

not interfere with continued maintenance of the 1997 ozone NAAQS in the Area Middle Tennessee Area, or with any other applicable CAA requirement, has been placed in the public docket for this action.

#### V. Legal Authority

The statutory authority for this action is granted to the EPA by Sections 211(h) and 301(a) of the Clean Air Act, as amended; 42 U.S.C. 7545(h) and 7601(a).

#### List of Subjects in 40 CFR Part 80

Environmental protection, Administrative practice and procedures, Air pollution control, Fuel additives, Gasoline, Incorporation by reference, Motor vehicle and motor vehicle engines, Motor vehicle pollution, Penalties, Reporting and recordkeeping requirements.

Dated: April 4, 2017.

**E. Scott Pruitt,**

*Administrator.*

[FR Doc. 2017-07399 Filed 4-11-17; 8:45 am]

**BILLING CODE 6560-50-P**

## ENVIRONMENTAL PROTECTION AGENCY

### 40 CFR Chapter I

[EPA-HQ-OPPT-2017-0038; FRL-9961-04]

#### Chlorinated Phosphate Ester (CPE) Cluster; 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 January 6, 2017. The petitioners requested that EPA issue an order under TSCA section 4, requiring that testing be conducted by manufacturers and processors of chlorinated phosphate esters ("CPE"). The CPE Cluster is composed of tris(2-chloroethyl) phosphate ("TCEP") (CAS No. 115-96-8), 2-propanol, 1-chloro-, phosphate ("TCPP") (CAS No. 13674-84-5), and 2-propanol, 1,3- dichloro-, phosphate ("TDCPP") (CAS No. 13674-87-8). 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 April 6, 2017.

#### FOR FURTHER INFORMATION CONTACT:

*For technical information contact:* Hannah Braun, Chemical Control Division (7405M), Office of Pollution Prevention and Toxics, Environmental Protection Agency, 1200 Pennsylvania Ave. NW., Washington, DC 20460-0001; telephone number: (202) 564-5614; email address: [braun.hannah@epa.gov](mailto:braun.hannah@epa.gov).

*For general information contact:* The TSCA-Hotline, ABVI-Goodwill, 422 South Clinton Ave., Rochester, NY 14620; telephone number: (202) 554-1404; email address: [TSCA-Hotline@epa.gov](mailto:TSCA-Hotline@epa.gov).

#### 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 or process the chemicals tris(2-chloroethyl) phosphate ("TCEP") (CAS No. 115-96-8), 2-propanol, 1-chloro-, phosphate ("TCPP") (CAS No. 13674-84-5), and 2-propanol, 1,3- dichloro-, phosphate ("TDCPP") (CAS No. 13674-87-8). 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-2017-0038, 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 an order 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 January 6, 2017, 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 and processors of the chlorinated phosphate esters (“CPE”) Cluster composed of tris(2-chloroethyl) phosphate (“TCEP”) (CAS No. 115–96–8), 2-propanol, 1-chloro-, phosphate (“TCPP”) (CAS No. 13674–84–5), and 2-propanol, 1,3- dichloro-, phosphate (“TDCPP”) (CAS No. 13674–87–8) (Ref. 1).

#### B. What support do the petitioners offer?

The petitioners cite to section 4(a)(1) of TSCA, which requires EPA to direct testing on a chemical substance or mixture if the Administrator 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 the CPE Cluster chemicals “may present an unreasonable risk of injury to health or the environment” because there is substantial evidence that chemicals in the CPE Cluster may be toxic, including:

- EPA’s TSCA Work Plan Chemical Problem Formulation and Initial Assessment—Chlorinated Phosphate Ester Cluster Flame Retardants (heretofore referred to as Problem Formulation and Initial Assessment), which cites multiple mammalian toxicity studies showing adverse effects caused by the cluster members such as reproductive and developmental effects, neurological effects, liver, kidney and thyroid effects and cancer (for certain cluster members) (Refs. 2–7).
- EPA’s Problem Formulation and Initial Assessment, which also states

that ecological toxicity from exposure to TCEP and TDCPP was exhibited in acute tests with fish resulting in loss of coordination, edema, darker pigmentation and hyperventilation (Ref. 2).

- EPA’s Design for the Environment in which the Agency conducted a hazard assessment of the chemicals in the CPE cluster and found that each of the three cluster members are considered a high hazard for more than one human health effect, as well as for aquatic toxicity, based on empirical data. Additionally, TCPP and TDCPP are considered to be highly persistent (Ref. 8).

- The state of California finds TDCPP to be a “known carcinogen,” and in 2011 California added TDCPP to the list of chemicals requiring warning labels under California Proposition 65 law (Ref. 9, 10).

- California’s Proposition 65 list of chemicals where TCEP was “known to the State to cause cancer” in 1992 (Ref. 11).

- The European Union (EU) classifying TCEP as a “Substance of Very High Concern” based on reproductive toxicity (Ref. 12).

- California’s Safer Consumer Products program listing TCPP as a candidate chemical based on carcinogenicity (Ref. 13).

The petitioners assert there are CPE Cluster chemicals exposure to humans and the environment based on the following information provided in EPA’s Problem Formulation and Initial Assessment (Ref. 2).

- Several studies of U.S. drinking water where CPEs have been detected (Refs. 14–16).

- Numerous studies where concentrations of CPEs in infant products such as high chairs, bath mats, car seats, nursing pillows, carriers, sofas, and camping tents have been measured (Refs. 17–21).

- Small children may have additional exposures through contact with baby products containing CPEs and via mouthing behaviors (Ref. 2).

- A number of published studies where levels of CPEs in indoor air and dust have been reported (Refs. 19–49).

- Several studies throughout the United States and abroad which reported levels of the CPEs in surface water. Collectively, these data indicate high potential for exposures to ecological receptors, and in particular, aquatic organisms (Refs. 50–77).

- A study where TCEP, TCPP, and TDCPP have all been measured in herring gull eggs from the Lake Huron area (Ref. 78).

With the evidence of toxicity and exposure the petitioners argue that the chemicals in the CPE Cluster meet the criteria for “may present an unreasonable risk of injury to health or the environment.”

The petitioners also assert there is “insufficient information” on the CPE Cluster chemicals. They indicate that EPA’s Problem Formulation and Initial Assessment (Ref. 2) “identifies seven critical data gaps around exposures and hazards of these flame retardants”. While EPA disagrees that the Problem Formulation and Initial Assessment specifically identifies those which the petitioners assert, the petition lists the following seven data gaps around exposures and hazard of CPE flame retardants:

Exposure pathways: Dermal and inhalation;

2. Hazard: Reproduction and endocrine toxicity;

3. Exposure: Environmental releases from non-industrial uses;

4. Exposure: Community and worker exposures from manufacturing, processing, industrial and non-industrial uses;

5. Exposure: Community and worker exposures recycling;

6. Exposure: Community, worker and environmental exposures from disposal; and

7. Hazard: Toxicity to birds, wildlife, sediment organisms.

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 the CPE Cluster chemicals.

### IV. Disposition of TSCA Section 21 Petition

#### A. What was EPA’s response?

After careful consideration, EPA 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. 79), 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 the CPE Cluster chemicals in August 2015 (Ref. 2). As stated on EPA’s Web site titled “Assessments for TSCA Work Plan Chemicals” (Ref. 80), “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 the CPE Cluster chemicals, EPA followed an approach developed for assessing chemicals under TSCA as it existed at that time. In addition, in Table 2–1 of the Problem Formulation and Initial Assessment (Ref. 2), EPA specified, in very general terms, the nature and type of information sought to inform this particular risk assessment, under the existing TSCA framework.

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 information and presented it in the Problem Formulation and Initial Assessment, EPA did not necessarily undertake a comprehensive search of available information 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 information had been identified. (Ref. 2).

As EPA explains on its Web site, “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’s approach and provide additional information/data to supplement or refine our assessment approach prior to EPA conducting detailed risk analysis and risk characterization” (Ref. 80).

EPA’s 2015 Problem Formulation and Initial Assessment for the CPE Cluster chemicals does not constitute a full risk assessment for the chemicals in the CPE Cluster, 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. 81). 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 the CPE Cluster chemicals.

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. 83). This interpretation of “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 evaluations. 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 Problem Formulation and Initial Assessment (Ref. 2).

EPA agrees that the manufacture, distribution in commerce, processing, use, or disposal of the CPE Cluster chemicals 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 the CPE Cluster chemicals, 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. 82 and 83). However, EPA does not find that the petitioners have demonstrated, for each exposure pathway and hazard endpoint presented in the petition, that the 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” (or any combination of such activities) of the CPE Cluster chemicals 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 that for each requested test the information on the individual exposure pathways and hazard endpoints identified by the petitioners do not demonstrate that there is insufficient information upon which the effects of the CPE Cluster chemicals 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 the CPE Cluster chemicals. The toxicokinetics test (Organization for Economic Co-operation and Development (OECD) Test Guideline 417) (Ref. 84), in vivo absorption test (OECD Test Guideline 427) (Ref. 85) and dermal toxicity test (OPPTS Test Guideline 870.1200) (Ref. 86) requested by the petitioners may not be needed. In the Problem Formulation and Initial Assessment, EPA stated that risk from the dermal exposure pathway could not be quantified for risk assessment because of a lack of route-specific toxicological data, but also indicated that an alternative approach, *i.e.*, development of a PBPK model for oral, inhalation and dermal routes of exposure would provide the ability to perform route-to-route extrapolation. The Problem Formulation and Initial Assessment indicated that adequate toxicokinetic data would be needed for each route of exposure and that these data are lacking for dermal exposures. However, since the publication of the Problem Formulation and Initial Assessment document, EPA has identified pharmacokinetic data including absorption, bioaccessibility

and absorption, distribution, metabolism and excretion (ADME) data (Refs. 7, 87–96) that could be used to perform route-to-route extrapolation from oral toxicity studies to predict effects from dermal exposure to the CPE Cluster chemicals.

