Annex XV dossier

PROPOSAL FOR IDENTIFICATION OF A SUBSTANCE AS A CMR CAT 1 OR 2, PBT, vPvB OR A SUBSTANCE OF AN EQUIVALENT LEVEL OF CONCERN

Proposal for identification of Hexabromocyclododecane as a SVHC

Substance Name: Hexabromocyclododecane

EC Number: 247-148-4

CAS Number: 25637-99-4

- *It is proposed to identify the substance as a PBT according to Article 57 (d).*

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PROPOSAL FOR IDENTIFICATION OF A SUBSTANCE AS A PBT SUBSTANCE

Substance Name: Hexabromocyclododecane

EC Number: 247-148-4

CAS number: 25637-99-4

- *It is proposed to identify the substance as a PBT according to Article 57 (d).*

Summary of how the substance meets PBT criteria

Hexabromocyclododecane (HBCDD) fulfils the vB-criterion based on experimental data (BCF=18100) and measured data from biota. With a NOEC of 3.1 µg/l for daphnia, the T-criterion is also met. The available soil degradation simulation tests indicate that the half-life of HBCDD in aerobic soil is > 120 d and thus the P-criterion in soil is met. The experimental data regarding persistence in sediment are varying. According to some of the sediment degradation simulation studies available the P-criterion is met, whereas other studies substance indicates that the substance is degradable in certain experimental conditions. However, data from dated sediment cores gives support to HBCDD being persistent also in sediment. Furthermore, HBCDD is found to be ubiquitously present in remote areas in abiotic samples and biota providing evidence, that the substance is persistent in the environment and undergoes long-range environmental transport. Overall it is concluded, that HBCDD is a PBT substance.

This Annex XV dossier mainly builds on the agreed European Union Risk Assessment Report (RAR) on HBCDD performed under regulation EEC 793/93 and the corresponding European Union Risk Reduction Strategy (RRS). Information from those documents is used in this dossier without giving full references in the dossier. Thus, the reader is referred to the RAR and the RRS (the latter is attached to this dossier). New information and new studies not used in the RAR and RRS are given as full references in the dossier.

Registration number(s) of the substance or of substances containing the substance:

The substance has not yet been registered.

JUSTIFICATION

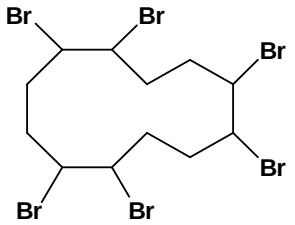
1 IDENTITY OF THE SUBSTANCE AND PHYSICAL AND CHEMICAL PROPERTIES

1.1 Name and other identifiers of the substance

Chemical Name:	Hexabromocyclododecane
EC Name:	247-148-4; 221-695-9[a]
CAS Number:	25637-99-4; 3194-55-6[a]
IUPAC Name:	Hexabromocyclododecane

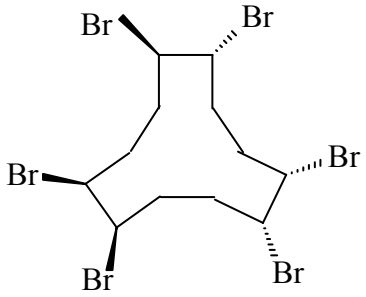
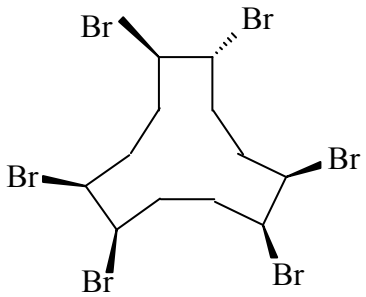
a: The latter no is more specific in terms of the diastereomeric composition of the substance (see below)

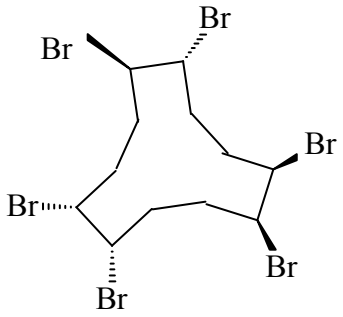
1.2 Composition of the substance

Chemical Name:	Hexabromocyclododecane
EC Number:	247-148-4
CAS Number:	25637-99-4[b]
IUPAC Name:	Hexabromocyclododecane
Molecular Formula:	$C_{12}H_{18}Br_6$
Structural Formula:	
Molecular Weight:	641.7
Synonyms	Cyclododecane, hexabromo; HBCD; Bromkal 73-6CD; Nikkafainon CG 1; Pyroguard F 800; Pyroguard SR 103; Pyroguard SR 103A; Pyrovatex 3887; Great Lakes CD-75P™; Great Lakes CD-75; Great Lakes CD75XF; Great Lakes CD75PC (compacted); Dead Sea Bromine Group Ground FR 1206 I-LM; Dead Sea Bromine Group Standard FR 1206 I-LM; Dead Sea Bromine Group Compacted FR 1206 I-CM;

Concentration range (% w/w):	Depending on the producer, technical grade HBCDD consists of approximately 70-95 % γ-HBCDD and 3-30 % of α- and β-HBCDD due to its production method (European Commission, 2007). Two additional stereoisomers, δ-HBCDD and ε–HBCDD have been found by Heeb et al. (2005) in commercial HBCDD in concentration of 0.5 % and 0.3 %, respectively

[b]: This number refers to unspecific isomer composition.

Chemical Name:	Hexabromocyclododecane
EC Number:	221-695-9
CAS Number:	3194-55-6 This number refers to (1,2,5,6,9,10-hexabromocyclododecane) composed of three main diastereomers. Each of these have a specific CAS No, namely: • (1R,2R,5R,6R,9R,10S)-rel-1,2,5,6,9,10-hexabromocyclododecane [beta-hexabromocyclododecane;CAS No 134237-51-7]. • (1R,2R,5S,6R,9R,10S)-rel-1,2,5,6,9,10-hexabromocyclododecane [alpha-hexabromocyclododecane;CAS No 134237-50-6] • (1R2R5R6S10R)-rel-1,2,5,6,9,10-hexabromocyclododecane [gamma-hexabromocyclododecane.;CAS No 134237-52-8]
IUPAC Name:	Hexabromocyclododecane
Molecular Formula:	$C_{12}H_{18}Br_6$
Structural Formula:	 +/- alpha-HBCDD CAS No: 134237-50-6  +/-beta-HBCDD CAS No: 134237-51-7

	 +/-gamma-HBCDD CAS No: 134237-52-8
Molecular Weight:	641.7
Synonyms	Cyclododecane, hexabromo; HBCD; Bromkal 73-6CD; Nikkafainon CG 1; Pyroguard F 800; Pyroguard SR 103; Pyroguard SR 103A; Pyrovatex 3887; Great Lakes CD-75P™; Great Lakes CD-75; Great Lakes CD75XF; Great Lakes CD75PC (compacted); Dead Sea Bromine Group Ground FR 1206 I-LM; Dead Sea Bromine Group Standard FR 1206 I-LM; Dead Sea Bromine Group Compacted FR 1206 I-CM;
Concentration range (% w/w):	Depending on the producer, technical grade HBCDD consists of approximately 70-95 % γ-HBCDD and 3-30 % of α- and β-HBCDD due to its production method (European Commission, 2007). Two additional stereoisomers, δ-HBCDD and ε–HBCDD have been found by Heeb et al. (2005) in commercial HBCDD in concentration of 0.5 % and 0.3 %, respectively

1.3 Physico-chemical properties

Table1-1: Summary of physico- chemical properties

REACH ref Annex, §	Property	Value	Comments
VII, 7.1	Physical state at 20 C and 101.3 Kpa	White colourless solid	
VII, 7.2	Melting / freezing point	Ranges from approximately: 172-184 °C to 201-205 °C 190 °C , as an average value, was used as input data in the EU risk assessment	Smith et al. (2005)
		179-181 °C α-HBCDD 170-172 °C β-HBCDD 207-209 °C γ-HBCDD	Smith et al. (2005)
VII, 7.3	Boiling point	Decomposes at >190 °C	Peled et al. (1995)
VII, 7.5	Vapour pressure	$6.3 \cdot 10^{-5}$ Pa (21 °C)	Stenzel and Nixon (1997)
VII, 7.7	Water solubility	See Table 1.2	
VII, 7.8	Partition coefficient n-octanol/water (log value)	5.625 (technical product) 5.07 ± 0.09 α-HBCDD 5.12 ± 0.09, β-HBCDD	MacGregor and Nixon (1997) Hayward et al. (2006)

		5.47 ± 0.10 γ-HBCDD	
	Dissociation constant	-	

Table1-2 Summary of the results of valid water solubility studies using generator column method, as evaluated by European Commission (2007)

Test substance	**Water**	**Water solubility (µg l^{-1})**	**Reference**
α -HBCDD	Water	48.8±1.9	MacGregor and Nixon (2004)
β -HBCDD		14.7±0.5	
γ -HBCDD		2.1±0.2	
HBCDD technical product, sum of above		65.6	
α -HBCDD	Salt-water medium	34.3	Desjardins et al. (2004)
β -HBCDD		10.2	
γ -HBCDD		1.76	
HBCDD technical product, sum of above		46.3	
γ -HBCDD	Water	3.4±2.3	Stenzel and Markley (1997)

2 MANUFACTURE AND USES

According to the producers, HBCDD is manufactured by bromination of the starting material *cis, trans, trans*-1,5,9-cyclododecatriene (EU RAR, 2008). The uses identified in the EU risk assessment are presented in Table 2.1.

Table 2.1 Uses of HBCDD identified in EU RAR (2008).

Material	Use/Function	End-products (examples)
Expandable polystyrene (EPS)	Insulation	Construction, insulation boards, (packaging material) Packaging material (minor use and not in food packaging) Insulation boards (against cold or warm) of transport vehicles e.g. lorries and caravans Insulation boards in building constructions e.g. houses' walls, cellars and indoor ceilings and "inverted roofs" (outdoor) Insulation boards against frost heaves of road and railway embankments
Extruded polystyrene (XPS)	Insulation	Construction, insulation boards, Insulation boards (against cold or warm) of transport vehicles e.g. lorries and caravans Insulation boards in building constructions e.g. houses' walls, cellars and indoor ceilings and "inverted roofs" (outdoor) Insulation boards against frost heaves of road and railway embankments
High impact polystyrene (HIPS)	Electrical and electronic parts	Electric housings for VCR Electrical and electronic equipment e.g. distribution boxes for electrical lines Video cassette housings
Polymer dispersion on cotton or cotton/synthetic blends	Textile coating agent	Upholstery fabric bed mattress ticking Flat and pile upholstered furniture (residential and commercial furniture), Upholstery seatings in transportation, draperies, and wall coverings, Interior textiles e.g. roller blinds automobile interior textiles

3 CLASSIFICATION AND LABELLING

3.1 Classification in Annex I of Directive 67/548/EEC

The substance is not classified under Directive 67/548/EEC.

Classification of HBCDD with N; R50/53 was agreed at a Technical Committee for Classification & Labelling (TC C&L)-meeting on 11-12 June, 2003. Classification for health effects has not yet been discussed and HBCDD is therefore not included in Annex I to Directive 67/548/EEC.

3.2 Self classification(s)

4 ENVIRONMENTAL FATE PROPERTIES

4.1 Degradation

Indirect photochemical degradation in the atmosphere is considered to be slow based on the estimated half-life of 3.2 days for the reaction with OH-radicals using AOP v1.91 (24 h day^{-1}; $5*10^5$ OH^- cm^{-3}). Wania (2003) estimated a photochemical degradation half-life of 51.2 hours using the same model but different settings.

Additionally, HBCDD has been observed to degrade in the abiotic controls of biodegradation tests described in the next section.

Hydrolysis is not likely to be a significant route of environmental degradation for HBCDD due to its very low water solubility.

4.1.1 Stability

4.1.2 Biodegradation

4.1.2.1 Biodegradation estimation

4.1.2.2 Screening tests

One reliable ready biodegradability test result is available for HBCDD. Schaefer and Haberlein (1996) observed no degradation in an OECD 301D –test with a test concentration of 7.7 mg l^{-1}. Based on the result, HBCDD is considered to be not readily biodegradable.

4.1.2.3 Simulation tests

Two large degradation simulation studies and supporting screening tests have been conducted by Davis et al. (2003a, b and 2004). Below the results and test conditions are briefly discussed. More details are presented in EU RAR, 2008.

Simulation tests, soil

In an aerobic soil-dissipation study according to OECD 307 (Davis et al., 2003a), γ-HBCDD disappeared with a half-life of approximately 4 months (119 days) at 12 ^oC from sandy loam soil amended with 5 mg kg^{-1} sewage sludge. The nominal test concentration was 25 µg technical HBCDD kg^{-1} dw. In abiotic soil samples almost no dissipation occurred during 119 days indicating that biotic mechanisms may be involved in the dissipation of γ-HBCDD from aerobic soil. However, no transformation products were detected and the fate of the α- and β-diastereomers was not studied. The extraction method was not completely reliable (recovery relatively low) and thus, the half-lives derived from this study may not solely represent biodegradation.

In an aerobic soil simulation study of Davis et al. (2004) conducted according to OECD 307, no indications of any transformation of ^{14}C-HBCDD during 112 days of incubation at 20±2^oC were observed. The nominal test concentration was 3.0 mg technical HBCDD kg^{-1} dw. The recovery of radioactivity was very good throughout the test. Even if metabolites would have been formed at levels below the detection limit (0.4 % of added radioactivity), such potential transformation is not considered to contradict the indicated persistence of HBCDD in soil. The result from this study also supports the assumption that the results of Davis et al. (2003a) may overestimate the degradability of HBCDD in soil.

