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ENVIRONMENTAL PROTECTION AGENCY

40 CFR Chapter I

[EPA-HQ-OPPT-2016-0770; FRL-9960-09]

Tetrabromobisphenol A (TBBPA); TSCA Section 21 Petition; Reasons for Agency Response

AGENCY: Environmental Protection Agency (EPA).

ACTION: Petition; reasons for Agency response.

SUMMARY: This document provides the reasons for EPA's response to a petition it received under the Toxic Substances Control Act (TSCA). The TSCA section 21 petition was received from Earthjustice, Natural Resources Defense Council, Toxic-Free Future, Safer Chemicals, Healthy Families, BlueGreen Alliance, and Environmental Health Strategy Center on December 13, 2016. The petitioners requested that EPA issue an order under TSCA section 4, requiring that testing be conducted by manufacturers (which includes importers) and processors on tetrabromobisphenol A ("TBBPA") (CAS No. 79-94-7). After careful consideration, EPA denied the TSCA section 21 petition for the reasons discussed in this document.

DATES: EPA's response to this TSCA section 21 petition was signed March 10, 2017.

FOR FURTHER INFORMATION CONTACT: *For technical information contact*: Virginia Lee, Chemical Control Division (7405M), Office of Pollution Prevention and Toxics, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460-0001; telephone number: (202) 564-4142; email address: lee.virginia@*epa.gov*.

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SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

This action is directed to the public in general. This action may, however, be of interest to those persons who are or may manufacture (which includes import) or process the chemical tetrabromobisphenol A ("TBBPA") (CAS No. 79-94-7). Since other entities may also be interested, the Agency has not attempted to describe all the specific entities that may be affected by this action.

B. How Can I Access Information About this Petition?

The docket for this TSCA section 21 petition, identified by docket identification (ID) number EPA-HQ-OPPT-2016-0770, is available at *http://www.regulations.gov* or at the Office of Pollution Prevention and Toxics Docket (OPPT Docket), Environmental Protection Agency Docket Center (EPA/DC), West William Jefferson Clinton Bldg., Rm. 3334, 1301 Constitution Ave., NW., Washington, DC. The Public Reading Room is open from 8:30 a.m. to 4:30 p.m., Monday through Friday, excluding legal holidays. The telephone number for the Public Reading Room is (202) 566-1744, and the telephone number for the OPPT Docket is (202) 566-0280. Please review the visitor instructions and additional information about the docket available at *http://www.epa.gov/dockets*.

II. TSCA Section 21

A. What is a TSCA Section 21 Petition?

Under TSCA section 21 (15 U.S.C. 2620), any person can petition EPA to initiate a

rulemaking proceeding for the issuance, amendment, or repeal of a rule under TSCA section 4, 6, or 8 or an order under TSCA section 4 or 5(e) or (f). A TSCA section 21 petition must set forth the facts that are claimed to establish the necessity for the action requested. EPA is required to grant or deny the petition within 90 days of its filing. If EPA grants the petition, the Agency must promptly commence an appropriate proceeding. If EPA denies the petition, the Agency must publish its reasons for the denial in the **Federal Register**. A petitioner may commence a civil action in a U.S. district court to compel initiation of the requested rulemaking proceeding within 60 days of either a denial or the expiration of the 90-day period.

B. What Criteria Apply to a Decision on a TSCA Section 21 Petition?

1. *Legal standard regarding TSCA section 21 petitions.* Section 21(b)(1) of TSCA requires that the petition "set forth the facts which it is claimed establish that it is necessary" to issue the rule or order requested. 15 U.S.C. 2620(b)(1). Thus, TSCA section 21 implicitly incorporates the statutory standards that apply to the requested actions. Accordingly, EPA has relied on the standards in TSCA section 21 and in the provisions under which actions have been requested to evaluate this TSCA section 21 petition. In addition, TSCA section 21 establishes standards a court must use to decide whether to order EPA to initiate rulemaking in the event of a lawsuit filed by the petitioner after denial of a TSCA section 21 petition. 15 U.S.C. 2620(b)(4)(B).

2. *Legal standard regarding TSCA section 4 rules.* EPA must make several findings in order to issue a rule or order to require testing under TSCA section 4(a)(1)(A)(i). In all cases, EPA must find that information and experience are insufficient to reasonably determine or predict the effects of a chemical substance on health or the environment and

that testing of the chemical substance is necessary to develop the missing information. 15 U.S.C. 2603(a)(1). In addition, EPA must find that the chemical substance may present an unreasonable risk of injury under section 4(a)(1)(A)(i). Id. If EPA denies a petition for a TSCA section 4 rule or order and the petitioners challenge that decision, TSCA section 21 allows a court to order EPA to initiate the action requested by the petitioner if the petitioner demonstrates to the satisfaction of the court by a preponderance of the evidence in a *de novo* proceeding that findings very similar to those described in this unit with respect to a chemical substance have been met.

III. Summary of the TSCA Section 21 Petition

A. What Action was Requested?

On December 13, 2016, Earthjustice, Natural Resources Defense Council, Toxic-Free Future, Safer Chemicals, Healthy Families, BlueGreen Alliance, and Environmental Health Strategy Center petitioned EPA to issue an order under TSCA section 4(a)(1), 90 days after the petition was filed, requiring that testing be conducted by manufacturers (which includes importers) and processors on tetrabromobisphenol A ("TBBPA") (CAS No. 79-94-7) (Ref. 1).

B. What Support Do the Petitioners Offer?

The petitioners state section 4(a)(1) of TSCA requires EPA to direct testing on a chemical substance or mixture if it finds the following criteria are met:

1. The manufacture, distribution in commerce, processing, use, or disposal of a chemical substance or mixture, or that any combination of such activities, may present an unreasonable risk of injury to health or the environment.

2. There is insufficient information and experience upon which the effects of such

manufacture, distribution in commerce, processing, use, or disposal of such substance or mixture, or of any combination of such activities on health or the environment can reasonably be determined or predicted.

3. Testing is necessary to develop such information.

The petitioners assert that TBBPA "may present an unreasonable risk of injury to health or the environment" because there is substantial evidence that TBBPA may be toxic, including conclusions from:

• EPA's TSCA Work Plan Chemical Problem Formulation and Initial Assessment (Ref. 2), which states TBBPA "can be considered hazardous to the environment" and that "there is some concern" for certain cancers and developmental effects.

• The International Agency for Research on Cancer (IARC) has identified TBBPA as probably carcinogenic to humans (Ref. 3).

• Multiple *in vitro* and animal tests, where TBBPA has been detected to cause endocrine effects, reproductive effects, neurological effects, and immunological effects (Refs. 4-9).

The petitioners also note that EPA, upon adding TBBPA in 1999 to the Toxics Release Inventory (TRI) established under the Emergency Planning and Community Right to Know Act, concluded that "TBBPA is toxic" because "[i]t has the potential to kill fish, daphnid, and mysid shrimp, among other adverse effects, based on chemical and/or biological interactions." 64 FR 58666, 58708. The petitioners assert there is TBBPA exposure to humans and the environment based on the following conclusions.

• TBBPA has the highest production volume of any brominated flame retardant and is extensively used in consumer products, including children's products (Ref. 2). The potential

for widespread exposure is extremely high.

• In 2012, TRI indicated that 127,845 pounds of TBBPA were released into the environment (Ref. 2). Such releases indicate the potential for widespread exposure in the population.

• The presence of TBBPA in people and the environment (biota and environmental media) is established and affirmed in EPA's TBBPA Problem Formulation and Initial Assessment (Ref. 2).

With the evidence of toxicity and exposure and EPA's addition of TBBPA to TRI (Ref. 10), the petitioners argue that TBBPA clearly meets the TSCA section 4 criteria for "may present an unreasonable risk of injury to health or the environment."

The petitioners also assert there is "insufficient information" on TBBPA based on EPA's TBBPA Problem Formulation (Ref. 2), which petitioners say cited lack of data for:

• dermal and inhalation exposures, diet and drinking water exposures, exposures to communities near facilities that manufacture and process TBBPA, exposures to communities near facilities where "e-waste" is disposed of and recycled, exposures to the workers in manufacturing, processing, disposal and recycling facilities, and exposures to degradation and combustion products.

• developmental, reproductive and neurological toxicity, endocrine disruption, and genotoxic effects.

The petitioners argue that the testing recommended in the petition is critical to address this allegedly insufficient information and for performing any TSCA section 6 risk evaluation of TBBPA, and they request EPA to not commence the risk evaluation for TBBPA until data generated to comply with the section 4 test order requested by the

petitioners have been received by EPA.

IV. Disposition of TSCA Section 21 Petition

A. What was EPA's Response?

After careful consideration, EPA has denied the petition. A copy of the Agency's response, which consists of two letters to the signatory petitioners from Earthjustice and Natural Resources Defense Council (Ref. 11), is available in the docket for this TSCA section 21 petition.

B. Background Considerations for the Petition

EPA published a Problem Formulation and Initial Assessment for TBBPA in August 2015 (Ref. 2). As stated on EPA's website titled "Assessments for TSCA Work Plan Chemicals" (Ref. 12), "As a first step in evaluating TSCA Work Plan Chemicals, EPA performs problem formulation to determine if available data and current assessment approaches and tools will support the assessments." During development of the Problem Formulation and Initial Assessment document for TBBPA, EPA followed an approach developed for assessing chemicals under TSCA as it existed at that time.

Under TSCA prior to the June amendments, EPA performed risk assessments on individual uses, hazards, and exposure pathways. The approach taken during the TSCA Work Plan assessment effort was to focus risk assessments on those conditions of use that were most likely to pose concern, *and* for which EPA identified the most robust readily available, existing, empirical data, located using targeted literature searches, although modeling approaches and alternative types of data were also considered. EPA relied heavily on previously conducted assessments by other authoritative bodies and well-established conventional risk assessment methodologies in developing the Problem Formulation

documents. Although EPA identified existing data and presented them in the problem formulations, EPA did not necessarily undertake a comprehensive search of available data or articulate a range of scientifically supportable approaches that might be used to perform risk assessment for various uses, hazards, and exposure pathways in the absence of directly applicable, empirical data prior to seeking public input. Rather, EPA generally elected to focus its attention on the uses, hazards, and exposure pathways that appeared to be of greatest concern and for which the most extensive relevant data had been identified. (Ref. 2).

As EPA explains on its website, "Based on on-going experience in conducting TSCA Work Plan Chemical assessments and stakeholder feedback, starting in 2015 EPA will publish a problem formulation for each TSCA Work Plan assessment as a stand-alone document to facilitate public and stakeholder comment and input prior to conducting further risk analysis. Commensurate with release of a problem formulation document, EPA will open a public docket for receiving comments, data or information from interested stakeholders. EPA believes publishing problem formulations for TSCA Work Plan assessments will increase transparency of EPA's thinking and analysis process, provide opportunity for public/stakeholders to comment on EPA approach and provide additional information/data to supplement or refine assessment approach prior to EPA conducting detailed risk analysis and risk characterization." (Ref. 12).

EPA's 2015 Problem Formulation and Initial Assessment for TBBPA does not constitute a full risk assessment for TBBPA, nor does it purport to be a final analysis plan for performing a risk assessment or to present the results of a comprehensive search for available data or approaches for conducting risk assessments. Rather, it is a preliminary step in the risk assessment process, which EPA desired to publish to provide transparency and the

opportunity for public input. EPA received comments from Earthjustice, Natural Resources Defense Council and others during the public comment period, which ended in November 2015 (Ref. 13). After the public comment period, EPA was in the process of considering this input in refining the analysis plan and further data collection for conducting a risk assessment for TBBPA.