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. 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 the CPE Cluster chemicals. The toxicokinetics test (OECD Test Guideline 417: Toxicokinetics) (Ref. 84) and inhalation toxicity test (OPPTS Test Guideline 870.1300: Acute Inhalation Toxicity) (Ref. 98) requested by the petitioners may not be needed. In the Problem Formulation and Initial Assessment, EPA stated that risk from the inhalation exposure pathway could not be quantified for risk assessment because of a lack of route-specific toxicological data, but also indicated that an alternative approach, *i.e.*, development of a PBPK model for oral, inhalation and dermal routes of exposure would provide the ability to perform route-to-route extrapolation. The Problem Formulation and Initial Assessment, indicated that adequate toxicokinetic data would be needed for each route of exposure and that these data are lacking for inhalation exposures. However, since the publication of the Problem Formulation and Initial Assessment, EPA has identified toxicological data including, acute toxicity, bioaccessibility and ADME data (Refs. 7, 87–89, 93, 99 and 100) that could be used in route-to-route extrapolation from oral toxicity studies to predict effects from inhalation exposure to the CPE Cluster chemicals. As proposed in the Problem Formulation and Initial Assessment, CPE Cluster chemicals that are absorbed to and inhaled associated with particles, once the particles are in the gastrointestinal tract, absorption would be the same as in the oral toxicity studies and hence, oral toxicity studies can be used to determine or predict effects to health from inhalation exposure to the CPE cluster substances. Current literature on bioaccessibility (Ref. 89) could also be used to refine the estimate of the amount of the CPE Cluster chemicals absorbed via ingestion of particles (via inhalation and translocation to the gut).

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. *Reproductive and Endocrine Toxicity. a. Reproductive Toxicity.* The petition does not set forth facts demonstrating that there is insufficient data available to EPA to reasonably determine or predict the reproductive toxicity of the CPE Cluster chemicals. The NTP Modified One Generation study (Ref. 102) or the alternatively suggested in vivo reproductive toxicity screening test (OPPTS 870.3800: Reproduction and Fertility Effects) (Ref. 103) based on two-generation reproduction toxicity test (OECD Test Guideline 416) (Ref. 104), requested by the petitioners, may not be needed. Although EPA states in the Problem Formulation and Initial Assessment that “given uncertainty surrounding the impact of long-term exposures and male reproductive toxicity, it would not be possible to quantify risks at this time,” EPA now believes, after further review and consideration of existing studies, that the Agency could use information identified in the Problem Formulation and Initial Assessment, as well as new information identified through comprehensive literature searches, data from alternative testing approaches, and read-across (in which data for one structurally similar chemical can be used to assess the toxicity of another) could be used to conduct an assessment of effects of the CPE Cluster chemicals on reproduction (Ref. 2). As presented in the Problem Formulation and Initial Assessment, EPA identified several studies for each chemical in the CPE Cluster to assess reproductive effects. Specifically, a multi-generation reproductive and developmental toxicity study in mice for TCEP (Ref. 105) and a two-generation reproductive and developmental study in rats for TCPP (Ref. 106, test data currently listed as CBI) were identified. For TDCPP, a reproduction study in male rabbits (Ref. 7), two developmental toxicity studies in female rats (Refs. 7 and 107) and a two-year cancer bioassay in rats, which included evaluation of effects on reproductive organs (Ref. 108), are already available.

Since the publication of the Problem Formulation Initial Assessment document, EPA identified additional reproductive studies. Specifically, TCPP has been evaluated in a developmental toxicity study (Ref. 109). The results of this study have not yet been released, but are expected to be available to EPA

prior to initiation of a Risk Evaluation for TCPP. EPA has also identified studies using alternative animal models and *in vitro* tests that could inform the evaluation of reproductive toxicity (Refs. 110–117). Finally, given the structural similarity of the three chemicals in the CPE Cluster, EPA could consider read-across approaches, using data from one chemical to characterize the hazards of another chemical. Collectively, the studies identified in the Problem Formulation and Initial Assessment document, the studies identified since the release of the Problem Formulation and Initial Assessment document, and read-across approaches, could be used to characterize reproductive toxicity for the CPE Cluster chemicals.

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. Endocrine Activity.* The petition does not set forth facts demonstrating that there is insufficient information available to EPA to reasonably determine or predict the effects of the CPE Cluster chemicals on endocrine activity. EPA believes that the Larval Amphibian Growth and Development Assay (OCSPP 890.2300) (Ref. 118) or the alternatively suggested NTP Modified One Generation Study (Ref. 102) requested by the petitioners may not be needed. EPA's Problem Formulation and Initial Assessment stated that data were conflicting with regard to endocrine activity, which made it difficult to make a determination in the pre-assessment phase. However, EPA did not consider the information to be insufficient; rather EPA intended to defer drawing conclusions until the assessment phase when additional, comprehensive review of all available data would be conducted.

A number of studies evaluating thyroidal and other endocrine effects are available, including the reproduction and developmental toxicity studies described in Unit IV.C.2.a. (Refs. 7, 105, 106 and 108), as well as studies using alternative animal models and *in vitro* tests (Refs. 110–117) identified since the Problem Formulation and Initial Assessment. An evaluation of each study as well as the full body of

evidence (*i.e.*, weight of evidence) would be undertaken to identify endocrine-related hazard concerns. 3. *Environmental Releases from Non-Industrial and Consumer Uses.* The petition does not set forth facts demonstrating that there is insufficient information available to EPA to reasonably determine or predict effects of the CPE Cluster chemicals associated with environmental releases from non-industrial and consumer uses nor specifically the potential contribution of down-the-drain releases of the CPE Cluster chemicals in United States waters. EPA agrees with the petitioner's suggestion that existing data (*e.g.*, effluent and influent of wastewater) could be used to estimate environmental concentrations of the CPE Cluster chemicals from consumer and down-the-drain uses. Hence, development of sampling plans for effluent waters from municipal treatment plants and analytical methods for measuring the CPE Cluster chemicals may not be needed.

While EPA's Problem Formulation and Initial Assessment indicated that contributions of non-industrial and consumer uses to water and wastewater were not quantifiable, EPA's conceptual model did indicate that exposures to water and wastewater (aggregated from all sources) would be assessed. EPA agrees, as the petition suggests, that existing effluent and influent from wastewater could likely be used to predict environmental concentrations of the CPE Cluster chemicals from consumer and other down-the-drain uses. As identified in the Problem Formulation and Initial Assessment, there are over 100 available monitoring studies that could be used to characterize concentrations of the CPE Cluster chemicals in water and wastewater. Monitoring studies range from nationwide studies with larger sample sizes and consistent analytical methods such as United States Geological Survey (USGS), to targeted studies with generally smaller sample sizes and variable analytical methods.

In addition, several studies from other countries are also available to characterize the CPE Cluster chemicals in water and wastewater. Since the publication and Problem Formulation and Initial Assessment document, an Australian study (Ref. 124), sampled for all three members of the CPE Cluster in

11 waste water treatment plants (Ref. 124). Another study, identified in the Problem Formulation and Initial Assessment, compares influent water concentrations between the U.S. and Sweden (Ref. 29) and indicates that U.S. concentration values are comparable to Sweden, suggesting that data from Sweden could also be considered in a U.S. assessment.

EPA has identified existing effluent data from municipal treatment plants for TCEP and TDCPP from the U.S. Geological Survey National Water Information System (Ref. 121) since the publication of the Problem Formulation and Initial Assessment document. Several other studies also indicate the presence of CPE Cluster chemicals in U.S. wastewater (Refs. 55 and 122). One study shows low levels of TCEP in a sample from U.S. industrial laundry wastewater (Ref. 123), a potential down-the-drain contributor to treatment plant effluent. Other wastewater samples in the industrial laundry study showed non-detect levels of TCEP. EPA agrees with the petitioners that these types of data may be especially useful to estimate potential contributions from down-the-drain uses to water and wastewater CPE concentrations. Hence, as the petitioners suggest, EPA could use a combination of existing occurrence data, especially effluent and influent of wastewater from municipal treatment plants (*e.g.*, U.S. effluent data and non-U.S. data) to determine or predict contributions from non-industrial and consumer uses, including the potential contribution of down-the-drain releases. EPA believes that the monitoring and effluent data described previously, as well as additional data that describes non-industrial or consumer sources to wastewater (Ref. 125) that may be identified during prioritization of the CPE Cluster for risk evaluation is likely sufficient for characterizing risk from exposures to water and wastewater and for assessing potential contributions from non-industrial and consumer down-the-drain releases of the CPE Cluster chemicals. As the petitioners point out, this approach of using existing monitoring data and especially wastewater effluent data has been used by others (*i.e.*, Environment and Climate Change Canada) to assess the potential contribution to down-the-drain releases (Ref. 2).

EPA believes that the development of analytical methods for the determination and quantification of the CPE Cluster chemicals in sampled waters and the development of a strategy for sampling effluent waters from municipal treatment plants as requested by the petitioners is not needed at this time. Analytical methods for TCEP, TCPP and TDCPP already exist as evidenced by measurements performed by the USGS and other laboratories (Refs. 119 and 120). The petition does not establish why these are insufficient. *4. Exposure from manufacturing, processing, industrial and non-industrial uses. a. Communities.* 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 air, soil and water in communities near manufacturing, processing, industrial and non-industrial use facilities of the CPE Cluster chemicals. The petitioners state that in the absence of facility specific Toxic Release Inventory (TRI) data, other information sources should be used to identify relevant facilities to monitor near. EPA agrees with the petitioners that other sources of information, such as Chemical Data Reporting (CDR), can be used to identify relevant facilities on which exposure estimates could be made.

