Substance name: Hexabromocyclododecane
and all major diastereoisomers identified
EC number: 247-148-4 and 221-695-9
CAS number: 25637-99-4 and 3194-55-6
Names of the major diastereoisomers identified:

- alpha-hexabromocyclododecane CAS No 134237-50-6
- beta-hexabromocyclododecane CAS No 134237-51-7
- gamma-hexabromocyclododecane CAS No 134237-52-8

MEMBER STATE COMMITTEE
SUPPORT DOCUMENT FOR IDENTIFICATION OF
HEXABROMOCYCLODODECANE AND ALL MAJOR
DIASTEREISOEROMERS IDENTIFIED
AS A SUBSTANCE OF VERY HIGH CONCERN

Adopted on 8 October 2008
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**Substance Name:** Hexabromocyclododecane and all major diastereoisomers identified

**EC Number:** 221-695-9, 247-148-4

**CAS number:** 3194-55-6, 25637-99-4

**Names of the major diastereoisomers identified:**

- alpha-hexabromocyclododecane  CAS No 134237-50-6
- beta-hexabromocyclododecane  CAS No 134237-51-7
- gamma-hexabromocyclododecane  CAS No 134237-52-8

The substance is identified as a PBT according to Article 57 (d) of Regulation (EC) No 1907/2006 (REACH).

**Summary of the evaluation:**

Hexabromocyclododecane (HBCDD) fulfills both the B and the vB-criteria 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 data show that the half-life of HBCDD in aerobic soil is > 120 d and thus the P-criterion in soil is met. In addition, degradation sediment simulation tests and dated sediment cores are available indicating slow degradation rates of HBCDD thus supporting the P criterion 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. **It is concluded that HBCDD is a PBT substance.**

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

The substance has not yet been registered.

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). The PBT-assessment builds on the PBT-fact sheet agreed by the TC NES PBT-subgroup. Information from those documents is used in this support document without giving references in this support document. Thus, the reader is referred to the RAR and the RRS. New information and new studies not used in the RAR and RRS are given as full references in this document.
1 JUSTIFICATION

IDENTITY OF THE SUBSTANCE AND PHYSICAL AND CHEMICAL PROPERTIES

1.1 Name and other identifiers of the substance

Chemical Name: Hexabromocyclododecane and 1,2,5,6,9,10-hexabromocyclododecane
EC Number: 247-148-4; 221-695-9\textsuperscript{a}; this number refers to hexabromocyclododecane (without specifying the bromine positions) and is used by some industry for the commercial substance.
CAS Number: 25637-99-4; 3194-55-6\textsuperscript{a}; this number refers to 1,2,5,6,9,10-hexabromocyclododecane and is thus the most correct one from a chemical point of view
IUPAC Name: Hexabromocyclododecane

\textsuperscript{a} The latter number is more specific in terms of the diastereomeric composition of the substance (1,2,5,6,9,10-HBCDD; see below). However, as the former numbers are used by industry (e.g., in SDS) for technical HBCDD, the dossier need to cover both numbers.

1.2 Composition of the substance

Chemical Name: Hexabromocyclododecane and 1,2,5,6,9,10-hexabromocyclododecane
EC Number: 247-148-4; 221-695-9\textsuperscript{a}
CAS Number: 25637-99-4\textsuperscript{b}; 3194-55-6\textsuperscript{a}
IUPAC Name: Hexabromocyclododecane
Molecular Formula: C\textsubscript{12}H\textsubscript{18}Br\textsubscript{6}
Structural Formula:

![structure formula for 1,2,5,6,9,10-HBCDD, i.e., CAS no 3194-55-6a]

Note that CAS no 25637-99-4 is also used for this substance, although not correct from a chemical point of view as this number is not specifying the positions of the bromine atoms.

As additional information, the structures and CAS numbers for the diastereomers making up 1,2,5,6,9,10-HBCDD is given below, although these diastereomers always occur as mixtures in the technical product.

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\textsuperscript{TM}; Great Lakes CD-75; Great Lakes CD75XF; Great
Concentration range (% w/w):

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); FR-1206; HBCD ILM; HBCD IHM

Depending on the producer, technical grade HBCDD consists of approximately 70-95 % \( \gamma \)-HBCDD and 3-30 % of \( \alpha \)- and \( \beta \)-HBCDD due to its production method (European Commission, 2007). Two additional diastereoisomers, \( \delta \)-HBCDD and \( \varepsilon \)-HBCDD have been found by Heeb et al. (2005) in commercial HBCDD in concentration of 0.5 % and 0.3 %, respectively. The only detailed information on composition available in the EU RAR (European Commission, 2007), concerns composites used for most testing purposes. The composites were prepared by mixing equal amounts of technical HBCDD obtained from the three manufacturers being on the EU market, generally giving composite compositions of approximately 80 % \( \gamma \)-HBCDD, 5-10 % of \( \alpha \)-HBCDD, 5-10 % of \( \beta \)-HBCDD. The amount of contaminants/unknown constituents varies (0-5 %) and one identified constituent is tetrabromocyclododecane. The composition is likely to differ between products from the different manufacturers, but also to differ between different products of a single manufacturer (e.g., HBCD-ILM (high-melting) and HBCD-IHM (low-melting)).

\( ^b \): This number refers to unspecific isomer composition.