On June 22, 2016, Congress passed the Frank R. Lautenberg Chemical Safety for the 21st Century Act. EPA has interpreted the amended TSCA as requiring that forthcoming risk evaluations encompass all manufacturing, processing, distribution in commerce, use, and disposal activities that the Administrator determines are intended, known, or reasonably foreseen (Ref. 14). This interpretation, encompassing "conditions of use" as defined by TSCA section 3(4), has prompted EPA to re-visit the scoping and problem formulation for risk assessments under TSCA. Other provisions included in the amended TSCA, including section 4(h) regarding alternative testing methods, have also prompted EPA to evolve its approach to scoping and conducting risk assessments. The requirement to consider all conditions of use in risk evaluations – and to do so during the three to three and a half years allotted in the statute – has led EPA to more fully evaluate the range of data sources and technically sound approaches for conducting risk evaluations. Thus, a policy decision articulated in a problem formulation under the pre-amendment TSCA not to proceed with risk assessment for a particular use, hazard, or exposure pathway does not necessarily indicate at this time that EPA will need to require testing in order to proceed to risk evaluation. Rather, such a decision indicates an area in which EPA will need to further evaluate the range of potential approaches – including generation of additional test data – for proceeding to risk evaluation. EPA is actively developing and evolving approaches for

implementing the new provisions in amended TSCA. These approaches are expected to address many, if not all, of the data needs asserted in the petition. Whereas under the Work Plan assessment effort, EPA sometimes opted not to include conditions of use for which data were limited or lacking, under section 6 of amended TSCA, EPA will evaluate all conditions of use and will apply a broad range of scientifically defensible approaches—using data, predictive models, or other methods—that are appropriate and consistent with the provisions of TSCA section 26, to characterize risk and enable the Administrator to make a determination of whether the chemical substance presents an unreasonable risk.

C. What was EPA's Reason for this Response?

For the purpose of making its decision on the response to the petition, EPA evaluated the information presented or referenced in the petition and its authority and requirements under TSCA sections 4 and 21. EPA also evaluated relevant information that was available to EPA during the 90-day petition review period that may have not been available or identified during the development of EPA's TBBPA Problem Formulation and Initial Assessment (Ref. 2).

EPA agrees that the manufacture, distribution in commerce, processing, use, or disposal of TBBPA may present an unreasonable risk of injury to health or the environment under TSCA section 4(a)(1)(A). EPA also agrees that the Problem Formulation and Initial Assessment was not comprehensive in scope with regard to the conditions of use of TBBPA, exposure pathways/routes, or potentially exposed populations. However, the Problem Formulation and Initial Assessment was not designed to be comprehensive. Rather, the Problem Formulation and Initial Assessment was developed under EPA's then-existing process, as explained previously. It was a fit-for-purpose document to meet a TSCA Work

Plan (i.e., pre-Lautenberg Act) need. Going forward under TSCA, as amended, EPA will conform its analyses to TSCA, as amended. EPA has explained elsewhere how the Agency proposes to conduct prioritization and risk evaluation going forward (Refs. 15 and 16). However, EPA does not find that the petitioners have demonstrated, for each exposure pathway and hazard endpoint presented in the petition, that the existing information and experience available to EPA are insufficient to reasonably determine or predict the effects on health or the environment from "manufacture, distribution in commerce, processing, use, or disposal" of TBBPA (or any combination of such activities) nor that the specific testing they have identified is necessary to develop such information.

The discussion that follows provides the reasons for EPA's decision to deny the petition based on the finding for each requested test that the information on the individual exposure pathways and hazard endpoints identified by the petitioners does not demonstrate that there is insufficient information upon which the effects of TBBPA can reasonably be determined or predicted or that the requested testing is necessary to develop additional information. The sequence of EPA's responses follows the sequence in which requested testing was presented in the petition (Ref. 1).

1. *Dermal and Inhalation Exposure Toxicity. a. Dermal toxicity.* The petition does not set forth facts demonstrating that there is insufficient information available to EPA to reasonably determine or predict effects to health from dermal exposure to TBBPA. Therefore, the toxicokinetics test (Organisation for Economic Co-operation (OECD) Test Guideline 417) (Ref. 17) via the dermal route and the skin absorption: in vivo test (OECD Test Guideline 427) (Ref. 18), requested by the petitioners, are not needed. The information already available includes oral toxicity studies and oral toxicokinetic studies identified in

EPA's Problem Formulation and Initial Assessment document (Ref. 2) and the dermal toxicokinetics study identified by the petitioners (Ref. 19). These available studies are sufficient to reasonably determine the internal doses of TBBPA for purposes of route-to-route (oral to dermal) extrapolation. The 2016 Yu et al. study, cited in the petition (Ref. 1), characterizes absorption and elimination, while distribution and metabolism characterization is available from studies using intravenous dosing (Ref. 20). Furthermore, the available studies do not indicate differential distribution, metabolism, and elimination specific to skin. Therefore, the dermal toxicokinetics study requested by the petitioners is not needed to inform or refine evaluation of dermal exposures.

b. Inhalation toxicity. The petition does not set forth facts demonstrating that there is insufficient information available to EPA to reasonably determine or predict effects to health from inhalation exposure to TBBPA. Therefore, the toxicokinetics test (OECD Test Guideline 417) (Ref. 17) via the inhalation route, requested by the petitioners, is not needed. As described in EPA's Problem Formulation and Initial Assessment (Ref. 2), EPA will use an alternative approach to evaluate risks from inhalation exposure to TBBPA. Because TBBPA is a solid, it may be reasonably predicted that particulates in the air are the primary form of TBBPA that would be inhaled. TBBPA particles in air that are inhaled are subsequently swallowed via the mucociliary escalator (Ref. 21). Once the particles are in the gastrointestinal tract, absorption can reasonably be assumed to be the same as in the oral toxicity studies and hence, oral toxicity studies can be used for risk assessment. Information is also available to estimate bioaccessibility of TBBPA from dust using an extraction test with an in vitro colon (Ref. 22). This additional information could also be considered when evaluating risks from TBBPA via the oral route. This approach would not require conducting

the requested toxicokinetics test (Ref. 17).

Although a small percent of TBBPA particles may be in the respirable range and may be absorbed directly through the lungs, existing tests show that no systemic effects were observed in a 14-day inhalation toxicity study (Ref. 23). Therefore, EPA considers that assuming all inhaled particles are eventually swallowed and using existing oral toxicity data should not underestimate effects from inhaling TBBPA particles and therefore would reasonably predict such effects.

Furthermore, EPA's use of available existing toxicity information reduces the use of vertebrate animals in the testing of chemical substances in a manner consistent with provisions described in TSCA section 4(h).

The petition does not set forth facts demonstrating that there is insufficient information available to EPA to reasonably determine or predict effects to the environment, specifically, toxicity to plants exposed to TBBPA via the air. Therefore, the early seedling growth toxicity test (OCSPP Test Guideline 850.4230) (Ref. 24), requested by the petitioners, is not needed. As previously mentioned, because TBBPA is a solid, it may be reasonably predicted that particulates in the air are the primary form of TBBPA that would exist in air. Furthermore, as stated on page 88 of EPA's Problem Formulation and Initial Assessment document (Ref. 2), "[u]ltimately air releases of TBBPA would be expected to undergo deposition to terrestrial and aquatic environments…" and "TBBPA tends to partition to soil and sediment…". These fate pathways for TBBPA are also shown in Figure 2-1 of EPA's Problem Formulation and Initial Assessment document (Ref. 2). Hence, exposure of plants to TBBPA is expected to occur primarily via soil and sediments after deposition from air, which is why EPA excluded this pathway from further assessment (Ref. 2, page 42),

although EPA in the Problem Formulation and Initial Assessment document mistakenly mentioned plants in another sentence addressing "[e]xposure via *directly inhaling* [emphasis added] TBBPA," even though direct inhalation is not applicable to plants and thereby may have caused potential confusion to readers. If toxicity of TBBPA to plants were to be included in an assessment, toxicity data following exposure via soil and/or sediment exposures, not air, would be the scientifically relevant data needed. To this end, as described in EPA's Problem Formulation and Initial Assessment (Ref. 2), existing data and information on phytotoxicity of TBBPA to six plant species is available (Ref. 25). EPA's Problem Formulation and Initial Assessment document (Ref. 2) included references for and a brief description of the existing plant toxicity data (page 105). While assessment of soil-dwelling organisms is included in EPA's Problem Formulation and Initial Assessment document (Ref. 2), as depicted in Figure 2-1 and described on page 40, EPA indicated that the environmental risk assessment for the soil exposure pathway would be based on concentrations of concern derived from data for soil invertebrates (Ref. 2; Figure 2-1; Table 2-6; Page 40). Support for EPA's selection of using species that are expected to be more sensitive to potential effects of TBBPA in soil is provided in EPA's summary of plant toxicity data, which states "…TBBPA is two to three orders of magnitude less toxic to terrestrial plants than to soil-dwelling organisms" (Ref. 2; Table_Apx F-2 and text on page 106).

The petition does not set forth facts demonstrating that there is insufficient information available to EPA to reasonably determine or predict toxicity of TBBPA to avian species. Hence, inhalation toxicokinetic studies (OECD Test Guideline 417) (Ref. 17) and the acute inhalation toxicity study (OCSPP Test Guideline 870.1300) (Ref. 26) modified for birds, requested by the petitioners, are not needed. Although the Problem Formulation and

Initial Assessment document states, "Exposure via directly inhaling TBBPA will not be assessed because no information is available on the toxicity of tetrabromobisphenol A to plants and other wildlife organisms (e.g., birds) exposed via the air." (Ref. 2; page 42), EPA's primary rationale for not including further elaboration of inhalation risks to avian species, as discussed in the Problem Formulation and Initial Assessment document (Ref. 2; page 32 and Appendix F) is TBBPA's low avian toxicity demonstrated in existing studies.

Halldin et al., 2001 and Berg et al., 2001 (Refs. 27 and 28) indicate no effects to egg-laying female quail nor embryos (except at very high doses). The Halldin et al. (Ref. 27) study also included toxicokinetic data indicating that TBBPA is rapidly metabolized and excreted in birds (both embryos and egg-laying females). In these studies, TBBPA was delivered by intravenous injection into females and direct injection into eggs. This dosing regimen assures full (100%) delivery of the dose into the animal, which does not occur in nature, and thus provides the most sensitive means to detect the toxicity of the TBBPA. Other routes of exposure (i.e., oral, inhalation, dermal) result in incomplete absorption limiting the systematic availability of TBBPA compared to the intravenous injection (i.e. less than 100% delivered dose). Hence, intravenous toxicity test designs provide a good understanding of the potential toxicity (or lack thereof) of a chemical. In addition to the low avian toxicity of TBBPA, demonstrated via intravenous injection, inhalation is not expected to be a substantial exposure pathway to wildlife for TBBPA (Refs. 29 and 30). The predominant route of exposure to terrestrial wildlife for a chemical with physical-chemical properties (i.e., Log K_{OW} = 5.90; water solubility = 4.16 mg/L) and partitioning parameters (i.e., low mobility in soil) such as TBBPA is not expected to be via inhalation, but rather through ingestion because the TBBPA will predominantly partition to soils and sediments

if/when released to the environment. The physical-chemical properties of TBBPA also indicate that the fate of TBBPA into water would result in preferential partitioning into sediments and biota (fish or other aquatic organism). Available monitoring data support this conclusion, with higher concentrations of TBBPA in soil and fish relative to concentrations in air.

