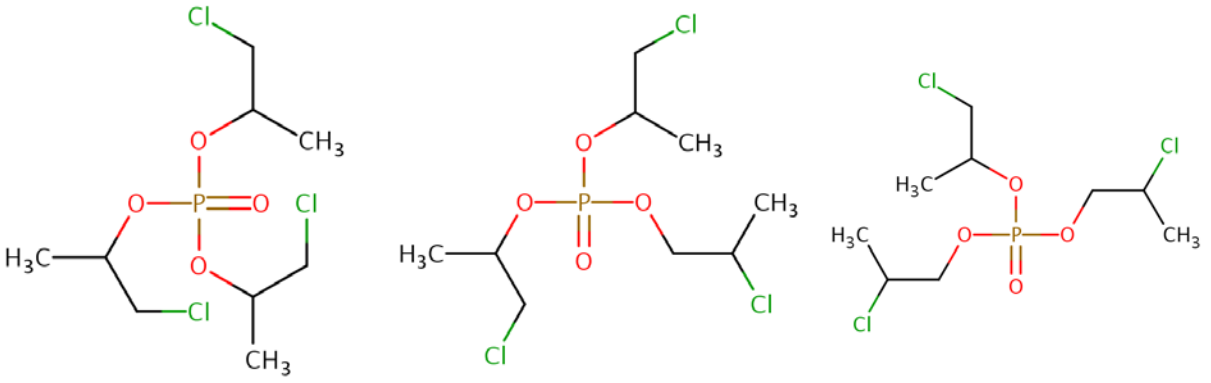
CAS RN 13674-84-5

Substance Name Tris (2-chloroisopropyl) phosphate (**TCPP** or TCIPP)



CAS 13674-84-5 (main isomer) | CAS 76025-08-6 (isomer) | CAS 76649-15-5 (Isomer)

Source: EPA Chemistry Dashboard

Uses

TCPP is an additive flame retardant used in flexible polyurethane foam, textiles, apparel, leather, and electronics. Uses in building construction materials include rigid polyisocyanurate foam insulation, adhesives and sealants, and roofing laminates. It is used for industrial manufacturing of plastic material and resins [1, 2].

TCPP has been detected in U.S. household furniture and in baby products that contain flexible polyurethane foam including: car seats, changing table pads, sleep positioners, portable mattresses, nursing pillows and children's furniture [3-5]. Detection rates in foam are reported to be 0.5-22% by weight in furniture foam and 1-14% in baby product foam [1, 5].

Manufacturers reported to U.S. Environmental Protection Agency (EPA) in 2016, that foam insulation for commercial and consumer uses has a maximum concentration of TCPP of 1- 30% by weight. Wood and engineered wood products used as building materials have less than 1% by weight TCPP. Another commercial use in an unspecified building construction material contains at least 90% by weight. Commercial use in fabrics, textile, and leather products results in maximum concentrations of TCPP of 30 - 60% by weight [2].

Manufacturing

The U.S. national production volume of TCPP was reported to be 54,673,933 pounds in 2012 [1, 6]. In the most recent round of reporting to EPA, the national production volume was given as a range

between 10-50 million pounds/year in 2012 and 2013 and between 50-100 million pounds/year in 2014 and 2015. Four domestic manufactures reported[1] making or importing TCPP in 2016. None are located in Washington State [2].

Toxicity

EPA classified TCPP as high hazard for reproductive and developmental effects based on decreased uterine weights, and increased number of offspring that were runts at the 99 mg/kg-d dose in a 2-generation oral rat study [7, 8]. Another study on this endpoint by Kawasaki et al. 1982, reported that oral dosing in pregnant rats on gestation days 0-20 had no significant effects on the number of implantations or resorptions, maternal or fetal weight, or pup survival and growth in the first 4 postnatal weeks. There was a dose-related 6% increase in missing 13th ribs observed that did not reach statistical significance but that the Agency for Toxic Substances and Disease Registry (ATSDR) and EPA scientists considered biologically significant. They considered the no observed adverse effect level (NOAEL) to be 69 mg/kg-day and the lowest observed adverse effect level (LOAEL) to be 670 mg/kg-day in this study [9, 10].

