eligible to apply time credits under 18 U.S.C. 3632(d)(4)—including the extent to which any of the statutory provisions listed in this notice might affect the ability of some or all D.C. Code offenders to apply time credits—and not on the other contents of the November 25, 2020, proposed rule.

Issued under rulemaking authority vested in the Attorney General in 5 U.S.C. 301; 28 U.S.C. 509, 510 and delegated to the Director, Bureau of Prisons in 28 CFR 0.96.

Michael D. Carvajal,

Director, Federal Bureau of Prisons.

[FR Doc. 2021–22613 Filed 10–15–21; 8:45 am]

BILLING CODE P

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 372

[EPA–HQ–TRI–2017–0434; FRL–5927–03–OCSPP]

RIN 2070–AK26

Addition of Certain Chemicals; Community Right-to-Know Toxic Chemical Release Reporting

AGENCY: Environmental Protection Agency (EPA).

ACTION: Proposed rule.

SUMMARY: In response to a petition filed under the Emergency Planning and Community Right-to-Know Act (EPCRA), EPA is proposing to add 12 chemicals to the list of toxic chemicals subject to the reporting requirements under EPCRA and the Pollution Prevention Act (PPA). EPA believes that each of the 12 chemicals meets the EPCRA criteria. In addition, based on the available bioaccumulation and persistence data, EPA believes that one chemical should be classified as a persistent, bioaccumulative, and toxic (PBT) chemical and designated as a chemical of special concern with a 100-pound reporting threshold.

DATES: Comments must be received on or before December 17, 2021.

ADDRESSES: Submit your comments, identified by docket identification (ID) number EPA–HQ–TRI–2017–0434, using the Federal eRulemaking Portal at *http://www.regulations.gov.* Follow the online instructions for submitting comments. Do not submit electronically any information you consider to be Confidential Business Information (CBI) or other information whose disclosure is restricted by statute.

Due to the public health concerns related to COVID–19, the EPA Docket Center (EPA/DC) and Reading Room is closed to visitors with limited exceptions. The staff continues to provide remote customer service via email, phone, and webform. For the latest status information on EPA/DC services and docket access, visit *https://www.epa.gov/dockets.*

FOR FURTHER INFORMATION CONTACT: *For technical information contact:* Daniel R. Bushman, Toxics Release Inventory Program Division (7410M), Office of Pollution Prevention and Toxics, Environmental Protection Agency, 1200 Pennsylvania Ave. NW, Washington, DC 20460–0001; telephone number: (202) 566–0743; email: *bushman.daniel@epa.gov.*

For general information contact: The Emergency Planning and Community Right-to-Know Hotline; telephone numbers: toll free at (800) 424–9346 (select menu option 3) or (703) 348–5070 in the Washington, DC Area and International; or go to *https://www.epa.gov/home/epa-hotlines.*

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this action apply to me?

You may be potentially affected by this action if you own or operate a facility that manufactures, processes, or otherwise uses any of the 12 chemicals included in this proposed rule. The following list of North American Industrial Classification System (NAICS) codes is not intended to be exhaustive, but rather provides a guide to help readers determine whether this document applies to them. Potentially affected facilities may include:

- *Facilities included in the following NAICS manufacturing codes (corresponding to Standard Industrial Classification (SIC) codes 20 through 39)*: 311*, 312*, 313*, 314*, 315*, 316, 321, 322, 323*, 324, 325*, 326*, 327, 331, 332, 333, 334*, 335*, 336, 337*, 339*, 111998*, 113310, 211130*, 212324*, 212325*, 212393*, 212399*, 488390*, 511110, 511120, 511130, 511140*, 511191, 511199, 512230*, 512250*, 519130*, 541713*, 541715* or 811490*. (*Exceptions and/or limitations exist for these NAICS codes.)
- *Facilities included in the following NAICS codes (corresponding to SIC codes other than SIC codes 20 through 39)*: 212111, 212112, 212113 (corresponds to SIC code 12, Coal Mining (except 1241)); or 212221, 212222, 212230, 212299 (corresponds to SIC code 10, Metal Mining (except 1011, 1081, and 1094)); or 221111, 221112, 221113, 221118, 221121, 221122, 221330 (limited to facilities that combust coal and/or oil for the purpose of generating power for distribution in commerce) (corresponds to SIC codes 4911, 4931, and 4939, Electric Utilities); or 424690, 425110, 425120 (limited to facilities previously classified in SIC code 5169, Chemicals and Allied Products, Not Elsewhere Classified); or 424710 (corresponds to SIC code 5171, Petroleum Bulk Terminals and Plants); or 562112 (limited to facilities primarily engaged in solvent recovery services on a contract or fee basis (previously classified under SIC code 7389, Business Services, NEC)); or 562211, 562212, 562213, 562219, 562920 (limited to facilities regulated under the Resource Conservation and Recovery Act, subtitle C, 42 U.S.C. 6921 *et seq.*) (corresponds to SIC code 4953, Refuse Systems).
- *Federal facilities:* To determine whether your facility would be affected by this action, you should carefully examine the applicability criteria in part 372, subpart B of Title 40 of the Code of Federal Regulations. If you have questions regarding the applicability of this action to a particular entity, consult the person listed under **FOR FURTHER INFORMATION CONTACT**.

B. What action is the Agency taking?

In response to a petition, EPA is proposing to add 12 chemicals to the EPCRA section 313 toxic chemical list. As discussed in more detail later in this document, EPA believes that each of the 12 chemicals meets the EPCRA section 313(d)(2)(B) and/or (C) criteria for listing. EPA is also proposing to classify one chemical as a PBT chemical of special concern with a 100-pound reporting threshold.

C. What is the Agency's authority for taking this action?

This action is issued under EPCRA sections 313(d), 313(e)(1) and 328, 42 U.S.C. 11023(d), 11023(e)(1) and 11048. EPCRA is also referred to as Title III of the Superfund Amendments and Reauthorization Act of 1986.

EPCRA section 313, 42 U.S.C. 11023, requires owners/operators of certain facilities that manufacture, process, or otherwise use listed toxic chemicals in amounts above reporting threshold levels to report their facilities' environmental releases and other waste management information on such chemicals annually. These facility owners/operators must also report pollution prevention and recycling data for such chemicals, pursuant to section 6607 of the PPA, 42 U.S.C. 13106.

Under EPCRA section 313(c), Congress established an initial list of toxic chemicals subject to EPCRA toxic chemical reporting requirements that

was comprised of 308 individually listed chemicals and 20 chemical categories.

EPCRA section 313(d) authorizes EPA to add or delete chemicals from the list and sets criteria for these actions. EPCRA section 313(d)(2) states that EPA may add a chemical to the list if any of the listing criteria in EPCRA section 313(d)(2) are met. Therefore, to add a chemical, EPA must determine that at least one criterion is met, but need not determine whether any other criterion is met. Conversely, to remove a chemical from the list, EPCRA section 313(d)(3) dictates that EPA must determine that none of the criteria in EPCRA section 313(d)(2) are met. The listing criteria in EPCRA section 313(d)(2)(A)–(C) are as follows:

- The chemical is known to cause or can reasonably be anticipated to cause significant adverse acute human health effects at concentration levels that are reasonably likely to exist beyond facility site boundaries as a result of continuous, or frequently recurring, releases.
- The chemical is known to cause or can reasonably be anticipated to cause in humans: Cancer or teratogenic effects, or serious or irreversible reproductive dysfunctions, neurological disorders, heritable genetic mutations, or other chronic health effects.
- The chemical is known to cause or can be reasonably anticipated to cause, because of its toxicity, its toxicity and persistence in the environment, or its toxicity and tendency to bioaccumulate in the environment, a significant adverse effect on the environment of sufficient seriousness, in the judgment of the Administrator, to warrant reporting under this section.

EPA often refers to the EPCRA section 313(d)(2)(A) criterion as the ''acute human health effects criterion;'' the EPCRA section 313(d)(2)(B) criterion as the ''chronic human health effects criterion;'' and the EPCRA section 313(d)(2)(C) criterion as the ''environmental effects criterion.''

Under EPCRA section 313(e)(1), any person may petition EPA to add chemicals to or delete chemicals from the list. EPA issued a statement of policy in the **Federal Register** of February 4, 1987 (52 FR 3479) (FRL–3101–6) providing guidance regarding the recommended content of and format for petitions. On May 23, 1991 (56 FR 23703) (FRL–3802–2), EPA issued guidance regarding the recommended content of petitions to delete individual members of the metal compounds categories reportable under EPCRA section 313. EPA published in the **Federal Register** of November 30, 1994 (59 FR 61432) (FRL–4922–2) a statement clarifying its interpretation of the EPCRA section 313(d)(2) and (d)(3) criteria for modifying the EPCRA section 313 list of toxic chemicals.