Hence, additional toxicokinetic studies by the inhalation route is not needed to conduct a reasoned determination or prediction of TBBPA risk to birds.

Furthermore, EPA's use of available existing toxicity information reduces the use of vertebrate animals in the testing of chemical substances in a manner consistent with provisions described in TSCA section 4(h).

2. *Diet and Drinking Water Exposures. a. Diet.* The petition does not set forth facts demonstrating that there is insufficient information available to EPA to reasonably determine or predict effects from exposure to TBBPA via diet. Testing of food products for TBBPA contamination, such as the plant uptake and translocation test (OCSPP Test Guideline 850.4800) (Ref. 31) and modified methods for TBBPA using the Food & Drug Administration's (FDA) Drug & Chemical Residues Methods (Ref. 32), requested by the petitioners, is not necessary because existing data are available to address this exposure pathway.

While a plant uptake study combined with soil concentrations could be used to estimate dietary exposures from plants, chemicals with low water solubility and higher log K_{OW} values similar to TBBPA are less likely to bioaccumulate in plants compared to other foods, such as meats, fish and dairy products (Ref. 33). Hence, other food items, such as meats, fish and dairy products would be expected to be primary contributors to dietary

exposures. Available market basket surveys for TBBPA support this, with most samples comprised of lipid-rich food groups (Ref. 34). There were 465 food samples collected in Europe between 2003 and 2010. Most of these were comprised of lipid-rich food groups; however, some vegetable and grain based food groups were sampled. All samples from this study were below the level of quantification, which was approximately <1 ng/g wet weight, although this varied by food group (Ref. 35). To address dietary exposure from TBBPA, EPA could use a combination of approaches. First, there are existing plant uptake studies available that could be used to estimate TBBPA concentrations in plants from modeled or measured near-facility soil concentrations (Refs. 36 and 37). These studies are not cited in the petition. This approach is supported by a study, that EPA identified since the Problem Formulation and Initial Assessment document was published, that compared a wide variety of plant uptake studies with available models that estimate soil to plant uptake (Ref. 38). Any modeled estimate can be compared to available measured data and a range of values informed by both approaches could be derived. EPA could model soil concentrations from TRI data; these concentrations along with available physical-chemical properties can be used to reasonably estimate plant concentrations and associated dietary exposures. There is also an existing study that quantified soil and plant TBBPA concentrations near a facility (Ref. 39). This data can be used to supplement and/or evaluate the modeling approach. Because existing approaches exist for estimating plant concentrations of TBBPA (modeling and market basket data), the plant uptake and translocation test (Ref. 31) is not necessary.

EPA recognizes that dietary exposures come from a wide variety of sources, not just plants. Market basket surveys provide food concentrations, which can be used to estimate dietary exposure. There are market basket surveys from other countries that measured

TBBPA in various food products (Refs. 40 to 42). Other studies are available that provide data on TBBPA concentrations in breast milk or edible fish (Refs. 43 to 48). Fish concentrations can also be estimated from combining modeled or measured surface water concentrations with bioaccumulation/bioconcentration factors (BAF/BCF). Ingestion from other dietary sources, in addition to fish, shellfish, and breast milk (dairy, meat, fruits and vegetables and grains), can be estimated individually and in total using existing data. It is expected that ingestion of foods with higher lipid content, such as fish and milk, will contribute more to dietary exposure (Ref. 49) than other foods, such as plants. Levels may vary based on proximity to point sources when compared to levels detected in market basket surveys, and this can be considered in developing exposure scenarios and/or background estimates.

b. Drinking Water. The petition does not set forth facts demonstrating that there is insufficient information available to EPA to reasonably determine or predict effects from exposure to TBBPA via drinking water. Sampling of waters in the vicinity of representative manufacturing and processing facilities known to discharge TBBPA, requested by the petitioners, is not necessary because an existing approach is available to address this exposure pathway.

EPA can use release data collected under EPA's TRI program to characterize TBBPA concentrations in surface water near TBBPA manufacturing and processing facilities.

In addition, while there are no data on TBBPA concentrations in finished drinking water, EPA can use surface water monitoring data as a surrogate for finished drinking water to assess potential risks posed by drinking TBBPA-contaminated water. EPA's Office of Water routinely derives Ambient Water Quality Criteria for the Protection of Human Health

(Ref. 50) using the assumption that people may ingest surface water as a drinking water source over a lifetime. There are existing data on TBBPA concentrations in surface water to conduct a drinking water exposure assessment using surface water as a surrogate (Refs. 51 to 53).

EPA believes these approaches are adequate, and likely conservative, to assess potential exposures to drinking water. First, the physical-chemical and fate properties of TBBPA, such as high sorption, low water solubility, and high K_{OC} indicate that concentrations of TBBPA in drinking water would be expected to be low prior to treatment. When sediment monitoring data is used with assumptions about K_{OC}, organic content, and density of water and sediment, surface water concentrations can be estimated to be generally low, below the highest levels reported in surface water (Refs. 54 to 56). This is supported by existing surface water monitoring data indicating the highest concentration of TBBPA in surface water is 4.87 ug/L with most data below 1 ug/L (Refs. 57 and 58). These same chemical and fate properties would indicate that drinking water treatment processes would further reduce TBBPA concentrations in finished drinking water. Overall, the contribution to exposure to TBBPA via drinking water is expected to be minimal.

3. *Exposure from Manufacturing and Processing. a. Communities.* The petition does not set forth facts demonstrating that there is insufficient information available to EPA to reasonably determine or predict exposure to TBBPA to communities near manufacturing and processing facilities. Air sampling, using methods, such as EPA Air Method Toxic Organics-9A (TO-9A, Determination Of Polychlorinated, Polybrominated And Brominated/Chlorinated Dibenzo-p-Dioxins And Dibenzofurans In Ambient Air) (Ref. 60), sampling of soils, and sampling of waters in the vicinity of representative manufacturing and

processing facilities known to discharge TBBPA, as requested by the petitioners, is not necessary because EPA could use an alternative approach to evaluate exposure to TBBPA to communities near manufacturing and processing facilities. EPA could use release data collected under EPA's TRI program and a Gaussian dispersion model, such as AERMOD, to quantify air concentrations and air deposition to soil, to water bodies and to sediments near manufacturing and processing facilities. AERMOD is an EPA model that has been extensively reviewed and validated based on comparisons with monitoring data (Ref. 60). Variability and uncertainty associated with variable emission rates and degradation over time can also be characterized using modeling approaches whereas one-time or limited sampling cannot provide temporal characterizations. In addition, EPA can use monitoring data from other countries as surrogate "near-facility" monitoring data along with modeled estimates. However, the petition does not address this possibility, let alone explain why a testing order under section 4 would be necessary on this point. There are several references with sampling locations near facilities that can be considered, many of which were cited in the Problem Formulation and Initial Assessment document (Ref. 2). EPA considers this approach to be reasonable to determine exposure to communities near manufacturing or processing facilities, but may decide to pursue targeted sampling in the future near manufacturing and processing facilities to reduce uncertainty.

b. Workers. The petition does not set forth facts demonstrating that there is insufficient information available to EPA to reasonably determine or predict exposure to TBBPA to workers in manufacturing and processing facilities.

Since publication of the Problem Formulation and Initial Assessment document, EPA identified exposure monitoring data for Europe, China and the United States for several

industries (the manufacture of epoxy resins and laminates; manufacture of printed circuit boards; and compounding of acrylonitrile butadiene styrene (ABS) resin) (Refs. 61 to 66).

As discussed previously, EPA is actively developing or evolving approaches for implementing the new provisions in amended TSCA. One such approach is to perform systematic literature reviews to identify and/or develop additional available data and modeling approaches for estimating worker inhalation exposure. EPA may also assess exposure concentration in the case of conversion of compounded ABS resin to finished products based on available monitoring data for other industries, such as manufacture of epoxy resins and laminates and manufacture of printed circuit boards. Furthermore, the National Institute of Occupational Safety and Health (NIOSH) has initiated a study titled: "Assessment of Occupational Exposure to Flame Retardants" that aims to quantify, characterize occupational exposure (inhalation, ingestion, or dermal) among workers, and to compare workers' exposures to those of the general population (Ref. 67). Data generated from the NIOSH study is expected to inform occupational exposures and will be considered in an occupational assessment of TBBPA. However, the petition fails to explain how it considered these points or why a testing order under section 4 would be necessary for additional information.

EPA considers the approach considered in the previous paragraph to be reasonable to determine exposure to workers in manufacturing and processing facilities, but may decide to pursue targeted sampling in the future near manufacturing and processing facilities to supplement or refine these approaches.

Dust. EPA believes the approaches described earlier in this unit are sufficient to characterize exposures to workers at manufacturing or processing facilities from external

doses/concentrations. Sampling of settled dust (surface wipe and bulk sampling) using the OSHA Technical Manual (Ref. 68), as specifically requested by the petitioners, is not needed. Presence of TBBPA in settled dust may indicate additional dermal and ingestion exposures are possible. However, surface wipe sampling does not provide a direct estimate of dermal or ingestion exposure. Surface wipe sampling would need to be combined with information on transfer efficiency between the surface, hands, and objects, as well as the number of events to estimate exposures from ingestion (Ref. 69). EPA notes that in the NIOSH study that is in progress surface wipe sampling is not included, which provides support for the conclusion that settled dust is not a customary measure for occupational exposure. EPA would, however, use any information generated from the NIOSH study considered relevant for this exposure pathway.

Biomonitoring. EPA believes the approaches described previously are sufficient to characterize exposures to workers at manufacturing or processing facilities from external doses/concentrations. Therefore, the biomonitoring data collected following the protocols of the current NIOSH study, as requested by the petitioners, is not needed. EPA would, however, consider any data or information generated from the NIOSH study deemed to be relevant and applicable for discerning exposures from any/all exposure routes.

4. *Exposure from recycling.* The petition does not set forth facts demonstrating that there is insufficient information available to EPA to reasonably determine or predict communities specifically located at or near and workers in facilities that recycle TBBPA-containing products. In the Problem Formulation and Initial Assessment document (Ref. 2), EPA identified three monitoring studies that describe concentrations of TBBPA in soil, sediment, and sludge near manufacturing and recycling facilities (Refs. 71, 72, 76). Since

publication of the Problem Formulation and Initial Assessment document (Ref. 2), EPA has identified four monitoring studies that describe concentrations of TBBPA in soil, sediment, indoor and outdoor dust from sampling locations in and near e-waste recycling facilities in other countries (Refs. 70, 73 to 75). These data may be useful for estimating exposures at or near U.S. recycling facilities.

However, EPA intends to further assess how comparable the nature and magnitude of these types of facilities and handling of TBBPA-containing products are to facilities within the U.S. EPA may collect available information related to estimating potential extent and magnitude of exposure. For example, the following could inform development of exposure scenarios for recycling facilities within the United States:

a. the number and location of recycling facilities in the United States,

b. the types and volumes of products that are accepted by these sites, and

c. the recycling and disposal methods employed at these facilities.