\( ^c \): Historical names of the products of ICL-IP. Current names of ICL-IP products are: FR-1206, HBCD ILM and HBCD IHM

**Additional information on the three main constituents of technical hexabromocyclododecane**

**CAS Number:**

Technical HBCDD is made up of three main chiral diastereomers. Each of these have a specific CAS No, namely:

- (1R,2R,5R,6S,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]
- (1R,2R,5R,6S,9S,10R)-rel-1,2,5,6,9,10-hexabromocyclododecane [gamma-hexabromocyclododecane;CAS No 134237-52-8]

**Structural Formula:**

- alpha-HBCDD CAS No: 134237-50-6
- beta-HBCDD CAS No: 134237-51-7
1.3 Physico-chemical properties

Table 1-1: Summary of physico-chemical properties

<table>
<thead>
<tr>
<th>REACH ref</th>
<th>Property</th>
<th>Value</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>VII, 7.1</td>
<td>Physical state at 20°C and 101.3 Kpa</td>
<td>White odourless solid</td>
<td></td>
</tr>
<tr>
<td>VII, 7.2</td>
<td>Melting / freezing point</td>
<td>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</td>
<td>Smith et al. (2005)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>179-181 °C α-HBCDD</td>
<td>Smith et al. (2005)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>170-172 °C β-HBCDD</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>207-209 °C γ-HBCDD</td>
<td></td>
</tr>
<tr>
<td>VII, 7.3</td>
<td>Boiling point</td>
<td>Decomposes at &gt;190 °C</td>
<td>Peled et al. (1995)</td>
</tr>
<tr>
<td>VII, 7.5</td>
<td>Vapour pressure</td>
<td>6.3·10⁻⁵ Pa (21 °C)</td>
<td>Stenzel and Nixon (1997)</td>
</tr>
<tr>
<td>VII, 7.7</td>
<td>Water solubility</td>
<td>See Table 1.2</td>
<td></td>
</tr>
<tr>
<td>VII, 7.8</td>
<td>Partition coefficient n-octanol/water</td>
<td>5.625 (technical product)</td>
<td>MacGregor and Nixon (1997)</td>
</tr>
<tr>
<td></td>
<td>(log value)</td>
<td>5.07 ± 0.09 α-HBCDD</td>
<td>Hayward et al. (2006)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5.12 ± 0.09, β-HBCDD</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>5.47 ± 0.10 γ-HBCDD</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Dissociation constant</td>
<td>-</td>
<td></td>
</tr>
</tbody>
</table>

Table 1-2 Summary of the results of valid water solubility studies using generator column method, as evaluated by European Commission (2007)

<table>
<thead>
<tr>
<th>Test substance</th>
<th>Water</th>
<th>Water solubility (µg l⁻¹)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>α-HBCDD</td>
<td>Water</td>
<td>48.8±1.9</td>
<td>MacGregor and Nixon (2004)</td>
</tr>
<tr>
<td>β-HBCDD</td>
<td></td>
<td>14.7±0.5</td>
<td></td>
</tr>
<tr>
<td>γ-HBCDD</td>
<td></td>
<td>2.1±0.2</td>
<td>Desjardins et al. (2004)</td>
</tr>
<tr>
<td>HBCDD technical product, sum of above</td>
<td></td>
<td>65.6</td>
<td></td>
</tr>
<tr>
<td>α-HBCDD</td>
<td>Salt-water medium</td>
<td>34.3</td>
<td>Desjardins et al. (2004)</td>
</tr>
<tr>
<td>Compound</td>
<td>Value</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-----------------------------------------------</td>
<td>-----------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>β-HBCDD</td>
<td>10.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>γ-HBCDD</td>
<td>1.76</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HBCDD technical product, sum of above</td>
<td>46.3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>γ-HBCDD Water</td>
<td>3.4±2.3</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Source: Stenzel and Markley (1997)
2 classification and labelling

2.1 classification in annex i of directive 67/548/EEC

The substance is not included in Annex I of 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.

2.2 self classification(s)

Members of EBFRIP (European Brominated Flame Retardant Industry Panel) are implementing the N;R50/53 classification and labelling for their HBCDD products.
3 ENVIRONMENTAL FATE PROPERTIES

3.1 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\(\times\)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.

3.1.1 Biodegradation

3.1.1.1 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.

3.1.1.2 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 (European Commission, 2008).

Simulation tests, soil

In an aerobic soil-dissipation study according to OECD 307 (Davis et al., 2003b), \(\gamma\)-HBCDD disappeared with a half-life of approximately 63 days at 20°C from sandy loam soil amended with sewage sludge at a rate of 5 mg g\(^{-1}\) dry soil. This half life is equivalent to 119 days at 12°C (recalculated with EUSES 2.03 (equation: \(DT_{50 \text{ temp env}} = DT_{50 \text{ temp test}} \times e^{(0.08(\text{temp test-Tempenv})})\)). The temperature of 12°C is a default value used in current risk assessment e.g. EUSES to reflect the average environmental conditions in the EU. The nominal test concentration was 25 µg technical HBCDD kg\(^{-1}\) dw. The test substance used had the following composition: 5.8 % \(\alpha\)-HBCDD, 19.3% \(\beta\)-HBCDD and 74.9% \(\gamma\)-HBCDD. In abiotic soil samples almost no dissipation occurred during 119 days indicating that biotic mechanisms may be involved in the dissipation of \(\gamma\)-HBCDD from aerobic soil. However, no transformation products were detected and the fate of the \(\alpha\)- and \(\beta\)-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°C were observed. The nominal test concentration was 3.0 mg technical HBCDD kg\(^{-1}\) dw. The test material was a composite sample from three manufacturers with a composition of 8.7%, 6.1% and 85.2% of \(\alpha\)-, \(\beta\)- and \(\gamma\)-HBCDD, respectively. 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. (2003b) 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 test substance used had the following composition: 5.8% α-HBCDD, 19.3% β-HBCDD and 74.9% γ-HBCDD.

The concentration decreased to 7-10% of the day 0 concentration during the 119 days of incubation in the aerobic sediments and decreased below the detection limit of 0.5 µg/kg within 7 days in the anaerobic sediments. The disappearance of γ-HBCDD from the aquatic water/sediment systems resulted in approximate DT50-values at 20°C of 11 and 32 days under aerobic conditions in the two systems (two different sediments), respectively. These half-lives are equivalent to 21 and 61 days at 12°C. 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 ¹⁴C-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. The test material was a composite sample from three manufacturers with a composition of 8.7%, 6.1% and 85.2% of α-, β- and γ-HBCDD, respectively. There were no indications of an influence of HBCDD on the biological activity of the samples. The HBCDD concentration decreased to 56% of the day 0 concentration during 112 days of incubation in aerobic sediment and to 38% of the day 0 concentration in anaerobic sediments. The resulting half-lives recalculated to 12°C were 191 and 125 days in aerobic and anaerobic sediment, respectively. The recalculated half-life for α-HBCDD was approximately 210 days under both conditions. Table 3.1 provides an overview of the results.