II. What is the description of the petition and EPA's response?

A. Who submitted the petition and what was requested?

On May 6, 2014, EPA received a petition from the Toxics Use Reduction Institute (TURI) requesting the addition of 25 chemicals to the EPCRA section 313 toxic chemicals list (Ref. 1). The petitioner believes that each of these 25 chemicals meets the EPCRA section 313(d)(2) listing criteria and that the 25 chemicals should be added to the EPCRA section 313 toxic chemical list so that releases can be monitored and reported. The 25 chemicals, listed by name and Chemical Abstracts Service Registry Number (CASRN), are shown here (note that some chemical names are different than those used in the petition because they are listed here using the EPA Registry Name):

- Azodicarbonamide; 123–77–3
- 1-Bromopropane; 106–94–5
- 4-Chlorobenzotrichloride; 5216–25–1
- Cyclododecane; 294–62–2
- Dibutyltin dichloride; 683–18–1
- 1,3-Dichloro-2-propanol; 96–23–1
- Dimethylacetamide; 127–19–5
- 2,3-Dinitrotoluene; 602–01–7
- 2,5-Dinitrotoluene; 619–15–8
- Formamide; 75–12–7
- 1,2,5,6,9,10-Hexabromocyclododecane; 3194–55–6
- 1,3,4,6,7,8-Hexahydro-4,6,6,7,8,8-hexamethylcyclopenta[g]-2-benzopyran; 1222–05–5
- Hexahydrophthalic anhydride; 85–42–7
- N-Hydroxyethylethylenediamine; 111–41–1
- N-Methylformamide; 123–39–7
- Methylhexahydrophthalic anhydride; 25550–51–0
- Nitrilotriacetic acid trisodium salt; 5064–31–3
- Nonylphenol; 25154–52–3
- Octabromodiphenyl ether; 32536–52–0
- p-(1,1,3,3-Tetramethylbutyl)phenol; 140–66–9
- 1,2,3-Trichlorobenzene; 87–61–6
- Triglycidyl isocyanurate; 2451–62–9
- Tris(2-chloroethyl) phosphate; 115–96–8
- Tris(1,3-dichloro-2-propyl) phosphate; 13674–87–8
- Tris(dimethylphenol) phosphate; 25155–23–1

B. How is EPA responding to the petition?

As discussed in Unit I.B., EPA is proposing to add 12 of the 25 chemicals included in the TURI petition to the EPCRA section 313 toxic chemicals list. In separate, unrelated actions, three of the 25 chemicals (1-bromopropane (November 23, 2015 (80 FR 72906) (FRL–9937–12–OEI)), nonylphenol (September 30, 2014 (79 FR 58686) (FRL–9915–59–OEI)) and 1,2,5,6,9,10-hexabromocyclododecane (November 28, 2016 (81 FR 85440) (FRL–9953–28)) have already been added to the EPCRA section 313 chemical list. Of the remaining 10 chemicals, EPA has determined that the available data for nine chemicals are not sufficient for EPA to find that the chemicals meet the EPCRA section 313 listing criteria for human health or ecological effects (Refs. 2 and 3). Therefore, EPA is not proposing to add the nine chemicals listed here:

- Azodicarbonamide; 123–77–3
- 4-Chlorobenzotrichloride; 5216–25–1
- Cyclododecane; 294–62–2
- Dimethylacetamide; 127–19–5
- 2,3-Dinitrotoluene; 602–01–7
- 2,5-Dinitrotoluene; 619–15–8
- Hexahydrophthalic anhydride; 85–42–7
- Methylhexahydrophthalic anhydride; 25550–51–0
- N-Methylformamide; 123–39–7

In addition, EPA is not proposing to add octabromodiphenyl ether (OctaBDE) (32536–52–0) to the EPCRA section 313 toxic chemical list. EPA issued a significant new use rule (SNUR) that requires notification to EPA 90 days prior to the intended manufacture or import for any use of OctaBDE ether after January 1, 2005 (June 13, 2006 (71 FR 34015) (FRL–7743–2); 40 CFR 721.10000). The lack of significant new use notices (SNUNs) under this SNUR indicates that there has been no non-exempt manufacture or import for any use of OctaBDE in the United States since January 1, 2005. There have also been no submissions for OctaBDE under the Chemical Data Reporting (CDR) Rule (*https://www.epa.gov/chemical-data-reporting*) since 2006. In a 2008 evaluation, the United Nations noted that as of 2005, the manufacture and import of OctaBDE had been phased out by industry and estimated that most of the remaining processing of OctaBDE in the United States was likely negligible and only occurring where remaining stockpiles were being used up or in waste processing facilities (*http://chm.pops.int/portals/0/repository/poprc4/unep-pops-poprc.4-6.english.pdf*). Given that the phase out occurred more than ten years ago, it is even more likely today that there is a negligible amount of OctaBDE remaining that is processed or otherwise

used by facilities in the United States. Therefore, EPA is not proposing to add octabromodiphenyl ether to the EPCRA section 313 list since EPA expects that no TRI reports would be filed for this chemical. EPCRA section 313(d)(2) provides EPA the discretion to add chemicals to the TRI list when there is sufficient evidence to establish any of the listing criteria. EPA can add a chemical that meets one criterion regardless of its production volume. However, consistent with the Agency's previously articulated position on the use of manufacturing volume thresholds (*e.g.,* 58 FR 63500, December 1, 1993) (FRL–4904–6) and as in past chemical reviews (*e.g.,* 59 FR 61432, November 30, 1994) (FRL–4922–2), EPA adopted a production volume screen for the development of this proposed rule to screen out those chemicals for which no reports are expected to be submitted. If chemicals that did not meet the production volume screen were listed, there would be an economic burden for firms that would have to determine that they did not exceed the reporting threshold. Since the production volume screen indicates that no reports would be filed for such chemicals, there would be no information provided to the public. EPA feels it is appropriate at this time to focus on chemicals for which reports are likely to be filed.

In addition to proposing to add 1,3,4,6,7,8-hexahydro-4,6,6,7,8,8-hexamethylcyclopenta[g]-2-benzopyran to the EPCRA section 313 toxic chemical list, EPA is proposing to add this chemical to the list of chemicals of special concern. There are several chemicals and chemical categories on the EPCRA section 313 chemical list that have been classified as chemicals of special concern because they are PBT chemicals (see 40 CFR 372.28(a)(2)). In a final rule published in the **Federal Register** of October 29, 1999 (64 FR 58666) (FRL–6389–11), EPA established the PBT classification criteria for chemicals on the EPCRA section 313 chemical list. For purposes of EPCRA section 313 reporting, EPA established persistence half-life criteria for PBT chemicals of 2 months in water, sediment and soil and 2 days in air, and established bioaccumulation criteria for PBT chemicals as a bioconcentration factor (BCF) or bioaccumulation factor (BAF) of 1,000 or higher. Most chemicals meeting the PBT criteria are assigned 100-pound reporting thresholds. EPA set lower reporting thresholds (10 pounds) for those PBT chemicals with persistence half-lives of 6 months or more in water, sediment, or soil and with BCF or BAF values of 5,000 or higher, since these chemicals are considered highly PBT chemicals. The data presented in this proposed rule support classifying 1,3,4,6,7,8-hexahydro-4,6,6,7,8,8-hexamethylcyclopenta[g]-2-benzopyran as a PBT chemical and designating it as a chemical of special concern with a 100-pound reporting threshold.

III. What are the 12 chemicals that EPA is proposing to add?

The 12 chemicals that EPA is proposing to add are shown here listed by name and CASRN (note that some chemical names are different than those used in the petition because they are listed here using the EPA Registry Name):

- Dibutyltin dichloride; 683–18–1
- 1,3-Dichloro-2-propanol; 96–23–1
- Formamide; 75–12–7
- 1,3,4,6,7,8-Hexahydro-4,6,6,7,8,8-hexamethylcyclopenta[g]-2-benzopyran; 1222–05–5
- N-Hydroxyethylethylenediamine; 111–41–1
- Nitrilotriacetic acid trisodium salt; 5064–31–3
- p-(1,1,3,3-Tetramethylbutyl)phenol; 140–66–9
- 1,2,3-Trichlorobenzene; 87–61–6
- Triglycidyl isocyanurate; 2451–62–9
- Tris(2-chloroethyl) phosphate; 115–96–8
- Tris(1,3-dichloro-2-propyl) phosphate; 13674–87–8
- Tris(dimethylphenol) phosphate; 25155–23–1

EPA has determined that each of these chemicals have production and use levels that would result in TRI reports being filed (Ref. 4).

IV. What is the Agency's evaluation of the toxicity of the 12 chemicals?

EPA prepared hazard assessment documents that reviewed the available data on human health (Ref. 5) and/or ecological effects (Ref. 6) associated with each of the 12 chemicals being proposed for addition to the EPCRA section 313 toxic chemical list. Brief summaries of the available human health and ecological effects information that support listing these chemicals under EPCRA section 313 are provided in this Unit. Readers should consult the support documents (Refs. 5 and 6) for more detailed information.

1. *Dibutyltin dichloride (CASRN 683–18–1*). Monkey, rat, and mouse studies indicate that dibutyltin dichloride (DBTC) exposure during early pregnancy may result in embryo/fetal lethality following exposure to doses as low as 2.5 milligrams per kilogram per day (mg/kg/day) (Refs. 7, 8, 9, 10, 11, and 12). In these studies, decreased pre/post implantation loss, increased resorption, and/or decreased number of live fetuses/pups were accompanied by maternal body weight effects and/or clinical signs of toxicity. However, Ema and Harazono (Ref. 7) indicated that body weight effects alone did not account for reproductive effects, as effects observed at 15.2 mg/kg/day from gestation day 0–3 or 4–7 were significantly different than those observed in pair-fed controls that had similar body weights.

