CPSC Staff Statement on Toxicology Excellence for Risk Assessment (TERA) Report "Environmental Concentrations and Consumer Exposure Data for Tris(2-Chloroethyl)Phosphate (TCEP)"[1]

June 2015

The report titled, "Environmental Concentrations and Consumer Exposure Data for Tris(2-Chloroethyl)Phosphate (TCEP)," presents environmental and consumer exposure data on TCEP conducted by TERA under Contract CPSC-D-12-0001, Task Order 0008.

First, TERA provides information on TCEP's chemical and physical properties and its use. Next, TERA provides human exposure data, both domestic and international, from the following media: indoor and ambient air, water, food, dust, and consumer products. Absorption, distribution, metabolism, excretion, and biomonitoring studies are summarized next. This report concludes with existing exposure assessment and exposure estimate summaries and a report discussion.

Based on this report, TCEP has been detected in outdoor and indoor air, surface water, groundwater, house dust, food, and consumer products. TCEP and its metabolites have also been detected in breast milk and urine samples. V6, another flame retardant, contains a significant level of TCEP as an impurity and may be an additional source of exposure to TCEP. The main sources of consumer exposure to TCEP appear to be from indoor air and dust. The exposure routes of importance are dust ingestion and inhalation of vapors and particulates in indoor air.

Limited U.S. data exist for these exposure sources, and data from other countries may not be representative of U.S. levels. Additionally, due to California's regulations, levels of TCEP in dust may not be representative of nationwide levels, and using this data for exposure estimating may result in conservative estimates for other parts of the country.

While the general mechanism for TCEP diffusion out of treated plastics and into dust is relatively well understood, dermal exposure from contact with or ingestion from mouthing of objects containing TCEP, such as changing table pads, infant sleep positioners, portable crib mattresses, and upholstered furniture, is not.

Given the limited data available and lack of information on the migration and degradation from indoor air media and dermal exposure factors, performing an exposure assessment is difficult. However, reasonable worst case estimates of exposure could be made using the media concentrations presented in this document along with age-specific estimates of inhalation or ingestion rates of these media.

[1] This statement was prepared by the CPSC staff, and the attached report was produced by TERA for CPSC staff. The statement and report have not been reviewed or approved by, and do not necessarily represent the views of, the Commission.

TERA

INDEPENDENT
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FOR PUBLIC HEALTH PROTECTION

Environmental Concentrations and Consumer Exposure Data for Tris(2-Chloroethyl) Phosphate (TCEP)

Task Order 8
Contract Number CPSC-D-12-0001

Final

June 26, 2015

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Abbreviations and Acronyms

AC	Activity coefficient
ADME	Absorption, Distribution, Metabolism, and Excretion
ATSDR	Agency for Toxic Substances and Disease Registry
AUC	Area under the plasma concentration-time curve
BCCP	Bis(2-chloroethyl) carboxymethyl phosphate
BCEP	Bis(2-chloroethyl) phosphate
BCHP	Bis(2-chloroethyl) hydrogen phosphate
CPSC	Consumer Product Safety Commission
DCEP	Di(2-chloroethyl) phosphate
ECHA	European Chemicals Agency
EURAR	European Union Risk Assessment Report
HSDB	Hazardous Substances Data Bank
IARC	International Agency for Research on Cancer
ICRP	International Commission on Radiological Protection
IPCS	International Programme on Chemical Safety
LD_{50}	Median lethal dose
MBR	Membrane bioreactor
MSDS	Material Safety Data Sheet
NF	Nanofiltration
NRC	National Research Council
NTP	National Toxicology Program
OECD	Organisation for Economic Co-operation and Development
PU	Polyurethane
PUF	Polyurethane foam
PVC	Polyvinylchloride
RO	Reverse osmosis
SPE	Solid phase extraction
SPME	Solid-phase microextraction
SVOCs	Semi-Volatile Organic Compounds
TCEP	Tris(2-Chloroethyl) Phosphate
TERA	Toxicology Excellence for Risk Assessment
TPP	Triphenyl Phosphate
U.S.	United States
U.S. EPA	U.S. Environmental Protection Agency
U.S. FDA	U.S. Food and Drug Administration
USGS	U.S. Geological Survey

UV	Ultraviolet
VP	Vapor pressure

1 Introduction

This document compiles available human exposure information on Tris(2-Chloroethyl) Phosphate (TCEP). Flame retardant chemicals are added to materials to increase a product's resistance to ignition or to decrease the spread of flames. They are used in many different types of consumer products, including upholstery and mattresses, toys and children's products, electrical devices, appliances, building materials, and apparel. Various flame retardants or their metabolites have been detected in human fluids or tissues, indicating human exposure and absorption of at least some of these chemicals.

Toxicology Excellence for Risk Assessment (TERA) reviewed available literature and data relating to human exposure of the TCEP. Data were obtained from assessments of the flame retardants prepared by United States (U.S.) government agencies and other authoritative bodies. We also searched the scientific literature for available data on human exposures and environmental measurements of flame retardants. In particular, data on concentrations in indoor air and dust, concentration in potential drinking water as well as consumer products, including children's products, upholstered furniture, mattresses, apparel, household products, building materials, and electronics were sought. The information is compiled by "media" with data on measured concentrations identified in ambient air, indoor air, household dust, drinking water (and surface and groundwater), and food presented in tables. Available measurements of concentrations in consumer products such as electronics, furniture, mattresses, toys, and building materials are also presented. Results of available biomonitoring studies and exposure estimates, if any, are also included. Appendix A includes a description of the literature search strategy, key words, and databases searched.

It should be noted that this report compiles data from a variety of sources. We have not evaluated the quality of the studies and their results; rather we included all the relevant data we found. The estimates described in the exposure assessment sections are presented "as is" without a detailed analysis and critique of the methodology, assumptions, or underlying data quality. Further review on the quality and representativeness of these studies would be needed before using these data to estimate human exposures.

In reviewing the results and compilation of the available literature for this report as well as the report on four other flame retardants, it is apparent that there is a dearth of quality exposure information for these flame retardants to quantify human exposure. We observed several common elements from our review.

There is a basic lack of a lot of monitoring data in environmental media for these chemicals.

The U.S. Geological Survey (USGS) has conducted sampling for some of the flame retardants in drinking water and U.S. waters, but there is no systematic or routine monitoring in place by the federal government. Most of the studies we identified focused on measurements in heavily industrialized rivers where presumably contamination was suspected due to manufacturing. Several studies noted the ability of treatment to remove particular flame retardants from drinking water influent or manufacturing effluent. For some flame retardants, treatment reduced concentrations and in other cases, it did not.

Much of the available information is from studies outside of the U.S.

We found limited U.S. data for these flame retardants. Data from other countries may introduce uncertainty into exposure estimates because particular flame retardant levels in other countries may not be representative of products, flame retardants, and building parameters found in the U.S. Within the U.S., California has traditionally had more stringent flame retardant regulations, and levels of flame retardants in dust in California may be higher than in other parts of the country.

Limited data on emission or migration rates from products and materials containing the flame retardant chemicals.

Unlike concentrations in environmental media (e.g., air, water, dust), flame retardant concentrations measured in consumer products and building materials cannot be used directly as a proxy for concentration levels to which consumers are exposed. Any product that contains a flame retardant has the potential to contribute to household dust levels. To develop realistic consumer exposure concentrations, the flame retardant levels in these products would need to be paired with experimental or monitoring results that reflect the availability of the specific compound to leave these products and be available to enter the body. These data are not generally available. Alternatively, emission or migration rates could be used to model or estimate exposures for:

- Near-field exposure for persons proximate to the device
- Far-field exposure in the room and building
- Long term rate of flame retardant input to the space

For example, the work of Carlsson et al. (2000) detecting and measuring Triphenyl Phosphate (TPP) in air sampled in the breathing zone of a computer user could be used to estimate exposure. Another example is the work of Saito et al. (2007) measuring the actual rate of migration of TPP from the outer case of an electronic device to a solid extraction disk.

Without the data to make the link between concentrations and exposure levels, the data on flame retardant content in a product provides only a qualitative indication of the ultimate availability of that flame retardant to be released into the indoor environment, either into the air or dust. The rate of release will depend upon the individual products, their physical and chemical properties, and how they are used.

Flame retardants used in many products that come in contact with people's skin, but there is uncertainty associated with percutaneous exposure from a lack of testing,

An area of uncertainty is exposure potential from dermal contact with or without ingestion from mouthing of objects containing a flame retardant. The general mechanism for diffusion out of treated plastics and into dust is relatively well understood based on first principle models (e.g., http://www.epa.gov/nrmrl/appcd/mmd/i-sovc.html). Similarly, the potential for hand-to-mouth transfer of dust is understood and established. This is not the case for dermal or mouthing transfer of this flame retardant from contact with treated objects. The potential dermal or mouth exposure to a flame retardant as it is diffusing and being "expressed" from the treated plastic is not well understood or documented. The flame retardant molecules will have some rate of dermal penetration if they are in contact with the skin. This exposure route may be particularly important for children's' items, such as changing table pads, infant sleep positioners, portable crib mattresses, and nursing pillows, as well as clothing. Transfer and ingestion via child-mouthing of these items, toys, and treated furniture in general is another area of uncertainty.

Changes in usage of particular flame retardants as manufacturers and government agencies identify potential problems and substitute alternative flame retardants.

As government agencies, manufacturers, and consumers are more aware of flame retardant usage and potential exposures, usage patterns change, with new chemicals substituting for old or different concentrations and chemicals used in new products. Past measurements may not be representative of current conditions, with changing product content or usage patterns. Also, the time frame of these exposure events suggests that the exposure potential could continue to grow for months or years after the initial use and placement of treated objects indoors.

Both dust and vapors may contribute to total concentrations in indoor air.

Depending upon the flame retardant's properties, a particular flame retardant may or may not be anticipated to become airborne as a vapor out of organic substrates indoors. However, particulates, such as dust, are quite mobile and can become airborne. The flame retardant could be distributed within the indoor environment in dust on surfaces with a lesser amount of the flame retardant containing dust being airborne. An exception to this rule of low airborne levels would be relatively "dusty" rooms where the dust has been allowed to accumulate and/or activities occurs that tend to continually entrain the dust into the air. In these indoor

environments, relatively high levels of airborne flame retardant (dissolved in dust) may be found. This as well as computer equipment and furniture may explain some rare reports of relatively high levels of flame retardant in the indoor air of an office or hospital ward (see for example, Marklund et al., 2005a).

