BILLING CODE 6560-50-P

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 372

[EPA-HQ-TRI-2015-0607; FRL-9943-55]

RIN 2025-AA42

Addition of Hexabromocyclododecane (HBCD) Category; Community Right-to-Know Toxic Chemical Release Reporting

AGENCY: Environmental Protection Agency (EPA).

ACTION: Proposed rule.

SUMMARY: EPA is proposing to add a hexabromocyclododecane (HBCD) category to the list of toxic chemicals subject to reporting under section 313 of the Emergency Planning and Community Right-to-Know Act (EPCRA) and section 6607 of the Pollution Prevention Act (PPA). EPA is proposing to add this chemical category to the EPCRA section 313 list because EPA believes HBCD meets the EPCRA section 313(d)(2)(B) and (C) toxicity criteria. Specifically, EPA believes that HBCD can reasonably be anticipated to cause developmental and reproductive effects in humans and is highly toxic to aquatic and terrestrial organisms. In addition, based on the available bioaccumulation and persistence data, EPA believes that HBCD should be classified as a persistent, bioaccumulative, and toxic (PBT) chemical and assigned a 100-pound reporting threshold. Based on a review of the available production and use information, members of the HBCD category are expected to be manufactured, processed, or otherwise used in quantities that would exceed a 100-pound EPCRA section 313 reporting threshold.

DATES: Comments must be received on or before **[***insert date 60 days after date of*

publication in the **Federal Register].**

ADDRESSES: Submit your comments, identified by Docket ID No. EPA-HQ-TRI-2015-0607, by one of the following methods:

• *Federal eRulemaking Portal: 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.

• *Mail:* Document Control Office (7407M), Office of Pollution Prevention and Toxics (OPPT), Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460-0001.

• *Hand Delivery:* To make special arrangements for hand delivery or delivery of boxed information, please follow the instructions at *http://www.epa.gov/dockets/where-send-comments-epa-dockets#hq.*

Additional instructions on commenting or visiting the docket, along with more information about dockets generally, is available at *http://www.epa.gov/dockets/commenting-epa-dockets*.

FOR FURTHER INFORMATION CONTACT: *For technical information contact:* Daniel R. Bushman, Toxics Release Inventory Program Division (7409M), 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) 412-9810 in Virginia and Alaska; or toll free, TDD (800) 553-7672; or go to

http://www.epa.gov/superfund/contacts/infocenter/.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this notice apply to me?

You may be potentially affected by this action if you manufacture, process, or otherwise use HBCD. 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 entities 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*, 211112*, 212324*, 212325*, 212393*, 212399*, 488390*, 511110, 511120, 511130, 511140*, 511191, 511199, 512220, 512230*, 519130*, 541712*, 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, 212231, 212234, 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?

EPA is proposing to add a hexabromocyclododecane (HBCD) category to the list of toxic chemicals subject to reporting under EPCRA section 313 and PPA section 6607. As discussed in more detail later in this document, EPA is proposing to add this chemical category to the EPCRA section 313 list because EPA believes HBCD meets the EPCRA section 313(d)(2)(B) and (C) toxicity criteria.

C. What is the Agency's Authority for Taking this Action?

This action is issued under EPCRA sections 313(d) and 328, 42 U.S.C. 11023 *et seq.*, and PPA section 6607, 42 U.S.C. 13106. EPCRA is also referred to as Title III of the Superfund Amendments and Reauthorization Act of 1986.

Section 313 of EPCRA, 42 U.S.C. 11023, requires certain facilities that manufacture, process, or otherwise use listed toxic chemicals in amounts above reporting threshold levels

to report their environmental releases and other waste management quantities of such chemicals annually. These facilities must also report pollution prevention and recycling data for such chemicals, pursuant to section 6607 of the PPA, 42 U.S.C. 13106. Congress established an initial list of toxic chemicals that comprised 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 demonstrate 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 demonstrate that none of the following listing criteria in EPCRA section 313(d)(2)(A)-(C) are met:

• 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."

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. Background Information

A. What is HBCD?

HBCD is a cyclic aliphatic hydrocarbon consisting of a 12-membered carbon ring with 6 bromine atoms attached (molecular formula $C_{12}H_{18}Br_6$). HBCD has 16 possible stereoisomers. Technical grades of HBCD consist predominantly of three diastereomers, α-, ß- and γ-HBCD (Ref. 1). HBCD may be designated as a non-specific mixture of all isomers (hexabromocyclododecane, Chemical Abstracts Service Registry Number (CASRN) 25637-99-4) or as a mixture of the three main diastereomers (1,2,5,6,9,10-hexabromocyclododecane, CASRN 3194-55-6) (Ref 1). The main use of HBCD is as a flame retardant in expanded polystyrene foam (EPS) and extruded polystyrene foam (XPS) (Ref. 2). EPS and XPS are used primarily for thermal insulation boards in the building and construction industry. HBCD may also be used as a flame retardant in textiles including: upholstered furniture, upholstery seating in transportation vehicles, draperies, wall coverings, mattress ticking, and interior textiles, such as roller blinds (Ref. 2). In addition, HBCD is used as a flame retardant in high-impact polystyrene for electrical and electronic appliances

such as audio-visual equipment, as well as for some wire and cable applications (Ref. 2).

Concerns for releases and uses of HBCD have been raised because it is found world-wide in the environment and wildlife and has also been found in human breast milk, adipose tissue and blood (Ref. 1). HBCD is known to bioaccumulate and biomagnify in the food chain and has been detected over large areas and in remote locations in environmental monitoring studies (Ref. 1).

B. How is EPA proposing to list HBCD under EPCRA section 313?

HBCD is identified through two primary CASRNs 3194-55-6 (1,2,5,6,9,10-hexabromocyclododecane) and 25637-99-4 (hexabromocyclododecane) (Ref. 1). EPA is proposing to create an HBCD category that would cover these two chemical names and CASRNs. The HBCD category would be defined as: Hexabromocyclododecane and would only include those chemicals covered by the following CAS numbers:

- 3194-55-6; 1,2,5,6,9,10-Hexabromocyclododecane
- 25637-99-4; Hexabromocyclododecane.

As a category, facilities that manufacture, process or otherwise use HBCD covered under both of these names and CASRNs would file just one report.

In addition to listing HBCD as a category, EPA is proposing to add the HBCD category 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. Chemicals meeting the PBT criteria were assigned 100-pound reporting thresholds. With regards to setting the EPCRA section 313 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, these chemicals were considered highly PBT chemicals. The data presented in this proposed rule support classifying the HBCD category as a PBT chemical category with a 100-pound reporting threshold.

III. What is EPA's evaluation of the toxicity, bioaccumulation, and environmental persistence of HBCD?

EPA evaluated the available literature on the human health toxicity, ecological toxicity, bioaccumulation potential, and environmental persistence of HBCD (Ref. 1). Unit III.A. provides a review of the human health toxicity studies and EPA's conclusions regarding the human health hazard potential of HBCD. Unit III.B. discusses the ecological toxicity of HBCD, Unit III.C. contains information on the bioaccumulation potential of HBCD, and Unit III.D. provides information on the environmental persistence of HBCD.

A. What is EPA's review of the human health toxicity data for HBCD?

1. *Toxicokinetics.* HBCD is absorbed via the gastrointestinal tract and metabolized in rodents (Refs. 3, 4, 5, and 6). Once absorbed, HBCD is distributed to a number of tissues, including fatty tissue, muscle, and the liver (Refs. 7, 8, 9, 10, 11, and 12). Elimination of HBCD is predominantly via feces (as the parent compound), but it is also eliminated in urine (as secondary metabolites) (Refs. 3, 4, and 5). HBCD has been detected in human milk,

adipose tissue, and blood (Refs. 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, and 24). The composition of HBCD isomers in most rodent toxicity studies resembles that of industrial grade HBCD, which may differ from human exposure to certain foods that have been shown to contain elevated fractions of α-HBCD (Ref. 25).

2. *Effects of acute exposure.* HBCD was not found to be highly toxic in acute oral, inhalation, and dermal studies in rodents. One study reported an oral median lethal dose (LD_{50}) of >10,000 milligrams per kilogram (mg/kg) in Charles River rats (Ref. 26). Another study by the same researchers, however, reported an LD_{50} of 680 mg/kg for females and 1,258 mg/kg for males in Charles River CD rats (Ref. 27). Two other studies reported an oral LD_{50} of >5,000 mg/kg in Sprague-Dawley rats and >10,000 mg/kg in NR rats (Refs. 28 and 29). An oral study in NR mice reported an LD_{50} of >6,400 mg/kg (Ref. 30). Acute inhalation studies in rats have generally concluded that HCBD is not highly toxic, with a median lethal concentration (LC_{50}) reported by Gulf South Research Institute of >200 milligrams per liter (mg/L) (Refs. 26, 27, 29, 31). Acute dermal toxicity studies have generally shown HBCD not to be highly toxic in rabbits (Refs. 27, 29, 31, and 32). One dermal study reported an LD_{50} of 3,969 mg/kg (Ref. 27). Additionally, HBCD is not a dermal irritant in rabbits (Refs. 27, 29, and 31), but it is a mild skin allergen in guinea pigs (Ref. 32). Acute eye irritation studies have concluded that HBCD is a primary eye irritant (Ref. 27) and a mild, transient ocular irritant (Ref. 29).

3. *Effects of short-term and subchronic exposure.* In subacute and subchronic studies, HBCD demonstrated effects on the thyroid and liver (Refs. 8, 33, 34, and 35). In a subacute study, van der Ven *et al*. (Ref. 8) exposed Wistar rats (5/sex/dose) by gavage to a mixture of HBCD dissolved in corn oil at concentrations resulting in doses of 0.3, 1.0, 3.0, 10, 30, 100,

and 200 milligrams per kilogram per day (mg/kg/day) for 28 days. The isomeric composition of the HBCD was 10.3% α, 8.7% β, and 81.0% γ. The authors reported a benchmark dose lower bound confidence limit (BMDL) of 29.9 mg/kg/day for an increase in pituitary weight, a BMDL of 1.6 mg/kg/day for an increase in thyroid weight, and a BMDL of 22.9 mg/kg/day for an increase in liver weight. The increase in thyroid weight was the most sensitive end point observed and, according to research by EPA, is considered relevant to humans (Ref. 36). Additionally, histopathology of the thyroid demonstrated that thyroid follicles were smaller, depleted, and had hypertrophied epithelium in female rats.

In another subacute study, HBCD was administered orally by gavage in corn oil to Sprague-Dawley Crl:CD BR rats for 28 days at doses of 0, 125, 350, or 1,000 mg/kg/day (6 rats/sex/dose in 125 and 350 mg/kg/day groups and 12 rats/sex/dose in the control and 1,000 mg/kg/day groups) (Ref. 33). At the end of 28 days, 6 rats/sex/dose were necropsied, while the remaining rats in the control and 1,000 mg/kg/day groups were untreated for a 14-day recovery period prior to necropsy. The authors reported increased absolute and liver to body weight ratios in females, but the authors considered the findings to be adaptive and not adverse. This study also identified a no-observed-adverse-effect level (NOAEL) of 1,000 mg/kg/day.

In an older subacute study (Ref. 37), an HBCD product was administered to Sprague-Dawley rat (10/sex/group) at doses of 0, 1, 2.5, and 5% of the diet for 28 days. Doses were calculated to be 0, 940, 2,410, 4,820 mg/kg/day. Mean liver weight (both absolute and relative) was increased in all dose groups, but no microscopic pathology was detected. Thyroid hyperplasia was observed in some animals at all doses in addition to slight numerical development of the follicles and ripening follicles in the ovaries at the high dose. The authors

concluded that these observed effects were not pathologic and reported a NOAEL of 940 mg/kg/day (Ref. 37).