EPA derived a provisional reference dose (p-RfD) for TCPP to use as a screening value at Superfund sites. They selected a 14 week dietary study in mice conducted by the National Toxicology Program in 2011 as the key study. Male mice exposed orally to TCPP showed increased relative liver weight and decreased body weight (despite increased food consumption) at 456 mg/kg-d (NOAEL was 219 mg/kg-day). At higher doses, hepatocyte hypertrophy and declines in a number of blood cells involved in immune defense were observed. EPA used a bench mark ($BMDL_{10}$) of 138 mg/kg-day and uncertainty factors were used to calculate a subchronic screening level of 0.1 mg/kg-d and a chronic screening level of 0.01 mg/kg-d for TCPP [10].

TCPP has not been tested for cancer but is structurally similar to TDCPP and TCEP which are both demonstrated animal carcinogens [8]. The National Toxicology Program has a cancer assay underway to fill this important data gap [1].

Based on preliminary data, EPA considered TCPP to be a potential inhibitor of cholinesterase and EPA's Office of Research and Development and the National Toxicology Program have studies underway to further assess neurotoxicity and developmental toxicity [1].

TCPP increased testosterone and estradiol levels in a human steroid-producing cell line, H295R [11]. Increased hormone production was accompanied by alterations to genes involved in the hormone's biosynthesis pathway (transcription of steroidogenic genes were up-regulated and sulfotransferase genes were down-regulated) [11]. No direct estrogen receptor agonist activity of TCPP was observed in

[1] Manufacturers of chemicals listed on the Toxic Substances Control Act (TSCA) Inventory were required to report to EPA in 2016 if they produced or imported the chemical in volumes ≥25,000 pounds at a U.S. site during any of the calendar years 2012, 2013, 2014, or 2015. https://www.epa.gov/chemical-data-reporting/2016-chemical-data-reporting-results#overview

MVLN cells [11], or an MCF-7 proliferation assay[12] or in endometrial cancer (ECC-1) cells [13] suggesting that the mechanism is estrogen receptor-independent.

Recent investigations into broader biological activity of TCPP *in vitro* reported that TCPP was active in genes related to xenobiotic metabolism and defense responses in the human fetal hepatocyte cell line, L02 [14]. TCPP administered at 2.5 µM in HepG2 cells, affected a wide array of genes involved in immune response, inflammation, steroid hormone biosynthesis, and xenobiotic metabolism. TCPP showed similar types of activity as TCEP but was active at concentrations that were 10x lower than TCEP. Also of interest, the low dose for both compounds produced more changes in gene expression than the higher dose. Neither of the doses were cytotoxic [15].

Exposure

TCPP use in rigid foam insulation installed in building roofs and sidewalls may be a source of TCPP in indoor air. Although building occupants generally have no direct contact with wall insulation, TCPP is not chemically bound to this material and slowly migrates into the gas phase of surrounding air over time [16, 17]. TCPP in furniture foam can similarly escape over time into indoor air [16]. Emission rates of 50-140 µg TCPP per square meter of foam per hour were reported from furniture assembly foams that contained 20% TCPP [16]. Because of its semi-volatile nature, TCPP that escapes household products will partition to airborne particles and indoor dust. TCPP is also water soluble and children may be exposed to TCPP by mouthing and sucking on treated products and by skin contact with treated products. One study investigated TCPP migration into a test substance that mimics human sweat from a car seat, a baby mattress and a baby sling. TCPP migrated from these children's products into the artificial sweat up to 1,100 mg/m^2 during a 3-hour test [18]. Levels in the environment and in urine have been observed to be higher during warmer months [19-21] indicating that escape from products is sensitive to seasonal temperatures.

TCPP has repeatedly been reported as one of the most abundant organophosphate flame retardants measured in indoor air and in personal air samples from building occupants [22-25]. Two Seattle area studies used personal air monitors clipped to their participants' shirt to collect relevant inhalation measurements for TCPP. Inhalable particulate (defined as > 4µm) carried the bulk of TCPP. Mean inhalable TCPP was 536 ng/m3 in gymnastic coach's homes and 371 ng/m3 in mixed office and home settings. Maximum detected was 1,360 ng/m3 [26]. Less TCPP was associated with respirable airborne particulate (defined as < 4 um). The mean respirable TCPP was 12.3 ng/m3 in mixed office and home settings [26]. The authors assumed that smaller particles are absorbed across the lungs while larger particulate would deposit in the upper respiratory tract and be swallowed. Their estimated total daily intake (ingestion + inhalation) from TCPP in air was 99.4 ng/kg-d in coaches' homes and 71.5 ng/kg-d in mixed residential and office settings.