2 Tris(2-Chloroethyl) Phosphate (TCEP) (CAS 115-96-8)

2.1 TCEP Chemical and Physical Properties

TCEP is a widely used flame retardant and is a clear, viscous liquid with little odor (HSDB, 2013). The water solubility value of 7.0 g/L at 24°C indicates that TCEP has low solubility in water but it is soluble in most solvents and will adsorb to solids and sediments in water based on an estimated soil-water coefficient (K_{oc}) value of 390 (HSDB, 2013). A measured vapor pressure for TCEP of 6.1 x 10^{-2} torr at 25 °C indicates that TCEP will exist in both the vapor and particulate phases in the atmosphere (HSDB, 2013). Significant volatilization from water surfaces is not expected based on an estimated Henry's Law constant of 3.29 x 10^{-6} atm-m^3/mol at 25 °C (HSDB, 2013).

Figure 2-1. Molecular Structure of TCEP (ChemIDPlus, 2014)

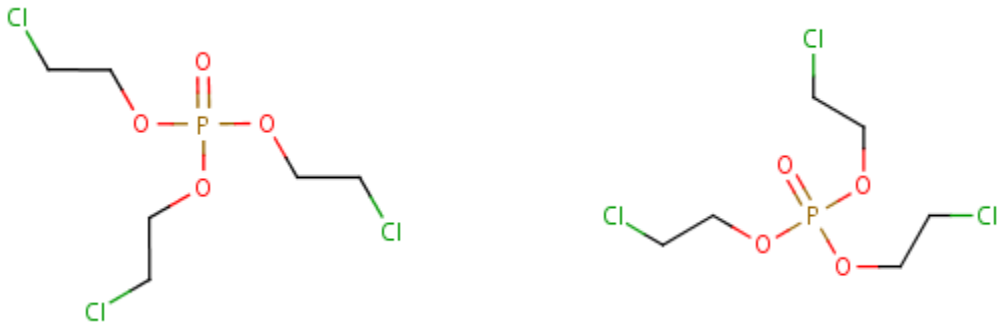

Table 2-1. CAS Registry Number and Synonyms for TCEP (ChemIDPlus, 2014; CalEPA, 2011)

CAS registry/RN	115-96-8 and 29716-44-7
Synonyms	2-Chloroethanol phosphate, 4-01-00-01379 (Beilstein Handbook Reference), AI3-15023, Antiblaze 100, BRN 1710938, CCRIS 1302, Celluflex, Celluflex CEF, Disflamoll TCA, EINECS 204-118-5, EINECS 249-806-6, Ethanol, 2-chloro-, phosphate (3:1), Fyrol CEF, HSDB 2577, NCI-C60128, Niax 3CF, Niax Flame Retardant 3CF, NSC 3213, Phosphoric acid, tris(2-chloroethyl)ester, TCEP, Tri(2-chloroethyl) phosphate, Tri(2-chloroethyl)phosphate, Tri-beta-chloroethyl phosphate, Trichlorethyl phosphate, Tris(2-chloroethyl) orthophosphate, Tris(2-chloroethyl) phosphate, Tris(2-chloroethyl)phosphate, Tris(beta-chloroethyl) phosphate, Tris-(2-chlorethyl)fosfat [Czech], Tris-(2-chloroethyl)fosfat [Czech], UNII-32IVO568B0

Table 2-2. Physical and Chemical Properties of TCEP (ChemIDPlus, 2014; HSDB, 2013)

Molecular Formula	C6-H12-Cl3-O4-P
Molecular Weight	285.4898
Melting Point	-35˚C (ChemIDPlus, 2014) -55˚C (HSDB, 2013)
Boiling Point	330˚C (ChemIDPlus, 2014) 194˚C at 10 mm Hg (HSDB, 2013)
Density	1.39 g/cu cm at 25 ˚C (HSDB, 2013)
Solubility (in water)	7.0 g/L
Log K_{ow}	1.44
Vapor Pressure	6.1 x 10^{-2} torr at 25˚C
Henry's Law Constant	3.29x10^{-6} atm-m^3/mole at 25˚C

The primary properties of TCEP that are relevant to exposure are its molecular weight (285.5g/mole), vapor pressure (6.1 x 10^{-2} torr), water solubility (7.0 g/L) and Log K_{ow} (1.44) (HSDB, 2009, 2011 as cited in ATSDR, 2012). This vapor pressure translates to a room temperature (25^{o}C) saturation vapor concentration for pure TCEP of 940 mg/m^3.

Semi-Volatile Organic Compounds (SVOCs), such as TCEP, are typically characterized by relatively high molecular weights, low vapor pressure/volatility, low or moderate solubility in water and a high octanol-water partition coefficient. To a significant degree, these properties determine the fate of TCEP in the environment, which impacts the potential for human exposure. The high octanol-water partition coefficient means it will be lipophilic or "fat loving"; that is, it will partition into any sediment layers in an environmental water column. Unless there are large areas of volatilizing surface in the indoor environment, it will not be highly present in the air as a vapor but will ultimately partition out of treated objects and into and be present in available organic rich substrates like house dust. If associated with food packaging it could partition into the food.

Because of its properties and fate in the indoor environment, the vapor pressure (VP) of pure TCEP can be misleading in estimating its potential to become airborne as a vapor. Using the measured vapor pressure at typical room temperature of 25°C (73°F) results in the following estimation of a maximum or saturated airborne concentration:

$(6.1x10^{-2}$ torr/760 torr) (1,000,000) (285.5/24.4) = 940 $\mu g/m^3$

All monomers (including monomeric flame retardants) embedded within polymer matrices will move out of that matrix into surrounding media following classic laws of diffusion. Given a time frame of years, a significant portion of the monomer will diffuse out of the polymer and into the residential environment. In reality, TCEP would never be expected to exist as a pure material indoors. Initially, it is in the polymer (typically polyvinyl chloride [PVC] and polyurethane [PU]) matrix and after it diffuses out of the matrix, it comes to the surface. TCEP is then associated with house dust, which is primarily composed of human skin cells that have previously been shed. As such, house dust is essentially an organic substrate. In this case, an estimated $6.1x10^{-2}$ mmHg VP at 25°C (73°F) of pure TCPP is highly attenuated via what is known as Raoult's Law:

(VP of Pure TCEP)(Mole Fraction of TCEP in substrate) = VP over the substrate

This is for "ideal mixtures" of TCEP in various substrates (e.g., in plastic products or typical house dust, which is made of skin cells that are continuously shed by the occupants). For real world mixtures a thermodynamic activity coefficient (AC<<1) is added.

(VP of Pure TCEP)(Mole Fraction of TCEP in substrate)(AC) = VP over the substrate

Thus, one would not expect but a small portion of the above saturation airborne concentration to occur in indoor air.

2.2 TCEP Uses

TCEP has been used as a flame retardant in a variety of commercial products, as well as in paints/glue and in industrial environments (Marklund et al., 2003, as cited in ATSDR, 2012). ATSDR (Agency for Toxic Substances and Disease Registry) reports that it has largely been used as a flame retardant for flexible and rigid polyurethane foams and for some textiles and clothing (Anderson et al., 2004, as cited in ATSDR, 2012). However, Health Canada reports that TCEP is being substituted with other flame-retardant substances (IPCS, 1998; EURAR, 2006, as cited in Health Canada, 2009) and TCEP is not recommended for use as a flame retardant in apparel fabrics (IPCS, 1998, as cited in Health Canada, 2009). TCEP is used in many types of products, including furniture, in building materials, such as roofing insulation, and as back-coatings for carpets and upholstery (Health Canada, 2009). Polymer products containing TCEP are used in car, railway car and aircraft manufacturing (Health Canada, 2009). Additional uses include fire resistant coatings in epoxy, phenolic and amino resins; and in wood resin composites, such as particleboards, adhesives and lacquers (IARC, 1990; IPCS, 1998; EURAR, 2006; OECD, 2006).

2.3 TCEP Human Exposure

TCEP has been detected in ambient and indoor air, surface and groundwater, food, house dust, condensed on the inside of car windows, acoustical tiles, paints and finishes, foam sealants, and in PU foam containing consumer products. Concentrations of TCEP in various media and a summary of exposure assessments and estimates found in the literature are presented below.

According to ATSDR, food and/or water consumption is likely the main source of exposure to phosphate ester flame retardants (ATSDR, 2012). However, for children, incidental oral exposure (non dietary exposures, such as dust ingestion and mouthing of objects) may be the primary exposure pathway (ATSDR, 2012).

2.3.1 TCEP in Indoor and Ambient Air

Likely sources of phosphate ester flame retardants like TCEP in indoor air include: PVC plasticizers, floor polishes, electronics (plastic cabinets), polyurethane foams, upholstery, furniture, and textiles (ATSDR, 2012, Reemtsma et al., 2008; Canada Gazette, 2011; Marklund et al., 2005). Both particulates and vapors contribute to exposure (Garcia et al., 2007 as cited by ATSDR, 2012).

TCEP particulate and vapor concentrations have been measured in many different types of indoor environments, including child care centers, houses and apartments, offices and other work places, schools, health care facilities, other public buildings, and cars. Only one study was located that reported measurement of ambient air concentrations (Bradman et al., 2012, 2014).

Bradman et al. (2012, 2014) measured TCEP in indoor and outdoor air, as well as dust in 40 childcare facilities in California. TCEP levels in indoor air were significantly higher than those found in the outdoor air. Of the indoor air samples collected, 65% were at levels above the detection limit (0.3 ng/m^3) (Bradman, 2012, 2014).

Several studies analyzed indoor air in a variety of locations in Sweden (Bergh, 2011; Bergh et al., 2011; Marklund et al., 2005; Carlsson et al., 2000, as cited by Destaillats et al., 2007; Staff and Ostman, 2005). Measurements ranged from less than 1 ng/m^3 in several settings (Marklund et al., 2005) to 870 ng/m^3 in office air (Staaf and Ostman, 2005). Marklund et al. (2005) analyzed indoor air samples in homes, a day care center, a hospital ward, a hotel, a prison, a library, shops, and factories. TCEP was one of more frequently detected compounds. Marklund et al. (2005) noted that TCEP air levels were correlated with dust levels found in the same environments. TCEP levels in public buildings were three to four times the concentrations found in residential buildings (Marklund et al., 2005). Staff and Ostman (2005) measured TCEP in indoor air at 24 locations in Sweden where humans may be exposed on a daily basis. These locations were grouped into five categories: private homes, workplaces, stores, health care facilities, and transportation. TCEP was detected in at least one location in each of the five categories. Hartmann et al. (2004) measured air in public buildings and cars in Switzerland. Yang et al. (2014) measured concentrations in office buildings in China.

TCEP was measured in the particulate matter of indoor air of two elementary school classrooms in Austria with concentrations of $PM_{2.5}$ ranging from below the limit of detection to 11,000,000 ng/g (median 522,000 mg/kg (Hutter et al., 2013). PM_{10} concentrations were lower. The authors reported a correlation between TCEP in indoor air samples and impaired cognitive performance in the children. These particulate matter levels are substantially greater than the range of TCEP dust concentrations (600-35,000 ng/g) measured in the same study (Hutter et al., 2013) (Table 2-7). See Table 2-3 for indoor air concentrations.