Simulation tests, sediment

In a simulation study by Davis et al. (2003a) only the disappearance of the γ-diastereomer was followed, since the test concentration was too low to allow for quantification of the α- and β-diastereomers. The test was performed at 20±1°C with nominal test concentrations of 34 and 60 µg technical HBCDD kg^{-1} dw in two different sediments. The disappearance of γ-HBCDD from the aquatic water/sediment systems resulted in approximate DT50-values of 21 and 61 days (recalculated to 12 °C) under **aerobic** conditions in the two systems, respectively. The disappearance half-lives under **anaerobic** conditions were around 2 days in both systems (recalculated to 12 °C). Lack of disappearance in abiotic samples (steam sterilisation at 120° C; 15 psi; 60 minutes) indicates that biotic mechanisms were probably involved. No degradation products were detected, neither in the headspace of the microcosms nor in the water or sediment phases. Since radiolabelled substance was not used and test concentrations were very low, mineralisation of HBCDD could not be followed and no mass balance could be established. It is noted that the recovery varied significantly (33-125 %) indicating problems with the extraction method. Therefore, it is not certain that the disappearance in this study only reflects biodegradation. The half-life values obtained from this study may overestimate the degradability of γ-HBCDD.

In the second sediment simulation study (Davis et al., 2004), the aim was to identify potential metabolites by means of using ^{14}C-labelled HBCDD and optimised methods for the extraction and analyses. By using approximately 100-fold higher HBCDD concentrations than in the simulation study of Davis et al. (2003a) (4.7 mg kg^{-1} dw in aerobic sediment, 4.3 mg kg^{-1} dw in anaerobic sediment) the disappearance of the α- and β-diastereomers could also be followed. There were no indications of an influence of HBCDD on the biological activity of the samples. Table 4.1 provides an overview of the results.

Table 4-1 Estimated primary degradation half-lives of HBCDD derived from the results of the degradation simulation tests of Davis et al. (2004) for the EU risk assessment (EU RAR, 2008).

Medium/Standard	**Sampling site**	**Degradation half-life of HBCDD in viable flasks at 20°C (value in parenthesis corrected to 12 °C)**	**Degradation half-life of HBCDD in abiotic flasks at 20°C (value in parenthesis corrected to 12 °C)**
Aerobic sediment/OECD 308	Schyukill River, Valley Forge, Pennsylvania, U.S.	Total HBCDD: 101 d (191 d) α-HBCDD: 113 d (214 d) β-HBCDD: 68 d (129 d) γ-HBCDD: 104 d (197 d)	Not estimated
Anaerobic sediment/ OECD 308	Schyukill River, Valley Forge, Pennsylvania, U.S.	Total HBCDD: 66 d (125 d) α-HBDD: 113 d (ca. 210 d) β-HBCDD: 44 d (ca. 80 d) γ-HBCDD: 65 d (ca. 125 d)	Not estimated

The study of Davis et al. (2004) also showed that HBCDD undergoes a step-wise reductive dehalogenation via tetrabromocyclododecene and dibromocyclododecadiene to 1,5,9-cyclododecatriene in aerobic as well as anaerobic sediment (see Figure 4.1). There were no indications of further transformation of 1,5,9-cyclododecatriene as no CO_2 or other volatiles were formed during the course of the study.

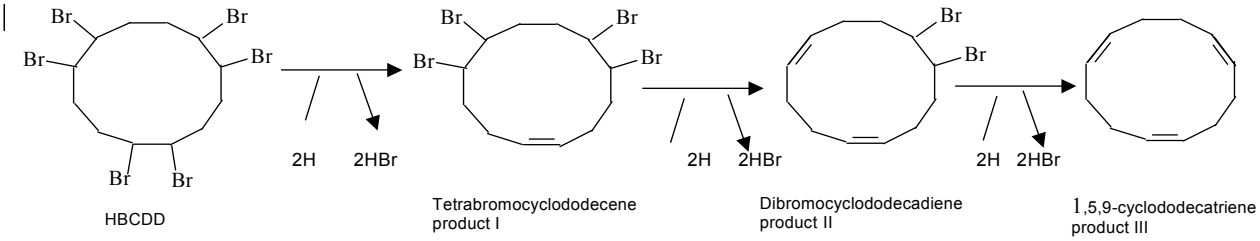


Figure 4-1 Stepwise dehalogenation of HBCDD (Davis et al., 2004).

Degradability of 1,5,9-cyclododecatriene (CDT) has been studied in two reliable modified ready biodegradation tests (Davis et al., 2006a, Davis et al., 2006b). CDT is clearly not ready biodegradable, but does not fulfil the P criterion of the TGD. Despite the fact, that primary degradation and even mineralisation was observed in two reliable biodegradation screening tests with CDT, no mineralisation was observed in the simulation and screening degradation studies with HBCDD. This may be due to: Firstly, the duration of HBCDD-experiments could not be long enough to discover any mineralisation even in those favourable conditions, where HBCDD was degraded in relevant amounts to CDT. Secondly, significant amounts of HBCDD were observed to degrade to CDT only in anaerobic conditions, whereas it is likely, that further degradation of CDT would need aerobic conditions. Hence, the available degradation data on CDT cannot be directly used to judge the overall degradation potential of HBCDD in the environment and vice versa.

Other information

Kohler et al. (2006) found HBCDD in one Lake Greifensee (CH) sediment core, sampled at a depth of 31 m, at concentrations of 2.5 µg kg^{-1} dw at the surface (year 2001), 1.8 µg kg^{-1} dw in a layer sedimented in 1995, 1.2 µg kg^{-1} dw in a layer sedimented in 1989 and 0.25 µg kg^{-1} dw (LOD) or lower in layers sedimented in 1982 and 1974. The initial exposure of sediment for the same years cannot be estimated retrospectively, and therefore it is not possible to estimate degradation half-life from the sediment core. It is nevertheless likely, that the exposure has not been considerably higher in the earlier years than in the year 2001, but more likely lower due to the increased market volumes of brominated flame retardants in the last decades. Christensen et al. (2004), Fjeld et al (2006b), Remberger et al. (2004) and Sternbeck et al. (2001) have also measured HBCDD in sediment core samples.) Also sediment cores from Tokyo bay in Japan (Minh et al, 2007) shows increasing HBCDD concentrations in sediment from the early 80-ies until early 2000s.
Although there are some uncertainties embedded to the dating of the sediment samples, the results show a significantly slower apparent decrease of HBCDD concentrations with time compared to what would be expected based on the half-lives obtained from some of the sediment biodegradation simulation tests.
HBCDD has been found in abiotic and biotic samples of even the most remote areas (see Table 4.2) and concentrations in biota have been increasing based on several temporal series (see section 4.3.3). These findings indicate that HBCDD behaves in the environment like a persistent substance.

4.1.3 Summary and discussion of persistence

Two large standard degradation simulation studies on HBCDD are available for sediment and soil (Davis et al., 2003a, b and Davis et al., 2004). No degradation was observed in the study of Davis et al. (2004) in aerobic soil. A significantly faster disappearance was observed in the sediment tests of Davis et al. (2003a) than in the study of Davis et al. (2004). Degradation half-lives calculated based on the results of Davis et al. (2004) are for aerobic sediment at 12 ºC 214 d (α-HBCDD), 129 d (β-HBCDD) and 197 d (γ-HBCDD) and for anaerobic sediment ca. 210 d, 80 d and 125 d, respectively.
Despite significantly higher test concentrations in the study of Davis et al. (2004) compared to the study of Davis et al. (2003a), there are several reasons for considering the results of Davis et al. (2004) more reliable. Firstly, no mass balance could be made and the recovery was generally bad at the start in the tests of Davis et al. (2003a). Dissipation to non-extractable residues and problems with extraction may have influenced the results. Furthermore, brominated degradation products were not detected at any time in the microcosms according to the authors. In the degradation simulation tests of Davis et al. (2004) a mass balance could be derived. Non-extractable adsorption to soil occurred only in the viable aerobic microcosms, which encountered for the ^{14}C-HBCDD losses observed in the extract. In abiotic control of the aerobic soil test and in the sediment tests the radioactivity was recovered in the extracts at a very good level throughout the study. The authors could also follow the emergence of several degradation products. The amount of HBCDD mineralised (measured as $^{14}CO_2$) and other volatile ^{14}C-degradation products were monitored and remained negligible in all tests. Davis et al. (2004) observed that α-HBCDD was degraded more slowly in the sediment test than β- and γ-HBCDD.
1,5,9-cyclododecatriene (CDT) was observed by Davis et al (2004) to be the main degradation product of HBCDD. Despite the fact, that primary degradation and even mineralisation has been observed in two reliable biodegradation screening tests with CDT, no mineralisation was observed in the simulation and screening degradation studies with HBCDD. This may be explained by the duration of HBCDD-experiments which could not be long enough to discover any mineralisation even in those favourable conditions, where HBCDD was degraded in relevant amounts to CDT. In addition, significant degradation of HBCDD to CDT was observed only in anaerobic conditions, whereas it is likely, that further degradation of CDT would need aerobic conditions. Hence, the available degradation data on HBCDD cannot be directly used to judge on the overall degradation potential of CDT in the environment and vice versa.
In addition to the experimental data, HBCDD has been found in abiotic and biotic samples of even the most remote areas and concentrations in biota have been increasing based on several temporal series. Furthermore, sediment core samples analysed indicate a slower degradation of HBCDD in sediment than what would be expected based on the simulation studies. It is concluded, that HBCDD is persistent in the environment, although it has been observed to degrade in certain experimental conditions in the aquatic environment.

4.2 Environmental distribution

4.2.1 Adsorption/desorption

No experimental data on adsorption are available. A logKoc of 4.66 has been derived in the EU RAR, 2008 indicating very high adsorption potential. HBCDD's mobility in soil and sediment can be expected to be very low.

4.2.2 Volatilisation

Based on the measured vapour pressure (6.3×10^{-5} Pa at 20 °C), HBCDD is very slightly volatile. Henry's law constant at 20-25 °C is 0.75 Pa m^3 mol^{-1} based on the sum of the water solubilities of the individual diastereomers (66 µg l^{-1}). Hence, HBCDD has a low potential to evaporate from aqueous surfaces. Due to the low volatility and high adsorption potential to suspended matter, evaporation of HBCDD seems to be a less important route of distribution.

4.2.3 Distribution modelling

Long range transport

HBCDD has a very slow atmospheric degradation rate (half-life > 2 days, see section 4.1.1), which indicates potential for long-range atmospheric transport in vapour phase. Despite of this, due to the low volatility and high adsorption potential, the majority of long-range environmental transport of HBCDD is likely to occur in aerosol form (Wania, 2003).

Measured data from remote regions provide evidence that HBCDD is subject to long-range environmental transport (see Table 4.2). In addition to data in Table 4.2, HBCDD has also been found in birds (i.e., in eggs, liver, blood) in remote Arctic areas in several studies. HBCDD has been found in these studies in the majority of samples (see EU RAR, 2008 for references).

Table 4-2 Measured environmental concentrations of HBCDD in remote Arctic areas (bird data excluded).

Species, sample type/	Location; sampling yeasr	Concentration	Reference
Air	Ammarnäs, northern Sweden	5.7 pg HBCDD/m^3 in particulate phase 0.2 pg HBCDD/m^3 in vapour phase	Bergander et al. (1995)
	Pallas, Finland	0.003 ng HBCDD/m^3 (autumn 2000), total conc. 0.002 ng HBCDD/m^3 (winter 2001), total conc.	Sternbeck et al. (2001)
Deposition	Pallas, Finland	13 ng/m^2 d, precipitation 21 mm (autumn 2000) 5.1 ng/m^2 d. precipitation 4 mm (winter 2001)	Sternbeck et al. (2001)
Sediment	Ellasjøen, Bjørnøya, Svalbard, Norway	3.8 ng γ-HBCDD /g dw in a sediment layer corresponding years 1973-1987. α- and β-HBCDD were below LOD. All diastereomer concentrations in top layer (1987-2001) and earlier than 1973 were <	Christensen et al. (2004)

Species, sample type/	Location; sampling yeasr	Concentration	Reference
		LOD.	
Invertebrates			
Gammarus wilkitzkii	North Atlantic, Svalbard area, Norway; 2003	Not detected	Sørmo et al. (2006)
Fish			
Polar cod (*Boreogadus saida*); whole fish	Svalbard, Norway; 2003	1.73 µg HBCDD/kg lw (median); min-max: 1.38-2.87, n = 7	Sørmo et al. (2006)
Polar cod (*Boreogadus saida*); whole fish	Bjørnøya, Svalbard, Norway; 2003	11.7 ±7.2 µg HBCDD/kg lw (mean±SD), n = 6	Jenssen et al. (2007)
Mammals			
Polar bear (*Ursus maritimus*), adipose tissue (females)	Svalbard, Norway; 2002	26±9.0 µg HBCDD/kg ww (mean±SD), min-max: 9.7-45, n = 15	Gabrielsen et al. (2004)
Polar bear (*Ursus maritimus*), adipose tissue (males)	Svalbard, Norway; 2002-2003	12.6 µg HBCDD/ kg lw (median); min-max: 5.31-16.51, n = 4	Sørmo et al. (2006)
Harbor seal (*Phoca vitulina*), blubber	Svalbard, Norway; 2003	3.66±1.54 µg HBCDD/kg lw (mean±SD), n=5	Jenssen et al. (2007)
Ringed seal (*Pusa hispida*), blubber	Svalbard, Norway; 2003	16.96 µg HBCDD/kg lw (median); min-max: 14.6-34.5, n = 6	Sørmo et al. (2006)

Additionally, Ueno et al. (2006) have determined half-distances for HBCDD, polybrominated diphenyl ethers and "existing" POPs (see Table 4.3).

Table4-3 Calculated half-distances for HBCDD, PBDEs and POPs in the North Pacific based on skipjack tuna monitoring (compiled in Ueno et al., 2006).

Substance	Number of sites	Correlation coefficient (r^2)	Half-distance±SE (km)
α –HCH	5	0.83	-1700±480
α-HBCDD	4	0.45	8500 ±6700
γ-HBCDD	4	0.73	1600±680
BDE-99	5	0.87	1400±320
BDE-153	5	0.79	1200±380
2378-T4CDF	5	0.93	3200±530
23478-P5CDF	5	0.87	2100±470
∑PCBs	5	0.77	1500±480
p,p'-DDT	5	0.91	950±170

Half-distance was in this study defined as the distance from the source (Japan), where the concentration in tuna muscle drops to 50 % of the concentration at/near the source. Although the authors state, that concentration in tuna muscle lipids well reflects the concentration of pollutants in water at the sampling site, it must be noted, that this method cannot distinguish between long-range transport via air and water, although it can apparently exclude the impact of migration.