Although the Problem Formulation and Initial Assessment states that chemical-specific environmental release data to air, soil and water from industrial sites could not be found (Ref. 2), EPA believes that approaches other than site-specific monitoring could be used to assess potential exposures from manufacturing, processing, industrial and non-industrial uses. EPA believes it could be reasonable to estimate or model releases from facilities and concentrations in the surrounding environments using established EPA models such as ChemSTEER, E-FAST and AERMOD. ChemSTEER is a model to estimate workplace exposure and environmental releases (Ref. 126). E-FAST is a tool to estimate concentrations of chemicals released to air, water, landfills and consumer products (Ref. 127). AERMOD is a model to estimate chemical emissions from stationary industrial sources (Ref. 128). All of these models have been extensively reviewed and validated based on comparisons with monitoring data. These modeled estimates could be compared to existing U.S. monitoring data, which is not site-specific, and non-U.S. data associated with industrial facilities to assess the modeling

approaches. Monitoring data exist for the CPE Cluster chemicals. As identified in the Problem Formulation Initial Assessment, there are over 100 available monitoring studies that could be used to characterize concentrations of the CPE Cluster chemicals in various media (Ref. 2).

*Air.* The petition does not set forth facts demonstrating that there is insufficient information available to EPA to reasonably determine or predict effects from exposure through air in communities near manufacturing, processing, industrial and non-industrial use facilities of the CPE Cluster chemicals. 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. 129), in the vicinity of representative manufacturing and processing facilities, as requested by the petitioners may not be necessary. EPA could use existing approaches, such as modeling (ChemSTEER, E-FAST and AERMOD) (Refs. 126–128) along with existing data to estimate releases and air concentrations near facilities for the CPE Cluster chemicals.

The modeled data in combination with measurements of the CPE Cluster chemicals in ambient air as identified in the Problem Formulation and Initial Assessment for the U.S. and abroad (Refs. 40, 49, 130 and 131), could be used to estimate air concentrations in communities near manufacturing and processing facilities. However, the petition does not address these possibilities, let alone explain why a testing order under section 4 would be necessary at this point. EPA considers this approach to be reasonable to determine exposure to communities near manufacturing and processing facilities, but may decide to pursue targeted sampling in the future near manufacturing and processing facilities to reduce uncertainty.

*Soil.* The petition does not set forth facts demonstrating that there is insufficient information available to EPA to reasonably determine or predict effects from exposure through soil in communities near manufacturing, processing, industrial and non-industrial use facilities of the CPE Cluster chemicals. Soil sampling, using EPA methods, in the vicinity of representative manufacturing and processing facilities, as requested by the petitioners may not be necessary. Although the Problem Formulation and Initial Assessment stated that “Studies of soil with measured U.S. values are not readily available” (Ref. 2 Page 67),

EPA could use a combination of models (e.g. ChemSTEER and AERMOD) to predict deposition to soil near facilities in conjunction with predicted environmental releases to air. The modeled data in combination with measurements of the CPE Cluster chemicals in other media such as sludge, biosolids, and effluent as identified in the Problem Formulation and Initial Assessment (Refs. 40, 55, 122, 132 and 133) could be used to estimate soil concentrations from land application of sludge and effluent. There is also a study in Germany, identified since the publication of the Problem Formulation and Initial Assessment, showing concentrations (ranging from approximately 2–20 µg/kg dry weight) of TCEP and TCPP in soil from grasslands and two urban sites (Ref. 134) which also could be evaluated for use in predicting soil concentrations in communities near manufacturing and processing facilities. However, the petition does not address these possibilities, let alone explain why a testing order under section 4 would be necessary at this point. EPA considers this approach to be reasonable to determine exposure to communities near manufacturing and processing facilities, but may decide to pursue targeted sampling in the future near manufacturing and processing facilities to reduce uncertainty.

*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 through water in communities near manufacturing, processing, and industrial and non-industrial use facilities of the CPE Cluster chemicals. Sampling studies, especially for various types of water (e.g., drinking water, surface water, and ground water) may not be necessary. EPA could use existing measured chemical-specific environmental data and modeling to estimate releases and water concentrations near facilities.

For example, surface water concentrations near known facilities can be estimated using existing approaches, such as E-FAST and ChemSTEER along with estimated releases from these activities (Refs. 126 and 127). As identified in the Problem Formulation and Initial Assessment, data are available for surface water concentrations of TCEP and TDCPP from USGS NWIS as well as other studies. Surface water monitoring data for TCPP are available in the open literature (Refs. 50, 55 and 135). Groundwater concentrations near known facilities can also be characterized using models such as E-

FAST and ChemSTEER (Refs. 126 and 127).

Furthermore, groundwater data are available for TCEP and TDCPP from USGS NWIS in addition to other monitoring studies that have reported concentrations (generally ranging from non-detect to approximately 1 µg/L) for all three CPE Cluster chemicals (Refs. 65 and 136).

As with surface and groundwater, drinking water concentrations near known facilities could also be estimated from releases using modeling (e.g., E-FAST and ChemSTEER). Furthermore, drinking water data from samples taken at drinking water treatment plants are available for TCEP, TCEP and TDCPP from several studies that have reported concentrations generally ranging from non-detect to approximately 1 µg/L (Refs. 14–16 and 137).

In summary, EPA could use modeled data in combination with measurements of the CPE Cluster chemicals in water to estimate water concentrations in communities near manufacturing and processing facilities. However, the petition does not address these possibilities, let alone explain why a testing order under section 4 would be necessary at this point. EPA considers this approach to be reasonable to determine exposure to communities near manufacturing and processing facilities, but may decide to pursue targeted sampling in the future near manufacturing and processing facilities to reduce uncertainty.

*b and c. Workers (Industrial and Non-Industrial).* The petition states that “Occupational assessments, including biological and environmental monitoring, should be conducted in representative manufacturing, processing and industrial use facilities” and that “Occupational assessments based on personal monitoring should be used for non-industrial workers” (Ref. 1).

*Air Sampling.* 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 the CPE Cluster chemicals through air for workers in manufacturing, processing, industrial and non-industrial use facilities. EPA believes that a combination of modeled data and existing data (e.g., non-U.S. data for similar activities/scenarios) could be used to determine or predict effects on workers exposed to air containing the CPE Cluster chemicals in an industrial and non-industrial environment.

The CPE Problem Formulation and Initial Assessment document states that EPA’s lack of toxicity data for inhalation

and dermal routes of exposure as the basis for not further elaborating these exposure pathways. However, as described in Unit IV.C.1., EPA has described data and approaches that may be useful in filling these data gaps such that this may not be a critical data gap going forward. Additionally, the petitioners cited a report from the National Institute of Occupational Safety and Health (NIOSH) titled: “Assessment of Occupational Exposure to Flame Retardants” that aims to quantify and characterize occupational exposure routes (inhalation, ingestion, or dermal) for CPE Cluster chemicals as potentially useful for EPA to consider (Ref. 138). EPA agrees that this report appears to include a number of scenarios and measurements for which the petitioners are asking for testing and that EPA would consider any relevant information that results from this ongoing study. However, the petition fails to explain how it considered worker exposure or why a testing order under section 4 would be necessary for additional information.

If measured data are not available, it is still possible to assess exposure using modelling approaches. Specifically, EPA’s ChemSTEER could be used to estimate worker exposure under a number of manufacturing, processing and use scenarios (Ref. 126). In addition, EPA may be able to use air concentration information or an estimation approach for a structurally similar chemical to estimate work exposures under specific industrial or non-industrial scenarios. However, the petition does not address these possibilities, let alone explain why a testing order under section 4 would be necessary at this point. EPA considers these approaches to be reasonable to determine exposure to workers of manufacturing and processing facilities, but may decide to pursue targeted sampling in the future for workers in manufacturing and processing facilities to reduce uncertainty.

*Dust Sampling.* 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 the CPE Cluster chemicals through dust for workers in manufacturing, processing, industrial and non-industrial use facilities. EPA believes that a combination of modelling and existing data (e.g., non-U.S. data) could allow EPA to determine or predict effects on workers exposed to dust containing the CPE Cluster chemicals in an industrial and non-industrial environment.

EPA believes the approaches described earlier, Unit IV.C.4.b. and c.

regarding Air Sampling, are sufficient to characterize exposures to workers at manufacturing or processing facilities from exposure to dust. Sampling of settled dust (surface wipe and bulk sampling) using the OSHA Technical Manual (Ref. 139), as requested by the petitioners, may not be necessary. During Problem Formulation and Initial Assessment, EPA stated that inhalation and dermal exposure were the primary routes of occupational exposure for the CPE Cluster chemicals. Presence of the CPE Cluster chemicals 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. 140).

EPA notes that in the ongoing NIOSH study (Ref. 138) surface wipe sampling is not included, which provides support for the conclusion that settled dust is not a customary measure for occupational exposure. Furthermore, EPA would 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. The biomonitoring data collected following the protocols of the ongoing NIOSH study or other peer-reviewed studies, 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 all exposure routes. *5. Exposures from recycling.* The petition does not set forth facts demonstrating that there is insufficient information available to EPA to reasonably determine or predict effects to communities and workers specifically located at or near facilities that recycle the CPE Cluster chemical-containing products. EPA believes that the approaches requested by the petitioners to measure exposure to the CPE Cluster chemicals from recycling facilities may not be needed. These are the same approaches referenced in Unit IV.C.4.a.b. and c. EPA did not include in the Problem Formulation and Initial Assessment a search for data associated with the recycling of the CPE Cluster chemicals. Going forward, EPA would initiate a comprehensive search of



available data. EPA could then assess the nature of the data, including those cited by the petitioners (Refs. 141–143) to determine feasibility of conducting an assessment. 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 information, the recycling processes used in the U.S. could potentially be assessed. However, the petition does not address this possibility, let alone explain why a testing order under section 4 would be necessary on this point.

