Summary of Technical Information and Scientific Conclusions for Designating Children's Foam-Padded Sleeping Products Containing Tris(1,3-dichloro-2-propyl) Phosphate (TDCPP) or Tris(2-chloroethyl) Phosphate (TCEP) as a Priority Product

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I. Executive Summary

The purpose of this document is to illustrate how DTSC identified and prioritized children's foam-padded sleeping products containing tris(1,3 dichloro-2-propyl) phosphate (TDCPP) or tris(2-chloroethyl) phosphate (TCEP) for listing as a Priority Product. DTSC conducted an extensive literature review on the associated hazard traits and exposure potential of TDCPP and TCEP and the potential for these chemical flame retardants in children's foam-padded sleeping products to contribute to or cause significant or widespread adverse impacts. This report summarizes the technical information evaluated and presents the conclusions of this evaluation.

A. Summary of Technical Information for TDCPP

TDCPP is a high production volume chemical that is commonly used as an additive flame retardant. TDCPP is a replacement for pentabromodiphenyl ether (pentaBDE) flame retardants in polyurethane foam. The pentaBDE mixture was banned in California in 2006 (California Health and Safety Code section 108922) (OEHHA 2011b). Additive flame retardants are not chemically bonded to polyurethane foam and can migrate into indoor and outdoor environments (Marklund et al. 2003). TDCPP was removed from children's pajamas in the 1970s due to concerns regarding mutagenicity, but it is still used in baby and children's products containing polyurethane foam (Stapleton et al. 2011). Following the national phase-out of pentaBDE flame retardants and California's ban of pentaBDEs in 2006, the use of TDCPP grew significantly in flexible polyurethane foam. TDCPP is currently one of the most commonly used flame retardants found in baby products containing polyurethane foam (Stapleton et al. 2011). Exposure to TDCPP from polyurethane foam contained in consumer products may occur through dermal contact, inhalation, or ingestion of TDCPP-laden dust. Infant and toddler hand-to-mouth behavior plays a significant role in exposure to flame retardants in dust (ATSDR 2012; Stapleton et al. 2014).

TDCPP is known to the State of California to cause cancer (OEHHA 2011a). Evidence of carcinogenicity includes increased incidence of liver and kidney tumors in male and

female rats and testicular tumors in male rats (ATSDR 2012; Bio/dynamics 1980; Freudenthal and Henrich 2000; OEHHA 2011b; OEHHA 2012; WHO 1998). TDCPP is metabolized in the body to several compounds that are also known to the State of California to cause cancer (OEHHA 2011b). TDCPP is associated with other adverse health effects including kidney, liver, and testicular abnormalities (ATSDR 2012; OEHHA 2011b). Research has also shown evidence of genotoxicity, developmental toxicity, reproductive toxicity, endocrine toxicity, and neurotoxicity related to TDCPP exposure (see Section IV. Hazard Traits).

In biomonitoring studies, TDCPP has been found in human fat, breast milk and seminal fluid, and metabolites of TDCPP have been detected in urine (Butt et al. 2014; Hoffman et al. 2014; Hudec et al. 1981; LeBel and Williams 1983; LeBel and Williams 1986; LeBel et al. 1989; Sundkvist et al. 2010). TDCPP has also been detected in hand wipe samples taken from children and adults (Hoffman et al. 2015b; Stapleton et al. 2014).

TDCPP has been detected in dust in homes, offices, automobiles, commercial airplanes, hospitals, and day care centers in California and other locations around the world (see Section VIII. Exposure Potential). In an air and dust monitoring study of California early childhood education (ECE) facilities[1], TDCPP was detected at higher concentrations in ECE facilities with foam-filled nap mats than those without (Bradman et al. 2014). The U.S. Environmental Protection Agency (U.S. EPA) estimates that children ingest on average approximately 60 mg dust/day. This is twice as much as adults, who on average ingest approximately 30 mg dust/day (U.S. EPA 2011). Further, children have a smaller body mass relative to adults, so their dosage in terms of mg dust/kg of body mass will be even greater compared to adults.

In a Consumer Product Safety Commission (CPSC) staff preliminary risk assessment report, it was calculated that adult and children's TDCPP exposures are above the acceptable daily intake (ADI) of 0.005 mg/kg/day for non-cancer health effects. It was estimated that TDCPP in furniture foam alone exposes adults to twice the ADI, and exposes children to five times the ADI. Further, the cancer risk for a lifetime of exposure to TDCPP-treated foam-filled furniture was estimated to be 300 per million; a substance may be considered hazardous if the lifetime individual cancer risk exceeds one per million. In children, the estimated cancer risk from exposure to upholstered furniture during the first two years of life was 20 per million (Babich 2006).

[1] Bradman's studies use the term ECE facilities which can include home-based child care providers, private for-profit or non-profit preschools, and programs run by government agencies (e.g., preschools in school districts or Head Start) or religious institutions. For the purposes of this document, the term ECE is used when referring to Bradman's studies while the term "day care center" is used for all other study citations.

TDCPP contamination exists in surface water, sediment, and wastewater. TDCPP has been detected in San Francisco Bay waters and sediment (Klosterhaus et al. 2012; SFEI 2013). TDCPP was detected in surface water in more than half of 139 freshwater streams tested across the U.S. including in California (Kolpin et al. 2002). TDCPP was measured in influents, effluents, and sludge of Swedish sewage facilities (Marklund et al. 2005b). TDCPP has also been detected in U.S. laundry wastewater samples from homes, as well as in the influents and effluents from the wastewater treatment plants associated with those homes, thus indicating the release of TDCPP to waterways from wastewater effluent (Schreder and La Guardia 2014).

TDCPP has been detected in samples of fish, mussels, birds, and bird eggs (Evenset et al. 2009; Green et al. 2008; Leonards et al. 2011; Sundkvist et al. 2010; Takahashi et al. 2013).

Based on these factors, DTSC has determined that the potential exposure to TDCPP in children's foam-padded sleeping products may contribute to or cause significant and widespread adverse impacts to human health and the environment within California.

B. Summary of Technical Information for TCEP

TCEP is an organophosphate chemical that is used as an additive flame retardant. TCEP is structurally similar to TDCPP (OEHHA 2011b). Like TDCPP, TCEP can migrate from foam products to indoor and outdoor environments (Marklund et al. 2003). Exposure to TCEP in consumer products containing polyurethane foam may occur through dermal absorption, inhalation, or ingestion of TDCPP-laden dust. Infant and toddler hand-to-mouth behavior plays a significant role in exposure to flame retardants in dust (EC 2009; Stapleton et al. 2014). TCEP has been detected in polyurethane foam in several children's foam-padded products (Stapleton et al. 2011).

TCEP is a carcinogen and reproductive toxicant and is also associated with other potential adverse health effects. TCEP is known to the State of California to cause cancer and is classified by the European Commission as a reproductive toxicant (ECHA 2012; OEHHA 2011a). Evidence of carcinogenicity includes increased incidence of kidney tumors in male and female rats, while follicular thyroid cancer was increased in rats but not clearly related to chemical exposure (Matthews et al. 1993; NTP 1991). Evidence of reproductive toxicity in mice includes decreased number of pups per litter and number of litters per breeding pair, as well as decreased sperm parameters in exposed male mice (Gulati et al. 1991). Research has also shown evidence of kidney toxicity, liver toxicity, and neurotoxicity related to TCEP exposure (EC 2009; Gulati et al. 1991; Matthews et al. 1990; Matthews et al. 1993; NTP 1991).

In biomonitoring studies, TCEP has been detected in human breast milk (Kim et al. 2014; Sundkvist et al. 2010) and metabolites have been found in human urine samples (Hoffman et al. 2014; Schindler et al. 2009). TCEP has also been detected in baby products containing polyurethane foam (Stapleton et al. 2011) and in hand wipe samples taken from children (Stapleton et al. 2014). TCEP has been detected in dust in various indoor environments including homes, offices, and day care centers worldwide (see Section VIII. Exposure Potential).

TCEP contamination in the environment has been documented in multiple studies. TCEP has been detected worldwide in rivers and streams, wildlife, sediment, and Antarctic ice. In California, TCEP has been detected in both drinking and surface waters (see Section VIII. Exposure Potential).

TCEP has been detected in samples of fish, mussels, crabs, birds, and bird eggs (Green et al. 2008; Leonards et al. 2011; Sundkvist et al. 2010).

Based on consideration of these factors, DTSC has determined that there is potential exposure to TCEP from children's foam-padded sleeping products that may contribute to or cause significant or widespread adverse impacts to human health and the environment within California.

II. Identification of the Priority Product and the Chemicals of Concern

DTSC has identified as a Priority Product children's foam-padded sleeping products containing the following Candidate Chemicals: tris(1,3-dichloro-2-propyl) phosphate (TDCPP) or tris(2-chloroethyl) phosphate (TCEP).

