA relied upon are outdated, are skewed, and overestimate current PFOA and PFOS occurrence. These commenters suggest that EPA should revise its occurrence analysis with more recent data prior to making a final determination.

EPA disagrees with those commenters who assert that UCMR 3 are not the best available occurrence data. EPA also disagrees that the UCMR 3 excludes small water systems and disagrees that the minimum reporting levels were too high. The UCMR 3 assured a nationally representative sample of 800 small drinking water systems and established minimum reporting levels based upon laboratory performance data that are lower than the HRLs for PFOA and PFOS. The UCMR 3 data are the best available information to assess the frequency and level of occurrence of PFOA and PFOS in the nation's public water systems. After considering the public comments and additional occurrence data provided by commenters, EPA continues to find that PFOA and PFOS meet the second statutory criterion for regulatory determinations under Section 1412(b)(1)(A) of SDWA that "the contaminant is known to occur or there is a substantial

likelihood that the contaminant will occur in public water systems with a frequency and at levels of public health concern." Nonetheless, EPA agrees with commenters who recommend that the Agency consider other existing available occurrence data to inform its final regulatory determination and PFOA and PFOS rulemaking. As discussed previously, the Final Regulatory Determination 4 Support Document presents a detailed discussion of state PFOA and PFOS occurrence information that were analyzed and used to further support the Agency's finding that PFOA and PFOS occur in public water systems with a frequency and at levels of public health concern (USEPA, 2021a).

EPA also received many comments on the Agency's evaluation of the third statutory criterion under section 14121412(b)(1)(A) of SDWA. Many commenters, including multiple state regulators and organizations representing states, agree with EPA's evaluation that regulation of PFOA and PFOS presents a meaningful opportunity for health risk reduction for persons served by PWSs. These commenters highlight the extensive amount of work associated with developing their own drinking water standards for several PFAS compounds. These commenters also noted the need for a consistent national standard for use in states where a state-specific standard has not yet been developed. Many commenters have also noted that although some states have developed or are in the process of developing their own state-level PFAS drinking water standards, regulatory standards currently vary across states. These commenters expressed concern that absence of a national drinking water standard has resulted in risk communication challenges with the public and disparities with PFAS exposure. Some commenters noted there are populations particularly sensitive or vulnerable to the health effects of PFAS, including newborns, infants and children. One commenter did not support EPA's evaluation of the third statutory criterion, noting that in their opinion, the toxicity assessment for PFOA and PFOS and existing occurrence data do not suggest that establishing drinking water standards presents a meaningful opportunity for health risk reduction.

EPA acknowledges commenter concerns regarding sensitive and vulnerable subpopulations and notes that the Agency has been particularly mindful that PFOA and PFOS are known to be transmitted to the fetus via cord blood and to the newborn, infant and child via breast milk. EPA agrees with commenters that there is a need for protective drinking water regulations across the United States and that moving forward with a national-level regulation for PFOA and PFOS would provide improved national consistency in protecting public health and may reduce regulatory uncertainty for stakeholders across the country. The Agency disagrees with the commenter's assertion that PFOA and PFOS health and occurrence information are insufficient to justify a drinking water standard, and the Agency finds that there is a meaningful opportunity for health risk reduction potential based upon consideration the population exposed to PFOA and PFOS including sensitive populations and lifestages, such as newborns, infants and children.

3. Considerations for Additional PFAS

As EPA begins the process to promulgate the NPDWR for PFOA and PFOS, the Agency recognizes that there is additional information to consider regarding a broader range of PFAS, including new monitoring and occurrence data, and ongoing work developing toxicity assessments by EPA, other federal agencies, state governments, international organizations, industry groups, and other stakeholders. While the Agency is not making regulatory determinations for additional PFAS at this time, the Agency remains committed to filling information gaps, including those identified in the PFAS Action Plan, by completing peer reviewed toxicity assessments and collecting nationally representative occurrence data for additional PFAS to support future regulatory determinations as part of the UCMR monitoring program (see discussion below).

EPA committed in the PFAS Action Plan to characterize potential health impacts and develop more drinking water occurrence data for a broader set of PFAS (USEPA, 2019b). EPA has followed through on its commitments and as a result expects to have peer-reviewed health

assessments and national occurrence data for more PFAS becoming available over the next few years. EPA notes that although SDWA does not require the Agency to complete regulatory determinations for the contaminants from the fifth CCL until 2026, because of the significant progress related to developing new high-quality PFAS information, combined with the Agency's commitment in the PFAS Action Plan to assist states and communities with PFAS contaminated drinking water, EPA will continue to prioritize regulatory determinations of additional PFAS in drinking water. The Agency is committing to making regulatory determinations in advance of the next SDWA deadline for additional PFAS for which the Agency has a peer reviewed health assessment, has nationally representative occurrence data in finished drinking water, and has sufficient information to determine whether there is a meaningful opportunity for health risk reduction for persons served by public water systems.

EPA is currently developing scientifically rigorous toxicity assessments for seven PFAS chemicals. The chemicals currently undergoing assessment include PFBS, PFBA, PFHxS, PFHxA, PFNA, PFDA, and HFPO-DA (GenX chemicals), all of which are currently scheduled to be completed by 2023. These assessments all include public comment periods, independent scientific external peer review, and a robust interagency review process. Furthermore, these toxicity assessments will provide critical health information for PFAS with varying chain lengths and functional groups. When complete, these assessments will summarize available scientific information regarding the anticipated human dose-response relationship for these chemicals, which is a key information need for informing a variety of Agency decisions.

To inform EPA's understanding of PFAS occurrence in drinking water as discussed in EPA's PFAS Action Plan (USEPA, 2019b), the Agency is also leading efforts to gather additional monitoring data for 29 PFAS contaminants in finished drinking water. EPA recently announced its proposal for nationwide drinking water monitoring for PFAS under the next UCMR monitoring cycle (UCMR 5) utilizing Methods 537.1 and 533 to detect more PFAS chemicals and at lower reporting limits than previously possible.

EPA is also is generating new PFAS toxicology data for a much larger set of less-studied PFAS through new approach methods (NAMs)[4] such as high throughput screening, computational toxicology tools, and chemical informatics for chemical prioritization, screening, and risk assessment. EPA will continue research on methods for using these data to support risk assessments using NAMs such as read-across (i.e., an effort to predict biological activity based on similarity in chemical structure) and transcriptomics (i.e., a measure of changes in gene expression in response to chemical exposure or other external stressors), and to make inferences about the toxicity of PFAS mixtures that commonly occur in real world exposures. This research can inform a more complete understanding of PFAS toxicity for the large set of PFAS chemicals without conventional toxicity data and can allow prioritization of actions to potentially address groups of PFAS. For additional information on the NAMs for PFAS toxicity testing, please visit: https://www.epa.gov/chemical-research/pfas-chemical-lists-and-tiered-testing-methods-descriptions. These EPA actions, in addition to other research, may provide useful information for future EPA evaluations of additional PFAS.

a) Summary of Public Comments on Considerations for Additional PFAS and Agency Responses

EPA requested comment on potential regulatory constructs the Agency may consider for PFAS chemicals including PFOA and PFOS. EPA specifically requested input on a regulatory approach to evaluate PFAS by different grouping approaches.