In a subchronic study, Chengelis (Refs. 34 and 35) administered HBCD by oral gavage in corn oil daily to Crl:CD(SD)IGS BR rats (15/sex/dose) at dose levels of 0, 100, 300, or 1,000 mg/kg/day for 90 days. At the end of 90 days, 10 rats/sex/dose were necropsied, while the remaining rats were untreated for a 28-day recovery period prior to necropsy. The authors reported significant treatment-related changes in rats, including decreased liver weight and histopathological changes, but the authors considered these changes mild, reversible, and adaptive. Decreased liver weight accompanied by the observed histopathological changes, however, can be considered an adverse effect. Therefore, EPA identified a lowest-observed-adverse-effect level (LOAEL) of 100 mg/kg/day based on these changes.

In an older subchronic study (Ref. 38) an HBCD product was administered to Sprague-Dawley rats (10/sex/group) at doses of 0, 0.16, 0.32, 0.64, and 1.28% of the diet for 90 days. Doses were calculated to be 0, 120, 240, 470, and 950 mg/kg/day. An increase in relative liver weight was observed and was accompanied by fatty accumulation. The pathology report concluded that although fat was visible microscopically in treated rats, the change was not accompanied by any pathology, and therefore could not be defined as "fatty liver." No histological changes were found in any other organ. The authors concluded that the increased liver weight and the fat deposits, both of which were largely reversible when administration of HBCD was stopped, were the result of a temporary increase in the activity of the liver. They identified a NOAEL of 950 mg/kg/day.

4. *Carcinogenicity.* No adequate studies were found evaluating the carcinogenicity of

HBCD in animals or humans. One non-guideline study (Ref. 39) was cited in the U.S. EPA's Flame Retardant Alternatives for Hexabromocyclododecane (HBCD): Final Report (Ref. 40), but this study was not adequate to draw conclusions regarding carcinogenicity.

5. *Developmental and reproductive toxicity.* The developmental and reproductive toxicity of HBCD have been investigated in several studies. In a 1-generation study that included additional immunological, endocrine and neurodevelopmental endpoints, van der Ven *et al.* (Ref. 9) exposed Wistar rats (10/sex/dose) to a composite mixture of technical-grade HBCD (10.3% α, 8.7% β, and 81.0% γ) in the diet at concentrations resulting in doses of 0.1, 0.3, 1.0, 3.0, 10, 30, or 100 mg/kg/day. In the highest dose group (100 mg/kg/day) body weight decreases of 7-36% in males and 10-20% in females were observed in first generation (F1) pups. The authors observed decreases in kidney and thymus weight in both F1 males and females. Decreases in testes, adrenal, prostate, heart, and brain weights in F1 males were also observed. No histopathological changes, however, were observed in any of these organs. Other developmental effects were observed, including: Immune system effects, indications of liver toxicity, and decreases in bone mineral density at very low doses (i.e., <1.3 mg/kg/day). The authors noted that the vehicle used (corn oil) may have affected some observations at higher doses, including: Increased mortality during lactation, decreased liver weight in males, decreased adrenal weight in females, decreased plasma cholesterol in females, and other immunological markers of toxicity. Increased anogenital distance was observed in males at 100 mg/kg on postnatal day (PND) 4, but not on PND 7 or 21. There was no effect on preputial separation. The time to vaginal opening was delayed in females at the 100 mg/kg dose. There were no effects of HBCD exposure on thyroid hormones triiodothyronine (T3) and thyroxine (T4) in either the parental or F1 animals. There were no

effects on thyroid weight or thyroid pathology in the F1 animals (parents were not examined). The most sensitive endpoints with valid benchmark dose (BMD)/BMDL ratios for female rats were decreased bone mineral density with a BMDL of 0.056 mg/kg/day (BMD of 0.18 mg/kg/day) at a benchmark response (BMR) of 10% and decreased concentrations of apolar retinoids in the liver with a BMDL of 1.3 mg/kg/day (BMD = 5.1 mg/kg/day) at a BMR of 10%. The most sensitive endpoint with a valid BMD/BMDL ratio for male rats was an increased IgG response to sheep red blood cells with a BMDL of 0.46 mg/kg/day (BMD = 1.45 mg/kg/day) at a BMR of 20%. There were no significant effects of HBCD exposure on any measure of reproduction, including: Mating success, time to gestation, duration of gestation, number of implantation sites, pup mortality (at birth and throughout lactation), or sex ratios within a litter. Therefore, a BMDL for reproductive toxicity could not be derived for this study.

Saegusa *et al.* (Ref. 41) exposed pregnant Sprague-Dawley rats (10/sex/dose) to HBCD from gestation day 10 until PND 20 at dietary concentrations of 0, 100, 1,000, or 10,000 parts per million (ppm) in a soy-free diet. The authors observed increased relative thyroid weight and decreased T3 levels in F1 male Sprague-Dawley rats at postnatal week (PNW) 11 following dietary exposure to 1,000 ppm (approximately 146.3 mg/kg/day) HBCD. The authors also reported a significant reduction in the number of CNPase-positive oligodendrocytes at 10,000 ppm (approximately 1,504.8 mg/kg/day). EPA identified a maternal LOAEL of 10,000 ppm (about 1,504.8 mg/kg/day) based on increased incidence of thyroid follicular cell hypertrophy, and a developmental LOAEL of 1,000 ppm (about 146.3 mg/kg/day) based on increased relative thyroid weight and decreased T3 levels in F1 males at PNW 11. Changes in reproductive endpoints (e.g., the number of implantation sites, live

offspring, sex ratio) were not observed. Therefore, a LOAEL for reproductive toxicity could not be determined for this study.

Ema *et al.* (Ref. 42) administered HBCD to groups of male and female Crl:CD(SD) rats (24/sex/dose, as a mixture of α-HBCD, β -HBCD, and γ-HBCD with proportions of 8.5, 7.9, and 83.7%, respectively) in the diet at concentrations of 0, 150, 1,500, or 15,000 ppm from 10 weeks prior to mating through mating, gestation, and lactation. The authors reported a decrease in the number of primordial follicles in F1 female rats at 1,500 ppm (approximately 138 mg/kg/day) and a significant increase in the number of litters lost in the F1 generation at 15,000 ppm (approximately 1,363 mg/kg/day). These authors reported no other significant treatment-related effects in any generation for indicators of reproductive health, including: Estrous cyclicity, sperm count and morphology, copulation index, fertility index, gestation index, delivery index, gestation length, number of pups delivered, number of litters, or sex ratios. The authors reported a reduced viability index on day 4 and day 21 of lactation among second generation (F2) offspring at 15,000 ppm (approximately 1,363 mg/kg/day). They observed additional developmental effects at doses as low as 1,500 ppm (approximately 115 and 138 mg/kg/day for F1 males and females, respectively), including: An increase in dihydrotestosterone (DHT) in F1 males and an increased incidence of animals with decreased thyroid follicle size in both sexes and generations. These authors reported no effects on sexual development indicated by anogenital distance, vaginal opening, or preputial separation among F1 or F2 generations. The percentage of pups with completed eye opening on PND 14 was significantly decreased compared to controls in F2 females at 1,500 ppm and in F2 males and females at 15,000 ppm. Fewer F2 females exposed to 15,000 ppm HBCD completed the mid-air righting reflex (76.9%) than control F2 females (100%). These

findings were not consistent over generations or sexes and were not considered treatment related. No other effects of HBCD exposure on the development of reflexes were observed in either F1 or F2 progeny. EPA identified a maternal LOAEL of 150 ppm (about 14 mg/kg/day) based on increased thyroid-stimulating hormone (TSH). A reproductive LOAEL of 1,500 ppm (about 138 mg/kg/day) was identified based on a decreased number of primordial follicles in the ovary observed in F1 females. A developmental LOAEL of 15,000 ppm (about 1,142 mg/kg/day for males and 1,363 mg/kg/day for females) was identified based on increased pup mortality during lactation in the F2 generation.

Murai *et al.* (Ref. 43) fed female Wistar rats HBCD in the diet at concentrations of 0, 0.01, 0.1, or 1% throughout gestation (Days 0 – 20). Dams in the high-dose group demonstrated a statistically significant decrease (8.4%) in food consumption and increase in liver weight (13%) in comparison with controls. There were no treatment-related effects on maternal or fetal body weight. There were no effects on the number of implants; number of resorbed, dead, or live fetuses; body weight of live fetuses; or incidence of external or visceral abnormalities. A few skeletal variations were present but were also observed in controls and not considered significant. There were no effects on weaning or survival. The European Commission (Ref. 44) used the study's data to calculate the doses to be 0, 7.5, 75, and 750 mg/kg/day (based on the assumption of a mean animal weight of 200 grams (g) and food consumption of 15 g/day). They concluded that the offspring NOAEL was 750 mg/kg/day and the maternal LOAEL was 750 mg/kg/day based on a 13% liver weight increase in the high dose group.

Eriksson *et al.* (Ref. 45) conducted a study that examined behavior, learning, and memory in adult mice following exposure to HBCD on PND 10. The authors administered a

single oral dose of HBCD (mixture of, α-, β-, and γ-diastereoisomers) dissolved in a fat emulsion at 0, 0.9, or 13.5 mg/kg/day on PND 10 to male and female NMRI mice. The authors concluded that exposure on PND 10 affected spontaneous motor behavior, learning, and memory in adult mice in a dose-dependent manner. The authors identified the lowest exposure level, 0.9 mg/kg, as the LOAEL based on significantly reduced mean locomotor activity compared with controls during the first 20-minute interval of testing. EPA, however, identified a LOAEL of 13.5 mg/kg/day based on decreased habituation, locomotion, and rearing during all intervals. This study was not conducted according to current guidelines (Ref. 46) and Good Laboratory Practices; therefore, EPA reserves judgment on the significance of these findings.

6. *Genotoxicity.* A limited number of studies investigated the genotoxicity of HBCD. These studies indicate that HBCD is not likely to be genotoxic (Refs. 47, 48, 49, 50, 51, 52, 53, and 54).

7. Conclusions regarding the human hazard potential of HBCD. The available evidence indicates that HBCD has the potential to cause developmental and reproductive toxicity at moderately low to low doses. While there were some indications of liver toxicity in some short-term and subchronic studies, the evidence for these effects is not sufficient to support listing. The available evidence for developmental and reproductive toxicity, however, is sufficient to conclude that HBCD can be reasonably anticipated to cause moderately high to high chronic toxicity in humans based on the EPCRA section 313 listing criteria published in the **Federal Register** of November 30, 1994 (59 FR 61432) (FRL-4922-2).

B. What is EPA's review of the ecological toxicity of HBCD?

HBCD can cause effects on survival, growth, reproduction, development, and behavior in aquatic and terrestrial species. Observed acute toxicity values as low as 0.009 mg/L for a 72-hour EC_{50} (i.e., the concentration that is effective in producing a sublethal response in 50% of test organisms) based on reduced growth in the marine algae *Skeletonema costatum* (Ref. 55) indicate high acute aquatic toxicity. Observed chronic aquatic toxicity values as low as 0.0042 mg/L (maximum acceptable toxicant concentration (MATC)) for reduced size (length) of surviving young in water fleas (*Daphnia magna)* (Ref. 56) indicate high chronic aquatic toxicity. Reduced chick survival in Japanese quails (*Coturnix coturnix japonica*) fed a 15 parts per million (ppm) HBCD diet (2.1 mg/kg/day) (Ref. 57 as cited in Ref. 58) and altered reproductive behavior (reduced courtship and brood-rearing activity) and reduced egg size in American kestrels (*Falco sparverius*) fed 0.51 mg/kg/day (Refs. 59, 60, 61, and 62) indicate high toxicity to terrestrial species as well.