This Priority Product includes the following sleeping products containing polyurethane foam and the additive flame retardants TDCPP or TCEP:

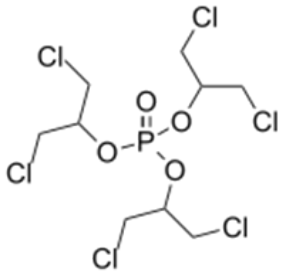
- Nap mat
- Soft-sided portable cribs
- Play pen
- Play yard (or playard)
- Infant travel bed
- Portable infant sleeper
- Bassinet
- Nap cot
- Infant sleep positioner

- Bedside sleeper
- Co-sleeper
- Baby or toddler foam pillow

This Priority Product listing does not include: (1) mattresses (as defined and covered by the requirements of CPSC 1632/1633) or products containing mattresses;
(2) upholstered furniture covered by the requirements of California Technical Bulletin 117-2013; and (3) add-on child restraint systems for use in motor vehicles and aircraft that are required to meet federal flammability standards.

A. Tris(1,3-dichloro-2-propyl) phosphate (TDCPP)

- Chemical Abstract Service (CAS) Registry Number: 13674-87-8
- Molecular formula: $C_9H_{15}Cl_6O_4P$
- Chemical structure:



- IUPAC and common names (ATSDR 2012; ChemSpider 2013; ECHA 2014; NRC 2000)

  Chlorinated Tris
  Chloroalkyl phosphate
  1,3-dichloro-2-propanol phosphate (3:1)
  Phosphoric acid tris(1,3-dichloro-2-propyl) ester
  2-propanol, 1,3-dichloro-, phosphate
  Tris(β, β'-dichloroisopropyl) phosphate
  Tris(1-chloromethyl-2-chloroethyl) phosphate
  Tris(1,3-dichloroisopropyl) phosphate
  Tris(1,3-dichloropropan-2-yl) phosphate
  Tris(1,3-dichloro-2-propanyl) phosphate
  Tris(1,3-dichloro-2-propyl) phosphate
  Tris(2-chloro-1-(chloromethyl)ethyl) phosphate
  Tris(2,2′-dichloroisopropyl) phosphate
  TCCP

TDCP
TDCPP
TDClPP

- Trade names (ATSDR 2012; ChemSpider 2013; ECHA 2014; NRC 2000)

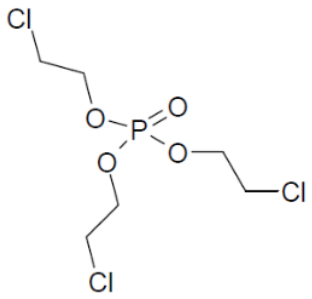
  Amgard
  Amgard TDCP
  Antiblaze 195
  Apex Flame Proof Emulsion 197 or 212
  CRP
  Emulsion 212
  Firemaster T33P
  Foforan Troj-(1,3-dwuchloroizopropylowy) [Polish]
  FR2
  Fyrol FR2
  Fyrol FR-2
  MDL number MFCD00083121
  PF 38
  PF 38/3
  Tolgard TDCP
  Tolgard TDCP MK1

TDCPP meets the conditions specified in California Code of Regulations, Title 22, section 69503.6(a) in that it appears on one or more of the authoritative lists in California Code of Regulations, Title 22, section 69502.2(a)(1) and is a chemical listed in California Code of Regulations, Title 22, section 69502.2(a)(2):

- TDCPP is listed as known to the State of California to cause cancer under Proposition 65 (Safe Drinking Water and Toxic Enforcement Act of 1986) (OEHHA 2011a).
- TDCPP is listed as a priority chemical by the California Environmental Contaminant Biomonitoring Program (CECBP 2014).

B. Tris(2-chloroethyl) phosphate (TCEP)

- Chemical Abstract Service (CAS) Registry Number: 115-96-8
- Molecular formula: $C_6H_{12}Cl_3O_4P$
- Chemical structure:



- IUPAC and common names (ATSDR 2012; EC 2009)

  Ethanol, 2-chloro-, phosphate (3:1)
  Phosphoric acid tris-(2-chlorethyl) ester
  Tri(2-chloroethyl) phosphate
  Trichloroethyl phosphate
  Tris(β-chloroethyl) phosphate
  Tris(beta-2-chloroethyl) phosphate
  Tris(2-chloroethyl) orthophosphate
  Tris(2-chlorethyl) phosphate
  Tris(2-chloroethyl) phosphate
  TRCP
  TCEP

- Trade Names (ATSDR 2012; EC 2009; Stapleton et al. 2011)

  Antiblaze 100
  Celluflex CEF
  Disflamoll TCA
  Fyrol CEF
  Genomoll P
  Hostaflam UP810
  Levagard EP
  Niax 3CF
  NIAX flame retardant
  Tolgard TCEP
  V6 (contains approx. 4.5 – 7.5 % TCEP as an impurity)
  Antiblaze V6 (contains approx. 10% TCEP as an impurity)

TCEP meets the conditions specified in California Code of Regulations, Title 22, section 69503.6(a) in that it appears on one or more of the authoritative lists in California Code of Regulations, Title 22, section 69502.2(a)(1) and is a chemical listed in California Code of Regulations, Title 22, section 69502.2(a)(2):

- TCEP is listed as known to the State of California to cause cancer under Proposition 65 (Safe Drinking Water and Toxic Enforcement Act of 1986) (OEHHA 1992).
- TCEP is classified by the European Commission as a reproductive toxicant (ECHA 2012).
- TCEP is listed as a priority chemical by the California Environmental Contaminant Biomonitoring Program (CECBP 2014).

III. Physicochemical Properties

A. Physicochemical Properties of TDCPP

- Physical description: Viscous, clear liquid (HSDB 2015)
- Molecular weight: 430.90 g/mol (ChemSpider 2013; HSDB 2015)
- Density: 1.48 kg/L at 25 °C (HSDB 2015)
- Boiling point: Between 236 and 237 °C at 5 mmHg (HSDB 2015)
- Melting point: 27 °C (OEHHA 2011b)
- Flashpoint: 252 °C (HSDB 2015)
- Octanol/water partition coefficient: log K_{ow} = 3.65 (HSDB 2015)
- Soil organic carbon/water partition coefficient: K_{oc} of 1,100 (estimate) (HSDB 2015)
- Water Solubility: 7 mg/L at 24 °C (HSDB 2015)
- Solubility: Soluble in most organic solvents (HSDB 2015)
- Vapor Pressure: 2.86 x 1^{0-7} mmHg at 25 °C (estimate) (HSDB 2015)
- Henry's Law constant = 2.61X10^{-9} atm-m^3/mol at 25 °C (estimate) (HSDB 2015)

B. Physicochemical Properties of TCEP

- Physical description: Clear, transparent low viscosity liquid (ATSDR 2012; HSDB 2014)
- Molecular weight: 285.49 g/mol (HSDB 2015)
- Density: 1.425 g/cm^3 at 20°C (ATSDR 2012)
- Boiling point: 330 °C at 1 atm (ATSDR 2012; HSDB 2014)
- Melting point: -55 °C (ATSDR 2012; HSDB 2014)
- Flash point: 216 °C (ATSDR 2012; HSDB 2015)

- Octanol/water partition coefficient: log K_{ow} = 1.44 (ATSDR 2012)
- Soil organic carbon/water partition coefficient: K_{oc} = 390 (estimate) (HSDB 2014)
- Water Solubility: 7.82 g/L at 20 °C (HSDB 2015)
- Solubility: Insoluble in benzene. Soluble in most organic solvents (HSDB 2015)
- Vapor pressure: 6.125 x 10^{-2} mmHg at 25 °C (ATSDR 2012)
- Henry's Law Constant: 3.3 x 10^{-6} atm-m^3/mol at 25 °C (estimate) (HSDB 2015)

IV. Hazard Traits

A. Hazard Traits of TDCPP

TDCPP exposure has been shown to cause a number of hazard traits including carcinogenicity, genotoxicity, developmental toxicity, reproductive toxicity, neurotoxicity, endocrine toxicity, and hematotoxicity. These are summarized below.

1. Carcinogenicity

a. TDCPP is known to the State of California to cause cancer under California's Proposition 65 law (Safe Drinking Water and Toxic Enforcement Act of 1986) (OEHHA 2011a). Exposure to TDCPP above 5.4 µg/day exceeds the Office of Environmental Health Hazard Assessment's (OEHHA) No Significant Risk Level (NSRL) for TDCPP. The NSRL is the estimated intake per day over a 70-year lifetime that results in a risk of one excess cancer in a population of 100,000 people and for TDCPP is based on liver, kidney, and testicular tumor incidence data in experimental animals (OEHHA 2012).
b. TDCPP is classified as a Category 2 Carcinogen (H351 - Suspected of causing cancer) by the European Chemical Agency (ECHA) Committee for Risk Assessment under Regulation (EC) No 1272/2008 (ECHA 2010; ECHA 2014).
c. A U.S. Consumer Product Safety Commission (CPSC) 2006 staff preliminary risk assessment report on flame retardants concluded that TDCPP is a probable human carcinogen based on evidence in animal studies (Babich 2006).
d. Two-year studies in male and female rats showed statistically significant increases in the incidence of tumors at multiple sites including liver, kidneys, testes, and adrenal gland (ATSDR 2012; Bio/dynamics 1980; Freudenthal and Henrich 2000; OEHHA 2011b; WHO 1998).