**Table 3-1 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).**

<table>
<thead>
<tr>
<th>Medium/Standard</th>
<th>Sampling site</th>
<th>Degradation half-life of HBCDD in viable flasks at 20°C (value in parenthesis recalculated to 12 °C)</th>
<th>Degradation half-life of HBCDD in abiotic flasks at 20°C (value in parenthesis recalculated to 12 °C)</th>
</tr>
</thead>
</table>
| 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 |
The data for the diastereomers indicate that there seems to be both a difference related to the environment and also a difference between the diastereomers. α-HBCDD seems to biodegrade at a slower rate compared to β- and γ-HBCDD. α-HBCDD does not seem to be influenced by an anaerobic environment, whereas both β- and γ-HBCDD biodegrade faster in an anaerobic environment. 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 3.1). There were no indications of further transformation of 1,5,9-cyclododecatriene as no CO₂ or other volatiles were formed during the course of the study.

![Figure 3-1 Stepwise dehalogenation of HBCDD (Davis et al., 2004).](image)

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 3.2) 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.

### 3.1.2 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 half-life of 119 days was observed in Davis et al (2003b), but this value may underestimate the half-life as only disappearance of HBCDD was studied.

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 °C 214 d (α-HBCDD), 129 d (β-HBCDD) and 197 d (γ-HBCDD) and for anaerobic sediment 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, both with regard to the soil and sediment studies. 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 14C-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 14CO2) and other volatile 14C-degradation products were monitored and remained negligible in all tests. Although the difference is not statistically significant the results from Davis et al. (2004) indicate that α-HBCDD is 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, sediment core samples analysed indicate a slower disappearance of HBCDD in sediment than what would be expected based on the simulation...
studies. Furthermore, 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.

It is concluded, that HBCDD meets the P-criteria in soil and sediment, although it has been observed to degrade/disappear under certain experimental conditions.

### 3.2 Environmental distribution

#### 3.2.1 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.

#### 3.2.2 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.

#### 3.2.3 Distribution modelling

The EUSES modelling performed for the EU risk assessment of HBCDD (EU Commission, 2008) gave the following steady state mass fractions for HBCDD at the regional scale:

- Freshwater: 0.003%
- Sea water: 0.0003%
- Air: 0.00003%
- Agricultural soil: 45%
- Natural soil: 0.015%
- Industrial soil: 0.005%
- Freshwater sediment: 0.02%
- Sea water sediment: 0.0003%

The level III fugacity model Epiwin 3.20 calculates the following distribution of HBCDD assuming equal emissions to air, water and soil:

- Air: 0.03%
- Water: 8.1%
- Soil: 83%
- Sediment: 9.1%

**Long range transport**

HBCDD has a very slow atmospheric degradation rate (half-life > 2 days, see section 3.1.1), 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 3.2). In addition to data in Table 3.2, 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 EURAR, 2008 for references).

**Table 3-2 Measured environmental concentrations of HBCDD in remote Arctic areas (bird data excluded).**

<table>
<thead>
<tr>
<th>Species, sample type</th>
<th>Location; sampling year</th>
<th>Concentration</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Air</td>
<td>Ammamås, northern Sweden</td>
<td>5.7 pg HBCDD/m³ in particulate phase</td>
<td>Bergander et al. (1995)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.2 pg HBCDD/m³ in vapour phase</td>
<td></td>
</tr>
<tr>
<td>Pallas, Finland</td>
<td>0.003 ng HBCDD/m³ (autumn 2000), total conc.</td>
<td>Sternbeck et al. (2001)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.002 ng HBCDD/m³ (winter 2001), total conc.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deposition</td>
<td>Pallas, Finland</td>
<td>13 ng/m²·d, precipitation 21 mm (autumn 2000)</td>
<td>Sternbeck et al. (2001)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5.1 ng/m²·d, precipitation 4 mm (winter 2001)</td>
<td></td>
</tr>
<tr>
<td>Sediment</td>
<td>Ellasjøen, Bjørnøya, Svalbard, Norway</td>
<td>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 &lt; LOD.</td>
<td>Christensen et al. (2004)</td>
</tr>
<tr>
<td>Invertebrates</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gammarus wilkitzki</td>
<td>North Atlantic, Svalbard area, Norway; 2003</td>
<td>Not detected</td>
<td>Sørmo et al. (2006)</td>
</tr>
<tr>
<td>Fish</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Polar cod (Boreogadus saida); whole fish</td>
<td>Svalbard, Norway; 2003</td>
<td>1.73 µg HBCDD/kg lw (median); min-max: 1.38-2.87, n = 7</td>
<td>Sørmo et al. (2006)</td>
</tr>
<tr>
<td>Polar cod (Boreogadus saida); whole fish</td>
<td>Bjørnøya, Svalbard, Norway; 2003</td>
<td>11.7 ±7.2 µg HBCDD/kg lw (mean±SD), n = 6</td>
<td>Jenssen et al. (2007)</td>
</tr>
<tr>
<td>Mammals</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Polar bear (Ursus maritimus), adipose tissue (females)</td>
<td>Svalbard, Norway; 2002</td>
<td>26±9.0 µg HBCDD/kg ww (mean±SD), min-max: 9.7-45, n = 15</td>
<td>Gabrielsen et al. (2004)</td>
</tr>
<tr>
<td>Polar bear (Ursus maritimus), adipose tissue (males)</td>
<td>Svalbard, Norway; 2002-2003</td>
<td>12.6 µg HBCDD/ kg lw (median); min-max: 5.31-16.51, n = 4</td>
<td>Sørmo et al. (2006)</td>
</tr>
<tr>
<td>Harbor seal (Phoca vitulina), blubber</td>
<td>Svalbard, Norway; 2003</td>
<td>3.66±1.54 µg HBCDD/kg lw (mean±SD),n=5</td>
<td>Jenssen et al. (2007)</td>
</tr>
<tr>
<td>Ringed seal (Pusa hispida), blubber</td>
<td>Svalbard, Norway; 2003</td>
<td>16.96 µg HBCDD/kg lw (median); min-max: 14.6-34.5, n = 6</td>
<td>Sørmo et al. (2006)</td>
</tr>
</tbody>
</table>

Additionally, Ueno et al. (2006) have determined half-distances for HBCDD, polybrominated diphenyl ethers and “existing” POPs (see Table 3.3).
Table 3-3 Calculated half-distances for HBCDD, PBDEs and POPs in the North Pacific based on skipjack tuna monitoring (compiled in Ueno et al., 2006).