EPA received multiple comments on how the Agency could consider additional PFAS for potential future rulemaking. Many commenters support a class-based approach for regulating PFAS based on one or more characteristics such as chain length, functional group, treatment processes, health effects, toxicity, common analytical methods, and/or shared occurrence with

[4] New approach methods (NAMs) refer to any technologies, methodologies, approaches, or combinations thereof that can be used to provide information on chemical hazard and potential human exposure that can avoid or significantly reduce the use of testing on animals.

other contaminants within a group. Additionally, many commenters also urge EPA to make additional regulatory determinations for PFAS that have a proposed or final drinking water standard in at least one state; PFAS that have been measured in water systems through monitoring programs such as UCMR; and/or PFAS for which EPA or the Agency for Toxic Substances and Disease Registry (ATSDR) has established a toxicity value. Some commenters suggest that EPA should make positive regulatory determinations for PFHxS and PFNA as well as in combination with PFOA, PFOS, and other PFAS such as PFBS. Many commenters recommend EPA consider various grouping and treatment technique approaches for PFAS beyond PFOA and PFOS that may not have sufficient health and occurrence data. Some of these commenters recommend approaches that consider acute and chronic health effects, long-term compared to short-term exposures, exposures during sensitive lifestages, and type of water systems and vulnerable populations such as vulnerable workers. Many commenters stated that the data may not be robust enough for each PFAS and therefore support a class-based approach for regulating PFAS in drinking water. In contrast, two commenters did not support a class-based approach for regulating PFAS. In summary, these commenters suggest that regulation without assessing each chemical's individual traits "would be contrary to the intent of SDWA" and that the Agency should address outstanding data and knowledge gaps regarding PFAS of concern prior to determining a regulatory grouping approach.

With respect to comments received on regulatory determinations for additional PFAS compounds other than PFOA and PFOS, EPA remains committed to filling information gaps by completing peer reviewed health assessments where appropriate and collecting nationally representative occurrence data. As discussed above, in response to public comments advocating timely regulation of additional PFAS in drinking water, where sufficient information is available, EPA intends to make regulatory determinations for additional PFAS prior to the fifth Regulatory Determination's statutory deadline (2026).

The Agency acknowledges many commenters' support for a class-based approach for regulating PFAS and appreciates commenter recommendations regarding potential regulatory constructs. EPA acknowledges commenters' recommendations to evaluate whether PFAS can be regulated as groups, and the Agency is developing the science necessary to consider whether such regulation is necessary and appropriate for PFAS. Regarding commenters' assertions that regulation without assessing each chemical's individual traits "would be contrary to the intent of SDWA," the Agency notes that the Safe Drinking Water Act establishes a robust scientific and public participation process that guide EPA's development of regulations for unregulated contaminants that may present a risk to public health. Regulation by groups is a regulatory strategy that is already used for certain regulated contaminants like disinfection byproducts, polychlorinated biphenyls, and radionuclides. EPA will continue to use best available science and available statutory authorities to guide Agency decision making with respect to how the Agency evaluates and potentially regulates additional PFAS.

b) Summary of Public Comments on Potential PFAS Monitoring Approaches and Agency Responses

As part of the proposed preliminary regulatory determination for PFOA and PFOS, EPA solicited comment on potential monitoring approaches if the Agency were to finalize a positive regulatory determination for these contaminants. EPA presented two monitoring approaches in the Agency's preliminary Regulatory Determination for CCL 4 contaminants. Under the Standardized Monitoring Framework (SMF) for synthetic organic chemicals, monitoring schedules are based around the detection levels of the regulated contaminants, and state primacy agencies can also issue waivers for monitoring. The Agency also presented an alternative monitoring approach to allow state primacy agencies to require monitoring at PWSs where information indicates potential PFAS contamination, such as proximity to facilities with historical or on-going uses of PFAS.

Many commenters supported the Agency's goal of reducing potential monitoring burden for PWSs without compromising public health protection. While there were differing views among commenters regarding which monitoring approach is best for PFAS, many urged EPA to keep evaluating different approaches as the Agency promulgates the NPDWR for PFOA and PFOS.

The Agency appreciates commenter recommendations on monitoring approaches. As the Agency promulgates the regulatory standard for PFOA and PFOS, EPA will continue to work to establish monitoring requirements in the rule that minimize burden while ensuring public health protection.

B. 1,1-Dichloroethane

1. Description

1,1-Dichloroethane is a halogenated alkane. It is an industrial chemical and is used as a solvent and a chemical intermediate. 1,1-Dichloroethane is expected to have moderate to high persistence in water (USEPA, 2021a).

2. Agency Findings

The Agency is making a determination not to regulate 1,1-dichloroethane with an NPDWR. It does not occur with a frequency and at levels of public health concern. As a result, the Agency finds that an NPDWR does not present a meaningful opportunity for health risk reduction.

a) Adverse Health Effects

The Agency finds that 1,1-dichloroethane may have adverse effects on the health of persons. Based on a 13-week gavage study in rats (Muralidhara et al., 2001), the kidney was identified as a sensitive target for 1,1-dichloroethane, and no-observed-adverse-effect level (NOAEL) and lowest-observed-adverse-effect level (LOAEL) values of 1,000 and 2,000

mg/kg/day, respectively, were identified based on increased urinary enzyme markers for renal damage and central nervous system (CNS) depression (USEPA, 2006a).

The only available reproductive or developmental study with 1,1-dichloroethane is an inhalation study where pregnant rats were exposed on days 6 through 15 of gestation (Schwetz et al., 1974). No effects on the fetuses were noted at 3,800 ppm. Delayed ossification of the sternum without accompanying malformations was reported at a concentration of 6,000 ppm.

A cancer assessment for 1,1-dichloroethane is available on IRIS (USEPA, 1990a). That assessment classifies the chemical, according to EPA's 1986 Guidelines for Carcinogenic Risk Assessment (USEPA, 1986), as Group C, a possible human carcinogen. This classification is based on no human data and limited evidence of carcinogenicity in two animal species (rats and mice), as shown by increased incidences of hemangiosarcomas and mammary gland adenocarcinomas in female rats and hepatocellular carcinomas and benign uterine polyps in mice (NCI, 1978). The data were considered inadequate to support quantitative assessment. The close structural relationship between 1,1-dichloroethane and 1,2-dichloroethane, which is classified as a B2 probable human carcinogen and produces tumors at many of the same sites where marginal tumor increases were observed for 1,1-dichloroethane, supports the suggestion that the 1,1-isomer could possibly be carcinogenic to humans. Mixed results in initiation/promotion studies and genotoxicity assays are consistent with this classification. On the other hand, the animals from the 1,1-dichloroethane National Cancer Institute (NCI, 1978) study were housed with animals being exposed to 1,2-dichloroethane providing opportunities for possible co-exposure impacting the 1,1-dichloroethane results. The following groups of individuals may have an increased risk from exposure to 1,1-dichloroethane (NIOSH, 1978; ATSDR, 2015):

- Those with chronic respiratory disease,
- Those with liver diseases that impact hepatic microsomal cytochrome P-450 functions,

- Individuals with impaired renal function and vulnerable to kidney stones
- Individuals with skin disorders vulnerable to irritation by solvents like 1,1-dichloroethane,
- Those who consume alcohol or use pharmaceuticals (e.g., phenobarbital) that alter the activity of cytochrome P-450s.

A provisional chronic RfD was derived from the 13-week gavage study in rats based on a NOAEL of 1,000 mg/kg/day administered for five days/week and adjusted to 714.3 mg/kg/day for continuous exposure (an increase in urinary enzymes was the adverse impact on the kidney). The chronic oral RfD of 0.2 mg/kg/day was derived by dividing the normalized NOAEL of 714.3 mg/kg/day in male Sprague-Dawley rats by a combined UF of 3,000. The combined UF includes factors of 10 for interspecies extrapolation, 10 for extrapolation from a subchronic study, 10 for human variability, and 3 for database deficiencies (including lack of reproductive and developmental toxicity tests by the oral route). This assessment noted several limitations in the critical study and database as a whole. Specifically, that the reporting of the results in the critical study were marginally adequate and that the database lacks information on reproductive and developmental and nervous system toxicity.