Table 2-3. TCEP Concentrations in Indoor Air

Country	Location	Media	TCEP Concentrations[1]	Reference	Notes
United States	California child care centers	Indoor air	Mean: 2.69 ng/m^3 Median: 0.91 ng/m^3 Max: 15.34 ng/m^3	Bradman et al., 2012, 2014	PUF cartridges, room conditions recorded during sampling
Sweden	Houses (n=2)	Indoor air	House 1: 0.4 ng/m^3 House 2: 3.0 ng/m^3	Marklund et al., 2005	Stationary sampler with SPE Cartridge

Country	Location	Media	TCEP Concentrations[1]	Reference	Notes
Sweden	Work (daycare, hospital, radio and textile shop, office, plastics factories, laboratory)	Indoor air	Range: 0.7-730 ng/m^3		
	Public places (hotel, prison, university lobby, library, dance hall, furniture shop, bowling alley)	Indoor air	Range: 2-590 ng/m^3		
	Homes, day care centers, work places, apartments	Indoor air	*Median Values* Homes: 4.8 ng/m^3 Daycare centers: 25 ng/m^3 Work places: 10 ng/m^3 Apartments: 3.7 ng/m^3, Range: ND-230 ng/m^3	Bergh, 2011 Bergh et al., 2011	
	Schools, office buildings	Indoor air	Schools:18-250 ng/m^3 Offices: 7.4-11 ng/m^3	Carlsson et al., 2000	
	Private homes, workplaces, stores, health care facilities, transportation	Indoor air	Private homes: 1-115 ng/m^3 Car: 20 ng/m^3 Garage:320 ng/m^3 Office: 6-870 ng/m^3 Workshop: 3-29 ng/m^3 Stores: 11-56 ng/m^3 Health care facility: 9-350 ng/m^3	Staaf and Ostman, 2005b	Stationary sampler with SPE cartridge containing an aminopropyl silica phase (25 mg, 1 ml)

Country	Location	Media	TCEP Concentrations[1]	Reference	Notes
Switzerland	Public buildings Cars	Indoor air	23-56 ng/m^3	Hartmann et al., 2004	
Germany	Daycare centers	Indoor air	Mean: 2.2 ng/m^3 Median: <0.20 ng/m^3 Range: <0.20-33 ng/m^3 95th: 7.5 ng/m^3	Fromme et al., 2014	Gaseous and particulates sampled with glass filter followed by a PUF plug
Austria	Elementary schools; 36 samples,2 classrooms, Spring and fall	Indoor air	*PM_{10}: 35/36* Median: 141,000 ng/g (141 mg/kg) Range: <LOD-4,700,000 ng/g (<LOD-4,700 mg/kg) *$PM_{2.5}$: 35/36* Median: 522,000 ng/g (522.0 mg/kg) Range: <LOD-11,000,000,000 ng/g (<LOD-11,000 mg/kg)	Hutter et al., 2013	Glass filter attached to Digital High Volume sampler for $PM_{2.5}$ and $_{10;}$ Concentrations reported as mg/kg
China	Hangzhou, Offices	Indoor air	Mean: 4.91 ng/m^3 Median: 3.11 ng/m^3 Range: 1.03-13.38 ng/m^3	Yang et al., 2014	Particulate matter filter
Japan	Homes and offices, Tokyo	Indoor air	Homes: 0-136 ng/m^3 Offices: 0-42.1 ng/m^3	Saito et al., 2007	Sampled using quartz fiber Filter (47 mm), first stage and a solid phase extraction disk (empore^{tm} Disk C18, 47mm), second stage.

Country	Location	Media	TCEP Concentrations[1]	Reference	Notes
Japan	Houses, Sapporo	Indoor air	Median: 15.5 ng/m^3 Range: <MDL-297 ng/m^3	Kanazawa et al., 2010	Sampling at 1-1.5 m from floor and 1 m from floor; empore^{tm} Disk C18, 47mm

[1]For ease of comparison, all units are converted to ng equivalent. Original study units, if different, are shown in parentheses. PUF- polyurethane foam, MDL – method detection limit, LOD – limit of detection, SPE – Solid Phase Extraction

The only study in the U.S. measured indoor and outdoor air concentrations at California child care centers. The mean concentration in outdoor air was 0.72 ng/m^3 and the maximum concentration was 1.60 ng/m^3 (Bradman et al., 2012). See Table 2-4.

Table 2-4. TCEP Concentrations in Ambient Air

Country	Location	Media	TCEP Concentrations[1]	Reference	Notes
United States	California, Child care centers	Outdoor air	Mean: 0.72 ng/m^3 Median: 0.19 ng/m^3 Range: <MDL-1.60 ng/m^3	Bradman et al., 2012	MDL = 0.3 ng/m3; PU foam cartridges, collected in outdoor play area

[1]For ease of comparison, all units are converted to ng equivalent. Original study units, if different, are shown in parentheses.
MDL – method detection limit, PU - polyurethane

2.3.1 TCEP Concentrations in Water

Available water concentrations are presented in Table 2-5.

2.3.1.1 Drinking Water

ATSDR (2012) notes that conventional water treatment may not be effective in removing TCEP from drinking water (Meyer and Bester, 2004; Reemtsma et al., 2006; Watts and Linden, 2008; Watts and Linden, 2009, as cited by ATSDR, 2012). A study by Westerhoff et al. (2005) on treatment effectiveness supports the conclusion that water treatment methods may not be effective in TCEP removal. Bennotti et al. (2009) analyzed source water, finished drinking water, and distribution system (tap) water from 19 U.S. water facilities for 51 compounds in 2006 and 2007. TCEP was one of the most frequently detected compounds and was detected in

all three types of water (Bennotti et al., 2009). Similar to studies cited by ATSDR, these data also suggest that conventional water treatment methods are not effective at removing or reducing TCEP levels in drinking water (Bennotti et al., 2009). Stackelberg et al. (2007) evaluated the effectiveness of water treatment methods in removing compounds from drinking water. Only one method tested, granulated activated charcoal filtration, was effective in reducing TCEP concentrations in finished water. Kim et al. (2007) compared several drinking water treatment technologies for the removal of TCEP. Membrane bioreactor (MBR) treatments were the least effective at removing TCEP; reverse osmosis (RO) and nanofiltration (NF) were the most effective and adding ultraviolet (UV) radiation to RO and NF did not increase their effectiveness (Kim et al., 2007) TCEP was detected in 100% of source water and 8% of finished water (Stackelberg et al., 2007). TCEP was detected in 88% of source water samples taken from a large urban treatment plant (Padhye et al., 2014). Removal of TCEP from water was 39% effective for intermediate ozonation treatment and 32% for filtration plus chlorination treatment (Padhye et al., 2014).

TCEP was detected in three out of 22 samples collected from 20 Cape Cod public drinking water wells. The maximum concentration was 20 ng/L (Schaider, 2010). In an earlier study also conducted on Cape Cod, samples were collected from monitoring wells located near a waste water treatment facility, three public water supply wells, a semiprivate well, and four private drinking water wells. Samples were also collected in a standard septic-tank leachfield and a sand recirculating system. TCEP was detected in three of the monitoring wells; however, the concentrations, although reported, were less than the minimum reporting limit of 0.5 µg/L. TCEP was detected in one private water supply well but was below the minimum reporting limit of 0.5 µg/L (Zimmerman, 2004).

USGS sampled nine water supplies using surface water as their source. Both raw and treated samples were analyzed for TCEP. TCEP was detected in 33% of source water samples and 31% of finished water samples (Kingsbury, 2008).

2.3.1.2 Surface and Groundwater

TCEP has been detected in streams across the U.S. (Lee and Rasmussen, 2006; Kolpin et al., 2002). TCEP was detected in 57.6% of the samples (Kolpin et al., 2002). Studies in Wisconsin and Kansas detected TCEP in streams and surface water (Kolpin et al., 2002; Peterman et al., 1980; Lee and Rasmussen, 2006).

The USGS also analyzed groundwater samples from 47 locations in 18 states. TCEP was detected in 29.5% of the samples collected (Barnes et al., 2008). The USGS also collected both groundwater and surface water samples used as drinking water from 25 states and Puerto Rico. Although TCEP was detected in 20.3% of the samples, all levels were below the reporting limit of 0.5 µg/L (Focazio et al., 2008).

TCEP has also been detected in rivers in Japan and South Korea, rainwater collected in Germany, and groundwater in Canada (Andresen et al., 2004; Fukushima et al., 1992; Williams et al., 1981; Fries and Puttmann, 2001 as cited by ATSDR, 2012; Kim et al., 2007).

Table 2-5. TCEP Concentrations in Water

Country	Location	Media	TCEP Concentrations	Reference	Notes
United States	19 Drinking water treatment plants	Source, finished, and distribution water	*Median values* Source: 120 ng/L Finished: 120 ng/L Distribution:150 ng/L *Max values* Source: 530 ng/L Finished: 470 ng/L Distribution:200 ng/L	Benotti et al., 2009	
	Cape Cod public wells	Water	Max: 20 ng/L	Schaider et al., 2010	
	Cape cod	Monitoring wells; drinking water	Monitoring wells: 81-240[a] ng/L Private well: 110[a] ng/L	Zimmerman, 2004	
	Drinking water supplies	Surface water: raw and finished	Max Source: 260 ng/L (estimated) Max Finished 220 ng/L (estimated)	Kingsbury et al., 2008	
	Kansas	Streams	Avg: 500 ng/L	Lee and Rasmussen, 2006	
	Multiple locations	Streams	Max: 540 ng/L	Kolpin et al., 2002	
	Multiple locations	Groundwater	Max: 737 ng/L	Barnes et al., 2008	Untreated drinking water sources
	Drinking water supplies	Groundwater and surface water	<500[b] ng/L	Focazio et al., 2008	Untreated drinking water sources
	Drinking water treatment plants	Drinking water	*Max values* Source:120 ng/L Finished: 50 ng/L	Stackelberg et al., 2007	

Country	Location	Media	TCEP Concentrations	Reference	Notes
United States	Drinking water treatment plant	Surface water and finished drinking water	*Median values* Source: 5.6 ng/L Finished: 3.7 ng/L *Range values* Source: 0-51.7 ng/L Finished: 0-20.4 ng/L	Padhye et al., 2014	Large urban treatment plant in southeast United States.
Germany	Oder River	Municipal waste water influent and effluent, river water, groundwater	*Mean* Effluent:352 ng/L Influent: 986 ng/L *Range* River: ND-1,036 ng/L Ground: ND-312 ng/L	Fries and Puttmann, 2003 as cited by ATSDR, 2012	ND = 1 ng/L
	N/A	River water untreated and finished	Untreated:10-130 ng/L Finished:0.3-30 ng/L	Andresen and Bester, 2006	
Spain	Northwest area	Surface water	Median: 5 ng/L	Rodil et al., 2012	
Italy	N/A	Volcanic lakes	Mean monthly range: ND-64 ng/L	Bacaloni et al., 2008	Detection limit not reported
South Korea	Rivers and lakes	Surface water	Mean: 42 ng/L Range: 14-81 ng/L	Kim et al., 2007	
		Waste water treatment	Mean Influent: 284 ng/L Effluent Means: MBR method: 283-303 ng/L RO method: 14 ng/L NF method: 13 ng/L		MBR system was not effective for TCEP. Adding UV radiation to the RO and NF method did not increase effectiveness.