<table>
<thead>
<tr>
<th>Substance</th>
<th>Number of sites</th>
<th>Correlation coefficient ($r^2$)</th>
<th>Half-distance±SE (km)</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\alpha$-HCH</td>
<td>5</td>
<td>0.83</td>
<td>-1700±480</td>
</tr>
<tr>
<td>$\alpha$-HBCDD</td>
<td>4</td>
<td>0.45</td>
<td>8500 ±6700</td>
</tr>
<tr>
<td>$\gamma$-HBCDD</td>
<td>4</td>
<td>0.73</td>
<td>1600±680</td>
</tr>
<tr>
<td>BDE-99</td>
<td>5</td>
<td>0.87</td>
<td>1400±320</td>
</tr>
<tr>
<td>BDE-153</td>
<td>5</td>
<td>0.79</td>
<td>1200±380</td>
</tr>
<tr>
<td>2378-T4CDF</td>
<td>5</td>
<td>0.93</td>
<td>3200±530</td>
</tr>
<tr>
<td>23478-P5CDF</td>
<td>5</td>
<td>0.87</td>
<td>2100±470</td>
</tr>
<tr>
<td>$\sum$PCBs</td>
<td>5</td>
<td>0.77</td>
<td>1500±480</td>
</tr>
<tr>
<td>p,p'-DDT</td>
<td>5</td>
<td>0.91</td>
<td>950±170</td>
</tr>
</tbody>
</table>

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.

3.3 Bioaccumulation

3.3.1 Aquatic bioaccumulation

3.3.1.1 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 $\alpha$-, 5.12 for $\beta$- and 5.47 for $\gamma$-HBCDD. Using these log Kow values BCFwin (v 2.17) estimates BCF values of 4240, 1600, 1750 and 3250 for the technical product and the $\alpha$-, $\beta$-, and $\gamma$-diastereomer, respectively.

3.3.1.2 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 °C. The steady-state BCF was calculated to be 18100.
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 °C. 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 5000. There is some difference between the BCF for low and high exposure groups, but overall they are in agreement with the value obtained in the Veith et al (1979) study.

Law et al. (2006a) exposed juvenile rainbow trout (*Oncorhynchus mykiss*) via diet to α-, β- 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-, β- 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 β-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.

### 3.3.2 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, 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 3-4).

### Table 3-4 Concentration of HBCDD in soil and earthworm tissue after 28 day of exposure and corresponding bioaccumulation factors (BAF) at different levels of exposure.

<table>
<thead>
<tr>
<th>Mean measured concentration of HBCDD in soil day 28 (mg/kg dwt)</th>
<th>Mean measured concentration of HBCDD in soil day 28 (mg/kg wwt)</th>
<th>HBCDD in worm tissue day 2 (mg/kg wwt)</th>
<th>BAF (wwt/wwt)</th>
</tr>
</thead>
<tbody>
<tr>
<td>61</td>
<td>54</td>
<td>3.4</td>
<td>0.06</td>
</tr>
</tbody>
</table>
In Table 3-5 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 α-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 (0.3-0.8) than for γ-HBCDD (0.005-0.02). 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 3-5 Concentration of α-, β- and γ- HBCDD in soil and earthworm tissue after 28 days of exposure, and diastereomer specific bioaccumulation factors (BAF) at different levels of exposure.

<table>
<thead>
<tr>
<th>Mean measured concentration of α, β, γ-HBCDD in soil day 28 (mg/kg dwt)</th>
<th>Concentration of α, β, γ-HBCDD in worm tissue day 28 (mg/kg wwt)</th>
<th>Diastereomer specific BAF. (dwt/wwt)</th>
</tr>
</thead>
<tbody>
<tr>
<td>α</td>
<td>β</td>
<td>γ</td>
</tr>
<tr>
<td>3.55</td>
<td>11.8</td>
<td>45.8</td>
</tr>
<tr>
<td>14.2</td>
<td>47.1</td>
<td>183</td>
</tr>
<tr>
<td>66.7</td>
<td>222</td>
<td>861</td>
</tr>
<tr>
<td>126</td>
<td>421</td>
<td>1633</td>
</tr>
<tr>
<td>243</td>
<td>809</td>
<td>3138</td>
</tr>
</tbody>
</table>

3.3.3 Others

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 3-6 provides an overview of the measured concentrations in freshwater fish muscle in Europe.

**Table 3-6 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).**

<table>
<thead>
<tr>
<th></th>
<th>Conc.</th>
<th>n</th>
<th>Median</th>
<th>Geometric mean</th>
<th>Arithmetic mean ± SD</th>
<th>90P</th>
<th>Min</th>
<th>Max</th>
</tr>
</thead>
<tbody>
<tr>
<td>All values</td>
<td>µg HBCDD kg⁻¹ ww¹</td>
<td>151</td>
<td>5.5</td>
<td>4.64</td>
<td>321 ± 1130</td>
<td>834</td>
<td>0.005</td>
<td>9432</td>
</tr>
<tr>
<td>µg HBCDD kg⁻¹ lw²</td>
<td>151</td>
<td>120</td>
<td>171</td>
<td>5223 ± 18745</td>
<td>7927</td>
<td>0.52</td>
<td>160905</td>
<td></td>
</tr>
</tbody>
</table>

¹: ww = wet weight  
²: lw = lipid weight

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

Table 3-7 provides an overview of measured concentrations of HBCDD in fish and marine mammals in Europe.