Assessment of HBCD's aquatic toxicity is complicated by its low water solubility and differences in the solubility of the three main HBCD isomers, which makes testing difficult and interpretation uncertain for studies conducted above the water solubility. Studies conducted at concentrations above the water solubility of HBCD are essentially testing the effects at the maximum HBCD concentration possible. In some acute and chronic aquatic toxicity studies conducted using methods, test species, and endpoints recommended by EPA, no effects were reported at or near the limit of water solubility. However, water solubility is not considered a limiting factor for hazard determination for aquatic species since there are studies showing adverse effects at or below the water solubility of HBCD. In addition, the potential for HBCD to bioaccumulate, biomagnify, and persist in the environment, significantly increases concerns for effects on aquatic organisms.

A wide range of effects of HBCD have been reported in fish (e.g., developmental toxicity, embryo malformations, reduced hatching success, reduced growth, hepatic enzyme and biomarker effects, thyroid effects, deoxyribonucleic acid (DNA) damage to erythrocytes, and oxidative damage) and in invertebrates (e.g., degenerative changes, morphological abnormalities, decreased hatching success, and altered enzyme activity) (Refs. 63, 64, 65, 66, 67, 68, 69, 70, 71, 72, 73, and 74). Reduced thyroid hormone (triiodothyronine, T3, and thyroxine, T4) levels in rainbow trout (*Oncorhynchus mykiss*) (Refs. 68 and 69), are similar to those observed in mammals. Reduced T4 levels were also reported in birds exposed to HBCD (Ref. 61).

1. *Acute aquatic toxicity.* Adverse effects observed following acute exposure were found in studies with marine algae, including EPA-recommended estuarine/marine algae species *Skeletonema costatum* (Ref. 75 as cited in Refs. 44 and 76, Refs. 55 and 77), a series of short-term (72 to 120-hour) early life stage tests with zebrafish (*Danio rerio*) embryos (Refs. 64, 65, 67, and 72), and short-term (72-hour) results from an early life stage test with sea urchin embryos (Ref. 63). Effects in these studies, reported at concentrations as low as 0.009 mg/L (measured) in algae, 0.01 mg/L (nominal) in zebrafish embryos, and 0.064 mg/L (nominal) in sea urchin embryos, indicate high acute toxicity. Walsh *et al.* (Ref. 55) reported measured 72-hour EC_{50} values in *Skeletonema costatum* ranging from 0.009 to 0.012 mg/L based on reduced growth rate in five different types of saltwater media (0.010 mg/L in seawater itself). The study tested two other marine algal species, *Chlorella sp.* and *Thalassiosira pseudonana*, that were also found to be inhibited by HBCD, albeit at higher concentrations than *Skeletonema costatum*. EC_{50} values for reduced growth in these species were 0.05 - 0.37 mg/L (0.08 mg/L in seawater) for *Thalassiosira pseudonana* and >1.5 mg/L

for *Chlorella sp*.

Subsequent studies by Desjardins *et al.* (Ref. 75) confirmed the high acute toxicity of HBCD to *Skeletonema costatum*. In these studies, single concentrations were tested, but the assays were conducted without solvent and the concentrations were measured. Desjardins *et al.* (Ref. 75) reported approximately 10% inhibition of growth in *Skeletonema costatum* exposed to 0.041 mg/L for 72 hours. Desjardins *et al.* (Ref. 77) found that a saturated solution of 0.0545 mg/L resulted in 51% growth inhibition after 72 hours of exposure. The latter result corresponds to an approximate EC_{50} of 0.052 mg/L.

Zebrafish embryo studies reported a variety of effects on embryos and larvae at low HBCD concentrations. In the Deng *et al.* (Ref. 64) study, developmental toxicity endpoints were assessed at 96 hours post-fertilization in embryos/larvae exposed to HBCD starting 4 hours post-fertilization. Survival of embryos/larvae was significantly reduced at all tested concentrations, making the low concentration of 0.05 mg/L the lowest-observed-effect-concentration (LOEC) in this study; a no-observed-effect-concentration (NOEC) was not established. Embryonic malformation rate was significantly increased and larval growth significantly decreased at ≥0.1 mg/L. Malformations included epiboly deformities, yolk sac and pericardial edema, tail and heart malformations, swim bladder inflation, and spinal curvature. Embryo hatching rate was reduced only at the high concentration of 1 mg/L. Heart rate, a marker for cardiac developmental toxicity, was significantly decreased at all tested concentrations. Associated mechanistic studies suggest the mechanism for developmental toxicity involves the generation of reactive oxygen species (ROS) and the consequent triggering of apoptosis genes. Increased ROS formation (indicative of oxidative stress) was observed at a nominal concentration of 0.1 mg/L. In the same study, zebrafish

embryos exposed to HBCD exhibited increased expression of pro-apoptotic genes (Bax, P53, Puma, Apaf-1, caspase 3, and caspase-9), decreased expression of anti-apoptotic genes (Mdm2 and Bcl-2), and increased activity of enzymes involved in apoptosis (caspase-3 and caspase-9) with LOECs of 0.05 - 1 mg/L.

Hu *et al.* (Ref. 67) found that hatching of zebrafish embryos was delayed at 0.002 mg/L, the lowest concentration tested, and other concentrations up to and including 0.5 mg/L, but not the two high concentrations of 2.5 and 10 mg/L. The same authors observed an increase in heat shock protein (Hsp70) at 0.01 mg/L and an increase in malondialdehyde activity, used as a measure of lipid peroxidation, at 0.5 mg/L. The activity of superoxide dismutase was increased at 0.1 mg/L, but decreased at 2.5 and 10 mg/L. The authors concluded that HBCD can cause oxidative stress and over expression of Hsp70 in acute exposures of zebrafish embryos.

Du *et al.* (Ref. 65) exposed zebrafish embryos 4 hours post-fertilization to each of three diastereomers of HBCD (α-, β-, and γ-HBCD) individually at nominal concentrations of 0.01, 0.1, and 1.0 mg/L. Hatching success was reduced after 68 hours of exposure to γ-HBCD at the lowest concentration (0.01 mg/L), but a higher concentration of α- or β-HBCD (0.1 mg/L) was necessary to reduce hatching success. After 92 hours, survival was reduced at concentrations of 0.01, 0.1, and 1 mg/L of γ-, β-, and α-HBCD, respectively. Growth, measured as body length of larvae after 92 hours of exposure, was reduced at 0.1 mg/L of β- and γ-HBCD and at 1 mg/L of α-HBCD. After 116 hours of exposure, malformations were observed at all test concentrations of β- and γ-HBCD and at 0.1 mg/L and above for α-HBCD. Effects on heart rate varied depending upon the length of exposure; reduced heart rate was observed at 0.1 mg/L of β- and γ-HBCD or 1 mg/L of α-HBCD at 44 hours and at

0.1 mg/L of α- and β-HBCD at 92 hours, whereas γ HBCD resulted in an increase in heart rate at 1 mg/L at 92 hours. An increase in generation of ROS was observed after 116 hours at 0.1 mg/L of β- and γ-HBCD and at 1 mg/L of α-HBCD. Activities of caspase-3 and caspase-9 enzymes, indicative of apoptosis, were increased after 116 hours at 0.1 mg/L of γ-HBCD and at 1 mg/L of α- and β-HBCD. The authors ranked the HBCD diastereomers in the following order for developmental toxicity to zebrafish: γ HBCD > β HBCD > α-HBCD.

Effects indicative of oxidative stress, as seen in the zebrafish embryo studies, were also found in clams. Zhang *et al.* (Ref. 74) measured parameters indicative of antioxidant defenses and oxidative stress after 1, 3, 6, 10, and 15 days of exposure to low nominal concentrations of HBCD ranging from 0.000086 to 0.0086 mg/L in the clam *Venerupis philippinarum*. Increases in ethyoxyresorufin-o-deethylase (EROD) activity, glutathione (GSH) content, and DNA damage were observed in clams exposed to 0.00086 mg/L, while increased lipid peroxidation (LPO) was observed at 0.0086 mg/L. These same effects were observed at lower concentrations as the length of exposure increased.

Anselmo *et al.* (Ref. 63) exposed sea urchin (*Psammechinus miliaris*) embryos to HBCD in an early life stage test. Newly-fertilized embryos were exposed to HBCD at nominal concentrations of 0, 9, 25, 50, and 100 nanomolar (nM) (0, 0.0058, 0.016, 0.032, and 0.064 mg/L, respectively) in dimethyl sulfoxide solvent and evaluated at 72 hours post-fertilization. A significant increase in morphological abnormalities was found at a nominal concentration of 100 nM HBCD (0.064 mg/L), the highest concentration tested. Observed malformations included short or deformed larval arms and slight edema around the larval body. The NOEC for this effect at 72 hours was 0.032 mg/L.

2. *Chronic aquatic toxicity.* A measured MATC of 0.0042 mg/L, based on reduced

size (length) of surviving young water fleas (*Daphnia magna*), indicates high chronic toxicity (Ref. 56). This study reported additional effects, including decreased reproductive rate and decreased mean weight of surviving young at 0.011 mg/L. Other effects reported following chronic exposure to HBCD included degenerative changes in the gills of clams (*Macoma balthica*), manifested by the increased frequency of nuclear and nucleolar abnormalities and the occurrence of dead cells, at nominal concentrations of ≥0.1 mg/L (50-day LOEC) (Ref. 71), a nominal MATC of 0.045 mg/L for increased morphological abnormalities in sea urchin (*P. miliaris*) embryos exposed to HBCD for up to 16 days in an early life stage test (Ref. 63), and a nominal MATC of 0.03 mg/L for increased malformation rate in marine medaka (*Oryzias melastigma*) embryos exposed to HBCD for 17 days in an early life stage test (Ref. 66). The developmental abnormalities in medaka included yolk sac edema, pericardial edema, and spinal curvature (Ref. 66). Mechanistic findings in this study included increases in heart rate and sinus venosus-bulbus arteriosus (SV-BA) distance, which are markers for cardiac development, induction of oxidative stress and apoptosis, and suppression of nucleotide and protein synthesis.

Thyroid effects were reported in juvenile rainbow trout (*Oncorhynchus mykiss*) following dietary exposure to HBCD (Refs. 68 and 69). Each of the diastereomers of HBCD (administered separately via diet at concentrations of 5 ng/g of α-, β-, or γ-HBCD for up to 56 days) disrupted thyroid homeostasis, as indicated by lower free circulating T3 and T4 levels.

The mechanisms of the effects on fish and invertebrates following chronic exposure were similar to those found in acute studies. Effects observed in fish include increased formation of ROS resulting in oxidative damage to lipids, proteins, and DNA, decreased

antioxidant capacities in fish tissue (e.g., brains, hepatocytes, or erythrocytes), and increasing levels of EROD (detoxification enzyme) and PentoxyResorufin-O-Deethylase (PROD, detoxification enzyme) levels in hepatocytes of fish exposed to the nominal concentration of ≥0.1 mg/L (corresponds to ~0.2 mg/g whole fish (wet weight)) for 42 days (Ref. 73). Ronisz *et al.* (Ref. 70) found a significant increase in hepatic cytosolic catalase activity in rainbow trout (*Oncorhynchus mykiss*) 5 days after a single intraperitoneal injection of 50 mg/kg was administered. The same authors observed reductions in liver somatic index (LSI) and EROD activity in a 28-day study in which rainbow trout were injected intraperitoneally with HBCD on days 1 and 14 at a dose somewhat less than 500 mg/kg. Zhang *et al.* (Ref. 74) observed the following signs of oxidative stress in clams (*V. philippinarum*) after 15 days of exposure to HBCD: the activities of antioxidant enzymes (EROD, superoxide dismutase (SOD), and glutathione-S-transferase (GST)), as well as GSH content, were increased at 0.000086 mg/L, the lowest concentration tested. In addition, LPO was increased at 0.00086 mg/L and DNA damage was increased at 0.0086 mg/L.