According to the authors, the half-distance of HBCDD reflected one of the highest long-range transportabilities among the substances investigated. However, it must be noted, that for HBCDD, significance of the distance-to-concentration correlation was very low (r^2 = 0.45; p=0.33) and standard errors of the estimates were rather high, probably due to the low amount of sites included (four sites used as the basis of the regression). Nevertheless, when the results for HBCDD are considered together with the results of other organohalogen compounds studied, the findings of Ueno et al. (2006) can be taken as evidence of a high long-range transport potential for HBCDD.

4.3 Bioaccumulation

4.3.1 Aquatic bioaccumulation

4.3.1.1 Bioaccumulation estimation

A measured logKow of 5.625 is available for the technical product. In another study (Hayward, et al. 2006) logKow was estimated for the individual diastereomers to be 5.07 for α- , 5.12 for β- and 5.47 for γ-HBCDD.

4.3.1.2 Measured bioaccumulation data

Bioconcentration in fish has been determined in two reliable flow-through tests.

Veith et al. (1979) carried out a 32-day flow-through test with *Pimephales promelas*. Mean test concentration was 6.2 µg l^{-1} and test temperature 25 ± 0.5 ºC. The steady-state BCF was calculated to be 18 100.

Drottar and Krueger (2000) conducted a flow-through test according to OECD 305 (and corresponding ASTM and U.S. EPA –standards) with *Oncorhynchus mykiss*. Two exposure groups (0.34 and 3.4 µg l^{-1} nominal) and a solvent control group were run containing 85 fish per group. As test substance, HBCDD with diastereomer composition typical for a commercial product was used. Acetone was used as solvent. Duration of exposure and depuration phases was 35 days each. The aquaria were kept in a temperature of 12 ± 1 ºC. Mean measured exposure concentrations during the uptake phase were 0.18 and 1.8 µg l^{-1}. Apparent steady-state whole fish BCFs of 13 085 and 8 974 were calculated for the low and high exposure group, respectively. Corresponding kinetic BCFs were 21 940 and 16 450. BCFs calculated for muscle were also all above 5 000.

Law et al. (2006a) exposed juvenile rainbow trout (*Oncorhynchus mykiss*) via diet to α-, ß- and γ-HBCDD (separate aquaria for each diastereomer). Additionally, a control aquarium was run. The uptake phase lasted 56 days followed by a 112-day depuration period. Muscle samples were analysed at various points of uptake and depuration phases. No peaks of debrominated or OH-HBCDD metabolites were found in either the muscle or liver tissue extracts. The BMFs for the α-, ß- and γ-diastereomers were calculated to be 9.2, 4.3 and 7.2, respectively.

After the termination of the biomagnification test (day 168) the authors observed, that a major part of HBCDD in muscle samples of fish exposed solely to ß-HBCDD was in the form of α- and γ-HBCDD. In the fish exposed to γ-HBCDD a major part of HBCDD found was α-HBCDD. In the fish exposed to α-HBCDD, no shift to other diastereomers was found. The study shows, that the diastereomeric distribution of HBCDD can be changed by way of bioisomerisation in biological material.

Additionally, Janák et al. (2005b) observed diastereomer and enantiomer selective metabolisation rates in microsomal liver preparations of common dab (*Limanda limanda*). According to the authors, α-HBCDD was least biotransformed of the main three diastereomers tested.

4.3.2 Terrestrial bioaccumulation

There are no earthworm BCF studies available. There is, however, a study on the survival and reproduction of earthworm (Aufderheide *et al.*, 2003) were the concentration of HBCDD in earthworms has been measured.

The earthworms were exposed to HBCDD for a total of 56 days to nominal test concentrations of 78.5,157, 313, 625, 1250, 2500 or 5000 mg HBCDD/kg soil (dwt). After 28 days of exposure adult earthworms were collected, placed on glass dishes and allowed to purge their gut contents for 48 hours. After that they were rinsed in deionised water and stored frozen until analysis. Composite samples of the worms from each exposure group were analysed for the separate diastereomers using HPLC.

The total concentration of HBCDD in worm tissue in the different exposure groups after 28 days of exposure was 3.4, 7.3, 16.8, 15.3, 53, 71.2, and 150 µg per worm tissue (wwt). The bioaccumulation factors based on soil and worm wet weight concentrations ranged between 0.03 and 0.08 (see Table 4-4)

Table 4-4 Concentration of HBCDD in soil and earthworm tissue after 28 day of exposure and corresponding bioaccumulation factors (BAF) at different levels of exposure.

Mean measured concentration of HBCDD in soil day 28 (mg/kg dwt)	**Mean measured concentration of HBCDD in soil day 28 (mg/kg wwt)***	**HBCDD in worm tissue day 28 (mg/kg wwt)**	**BAF (wwt/wwt)**
61	54	3.4	0.06
145	128	7.3	0.06
244	215	16.8	0.08
578	509	15.3	0.03
1150	1012	53	0.05
2180	1918	71.2	0.04
4190	3687	150	0.04

*Recalculated from dry weight using the default conversion factor from EUSES between dry and wet soil of 0.88.

In Table 4-5 the concentrations of the diastereomers α-, β- and γ-HBCDD in soil and worm tissue are presented together with diastereomer specific BAFs. The concentration of the diastereomer is relatively higher in the worm tissue than in soil. In soil the α-diastereomer makes up approx 6 % of the total HBCDD concentration whereas in worm tissue the α-HBCDD fraction is approx 60 % of the total concentration. The diastereomer specific BAF is more than one order of magnitude higher for α-HBCDD than for γ-HBCDD. This is in line with what has been observed also for other biota e.g. mammals and fish where the α-HBCDD is the dominating diastereomer.

The reason for this difference is not known. It could be due to e.g. higher uptake of the α-diastereomer or differences in metabolism between the diastereomers.

Table 4-5 Concentration of α-, β- and γ- HBCDD in soil and earthworm tissue after 28 day of exposure, and diastereomer specific bioaccumulation factors (BAF) at different levels of exposure.

Mean measured concentration of α-, β-, γ- HBCDD in soil day 28 (mg/kg dwt)			**Concentration of α-, β-, γ- HBCDD in worm tissue day 28 (mg/kg wwt)**			**Diastereomer specific BAF. (dwt/wwt)**		
α	**β**	**γ**	**α**	**β**	**γ**	**α**	**β**	**γ**
3.55	11.8	45.8	2.09	0.352	0.953	0.6	0.03	0.02
8.41	28.0	109	4.55	0.769	2.00	0.5	0.03	0.02
14.2	47.1	183	10.7	1.91	4.15	0.8	0.04	0.02
33.5	112	433	11.2	2.01	2.12	0.3	0.02	0.005
66.7	222	861	29.0	6.10	17.9	0.4	0.03	0.01
126	421	1633	41.1	12.1	18.0	0.3	0.03	0.01
243	809	3138	72.9	23.8	53.0	0.3	0.01	0.02

Other supporting information

A large set of data on measured concentrations in biota and few trophic transfer studies are available and have been presented comprehensively in the EU RAR (2008). In the following, only a small part of that information is presented.

Measured concentrations in European surface waters and in freshwater fish as compiled in the EU RAR (2008) indicate, that HBCDD accumulates in fish in the field. The recent very few measurements of HBCDD in filtered water samples in European surface waters (n=14) show a range from 0.016 (or below detection limit) to 1.5 µg l^{-1} (point source recipient site, River Skerne). Table 4-6 provides an overview of the measured concentrations in freshwater fish muscle in Europe.

Table 4-6 Statistical overview of measured HBCDD concentrations in muscle of freshwater fish in the EU and Norway. The percentiles were calculated using weighted average at X(n+1)p (EU RAR, 2008).

	Conc.	**n**	**Median**	**Geometric mean**	**Arithmetic mean ± SD**	**90P**	**Min**	**Max**
All values	µg HBCDD kg^{-1} ww	151	5.5	4.64	321 ± 1130	834	0.005	9432

	µg HBCDD kg $^{-1}$ lw	151	120	171	5223 ± 18745	7927	0.52	160905

It is noted, that concentration in whole fish can be expected to be even higher.

Table 4-7 provides an overview of measured concentrations of HBCDD in fish and marine mammals in Europe.

Table 4-7 Median concentrations of HBCDD in marine mammals and fish muscle collected from specific European regions. As for marine mammals the concentration in blubber is reported conventionally, the data have been converted to whole body concentrations assuming a 1/3 lipid/whole body ratio (EU RAR, 2008).

Region	**Species**	**n**	**Median concentration**	**Concentration ratios (marine mammals/fish muscle)**	
				ww bw/ ww	**lw/lw**
Western Europe	Fish	102	0.40 µg HBCDD kg^{-1} ww	272	28
		100	13 µg HBCDD kg $^{-1}$ lw		
	Marine mammals	225	109 µg HBCDD kg^{-1} ww		
		225	368 µg HBCDD kg $^{-1}$ lw		
Baltic Sea	Fish	42	0.31 µg HBCDD kg^{-1} ww	61	5.8
		38	11.5 µg HBCDD kg $^{-1}$ lw		
	Marine mammals	2 (representing 20 + 30 individuals)	19 µg HBCDD kg^{-1} ww		
		2	67 µg HBCDD kg $^{-1}$ lw		
WesternScheldt (approx. region)	Fish	18	1.8 µg HBCDD kg^{-1} ww	187	11
		16	107 µg HBCDD kg $^{-1}$ lw		
	Marine mammals	19	336 µg HBCDD kg^{-1} ww		
		19	1144 µg HBCDD kg $^{-1}$ lw		
U.K.	Fish	300 (5 dietary relevant species; each species pooled data of 60 individuals)	0.44 µg HBCDD kg^{-1} ww	1859	44
		300	63 µg HBCDD kg $^{-1}$ lw		
	Harbour porpoise	34	818 µg HBCDD kg^{-1} ww		
		34	2780 µg HBCDD kg $^{-1}$ lw		

The concentration ratios presented above may overestimate the actual field biomagnification as concentrations in fish muscle have been used for the calculations instead of whole fish. Therefore, EU RAR, 2008 estimated additionally for the U.K. dataset a ratio based on HBCDD concentration in whole fish. The ratio between harbour porpoise and its diet was calculated at 254.

Temporally increasing concentrations have been observed for several species. Law et al. (2006) measured HBCDD in blubber of 85 harbour porpoises stranded or dying in the U.K. during 1994-2003. The mean concentration in the mid-1990 was 100 µg kg^{-1} lw and increased to 9 400 µg kg^{-1} lw in 2003. Knudsen et al. (2005) found a statistically significant, increasing trend of HBCDD concentrations between 1983 and 2003 in eggs of six marine bird populations (Atlantic puffin, herring gull, kittiwake; n = 89 in total) from two remote locations in the Norwegian Arctic. Concentrations have risen from 1.1-2.9 µg kg^{-1} ww in 1983 to 6.1-17.3 µg kg^{-1} ww in 2003. Sellström et al. (2003) found a temporally increasing trend in Baltic Sea guillemot eggs, although the concentrations seem, according to the author, to have levelled off in the last decade (see Figure 4.2).

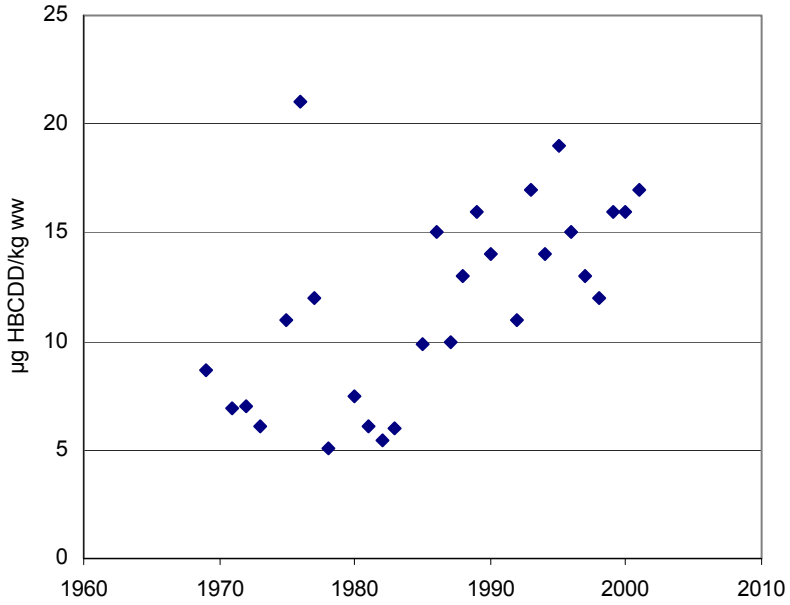


Figure 4-2 Concentration of HBCDD over time in guillemot (Uria aalge) eggs in the Baltic Sea (data from Sellström et al., 2003)

In addition a recent Swedish study (Swedish Museum of Natural History, 2007) shows an ongoing increase of the HBCDD-levels in Guillemot eggs from the Baltic Sea (Stora Karlsö) of about 3% per year during the recent 10 years period (1994-2004) see Figure 4.3.