[1]For ease of comparison, all units are converted to ng equivalent. Original study units, if different, are shown in parentheses. [a]Reported levels were below the minimum reporting limit (0.5 µg/L or 500 ng/L).[b]All samples were detected below the reporting limit. ND = non-detect; N/A = not applicable; MBR – membrane bioreactor; UV – ultraviolet; RO – reverse osmosis membrane filtration method; NF – nanofiltration membrane filtration method, max – maximum, Avg – average

2.3.2 TCEP Concentrations in Food

Results from the U.S. Food and Drug Administration's (U.S. FDA) Total Dietary Study from 1991-2003 and 2004-2005 are presented in Table 2-5. The source of TCEP in food has not been determined; however, Daft (1982, as cited by ATSDR, 2012) suggested that the wrapping material used in food packaging may be the source.

Another potential source of TCEP in food may be from plant uptake. Two recent studies evaluated TCEP uptake in plants (Trapp and Eggen, 2013; Eggen et al., 2013). TCEP was added to the soil in known concentrations and both roots and leaves of plants were analyzed for TCEP. TCEP was found to accumulate in the leaves of plants (Trapp and Eggen, 2013; Eggen et al., 2013). This finding may be relevant where biosolids are applied to agricultural land that is used for food crops (Trapp and Eggen, 2013; Eggen et al., 2013). See Table 2-6.

Table 2-6. TCEP Concentrations in Food

Country	Study Type	Food	TCEP Concentrations[1]	Reference	Notes
United States	Total Diet Study Market Baskets 1991-1993, 2003-2004	Peas, green, frozen, boiled	1.82 ng/g[a]	U.S. FDA, 2006 as cited by ATSDR 2009	234 food items were evaluated over a 10-year period between 1982 and 1991.
		Oatmeal, plain, cooked	0.02 ng/g[b]		
		Cream of wheat (farina), enriched, cooked	2.59 ng/g[a]		
		Rolls, white, soft, enriched	0.08 ng/g[b]		
		Broccoli, fresh/frozen, boiled	0.14 ng/g[a]		
		Green beans, fresh/frozen, boiled	1.59 ng/g[a]		
		BF turkey and rice	0.48 ng/g[a]		
		BF peas	0.02 ng/g[b]		
		Bread, cracked wheat	0.02 ng/g[b]		
		Eggplant, fresh, peeled, boiled	1.75 ng/g[a]		
		Candy, hard, any flavor	0.02 ng/g[b]		
		Sweet cucumber pickles	0.05 ng/g[b]		

Country	Study Type	Food	TCEP Concentrations[1]	Reference	Notes
		BF teething biscuits	0.06 ng/g[b]		
		Soup, Oriental noodles (ramen noodles), prepared with water	7.25 ng/g[a]		
		BF pears, and pineapple	0.02 ng/g[b]		
United States	Total Diet Study Market Baskets 2004-2005	BF custard/pudding	28 ng/g[a]	U.S. FDA, 2006 as cited by ATSDR, 2009	
		BF, juice, apple-banana	1.05 ng/g[a]		
		BF, juice, apple-cherry	4.63 ng/g[a]		
		BF, oatmeal w/fruit	2.37 ng/g[a]		
		BF, veg w/turkey	0.88 ng/g[a]		

[1]For ease of comparison, all units are converted to ng equivalent. Original study units, if different, are shown in parentheses.
[a]Only one sample >=LQ; [b]Trace amounts only; BF – baby food

2.3.1 TCEP Concentrations in Dust

Given TCEP's properties and the quantity of data found on dust, it would appear that the dominant mechanism for human exposure is diffusion of TCEP out of materials to the surface where it partitions into dust, which can then be ingested. Studies have measured TCEP in dust collected from homes in the U.S., and in homes, day care centers, motels and hotels, cars, schools, shops and other work environments, and public places in many countries. Concentrations measured ranged vary widely, from less than 1 to the highest level of 330,000 ng/g reported by Haumann and Thumulla (2002) in 1569 samples from various types of buildings in Germany.

In California, two rounds of sampling were conducted in 16 homes; the first round in 2006 and the second in 2011. Maximum concentrations of TCEP in house dust exceeded 0.01% (100 ng/mg). In 2006, TCEP concentrations ranged from 610-160,000 ng/g and in 2011 TCEP concentrations ranged from 330-110,000 ng/g. After a new roof was installed on one home, TCEP levels in house dust increased 20-fold (Dodson et al., 2012).

Fang et al. (2013) collected dust samples from 20 homes and cars in Boston and found a range of less than 20 to 1350 ng/g in house dust and less than 20 to 50,120 ng/g in car dust. The study authors purchased a V6 commercial standard to develop the analytical methodology. While V6, another flame retardant, was the focus of this study, TCEP was found in the commercial standard

as an impurity at a concentration of 14% by weight. Both V6 and TCEP were found in the dust samples and were highly correlated. TCEP was detected in 48% of house dust samples and 95% of the car dust samples. The authors suggest that use of V6 may be an important source of TCEP. The authors also suggested that, since TCEP has a higher vapor pressure than V6, TCEP may result in higher dust concentrations and greater migration from consumer products with respect to V6 (Fang et al., 2013). In another Boston study, Stapleton et al. (2014) found correlations between house dust and hand wipe samples in a study of children in Boston. They measured concentrations ranging from 20-6,920 ng/g in dust collected with vacuum cleaners and concentrations ranging from 24-197 ng/g in dust from hand wipe samples.

In Canada, Fan et al. (2014) measured dust in 134 urban homes. Concentrations from samples collected using a Pullman Holt vacuum sampler by a trained technician ranged up to 33,000 ng/g (median 800 ng/m). Samples from household vacuum cleaner collection ranged up to 7000 ng/g (median 600 ng/g) (Fan et al., 2014).

Contact with house dust by adults and children may lead to incidental oral ingestion of TCEP by hand to mouth activities. Young children typically engage in more hand-to-mouth activities than adults, which increases their exposure to TCEP in dust.

See Table 2-7 below for data on dust concentrations in the U.S. and other countries

Table 2-7. TCEP Concentrations in Dust

Country	Location	Media	TCEP Concentrations		Reference	Notes
United States	California homes	Dust	2006 sampling Median: 5100 ng/g Range: 610-160,000 ng/g	2011 sampling Median: 2700 ng/g Range: 330-110,000 ng/g	Dodson et al., 2012	2006 collection, vacuum cleaner with cellulose extraction thimble. 2011 collection, vacuum cleaner with cellulose extraction thimble
	Boston MA 20 homes and cars	Dust	*Medians* House: 50.2 ng/g Car: 1080.0 ng/g	*Ranges* House: <20-1350 ng/g Car: <20-50120 ng/g	Fang et al., 2013	
	Boston, MA, house	Dust	Geo mean: N/A Range: 24-197 ng/g		Stapleton et al., 2014	Hand wipe samples

Country	Location	Media	TCEP Concentrations		Reference	Notes
		Dust	Geo mean: 348 ng/g Range: 20-6,920 ng/g			Household vacuum cleaner bag collection
Canada	134 Urban homes	Dust	Median: 800 ng/g (0.8 µg/g) Max: 33,000 ng/g (33 µg/g) 95th: 4,400 ng/g (4.4 µg/g)		Fan et al., 2014	Fresh/active dust collected using a Pullman Holt vacuum sampler by a trained technician
Canada	House	Dust	Median: 600 ng/g (0.6 µg/g) Max: 7,000 ng/g (7 µg/g) 95th: 3,700 ng/g (3.7 µg/g)			Household vacuum cleaner collection
Philippines	Malate and Payatas, House	Dust	*Malate* Median: 34 ng/g Range: <0.44-1200 ng/g	*Payatas* Median: 16 ng/g Range: <0.44-140 ng/g	Kim et al., 2012	
Japan	182 single family homes	Floor, Multi surface dust	Floor Median:5830 ng/g Range: <MDL-338,450 ng/g	Multi surface dust Median: 8260 ng/g Range: <MDL-2,320,000 ng/g	Araki et al., 2013	Hand held vacuum cleaner
Germany	Homes, schools commercial buildings	Dust	Median: 600 ng/g (0.6 mg/kg) 95th: 8,400 ng/g (8.4 mg/kg) Max: 330,000 ng/g (330 mg/kg)		Haumann and Thumulla, 2002	1569 Samples, 90% of samples above limit of determination; vacuum cleaner collection

Country	Location	Media	TCEP Concentrations		Reference	Notes
	Daycare centers	Dust	Mean: 1,350 ng/g (1.35 mg/kg) Median: 400 ng/g (0.40 mg/kg) Range: 100-8,300 ng/g (0.1-8.3 mg/kg) 95th: 4,900 ng/g (4.9 mg/kg)		Fromme et al., 2014	ALK dust filter mounted on a sampler connected to a vacuum cleaner, vacuumed for 5-10 minutes
Germany	Pooled homogen-ized sample from 20 buildings, mostly residences	Dust	Arith Mean: 2,230-3,750 ng/g (2.23-3.75 mg/kg) Geo Mean: 640-890 ng/g (0.64-0.89 mg/kg) Max: 64,000-121,000ng/g (64-121 mg/kg)		Ingerowski et al., 2001	Conventional vacuum with filter from clients with health problems; results from 3 laboratories
New Zealand	House, floor and mattress	Dust	Medians Floor:110 ng/g Mattress:10 ng/g		Ali et al., 2012	
Netherlands	House, hotel, motel	Dust	Mean: 792 ng/g		Brandsma et al., 2013	Composite reference dust from NIST (SRM2585), collected from vacuum cleaner bags
	House	Dust	Around electronics Median: 1300 ng/g Range: 220-6900 ng/g	On electronics Median: 880 ng/g Range: 520-2200 ng/g	Brandsma et al., 2014	Collected using dustream™ dust collector
	Car	Dust	Dashboard Median: 2800 ng/g Range: 1100-5700 ng/g	Seats Median: 600 ng/g Range: 240-5600 ng/g		
Sweden	Houses	Dust	House 1: 0.27 ng/g House 2: 0.19 ng/g		Marklund et al., 2003	Vacuum cleaner bag collection, 2 houses