EPA also notes that the NIOSH study (Ref. 138) may inform occupational exposures from recycling facilities and could be considered in an occupational assessment of CPE Cluster chemicals. 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.4.b. and c. EPA would consider any data or information generated from the NIOSH study deemed to be relevant and applicable for discerning exposures from all exposure routes. 6. *Exposure from disposal*. The petition does not set forth facts demonstrating that there is insufficient information available to EPA to reasonably determine or predict effects to communities and workers specifically located at or near facilities that dispose of CPE Cluster chemical-containing products. EPA believes that the approaches requested by the petitioners to measure exposure to the CPE Cluster chemicals from disposal facilities may not be needed. These are the same approaches referenced in Unit IV.C.4.a.b. and c. EPA did not include in the Problem Formulation and Initial Assessment a search for data associated with the disposal of the CPE Cluster chemicals. Going forward, EPA would initiate a comprehensive search of available data. EPA could then assess the nature of the data to determine feasibility of conducting an assessment. 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 potentially be assessed. However, the petition does not address this possibility, let alone explain why a testing order under section 4 would be necessary at this point.

EPA also notes that the NIOSH study (Ref. 138), may inform occupational exposures from disposal facilities and could be considered in an occupational assessment of the CPE Cluster chemicals. 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.4.b. and c., 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. 7. *Exposures of birds, wildlife and sediment organisms*.

*Terrestrial organism toxicity*. The petition does not set forth facts demonstrating that there is insufficient information available to EPA to reasonably determine or predict CPE Cluster chemicals' effects to terrestrial organisms. The avian toxicity test (OCSPP 850.2100: Avian Acute Oral Toxicity Test) (Ref. 144) as requested by the petitioners is not necessary. Although the Problem Formulation and Initial Assessment previously stated that there was limited ability to quantify risks because of a lack of monitoring data and hazard endpoints (Ref. 2), studies have been identified since the publication of the Problem Formulation and Initial Assessment document including a study by Fernie et al. (2013) measuring toxicity of all three CPE Cluster chemicals to American Kestrels (Ref. 145) using a modified Avian Dietary Toxicity Test (OCSPP 850.2200) (Ref. 146), and a study on the toxicity of TCEP to hens (Ref. 147).

EPA considers the three chemicals in the CPE Cluster to have similar hazard profiles from an ecological perspective and hence, read-across, in which data for one structurally similar chemical can be used to assess the toxicity of another, could be appropriately applied. EPA's conclusion regarding this approach is supported by its use in risk assessments performed by the European Union (Refs. 96, 97 and 148). Collectively, the available data could be used to determine or predict the effects of the CPE Cluster chemicals on terrestrial organism, specifically birds, from repeated exposures.

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).

*Soil/Sediment dwelling organisms*. The petition does not set forth facts demonstrating that there is insufficient information available to EPA to reasonably determine or predict the CPE Cluster chemicals' effects to soil/sediment dwelling organisms. The Earthworm Subchronic Toxicity Test (OCSPP 850.3100) (Ref. 152) as requested by petitioners is not needed. Although the Problem Formulation and Initial Assessment states that data was not available to characterize risk for sediment dwelling organisms (Ref. 2), adequate sediment toxicity studies exist for TDCPP and this data could also be used to evaluate and characterize the effects of the other CPE Cluster chemicals to sediment dwelling organisms using read-across. There are chronic toxicity studies on three sediment-dwelling species, *Chironomus riparius* (midge), *Hyallela Azteca* (amphipod) and *Lumbriculus variegatus* (oligochaete) (Refs. 150–152). Since publication of the Problem Formulation and Initial Assessment, EPA identified additional data on soil/sediment dwelling organisms that could be used to assess risks to these organisms (Refs. 153–155).

EPA considers the three chemicals in the CPE Cluster to have similar hazard profiles from an ecological perspective and hence, read-across, in which data for one structurally similar chemical can be used to assess the toxicity of another, could be appropriately applied. EPA's conclusion regarding this approach is supported by its use in risk assessments performed by the European Union (Refs. 96, 97, and 148). Collectively, the available data could be used to determine or predict the effects of the CPE Cluster chemicals on soil/sediment dwelling organisms.

*Plant toxicity*. The petition does not set forth facts demonstrating that there is insufficient information available to EPA to reasonably determine or predict the CPE Cluster chemicals effects on plants. The Early Seedling Growth Toxicity Test (OCSPP 850.4230) (Ref. 156) as requested by the petitioners is not needed. Since publication of the Problem Formulation and Initial Assessment document, EPA identified data on the toxicity to terrestrial plants from TDCPP (Ref. 157), TCEP (Ref. 158) and TCPP (Ref. 159). The data could be used to determine or predict the effects of the CPE Cluster chemicals on plants.

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 the CPE Cluster chemicals as well as alternate approaches for risk evaluation is insufficient to permit a reasoned determination or prediction of the health or environmental effects of the CPE Cluster chemicals 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 TSCA section 4(h), 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 the Chlorinated Phosphate Ester Cluster Flame Retardants (TCEP, TCPP and TDCPP) under Section 4(a) of the Toxic Substances Control Act. January 6, 2017.
2. EPA. 2015a. TSCA Work Plan Chemical Problem Formulation and Initial Assessment Chlorinated Phosphate Ester Cluster Flame Retardants.
3. NTP (National Toxicology Program). 1991a. Toxicology and Carcinogenesis Studies of Tris(2-Chloroethyl) Phosphate (CAS No. 115-96-8) in F344/N Rats and B6c3f1 Mice (Gavage Studies). Department of Health and Human Services. Research Triangle Park, NC. NTP Technical Report 391.
4. Freudenthal, R.I., and R.T. Henrich. 2000. Chronic Toxicity and Carcinogenic Potential of Tris(1,3-Dichloro-2-Propyl) Phosphate in Sprague-Dawley Rat. *International Journal of Toxicology*. 19, 119-125.
5. Freudenthal, R.I., and R.T. Henrich. 1999. A Subchronic Toxicity Study of Fyrol Pcf in Sprague-Dawley Rats. *International Journal of Toxicology*, 18(3), 173-176.
6. Tilson, H., B. Veronesi, R. McLamb, and H. Matthews. 1990. Acute Exposure to Tris(2-Chloroethyl) Phosphate Produces Hippocampal Neuronal Loss and Impairs Learning in Rats. *Toxicology and Applied Pharmacology*, 106(2), 254-269.
7. Anonymous. 1977. Health and safety data for 4 chemicals with cover letter dated 021089 (sanitized). Submitted to the U.S. Environmental Protection Agency under TSCA Section 8D. EPA86-8900001189. OTS0516689.
8. EPA. 2015b. Flame Retardants Used in Flexible Polyurethane Foam: An Alternatives Assessment Update. Doc. No. 744-R-15-002. [https://www.epa.gov/sites/production/files/2015-08/documents/ffr\\_final.pdf](https://www.epa.gov/sites/production/files/2015-08/documents/ffr_final.pdf).
9. Cal. EPA. Office of Environmental Health Hazard Assessment (OEHHA). Oct. 21, 2016. Chemicals Known to the State to Cause Cancer or Reproductive Toxicity 21 <http://oehha.ca.gov/media/downloads/proposition-65//p65single10212016.pdf>.
10. OEHHA. July 2011. Reproductive and Cancer Hazard Assessment Branch, Evidence on the Carcinogenicity of Tris(1,3-Dichloro-2-Propyl) Phosphate. <http://oehha.ca.gov/media/downloads/proposition-65/chemicals/tdcpp070811.pdf>.
11. OEHHA. Oct. 21, 2016. Chemicals Known to the State to Cause Cancer or Reproductive Toxicity. <http://oehha.ca.gov/media/downloads/proposition-65//p65single10212016.pdf>.
12. European Chemicals Agency. Nov. 27, 2009. Support Document for Identification of Tris(2-Chloroethyl) Phosphate as a Substance of Very High Concern Because of its CMR Properties. <https://echa.europa.eu/documents/10162/6d09755f-7fcb-4a00-b7ce-91ab45a2e5af>.
13. See Cal SAFER. Candidate Chemical Details (last visited Jan. 4, 2017), <https://calsafer.dtsc.ca.gov/chemical/ChemicalDetail.aspx?chemid=20838>.
14. Benotti, M., R. Trenholm, B. Vanderford, J. Holady, B. Stanford, and S. Snyder. 2009. Pharmaceuticals and Endocrine Disrupting Compounds in U.S. Drinking Water. *Environmental Science & Technology*, 43(3), 597-603.
15. Snyder, S.A., E.C. Wert, H. Lei, P. Westerhoff, and Y. Yoon. 2007. Removal of Edcs and Pharmaceuticals in Drinking and Reuse Treatment Processes.
16. Stackelberg, P.E., J. Gibbs, E.T. Furlong, M.T. Meyer, S.D. Zaugg, and R.L. Lippincott. 2007. Efficiency of Conventional Drinking-Water-Treatment Processes in Removal of Pharmaceuticals and Other Organic Compounds. *Science of the Total Environment*, 377(2-3), 255-272.
17. Fang, M., T. Webster, D. Gooden, E. Cooper, M. McClean, C. Carignan, C. Makey, and H. Stapleton. 2013. Investigating a Novel Flame Retardant Known as V6: Measurements in Baby Products, House Dust, and Car Dust. *Environmental Science & Technology*, 47(9), 4449-4454.
18. Stapleton, H.M., S. Klosterhaus, A.S. Keller, P.L. Ferguson, S. van Bergen, E.M. Cooper, T.F. Webster, and A. Blum. 2011. Identification of Flame Retardants in Polyurethane Foam Collected from Baby Products. *Environmental Science & Technology*, 45(12), 5323-5331.
19. Stapleton, H.M., S. Klosterhaus, S. Eagle, J. Fuh, J.D. Meeker, A. Blum, and T.F. Webster. 2009. Detection of Organophosphate Flame Retardants in Furniture Foam and U.S. House Dust. *Environmental Science & Technology*, 43(19), 7490-7495.
20. Stapleton, H.M., S. Sharma, G. Getzinger, P.L. Ferguson, M. Gabriel, T.F. Webster, and A. Blum. 2012. Novel and High Volume Use Flame Retardants in U.S. Couches Reflective of the 2005 Pentabde Phase Out. *Environmental Science & Technology*, 46(24), 13432-13439.
21. Keller, A.S., N.P. Raju, T.F. Webster, and H.M. Stapleton. 2014. Flame Retardant Applications in Camping Tents and Potential Exposure. *Environmental Science and Technology Letters*(1), 152-155.
22. Ali, N., A.C. Dirtu, N. Van den Eede, E. Goosey, S. Harrad, H. Neels, A. Mannetje, J. Coakley, J. Douwes, and A. Covaci. 2012. Occurrence of Alternative Flame Retardants in Indoor Dust from New Zealand: Indoor Sources and Human Exposure Assessment. *Chemosphere*, 88(11), 1276-1282.
23. Ali, N., N. Van den Eede, A.C. Dirtu, H. Neels, and A. Covaci. 2012. Assessment of Human Exposure to Indoor Organic Contaminants Via Dust Ingestion in Pakistan. *Indoor Air*, 22(3), 200-211.
24. Allen, J.G., H.M. Stapleton, J. Vallarino, E. McNeely, M.D. McClean, S.J. Harrad, C.B. Rauert, and J.D. Spengler. 2013. Exposure to Flame Retardant Chemicals on Commercial Airplanes. *Environmental Health*, 12(17), 13.
25. Bergh, C., R. Torgrip, G. Emenius, and C. Ostman. 2011. Organophosphate and Phthalate Esters in Air and Settled Dust—a Multi-Location Indoor Study. *Indoor Air*, 21, 67-76.
26. Brommer, S., S. Harrad, N. Van den Eede, and A. Covaci. 2012. Concentrations of Organophosphate Esters and Brominated Flame Retardants in German Indoor Dust Samples. *Journal of Environmental Monitoring*, 14(9), 2482-2487.
27. Carignan, C.C., M.D. McClean, E.M. Cooper, D.J. Watkins, A.J. Fraser, W. Heiger-Bermays, H.M. Stapleton, and T.F. Webster. 2013. Predictors of Tris(1,3-Dichloro-2-Propyl) Phosphate Metabolite in the Urine of Office Workers. *Environmental International*, 55, 56-61.
28. Dodson, R.E., L.J. Perovich, A. Covaci, N. Van den Eede, A.C. Ionas, A.C. Dirtu, J.G. Brody, and R.A. Rudel. 2012. After the Pbde Phase-Out: A Broad Suite of Flame Retardants in Repeat House Dust Samples from California. *Environmental Science and Technology*, 46(24), 13056-13066.
29. Marklund, A., B. Andersson, and P. Haglund. 2003. Screening of