EPA calculated an HRL for 1,1-dichloroethane of 1,000 µg/L, based on EPA oral RfD of 0.2 mg/kg/day, using 2.5 L/day drinking water ingestion, 80 kg body weight and a 20% relative source contribution (RSC) factor.

b) Occurrence

EPA has determined that 1,1-dichloroethane does not occur with a frequency and at levels of public health concern at PWSs based on the Agency's evaluation of available occurrence information. The primary occurrence data for 1,1-dichloroethane are the 2013-2015 nationally representative drinking water monitoring data generated through EPA's UCMR 3. 1,1-Dichloroethane was not detected in any of the 36,848 UCMR 3 samples collected by 4,916

PWSs (serving ~ 241 million people) at levels greater than ½ the HRL (500 µg/L) or the HRL (1,000 µg/L). 1,1-Dichloroethane was detected in about 2.3% samples at or above the MRL (0.03 µg/L) (USEPA, 2019a; USEPA, 2021a).

Other supplementary sources of finished water occurrence data from UCM Rounds 1 and 2 indicate that the occurrence of 1,1-dichloroethane in PWSs is likely to be low to non-existent (USEPA, 2021a). 1,1-Dichloroethane occurrence data for ambient water from NAWQA and NWIS are consistent with those for finished water (USEPA, 2021a).

c) Meaningful Opportunity

The Agency has determined that regulation of 1,1-dichloroethane does not present a meaningful opportunity for health risk reduction for persons served by PWSs based on the estimated exposed populations, including sensitive populations. UCMR 3 findings indicate that the estimated population exposed to 1,1-dichloroethane at levels of public health concern is 0%, based on lack of detections at levels greater than ½ the HRL (500 µg/L) or the HRL (1,000 µg/L). As a result, the Agency finds that an NPDWR for 1,1-dichloroethane does not present a meaningful opportunity for health risk reduction.

d) Summary of Public Comments on 1,1-Dichloroethane and Agency Responses

EPA received several comments on the Agency's evaluation of 1,1-dichloroethane under section 1412(b)(1)(A) of SDWA, all of which were in support of its preliminary determination not to regulate 1,1-dichloroethane. EPA agrees with the comments that are in support of the negative regulatory determination.

C. Acetochlor

1. Description

Acetochlor is a chloroacetanilide pesticide that is used as an herbicide for pre-emergence control of weeds. It is registered for use on corn crops (field corn and popcorn) and has been approved for use on cotton as a rotational crop. Synonyms for acetochlor include 2-chloro-2'-

methyl-6-ethyl-N-ethoxymethylacetanilide (USEPA, 2021a). Acetochlor is expected to have low to moderate persistence in water due to its biodegradation half-life, as well as susceptibility to photolysis (USEPA, 2021a).

2. Agency Findings

The Agency is making a determination not to regulate acetochlor with an NPDWR. Acetochlor does not occur with a frequency and at levels of public health concern. As a result, the Agency finds that an NPDWR does not present a meaningful opportunity for health risk reduction.

a) Adverse Health Effects

The Agency finds that acetochlor may have adverse effects on the health of persons. Subchronic and chronic oral studies have demonstrated adverse effects on the liver, thyroid (secondary to the liver effects), nervous system, kidney, lung, testes, and erythrocytes in rats and mice (USEPA, 2006b; USEPA, 2018c). There was evidence of carcinogenicity in studies conducted with acetochlor in rats and mice and a non-mutagenic mode of action was demonstrated for nasal and thyroid tumors in rats (USEPA, 2006b). Cancer effects include nasal tumors and thyroid tumors in rats, lung tumors and histiocytic sarcomas in mice, and liver tumors in both rats and mice (Ahmed and Seely, 1983; Ahmed et al., 1983; Amyes, 1989; Hardisty, 1997a; Hardisty, 1997b; Hardisty, 1997c; Naylor and Ribelin, 1986; Ribelin, 1987; USEPA, 2004b; USEPA, 2006b; and Virgo and Broadmeadow, 1988). No biologically sensitive human subpopulations have been identified for acetochlor. Developmental and reproductive toxicity studies do not indicate increased susceptibility to acetochlor exposure at early life stages in test animals (USEPA, 2006b).

The study used to derive the oral RfD is a 1-year oral chronic feeding study conducted in beagle dogs. This study describes a NOAEL of 2 mg/kg/day, and a LOAEL of 10 mg/kg/day, based on the critical effects of increased salivation; increased levels of alanine aminotransferase

(ALT) and ornithine carbamoyl transferase (OTC); increased triglyceride levels; decreased blood glucose levels; and alterations in the histopathology of the testes, kidneys, and liver of male beagle dogs (USEPA, 2018c; ICI, Inc., 1988). The UF applied was 100 (10 for intraspecies variation and 10 for interspecies extrapolation). The EPA OPP RfD for acetochlor of 0.02 mg/kg/day, based on the NOAEL of 2 mg/kg/day from the 1-year oral chronic feeding study in beagle dogs, is expected to be protective of both noncancer and cancer effects.

EPA calculated an HRL of 100 µg/L based on the EPA OPP RfD for non-cancer effects for acetochlor of 0.02 mg/kg/day (USEPA, 2018c) using 2.5 L/day drinking water ingestion, 80 kg body weight, and a 20% RSC factor.

b) Occurrence

EPA has determined that acetochlor does not occur with a frequency and at levels of public health concern at PWSs based on the Agency's evaluation of available occurrence information. The primary occurrence data for acetochlor are from the first Unregulated Contaminant Monitoring Regulation (UCMR 1) assessment monitoring (AM) (2001-2003) and the second Unregulated Contaminant Monitoring Regulation (UCMR 2) screening survey (SS) (2008-2010). Acetochlor was not detected at levels greater than ½ the HRL (50 µg/L), the HRL (100 µg/L), or the MRL (2 µg/L) in any of the 33,778 UCMR 1 assessment monitoring samples from 3,869 PWSs (USEPA, 2008; USEPA, 2021a) or in any of the 11,193 UCMR 2 screening survey samples from 1,198 PWSs (USEPA, 2015; USEPA, 2021a).

Findings from the available ambient water data for acetochlor are consistent with the results in finished water. Ambient water data in NAWQA show that acetochlor was detected in between 13% and 23% of samples from between 3% and 10% of sites. While maximum values in NAWQA Cycle 2 (2002-2012) and Cycle 3 (2013-2017) monitoring exceeded the HRL (215 µg/L in 2004 and 137 µg/L in 2013) (only one sample in each of those two cycles exceeded the HRL), 90th percentile levels of acetochlor remained below 1 µg/L. More than 10,000 samples

were collected in each cycle. Non-NAWQA NWIS data (1991-2016), which included limited finished water data in addition to the ambient water data, show no detected concentrations greater than the HRL (USEPA, 2021a).

c) Meaningful Opportunity

The Agency has determined that regulation of acetochlor does not present a meaningful opportunity for health risk reduction for persons served by PWSs based on the estimated exposed populations, including sensitive populations. The estimated population exposed to acetochlor at levels of public health concern is 0% based on UCMR 1 finished water data gathered from 2001 to 2003 and UCMR 2 finished water data gathered from 2008 to 2010. As a result, the Agency finds that an NPDWR for acetochlor does not present a meaningful opportunity for health risk reduction.

d) Summary of Public Comments on Acetochlor and Agency Responses

EPA received several comments on the Agency's evaluation of acetochlor under section 1412(b)(1)(A) of SDWA, all of which were in support of its preliminary determination not to regulate acetochlor. EPA agrees with the comments that are in support of the negative regulatory determination.

D. Methyl Bromide

1. Description

Methyl bromide is a halogenated alkane and occurs as a gas. Methyl bromide has been used as a fumigant fungicide applied to soil before planting, to crops after harvest, to vehicles and buildings, and for other specialized purposes. Use of the chemical in the United States was phased out in 2005, except for specific critical use exemptions and quarantine and pre-shipment exemptions in accordance with the Montreal Protocol. Critical use exemptions have included strawberry cultivation and production of dry cured pork. Synonyms for methyl bromide include bromomethane, monobromomethane, curafume, Meth-O-Gas, and Brom-O-Sol. Methyl bromide

is expected to have moderate persistence in water due to its susceptibility to hydrolysis (USEPA, 2021a).