2. Genotoxicity

TDCPP has tested positive for genotoxicity in both *in vitro* and *in vivo* assay systems (OEHHA 2011b). Evidence of genotoxicity includes findings of induction of mutations, chromosomal aberrations, and DNA binding in animal assays. Selected genotoxicity studies are summarized below.

a. TDCPP readily bound to DNA and proteins in liver, kidney, and muscle in mice treated intravenously with TDCPP (Morales and Matthews 1980; OEHHA 2011b).
b. Studies in Salmonella strains (TA 97, TA 98, TA1537, and TA 1538) indicate that TDCPP induces frameshift mutations (i.e., a genetic mutation caused by a deletion or insertion in a DNA sequence that shifts the way the sequence is read), with or without metabolic activation (Gold et al. 1978; OEHHA 2011b)
c. Treatment of Salmonella strains TA 100 and TA 1535 (sensitive to base pair substitution mutations) with TDCPP resulted in mutations (Gold et al. 1978; OEHHA 2011b)
d. TDCPP caused an increase in chromosomal aberrations (i.e., any irregularity or abnormality of chromosome distribution, number, structure, or arrangement) *in vitro* in mouse lymphoma and Chinese hamster fibroblast cells, but not in Chinese hamster ovary cells (Brusick et al. 1979; Covance 2004; Ishidate 1983; OEHHA 2011b).
e. In one study, TDCPP weakly induced sister chromatid exchanges (i.e., genetic damage demonstrated by the exchange of genetic material between sister chromatids during mitosis) in mouse lymphoma cells; another study did not reveal such changes (Brusick et al. 1979; OEHHA 2011b; Stauffer 1977).
f. In an *in vitro* rat hepatocyte DNA repair synthesis (UDS) assay TDCPP induced a weakly positive response in the absence of, but not in the presence of, phenobarbital induction (OEHHA 2011b).
g. Studies of TDCPP in *in vitro* mammalian cell assays for gene mutation gave both positive and negative results (ATSDR 2012; Brusick et al. 1979; Inveresk 1985; OEHHA 2011b; Soderlund et al. 1985).
h. TDCPP did not induce mutations in *Saccharomyces cerevisiae* (OEHHA 2011b).

3. Developmental Toxicity

Several recent studies on the effects of TDCPP exposure on embryonic development are summarized below.

a. *In Vitro* - An *in vitro* study in PC12[2] cells indicated that TDCPP has the potential to cause developmental neurotoxicity, as evidenced by inhibited DNA synthesis, decreased cell number, and altered neurodifferentiation. (Dishaw et al. 2011).

b. Studies in Zebrafish -
   - Exposure of zebrafish embryos to various concentrations of TDCPP resulted in dose-dependent developmental toxicity, including decreased body weight, reduced hatching, reduced survival and heartbeat rates, and increased malformation (e.g., spinal curvature) (Wang et al. 2013).
   - TDCPP exposure resulted in significantly smaller rates of hatching and survival in a dose- and time-dependent manner (Liu et al. 2013a).
   - TDCPP exposure post-fertilization negatively affected zebrafish embryo development and formation. This was demonstrated by increased mortality, inhibited cell rearrangement, delay in epiboly[3], and abnormal fetal development (e.g., short tail, reduced body size, trunk curvature, tail malformations, craniofacial malformations, decreased body length) (Fu et al. 2013; McGee et al. 2012).

c. Studies in Chickens - TDCPP exposure in chicken eggs was associated with decreases in head- plus-bill length, embryo mass, and gall bladder size in chicken embryos (Farhat et al. 2013).

d. Studies in Rats -
   - Two studies found that when pregnant rats were exposed to high doses of TDCPP, there were high mortality rates in the pregnant dams, decreased live births, and an increased incidence of fetal death (EC 2008; Kawashima et al. 1983). Maternal toxicity was also demonstrated by decreased body weight and decreased food consumption (EC 2008; Kawashima et al. 1983).
   - One study in rats exposed to TDCPP during pregnancy showed no developmental effects in the offspring at dose levels that significantly reduced weight gain in the dams. However, fetal viability was significantly decreased in high dose rats. Maternal toxicity was noted as increased

[2] PC 12 cells are a clonal cell line derived from a pheochromocytoma of the rat adrenal medulla.

[3] Epiboly is a cell movement that occurs in the early embryo. It is one of many coordinated movements in early embryonic development that allows for dramatic physical restructuring. The movement is generally characterized as being a thinning and spreading of cell layers.

mortality at the high dose and decreased body weight and decreased food consumption at the mid- and high doses (ATSDR 2012; Stauffer 1981b).

4. Reproductive Toxicity

Some studies suggest that TDCPP exposure may be associated with male reproductive toxicity. Below are summaries of findings from studies that are relevant to male reproductive toxicity; both positive and negative studies are discussed.

a. A 2010 study reported evidence that TDCPP concentrations in house dust may be associated with decreased sperm concentration in men recruited from an infertility clinic (Meeker and Stapleton 2010).
b. A two-year study found a higher incidence of small seminal vesicles and testicular enlargement in male rats treated with TDCPP at the mid- and high-dose as compared to control males (EC 2008; Freudenthal and Henrich 2000; OEHHA 2011b; Stauffer 1981a).
c. Fertility was not affected and significant alterations of sperm were not observed in male rabbits dosed with TDCPP and then mated with untreated female rabbits (Anonymous 1977; ATSDR 2012).
d. No changes in mating behavior, fertility, or sperm quality or quantity were noted in rabbits exposed to TDCPP via oral gavage (Babich 2006; Brandwene 2001; Wilczynski et al. 1983).
e. A 2008 risk assessment by the European Union concluded that there is no concern for male fertility due to TDCPP exposure based on a weight of evidence approach. The report further stated that there is a lack of data regarding female reproductive toxicity related to TDCPP exposure (EC 2008).

5. Endocrine Toxicity

Recent studies using human cells and zebrafish have indicated that TDCPP has the potential to disrupt normal endocrine function, including thyroid abnormalities and alterations in steroid hormone metabolism.

a. TDCPP could potentially disrupt endocrine function through multiple mechanisms, including effects on steroidogenesis or estrogen metabolism, as suggested by studies in human cell lines and zebrafish (Liu et al. 2012; Liu et al. 2013b).
b. Exposure to various concentrations of TDCPP resulted in altered thyroid hormone levels in zebrafish embryos (Wang et al. 2013).
c. Chicken embryos exposed to TDCPP had lower thyroid hormone levels compared to controls (Farhat et al. 2013).

d. Endocrine disruption potential of TDCPP via human nuclear receptors was reported in an *in vitro* study, showing activity against the pregnane X receptor, androgen receptor, and glucocorticoid receptor (Kojima et al. 2013).
e. TDCPP has the potential to induce estrogenic effects as demonstrated in a combination of *in vitro* assays, such as the E-screen and luciferase reporter gene assays in XX cells (Zhang et al. 2014).
f. Concentrations of TDCPP in house dust correlated with decreased concentrations of circulating thyroid hormone in men recruited from an infertility clinic (Meeker and Stapleton 2010).

6. Neurotoxicity

Most studies that assessed neurotoxicity as an endpoint report TDCPP-induced neurotoxicity. Located studies are summarized below.

a. In an *in vitro* study in PC12 cells, TDCPP displayed concentration-dependent neurotoxicity as indicated by inhibited DNA synthesis, decreased cell number, and altered neurodifferentiation. In this study, TDCPP was a more potent neurotoxicant than chlorpyrifos, an insecticide whose use has been restricted since 2001 (Dishaw et al. 2011).
b. Long-term exposure to TDCPP in zebrafish led to reductions of dopamine and serotonin levels in female brains, and downregulation of genes involved in nervous system development[4] in male and female brain tissues (Wang et al. 2015).
c. Acute exposure to high doses of TDCPP in rats led to clinical signs suggestive of neurotoxicity, such as hyperactivity and convulsions (Babich 2006; Stauffer 1981b).
d. In a two-year dietary study in rats, TDCPP did not induce clinical signs or morphological alterations in the brain or spinal cord. In the same study, changes in measured red blood cell cholinesterase levels were inconsistent (ATSDR 2012; Stauffer 1981b).

7. Other Hazard Traits

a. Acute Toxicity - TDCPP also induces non-cancer chronic health effects in animals and is classified as “acutely toxic” under the Federal Hazardous Substances Act (FHSA) regulations. This includes acute oral and dermal toxicity as well as eye irritation (Babich 2006).