Country	Location	Media	TCEP Concentrations		Reference	Notes
	Work (daycare, hospital, radio and textile shop, office)	Dust	0.37-48 ng/g			Vacuum cleaner bag collection, except textile shop and hospital samples were handpicked
Sweden	Public Places (hotel, prison, university lobby, aircraft, library, cinema, dance hall)	Dust	0.85-94 ng/g			Vacuum cleaner bag collection
	Computer screen and cover	Dust	Screen: 220 ng/m^2	Cover: 210 ng/m^2		Wipe test samples; location of computer not reported
Belgium, Flemish region	House	Dust	Mean: 490 ng/g (0.49 µg/g) Median: 230 ng/g (0.23 µg/g)	Range: 80-2,650 ng/g (<0.08-2.65 µg/g)	Van den Eede et al., 2011	Vacuum dust samples
	Carpenter workshop, second-hand store, electronics stores, laboratory	Dust	Mean: 1,170 ng/g (1.17 µg/g) Median: 590 ng/g (0.59 µg/g)	Range: 80-5,460 ng/g (<0.08-5.46 µg/g)		
Belgium	House	Dust	Range: 75-1310 ng/g		Van den Eede et al., 2012	Vacuum dust samples
Romania	House	Dust	Range: 40-1450 ng/g		Van den Eede et al., 2012	Vacuum dust samples

Country	Location	Media	TCEP Concentrations		Reference	Notes
Spain	House	Dust	82 ng/g		Van den Eede et al., 2012	Vacuum dust samples
	House	Dust	Mean: 757 ng/g (0.757 µg/g)		Cristale and Lacorte, 2013	Vacuum cleaner bag collection
Austria	Elementary schools	Dust	Median: 2,500 ng/g (2.5 µg/m^3)	Range: 600-35,000 ng/g (<0.6-35 µg/m^3)	Hutter et al., 2013	Sampled classrooms in spring and fall; used industrial vacuum cleaner collection
Egypt, Assiut	Car	Dust	Avg: 198 ng/g Median: 127 ng/g	Max: 572 ng/g	Abdallah and Covaci, 2014	Collected with a dust buster vacuum
	House	Dust	Avg: 49 Median: 22	Max: 132 ng/g		
	Office	Dust	Avg: 61 ng/g Median: 31 ng/g	Max: 125 ng/g		
	Micro-environment (coffee shops, restaurants, supermarkets)	Dust	Avg: 277 ng/g Median: 234 ng/g	Max: 538 ng/g		
Not specified	House	Dust	Mean: 820 ng/g (0.82 µg/g)		Ionas and Covaci, 2013 as cited in Fan et al., 2014	
Not specified	House	Dust	Mean: 820 ng/g (0.82 µg/g)		Murray et al., 2013 as cited in Fan et al., 2014	

[1]For ease of comparison, all units are converted to ng equivalent. Original study units, if different, are shown in parentheses.
NIST- National Institute of Standards and Technology, geo mean – geometric mean; N/A – not available; avg– average; max – maximum, min- minimum, MDL – method detection limit

2.3.1 TCEP in Consumer Products

Unlike concentrations in environmental media (e.g., air, water, dust), TCEP levels in consumer products cannot be used as a proxy for concentration levels to which consumers are exposed. To develop realistic consumer exposure concentrations, TCEP levels in consumer products would need to be paired with experimental or monitoring results that reflect the availability of TCEP compound to leave these products and enter the body. In the absence of such data, this review is limited to a discussion of levels measured in consumer products. Table 2-8 presents data on TCEP in various consumer products, including furniture, baby products toys, mattresses, foam sealants, paints, and acoustical panels. Any product that contains TCEP has the potential to contribute to household dust levels. See Table 2-8.

2.3.1.1 TCEP in Children and Baby Products

Stapleton et al. (2011) analyzed 101 polyurethane foam samples collected from baby products. TCEP was detected at concentrations greater than 1 mg/g of foam in a car seat, one changing table pad, one sleep positioner, one portable mattress, 10 nursing pillows, one baby carrier, and two infant bath mats/slings. Of note is that V6 was detected along with TCEP in 15 of the 16 samples with a mean concentration of 5.91 mg/g. The authors suggested that the results indicate that the products may have been treated with V6 and, since TCEP is an impurity of V6, its presence may be from the use of V6. TCEP was detected in lower levels than other flame retardants measured (Stapleton et al., 2011).

The Washington Toxics Coalition and Safer States purchased 20 baby products in 2011. The purchased products were tested for the presence of flame retardants. TCEP was detected in one product, a co-sleeper at 2.99 mg/g. TCEP was below the detection limit (0.04 mg/g) in all other products (Schreder, 2012).

Fang et al. (2013) reanalyzed 12 foam samples from baby products collected in a previous study where V6 was identified but not quantified. Both V6 and TCEP were detected in the 12 foam samples. The reported concentrations in foam (1.1 – 5.9 mg/g) are consistent with reported application rates of 5.3 weight % for V6 in automobile foam (Fang et al., 2013).

2.3.1.2 TCEP Concentrations in Furniture

ATSDR (2012) briefly summarized data on TCEP in products. TCEP was detected in polyurethane foam samples in concentrations ranging from 0.8-3.1 µg/g (Nagase et al., 2003 as cited by ATSDR, 2012). Stapleton et al. (2012) collected 102 foam samples from couches purchased between 1985 and 2012 in the U.S. TCEP was detected in one sample (5.47 mg/g) along with V6. According to the authors, the Material Safety Data Sheet (MSDS) for Antiblaze,

a product containing V6, indicates that TCEP is present at 10% by weight. As such, V6 may be the source of TCEP in the sample (Stapleton et al., 2012).

There is a dearth of data from the U.S. on emission rates from consumer products. The European Union Risk Assessment Report (EU RAR) for TCEP (2009) states that migration from various consumer products is generally unknown (ECHA, 2009). According to an unpublished study presented in the EU RAR, 217 ng/cm^2/hr of TCEP may be released from upholstered furniture originally containing 8 mg/cm^2 of this flame retardant (Bruckert and Schoene, 1990 as cited by ECHA, 2009)

2.3.1.3 TCEP Concentrations in Electronic Products

McKone et al. (2009) tested TCEP emissions from computers in test chambers. With a computer running for seven days, the air concentration of TCEP in the test chamber was over 20 ng/m^3. McKone also measured the emission rate of TCEP from five computers. The emission rates ranged from 50 ng/hr/unit to over 200 ng/hr/unit (McKone et al., 2009).

Several studies have measured TCEP in televisions. TCEP migration rate from the surface area of plastic housings of television sets was measured at 13 µg/m^2-hr at ambient air temperatures (Saito et al., 2007 as cited by ATSDR, 2012).

Wensing et al. (2005) also measured emission rates from televisions sets in a 1 m^3 test chamber with an air exchange rate of 0.5/hr over the course of 550 hours. The concentration of TCEP in the chamber rose steeply over the first 100 hours of testing, then rose at a more gradual rate (Figure 6 in Wensing et al., 2005). TCEP concentrations from computer monitors were measured in a 1 m^3 test chamber with an air exchange rate of 0.5/hr on different days (Wensing et al., 2005). TCEP levels ranged from 10 to 121 ng/m^3 over the course of 3 to 14 days (Wensing et al., 2005).

Wensing (1999, as cited in Malmgren-Hansen et al., 2003) conducted a study on chemicals found in electrical and electronic products. While new television sets did not emit TCEP above 0.01 µg/set-hr, aged TV sets emitted TCEP at levels ranging from <0.01 to 0.30 µg/set-hr. TCEP reached 75% of equilibrium after 100 hours (Wensing, 1999 as cited in Malmgren-Hansen et al., 2003). The data strongly indicate that TCEP can migrate to the surface where it is subsequently released to the air or available for dermal transfer. TCEP migrates to the outer surface of plastic material via diffusion, a process known as "blooming." However, the rate of migration is not known (SCHER, 2012). TCEP is a non-volatile compound and, therefore, is unlikely to be present in a gaseous state. Release of TCEP from consumer products likely occurs via abrasion, which contributes to TCEP levels in dust (ECHA, 2009).

Marklund et al. (2003) using wipe tests, measured TCEP levels of 220 ng/m^2 and 210 ng/m^2, in dust on a computer screen and cover, respectively (see Table 2.7).

2.3.1.4 TCEP Concentrations in Non-U.S. Products

In 2009, Health Canada tested the following products for TCEP: 14 sofas, 4 mattresses, 10 children's products, 4 acoustical panels, and a seat from a car. TCEP was detected in four sofas, a car seat, and two children's products. The only reported TCEP levels were for the children's products (13,000 – 21,000 ng/g). In 2010, additional testing on 30 children's products was conducted for Health Canada's Priority Substances List evaluation. Again, TCEP was detected in a polyurethane foam book (3,800 ng/g) and a sleep positioner (34 ng/g). Three other products contained TCEP levels below the quantitation limit (Canada Gazette, 2011).

In Belgium, 64 toys were sampled and analyzed for flame retardants. TCEP was detected in 13% of the samples (Ionas et al., 2012). A Danish study (Borling et al., 2006) tested the foam matrix of various children's products (a sword, 2 floor puzzles, a swim board, a mask, a ball, a book, and an activity carpet) for the presence of TCEP. The content of TCEP was found above the detection limit of 0.1% by content; no other values were reported in the study (Borling et al., 2006. Another Danish study (Tonnig et al., 2008) measured the TCEP content of various baby products. All the measured amounts were below 1000 ng/g (1 µg/g) (Tonnig et al., 2008). In Germany, Haumann and Thumulla (2002) report concentrations of TCEP in various products; the highest concentration of $8.9x10^8$ ng/g was measured in mattresses.

Kajiwara et al. (2011) analyzed consumer electronic components and building materials and found low concentrations of TCEP (below detection to < 120 ng/g) relative to what has been measured in PU foam.

Table 2-8. TCEP Concentrations in Consumer Products

Country	Location or item	Media	TCEP Concentrations[1]	Reference	Notes
United States	Couch	Foam	5,470,000 ng/g (5.47 mg/g)	Stapleton et al., 2012	102 Foam samples collected all over U.S. from couches purchased between 1985 and 2010. TCEP detected in 1.