Several studies in rats indicate that maternal exposure to DBTC during the period of organogenesis causes external, skeletal, and/or visceral malformations and decreased body weight in fetuses at oral doses ≥5 mg/kg/day (Refs. 8, 9, 10, and 13). An increased incidence of external and skeletal malformations was observed in fetuses from dams exposed to doses as low as 5 mg/kg/day DBTC (lowest dose tested) from gestation day 7–15 (Ref. 8). Maternal toxicity was not observed in this study until 7.5 mg/kg/day (Ref. 8).

In summary, the available literature provides evidence that DBTC can be reasonably anticipated to cause serious or irreversible reproductive and developmental toxicity in humans. Based on the observed effects and dose levels, EPA considers DBTC to have moderately high to high toxicity. EPA believes that there is sufficient evidence for listing DBTC on the EPCRA section 313 toxic chemicals list pursuant to EPCRA section 313(d)(2)(B) based on the available reproductive and developmental toxicity data.

DBTC is toxic to aquatic organisms with experimentally determined acute and chronic toxicity values lower than 1 milligram per liter (mg/L). The acute aquatic toxicity values for DBTC are as low as 16.7 µg DBTC/L (96-hour median effect concentration (EC_{50}) for growth) in the green algae (*Scenedesmus obliquus*) (Ref. 14) and chronic aquatic toxicity values are as low as 20 µg/L for dibutyltin (DBT) (33-day lowest-observed-effect-concentration (LOEC) for reduction in shell length) in larvae of the blue mussel (*Mytilus edulis*), and 38 µg DBTC/L (210-day LOEC for reduced body weight and reduced stores of energy substrates) in the duck mussel (*Anodonta anatina*) (Ref. 15).

Several studies reported effects of short-term exposure to DBTC on estuarine and marine invertebrates. Salazar and Salazar (Ref. 16) observed a significant effect on mortality in mysids (*Metamysidopsis elongata*) exposed to DBTC at 56 µg/L for 96 hours, while no effect on mortality was observed at concentrations of ≤11 µg DBTC/L; the

96-hour LC_{50} was between 11 and 56 µg DBTC/L. Thom *et al.* (Ref. 17) exposed the embryos of Pacific oysters (*Crassostrea gigas*) to DBTC and found a 48-hour EC_{50} of 142 µg DBTC/L (55.5 µg tin (Sn)/L), based on abnormal larval development, and a 48-hour LC_{50} of 171 µg DBTC/L (66.9 µg Sn/L). In addition to affecting the survival and growth of aquatic organisms, DBTC has been shown to have adverse effects on the development of aquatic invertebrates at concentrations of 1 mg/L or less by causing abnormalities in the embryos of the Pacific oyster (*C. gigas*) (Ref. 18), preventing development of embryos of the tunicate (*Styela plicata*) to the larval stage (Ref. 19), and increasing the duration of zoeal development and reducing the dry weight of megalops larvae of the mud crab (*Rhithropanopeus harrisii*) (Ref. 20). Additionally, fish have been found to be more sensitive to DBTC in early life stages than as adults (Ref. 21). DBTC has been observed to cause histological changes in the liver, kidney, thymus, eye, and/or skin of Japanese medaka (*Oryzias latipes*) and guppy (*Poecilia reticulata*) (Refs. 22 and 23), reduced resistance to bacterial challenge in the rainbow trout (*Oncorhynchus mykiss*) (Ref. 21), and increased chromosomal aberrations in the land snail (*Truncatella subcylindrica*) (Ref. 24).

In summary, there is evidence for both acute and chronic toxicity to aquatic organisms exposed to DBTC. DBTC has been shown to cause lethality and impair growth and development in a wide range of aquatic species. The acute and chronic aquatic toxicity values indicate that DBTC is toxic at low concentrations and thus is highly toxic to aquatic organisms. EPA believes that the evidence is sufficient to list DBTC on the EPCRA section 313 toxic chemicals list pursuant to EPCRA section 313(d)(2)(C) based on the available ecotoxicity information for this chemical.

2. *1,3-Dichloro-2-propanol (CASRN 96–23–1).* Evidence from an unpublished 2-year bioassay indicates that 1,3-dichloro-2-propanol (DC2P) is carcinogenic in male and female rats (Refs. 25 and 26) following exposure to 240 mg/L in drinking water in rats of both sexes (19.31 mg/kg/day in males; 29.83 mg/kg/day in females). At the 78-week interim sacrifice, hepatocellular carcinomas were significantly increased in the high-dose male and female groups. At the termination of the study, exposure-related increases in neoplastic lesions were observed in the liver, kidney, and tongue; neoplasms observed in the thyroid may also be exposure-related. Additionally, 25 percent of liver carcinomas in high-dose females metastasized to the lung. Survival was reduced in both sexes at 240 mg/L over the second year of the study. Significant exposure-related changes in clinical chemistry observed predominantly in high-dose animals were indicative of liver damage and multiple non-neoplastic lesions were observed in both sexes at all doses in a dose- and duration-dependent manner.

It is reasonable to conclude that DC2P is genotoxic because of the preponderance of positive *in vitro* assays, though a limited number of *in vivo* studies reported negative results (Refs. 27, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37, 38, 39, 40 (as cited in Ref. 41), and 42 (as cited in Ref. 39)). The California EPA concluded that DC2P was ''clearly shown through scientifically valid testing according to generally accepted principles to cause cancer.'' (Ref. 43). Under the 2005 U.S. EPA guidelines (Ref. 44), DC2P is considered likely to be carcinogenic to humans based on strong evidence of carcinogenicity in male and female rats in a single adequate study and supporting mutagenicity data.

In summary, the available literature provides evidence that DC2P can be reasonably anticipated to cause cancer in humans. EPA considers chemicals that can reasonably be anticipated to cause cancer to have moderately high to high chronic toxicity. EPA believes that there is sufficient evidence for listing DC2P on the EPCRA section 313 toxic chemicals list pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data.

3. *Formamide (CASRN 75–12–7).* Available data from oral studies, including a 2-generation study, indicate that formamide is both a reproductive and developmental toxicant at doses ≥87 mg/kg/day (Refs. 45, 46, 47, 48, 49, 50, 51, and 52). These effects, including decreased pregnancy rates, increased days to litter, decreased live pups/litter, increased post implantation loss, and fetal variations, were observed in rats, mice, and rabbits, which serves to strengthen the conclusion on the potential reproductive and developmental toxicity of formamide. In two of the gestational exposure studies, fetal effects occurred at doses lower than overt maternal toxicity (decreased fetal body weights were observed in Sprague Dawley rats at 100 mg/kg/day and increased postimplantation loss and fetal variations were observed in NZ white rabbits at 113 mg/kg/day), suggesting that the developing organism is a sensitive target for formamide. The available dermal toxicity data suggest that formamide can cause developmental effects, including decreased fetal body weight and increased fetal variations and malformations at ≥310 mg/kg/day in rats (Refs. 45, 53, 54, and 55).

In summary, the available literature provides evidence that formamide can be reasonably anticipated to cause serious or irreversible reproductive and developmental toxicity in humans. Based on the observed effects and dose levels, EPA considers formamide to have moderately high to high toxicity. EPA believes that there is sufficient evidence for listing formamide on the EPCRA section 313 toxic chemicals list pursuant to EPCRA section 313(d)(2)(B) based on the available reproductive and developmental toxicity data.

4. *1,3,4,6,7,8-Hexahydro-4,6,6,7,8,8-hexamethylcyclopenta[g]-2-benzopyran (CASRN 1222–05–5).* 1,3,4,6,7,8-Hexahydro-4,6,6,7,8,8-hexamethylcyclopenta[g]-2-benzopyran (HHCB) is toxic to aquatic organisms, with experimentally determined acute and chronic toxicity values lower than 1 mg/L. The experimental data for HHCB from aquatic toxicity studies includes acute toxicity endpoint values as low as 723 µg/L in algae (72-hour EC_{50} for inhibition of biomass in the microalgal species (*Pseudokirchneriella subcapitata*) (Ref. 56 as cited in Ref. 57), 153 µg/L in aquatic invertebrates (96-hour EC_{50} in the mussel (*Lampsilis cardium*) (Ref. 58)), and 950 µg/L (concentration that is lethal to 50% of the test organisms (LC_{50})) in fish (*O. latipes*) larvae (Ref. 59). Chronic studies also indicate a high concern for environmental hazard with maximum acceptable toxicant concentration (MATC) values as low as 98 µg/L (36-day MATC for effects on larval survival, growth, and development in the fathead minnow (*Pimephales promelas*) (Ref. 60 as cited in Ref. 57)) and 4.7 µg/L in fish (14-day MATC for oxidative stress in goldfish (*Carassius auratus*) (Ref. 61)). Chronic studies in aquatic invertebrates have found a MATC as low as 53 µg/L (6-day MATC based on inhibition of larval development rate in the copepod (*Acartia tonsa*) (Ref. 62 as cited in Ref. 63)).