TCPP has been detected, often with high frequency, in indoor house dust and air by multiple studies in North America [5, 22, 23, 27-30]. Median and mean levels in indoor dust are frequently in the low parts per million (µg/g). The maximum detection of TCPP in these household dust studies was 469 µg/g dust [29]. Increasing levels of TCPP in residential indoor dust correlated with higher TCPP in hand wipes of

toddlers hands (3-6 years old) and with increased urine concentration of TCPP metabolites, indicating that ingestion and dermal exposure to dust is an important exposure source for this age group.[29] TCPP was detected in 100% of car dust samples in a European study [31]. Mean level of TCPP in the dust of 25 cars in Greece was 8062 ng/g (range 110-101,800 ng/g).

Xu et al., 2016, investigated the pathways of residential exposure to TCPP [25]. Multiple TCPP isomers were measured in indoor air (median 128 ng/m^3), 24 hour-personal air samples (median 28 ng/m^3), dust from living room floors (median 1997 ng/g) and dust from other surfaces (median 5241 ng/g), and hand wipes (median 37 ng) from 61 participants and their homes in Norway. The authors then estimated daily exposures for the general adult population for each route of exposure based on mean exposures in their study. Use of stationary air measurements generated an intake estimate of 43.5 ng/kg-d from inhalation. When the authors used personal air measurements of TCPP, the air intake estimate was lower - 9.5 ng/kg-d. Other significant routes of exposures for adults were: surface dust ingestion (15.6 ng/kg-d), floor dust ingestion (3.4 ng/kg-d), dermal absorption from surface dust (3.6 ng/kg-d), and dermal absorption from hands (0.5 ng/kg-d) [25]. Childhood exposures were not estimated.

TCPP has been detected in a variety of foods including baby foods in the U.S. Food and Drug Administration's (FDA) Total Diet Studies conducted between 1991 and 2003. TCPP was detected at low levels (range 0.05 – 0.82 ng/g) [9]. More recently, dietary surveys in Sweden and Belgium detected two TCPP isomers in a wide range of retail market foods (range 0.02 – 109.87 ng/g wet weight). The highest concentrations were in fats and oils (including fish oil) and in cereal products such as flour, grain, corn flakes, pasta, and bread. The per capita dietary consumption for an average Swedish adult was 8.5 ng TCPP/kg body weight per day and for an average Belgian adult was 18.5 ng/kg-day [32, 33].

TCPP is water soluble and has been detected in U.S. surface water used for drinking water. In 2006-07, water testing was conducted at 19 drinking water treatment plants across the U.S., representing the drinking water for more than 28 million Americans [34]. TCPP was detected in 42% of the source water samples with a mean concentration of 180 ng/L and in 28% of finished water samples with a mean of 210 ng/L. Maximum detected in finished water was 510 ng/L [34]. A more recent study in New York state detected TCPP in 91% of tap water samples with a mean concentration of 11.6 ng/L and a maximum of 67.1 ng/L [19]. Waste water treatment plants and atmospheric deposition in rainwater appear to contribute to TCPP in surface waters [19, 35]. Neither primary nor secondary treatment technology were effective at removing TCPP at wastewater treatment plants in New York [35]. A small study in Washington State provided evidence that TCPP in residential indoor dust, picked up on clothing, flows to the wastewater treatment plant in laundry water and may actually be a significant source of TCPP wastewater treatment plants [36].

In biomonitoring studies, two metabolites of TCPP have been measured and detected in human urine: bis (1-chloro-2-propyl) phosphate (BCIPP) and 1-hydroxy-2-propyl bis (1-chloro-2-propyl) phosphate (BCIPHIPP). One or both were detected in urine from toddlers and their mothers in New Jersey [37]; infants, toddlers, and pregnant women in North Carolina [20, 29, 38]; mothers and their children in California [39]; and in adults in Northern California [40]. Relative to the BCIPP metabolite, BCIPHIPP has

been more widely detected (>97%) in adults and children and often at slightly higher mean levels (GMs range 1.1- 3.4 ng/ml) [20, 21, 29, 39, 41].