- Organophosphorus Compounds and Their Distribution in Various Indoor Environments. *Chemosphere*, 53(9), 1137–1146.
30. Meeker, J.D., and H.M. Stapleton. 2010. House Dust Concentrations of Organophosphate Flame Retardants in Relation to Hormone Levels and Semen Quality Parameters. *Environmental Health Perspectives*, 118(3), 318–323.
31. Takigami, H., G. Suzuki, Y. Hirai, Y. Ishikawa, M. Sunami, and S. Sakai. 2009. Flame Retardants in Indoor Dust and Air of a Hotel in Japan. *Environment International*, 35(4), 688–693.
32. Cao, S., X. Zeng, H. Song, H. Li, Z. Yu, G. Sheng, and J. Fu. 2012. Levels and Distributions of Organophosphate Flame Retardants and Plasticizers in Sediment from Taihu Lake, China. *Environmental Toxicology and Chemistry*, 31(7), 1478–1484.
33. Stapleton, H.M., J. Misenheimer, H.K., and T.F. Webster. 2014. Flame Retardant Associations between Children's Handwipes and House Dust. *Chemosphere*, 116, 54–60.
34. Bergh, C., R. Torngrip, G. Emenius, and C. Ostman. 2011. Organophosphate and Phthalate Esters in Air and Settled Dust—a Multi-Location Indoor Study. *Indoor Air*, 21, 67–76.
35. Bjorklund, J., S. Isetun, and U. Nilsson. 2004. Selective Determination of Organophosphate Flame Retardants and Plasticizers in Indoor Air by Gas Chromatography, Positive-Ion Chemical Ionization and Collision-Induced Dissociation Mass Spectrometry. *Rapid Communications in Mass Spectrometry*, 18(24), 3079–3083.
36. Green, N., M. Schlabach, T. Bakke, E. Brevik, C. Dye, D. Herzke, S. Huber, B. Plosz, M. Remberger, M. Schoyen, H. Uggerud, and C. Vogelsang. 2008. Screening of Selected Metals and New Organic Contaminants, 2007. 5569–2008. Norwegian Pollution Control Agency, Oslo, Norway.
37. Hartmann, P.C., D. Burgi, and W. Giger. 2004. Organophosphate Flame Retardants and Plasticizers in Indoor Air. *Chemosphere*, 57(8), 781–787.
38. Ingerowski, G., A. Friedle, and J. Thumulla. 2001. Chlorinated Ethyl and Isopropyl Phosphoric acid Triesters in the Indoor Environment—an Inter-Laboratory Exposure Study. *Indoor Air*, 11, 145–49.
39. Makinen, M.S.E., M.R.A. Makinen, J.T.B. Koistinen, A.L. Pasanen, P.O. Pasanen, P.J. Kalliokoski, and A.M. Korpi. 2009. Respiratory and Dermal Exposure to Organophosphorus Flame Retardants and Tetrabromobisphenol A at Five Work Environments. *Environmental Science & Technology*, 43(3), 941–947.
40. Marklund, A., B. Andersson, and P. Haglund. 2005. Traffic as a Source of Organophosphorus Flame Retardants and Plasticizers in Snow. *Environmental Science & Technology*, 39(10), 3555–3562.
41. Sanchez, C., M. Ericsson, H. Carlsson, and A. Colmsjo. 2003. Determination of Organophosphate Esters in Air Samples by Dynamic Sonication-Assisted Solvent Extraction Coupled on-Line with Large-Volume Injection Gas Chromatography Utilizing a Programmed-Temperature Vaporizer. *Journal of Chromatography A*, 993(1–2), 103–110.
42. Staaf, T., and C. Ostman. 2005a. Indoor Air Sampling of Organophosphate Triesters Using Solid Phase Extraction (Spe) Adsorbents. *Journal of Environmental Monitoring (JEM)*, 7(4), 344–348.
43. Staaf, T., and C. Ostman. 2005b. Organophosphate Triesters in Indoor Environments. *Journal of Environmental Monitoring (JEM)*, 7(9), 883–887.
44. Tollback, J., D. Tamburro, C. Crescenzi, and H. Carlsson. 2006. Air Sampling with Empore Solid Phase Extraction Membranes and Online Single-Channel Desorption/Liquid Chromatography/Mass Spectrometry Analysis: Determination of Volatile and Semi-Volatile Organophosphate Esters. *Journal of Chromatography A*, 1129(1), 1–8.
45. Kanazawa, A., I. Saito, A. Araki, M. Takeda, M. Ma, Y. Saijo, and R. Kishi. 2010. Association between Indoor Exposure to Semi-Volatile Organic Compounds and Building-Related Symptoms among the Occupants of Residential Dwellings. *Indoor Air*, 20(1), 72–84.
46. Ohura, T., T. Amagai, Y. Senga, and M. Fusaya. 2006. Organic Air Pollutants Inside and Outside Residences in Shimizu, Japan: Levels, Sources and Risks. *Science of The Total Environment*, 366(2–3), 485–499.
47. Otake, T., J. Yoshinaga, and Y. Yanagisawa. 2004. Exposure to Phthalate Esters from Indoor Environment. *Journal of Exposure Analysis and Environmental Epidemiology*, 14, 524–528.
48. Otake, T., J. Yoshinaga, and Y. Yukio Yanagisawa. 2001. Analysis of Organic Esters of Plasticizer in Indoor Air by GC-MS and GC-Fpd. *Environmental Science & Technology*, 35, 3099–3102.
49. Saito, I., A. Onuki, and H. Seto. 2007. Indoor Organophosphate and Polybrominated Flame Retardants in Tokyo. *Indoor Air*, 17(1), 28–36.
50. Alvarez, D., K. Maruya, N. Dodder, W. Lao, E. Furlong, and K. Smalling. 2013. Occurrence of Contaminants of Emerging Concern Along the California Coast (2009–10) Using Passive Sampling Devices. *Marine Pollution Bulletin*, 81(2), 347–354.
51. Hoppe-Jones, C., G. Oldham, and J.E. Drewes. 2010. Attenuation of Total Organic Carbon and Unregulated Trace Organic Chemicals in U.S. Riverbank Filtration Systems. *Water Research*, 44(15), 4643–4659.
52. Kolpin, D.W., E.T. Furlong, M.T. Meyer, E.M. Thurman, S.D. Zaugg, L.B. Barber, and H.T. Buxton. 2002. Pharmaceuticals, Hormones, and Other Organic Wastewater Contaminants in U.S. Streams, 1999–2000: A National Reconnaissance. *Environmental Science & Technology*, 36(6), 1202–1211.
53. Oros, D.R., W.M. Jarman, T. Lowe, N. David, S. Lowe, and J.A. Davis. 2003. Surveillance for Previously Unmonitored Organic Contaminants in the San Francisco Estuary. *Marine Pollution Bulletin*, 46(9), 1102–1110.
54. Vanderford, B.J., R.A. Pearson, D.J. Rexing, and S.A. Snyder. 2003. Analysis of Endocrine Disruptors, Pharmaceuticals, and Personal Care Products in Water Using Liquid Chromatography/Tandem Mass Spectrometry. *Analytical Chemistry*, 75(22), 6265–6274.
55. Vidal-Dorsch, D.E., S.M. Bay, K. Maruya, S.A. Snyder, R.A. Trenholm, and B.J. Vanderford. 2012. Contaminants of Emerging Concern in Municipal Wastewater Effluents and Marine Receiving Water. *Environmental Toxicology and Chemistry*, 31(12), 2674–2682.
56. Andresen, J., and K. Bester. 2006. Elimination of Organophosphate Ester Flame Retardants and Plasticizers in Drinking Water Purification. *Water Research*, 40(3), 621–629.
57. Andresen, J., A. Grundmann, and K. Bester. 2004. Organophosphorus Flame Retardants and Plasticizers in Surface Waters. *Science of The Total Environment*, 332(1–3), 155–166.
58. Andresen, J., D. Muir, D. Ueno, C. Darling, N. Theobald, and K. Bester. 2007. Emerging Pollutants in the North Sea in Comparison to Lake Ontario, Canada, Data. *Environmental Toxicology and Chemistry*, 26(6), 1081–1089.
59. Bacaloni, A., F. Cucci, C. Guarino, M. Nazzari, R. Samperi, and A. Lagana. 2008. Occurrence of Organophosphorus Flame Retardant and Plasticizers in Three Volcanic Lakes of Central Italy. *Environmental Science and Technology*, 42(6), 1898–1903.
60. Bendz, D., N.A. Paxeus, T.R. Ginn, and F.J. Jloge. 2005. Occurrence and Fate of Pharmaceutically Active Compounds in the Environment, a Case Study: HüOje River in Sweden. *Journal of Hazardous Materials*, 122, 195–204.
61. Bollmann, U., A. Moeler, Z. Xie, R. Ebinghaus, and J. Einax. 2012. Occurrence and Fate of Organophosphorus Flame Retardants and Plasticizers in Coastal and Marine Surface Waters. *Water Research*, 46(2), 531–538.
62. Clara, M., M. Kralik, H. Miesbauer, M. Schabuss, S. Scharf, B. Vallant, S. Weiss, and B. Grillitsch (Environment Agency Austria). 2010. Pollutants of Priority Concern in Austrian Rivers Mercury and Its Compounds Triphosphates. REP–0253. Vienna, Austria.
63. Cristale, J., A. Katsoyiannis, C. Chen, K.C. Jones, and S. Lacorte. 2013a. Assessment of Flame Retardants in River Water Using a Ceramic Dosimeter Passive Sampler. *Environmental Pollution*, 172, 163–169.
64. Cristale, J., A. Katsoyiannis, A.J. Sweetman, K.C. Jones, and S. Lacorte. 2013b. Occurrence and Risk Assessment of Organophosphorus and Brominated Flame Retardants in the River Aire (Uk). *Environmental Pollution*, 179, 194–200.
65. Fries, E., and W. Puttmann. 2003. Monitoring of the Three