3. *Terrestrial toxicity and phytotoxicity.* Japanese quail (*Coturnix coturnix japonica*) exposed for 6 weeks to an isomeric mixture of HBCD in the diet experienced a reduction in hatchability at all tested concentrations (12 - 1,000 ppm) (Ref. 57). Additional effects included a significant reduction in egg shell thickness starting at 125 ppm, decreases in egg weights and egg production rates starting at 500 ppm, increases in cracked eggs starting at 500 ppm, and adult mortality at 1,000 ppm. A subsequent test, conducted at lower dietary concentrations, determined LOAEL and NOAEL values of 15 and 5 ppm, respectively, based on significant reduction of survival of chicks hatched from eggs of quails fed HBCD (Ref. 57).

Several studies have been conducted examining effects of HBCD on American kestrels (*Falco sparverius*). Kobiliris (Ref. 78) reported a reduced "corticosterone response" (where "corticosterone response" was defined as a stimulation of the adrenal cortex to produce and release corticosterone into the bloodstream), reduced flying activities of juvenile males during hunting behavior trials, and delayed response times of juvenile females during predator avoidance behavior trials in American kestrels exposed in ovo to 164.13 ng/g wet weight. Kestrels exposed via the diet to 0.51 mg/kg/day beginning 3 weeks prior to pairing and continuing until the first chick hatched began to lay eggs 6 days earlier than controls and laid larger clutches of smaller eggs (Ref. 59). Although the technical mixture of HBCD stereoisomers contained predominantly γ-HBCD (80% of the mixture), the main isomer found in eggs was α-HBCD (>90% of the total HBCD in eggs). In a subsequent study, Marteinson *et al.* (Ref. 61) exposed kestrels to dietary HBCD at the same dose (0.51 mg/kg/day) and found increased testes weight in unpaired males, a marginally significant effect on testis histology in unpaired males (increased number of seminiferous tubules containing elongated spermatids; $p = 0.052$), marginally increased testosterone levels in breeding males (increased at the time the first egg was laid; $p = 0.054$), and no significant effect on sperm counts. Plasma T4 levels were reduced in breeding males throughout the study, which the authors took to suggest that thyroid disruption that may have contributed to the observed increase in testes weight. Marteinson *et al.* (Ref. 62) found altered reproductive behavior in both sexes of kestrels fed 0.51 mg/kg/day, including reduced activity in both sexes during courtship and in males during brood rearing, which may have contributed to the observed reduction in incubation nest temperature and also to the reduced egg size reported previously by Fernie *et al.* (Ref. 58). In a 22-day study of chickens (*Gallus gallus*

domesticus) exposed to HBCD in ovo, reduced pipping success was observed at 100 ng/g egg (Ref. 79).

The accumulation and toxicity of α-, β-, and γ-HBCDs in maize have been studied (Ref. 80). The order of accumulation in roots was β-HBCD > α-HBCD > γ-HBCD and in shoots it was β-HBCD > γ-HBCD > α-HBCD. In maize exposed to 2 μg/L HBCD, the inhibitory effect of the diastereomers on the early development of maize as well as the intensities of hydroxyl radical and histone H2AX phosphorylation followed the order α-HBCD > β-HBCD > γ-HBCD, which indicates diastereomer-specific oxidative stress and DNA damage in maize. The study confirmed that for maize exposed to HBCDs, the generation of reactive oxygen species was one, but not the only, mechanism for DNA damage.

4. *Conclusions regarding the ecological hazard potential of HBCD.* HBCD has been shown to cause acute toxicity to aquatic organisms at concentrations as low as 0.009 mg/L and chronic toxicity at concentrations as low as 0.0042 mg/L. Toxicity to terrestrial species has been observed at doses as low as 0.51 mg/kg/day. The available evidence shows that HBCD is highly toxic to aquatic and terrestrial species.

C. What is EPA's review of the bioaccumulation data for HBCD?

HBCD has been shown in numerous studies to bioaccumulate in aquatic species and biomagnify in aquatic and terrestrial food chains (Ref. 1). BCFs for HBCD in fish in the peer-reviewed literature range as high as 18,100 (Refs. 81, 82, and 83). Some of the bioaccumulation values for fish species and a freshwater food web are shown in Table 1. The complete listing of the available bioaccumulation data and more details about the studies can be found in the ecological assessment (Ref. 1).

Table 1. HBCD BCF and BAF Data for Fish and Freshwater Food Web

Species	Duration and test endpoint	Value	Reference
Rainbow trout (*Oncorhynchus mykiss*)	35-day BCF	8,974 and 13,085	Ref. 81
Fathead minnow (*Pimephales promelas*)	32-day BCF	18,100	Ref. 82
Mirror carp (*Cyprinus carpio morpha noblis*)	30-day exposure and 30-day depuration BCF	α-HBCD: 5,570 – 11,500 β-HBCD: 187 – 642 γ-HBCD: 221 – 584	Ref. 83
Mud carp (*Cirrhinus molitorella*), nile tilapia (*Tilapia nilotica*), and suckermouth catfish (*Hypostomus plecostomus*)	Log BAF	4.8 – 7.7 for HBCD isomers (α-HBCD had higher BAFs than β- and γ-HBCD) (BAFs ranged from ~63,000 to 50,000,000)	Ref. 84
Freshwater food web	Log BAF	α-HBCD: 2.58 – 6.01 β-HBCD: 3.24 – 5.58 γ-HBCD: 3.44 – 5.98 ΣHBCDs: 2.85 – 5.98 (BAFs range from ~700 to 950,000)	Ref. 85

Drottar and Kruger (Ref. 81) provided strong evidence that HBCD bioaccumulates in a study conducted according to established guidelines (OECD Test Guideline (TG) 305 and Office of Prevention, Pesticides and Toxic Substances (OPPTS) 850.1730). In this study, BCFs of 13,085 and 8,974 were reported in rainbow trout (*O. mykiss*) exposed to 0.18 and 1.8 µg/L, respectively. Concentrations of HBCD in tissue reached steady-state at day 14 for fish exposed to 1.8 µg/L and, during the subsequent depuration stage, a 50% reduction of HBCD from edible and non-edible tissue and whole fish was reported on days 19 and 20 post-exposure. In fish exposed to 0.18 µg/L, an apparent steady-state was reached on day 21, but on day 35, the tissue concentration of HBCD in fish increased noticeably; thus, steady-

state was not achieved according to study authors, and BCF values (for the exposure concentration of 0.18 µg/L) were calculated based on day 35 tissue concentrations. Clearance of 50% HBCD from tissue of 0.18 µg/L exposed fish occurred 30-35 days post-exposure.

Veith *et al*. (Ref. 82) further supports the conclusion that HBCD bioaccumulates in a study conducted prior to the establishment of standardized testing guidelines for bioconcentration studies. The study reported a BCF of 18,100 following exposure of fathead minnows to 6.2 µg/L; the BCF was identified as a steady-state BCF, but the report does not indicate the time when steady-state was reached. A depuration phase was not included in this study. Zhang *et al*. (Ref. 83) calculated BCFs for each HBCD diastereomer in mirror carp and found strong evidence that α-HBCD (BCF of 5,570-11,500) is much more bioaccumulative than β- and γ-HBCD (BCF of 187-642); BCF values that were normalized to lipid content were much higher (30,700-45,200 for α-HBCD, 1,030-1,900 for β-HBCD, and 950-1,730 for γ-HBCD) than non-normalized BCFs.

BAFs, which capture accumulation of HBCD from diet as well as water and sediment, were calculated for freshwater food webs in industrialized areas of Southern China in two separate field studies. He *et al*. (Ref. 84) calculated log BAFs of 4.8-7.7 (corresponding to BAFs of 63,000-50,000,000) for HBCD isomers in carp, tilapia, and catfish, and found higher BAFs for α-HBCD than β- and γ-HBCD. In a pond near an e-waste recycling site, Wu *et al*. (Ref. 85) calculated log BAFs of 2.85 - 5.98 for ΣHBCD (corresponding to BAFs of 700-950,000) in a freshwater food web. Log BAFs for each diastereomer in this study were comparable to one another (see Table 1). La Guardia *et al*. (Ref. 86) calculated log BAFs in bivalves and gastropods collected downstream of a textile

manufacturing outfall; these ranged from 4.2 to 5.3 for α- and β-HBCD (BAFs of 16,000-200,000), and from 3.2 to 4.8 for γ-HBCD (BAFs of 1,600-63,000).

In general, α-HBCD bioaccumulates in organisms and biomagnifies through food webs to a greater extent than the β- and γ- diastereomers. Uncertainty remains as to the balance of diastereomer accumulation in various species and the extent to which bioisomerization and biotransformation rates for each isomer affect bioaccumulation potential. Some authors (e.g., Law *et al*., Ref. 87) have proposed that γ-HBCD isomerizes to α-HBCD under physiological conditions, rather than uptake being diastereisomer-specific. To test this theory, Esslinger *et al*. (Ref. 88) exposed mirror carp (*Cyprinus carpio morpha noblis*) to only γ-HBCD and found no evidence of bioisomerization. In contrast, when Du *et al*. (Ref. 89) exposed zebrafish (*Danio rerio*) to only γ-HBCD, they found detectable levels of α-HBCD in fish tissue, suggesting that bioisomerization occurred. Marvin *et al*. (Ref. 90) hypothesized that differences in accumulation could also be due in part to a combination of differences in solubility, bioavailability, and uptake and depuration kinetics.

Zhang *et al*. (Ref. 91) calculated diastereomer-specific BCFs in algae and cyanobacteria ranging from 174 to 469. For the cyanobacteria (*Spirulina subsalsa*), the BCF for α-HBCD (350) was higher than the BCFs for β-HBCD (270) and γ-HBCD (174). However, for the tested alga (*Scenedesmus obliquus*), the BCF for β-HBCD (469) was higher than that for the other isomers (390-407).

In summary, HBCD has been shown in numerous studies to be highly bioaccumulative in aquatic species and biomagnify in aquatic and terrestrial food chains; however, diastereomer- and enantiomer-specific mechanisms of accumulation are still unclear.

D. What is EPA's review of the persistence data for HBCD?

There are limited data available on the degradation rates of HBCD under environmental conditions. A short summary of the environmental fate and persistence data for HBCD is presented in Table 2; additional details about this data can be found in the HBCD hazard assessment (Ref. 1).