Country	Location or item	Media	TCEP Concentrations[1]	Reference	Notes
United States	1 Car seat, 1 changing table pad, 1 sleep positioner, 1 portable mattress, 10 nursing pillows, 1 baby carrier, 2 infant bath mats/slings	Foam	Mean: 5,910,000 ng/g (5.91 mg/g) Range: 1,080,000-5,940,000 ng/g (1.08-5.94 mg/g)	Stapleton et al., 2011	PUF samples from 101 baby products. TCEP > 1,000,000 ng/g in 17.
	Co-sleeper	Foam	2,990,000 ng/g (2.99 mg/g)	Schreder, 2012	20 baby products several states in 2011, all but 1 co-sleeper below DL of 40,000 ng/g
	Boston, MA Baby products	Foam	1,100,000 -5,900,000 ng/g	Fang et al., 2013	12 foam samples
Canada	PUF books and sleep positioners	Foam	2009 PUF book: 13,000 ng/g Sleep positioner: 21,000 ng/g 2010 PUF book 3800 ng/g Sleep positioner 34 ng/g	Canada Gazette, 2011	2009: sofas, mattresses, acoustical panels, seat from car; but only children's products reported
Belgium	64 toys	Foam	75th percentile: 70 ng/g Median: < LOQ	Ionas et al., 2012	

Country	Location or item	Media	TCEP Concentrations[1]	Reference	Notes
Germany	Various products	Foams, paints, mattresses, sealants	Soft foams: 6,400,000,000 ng/g (6400 mg/kg) Paints/finishes: 840,000,000 ng/g (840 mg/kg) Mattresses: 890,000,000 ng/g (890 mg/kg) 89,000,000,000 ng/g Foam sealants: (89,000 mg/kg)	Haumann and Thumulla, 2002	
Japan	Laptop computer	Chassis	16 ng/g	Kajiwara et al., 2011	
		Keyboard top	<4 ng/g		
		PC boards	14 ng/g		
		Cooling fan and speakers	120 ng/g		
		AC adapter	<4 ng/g		
		LCD panel	<4 ng/g		
	LCD TVs; purchased new in Japan in 2008	Rear cover	7.0 ng/g TV1 <4 ng/g TV2	Kajiwara et al., 2011	Two TVs sampled
		Front cover	4.0 ng/g TV1 <4 ng/g TV2		
		Power board	<4 ng/g TV1		
		PC board for power and fluorescent	4.0 ng/g TV1 5.5 ng/g TV2		
		Other PC boards	9.0 ng/g TV1 7.0 ng/g TV2		
		LCD panel	<4 ng/g TV1 <4 ng/g TV2		
	Other products: purchased new in Japan in 2008	Curtains	4.0 ng/g 6.0 ng/g	Kajiwara et al., 2011	Two samples
		Electrical outlets	4.0 ng/g <8 ng/g		Two samples
		Insulation boards	<9.0 ng/g 10 ng/g		Two samples
		Wallpaper	<2-<20 ng/g		Four samples

Country	Location or item	Media	TCEP Concentrations[1]	Reference	Notes
Denmark	Electrical and electronic products	10 New TVs	10 ng/set-hr (0.01 µg/set-hr)	Wensing, 1999 as cited in Malmgren-Hansen et al., 2003	75% of equilibrium after 100 hours
		10 Old TVs	10-300 ng/set-hr (<0.01 to 0.30 µg/set-hr)		
	Children's products	1 Sword, 2 floor puzzles, 1swim board, 1 mask, 1 ball, 1 book, 1 activity carpet	>0.1% by content (specific concentrations not reported)	Borling et al., 2006	Tested foam matrix
	Baby Products	3 Foam wash cloths, 2 feeding pillows, 2 covered mattresses, 2 nursing pillows, 2 baby carriers, 2 perambulator aprons	All: <1,000 ng/g (<1 µg/g)	Tonnig et al., 2008	Detection limit = 1,000 ng/g (<1 µg/g)
Not Specified	Not specified	Poly-urethane foam	800-3,100 ng/g (0.8-3.1 µg/g)	Nagase et al., 2003, as cited by ATSDR, 2012	

[1]For ease of comparison, all units are converted to ng equivalent. Original study units, if different, are shown in parentheses. LOQ – limit of quantification, AC – alternating current, LCD – liquid crystal display, PC – personal computer

2.4 TCEP ADME and Biomonitoring Studies

No *in vivo* human data for absorption, distribution, metabolism, or elimination of TCEP by any route of exposure were located, although there are some limited *in vitro* data on metabolism in liver slices or via microsomes.

2.4.1 TCEP Absorption

Oral studies in rats and mice indicate that TCEP is well absorbed orally and rapidly quantifiable in blood and plasma (Burka et al., 1991; Herr et al., 1991). Studies with ^{14}C-labeled TCEP showed >90% absorption based on quantification of radioactivity in urine, feces, and expired air (Burka et al., 1991; Herr et al., 1991). Maximum concentrations of TCEP have been observed in the plasma within 15 minutes of gavage administration, with the maximum plasma concentration achieved by 2-hours post dose (Herr et al., 1991). Absolute bioavailability via the oral route has not been evaluated.

While no animal studies were identified that directly assessed toxicokinetics following inhalation exposure for TCEP, indirect evidence of absorption is provided by several medial lethal concentrations (LC_{50}) and longer-term studies showing adverse systemic effects (Stauffer Chemical Company, 1974, 1979 as cited in IUCLID, 2000; Smyth et al., 1951 as cited in IUCLID, 2000; ATSDR, 2012; Shepel'skaia and Dyschinegvich, 1981 as cited in ECHA, 2009). TCEP is also well absorbed by inhalation. A number of studies reported systemic toxicity following inhalation exposure of rats to TCEP; however, no information is available on the extent or rate of absorption. Systemic toxicity, including lethargy, depression and decreased body weight were reported in one 4-hour inhalation study conducted in rats (Stauffer Chemical Company, 1974, 1979 as cited in IUCLID, 2000). In a 4 month inhalation study, male rats showed histopathological changes in the testes (NTP, 1991; Shepel'skaia and Dyschinegvich, 1981 as cited in ECHA, 2009). Taken together these studies provide indirect evidence as to the absorption of TCEP via the inhalation route. Absolute bioavailability associated with inhalation exposure has not been established and systemic concentrations associated with toxicological effects (AUC- Area Under the Curve and/or $Concentration_{max}$) were not reported.

No quantitative data are available on the extent or rate of dermal absorption in any species. Some information about dermal absorption can be inferred, however, from dermal toxicity studies, including those discussed in section 5.7 (U.S. EPA, 1989; section 5.7). There are limited data on other alkyl phosphate flame retardants to provide support for dermal absorption of TCEP (ATSDR, 2012). An unpublished study submitted under the Toxic Substances Control Act (TSCA) rule reported a median lethal dose (LD_{50}) greater than 200 mg/kg but less than 5000 mg/kg in albino New Zealand rabbits (U.S. EPA, 1989). The sex, dose, number of animals, and lethality incidence were not reported. However, the observation of some death(s) at the tested dose(s) indicates that toxicity (and death) was observed, and, therefore, that TCEP was dermally absorbed in rabbits. More definitive data on absorption in rabbits comes from a primary skin irritation study, where the applied doses were not reported, but sufficient TCEP was absorbed to cause narcosis and paralysis in 4/6 of the treated rabbits (U.S. EPA, 1989). Overall, the data in rabbits indicate that TCEP is systemically available following dermal application.

2.4.2 TCEP Distribution

Distribution studies via oral (Minegishi et al., 1988; Chadwick et al., 1989 as cited in ECHA, 2009; Herr et al., 1991) and intravenous routes (Dix et al., 1994) show wide and rapid distribution throughout the body. In rodents, enterohepatic circulation was identified.

Following oral administration of ^{14}C-TCEP to rats, radioactivity was reported in all organs measured, indicating wide distribution of TCEP or its metabolites (Chadwick et al., 1989 as cited in ECHA, 2009). Low tissue/blood ratios of radioactivity for brain, heart, muscle, and testes were identified indicating poor distribution to these tissues. Peak radioactivity was reported in the liver and kidneys at 6 hours. At 168 hours post-dosing, the highest remaining ^{14}C level was found in the liver suggesting TCEP and/or its metabolites undergo enterohepatic circulation (Minegishi et al., 1988). Herr et al. (1991) compared blood and brain radioactivity in male and female rats at 2 hours (peak seizure time point) and 24 hours following a single oral gavage administration at doses of 0 (corn oil vehicle), 175, 350, or 700 mg/kg TCEP. At 2 hours post-dosing, the concentration of ^{14}C in blood significantly increased with dose and differed by sex. Males had somewhat higher blood levels of radioactivity than females at 2 hours post administration of the single dose. Radioactivity was detected in the blood and brain after 24 hours in both dosing regimens. The radiolabel was distributed to all regions of the brain, and there were no dose-related differences in brain TCEP levels at 24 hours after treatment, although female rats had higher parent TCEP/metabolite ratios in brain cortical tissue compared to males. In the repeated dosing scenario in which females rats received 14 consecutive doses of 175 mg/kg, levels of ^{14}C were evenly distributed throughout all brain regions evaluated. The similarity of the blood/brain ratios, independent of dose in males and females, suggests that TCEP does not accumulate in the brain.

Distribution of TCEP in rats was compared by Dix et al. (1994) in male and female F344 rats administered a single intravenous dose of 20 mg/kg TCEP. Values for free TCEP area under the curve (AUC) and other pharmacokinetic parameters were not statistically different between males and females using the conventional methods. The authors also noted that, at high TCEP plasma concentrations, binding sites are saturated leading to a higher unbound TCEP fraction in blood. The free-fraction of TCEP was about 0.4 to 0.5 (depending on the method) at 5-10 mg/L, and ~0.56-0.58 at 220 to 400 mg total TCEP/L plasma.

2.4.3 TCEP Metabolism

Very limited information about the metabolism of TCEP is available (Van den Eede et al., 2013). *In vivo* (oral administration) and *in vitro* studies show that metabolism of TCEP involves phase I and phase II pathways (Herr et al., 1991; Burka et al., 1991). Phase I metabolism occurs via both an oxidative pathway, likely via a cytochrome P450, and via a hydrolytic pathway via a

beta-esterase. Some products of the oxidative pathway undergo glucuronidation, a phase II process. Burka et al. (1991) evaluated the presence or TCEP metabolites in urine and feces of male B6C3F1 mice and male and female F344 rats. They showed that TCEP undergoes extensive metabolism and is excreted primarily in the form of metabolites. Metabolism was not induced or inhibited by nine consecutive daily doses. Qualitative evidence indicates that the metabolic pathways in rats and mice are similar, although quantitative differences in the amounts of different metabolites were observed. A proposed metabolic scheme was presented to account for identified metabolites based on female rat metabolism and confirmed in male rats and mice. TCEP can be metabolized via a hydrolytic pathway or via an oxidative pathway, and some products of the oxidation pathway undergo glucuronidation (a phase II process) prior to elimination in urine. The enzymes responsible for the oxidative pathway and hydrolytic pathways were considered to be a cytochrome P450 and a beta-esterase, respectively. One metabolite [bis(2-chloroethyl) hydrogen phosphate - BCHP] can be produced either by direct hydrolysis of TCEP or by oxidation followed by hydrolysis. Bis(2-chloroethyl) carboxymethyl phosphate, bis(2-chloroethyl) 2-hydroxyethyl phosphate glucuronide, and the bis(2-chloroethyl) phosphate (BCEP) diester were reported as urinary metabolites of TCEP in mice (Van den Eede, et al., 2013).