[4] Downregulation of nervous system development is any process that stops, prevents, or reduces the frequency, rate or extent of nervous system development, the origin and formation of nervous tissue.

b. Hepatotoxicity - An increased incidence of altered hepatocellular foci (i.e., altered liver cells) in high-dose female rats was reported following 24 months of dosing (Bio/dynamics 1980; Freudenthal and Henrich 2000; OEHHA 2011b).
c. Nephrotoxicity - An increased incidence of hyperplasia[5] of the convoluted tubules of the kidney was reported in male and female rats in a two-year study (Bio/dynamics 1980; Freudenthal and Henrich 2000; OEHHA 2011b).
d. Hematotoxicity - Decreases in hemoglobin, hematocrit, and total erythrocyte counts were reported following high dose treatment of TDCPP in male and female rats in a two-year study (Bio/dynamics 1980; Freudenthal and Henrich 2000; OEHHA 2011b).
e. Ocular toxicity - An increased number of sacculations (i.e., pouches) along the course of the retinal arterioles were observed (ATSDR 2012; Stauffer 1981a).
f. Dermatotoxicity - A higher prevalence of dermatitis was reported for TDCPP-exposed workers compared to non-exposed workers in a study submitted to U.S. EPA (ATSDR 2012; EC 2009; Stauffer 1983).

B. Hazard Traits of TCEP

TCEP exposure has been shown to cause carcinogenicity, reproductive toxicity, hepatotoxicity, nephrotoxicity, and neurotoxicity. These are summarized below.

1. Carcinogenicity

a. TCEP is known to the State of California to cause cancer under Proposition 65 (Safe Drinking Water and Toxic Enforcement Act of 1986) (OEHHA 1992).
b. The National Toxicology Program (NTP) has concluded that there is clear evidence of carcinogenic activity of TCEP in F344/N rats administered TCEP by gavage (NTP 1991).
c. The International Agency for Research on Cancer (IARC) found that there is limited evidence for carcinogenic activity of TCEP in experimental animals and concluded that TCEP is not classifiable as to its carcinogenicity to humans (IARC 1999a).
d. In a two-year study of rodents administered TCEP by oral gavage, renal tubule adenomas[6] in the kidneys were significantly increased in male rats. Female rats appeared to be relatively more resistant to this effect than males.

[5] Hyperplasia is the enlargement of an organ or tissue caused by an increase in the reproduction rate of its cells, often as an initial stage in the development of cancer.
[6] An adenoma is a benign tumor of epithelial tissue with glandular origin, glandular characteristics, or both; they have the potential to become adenocarcinomas that are malignant or cancerous.

Cancers that may have been related to TCEP exposure include thyroid follicular cell cancers and mononuclear cell leukemia in rats. Findings in mice were equivocal (Matthews et al. 1993).

2. Reproductive Toxicity

Reproductive toxicity resulting from TCEP exposure has been demonstrated in laboratory animals.

a. TCEP is classified by the European Commission as a reproductive toxicant (ECHA 2012).
b. NTP reported that TCEP treatment in mice adversely affected both the number of pups per litter and number of litters per breeding pair in a continuous breeding protocol. The study found that a number of sperm parameters were decreased in exposed male mice. Adverse impacts on the reproductive capacity of mice were also seen at low doses (Gulati et al. 1991).

3. Hepatotoxicity

TCEP exposure resulted in liver toxicity in animal studies.

a. The European Union concluded that liver weight was significantly increased following short and long term oral exposure to TCEP (EC 2009).
b. Liver toxicity was seen in F344/N rats and B6C3F1 mice administered TCEP by gavage. In 14-day exposure studies, increases in female rat liver weights were seen following 16-week exposures, increased liver weights in both sexes of rats and mice were seen (Matthews et al. 1990; NTP 1991).

4. Nephrotoxicity

TCEP exposure resulted in kidney toxicity in animal studies.

a. Significant kidney weight increase was observed in rats following both short- (16 day) and long-term (16-18 weeks) oral exposure to TCEP (EC 2009; NTP 1991).
b. In a two-year study of oral TCEP administration, karyomegaly (i.e., a condition of having an enlarged cell nucleus) was recorded in kidney cells in both sexes of B6C3F1 mice. Rats displayed renal tissue damage in both sexes following repeat exposure (NTP 1991).

5. Neurotoxicity

TCEP-induced neurotoxicity has been shown in animal studies.

a. Neuronal necrosis was seen in rats after both short- and long-term TCEP exposures. The neuronal damage was both dose and sex dependent, with female rats appearing more susceptible than male rats. In some rats, hemorrhages and other neuronal tissue damage were seen (Matthews et al. 1990; Matthews et al. 1993).
b. Brain lesions were identified following both 2 year, 66 week, and 16 week oral TCEP exposure in rats (EC 2009; NTP 1991).

6. Endocrine Toxicity

a. A recent study in male mice indicated the potential of TCEP to induce oxidative stress and affect endocrine function, as indicated by decreased hormone levels and the down regulation of genes related to testosterone function (Chen et al. 2015).

V. Environmental Fate

A. Environmental Fate of TDCPP

TDCPP production and use as an additive flame retardant for polyurethane foams may result in its release to the environment.

1. Air

Based on an estimated vapor pressure of 2.9 x 10^{-7} mmHg at 25 °C and as a function of TDCPP's physical properties, TDCPP volatilizes into the ambient atmosphere, and can adsorb onto dust particles (HSDB 2015). Vapor-phase TDCPP is degraded in the atmosphere by reaction with photochemically produced hydroxyl radicals with an estimated half-life of 21.3 hours. Particulate-phase TDCPP is removed from the atmosphere by wet and dry deposition (HSDB 2015).

2. Soil

In soil, TDCPP is expected to have slight mobility based upon an estimated K_{oc} of 1,100. Volatilization from moist soil surfaces is not expected to be an important pathway for removal of TDCPP based upon an estimated Henry's Law constant of 2.61 x 10^{-9} atm-m^3/mole. Biodegradation is not expected to be an important environmental fate

process in soil based upon a 0-4% biological oxygen demand as determined using the Japanese MITI test (HSDB 2015).

3. Water/Sediment

In water, TDCPP is expected to adsorb to suspended solids and sediment based upon its estimated log K_{ow} of 3.6. Volatilization from water surfaces is not expected to be an important environmental fate process based upon TDCPP's estimated Henry's Law constant of 2.6 x 10^{-9} atm-m^3/mole at 25 °C. Limited data suggests that TDCPP will be resistant to hydrolysis in most environmental waters such as ground water, surface water, and drinking water. Bioconcentration factors (BCFs) ranging from 0.3 to 113 suggest that the potential for bioconcentration in aquatic organisms is low to moderate (HSDB 2015).

B. Environmental Fate of TCEP

TCEP production and use as an additive flame retardant for polyurethane foam may result in its release to the environment.

1. Air

Based on a vapor pressure of 6.13 x 10^{-2} mmHg at 25 °C and as a function of TCEP's physical properties, TCEP will volatilize into the ambient atmosphere, and can adsorb onto dust particles. Vapor-phase TCEP will degrade in the atmosphere by reaction with photochemically produced hydroxyl radicals with an estimated half-life of 16 hrs. TCEP is not expected to be susceptible to direct photolysis by sunlight (HSDB 2015).

2. Soil

In soil, TCEP is expected to have moderate mobility based upon an estimated K_{oc} of 390. Volatilization from moist soil surfaces is not expected to be an important pathway for removal of TCEP based upon an estimated Henry's Law constant of 3.3 x 10^{-6} atm-m^3/mole. Based on results from the Japanese MITI test, biodegradation in soil is not considered an important environmental fate process (HSDB 2015).

3. Water/Sediment

In water, TCEP is expected to adsorb to suspended solids and sediment based upon the estimated K_{oc} of 390. Volatilization from water surfaces is expected to be an important environmental fate process based upon this compound's estimated Henry's Law constant of 3.3 x 10^{-6} atm-m^3/mole. Estimated volatilization half-lives for a model river and model lake are 19 and 140 days, respectively. BCFs ranging from 0.6 to 5.1 suggest bioconcentration in aquatic organisms is low. TCEP may undergo hydrolysis in

the environment based on an estimated hydrolysis half-life of 20 days at pH 5 to 9 (HSDB 2015).

VI. Potential for TDCPP to Degrade, Form Reaction Products, or Metabolize into Another Candidate Chemical or a Chemical that Exhibits One or More Hazard Traits

A number of metabolites and putative metabolites of TDCPP have been reported to be carcinogenic or exhibit other hazard traits. The carcinogenic and mutagenic metabolites of TDCPP include 1,3-dichloro-2- propanol, 3-chloro-1,2-propanediol (3-MCPD), 1,3-dichloro-2-propanone, dichloroacetone, epichlorohydrin, and glycidol.