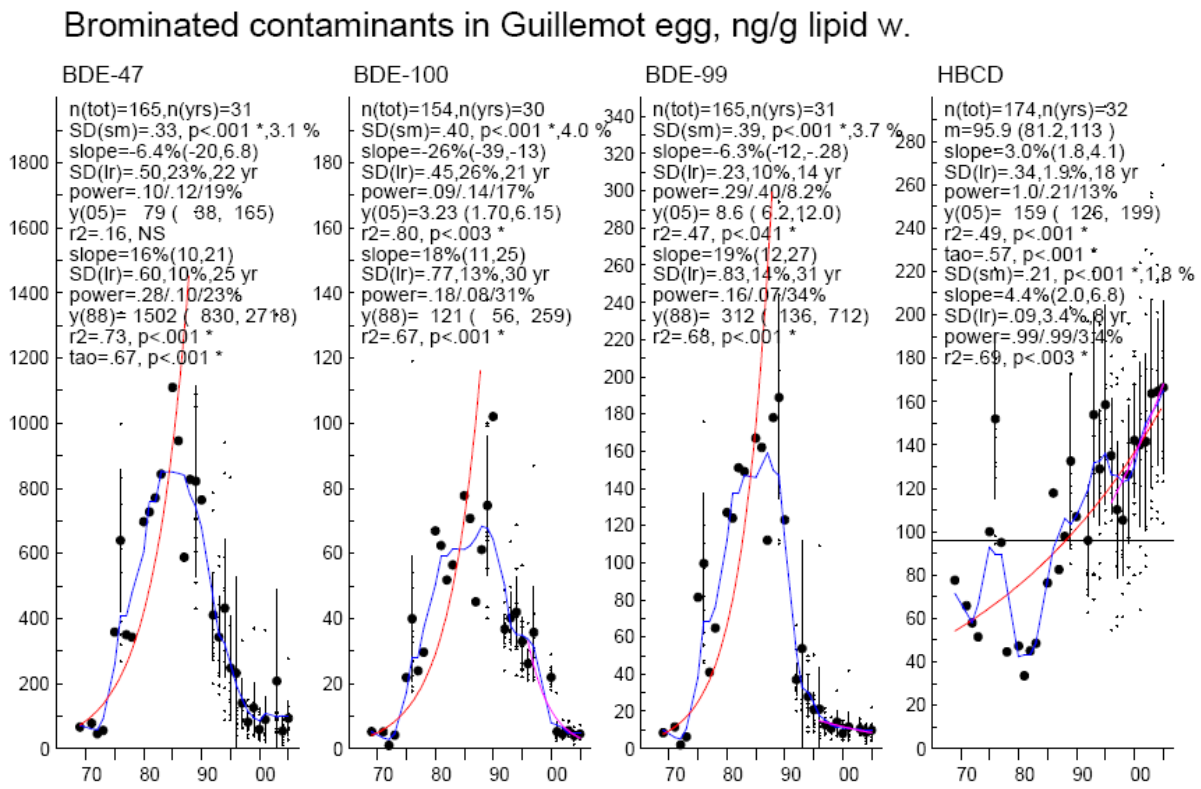


Figure 4-3Concentration of BDE-47, BDE-100, BDE-99 and HBCDD over time in guillemot (*Uria aalge*) eggs in the Baltic Sea (data from Swedish Museum of Natural History, 2007).

Increasing temporal trends have been reported also from other parts of the world (e.g., Kajiwara et al., 2006b; Stapleton et al., 2006).

Although α-HBCDD is present at a low concentration in the commercial product, it is in general found at the highest concentrations of the three diastereomers in biota (e.g., de Boer et al., 2002; Schlabach et al., 2002; Gerecke et al., 2003; Tomy et al., 2004a; Janák et al., 2005a; Zegers et al., 2005; Law et al., 2006b; Ueno et al., 2006). Furthermore, α-HBCDD is not a generally dominant species in abiotic samples. Several factors may lead to the dominance of α-HBCDD in biota. Firstly, the mass-transfer limitations are lowest for α-HBCDD of the three diastereomers based on its higher water solubility and lower logKow -value. These properties make it more readily available for uptake from environmental compartments and from gastrointestinal tract. Secondly, α-HBCDD seems to have the lowest potential to be metabolised based on *in vitro* tests with mammals and fish (Zegers et al., 2005; Janák et al., 2005b). The simulation degradation tests of Davis et al. (2004) also indicate, that α-HBCDD would be degraded slowest of the three diastereomers. Additionally, bioisomerisation of γ-HBCDD and β-HBCDD to α-HBCDD has been observed to occur in fish (Law et al., 2006a).

4.3.3 Terrestrial bioaccumulation

4.3.4 Summary and discussion of bioaccumulation

Reliable experimental BCFs from two flow-through bioconcentration tests with fish are available. As a representative BCF-value 18 100 was chosen in the EU risk assessment (EU RAR, 2008). Furthermore, a large set of measured data in biota in the field show, that HBCDD is biomagnified in the environment. Increasing concentrations of HBCDD have been found in several time series of, e.g. birds and marine mammals. No diastereomer specific BCFs are available. Despite being present in commercial HBCDD at the lowest concentration, α-HBCDD generally has the highest concentration of the three main diastereomers in biota. However, several reasons may have lead to this difference in diastereomeric distribution in biota compared to technical product. It is concluded, that HBCDD has a very high bioaccumulation potential.

4.4 Secondary poisoning

Due to accumulation of HBCDD in organisms such as fish (BCF = 18 000) fish feeding mammals and birds are exposed to HBCDD. In addition, predators feeding on marine mammals and birds are another group of animals that may be highly exposed to HBCDD. In line with the TGD it is acknowledged that a regional assessment of secondary poisoning for PBT substances can not be done with any certainty. A strict comparison of measured levels in fish and marine mammals indicate that they are mostly below the estimated PNEC for secondary poisoning of 5 mg HBCDD/kg wwt food. It must be pointed out though, that this PNEC is uncertain. However, in the vicinity of point sources such as the river Skerne in UK and the river Scheldt basin in Belgium HBCDD concentrations higher than 5 mg/kg wwt have been measured in eel and brown trout. The highest measured concentration in fish is 9.4 mg/kg wwt (eel in river Skerne). Also in marine mammals concentrations higher than the PNEC has been measured, the highest being 6.4 mg/kg wwt whole body weight in harbour porpoise from the UK.

To conclude, even though the PNEC for secondary poisoning is uncertain there is a potential for secondary poisoning of e.g., predatory mammals and birds as indicated by measured concentrations in fish and mammals being higher than the PNEC.

5 HUMAN HEALTH HAZARD ASSESSMENT

5.1 Toxicokinetics (absorption, metabolism, distribution and elimination)

Information on the toxicokinetics of HBCDD is limited.

Properly dissolved HBCDD is probably readily absorbed from the gastro-intestinal tract with the highest concentrations subsequently reached in adipose tissue and muscle, followed by liver and to a much lower extent the lung, kidney, blood and brain, in rodents. Although the exact extent of oral absorption is unknown, it is probably in the order of 50-100 %. However, 100% oral absorption is assumed for derivation of DNEL. Higher concentrations are achieved in females than in males, but the substance is accumulating in both sexes. Among the three diastereoisomers of HBCDD present in the technical product, the accumulation of the α-diastereomer is much higher than of the others, especially at higher exposure levels. The time to reach steady-state seems to be in the order of months. HBCDD can be metabolised, and three polar metabolites as well as unextractable substance in faeces and urine have been detected after exposure to γ-HBCDD, although the overall extent of metabolism of technical HBCDD is unknown. In environmental biodegradation studies, the only biodegradation pathway so far identified is a step-wise reductive debromination of HBCDD, via tetrabromocyclododecene and dibromocyclododecadiene, to 1,5,9-cyclododecatriene, which seemed to be the final degradation product in the environmental samples.

For an initial period of 3 days post dosing of rats, elimination of HBCDD and its metabolites occurs mainly via faeces with a minor part excreted in urine. Elimination from body fat appears to be markedly slower than from other tissues, with an elimination half-life of the three diastereoisomers possibly being in the order of weeks to months.

Data on absorption by inhalation exposure is lacking. However, the efficiency of inhalation uptake can be considered equal to uptake by the oral route (100 %). A value of 4 % is assumed to be applicable for uptake of powder by the dermal route.

5.2 Acute toxicity

5.2.1 Acute toxicity: oral

The minimum lethal dose is greater than 20 g/kg

5.2.2 Acute toxicity: inhalation

The minimum lethal dose is greater than 200 mg/l

5.2.3 Acute toxicity: dermal

The minimum lethal dose is greater than 20 g/kg

5.2.4 Acute toxicity: other routes

5.2.5 Summary and discussion of acute toxicity

The data available on acute toxicity do not suggest a classification of HBCDD according to EU criteria.

5.3 Irritation

The substance is mildly irritating to the eye, but should not be classified as an eye irritant according to EU criteria. HBCDD is not irritating to skin.

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5.4 Corrosivity

The substance is not corrosive to skin.

5.5 Sensitisation

Available data indicates that at least certain commercial (Japanese) brands of HBCDD are potential skin sensitizers. However, the HBCDD available on the EU-market has been negative in both a Magnuson-Kligman test and in a Local Lymph Node assay, leading to the conclusion that there is no concern for sensitisation for the HBCDD occurring in the EU.

No information is available on respiratory sensitisation.

5.6 Repeated dose toxicity

5.6.1 Repeated dose toxicity: oral

Results from several studies on repeated dose toxicity are available
The most recent conducted study is a 28 days study (van der Ven *et al.*, 2006), using a benchmark model design and oral administration of dissolved HBCDD. The study mainly shows effects on the liver, the thyroid, and the pituitary. A NOAEL/BMD-L of 22.9 mg/kg/day for liver weight increase is deduced from this study. The earlier conducted studies show similar effects and a LOEAL of 100mg/kg/day is deduced from those studies.
Overall, a NOAEL/BMD-L of 22 mg/kg/day for liver weight is deduced for repeated dose toxicity.
It has been suggested that the liver weight increase is caused by hepatic enzyme induction, as indicated by histopathology (proliferation of SER) and induced hepatic enzyme activities/mRNA/protein. There is no consistent difference in sensitivity towards hepatic enzyme induction between males and females. However, it is noteworthy that in spite of similar enzyme induction in females and males, the concentration of HBCDD was higher in females than in males, indicating little relationship between enzyme induction and accumulation of HBCDD in the animals. Enzyme induction is clearly involved, and is likely the most important reason for the liver weight increase, but it cannot be ruled out that other mechanisms also are involved.
With regard to effects on the thyroid system, the studies have shown either no effects, effects only in females, or effects in both sexes. However, in the early studies, the thyroid system was not

studied that thoroughly. The latest studies showed effects on the thyroid weight (increases) only in females. In contrast, Chengelis (2001) indicated decreased serum T4 and increased serum TSH in both sexes, whereas (van der Ven *et al.*, 2006) only observed effects in females.
The mechanism for the thyroid effects is not clear but is thoroughly discussed in the EU RAR.

Table5-1 Summary of findings related to the liver and the thyroid system in the RdT studies.

Studies on undissolved HBCDD (particles in suspension)		
Study	Liver effects	Thyroid effects
28-days (Zeller and Kirsch 1969)	Liver weight increase as from the lowest dose (940 mg/kg/day) in both sexes	Thyroid hyperplasia as from the lowest dose (940 mg/kg/day) in both sexes
90-days (Zeller and Kirsch 1970)	Liver weight increases as from the lowest dose (120 mg/kg/day) in both sexes.	No histopathological effects were reported.
28-days (Chengelis 1997)	Liver weight increase in females as from the lowest dose (125 mg/kg/day) and in males from the mid dose (350 mg/kg/day).	No histological effects were observed in the thyroids in either sex.
90-days (Chengelis 2001)	Liver weight increase as from the lowest dose (100 mg/kg/day) in both sexes.	Thyroid weight was increased from mid dose in females (300 mg/kg/day), but not in males. Serum T4 was decreased and TSH increased in all dose groups of both sexes.
Studies on dissolved HBCDD (using a Benchmark method)		
Study	Liver effects	Thyroid effects
28-days (van der Ven *et al.*, 2006)	Liver weight increase only in females; BMD-L 23 mg/kg/day BMD-L (mg/kg/day) for; hepatic T4-conjugation - females 4 - males 0.1 (uncertain) Hepatic CYP2B-activity (PROD) was only induced in males (as from 10 mg/kg/day), whereas mRNA and protein for CYP2B was increased also in females. Hepatic CYP3A4-induktion (LBD) was only observed in females (as from 10 mg/kg/day).	Thyroid weight effects only in females. BMD-L for weight increase 2 mg/kg/day BMD-L for decreased serum T4 55 mg/kg/day

5.6.2 Repeated dose toxicity: inhalation

No data are available

5.6.3 Repeated dose toxicity: dermal

No data are available

5.6.4 Other relevant information

5.6.5 Summary and discussion of repeated dose toxicity:

The data available on repeated dose toxicity do not suggest a classification of HBCDD according to EU criteria.

5.7 Mutagenicity

HBCDD did not induce mutations in the Ames test, and was negative in both an *in vitro* chromosome aberration test and an *in vivo* micronucleus test. Therefore, it can be concluded that HBCDD lacks significant genotoxic potential *in vitro* as well as *in vivo*.

5.7.1 In vitro data

5.7.2 In vivo data

5.7.3 Human data

5.7.4 Other relevant information

5.7.5 Summary and discussion of mutagenicity

The data available on mutagenicity do not suggest a classification of HBCDD according to EU criteria.

5.8 Carcinogenicity

5.8.1 Carcinogenicity: oral

Data from one lifetime bioassay with oral exposure for 18 month in mice, is available. This study is not reported according to current guideline, it is only available as a study summary lacking significant details.

The main change in this test was liver lesions such as hepatocytic swelling; degeneration, necrosis, vacuole formation and fatty infiltration in the experimental groups in comparison with the control group. Such changes might indicate induction of liver enzymes, but there was a poor correlation between these effects and the dosage. The changes in the liver are difficult to interpret due to lack

of description of severity and absence of a clear-cut dose-response relationship, but it supports that the liver is an HBCDD target organ. An increased frequency of liver carcinomas is suggested in females. The incidences of total liver tumours are, nevertheless, within the normal range observed for this mouse strain.

5.8.2 Carcinogenicity: inhalation

No data are available

5.8.3 Carcinogenicity: dermal

No data are available

5.8.4 Carcinogenicity: human data

No data are available

5.8.5 Other relevant information

5.8.6 Summary and discussion of carcinogenicity

The data available on carcinogenicity do not suggest a classification of HBCDD according to EU criteria.

5.9 Toxicity for reproduction

5.9.1 Effects on fertility

Data from one two-generation reproductive toxicity study in rats is available (Ema, *in press*). The study was conducted according to OECD guideline 416 and in accordance with the principles for good laboratory practice.

The main effects seen were a dose-dependent decrease (8-14%) in fertility index in both generations. A significantly reduced number of primordial follicles in the mid and high dose groups was also evident (30 %, only measured in F1). In addition, a high and dose-dependent pup mortality during lactation was observed in the F2 generation (increased by 35 % in the high dose group and 15 % in the mid dose group), although only being statistically significant in the high dose group. There were indications of effects on liver and thyroid weights. The effect of HBCDD on the primordial follicles could be an indication of toxic effects on development as well as on fertility. However, the reduced number of follicles can cause reduced fertility, and thus also be considered a fertility endpoint.