Burka et al. (1991) used enzyme inhibitors to determine the relationship between acute neurotoxicity (as manifested by clinical signs of “wet dog shakes”) and metabolism, and to identify the toxic form(s). Pretreatment of male and female rats with inhibitors of mixed function oxidases and aldehyde dehydrogenase altered the metabolic profile of TCEP by *increasing* the hydrolysis product (BCHP) compared to the oxidative product [bis(2-chloroethyl) carboxymethyl phosphate – BCCP]. Clinical signs of toxicity were substantially increased by the two aldehyde dehydrogenase inhibitors. The authors suggested that the increased toxicity was related to increased levels of a reactive metabolite, possibly the aldehyde. Inhibition of the mixed function oxidase pathway did not increase toxicity, but did slow the elimination of radioactivity in the urine and inhibited production of BCCP. The results were interpreted as suggesting that a metabolite, rather than the parent TCEP, is responsible for the acute neurotoxicity. However, the authors also noted that inhibition of the oxidative pathway lead to increased metabolism via the hydrolysis pathway. A definitive assignment of toxicity to the metabolite is precluded by the lack of data on parent compound TCEP plasma levels in animals administered metabolism inhibitors.

The involvement of a metabolite as the toxic form appears contrary to the results from the same laboratory published by Herr et al. (1991). In these studies, radio-labeled TCEP was orally administered to F344 rats and radioactivity form isolated form liver and brain tissues was primarily parent compound (Herr et al., 1991). The implication is that that most of the radioactivity in the brain cortex at the time of seizures was in the form of the parent (therefore implicating the parent compound TCEP as the toxic agent) ATSDR (2012).

In vitro data provide some information on the human metabolites of TCEP. Although the data are less complete than the rodent data, the available data are consistent with at least a portion of the human metabolic pathway and possibly the entire pathway being the same as in rats. Human liver slices (which contain cytosolic enzymes) and microsomes (only enzymes from the rough endoplasmic reticulum) metabolize TCEP to BCHP, 2-chloroethanol (formed by the hydrolysis of TCEP to BCHP), and three unknown metabolites (Chapman et al., 1991 as cited in ATSDR, 2012). The same spectrum of metabolites was seen with male rat liver slices and microsomes. Female liver slices but not microsomes also metabolized TCEP, and sex-specific differences were identified, as male liver slices metabolized TCEP 1.7 times faster than females. TCEP hydrolysis was localized primarily in the liver cytosol, but the difference between the metabolic profile described by Chapman et al. (1991 as cited in ATSDR, 2012) and that described by Burka et al. (1991) indicates that *in vivo* oxidation may occur extrahepatically. ATSDR (2012) further indicated that cytochrome P-450 was responsible for approximately 38% of the microsomal hydrolytic activity, while the majority of activity was associated with beta-esterase (Chapman et al., 1991 as cited in ATSDR, 2012). The presence of beta-esterase in rat serum but not in human serum is consistent with the observation by Chapman et al. (1991 as cited in ATSDR, 2012) that TCEP was metabolized by rat plasma but not human plasma or whole blood.

2.4.4 TCEP Excretion

Elimination of TCEP and/or its metabolites from blood was biphasic (Chadwick et al., 1989 as cited in ECHA, 2009; Minegishi et al., 1988). The maximum average concentration in tissues occurred by six hours post exposure, with adipose tissue having the longest tissue elimination half-life of 87 hours; no accumulation was expected (Minegishi et al., 1988). No indication of long-term sex differences in clearance from the brain were identified (Herr et al., 1991).

Urine is the main route of excretion for TCEP in rodent studies following oral and i.v. administration, with minimal excretion in exhaled air and feces. Following a single gavage dose of ^{14}C-TCEP, more than 75% of the radiolabel excreted within the first 24 hours was in the urine and less than 10% was excreted in the feces (Burka et al., 1991; Herr et al., 1991). In another study of ^{14}C-TCEP orally administered to rats, >90% was excreted in the urine, 7% in feces, and 1% as CO_2 within 72 hours (Chadwick et al., 1989 as cited in ECHA, 2009), and about 90% of radioactivity was excreted in the urine within seven days, with minimal excretion in feces or expired air (Minegishi et al., 1988).

At high oral TCEP doses (350 mg/kg), Herr et al. (1991) observed longer excretion half-life for females at higher doses than observed at lower doses (175 mg/kg), a result that Burka et al. (1991) interpreted as reflecting saturation of metabolism in females. This observation is consistent with less cumulative excretion of ^{14}C in the urine and lower fecal excretion ^{14}C relative to males over a 24 hour period (Herr et al., 1991). Daily dosing for nine consecutive days

did not change the elimination or metabolic profile in male or female rats (Burka et al., 1991). In female rats, elimination in urine followed first-order kinetics with averaged half-lives (across 1, four and seven days of dosing) of roughly 6.3 hrs; the elimination half life in males was roughly 7.5 hrs. These differences were significant between sexes after one and four consecutive daily doses with females more rapidly excreting than males, but not after seven consecutive daily doses (Burka et al., 1991).

Peak biliary excretion occurred two hours post-dosing with 25% TCEP excreted in the bile within 48 hours. The biliary/fecal excretion ratio reported for TCEP was 4.62 after 48 hours, suggesting enterohepatic circulation (Minegishi et al., 1988). Biphasic plasma elimination half-lives of 3 and 3.4 hours and red blood cell elimination half lives of 1.8 and 10.8 days were reported (Chadwick et al., 1989 as cited in ECHA, 2009).

Species differences in elimination were noted between rats and mice, with excretion rate of ^{14}C in the urine significantly greater in mice (three times faster than rats) (Burka et al., 1991). There were also quantitative differences in the urinary excretion between species, with mice eliminating about 70% of the radioactivity as the major metabolite (identified as BCCP - bis(2-chloroethyl) carboxymethyl phosphate), while this metabolite represented 46% (females) or 55% (males) of the radioactivity in rats. TCEP was not consistently detected in blood sampled at 30-45 minutes after a single i.v. dose of 20 mg/kg TCEP, suggesting rapid elimination of TCEP (Dix et al., 1994). The authors calculated that the clearance was 53 mL/min per kg and 74 mL/min per kg in females and males, respectively, with a volume of distribution of 1.634 and 1.41 respectively (Dix et al., 1994).

TCEP was detected in human breast milk in 3-8% of the samples from women of Vietnam, the Philippines, and Japan (Kim et al., 2014). The TCEP metabolite, BCEP, was detected in urine samples from non-smoking adults living in California (Dodson et al., 2014). The TCEP metabolite, di(2-chloroethyl) phosphate (DCEP), was detected in urine samples from 312 children attending daycare centers in Germany; the daycare centers were also being measured for air and dust concentrations (Fromme et al., 2014). See Table 2-9.

Table 2-9. TCEP Biomonitoring Data

Country (location)	Tissue/fluid	Concentrations[1]	Reference	Notes
United States	Urinary metabolite BCEP	Mean: 0.76 ng/l Median: 0.63 ng/l Max: 2.1 ng/l	Dodson et al., 2014	Samples collected from 16 non-smoking adults living in northern California.

Country (location)	Tissue/fluid	Concentrations[1]	Reference	Notes
Germany	Urine metabolite DCEP	Mean: 400 ng/l (0.4 µg/l) Median: 200 ng/l (0.2 µg/l) Range: 100-13,100 ng/l <0.1-13.1 µg/l 95th: 1,600 ng/l (1.6 µg/l)	Fromme et al., 2014	Spot urine samples from 312 children attending daycare centers that were also measured for air and dust concentrations
Philippines (Payatas and Malate)	Human breast milk	Payatas Median: 41 ng/g Range: ND-512 ng/g Malate Median: 42 ng/g Range: ND-153 ng/g	Kim et al., 2014	Detection limits were between 0.01 (2.7%) and 0.08 (7.9%) ng/g lipid weight.
Japan (Kanagawa)	Human breast milk	Median: 0.14 ng/g Range: ND-20 ng/g	Kim et al., 2014	Detection limits were between 0.01 (2.7%) and 0.08 (7.9%) ng/g lipid weight.
Vietnam (Hanoi, Bui Dau, Trang Minh)	Human breast milk	All locations Median: ND Bui dau and trang minh Range: ND-18 ng/g	Kim et al., 2014	Detection limits were between 0.01 (2.7%) and 0.08 (7.9%) ng/g lipid weight.

[1]For ease of comparison, all units are converted to ng equivalent. Original study units, if different, are shown in parentheses.
BCEP – bis(2-chloroethyl) phosphate; max – maximum; DCEP – di(2-chloroethyl) phosphate; ND – non detect

2.5 TCEP Exposure Assessments and Estimates

Several authoritative agencies have estimated exposures to TCEP for adults and/or children (ECHA, 2008; Health Canada, 2009; NRC, 2000). Health Canada estimated oral exposures for infants and toddlers from dust and mouthing of foam in their 2009 screening assessment (Health Canada, 2009) and ECHA (2009) estimated exposure for oral, dermal and inhalation routes of exposure. In addition, several publications were reviewed in which the authors estimated exposure levels (e.g., Brommer et al, 2012; Yang et al., 2014; Van den Eede et al., 2011).

It should be noted that multiple approaches exist to calculate the average daily intake, each utilizing different values, institutional practices and accepted assumptions about many factors (e.g., safety factors, using high end or average values for intake estimates, assumptions about

food intake, derivation of those values for subpopulations such as children, toddlers). In an exposure assessment, choices for those values and the assumptions and approaches should be discussed and defended.

Health Canada published its Screening Assessment for the Challenge on TCEP in 2009 (Health Canada, 2009). As part of this assessment, Health Canada derived exposure estimates for infants and toddlers who mouthed foam. The Canadian calculations are based on the U.S. EPA's Voluntary Children's Chemical Evaluation Program for two other flame retardants, pentabromodiphenyl ether and octabromodiphenyl ether (Health Canada, 2009). For toddlers and infants, mouthing of foam is a significant exposure route (Health Canada, 2009). For infants the upper bound estimate of daily intake from dust was 0.2 µg/kg-day for infants and 0.3 µg/kg-day for 0.5 to 4 year olds. The upper bound estimate for mouthing was 39 µg/kg-day for infants and 19 µg/kg-day for 0.5-4 year olds (Health Canada, 2009). The default values used by Health Canada were as follows: water solubility of TCEP is 7820 mg/L, salivary flow rate in child's mouth is 0.22 mL/min, saliva extraction rate is 0.038, absorption factor is 0.5, mouthing behavior frequency is 9 min/day, and body weight is 7.5 kg for infants and 15.5 for toddlers (Health Canada, 2009). For dermal exposure to dust, Health Canada used a dermal adherence rate of 0.05 mg/cm^2 -day for infants and toddlers and 0.07 mg/cm^2 -day for older children and adults. The dermal absorption factor was set to 1 since it is not known for TCEP (Health Canada, 2009).