The U.S. Centers for Disease Control recently included the BCIPP urinary metabolite in the 2013-2014 National Health and Nutrition Examination Study biomonitoring survey. This is the largest and most representative survey available for the U.S. population. Toddlers were not included but younger participants had slightly higher levels or urinary BCIPP than adults. Median and 95th percentile values were 0.25 and 2.12 ng/ml for children 6-11 years old, 0.16 and 1.04 ng/ml for children 12-19 years old, and 0.15 and 1.25 ng/ml for adults 20 years old and older [42]. Total detection frequency was 61% of participants and the maximum level reported was 46.7 ng/ml [43].

TCPP has also been detected in breast milk in Sweden at concentrations up to 82 ng/g lipid [44]. TCPP was detected in 90% of blood samples collected in 2012 from 257 adults in the general population of Shenzhen, China. The median concentration in whole blood was 0.71 ng/ml, the 95th percentile was 1.85 ng/ml, the maximum detected was 21.61 ng/ml [45].

Environmental Fate and Transport

EPA considers TCPP to have high hazard for persistence and low hazard for bioaccumulation[7].

If released to air	• TCPP is expected to exist in the vapor phase and particulate phase, based on its vapor pressure. • Based on a similar structure, predicted half-life for TCPP associated with particle phase is 5 - 10 days. • Deposition may occur with precipitation. TCPP was measured in 100% of rainwater samples in New York State. Mean levels were 61.8 ng/L [19]. • TCPP is detected globally in air and marine waters including in remote areas [46].
If released to soil	• Empirical studies indicate that TCPP is not rapidly biodegradable in soil. • TCPP isomers are expected to have high mobility in soil, based on measured or estimated K_{OC} values. • Leaching through soil to groundwater may occur [7].
If released into water	• TCPP is highly soluble in water and has a low octanol-water partition coefficient. It is considered to be very stable in water and sediments. • TCPP is routinely found in rivers, lakes, and seawater [19].
Bioconcentration and bioaccumulation	• TCPP can be metabolized by aquatic biota and bioconcentration is expected to below.

	• An estimated BAF of 13.26 was calculated in fish, using an estimated log K_{ow} of 2.68.

Physical-Chemical Properties of TCPP from EPA Chemistry Dashboard:

Commercial TCPP is a mixture of isomers: primarily CAS numbers 13674-84-5, 76025-08-6, and 76649-15-5 [1]. Commercial manufacture produces TCPP with 70 – 85% of the first listed isomer [7].

Property	Average		Median		Range		Unit
	Experimental	Predicted	Experimental	Predicted	Experimental	Predicted	
LogP: Octanol-Water	2.59 (1)	2.44 (5)	-	2.69	2.59	1.53 to 2.89	-
Water Solubility	3.66e-03 (1)	9.99e-04 (4)	-	8.44e-04	3.66e-03	1.58e-04 to 2.15e-03	mol/L
Density	-	1.30 (2)	-	1.30	-	1.28 to 1.32	g/cm^3
Flash Point	-	214 (2)	-	214	-	178 to 250	°C
Melting Point	-40.0 (2)	-4.95 (4)	-40.0	-26.3	-40.0	-39.4 to 72.3	°C
Boiling Point	-	328 (5)	-	318	-	283 to 365	°C
Surface Tension	-	35.4 (2)	-	35.4	-	34.2 to 36.6	dyn/cm
Thermal Conductivity	-	132 (1)	-	-	-	-	mW/(m*K
Vapor Pressure	-	4.44e-03 (4)	-	2.02e-03	-	5.25e-05 to 1.37e-02	mmHg
Viscosity	-	5.74 (1)	-	-	-	-	cP
LogKoa: Octanol-Air	-	8.85 (1)	-	-	-	-	-
Henry's Law	-	1.58e-06 (1)	-	-	-	-	$atm\text{-}m^3/m$
Index of Refraction	-	1.46 (1)	-	-	-	-	-
Molar Refractivity	-	70.3 (1)	-	-	-	-	cm^3
Molar Volume	-	256 (1)	-	-	-	-	cm^3
Polarizability	-	27.9 (1)	-	-	-	-	$Å^3$

Numbers in parentheses indicate the number of measurements or model predictions identified by EPA.

Regulatory

The European Commission, Safety of Toys Directive (Directive 2014/79/EC) sets a limit of 5 mg/kg for the content of TCEP, TCPP and TDCP in toys intended for children under 36 months and in toys intended to be put in the mouth, applicable to each of the three substances. This directive became effective December 2015.

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