1. Metabolites of TDCPP

a. Diester, bis(1,3-dicholoro-2-propyl) phosphate (BDCPP) (Lynn et al. 1981; Nomeir et al. 1981; OEHHA 2011b; Sasaki et al. 1984).
b. Monoester, 1,3-dichloro-2-propyl phosphate (MDCPP) (Lynn et al. 1981; OEHHA 2011b).
c. 1,3-Dichloro-2- propanol (Lynn et al. 1981; Nomeir et al. 1981; OEHHA 2011b; Ulsamer et al. 1980).
   - Mutagenic (Gold et al. 1978; Lynn et al. 1981; OEHHA 2010a; OEHHA 2011b).
   - Listed as known to the State of California to cause cancer under Proposition 65 (OEHHA 2011a; OEHHA 2011b).
   - Identified as a Group 2B carcinogen (i.e., possibly carcinogenic to humans) by the International Agency for Research on Cancer (IARC) (IARC 2012a).
   - On the DTSC Candidate Chemicals list (DTSC 2014).
     - Hazard trait: Carcinogenicity
d. 3-MCPD (ATSDR 2012; Nomeir et al. 1981; OEHHA 2011b).
   - Mutagenic (OEHHA 2010b; OEHHA 2011b).
   - Listed as known to the State of California to cause cancer under Proposition 65 (OEHHA 2011a; OEHHA 2011b).
   - Identified as a Group 2B carcinogen (i.e., possibly carcinogenic to humans) by IARC (IARC 2012b).
   - On the DTSC Candidate Chemicals list (DTSC 2014).
     - Hazard trait: Carcinogenicity

2. Proposed metabolite of TDCPP

a. 1,3-Dichloroacetone (aka 1,3-dichloro-2-propanone) (Gold et al. 1978; Nomeir et al. 1981; OEHHA 2011b).
    - Strong, direct-acting mutagen (Gold et al. 1978; OEHHA 2011b).

3. Metabolites of 1,3-dichloro-2- propanol

a. 1,3-Dichloroacetone (OEHHA 2010a; OEHHA 2011b).
    - Mutagen and tumor initiator (OEHHA 2010a; OEHHA 2011b).
b. Epichlorohydrin (OEHHA 2010a; OEHHA 2011b).
    - Listed as known to the State of California to cause cancer under Proposition 65 (OEHHA 2011a; OEHHA 2011b).
    - Identified as a Group 2A carcinogen (i.e., probably carcinogenic to humans) by IARC (IARC 1999b).
    - On the DTSC Candidate Chemicals list (DTSC 2014).
        - Hazard traits: Carcinogenicity, ocular toxicity, reproductive toxicity, respiratory toxicity

4. Metabolites of 3-MCPD

a. Glycidol (OEHHA 2010b; OEHHA 2011b).
    - Listed as known to the State of California to cause cancer under Proposition 65 (OEHHA 2011a; OEHHA 2011b).
    - Identified as a Group 2A carcinogen (i.e., probably carcinogenic to humans) by IARC (IARC 2000).
    - On the DTSC Candidate Chemicals list (DTSC 2014).
        - Hazard traits: Carcinogenicity, reproductive toxicity
b. β-Chlorolactaldehyde (OEHHA 2011b).

VII. Adverse Impacts Associated with Structurally/Mechanistically Similar Chemicals

DTSC may also evaluate and consider the adverse impacts associated with structurally or mechanistically similar chemicals for which there is a known toxicity profile. The compounds listed below have been identified as structurally similar to TDCPP and TCEP (OEHHA 2011). Each of these compounds can be used as a flame retardant. Research studies have demonstrated similar hazard traits and exposure potential for TDCPP, TCEP, and tris(2,3-dibromopropyl) phosphate (TDBPP aka Tris). While long-term carcinogenicity studies have not been conducted on tris(1-chloro-2-propyl) phosphate (TCPP), it is structurally similar to TDCPP and TCEP, has demonstrated genotoxicity in *in vitro* studies, and is listed on DTSC's Candidate Chemicals list.

1. Tris(2,3-dibromopropyl) phosphate (TDBPP; Tris) - a brominated analogue of TDCPP

a. Carcinogenic in rats and mice (Gold et al. 1978; IARC 1999b; OEHHA 2011b).
b. Listed as known to the State of California to cause cancer under Proposition 65 (DTSC 2014; OEHHA 2011a).
c. Identified as a 2A Carcinogen by IARC (DTSC 2014; IARC 2015).
d. Listed as *reasonably anticipated to be a human carcinogen* in the National Toxicology Program's 12th Report on Carcinogens (DTSC 2014; NTP 2010).
e. Identified as a priority chemical by the California Environmental Contaminant Biomonitoring Program (CECBP 2014; DTSC 2014).
f. Genotoxic *in vitro* and *in vivo* (Blum and Ames 1977; Gold et al. 1978; IARC 1999b; OEHHA 2011b).
g. On the Candidate Chemicals list (DTSC 2014).
h. Causes sterility in animals (Blum et al. 1978; Gold et al. 1978).
i. Absorbed through human skin (Gold et al. 1978).

2. Tris(1-chloro-2-propyl) phosphate (TCPP) – a chlorinated phosphate triester

a. Genotoxic in *in vitro* but not *in vivo* assays (EC 2008; OEHHA 2011b).
b. Has not been tested in long-term studies for carcinogenicity (OEHHA 2011b).
c. On the Candidate Chemicals list (DTSC 2014).
d. Identified as a priority chemical by the California Environmental Contaminant Biomonitoring Program (CECBP 2014; DTSC 2014).

VIII. Exposure Potential of People or Wildlife to TDCPP or TCEP in Children's Foam-Padded Sleeping Products

Pursuant to the SCP Regulations, DTSC may draw from a large number of information sources to evaluate exposure including, but not limited to, biomonitoring data, market share data, data on the volume of a chemical or product in commerce, the physicochemical properties of the chemical under evaluation, data indicating a chemical's presence in household dust, on interior surfaces, indoor air, drinking water, surface waters or sediments, or data showing a chemical to be present in (or released from) products present in homes, schools, or places of employment. In evaluating the potential for exposure to TDCPP or TCEP in children's foam-padded sleeping products, DTSC considered the factors below.

A. Exposure Potential to TDCPP in Children's Foam-Padded Sleeping Products

1. Routes of Exposure

a. Routes of exposure include inhalation, ingestion, and dermal absorption (ATSDR 2012).
b. Children's overall exposure to flame retardants may be influenced by their hand-to-mouth behavior (Stapleton et al. 2014).
c. Both inhalation and dust ingestion have been identified as important routes of exposure (Babich 2006; Stapleton et al. 2014).
d. TDCPP is readily absorbed through skin and the gastrointestinal tract in laboratory animals (Nomeir et al. 1981).
e. Occupational exposure to TDCPP may occur through dermal contact and inhalation at workplaces where TDCPP is produced or used (HSDB 2015).
f. Monitoring data indicate that the general population may be exposed to TDCPP via ingestion of drinking water (HSDB 2015).
g. Adult exposures to TDCPP have been confirmed by detection of TDCPP in breast milk (Kim et al. 2014; Sundkvist et al. 2010) and hand wipe samples (Hoffman et al. 2015b), and the detection of urinary metabolites (Butt et al. 2014; Hoffman et al. 2014).
h. Children's exposures to TDCPP have been confirmed by hand wipe samples (Stapleton et al. 2014) and detection of urinary metabolites (Butt et al. 2014).
i. Infant exposure has been confirmed through by detection of TDCPP in breast milk (Kim et al. 2014; Sundkvist et al. 2010).
j. Presence of TDCPP contamination in surface water and wildlife has been confirmed in California and in several countries (Evenset et al. 2009; Kim et

al. 2007; Klosterhaus et al. 2012; Kolpin et al. 2002; SFEI 2013; Sundkvist et al. 2010).

2. Market Presence

a. TDCPP is a high production volume chemical (OEHHA 2011b; U.S. EPA 2006). Approximately 10 to 50 million pounds/year of TDCPP is produced in the U.S. (U.S. EPA 2013).
b. TDCPP is one of the most widely used flame retardants in polyurethane foam (Markets and Markets 2012).
c. Chlorinated flame retardants such as TDCPP are widely used in infant products (Markets and Markets 2012).
d. The global market for chlorinated flame retardants was estimated at approximately 360 million pounds in 2011 and is expected to reach approximately 440 million pounds by 2017 (Markets and Markets 2012).
e. Several manufacturers in China list TDCPP on their websites as one of multiple flame retardant chemicals available for purchase.
f. In a survey of 63 U.S. companies that manufacture or import and distribute infant products, approximately 1.8 million play yards and greater than 2 million play yards were sold in 2011 and 2012 in the U.S., respectively. Approximately 500,000 and 570,000 cradles and bassinets were sold in the U.S. in 2011 and 2012, respectively (JMPA 2013).

3. Studies on the Presence of TDCPP in Foam-Padded Products

a. TDCPP was the most common flame retardant detected in a study which analyzed 101 polyurethane foam samples from commonly used baby products in the U.S. including sleep positioners, portable mattresses, nursing pillows, baby carriers, high chairs, car seats, changing table pads, and baby walkers (Stapleton et al. 2011).
b. The Center for Environmental Health (CEH) had foam samples from 24 children's nap mats analyzed for flame retardants. TDCPP was detected in 9 of the 24 nap mats (Cox 2013).