Table 2. Environmental Degradation of HBCD

Property	Value	Reference
Air		
Photodegra-dation	Photo-induced isomerization of γ-HBCD to α-HBCD in indoor dust with a measured decrease in HBCD concentration concurrent with an increase of pentabromocyclododecenes (PBCDs) in indoor dust	Ref. 92
	Indirect photolysis half-life: 26 hours AOPWIN v1.92 (estimated)	Ref. 93
Water		
Hydrolysis	Not expected due to lack of functional groups that hydrolyze under environmental conditions and low water solubility (estimated)	Ref. 44
Sediment		
Aerobic conditions	No biodegradation observed in 28-day closed-bottle test	Refs. 76 and 94
	Half-life: 128, 92, and 72 days for α-, γ-, and β-HBCD, respectively (estimated), based on a 44% decrease in total initial radioactivity in viable freshwater sediment	Ref. 95
	Half-life: >120 days (estimated), based on a 15% decrease in total initial radioactivity in abiotic freshwater sediment	
	Half-life: 11 and 32 days (estimated) in viable sediment collected from Schuylkill River and Neshaminy creek, respectively	Ref. 96
	Half-life: 190 and 30 days (estimated) in abiotic sediment collected from Schuylkill River and Neshaminy creek	
Anaerobic conditions	Half-life: 92 days (estimated), based on a 61% decrease in total initial radioactivity in viable freshwater sediment	Ref. 95
	Half-life: >120 days (estimated), based on a 33% decrease in total initial radioactivity in abiotic freshwater sediment	
	Half-life: 1.5 and 1.1 days (estimated) in viable sediment collected from Schuylkill River and Neshaminy creek	Ref. 96

	Half-life: 10 and 9.9 days (estimated) in abiotic sediment collected from Schuylkill River and Neshaminy creek	
Soil		
Aerobic conditions	Half-life: >120 days (estimated), based on a 10% decrease in total initial radioactivity in viable soil	Ref. 95
	Half-life: >120 days (estimated), based on a 6% decrease in total initial radioactivity in abiotic soil	
	Half-life: 63 days (estimated) in viable soil amended with activated sludge	Ref. 96
	Half-life: >120 days (estimated) in abiotic soil	
Anaerobic conditions	Half-life: 6.9 days (estimated) in viable soil amended with activated sludge	Ref. 96
	Half-life: 82 days (estimated) in abiotic soil using a nominal HBCD concentration of 0.025 mg/kg dry weight	

1. *Abiotic degradation*. HBCD is not expected to undergo significant direct photolysis since it does not absorb radiation in the environmentally available region of the electromagnetic spectrum that has the potential to cause molecular degradation (Ref. 97). Although HBCD is expected to exist primarily in the particulate phase in the atmosphere, a small percentage may also exist in the vapor phase based on its vapor pressure (Refs. 22, 90, 98, and 99). HBCD in the vapor phase will be degraded by reaction with photochemically produced hydroxyl radicals in the atmosphere. An estimated rate constant of 5.01×10^{-12} cubic centimeters (cm^3)/molecules-second at 25°C for this reaction corresponds to a half-life of 26 hours, assuming an atmospheric hydroxyl radical concentration of 1.5×10^6 molecules/cm^3 and a 12-hour day (Refs. 93 and 100).

Photolytic isomerization of HBCD has been described in both indoor dust samples and in samples of HBCD standards dissolved in methanol using artificial light (Ref. 92). After 1 week in the presence of light, indoor dust containing predominantly γ-HBCD was found to decrease in γ-HBCD and increase in α-HBCD concentration. There was a measured

decrease in HBCD concentration concurrent with an increase in PBCDs in the indoor dust exposed to artificial light. The three diastereomerically-pure HBCD standards (α-, β-, and γ-HBCD) that were dissolved in methanol also began to interconvert within 1 week, resulting in a decrease in γ-HBCD concentration and an increase in α-HBCD concentration.

HBCD is not expected to undergo hydrolysis in environmental waters due to lack of functional groups that hydrolyze under environmental conditions and the low water solubility of HBCD (Ref. 44).

Observed abiotic degradation of HBCD during simulation tests based on Organisation for Economic Cooperation and Development (OECD) methods 307 and 308 was approximately 33% in anaerobic freshwater sediment, 15% in aerobic freshwater sediment, and 6% in aerobic soil after 112-113 days (Refs. 44 and 95). The results from these studies correspond to estimated half-lives >120 days in soil and sediment due to minimal degradation being observed. Initial concentrations of ^{14}C radiolabeled HBCD (α-, β-, and γ- ^{14}C-HBCD in a ratio of 7.74:7.84:81.5) were 3.0-4.7 mg/kg dry weight in the sediment and soil systems. HBCD degradation observed under abiotic conditions was attributed to abiotic reductive dehalogenation (Refs. 44, 76, and 95). Degradation proceeded through a stepwise process to form tetrabromocyclododecene, dibromocyclododecadiene (DBCD), and 1,5,9-cyclododecatriene (Refs. 44 and 95). Further degradation of 1,5,9-cyclododecatriene was not observed. In this study, HBCD degradation occurred faster in sediment than in soil and faster under anaerobic conditions compared to aerobic conditions (Refs. 44 and 95).

Previous OECD 308 and 307 based simulation tests from the same authors (Davis *et al*. 2005, Ref. 96) presented results suggesting faster abiotic degradation, particularly in sediment under anaerobic conditions, but were performed at much lower HBCD

concentrations and measured only γ-HBCD (Refs. 44, 76, 90, 96, and 101). In this study, abiotic degradation half-lives in freshwater sediments were 30-190 days under aerobic conditions and 9.9-10 days under anaerobic conditions. Estimated half-lives in abiotic soil were >120 days under aerobic conditions and 82 days under anaerobic conditions. This study evaluated γ-HBCD only and did not address interconversion of HBCD isomers or α- and β-HBCD degradation. The initial concentrations of HBCD were 0.025-0.089 mg/kg dry weight in the sediment and soil systems, nearly 100 times less than the HBCD concentrations used in the subsequent Davis *et al*. 2006 study (Ref. 95). Higher concentrations of HBCD (3.0-4.7 mg/kg dry weight) in the Davis *et al*. 2006 study (Ref. 95) allowed for quantification of individual isomers, metabolite identification and mass balance evaluation (Refs. 95 and 101). Additionally, the Davis *et al*. 2005 study (Ref. 96) was considered to be of uncertain reliability for quantifying HBCD persistence because of concerns regarding potential contamination of sediment samples, an interfering peak corresponding to γ-HBCD in the liquid chromatography/mass spectrometry (LC/MS) chromatograms, and poor extraction of HBCD leading to HBCD recoveries of 33-125% (Refs. 44 and 101).

2. *Biotic degradation*. A few studies on the biodegradation of HBCD were located. A closed bottle screening-level test for ready biodegradability (OECD Guideline 301D, EPA OTS 796.3200) was performed using an initial HBCD concentration of 7.7 mg/L and an activated domestic sludge inoculum (Refs. 76 and 94). No biodegradation was observed (0% of the theoretical oxygen demand) over the test period of 28 days under the stringent guideline conditions of this test.

Degradation of HBCD during simulation tests with viable microbes, based on OECD methods 307 and 308, was approximately 61% in anaerobic freshwater sediment, 44% in

aerobic freshwater sediment, and 10% in aerobic soil after 112-113 days (Refs. 44 and 95). The results from this study correspond to estimated HBCD half-lives of 92 days in anaerobic freshwater sediment, 128, 92, and 72 days for α-, γ-, and β-HBCD, respectively in aerobic freshwater sediment, and >120 days in aerobic soil. An initial total ^{14}C-HBCD concentration of 3.0-4.7 mg/kg dry weight in the sediment and soil systems was used, allowing for quantification of individual isomers, metabolite identification, and mass balance evaluation (Refs. 95 and 101). Although very high spiking rates can be toxic to microorganisms in biodegradation studies and lead to unrealistically long estimated half-lives, the results of this study did not suggest toxicity to microorganisms. Tests with viable microbes demonstrated increased HBCD degradation compared to the biologically-inhibited control studies. In combination, these studies suggest that HBCD will degrade slowly in the environment, although faster in sediment than in soil, faster under anaerobic conditions than aerobic conditions, faster with microbial action than without microbial action, and at different rates for individual HBCD diastereomers (slower for α-HBCD than for the γ- and β-stereoisomers).

The same researchers (Ref. 76) previously conducted a water-sediment simulation test for commercial HBCD based on OECD guideline 308 using nominal HBCD concentrations of 0.034-0.089 mg/kg dry weight (Refs. 44, 76, and 102). Aerobic and anaerobic microcosms were pre-incubated at 20 °C for 49 days and at 23 °C for 43-44 days, respectively. HBCD was then added to 14-37 g dry weight freshwater sediment samples in 250 ml serum bottles (water:sediment ratio of 1.6-2.9) and the microcosms were sealed and incubated in the dark at 20 °C for up to 119 days. For the aerobic microcosms, the headspace oxygen concentration was kept above 10-15%. This study evaluated only γ-HBCD and did

not address interconversion of HBCD isomers or α- and β-HBCD degradation. Disappearance half-lives of HBCD with sediment collected from Schuylkill River and Neshaminy creek were 11 and 32 days in viable aerobic sediments, respectively (compared to 190 and 30 days in abiotic aerobic controls, respectively), and 1.5 and 1.1 days in viable anaerobic sediments, respectively (compared to 10 and 9.9 days in abiotic anaerobic controls).

Data from these tests suggest that anaerobic degradation is faster than aerobic degradation of HBCD in viable and abiotic sediments and that degradation is faster in viable conditions than abiotic conditions. While these findings are consistent with Davis *et al*. 2006 (Ref. 95), the actual degradation rates in this study are much faster. However, results from this study do not provide a reliable indication of HBCD persistence. A mass balance could not be established because only γ-HBCD was used to quantify HBCD concentrations, ^{14}C-radiolabelled HBCD was not used, and degradation products were not identified; therefore, apparent disappearance of HBCD in this study may not reflect biodegradation. In addition, there were concerns that contaminated sediment may have been used, HBCD extraction was incomplete (HBCD recovery varied from 33 to 125%), and an interfering peak was observed in the LC/MS chromatograms corresponding to γ-HBCD (Refs. 44 and 101).

Similarly, a soil simulation test was conducted based on OECD guideline 307 for commercial HBCD using 50 g dry weight sandy loam soil samples added to 250 ml serum bottles (Refs. 44, 76, 96, and 103). The moisture content was 20% by weight. Aerobic and anaerobic microcosms were pre-incubated at 20 °C for 35 days and at 23 °C for 43 days, respectively. Activated sludge was added to the soil at 5 mg/g, and HBCD was added to the soil to achieve a nominal concentration of 0.025 mg/kg dry weight. The microcosms were

then incubated in the dark at 20 °C for up to 120 days. The disappearance half-lives were 63 days in viable aerobic soil (compared to >120 days in abiotic aerobic controls) and 6.9 days in viable anaerobic soil (compared to 82 days in abiotic anaerobic controls). As in the sediment studies, HBCD degradation in soil occurred faster under anaerobic conditions compared to aerobic conditions, and faster in viable conditions than abiotic conditions. The disappearance half-lives in soil were slower than those in sediment.

Biological processes were suggested to be responsible for the increased degradation of HBCD in this study using viable conditions, relative to abiotic conditions; however, degradation was not adequately demonstrated in soil because no degradation products were detected and only γ-HBCD was used to quantify HBCD concentrations, making it impossible to calculate a mass balance. HBCD recoveries on day 0 of the experiment were well below (0.011–0.018 mg/kg dry weight) the nominal test concentrations (0.025 mg/kg dry weight), suggesting rapid adsorption of HBCD to soil and poor extraction methods (Refs. 44 and 101).

In studies using 0.025-0.089 mg/kg HBCD (Davis *et al*. 2005, Ref. 96), the estimated half-life values were shorter than studies using 3.0-4.7 mg/kg HBCD (Davis *et al*. 2006, Ref. 95) by approximately one order of magnitude for aerobic viable sediment (11–32 days compared to72-128 days) and anaerobic viable sediment (1.1-1.5 days compared to 92 days). The viable aerobic soil half-life using lower concentrations of HBCD (Davis *et al*. 2005, Ref. 96) was less than half of the half-life based on the higher HBCD concentration (63 days compared to >120 days) (Davis *et al*. 2006, Ref. 95). Both Davis *et al*. studies (Refs. 95 and 96) suggest that HBCD degrades faster in sediment than in soil, faster under anaerobic conditions than aerobic conditions, and faster with microbial action than without microbial action. HBCD is poorly soluble, and it was suggested that at higher concentrations of

HBCD, degradation is limited by mass transfer of HBCD into microbes. However, results from the Davis *et al*. 2005 study (Ref. 96) likely overestimate the rate of HBCD biodegradation, for the reasons noted previously (primarily, failure to use ^{14}C-radiolabelled HBCD, quantify isomers other than γ-HBCD, identify degradation products, or establish a mass balance, but also procedural problems with contamination of sediment, incomplete HBCD extraction, and occurrence of an interfering peak in the LC/MS chromatograms corresponding to γ-HBCD).