2. Agency Findings

The Agency is making a determination not to regulate methyl bromide with an NPDWR. Methyl bromide does not occur with a frequency and at levels of public health concern. As a result, the Agency finds that an NPDWR does not present a meaningful opportunity for health risk reduction.

a) Adverse Health Effects

The Agency finds that methyl bromide may have adverse effects on the health of persons. The limited number of studies investigating the oral toxicity of methyl bromide indicate that the route of administration influences the toxic effects observed (USEPA, 2006c). The forestomach of rats (forestomachs are not present in humans) appears to be the most sensitive target of methyl bromide when it is administered orally by gavage (ATSDR, 1992). Acute and subchronic oral gavage studies in rats identified stomach lesions (Kaneda et al., 1998), hyperemia (excess blood) (Danse et al., 1984), and ulceration (Boorman et al., 1986; Danse et al., 1984) of the forestomach. However, forestomach effects were not observed in rats and stomach effects were not observed in dogs that were chronically exposed to methyl bromide in the diet, potentially because methyl bromide degrades to other bromide compounds in the food (Mertens, 1997). Decreases in food consumption, body weight, and body weight gain were noted in the chronic rat study when methyl bromide was administered in capsules (Mertens, 1997).

In a subchronic (13-week) rat study (Danse et al., 1984), a NOAEL of 1.4 mg/kg/day (a time weighted average, 5/7 days, of the 2 mg/kg/day dose group) was selected in the EPA IRIS assessment based on severe hyperplasia of the stratified squamous epithelium in the forestomach, in the next highest dose group of 7.1 mg/kg/day (USEPA, 1989). In ATSDR's Toxicological Profile (ATSDR, 1992), a lower dose of 0.4 mg/kg/day is selected as the NOAEL because "mild

focal hyperemia" was observed at the 1.4 mg/kg/day dose level. It is worth noting that authors of this study reported neoplastic changes in the forestomach. However, EPA and others (USEPA, 1985; Schatzow, 1984) re-evaluated the histological results, concluding that the lesions were hyperplasia and inflammation, not neoplasms. ATSDR notes that histological diagnosis of epithelial carcinomas in the presence of marked hyperplasia is difficult (Wester and Kroes 1988; ATSDR 1992). Additionally, the hyperplasia of the forestomach observed after 13 weeks of exposure to bromomethane regressed when exposure ended (Boorman et al. 1986; ATSDR 1992).

EPA selected an OPP Human Health Risk Assessment from 2006 as the basis for developing the HRL for methyl bromide (USEPA, 2006c). As described in the OPP document, the study was of chronic duration (two years) with four groups of male rats and four groups of female rats treated orally via encapsulated methyl bromide. In the OPP assessment (USEPA, 2006c), Mertens (1997) was identified as the critical study and decreased body weight, decreased rate of body weight gain, and decreased food consumption were the critical effects in rats orally exposed to methyl bromide (USEPA, 2006c). The NOAEL was 2.2 mg/kg/day and the LOAEL was 11.1 mg/kg/day. The RfD derived in the 2006 OPP Human Health Assessment is 0.022 mg/kg/day, based on the point of departure (POD) of 2.2 mg/kg/day (the NOAEL) and a combined uncertainty factor (UF) of 100 for interspecies variability (10) and intraspecies variability (10). No benchmark dose modeling was performed.

Neurological effects reported after inhalation exposures have not been reported after oral exposures, indicating that route of exposure may influence the most sensitive adverse health endpoint (USEPA, 1988).

Limited data are available regarding the developmental or reproductive toxicity of methyl bromide, especially via the oral route of exposure. ATSDR (1992) found no information on developmental effects in humans with methyl bromide exposure. An oral developmental toxicity

study of methyl bromide in rats (doses of 3, 10, or 30 mg/kg/day) and rabbits (doses of 1, 3, or 10 mg/kg/day) found that there were no treatment-related adverse effects in fetuses of the treated groups of either species (Kaneda et al., 1998). ATSDR's 1992 Toxicological Profile also did not identify any LOAELs for rats or rabbits in this study. In rats exposed to 30 mg/kg/day, there was an increase in fetuses having 25 presacral vertebrae; however, ATSDR notes that there were no significant differences in the number of litters with this variation and the effect was not exposure-related (ATSDR, 1992). No significant alterations in resorptions or fetal deaths, number of live fetuses, sex ratio, or fetal body weights were observed in rats and no alterations in the occurrence of external, visceral, or skeletal malformations or variations were observed in the rabbits. Some inhalation studies reported no effects on development or reproduction, but other inhalation studies show adverse developmental effects. For example, Hardin et al. (1981) and Sikov et al. (1980) conducted studies in rats and rabbits and found no developmental effects, even when maternal toxicity was severe (ATSDR, 1992). However, another inhalation study of rabbits found increased incidence of gallbladder agenesis, fused vertebrae, and decreased fetal body weights in offspring (Breslin et al., 1990). Decreased pup weights were noted in a multigeneration study in rats exposed to 30 ppm (Enloe et al., 1986). Reproductive effects were noted in intermediate-duration inhalation studies in rats and mice (Eustis et al., 1988; Kato et al., 1986), which indicated that the testes may undergo degeneration and atrophy at high exposure levels.

In the OPP HHRA for methyl bromide (USEPA, 2006c), methyl bromide is classified as “not likely to be carcinogenic to humans”. In 2007, EPA published a PPRTV report which stated that there is “inadequate information to assess the carcinogenic potential” of methyl bromide in humans (USEPA, 2007a). The PPRTV assessment agrees with earlier National Toxicology Program (NTP) conclusions that the available data indicate that methyl bromide can cause genotoxic and/or mutagenic changes. The PPRTV assessment states that the results in studies by Vogel and Nivard (1994) and Gansewendt et al. (1991) clearly indicate methyl bromide is

distributed throughout the body and is capable of methylating DNA in vivo. However, the PPRTV assessment also summarizes the results of several studies in mice and rats that have not demonstrated evidence of methyl bromide-induced carcinogenic changes (USEPA, 2007a; NTP, 1992; Reuzel et al. 1987; ATSDR, 1992). In 2012, an epidemiology study was published that concluded there was a significant monotonic exposure-dependent increase in stomach cancer risk among 7,814 applicators of methyl bromide (Barry et al., 2012). In OPP's Draft HHRA for Methyl Bromide, OPP reviews all the epidemiological studies for methyl bromide, including the Barry et al. (2012) Agricultural Health Study. OPP concludes that "based on the review of these studies, there is insufficient evidence to suggest a clear associative or causal relationship between exposure to methyl bromide and carcinogenic or non-carcinogenic health outcomes."

According to ATSDR (1992) and the EPA OPP assessment (USEPA, 2006c), no studies suggest that a specific subpopulation may be more susceptible to methyl bromide, though there is little information about susceptible lifestages or subpopulations when exposed via the oral route. Because the critical effects of decreased body weight, decreased rate of body weight gain, and decreased food consumption in this study are not specific to a sensitive subpopulation or life stage, the target population of the general adult population was selected in deriving the HRL for regulatory determination. EPA's OPP assessment conducted additional exposure assessments for lifestages that may increase exposure to methyl bromide and concluded that no lifestages have expected exposure greater than 10% of the chronic population-adjusted dose (cPAD), including children.

EPA calculated an HRL of 100 µg/L (rounded from 140.8 µg/L) based on an EPA OPP assessment cPAD of 0.022 mg/kg/day and using 2.5 L/day drinking water ingestion, 80 kg body weight, and a 20% RSC factor (USEPA, 2006d; USEPA, 2011, Table 8-1 and 3-33).

b) Occurrence

EPA has determined that methyl bromide does not occur with a frequency and at levels of public health concern at PWSs based on the Agency's evaluation of available occurrence information. The primary data occurrence data for methyl bromide are the 2013-2015 nationally representative drinking water monitoring data generated through EPA's UCMR 3. Methyl bromide was not detected in any of the 36,848 UCMR 3 samples collected by 4,916 PWSs (serving ~ 241 million people) at levels greater than ½ the HRL (50 µg/L) or the HRL (100 µg/L). Methyl bromide was detected in about 0.3% samples at or above the MRL (0.2 µg/L) (USEPA, 2019a; USEPA, 2021a).