4. Containment of the Chemical of Concern within the Product

a. TDCPP in polyurethane foam is not chemically bonded to the foam and can migrate into air and dust throughout the lifetime of the product. Losses to the environment may occur through volatilization, leaching, or abrasion (Marklund et al. 2003). TDCPP can also migrate to the surface of the product where people can be dermally exposed

5. Studies on the Presence of TDCPP in Indoor Dust and Air

a. Flame retardant concentrations were measured in air and dust from 40 California ECE facilities. Detected concentrations of TDCPP in dust were higher in ECE facilities where foam nap mats were used compared to ECE facilities where foam nap mats were not used. Levels of TDCPP were higher indoors compared to outdoors. Child TDCPP exposure estimates in this study exceeded the age-adjusted NSRL for carcinogenicity in 51% of the facilities for children less than six years old (Bradman et al. 2014; Bradman et al. 2012).
b. TDCPP has been detected in indoor dust samples from multiple locations in the United States and abroad including homes, offices, hotels, retail spaces, automobiles, and commercial airplanes (Abdallah and Covaci 2014; Ali et al. 2012a; Ali et al. 2012b; Allen et al. 2013; Bergh et al. 2011; Brandsma et al. 2014; Brommer et al. 2012; Cao et al. 2014; Carignan et al. 2013; Dirtu et al. 2012; Dodson et al. 2012; Hoffman et al. 2015b; Marklund et al. 2003; Marklund et al. 2005a; Meeker and Stapleton 2010; OEHHA 2011b; Schreder and La Guardia 2014; Staaf and Ostman 2005; Stapleton et al. 2009; Takigami et al. 2009; Van den Eede et al. 2011).
c. In a study of 30 homes in North Carolina in which children ages 2-5 lived, TDCPP was detected in 100% of house dust samples (Stapleton et al. 2014).
d. In a study of dust collected from 16 homes in California in 2006 and 2011, TDCPP was detected at concentrations higher than previously reported in the U.S. (Dodson et al. 2012).
e. TDCPP was detected in the dust from 96% of the homes included in a study of 50 homes in Boston, MA (Stapleton et al. 2009).
f. In a study conducted in Sweden, TDCPP was detected in dust and air samples taken from homes, day care centers, hospitals, and offices (Marklund et al. 2003).
g. TDCPP was detected in air and dust samples taken from day care centers, workplaces, and homes in Sweden. The air concentrations of TDCPP were approximately 2-8 times higher in day care centers and workplaces than in homes (Bergh et al. 2011).
h. Inhalation exposure was assessed using active personal air samplers in Washington State with both respirable and inhalable particulate fractions collected to assess the likelihood particles penetrate deep into the lungs. TDCPP was detected in three of nine (33%) of the inhalable fraction samples and 50% of the respirable fraction samples. In general, higher levels of TDCPP were detected in the inhalable particulate fraction. Total intake of chlorinated flame retardants via inhalation exposure was estimated to exceed

intake via dust ingestion, indicating that inhalation is an important route of exposure (Schreder et al. 2016).

6. Studies on the Presence of TDCPP in Hand Wipe Samples

a. In a study of 30 North Carolina homes, hand wipe samples were taken from 43 children ages 2-5 years old and 96% contained TDCPP. Further, higher levels of flame retardants detected in house dust were consistently associated with higher hand wipe levels (Stapleton et al. 2014).
b. In a study of indoor exposure to TDCPP in North Carolina homes, TDCPP was detected in 90.6% of hand wipe samples taken from 53 adults (Hoffman et al. 2015b).

7. Biomonitoring

a. TDCPP is listed as a priority chemical by the California Environmental Contaminant Biomonitoring Program (CECBP 2014).
b. TDCPP collects in adipose (fat) tissue (CECBP 2008). TDCPP has been detected in adipose tissue (LeBel and Williams 1983; LeBel and Williams 1986; LeBel et al. 1989) and in human seminal plasma (Hudec et al. 1981).
c. TDCPP has been detected in the lipids of human breast milk in Sweden (Sundkvist et al. 2010).
d. TDCPP was detected in human breast milk in Japan (Kim et al. 2014).
e. The primary metabolite of TDCPP, BDCPP, was detected in 38 out of 39 urine samples from a cohort of pregnant women in North Carolina (Hoffman et al. 2014).
f. BDCPP was detected in 100% of urine samples taken from 21 mother-toddler pairs (Butt et al. 2014). Further, BDCPP urinary levels in children were 4.9 times those of the mothers (Butt et al. 2014), suggesting that children had greater exposure to TDCPP, or a greater dose due to their smaller body mass.
g. BDCPP was detected in 94% of urine samples taken from 16 adults living in northern California homes (Dodson et al. 2014).
h. BDCPP was detected in urine samples taken from seven men in the U.S. over the course of 3 months. TDCPP in house dust was measured in the same study and a correlation between urinary BDCPP and TDCPP concentrations in house dust was noted. This study concluded that house dust might be an important source of exposure to TDCPP (Meeker et al. 2013).
i. A recent study measured the metabolite BDCPP in urine from children ages 2-18 months, and determined that BDCPP levels were strongly associated

with the number of foam-containing infant products (e.g., play yards, sleep positioners, and bassinets) the parents reported owning in the home. Children with greater than 16 products in the home had BDCPP levels that were 6.8 times higher than those with less than 13 products (Hoffman et al. 2015a).

8. Human Exposure Estimates

a. The U.S. EPA estimates that children ingest an approximate average of 60 mg dust/day, whereas adults ingest an approximate average of 30 mg dust/day (U.S. EPA 2011).
b. The calculated cumulative average exposure to flame retardants from dust is 1.6 µg/day for children and 0.325 µg/day for adults (Stapleton et al. 2009).
d. An analysis of potential dust exposures to several flame retardants, including TDCPP, suggests that an adult consumer may be exposed to a median concentration of 0.05 ng TDCPP/kg bw/day and a toddler may be exposed to a median concentration of 0.73 ng TDCPP/kg bw/day using mean dust ingestion assumptions. (Ali et al. 2012a). Thus, children may be receiving much higher exposures to TDCPP than adults due to ingestion of dust. Infant exposure has been confirmed through by detection of TDCPP in breast milk (Kim et al. 2014; Sundkvist et al. 2010).
c.
e. Stapleton et al. have predicted that infants may receive greater exposure to TDCPP from products containing polyurethane foam than the average child or adult receives from upholstered furniture. Infants have smaller body mass than adults, and spend a greater portion of their time in intimate contact with foam-padded sleeping products (Stapleton et al. 2011).
f. It has been estimated that children's exposure to TDCPP from treated furniture foam is five times higher than the ADI for non-cancer endpoints (Babich 2006). Scrap foam from furniture is sometimes used in children's foam-padded sleeping products.
g. It has been estimated that the cancer risk for a lifetime of exposure to TDCPP-treated upholstered furniture is 300 per million. In children, the estimated cancer risk from exposure to upholstered furniture during the first two years of life is 20 per million (Babich 2006). Consumer Product Safety Commission (CPSC) staff considers cancer risks greater than one in a million relevant for regulatory consideration (Babich 2006).

9. Potential for the Chemical of Concern to be Released into Environmental Media

Published reports indicate that TDCPP has been found in wastewater treatment plant influent and effluent, laundry wastewater, surface water, drinking water, sediment, and wildlife.

a. TDCPP in Water

- TDCPP has been detected in San Francisco Bay waters and sediment. Further, TDCPP is relatively abundant in San Francisco Bay sediment, with concentrations comparable to those of polybrominated biphenyl ethers (PBDEs) and polychlorinated biphenyls (PCBs) in the same samples (Klosterhaus et al. 2012; SFEI 2013). PBDEs and PCBs have been banned or phased out of production due to their environmental persistence and high concentration levels previously detected in the environment.
- TDCPP has been detected in surface water in over half of 139 freshwater streams studied across the U.S., including streams in California, suggesting significant releases of TDCPP to the environment (Kolpin et al. 2002; OEHHA 2011b).
- Samples from Lake Mead, NV have been found to contain TDCPP and other organophosphorus compounds. There was evidence to suggest that the water infiltrated into the sediment had a different chemical composition than the rest of the water column and could be a potential exposure risk to bottom-dwelling aquatic organisms (Alvarez et al. 2012).
- TDCPP has been detected in river water, drinking water, treated wastewater, and influents, effluents, and sludge of sewage treatment facilities (Andresen et al. 2004; Green et al. 2008; Marklund et al. 2005b; Martinez-Carballo et al. 2007; OEHHA 2011b; Rodil et al. 2012; Stackelberg et al. 2004).
- TDCPP was detected in 100% of household laundry wastewater samples taken from 20 Washington state homes, as well as in influents and effluents of two wastewater treatment plants associated with those homes (Schreder and La Guardia 2014), indicating the release of TDCPP to waterways from effluent.
- Precipitation and in storm water runoff samples in Germany were found to contain TDCPP; however, the concentrations of TDCPP were below the analytical limit of detection in several precipitation samples (Regnery and Puttmann 2010).