It is important to note that the rapid biodegradation rates from Davis *et al*. 2005 (Ref. 96) are not consistent with environmental observations. HBCD has been detected over large areas and in remote locations in environmental monitoring studies (Refs 1 and 104). Dated sediment core samples indicate slow environmental degradation rates (Refs. 44, 90, 96, and 101). For example, HBCD was found at concentrations ranging from 112 to 70,085 µg/kg dry weight in sediment samples collected at locations near a production site in Aycliffe, United Kingdom two years after the facility was closed down (Ref. 44). Monitoring data do not provide a complete, quantitative determination of persistence because HBCD emission sources, rates, and quantities are typically unknown, and all environmental compartments are not considered. However, the monitoring data do provide evidence in support of environmental persistence. In addition, the widespread presence of HBCD in numerous terrestrial and aquatic species indicates persistence in the environment sufficient for bioaccumulation to occur (Ref. 1).

IV. Rationale for listing HBCD and lowering the reporting threshold.

A. What is EPA's rationale for listing the HBCD category?

HBCD has been shown to cause developmental effects at doses as low as 146.3

mg/kg/day (LOAEL) in male rats. Developmental effects have also been observed with a BMDL of 0.056 mg/kg/day (BMD of 0.18 mg/kg/day) based on effects in female rats and a BMDL of 0.46 mg/kg/day (BMD of 1.45 mg/kg/day) based on effects in male rats. HBCD also causes reproductive toxicity at doses as low 138 mg/kg/day (LOAEL) in female rats. Based on the available developmental and reproductive toxicity, EPA believes that HBCD can be reasonably anticipated to cause moderately high to high chronic toxicity in humans. Therefore, EPA believes that the evidence is sufficient for listing the HBCD category on the EPCRA section 313 toxic chemical list pursuant to EPCRA section 313(d)(2)(B) based on the available developmental and reproductive toxicity data.

HBCD has been shown to be highly toxic to both aquatic and terrestrial species with acute aquatic toxicity values as low as 0.009 mg/L and chronic aquatic toxicity values as low as 0.0042 mg/L. HBCD is highly toxic to terrestrial species as well with observed toxic doses as low as 0.51 and 2.1 mg/kg/day. In addition to being highly toxic, HBCD is also bioaccumulative and persistent in the environment, which further supports a high concern for the toxicity to aquatic and terrestrial species. EPA believes that HBCD meets the EPCRA section 313(d)(2)(C) listing criteria on toxicity alone but also based on toxicity and bioaccumulation as well as toxicity and persistence in the environment. Therefore, EPA believes that the evidence is sufficient for listing the HBCD category on the EPCRA section 313 toxic chemical list pursuant to EPCRA section 313(d)(2)(C) based on the available ecological toxicity data as well as the bioaccumulation and persistence data.

HBCD has the potential to cause developmental and reproductive toxicity at moderately low to low doses and is highly toxic to aquatic and terrestrial organisms; thus, EPA considers HBCD to have moderately high to high chronic human health toxicity and

high ecological toxicity. 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 HBCD meets the criteria of EPCRA section 313(d)(2)(B) or (C).

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

EPA believes that the available bioaccumulation and persistence data for HBCD support a classification of HBCD as a PBT chemical. HBCD has been shown to be highly bioaccumulative in aquatic species and to also biomagnify in aquatic and terrestrial food chains. While there is limited data on the half-life of HBCD in soil and sediment, the best available data supports a determination that the half-life of HBCD in soil and sediment is at least 2 months. This determination is further supported by the data from environmental monitoring studies, which indicate that HBCD has significant persistence in the environment. The widespread presence of HBCD in numerous terrestrial and aquatic species also supports the conclusion that HBCD has significant persistence in the environment. 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 the

HBCD category.

V. 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. USEPA, OEI. 2016. Technical Review of Hexabromocyclododecane (HBCD) CAS Registry Numbers 3194-55-6 and 25637-99-4. January 25, 2016.

2. USEPA, OEI. 2014. Economic Analysis of the Proposed Rule to add HBCD to the List of TRI Reportable Chemicals. March 28, 2014.

3. Arita, R., Miyazaki, K., Mure, S. 1983. Metabolic test of HBCD. Test on chemical substances used in household items. Studies on pharmacodynamics of HBCD (unpublished). In: Toxicology summary: HBCD (HBCD), Albemarle, S.A. Department of Pharmacy, Hokkaido University Hospital, Japan.

4. Yu, C.C., Atallah, Y.H. 1980. Pharmacokinetics of HBCD in rats (unpublished). Vesicol Chemical Corporation, Rosemont, IL.

5. Szabo, D.T., Diliberto, J.J., Hakk, H. *et al*. 2010. Toxicokinetics of the flame retardant HBCD gamma: Effect of dose, timing, route, repeated exposure, and metabolism. Toxicol. Sci. 117(2):282-293.

6. Szabo, D.T., Diliberto, J.J., Hakk, H., Huwe, J.K., Birnbaum, L.S. 2011. Toxicokinetics of the flame retardant hexabromocyclododecane alpha: Effect of dose, timing,

route, repeated Exposure, and metabolism. Toxicol. Sci. 121(2):234-244.

7. Reistad, T., Fonnum, F., Mariussen, E. 2006. Neurotoxicity of the pentabrominated diphenyl ether mixture, DE-71, and HBCD (HBCD) in rat cerebellar granule cells *in vitro*. Arch. Toxicol. 80(11):785-796.

8. van der Ven, L.T.M., Verhoef, A., van de Kuil, T., Slob, W., Leonards, P.E.G., Visser, T.J., Hamers, T., Herlin, M., Hakansson, H., Olausson, H., Piersma, A.H., Vos, J.G. 2006. A 28-day oral dose toxicity study enhanced to detect endocrine effects of hexabromocyclododecane in Wistar rats. Toxicological Sciences 94(2): 281-292.

9. van der Ven, L.T.M., van de Kuil, T., Leonards, P.E., *et al*. 2009. Endocrine effects of HBCD (HBCD) in a one-generation reproduction study in Wistar rats. Toxicol Lett 185:51-62. Including supplementary tables.

10. Brandsma, S.H., van der Ven, L.T.M., De Boer, J. and Leonards, P.E. 2009. Identification of hydroxylated metabolites of hexabromocyclododecane in wildlife and 28-days exposed Wistar rats. Environ. Sci. Technol. 43, 6058-6063.

11. Hakk, H., Szabo, D.T., Huwe, J., Diliberto, J. and Birnbaum, L.S. 2012. Novel and distinct metabolites identified following a single oral dose of α- or γ-hexabromocyclododecane in mice. Environ. Sci. Technol. 46:13494-13503.

12. Sanders, J.M., Knudsen, G.A. and Birnbaum, L.S. 2013. The fate of β-hexabromocyclododecane in female C57BL/6 mice. Toxicological Sciences 134(2): 251–257.

13. Antignac, J.P., Cariou, R., Maume, D., *et al*. 2008. Exposure assessment of fetus and newborn to brominated flame retardants in France: preliminary data. Mol. Nutr. Food Res. 52(2):258-265.

14. Weiss, J., Wallin, E., Axmon, A., *et al*. 2006. Hydroxy-PCBs, PBDEs, and HBCDDs in serum from an elderly population of Swedish fishermen's wives and associations with bone density. Environ. Sci. Technol. 40(20):6282-6289.

15. Kakimoto, K., Akutsu, K., Konishi, Y., *et al*. 2008. Time trend of HBCD in the breast milk of Japanese women. Chemosphere 71(6):1110-1114.

16. Rawn, D.F.K., Ryan, J.J., Sadler, A.R. *et al*. 2014. Brominated flame retardant concentrations in sera from the Canadian Health Measures Survey (CHMS) from 2007 to 2009. Environment International 63: 26-34.

17. Abdallah, M. and Harrad, S. 2011. Tetrabromobisphenol-A, hexabromocyclododecane and its degradation products in UK human milk: Relationship to external exposure. Environment International, 37: 443-448.

18. Meijer, L., Weiss, J., Van Velzen, M., *et al*. 2008. Serum concentrations of neutral and phenolic organohalogens in pregnant women and some of their infants in The Netherlands. Environ. Sci. Technol. 42(9):3428-3433.

19. Thomsen, C., Molander, P., Daae, H.L., *et al*. 2007. Occupational exposure to HBCD at an industrial plant. Environ. Sci. Technol. 41(15):5210-5216.

20. Fangstrom, B., Strid, A., Bergman, A. 2005. Temporal trends of brominated flame retardants in milk from Stockholm mothers, 1980-2004. Department of Environmental Chemistry, Stockholm University, Stockholm, Sweden. Available online at: *http://www.imm.ki.se/Datavard/PDF/mj%C3%B6lk_poolade_NV%20rapport%202005%20modersmjolk.pdf.*

21. Fangstrom, B., Athanassiadis, I., Odsjo, T., *et al*. 2008. Temporal trends of polybrominated diphenyl ethers and HBCD in milk from Stockholm mothers, 1980-2004.

Mol. Nutr. Food Res. 52(2):187-193.

22. Covaci, A., Gerecke, A.C., *et al*. 2006. Hexabromocyclododecanes (HBCDs) in the Environment and Humans: A Review. Environ. Sci. Technol. 40: 3679-3688.

23. Johnson-Restrepo, B., Adams, D.H., *et al*. 2008. Tetrabromobisphenol A (TBBPA) and Hexabromocyclododecanes (HBCDs) in tissues of humans, dolphins, and sharks from the United States. Chemosphere 70: 1935-1944.

24. Toms, L-M.L., Guerra, P., Eljarrat, E., Barceló, D., Harden, F.A., Hobson, P., *et al*. 2012. Brominated flame retardants in the Australian population: 1993–2009. Chemosphere 89:398-403.

25. Schecter, A., Szabo, D.T., Miller, J., Gent, T.L., Malik-Bass, N., Petersen, M., Paepke, O., Colacino, J.A., Hynan L.S., Harris, T.R., Malla, S., Birnbaum, L.S. 2012. Hexabromocyclododecane (HBCD) stereoisomers in U.S. food from Dallas, TX. Environmental Health Perspectives 120(9): 1260-1264.

26. IRDC (International Research and Development Corporation). 1977. Acute toxicity studies in rabbits and rats with HBCD with attachments. Submitted under TSCA Section 8E; EPA Document No. 88-7800065; NTIS No. OTS0200051.

27. IRDC (International Research and Development Corporation). 1978. Acute toxicity studies in rabbits and rats with residue of HBCD with attachments and cover letter dated 030178. Submitted under TSCA Section 8E; EPA Document No. 88-7800088; NTIS No. OTS0200466.

28. Pharmakon Research International Inc. 1990. Acute exposure oral toxicity study in rats (83 EPA/OECD) with attachments and cover letter dated 030890. Submitted under TSCA Section 8D; EPA Document No. 86-900000166; NTIS No. OTS0522237.

29. Gulf South Research Institute. 1988. Initial submission: Letter from Ethyl Corp to USEPA regarding technical and toxicity data on brominated flame retardants including HBCD. EPA Document No. FYI-OTS-0794-0947; NTIS No. OTS0000947.

30. BASF. 1990. Report on the study of the acute oral toxicity of HBCD in the mouse with cover letter dated 03-12-90. Submitted under TSCA Section 8D; EPA Document No. 86-900000383; NTIS No. OTS0522946.