**Table 3-7 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).**

<table>
<thead>
<tr>
<th>Region</th>
<th>Species</th>
<th>n</th>
<th>Median concentration</th>
<th>Concentration ratios (marine mammals/fish muscle)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>ww/lwm</td>
</tr>
<tr>
<td>Western Europe</td>
<td>Fish</td>
<td>102</td>
<td>0.40 µg HBCDD kg⁻¹ ww</td>
<td>272</td>
</tr>
<tr>
<td></td>
<td></td>
<td>100</td>
<td>13 µg HBCDD kg⁻¹ lw</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Marine mammals</td>
<td>225</td>
<td>109 µg HBCDD kg⁻¹ ww</td>
<td>28</td>
</tr>
<tr>
<td></td>
<td></td>
<td>225</td>
<td>368 µg HBCDD kg⁻¹ lw</td>
<td></td>
</tr>
<tr>
<td>Baltic Sea</td>
<td>Fish</td>
<td>42</td>
<td>0.31 µg HBCDD kg⁻¹ ww</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>38</td>
<td>11.5 µg HBCDD kg⁻¹ lw</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Marine mammals</td>
<td>2 (representing 20 + 30 individuals)</td>
<td>19 µg HBCDD kg⁻¹ ww</td>
<td>61</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>2</td>
<td>67 µg HBCDD kg⁻¹ lw</td>
<td>5.8</td>
</tr>
<tr>
<td>Western Scheldt</td>
<td>Fish</td>
<td>18</td>
<td>1.8 µg HBCDD kg⁻¹ ww</td>
<td>187</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
The concentration ratios presented above may overestimate the “true” whole body weight ratios since the fish species used mainly store their lipids in the liver, and the concentrations used represent muscle concentrations which are lower. 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. The increase was especially pronounced between 2000 and 2003.

Law et al are in the process of publishing a follow up study looking at HBCDD levels in the blubber of porpoises collected all around the UK coast during 2003-2006. These yet unpublished data, indicate a sharp decrease in concentrations in blubber from 2003 to 2004 (median conc. dropped nearly 4 times) followed by a much slower decline (if any) from 2004 to 2006, the concentration of HBCDD in blubber 2006 being 6 times higher than the mean concentration for the years 1994-2000. The apparent rapid increase from 2000-2003 and almost equally rapid decrease from 2004 seems hard to explain. However, the large variation in concentration within each year (up to six orders of magnitude) is a reason not to over interpret the data by making trends out of 1, 2 or 3 years of observations.

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 3.2).
Figure 3-2 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 3.3.
Brominated contaminants in Guillemot egg, ng/g lipid w.

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). Janák et al. (2005b) observed that α-HBCDD was least bio-transformed of the main three diastereomers tested in microsomal liver preparations of common dab (Limanda limanda). 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).

Since HBCDD is a rather persistent and bioaccumulating substance emitted from both point sources and diffuse sources, exposure to man via food is a relevant route of exposure.
Based on studies on food-samples bought in food-stores in Sweden, representing fish, meat, chicken, milk, and egg a maximum intake of 22 ng HBCDD/kg/day was calculated in the EU RAR. The medium value was 10-fold lower (European Commission, 2008).

The mean dietary intake of a number of brominated flame retardants by the Dutch population was estimated using analytical and consumption data from different surveys conducted in the Netherlands. The concentration of HBCDD was determined in 91 samples from the food categories dairy, meat, animal fat, eggs, fish and vegetable oil. HBCDD was present in 15 out of 18 food categories. The percentage of non-detects was high; HBCDD could not be detected in 54 % of the samples. The total average dietary intake of HBCDD by the Dutch population was 2.9 ng/kg bwt/day.

HBCDD levels in blood have been detected in a number of surveys. Plasma from 10 pregnant women living in Bodø, Norway and from 10 women living in Taimyr, Russia were collected 2002 and analysed by LC-MS. HBCDD was detected in more than half of the samples but at low concentrations, close to the limit of detection. The Norwegian samples median and range values were (pg/ml plasma): α-HBCDD 19 (<11-345), β-HBCDD 7 (5-343), γ-HBCDD 23 (7-317) and the Russian samples median and range values were: α-HBCDD 21(<11-51), β-HBCDD 8 (<5-126), γ-HBCDD 33 (13-160).

HBCDD has, in a number of studies, been detected also in breast milk at various concentrations. One study with the objective to assess the temporal trends of polybrominated diphenyl ethers and HBCDD in mothers’ milk from the Stockholm area shows an increase of HBCDD in mothers’ milk over time. From 1980 the average concentrations of HBCDD in mothers’ milk has increased from 0.13 pmol/g (0.084 ng/g) to 0.60 pmol/g (0.39 ng/g) lipid in 2004. The highest values were found in 2001 and in 2002 (0.83 and 0.93 pmol/g). During the last 10 years the concentrations have varied between 0.6 and 0.93 pmol/g lipid. In 1986, 1993 and 2001, Norwegian breast milk samples were obtained from 10-12 primiparous mothers living in a coastal area in the North (Tromsø), in a rural inland area (Hamar), and in an industrialized area in the South Norway (Skin/Porsgrunn). HBCDD was found in all samples, but at very varying levels, range 0.25-2 ng/g lipids. HBCDD concentrations in blood and breast milk are more thoroughly discussed in the EU Risk assessment Report (European Commission, 2008). The risk assessment report concludes that there is at present no concern for repeated dose toxicity as well as no concern for reproductive toxicity for breast feeding infants.

### 3.3.4 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 (European Commission, 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.

### 3.4 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.
4 HUMAN HEALTH HAZARD ASSESSMENT

4.1 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 $\alpha$-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 $\gamma$-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. This refers to an estimated half-life after steady state was reached in a 90-day oral gavage study. Data on absorption by inhalation exposure is lacking. Therefore, as a worst case assumption, the efficiency of inhalation uptake applied in the risk assessment is 100%. For dermal absorption the EU risk assessment concluded 4% for fine particles (powder) and 2% for granular particles.

4.2 Acute toxicity

4.2.1 Acute toxicity: oral

The minimum lethal dose, in rats, is greater than 20 g/kg

4.2.2 Acute toxicity: inhalation

The minimum lethal dose, in rats, is greater than 200 mg/l

4.2.3 Acute toxicity: dermal

The minimum lethal dose, in rabbits, is greater than 20 g/kg

4.2.4 Summary and discussion of acute toxicity.

The data available on acute toxicity show a very low acute toxicity and do not suggest a classification of HBCDD according to EU criteria.
4.3 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.

4.4 Corrosivity

The substance is not corrosive to skin.

4.5 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.