With such data or information, the recycling processes used in the U.S. could be compared with the processes used in the studies characterizing the foreign facilities. However, the petition does not address this possibility, let alone explain why a testing order under section 4 would be necessary on this point. If the processes are similar, EPA could extrapolate from foreign facilities to U.S. facilities. If EPA determines these previously indicated approaches are not reasonable to determine exposures, then sampling of soils, sediments and waters in the vicinity of facilities and air to which workers may be exposed at facilities known to recycle TBBPA-containing products, as requested by the petitioners, may become necessary. EPA also notes that the NIOSH study, “Assessment of Occupational Exposure to Flame Retardants,” (Ref. 67) may inform occupational exposures from recycling facilities and will

be considered in an occupational assessment of TBBPA. EPA also notes that the settled dust sampling and biomonitoring data, as requested by the petitioners, may not be the most appropriate data to collect for the reasons provided previously in Unit IV.C.3.b., but that EPA would consider any data or information generated from the NIOSH study deemed to be relevant and applicable for discerning exposures from any/all exposure routes.

5. Exposure from disposal. a. Landfills, wastewater treatment plants, and sewage sludge. The petition does not set forth facts demonstrating that there is insufficient information available to EPA to reasonably determine or predict movement of TBBPA from landfills in soil columns. Leaching studies (OCSPP Testing Guideline 835.1240) (Ref. 77), requested by the petitioners, are not necessary because an existing approach is available to address this fate pathway. Studies measuring the sorption of TBBPA to soil, sand columns, and sediment are available as discussed in Appendix C of the Problem Formulation and Initial Assessment document (Ref. 2). Larsen et al. (2001) reported negligible leaching potential of TBBPA applied to soil and sand columns. (Ref. 78). The adsorption of TBBPA to sediment has been reported (Ref. 79) and suggest low mobility in soil and partitioning to sediments. Data from these existing studies can also serve as input to soil transport models to estimate mobility.

The petition does not set forth facts demonstrating that there is insufficient information available to EPA to reasonably determine or predict transformation processes of TBBPA, which would be episodically and/or continuously released to wastewater. The simulation tests to assess the primary and ultimate biodegradability of chemicals discharged to wastewater (OPPTS Test Guideline 835.3280) (Ref. 80), requested by the petitioners, is not needed because primary degradation and major transformation products can be

determined from existing studies on the ultimate biodegradability of TBBPA in aerobic and anaerobic sludge. One of the studies (Ref. 81) was discussed in Appendix C of EPA's Problem Formulation and Initial Assessment (Ref. 2). Two additional studies (Refs. 82 and 83) were identified after publication of EPA's document (Ref. 2). Li, et al. (2015) (Ref. 82) studied TBBPA transformation in nitrifying activated sludge (NAS). TBBPA transformation was accompanied by mineralization. Twelve metabolites, including those with single benzene ring, O-methyl TBBPA ether, and nitro compounds, were identified during the study. Potvin et al. (2012) (Ref. 83) measured the removal of TBBPA from influent to conventional activated sludge, submerged membrane and membrane aerated biofilm reactors. Removal of TBBPA from these wastewater treatment systems was found to be due to a combination of adsorption and biological degradation. Nyholm, et al. 2010 (Ref. 81) reported transformation as biodegradation half-lives for TBPPA in aerobic activated sludge, aerobic digested sludge, and anaerobic activated sludge amended soils.

The petition does not set forth facts demonstrating that there is insufficient information available to EPA to reasonably determine or predict effects from dietary exposure to crops where TBBPA contaminated sewage sludge is applied. A plant uptake and translocation test (OCSPP Test Guideline 850.4800) (Ref. 31), requested by the petitioners, is not necessary because existing data are available to address this fate pathway. As explained in the dietary exposure section, there are existing plant uptake studies available (Refs. 36 and 37). These data are also available to be used to estimate plant concentrations of agricultural crops where TBBPA-containing sewage sludge is applied. While a plant uptake study combined with sewage sludge concentrations could be used to estimate dietary exposures from plants, chemicals with low water solubility and higher log K_{OW} values similar to

TBBPA, are less likely to bioaccumulate in plants compared to other foods, such as meats, fish and dairy products (Ref. 33). Hence, other food items, such as meats, fish and dairy products, would be expected to be primary contributors to dietary exposures. Available market basket surveys for TBBPA support this, with most samples comprised of lipid-rich food groups (Ref. 34). To address dietary exposure from TBBPA, EPA could use a combination of approaches as described in the dietary exposure section. EPA believes this approach can provide a reasonable estimate of plant concentrations of agricultural crops grown where TBBPA-containing sewage sludge was applied.

b. Incineration. The petition does not set forth facts demonstrating that there is insufficient information available to EPA to reasonably determine or predict communities specifically located near facilities that incinerate TBBPA or TBBPA-containing products.

Electronic waste can be sent to waste-to-energy incinerators (Ref. 84). EPA's Problem Formulation and Initial Assessment for TBBPA (Ref. 2) included a study that measured TBBPA emissions (0.008 ng/L to air) from a mixed household and commercial waste incinerator in Japan (Ref. 85). These data may be useful for estimating exposures at or near U.S. facilities that incinerate TBBPA or TBBPA-containing products.

EPA intends to further assess these facilities and could use an approach that combines existing data to estimate the amount of combustion products at incineration facilities that could have formed from incinerating products that contain TBBPA. Such an approach could combine information on:

i. the types of by-products using data from EU (2006) (Ref. 62) and U.S. EPA (Ref. 87);

ii. information regarding types of consumer waste that contains TBBPA and may be

sent to incinerators;

iii. information on the concentrations of TBBPA in various types of consumer waste; some of these data are available (Refs. 86 to 91);

iv. Toxics Release Inventory data on emissions of the dioxin, furan and polycyclic aromatic hydrocarbons (PAH) by-products from incinerators.

The emissions of dioxins, furans and PAHs could then be modeled using EPA's AERMOD air dispersion model (Ref. 60) and the amount of these by-products that might be attributed to TBBPA could be determined.

Another approach that EPA could take is to estimate exposures near facilities by grouping all near-facility data for a variety of facilities (manufacturing, processing, e-waste, disposal) to estimate a generic "near-facility" exposure. By estimating exposure in this manner, EPA could take advantage of the larger number of monitoring studies or modeled estimates.

However, EPA intends to further assess how comparable locations around incineration sites would be to those around manufacturing, processing, e-waste, and other disposal facilities. There are factors that may either increase and decrease emissions and potential concentrations around these facilities. For example, elevated temperatures are likely to eliminate some amount of possible TBBPA and its combustion products which could reduce overall exposures. The waste stream and content of TBBPA in materials as part of this waste stream are likely to be highly variable and could result in emissions that are higher or lower than those in manufacturing and processing facilities. Comparison of facility specific information could inform which categories of incineration may be sufficiently different from manufacturing and processing facilities to potentially warrant environmental

sampling.

Therefore, to complement the existing data, EPA could collect available information related to estimating potential extent and magnitude of exposure (for example, the number and location of incineration facilities in the U.S. and the types and volumes of products that are accepted by these sites). Waste disposal by incineration as used in the United States could be then compared with the processes used in the studies assessing the foreign facilities. However, the petition does not address this possibility, let alone explain why a testing order under section 4 would be necessary on this point. If the processes are similar, EPA could extrapolate from foreign facilities to U.S. facilities. If EPA determines these previously indicated approaches are not reasonable to determine exposures, then sampling of soils, sediments and waters in the vicinity of facilities and air to which workers may be exposed at facilities known to incinerate TBBPA or TBBPA-containing products, as requested by the petitioners, may be necessary, but could be more strategic and better targeted when based on deliberate evaluation of available existing data and information.

6. *Exposure to degradation by-products. a. Degradation in water or soil.* The petition does not set forth facts demonstrating that there is insufficient information available to EPA to reasonably determine or predict degradation of TBBPA in water by direct photolysis. Studies identifying photodegradation products of TBBPA formed by direct photolysis in water under laboratory conditions (Ref. 92) were identified after the Problem Formulation and Initial Assessment document was published. Therefore, the photodegradation in water test (OCSPP Test Guideline 835.2240) (Ref. 93), requested by the petitioners, is not needed.

The petition does not set forth facts demonstrating that there is insufficient information available to EPA to reasonably determine or predict reactions resulting from

chemical or electronic excitation transfer from light-absorbing humic species rather than from direct sunlight for TBBPA. A study identifying indirect photodegradation products of TBBPA formed by indirect photolysis in water under laboratory conditions (Ref. 94) was identified after the Problem Formulation and Initial Assessment document was published. Therefore, the indirect photolysis in water test (OCSPP 835.5270) (Ref. 95), requested by the petitioners, is not needed.

The petition does not set forth facts demonstrating that there is insufficient information available to EPA to reasonably determine or predict degradation of TBBPA in soil by photolysis. Photolysis of TBBPA deposited on soil or applied to soil with sludge is a possible fate pathway, which could involve different pathways and mechanisms other than photolysis in water. Existing aqueous photolysis studies and/or predictive models can be used to reasonably predict the degradation products of TBBPA. Environmental transport and exposure modeling could be conducted using available measured or estimated physical-chemical properties to estimate exposure of degradation products. This approach has been used by others (Ref. 96) to estimate PBT properties for degradation products. Therefore, the photodegradation in soil test (OCSPP Test Guideline 835.2410) (Ref. 97), requested by the petitioners, is not needed.

b. Microbial degradation. The petition does not set forth facts demonstrating that there is insufficient information available to EPA to reasonably determine or predict microbial degradation of TBBPA in soil in aerobic and anaerobic conditions. EPA has identified existing studies/data describing aerobic and anaerobic biodegradation pathways of TBBPA in both soil samples potentially pre-exposed and not pre-exposed to TBBPA. Some studies are discussed in Appendix C of EPA's Problem Formulation and Initial Assessment

document (Refs. 81, 98 and 99). EPA identified two additional studies after publication of the Problem Formulation and Initial Assessment document that also address this endpoint (Refs. 82 and 100). These studies allow EPA to reasonably determine transformation products and predict relative rates from aerobic and anaerobic microbial degradation in soil. Therefore, the aerobic and anaerobic transformation in soil test (OECD Test Guideline 307) (Ref. 101) and terrestrial soil-core microcosm test (OCSPP Test Guideline 850.4900) (Ref. 102), requested by the petitioner, are not needed.

The petition does not set forth facts demonstrating that there is insufficient information available to EPA to reasonably determine or predict aerobic aquatic biodegradation of TBBPA. Studies are available (Refs. 103 and 104) to reasonably determine aerobic aquatic biodegradation pathways and products as discussed in Appendix C of EPA's Problem Formulation and Initial Assessment document (Ref. 2). Therefore, the aerobic mineralization in surface water-simulation biodegradation test (OCSPP Test Guideline 835.3190) (Ref. 105), requested by the petitioner, is not needed.