31. Lewis, A.C., Palanker, A.L. 1978. A dermal LD_{50} study in albino rabbits and an inhalation LC_{50} study in albino rats. Test material GLS-S6-41A (unpublished). Consumer Product Testing, Fairfield, NJ; Experiment Reference No. 78385-2. Client: Saytech Inc.

32. Momma, J., Kaniwa, M., Sekiguchi, H., Ohno, K., Kawasaki, Y., Tsuda, M., Nakamura, A., Kurokawa, Y. 1993. Dermatological evaluation of a flame retardant, hexabromocyclododecane (HBCD) on guinea pig by using the primary irritation, sensitization, phototoxicity, and photosensitization of skin. (Article in Japanese; English abstract). Eisei Shikenjo Hokoku 111:18-24.

33. Chengelis, C. 1997. A 28-day repeated dose oral toxicity study of HBCD in rats. Study No. WIL-186004. WIL Research Laboratories, Inc. Ashland, OH.

34. Chengelis, C. 2001. An oral (gavage) 90-day toxicity study of HBCD in rats. Study No. WIL-186012. WIL Research Laboratories, Inc. Ashland, Ohio.

35. Chengelis, C. 2002. Amendment to the Final Report for: An oral (gavage) 90-day toxicity study of HBCD in rats. Study No. WIL-186012. WIL Research Laboratories, Inc. Ashland, Ohio.

36. Hill, R.N., Crisp, T.M., Hurley, P.M., Rosenthal, S.L., and Singh, D.V. 1998. Risk assessment of thyroid follicular cell tumors. Environ. Health Perspect. 106, 447–457.

37. Zeller, H. and Kirsch, P. 1969. Hexabromocyclododecane: 28-day feeding trials with rats. BASF unpublished laboratory study. As cited in USEPA. 2001. High Production Volume (HPV) data summary and test plan for hexabromocyclododecane (HBCD) CAS No. 3194-55-6. Prepared by the American Chemistry Council's Brominated Flame Retardant Industry Panel (BFRIP), Arlington, VA.

38. Zeller, H. and Kirsch, P. 1970. Hexabromocyclododecane: 90-day feeding trials with rats. BASF unpublished laboratory study. As cited in USEPA. 2001. High Production Volume (HPV) data summary and test plan for hexabromocyclododecane (HBCD) CAS No. 3194-55-6. Prepared by the American Chemistry Council's Brominated Flame Retardant Industry Panel (BFRIP), Arlington, VA.

39. Kurokawa, Y., Inoue, T., Uchida, Y., *et al*. 1984. Carcinogenesis test of flame retarder hexabromocyclododecane in mice. Hardy, M.; Albemarle Corporation, personal communication, Department of Toxicology, National Public Health Research Institute, Biological Safety Test Research Center. Unpublished, translated from Japanese. As cited in reference 40.

40. USEPA. 2014. Flame Retardant Alternatives for Hexabromocyclododecane (HBCD): Final Report.

41. Saegusa, Y., Fujimoto, H., Woo, G., *et al*. 2009. Developmental toxicity of brominated flame retardants, tetrabromobisphenol A and 1,2,5,6,9,10-HBCD, in rat offspring after maternal exposure from mid-gestation through lactation. Reprod. Toxicol. 28(4):456-67.

42. Ema, M., Fujii, S., Hirata-Koizumi, M., *et al*. 2008. Two-generation reproductive toxicity study of the flame retardant HBCD in rats. Reprod. Toxicol. 25(3):335-

351.

43. Murai, T., Kawasaki, H., Kanoh, S. 1985. Studies on the toxicity of insecticides and food additives in pregnant rats (7). Fetal toxicity of HBCD. Oyo Yakuri (Pharmacometrics) 29:981-986 (in Japanese with English abstract).

44. European Commission. 2008. Risk Assessment: Hexabromocyclododecane CAS-No.: 25637-99-4 EINECS No.: 247-148-4, Final Report May 2008. Luxembourg: Office for Official Publications of the European Communities.

45. Eriksson, P., Fischer, C., Wallin, M., *et al*. 2006. Impaired behaviour, learning and memory, in adult mice neonatally exposed to HBCD (HBCDD). Environ. Toxicol. Pharmacol. 21(3):317-322.

46. USEPA. 1998. Guidelines for neurotoxicity risk assessment. Risk Assessment Form. **Federal Register**. 63 FR 26926, May 14, 1998 (FRL-6011-3).

47. Industrial Bio-Test Labs. 1990. Mutagenicity of two lots of FM-100 lot 53 and residue of lot 3322 in the absence and presence of metabolic activation with test data and cover letter. Submitted under TSCA Section 8D; EPA Document No. 86-900000267; NTIS No. OTS0523259.

48. Litton Bionetics Inc. 1990. Mutagenicity evaluation of 421-32b (Final report) with test data and cover letter. Submitted under TSCA Section 8D; EPA Document No. 86-900000265; NTIS No. OTS0523257.

49. SRI Research Institute. 1990. In vitro microbiological mutagenicity studies of four CIBA-GEIGY corporation compounds (Final report) with test data and cover letter. Submitted under TSCA Section 8D; EPA Document No. 86-900000262; NTIS No. OTS0523254.

50. Zeiger, E., Anderson, B., Haworth, S., *et al*. 1987. Salmonella mutagenicity tests: III. Results from the testing of 255 chemicals. Environ. Mutagen. 9 (Suppl. 9):1-110.

51. Huntingdon Research Center. 1978. Ames metabolic activation test to assess the potential mutagenic effect of compound no. 49 with cover letter dated 031290. Submitted under TSCA Section 8D; EPA Document No. 86-900000385; NTIS No. OTS0522948.

52. Pharmakologisches Institute. 1978. Ames test with hexabromides with cover letter dated 031290. Submitted under TSCA Section 8D; EPA Document No. 86-900000379; NTIS No. OTS0522942.

53. Ethyl Corporation. 1985. Genetic toxicology Salmonella/microsomal assay on HBCD with cover letter dated 030890. Submitted under TSCA Section 8D; EPA Document No. 86-900000164; NTIS No. OTS0522235.

54. Microbiological Associates Inc. 1996. HBCD (HBCD): chromosome aberrations in human peripheral blood lymphocytes with cover letter dated 12/12/1996. Submitted under TSCA Section 8D; EPA Document No. 86970000358; NTIS No. OTS0573552.

55. Walsh, G.E., Yoder, M.J., McLaughlin, L.L., *et al*. 1987. Responses of marine unicellular algae to brominated organic compounds in six growth media. Ecotoxicol. Environ. Saf. 14:215-222.

56. Drottar, K.R., Krueger, H.O. 1998. Hexabromocyclododecane (HBCD): A flow-through life-cycle toxicity test with the cladoceran (*Daphnia magna*). Report #439A-108. Wildlife International Ltd, Easton, MD, pp 78. Submitted under TSCA Section 8D; EPA Document No. 86980000152; OTS0559490.

57. MOEJ (Ministry of the Environment, Japan). 2009. 6-Week administration study of 1,2,5,6,9,10-hexabromocyclododecane for avian reproduction toxicity under long-day

conditions using Japanese quail. Report. Ministry of the Environment, Japan. Research Institute for Animal Science in Biochemistry & Toxicology (as cited in Ref. 58).

58. UNEP (United Nations Environmental Program). 2010. Hexabromocyclododecane draft risk profile. United Nations Environmental Program, Stockholm Convention.

59. Fernie, K.J., Marteinson, S.C., Bird, D.M., *et al*. 2011. Reproductive changes in American kestrels (*Falco sparverius*) in relation to exposure to technical hexabromocyclododecane flame retardant. Environ. Toxicol. Chem. 30:2570-2575.

60. Marteinson, S.C., Bird, D.M., Shutt, J.L., *et al*. 2010. Multi-generational effects of polybrominated diphenylethers exposure: embryonic exposure of male American kestrels (*Falco sparverius*) to DE-71 alters reproductive success and behaviors. Environ. Toxicol. Chem. 29: 1740-1747.

61. Marteinson, S.C., Kimmins, S., Letcher, R.J., *et al*. 2011. Diet exposure to technical hexabromocyclododecane (HBCD) affects testes and circulating testosterone and thyroxine levels in American kestrels (*Falco sparverius*). Environ. Res. 111:1116-1123.

62. Marteinson, S.C., Bird, D.M., Letcher, R.J., *et al*. 2012. Dietary exposure to technical hexabromocyclododecane (HBCD) alters courtship, incubation and parental behaviors in American kestrels (*Falco sparverius*). Chemosphere 89:1077-1083.

63. Anselmo, H.M.R., Koerting, L., Devito, S., *et al*. 2011. Early life developmental effects of marine persistent organic pollutants on the sea urchin Psammechinus miliaris. Ecotox. Environ. Safe. 74:2182-2192.

64. Deng, J., Yu, L., Liu, C., *et al*. 2009. Hexabromocyclododecane-induced developmental toxicity and apoptosis in zebrafish embryos. Aquat. Toxicol. 93(1):29–36.

65. Du, M., Zhang, D., Yan, C., *et al*. 2012. Developmental toxicity evaluation of three hexabromocyclododecane diastereoisomers on zebrafish embryos. Aquat. Toxicol. 112-113:1-10.

66. Hong, H., Li, D., Shen, R., *et al*. 2014. Mechanisms of hexabromocyclododecanes induced developmental toxicity in marine medaka (*Oryzias melastigma*) embryos. Aquat. Toxicol. 152:173-185.

67. Hu, J., Liang, Y., Chen, M., *et al*. 2009. Assessing the toxicity of TBBPA and HBCD by zebrafish embryo toxicity assay and biomarker analysis. Environ. Toxicol. 24:334-342.

68. Palace, V.P., Pleskach, K., Halldorson, T., *et al*. 2008. Biotransformation enzymes and thyroid axis disruption in juvenile rainbow trout (*Oncorhynchus mykiss*) exposed to hexabromocyclododecane diastereoisomers. Environ. Sci. Technol. 42(6):1967-1972.

69. Palace, V., Park, B., Pleskach, K., *et al*. 2010. Altered thyroxine metabolism in rainbow trout (*Oncorhynchus mykiss*) exposed to hexabromocyclododecane (HBCD). Chemosphere 80(2):165-169.

70. Ronisz, D., Farmen Finne, E., Karlsson, H., *et al*. 2004. Effects of the brominated flame retardants hexabromocyclododecane (HBCDD), and tetrabromobisphenol A (TBBPA), on hepatic enzymes and other biomarkers in juvenile rainbow trout and feral eelpout. Aquat. Toxicol. 69:229-245.

71. Smolarz, K. and Berger, A. 2009. Long-term toxicity of hexabromocyclododecane (HBCDD) to the benthic clam *Macoma balthica* (L.) from the Baltic Sea. Aquat. Toxicol. 95(3):239-247.

72. Wu, M., Zuo, Z., Li, B., *et al*. 2013. Effects of low-level hexabromocyclododecane (HBCD) exposure on cardiac development in zebrafish embryos. Ecotoxicology 22:1200-1207.

73. Zhang, X., Yang, F., Zhang, X., *et al*. 2008. Induction of hepatic enzymes and oxidative stress in Chinese rare minnow (*Gobiocypris rarus*) exposed to waterborne hexabromocyclododecane (HBCDD). Aquat. Toxicol. 86(1):4-11.

74. Zhang, H., Pan, L., Tao, Y. 2014. Antioxidant responses in clam *Venerupis philippinarum* exposed to environmental pollutant hexabromocyclododecane. Environ. Sci. Pollut. Res. 21:8206-8215.