HHCB bioaccumulates in aquatic organisms. Experimentally-derived BCFs as high as 1,584 in fish (*Lepomis macrochirus*) (Ref. 64) and 2,692 in benthic worms (*Lumbriculus variegatus*) (Ref. 65 as cited in Ref. 63) have been reported. BCFs for HHCB calculated using the Estimation Programs Interface Suite™ (EPI Suite™) (Ref. 66) were 3,629 using the regression-based method and 1,231 using the Arnot-Gobas model for upper trophic level species, while the bioaccumulation factor (BAF)

calculated by EPI Suite™ was 1,826 (Ref. 67). There are no data available to evaluate the potential for HHCB to biomagnify through the food chain. Studies have consistently found half-lives longer than two months for HHCB in soils and sediments (Ref. 68). Envirogen (Ref. 69 as cited in Ref. 63) reported half-lives in river sediment at 79 days, forest soil at 95 days, sludge amended soil at 105 days, and agricultural soil at 239 days. DiFrancesco *et al.* (Ref. 70 as cited in Ref. 63) reported half-lives between 140–145 days in four types of sludge-amended soils.

In summary, the available data demonstrate that HHCB can cause acute and chronic toxicity to aquatic organisms at concentrations at or below 1 mg/L. The acute and chronic aquatic toxicity values indicate that HHCB is highly toxic to aquatic organisms. In addition, HHCB bioaccumulates and is persistent in the environment. EPA believes that the evidence is sufficient to list HHCB on the EPCRA section 313 toxic chemicals list pursuant to EPCRA section 313(d)(2)(C) based on the available ecotoxicity information for this chemical alone and also based on its toxicity and persistence in the environment, and toxicity and tendency to bioaccumulate.

EPA believes that the available bioaccumulation and persistence data for HHCB support a classification of HHCB as a persistent, bioaccumulative, and toxic (PBT) chemical. HHCB has been shown to be bioaccumulative in aquatic species with BCF values greater than 1,000 and to be persistent in soil and sediment for at least 2 months. Therefore, consistent with EPA's established policy for PBT chemicals (See 64 FR 58666, October 29, 1999) (FRL–6389–11), EPA is proposing to designate HHCB as a chemical of special concern with a 100-pound reporting threshold.

5. *N-Hydroxyethylethylenediamine (CASRN 111–41–1).* Several rat studies, including pre-mating though early lactation oral exposure and gestational oral exposure, indicate that maternal exposure to N-hydroxyethylethylenediamine can cause malformations of the great vessels in offspring at gavage doses ≥10 mg/kg/day, particularly aortic aneurysms (Refs. 71, 72, 73, 74, and 75). Other observed malformations included aneurysms of the pulmonary trunk, dilations of the carotids and descending aorta, and abnormal course of the carotids. While some of these studies (Refs. 71, 73, and 74) presented a limited consideration of material endpoints and lacked litter-based statistics, studies incorporating these elements reported similar developmental effects (Refs. 72 and 75). Aortic aneurysms were also observed at intraperitoneal injection doses ≥10 mg/kg/day (Refs. 71 and 76). Available evidence indicates that, at high enough doses, prenatal exposure is adequate to induce great vessel malformations; however, the critical period appears to extend into the early postnatal period since incidence and severity of great vessel malformations was increased when exposure extended into the postnatal period (Refs. 77, 78, 79, 80, and 81). This may, in part, explain why no vessel malformations were observed at doses up to 50 mg/kg-day on GD 6–19 and examination of fetuses on GD 20 in the EPSDG study (Ref. 75), while aneurysms were observed with dosing at ≥10 mg/kg-day on GD 14–20 and examination of pups on PND 1 in the Xu *et al.* study (Ref. 71).

Mechanistic studies indicate that great vessel malformation may be due to decreased expression of collagen type 1 and 3 in the walls of the great vessels (Ref. 71). A recent study by Chen *et al.* (Ref. 82) concluded that HEED causes significant morphological, biochemical, and biomechanical alterations in the extracellular matrix in neonatal aortic vascular smooth muscle cells. Additionally, Moore *et al.* (Ref. 83) exposed dams to HEED and confirmed exposure of offspring both *in utero* and during lactation. HEED did not, however, appear to specifically concentrate in the great vessels of offspring.

In summary, the available literature provides evidence that N-hydroxyethyl-ethylenediamine can be reasonably anticipated to cause serious or irreversible developmental toxicity in humans. Based on the observed effects and dose levels, EPA considers N-hydroxyethyl-ethylenediamine to have moderately high to high toxicity. EPA believes that there is sufficient evidence for listing N-hydroxyethylethylenediamine on the EPCRA section 313 toxic chemicals list pursuant to EPCRA section 313(d)(2)(B) based on the available developmental toxicity data.

6. *Nitrilotriacetic acid trisodium salt (CASRN 5064–31–3).* Evidence from bioassays of 18–24 months indicates that nitrilotriacetic acid trisodium salt (NTA) compounds are carcinogenic in rats and mice (Refs. 84 and 85). Tumors were significantly increased at dietary doses ≥1,200 mg/kg/day in rats of both sexes, ≥590 mg/kg/day in male mice, and 2,600 mg/kg/day in female mice, and at drinking water doses of 81 mg/kg/day in male rats (only dose tested, females not evaluated). Exposure-related increases in neoplastic lesions were observed in the urinary tract of male and female rats and mice (kidney, ureter, and/or bladder), adrenal glands (female rats), liver (female rats), pituitary gland (male rats), and hematopoietic system (male mice). Significant non-neoplastic and pre-neoplastic lesions were also observed in the kidney, lung, bladder, and ureter, especially at the highest doses (at dietary doses ≥1,200 mg/kg/day in rats and at drinking water doses of 81 mg/kg/day in male rats). In rats, nitrilotriacetic acid trisodium salt monohydrate ($Na_3NTA{\cdot}H_2O$) was a renal and bladder tumor promoter, but NTA did not promote bladder tumors (Refs. 86, 87, 88, 89, 90, and 91). In both the cancer bioassays and promotion studies featuring multiple dose levels, NTA compounds were effective at higher doses while showing no activity at lower doses. This suggests that high levels may be required for promotion or tumorigenicity. Specific doses that induce activity, however, appear to differ with route (*i.e.,* carcinogenicity seen at lower doses via drinking water than via diet). Genotoxicity data, in general, indicate that NTA compounds do not induce direct genetic effects, although there is some evidence that they may interfere with normal segregation of chromosomes (Refs. 92, 93, and 94).

Under the U.S. EPA 2005 guidelines (Ref. 44), NTA is considered likely to be carcinogenic to humans, based on evidence of carcinogenicity in male and female rats and mice in three adequate dietary bioassays reported by the National Cancer Institute (Ref. 85), along with supporting evidence of carcinogenicity from a drinking water study using only one dose level (Ref. 84) and tumor promoting activity of Na3NTA·H2O (Refs. 86, 87, 88, 89, 90, and 91). In addition, the National Toxicology Program concluded that ''Nitrilotriacetic acid is *reasonably anticipated to be a human carcinogen* based on sufficient evidence of carcinogenicity from studies in experimental animals.'' and noted that ''exposure to the trisodium salt had the same effects in rats and also caused kidney tumors and cancer of the ureter in female rats (Refs. 84 and 85).''

In summary, the available literature provides evidence that nitrilotriacetic acid trisodium salt can be reasonably anticipated to cause cancer in humans. EPA considers chemicals that can reasonably be anticipated to cause cancer to have moderately high to high chronic toxicity. EPA believes that there is sufficient evidence for listing nitrilotriacetic acid trisodium salt on the

EPCRA section 313 toxic chemicals list pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data.

7. *p-(1,1,3,3-Tetramethylbutyl)phenol (CASRN 140–66–9).* p-(1,1,3,3-Tetramethylbutyl)phenol (TMBP) is toxic to aquatic organisms with experimentally determined acute and chronic toxicity values lower than 1 mg/L. The experimental data for TMBP include acute toxicity endpoint values as low as 47.9 µg/L in aquatic invertebrates (96-hour LC_{50} in mysid shrimp (*Mysidopsis bahia*) (Ref. 95)), 120 µg/L in fish (14-day LC_{50} in rainbow trout (*O. mykiss*) (Ref. 96)), and 0.2 µg/L in amphibians (24-hour LOEC for early sexual differentiation in bullfrog tadpoles (*Rana catesbeiana*) (Ref. 97)). Chronic toxicity endpoint values are as low as 0.03 µg/L in aquatic invertebrates (21-day MATC for delayed nauplii development in the copepod (*Tigriopus japonicas*) (Ref. 98)), 1 µg/L in fish (35-day LOECs for reduced growth in rainbow trout larvae (*O. mykiss*) (Ref. 99)), and 0.002 µg/L in amphibians (48-week LOEC for malformations and abnormalities and developmental delay in Northern leopard frog tadpoles (*Rana pipiens*) (Refs. 100 and 101)). The majority of chronic toxic effects on aquatic organisms were due to endocrine disruption. For example, TMBP mimics the effects of 17β-estradiol by binding to the estrogen receptor and acting as an estrogen agonist (Refs. 99, 102, 103, 104, and 105). Examples of estrogenic effects caused by TMBP in male fish include induction of synthesis of vitellogenin (an egg yolk protein precursor that is not usually synthesized in male fish, but can be induced by estrogen), inhibition of testicular growth and spermatogenesis, and reduction of the gonadosomatic index (gonad mass as a percentage of total body mass) (Refs. 106, 107, and 108).