According to a report on toys from the Scientific Committee on Health and Environmental Risks (SCHER, 2012): "It is not possible to give an adequate estimate of the TCEP exposure of children sucking on toys containing TCEP due to the scant representativeness and reliability of the available data."

The EU RAR for TCEP (2009) also states that there are no data available for estimating TCEP exposure from children sucking on toys (ECHA, 2009). However, ECHA (2009) calculated worst case exposure estimate for adults and children via inhalation, dermal and oral exposure routes and calculated body burden. For inhalation, model estimates of 0.4 µg/kg bw/day (adults) and 0.96 µg/kg bw/day (children) were calculated based on data from Ingerowki et al. (2001) and assumption of 100% absorption. Dermal exposure likewise assumed 100% absorption and considered different sources for an estimate of 4 µg/kg bw/day for adults. ECHA noted that childrens' dermal exposure as related to body weight can exceed that of adults. Oral exposure considered hand-to-mouth behavior resulting in ingestion of dust. For adults, they estimate 0.0033 µg/kg bw/day for adults (99^{th} percentile) and 0.2 µg/kg bw/day for a 3-year old child (99^{th} percentile). They also considered an infant sucking on a toy and estimated a worst case exposure value of up to 240 µg/kg bw/day for a 3-month old baby. The resulting total body burden for females under reasonable worst case conditions and accounting for all exposure pathways is approximately 4.5 µg/kg bw/day; for 1-3 year-olds it is 11 µg/kg bw/day; and for a

3-month old baby the body burden would account for up to 240 µg/kg bw/day, solely from sucking on toys (ECHA, 2009).

In 2000, The National Academy of Sciences published Toxicological Risks of Selected Flame-Retardant Chemicals (NRC, 2000). While TCEP was not one of the flame retardants included in this report, the report included trismonochloropropyl phosphate, another chloroalkyl phosphate. For the dermal and oral assessments, the following factors were used: application rate to the upholstery of 5 mg/cm^2, extraction rate of 0.038, and a release rate of 0.06/day. For particulate inhalation, the following values were used: application rate to the upholstery of 5 mg/cm^2 and release rate of 2.3x10^{-7}/day. For vapor inhalation, the following values were used: application rate to the upholstery of 5 mg/cm^2 and saturated vapor concentration of 35,300 mg/m^3 (NRC, 2000).

Average daily intakes for TCEP in foods are presented in ATSDR (2012). However, these values are calculated from data from 1979 and 1980 and are based solely on fruit consumption and calculated only for infants and toddlers. Average daily intakes for infants were 0.016 µg/kg (1979) and 0.004 µg/kg (1980). For toddlers, the 1979 average daily intake was 0.009 µg/kg and in 1980 was not detected (Gartrell et al., 1985 as cited by ATSDR, 2012).

Brommer et al. (2012) estimated exposure via dust ingestion in subpopulations with various exposure scenarios using TCEP concentrations from their study of cars, offices and residences. Toddler exposure was estimated to range from 0.4 ng/kg bw/day (low, based on 5th percentiles) to 0.33 ng/kg bw/day (typical, based on median concentrations), to 1.7 ng/kg bw/day (high, based on 95th percentiles), with adult exposure ranging from 0.24 ng/kg bw/day (low, 5th percentiles), to 0.05 ng/kg bw/day (typical, medians), to 0.28 ng/kg bw/day (high, 95th percentiles). Exposure projections were based on body weights of 12.3 kg and 70 kg and dust ingestion of 200 mg and 50 mg for toddlers and adults, respectively. Adults were assumed to spend 4.2%, 23.8%, and 72% of their time in cars, offices, and home, respectively. Toddlers were assumed to spend 4.2% of time in cars and the remainder of the day at home (Brommer et al., 2012).

Yang et al. (2014) measured suspended particulate matter collected from offices for a number of organophosphate flame retardants, including TCEP. The measured concentrations of TCEP in airborne dust were used to estimate inhalation exposure for adults using U.S. EPA and International Commission on Radiological Protection (ICRP) models for deposition efficiency and flux of inhaled particles in the respiratory tract. The authors assumed a dust inhalation rate of 16 m^3/day, a body weight of 70 kg, and an 8-hour exposure (Yang et al., 2014). The authors reported a median exposure of 0.24 ng/kg/day and a 95th percentile exposure of 1.02 ng/kg/day for adults using the U.S. EPA model and a median exposure of 0.15 ng/kg/day and a 95th percentile exposure of 0.59 ng/kg/day for adults using the ICRP model (Yang et al., 2014).

Van den Eede et al. (2011) analyzed dust samples from Flemish homes and shops for multiple organophosphate flame retardants, including TCEP (median 2940 ng/g). For adults the authors assumed 20 mg/day or 50 mg/day (average and high ingestion rates, respectively) and an average body weight of 70 kg. For toddlers they assumed a dust ingestion rate of 50 mg/day (average) and 200 mg/day (high), and an average toddler body weight of 12.3 kg. Using median exposure concentrations, the authors calculated a daily ingestion exposure for toddlers of 1.0 ng/kg/day for average ingestion, and 3.7 ng/kg/day for high ingestion; a worst case exposure scenario combined the upper 95th percentile concentration with high ingestion rate resulting in an intake rate of 19.8 ng/kg/day. Using median concentrations for non working adult exposure resulted in 0.1 ng/kg/day for average ingestion and 0.2 ng/kg/day for high ingestion; the worst case for working adults was calculated at 1.5 ng/kg/day.

2.6 TCEP Discussion

TCEP has been detected in many media including outdoor and indoor air, surface water, groundwater, house dust, food, and consumer products. V6, another flame retardant, contains a significant level of TCEP as an impurity and may be an additional source of exposure to TCEP.

The primary sources of exposure to TCEP for consumers appear to be dust and indoor air. The exposure routes are dust ingestion and inhalation of vapors and particulates in indoor air. However, there are limited U.S. data for these exposure sources. There are data from other countries, but use of these data may introduce uncertainty into exposure estimates because TCEP levels in other countries may not be representative of U.S. levels. Due to California's stringent flame retardant regulations, levels of flame retardants in dust are higher than in other parts of the country. Using California data may result in conservative estimates for other areas of the country where TCEP levels may be lower. A separate assessment for California residents may be warranted. There are U.S. PU foam data available for use in calculating exposures from mouthing (see Table 2-8).

TCEP was detected in human breast milk in 3-8% of the samples from women of Vietnam, the Philippines, and Japan (Kim et al., 2014). The TCEP metabolite, BCEP, was detected in urine samples from non-smoking adults living in California (Dodson et al., 2014). The TCEP metabolite, di(2-chloroethyl) phosphate (DCEP), was detected in urine samples from 312 children attending daycare centers in Germany (Fromme et al., 2014).

An area of uncertainty is the percutaneous exposure potential from dermal contact with or ingestion from mouthing of objects containing TCEP. The general mechanism for TCEP diffusion out of treated plastics and into dust is relatively well understood. Similarly, the potential for hand-to-mouth transfer of dust is understood and established. This is not the case for dermal or mouthing transfer of this flame retardant from contact with treated objects. The potential dermal or mouth exposure to TCEP as it is diffusing and being ”expressed” from the

treated foam is not well understood or documented. TCEP molecules would be expected to have a reasonable rate of dermal penetration if they were in contact with the skin. This could be particularly important for children's' items such as changing table pads, infant sleep positioners, portable crib mattresses, and nursing pillows. Transfer and ingestion via child-mouthing of these items and treated furniture in general is another area of uncertainty.

There are limited data available to perform an exposure assessment. Most notably, information on migration and degradation from indoor media and dermal exposure factors are lacking. However, reasonable worst case estimates of exposure can be made using the media concentrations presented herein along with age-specific estimates of inhalation or ingestion rates of these media.

It should be noted that this report compiles data from a variety of sources. We have not evaluated the quality of the studies and their results; rather we included all the relevant data we found. The estimates described in the exposure assessments are presented "as is" without a detailed analysis and critique of the methodology, assumptions, or underlying data quality.

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Appendix A – Flame Retardant Exposure Literature Search Strategy

TERA conducted a thorough literature search that included: Pubmed, Google Scholar, Science Direct, TOXNET (including Toxline), CAB abstracts databases and a general web search. The search terms that were used are listed below.

Search terms

Chemical name OR CAS number

Chemical name OR CAS number AND human

Chemical name OR CAS number AND children

Chemical name OR CAS number AND consumer

Chemical name OR CAS number AND consumers

Chemical name OR CAS number AND residential

Chemical name OR CAS number AND residential AND children

Chemical name OR CAS number AND residential AND consumers

Chemical name OR CAS number AND dust

Chemical name OR CAS number AND "hand to mouth"

Chemical name OR CAS number AND mouthing

Chemical name OR CAS number AND dislodgeable residue

Chemical name OR CAS number AND dermal

Chemical name OR CAS number AND oral

Chemical name OR CAS number AND inhalation

Chemical name OR CAS number AND ingestion

Chemical name OR CAS number AND indoor air

Chemical name OR CAS number AND products

Chemical name OR CAS number AND toys

Chemical name OR CAS number AND pillows

Chemical name OR CAS number AND baby carriers

Chemical name OR CAS number AND baby products

Chemical name OR CAS number AND human exposure assessment

Chemical name OR CAS number AND human risk assessment

Chemical name OR CAS number AND migration

Chemical name OR CAS number AND electronics

Chemical name OR CAS number AND plastic

Chemical name OR CAS number AND food

Chemical name OR CAS number AND air

Chemical name OR CAS number AND soil

Chemical name OR CAS number AND water

Chemical name OR CAS number AND bedding

Chemical name OR CAS number AND mattress

Chemical name OR CAS number AND foam

Chemical name OR CAS number AND carpet

Chemical name OR CAS number AND furniture

Chemical name OR CAS number AND biomonitoring

Chemical name OR CAS number AND breast milk

Inclusion criteria

According to the SOW, exposure should be human with an emphasis on residential or consumer exposures. Therefore, preference will be given to those articles that describe residential or consumer exposures. Articles will be included if human exposures or children's exposures to other sources are included. Articles will also be included if they describe levels in the environment and /or other media because they represent potential sources of exposure. If found, biomonitoring data will be included as they are represented of an exposed population. Also to be included is "grey" literature, such as white papers, poster, or presentations. More focus will be placed on references published two to three years prior to and after the publication of any identified major secondary references (i.e., ATSDR, EPA) for flame retardants because it is assumed that they did a thorough literature search. We will not apply any time exclusions.