As noted in the exposure from disposal discussion, the petition does not set forth facts demonstrating that there is insufficient information available to EPA to reasonably determine or predict degradation processes of TBBPA, which would be episodically and/or continuously released to wastewater. The simulation tests to assess the primary and ultimate biodegradability of chemicals discharged to wastewater (OPPTS Test Guideline 835.3280) (Ref. 80), which the petitioner cited in the discussion about exposure to degradation by-products, is not needed.

c. Combustion products. The petition does not set forth facts demonstrating that there is insufficient information available to EPA to reasonably determine or predict potential

combustion products of TBBPA. The reference to combustion testing cited by the petitioners and others is available (Refs. 62 and 106). However, knowledge of the types and volumes of TBBPA-containing products is needed to use this data to estimate potential exposures to combustion products. As stated in the Problem Formulation and Initial Assessment document (Ref. 2; page 91), "… contribution of TBBPA to combustion byproducts is not possible to determine." However, EPA could acquire this information from recycling and incineration facilities using approaches described in Units IV.C.4. and IV.C.5.b. The petition does not address this possibility, let alone explain why a testing order under section 4 would be necessary on this point.

d. Toxicity of degradation products. The petition does not set forth facts demonstrating that there is insufficient information available to EPA to reasonably determine or predict characterization of TBBPA degradation products, and, as stated in Units IV.C.5.a, IV.C.6.a, and IV.C.6.b., EPA has an understanding of the products potentially formed from TBBPA degradation (e.g., tri-, di-, and monobromobisphenol A, bisphenol A, TBBPA – bis(methyl ether), isopropyl dibromophenols). EPA can use predictive models (e.g., EPA's EPISuite models (Ref. 107) to estimate the key physical-chemical properties of these degradants. EPISuite models have been validated and peer reviewed, and TBBPA degradates are chemicals for which EPISuite models are suitable for estimating (i.e., are within applicability domains of EPISuite models). EPISuite has been used for estimating chemical properties in risk assessments conducted by the USEPA, the EU, and Canada. Therefore, the use of the EPA series 830 Group B testing guidelines (Ref. 108), requested by the petitioners, is not needed.

The petition does not set forth facts demonstrating that there is insufficient

information available to EPA to reasonably determine or predict toxicity effects of TBBPA degradation products to mammals and birds. The petition did not reflect a comprehensive search and review for existing toxicity data on potential degradation products, and EPA's Problem Formulation and Initial Assessment document (Ref. 2) did not purport to represent such a comprehensive search for degradation products. To address the need for mammal or avian toxicity under EPA's current approach, EPA would conduct a comprehensive literature review to identify existing data for these chemicals or for analogs. Following identification and review of existing data, if EPA deemed specific testing necessary to fill identified data gaps, EPA would consider testing according to EPA series 850 Ecological Effects Test Guidelines (Ref. 109), EPA series 870 Health Effects Test Guidelines (Ref. 110), or appropriate OECD Guidelines.

The petition does not set forth facts demonstrating that there is insufficient information available to EPA to reasonably determine or predict the toxicity effects of TBBPA degradation products to aquatic organisms. The petition did not reflect a comprehensive search and review for existing toxicity data on potential degradation products, and EPA's Problem Formulation and Initial Assessment document (Ref. 2) did not purport to represent such a comprehensive search. To address the need for aquatic toxicity under EPA's current approach, EPA would conduct a comprehensive literature review to identify existing data for these chemicals or for analogs. EPA also believes there are alternative approaches available to EPA regarding ecological effects of TBBPA degradation products on aquatic organisms. EPA could use EPA's ECOSAR (Ref. 111) to estimate the aquatic toxicity of these degradants. ECOSAR is an expert system and collection of models (i.e., Quantitative Structure Activity Relationships) that estimate toxicity from structure and physical-chemical

properties of a chemical. The models incorporated into ECOSAR have been validated and peer reviewed. ECOSAR models are suitable for estimating toxicity of potential TBBPA degradates (i.e., TBBPA degradation product chemicals are within the applicability domains of ECOSAR models). Therefore, the use of the EPA series 850 testing guidelines (Ref. 109), requested by the petitioners, is not needed for aquatic organisms.

Furthermore, EPA's use of available existing toxicity information and modeling approaches reduces the use of vertebrate animals in the testing of chemical substances in a manner consistent with provisions described in TSCA section 4(h).

7. *Hazard endpoints. a. Reproductive toxicity, developmental toxicity and neurotoxicity*. The petition does not set forth facts demonstrating that there is insufficient information available to EPA to reasonably determine or predict reproductive, developmental and neurotoxicity of TBBPA. Therefore, the reproductive/developmental toxicity screening test (OECD Test Guideline 421) (Ref. 112), NTP's Modified One-Generation Reproduction Study (Ref. 113) and the complementing Developmental Neurotoxicity Study (OECD Test Guideline 426) (Ref. 114), requested by the petitioners, are not necessary. EPA has identified 15 reproductive/developmental toxicity tests conducted by the oral route of which some include evaluation of neurotoxicity endpoints. The available studies include: a one-generation reproduction toxicity test (Refs. 115 and 9); two 2-generation reproduction tests (Refs. 116 to 118); four prenatal developmental toxicity tests, including a developmental neurotoxicity test (Refs. 119 to 122); and six postnatal developmental toxicity tests, with some that also include a prenatal component (Refs. 123 to 128). All of these studies, except Hass et al. (2003) (Ref. 119) and Kim et al. (2015) (Ref. 126), were described in Appendix G of the published Problem Formulation and Initial Assessment document for TBBPA (Ref. 2).

These studies are either equivalent or superior to the methods used in the reproductive/developmental toxicity screening test (OECD Test Guideline 421) (Ref. 112) and the NTP Modified One-Generation Reproduction Study (Ref. 113).

For developmental neurotoxicity, a study for this endpoint by the oral route is available (Ref. 119), and EPA would consider the results of this study when evaluating risks from TBBPA. Although the study was conducted when the Developmental Neurotoxicity Study OECD Test Guideline 426 (Ref. 114) was a draft guideline, the study is adequate for consideration as part of a weight-of-evidence analysis along with the results of a 2-generation reproduction toxicity study that included a neurotoxicity component (Ref. 121).

Furthermore, EPA conducted an in-depth review of the existing dataset of reproductive and developmental toxicity studies identified, as well as additional animal and human data that evaluated neurotoxicity endpoints (Refs. 131 and 116) following the publication of the Problem Formulation and Initial Assessment document (Ref. 2) and determined that the developmental, reproductive and neurotoxicity endpoints are adequately addressed. Therefore, EPA could use this body of existing data in selecting studies for use in risk evaluation.

Furthermore, EPA's use of available existing toxicity information reduces the use of vertebrate animals in the testing of chemical substances in a manner consistent with provisions described in TSCA section 4(h).

b. Amphibian endocrine system. The petition does not set forth facts demonstrating that there is insufficient information available to EPA to reasonably determine or predict adverse endocrine-related effects from exposure to TBBPA. Therefore, the larval amphibian growth and development assay (LAGDA) (OCSPP Test Guideline 890.2300) (Ref. 132) is

not necessary. Data are available that address thyroid effects of TBBPA for both bioactivity and dose response (Refs. 57 and 133 to 139). These data include mixed results in amphibians and more consistent results in mammals indicating that changes in thyroid hormones are associated with developmental effects (specifically neurobehavioral effects). Given the weight-of-evidence, EPA does not believe that the LAGDA would significantly change this conclusion. Furthermore, EPA's use of this available existing toxicity information reduces the use of vertebrate animals in the testing of chemical substances in a manner consistent with provisions described in TSCA section 4(h).

8. *EPA's conclusions.* EPA denied the request to issue an order under TSCA section 4 because the TSCA section 21 petition does not set forth sufficient facts for EPA to find that the information currently available to the Agency, including existing studies (identified prior to or after publication of EPA's Problem Formulation and Initial Assessment) on TBBPA and analogs, as well as alternate approaches for risk evaluation, is insufficient to permit a reasoned determination or prediction of the health or environmental effects of TBBPA at issue in the petition nor that the specific testing the petition identified is necessary to develop additional information, as elaborated throughout Unit IV of this notice.

Furthermore, to the extent the petitioners request vertebrate testing, EPA emphasizes that future petitions should discuss why such testing is appropriate, considering the reduction of testing on vertebrates encouraged by section 4(h) of TSCA, as amended.

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 physically located in the docket. For assistance in locating these other documents, please consult the technical person listed under **FOR FURTHER INFORMATION CONTACT**.

1. Earthjustice, Natural Resources Defense Council, Toxic-Free Future, Safer Chemicals, Healthy Families, BlueGreen Alliance, Environmental Health Strategy Center; Eve Gartner, Earthjustice; and Veena Singla, Natural Resources Defense Council to Gina McCarthy, Administrator, Environmental Protection Agency. Re: Petition to Order Testing of Tetrabromobisphenol A (CAS No. 79-94-7) under Section 4(a) of the Toxic Substances Control Act. December 13, 2016.

2. EPA. TSCA Work Plan Chemical Problem Formulation and Initial Assessment Tetrabromobisphenol A and Related Chemicals Cluster Flame Retardants. 2015.

3. World Health Organization International Agency for Research on Cancer. *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans*. 2014. (retrieved on February 4, 2017) *https://monographs.iarc.fr/ENG/Publications/internrep/14-002.pdf*

4. Hamers, T. et al. In Vitro Profiling of the Endocrine-Disrupting Potency of Brominated Flame Retardants. *Toxicological Sciences.* 92:157. 2006.

5. Shi, H. et al. Teratogenic effects of tetrabromobisphenol A on Xenopus tropicalis embryos. *Comp. Biochemistry & Physiology Part C: Toxicology & Pharmacology*. 152:62-68. 2010.

6. Zatecka, E. et al. Effect of tetrabrombisphenol A on induction of apoptosis in the testes and changes in expression of selected testicular genes in CD1 mice. *Reproductive Toxicology.* 35:32 2013.

7. Meerts, I. et al. In vitro estrogenicity of polybrominated diphenyl ethers,

hydroxylated PDBEs, and polybrominated bisphenol A compounds. *Environmental Health Perspective.* 2001.

8. Pullen, S. et al. The flame retardants tetrabromobisphenol A and tetrabromobisphenol A /bisallylether suppress the induction of interleukin-2 receptor a chain (CD25) in murine splenocytes. *Toxicology.* 2003.

9. Van der Ven, L. et al. Endocrine effects of tetrabromobisphenol-A (TBBPA) in Wistar rats as tested in a one-generation reproduction study and a subacute toxicity study. *Toxicology.* 2008.

10. EPA. Persistent Bioaccumulative Toxic (PBT) Chemicals; Lowering of Reporting Thresholds for Certain PBT Chemicals; Addition of Certain PBT Chemicals; Community Right-to-Know Toxic Chemical Reporting; Final Rule. **Federal Register**. (Oct. 29, 1999, 64 FR 58666) (FRL-6389-11).

11. EPA. Response to Petition to Order Testing of Tetrabromobisphenol A (CAS No. 79-94-7) Under Section 4(a) of the Toxic Substances Control Act. 2017.

12. EPA. Assessments for TSCA Work Plan Chemicals. *https://www.epa.gov/assessing-and-managing-chemicals-under-tsca/assessments-tsca-work-plan-chemicals* (retrieved on February 21, 2017)

13. EPA. Work Plan Chemical Problem Formulation and Initial Assessment and Data Needs Assessment Documents for Flame Retardant Clusters. 2015. *https://www.epa.gov/assessing-and-managing-chemicals-under-tsca/tsca-work-plan-chemical-problem-formulation-and-2*

14. EPA. Procedures for Chemical Risk Evaluation under the Amended Toxic Substances Control Act; Proposed Rule. **Federal Register** (82 FR 7565, January 19, 2017)

(FRL-9957-75). *https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0654-0001*

15. EPA. Procedures for Prioritization of Chemicals for Risk Evaluation under Toxic Substances Control Act; Proposed Rule. **Federal Register** (82 FR 4826, January 17, 2017) (FRL-9957-74). *https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0636-0001*

16. EPA. Docket EPA-HQ-OPPT-2016-0654. 2016. *https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0654-0001*

17. OECD. Test No 417: *Toxicokinetics. Guideline for the testing of chemicals.* OECD Guidelines for the Testing of Chemicals, Section 4: Health Effects. OECD Publishing, Paris. 2010.