TMBP bioaccumulates in aquatic organisms. Whole fish wet weight based BCFs determined under controlled experimental conditions at steady state were 471 in rainbow trout (*O. mykiss*) and 261 in Japanese medaka (*O. latipes*) (Refs. 109 and 110). Wet weight based field BAFs in fish were similar, ranging from 46 to 297 (Ref. 111). Maximum BAF values for the blue mussel (*M. edulis*) were 1,280 when converted to a wet weight basis (Refs. 112 and 113). A maximum value for phytoplankton was 2,510 when converted to a wet weight basis (Refs. 112 and 113). BCFs for TMBP calculated using the Estimation Programs Interface Suite™ (EPI Suite™) (Ref. 66) were also similar: 243 using the regression-based method and 302 using the Arnot-Gobas model for upper trophic level species. There was some evidence of biomagnification in fish species preying on mussels and in herring gulls feeding on fish (Ref. 112).

In summary, the available data demonstrate that TMBP can cause acute and chronic toxicity to aquatic organisms at low concentrations indicating that TMBP is highly toxic to aquatic organisms. TMBP can cause lethality and impair growth and reproduction and is also an endocrine disruptor that may lead to estrogenic effects. TMBP has the potential to bioaccumulate in aquatic organisms and there is limited evidence for biomagnification of TMBP. EPA believes that the evidence is sufficient to list TMBP on the EPCRA section 313 toxic chemicals list pursuant to EPCRA section 313(d)(2)(C) based on the available ecotoxicity information for this chemical alone and also based on its toxicity and tendency to bioaccumulate.

8. *1,2,3-Trichlorobenzene (CASRN 87–61–6).* 1,2,3-Trichlorobenzene (1,2,3-TCB) is toxic to aquatic organisms with experimentally determined acute and chronic toxicity values lower than 1 mg/L. The experimental data for 1,2,3-TCB include acute toxicity endpoint values as low as 330 µg/L in aquatic invertebrates (96-hour LC_{50} in the mysid shrimp (*M. bahia*) (Ref. 114)) and 350 µg/L in fish (96-hour LC_{50} in the guppy (*P. reticulata*) (Ref. 115)). Chronic toxicity endpoint values are as low as 22 µg/L in aquatic invertebrates (28-day MATC for inhibition of reproduction and growth in *M. bahia* (Ref. 116)) and 44 µg/L in fish (42-day MATC for reduced growth in the mosquitofish (*Gambusia affinis*) (Ref. 117)).

1,2,3-TCB bioaccumulates in aquatic organisms. There are experimentally-derived BCF values in fish over 1,000 and as high as 5,600 for the fathead minnow (*P. promelas*) (Ref. 118). A biomagnification factor (BMF) of 2.3 was estimated by Hendriks *et al.* (Ref. 119) for an aquatic food chain.

In summary, based on experimental data from both acute and chronic studies of aquatic organisms, 1,2,3-TCB is toxic to aquatic organism at low concentrations. The acute and chronic aquatic toxicity values indicate that 1,2,3-TCB is highly toxic to aquatic organisms. In addition, 1,2,3-TCB has been shown to be highly bioaccumulative in fish. EPA believes that the evidence is sufficient to list 1,2,3-TCB on the EPCRA section 313 toxic chemicals list pursuant to EPCRA section 313(d)(2)(C) based on the available ecotoxicity information for this chemical alone and also based on its toxicity and tendency to bioaccumulate.

9. *Triglycidyl isocyanurate (CASRN 2451–62–9).* Available animal toxicology studies on triglycidyl isocyanurate (TGIC) provide evidence of male reproductive toxicity. For example, a subchronic (13 week) oral exposure study in rats exposed to 0, 0.72, 2.08, and 7.32 mg/kg/day TGIC reported a dose-dependent decrease in the mean number of spermatozoa (0.0%, 5.1%, 13.5%, and 23.1%, respectively) with statistical significance at the high dose (Ref. 120). No mortalities, clinical signs of toxicity, or effects on any fertility parameters were observed during the study. However, although no significant effects on male rat fertility were observed, a decrease in sperm count could have biological significance in humans since it is well-known that the human male is of relatively low fertility and thus may be at greater risk from effects on sperm parameters than are males of the common laboratory animal model species (Ref. 121).

Supplemental data from shorter-term exposure studies in mice also provide some additional supporting evidence for male reproductive effects following exposure to TGIC. For example, in spermatogonial cytogenics assays, decreased spermatogonial cell survival was reported in NS mice exposed orally to a single dose of 115 mg/kg/day (Ref. 122), but not in CD–1 mice exposed by inhalation (Ref. 122). In a dose-range finding study, ICR mice demonstrated decreased spermatogonial cell survival at 667 mg/kg/day administered via oral gavage (Ref. 123). The differences in responses among these studies may be due to differences in sensitivity between mice strain and route of exposure. In dominant lethal assays, although impairment of reproductive performance (decreased mating index) in CD–1 mice exposed via inhalation was reported, it occurred at the same level (49.6 mg/m^3) exhibiting 10% mortality, decreased body weight, as well as clinical signs of toxicity, and may not be indicative of reproductive effects (Ref. 124). Likewise, ICR mice exposed orally failed to show an impairment of male mice impregnating unexposed females at 550 mg/kg/day (Ref. 125). Of the few genotoxicity studies of TGIC identified in the literature, TGIC did not induce chromosomal aberrations in spermatogonial cells in mice (Ref. 126) but did induce both sister chromatid exchange and chromosomal aberrations in Chinese hamster ovary cells in vitro (Ref. 127 and Ref. 128).

In summary, the available data indicate that the male reproductive

system, particularly spermatogonia and spermatozoa, may be a target of TGIC toxicity. Effects on sperm measurements were seen across two species (rats and mice) and routes of exposure (oral and inhalation) following subchronic and shorter-term exposures and collectively provide sufficient evidence of male reproductive toxicity. Based on the observed effects and dose levels, EPA considers TGIC to have moderately high to high toxicity. Therefore, EPA believes there is sufficient evidence for listing TGIC on the EPCRA section 313 toxic chemicals list pursuant to EPCRA section 313(d)(2)(B) based on the available reproductive toxicity data.

10. *Tris(2-chloroethyl) phosphate (CASRN 115–96–8).* The National Toxicology Program (NTP) (Ref. 129) performed 2-year oral bioassays of tris(2-chloroethyl) phosphate (TCEP) in male and female rats and mice. The NTP concluded there is clear evidence of carcinogenicity in both male and female rats based on renal tubule adenomas observed at 88 mg/kg/day and noted that mononuclear cell leukemia and thyroid follicular cell neoplasms in both sexes may also be exposure related. A significant increase in the incidence of renal tubule adenomas in male and female rats was observed at 88 mg/kg/day. From the mouse bioassay, the NTP concluded that there was equivocal evidence for carcinogenicity in male mice based on a marginal increase in renal tubule cell neoplasms and in female mice based on a marginal increase in harderian gland neoplasms in the main study group (14% incidence at 350 mg/kg/day versus 6% incidence in controls). The incidence of harderian gland tumors in females (main study and interim sacrifice groups combined) was statistically increased at the high dose of 350 mg/kg/day ($p \leq 0.05$) with a significant dose-related trend ($p \leq 0.05$). Significant non-neoplastic and pre-neoplastic lesions occurred in both male and female rats at 88 mg/kg/day (in the brain stem, cerebrum, and kidney) and in both male and female mice at ≥175 mg/kg/day (in the kidney). Genotoxicity data indicate that TCEP is not mutagenic, and evidence for clastogenicity and cell transformation is limited and inconsistent (Refs. 130, 131, 132, 133, 134, 135, 136 as cite in Ref. 129, 137, and 138).

Under the U.S. EPA 2005 guidelines (Ref. 44), TCEP is considered likely to be carcinogenic to humans, based on clear evidence of carcinogenicity in male and female rats and equivocal evidence in male and female mice in adequate studies performed by NTP (Ref. 129). In 2009, EPA's Office of Research and Development reached the same conclusion when it derived the provisional peer-reviewed toxicity values for TCEP (Ref. 139).

Available data indicate that TCEP causes reproductive toxicity in mice, including sperm alterations and decreases in fertility in treated males and altered sex ratios in pups. A two-generation study with continuous breeding protocol showed that oral exposure to TCEP caused a decrease in the number of live male pups/litter and an altered sex ratio at 175 mg/kg/day and decreases in the numbers of litters/pair and live pups/litter at 350 mg/kg/day; a crossover breeding trial indicated that these effects were predominantly due to effects in male mice, including decreased fertility and sperm alterations (Ref. 140). Dose-related sperm alterations in mice have also been reported following oral exposure to 700 mg/kg/day TCEP for 16 weeks (Ref. 141). Sperm effects were also noted in an inhalation study in male rats continuously exposed to ≥0.5 mg/m^3 for 4 months, with decreased litter size and increased pre- and post-implantation loss observed when males exposed to 1.5 mg/m^3 were mated to naïve females (Ref. 142 as cited in Ref. 140). There was no evidence of adverse effects in the female reproductive system in either the two-generation study with crossover trial or the subchronic reproductive screen (Refs. 129 and 141). A gestational exposure study found no evidence for developmental toxicity resulting from TCEP exposure (Refs. 143 and 144).