- Organophosphate Esters Tbp, TCEP and Tbp in River Water and Ground Water (Oder, Germany). *Journal of Environmental Monitoring*, 5(2), 346–352.
66. Garcia-Lopez, M., I. Rodriguez, and R. Cela. 2010. Mixed-Mode Solid-Phase Extraction Followed by Liquid Chromatography-Tandem Mass Spectrometry for the Determination of Tri- and Di-Substituted Organophosphorus Species in Water Samples. *Journal of Chromatography. A*, 1217(9), 1476–1484.
67. Kim, S.D., J. Cho, I.S. Kim, B.J. Vanderford, and S.A. Snyder. 2007. Occurrence and Removal of Pharmaceuticals and Endocrine Disruptors in South Korean Surface, Drinking, and Waste Waters. *Water Research*, 41(5), 1013–1021.
68. Martinez-Carballo, E., C. Gonzalez-Barreiro, A. Sitka, S. Scharf, and O. Gans. 2007. Determination of Selected Organophosphate Esters in the Aquatic Environment of Austria. *Science of The Total Environment*, 388(1–3), 290–299.
69. Matamoros, V., C.A. Arias, L.X. Nguyen, V. Salvado, and H. Brix. 2012. Occurrence and Behavior of Emerging Contaminants in Surface Water and a Restored Wetland. *Chemosphere*, 88(9), 1083–1089.
70. Quednow, K., and W. Puttmann. 2009. Temporal Concentration Changes of Deet, TCEP, Terbutryn, and Nonylphenols in Freshwater Streams of Hesse, Germany: Possible Influence of Mandatory Regulations and Voluntary Environmental Agreements. *Environmental Science and Pollution Research*, 16(6), 630–640.
71. Quednow, K., and W. Puttmann. 2008. Organophosphates and Synthetic Musk Fragrances in Freshwater Streams in Hessen/Germany. *CLEAN—Soil, Air, Water*, 36(1), 70–77.
72. Regnery, J., and W. Puttmann. 2010. Occurrence and Fate of Organophosphorus Flame Retardants and Plasticizers in Urban and Remote Surface Waters in Germany. *Water Research*, 44(14), 4097–4104.
73. Rodil, R., J.B. Quintana, E. Concha-Grana, P. Lopez-Mahia, S. Muniategui-Lorenzo, and D. Prada-Rodriguez. 2012. Emerging Pollutants in Sewage, Surface and Drinking Water in Galicia (Nw Spain). *Chemosphere*, 86(10), 1040–1049.
74. Schwarzbauer, J., and S. Heim. 2005. Lipophilic Organic Contaminants in the Rhine River, Germany. *Water Research*, 39(19), 4735–4748.
75. Stepien, D.K., J. Regnery, C. Merz, and W. Puttmann. 2013. Behavior of Organophosphates and Hydrophilic Ethers During Bank Filtration and Their Potential Application as Organic Tracers. A Field Study from the Oderbruch, Germany. *Science of The Total Environment*, 458–460, 150–159.
76. Weigel, S., K. Bester, and H. Huhnerfuss. 2005. Identification and Quantification of Pesticides, Industrial Chemicals, and Organobromine Compounds of Medium to High Polarity in the North Sea. *Marine Pollution Bulletin*, 50(3), 252–263.
77. Yoon, Y., J. Ryu, J. Oh, B.G. Choi, and S.A. Snyder. 2010. Occurrence of Endocrine Disrupting Compounds, Pharmaceuticals, and Personal Care Products in the Han River (Seoul, South Korea). *Science of The Total Environment*, 408(3), 636–643.
78. Chen, D., R. Letcher, N. Burgess, L. Champoux, J. Elliott, C. Hebert, P. Martin, M. Wayland, D. Weseloh, and L. Wilson. 2012. Flame Retardants in Eggs of Four Gull Species (Laridae) from Breeding Sites Spanning Atlantic to Pacific Canada. *Environmental Pollution*, 168, 1–9.
79. EPA. Response to Petition to Order Testing of the Chlorinated Phosphate Ester (CPE) Cluster Under Section 4(a) of the Toxic Substances Control Act. 2017.
80. EPA. Assessments for TSCA Work Plan Chemicals. <https://www.epa.gov/assessing-and-managing-chemicals-under-tsca/assessments-tsca-work-plan-chemicals>. (retrieved on March 15, 2017).
81. EPA. Docket EPA–HQ–OPPT–2015–0068. 2016. <https://www.regulations.gov/docket?D=EPA-HQ-OPPT-2015-0068>.
82. 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>.
83. 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>.
84. OECD. 2010. Test No 417: Toxicokinetics. Guideline for the testing of chemicals. OECD Guidelines for the Testing of Chemicals, Section 4: Health Effects. OECD Publishing, Paris.
85. OECD. 2004. Test No. 427: Skin Absorption: In Vivo Method. OECD Guidelines for the Testing of Chemicals, Section 4: Health Effects. OECD Publishing, Paris.
86. EPA. 1998a. Acute Dermal Toxicity (OPPTS Test Guideline 870.1200).
87. Lynn, R.K.; Wong, C; Garvie-Gould, Kennish J.M. 1981 Disposition of the Flame Retardant, Tris(1,3-dichloro-2-propyl)phosphate, in the Rat. *Drug Metab. Disp.* 9(5):434–441.
88. Burka L.T., Sanders J.M., Herr D.W., Matthews H.B. 1991. Metabolism of Tris(2-chloroethyl)phosphate in Rats and Mice. *Drug Metab Disp.* 19(2):443–447.
89. Fang M., Stapleton H.M. 2014. Evaluating the Bioaccessibility of Flame Retardants in House Dust Using an In Vitro Tenax Bead Assisted Sorptive Physiologically Based Method. *Environmental Science & Technology*. 48:13323–13330.
90. Herr D.W., Sanders J.M., Matthews H.B. 1991. Brain Distribution and Fate of Tris(2-chloroethyl)phosphate in Fischer 344 Rats. *Drug Metab Disp.* 19(2):436–442.
91. Hughes M.F., Edwards B.C., Mitchell C., T. Bhooshan B. 2001. In Vitro Dermal Absorption of Flame Retardant Chemicals. *Food Chem Toxicol.* 39:1263–1270.
92. Marzulli F.N., Callahan B.S., Brown D.W.C. 1965. Chemical Structure and Skin Penetrating Capacity of a Short Series of Organic Phosphates and Phosphoric Acid. *J Invest Derm.* 44(5):339–344.
93. Minegashi K–I, Kurebayashi H., Seiichi, Namaru, Morimot K, Takahashi T, Yamaha T. 1988. Comparative Studies on Absorption, Distribution, and Excretion of Flame Retardants Halogenated Alkyl Phosphate in Rats. *Eisei Kagaku* 34(2):102–114.
94. Nomeir A.A., Kato D., Matthews H.B. 1981. The Metabolism and Disposition of Tris(1,3-dichloro-2-propyl)phosphate (Fryol FR–2) in the Rat. *Toxicol Appl Pharm.* 57:401–413.
95. Pawar G. Abdallah A–E, Villaverde de Saa E., Harrad S. 2017. Dermal Bioaccessibility of Flame Retardants from Indoor Dust and the Influence of Topically Applied Cosmetics. *J Exp Scie Environ Epid.* 27:100–105.
96. EU (European Union). 2008a. European Union Risk Assessment Report: Tris(2-Chloro-1-Methylethyl) Phosphate (TCPP) CAS No: 13674–84–5. Ireland and United Kingdom, Luxembourg. [http://echa.europa.eu/documents/10162/6434698/orats\\_final\\_rar\\_tris2-chloro-1-methylethylphos\\_en.pdf](http://echa.europa.eu/documents/10162/6434698/orats_final_rar_tris2-chloro-1-methylethylphos_en.pdf).
97. EU (European Union). 2008b. European Union Risk Assessment Report: Tris[2-Chloro-1-(Chloromethyl)Ethyl] Phosphate (TDCP) CAS No: 13674–87–8. Ireland and United Kingdom, Luxembourg. [http://echa.europa.eu/documents/10162/6434698/orats\\_final\\_rar\\_tris2-chloro-1-chloromethyleth\\_en.pdf](http://echa.europa.eu/documents/10162/6434698/orats_final_rar_tris2-chloro-1-chloromethyleth_en.pdf).
98. EPA. 1998b. Acute Inhalation Toxicity (OPPTS Test Guideline 870.1300).
99. Smyth H.F., Carpenter C.P. and Weil C.S. 1951. Range-finding toxicity data: List IV. *Arch. Ind. Hyg. Occup. Med.*, 4, 119–122.
100. Environmental Affairs and Toxicology Department. 1981. Four hour acute inhalation toxicity study in Sprague-Dawley rats with 2425–80 (Unpublished report).
101. Klassen, C.D. 2008. Editor: Cassarett and Doull’s Toxicology: The Basic Science of Poisons. Seventh Edition. McGraw-Hill Medical Publishing Division. New York.
102. NTP (National Toxicology Program). n.d. Modified One-Generation Studies. <https://ntp.niehs.nih.gov/testing/types/mog/index.html> (retrieved in 2016).
103. EPA. 1998c. Reproduction and fertility effects (OPPTS Test Guideline 870.3800).
104. OECD. 2001. Test No 416: Two-Generation Reproduction Toxicity. OECD Guidelines for the Testing of Chemicals, Section 4: Health Effects. OECD Publishing, Paris.
105. NTP (National Toxicology Program). 1991b. Final Report on the Reproductive Toxicity of Tris (2-chloroethyl) phosphate (CAS No. 115–96–8) in CD–1 Swiss Mice. RACB9204.
106. TNO Quality of Life. 2007. Oral two-generation reproduction toxicity study