18. OECD. Test No. 427: Skin Absorption: In Vivo Method. *OECD Guidelines for the Testing of Chemicals, Section 4: Health Effects*. OECD Publishing, Paris. 2004.

19. Knudsen, G. A., J. M. Sanders, A. M. Sadik, and L. S. Birnbaum. Disposition and kinetics of tetrabromobisphenol A in female Wistar Han rats. *Toxicology reports*. 1, 214-223.2014.

20. Yu et al. Absorption and excretion of tetrabromobisphenol A in male Wistar rats following subchronic dermal exposure. *Chemosphere*. 146:189-194. 2016.

21. Klassen, C.D. Editor: Cassarett and Doull's *Toxicology: The Basic Science of Poisons*. Seventh Edition. McGraw-Hill Medical Publishing Division. New York. 2008.

22. Abdallah, M. A-E., Tilston, E., Harrad, S. and C. Collins. *In vitro* assessment of the bioaccessibility of brominated flame retardants in indoor dust using a colon extended model of the human gastrointestinal tract. *Journal of Environmental Monitoring.* 14:3276-

3283. 2012.

23. IRDC (International Research and Development Corporation). 1975. *Fourteen-Day Inhalation Toxicity Study in Rats (Unpublished)*. (as cited in EC, 2006).

24. EPA. Early Seedling Growth Toxicity. OCSPP Test Guideline 850.4230. 1998.

25. ACC-BFRIP (American Chemistry Council Brominated Flame Retardant Industry Panel). Tetrabromobisphenol A: A Toxicity Test to Determine the Effects of the Test Substance on Seedling Emergence of Six Species of Plants. Study conducted by Wildlife International Ltd., March 5. Project No 439-102. 2002.

26. EPA. Acute Inhalation Toxicity (OCSPP Test Guideline 870.1300). 1998.

27. Halldin, K., C. Berg, A. Bergman, I. Brandt, and B. Brunstrom. Distribution of Bisphenol a and Tetrabromobisphenol a in Quail Eggs, Embryos and Laying Birds and Studies on Reproduction Variables in Adults Following in Ovo Exposure. *Archives of Toxicology.* 75, 597-603. 2001.

28. Berg, C., K. Halldin, and B. Brunstrom. Effects of Bisphenol a and Tetrabromobisphenol a on Sex Organ Development in Quail and Chicken Embryos. *Environmental Toxicology and Chemistry*. 20(12), 2836-2840. 2001.

29. Hudson, R. H., et al. Handbook of toxicity of pesticides to wildlife. *Resource Publication*. Washington, D.C. (1984).

30. Driver, C. J., Drown, D. B., Ligotke, M. W., Van Voris, P., McVeety, B. D. and Greenspan, B. J. Routes of uptake and their relative contribution to the toxicologic response of Northern bobwhite (*Colinus virginianus*) to an organophosphate pesticide. *Environmental Toxicology and Chemistry*. 10: 21–33. 1991.

31. EPA. Plant Uptake and Translocation (OCSPP Test Guideline 850.4800). 1998.

32. FDA. *DIOXINS: FDA Strategy for Monitoring, Method Development, and Reducing Human Exposure*. 2002, Feb. 7. Retrieved from *http://www.fda.gov/Food/FoodborneIllnessContaminants/ChemicalContaminants/ucm077432.htm*

33. She, Ya-Zhe, et al. "Bioaccumulation of polybrominated diphenyl ethers and several alternative halogenated flame retardants in a small herbivorous food chain." Environmental pollution 174 (2013): 164-170.

34. Shi, Z. X., Wu, Y. N., Li, J. G., Zhao, Y. F., & Feng, J. F. (2009). Dietary exposure assessment of Chinese adults and nursing infants to tetrabromobisphenol-A and hexabromocyclododecanes: occurrence measurements in foods and human milk. Environmental science & technology, 43(12), 4314-4319.

35. EFSA 2011: Scientific Opinion on TBBPA and its derivatives in Food. EFSA Panel on Contaminants in the Food Chain.

36. Li, Y., Q. Zhou, Y. Wang, and X. Xie. Fate of Tetrabromobisphenol a and Hexabromocyclododecane Brominated Flame Retardants in Soil and Uptake by Plants. *Chemosphere*. 82(2), 204-209. 2011.

37. Suominen, K., Verta, M., and Marttinen, S. Hazardous organic compounds in biogas plant end products—Soil burden and risk to food safety. *Science of the Total Environment.* 491:192-199. 2014.

38. Takaki K, Wade AJ, Collins CD. Assessment of plant uptake models used in exposure assessment tools for soils contaminated with organic pollutants. *Environmental Science and Technology*. 48(20):12073-82. 2014.

39. Wang, J., L. Liu, J. Wang, B. Pan, X. Fu, G. Zhang, L. Zhang, and K. Lin.

Distribution of Metals and Brominated Flame Retardants (BFRs) in Sediments, Soils and Plants from an Informal E-Waste Dismantling Site, South China. *Environmental Science Pollution Research International*. 22(2), 1020-1033. 2015.

40. de Winter-Sorkina, R., Bakker, M. I., Van Donkersgoed, G., and Van Klaveren, J. D. Dietary intake of brominated flame retardants by the Dutch population. RIVM report 31305001/2003. RIVM – Netherlands Institute of Public Health and the Environment. 2003.

41. Murata, S., Nakagawa, R., Ashizuka, Y., Hori, T., Yasutake, D., Tobiishi, K., and Sasaki, K. Brominated flame retardants (HBCD, TBBPA and ΣPBDEs) in market basket food samples of Northern Kyushu district in Japan. *Organohalogen Compounds*. 69:1985-1988. (2007).

42. Nakao, T., Kakutani, H., Akiyama, E., and Ohta, S. Levels of tetrabromobisphenol A and its related compounds in infant foods in Japan. *Organohalogen Compounds*. 75:169-172. 2013.

43. Luigi, V., M. Giuseppe, and R. Claudio. Emerging and priority contaminants with endocrine active potentials in sediments and fish from the river Po (Italy). *Environmental Science and Pollution Research*. 22(18):14050-14066. 2015.

44. He, M.-J., X.-J. Luo, L.-H. Yu, J.-P. Wu, S.-J. Chen, and B.-X. Mai. 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. *Environmental Pollution*. 179:105-110. 2013.

45. Ohta, S., T. Okumura, H. Nishimura, T. Nakao, A. Osamau, and H. Miyata. Characterization of Japanese Pollution by PBDEs, TBBPA, PCDDs/DFs, PBDDs/DFs and

PXDDs/DFs Observed in the Long-Term Stock- Fishes and Sediments. Abstracts of the 3rd International Workshop on Brominated Flame Retardants. 2004.

46. Harrad, S., and Abdallah, M. A.-E. Concentrations of Polybrominated Diphenyl Ethers, Hexabromocyclododecanes and Tetrabromobisphenol-A in Breast Milk from United Kingdom Women Do Not Decrease Over Twelve Months of Lactation. *Environmental Science and Technology*. 49(23):13899-13903. 2015.

47. Lankova, D., O. Lacina, J. Pulkrabova, and J. Hajslova. The determination of perfluoroalkyl substances, brominated flame retardants and their metabolites in human breast milk and infant formula. *Talanta*. 117:318-325. 2013.

48. Shi, Z., Y. Jiao, Y. Hu, Z. Sun, X. Zhou, J. Feng, J. Li, and Y. Wu. Levels of tetrabromobisphenol A, hexabromocyclododecanes and polybrominated diphenyl ethers in human milk from the general population in beijing, china. *Science of the Total Environment*. 452:10-18. 2013.

49. Shi, Z., Zhang, L., Li, J., Zhao, Y., Sun, Z., Zhou, X., and Wu, Y. Novel brominated flame retardants in food composites and human milk from the Chinese Total Diet Study in 2011: Concentrations and a dietary exposure assessment. *Environment International*. 96:82-90. 2016.

50. U.S. EPA (OW). Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health. October. EPA-822-B-00-004.2000.

51. Quade, S. C. 2003. Determination of Tetrabromobisphenol a in Sediment and Sludge. (M.Sc.), University of Guelph, Guelph, Ontario.

52. Xiong, J., T. An, C. Zhang, and G. Li. 2015. Pollution Profiles and Risk Assessment of Pbdes and Phenolic Brominated Flame Retardants in Water Environments

within a Typical Electronic Waste Dismantling Region. Environ Geochem Health, 37(3), 457-473.

53. Yang, S., S. Wang, H. Liu, and Z. Yan. 2012. Tetrabromobisphenol A: Tissue Distribution in Fish, and Seasonal Variation in Water and Sediment of Lake Chaohu, China. Environmental Science and Pollution Research, 19(9), 4090-4096.

54. ECHA 2016. Guidance on information requirements and chemical safety assessment. Chapter R.16: Environmental exposure assessment.

55. Guerra, P., E. Eljarrat, and D. Barcelo. 2010. Simultaneous Determination of Hexabromocyclododecane, Tetrabromobisphenol a, and Related Compounds in Sewage Sludge and Sediment Samples from Ebro River Basin (Spain). Analytical and Bioanalytical Chemistry, 397, 2817-2824.

56. Zhang, X. L., X. J. Luo, S. J. Chen, J. P. Wu, and B. X. Mai. 2009. Spatial Distribution and Vertical Profile of Polybrominated Diphenyl Ethers, Tetrabromobisphenol a, and Decabromodiphenylethane in River Sediment from an Industrialized Region of South China. Environ Pollut, 157(6), 1917-1923.

57. Yang, S., Z. Yan, F. Xu, S. Wang, and F. Wu. Development of freshwater aquatic life criteria for tetrabromobisphenol A in China. *Environmental Pollution*. 169:59-63. 2012.

58. Harrad, S., M. A. Abdallah, N. L. Rose, S. D. Turner, and T. A. Davidson. Current-Use Brominated Flame Retardants in Water, Sediment, and Fish from English Lakes. *Environmental Science and Technology*. 43(24), 9077-9083. 2009.

59. EPA. Office of Research and Development. *Compendium Method TO-9A: Determination Of Polychlorinated, Polybrominated And Brominated/Chlorinated Dibenzo-p-Dioxins And Dibenzofurans In Ambient Air*. 1999.

https://www3.epa.gov/ttnamti1/files/ambient/airtox/to-9arr.pdf

60. EPA. AERMOD. Technology Transfer Network Support Center for Regulatory Atmospheric Modeling, Meteorological Processors and Accessory Programs. Air dispersion software. 2016. *https://www3.epa.gov/ttn/scram/dispersion_prefrec.htm#aermod.*

61. ACC. 2000. Brominated Flame Retardant End-User Survey - Phase 1. Study conducted by Breysse, P., and J. Kacergis, Johns Hopkins School of Hygiene and Public Health. Baltimore, MD. Doc ID 84010000001. May 19, 2000.