A low number of follicles and ripening follicles in the ovaries were reported at high doses in one old 28 days study (Zeller and Kirch 1969), and this finding could possibly support the effects on primordial follicles and the decrease in fertility index seen in the Ema study.

A NOAEL of 10 mg/kg/day is deduced from the two-generation reproductive toxicity study.

5.9.2 Developmental toxicity

Two developmental toxicity studies, one according to OECD Guideline 414, have failed to demonstrate any fetotoxicity, teratogenic potential or adverse effects from HBCDD on development of rats.

Developmental neurotoxicity

One study (Eriksson et al, 2006) indicates that neonatal HBCDD exposure may cause developmental neurotoxic effects as illustrated by statistically significant changes in spontaneous behaviour, learning and memory defects. An indicative LOAEL of 0.9 mg/kg/day can be deduced from this study, but the results need to be confirmed by other laboratories before any conclusions can be drawn.

5.9.3 Human data

No data are available

5.9.4 Other relevant information

5.9.5 Summary and discussion of reproductive toxicity

The data available on reproductive toxicity suggest a classification; Repr Cat 3; R62 (Possible risk of impaired fertility) for HBCDD, according to EU criteria.

5.10 Other effects

5.11 Derivation of DNEL(s) or other quantitative or qualitative measure for dose response

5.11.1 Overview of typical dose descriptors for all endpoints

The majority of studies with oral administration of the test substance have been performed with HBCDD-particles suspended in oil. The mean particle size of the tested technical HBCDD (composite from three manufacturers) is presently reported to be 0.14 mm (10 % <3.6 μm, 10 % >280 μm). The absorption may have been low (especially at high dose levels) in these studies, and the internal dose is likely to have been lower than the administered dose. These studies may thus underestimate the toxicity of HBCDD.

Van der Ven, 2006 is the only study available where the rats were exposed to properly dissolved HBCDD.

HBCDD has been shown to cause increased liver weight in all repeated dose toxicity studies. It is probably caused by hepatic enzyme induction, as indicated by histopathology and induced hepatic enzyme activities/mRNA/protein. In the EU RAR , a NOAEL of 22.9 mg/kg/day was identified for liver weight increase (van der Ven, 2006). Effects on the thyroid system have also been identified. The mechanism for the thyroid effects is not clear but is in depth discussed in the EU RAR.

Effects on fertility parameters are evident in a two generation reproductive toxicity study (Ema, in press). The main effects seen were a dose-dependent decrease (8-14%) in fertility index in both generations. A significantly reduced number of primordial follicles in the mid and high dose groups

was also evident (30 %, only measured in F1). In addition, a high and dose-dependent pup mortality during lactation was observed in the F2 generation (increased by 35 % in the high dose group and 15 % in the mid dose group), although only being statistically significant in the high dose group.

Table 5-2Studies showing the critical endpoints and NOAELs for HBCDD

Species	**Study Protocol; Quality**	**Effects observed at LOAEL**	**LOAEL** (mg/kg bw/day)	**NOAEL/BMD-L** (mg/kg bw/day)	**Ref.**
Repeated-dose toxicity					
Rat, Wistar, males and females	Diet, 28 days; OECD Guideline 407 with focus on effects on the thyroid hormone axis, haematology and bone parameters.	**Repeated dose toxicity**, increased liver weight		22.9	van der Ven, et al. 2006
Reproductive toxicity					
Rat, Crl:CD(SD) rats males and females	Diet, 2-Generation reproductive toxicity study, GLP, OECD Guideline 416	Reproductive toxicity/fertility, dose-dependent decrease in fertility-index and a reduced number of primordial follicles.	100	10	Ema, in press

5.11.2 Correction of dose descriptors if needed (for example route-to-route extrapolation)

An oral absorption of 100 % is deduced for *dissolved* HBCDD. Data on absorption by inhalation exposure is lacking, but for the purpose of modelling, the efficiency of inhalation uptake can be considered equal to uptake by the oral route (100 %). A value of 4 % is assumed to be applicable for uptake of powder by the dermal route. Depending on the particle size occurring in the exposure situation, a value of 2 % is used when granules are used.

The oral NOAELs can be transformed using route to route extrapolation into NOAELs also for inhalation and dermal exposure, but that has not been done in the context of this Annex XV SVHC dossier.

No recalculation of the NOAEL/BMD-Ls is needed as the exposure in a couple of key studies occurs during life stages with an assumed oral absorption of 100%.

5.11.3 Application of assessment factors

In the EU RAR on HBCDD, total assessment factors for the fertility end-point of 50 to 100 have been used. For the general population, a total factor of 100 was made up of a factor 10 for intraspecies differences, a factor of 10 for interspecies differences. The factor for interspecies

differences was made up of a factor of 4 representing the difference in caloric demand between rats and humans and a factor of 2.5 for remaining uncertainties (as the mechanism of action is unknown, it cannot be excluded that there are differences in sensitivity between rats and humans). Concerning workers, a factor of 5 for intraspecies differences and the same factor 10 for interspecies were used.

Concerning assessment factors for repeated dose toxicity a total factor of 20 to 40 have been used. For the general population, a total factor of 40 was made up of a factor 10 for intraspecies differences and a factor of 4 for interspecies differences, representing the difference in caloric demand between rats and humans. As enzyme induction is the present explanation of the liver and thyroid weight increase and that humans are not expected to be more sensitive than rats to the enzyme induction/liver weight increase, no factor for differences in sensitivity is thus needed.
The overall AFs are given in Table 5-3 below for the general population and workers. The table also gives the oral DNELs for the different endpoints.

The overall AFs are given in

Table 5-3Derivation of oral DNELs for the different endpoints

Endpoint	**oral dose descriptor[1] (mg/kg/day)**	**Overall AF applied**		**Endpoint specific oral DNEL**	
		General population	**Workers**	**General population (mg/kg/day)**	**Workers (mg/kg/day)**
RDT liver	22.9	40	20	0.6	1.1
Fertility	10	100	50	0.1	0.2

1: no local effects are of relevance for HBCDD

5.11.4 Selection/ identification of the critical DNEL(s)/ the leading health effect

In this Annex XV SVHC dossier, only oral DNELs have been set. Two different subpolulations have been assessed, and the critical DNELs are presented in the table Table 5-4 below.

Table 5-4Summary of the leading population-specific oral DNELs

Population	**End-point(s)**	**Oral DNEL**
Workers	fertility / developmental toxicity	0.2 mg/kg/day
General population (consumers and humans exposed via the environment)	fertility / developmental toxicity	0.1 mg/kg/day

6 HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Not relevant for this type of dossier.

7 ENVIRONMENTAL HAZARD ASSESSMENT

The results of ecotoxicity tests, which have been considered reliable by EU RAR, 2008, are presented in Table 7.1.

Table7-1 Acute and chronic ecotoxicity data, which are considered reliable according to EU RAR (2008)

Compartment/Species	Method	Results	Remark and reference
AQUATIC COMPARTMENT			
FISH			
Onchorhyncus mykiss	OECD 203 and TSCA 40/797/1400, and ASTM Standard E729-88a	No mortalities or other effects around 2.5 µg/l.	Graves and Swigert (1997a)
Onchorhyncus mykiss	Flow-through OECD 210 and OPPTS 850.1400	NOEC: Hatching success ≥3.7 µg/l Swim-up ≥3.7 µg/l Larvae and fry survival ≥3.7 µg/l Growth ≥3.7 µg/l	Drottar et al. (2001)
INVERTEBRATES			
Daphnia magna	OECD 202. Static immobilisation test, and TSCA 40/797/1300, and ASTM Standard E729-88a	48 h EC_{50} >3.2 µg/l	Graves and Swigert (1997b)
Daphnia magna	TSCA , OECD Flow through 21 day test.	NOEC 3.1 µg/l LOEC length 5.6 µg/l	Drottar and Krueger (1998)
ALGAE			
Selenastrum capricornutum	OECD 201 and TSCA40/797/1050	96 h EC_{50} >2.5 µg/l	Roberts and Swigert (1997)
Skeletonema costatum *Thallassiosira pseudonana* Chlorella sp.	Marine algal bioassay method, different marine growth media	72 h EC_{50} = 9 µg/l (lowest value) 72 h EC_{50} = 40 µg/l (lowest value) 96h EC_{50} >water solubility	Walsh et al. (1987) Not according to guidelines, results only used as supportive
Skeletonema costatum	OECD 201, ISO 10253:1995 and EU Directive 92/69/EEC – Method C.3. One test concentration at the limit of respective water solubilites of each diastereomer.	NOEC <40.6 µg/l EC_{50} >40.6	Desjardins et al. (2004)
Skeletonema costatum	OECD 201. EC50 obtained from a limit test with one test concentration (54.5 µg/l) at the limit of respective water solubilites of each diastereomer.	NOEC >10 µg/l EC_{50} = 52 µg/l	Desjardins et al. (2005)
SEWAGE TREATMENT PLANT, MICRO-ORGANISMS			
Acivated sludge	Respiration inhibition OECD 209	EC_{50} = 15 mg/l	Limit test with one test concentration, EC_{50} is an estimated value. Schaefer and Siddiqui (2003)
SEDIMENT COMPARTMENT			
INVERTEBRATES			
Hyalella azteca	Sediment toxicity test 28-day	LOEC >1000 mg/kg	Thomas et al.

Compartment/Species	Method	Results	Remark and reference
(Amphipod)	exposure period under flow-through conditions.	dw of sediment NOEC 1000 mg/kg dw of sediment.	(2003b)
Lumbriculus variegatus (Worm)	28-day sediment bioassay	LOEC = 28.7 mg/kg dw NOEC = 3.1 mg/kg dw Normalized: NOEC = 8.61 mg/kg dw	Oetken et al. (2001)
Chironomus riparius (Mosquito)	28-day sediment bioassay Egg production of F generation	LOEC = 159 mg/kg dw NOEC = 13.6 mg/kg dw Normalized: NOEC = 37.8 mg/kg dw	Oetken et al. (2001)
TERRESTRIAL COMPARTMENT			
PLANTS			
Plants: corn (*Zea mays*), cucumber (*Cucumis sativa*), onion (*Allium cepa*), ryegrass, (*Lolium perenne*), soybean (*Glycine max*), and tomato (*Lycopersicon esculentum*)	Seedling emergence, suvival, height 21 days OECD 308 (proposal for revision), 850.4100 and 850.4225 (public drafts)	NOEC >5000 mg/kg dry soil	Porch et al. (2002)
INVERTEBRATES			
Eisenia fetida (Earthworm)	Survival and reproduction, 56 days OECD prosal and 207 and OPPTS 850.6200	NOEC 128 mg/kg dry soil Normalized: NOEC 59 mg/kg dry soil (EC_{50} 771 mg/kg dry soil)	Aufderheide et al. (2003)

7.1 Aquatic compartment (including sediment)

7.1.1 Toxicity test results

7.1.1.1 Fish

Short-term toxicity to fish

The acute toxicity of HBCDD to rainbow trout, *Oncorhynchus mykiss*, was studied in a 96 h flow through test by Graves and Swigert, 1997b.

The acute toxicity of the substance was studied in five nominal test concentrations (1.5, 2.2, 3.2, 4.6 and 6.8 µg HBCDD/l) and compared to control and solvent control.

No mortalities or other effects were observed throughout the test. The results indicate that HBCDD is not acutely toxic to fish at a nominal concentration of about 6.8 µg/l (mean measured concentration 2.5 µg/l).

Long-term toxicity to fish

An early life-stage toxicity test was performed with the rainbow trout (*Oncorhynchus mykiss*) (Drottar *et al.*, 2001). Endpoints examined were: hatching success, time to hatch, time for larvae to swim-up, and post-hatch growth and survival.

The test was performed with newly-fertilised eggs. The nominal test concentrations were 0.43, 0.85, 1.7, 3.4 and 6.8 µg/l. Test concentrations were measured every 7th day from day 0 to day 84 and also day 88 resulting in the following mean measured test concentrations: 0.25, 0.47, 0.83, 1.8, and 3.7 µg/l. A negative control and a solvent control were also run. The total exposure period was 88 days, including a 27-day hatching period and a 61-day post-hatch period.

The hatching success ≥83 % in the exposed groups was not statistically different ($p > 0.05$) from the pooled controls. There were no statistically significant reductions in the numbers of fish swimming up in any HBCDD treatment group compared to the pooled control groups. There was no significant difference in survival between the different groups. There was no significant difference in growth between the different groups.

Hence, NOEC was ≥3.7 µg/l.

7.1.1.2 Aquatic invertebrates

Short-term toxicity to aquatic invertebrates

An acute flow through toxicity study on *Daphnia magna* (neonates) was performed with duplicates for each test concentration with 10 animals per replicate, at 20±2 °C (Graves and Swigert, 1997a).

The nominal HBCDD concentrations were: 1.5, 2.2, 3.2, 4.6, and 6.8 µg/1, solvent control, and negative (dilution water) control. The measured test concentrations day 0 were: 2.17/2.26, 1.74/1.85, 2.16/1.55, 2.73/2.47, 2.99/3.33 µg/l; and at day 2 they were: 2.48/2.50, 1.75/1.70, 2.48/2.27, 1.55, 3.41 µg/l.

The EC_{50} (48h) was >3.2 µg/l, which is the mean of the measured values at the highest nominal test concentration.

Long-term toxicity to aquatic invertebrates

A flow-through 21 day life-cycle toxicity test was performed with the cladoceran *Daphnia magna* (Drottar and Krueger, 1998). Survival of the first and second generation daphnids, the number of young produced per reproductive day, and the length and dry weight of surviving first-generation daphnids were evaluated.

The nominal test concentrations were: 0.85, 1.7, 3.4, 6.8 and 13.6 µg HBCDD/l, solvent control, and negative (dilution water) control. Test concentrations were measured day 0, 7, 14 and 21 resulting in the following mean measured test concentrations (range): negative control <LOQ, solvent control <LOQ, 0.87 (0.72-1.02), 1.6 (1.34-1.85), 3.1 (2.69-3.63), 5.6 (4.75-6.38), and 11 (9.82-12.3) µg/l.

Daphnids exposed to 11 µg/l for 21 days had statistically significant reduced lengths, dry weight and fewer young. Daphnids exposed to 5.6 µg/l for 21 days had statistically significant reduced mean lengths. The used test concentrations are below the maximum water solubility of HBCDD. Thus, the LOEC was determined to 5.6 µg/l.