- (including a dose range finding study) with Tris(2-chloro-1-methylethyl)-phosphate in rats. (Unpublished report).
107. Tanaka S., Nakaura S., Kawashima K., Nagao S., Endo T., Onoda K., Kasuya Y. and Omori Y. 1981. Effect of oral administration of tris(1,3-dichloroisopropyl) phosphate to pregnant rats on prenatal and post natal developments. *Eisei Shikenjo Hokoku* 99, 50–55. (in Japanese).
  108. Stauffer Chemical Company. 1981b. Toxicology reports on FYROL FR–2 (volume I–II) with attachments and cover letters dated 020381. Submitted to the U.S. Environmental Protection Agency under TSCA Section 8E. EPA88–8100271. OTS0204911.
  109. NTP, (National Toxicology Program). n.d. Testing Status of Tris(Chloropropyl)phosphate—M20263. <https://ntp.niehs.nih.gov/testing/status/agents/ts-m20263.html> (retrieved in 2017).
  110. Chen G., Jin Y., Wu Y., Liu L., Fu Z. 2015. Exposure of male mice to two kinds of organophosphate flame retardants (OPFRs) induced oxidative stress and endocrine disruption. *Environ Toxicol Pharmacol.* 40:310–318
  111. Farhat A., Crump D., Chiu S., Williams K.L., Letcher R.J., Gauthier L.T., Kennedy S.W. 2013. In Ovo effects of two organophosphate flame retardants—TCPP and TDCPP—on pipping success, development, mRNA expression, and thyroid hormone levels in chicken embryos. *Tox Sci* 134(1):92–102.
  112. Kojima H., Takeuchi S., Itoh T., Iida M., Kobayashi S., Yoshida T. 2013. In vitro endocrine disruption potential of organophosphate flame retardants via human nuclear receptors. *Toxicology.* 14(1):76–83.
  113. Wang Q., Lai N.L., Wang X., Guo Y., Lam P.K., Lam J.C., Zhou B. 2015a. Bioconcentration and transfer of the organophosphorus flame retardant 1,3-dichloro-2-propyl phosphate causes thyroid endocrine disruption and developmental neurotoxicity in zebrafish larvae. *Environmental Science & Technology.* 49(8):5123–32.
  114. Wang Q., Lam J.C., Han J., Wang X., Guo Y., Lam P.K., Zhou B. 2015b. Developmental exposure to the organophosphorus flame retardant tris(1,3-dichloro-2-propyl) phosphate: Estrogenic activity, endocrine disruption and reproductive effects on zebrafish. *Aquatic Toxicol.* 160:163–171.
  115. Wang Q., Liang K., Liu J., Yang L., Guo Y., Liu C., Zhou B. 2015c. Exposure of zebrafish embryos/larvae to TDCPP alters concentrations of thyroid hormones and transcriptions of genes involved in the hypothalamic-pituitary-thyroid axis. *Aquatic Toxicol.* 126:207–213.
  116. Xu T., Wang Q., Shi Q., Fang Q., Guo Y., Zhou B. 2015. Bioconcentration, metabolism and alterations of thyroid hormones of Tris(1,3-dichloro-2-propyl) phosphate (TDCPP) in Zebrafish. 81–586. *Environ Toxicol. Pharm.* 40(2):5.
  117. Zhang Q., Lu M., Dong X., Wang C., Zhang C., Liu W., Zhao M. 2014. Potential estrogenic effects of phosphorus-containing flame retardants. *Environmental Science & Technology.* 48(12):6995–7001.
  118. EPA. 2015. Larval amphibian growth and development assay (LAGDA) (OCSPP Test Guideline 890.2300).
  119. Zaugg, S.D., Smith, S.G., Schroeder, M.P., Barber, L.B., Burkhardt, M.R. 2002. *Methods of Analysis by the U.S. Geological Survey National Water Quality Laboratory—Determination of Wastewater Compounds by Polystyrene—Divinylbenzene Solid-Phase Extraction and Capillary-Column Gas Chromatography/Mass Spectrometry, Water-Resources Investigations Report 01–4186.* U.S. Department of the Interior.
  120. Rodil, R., Quintana, J.B., Reemtsma, T. 2005. *Liquid Chromatography—Tandem Mass Spectrometry Determination of Nonionic Organophosphorus Flame Retardants and Plasticizers in Wastewater Samples.* *Anal. Chem.* (77).
  121. Unites States Geological Survey (USGS). National Water Information System. <https://waterdata.usgs.gov/nwis> (Retrieved on March 21, 2017).
  122. Glassmeyer, S.T., Furlong, E.T., Kolpin, D.W., Cahill, J.D., Zaugg, S.D., Werner, S.L., Meyer, M.T., Kryak, D.D. 2005. Transport of Chemical and Microbial Compounds from Known Wastewater Discharges: Potential for Use as Indicators of Human Fecal Contamination, *Environmental Science & Technology.* (39).
  123. Jackson, J., Sutton, R. 2008 *Sources of Endocrine-Disrupting Chemicals in Urban Wastewater, Oakland, CA Sci. Tot. Environ.* (405).
  124. O'Brien, J.W., Thai, P.K., Brandsma, S.H., Leonards, P.E.G., Ort, C., Mueller, J.F. 2015. Wastewater analysis of Census Day Samples to Investigate per Capita Input of Organophosphorus Flame Retardants and Plasticizers into Wastewater. *Chemosphere* (138).
  125. Schreder, E.D. and La Guardia, M.J. 2014 *Flame Retardant Trnsfers from U.S. Households (Dust and Laundry Wastewater) to the Aquatic Environment, Environmental Science & Technology* 48, 11575–11583.
  126. EPA. ChemSTEER-chemical screening tool for exposures and environmental releases. 2017. <https://www.epa.gov/tsc-screening-tools/chemsteer-chemical-screening-tool-exposures-and-environmental-releases>.
  127. EPA. E-FAST-exposure and fate assessment screening tool version 2014. 2017. <https://www.epa.gov/tsc-screening-tools/e-fast-exposure-and-fate-assessment-screening-tool-version-2014>.
  128. 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](https://www3.epa.gov/ttn/scram/dispersion_prefrec.htm#aermod).
  129. 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>.
  130. Bradman, A., Castorina, R., Gaspar, F., Nishioka, M., Colon, M., Weathers, W., Egeghy, P.P., Maddalena, R., Williams, J., Jenkins, P.L., McKone, T.E. 2014. *Flame Retardant Exposures in California Early Childhood Education Environments.* *Chemosphere* (116).
  131. Wei, G.-L., Li, D.-Q., Zhuo, M.-N., Liao, Y.-S., Xie, Z.-Y., Guo, T.-L., Li, J.-J., Zhang, S.-Y., Liang, Z.-Q. 2015. *Organophosphorus Flame Retardants and Plasticizers: Sources, Occurrence, Toxicity and Human Exposure.* *Environ. Poll.* (196).
  132. Bester, K. 2005. Comparison of TCPP concentrations in sludge and wastewater in a typical German sewage treatment plant—comparison of sewage sludge from 20 plants. 7: 509–513.
  133. Olofsson, U., Bignert, A., Haglund, P. 2012. Time-trends of metals and organic contaminants in sewage sludge. *Water research* 46: 4841–4851. <http://www.ncbi.nlm.nih.gov/pubmed/22763293>.
  134. Fries, E., Mihajlovic, I. 2011. Pollution of Soils with Organophosphorus Flame Retardants and Plasticizers. *J. Environ. Monit.* (13).
  135. Klosterhaus, S.L., Stapleton, H.M., LaGuardia, M.J., Greig, D.J. 2012. Brominated and Chlorinated Flame Retardants in San Francisco Bay Sediments and Wildlife. *Environ. Intl.* (47).
  136. Regnery, J., Puttmann, W., Merz, C., Berthold, G., 2011. Occurrence and distribution of organophosphorus flame retardants and plasticizers in anthropogenically affected groundwater. *J. Environ. Monit.* 13, 347–354.
  137. Stackelberg, P.E., Furlong, E.T., Meyer, M.T., Zaugg, S.D., Henderson, A.K., Reissman, D.B. 2004. Persistence of pharmaceutical compounds and other organic wastewater contaminants in a conventional drinking-water-treatment plant. *Sci. Tot. Environ.* 329, 99–113.
  138. NIOSH. Assessment of Occupational Exposure to Flame Retardants. 2014. [https://ntp.niehs.nih.gov/ntp/about\\_ntp/bsc/2014/dec/nioshupdate\\_508.pdf](https://ntp.niehs.nih.gov/ntp/about_ntp/bsc/2014/dec/nioshupdate_508.pdf).
  139. 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](https://www.osha.gov/dts/osta/otm/otm_ii/otm_ii_2.html).
  140. Gorman Ng, M., Semple, S., Cherrie, J.W., Christopher, Y., Northage, C., Tielemans, E., Veroughstraete, V. and Von Tongeren, M. 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).
  141. Mäkinen, M.S., Mäkinen, M.R., Koistinen, J.T., Pasanen, A.L., Pasanen, P.O., Kalliokoski, P.J., & Korpi, A.M. 2009. Respiratory and dermal exposure