62. EC (European Commission). European Union Risk Assessment Report for 2,2',6,6'-Tetrabromo-4,4'-Isopropylidenediphenol (Tetrabromobispheonl-A or TBBP-A) Part II – Human Health, CAS No. 79-94-7, EINECS No. 201-236-9. 4th Priority List, Volume: 63, EUR22161 EN. Institute for Health and Consumer Protection, Joint Research Centre, Luxembourg. 2006. *http://esis.jrc.ec.europa.eu/doc/risk_assessment/REPORT/tbbpaHHreport402.pdf.*

63. Mäkinen, M.S.E., Mäkinen, M.R.A., Koistinen, J.T.B., Pasanen, A., Pertti, O.P., Kalliokoski, P.J., and Korpi, A.M. Respiratory and Dermal Exposure to Organophosphorus Flame Retardants and Tetrabromobisphenol A at Five Work Environments. *Environmental Science and Technology. 43* (3), pp 941–947. 2009.

64. Rosenberg, C., M. Hameila, J. Tornaeus, K. Sakkinen, K. Puttonen, A. Korpi, M. Kiilunen, M. Linnainmaa, and A. Hesso. 2011. Exposure to Flame Retardants in Electronics Recycling Sites. Ann Occup Hyg, 55(6), 658-665.

65. Thuresson K, Bergman A, Jakobsson K. Occupational exposure to commercial decabromodiphenyl ether in workers manufacturing or handling flame-retarded rubber. *Environmental Science and Technology.* 39:1980–1986. 2005.

66. Zhou, X., J. Guo, W. Zhang, P. Zhou, J. Deng, and K. Lin. 2014. Tetrabromobisphenol A Contamination and Emission in Printed Circuit Board Production and Implications for Human Exposure. J Hazard Mater, 273(2014), 27-35.

67. NIOSH. Assessment of Occupational Exposure to Flame Retardants. 2014. *https://ntp.niehs.nih.gov/ntp/about_ntp/bsc/2014/dec/nioshupdate_508.pdf*

68. OSHA. OSHA Technical Manual (OTM), OSHA Instruction TED 01-00-015 [TED 1-0.15A]. *https://www.osha.gov/dts/osta/otm/otm_ii/otm_ii_2.html*

69. Gorman Ng, M., Semple, S., Cherrie, J.W., Christopher, Y., Northage, C., Tielemans, C., Veroughstraete, V. and M. Van Tongeren. 2012. The Relationship Between Inadvertent Ingestion and Dermal Exposure Pathways: A New Integrated Conceptual Model and a Database of Dermal and Oral Transfer Efficiencies. Annals of Occupational Hygiene. 56(9):1000-1012.

70. Matsukami, H., N. M. Tue, G. Suzuki, M. Someya, H. Tuyen le, P. H. Viet, S. Takahashi, S. Tanabe, and H. Takigami. Flame retardant emission from e-waste recycling operation in northern Vietnam: Environmental occurrence of emerging organophosphorus esters used as alternatives for PBDEs. *Science of The Total Environment*. 514, 492-499. 2015.

71. Qu, G., A. Liu, T. Wang, C. Zhang, J. Fu, M. Yu, J. Sun, N. Zhu, Z. Li, G. Wei, Y. Du, J. Shi, S. Liu, and G. Jiang. Identification of tetrabromobisphenol A allyl ether and tetrabromobisphenol A 2,3-dibromopropyl ether in the ambient environment near a manufacturing site and in mollusks at a coastal region. *Environmental Science and Technology.* 47(9), 4760-4767. 2013.

72. Schlabach, M., M. Remberger, E. Brorstrom-Lunden, K. Norstrom, L. Kaj, H.

Andersson, D. Herzke, A. Borgen, and M. Harju. Brominated Flame Retardants (BFR) in the Nordic Environment. TemaNord. 2011:528. Nordic Council of Ministers, Copenhagen, Denmark. 2011. *http://www.norden.org/en/publications/publikationer/2011-528*.

73. Wang, J., Liu, L., Wang, J., Pan, B., Fu, X., Zhang, G., ... and Lin, K. Distribution of metals and brominated flame retardants (BFRs) in sediments, soils and plants from an informal e-waste dismantling site, South China. *Environmental Science and Pollution Research*. *22*(2):1020-1033. 2015.

74. Wang, W., K. O. Abualnaja, A. G. Asimakopoulos, A. Covaci, B. Gevao, B. Johnson-Restrepo, T. A. Kumosani, G. Malarvannan, T. B. Minh, H.-B. Moon, H. Nakata, R. K. Sinha, and K. Kannan. A comparative assessment of human exposure to tetrabromobisphenol A and eight bisphenols including bisphenol A via indoor dust ingestion in twelve countries. *Environment International.* 83, 183-191. 2015.

75. Wu, Y, et al. Tetrabromobisphenol A and heavy metal exposure via dust ingestion in an e-waste recycling region in southeast China. *Science of the Total Environment*. 541: 356-364. 2016.

76. Xu, T., J. Wang, S.-z. Liu, C. Lu, W. L. Shelver, Q. X. Li, and J. Li. A highly sensitive and selective immunoassay for the detection of tetrabromobisphenol A in soil and sediment. *Analytica Chimica Acta*. 751, 119-127. 2012.

77. EPA. Leaching studies (OCSPP Test Guideline 835.1240). 2008.

78. Larsen, G., F. Casey, A. Bergman, and H. Hakk. Mobility, Sorption and Fate of Tetrabromobisphenol a (TBBPA) in Loam Soil and Sand. Abstracts of the 2nd International Workshop on Brominated Flame Retardants. Part 2 - Analysis and Fate, Products, Standards and Uses. Stockholm, Sweden. 2001.

https://www.researchgate.net/profile/Tom_Harner/publication/244465065_Measurements_of_OctanolAir_Partition_Coefficients_(_K_OA_)_for_Polybrominated_Diphenyl_Ethers_(PBDEs)_Predicting_Partitioning_in_the_Environment/file/9c960526a70f0039a2.pdf

79. GLCC (Great Lakes Chemical Corporation). The Subchronic Toxicity of Sediment-Sorbed Tetrabromobisphenol a to Chironomus tentans under Flow-through Conditions (Final Report) with Cover Sheet and Letter Dated 101689. Study conducted by Breteler, R. J., J. R. Hoberg, N. Garvey, S. R. Connor, D. A. Hartley, S. P. Shepherd, P. H. Fackler , and P. D. Royal, Springborn Laboratories, Inc., Wareham, MA. OTS# 0525507. Doc ID 40-8998109. 1989.

80. EPA. Simulation tests to assess the primary and ultimate biodegradability of chemicals discharged to wastewater (OPPTS Test Guideline 835.3280). 2008.

81. Nyholm, J. R., C. Lundberg, and P. L. Andersson. Biodegradation kinetics of selected brominated flame retardants in aerobic and anaerobic soil. *Environmental Pollution*. 158(6):2235-2240. 2010.

82. Li, F., J. Wang, B. Jiang, X. Yang, P. Nastold, B. Kolvenbach, L. Wang, Y. Ma, P. F. Corvini, and R. Ji. Fate of tetrabromobisphenol A (TBBPA) and formation of ester- and ether-linked bound residues in an oxic sandy soil. *Environmental Science and Technology*. 49(21):12758-12765. 2015.

83. Potvin, C. M., Z. Long, and H. Zhou. Removal of tetrabromobisphenol a by conventional activated sludge, submerged membrane and membrane aerated biofilm reactors. *Chemosphere*. 89(10):1183-1188. 2012.

84. EPA. Electronics Waste Management in the United States through 2009. EPA 530-R-11-002. Office of Resource Conservation and Recovery, Washington, D. C. 2011.

http://www.epa.gov/osw/conserve/materials/ecycling/docs/fullbaselinereport2011.pdf.

85. Borgnes, D., and B. Rikheim. Decomposition of BFRs and Emission of Dioxins from Co-Incineration of MSW and Electrical and Electronic Plastics Waste. *Organohalogen Compounds*. 66, 890-898. 2004.

86. Gallen, C., A. Banks, S. Brandsma, C. Baduel, P. Thai, G. Eaglesham, A. Heffernan, P. Leonards, P. Bainton, and J. F. Mueller. 2014. Towards Development of a Rapid and Effective Non-Destructive Testing Strategy to Identify Brominated Flame Retardants in the Plastics of Consumer Products. Sci Total Environ, 491-492, 255-265.

87. Guo, Q., Z. Du, Y. Zhang, X. Lu, J. Wang, and W. Yu. 2013. Simultaneous Determination of Bisphenol a, Tetrabromobisphenol a, and Perfluorooctanoic Acid in Small Household Electronics Appliances of "Prohibition on Certain Hazardous Substances in Consumer Products" Instruction Using Ultra-Performance Liquid Chromatography-Tandem Mass Spectrometry with Accelerated Solvent Extraction. J Sep Sci, 36(4), 677-683.

88. Puype, F., J. Samsonek, J. Knoop, M. Egelkraut-Holtus, and M. Ortlieb. 2015. Evidence of Waste Electrical and Electronic Equipment (Weee) Relevant Substances in Polymeric Food-Contact Articles Sold on the European Market. Food Addit Contam Part A Chem Anal Control Expo Risk Assess, 32(3), 410-426.

89. Rani, M., W. J. Shim, G. M. Han, M. Jang, Y. K. Song, and S. H. Hong. 2014. Hexabromocyclododecane in Polystyrene Based Consumer Products: An Evidence of Unregulated Use. Chemosphere, 110, 111-119.

90. Samsonek, J., and F. Puype. 2013. Occurrence of Brominated Flame Retardants in Black Thermo Cups and Selected Kitchen Utensils Purchased on the European Market. Food Addit Contam Part A Chem Anal Control Expo Risk Assess, 30(11), 1976-1986.

91. Washington State DE (Department of Ecology). 2016. Children's Safe Product Act Reports. h*ttps://fortress.wa.gov/ecy/cspareporting/Reports/ReportViewer.aspx?ReportName=ChemicalReportByCASNumber*.

92. Wang, X., X. Hu, H. Zhang, F. Chang, and Y. Luo. Photolysis kinetics, mechanisms, and pathways of tetrabromobisphenol A in water under simulated solar light irradiation. *Environmental Science and Technology*. 49(11):6683-6690. 2015.

93. EPA. Photodegradation in water test (OCSPP Test Guideline 835.2240). 2008.

94. Bao, Y., and J. Niu. Photochemical transformation of tetrabromobisphenol A under simulated sunlight irradiation: Kinetics, mechanism and influencing factors. *Chemosphere*. 134:550-556. 2015.

95. EPA. Indirect photolysis in water test (OCSPP Test Guideline 835.5270). 2008.

96. EC (European Commission). Risk Assessment of 2,2',6,6-Tetrabromo-4,4'-Isopropylidene Diphenol (Tetrabromobisphenol-A): CAS Number: 79-94-7; EINECS Number: 201-236-9; Final Environmental Risk Assessment Report of February 2008. R402_0802_env. Rapporteur: United Kingdom. 2008. *http://echa.europa.eu/documents/10162/17c7379e-f47b-4a76-aa43-060da5830c07*.

97. EPA. Photodegradation in soil test (OCSPP Test Guideline 835.2410). 2008.

98. GLCC (Great Lakes Chemical Corporation). 1989. Determination of the Biodegradability of Tetrabromobisphenol A in Soil under Aerobic Conditions. Final Report. Study conducted by, Springborn Life Sciences, Inc., (January 20, 1989), Wareham, MA. OTS# 0525513. Doc ID 42083 G3-2.