- TDCPP has been detected in groundwater and surface water sources used for drinking water at very low concentration levels, typically below the analytical reporting limit (Barnes et al. 2008; Focazio et al. 2008; Schaider et al. 2014).
- In a 2006 study in Germany, TDCPP was detected in surface water used for drinking; however, after the water went through the purification process, it was detected at very low concentrations (Andresen and Bester 2006).

b. TDCPP in Sediment
   - TDCPP was found in sediments from Taihu Lake, one of the largest freshwater lakes in China (Cao et al. 2012).
   - Bottom sediment in Lake Mead, NV contained TDCPP (Alvarez et al. 2012).

c. TDCPP in Leachate
   - Leachate from a solid waste disposal site near Osaka, Japan was found to contain TDCPP (Kawagoshi et al. 2002).

d. TDCPP in Wildlife
   - TDCPP has been detected in fishes, mussels (Evenset et al. 2009; Green et al. 2008; Takahashi et al. 2013) and bird blood/plasma and eggs in Norway (Leonards et al. 2011; Takahashi et al. 2013).
   - In Sweden, TDCPP has been detected in freshwater fishes from lakes close to emission sources (Sundkvist et al. 2010; Takahashi et al. 2013).

B. Exposure Potential of TCEP in Children's Foam-Padded Sleeping Products

1. Routes of Exposure

a. Occupational exposure to TCEP may occur through inhalation and dermal contact with this compound at workplaces where TCEP is produced or used (HSDB 2015).
b. A 2009 risk assessment identified inhalation, ingestion, and dermal absorption as potential routes of occupational and consumer exposure to TCEP (EC 2009).
c. Monitoring data indicate that the general population may be exposed to TCEP via inhalation of ambient air, ingestion of contaminated food and drinking water, and dermal contact with consumer products containing TCEP (HSDB 2015).

d. Children's overall exposure to flame retardants may be influenced by their hand-to-mouth behavior (Stapleton et al. 2014).
e. The presence of TCEP in surface water and wildlife has been confirmed in several countries (van der Veen and de Boer 2012).
f. Adult exposures have been confirmed by detection of TCEP in human breast milk (Kim et al. 2014; Sundkvist et al. 2010) and the detection of urinary metabolites (Dodson et al. 2014; Schindler et al. 2009).
g. Children's exposures to TCEP have been confirmed by hand wipe samples (Stapleton et al. 2014).
h. Infant exposure has been confirmed through by detection of TCEP in breast milk (Kim et al. 2014; Sundkvist et al. 2010).

2. Market Presence

a. Production volume for TCEP of 500,000-1,000,000 lbs. was reported to the US EPA in 2006 under the Inventory Update Rule (TOXNET 2014).
b. Global production appears to have peaked with 1.8 million pounds produced in 1989 and declining amounts in subsequent years (WHO 1998).
c. TCEP is no longer produced within the European Union, as of 2009 (EC 2009).
d. Chlorinated flame retardants such as TCEP are widely used in infant products (Markets and Markets 2012).
e. The global market for chlorinated flame retardants was estimated at approximately 360 million pounds in 2011 and is expected to reach approximately 440 million pounds by 2017 (Markets and Markets 2012).
f. Several manufacturers in China list TCEP as one of multiple flame retardant chemicals available for purchase on their websites.
g. In a survey of 63 U.S. companies that manufacture or import and distribute infant products, approximately 1.8 million play yards and greater than 2 million play yards were sold in 2011 and 2012 in the U.S., respectively. Approximately 500,000 and 570,000 cradles and bassinets were sold in the U.S. in 2011 and 2012, respectively (JMPA 2013).

3. Studies on the Presence of TCEP in Foam-Padded Products

a. Analysis of multiple consumer products has identified TCEP in sleep positioners, portable mattresses, nursing pillows, baby carriers, children's car seats, changing table pads, and infant bath mats (Stapleton et al. 2011).

4. Containment of the Chemical of Concern within the Product

a. TCEP in polyurethane foam is not chemically bonded to the foam and can migrate into air and dust throughout the lifetime of the product. Factors such as volatilization, leaching, or abrasion may contribute to this (Marklund et al. 2003). TCEP can also migrate to the surface of the product where people can be dermally exposed.

5. Studies on the Presence of TCEP in Indoor Dust and Air

a. Flame retardant concentrations were measured in air and dust from 40 California ECE facilities. Detected concentrations of TCEP in dust were higher in ECE facilities where foam nap mats were used compared to ECE facilities where foam nap mats were not used. Levels of TCEP were higher indoors compared to outdoors (Bradman et al. 2014).
b. Multiple indoor locations were sampled in the Stockholm area of Sweden, and TCEP was detected in home, work, and day care environments. Samples taken from day care centers had the highest TCEP concentrations among the indoor environments (Bergh et al. 2011).
c. In a study exploring associations of flame retardants in children's hand wipes to house dust, TCEP was found in both dust and hand wipe samples (Stapleton et al. 2014).
d. TCEP has been detected in indoor dust samples from multiple locations in the United States and abroad including homes, hotels, offices, retail spaces, and automobiles (Abdallah and Covaci 2014; Ali et al. 2012a; Ali et al. 2012b; Bradman et al. 2014; Brandsma et al. 2014; Brommer et al. 2012; Cao et al. 2014; Dirtu et al. 2012; Dodson et al. 2012; Ingerowski et al. 2001; Marklund et al. 2003; Schreder and La Guardia 2014; Stapleton et al. 2014; Takigami et al. 2009; Van den Eede et al. 2011).
e. TCEP is present as an impurity, at about 5-10%, in the flame retardant tetrekis (2-chloroetheyl) dichloroisopentyldiphosphate, known as V6. Production and use of V6 could lead to environmental releases of TCEP (EC 2009). In one study, TCEP was found as an impurity in a V6 commercial mixture at levels of 14% by weight. In the same study, TCEP was found in house and automobile dust samples, a significant correlation between the concentrations of TCEP and the flame retardant V6 was observed in the dust samples, suggesting that the use of V6 is a significant source of TCEP in indoor environments (Fang et al. 2013).
f. Indoor air environments sampled around the world have also been found to be contaminated with TCEP including theaters, offices, retail establishments, and homes (Bergh et al. 2011; Bradman et al. 2014; Hartmann et al. 2004;

Ingerowski et al. 2001; Makinen et al. 2009; Marklund et al. 2003; Marklund et al. 2005a; Staaf and Ostman 2005).

g. Inhalation exposure was assessed using active personal air samplers in Washington State with both respirable and inhalable particulate fractions collected to assess the likelihood particles penetrate deep into the lungs. TCEP was deterred in eight of nine (89%) of the inhalable fraction samples and none (0%) of the respirable fraction samples. Higher levels of TCEP were detected in the inhalable particulate fraction. Total intake of chlorinated flame retardants via inhalation exposure was estimated to exceed intake via dust ingestion, indicating that inhalation is an important route of exposure (Schreder et al. 2016).

6. Studies on the Presence of TCEP in Children's Hand Wipe Samples

a. In a recent study, 43 children from 30 families were sampled for the presence of multiple flame retardants including TCEP on hand wipes and in house dust. TCEP was found on 47% of hand wipe samples and in 100% of house dust samples (Stapleton et al. 2014).

7. Biomonitoring

a. TCEP is listed as a priority chemical by the California Environmental Contaminant Biomonitoring Program (CECBP 2014).
b. Samples of human breast milk in women from 4 urban areas in Sweden contained TCEP as well as other flame retardants (Sundkvist et al. 2010).
c. TCEP has been detected in human breast milk in Japan, Vietnam and the Philippines (Kim et al. 2014).
d. The urinary metabolite of TCEP, bis(2-chloroethyl) phosphate (BCEP), was detected in 75% of urine samples taken from 16 adults living in northern California homes and TCEP was detected in 13% of the same samples (Dodson et al. 2014).
e. The urinary metabolite BCEP was detected in 50% of urine samples taken in Germany from persons ranging in age from 11 to 68 years (Schindler et al. 2009).

8. Human Exposure Estimates

a. The U.S. EPA estimates that children ingest an approximate average of 60 mg dust/day, whereas, adults ingest an approximate average of 30 mg dust/day (U.S. EPA 2011).
b. The calculated cumulative average exposure to flame retardants from dust is 1.6 µg/day for children and 0.325 µg/day for adults (Stapleton et al. 2009).

c. An analysis of potential dust exposures to several flame retardants, including TCEP, suggests that an adult consumer may be exposed to a median concentration of 0.02 ng TCEP/kg bw/day and a toddler may be exposed to a median concentration of 0.34 ng TCEP/kg bw/day using mean dust ingestion assumptions (Ali et al. 2012a). Thus, children may be receiving much higher exposures to TCEP than adults due to ingestion of dust.
d. Dietary intake estimates of TCEP have been calculated to be 4.9 ng/kg and 6.5 ng/kg for children aged 6-11 months and 2 years old, respectively. Estimates for adults range from 1.3 - 3.1 ng/kg (ATSDR 2012; Gunderson 1995). Thus, children may be receiving much higher exposures to TCEP than adults due to ingestion from food sources.