Findings from the available ambient water data for methyl bromide are consistent with the results in finished water. Ambient water data in NAWQA show that methyl bromide was detected in fewer than 1% of samples from fewer than 2% of sites. No detections were greater than the HRL in any of the three cycles. The median concentration among detections were 0.5 µg/L and 0.8 µg/L in Cycle 1 and Cycle 3, respectively. There were no detections in Cycle 2. The results of the NWIS analysis show that methyl bromide was detected in approximately 0.1% of samples at approximately 0.1% of sites. The median concentration among detections was 0.6 µg/L.

c) Meaningful Opportunity

The Agency has determined that regulation of methyl bromide does not present a meaningful opportunity for health risk reduction for persons served by PWSs based on the estimated exposed populations, including sensitive populations. UCMR 3 findings indicate that the estimated population exposed to methyl bromide at levels of public health concern is 0%. As a result, the Agency finds that an NPDWR for methyl bromide does not present a meaningful opportunity for health risk reduction.

d) Summary of Public Comments on Methyl Bromide and Agency Responses

EPA received several comments on the Agency's evaluation of methyl bromide under section 1412(b)(1)(A) of SDWA, including several comments in support of its preliminary determination not to regulate methyl bromide. Three anonymous members of the public opposed the negative determination of methyl bromide because of their perceptions about its production and use. Specifically, commenters appear to be seeking to prohibit the production and use of methyl bromide.

EPA agrees with the comments that are in support of the negative regulatory determination. Regarding comments that oppose the negative determination because of methyl bromide's production and use; the production, importation, use, and disposal of specific chemicals are not regulated by SDWA and therefore are not relevant to this determination. As discussed above, methyl bromide was not found above ½ the HRL in drinking water in any UCMR 3 samples. Furthermore, commenters did not provide any data or other information that suggested that their concerns had impacts on the occurrence of methyl bromide in drinking water or discuss any other methyl bromide issues that specifically related to drinking-water. Hence, commenters concerns are not addressable by this decision not to regulate methyl bromide under SDWA.

E. Metolachlor

1. Description

Metolachlor is a chloroacetanilide pesticide that is used as an herbicide for weed control. Initially registered in 1976 for use on turf, metolachlor has more recently been used on corn, cotton, peanuts, pod crops, potatoes, safflower, sorghum, soybeans, stone fruits, tree nuts, non-bearing citrus, non-bearing grapes, cabbage, certain peppers, buffalograss, guymon bermudagrass for seed production, nurseries, hedgerows/fencerows, and landscape plantings.

Synonyms for metolachlor include dual and bicep (USEPA, 2021a). Metolachlor is expected to have moderate to high persistence in water due to its biodegradation half-life (USEPA, 2021a).

2. Agency Findings

The Agency is making a determination not to regulate metolachlor with an NPDWR. Metolachlor does not occur with a frequency and at levels of public health concern. As a result, the Agency finds that an NPDWR does not present a meaningful opportunity for health risk reduction.

a) Adverse Health Effects

The Agency finds that metolachlor may have adverse effects on the health of persons. The existing toxicological database includes studies evaluating both metolachlor and S-metolachlor. When combined with the toxicology database for metolachlor, the toxicology database for S-metolachlor is considered complete for risk assessment purposes (USEPA, 2018d). In subchronic (metolachlor and S-metolachlor) (USEPA, 1995b; USEPA, 2018d) and chronic (metolachlor) (Hazelette, 1989; Tisdel, 1983; Page, 1981; USEPA, 2018d) toxicity studies in dogs and rats, decreased body weight was the most commonly observed effect. Chronic exposure to metolachlor in rats also resulted in increased liver weight and microscopic liver lesions in both sexes (USEPA, 2018d). No systemic toxicity was observed in rabbits when metolachlor was administered dermally, though dermal irritation was observed at lower doses (USEPA, 2018d). Portal of entry effects (e.g., hyperplasia of the squamous epithelium and mucous cell) occurred in the nasal cavity at lower doses in a 28-day inhalation study in rats (USEPA, 2018d). Systemic toxicity effects were not observed in this study. Immunotoxicity effects were not observed in mice exposed to S-metolachlor (USEPA, 2018d).

While some prenatal developmental studies in the rat and rabbit with both metolachlor and S-metolachlor revealed no evidence of a qualitative or quantitative susceptibility in fetal animals, decreased pup body weight was observed in a two-generation study (Page, 1981,

USEPA, 2018d). Though there was no evidence of maternal toxicity, decreased pup body weight in the F1 and F2 litters was observed, indicating developmental toxicity (Page, 1981; USEPA, 1990b). Therefore, sensitive lifestages to consider include infants, as well as pregnant women and their fetus, and lactating women.

Although treatment with metolachlor did not result in an increase in treatment-related tumors in male rats or in mice (both sexes), metolachlor caused an increase in liver tumors in female rats (USEPA, 2018d). There was no evidence of mutagenic or cytogenetic effects in vivo or in vitro (USEPA, 2018d). In 1994 (USEPA, 1995b), EPA classified metolachlor as a Group C possible human carcinogen, in accordance with the 1986 Guidelines for Carcinogen Risk Assessment (USEPA, 1986). In 2017 (USEPA, 2018d), EPA re-assessed the cancer classification for metolachlor in accordance with EPA's final Guidelines for Carcinogen Risk Assessment (USEPA, 2005), and reclassified metolachlor/S-metolachlor as ''Not Likely to be Carcinogenic to Humans'' at doses that do not induce cellular proliferation in the liver. This classification was based on convincing evidence of a constitutive androstane receptor (CAR)-mediated mitogenic MOA for liver tumors in female rats that supports a nonlinear approach when deriving a guideline that is protective for the tumor endpoint (USEPA, 2018d).

A recent OPP HHRA identified a two-generation reproduction study in rats as the critical study (USEPA, 2018d). OPP proposed an RfD for metolachlor of 0.26 mg/kg/day, derived from a NOAEL of 26 mg/kg/day for decreased pup body weight in the F1 and F2 litters. A combined UF of 100 was used based on interspecies extrapolation (10), intraspecies variation (10), and an FQPA Safety Factor of 1. This RfD is considered protective of carcinogenic effects as well as effects observed in chronic toxicity studies (USEPA, 2018d). The decreased F1 and F2 litter pup body weights in the absence of maternal toxicity were considered indicative of increased susceptibility to the pups. Therefore, a rate of 0.15 L/kg/day was selected from the Exposure Factors Handbook (USEPA, 2011) to represent the consumers-only estimate of DWI based on the combined direct and indirect community water ingestion at the 90th percentile for bottle fed

infants. This estimate is more protective than the estimate for pregnant women (0.033 L/kg/day) or lactating women (0.054 L/kg/day). DWI and BW parameters are further outlined in the Exposure Factors Handbook (USEPA, 2011).

EPA OW calculated an HRL for metolachlor of 300 µg/L (rounded from 0.347 mg/L). The HRL was derived from the oral RfD of 0.26 mg/kg/day for bottle fed infants ingesting 0.15 L/kg/day water, with the application of a 20% RSC.

b) Occurrence

EPA has determined that metolachlor does not occur with a frequency and at levels of public health concern at PWSs based on the Agency's evaluation of available occurrence information. The primary occurrence data for metolachlor are from the UCMR 2 screening survey. A total of 11,192 metolachlor samples were collected from 1,198 systems. Of these systems, three (0.25%) had metolachlor detections (1 µg/L) and none of the detections were greater than ½ the HRL (150 µg/L) or the HRL (300 µg/L) (USEPA, 2015; USEPA, 2021a).