4.6 Repeated dose toxicity

4.6.1 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 of action for the thyroid effects is thoroughly discussed in the EU RAR. It is plausible to assume that the thyroid effects are caused indirectly by liver enzyme induction, although some uncertainty remains regarding the mechanism of action.
Table 4-1 Summary of findings related to the liver and the thyroid system in the RdT studies.

**Studies on undissolved HBCDD (particles in suspension)**

<table>
<thead>
<tr>
<th>Study</th>
<th>Liver effects</th>
<th>Thyroid effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>28-days (Zeller and Kirsch 1969)</td>
<td>Liver weight increase as from the lowest dose (940 mg/kg/day) in both sexes</td>
<td>Thyroid hyperplasia as from the lowest dose (940 mg/kg/day) in both sexes</td>
</tr>
<tr>
<td>90-days (Zeller and Kirsch 1970)</td>
<td>Liver weight increases as from the lowest dose (120 mg/kg/day) in both sexes.</td>
<td>Histopathology of the thyroid was performed and revealed no significant lesions.</td>
</tr>
<tr>
<td>28-days (Chengelis 1997)</td>
<td>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).</td>
<td>No histological effects were observed in the thyroids in either sex.</td>
</tr>
<tr>
<td>90-days (Chengelis 2001)</td>
<td>Liver weight increase as from the lowest dose (100 mg/kg/day) in both sexes.</td>
<td>Thyroid weight was increased from mid dose in females (300 mg/kg/day), but not in males. Minimal follicular hypertrophy of the thyroid was observed in mid dose females and mild follicular hypertrophy in the thyroid of the high dose group males and females. Serum T4 was decreased and TSH increased in all dose groups of both sexes.</td>
</tr>
</tbody>
</table>

**Studies on dissolved HBCDD**

(using a Benchmark method)

<table>
<thead>
<tr>
<th>Study</th>
<th>Liver effects</th>
<th>Thyroid effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>28-days (van der Ven et al., 2006)</td>
<td>Liver weight increase only in females; BMD-L 23 mg/kg/day</td>
<td>Thyroid weight effects only in females.</td>
</tr>
<tr>
<td></td>
<td>BMD-L (mg/kg/day) for; hepatic T4-conjugation</td>
<td>BMD-L for weight increase</td>
</tr>
<tr>
<td></td>
<td>- females 4</td>
<td>2 mg/kg/day</td>
</tr>
<tr>
<td></td>
<td>- males 0.1 (uncertain)</td>
<td>BMD-L for decreased serum T4 55 mg/kg/day</td>
</tr>
<tr>
<td></td>
<td>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.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hepatic CYP3A4-induktion (LBD) was only observed in females (as from 10 mg/kg/day).</td>
<td></td>
</tr>
</tbody>
</table>

¹: In EU RAR the thyroid weight increase and the suggested BMDL was evaluated and, due to the high variability of the data, a clear increase was only observed in female animals at doses of 30 mg/kg and above. It was concluded that the BMDL for liver (22.9 mg/kg/day) is the most robust effect level and will most likely also cover the effects on the thyroid. Furthermore, assuming that hepatic enzyme induction is one factor contributing to the effects on the thyroid, it does not make sense with a BMD-L
for the thyroid effect being lower than that for enzyme induction (4.1 mg/kg/day for T4-UDT in females).

4.6.2 Repeated dose toxicity: inhalation

No data are available

4.6.3 Repeated dose toxicity: dermal

No data are available

4.6.4 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.

4.7 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.

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

4.8 Carcinogenicity

4.8.1 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.

4.8.2 Carcinogenicity: inhalation

No data are available

4.8.3 Carcinogenicity: dermal

No data are available
4.8.4 Carcinogenicity: human data

No data are available

4.8.5 Summary and discussion of carcinogenicity

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

4.9 Toxicity for reproduction

There are studies available in the Risk Assessment Report (European Commission, 2008) and these will be discussed by the Risk Assessment Committee for classification purpose as an Annex XV dossier for Harmonised Classification and labelling has been submitted.
## 5 ENVIRONMENTAL HAZARD ASSESSMENT

The results of ecotoxicity tests, which have been considered reliable by EU RAR, 2008, are presented in Table 5.1.