In summary, the available literature provides evidence that TCEP can be reasonably anticipated to cause cancer and serious or irreversible reproductive toxicity in humans. EPA considers chemicals that can reasonably be anticipated to cause cancer to have moderately high to high chronic toxicity. In addition, based on the observed reproductive effects and dose levels causing those effects, EPA considers TCEP to have moderately high to high toxicity. EPA believes that there is sufficient evidence for listing TCEP on the EPCRA section 313 toxic chemicals list pursuant to EPCRA section 313(d)(2)(B) based on the available cancer and reproductive toxicity data.

11. *Tris(1,3-dichloro-2-propyl) phosphate (CASRN 13674–87–8).* Evidence from a 2-year bioassay indicates that tris(1,3-dichloro-2-propyl) phosphate (TDCPP) is carcinogenic in male and female rats (Ref. 145). Tumors were significantly increased at ≥20 mg/kg/day in rats of both sexes. Exposure-related increases in neoplastic lesions were observed in the kidney (both sexes at high dose), liver (both sexes), testes (males), and adrenal glands (females). Significant non-neoplastic lesions were also observed in the kidney and liver of male and female rats and in the epididymides and seminal vesicles of male rats. Genotoxicity data indicate that TDCPP is mutagenic in bacteria with metabolic activation, although assays for mutagenicity in mammalian cells and fruit flies were negative (Refs. 146, 147, 148, 149, 150 and 151). Assays for clastogenicity in mammalian cells *in vitro* were positive with activation, but *in vivo* studies were negative (Refs. 146, 148, and 152). Results for cell transformation were mixed (Refs. 146 and 151).

The California EPA concluded that TDCPP was ''clearly shown through scientifically valid testing according to generally accepted principles to cause cancer.'' (Ref. 153). Under the U.S. EPA 2005 guidelines (Ref. 44), TDCPP is considered likely to be carcinogenic to humans, based on strong evidence of carcinogenicity in male and female rats with multiple tumors in a single yet largely adequate chronic cancer bioassay study and supporting mutagenicity data of both the primary compound and metabolites, in bacteria.''

In summary, the available literature provides evidence that TDCPP can cause cancer at multiple sites in rats and can be reasonably anticipated to cause cancer in humans based on the animal data and the overall weight of mutagenicity and genotoxicity in bacteria and mammalian cells. EPA considers chemicals that can reasonably be anticipated to cause cancer to have moderately high to high chronic toxicity. EPA believes that there is sufficient evidence for listing TDCPP on the EPCRA section 313 toxic chemicals list pursuant to EPCRA section 313(d)(2)(B) based on the available carcinogenicity data.

TDCPP is toxic to aquatic organisms both from acute and chronic exposures with acute toxicity below 10 mg/L and chronic toxicity below 0.1 mg/L. Observed acute aquatic toxicity values are as low as 1,400 µg/L (96-hour LC_{50}) in rainbow trout (*O. mykiss*) (Ref. 154). Chronic aquatic toxicity values are below 0.1 mg/L and are as low as 22 µg/L (142-hour MATC for decreases in body weight and whole-body thyroxin (T4) content) in zebrafish (*Danio rerio*) (Ref. 155) and 20 µg/L (116-hour LOEC for effects on mRNA expression of genes for estrogen and progesterone receptors and vitellogenin) in *D. rerio* (Ref. 156). EPA has previously determined that TDCPP is persistent in the environment with a half-life >60 days (Ref. 157).

In summary, the acute toxicity data for TDCPP for fish range from 1 to 10 mg/L and chronic aquatic toxicity values range from 20 to 1,000 µg/L. TDCPP has also been shown to be persistent in the environment. Based on experimental data from both acute and chronic studies of aquatic organisms, TDCPP is toxic to aquatic organism at low concentrations. The acute and chronic aquatic toxicity values along with the persistence data indicate that TDCPP is highly toxic to aquatic organisms. EPA believes that the evidence is sufficient to list TDCPP on the EPCRA section 313 toxic chemicals list pursuant to EPCRA section 313(d)(2)(C) based on the available ecotoxicity data and its persistence in the environment.

12. *Tris(dimethylphenol) phosphate (CASRN 25155–23–1).* In a one-generation reproductive/developmental toxicity screening study in rats, the pregnancy index was significantly decreased by tris(dimethylphenol) phosphate (TDMPP) at gavage doses as low as 200 mg/kg/day as demonstrated by the reduced number of implantations and the decreased number of gravid dams and successful parturitions (Ref. 158 as cited in Ref. 159). While these effects were shown to be reversible in the recovery group (*i.e.,* animals maintained for 4 weeks without exposure, after which rats were mated), they were accompanied by significant effects on organ weight and histological changes at doses as low as 25 mg/kg/day. These treatment-related organ weight and histological changes were also partly reversible in the recovery group.

In summary, the available data provides evidence that TDMPP can be reasonably anticipated to cause serious or irreversible reproductive toxicity in humans. Based on the observed effects and dose levels, EPA considers TDMPP to have moderately high to high toxicity. EPA believes that there is sufficient evidence for listing TDMPP on the EPCRA section 313 toxic chemicals list pursuant to EPCRA section 313(d)(2)(B) based on the available reproductive toxicity data.

V. Why is EPA proposing to list the 12 chemicals and lower the reporting threshold for HHCB?

A. What is EPA's rationale for listing the 12 chemicals?

Based on EPA's review of the available toxicity data, EPA believes that the 12 chemicals EPA is proposing to add to the EPCRA section 313 toxic chemical list can reasonably be anticipated to cause either adverse chronic human health effects at moderately low to low doses and/or environmental effects at low concentrations. EPA believes that the data show that these 12 chemicals have moderately high to high human health toxicity and/or are highly toxic to aquatic organisms. Therefore, EPA believes that the evidence is sufficient for listing all 12 of the chemicals in this proposed rule on the EPCRA section 313 toxic chemicals list pursuant to EPCRA section 313(d)(2)(B) and/or (C).

EPA does not believe that it is appropriate to consider exposure for chemicals that are moderately high to highly toxic based on a hazard assessment when determining if a chemical can be added for chronic human health effects pursuant to EPCRA section 313(d)(2)(B) (see 59 FR 61440–61442). EPA also does not believe that it is appropriate to consider exposure for chemicals that are highly toxic based on a hazard assessment when determining if a chemical can be added for environmental effects pursuant to EPCRA section 313(d)(2)(C) (see 59 FR 61440–61442). Therefore, in accordance with EPA's standard policy on the use of exposure assessments (see November 30, 1994 (59 FR 61432, FRL–4922–2), EPA does not believe that an exposure assessment is necessary or appropriate for determining whether any of the chemicals in this proposed rule meet the criteria of EPCRA section 313(d)(2)(B) or (C).

B. What is EPA's rationale for lowering the reporting threshold for HHCB?

EPA believes that the available bioaccumulation and persistence data for HHCB support a classification of HHCB as a PBT chemical. HHCB has been shown to be bioaccumulative in aquatic species with BCF values greater than 1,000 and to persist in soils and sediments with half-lives greater than 2 months. Therefore, consistent with EPA's established policy for PBT chemicals (see 64 FR 58666, October 29, 1999) (FRL–6389–11), EPA is proposing to establish a 100-pound reporting threshold for HHCB.

VI. References

The following is a listing of the documents that are specifically referenced in this document. The docket includes these documents and other information considered by EPA, including documents that are referenced within the documents that are included in the docket, even if the referenced document is not itself physically located in the docket. For assistance in locating these other documents, please consult the person listed under **FOR FURTHER INFORMATION CONTACT**.

1. Petition from the Massachusetts Toxics Use Reduction Institute (TURI), University of Massachusetts Lowell, 600 Suffolk St., Suite 501, Lowell, MA 01854, May 6, 2014.
2. USEPA, OPPT. Memorandum from Jocelyn Hospital, Toxicologist, Regulatory Development Branch to David Turk, Chief, Regulatory Development Branch. December 8, 2016. Subject: Review of Toxics Use Reduction Institute (TURI) Petition Chemicals.
3. USEPA, OPPT. Memorandum from Kara Koehrn and Thomas Forbes, Regulatory Development Branch, to David Turk, Chief, Regulatory Development Branch. February 16, 2017. Subject: Review of Toxics Use Reduction Institute (TURI) Petition Chemicals.
4. USEPA, OPPT. 2018. Economic Analysis of the Proposed Rule to Add Twelve Chemicals Identified in a Petition from the Toxics Use Reduction Institute to the EPCRA Section 313 List of Toxic Chemicals. November 7, 2018.
5. USEPA, OPPT. 2016. Human Health Review of Chemicals from the Toxics Use Reduction Institute (TURI) Petition. Office of Pollution Prevention and Toxics, Toxics Release Inventory Program Division, Regulatory Developmental Branch. March 29, 2016.
6. USEPA, OPPT. 2017. Ecological Toxicity Review of Chemicals from the Toxics Use Reduction Institute (TURI) Petition. Office of Pollution Prevention and Toxics, Toxics Release Inventory Program Division, Regulatory Developmental Branch. July 18, 2017.
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organometallic compounds on algae. Appl. Organomet. Chem. 7: 373–380.
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VII. What are the statutory and Executive Orders reviews associated with this action?

