

2016 Children's Safe Products - Reporting Rule update
Draft Chemical Evaluation

CAS 84852-53-9

Substance Name Decabromodiphenyl ethane (**DBDPE** or DecaBDE-ethane)

Toxicity

EPA classified decabromodiphenylethane (DBDPE) as a high hazard for developmental toxicity based on its structural similarity to decabromodiphenylether (decaBDE) [1]. Available toxicity data has been reviewed by the United Kingdom, Environment Agency in 2007; by EPA in 2014; and by Health Canada and Environment Canada in 2016 [1-3]. Briefly, DBDPE had low acute toxicity in animals, either orally and dermally, and is predicted to have low acute inhalation toxicity. In a 90-day study in rats, minimal systemic effects were reported at the highest dose tested including increased liver size and hepatic cell hypertrophy at 1,000 mg/kg-day (LOAEL). No effects were reported at 320 mg/kg-day (NOAEL). These liver changes were reversible after 14 days post-exposure and the effects were interpreted as an adaptive response to increased demand on the liver to metabolize and excrete DBDPE [1].

In another 90-day oral assay in rats, Wang et al. dosed male rats for 90 days with 100 mg/kg-day DBDPE [4]. No alteration in liver, kidney or body weights was observed indicating no overt toxicity. Authors reported indications of organ impairment in DBDPE-treated rats (decreased serum creatinine, decreased serum liver enzymes alanine transferase and alkaline phosphatase, and increased total bile acids). Liver tissue was not examined for signs of pathology in this study to investigate this observation. DBDPE-treated rats also showed increased serum thyroid hormones T3 and T4 although the difference was not statistically significant for T4 [4]. Thyroid hormones are central to proper mammalian development, including the brain and reproductive organs, so this observation should be further investigated in assays involving prenatal exposure.

Reproductive toxicity testing has not been conducted. In two developmental toxicity tests in rats and rabbits, neither reported treatment-related malformations at birth or altered pup weight or decrease in survivability. The NOAEL was 1,250 mg/kg-day [1, 2]. The developmental tests did not include observations for neurobehavioral effects as the pups matured. DBDPE is structurally similar to decabromodiphenyl ether (decaBDE) and has a similar toxicity profile in acute and short-term toxicity testing [4]. In further investigations of developmental exposures, however, decaBDE has been shown to produce neurodevelopmental toxicity and endocrine disruption in rodents in at much lower doses [5-12]. In fact, EPA used a NOAEL of 2.2 mg/kg-day to establish a reference dose for decaBDE based on neurobehavioral effects of prenatal exposure. Lack of testing for neurodevelopmental outcomes and endocrine disruption are important data gaps for DBDPE given its very close structural similarity to decaBDE. EPA use of toxicity data from decaBDE to score DBDPE's potential for development toxicity is a reasonable approach to address this important gap in toxicity testing.

No cancer testing was identified. DBDPE was negative in two genotoxicity tests [1].

2016 Children's Safe Products - Reporting Rule update Draft Chemical Evaluation

Exposure

DBDPE is a general purpose additive flame retardant for a variety of polymer applications and for textiles. It is a commercially important alternative to decaBDE. It typically comprises 10-15% of the weight of treated plastics (e.g., ABS, HIPS, PVC, polypropylene and polyethylene, etc.). It is used in wire and cable coatings for telecommunications, electrical, and the automotive industry. To a lesser extent it can be used in the latex-based back coating for drapery and upholstery fabrics [2]. DBDPE has been manufactured for more than 20 years and is a High Production Volume (HPV) chemical in the United States today; as of 2012, the National Production volume was 50 to 100 million pounds per year [13].

DBDPE was detected in 1/3 of baby formula and about ¼ of baby cereals collected from the U.S. in 2013 [14]. Median levels of DBDPE detected were 22 and 11 pg/g fresh weight, respectively. The daily median intake for U.S. infants consuming formula and cereal was estimated by authors to be 2.2 – 3.44 ng DBDPE/day.

DBDPE was detected in a child's tablet and plastics of other consumer products by the Washington Department of Ecology at levels of 1000 ppm or lower [15]. It was also detected at lower levels (<100 ppm) in foam, stuffing, and padding of children's products collected by the Washington Department of Ecology [16]. A study that tested a variety of children's toys for sale in China found DBDPE in 80% of hard plastic toys, 89% of foam toys, 50% of the stuffed toys, and 40% of rubber or soft plastic toys including baby teethingers. Maximum levels detected was 237 ppm [17]. Potential migration into saliva was tested by volunteers in this study. One out of 5 volunteers had measurable DBDPE in saliva after lightly chewing a segment of a hard plastic toy in the mouth for 15 min [17].

Because DBDPE is not chemically bound to the treated materials, it can escape into the environment. DBDPE has been widely detected in studies of U.S. house dust [18-21]. The dust levels of DPDPE reported ranged <2.6 -11,070 ng/g dust. DBDPE has also been detected in residential indoor air (mean 5 ng/m³) and at higher levels in a gymnastics facility in Seattle (50 ng/m³)[22]. In addition to U.S. studies, Harrad et al., 2008, studied DPDPE in dust samples from UK homes, offices, and cars. Average (and maximum) concentrations of DPDPE were found to be 270 (3,400), 170 (860) and 900 (2,900) ng/g dust respectively [23].

Only very limited human biomonitoring data are available in the literature for DBDPE. It was measured but not detected in maternal serum in Norway in 2012 [24]. It was detected at low frequency in maternal serum and breast milk collected between 2008 and 2009 in the Sherbrooke region of Canada [25]. Low dermal and oral absorption may explain the low detections in people [1]. DBDPE is listed as a priority for biomonitoring by the California Biomonitoring Program [26].

Two recent government assessments predict that DBDPE has high environmental persistence but came to different conclusions regarding potential for bioaccumulation [2-3]. In a 90-day oral rat study DBDPE and its metabolic products accumulated in adipose, liver and kidney tissue [3]. DBDPE has been detected in environmental media from various parts of the world and in wildlife including birds, dolphins and pandas. There is limited but positive evidence that DPDPE biomagnifies in aquatic food chains [2, 27-28]. More testing is needed to characterize environmental fate, bioavailability and metabolism of DBDPE in different species. If debromination to nona-, octa-, and hepta-bromodiphenyl ethane occurs following the pathway

2016 Children's Safe Products - Reporting Rule update Draft Chemical Evaluation

of debromination established for decaBDE, then degradation products are likely to have high potential for bioaccumulation [3].

References

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2016 Children's Safe Products - Reporting Rule update
Draft Chemical Evaluation

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