### Table 5-1 Acute and chronic ecotoxicity data, which are considered reliable according to EU RAR (2008)

<table>
<thead>
<tr>
<th>Compartment/Species</th>
<th>Method</th>
<th>Results</th>
<th>Remark and reference</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>AQUATIC COMPARTMENT</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>FISH</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Onchorhyncus mykiss</em></td>
<td>OECD 203 and TSCA 40/797/1400, and ASTM Standard E729-88a</td>
<td>No mortalities or other effects around 2.5 µg/l.</td>
<td>Graves and Swigert (1997b)</td>
</tr>
<tr>
<td><em>Onchorhyncus mykiss</em></td>
<td>Flow-through OECD 210 and OPPTS 850.1400</td>
<td>NOEC: Hatching success ≥3.7 µg/l&lt;br&gt;Swim-up ≥3.7 µg/l&lt;br&gt;Larvae and fry survival ≥3.7 µg/l&lt;br&gt;Growth ≥3.7 µg/l</td>
<td>Drottar et al. (2001)</td>
</tr>
<tr>
<td><strong>INVERTEBRATES</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Daphnia magna</em></td>
<td>OECD 202. Static immobilisation test, and TSCA 40/797/1300, and ASTM Standard E729-88a</td>
<td>48 h EC₅₀ ≥3.2 µg/l</td>
<td>Graves and Swigert (1997a)</td>
</tr>
<tr>
<td><em>Daphnia magna</em></td>
<td>TSCA 40/797/1330, OECD 202 Flow through 21 day test.</td>
<td>NOEC 3.1 µg/l&lt;br&gt;LOEC length 5.6 µg/l</td>
<td>Drottar and Krueger (1998)</td>
</tr>
<tr>
<td><strong>ALGAE</strong></td>
<td></td>
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<tr>
<td><em>Selenastrum capricornutum</em></td>
<td>OECD 201 and TSCA40/797/1050</td>
<td>96 h EC₅₀ &gt;2.5 µg/l</td>
<td>Roberts and Swigert (1997)</td>
</tr>
<tr>
<td><em>Skeletonema costatum</em></td>
<td>Marine algal bioassay method, different marine growth media</td>
<td>72 h EC₅₀ = 9 µg/l (lowest value)</td>
<td>Walsh et al. (1997) Not according to guidelines, results only used as supportive</td>
</tr>
<tr>
<td><em>Chlorella</em> sp.</td>
<td></td>
<td>72 h EC₅₀ = 40 µg/l (lowest value)</td>
<td></td>
</tr>
<tr>
<td><em>Skeletonema</em> costatum</td>
<td>OECD 201. EC₅₀ obtained from a limit test with one test concentration (54.5 µg/l) at the limit of respective water solubilities of each diastereomer.</td>
<td>NOEC &gt;10 µg/l&lt;br&gt;EC₅₀ = 52 µg/l</td>
<td>Desjardins et al. (2005)</td>
</tr>
<tr>
<td><strong>SEWAGE TREATMENT PLANT, MICRO-ORGANISMS</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Activated sludge</td>
<td>Respiration inhibition OECD 209</td>
<td>EC₅₀ = 15 mg/l</td>
<td>Limit test with one test concentration, EC₅₀ is an estimated value. Schaefer and Siddiqui (2003)</td>
</tr>
<tr>
<td><strong>SEDIMENT COMPARTMENT</strong></td>
<td></td>
<td></td>
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<tr>
<td><strong>INVERTEBRATES</strong></td>
<td></td>
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<td></td>
</tr>
<tr>
<td><em>Hyalithea azteca</em> (Amphipod)</td>
<td>Sediment toxicity test 28-day exposure period under flow-through conditions. ASTM E 1706-89b, OPPTS 850.1735</td>
<td>LOEC &gt;1000 mg/kg dw of sediment&lt;br&gt;NOEC 1000 mg/kg dw of sediment.</td>
<td>Thomas et al. (2003b)</td>
</tr>
<tr>
<td><em>Lumbriculus variegatus</em> (Worm)</td>
<td>28-day sediment bioassay</td>
<td>LOEC = 28.7 mg/kg dw&lt;br&gt;NOEC = 3.1 mg/kg dw&lt;br&gt;Normalized: NOEC = 8.61 mg/kg dw</td>
<td>Oetken et al. (2001)</td>
</tr>
<tr>
<td><em>Chironomus riparius</em> (Mosquito)</td>
<td>28-day sediment bioassay Egg production of F generation</td>
<td>LOEC = 159 mg/kg dw&lt;br&gt;NOEC = 13.6 mg/kg dw&lt;br&gt;Normalized: NOEC = 37.8 mg/kg dw</td>
<td>Oetken et al. (2001)</td>
</tr>
<tr>
<td><strong>TERRESTRIAL COMPARTMENT</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
### Compartment/Species | Method | Results | Remark and reference
--- | --- | --- | ---
**PLANTS**
Plants: corn (Zea mays), cucumber (Cucumis sativa), onion (Allium cepa), ryegrass, (Lolium perenne), soybean (Glycine max), and tomato (Lycopersicon esculentum)  
Seedling emergence, survival, height 21 days  
OECD 308 (proposal for revision), OPPTS 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 (EC50 771 mg/kg dry soil)  
Aulderheide et al. (2003)

**MICROORGANISMS**
Soil microorganisms  
Nitrate production  
NOEC≥ 750 mg  
Fürster (2007)

### 5.1 Aquatic compartment (including sediment)

#### 5.1.1 Toxicity test results

Studies with low reliability and/or test concentrations well above the water solubility are not described in this chapter.

##### 5.1.1.1 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 test substance consisted of a composite of HBCDD samples from three manufacturers. The composite contained 6.0 % α- diastereomer, 8.5 % β- diastereomer, and 79.1 % γ- diastereomer. 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 purity of HBCDD was 100 %, assumed to be technical product, with the following composition: α- diastereomer 9.4 %, β- diastereomer 6.3 %, and γ- diastereomer 84.3 %. 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 $\geq 83\%$ 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 based on measured concentration was $\geq 3.7\, \mu g/l$.

### 5.1.1.2 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 test substance consisted of a composite of HBCDD samples from three manufacturers. The
composite contained 8.5 % $\beta$-diastereomer, 6.0 % $\alpha$-diastereomer and 79.1 % $\gamma$-diastereomer (total
HBCDD 93.6 %). The nominal HBCDD concentrations were: 1.5, 2.2, 3.2, 4.6, and 6.8 µg/l,
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 test substance consisted of a composite of HBCDD samples from three manufacturers. The
composite contained 8.5 % $\beta$-diastereomer, 6.0 % $\alpha$-diastereomer and 79.1 % $\gamma$-diastereomer (total
HBCDD 93.6 %). 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 a measured concentration of 3.1 µg/l, and hence, the NOEC was 3.1 µg/l.

### 5.1.1.3 Algae and aquatic plants

Data are available from four reliable algal growth inhibition studies.
Study 1

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 test substance consisted of a composite of HBCDD samples from three manufacturers. The composite contained 6.0 % α-diastereomer, 8.5 % β-diastereomer, and 79.1 % γ-diastereomer (total HBCDD 93.6 %). 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 measured test concentration. Thus, the 72-hour EC\textsubscript{50} is >2.5 µg/l and the LOEC is >2.5 µg/l.

Study 2

The algal growth inhibition of HBCDD was also studied in six marine media (Walsh et al., 1987). The test substance HBCDD with unknown diastereomeric composition was obtained from one manufacturer, Great Lakes Chemical Inc. The studied test organisms were *Skeletonema costatum*, *Thalassiosira pseudonana* and *Chlorella sp.* Population density was estimated by cell counts on a haemocytometer. Toxicity, EC\textsubscript{50}, was based upon cell numbers after incubation for 72 hr for *S. costatum* and *T. pseudonana* and for 96 h for *C. sp*.

EC\textsubscript{50}s:

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

\textsuperscript{*} 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.

Study 3

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\textsubscript{50} >40.6 µg HBCDD/l.

Study 4

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. The test substance HBCDD used in the test was a 1:1:1 composite of three samples received from three different manufacturers.