75. Desjardins, D., MacGregor, J.A., Krueger, H.O. 2004. Hexabromocyclododecane (HBCD): A 72 hour toxicity test with the marine diatom (*Skeletonema costatum*), Final report. Wildlife Internation Ltd, Easton, MD, pp 66. As cited in Refs. 44 and 76.

76. IUCLID. 2005. Hexabromocyclododecane IUCLID dataset. Submitted to U.S. EPA's High Production Volume (HPV) Chemical Program.

77. Desjardins, D., MacGregor, J.A., Krueger, H.O. 2005. Final report. Chapter 1, Hexabromocyclododecane (HBCD): A 72-hour toxicity test with the marine diatom (*Skeletonema costatum*) using a co-solvent. Chapter 2, Hexabromocyclododecane (HBCD): A 72-hour toxicity test with the marine diatom (*Skeletonema costatum*) using generator column saturated media. Wildlife International Ltd, Easton, MD, pp19. As cited in Ref. 44.

78. Kobiliris, D. 2010. Influence of embryonic exposure to hexabromocyclododecane (HBCD) on the corticosterone response and "fight or flight" behaviors of captive American kestrels. Thesis submitted to McGill University in partial

fulfilment of the requirements of the degree of Masters of Science. Department of Natural Resource Sciences, McGill University, Montreal, Canada.

79. Crump, D., Egloff, C., Chiu, S., *et al*. 2010. Pipping success, isomer-specific accumulation, and hepatic mRNA expression in chicken embryos exposed to HBCD. Toxicol. Sci. 115:492-500.

80. Wu, T., Wang, S., Huang, H., *et al*. 2012. Diastereomer-specific uptake, translocation, and toxicity of hexabromocyclododecane diastereoisomers to maize. J. Agr. Food Chem. 60:8528-8534.

81. Drottar, K.R. and Krueger, H.O. 2000. Hexabromocyclododecane (HBCD): A flow-through bioconcentration test with the rainbow trout (*Oncorhynchus mykiss*). Report# 439A-111. Wildlife International Ltd, Easton, MD, pp 1-137. Submitted under TSCA Section FYI; EPA Document No. 84010000001; OTS0001392.

82. Veith, G.D., Defoe, D.L., Bergstedt, B.V. 1979. Measuring and estimating the bioconcentration factor of chemicals in fish. J. Fish Res. Board Can. 36:1040-1048.

83. Zhang, Y., Sun, H., Ruan, Y. 2014. Enantiomer-specific accumulation, depuration, metabolization and isomerization of hexabromocyclododecane (HBCD) diastereomers in mirror carp from water. J. Haz. Mater. 264:8-15.

84. He, M., Luo, X., Yu, L., *et al*. 2013. Diasteroisomer and enantiomer-specific profiles of hexabromocyclododecane and tetrabromobisphenol A in an aquatic environment in a highly industrialized area, South China: vertical profile, phase partition, and bioaccumulation. Environ. Poll. 179:105-110.

85. Wu, J., Guan, Y., Zhang, Y., et al. 2011. Several current-use, non-PBDE brominated flame retardants are highly bioaccumulative: evidence from field determined

bioaccumulation factors. Environ. Int. 37:210-215.

86. La Guardia, M.J., Hale, R.C., Harvey, E., *et al*. 2012. In situ accumulation of HBCD, PBDEs, and several alternative flame-retardants in the bivalve (*Corbicula fluminea*) and gastropod (*Elimia proxima*). Environ. Sci. Technol. 46:5798-5805.

87. Law, K., Palace, V.P., Halldorson, T., *et al*. 2006. Dietary accumulation of hexabromocyclododecane diastereoisomers in juvenile rainbow trout (*Oncorhynchus mykiss*) I: bioaccumulation parameters and evidence of bioisomerization. Environ. Toxicol. Chem. 25(7):1757-1761.

88. Esslinger, S., Becker, R., Müller-Belecke, A., *et al*. 2010. HBCD stereoisomer pattern in mirror carps following dietary exposure to pure γ-HBCD enantiomers. J. Agric. Food Chem. 58:9705-9710.

89. Du, M., Lin, L., Yan, C., et al. 2012. Diastereoisomer- and enantiomer-specific accumulation, depuration, and bioisomerization of hexabromocyclododecanes in zebrafish (*Danio rerio*). Environ. Sci. Technol. 46:11040-11046.

90. Marvin, C.H., Tomy, G.T., Armitage, J.M., *et al*. 2011. Hexabromocyclododecane: current understanding of chemistry, environmental fate and toxicology and implications for global management. Environ. Sci. Technol. 45:8613-8623. Including supporting information document.

91. Zhang, Y., Sun, H., Zhu, H., *et al*. 2014. Accumulation of hexabromocyclododecane diastereomers and enantiomers in two microalgae, *Spirulina subsalsa* and *Scenedesmus obliquus*. Ecotox. Environ. Safe. 104:136-142.

92. Harrad, S; Abdallah, MA; Covaci, A. (2009a) Causes of variability in concentrations and diastereomer patterns of Hexabromocyclododecanes in indoor dust.

Environment International 35:573–579.

93. USEPA. 2011. EPI Suite results for CAS 003194-55-6. Download EPI SuiteTM v4.0. U.S. Environmental Protection Agency. Available online at *http://www.epa.gov/opptintr/ exposure/pubs/episuitedl.htm* (see section 2, attachment A in Ref. 1).

94. Schaefer, E.C. and Haberlein, D. 1996. Hexabromocyclododecane (HBCD): Closed bottle test. 439E-102, Wildlife International Ltd, Easton, MD, USA (as cited in Ref. 44).

95. Davis, J.W., Gonsior, S.J., Markham, D.A., *et al*. 2006. Biodegradation and product identification of [^{14}C]hexabromocyclododecane in wastewater sludge and freshwater aquatic sediment. Environ. Sci. Technol. 40:5395-5401. Including supporting information document.

96. Davis, J.W., Gonsior, S.J., Marty, G.T., *et al*. 2005. The transformation of hexabromocyclododecane in aerobic and anaerobic soils and aquatic sediments. Water Res. 39:1075-1084.

97. Hazardous Substance Data Bank. 2011. 1,2,5,6,9,10-Hexabromocyclododecane. Hazardous Substances Data Bank. Part of the National Library of Medicine's Toxicology Data Network (TOXNET7). Bethesda, MD. Available online at *http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB* (accessed May 31, 2011).

98. Bidleman, T.F. 1988. Atmospheric processes. Environ. Sci. Technol. 22(4):361-367.

99. Stenzel, J.I., Nixon, W.B. 1997. Hexabromocyclododecane (HBCD): determination of the vapor pressure using a spinning rotor gauge with cover letter dated

08/15/1997. Chemical Manufacturers Association. Submitted under TSCA Section 8D. OTS0573702.

100. USEPA. 1993. Determination of rates of reaction in the gas-phase in the troposphere. 5. Rate of indirect photoreaction: Evaluation of the atmospheric oxidation computer program of Syracuse Research Corporation for estimating the second-order rate constant for the reaction of an organic chemical with hydroxyl radicals. Washington, DC: U.S. Environmental Protection Agency. EPA744R93001.

101. National Industrial Chemicals Notification and Assessment Scheme. 2012. Hexabromocyclododecane. Priority existing chemical assessment report. Volume 34. Commonwealth of Australia: Australia. National Industrial Chemicals Notification and Assessment Scheme. PEC34.

102. Davis, J.W., Gonsior, S.J., Marty, G.T. 2003. Evaluation of aerobic and anaerobic transformation of hexabromocyclododecane in aquatic sediment systems. Project Study ID 021081, 87 pp. DOW Chemical Company: Midland, MI, USA. Submitted under TSCA Section FYI; EPA Document No. 84040000010; FYI-1103-01472, pg. 440.

103. Davis, J.W., Gonsior, S.J., Marty, G.T. 2003. Evaluation of aerobic and anaerobic transformation of hexabromocyclododecane in soil. Project Study ID 021082, 61 pp. DOW Chemical Company: Midland, MI, USA. Submitted under TSCA Section FYI; EPA Document No. 84040000010; FYI-1103-01472, pg. 379.

104. USEPA. 2010. Hexabromocyclododecane (HBCD) action plan. U.S. Environmental Protection Agency. August 18, 2010.

VI. 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 requirements 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 2025-0009 and 2050-0078. Currently, the facilities subject to the reporting requirements under EPCRA section 313 and PPA section 6607 may use either EPA Toxic Chemicals Release Inventory Form R (EPA Form 1B9350-1), or EPA Toxic Chemicals Release Inventory Form A (EPA Form 1B9350- 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. Since the HBCD category would be classified a PBT category, it is designated as a chemical of special concern, for which Form A reporting is not

allowed. 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 2025-0009 (EPA Information Collection Request (ICR) No. 1363) and those related to trade secret designations under OMB Control 2050-0078 (EPA ICR No. 1428). 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 or 48 CFR chapter 15, 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 55 entities estimated to be impacted by this action, 42 are small businesses; no small governments or small organizations are expected to be affected by this action. All 42 small businesses affected by this action are estimated to incur annualized cost impacts of less than 1%. 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 located in EPA's economic analysis (Ref. 2).

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. Small governments are not subject to the EPCRA section 313 reporting requirements. EPA's economic analysis indicates that the total cost of this action is estimated to be $372,973 in the first year of reporting (Ref. 2).

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 technical standards and is therefore not subject to considerations under section 12(d) of NTTAA, 15 U.S.C. 272 note.

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

EPA has determined that this action will not have disproportionately high and adverse human health or environmental effects on minority or low-income populations as specified in Executive Order 12898 (59 FR 7629, February 16, 1994). 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. This action adds an additional chemical to the EPCRA section 313 reporting requirements. By adding a chemical to the list of toxic chemicals subject to reporting under section 313 of EPCRA, EPA would be providing communities across the United States (including minority populations and low income populations) with access to data which they may use to seek lower exposures and consequently reductions in chemical risks for themselves and their children. This information can also be used by government agencies and others to identify potential problems, set priorities, and take appropriate steps to reduce any potential risks to human health and the environment. Therefore, the informational benefits of the action will have positive human health and

environmental impacts on minority populations, low-income populations, and children.

List of Subjects in 40 CFR Part 372

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

Dated: May 16, 2016.

Gina McCarthy,
Administrator.

Therefore, it is proposed that 40 CFR chapter I be amended as follows:

PART 372—[AMENDED]

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)(2) as follows:

a. Revise the heading for the second column, and

b. Alphabetically add the category "Hexabromocyclododecane (This category includes only those chemicals covered by the CAS numbers listed here)" and list "3194-55-6 (1,2,5,6,9,10-Hexabromocyclododecane)" and "25637-99-4 (Hexabromocyclododecane)"

The additions to read as follows:

§ 372.28 Lower thresholds for chemicals of special concern.

(a) * * *

(2) * * *

Category name	Reporting threshold (in pounds unless otherwise noted)
* * * * * * *	
Hexabromocyclododecane (This category includes only those chemicals covered by the CAS numbers listed here)	100
3194-55-6 1,2,5,6,9,10-Hexabromocyclododecane	
25637-99-4 Hexabromocyclododecane	
* * * * * * *	

* * * * *

3. In § 372.65, paragraph (c) is amended by adding alphabetically an entry for "Hexabromocyclododecane (This category includes only those chemicals covered by the CAS numbers listed here)" to the table to read as follows:

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

* * * * *

(c) * * *

Category name	Effective date
* * * * * * *	
Hexabromocyclododecane (This category includes only those chemicals covered by the CAS numbers listed here)	1/1/17
3194-55-6 1,2,5,6,9,10-Hexabromocyclododecane	
25637-99-4 Hexabromocyclododecane	
* * * * * * *	

[FR Doc. 2016-12464 Filed: 6/1/2016 8:45 am; Publication Date: 6/2/2016]