Supplementary sources of finished water occurrence data from UCM Round 2 indicate that the occurrence of metolachlor in PWSs is likely to be low to non-existent (USEPA, 2021a). Metolachlor occurrence data for ambient water from NAWQA and NWIS are consistent with those for finished water (USEPA, 2021a).

c) Meaningful Opportunity

The Agency has determined that regulation of metolachlor does not present a meaningful opportunity for health risk reduction for persons served by PWSs based on the estimated exposed populations, including sensitive populations. UCMR 2 findings indicate that the estimated population exposed to metolachlor at levels of public health concern is 0%. As a result, the Agency finds that an NPDWR for metolachlor does not present a meaningful opportunity for health risk reduction.

d) Summary of Public Comments on Metolachlor and Agency Responses

EPA received several comments on the Agency's evaluation of metolachlor under section 1412(b)(1)(A) of SDWA, all of which were in support of its preliminary determination not to regulate metolachlor. EPA agrees with the comments that are in support of the negative regulatory determination.

F. Nitrobenzene

1. Description

Nitrobenzene is a synthetic aromatic nitro compound and occurs as an oily, flammable liquid. It is commonly used as a chemical intermediate in the production of aniline and drugs such as acetaminophen. Nitrobenzene is also used in the manufacturing of paints, shoe polishes, floor polishes, metal polishes, aniline dyes, and pesticides. Nitrobenzene is expected to have a moderate to high likelihood of partitioning to water and moderate persistence in water (USEPA, 2021a).

2. Agency Findings

The Agency is making a determination not to regulate nitrobenzene with an NPDWR. Nitrobenzene does not occur with a frequency and at levels of public health concern. As a result, the Agency finds that an NPDWR does not present a meaningful opportunity for health risk reduction.

a) Adverse Health Effects

The Agency finds that nitrobenzene may have adverse effects on the health of persons. NTP (1983) conducted a 90-day oral gavage study of nitrobenzene in F344 rats and B6C3F1 mice. The rats were more sensitive to the effects of nitrobenzene exposure than the mice, and changes in absolute and relative organ weights, hematologic parameters, splenic congestion, and histopathologic lesions in the spleen, testis, and brain were reported. Based on statistically significant changes in absolute and relative organ weights, splenic congestion, and increases in

reticulocyte count and methemoglobin (metHb) concentration, a LOAEL of 9.38 mg/kg/day was identified for the subchronic oral effects of nitrobenzene in F344 male rats (USEPA, 2009). This was the lowest dose studied, so a NOAEL was not identified. The mice were treated with higher doses and were generally more resistant to nitrobenzene toxicity, the toxic endpoints were similar in both species.

The testis, epididymis, and seminiferous tubules of the male reproductive system are targets of nitrobenzene toxicity in rodents. In male rats (F344/N and CD) and mice (B6C3F1), nitrobenzene exposure via the oral and inhalation routes results in histopathologic lesions of the testis and seminiferous tubules, testicular atrophy, a large decrease in sperm count, and a reduction of sperm motility and/or viability, which contribute to a loss of fertility (NTP, 1983; Bond et al., 1981; Koida et al., 1995; Matsuura et al., 1995; Kawashima et al., 1995). These data suggest that nitrobenzene is a male-specific reproductive toxicant (USEPA, 2009).

Under the Guidelines for Carcinogen Risk Assessment (USEPA, 2005), nitrobenzene is classified as "likely to be carcinogenic to humans" by any route of exposure (USEPA, 2009). A two-year inhalation cancer bioassay in rats and mice (Cattley et al., 1994; CIIT, 1993) reported an increase in several tumor types in both species. However, the lack of available data, including a physiologically based biokinetic or model that might predict the impact of the intestinal metabolism on serum levels of nitrobenzene and its metabolites following oral exposures, precluded EPA's IRIS program from deriving an oral CSF (USEPA, 2009). Additionally, a metabolite of nitrobenzene, aniline, is classified as a probable human carcinogen (B2) (USEPA, 1988).

Nitrobenzene has been shown to be non-genotoxic in most studies and was classified as, at most, weakly genotoxic in the 2009 USEPA IRIS assessment (ATSDR, 1990; USEPA, 2009).

Of the available animal studies with oral exposure to nitrobenzene, the 90-day gavage study conducted by NTP (1983) is the most relevant study for deriving an RfD for nitrobenzene.

This study used the longest exposure duration and multiple dose levels. Benchmark dose software (BMDS) (version 1.4.1c; USEPA, 2007b) was applied to estimate candidate PODs for deriving an RfD for nitrobenzene. Data for splenic congestion and increases in reticulocyte count and metHb concentration were modeled. The POD derived from the male rat increased metHb data with a benchmark response (BMR) of 1 standard deviation (SD) was selected as the basis of the RfD (see USEPA, 2009 for additional detail). Therefore, the benchmark dose level (BMDL) used as the POD is a BMDL1SD of 1.8 mg/kg/day.

In deriving the RfD, EPA's IRIS program applied a composite UF of 1,000 to account for interspecies extrapolation (10), intraspecies variation (10), subchronic-to-chronic study extrapolation (3), and database deficiency (3) (USEPA, 2009). Thus, the RfD calculated in the 2009 IRIS assessment is 0.002 mg/kg/day. The overall confidence in the RfD was medium because the critical effect is supported by the overall database and is thought to be protective of reproductive and immunological effects observed at higher doses; however, there are no chronic or multigenerational reproductive/developmental oral studies available for nitrobenzene. Because the critical effect in this study (increased metHb in the adult rat) is not specific to a sensitive subpopulation or lifestage, the general adult population was selected in deriving the HRL for regulatory determination.

EPA calculated an HRL for the noncancer effects of nitrobenzene of 10 µg/L (rounded from 12.8 µg/L), based on the RfD of 0.002 mg/kg/day, using 2.5 L/day drinking water ingestion, 80 kg body weight, and a 20% RSC factor.

b) Occurrence

EPA has determined that nitrobenzene does not occur with a frequency and at levels of public health concern at PWSs based on the Agency's evaluation of available occurrence information. The primary occurrence data for nitrobenzene are nationally representative finished water monitoring data generated through EPA's UCMR 1 AM (2001-2003). UCMR 1 collected

33,576 finished water samples from 3,861 PWSs (serving ~226 million people) for nitrobenzene and it was detected in only a small number of those samples (0.01%) above the HRL (10 µg/L), which is the same as the MRL (10 µg/L).

Findings from the available ambient water data for nitrobenzene are consistent with the results in finished water. Ambient water data in NAWQA show that nitrobenzene was not detected in any of the samples collected under any of the three monitoring cycles, while NWIS data show that nitrobenzene was detected in approximately 1% of samples.

c) Meaningful Opportunity

The Agency has determined that regulation of nitrobenzene does not present a meaningful opportunity for health risk reduction for persons served by PWSs based on the estimated exposed populations, including sensitive populations. UCMR 1 data indicate that the estimated population exposed to nitrobenzene above the HRL is 0.1%. The Agency finds that an NPDWR for nitrobenzene does not present a meaningful opportunity for health risk reduction.

d) Summary of Public Comments on Nitrobenzene and Agency Responses

EPA received several comments on the Agency's evaluation of nitrobenzene under section 1412(b)(1)(A) of SDWA, all of which were in support of its preliminary determination not to regulate nitrobenzene. EPA agrees with the comments that are in support of the negative regulatory determination.

G. RDX

1. Description

RDX is a nitrated triazine and is an explosive. The name RDX is an abbreviation of "Royal Demolition eXplosive." The formal chemical name is hexahydro-1,3,5-trinitro-1,3,5-triazine. RDX is expected to have a moderate to high likelihood of partitioning to water and low to moderate persistence in water (USEPA, 2021a).