99. GLCC (Great Lakes Chemical Corporation). 1989. Determination of the

Biodegradability of Tetrabromobisphenol A in Soil under Anaerobic Conditions (Final Report) with Attachments and Cover Letter Dated 013189. Study conducted by, Springborn Life Sciences, Inc., (January 19, 1989), Wareham, MA. OTS# 0525513. Doc ID 42083 G3-2.

100. Liu, J., Y. Wang, B. Jiang, L. Wang, J. Chen, H. Guo, and R. Ji. Degradation, metabolism, and bound-residue formation and release of tetrabromobisphenol A in soil during sequential anoxic-oxic incubation. *Environmental Science and Technology*. 47(15):8348-8354. 2013.

101. EPA. Aerobic and anaerobic transformation in soil test (OECD Test Guideline 307). 2008.

102. EPA. Terrestrial soil-core microcosm test (OCSPP Test Guideline 850.4900). 2008.

103. NITE (National Institute of Technology and Evaluation). #32: Bioaccumulation: Aquatic/Sediment for TBBPA (CASRN 79-94-7). Japan Chemicals Collaborative Knowledge Database, Ministry of Economy, Trade and Industry and Ministry of the Environment, Japan. 2010. *http://www.safe.nite.go.jp/jcheck/template.action?ano=849andmno=4-0205andcno=79-94-7andrequest_locale=en* (retrieved on November 14, 2014)

104. Fackler, P. Tetrabromobisphenol A. Determination of Biodegradability in a Sediment/Water Microbial System. SLS Report 89-8-3070. Springborn Life Sciences, Inc., Wareham, MA. 1989. *http://www.epa.gov/chemrtk/pubs/summaries/phenolis/c13460rr3.pdf.* (retrieved in 2006)

105. EPA. Aerobic mineralization in surface water-simulation biodegradation test (OCSPP Test Guideline 835.3190). 2008.

106. EPA. Flame Retardants in Printed Circuit Boards: Final Report. EPA Publication 744-R-15-001. Design for the Environment (now Safer Choice), Washington, DC. 2015. *https://www.epa.gov/sites/production/files/2015-08/documents/pcb_final_report.pdf.* (retrieved in 2017)

107. EPA. Episuite (Estimation Programs Interface). 2000-2012. *https://www.epa.gov/tsca-screening-tools/epi-suitetm-estimation-program-interface* (retrieved in 2017)

108. EPA (n.d.). *Series 830 - Product Properties Test Guidelines. https://www.epa.gov/testguidelines-pesticides-and-toxic-substances/series-830-product-properties-test-guidelines* (retrieved in 2016)

109. EPA (n.d.). *Series 850 – Ecological Effects Test Guidelines. https://www.epa.gov/test-guidelines-pesticides-and-toxic-substances/series-850-ecological-effects-test-guidelines* (retrieved in 2016)

110. EPA (n.d.). *Series 870 – Health Effects Test Guidelines. https://www.epa.gov/test-guidelines-pesticides-and-toxic-substances/series-870-health-effects-test-guidelines* (retrieved in 2016)

111. EPA. ECOSAR v1.11. 2012. *https://www.epa.gov/tsca-screening-tools/ecological-structure-activity-relationships-ecosar-predictive-model* (retrieved in 2017)

112. OECD Test No. 421: Reproductive/Developmental Toxicity Screening Test. *OECD Guidelines for the Testing of Chemicals, Section 4: Health Effects*. OECD Publishing, Paris. 2007.

113. NTP (National Toxicology Program) (n.d.). *Modified One-Generation Studies.*

https://ntp.niehs.nih.gov/testing/types/mog/index.html (retrieved in 2016)

114. OECD Test No. 426: Developmental Neurotoxicity Study. *OECD Guidelines for the Testing of Chemicals, Section 4: Health Effects*. OECD Publishing, Paris. 2007.

115. Lilienthal, H., C. M. Verwer, L. T. van der Ven, A. H. Piersma, and J. G. Vos. Exposure to Tetrabromobisphenol a (TBBPA) in Wistar Rats: Neurobehavioral Effects in Offspring from a One-Generation Reproduction Study. *Toxicology*. 246(1), 45-54. 2008.

116. MPI Research. An Oral Two Generation Reproductive, Fertility and Developmental Neurobehavioral Study of Tetrabromobisphenol-A in Rats (Unpublished). 2002.

117. MPI Research. Amendment to the Final Report. An Oral Two Generation Reproductive, Fertility and Developmental Neurobehavioural Study of Tetrabromobisphenol-A in Rats (Unpublished Report). 2003.

118. Zatecka, E., L. Ded, F. Elzeinova, A. Kubatova, A. Dorosh, H. Margaryan, P. Dostalova, and J. Peknicova. Effect of tetrabromobisphenol A on induction of apoptosis in the testes and changes in expression of selected testicular genes in CD1 mice. *Reproductive Toxicology*. 35:32-39. 2013.

119. Hass, H., C. Wamberg, O. Ladefoged, M. Dalgaard, H. Rye Lam, and A. Vinggard. Developmental Neurotoxicity of Tetrabromobisphenol A in Rats (Unpublished; Cited in EC, 2006). 2003.

120. MPI Research. Final Report - an Oral Prenatal Developmental Toxicity Study with Tatrabromobisphenol-A in Rats (Unpublished). 2001.

121. Noda, T., S. Morita, S. Ohgaki, and M. Shimizu. Safety Evaluation of Chemicals for Use in Household Products (VII) Teratological Studies on

Tetrabromobisphenol-A in Rats. Annual Report of the Osaka Institute of Public Health and Environmental Sciences, 48, 106-112. 1985.

122. VCC (Velsicol Chemical Corporation). Pilot Teratology Study in Rats with Tetrabromobisphenol A with Cover Letter Dated 04/17/78. 0200479. 1978.

123. Eriksson, P., E. Jakobsson, and A. Fredriksson. Developmental Neurotoxicity of Brominated Flame Retardants, Polybrominated Diphenyl Ethers, and Tetrabromobisphenol A. Organohalogen Compounds, 35, 375-377. 1998.

124. Eriksson, P., E. Jakobsson, and A. Frederiksson. Brominated Flame Retardants: A Novel Class of Developmental Neurotoxicants in Our Environment? *Environmental Health Perspectives*. 109, 903-908. 2001.

125. Fukuda, N., Y. Ito, M. Yamaguchi, K. Mitumori, M. Koizumi, R. Hasegawa, E. Kamata, and M. Ema. Unexpected Nephrotoxicity Induced by Tetrabromobisphenol a in Newborn Rats. *Toxicology Letters*. 150, 145-155. 2004.

126. Kim, B., E. Colon, S. Chawla, L. N. Vandenberg, and A. Suvorov. Endocrine disruptors alter social behaviors and indirectly influence social hierarchies via changes in body weight. *Environmental health : a global access science source*. 14, 64. 2015.

127. Saegusa, Y., H. Fujimoto, G. H. Woo, K. Inoue, M. Takahashi, K. Mitsumori, A. Nishikawa, and M. Shibatani. Developmental Toxicity of Brominated Flame Retardants, Tetrabromobisphenol a and 1,2,5,6,9,10-Hexabromocyclododecane, in Rat Offspring after Maternal Exposure from Mid-Gestation through Lactation. *Reproductive Toxicology*. 28, 456-467. 2009.

128. Saegusa, Y., H. Fujimoto, G. H. Woo, T. Ohishi, L. Wang, K. Mitsumori, A. Nishikawa, and M. Shibutani. Transient Aberration of Neuronal Development in the

Hippocampal Dentate Gyrus after Developmental Exposure to Brominated Flame Retardants in Rats. *Archives of Toxicology*. 86(9), 1431-1442. 2012.

129. Tada, Y., T. Fujitani, N. Yano, H. Takahashi, K. Yuzawa, H. Ando, Y. Kubo, A. Nagasawa, A. Ogata, and H. Kamimura. Effects of Tetrabromobisphenol a, a Brominated Flame Retardant, in ICR Mice after Prenatal and Postnatal Exposure. *Food and Chemical Toxicology*. 44(8), 1408-1413. 2006.

130. Viberg, H., and P. Eriksson. Differences in Neonatal Neurotoxicity of Brominated Flame Retardants, PBDE 99 and TBBPA, in Mice. *Toxicology*. 289(1), 59-65. 2011.

131. Kicinski, M., M. K. Viaene, E. D. Hond, G. Schoeters, A. Covaci, A. C. Dirtu, V. Nelen, L. Bruckers, K. Croes, I. Sioen, W. Baeyens, N. Van Larebeke, and T. S. Nawrot. 2012. Neurobehavioral Function and Low-Level Exposure to Brominated Flame Retardants in Adolescents: A Cross-Sectional Study. Environmental Health, 11, 1-12.

132. EPA. Larval amphibian growth and development assay (LAGDA) (OCSPP Test Guideline 890.2300). 2002.

133. ACC. HPV Data Summary and Test Plan for Phenol, 4,4'-Isopropylidenbis[2,6-Dibromo- (Tetrabromobisphenol a, TBBPA). Test plan revision/updates, revised test plan. Robust summaries & test plans: Phenol, 4,4'-isopropylidenbis[2,6-dibromo-. 2006. (retrieved in 2013) http://www.epa.gov/chemrtk/pubs/summaries/phenolis/c13460rt3.pdf.

134. Garber, E. A. E., G. L. Larsen, H. Hakk, and A. Bergman. Frog Embryo Teratogenic Assay: Xenopus (FETAX) Analysis of the Biological Activity of

Tetrabromobisphenol a (TBBPA). Poster presentation at Second International Workshop on Brominated Flame Retardants, May 14-16, Stockholm University, Sweden. 2001.

135. Balch, G. C., and C. D. Metcalfe. *In Vivo* Toxicity Testing of PBDEs Using Early Life Stages of the Japanese Medaka and the Xenopus Tail Resorption Model. 3rd Annual Workshop on Brominated Flame Retardants in the Environment. Canada Centre for Inland Waters, August 23-24, pp. 59-60. 2001. (as cited in EC, 2006 and ACC, 2006).

136. Brown, D. D., Z. Wang, J. D. Furlow, A. Kanamori, R. A. Schawartzman, B. F. FRemo, and A. Pinder. The thyroid hormone-induced tail resorption program during *Xenopus laevis* metamorphosis. *Developmental Biology*. 93:1924-1929. 1996.

137. Hanada, H., K. Katsu, T. Kanno, E. F. Sato, A. Kashiwagi, J. Sasaki, M. Inoue, and K. Utsumi. Cyclosporin a Inhibits Thyroid Hormone-Induced Shortening of the Tadpole Tail through Membrane Permeability Transition. *Comparative Biochemistry and Physiology*. Part B, 135, 473-483. 2003.

138. Kashiwagi, A., H. Hanada, M. Yabuki, T. Kanno, R. Ishisaka, J. Sasaki, M. Inoue, and K. Utsumi. Thyroxine Enhancement and the Role of Reactive Oxygen Species in Tadpole Tail Apoptosis. *Free Radical Biology and Medicine*. 26(7/8), 1001-1009. 1999.

139. Veldhoen, N., A. Boggs, K. Walzak, and C. C. Helbing. Exosure to Tetrabromobisphenol-a Alters Th-Associated Gene Expression and Tadpole Metamorphosis in the Pacific Tree Frog Pseudacris regilla. *Aquatic Toxicology*. 78, 292-302. 2006.

List of Subjects in 40 CFR Chapter I

Environmental protection, Flame retardants, Hazardous substances, tetrabromobisphenol A.

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