No statistical effects on survival, reproduction or growth were observed in *Daphnia magna* exposed for 21 days to 3.1 µg/l, and hence, the NOEC was 3.1 µg/l.

7.1.1.3 Algae and aquatic plants

Data are available from four reliable algal growth inhibition studies.

The toxicity of HBCDD to the freshwater alga, *Selenastrum capricornutum,* was studied in a static 96 h growth inhibition test (Roberts and Swigert, 1997).

The effects on growth rate and biomass were studied in five nominal test concentrations (1.5, 2.2, 3.2, 4.6 and 6.8 µg HBCDD/l). The measured test concentrations (corrected for a mean procedural recovery of 113 %) on day 0 were: 1.30, 2.25, 3.38, 4.28 and 6.44 µg/l, and on day 4 (in the abiotic test solution): <0.571 (detection limit), 1.20, 1.90, 1.64 and 2.47 µg/l.

No effects were seen at the highest tested concentration, i.e NOEC =2.5 µg/l (day 4). Thus, the 72-hour EC_{50} is >2.5 µg/l and the LOEC is >2.5 µg/l.

The algal growth inhibition of HBCDD was also studied in six marine media (Walsh *et al.*, 1987). The studied test organisms were *Skeletonema costatum, Thalassiosira pseudonana* and *Chlorella sp.* Population density was estimated by cell counts on a haemocytometer. Toxicity, EC_{50}, was based upon cell numbers after incubation for 72 hr for *S. costatum* and *T. pseudonana* and for 96 h for *C. sp*.

The EC_{50}s:

*Skeletonema costatum**	EC_{50} (72h) 9-12.2 µg/l
Thalassiosira pseudonana	EC_{50} (72h) 40-380 µg/l
Chlorella sp.	EC_{50} (96h) >1500 µg/l

* Only results from tests in five different media

No NOEC was determined in the test.

There are some question marks regarding the methodology used in this study. For instance, it is not shown that the growth rate is calculated during exponential growth. Since this study appears to deviate from standard methods, the results will only be used as supportive to more recent studies, performed more in line with standard methods.

A 72 hours growth inhibition study was performed with *Skeletonema costatum* (Desjardins *et al.*, 2004). The test was performed to study effects on algal growth of the mixed diastereomers of HBCDD at the limit of their respective water solubility.

Passing saltwater algal medium through a generator column saturated with HBCDD produced the single test concentration (40.6 µg/l). In this way the composition of HBCDD in the saltwater algal medium became 74.6 % α-, 21.5 % β- and 3.97 % γ- diastereomer which is different from that of the technical product.

There was a 10 % inhibition of the growth rate at the measured test concentration of HBCDD 40.6 µg/l. NOEC is <40.6 µg HBCDD/l and EC_{50} >40.6 µg HBCDD/l.

Desjardins *et al.*, 2005 performed a 72 hours study with HBCDD on the marine diatom alga *Skeletonema costatum* using (i) a co-solvent, and (ii) a saturated solution. Both the biomass and the growth rate were derived.

i) Study with a co-solvent

Nominal test concentrations of 0.64, 1.6, 4.0 and 10 µg HBCDD/l, were prepared by diluting a stock solution in dimethylformamide (DMF) with saltwater medium. The analytical results performed at the beginning of the test corresponded to 332, 131, 94 and 108 % of the nominal concentration, respectively. The solvent concentration in the solvent control and treatment groups was 0.1 ml/l.

There were no statistically significant effects at any of the test concentrations. It is probable that the actual test concentrations were almost equal, i.e. about the solubility of γ-HBCDD at all four nominal test concentrations. The other diastereomers would still not have reached significant concentrations at these nominal concentrations of technical HBCDD. Hence, it can be concluded that there are no significant effects at the solubility of γ-HBCDD, and that the NOEC of technical HBCDD in this study was >10 µg/l.

ii) Study at saturated solution

The test was performed to study effects on algal growth of the mixed diastereomers of HBCDD at the limit of their respective water solubility. Only one test concentration was used. The test solution used in this study corresponded to the saturated solution of HBCDD in saltwater. The mean measured HBCDD concentration as a sum of the diastereomers was 54.5 µg/l.

The growth rate inhibition rose during the study and was 17% compared to the column control after 24 hours, 29 % after 48 hours and 51% after 72 hours. The authors of the study used non-linear regression fitting to cumulative normal distribution to calculate EC_{50}. The 72-hr EC_{50} for biomass and growth rate was calculated to be 27 and 52 µg/l respectively. The relevance of calculating an EC_{50} from a study where only one test concentration has been used can be questioned. However, as the growth rate inhibition (0-72 h) was 51% at a test concentration of 54.5 µg HBCDD/l, the calculated EC_{50}-value of 52 µg/l seems adequate. Furthermore, this EC_{50}-value is in line with the result obtained with the saturated solution where EC_{10} was around 40.6 µg/l (Desjardins *et al.*, 2004).

Summary of algal toxicity

Based on the most reliable algal toxicity study (Desjardins *et al.*, 2005) the EC_{50} for algae based on growth rate, is concluded to be 52 µg HBCDD/l. The 72-hr NOEC is determined to be between 10µg/l and 40 µg/l (EU RAR, 2008).

7.1.1.4 Sediment organisms

Two toxicity tests have been performed on the amphipod *Hyalella azteca* (Thomas *et al.*, 2003a-b). Groups of amphipods were exposed to six test concentrations and a control in each study. Eight replicate test compartments were maintained in each treatment and control group, with 10 amphipods in each test compartment. Additional replicates were added in the control group, low and high treatment groups for analytical sampling of water and sediment at day 0, 7 and at the end of the test. Nominal test concentrations were 31, 63, 125, 250 500 and 1000 HBCDD mg/kg of

sediment based on dry weight of sediment. Results of "the analytical replicates" were used to confirm the lowest and the highest test concentration. The results of the studies are based on the nominal test concentrations. The measured endpoints were survival and growth as determined by dry weight measurements.

In both studies LOEC was concluded to be >1000 mg/kg dwt of sediment and NOEC was concluded to be 1000 mg/kg dwt of sediment.

Chronic tests (28 days, static) were also performed with *Lumbriculus variegatus* and *Chironomus riparius* in spiked sediment with an organic matter content of about 1.8 % (Oetken *et al.*, 2001). For *L. variegatus*, different endpoints resulted in different NOECs. The lowest NOEC, 8.6 mg/kg dwt (normalized to standard organic carbon content, *i.e.* 5 %), was obtained for the total number of worms.

Most of the results from the test with *C. riparius* are considered invalid. However, based on the endpoint number of eggs from the F1 generation a NOEC of 13.6 mg/kg dwt was determined for *C. riparius.*

7.1.1.5 Other aquatic organisms

7.1.2 Calculation of Predicted No Effect Concentration (PNEC)

7.1.2.1 PNEC water

Long term studies are in general considered more relevant than short term studies particularly for substances with low water solubility. Reliable long term studies are available for all three trophic levels, but all studies, except the 21d-study with *Daphnia magna*, resulted in larger-than values. None of the larger-than values, is below the 3.1 µg/l NOEC-value for *Daphnia*, except for the LOEC-value of >2.5 µg/l from the 72(96) h growth inhibition test with *Selenastrum capricornutum*. This may indicate that the NOEC for algae could be <3.1 µg/l, i.e. the NOEC-value for *Daphnia*. The lowest NOEC, the 21d-NOEC 3.1 µg/l for *Daphnia magna*, will be used for derivation of PNEC.

According to the revised TGD (Table 20) an assessment factor of 10 can be applied on the lowest NOEC, when reliable NOEC values are available for three trophic levels to derive the $PNEC_{aquatic}$.

Thus, the predicted no effect concentration for the aquatic compartment is 3.1/10 = 0.31 µg/l.

For intermittent releases to the aquatic environment the lowest $L(EC)_{50}$ of at least three short-term tests from three trophic levels is recommended in the revised TGD with applying an assessment factor of 100 for calculation of PNEC. The lowest EC_{50} is the one from the algae growth inhibition test with *Skeletonema costatum,* which is 52 µg/l.

Thus the PNEC for intermittent releases in the water phase is 52/100 = 0.52 µg/l.

7.1.2.2 PNEC sediment

Two toxicity tests have been performed on the amphipod *Hyalella azteca* to determine the effects of sediment-incorporated HBCDD during a 28-day exposure period under flow-through conditions. The results from the two tests were similar and the NOEC for Hyalella was 1000 mg/kg dwt.

Chronic tests (28 days, static) were also performed with *Lumbriculus variegatus* and *Chironomus riparius* in spiked sediment (organic matter content about 1.8 %). The lowest NOEC, 8.6 mg/kg dwt (normalized to standard organic carbon content, *i.e*. 5 %), was obtained for the total number of worms.

Most of the results from the test with *C. riparius* are considered invalid. However, based on the endpoint number of eggs from the F1 generation a NOEC of 13.6 mg/kg dwt was determined for *C. riparius.*

According to the revised TGD an assessment factor can be used on the lowest NOEC for the calculation of $PNEC_{sed}$. In this case there are chronic results from three species with different feeding regimes. Therefore, an assessment factor of 10 is used on the lowest NOEC above (Table 19, revised TGD).

Thus $PNEC_{sed}$, based on chronic test data, is 8.6/10 = 0.86 mg/kg dwt.

7.2 Terrestrial compartment

7.2.1 Toxicity test results

7.2.1.1 Toxicity to soil macro organisms

Acute toxicity

There are no studies on the acute toxicity of HBCDD to earthworms available.

Long term toxicity

A test on the survival and reproduction of earthworm was performed by Aufderheide *et al.*, 2003. The test species was earthworm, *Eisenia fetida* (clitellate adults). Control worms had an initial mean weight of 433.2 mg/worm and the weight of the test worms ranged from 354.0 to 502.6 mg/worm.

The NOEC was estimated to 128 mg HBCDD/kg dry soil and the LOEC to 235 mg HBCDD/kg dw.

In the study the weight fraction of organic matter content was 7.4 %, whereas in a standard soil the organic matter content is 3.4 %, according to the TGD. The NOEC (NOEC = 128 mg HBCDD/kg dry soil) is therefore normalized with the equation 71 in TGD:

$NOEC_{standard} = NOEC_{exp} \times (Fom_{soil(standard)}/Fom_{soil(exp)})$

where Fom is fraction of organic matter.

The normalized NOEC is 59 mg/kg dry soil.

7.2.1.2 Toxicity to terrestrial plants

Porch *et al.*, 2002 performed a seedling emergence test with six plant species.

The test species were corn (*Zea mays*), cucumber (*Cucumis sativa*), onion (*Allium cepa*), ryegrass, (*Lolium perenne*), soybean (*Glycine max*), and tomato (*Lycopersicon esculentum*).

For the onion seedlings there were seemingly a decrease in dry weight and height at 725 mg/kg and above. The decrease was however not significant according to the Dunnett's test. With a post-test for trends it should be possible to show the decreasing trend. Since there is another terrestrial test, survival and reproduction of earthworm, with a lower PNEC there is no need to make any further effort with a trend test.

No NOEC could be determined for the tested plants.

7.2.1.3 Toxicity to soil micro-organisms

A study on the effects of HBCDD on micro-organisms in soil has been performed by Förster, 2007). HBCDD was dissolved in acetone and mixed into quartz sand. After evaporation of the acetone the sand was mixed into sieved (2 mm) field soil (Lufa standard soil 2.3 containing 1.02% organic carbon and 61% sand based on dry weight) that was amended with ground Lucerne meal (5 g/kg soil). The water content of the soil was adjusted to 50% of the maximum water holding capacity. The nominal concentrations of HBCDD were 10.0, 31.6, 100.0, 316.2 and 1000 mg/kg soil dw. Three replicates were set up for each test concentration and control (including a solvent control). The soil was incubated in glass jars in the dark for 28 days at 20 ± 2°C. Soil nitrate concentration was measured day 0 and day 28. The concentration of HBCDD was measured in the 10, 100 and 1000 mg/kg test concentrations and was 104%, 83.1% and 75% of the nominal concentrations, respectively.

No statistically significant differences in nitrate production between the controls and HBCDD treated soil samples were detected. (ANOVA, $p \leq 0.05$).

Thus the NOEC from this study was ≥750 mg HBCDD/kg dw.

7.2.1.4 Toxicity to other terrestrial organisms

Toxicity to birds

Toxicity to other above ground organisms

7.2.2 Calculation of Predicted No Effect Concentration (PNEC_soil)

There are studies on terrestrial organisms from three trophic levels available. Thus an assessment factor of 10 can be applied (revised TGD Table 20). The normalized NOEC value for reproduction of earthworms is used to calculate the PNEC for the terrestrial environment.

Applying an assessment factor of 10 results in a predicted no effect concentration for the terrestrial compartment $PNEC_{soil}$ of 59/10 = 5.9 mg/kg dry soil.

7.3 Atmospheric compartment

There are no effect data available for the atmospheric environment and therefore it is not possible to calculate a $PNEC_{air}$.

7.4 Microbiological activity in sewage treatment systems

7.4.1 Toxicity to aquatic micro-organisms

An oxygen consumption test using *Pseudomonas putida* was carried out by Siebel-Sauer (1990). The nominal test concentrations were between 1250-10000 mg/l. No toxic effects compared to control were observed at the maximum nominal concentration of 10000 mg/l. The results from this study indicate that HBCDD has a low toxicity to micro-organisms.

However, the nominal test concentrations were much above the water solubility of HBCDD. Furthermore, the study was shortly described which makes the reliability difficult to assess. According to the TGD tests on individual bacterial populations are considered less relevant. It has therefore not been considered relevant to base a $PNEC_{STP}$ on the results from this study.

An activated sludge respiration inhibition test has been performed (Schaefer and Siddiqui, 2003).

The test substance was a composite sample from three manufacturers of hexabromocyclododecane and had a purity of 95.86 %. The activated sludge used in the test was from a wastewater treatment plant that receives mainly domestic sewage. The test was carried out at 20-21 °C and the sludge used had a total suspended solids content of 4213 mg/l and a pH of 7.8. The test substance, HBCDD, was dosed at a limit concentration of 15 mg/l being tested in triplicate. Two controls were run and a reference substance (3,5-dichlorophenol) was also tested at concentrations of 3, 15 and 50 mg/l. The respiration rate after 3 hours in the three replicate HBCDD treatments were 42.4, 41.0 and 40.0 mg O_2/l/hour, which was equivalent to approximately 29.1 % inhibition when compared to the controls. Thus only an approximate EC_{30} value of 15 mg/l can be estimated.