2. Agency Findings

The Agency is making a determination not to regulate RDX with an NPDWR. RDX does not occur with a frequency and at levels of public health concern. As a result, the Agency finds that an NPDWR does not present a meaningful opportunity for health risk reduction.

a) Adverse Health Effects

The Agency finds that RDX may have adverse effects on the health of persons. Available health effects assessments include an IRIS toxicological review (USEPA, 2018e), and older assessments including an ATSDR toxicological profile (ATSDR, 2012) and an OW assessment published in the 1992 Drinking Water Health Advisory: Munitions (USEPA, 1992). The EPA IRIS assessment (2018e) presents an RfD of 0.004 mg/kg/day based on convulsions as the critical effect observed in a subchronic study in F-344 rats by Crouse et al. (2006). The POD for the derivation was a $BMDL_{0.05}$ of 1.3 mg/kg/day derived using a pharmacokinetic model that identified the human equivalent dose (HED) based on arterial blood concentrations in the rats as the dose metric. A 300-fold UF (3 for extrapolation from animals to humans, 10 for interindividual differences in human susceptibility, and 10 for uncertainty in the database) was applied in determination of the RfD.

Additionally, the EPA IRIS assessment (USEPA, 2018e) classified data from the Lish et al. (1984) chronic study in B6C3F1 as providing suggestive evidence of carcinogenic potential following EPA (USEPA, 2005) guidelines. The slope factor was derived from the lung and liver tumors' dose-response in the Lish et al. (1984) study. The POD for the slope factor was the $BMDL_{10}$ allometrically scaled to a HED yielding a slope factor of 0.08 per mg/kg/day.

In mice fed doses of 0 to 35 mg/kg/day for 24 months in the Lish et al. (1984) study, there were dose-dependent increases in adenomas or carcinomas of the lungs and liver in males and females (USEPA, 2018e). The formulation used contained 3 to 10% HMX, another munition ingredient. EPA assessed the toxicity of HMX (USEPA, 1988). No chronic-duration studies were

available to evaluate the carcinogenicity of HMX (USEPA, 1988). HMX is classified as Group D, or not classifiable as to human carcinogenicity (USEPA, 1992; USEPA, 1988). In the Levine et al. (1983) RDX dietary exposure study with Fischer 344 rats, a statistically significant increase in the incidence of hepatocellular carcinomas was observed in males but not in females (USEPA, 2018e). Although evidence of carcinogenicity included dose-dependent increases in two experimental animal species, two sexes, and two systems (liver and lungs), evidence supporting carcinogenicity in addition to the B6C3F1 mouse study was not robust; this factor contributed to the suggestive evidence of carcinogenic potential classification. EPA considered both the Lish et al. (1984) and Levine et al. (1983) studies to be suitable for dose-response analysis because they were well conducted, using similar study designs with large numbers of animals at multiple dose levels (USEPA, 2018e). EPA (2018e) concluded that insufficient information was available to evaluate male reproductive toxicity from experimental animals exposed to RDX. In addition, EPA (2018e) concluded that inadequate information was available to assess developmental effects from experimental animals exposed to RDX. EPA selected the 2018 EPA IRIS assessment to derive two HRLs for RDX: the RfD-derived HRL (based on Crouse et al., 2006) and the oral cancer slope factor-derived HRL (based on Lish et al., 1984). EPA has generally derived HRLs for "possible" or Group C carcinogens using the RfD approach in past Regulatory Determinations. However, for RDX, EPA decided to show both an RfD-derived and oral-cancer-slope-factor-derived HRL since the mode of action for liver tumors is unknown and the 1 x 10-6 cancer risk level provides a more health protective HRL to evaluate the occurrence information.

The RfD-derived HRL for RDX was calculated using the RfD of 0.004 mg/kg/day based on a subchronic study in F-344 rats by Crouse et al. (2006) with convulsions as the critical effect (USEPA, 2018e). The point of departure for the RfD calculation was a human equivalent $BMDL_{0.05}$ of 1.3 mg/kg/day. The HED was derived using a pharmacokinetic model based on arterial blood concentrations in the rats as the dose metric. A 300-fold uncertainty factor (3 for extrapolation from animals to humans, 10 for interindividual differences in human susceptibility,

and 10 for uncertainty in the database) was applied in determination of the RfD. EPA calculated a RfD-derived HRL of 30 µg/L (rounded from 25.6 µg/L), for the noncancer effects of RDX based on the RfD of 0.004 mg/kg/day, using 2.5 L/day drinking water ingestion, 80 kg body weight, and a 20% RSC factor.

The oral-cancer-slope-factor-derived HRL for RDX was also based on values presented in the 2018 EPA IRIS assessment. The slope factor is derived from the dose-response for lung and liver tumors in the Lish et al. (1984) study, with elimination of the data for the high dose group due to high mortality. The point of departure for the slope factor of 0.08 (mg/kg/day)-1 was the $BMDL_{10}$ which was allometrically scaled to a HED. EPA calculated an oral cancer slope factor-derived HRL of 0.4 µg/L for RDX based on the cancer slope factor of 0.08 (mg/kg/day)-1, using 2.5 L/day drinking water ingestion, 80 kg body weight, and a 1 in a million cancer risk level.

EPA's (USEPA, 2018e) derivation of an oral slope factor for cancer is in accordance with the Guidelines for Carcinogen Risk Assessment (USEPA, 2005) while RDX is classified as having "suggestive evidence of carcinogenic potential." Specifically, the guidelines state "when the evidence includes a well-conducted study, quantitative analyses may be useful for some purposes, for example, providing a sense of the magnitude and uncertainty of potential risks, ranking potential hazards, or setting research priorities" (USEPA, 2005). The EPA IRIS assessment concluded that the database for RDX contains well-conducted carcinogenicity studies (Lish et al., 1984; Levine et al., 1983) suitable for dose response and that the quantitative analysis may be useful for providing a sense of the magnitude and uncertainty of potential carcinogenic risk (USEPA, 2018e). Therefore, EPA felt it was important to evaluate the occurrence information against both the RfD-derived HRL and the oral cancer slope factor-derived HRL.

b) Occurrence

EPA has determined that RDX does not occur with a frequency and at levels of public health concern at PWSs based on the Agency's evaluation of available occurrence information. The primary data for RDX are nationally representative drinking water monitoring data generated through EPA's UCMR 2 AM (2008-2010). UCMR 2 collected 32,150 finished water samples from 4,139 PWSs (serving ~229 million people) for RDX and it was detected in only a small number of those samples (0.01%) at or above the MRL. The detections occurred in three large surface water systems; the maximum detected concentration of RDX was 1.1 µg/L. The MRL is 1 µg/L, which is about 2.5 times higher than the oral cancer slope factor-derived HRL (0.4 µg/L). The RfD-derived HRL (30 µg/L) is 30 times higher than the MRL and 75 times higher than the cancer slope factor-derived HRL.

Findings from the available ambient water data for RDX in ambient water, available from NWIS, show that RDX was detected in approximately 46% of samples and at approximately 29% of sites; RDX data are not available from the NAWQA program.

c) Meaningful Opportunity

The Agency has determined that regulation of RDX does not present a meaningful opportunity for health risk reduction for persons served by PWSs based on the estimated exposed populations, including sensitive populations. UCMR 2 findings indicate that the estimated population exposed to RDX at or above the MRL is 0.04%. There were no detections greater than the non-cancer HRL (30 µg/L) or the one-half the non-cancer HRL (15 µg/L). Because the MRL of 1 µg/L is higher than the cancer HRL of 0.4 µg/L, the population exposed relative to the cancer HRL and ½ the cancer HRL is not presented here. As a result, the Agency finds that an NPDWR for RDX does not present a meaningful opportunity for health risk reduction. Based on the small number of samples measured at or marginally above the MRL, EPA does not believe that there would be enough occurrence in the narrow range between the HRL and the MRL to change the meaningful opportunity determination.

d) Summary of Public Comments on RDX and Agency Responses

EPA received several comments on the Agency's evaluation of RDX under section 1412(b)(1)(A) of SDWA, all of which were in support of its preliminary determination not to regulate RDX. EPA agrees with the comments that are in support of the negative regulatory determination.