9. Potential for the Chemical of Concern to be Released into Environmental Media

Published reports indicate that TCEP can be found in wastewater treatment plant effluents, surface water, finished drinking water, wildlife, sediments, and Antarctic ice.

a. TCEP in Water
   - Samples from urban river systems and lakes have been found to contain TCEP and other organophosphorus compounds within California and Nevada (Alvarez et al. 2012; Sengupta et al. 2014). In one study, there was evidence to suggest that the water infiltrated into the sediment had a different chemical composition than the rest of the water column and could be a potential exposure risk to bottom-dwelling aquatic organisms (Alvarez et al. 2012).
   - Streams, drinking water, ground water, wastewater and laundry effluent were found to contain TCEP in several US states (Barnes et al. 2008; Kolpin et al. 2002; Schreder and La Guardia 2014; Stackelberg et al. 2004).
   - Outside of the US, TCEP has been found in river water, rain water, storm water runoff, aquifers, drinking water, treated waste water, and influents, effluents, and sludge of sewage treatment facilities (Andresen and Bester 2006; Andresen et al. 2004; Bacaloni et al. 2007; Dsikowitzky et al. 2004; Fries and Puttmann 2001; Green et al. 2008; Kim et al. 2007; Marklund et al. 2005b; Martinez-Carballo et al. 2007; Matamoros et al. 2012; Meyer and Bester 2004; Regnery and Puttmann 2010; Rodil et al. 2012; Rodriguez et al. 2006).
   - TCEP was detected in 100% of household laundry wastewater samples taken from 20 Washington state homes, as well as in influents and effluents of two wastewater treatment plants associated with those homes

(Schreder and La Guardia 2014), indicating the release of TCEP to waterways from effluent.

b. TCEP in Sediment
- In Austria, TCEP was detected in river sediment within the Schwechat River (Martinez-Carballo et al. 2007).
- Sediment samples from four locations in Norway contained TCEP as well as other contaminants (Leonards et al. 2011).
- TCEP was found in sediments from Taihu Lake, one of the largest freshwater lakes in China (Cao et al. 2012).

c. TCEP in Antarctic Ice
- Analysis of the East Antarctic Ice Sheet identified TCEP as one of several contaminants found in fresh snow samples (Cheng et al. 2013).

d. TCEP in Leachate
- Leachate from a solid waste disposal site near Osaka, Japan was found to contain TCEP (Kawagoshi et al. 2002).

e. TCEP in Wildlife
- A study in Sweden found TCEP in herring, perch, mussels, and salmon (Sundkvist et al. 2010).
- A separate analysis in Sweden also found TCEP in mussels, crab, fish, and eagles (Leonards et al. 2011).

IX. Potential Exposure of Sensitive Subpopulations to TDCPP or TCEP

1. Infants and children

a. Infants may be exposed to TDCPP and TCEP through breast milk, as evidenced by studies detecting TDCPP and TCEP in human breast milk (Kim et al. 2014; Sundkvist et al. 2010).
b. Children's exposures to TDCPP and TCEP have been confirmed by hand wipe samples (Stapleton et al. 2014) and detection of urinary metabolites (Butt et al. 2014).
c. The primary urinary metabolite of TDCPP (BDCPP) has been detected in urine samples taken from toddlers (Butt et al. 2014). Further, BDCPP urinary levels in children were 4.9 times those of the mothers tested in the same study (Butt et al. 2014), suggesting that either children were exposed to a

greater amount of TDCPP or had higher metabolite levels due to their smaller body mass.

d. Children's overall exposure to flame retardants may be influenced by their hand-to-mouth behavior (Stapleton et al. 2014).
e. TDCPP and TCEP have been detected in numerous foam-filled children's products (Stapleton et al. 2011).
f. TDCPP has been detected in dust samples in homes where children ages 2-5 lived (Stapleton et al. 2014).
g. The U.S. Environmental Protection Agency (U.S. EPA) estimates that children ingest on average approximately 60 mg dust/day; this is significantly more than adults, who on average ingest approximately 30 mg dust/day (U.S. EPA 2011). As a result, children may have a greater exposure to TDCPP in dust than adults. Further, due to children's smaller body mass relative to adults, the dosage received by children in mg/kg of body mass is substantially greater than this twofold dust ingestion rate difference with adults might suggest.
h. One study calculated a cumulative average exposure to flame retardants from dust of 1.6 µg/day for children and 0.325 µg/day for adults (Stapleton et al. 2009).
i. Based on human exposure estimates (Ali et al. 2012a; ATSDR 2012; Gunderson 1995), children may be receiving much higher exposures to TDCPP and TCEP than adults due to ingestion of dust and food sources. Thus, children's exposure to TDCPP and TCEP is of concern due to their greater dust ingestion rate and greater exposure on a mg/kg of body weight basis due to their smaller body mass as compared to adults.
j. Flame retardant concentrations were measured in air and dust from 40 California ECE facilities. Detected concentrations of TDCPP and TCEP in dust were higher in ECE facilities where foam nap mats were used compared to ECE facilities where foam nap mats were not used. Levels of TDCPP and TCEP were higher in indoor air as compared to outdoor air. Child TDCPP exposure estimates in this study exceeded the age-adjusted NSRL for carcinogenicity in 51% of the facilities for children less than six years old (Bradman et al. 2014; Bradman et al. 2012).
k. In children, the estimated cancer risk from exposure to upholstered furniture during the first two years of life is 20 per million (Babich 2006).

2. Pregnant women

a. The primary metabolite of TDCPP, BDCPP, was detected in 38 out of 39 urine samples from a cohort of pregnant women (Hoffman et al. 2014).

3. Workers

a. Occupational exposure to TDCPP may occur through dermal contact with and inhalation of this compound at workplaces where TDCPP is produced or used (HSDB 2015).
b. TDCPP and TCEP have been detected in dust and air in California day care centers (Bradman et al. 2014; Bradman et al. 2012).
c. TDCPP and TCEP have been detected in offices, retail spaces, automobiles, hospitals, commercial airplanes, day care facilities, and other public spaces (Ali et al. 2012a; Ali et al. 2012b; Allen et al. 2013; Bergh et al. 2011; Brommer et al. 2012; Cao et al. 2014; Carignan et al. 2013; Dirtu et al. 2012; Dodson et al. 2012; Hartmann et al. 2004; Hoffman et al. 2015b; Makinen et al. 2009; Marklund et al. 2003; Marklund et al. 2005a; Meeker and Stapleton 2010; OEHHA 2011b; Schreder and La Guardia 2014; Staaf and Ostman 2005; Stapleton et al. 2009; Takigami et al. 2009; Van den Eede et al. 2011).

X. Conclusions

DTSC identified children's foam-padded sleeping products containing TDCPP or TCEP as a Priority Product. This determination was based on a consideration of available, reliable scientific information regarding the potential exposure to TDCPP or TCEP in children's foam-padded sleeping products and the potential for these exposures to contribute to or cause significant or widespread adverse human health impacts.

TDCPP and TCEP are semi-volatile compounds used as additive flame retardants that are not chemically bonded to polyurethane foam and are easily released to indoor and outdoor environments. Both TDCPP and TCEP are ubiquitous compounds and have been detected worldwide, including in California, in dust sampled in indoor environments such as homes, offices, and daycare centers. TDCPP and TCEP have been detected in waterways and wastewater treatment influent and effluent in the U.S. and other nations. Further, TDCPP and TCEP have been detected in wildlife such as fish, mussels, and birds.

Both TDCPP and TCEP are known to the State of California to cause cancer, and carcinogenicity has been demonstrated in animal studies for both TDCPP and TCEP. Research studies suggest that TDCPP and TCEP exposure is associated with other hazard traits including reproductive toxicity, neurotoxicity, and kidney and liver toxicity. TDCPP exposure has also been linked to developmental toxicity and endocrine disruption.

Human exposure to TDCPP has been demonstrated by detection in human breast milk, adipose tissue, and seminal plasma, as well as the detection of primary metabolites in urine samples collected from adults, including pregnant women, and children. Human exposure to TCEP has been demonstrated by detection in human breast milk, as well as detection of primary metabolites in adult urine samples. Further, TDCPP has been detected in hand wipe samples from adults and children and TCEP has been detected in hand wipe samples from children, demonstrating an important route for potential exposure to these chemical flame retardants.

DTSC determined that exposure to TDCPP or TCEP through the normal use of children's foam- padded sleeping products may contribute to or cause significant or widespread adverse health impacts with the greatest risks borne by sensitive subpopulations such as pregnant women, children, infants, and day care center and school employees. This determination is based on the ubiquitous detection of TDCPP and TCEP in indoor and outdoor environments, the hazard traits associated with each compound, and the data showing widespread exposures to both TDCPP and TCEP in adults, children, and wildlife.

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