Additional information about these statutes and Executive Orders can be found at *http://www2.epa.gov/laws-regulations/laws-and-executive-orders.*

A. Executive Order 12866: Regulatory Planning and Review and Executive Order 13563: Improving Regulation and Regulatory Review

This action is not a significant regulatory action and was therefore not submitted to the Office of Management and Budget (OMB) for review under Executive Orders 12866 (58 FR 51735, October 4, 1993) and 13563 (76 FR 3821, January 21, 2011).

B. Paperwork Reduction Act (PRA)

This action does not contain any new information collection activities that require additional approval by OMB under the PRA, 44 U.S.C. 3501 *et seq.* OMB has previously approved the information collection activities contained in the existing regulations and has assigned OMB control numbers 2070–0212 (EPA ICR No. 2613.02, entitled ''Toxic Chemical Release Reporting'') and 2050–0078 (EPA ICR No. 1428.11, entitled ''Trade Secret Claims for Community Right-to-Know and Emergency Planning''). Currently, the facilities subject to the reporting requirements under EPCRA section 313 and PPA section 6607 may use either the EPA Toxic Chemicals Release Inventory Form R (EPA Form 9350–1), or the EPA Toxic Chemicals Release Inventory Form A (EPA Form 9350–2). The Form R must be completed if a facility manufactures, processes, or otherwise uses any listed chemical above threshold quantities and meets certain other criteria. For the Form A, EPA established an alternative threshold for facilities with low annual reportable amounts of a listed toxic chemical. A facility that meets the appropriate reporting thresholds, but estimates that the total annual reportable amount of the chemical does not exceed 500 pounds per year, can take advantage of an alternative manufacture, process, or otherwise use threshold of 1 million pounds per year of the chemical, provided that certain conditions are met, and submit the Form A instead of the Form R. In addition, respondents may designate the specific chemical identity of a substance as a trade secret pursuant to EPCRA section 322, 42 U.S.C. 11042, 40 CFR part 350.

OMB has approved the reporting and recordkeeping requirements related to Forms A and R, supplier notification, and petitions under OMB Control number 2070–0212 and those related to trade secret designations under OMB Control 2050–0078. As provided in 5 CFR 1320.5(b) and 1320.6(a), an Agency may not conduct or sponsor, and a person is not required to respond to, a collection of information unless it displays a currently valid OMB control number. The OMB control numbers relevant to EPA's regulations are listed in 40 CFR part 9 and displayed on the information collection instruments (*e.g.,* forms, instructions).

C. Regulatory Flexibility Act (RFA)

I certify that this action will not have a significant economic impact on a substantial number of small entities under the RFA, 5 U.S.C. 601 *et seq.* The small entities subject to the requirements of this action are small manufacturing facilities. The Agency has determined that of the 488 entities estimated to be impacted by this action, 449 are small businesses; no small governments or small organizations are expected to be affected by this action. All 449 small businesses affected by this action are estimated to incur annualized cost impacts of less than 1% of annual revenue or sales. Thus, this action is not expected to have a significant adverse economic impact on a substantial number of small entities. A more detailed analysis of the impacts on small entities is provided in EPA's economic analysis (Ref. 4).

D. Unfunded Mandates Reform Act (UMRA)

This action does not contain an unfunded mandate of $100 million or more as described in UMRA, 2 U.S.C. 1531–1538, and does not significantly or uniquely affect small governments. This action is not subject to the requirements of UMRA because it contains no regulatory requirements that might significantly or uniquely affect small governments. EPA did not identify any small governments that would be impacted by this action. EPA's economic analysis indicates that the total cost of this action is estimated to be $2,057,000 in the first year of reporting (Ref. 4).

E. Executive Order 13132: Federalism

This action does not have federalism implications as specified in Executive Order 13132 (64 FR 43255, August 10, 1999). It will not have substantial direct effects on the States, on the relationship between the national government and the States, or on the distribution of power and responsibilities among the various levels of government.

F. Executive Order 13175: Consultation and Coordination With Indian Tribal Governments

This action does not have tribal implications as specified in Executive Order 13175 (65 FR 67249, November 9, 2000). This action relates to toxic chemical reporting under EPCRA section 313, which primarily affects private sector facilities. Thus, Executive Order 13175 does not apply to this action.

G. Executive Order 13045: Protection of Children From Environmental Health Risks and Safety Risks

EPA interprets Executive Order 13045 (62 FR 19885, April 23, 1997) as applying only to those regulatory actions that concern environmental health or safety risks that EPA has reason to believe may disproportionately affect children, per the definition of ''covered regulatory action'' in section 2–202 of the Executive Order. This action is not subject to Executive Order 13045 because it does not concern an environmental health risk or safety risk.

H. Executive Order 13211: Actions Concerning Regulations that Significantly Affect Energy Supply, Distribution, or Use

This action is not subject to Executive Order 13211 (66 FR 28355, May 22, 2001), because it is not a significant regulatory action under Executive Order 12866.

I. National Technology Transfer and Advancement Act (NTTAA)

This rulemaking does not involve any technical standards subject to NTTAA section 12(d) (15 U.S.C. 272 note).

J. Executive Order 12898: Federal Actions To Address Environmental Justice in Minority Populations and Low-Income Populations

The EPA believes that this action is not subject to Executive Order 12898 (59 FR 7629, February 16, 1994) because it does not establish an environmental health or safety standard. This regulatory action adds additional chemicals to the EPCRA section 313 reporting requirements; it does not have

any impact on human health or the environment. This action does not address any human health or environmental risks and does not affect the level of protection provided to human health or the environment. The addition of these chemicals to the EPCRA section 313 reporting requirements will provide information that government agencies and others can use to identify potential problems, set priorities, and help inform activities.

List of Subjects in 40 CFR Part 372

Environmental protection, Community right-to-know, Reporting and recordkeeping requirements, and Toxic chemicals.

Dated: October 6, 2021.

Michal Freedhoff,

Assistant Administrator, Office of Chemical Safety and Pollution Prevention.

Therefore, for the reasons stated in the preamble, it is proposed that 40 CFR chapter I be amended as follows:

PART 372—TOXIC CHEMICAL RELEASE REPORTING: COMMUNITY RIGHT-TO-KNOW

■ 1. The authority citation for part 372 continues to read as follows:

Authority: 42 U.S.C. 11023 and 11048.

■ 2. In § 372.28, amend the table in paragraph (a)(1) by:

■ a. Revising the third column header to read ''Reporting threshold (in pounds),'' and

■ b. Adding the chemical ''1,3,4,6,7,8-Hexahydro-4,6,6,7,8,8-hexamethylcyclopenta[g]-2-benzopyran'' in alphabetical order.

The revision and addition read as follows:

§ 372.28 Lower thresholds for chemicals of special concern.

(a) * * *

(1) * * *

TABLE TO PARAGRAPH (a) (1)

Chemical name	CAS No.	Reporting threshold (in pounds)
* * * * * * *		
1,3,4,6,7,8-Hexahydro-4,6,6,7,8,8-hexamethylcyclopenta[g]-2- benzopyran	1222–05–5	100
* * * * * * *		

* * * * *

■ 3. Amend § 372.65 by:

■ a. Adding new entries in alphabetical order in table 1 to paragraph (a) for ''Dibutyltin dichloride,'' ''1,3-Dichloro-2-propanol,'' ''Formamide,'' ''1,3,4,6,7,8-Hexahydro-4,6,6,7,8,8-hexamethylcyclopenta[g]-2-benzopyran,'' ''N-Hydroxyethylethylenediamine,'' ''Nitrilotriacetic acid trisodium salt,'' ''p-(1,1,3,3-Tetramethylbutyl)phenol,'' ''1,2,3-Trichlorobenzene,'' ''Triglycidyl isocyanurate,'' ''Tris(2-chloroethyl) phosphate,'' ''Tris(1,3-dichloro-2-propyl) phosphate,'' and ''Tris(dimethylphenol) phosphate''; and

■ b. Adding new entries in alphabetical order in the table 2 to paragraph (b) for ''Formamide,'' ''1,2,3-Trichlorobenzene,'' ''1,3-Dichloro-2-propanol,'' ''N-Hydroxyethylethylenediamine,'' ''Tris(2-chloroethyl) phosphate,'' ''p-(1,1,3,3-Tetramethylbutyl)phenol,'' ''Dibutyltin dichloride,'' ''1,3,4,6,7,8-Hexahydro-4,6,6,7,8,8-hexamethylcyclopenta[g]-2-benzopyran,'' ''Triglycidyl isocyanurate,'' ''Nitrilotriacetic acid trisodium salt,'' ''Tris(1,3-dichloro-2-propyl) phosphate,'' and ''Tris(dimethylphenol) phosphate''.