- to organophosphorus flame retardants and tetrabromobisphenol A at five work environments. Environmental Science & Technology, 43(3), 941–947. <https://doi.org/10.1021/es802593t>.
142. Washington State Department of Ecology. 2014. Flame Retardants in General Consumer and Children's Products. (Publication No. 14–04–021). Washington State Department of Ecology: Olympia, WA. <https://fortress.wa.gov/ecy/publications/documents/1404021.pdf>.
143. Miller, G.Z. & Gearhart, J. 2016. Traveling with Toxics: Flame Retardants & Other Chemicals in Children's Car Seats. Ecology Center: Ann Arbor, MI. <http://www.ecocenter.org/healthy-stuff/pages/childrens-car-seat-study-2016-report>.
144. EPA. 2012a. Avian Acute Oral Toxicity Test (OCSPP Test Guideline 850.2100).
145. Fernie K., Palace V., Peters L., Basu Nil, Letcher R., Karouna-Renier N., Schultz S., Lazarus R. and Rattner B. 2015. Investigating Endocrine and Physiological Parameters of Captive American Kestrels Exposed by Diet to Selected Organophosphate Flame Retardants; Environmental Science & Technology, vol. 49, issue 12, pp. 7448–7455.
146. EPA. 2012b. Avian Dietary Toxicity Test (OCSPP Test Guideline 850.2200).
147. Sprague G.L., Sandvik L.L., Brookins-Hendricks M.J. and Bickford A.A. 1981. Neurotoxicity of two organophosphorus ester flame retardants in hens. J. Toxicol. Environ. Health, 8, 507–518.
148. EU (European Union). 2009. European Union Risk Assessment Report: Tris (2-Chloroethyl) Phosphate, (TCEP) CAS No: 115–96–8. Ireland and United Kingdom, Luxembourg. [http://echa.europa.eu/documents/10162/6434698/orats\\_final\\_rar\\_tris2-chloroethylphosphate\\_en.pdf](http://echa.europa.eu/documents/10162/6434698/orats_final_rar_tris2-chloroethylphosphate_en.pdf).
149. EPA. 2012c. Earthworm Subchronic Toxicity Test (OCSPP Test Guideline 850.3100).
150. Wildlife International, Ltd. 2006a. Tris[2-chloro-1-(chloromethyl)ethyl]-phosphate (TDCEP): A 28-Day Sediment Toxicity Test with *Chironomus riparius* Using Spiked Sediment. Final Report Project Number: 583A–104. Wildlife International, Ltd., Easton, Maryland 21601, U.S.A., as cited in EU (European Union), 2008b. (REF 106)
151. Wildlife International, Ltd. 2006b. Tris[2-chloro-1-(chloromethyl)ethyl]-phosphate (TDCEP): A Prolonged Sediment Toxicity Test with *Hyalella azteca* Using Spiked Sediment. Final Report Project Number: 583A–105. Wildlife International, Ltd., Easton, Maryland 21601, U.S.A., as cited in EU (European Union), 2008b. (Ref. 97)
152. Wildlife International, Ltd. 2006c. Tris[2-chloro-1-(chloromethyl)ethyl]-phosphate (TDCEP): A Prolonged Sediment Toxicity Test with *Lumbriculus variegatus* using Spiked Sediment. Final Report Project Number: 583A–106. Wildlife International, Ltd., Easton, Maryland 21601, U.S.A., as cited in EU (European Union), 2008b. (Ref. 97)
153. Wetton P.M. 1996. Acute toxicity to earthworms. Report of SPL Project Number: 071/458. SafePharm Laboratories Ltd., Derby. as cited in EU (European Union), 2008a (Ref. 96) and EU (European Union), 2008b (Ref. 97).
154. Servajean E. 2003a. Laboratory determination of the long-term toxicity of TCCP to earthworms (*Eisenia fetida*) using artificial soil substrate. Report of Phytosafe Study Number: 03–69–005–ES. PHYTOSAFE s.a.r.l., 2, rue Marx Dormoy, 64000 Pau, France. as cited in EU (European Union), 2008a (Ref. 96).
155. Van Ginkel C.G. 2005b. Toxicity of TDCP to soil micro-organisms: Nitrogen transformation inhibition test. Akzo Nobel Research and Technology Chemicals Arnheim. Report number CER F05047 T 05015 NTI, 20th October 2005. as cited in EU (European Union), 2008b (Ref. 97).
156. EPA. 2012d. Early Seedling Growth Toxicity Test (OCSPP Test Guideline 850.4230).
157. Servajean E. 2004b. Laboratory assessment of the side-effects of TDCP on plant growth. Study Number: 04–99–022–ES. PHYTOSAFE s.a.r.l. Pau, France. as cited in EU (European Union), 2008b (Ref. 97).
158. Römbke, J. Bauer, C. Brodessa, J. Brodsky, J. Danneberg, G. Heimann, D. Renner, I. and Schallnass, H.J. 1995. Basis for the assessment of the ecotoxicological potential of “existing chemicals” in the terrestrial environment—development of a testing strategy. Batelle Eur. Res. rept. 106 04 103 (UBA), UBA-Texte 53/95 (in German), as cited in EU (European Union), 2009 (Ref. 148).
159. Servajean E. 2003b. Laboratory assessment of the side-effects of TCCP on plant growth. Report of Phytosafe Study Number: 03–69–012–ES. PHYTOSAFE s.a.r.l., 2, rue Marx Dormoy, 64000 Pau, France. as cited in EU (European Union), 2008a (Ref. 96).

#### List of Subjects in 40 CFR Chapter I

Environmental protection, Flame retardants, Hazardous substances, chlorinated phosphate ester cluster.

Dated: April 6, 2017.

**Wendy Cleland-Hamnett, Acting,**  
Assistant Administrator, Office of Chemical Safety and Pollution Prevention.

[FR Doc. 2017–07404 Filed 4–11–17; 8:45 am]

**BILLING CODE 6560–50–P**

## FEDERAL COMMUNICATIONS COMMISSION

### 47 CFR Part 64

[CG Docket Nos. 10–51 and 03–123; FCC 17–26]

### Structure and Practices of the Video Relay Services Program

**AGENCY:** Federal Communications Commission.

**ACTION:** Proposed rule.

**SUMMARY:** In this document, the Commission seeks comment on establishing performance goals and service quality metrics to evaluate the efficacy of the video relay service (VRS) program and on the incidence of “phony” VRS calls and the handling of such calls. The Commission also proposes a four-year plan for VRS compensation and rule amendments to permit server-based routing of VRS and point-to-point video calls, provide safeguards regarding who may use VRS at enterprise and public videophones, allow customer service support centers to access the Telecommunications Relay Service (TRS) Numbering Directory for direct video calling, and make a technical change to per-call validation requirements. The Commission also seeks comment on whether to continue including research and development in the TRS Fund budget, prohibit non-service related inducements to register for VRS, and prohibit the use of non-compete provisions in VRS communications assistant (CA) employment contracts.

**DATES:** For VRS compensation rates, server-based routing, and research and development, comments are due April 24, 2017, and reply comments are due May 4, 2017. For performance goals and service quality metrics, the incidence and handling of “phony” VRS calls, VRS use of enterprise and public videophones, direct video calling customer support services, per-call validation procedures, non-service related inducements, and non-compete provisions in VRS employment contracts, comments are due May 30, 2017, and reply comments are due June 26, 2017.

**ADDRESSES:** You may submit comments, identified by CG Docket Nos. 10–51 and 03–123, by any of the following methods:

- **Electronic Filers:** Comments may be filed electronically using the Internet by accessing the Commission's Electronic Comment Filing System (ECFS), through the Commission's Web site <http://apps.fcc.gov/ecfs/>. Filers should follow the instructions provided on the Web