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 nominal 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\textit{50}. The 72-hr EC\textit{50} for biomass and growth rate was calculated to be 27 and 52 µg/l respectively. The relevance of calculating an EC\textit{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\textit{50}-value of 52 µg/l seems adequate. Furthermore, this EC\textit{50}-value is in line with the result obtained with the saturated solution where EC\textit{10} was around 40.6 µg/l (Desjardins \textit{et al.}, 2004).

**Summary of algal toxicity**

Based on the most reliable algal toxicity study (Desjardins \textit{et al.}, 2005) the EC\textit{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 (European Commission, 2008).

5.1.1.4 Sediment organisms

Two toxicity tests have been performed on the amphipod \textit{Hyalella azteca} to determine the effects of sediment incorporated HBCDD during 28-day period under flow through conditions. (Thomas \textit{et al.}, 2003a-b). Spiked sediment with 2% and 5% total organic carbon content were used. Range-finding studies were performed with 3 freshwater species associated with sediment. Hyalella was found to be the most sensitive species and therefore used in a definitive study with 2 sediment types. 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). The nominal test concentrations were: 0.05; 0.5; 5; 50 and 500 mg HBCDD/kg dwt for both test organisms. 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*.

5.1.1.5 Other aquatic organisms

5.2 Terrestrial compartment

5.2.1 Toxicity test results

5.2.1.1 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 test substance consisted of a composite of HBCDD samples from three manufacturers. The composite contained 5.8 % α-diastereomer, 19.3 % β-diastereomer, and 74.9 % γ-diastereomer. The nominal test concentrations were 78.5, 157, 313, 625, 1250, 2500 and 5000 mg HBCDD/kg dry soil.

The NOEC for survival was estimated to 4190 mg HBCDD/kg dry soil. The NOEC for reproduction 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:

\[ \text{NOEC}_{\text{standard}} = \text{NOEC}_{\text{exp}} \times \left( \frac{\text{Fom}_{\text{soil(standard)}}}{\text{Fom}_{\text{soil(exp)}}} \right) \]

where Fom is fraction of organic matter.
The normalized NOEC is 59 mg/kg dry soil.

5.2.1.2 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). The test substance consisted of a composite of HBCDD samples from three manufacturers. The composite contained 5.8 % α-diastereomer, 19.3 % β-diastereomer, and 74.9 % γ-diastereomer. The nominal test concentrations were 40, 105, 276, 725, 1904 and 5000 mg HBCDD/kg dry soil.

The NOEC was > 5000 mg HBCDD/kg dry soil for all species. 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.

5.2.1.3 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≤0.05).

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

5.3 Atmospheric compartment

There are no effect data available for the atmospheric environment. The major part of HBCDD emitted to and/or measured in the air, is in particulate form. Due to the low vapour pressure and the stability of HBCDD, it is not considered to present a risk of adding to ozone depletion in the stratosphere, global warming or acidification.

5.4 Microbiological activity in sewage treatment systems

5.4.1 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 O₂/l/hour, which was equivalent to approximately 29.1 % inhibition when compared to the controls. Thus only an approximate EC₃₀ 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 EC₅₀ 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.

5.5 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:

<table>
<thead>
<tr>
<th>Concentration limits of substance</th>
<th>Classification of preparation</th>
</tr>
</thead>
<tbody>
<tr>
<td>C ≥2.5 %</td>
<td>N; R50-53</td>
</tr>
<tr>
<td>C ≥0.25 %</td>
<td>N; R51-53</td>
</tr>
<tr>
<td>C ≥0.025 %</td>
<td>R52-53</td>
</tr>
</tbody>
</table>

The proposal is based on the toxic effects seen in a 72-hour study on the marine algae Skeletonema costatum (EC₅₀ 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 EC₅₀ 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 EC₅₀ >40.6 µg/l is obtained for HBCDD.
6 PBT, VPVB AND EQUIVALENT LEVEL OF CONCERN ASSESSMENT

6.1 Comparison with criteria from annex XIII

**Persistence:** There are two degradation simulation studies in soil. In the first one the half-life for the \( \gamma \)-HBCDD diastereomer was of 119 days when recalculated to 12°C. In the other study no transformation was observed after 112 days of incubation. Based on the two studies Hexabromocyclododecane (HBCDD) fulfils the P-criterion in soil.

In addition, there are two degradation sediment simulation tests available. For \( \alpha \)-HBCDD, which seems to be the least degradable diastereomer, an aerobic DT\(_{50}\) of approximately 210 days in sediment recalculated at 12°C was determined, which is above the P-criterion of 120 days in sediment. For \( \gamma \)-HBCDD the available studies indicate very different half-lives. In the first study, using very low concentrations of \( \gamma \)-HBCDD, the parent compound disappeared with a half-life of 21 and 61 days (recalculated to 12°C) in two different sediments and in the second study, where a concentration similar to what is measured close to polluted areas was tested, the DT\(_{50}\) for \( \gamma \)-HBCDD was 197 days (recalculated to 12°C) in aerobic sediment.

The measured data available from dated sediment cores indicate slow degradation rates of HBCDD and support the results of the second study. It is therefore considered that the P-criterion is also fulfilled 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. 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 \( \alpha \)-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.

**Toxicity:** HBCDD fulfils the T criterion. A 21d-NOEC of 3.1 \( \mu \)g 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 \( \geq 2 \) 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.

6.2 PBT/vPvB Assessment/Assessment of substances of an equivalent level of concern

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

Hexabromocyclododecane (HBCDD) fulfils both the B and vB-criteria based on experimental data (BCF=18100) and measured data from biota. With a NOEC of 3.1 \( \mu \)g/l for *Daphnia*, the T-criterion
is also met. The available soil degradation simulation data show that the half-life of HBCDD in aerobic soil is > 120 d and thus the P-criterion in soil is met. In addition, degradation sediment simulation tests and dated sediment cores are available indicating slow degradation rates of HBCDD thus supporting the P criterion 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. It is concluded that HBCDD is a PBT substance.
REFERENCES

Main sources:

The information and references used in sections 1 to 8 and section 1 of chapter 2 of this report were mainly taken from the following source:


The information and references used in section 2 of chapter 2 of this report were mainly taken from the following source:

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