The study is considered reliable. However, due to the use of a limit concentration no inhibition curve can be obtained and a true EC_{50} cannot be calculated. The test concentration 15 mg HBCDD/l activated sludge is above the water solubility of HBCDD. Activated sludge is however not pure water and the test concentration is therefore considered acceptable.

The EC_{30} of 15 mg/l will be used for calculation of PNEC.

7.4.2 PNEC for sewage treatment plant

The EC_{30} obtained at 15 mg/l in the respiration inhibition test (Schaefer and Siddiqui, 2003) discussed above, is taken as an estimate for the EC_{50} for the PNEC derivation. When deriving a PNEC for micro-organisms from an EC_{50} value an assessment factor of 100 should be used according to the revised TGD. Thus $PNEC_{STP}$ is 0.15 mg/l.

7.5 Calculation of Predicted No Effect Concentration for secondary poisoning (PNEC_oral)

For the assessment of secondary poisoning, the results have to be expressed as the highest concentration in food causing no effects. Equations and factors for the conversion from NOAEL to NOEC are given by TGD. In addition an extra assessment factor, accounting for interspecies

variation, lab-to-field extrapolation and acute/subchronic to chronic extrapolation should be applied to derive a PNEC. According to the human health risk assessment, two effects could be relevant for the derivation of this PNEC, i.e., repeated dose toxicity on liver and the thyroid with an oral NOAEL of 22.9 mg/kg/day from a 28 days study in rats, and reproductive toxicity with a diet NOAEC of 150 ppm HBCDD dry weight (corresponding to a dose of 10 mg/kg/day).

According to TGD, an assessment factor of 300 can be applied to the NOEC for a 28 day repeated dose test on mammalian species. However, in this case a factor of 30 is chosen because there is no need to use an assessment factor for subchronic to chronic extrapolation. In the human risk assessment a factor of 1 is chosen to account for the differences between the 28 day study and chronic exposure. The reason for this is that there is no indication that the liver weight will increase more with time of exposure (similar liver weight increases are observed after 28 days and 90 days exposure). In addition, if assuming that enzyme induction is the primary event triggering the other effects, enzyme induction is neither likely to increase with time. There is some uncertainty as to whether the thyroid effects could become more severe after chronic exposure, but on balance, it is decided not to use an extra assessment factor for subchronic to chronic extrapolation.

A 2-generation study has recently been performed according to OECD TG 416 (Ema et al, 2008). HBCDD was administered via the diet by mixing HBCDD-particles with ground dry feed, at concentrations of 150, 1.500, and 15.000 ppm (dry weight). Because of the dosing of HBCDD particles, with the bioavailability likely being dependent on particle size and dose, there is some uncertainty regarding the actual systemic doses obtained especially in the higher dose groups. A significantly reduced number of primordial follicles in the mid and high dose groups was evident (30 %, only measured in F1). A dose-dependent decrease (8-14%) in fertility index was indicated in both generations, although statistically significant only in F0. In addition, a high and dose-dependent pup mortality during lactation was observed in the F2 generation (increased by 35 % in the high dose group and 15 % in the mid dose group), although only being statistically significant in the high dose group. Overall, a NOAEL of 150 ppm dry weight (10 mg/kg/day) can be deduced based on ecologically relevant effects at 1.500 ppm. As no assessment factor is needed for duration correction when the data come from a 2-generation study, the total assessment factor to be used is 30.

As reproductive toxicity may be more ecologically relevant than liver and thyroid effects, and also give the lowest NOAEC/NOAEL, the PNEC will be calculated based on the reproductive toxicity NOAEC of 150 ppm.

However, the derived PNEC is considered to be uncertain. There are indications that HBCDD may have developmental neurotoxicity effects at lower exposure levels than those cited above, although this needs to be confirmed. Consequently, the results from the neurotoxicity study cannot be used to derive a PNEC for secondary poisoning. The uncertainties in the mammalian toxicity database are also acknowledged in the human health risk characterization where a conclusion (i) on hold (awaiting results from ongoing studies) is drawn with regards to the need for a developmental neurotoxicity study in rodents.

7.6 Conclusion on the environmental classification and labelling

The proposed classification for the environment is:

N; R50-53 Very toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.

Concentration limits:

According to the proposal on specific concentration limits for very toxic substances (ECBI/65/99 Add.10), the reported L(E)C50 range of 10-100 µg/l will give rise to the following concentration limits of preparations:

Concentration limits of substance	*Classification of preparation*
C ≥2.5 %	N; R50-53
C ≥0.25 %	N; R51-53
C ≥0.025 %	R52-53

The proposal is based on the toxic effects seen in a 72-hour study on the marine algae *Skeletonema costatum* (EC_{50} 52 µg/l), the lack of biodegradation seen in a standard test and the very high bioconcentration factor (18 100) determined in a BCF study on fish. The proposed classification is supported by the results from a 21-day life cycle test on *Daphnia magna,* in which the LOEC, based on reduced mean lengths, was determined to 5.6 µg/l. The proposed classification is further supported by the results from two other 72-hour studies on the marine algae *Skeletonema costatum*: In one study an EC_{50} of about 10 µg/l is obtained, however this study is older and appears to deviate from standard methods and therefore the results are only used as supportive to the result above. In the other study a NOEC <40.6 µg/l and EC_{50} >40.6 µg/l is obtained for HBCDD.

8 PBT, VPVB AND EQUIVALENT LEVEL OF CONCERN ASSESSMENT

8.1 Comparison with criteria from annex XIII

Persistence: Hexabromocyclododecane (HBCDD) fulfils the P-criterion. Based on a standard degradation simulation study, HBCDD seems to be persistent in aerobic soil. No firm conclusion can be drawn solely from the performed simulation degradation studies regarding whether or not HBCDD fulfils the P-criterion for sediment. The assessment is complicated by the fact that available data indicate that the different diastereomers have different degradability. For α-HBCDD, which seems to be the least degradable, an aerobic DT_{50} of approximately 210 days in sediment at 12°C was determined, which is above the P-criterion of 120 days. For γ-HBCDD the available data indicate very different half-lives depending on test concentration. When tested at a concentration similar to what is measured close to polluted areas, the DT_{50} was 190 days (12°C).

The measured data available from dated sediment cores indicate, that HBCDD has degraded in these sediments more slowly than what would be expected based on some of the available experimental sediment degradation half-lives. Furthermore, HBCDD is found to be ubiquitously present in remote areas in abiotic samples and biota providing evidence, that the substance is persistent in the environment. Also the temporally increasing concentrations found in biota support the picture of HBCDD as a persistent substance.

Bioaccumulation: HBCDD meets the vB criterion based on reliable experimental BCFs from two flow-through bioconcentration tests with fish. A BCF of 18 100 was chosen as a representative value in the EU risk assessment (European Commission, 2007). Furthermore, a large set of measured data in biota in the field indicate, that HBCDD is biomagnified in the environment. No diastereomer specific BCFs are available. However, the concentration of α-HBCDD in biota is generally much higher than the concentration of the other two main diastereomers despite it being present in commercial HBCDD in a relatively low concentration. Several reasons may have lead to this difference in diastereomeric accumulation

Toxicity: HBCDD fulfils the T criterion. A 21d-NOEC of 3.1 μg l^{-1} has been derived for *Daphnia magna* in a flow-through test. It is noted, that ecotoxicity testing of HBCDD is highly complicated due to its very low water solubility.

Other: HBCDD has a high potential for long-range environmental transport. Its half-life in the atmosphere is > 2 days and it has been found in remote areas in abiotic samples (air, deposition, sediment) and biota (polar bears, bird eggs, seals) in the majority of samples of the last years. Additionally, a study comparing long-range transport potential of "existing" POPs and HBCDD with the help of tuna fish samples, found HBCDD to have a very high potential for long-range environmental transport.

8.2 Assessment of substances of an equivalent level of concern

8.3 Emission characterisation

8.4 Conclusion of PBT and vPvB or equivalent level of concern assessment

Hexabromocyclododecane (HBCDD) fulfils the vB-criterion based on experimental data (BCF=18100) and measured data from biota. With a NOEC of 3.1 μg/l for daphnia, the T-criterion

is also met. The available soil degradation simulation tests indicate that the half-life of HBCDD in aerobic soil is > 120 d and thus the P-criterion in soil is met. The experimental data regarding persistence in sediment are varying. According to some of the sediment degradation simulation studies available the P-criterion is met, whereas other studies substance indicates that the substance is degradable in certain experimental conditions. However, data from dated sediment cores gives support to HBCDD being persistent also in sediment. Furthermore, HBCDD has potential for long-range environmental transport based on environmental monitoring data and modelling. Overall it is concluded, that HBCDD is a PBT substance.

INFORMATION ON USE, EXPOSURE, ALTERNATIVES AND RISKS

1 INFORMATION ON EXPOSURE

This section is mainly based on information from the risk assessment performed under Council Regulation (EEC) 793/93. (Eu RAR, 2008)

Exposure

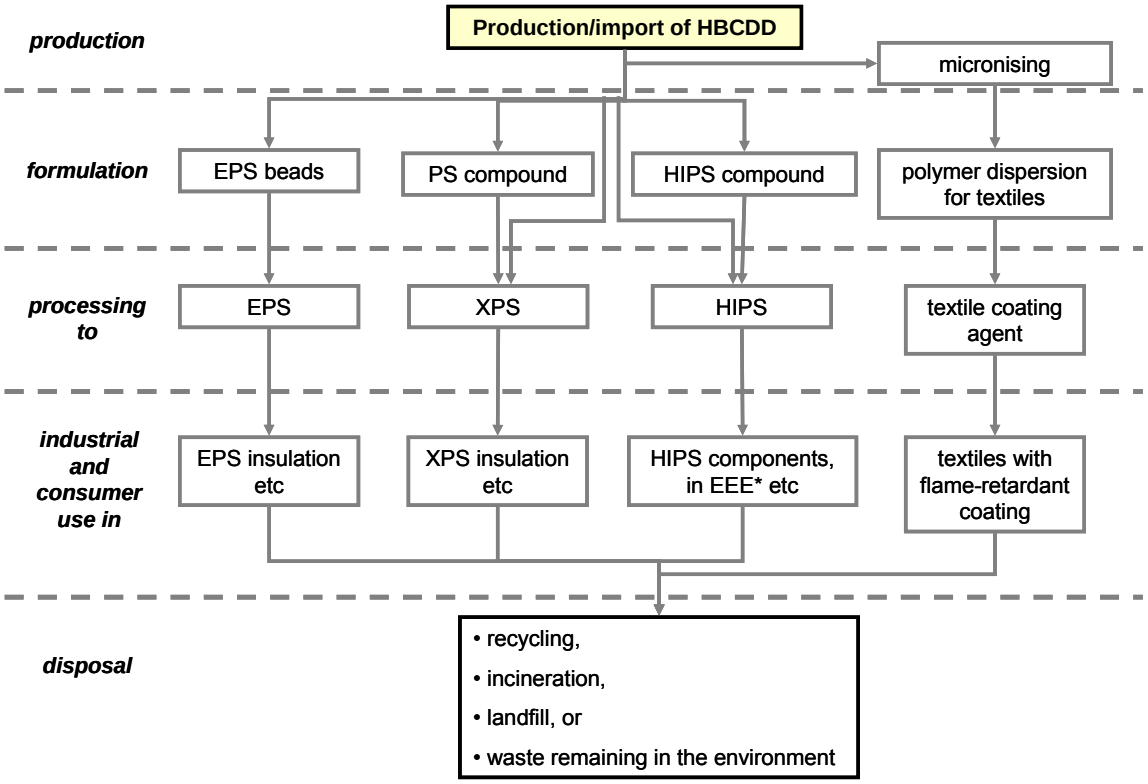
Due to the production and use of HBCDD humans may be exposed from different sources. Workers could be exposed to HBCDD at industrial sites during; production of HBCDD, use of HBCDD in formulation, industrial use of HBCDD as an additive and at industrial uses of articles containing HBCDD. The general population can be exposed via diffuse emissions from products containing HBCDD and indirectly via the environment via food, soil, water and air. HBCDD is present in breast-milk and blood of the general population. HBCDD is also present in the environment and in biota.

Production and use

Among those countries which constituted the European Union before 2004 (EU 15) HBCDD is presently only produced at one site. Two other production sites were closed for production in the autumn of 2003 and June 1997 respectively. HBCDD is imported to and probably exported from EU, both as a chemical (on its own or in formulations) and in articles. The total consumption of HBCDD in EU15 (1999) is estimated to nine to ten thousand tons. HBCDD is also extensively used in several of the countries that joined EU in 2004.

The main part (90 %) of HBCDD is used as flame retardant in polystyrene (PS). PS-containing HBCDD, in the form of Expanded PS (EPS) or Extruded PS (XPS), is mainly used as rigid thermal insulation panels/boards for buildings and for road and railway constructions to prevent frost heaves and provide a lightweight load-spreading construction material. HBCDD is also used to flame-retard textiles (for furniture, automobile interiors etc) and in smaller quantities in High Impact PS (HIPS). The latter polymer material is typically used in electronic and electrical equipment. Some other minor uses have been reported, but it is not clear whether they are relevant for EU. An overview of the life-cycle stages of HBCDD is shown in Figure 1. A summary of the number of sites and quantities relevant for the various stages is shown in Table 1.1.

Figure 1 Overview of life-cycle stages for HBCDD (micronising no longer takes place in EU)



* Electrical and Electronic Equipment

Use in EPS and XPS

Nearly all EPS containing HBCDD is used in building and construction industry, with smaller quantities used in (non-food) packaging. In Europe some 420 000 tonnes of EPS is used for construction applications, 170 000 of this is used in Eastern Europe. In Western Europe approximately 70 % of this EPS is flame-retarded grades, in Eastern Europe more than 99 %. Packaging uses some 250 000 tonnes of EPS in Western Europe of which approximately 10 % is flame-retarded grade.