Summary of Public Comments on Strontium, 1,4-Dioxane, and 1,2,3-Trichloropropane, and the Agency's Responses

H. Strontium

Strontium is an alkaline earth metal. On October 20, 2014 the Agency published its preliminary regulatory determination to regulate strontium and requested public comment on the determination and supporting technical information (USEPA, 2014). Informed by the public comments received, rather than making a final determination for strontium in 2016, EPA delayed the final determination to consider additional data, and to decide whether there is a meaningful opportunity for health risk reduction by regulating strontium in drinking water (USEPA, 2016f). Specifically, the publication on the delayed final determination mentioned that EPA would evaluate additional studies on strontium exposure and health studies related to strontium exposure. Since 2016, EPA has worked to identify and evaluate published studies on health effects associated with strontium exposure, sources of exposure to strontium, and treatment technologies to remove strontium from drinking water. In its March 10, 2020 document (USEPA, 2020a), EPA clarified that it is continuing with its previous 2016 decision (USEPA, 2016f) to delay a final determination for strontium in order to further consider additional studies related to strontium exposure.

The Agency received several comments in support of a continued evaluation of strontium and not making a final determination for strontium in this action. One commenter requested that EPA complete its evaluation of strontium in a more timely manner. EPA agrees with the

comments that are in support of the continued evaluation prior to making a final regulatory determination for strontium. Regarding making a regulatory determination for strontium in this rulemaking, EPA notes that there continues to be a need for additional information and analyses before a regulatory determination can be made for strontium. While EPA determined in 2014 that strontium may have adverse effects on the health of persons including children, the Agency continues to consider additional data, consult existing assessments (such as Health Canada's Drinking Water Guideline from 2018), and evaluate whether there is a meaningful opportunity for health risk reduction by regulating strontium in drinking water. Additionally, EPA understands that strontium may co-occur with beneficial calcium in some drinking water systems and treatment technologies that remove strontium may also remove calcium. The Agency is evaluating the effectiveness of treatment technologies under different water conditions, including calcium concentrations. EPA intends to make a determination after these data needs have been resolved as part of its regulatory determination process.

I. 1,4-Dioxane

1,4-Dioxane is used as a solvent in cellulose formulations, resins, oils, waxes, and other organic substances; also used in wood pulping, textile processing, degreasing; in lacquers, paints, varnishes, and stains; and in paint and varnish removers.

While the health effects data suggest that 1,4-dioxane may have an adverse effect on human health and the occurrence data indicate that 1,4-dioxane is occurring in finished drinking water above the current HRL in some systems, EPA has not made a preliminary determination for 1,4-dioxane, as the Agency has not determined whether 1,4-dioxane occurs in public water systems with a frequency and at levels of public health concern and whether there is a meaningful opportunity for public health risk reduction by establishing an NPDWR for 1,4-dioxane (USEPA, 2020a). The *Final Regulatory Determination 4 Support Document* (USEPA, 2021a) and the *Occurrence Data from the Third Unregulated Contaminant Monitoring Rule*

(UCMR 3) (USEPA, 2019a) present additional information and analyses supporting the Agency's evaluation of 1,4-dioxane.

The Agency received several comments in support of a continued evaluation and not making a 1,4-dioxane determination at this time. One commenter provided information summarizing their belief that 1,4 dioxane has a non-linear mode of action. Another commenter requested that EPA complete its evaluation of 1,4-dioxane in a more-timely manner. EPA agrees with the comments that are in support of the continued evaluation. Regarding making a regulatory determination for 1,4-dioxane today, EPA notes that there is a need for additional information and analyses before a regulatory determination can be made for 1,4-dioxane. Based on UCMR 3 data, EPA derived a national estimate of less than two baseline cancer cases per year attributable to 1,4-dioxane in drinking water (USEPA, 2021a). However, while the number of baseline cancer cases is relatively low, other adverse health effects following exposure to 1,4-dioxane may also contribute to potential risk to public health, and these analyses under SDWA have not yet been completed. The Agency recently completed its new TSCA risk evaluation for 1,4-dioxane by the Office of Chemical Safety and Pollution Prevention (OCSPP) (USEPA, 2020c) and intends to consider it and the Canadian guideline technical document, once finalized, (Health Canada, 2018) and other relevant new science relevant to drinking water contamination prior to making a regulatory determination. This evaluation may provide clarity as to whether a new HRL is appropriate for evaluating the occurrence of 1,4-dioxane and whether there is a meaningful opportunity for an NPDWR to reduce public health risk.

J. 1,2,3-Trichloropropane

1,2,3-Trichloropropane is a man-made chemical used as an industrial solvent, cleaning and degreasing agent, and synthesis intermediate.

While the UCMR 3 data indicated 1,2,3-trichloropropane occurrence was relatively low at concentrations above the MRL, the MRL (0.03 µg/L) is more than 75 times the HRL (0.0004

µg/L) for 1,2,3-trichloropropane. This discrepancy allows for a broad range of potential contaminant concentrations that could be in exceedance of the HRL but below the MRL. EPA did not make a preliminary determination for 1,2,3-trichloropropane due to these analytical method-based limitations. The Agency noted that it needs additional lower-level occurrence information prior to making a preliminary regulatory determination for 1,2,3-trichloropropane. The *Final Regulatory Determination 4 Support Document* (USEPA, 2021a) and the *Occurrence Data from the Third Unregulated Contaminant Monitoring Rule (UCMR 3)* (USEPA, 2019a) present additional information and analyses supporting the Agency's evaluation of 1,2,3-trichloropropane.

The Agency received several comments in support of a continued evaluation and not making a 1,2,3-trichloropropane determination at this time. In addition, EPA notes that several comments requested that EPA find solutions to the analytical method limitations and collect additional monitoring data with an MRL adequate to support decision-making. EPA agrees with the comments that are in support of the continued evaluation. EPA also agrees that further evaluation of 1,2,3-tricholoropropane is warranted when new methods or other tools are available to do so.

V. Next Steps

As required by SDWA, EPA will initiate the process to propose a NPDWR for PFOA and PFOS within 24 months of the publication of this document in the *Federal Register*. For this rulemaking effort, in addition to using the best available science, the Agency will seek recommendations from the EPA Science Advisory Board and consider public comment on the proposed rule. Therefore, EPA anticipates further scientific review of new science and an opportunity for additional public input prior to the promulgation of the regulatory standard for PFOA and PFOS. Additionally, the Agency will continue to collect and review additional state and other occurrence information during the development of the proposed NPDWR for PFOA

and PFOS. The Agency will not be taking any further regulatory action under SDWA for the six negative determinations at this time.

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and Tuber Vegetables), except Sugar Beets; (3) Tolerance Conversions (i) from Crop Subgroup 4B to Crop Subgroup 22B (Leaf Petiole Vegetable), (ii) from Crop Subgroup 5A to Crop Group 5–16 (Brassica, Head and Stem Vegetable) and (iii) from Crop Subgroup 5B to Crop Subgroup 4–16B (Brassica Leafy Greens); and (4) Tolerance Expansions of Representative Commodities to (i) Cottonseed Subgroup 20C, and (ii) Stalk and Stem Vegetable Subgroup 22A, except Kohlrabi. Human Health Risk Assessment. EPA–HQ–OPP–2017–0465. September.

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Signing Statement

This document of the Environmental Protection Agency was signed on January 15, 2021, by Andrew Wheeler, Administrator, pursuant to the statutory requirements of the Safe Drinking Water Act, Section 1412(b). That document with the original signature and date is maintained by EPA. For administrative purposes only, and in compliance with requirements of the Office of the Federal Register, the undersigned EPA Official re-signs the document for publication, as an official document of the Environmental Protection Agency. This administrative process in no way alters the legal effect of this document upon publication in the *Federal Register*.

Jane Nishida,
Acting Administrator.

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