The additions read as follows:

§ 372.65 Chemicals and chemical categories to which this part applies.

* * * * *

(a) * * *

TABLE 1 TO PARAGRAPH (a)

Chemical name	CAS No.	Effective date
* * * * * * *		
Dibutyltin dichloride	683–18–1	1/1/23
* * * * * * *		
1,3-Dichloro-2-propanol	96–23–1	1/1/23
* * * * * * *		
Formamide	75–12–7	1/1/23
* * * * * * *		
1,3,4,6,7,8-Hexahydro-4,6,6,7,8,8-hexamethylcyclopenta[g]-2- benzopyran	1222–05–5	1/1/23
* * * * * * *		
N-Hydroxyethylethylenediamine	111–41–1	1/1/23
* * * * * * *		
Nitrilotriacetic acid trisodium salt	5064–31–3	1/1/23
* * * * * * *		
p-(1,1,3,3-Tetramethylbutyl)phenol	140–66–9	1/1/23

TABLE 1 TO PARAGRAPH (a)—Continued

Chemical name	CAS No.	Effective date
* * * * * * *		
1,2,3-Trichlorobenzene	87–61–6	1/1/23
* * * * * * *		
Triglycidyl isocyanurate	2451–62–9	1/1/23
* * * * * * *		
Tris(2-chloroethyl) phosphate	115–96–8	1/1/23
* * * * * * *		
Tris(1,3-dichloro-2-propyl) phosphate	13674–87–8	1/1/23
* * * * * * *		
Tris(dimethylphenol) phosphate	25155–23–1	1/1/23
* * * * * * *		

* * * * * (b) * * *

TABLE 2 TO PARAGRAPH (b)

CAS No.	Chemical name	Effective date
*	* * * * * *	
75–12–7	Formamide	1/1/23
*	* * * * * *	
87–61–6	1,2,3-Trichlorobenzene	1/1/23
*	* * * * * *	
96–23–1	1,3-Dichloro-2-propanol	1/1/23
*	* * * * * *	
111–41–1	N-Hydroxyethylethylenediamine	1/1/23
*	* * * * * *	
115–96–8	Tris(2-chloroethyl) phosphate	1/1/23
*	* * * * * *	
140–66–9	p-(1,1,3,3-Tetramethylbutyl)phenol	1/1/23
*	* * * * * *	
683–18–1	Dibutyltin dichloride	1/1/23
*	* * * * * *	
1222–05–5	1,3,4,6,7,8-Hexahydro-4,6,6,7,8,8-hexamethylcyclopenta[g]-2- benzopyran	1/1/23
*	* * * * * *	
2451–62–9	Triglycidyl isocyanurate	1/1/23
*	* * * * * *	
5064–31–3	Nitrilotriacetic acid trisodium salt	1/1/23
*	* * * * * *	
13674–87–8	Tris(1,3-dichloro-2-propyl) phosphate	1/1/23
*	* * * * * *	
25155–23–1	Tris(dimethylphenol) phosphate	1/1/23
*	* * * * * *	

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DEPARTMENT OF COMMERCE

National Oceanic and Atmospheric Administration

50 CFR Part 622

[Docket No. 211006–0204]

RIN 0648–BK36

Fisheries of the Caribbean, Gulf of Mexico, and South Atlantic; Reef Fish Fishery of the Gulf of Mexico; Lane Snapper Management Measures and Proposed Rule

AGENCY: National Marine Fisheries Service (NMFS), National Oceanic and Atmospheric Administration (NOAA), Commerce.

ACTION: Proposed rule; request for comments.

SUMMARY: NMFS proposes to implement management measures described in a framework action to the Fishery Management Plan for the Reef Fish Resources of the Gulf of Mexico (FMP) as prepared by the Gulf of Mexico Fishery Management Council (Council). For Gulf of Mexico (Gulf) lane snapper, this proposed rule would modify the annual catch limit (ACL) and revise an accountability measure (AM). The purposes of this proposed rule are to prevent overfishing of lane snapper and achieve optimum yield (OY). This proposed rule would also make minor administrative changes to replace outdated NMFS website addresses and language about required software for the Individual Fishing Quota (IFQ) programs.

DATES: Written comments must be received by November 2, 2021.

ADDRESSES: You may submit comments on the proposed rule identified by ''NOAA–NMFS–2021–0073'' by any of the following methods:

- *Electronic Submission:* Submit all electronic public comments via the Federal e-Rulemaking Portal. Go to *www.regulations.gov* and enter [NOAA–NMFS–2021–0073] in the Search box. Click on the ''Comment'' icon, complete the required fields, and enter or attach your comments.
- *Mail:* Submit all written comments to Dan Luers, NMFS Southeast Regional Office, 263 13th Avenue South, St. Petersburg, FL 33701.

Instructions: Comments sent by any other method, to any other address or individual, or received after the end of the comment period, may not be considered by NMFS. All comments received are a part of the public record and will generally be posted for public viewing on *www.regulations.gov* without change. All personal identifying information (*e.g.,* name, address), confidential business information, or otherwise sensitive information submitted voluntarily by the sender will be publicly accessible. NMFS will accept anonymous comments (enter ''N/A'' in the required fields if you wish to remain anonymous).

Electronic copies of the framework action, which includes an environmental assessment, a fishery impact statement, a Regulatory Flexibility Act analysis, and a regulatory impact review, may be obtained from the Southeast Regional Office website at *https://www.fisheries.noaa.gov/action/.*

FOR FURTHER INFORMATION CONTACT: Dan Luers, NMFS Southeast Regional Office, telephone: 727–824–5305, email: *daniel.luers@noaa.gov.*

SUPPLEMENTARY INFORMATION: NMFS and the Council manage the Gulf reef fish fishery, which includes lane snapper, under the FMP. The Council prepared the FMP and NMFS implements the FMP through regulations at 50 CFR part 622 under the authority of the Magnuson-Stevens Fishery Conservation and Management Act (Magnuson-Stevens Act).

Background

The Magnuson-Stevens Act requires NMFS and regional fishery management councils to prevent overfishing and achieve, on a continuing basis, the OY from federally managed fish stocks. These mandates are intended to ensure fishery resources are managed for the greatest overall benefit to the nation, particularly with respect to providing food production and recreational opportunities, and protecting marine ecosystems.

Unless otherwise noted, all weights in this proposed rule are in round weight.

Lane snapper occur in estuaries and shelf waters of the Gulf, and are particularly abundant off south and southwest Florida. Lane snapper in the Gulf exclusive economic zone (EEZ) are managed as a single stock with a stock ACL of 301,000 lb (136,531 kg) that was implemented in 2012 (76 FR 82044; December 29, 2011). This stock ACL is based on average landings from 1999 through 2008. The Council has also established a stock annual catch target (ACT) that is set 14 percent below the ACL, at 259,000 lb (117,480 kg), but the ACT is not codified in the regulations and is not linked to any specific management action, such as a closure. The fishing season is open year-round, January 1 through December 31. However, the current AM for lane snapper specifies that if combined commercial and recreational landings exceed the stock ACL in a fishing year, then during the following fishing year, if the stock ACL is reached or is projected to be reached, the commercial and recreational sectors will be closed for the remainder of the fishing year.

In 2016, a Southeast Data, Assessment, and Review (SEDAR) stock assessment (SEDAR 49 2016, ''SEDAR 49'') was completed for lane snapper and determined that the size of the lane snapper stock was similar to previous estimates. The Council's Scientific and Statistical Committee (SSC) reviewed SEDAR 49, accepted the assessment as the best scientific information available, and made recommendations to the Council to revise the catch limits. However, because the catch limits based on SEDAR 49 were similar to the established catch limits, the Council decided not to act on the SSC's recommendation.

As described in this framework action, Gulf lane snapper landings exceeded the stock ACL each year from 2016 through 2019. In 2017, NMFS notified the Council that landings in 2017 exceeded the overfishing limit (OFL), resulting in overfishing. Subsequently, NMFS estimated that 2018 landings did not exceed the 2018 OFL, but did exceed the ACL, and that a closure would be needed in 2019 should the ACL be projected to be met. On December 13, 2019, NMFS closed fishing for lane snapper for the remainder of the year based on a projection that the ACL would be caught. Despite this closure, the ACL was exceeded in 2019. Review of recent landings data indicate this ACL was also exceeded in 2020.

In 2019, in response to landings data that indicated lane snapper experienced overfishing in 2017 and exceeded its ACL in 2018, the Council requested that the NMFS Southeast Fisheries Science Center provide an updated, interim analysis to include landings data from 2015–2018 (SEDAR 49 Update 2019). However, the updated analysis used recreational catch estimate values that were calculated using landings from the previous Marine Recreational Information Program (MRIP) Coastal Household Telephone Survey rather than the newer MRIP Fishing Effort Survey (FES). Thus, the Council's SSC requested that the recreational data used to calculate the estimated catch limits for lane snapper be converted to values directly comparable to those collected