XPS with HBCDD is used in construction industry as rigid insulation boards in constructions and in road and railway embankments to protect against frost damage and as thermal insulation. It is also used as insulation in sandwich constructions in vehicles such as caravans and lorries for cold or warm transport of goods.

Use in HIPS

HIPS containing HBCDD is used mainly in electronic and electrical equipment such as video and stereo equipment, distribution boxes in electrical lines, and refrigerator lining.

Use in textiles

HBCDD is formulated to polymer-based dispersions (e.g. acrylic or latex) in water. This dispersion is then applied to the textile. The dispersion is applied to the textile by back coating, either as a paste which is applied to the textile and a scratch knife defines the final thickness, or as a foam layer which is pressed on the textile through a rotating screen. The use of rotation screen is very limited.

Textiles flame-retarded with HBCDD are typically technical textile and furniture fabric. HBCDD has certain particular advantages when used on synthetic fibres although this does not exclude its

use on cotton. Typical end products are upholstered furniture, draperies, interior textiles and automobile interior textiles. Draperies would only be treated by back-coating in specific (institutional) end-uses, and then typically only when there are specific fabric-related reasons for using HBCDD. DecaBDE is largely used for back coating, as HBCDD is more expensive. HBCDD is used mainly where companies find that only HBCDD can provide the performance that is required.

Service-life and disposal

Waste containing HBCDD is generated at each life-cycle step. In some cases the waste material can be recycled into the process. Wasted end products are incinerated, put on landfill, left in the environment or recycled. Waste ending up in the municipal waste streams is likely to be incinerated or put on landfill. Construction material on or under ground may be left in the environment after use or be part of wasted construction material used as filling material. Recycling of EPS occurs in several European countries. Wasted EPS boards are ground and put back into the moulding process together with virgin EPS to form new boards. The percentage of HBCDD that is part of incinerated, landfill or recycled material is not known.

The service time for many products containing HBCDD is long, in some cases, for example roads and railways up to 100 years and for buildings typically 30 to 100 years. Thus the amount of HBCDD in the society is accumulating. It also means that much of the HBCDD produced today does not end up as waste until many years from now.

Table1-1 Summary of production and use, EU15 data from 2002 (EU RAR,2008)

	number of sites	quantity handled tons / year	typical HBCDD content in end-product	typical form of HBCDD (note 1)
Production of HBCDD in 2005	1	6 000	100 %	powder or granulate
Formulation of flame-retarded EPS beads	> 18	3 400	0.7 % (in EPS beads)	powder
Formulation of flame-retarded PS compound for HIPS	4	> 200	not available	powder
Formulation of flame-retarded PS compound for XPS	> 14	1 700	40 % (in compound)	powder, granulate
Formulation of polymer dispersion for textile back-coating	16	1 100 (assumption)	10 to 15 % (in the dispersion)	micronised
Industrial use of EPS beads to produce flame-retarded EPS	hundreds	3 400	0.7 % (in the EPS)	embedded in EPS
Industrial use of HBCDD in PS compound to produce flame-retarded HIPS	not available	> 200	1 to 3% (in the XPS)	powder or embedded in compound
Industrial use of HBCDD in PS compound to produce flame-retarded XPS	17	1 700	1 to 3% (in the XPS)	embedded in compound
Industrial use of HBCDD as powder to produce flame-retarded XPS	18	3 200	0.5 to 3 % (in the XPS)	powder, granules
Industrial use of HBCDD in polymer dispersion for textile back-coating	24	1 000	25 % or 6 to 15 % (in final layer) (note 2)	micronised, in a dispersion
Disposal	not known, widely spread	not known	varying	varying

Notes to table:

(1) micronised typically 3 to 4 μm, powder typically 50 to 250 μm; granulates typically > 500 μm
(2) the lower span is if used together with antimony trioxide, which is a synergistic flame-retardant

Information on use and exposure is included in the EU risk assessment of HBCDD (European Commission, 2008). Identification of potential risk reduction measures has been carried out by Swedish Chemicals Agency (2007).

2 INFORMATION ON ALTERNATIVES

2.1 Alternative substances

General

Some of the most commercially used alternatives are listed in the tables below. Further information and alternatives can be found in for example UBA 2001 and Danish EPA 1999. Available information on health and environmental hazards, including classification and labelling, of the suggested alternative substances are also included in the tables. Decabromodiphenyl ether, diantimony trioxide and the chlorinated paraffin C10-C13 are substances which have been evaluated under Council Regulation (EEC) 793/93 on the evaluation and control of existing substances and for which European Union Risk assessment reports are available. Zinc borate is converted to zinc oxide and boric acid which are substances also evaluated under that Regulation.

Summaries of available toxicological and eco-toxicological information for flame retardants have also been compiled by the Danish Environmental Protection Agency (Danish EPA, 1999 and 2000) the German Federal Environmental Agency (Umweltbundesamt; UBA 2001), and the UK Environment Agency (2003). However for most substances full information/data sets on health- and environmental hazards are not available. Additional data will become available during the registration of these substances under REACH.

It has not been possible to assess the availability of alternative substances to HBCDD in every specific application. What follows is a summary of alternatives used in the three main categories of use for HBCDD; the use in XPS/EPS, the use in textiles and the use in HIPS.

Use in XPS and EPS

No information suggesting the availability of suitable alternative substances has been found for the use of HBCDD in EPS and XPS.

Use in textiles

There are alternatives to HBCDD when transparency is not important (see Table 2-1). For transparent back coating on textiles there are no suitable alternative substances according to available information.

Table 2-1 Alternative substances in textile applications

Textiles			
Alternative substance	**Cas-No; EC-No**	**Health- and environmental hazards**[1]	**References**
Decabromodiphenyl ether	1163-19-5; 214-604-9	Not classified. Health: Conclusion (i). A developmental neurotoxicity	UK 2003; RAR 1

		study is required. Environment: Conclusion (i). A decision on the significance of bioaccumulation could not be made and the significance of degradation to substances that meet the PBT/vPvB criteria has not been established.	
Ammonium polyphosphate	68333-79-9; 269-789-9	Not classified.	Keml 2006; Danish EPA 2000
Ammonium phosphate	10124-31-9; 233-330-0	Not classified.	Keml 2006
Reactive phosphorous constituents	Several different CAS-/EC-No		Keml 2006
Red phosphorous	7723-14-0; 231-768-7	Red phosphorous is classified as R52/53 (harmful to aquatic organisms/may cause long-term adverse effects in the aquatic environment).	UBA 2001
Intumescent systems	Several, variable components		Keml 2006

[1] Classification according to Council Directive 67/548/EEC on the classification, packaging and labelling of dangerous substances.

Possible results of the risk assessment for existing substances:

Conclusion (i) There is a need for further information and/or testing.

Conclusion (ii) There is at present no need for further information and/or testing and no need for risk reduction measures beyond those which are being applied already.

Conclusion (iii) There is a need for limiting the risks; risk reduction measures which are already being applied shall be taken into account.

Use in HIPS

Table2-2 Alternative substances in HIPS

HIPS			
Alternative substance	**Cas-No; EC-No**	**Health- and environmental hazards**[1]	**References**
Triphenylphosphate	115-86-6; 204-112-2	Not classified. According to a risk assessment made by the US EPA: There is a moderate hazard concern for human health (non-cancer; based on systemic effects and eye irritation) and a high hazard concern for the aquatic environment. The substance is not PBT.	Keml 2006; Danish EPA 2000; US EPA 2005
Resorcinol bis (diphenyl phosphate) /Tetraphenyl m-phenylene bis(phosphate)	57583-54-7; 260-830-6	Not classified.	Keml 2006; Danish EPA No 17/2000
Bisphenol A bis (biphenyl phosphate)	5945-33-5; No EC-No available	Not classified.	Keml 2006
Polymeric biphenyl phosphate	No CAS-/EC-No available		Keml 2006

Cresol diphenyl phosphate	26444-49-5; 247-693-8	Not classified.	Keml 2006
Zinc borate	1332-07-6, 51201-70-8; 215-566-6	Zinc borate is converted to zinc oxide and boric acid. Zinc oxide (Cas No. 1314-13-2): Zinc oxide is classified as N; R50/53 (very toxic to aquatic organisms/may cause long-term adverse effects in the aquatic environment). Health: Conclusion (iii) for one worker scenario; systemic effects after repeated dermal exposure. Environment: Conclusion (iii) for some local scenarios. Boric acid (Cas No. 10043-35-3): Not classified. Health: The risk assessment report is not finalized but there is a proposal to classify borates with Repr. Cat. 2 and assign risk phrases R60-61 (may impair fertility, may cause harm to the unborn child). This is in line with the voting during the TPC[2]-meeting held in February 2007. Environment: According to the draft risk assessment report no environmental classification or labelling is required.	Danish EPA 2000; RAR 2-5
Decabromodiphenyl ether*	1163-19-5; 214-604-9	See Table 2-1	Keml 2006
Decabromodiphenylethane*	84852-53-9; 284-366-9	Not classified.	Keml 2006
Ethene bis(tetrabromophthalimide)*	32588-76-4; 251-118-6	Not classified.	Keml 2006
Brominated epoxy resins*	Several different CAS-/EC-No		Keml 2006
Chlorinated paraffins, C10-13*	85535-84-8; 287-476-5	Classified as Canc. Cat. 3; R40 N; R50-53 (limited evidence of carcinogenic effect; dangerous for the environment, very toxic to aquatic organisms, may cause long-term adverse effects in aquatic environment). Environment: Conclusion (i). Applies to the sediment and soil compartments for a few scenarios. Also conclusion (iii) applies to a few scenarios.	Keml 2006; RAR 6
* used together with Antimony trioxide	1309-64-4; 215-175-0	Antimony trioxide is classified as Canc. Cat. 3; R40 (limited evidence of carcinogenic effect). It is also proposed to be classified as Xi; R38 (irritating to skin). Health: Conclusion (iii). Applies to repeated dose toxicity (local pulmonary toxicity after inhalation) and carcinogenicity (pulmonary carcinogenicity) for most occupational exposure scenarios. It also applies to skin irritation for all scenarios to indicate the need for classification. Environment: Conclusion (iii). Reached only for a few scenarios modelled on default data.	Keml 2006; RAR 7

[1] Classification according to Council Directive 67/548/EEC on the classification, packaging and labelling of dangerous substances.

Possible results of the risk assessment for existing substances:

Conclusion (i) There is a need for further information and/or testing.

Conclusion (ii) There is at present no need for further information and/or testing and no need for risk reduction measures beyond those which are being applied already.

Conclusion (iii) There is a need for limiting the risks; risk reduction measures which are already being applied shall be taken into account.

[2] TPC "Technical Progress Committee" (representatives from all member states).

2.2 Alternative techniques

Use in XPS and EPS

The need for improved flame retardant properties of EPS and XPS is being deemed differently in different countries. In for example Sweden and Denmark most of the EPS and XPS used in the building sector do not contain any flame retardant. The functionality is instead reached by encasing the insulation with fire-proof materials or if this is not possible by using alternative insulation materials. (Brandforsk 2002) (KemI 2007)

Other materials than EPS and XPS are also used as insulation in buildings. The other major materials are PUR, polyurethane rigid foam (used with added flame-retardants) and mineral-based products (inherently non-flammable). The materials have different costs and technical properties, for example in insulation performance, mechanical properties, weight and sensitivity to humidity. Insulation materials are therefore not universally interchangeable, but rather serve specific applications segments, for which they provide substance specific advantages. The substitution of one for the other is either not possible, or only possible under certain circumstances. (UBA 2001) A comparison on the various materials potential effects on human health and the environment is not readily available.

Use in textiles

In the case of textiles alternative techniques include the redesign of products to reduce their fuel load, inherently fire-resistant fabrics and barrier layers.

Use in HIPS

Re-designing the products so that the voltage supply is separated from ignitable plastic will decrease the requirement for flame-retarding. If housings need to be protected from external ignition sources the demand for flame-retarding will remain.

3 RISK-RELATED INFORMATION

The risk assessment under Council regulation (EEC) 793/93 concluded that there was a need for limiting the risks for human health when HBCDD is handled in certain work-place activities.

The risk assessment also concluded that HBCDD has PBT properties according to the criteria of the Technical Guidance Document, TGD, and that there are also concerns for the environment near sites using HBCDD in various industrial processes and for sewage treatment plants receiving releases from certain industrial processes using HBCDD.

The amount of HBCDD in society is increasing due to the long service-life of many products containing HBCDD, for example thermal insulation in constructions.

The EU RAR (2008) estimates the known environmental emissions of HBCDD during production and use to 0.1 % of the produced and imported volume of HBCDD (8.7 tonnes out of a total volume of 8-9000 tonne), and there are concern for some environmental compartments from this emission. Thus, 99.9 % of the produced/imported volume ends up in articles, i.e., mainly in polystyrene (XPS, EPS) used in the construction and building sector where a very long service-life is assumed for these articles. The likely future emissions from these constructions (e.g., at repair or demolishing of old constructions/buildings) have not been assessed in the RAR. The RAR acknowledges that future emissions are very likely but that there is no methodology for assessing future emissions.

It is even possible that future emissions at the end of service-life will be higher than those we have seen in the production/formulation steps (unless more than 99.8 % of used polystyrene will be 'recycled' when repairing or demolishing old constructions). Thus, it is a risk that the current RAR severely underestimates the long-term risks with the use of HBCDD in articles with a long service-life.

OTHER INFORMATION

Extensive consultations with industry and member states experts took place during the risk assessment (up to spring 2008) and the preparation of a strategy to limit risks (April 2007 to April 2008) under regulation (EEC) 793/93, including written communications, bilateral meetings with representatives of industry producing and using HBCDD, and discussions in meetings (meetings of Technical Committee of New and Existing Substances, TCNES, and Risk Reduction Strategy Meetings RRSM). The results from these consultations have been incorporated in the Risk Assessment Report and the Strategy to Limit Risks.

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4. European Union Risk assessment report of Boric Acid and Disodium Tetraborates, anhydrous – Human health part, draft of November 2007. Rapporteur: Austria

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6. European Union Risk assessment report of Alkanes, C10-13, Chloro. 1st priority list, volume 4, 2000. Rapporteur: United Kingdom

7. European Union Risk assessment report of Diantimony trioxide, final version of May 2008. Rapporteur: Sweden

ANNEX

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